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Neural mechanisms of reward and loss processing in a low-income sample of at-risk adolescents

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Abstract

Adolescence is a time of engagement in risky, reward-driven behaviors, with concurrent developmental changes within reward-related neural systems. As previous research has recruited mostly higher socioeconomic, European and European American participants, therefore limiting generalizability to the US population, especially for populations of color or low-income populations. The current study provided one of the first opportunities to examine the neural correlates of reward and loss functioning in a population-based sample of adolescents at increased risk for poverty-related adversities. The study investigated neural reward and loss processing and whether age, pubertal status and the social constructs of gender and race predicted individual differences in reward- and loss-related brain function. One hundred and twenty-eight primarily low-income adolescents (mean age: 15.9 years, 75% African American) from urban environments completed a modified monetary incentive delay task during functional magnetic resonance imaging (fMRI). Consistent with the previous research, reward and loss anticipation recruited similar motivational circuitry including striatal, insular, thalamic and supplementary motor areas. Race and gender were not associated with reward- or loss-related neural reactivity. Age and pubertal development were associated with differences in neural reactivity to reward and loss, suggesting that older and more mature adolescents had increased activity in sensory and motivational circuits, but decreased activity in regions responsible for error detection and behavior modification.

Key words: adolescence; reward; loss; fMRI

Introduction

Adolescence is a developmental period characterized by increased impulsive and reward-driven behaviors, often without consideration for long-term consequences (Steinberg, 2010). The onset of puberty brings rapid changes in hormone levels that impact brain development and increase motivation toward novel, rewarding and high-sensation activities (Crone and Dahl, 2012). Although these changes can increase prosocial behaviors, they may also increase vulnerability to unhealthy motivated behaviors, such as thrill-seeking, substance use and delinquency (Galván *et al.*, 2007; Bjork and Pardini, 2015).

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Reinforcement processing is a complex construct with distinct phases (e.g. reward and loss anticipation and consumption) that recruits a network of corticostriatal brain regions involved in valuation, decision-making and arousal (Berridge and Robinson, 2003). The ventral striatum (VS) is a reinforcement network hub and is involved in reward valuation and motivation (Heekeren et al., 2007). Beyond the VS, meta-analyses indicate that reward and loss anticipation activate a common set of regions including the insula, amygdala, supplementary motor area (SMA) and thalamus, whereas reward consumption additionally recruits the medial prefrontal cortex (mPFC) and posterior cingulate (Oldham et al., 2018). Although less studied, loss consumption has been linked to increased activation in the anterior cingulate (ACC)/mPFC, insula, striatum and putamen (Dugré et al., 2018).

The monetary incentive delay (MID; Knutson et al., 2001b) task is a widely used neuroimaging paradigm for examining reward processing because it allows for the investigation of anticipatory and consummatory phases of reward and loss processing (Lutz and Widmer, 2014). However, few studies have investigated both phase (anticipation vs consumption) and valance (loss and reward) components within the same sample. Moreover, there is a relative lack of research on the neural correlates of loss consumption, particularly among adolescents-a key gap given that loss processing may be as important as reward to some risky behaviors (Dugré et al., 2018). Finally, although the MID task is designed to parse neural response for anticipation and consumption, most functional magnetic resonance imaging (fMRI) studies that aim to separate these phases have little (and potentially insufficient) temporal separation between anticipation and consumption (e.g. 2000-2500 ms; Knutson et al., 2001a) or between trials (e.g. 0-2000 ms; Knutson et al., 2001b; Bjork et al., 2004; Schumann et al., 2010; Casey et al., 2018). These gaps in the literature highlight important targets for future research in order to improve the understanding of reward and loss processing.

Moreover, much of the existing adolescent neuroimaging research has focused on mostly European and European American adolescents and often samples of convenience (e.g. undergraduates), potentially undermining our knowledge of 'normative' brain functioning (Chiao, 2009; Chiao and Cheon, 2010; Button et al., 2013; Falk et al., 2013; Hyde et al., 2015). Although the MID has been implemented in some large samples (e.g. IMAGEN, Schumann et al., 2010), these studies lack substantial representation of adolescents of color or those living in poverty. This limitation is particularly problematic when studying the neural correlates tied to risky behavior, because 'risky behavior' may have different causes and consequences in adolescents facing adversity, particularly those with less resources (i.e. who may value \$5 differently than advantaged participants) and for African American adolescents (whose risky behavior is often met with more extreme consequences; Burch, 2015). Thus, to better understand risky behaviors in riskier contexts, studies must examine whether the MID task activates similar neural regions in these contexts, with attention to potential demographic (e.g. gender and race) effects.

Though several studies have charted age-related changes in reward-related neural activity (Galván *et al.*, 2006; Galván, 2013; Silverman *et al.*, 2015), few studies have examined puberty and gender. Even within a narrow age range, there are substantial individual differences in biological and psychological functioning during adolescence (Braams *et al.*, 2015). A recent study examined gender and puberty effects on reward-related neural response using the MID task. They found that boys had greater putamen, middle temporal gyrus and precuneus response during reward anticipation us girls, whereas puberty was not related to reward response (Cao et al., 2019). In contrast, Forbes and colleagues found that using a sample with a narrow age range (11–13 years), mid-late pubertal adolescents compared to pre-early pubertal adolescents had reduced caudate activity and increased ACC/mPFC activity during reward receipt but not anticipation (Forbes et al., 2010). Notably, neither study investigated whether gender or puberty is related to loss-related neural functioning, and it is unclear whether ageor puberty-related differences in reward function will replicate in more representative samples.

Current study

The primary aim of this study was to characterize patterns of neural reactivity during the anticipation and consumption of reward and loss in a well-sampled, primarily low-income, urban cohort of adolescents with substantial representation of youth identifying as Black or African American. The second aim was to examine age, pubertal status, gender and race (a social, not biological construct) as potential sources of individual variability in neural function. This study addressed limitations of previous research in several ways: (i) investigating neural response during both phase (anticipation and consumption) and valance (reward and loss) in the same MID paradigm; (ii) investigating reward and loss processing during middle adolescence, a time of significant biological and behavioral development; (iii) examining the influence of demographic variables (age, gender and race) on neural reactivity; (iv) examining puberty within a narrow age range where puberty varies substantially but age does not and (v) recruiting a populationbased sample with greater representation of adolescents of color and families from lower socioeconomic contexts, thus providing one of the first opportunities to investigate reinforcement processing in adolescents who are typically under-represented in neuroimaging research. We investigated these associations in the VS, the key region of interest (ROI) in previous investigations, and across the whole brain, given the robust literature linking the broader corticostriatal system to reinforcement processing.

Consistent with the previous literature (Dugré et al., 2018; Oldham et al., 2018), we hypothesized that reward and loss anticipation would recruit similar regions within the corticostriatal circuit including the VS, SMA, thalamus and cingulate, whereas reward outcomes would additionally recruit the mPFC and loss outcomes would recruit the insula and the posterior cingulate. Given research suggesting that adolescence is linked to increased sensitivity to reward cues (Steinberg, 2010; Silverman et al., 2015), age was hypothesized to be negatively related to VS reactivity during reward anticipation. Puberty was hypothesized to be associated with reduced VS reactivity during reward outcome (Forbes et al., 2010). Furthermore, since adolescence is linked to relatively reduced recruitment of cognitive control systems (Steinberg, 2005; Forbes et al., 2010), age and pubertal status were hypothesized to be positively associated with mPFC activity during reward outcomes and positively associated with ACC/mPFC activity during the loss outcomes. Based on Cao and colleagues (2019), boys were hypothesized to have greater putamen response during reward anticipation but not outcome. We did not make specific hypotheses regarding gender differences in response to loss.

Method

Participants

The study examined a subsample of 237 adolescents (128 with useable fMRI data) from the Study of Adolescent Neural Development (SAND) who participated in the longitudinal Fragile Families and Child Wellbeing Study (FFCWS; Reichman *et al.*, 2001). The FFCWS is a representative population-based sample of children born in hospitals in large US cities (>200000) between 1998 and 2000. The FFCWS was oversampled 3:1 for non-marital births. At initial recruitment in the hospital, 42.16% of families reported a household income of <\$25 000/year. At childbirth, maternal self-identified race was 21.1% White non-Hispanic, 47.5% Black non-Hispanic, 27.3% Hispanic and 4.0% other. FFCWS families were interviewed at the focal youth birth and at 1, 3, 5, 9 and 15 years of age.

At the age of 15 years, FFCWS families in Detroit, Toledo, and a subsample of those in Chicago were contacted to participate in the SAND study and screened for MRI counter-indications. Eligible participants visited the University of Michigan, and youth completed a practice MID task on a laptop to ensure task understanding and to determine the initial response window. Youth also completed questionnaires, a psychiatric interview and provided biological samples. Primary caregivers completed questionnaires and psychiatric interviews about their own and their child's mental health. Participants were compensated for their time and travel. The University of Michigan Medical School Institutional Review Board approved all procedures. 195 youth completed the MRI scan. After behavioral and fMRI pre-processing, the final sample included 128 participants (Table 1) and was 58% female, 76% African American, 12% Caucasian and 4% biracial/multiracial. Youth ranged in the age from 15.0–17.6 years (mean = 15.9; Table 2).

Measures

MID task. Participants completed a modified version of the MID task during fMRI. The task was presented in two, 45-trial, 9.4 min runs (Figure 1). During each trial, participants saw a trial-type cue (18 win, 18 loss and 9 neutral trials). After a variable fixation cross-hair delay, participants responded with their right index finger when the target (white square) appeared. After a fixation cross-hair delay, feedback was presented on the points won or lost, followed by a jittered inter-trial interval. A performance tracking algorithm adjusted task difficulty so that participants successfully responded to \sim 50% of the trials. Although participants received a summary of their point accumulation at the end of the task, participants did not earn performance-based compensation/money. The task offers some advantage over previous versions of the MID (e.g. Knutson et al., 2001a; Schumann et al., 2010; Casey et al., 2018) as it allows for sufficient temporal separation of reward anticipation and consumption phases (6 s) and between the target offset and feedback to account for motor responses (3 s), as well as a jittered inter-trial interval to separate trials.

Puberty. Pubertal development was measured using youth report on the Pubertal Development Scale (Petersen *et al.*, 1988), which has shown high correlations (0.61–0.67) with physician ratings (Brooks-Gunn *et al.*, 1987). It includes five questions about physical development rated from 1 ('has not started') to 4 ('development seems complete'). Four youth did not complete the measure and parental report on the Pubertal Development Scale was used instead. Pubertal development was calculated as a mean score of the five items [total = 3.29(0.59);

	Number lost	Participants with data
Original sample		237
Sample with imaging data		
- Refused MRI	7	
- Exceeded MRI table	5	
weight limit/couldn't		
fit in scanner - Medical restriction	3	
 Braces or other metal 	13	
in body		
 Risk of pregnancy 	1	
 Missed scanning 	1	
appointment - Excluded for diagnosis	2	
of autism spectrum		
disorder - Incomplete fMRI data	12	
Total lost	44	193
Sample with usable imaging		
data		
 Task administra- 	2	
tion issue (i.e. wrong		
version, wrong hand)		
 fMRI scan quality 	17	
issues (distortion,		
artifact, signal dropout)	_	
- Low VS coverage	7	
(<70%)	1	
- Motion outlier (>20%	1	
IRS WITH ARI)	26	
- Poor task performance	20	
(<6 trials per outcome		
- Poor task performance	6	
 1001 task performance (>10 consecutive trials 	0	
without a recorded		
hutton press)		
- Activation outlier	6	
Total lost	65	128
101011031	05	120

Table 2. Descriptive statistics of demographic measures

Measure	Count (%)
Adolescent gender	
- Male	54 (42.2)
- Female	74 (57.8)
Adolescent self-reported race	
- Black/African American	97 (75.8)
- White/Caucasian	15 (11.7)
- More than one race	5 (3.9)
- Other non-Hispanic Groups	5 (3.9)
Adolescent self-reported ethnicity	
- Not Hispanic	122 (95.3)
- Hispanic	6 (4.7)
Measure	Mean (s.d.)
- Age (years)	15.88 (0.53)

female = 3.62(0.48); male = 2.85(0.42); Figure 2] and were windsorized to three standard deviations from the mean.

Demographics. Youth gender and age in months were collected from an interview with the primary caregiver. Youth age was windsorized to three standard deviations from the mean. Race



Fig. 1. MID task: schematic of a single trial. Trial type was indicated by a up arrow, down arrow or a horizontal double arrow to indicate reward, loss and neutral trials, respectively (2 s). After a variable delay (2–2.5 s) a white square (target) appeared. Participants were instructed to respond as quickly as possible to the target. A fixation cross appeared that included a delay (2 s) and a catch-up period to account for variability in participant response. Feedback was presented (1.65 s), followed by a jittered inter-trial interval (2, 3 or 4 s).



Fig. 2. Pubertal development: (A) Distribution of pubertal development categories across age for entire sample (N = 128). (B) Distribution of pubertal development categories across age for girls (N = 74); (C) Distribution of pubertal development categories across age for boys (N = 54).

was assessed using youth-reported ethnic identity from the Multigroup Ethnic Identity Measure (Phinney, 2010). For two youth, self-reported ethnic identity was missing and was coded using the parent report of the child's ethnicity/race from the demographic interview. We examined race, a social construct, to examine potential differences in brain activity that may reflect exposure to systemic racism and the various unequal exposures to stress, trauma and opportunity for African Americans in the USA (Brondolo *et al.*, 2009). Thus, we do not interpret any potential differences between groups as underlying, static 'biological' differences that emerge from genetics. Instead, they reflect the adversities that many African Americans face, which may shape their experience of the world, and in this case, their neural response to rewards or losses.

Analysis

Behavioral data processing. To ensure task engagement, participants with fewer than six trials per outcome were excluded from analyses due to poor task responding. Additionally, participant data with fewer than 10 trials per outcome were visually inspected to ensure task engagement. Upon inspection, participants were included if they consistently responded to the target stimuli (i.e. no more than 10 consecutive trials without a response; Table 1).

Blood oxygenation level dependent (BOLD) fMRI acquisition and pre-processing. Youth were scanned with a GE Discovery MR750 3T MRI scanner with an 8-channel head coil. T1-weighted gradient echo images were taken before the functional scans (repetition time (TR)/echo time (TE) = 9.0/1.8 s, inversion time (TI) = 400 ms, flip angle = 15° , field of vieo (FOV) = 22 cm; slice thickness = 3 mm; 256×256 matrix; 40 slices), and a high-resolution T1 axial overlay was acquired after the functional scans (TR/TE = 250/5.7 s, TI = 400 ms, flip angle = 30° , FOV = 26 cm, slice thickness = 1.4 mm, 256 \times 256; 100 slices). Functional T2*-weighted BOLD images were acquired using a reverse spiral sequence with interleaved contiguous axial 3 mm slices (TR/TE = 2000/30 ms, flip angle = 90° , FOV = 22 cm, voxel size = 3.44 mm \times 3.44 mm \times 3 mm) aligned with the anterior commissure-posterior commissure (AC-PC) plane. Functional images were positioned to maximize limbic coverage. An auto-shimming procedure was conducted to reduce field inhomogeneity. The pre-processing procedure included removing outliers from the raw k-space data, reconstructing the kspace data to image space, field map correction and slice timing correction. Using SPM12 (http://www.fil.ion.ucl.ac.uk/spm/), high-resolution anatomical images were re-oriented to the AC-PC plane, gray matter segmented and functional images were realigned, co-registered, spatially normalized into Montreal Neurological Institute (MNI) space and smoothed with an 8 mm FWHM Gaussian filter.

Imaging processing and analyses. After pre-processing, Artifact detection Tools (ART) software (http://www.nitrc.org/ projects/artifact_detect/) was used to identify motion outliers (>2 mm movement or 3.5° rotation in any direction) within each participant's data. Participants with >20% outlier TRs were excluded. For remaining participants, outlier scans were covaried for in the individual model using a single regressor in first level person-specific models for each outlier (i.e. spike regression). Additionally, single-subject fMRI data were only included in analyses if there was a minimum of 70% coverage in the anatomically defined bilateral VS ROI mask. The VS ROI was constructed from two bilateral 10 mm spheres centered around the MNI coordinates $x = \pm 12$, y = 12, z = -10 (Murray et al., 2017) using the Talairach Daemon option of the WFU PickAtlas Tool v3.0.5. Additionally, data were visually inspected for signal drop out and poor whole brain coverage (particularly in reward-related limbic and prefrontal regions).

Following initial fMRI pre-processing, to examine potential signal outliers, parameter estimates for each contrast of interest were extracted across the whole brain volume to obtain an average global parameter estimate. Six subjects were identified with large outlier parameter estimates (\pm >3 s.d. from sample mean). These subjects' individual functional scans were further inspected to confirm either a few large or many small movements that caused abnormal parameter estimates, despite having<20% outlier scans identified using ART. These subjects were excluded and did not significantly differ from included youth on demographic or developmental measures. Supplementary analyses including these subjects yielded similar outcomes (Supplementary Table S1).

Analyses were conducted in SPM12 across the whole brain and in the VS ROI. The updated version of 3dClustSim (Cox et al., 2017) was used within the Analysis of Functional NeuroImages package for multiple comparison correction. 3dClustSim uses a Monte Carlo simulation to provide a threshold that will achieve a correction for multiple comparisons of P < 0.05. Spatial autocorrelation function (ACF) values for a random 10% of the sample were calculated from the residuals of individual first level models using 3dFWHMx in AFNI. The individual ACF values were averaged (mean values: 0.512, 6.737, 12.656) and input into 3dClustSim to estimate the noise smoothness using a Gaussian plus mono-exponential function. We used a voxel-wise correction of P < 0.001 to achieve a whole brain or ROI P < 0.05 corrected for multiple comparisons. The resulting cluster thresholds were k = 5 for the VS ROI and k = 131 for whole brain analyses.

Main effects of MID task. As the main goal of the study was to investigate anticipatory and consummatory neural response to reward and loss, analyses focused on the following contrasts: (i) reward anticipation > neutral anticipation; (ii) reward anticipation > reward outcome; (iii) reward outcome > neutral outcome; (iv) loss anticipation > neutral anticipation; (v) loss anticipation > loss outcome and (vi) loss outcome > neutral outcome. As an exploratory aim, neural responses to win > no win (i.e. fail to win) and loss > no loss (i.e. avoid loss) were also examined. Supplementary analyses controlling for annual family income yielded similar results (Supplementary Table S3).

Effects of demographic variables. To examine potential developmental effects, age and pubertal status were examined as predictors using multiple regressions while controlling for other demographic variables (i.e. gender, race and puberty or age). Potential gender differences were examined using t-tests (i.e. boys vs girls) while controlling for age, puberty and race. Potential racial differences were examined using t-tests for the two largest racial groups (i.e. African American vs European American), while controlling for gender, puberty and age. The goal of these analyses was to determine whether the task activated the brain similarly across these socially constructed groups.

Results

Main effects of the MID task

Reward anticipation us neutral anticipation. Reward anticipation>neutral anticipation was associated with greater reactivity in a cluster encompassing the SMA and superior frontal gyrus (SFG) and a cluster encompassing the putamen, midbrain and thalamus (Table 3, Figure 3). There were two clusters of decreased activation in the bilateral inferior frontal gyri (IFGs) and within several regions of the default mode network (DMN) including the bilateral angular gyri, bilateral SFG and mid-cingulate/precuneus.

Reward anticipation vs reward outcome. Reward anticipation > reward outcome was associated with increased reactivity in a large cluster encompassing the SMA and pre-central gyrus, supramarginal gyrus and bilateral middle frontal gyri, and the bilateral VS ROI. There were also several clusters of decreased activity in the DMN including the angular gyrus/inferior parietal lobule, visual cortex, SFG/middle frontal gyrus (MFG) and posterior cingulate (Table 3, Figure 4).

Reward outcome vs neutral outcome. Reward outcome > neutral outcome was associated with increased reactivity in the

Table 3. Main effects of task

Analysis	t	Cluster size	MNI coordinates	Direction	Brain region
Reward anticipat	tion > neutra	anticipation			
Whole brain	7.67	2550	-2 -2 56	Increased	SMA, pre-central gyrus, SFG
Whole brain	4 29	2350	-24 -10 14	Increased	Putamen midbrain thalamus
	6.60	1122	34 -64 42	Decreased	Angular gyrus middle temporal gyrus
	6.00	1205	-46 -62 38	Decreased	Angular gyrus, madule temporar gyrus
	5.44	1509	-40 -02 50	Decreased	Progunous gunous mid singulato
	5.44	1308	-2-04-50 52 56 9	Decreased	reculieus, culleus, filiu-ciligulate
	5.45 E 21	9 4 0 214	-J2 -J0 -0	Decreased	IFC nore triangularia
	5.31	1025	10 16 22	Decreased	In G-pais triangularis
	3.20	1055	-40 10 52	Decreased	menor nontal operculuit, MFG, SFG
	4.97	1/1	62 - 48 - 10	Decreased	
	4.47	138	4 - 52 6	Decreased	Posterior cingulate
	4.40	594	14 26 52	Decreased	SFG
Reward anticipat	tion > reward	l win			
Whole brain	15.74	21967	-4 -4 56	Increased	SMA, left pre-central gyrus,
	8.12	2626	52 - 42 30	Increased	Supramarginal gyrus
	7.06	884	-32 42 28	Increased	MFG
	6.26	612	30 42 28	Increased	MFG
	10.02	2921	30 -64 40	Decreased	Superior occipital gyrus, inferior parietal lobule
	9.61	474	18 –96 2	Decreased	Calcarine, lingual gyrus, fusiform gyrus
	6.52	1039	-36 -64 40	Decreased	Angular gyrus, inferior parietal lobule
	6.20	387	-22 -94 4	Decreased	Middle occipital gyrus, fusiform gyrus
	6.05	2227	16 38 44	Decreased	SFG. MFG
	5.54	177	0 - 34 32	Decreased	Posterior cingulate gyrus
	4.77	209	-42 14 36	Decreased	MFG
VS ROI	6 5 3	159	-18 12 -2	Increased	Putamen
101101	6 5 3	97	20.8 - 6	Increased	Putamen
		57	200 0	mercubeu	
Reward win>ne	utral outcon	ne			
Whole brain	5.90	385	48 – 36 52	Increased	Inferior parietal lobule
	8.38	14938	-8 -48 66	Decreased	Precuneus, calcarine, MFG
	6.54	3264	-60 -58 14	Decreased	Middle temporal gyrus, supramarginal gyrus
	4.99	1357	52 -60 6	Decreased	Middle temporal gyrus, STG, middle occipital gyrus
	4.85	249	56 - 18 - 8	Decreased	Middle temporal gyrus
	4.09	258	-50 18 14	Decreased	Inferior frontal operculum, IFG-pars triangularis
Reward win > no	win				
Whole brain	6.00	1628	42 - 40 46	Increased	Inferior and superior parietal lobule
	4 37	365	-40 -54 50	Increased	Inferior parietal lobule
	5 27	808	-62 - 30.28	Decreased	Supramarginal gyrus, rolandic operculum
	5.12	320	8 - 84 38	Decreased	
	4 75	230	30 46 30	Decreased	MFC
	4.69	506	58 - 32 34	Decreased	Supremarginal gyrus
	1.05	245	14 54 60	Decreased	Procupous
	4.02	262	20 0 50	Decreased	SEC .
	4.34	680	20 - 8 38	Decreased	Lingual gurus
	4.37	089	-10 -70 -4 F0 64 10	Decreased	Middle temporal grave
	4.09	126	10 00 16	Decreased	Cupous
	3.91	130	-10 -90 16	Decreased	Curieus
Loss anticipation	n>neutral a	nticipation			
Whole brain	5.38	1138	-38 -16 52	Increased	Pre-central gyrus, SMA
	4.36	243	-60 -54 -10	Decreased	ITG, fusiform gyrus
Loss anticipation	>loss outco	ome			
Whole brain	15 15	10826	_4 _4 56	Increased	SMA pre-central avrus mid-cingulate
whole brain	11 / 0	2100		Increased	Butamon incula
	0.41	2010	-220-4 2204	Increased	Putamon incula
	5.41	400	52 64 4	Increased	Middle temporal grave
	0.09	400 2011	-32 -04 4	Increased	Supromorginal grain middle terrescuel grant
	6.5/	2011	58 -40 30	Increased	Supramarginai gyrus, middie temporal gyrus
	5./6	603	-34 36 28	Increased	
	4.59	158	-14 -58 58	Increased	Precuneus
	10.62	11131	30 -62 44	Decreased	Parietal Lobe, calcarine, middle occipital gyrus
	7.27	2413	-2 48 32	Decreased	MFG, ACC
	7.24	879	-46 16 28	Decreased	IFG-pars triangularis
	6.62	733	-32 -58 40	Decreased	Inferior parietal lobule
	6.33	313	40 16 -12	Decreased	Insula

Table 3. (Continued)

Analysis	t	Cluster size	MNI coordinates	Direction	Brain region
	6.23	158	-54 -8 32	Decreased	Pre-central gyrus
	6.14	257	-42 16 -14	Decreased	IFG
	4.65	179	-60 -32 0	Decreased	Middle temporal gyrus
VS ROI	11.07	245	-20 12 -4	Increased	Putamen
	8.47	185	20 6 -10	Increased	Putamen
Loss outcome > n	eutral outcome				
Whole brain	5.88	280	42 16 -10	Increased	Insula
	5.19	1035	0 36 24	Increased	ACC
	4.89	224	-2 -30 -4	Increased	Midbrain
	4.82	323	52 - 34 50	Increased	Inferior parietal lobule
	5.70	4417	14 -40 58	Decreased	Paracentral lobule, precuneus, SMA
	4.66	415	-20 18 42	Decreased	MFG
	4.13	352	-46 -60 32	Decreased	Angular gyrus, middle temporal gyrus
	4.11	297	22 18 22	Decreased	Caudate
	3.83	176	2 -50 32	Decreased	Mid-cingulate
Loss outcome > n	io loss				
Whole brain	6.14	1933	-4 50 26	Increased	Superior medial frontal gyrus, ACC
	5.41	1910	2 -70 8	Increased	Lingual gyrus, calcarine
	4.88	342	-6 -26 -6	Increased	Midbrain
	4.09	369	26 -60 46	Increased	Angular gyrus, superior parietal lobule,
	4.34	159	18 -4 28	Decreased	Cingulate gyrus
	4.16	240	-18 12 -6	Decreased	Putamen, caudate
VS ROI	4.42	69	12 12 -8	Decreased	Caudate
	4.16	101	-18 12 -6	Decreased	Putamen



Fig. 3. Reward and loss anticipation vs outcome. (A) Multi-slice depiction of increased (red) and decreased (blue) activation during reward anticipation > reward win. (B) Increased reactivity in the VS ROI during reward anticipation > reward win (displayed at peak voxel, t = 6.53, k = 159, x = -18, y = 12, z = -2). (C) Multi-slice depiction of increased (red) and decreased (blue) activation during loss anticipation > loss outcome. (D) Increased reactivity in the VS ROI during loss anticipation > loss win (displayed at peak voxel, t = 11.07, k = 245, x = -20, y = 12, z = -4).

inferior parietal lobule and decreased reactivity in a large cluster with a peak in the precuneus and extending to the visual cortex and bilateral mPFC, a cluster in the IFG, and in several clusters within the bilateral middle temporal gyri (Table 3, Figure 5). Reward outcome vs no win. Win>no win was associated with increased bilateral inferior partial lobule reactivity, and decreased reactivity in the bilateral supramarginal gyrus, bilateral precuneus/cuneus, MFG, middle temporal gyrus and lingual gyrus (Figure 6).



Fig. 4. Reward and loss anticipation vs neutral anticipation. (A) Multi-slice depiction of increased (red) and decreased (blue) activation during reward anticipation > neutral anticipation. (B) Multi-slice depiction of increased (red) and decreased (blue) activation during loss anticipation > neutral anticipation.

Loss anticipation vs neutral anticipation. Loss Anticipation>neutral anticipation was associated with increased reactivity in the pre-central gyrus/SMA and inferior temporal gyrus (ITG; Table 3, Figure 3).

Loss anticipation vs loss outcome. Loss anticipation > loss outcome was associated with a similar pattern of reactivity as reward anticipation > reward outcome, including increased reactivity in a large cluster encompassing the SMA and precentral gyrus, bilateral clusters encompassing the putamen, thalamus, insula, middle temporal gyri, precuneus and the bilateral VS ROI. There were also several clusters of decreased activity including the inferior parietal lobule, IFG and insula (Table 3, Figure 4). Loss outcome vs neutral outcome. Loss outcome > neutral outcome was associated with increased reactivity in the right insula, ACC, midbrain and inferior parietal lobule, and decreased reactivity in a large cluster encompassing the paracentral lobule, precuneus and SMA, and clusters in the MFG, angular gyrus, caudate and mid-cingulate (Table 3, Figure 5).

Loss outcome vs no loss. Loss > no loss was associated with increased reactivity in superior MFG, visual cortex, midbrain and angular gyrus, and decreased reactivity in the cingulate/dorsal caudate, left putamen (Figure 7) and the bilateral VS ROI.



Fig. 5. Reward and loss outcome vs neutral outcome. (A) Multi-slice depiction of increased (red) and decreased (blue) activation during reward win > neutral outcome. (B) Multi-slice depiction of increased (red) and decreased (blue) activation during loss outcome > neutral outcome.

Demographic effects on reward- and loss-related neural response

Age. Participant age (controlling for puberty, gender and race) was associated with greater reactivity in the bilateral postcentral gyrus, supramarginal gyrus, ITG, MFG and vermis during reward anticipation > neutral anticipation. During reward anticipation > reward outcome, age was associated with increased post-central gyrus activity, but decreased post-central gyrus activity. Age was also associated with decreased reactivity in the mid-cingulate/precuneus during win > no-win trials.

During loss anticipation > neutral anticipation, age was positively correlated with SMA, inferior parietal lobule, IFG, postcentral gyrus, caudate and VS ROI activity (Figure 8). During loss anticipation > loss outcome, age was associated with increased paracentral gyrus and cingulate activity and decreased inferior occipital gyrus activity. During loss outcomes > neutral outcome, age was associated with increased activity in the ITG and decreased activity in the pre-central gyrus. Age was associated with increased activity in the ITG during loss > no loss (Table 4).

Puberty. Puberty (controlling for age, gender, and race) was associated with decreased mid-cingulate activity during reward anticipation > neutral anticipation and reward outcome > neutral outcome (Figure 9), as well as during loss anticipation > loss outcome and loss outcome > neutral outcome (Table 4).



Fig. 6. Loss outcome vs no loss. (A) Increased activity in the ACC, midbrain and lingual gyrus during loss outcome > no loss (displayed at peak voxel, t = 6.14, k = 1933, x = -4, y = 50, z = 26). (B) Increased activity in the left putamen/caudate during loss outcome > no loss (displayed at peak voxel, t = 4.16, k = 240, x = -18, y = 12, z = -6). (C) Depiction of increased (red) and decreased (blue) activation during Loss outcome > no loss.



Fig. 7. Reward win vs no win. (A) Increased activity in the inferior parietal lobule during reward win > no win. (B) Decreased activity in supramarginal gyrus during reward win > no win. (C) Depiction of increased (red) and decreased (blue) activation during reward win > no win.

Gender. There were no significant neural differences between boys and girls during reward- or loss-related processing.

Race. There were no significant neural differences between selfreported Black/African American and White/European American youth during reward or loss-related processing.

Table 4. Associations between age and puberty and reward and loss processing

Analysis	t	Cluster Size	MNI Coordinates	Direction	Brain Region
Reward anticipation > neu	ıtral anticipati	on			
Age whole brain	4.62	261	56 - 18 38	Increased	Post-central gyrus
	4.41	668	-48 -40 34	Increased	Supramarginal gyrus
	4.27	288	2 -46 14	Increased	Vermis
	4.23	157	32 - 36 60	Increased	Post-central gyrus
	4.05	260	-32 34 18	Increased	MFG
	4.04	174	48 - 40 - 12	Increased	ITG
Puberty whole brain	3.95	266	-6 -2 40	Decreased	Mid-cingulate
Reward anticipation > rew	ard win				
Age whole brain	4.31	187	-40 -38 60	Increased	Post-central gyrus
Reward win > neutral outc	come				
Age whole brain	3.78	195	-30 -26 48	Decreased	Post-central gyrus
Puberty whole brain	3.81	169	4 -12 40	Decreased	Mid-cingulate
Reward win>no win					
Age whole brain	4.79	1246	30 - 28 38	Decreased	Mid-cingulate, precuneus
Loss anticipation > neutra	l anticipation				
Age whole brain	4.17	195	6 16 0	Increased	Caudate head
	4.37	1197	12 –26 50	Increased	SMA
	4.24	275	-48 -42 36	Increased	Inferior parietal lobule
	3.81	222	38 28 26	Increased	IFG-pars triangularis
	3.73	144	54 -16 40	Increased	Post-central gyrus
Age VS ROI	4.15	13	8 16 -2	Increased	Caudate head
0	3.19	5	-8 10 -8	Increased	Caudate
Loss anticipation > loss ou	itcome				
Age whole brain	4.15	162	14 - 30 52	Increased	Paracentral gyrus
	3.85	252	14 -12 40	Increased	Cingulate gyrus
	3.51	156	32 - 74 - 4	Decreased	Inferior Occipital Gyrus
Loss outcome > neutral ou	itcome				
Age whole brain	4.01	172	44 - 44 - 14	Increased	ITG
	4.19	218	-32 -22 54	Decreased	Pre-central Gyrus
Puberty whole brain	3.96	269	-6 -2 40	Decreased	Mid-cingulate
Loss outcome > no loss					
Age whole brain	3.71	176	52 - 50 - 4	Increased	ITG

Discussion

The current study takes a significant step toward improving generalizability of neuroimaging research by investigating reward-related neural functioning in an urban, low-income and well-sampled cohort of primarily African American youth. The novel version of the MID task allowed for a more fine-grained approach for examining anticipatory and consummatory components of reward and loss processing. Overall, the study demonstrated that this version of the MID task robustly engaged the VS and is consistent with the previous research demonstrating common patterns of neural activity within striatum, insula, thalamus and SMA for reward and loss anticipation (Oldham et al., 2018). It also adds to the limited research investigating the neural correlates of loss (Dugré et al., 2018), by reporting that loss outcomes recruit regions responsible for performance monitoring and motor integration including the ACC, insula and inferior parietal lobe. Race and gender analyses were not significant, indicating that this task may be potentially be used across diverse populations. Finally, we found unique effects of age and puberty on reward and loss processing. Specifically, age (controlling for puberty) was associated with greater activity in sensory and motivation regions during reward and loss processing, whereas pubertal development (controlling for age) was associated with decreased mid-cingulate activity during reward and loss processing. Thus, during this period of rapid development, age and puberty independently contribute to individual differences in reward and loss processing.

Common neural reactivity during reward and loss anticipation

Consistent with prior research in adults (Oldham et al., 2018), our adolescent sample displayed similar patterns of neural activation in the striatum, insula, thalamus and SMA during reward and loss anticipation. Interestingly, the current study found smaller clusters and effects when reward/loss anticipation was contrasted with neutral anticipation, than when contrasted with reward/loss outcome (reward anticipation > neutral anticipation vs reward anticipation > reward outcome $Z_{observed} = 11.91$, P < 0.0001; loss anticipation > neutral anticipation vs loss anticipation > loss outcome $Z_{observed} = 25.98$, P < 0.0001). It may be that neutral trials, or task participation in general, was inherently motivating, thus minimizing differences on reward/loss anticipation vs neutral contrasts. Moreover, strong activation in the SMA for all anticipation trials, likely indicating motor preparation, may have minimized differences between anticipation trials. The similar activation patterns between reward and loss anticipation and the relatively small differences vs neutral anticipation provide further support for the notion of a valence-independent motivational system.



Fig. 8. Age is associated with increased VS reactivity during loss anticipation. Increased activity in the VS ROI during loss anticipation > neutral anticipation (displayed at peak voxel of the VS cluster, t = 4.17 k = 195 x = 6, y = 16, z = 0).

Brain reactivity to reward outcome

Reward outcome (vs neutral outcome) was associated with large clusters of DMN deactivation including the posterior cingulate and precuneus. The DMN is consistently deactivated during task engagement (Greicius *et al.*, 2003), suggesting that participants were more engaged during the outcome of incentivized vs nonincentivized trials. Few studies have examined both activation and deactivation during reward vs no-win outcomes. Cao and colleagues (2019) reported decreased VS activity during no-win trials, which was hypothesized to signal a negative prediction error. Although we did not find differences in VS reactivity, no-win trials (i.e. the negative contrast of win > no win) evoked increased activity in the dorsolateral prefrontal cortex, superior temporal gyrus and posterior insula, suggesting that unexpected negative outcomes recruit a network of regions responsible for attention and behavioral adaptation (Paulus *et al.*, 2005; Dixon *et al.*, 2018).

Brain reactivity to loss outcome

This study adds to the limited body of research investigating the neural correlates of loss. Loss outcome was associated with increased reactivity in the ACC, anterior insula and midbrain vs



Fig. 9. Pubertal status is associated with decreased mid-cingulate reactivity during reward and loss outcome. (A) Pubertal status was associated with decreased activity in the mid-cingulate reward outcome > neutral outcome (displayed at peak voxel, t = 3.81, k = 169, x = 4, y = -12, z = 40). (B) Pubertal status was associated with decreased activity in the mid-cingulate loss outcome > neutral outcome (displayed at peak voxel, t = 3.96, k = 269, x = -6, y = -2, z = 40).

neutral outcomes, which is consistent with a meta-analysis of loss processing in adults (Dugré *et al.*, 2018). Greater loss-related activation in the ACC, insula and midbrain that are involved in error monitoring, affective valuation and behavior change may indicate efforts to modulate behavior in response to negative feedback.

Loss avoidance (i.e. the negative contrast of loss > no loss) revealed increased caudate and putamen activity. Increased caudate and putamen activity may indicate a positive prediction error response to a 'better than expected' outcome (Schultz, 2017). Conversely, loss outcomes were associated with decreased caudate activation, suggesting a negative prediction error response to a worse than expected outcome (Schultz, 2017).

Development and neural reinforcement processing

We did not find the hypothesized associations between age and puberty and frontostriatal activity during reinforcement processing. Age was uniquely associated with increased activation in the post-central gyrus/somatosensory cortex and inferior parietal lobule/supramarginal gyrus during reward and loss anticipation; increased VS activation during loss anticipation; and decreased post-central gyrus activity during reward outcomes. Sensory systems are strongly modulated by motivational information, particularly when accurate stimulusresponse pairing is essential to obtain reward (Pantoja et al., 2007). Our findings suggest that older youth recruited brain regions implicated in motivated attention when viewing reinforcement cues, perhaps to optimize performance. Despite a small age range (15.0-17.6 years), age was uniquely related to differences in sensory and reward-related brain regions during reward and loss processing.

Pubertal development was linked to reduced activity in the mid-cingulate/dorsal ACC during reward and loss processing. The mid-cingulate/dorsal ACC has connections to the lateral prefrontal cortex, parietal lobe, striatum and motor systems (Vogt, 2016), regions that were activated during the main effects of this task and in prior meta-analyses of reinforcement processing (Silverman *et al.*, 2015; Dugré *et al.*, 2018; Oldham *et al.*, 2018).

Overall, these findings suggest that during middle adolescence, age uniquely predicts activation in sensory and motivational regions during reward and loss anticipation, whereas puberty uniquely predicts decreased activity in regions responsible for error detection and behavior modification during reward and loss processing. These findings may partially explain the increase in reward-seeking behaviors seen during midto-late adolescence, which can contribute to negative (e.g. risky behavior) and positive (e.g. social affiliation) outcomes (van Duijvenvoorde et al., 2016).

No race or gender differences in neural reinforcement processing

The study provided one of the first opportunities to examine whether this task activated similar frontostriatal circuitry in a sample that included a large proportion of African American youth. We found no significant differences between African American and European American youth in reward- and lossrelated neural response. However, these results should be interpreted with caution, as there were only 15 European American youth included in the analyses, limiting power. However, if these null findings are replicated, they indicate that this task may work equally well with European and African American youth—an important task equivalence if we are to use this task in population-based samples (e.g. the adolescent brain cognitive development study; Casey et al., 2018). Though many studies have examined the 'measurement equivalence' of surveys between race and other demographic factors, little attention has been paid to the idea that fMRI tasks may not function equivalently across these socially constructed groups and thus

results may not be comparable (Chiao, 2009; Chiao et al., 2013; Hyde et al., 2015).

Additionally, we found no significant gender differences. Although the previous research indicates gender differences in neural response to social reward (Spreckelmeyer *et al.*, 2009), and during reward-related decision-making under stress (Lighthall *et al.*, 2011), a recent study found only minor gender differences during a MID task (Cao *et al.*, 2019). The current study extends previous findings by suggesting that, in a primarily low-income sample with a large proportion of African American youth, boys and girls do not differ in their neural response to non-social reward under low-stress conditions.

Limitations

Although the current study has several strengths including a well-sampled cohort of mostly African American youth recruited at birth from urban hospitals and a carefully designed task, several limitations should be considered. First, due to the population-based sampling methodology used in the larger FFCWS, a significant portion of participants did not participate in the scanning session due to MRI contraindications (Table 1). Moreover, several participants had insufficient responding on the MID task. It is unclear whether youth with uncollected/unusable MRI data would display similar patterns of neural reactivity, although they did not differ based on age, gender, puberty or family income (P-values = 0.11-0.89). Though this data loss is substantial, given the sampling frame, we are, at least, able to document who was and was not scanned (whereas most studies only report participants who completed the scan). Second, our task did not record participant responses that occurred outside of the response window. Thus, we are not able to distinguish too slow responses from those that did not occur at all. Although there were no reaction time differences between successful reward and loss trials, we noted reaction time differences to neutral trials suggesting that comparisons to neutral conditions may not adequately control for motor response for some participants. Nevertheless, it is encouraging that our results are consistent with previous findings using neutral conditions (Dugré et al., 2018; Oldham et al., 2018). Third, we used point rewards to reduce potential income-related effects of monetary reward in this primarily low-income sample. However, points may be less salient than money and could have affected the pattern or strength of our findings. Additionally, although the unique features of our sample are a strength, the sample was primarily African American (76%) with only 11.8% European American youth and 4.7% Latinx youth. We were unable to examine more fine-grained racial differences (e.g. Latinx or other under-represented groups) and may be underpowered to detect small to medium differences between African American and European American youth. Finally, though we examined neural activity during mid-to-late adolescence, which limited variability in age while allowing for variability in puberty, analyses examining age may have been limited by the relatively restricted window of adolescence we sampled (3 years).

Conclusion

The current study is among the first to examine the neural correlates of reward- and loss-related functioning in a well-sampled cohort of primarily African American youth living in low-income urban environments. The study is consistent with the previous research demonstrating that reward and loss anticipation share common patterns of neural activity and adds to the limited body of research investigating the neural correlates of loss by reporting that loss outcomes recruit regions responsible for performance monitoring and motor integration. The study also identified several developmental effects suggesting that middle adolescence is characterized by increased activity in motivational regions, but decreased activity in regions responsible for error detection and behavior modification. Finally, the study found that boys and girls and African American and European American youth did not differ on their neural response to reward and loss.

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Supplementary data

Supplementary data are available at SCAN online.

Conflict of interest

None declared.

References

- Berridge, K.C., Robinson, T.E. (2003). Parsing reward. Trends in Neurosciences, 26(9), 507–13.
- Bjork, J.M., Knutson, B., Fong, G.W., Caggiano, D.M., Bennett, S.M., Hommer, D.W. (2004). Incentive-elicited brain activation in adolescents: similarities and differences from young adults. *Journal of Neuroscience*, 24(8), 1793–802.
- Bjork, J.M., Pardini, D.A. (2015). Who are those "risk-taking adolescents"? Individual differences in developmental neuroimaging research. Developmental Cognitive Neuroscience, 11, 56–64.
- Braams, B.R., van Duijvenvoorde, A.C., Peper, J.S., Crone, E.A. (2015). Longitudinal changes in adolescent risk-taking: a comprehensive study of neural responses to rewards, pubertal development, and risk-taking behavior. *Journal of Neuroscience*, 35(18), 7226–38.
- Brondolo, E., Gallo, L.C., Myers, H.F. (2009). Race, racism and health: disparities, mechanisms, and interventions. *Journal of* Behavioral Medicine, 32(1), 1.
- Brooks-Gunn, J., Warren, M.P., Rosso, J., Gargiulo, J. (1987). Validity of self-report measures of girls' pubertal status. Child Development, 58(3), 829–41.

- Burch, T. (2015). Skin color and the criminal justice system: beyond black-white disparities in sentencing. *Journal of Empirical Legal Studies*, 12(3), 395–420.
- Button, K.S., Ioannidis, J.P., Mokrysz, C., et al. (2013). Power failure: why small sample size undermines the reliability of neuroscience. Nature Reviews Neuroscience, 14(5), 365–76.
- Cao, Z., Bennett, M., Orr, C., et al. (2019). Mapping adolescent reward anticipation, receipt, and prediction error during the monetary incentive delay task. *Human Brain Mapping*, 40(1), 262–83.
- Casey, B., Cannonier, T., Conley, M.I., et al. (2018). The adolescent brain cognitive development (ABCD) study: imaging acquisition across 21 sites. Developmental Cognitive Neuroscience, 32, 43–54.
- Chiao, J.Y. (2009). Cultural neuroscience: a once and future discipline. Progress in Brain Research, 178, 287–304.
- Chiao, J.Y., Cheon, B.K. (2010). The weirdest brains in the world. Behavioral and Brain Sciences, 33(2–3), 88–90.
- Chiao, J.Y., Cheon, B.K., Pornpattananangkul, N., Mrazek, A.J., Blizinsky, K.D. (2013). Cultural neuroscience: progress and promise. Psychological Inquiry, 24(1), 1–19.
- Cox, R.W., Chen, G., Glen, D.R., Reynolds, R.C., Taylor, P.A. (2017). FMRI clustering in AFNI: false-positive rates redux. *Brain Connectivity*, 7(3), 152–71.
- Crone, E.A., Dahl, R.E. (2012). Understanding adolescence as a period of social-affective engagement and goal flexibility. *Nature Reviews Neuroscience*, 13(9), 636.
- Dixon, M.L., De La Vega, A., Mills, C., et al. (2018) Heterogeneity within the frontoparietal control network and its relationship to the default and dorsal attention networks. *Proceedings of the National Academy of Sciences*, 115(7), E1598–607.
- Dugré, J.R., Dumais, A., Bitar, N., Potvin, S. (2018). Loss anticipation and outcome during the monetary incentive delay task: a neuroimaging systematic review and meta-analysis. *PeerJ*, 6, e4749.
- Falk, E.B., Hyde, L.W., Mitchell, C., et al. (2013). What is a representative brain? Neuroscience meets population science. Proceedings of the National Academy of Sciences, 110(44), 17615–22.
- Forbes, E.E., Ryan, N.D., Phillips, M.L., et al. (2010). Healthy adolescents' neural response to reward: associations with puberty, positive affect, and depressive symptoms. *Journal of the American Academy of Child and Adolescent Psychiatry*, 49(2), 162–172. e165.
- Galván, A. (2013). The teenage brain: sensitivity to rewards. Current Directions in Psychological Science, 22(2), 88–93.
- Galván, A., Hare, T., Voss, H., Glover, G., Casey, B. (2007). Risktaking and the adolescent brain: who is at risk? *Developmental Science*, 10(2), F8–F14. . PMID: 17286837.
- Galván, A., Hare, T.A., Parra, C.E., et al. (2006). Earlier development of the accumbens relative to orbitofrontal cortex might underlie risk-taking behavior in adolescents. *Journal of Neuroscience*, 26(25), 6885–92.
- Greicius, M.D., Krasnow, B., Reiss, A.L., Menon, V. (2003). Functional connectivity in the resting brain: a network analysis of the default mode hypothesis. Proceedings of the National Academy of Sciences, 100(1), 253–8.
- Heekeren, H.R., Wartenburger, I., Marschner, A., Mell, T., Villringer, A., Reischies, F.M. (2007). Role of ventral striatum in reward-based decision making. *Neuroreport*, 18(10), 951–5.
- Hyde, L.W., Tompson, S., Creswell, J.D., Falk, E.B. (2015). Cultural neuroscience: new directions as the field matures. *Culture and Brain*, 3(2), 75–92.

- Knutson, B., Adams, C.M., Fong, G.W., Hommer, D. (2001a). Anticipation of increasing monetary reward selectively recruits nucleus accumbens. *Journal of Neuroscience*, 21(16), RC159.
- Knutson, B., Fong, G.W., Adams, C.M., Varner, J.L., Hommer, D. (2001b). Dissociation of reward anticipation and outcome with event-related fMRI. Neuroreport, 12(17), 3683–7.
- Lighthall, N.R., Sakaki, M., Vasunilashorn, S., et al. (2011). Gender differences in reward-related decision processing under stress. Social Cognitive and Affective Neuroscience, 7(4), 476–84.
- Lutz, K., Widmer, M. (2014). What can the monetary incentive delay task tell us about the neural processing of reward and punishment. Neurosciences Neuroeconomics, 3, 33–45.
- Murray, L., Shaw, D.S., Forbes, E.E., Hyde, L.W. (2017). Rewardrelated neural correlates of antisocial behavior and callous– unemotional traits in young men. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*, 2(4), 346–54.
- Oldham, S., Murawski, C., Fornito, A., Youssef, G., Yücel, M., Lorenzetti, V. (2018). The anticipation and outcome phases of reward and loss processing: a neuroimaging meta-analysis of the monetary incentive delay task. *Human Brain Mapping*, 39(8), 3398–3418.
- Pantoja, J., Ribeiro, S., Wiest, M., et al. (2007). Neuronal activity in the primary somatosensory thalamocortical loop is modulated by reward contingency during tactile discrimination. *Journal of Neuroscience*, 27(39), 10608–20.
- Paulus, M.P., Feinstein, J.S., Leland, D., Simmons, A.N. (2005). Superior temporal gyrus and insula provide response and outcome-dependent information during assessment and action selection in a decision-making situation. *Neuroimage*, 25(2), 607–15.
- Petersen, A.C., Crockett, L., Richards, M., Boxer, A. (1988). A self-report measure of pubertal status: reliability, validity, and initial norms. *Journal of Youth and Adolescence*, 17(2), 117–33.
- Phinney, J.S. (2010). Multigroup Ethnic Identity Measure (MEIM). In: Clauss-Ehlers C.S., editor. Encyclopedia of Cross-Cultural School Psychology, Boston, MA: Springer.
- Reichman, N.E., Teitler, J.O., Garfinkel, I., McLanahan, S.S. (2001). Fragile families: sample and design. Children and Youth Services Review, 23(4–5), 303–26.
- Schultz, W. (2017). Reward prediction error. Current Biology: CB, 27(10), R369–71.
- Schumann, G., Loth, E., Banaschewski, T., et al. (2010). The IMA-GEN study: reinforcement-related behaviour in normal brain function and psychopathology. *Molecular Psychiatry*, 15(12), 1128.
- Silverman, M.H., Jedd, K., Luciana, M. (2015). Neural networks involved in adolescent reward processing: an activation likelihood estimation meta-analysis of functional neuroimaging studies. *Neuroimage*, 122, 427–39.
- Spreckelmeyer, K.N., Krach, S., Kohls, G., et al. (2009). Anticipation of monetary and social reward differently activates mesolimbic brain structures in men and women. Social Cognitive and Affective Neuroscience, 4(2), 158–65.
- Steinberg, L. (2005). Cognitive and affective development in adolescence. Trends in Cognitive Sciences, 9(2), 69–74.
- Steinberg, L. (2010). A dual systems model of adolescent risktaking. Developmental Psychobiology, 52(3), 216–24.
- van Duijvenvoorde, A.C., Peters, S., Braams, B.R., Crone, E.A., Reviews, B. (2016). What motivates adolescents?

Neural responses to rewards and their influence on adolescents' risk taking, learning, and cognitive control. *Neuroscience*, 70, 135–47. Vogt, B.A. (2016). Midcingulate cortex: structure, connections, homologies, functions and diseases. *Journal of Chemical Neu*roanatomy, 74, 28–46.