Short Title: ABERRANT REWARD MAGNITUDE TRACKING IN DEPRESSED YOUTH 1

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Aberrant Striatal Tracking of Reward Magnitude in Youth with

Current or Past-Year Depression

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Reward dysfunction is often present in youth with major depressive disorder (MDD), but the specific neurobiological bases underlying reward valuation deficits remain unclear. The current study examined whether youth with MDD track brain and behavioral responses according to relative reward magnitude—a neurocognitive valuation process known as magnitude tracking. Female youth aged 15-20 years (n=56 with current or past-year MDD; n=26 healthy controls [HC]) completed a task during functional neuroimaging in which they could win or lose money at high-stakes (+ $\$1/-50\phi$) and low-stakes (+ $20\phi/-10\phi$). Behaviorally, HC accelerated button press responses on high-stakes compared to low-stakes trials, whereas MDD did not alter response speed across stakes. Neurally, HC increased recruitment of the ventral and dorsal striatum, canonical reward processing regions, for high-magnitude versus low-magnitude rewards. However, the MDD group did not exhibit striatal magnitude tracking for low versus high rewards—an effect independent of MDD recency, MDD symptom severity, comorbid anxiety and substance use disorders, and psychiatric medication use. In contrast, striatal recruitment for overall reward reactivity, measured by comparing striatal activity for reward and loss feedback, was similar in the MDD and HC groups. However, reward reactivity was negatively correlated with current depression symptom severity in the MDD group. Taken together, these findings suggest that while reward reactivity may vary with current depression severity, reward magnitude tracking may represent an important aberrant valuation process in youth with depression-independent of symptom severity and recency. This valuation deficit may have implications for maladaptive motivation and learning observed in youth with MDD.

Keywords: Adolescence; Depression; fMRI; Reward; Loss; Valuation; Striatum

General Scientific Summary: This study demonstrates that youth with current or past-year major depressive disorder (MDD) do not adjust behavioral or brain responses according to the magnitude of rewarding outcomes. Specifically, youth with MDD do not distinguish between high- and low-magnitude reward outcomes in the ventral and dorsal striatum—key regions involved in reward processing. Findings may explain why youth with MDD experience difficulties with motivation and learning from reward.

Major depressive disorder (MDD) is a leading cause of illness and disability worldwide (Global Burden of Disease, 2016; World Health Organization, 2017). Prevalence rates of MDD increase significantly during adolescence (Kessler et al., 2007; Lewinsohn & Essau, 2002; Rohde, Beevers, Stice, & O'Neil, 2009), and adolescent-onset MDD is associated with marked academic and social impairment and serious clinical outcomes such as suicidal thoughts and behaviors (Avenevoli, Swendsen, He, Burstein, & Merikangas, 2015; Birmaher et al., 1996). Youth with MDD often experience reward dysfunction, which may manifest in symptoms such as low positive affect, low energy, low motivation, and anhedonia (a lack of interest or pleasure in previously enjoyed activities; American Psychiatric Association, 2013; Forbes & Dahl, 2012). However, the neurobiological underpinnings of this reward dysfunction in youth remain unclear.

Blunted neural responses to rewards may underlie some of the self-reported and behavioral symptoms of MDD (Barch, Pagliaccio, & Luking, 2015; Baskin-Sommers & Foti, 2015; Dichter, Damiano, & Allen, 2012; Luking, Pagliaccio, Luby, & Barch, 2016; Zald & Treadway, 2017). In particular, much of the prior research in this area has examined aberrant signaling in the striatum—a complex, heterogeneous brain structure within the mesolimbic dopamine circuit that is implicated in reward processing, reinforcement learning, and motivated action selection (Delgado, 2007; O'Doherty, Dayan, Friston, Critchley, & Dolan, 2003; Schultz, 1998). Studies examining reward dysfunction in MDD have focused on aberrant *reward reactivity* in the striatum when individuals receive a reward (outcome phase; e.g. winning money), which is measured by comparing ventral and dorsal striatal responses to rewards relative to neutral (winning nothing) or loss (losing money) outcomes. Converging evidence in adults suggests that depressed individuals exhibit reduced striatal reactivity during reward outcome compared to non-depressed controls (Keren et al., 2018; however for exceptions see: Knutson, Bhanji, Cooney, Atlas, & Gotlib, 2008; Moutoussis et al., 2018; Ubl et al., 2015).

Due to a current dearth of neuroimaging research in youth with MDD, the neural bases of aberrant reward-related processing remain underexplored in this developmental population. Although there is some evidence for blunted striatal reactivity to reward receipt in youth with MDD (e.g., Forbes et al., 2009; Sharp et al., 2014), this pattern has not been consistently observed across studies (e.g., Stringaris et al., 2015). Previous research in youth has included small samples, wide age ranges, and the inclusion of both males and females. Heterogeneity among small samples in prior studies may mask understanding of whether a specific age range (e.g., adolescents) and/or sex (e.g., female) is driving these effects (i.e., prior evidence of blunted reward responding in the striatum). Given the higher rates of MDD observed among female youth (Rohde et al., 2009), a focus on females is warranted. The present study focuses on youth 15 to 20 years old and aims to identify how the developmental onset of MDD may coincide with aberrant reward processes.

Reward Magnitude Tracking

One largely unexplored reward process in youth with MDD is reward magnitude tracking—a function whereby activity in the ventral and dorsal striatum increases or decreases to represent the value of reward quantity relative to the range of possible rewarding outcomes (Tobler, Fiorillo, & Schultz, 2005). For example, in healthy individuals, responses in the striatum increase for outcomes that are high in value magnitude relative to the other potential outcomes available, and striatal activity is relatively greater for higher than lower available rewards (Seymour & McClure, 2008). Reward magnitude tracking allows an individual to represent the relative value of an outcome in context and guides strategic motivated behavior, such as valuebased decision making and learning from rewarding feedback (Diederen, Spencer, Vestergaard, Fletcher, & Schultz, 2016).

Prior work in MDD has primarily focused on reward reactivity, which captures a global response to positive valence outcomes and may enable an individual to approach positive and avoid negative outcomes. In contrast, reward magnitude tracking captures a more nuanced and distinct valuation process, as this function allows an individual to prioritize the best outcomes available in a given situation according to a value hierarchy built on recent experience. If reward magnitude tracking is disrupted in MDD, this could have implications for motivated behavior, such as invigorating responses towards high value rewards. For example, reward magnitude tracking deficits could result in difficulty prioritizing high relative to low value outcomes (Treadway, Bossaller, Shelton, & Zald, 2012). Further, because reward magnitude tracking contributes to successful reinforcement learning (Diederen et al., 2016), aberrant magnitude tracking could hinder the ability to adaptively titrate value-driven behavior, which may attenuate the likelihood of pursuing and obtaining high value goals (Pizzagalli, Iosifescu, Hallett, Ratner, & Fava, 2009; Vrieze et al., 2013). Initial research suggests that adults with MDD have difficulty using relative reward value to guide behavior and effort allocation (Treadway et al., 2012; Zald & Treadway, 2017), but it is unknown whether youth with MDD exhibit similar differences in reward valuation. Moreover, the neurobiological bases of reward magnitude tracking remain unexplored in youth with MDD.

Notably, magnitude tracking can also be measured in the loss domain (Seymour & McClure, 2008). Prior work in healthy individuals has demonstrated that loss value is primarily coded in the striatum, cingulate, and anterior insula, and activity in these regions scales to represent the relative value of a loss outcome in context (Bartra, McGuire, & Kable, 2013).

Recent evidence suggests that adults with MDD exhibit intact loss magnitude tracking, such that adults with and without MDD display similar neural coding of loss value magnitude (Ubl et al., 2015). However, few studies have examined loss processing in youth (Luking et al., 2016). Therefore, it is currently unclear whether youth with MDD exhibit intact loss magnitude tracking. If youth with MDD express intact loss magnitude tracking in conjunction with aberrant reward magnitude tracking, this would reflect valence-specific asymmetries in neural value representation.

Characterizing potential reward magnitude tracking difficulties in youth with MDD could have important implications for contemporary models of reward blunting in the striatum among those with MDD (Zald & Treadway, 2017), as well as etiological theories of MDD in youth specifically (Davey, Yücel, & Allen, 2008; Luking et al., 2016). There is sufficient reason to hypothesize that reward processes would be different in youth depression versus adult depression. Adolescence is characterized by functional maturation of the striatum (Somerville & Casey, 2010) and emerging connectivity with the prefrontal cortex that facilitates value-driven action selection (Insel, Kastman, Glenn, & Somerville, 2017). This phase of life may provide an important window for the normative tuning of adaptive reward signals in the striatum (Hartley & Somerville, 2015), as these neurodevelopmental changes are thought to support normative increases in motivated approach behaviors (Davidow, Insel, & Somerville, 2018; Insel & Somerville, 2018). Additionally, adolescence is also a key time period for the development of depression. The prevalence of MDD increases significantly during adolescence (Kessler et al., 2007) and first episodes of major depression are most likely to occur during this developmental period (Lewinsohn, Clarke, Seeley, & Rohde, 1994). Given the importance of adolescence for the development of reward-related processes, researchers have hypothesized that the impact of

depression on reward processing, specifically striatal responses, may be even more pronounced during this developmental period (Forbes & Dahl, 2012; Luking et al., 2016).

Current Study

In this study, we examined reward magnitude tracking in MDD using a modified reward reactivity task (Delgado, Locke, Stenger, & Fiez, 2003) with a value stakes manipulation that permitted investigation of the brain-bases of distinct valuation processes (Insel & Somerville, 2018). The first major aim was to examine whether youth with MDD exhibited aberrant reward magnitude tracking, as indexed by attenuated value-selective response vigor and aberrant tracking of reward value in the striatum (i.e., failure to discriminate responses between low-magnitude and high-magnitude rewards). Behaviorally, we examined the effect of incentive stakes on response vigor to assess whether high- versus low-stakes differentially motivated behavioral responding. Neurally, we hypothesized that youth without MDD (i.e., healthy controls) would exhibit intact reward magnitude tracking in the striatum (Insel & Somerville, 2018), whereas youth with MDD would exhibit attenuated magnitude tracking of striatal responses according to reward value.

The second major aim was to identify whether youth with MDD exhibited differences specific to reward magnitude tracking, or if this pattern generalized across other forms of value representation. Specifically, we were able to isolate reward and loss magnitude tracking—a key test of specificity of valenced incentive processing aberrations in MDD. Additional analyses quantified the more classic "reward reactivity" comparison of gain versus loss processing. We also examined neural and behavioral activity during stakes context cue periods that indicated the value of the forthcoming set of high- or low-stakes trials to index the representation of motivational contexts (Insel et al., 2017). These distinct analyses allowed us to identify the

specific neurocognitive processes that are uniquely implicated in reward processing deficits in youth with MDD.

Methods

Participants and Procedure

All participants (and legal guardians if youth were minors) provided assent/consent under the protocol approved by the local Institutional Review Board. Eighty-two youth (*M* age=17.49 years, *SD*=1.37) completed this study, including 56 participants with current or past-year major depressive disorder (MDD) and 26 healthy controls (HC). The inclusion criteria were: (a) female (given the marked sex differences in neural development; Blakemore, Burnett, & Dahl, 2010), (b) 15-20 years-old (this age range was selected to be consistent with Centers for Disease Control guidelines for what is considered "youth" [CDC, 2015] and research demonstrating that neural development continues into the early 20's [Casey, Jones, & Hare, 2008]), (c) estimated IQ>80, confirmed during the lab visit (Wechsler, 2011), and (d) mid-late pubertal development confirmed during the lab visit (Petersen, Crockett, Richards, & Boxer, 1998). In addition, youth were specifically recruited for either a history of MDD (current or past-year) or for an absence of any lifetime psychiatric disorder or treatment (HC group; Table 1).

Major depressive disorder (MDD). DSM-IV major psychiatric disorders were assessed with a structured clinical interview—the Mini-International Neuropsychiatric Interview for Children and Adolescents, Child Version (MINI-Kid; Sheehan, Shytle, Milo, Janavs, & Lecrubier, 2009). The MINI-Kid has demonstrated good psychometric properties (Sheehan et al., 2009) and has been used in prior studies to validly assess major psychiatric disorders in community and outpatient samples of youth (Glenn et al., 2017; Wilkinson, Kelvin, Roberts, Dubicka, & Goodyer, 2011). Youth in the MDD group reported at least one major depressive episode in the past year. Presence of current anhedonia and number of lifetime depressive episodes were also assessed with the MINI-Kid. The Children's Depression Inventory (CDI: Kovacs, 1992) was used as a continuous measure of current depressive symptoms.¹ Youth were excluded if they had a history of a bipolar disorder (given our focus on unipolar depression), psychotic disorder, or pervasive developmental disorder (to limit severe psychiatric conditions that would be more primary than MDD). Other diagnostic comorbidities were allowed (Table 1). Psychiatric medication use was assessed following the MINI-Kid and was not an exclusion criterion (Table 1).

Healthy control (HC). Youth in the HC group had no lifetime history of a psychiatric disorder or neurological illness (assessed with the MINI-Kid [Sheehan et al., 2009] or the Schedule for Affective Disorders and Schizophrenia for School-Age Children [Kaufman et al., 1997]), and no lifetime history of psychosocial or pharmacological psychiatric treatment.

Sample matching. There were no restrictions based on handedness, but MDD and HC groups were matched on the proportion right and left handed, p=.52 (Table 1). MDD and HC groups were also balanced on gender (100% female), age, and estimated IQ. There were group differences in racial composition (p=.02; Table 1), but covariate analyses confirmed that race did not relate to the key outcome measures of interest (see Supplementary Materials).

Brain Imaging

Participants were scanned on a Siemens 3.0T Tim Trio scanner with a 32-channel head coil (see Supplementary Materials for image acquisition details).

Magnitude tracking task. Participants performed a modified reward reactivity task (Delgado et al., 2003) with low-magnitude and high-magnitude reward and loss outcomes during functional neuroimaging (Figure 1). Each block of the task consisted of a stakes cue followed by four trials. The stakes cue instructed participants whether the forthcoming trials would be paid out with high-stakes (+\$1/-50¢) or low-stakes (+20¢/-10¢) amounts. On each trial, participants viewed a card turned over with a "?" and were instructed there was a number between 1 and 9 (but not 5) on the back of the card. Participants were instructed to press a button to indicate their guess of whether the number was higher or lower than 5 (index finger for lower; middle finger for higher). In high-stakes trials, correct feedback yielded a high-magnitude reward of \$1 and incorrect feedback and missed responses incurred a high-magnitude loss of 50¢. In low-stakes trials, correct feedback yielded a low-magnitude reward of 20¢ and incorrect feedback and missed responses incurred a low-magnitude loss of 10¢. Feedback was experimentally fixed and 50% of trials were rewarded for all participants. Participants were instructed that earnings would be paid out in full; however, all participants received \$15 as task payout at the end of the study, which was equivalent to the amount a participant could earn if no missed responses occurred.

In total, the task included 24 stakes cues (12 high-stakes/12 low-stakes), 96 guess trials (48 high/48 low), and 96 feedback events (24 high win +\$1.00, 24 high loss -\$0.50, 24 low win +\$0.20, 24 low loss -\$0.10). The cue was presented for 1 second, the card presentation (card with "?") and guess response window lasted 1.5 seconds, and the feedback screen was presented for 1 second. All task events were temporally separated by jittered inter-stimulus intervals ranging from 1.5 to 3.5 seconds, and 8 seconds of fixation separated each block. Feedback presentation was pseudo-randomized within a block and ranged from 1 correct trial to 3 correct trials per block. The order of low- and high-stakes blocks was pseudo-randomized within and across runs. Square frames surrounding trial stimuli (one-line frame for low-stakes and two-line frame for high-stakes trials) differentiated task from rest and provided a constant reminder of stakes conditions to reduce working memory demands.

The task was presented using Psychopy (version 1.80) and displayed on a screen visible through a mirror on the head coil (Peirce, 2007). Behavioral responses were collected with a MRI-compatible button box.

FMRI data processing. FMRI data were evaluated for motion and signal outliers given the negative impact they can have on signal quality and GLM estimates. The following rules were imposed for exclusion of functional data. Runs in which more than 10% of TRs were censored for motion (relative motion>1mm) or outlier signal intensity (exceeded the grand run median by 4.5 median absolute deviations) were excluded from analysis. Runs with a single relative movement exceeding 5mm were also excluded. Within the MDD group, three participants had one run excluded due to excessive motion; no runs were excluded from the HC group. There was no significant group difference in number of volumes censored for motion $(M_{HC}=1.75$ volumes per run, $M_{MDD}=2.17$ volumes per run, t(80)=0.69, p=.49).

FMRI preprocessing and analysis were conducted in FSL (version 5.0.4) (Smith et al., 2004) and implemented through the Lyman pipeline (version 0.0.7), based on Nipype framework (version 0.9.2; Gorgolewski et al., 2011). Preprocessing steps included slice-time correction, realignment, coregistration of functional to structural images using bbregister (Greve & Fischl, 2009), non-linear normalization of structural to FSL's MNI152 template space using ANTS (1.9.x, svn release 891; Avants, Tustison, & Song, 2009), and spatial smoothing with a 6mm Gaussian kernel.

Data Analysis

Behavioral analysis. To assess whether incentive stakes influenced response vigor, which can be measured by the relative speeding of button presses, behavioral analyses focused on guess reaction times (RT). RT was recorded for button responses during the guess period (1.5 seconds while "?" was displayed on screen), when participants indicated whether the number on the back of a card was higher or lower than 5. RT was z-scored within participants and averaged for low-stakes and high-stakes trials. Because participants could not anticipate the forthcoming feedback, RT analyses combined trials preceding reward and loss feedback within the stakes conditions.

Further behavioral analyses were conducted to evaluate subjective reports of hedonic experience during the task. Participants provided self-reported ratings of the stimuli at the end of the task to evaluate whether HC and MDD expressed different hedonic experience of the task cues and monetary outcomes. After participants completed the scan, valence (1=unpleasant to 9=pleasant) and arousal (1=low to 9=high arousal) ratings (Lang, 1980) were collected for low-and high-stakes cues and for low- and high-magnitude reward and loss outcomes. Ratings were compared across groups, and analyses assessed the effects of feedback valence (gain/loss outcome) and feedback magnitude (low/high).

Analyses of RT, valence ratings, and arousal ratings implemented separate linear mixed effects models with fixed effects of group (between subjects: HC/MDD) and stakes (within subjects: high/low), and a random effect for subject. Post-hoc contrasts were computed via pairwise *t* tests.

FMRI analysis. Preprocessed data were submitted to a general linear model using film_gls in FSL to estimate relevant task effects. Model regressors included temporal onsets for task events (low-stakes cues, high-stakes cues, guesses, low-magnitude rewards, high-magnitude rewards, low-magnitude losses, high-magnitude losses, missed-responses), and were convolved with the canonical hemodynamic response function. Nuisance regressors included 6-parameter

motion correction values (calculated using mcflirt in FSL), censored frames for deviant signal intensity, and censored frames for excessive motion (see Supplementary Materials).

Random-effects group analyses were conducted to identify task-based changes in functional activity (see Supplementary Materials for detailed procedure). The design of the task allowed for analysis of distinct valuation-relevant processes: (1) reward magnitude tracking, (2) loss magnitude tracking, (3) reward reactivity, and (4) value context representation. Reward magnitude tracking was measured by comparing functional activity to high-magnitude versus low-magnitude rewards ($+\$1>+20\phi$). Loss magnitude tracking was measured by comparing functional activity to low-magnitude versus high-magnitude losses ($-10\phi>-50\phi$ and $-50\phi>-10\phi$). Reward reactivity was measured by comparing all rewards to all losses ($[+\$1 & +20\phi]>[-50\phi & 10\phi]$). Whole-brain activity during cue periods (indicating whether the forthcoming trials would contain high or low stakes) was used to examine whether HC and MDD similarly tracked the relative value difference between prospective low- and high-stakes rewards and losses (i.e., stakes-context representation). This analysis identified whether HC and MDD groups both registered the expected value of the low- and high-stakes contexts, and specifically whether activity in the striatum increased for high-stakes cues relative to low-stakes cues.

Multiple group-level analyses were conducted: HC only, MDD only, and HC versus MDD. For the HC versus MDD analysis, group comparisons were computed at the whole-brain level to quantify group differences. Untresholded group maps are available on NeuroVault to view and download at the following link: http://neurovault.org/collections/3960 (Gorgolewski et al., 2015). Whole-brain analyses were conducted with FLAME 1+2, as implemented in FSL. Whole-brain images were initially thresholded at z>2.3, and then underwent cluster-extent correction, which relies on Gaussian random field theory. This procedure results in whole-brain

threshold of p<.05 family-wise error (FWE) corrected. Conjunction analyses were conducted to identify overlapping regions of activation between HC and MDD whole-brain maps and were computed using the easythresh_conj function in FSL, which thresholds the joint map (FWE corrected p<.05). Cluster tables (see Supplementary Materials) present cluster peaks, statistical values, and cluster sizes for the whole brain corrected maps, reflecting a FWE p<0.05 corrected threshold.

Striatum region of interest analyses. A region of interest (ROI) was defined from the high-magnitude>low-magnitude reward HC>MDD group map (striatal peak: 22, 10, -10, 4mm sphere in the right putamen). ROI parameter-estimates were extracted from a sphere surrounding the peak of the HC>MDD the reward magnitude tracking contrast (high-magnitude>low-magnitude reward) for MDD participants only. These ROI values were submitted to further analyses to query relationships with clinical variables. Note that ROI analyses for magnitude-tracking were only implemented for independent analyses to assess individual differences and subgroup effects within the MDD group to avoid issues of statistical non-independence, as the region was defined from the HC>MDD group comparison. For reward reactivity ROI analyses, the ROI was defined from the peak voxel of the combined HC and MDD reward>loss outcome analysis (4mm sphere surrounding the peak-voxel in the right ventral striatum at X=12, Y=10, Z=-4).

Clinical heterogeneity in the MDD sample was assessed in several ways using post-hoc tests. First, we assessed relationships between striatal ROI activity and MDD recency and severity. Individuals with a current depressive disorder (MDD or dysthymia n=35) were compared to those with past-year but not current MDD (n=21). In addition, effects of current anhedonia (MINI-Kid) were examined by comparing youth in the MDD group with current

(n=16) or past anhedonia (n=40). We also examined associations between striatal activity and MDD severity, including number of lifetime MDD episodes (MINI-Kid) and current MDD symptoms (CDI), using Pearson correlations. Second, we examined the associations between striatal ROI activity and the presence of comorbid anxiety disorders or substance use disorders (MINI-Kid). Finally, we assessed the effects of psychiatric medication use (medicated n=38 vs. unmedicated n=18). Welch's *t* tests were used for all group analyses; these tests account for uneven sample sizes, resulting in adjusted degrees of freedom.

Brain-behavior analyses. Additional ROI analyses were conducted to analyze the relationship between striatal activity during high- and low-magnitude reward outcomes and guess reaction times during high- and low-stakes trials of the task. Striatal responses for lowmagnitude outcome>baseline and high-magnitude outcome>baseline contrasts were extracted from the striatal region of interest (ROI) that was defined from the high-magnitude>lowmagnitude reward HC>MDD group map (striatal peak: 22, 10, -10, 4mm sphere in the right putamen). A linear mixed effects model was computed with z-scored reaction time (subject average) as the dependent variable and the following independent variables: striatal response (low-magnitude reward>baseline and high-magnitude reward>baseline), stakes-magnitude (high/low), group (fixed effect between subjects: HC/MDD), and a random effect for subject. The model computed main effects for each factor, all 2-way interaction terms, and the 3-way interaction for striatal response by stakes-magnitude by group. The goal of this analysis was to identify whether reward outcome responses in the striatum were associated with differential reaction times for high and low stakes across groups. Post-hoc analyses were conducted by comparing model estimated marginal means for low and high z-scored reaction time within each group, as a function of striatal response.

Results

HC but not MDD increased response vigor for high-stakes

Behavioral analyses measured the effect of incentive stakes on response vigor during the guessing period of the task to assess whether high- and low-stakes differentially motivated behavioral responding in HC and MDD. There was no main effect of group (HC/MDD) on RT, F(1,80)=0.93, $\beta=0.10$, p=.33, 95%CI=[-0.03, 0.05]) indicating that HC and MDD responded comparably overall (Supplementary Table 1). The key analyses tested whether high- versus low-stakes induced different levels of response vigor for HC and MDD. There was a significant interaction between group (HC/MDD) and stakes (high/low) on z-scored RT, F(1,80)=6.48, $\beta=0.20$, p=.01, 95%CI=[0.02, 0.18]; Figure 2). HC were significantly faster for high- than low-stakes trials, t(25)=2.09, p=.05, whereas MDD exhibited no difference between stakes conditions, t(55)=0.69, p=.49. The main effect of stakes (high/low), F(1,80)=0.20, $\beta=0.04$, p=.65, 95%CI=[-0.07, 0.02]) was not significant. This suggests that motivational stakes influenced behavioral response vigor for the HC group, but not the MDD group.

HC but not MDD exhibited reward magnitude tracking in the striatum

Whole-brain analyses identified neural responses consistent with reward magnitude tracking by comparing functional activity for high-magnitude versus low-magnitude reward outcomes (+\$1>+20¢; Figure 3). The HC>MDD comparison revealed that HC, relative to MDD, recruited greater differential activity in the thalamus and striatum (spanning the ventral striatum, caudate, and putamen) for high-magnitude than low-magnitude rewards (Figure 3B; Supplementary Table 2.1). For the MDD>HC comparison, no regions survived whole-brain correction. Descriptive plots displaying activity extracted from the peak-voxel in the striatum from the HC>MDD contrast (X=22, Y-10, Z=-10, 4mm sphere in the right putamen)

demonstrate that HC tracked striatal responses according to reward stakes, but MDD exhibited similar striatal recruitment for low-magnitude and high-magnitude rewards (Figure 3C; for illustration purposes only).

To quantify group similarities in reward magnitude-tracking, separate highmagnitude>low-magnitude reward maps were computed for the HC and MDD groups (Supplementary Table 2.2-2.3, Figure 3A), and then a conjunction analysis was computed to identify areas of overlap. Both HC and MDD increased functional activity for high>lowmagnitude rewards in the bilateral cingulate (Supplementary Table 2.4).

Reward magnitude tracking and MDD heterogeneity (ROI analysis). Striatum ROI (peak voxel from high-magnitude>low-magnitude reward HC>MDD contrast at X=22, Y=10, Z=-10; 4mm sphere in the right putamen) analyses were used to examine whether aberrant striatal reward magnitude tracking (high-magnitude>low-magnitude reward) varied according to MDD heterogeneity. There was no significant difference in striatal magnitude tracking for current vs. past-year depressive disorder (t[53.94=.56, p=.58, 95%CI=[-44.41, -144.86], d=.15). Additional analyses demonstrated that reward magnitude tracking ROI activity was not associated with the presence of current anhedonia, severity of current depressive symptoms, number of lifetime depressive episodes, diagnostic comorbidities (anxiety or substance use disorders), or psychiatric medication use (Supplementary Table 3.1).

Striatal activity during high-magnitude reward outcomes was associated with enhanced high stakes speeding for HC but not MDD

To assess brain-behavior relationships, ROI analyses (Striatum ROI defined from peak voxel for the high-magnitude>low-magnitude reward HC>MDD contrast at X=22, Y=10, Z=-10; 4mm sphere in the right putamen) were conducted to assess whether reward outcome responses

in the striatum were differentially associated with average response speeding (z-scored reaction times) for high and low stakes across groups. The goal of this analysis was to interrogate how reward-outcome responses in the brain related to the behavioral finding that HC exhibited faster responses for high relative to low-stakes conditions, whereas MDD did not. To test this possibility, we examined whether there was a group (HC/MDD) by magnitude (high/low) by striatal response interaction effect on z-scored reaction time. This three-way interaction was significant (F[1,76]=5.83, p=.018, Supplementary Figure 2, Supplementary Table 9). Post-hoc analyses revealed that HC with increased striatal recruitment for high-magnitude relative to low-magnitude reward outcomes were more likely to speed up responses for high-stakes relative to low-stakes, as increased striatal activity for high-magnitude reward outcomes was associated with a tendency to slow responses during high stakes trials. These analyses suggest that striatal activity during high-magnitude reward outcomes selectively influenced response speeding in HC during high stakes conditions, but this pattern was not observed in the MDD group.

MDD and HC expressed intact reward reactivity in the striatum

To measure whether MDD exhibited blunted reward reactivity suggested by prior work, whole-brain analyses were conducted by measuring differences between reward (+\$1 and +20¢) and loss (-50¢ and -10¢) outcomes. No regions exhibited group differences in the whole-brain corrected HC>MDD and MDD>HC maps. To further probe this null group difference, the reward>loss contrast was examined separately for HC and MDD whole-brain maps (Figures 4A, 4B), which were then submitted to a conjunction analysis to identify regions of overlap. Conjunction analyses confirmed that HC and MDD exhibited comparable reward reactivity in the bilateral striatum (caudate, ventral striatum, and putamen), bilateral ventromedial prefrontal cortex, cingulate, insula, and occipital cortex (Supplementary Tables 4.1-4.2). Descriptive plots from a 4mm sphere surrounding the peak-voxel in the right ventral striatum at X=12, Y=10, Z=-4 (Figure 4C, for illustration purposes only) suggest that both HC and MDD recruited greater striatal activation for rewards than losses. Therefore, both HC and MDD exhibited comparable levels of reward reactivity in the striatum.

To further interrogate the reward reactivity effect in the striatum, we examined how highmagnitude and low-magnitude reward and loss responses contributed to the contrast of reward versus loss. To visualize each condition separately, we plotted ROI activity in the ventral striatum (4mm sphere surrounding the peak-voxel in the right ventral striatum at X=12, Y=10, Z=-4) for low loss, high loss, low reward, and high reward for the HC and MDD groups (Figure 5). Visual inspection reveals that the HC group shows a strong increase for high-magnitude relative to low-magnitude rewards. In contrast, the MDD group shows comparable responses to low- and high-magnitude rewards. However, overall reward responses in MDD are not blunted (i.e., low- and high-magnitude reward responses are above zero), which demonstrates the comparable reward reactivity responses for HC and MDD. There was also no significant difference in ventral striatal response between groups for low-magnitude reward outcomes (t[47.77]=1.45, p=.15) or for high-magnitude reward outcomes (t[44.20]=-0.42, p=.68). Therefore, the reward reactivity effect is not driven by responses to either the low-magnitude or high-magnitude reward outcomes alone. Further, the reward reactivity effect is not driven by differential loss processing between the HC and MDD groups.

Reward reactivity and MDD heterogeneity (ROI analysis). Further analyses were conducted to assess differences in ventral striatal reactivity between the HC group and individuals within the broader MDD sample with a current depressive disorder (n=33; ROI

analysis from 4mm sphere in right ventral striatum at X=12, Y=10, Z=-4). Right ventral striatum reward reactivity also did not differ between HC and individuals with current depression, t(48.41)=.23, p=.82. Reward reactivity in the right ventral striatum was also not related to current anhedonia, number of lifetime MDD episodes, comorbid anxiety or substance use disorders, or psychiatric medication use (Supplementary Table 3.2). However, reward reactivity in the ventral striatum ROI was negatively associated with current depression symptom severity (CDI), r(54)=.32, p=.02, 95%CI=[-0.53, -0.09]. This suggests that, among the MDD group, individuals with greater depression severity exhibited decreased reward reactivity in the striatum.

MDD and HC exhibited comparable tracking of loss magnitude

Whole-brain analyses identified neural regions tracking loss magnitude by comparing functional activity for low-magnitude versus high-magnitude loss outcomes (-10ϕ >-50 ϕ contrast and -50ϕ >-10 ϕ contrast). When comparing HC to MDD, no regions survived whole brain correction for either loss contrast, suggesting that HC and MDD did not differ in loss magnitude tracking activity at the whole-brain level. Loss magnitude tracking was quantified separately for HC and MDD groups (Supplementary Tables 5.1-5.4), and conjunction analyses were performed to identify regions of overlap between HC and MDD. A whole-brain conjunction analysis for the -10ϕ >-50 ϕ contrast revealed significant overlap between HC and MDD recruitment in the precuneus extending into the insula/operculum (5554 voxels with peak at X=-4, Y=-56, Z=22; Supplementary Table 5.5). For the -50ϕ >-10 ϕ contrast, there were no regions of significant overlap.

MDD and HC similarly coded stakes-value context cues in the striatum

One possible explanation why MDD exhibited aberrant reward magnitude tracking in the striatum could be that the MDD group did not represent the relative value of the stakes contexts.

To assess whether MDD discriminated between high and low value contexts, analyses compared group activity for the stakes cues, which signaled upcoming high- or low-stakes trials. When comparing HC and MDD for the high-stakes>low-stakes cue contrast, no regions survived whole-brain correction, suggesting that HC and MDD exhibited comparable levels of recruitment for high-stakes versus low-stakes cues. A conjunction analysis revealed regions recruited by both groups, demonstrating significant overlap in the bilateral ventral striatum and caudate (1286 voxels at X=10, Y=12, Z=2, see Supplementary Figure 1). That is, both HC and MDD increased striatal recruitment for high-stakes relative to low-stakes cues, confirming that MDD exhibited intact stakes-context value representation. This suggests that aberrant magnitude tracking in MDD is specific to the moment of receiving reward outcomes and is not an artifact of aberrant neural value-context representation.

MDD and HC self-reported comparable hedonic experiences for task stimuli and outcomes

To characterize hedonic experience associated with the task stimuli and monetary outcomes, participants rated the valence and arousal of stakes cues, reward outcomes, and loss outcomes (Supplementary Materials for complete results). For cue ratings (signaling the upcoming stakes value), reward outcomes, and loss outcomes, there was a significant main effect of stakes (high/low) across groups (*p*s<.01). This suggests that MDD and HC rated the high-stakes cues and high-magnitude reward outcomes as significantly higher in arousal and pleasant valence than the low-magnitude cues and low-magnitude reward outcomes. MDD and HC also rated high-magnitude loss outcomes to be significantly higher in arousal and unpleasant valence than low-magnitude loss outcomes. Importantly, there were no significant main effects of group or group by stakes interactions. This suggests that the HC and MDD groups reported comparable hedonic experiences for the stakes cues and monetary outcomes.

Discussion

The current study examined whether relative reward value modulated brain and behavioral responses in youth with depression. Behavioral results demonstrated that healthy youth adjusted reaction times according to stakes value, such that they were faster to respond when high-value (vs. low-value) rewards and losses were at stake. Conversely, youth with current or past-year depression responded similarly in high- and low-stakes conditions. These behavioral effects could not be explained by differences in hedonic experience, as healthy and depressed youth similarly experienced the low- and high-stakes context cues, as well as lowmagnitude and high-magnitude reward and loss outcomes. Neuroimaging results revealed that whereas healthy participants increased striatal responses for high-relative to low-value reward outcomes (consistent with reward magnitude tracking), youth with depression exhibited similar striatal activity for low and high rewards (i.e., aberrant reward magnitude tracking). Group differences in reward magnitude tracking were paralleled by brain-behavior relationships, such that high-magnitude reward outcome responses were associated with faster high-stakes responses in healthy controls, but not in youth with depression. Importantly, we did not find evidence that striatal magnitude tracking was related to depression symptom severity or recency, diagnostic comorbidity, or psychiatric medication use. This pattern of aberrant magnitude tracking reflects a valence-selective valuation process, as these group differences were specific to reward, but not loss outcomes. At the group level, we observed similar reward reactivity (reward versus loss response) in the striatum for healthy and depressed youth. However, this effect was modified by depression symptom severity within the depression group, such that individuals with more severe current depression symptoms exhibited reduced reward reactivity responses. Taken together, this study suggests that while reward reactivity may vary with current depression severity, reward

magnitude tracking may represent an important aberrant valuation process in depression independent of symptom severity and recency (i.e., state-independent). As such, this specific valuation aberration could constrain how value guides adaptive behavior in youth with depression.

Behavioral results revealed that youth with depression are less likely to translate reward value into motivated action. We found that healthy youth enhanced response vigor for highstakes, a behavioral strategy that has been shown to adaptively guide successful reward learning and capture of high value (O'Doherty, Buchanan, Seymour, & Dolan, 2006). In contrast, youth with current and past-year depression did not alter behavioral speeding for high-stakes. Importantly, these findings could not be explained by differences in hedonic experience, as depressed and healthy youth reported comparable valence and arousal ratings for the high-stakes cues, rewards, and losses. Taken together, the stimuli ratings and behavioral results reveal that youth with and without depression similarly value the prospect and receipt of high-stakes rewards (hedonic experience), but youth with depression do not convert this stakes-valuation into enhanced motivated responses (behavioral speeding), which could reflect underlying difficulty orchestrating adaptive motivated approach behavior.

Neuroimaging results focused on reward magnitude tracking, a process whereby striatal activity increases for high relative to low reward outcomes to adaptively represent relative reward value. Results revealed that healthy youth scaled striatal responses according to reward magnitude, whereas youth with depression expressed similar striatal responses for low and high rewards. Group differences in reward magnitude tracking between healthy and depressed youth were localized to the dorsal striatum. Although there were group differences in reward magnitude tracking found in the striatum, youth with depression did exhibit comparable reward

magnitude tracking to healthy youth in the cingulate, a region implicated in affective valuation (Bartra et al., 2013). However, magnitude tracking in the striatum has been specifically linked to adaptive reward approach behavior (Tobler et al., 2005), and therefore group differences in the striatum are particularly notable. Future work should examine whether aberrant reward magnitude tracking in the striatum contributes to motivational deficits in depression.

Notably, for high- relative to low-stakes conditions, increased dorsal striatal activity during high- relative to low-magnitude reward outcomes was associated with enhanced highstakes speeding for the healthy controls. In contrast, the depression group exhibited the opposite pattern such that enhanced dorsal striatal activity was associated with a tendency to slow response times for high stakes relative to low stakes. These findings suggest that high-magnitude reward outcome responses in the striatum may selectively invigorate behavior for healthy individuals but not for youth with depression. One possible interpretation, and focus for future research, is that individuals with MDD are less likely to integrate value signals with motor demands to guide motivated coordination of motor output.

Although the depression group showed aberrant magnitude tracking in the dorsal striatum, youth with depression exhibited intact stakes representation in the ventral striatum during the cue period, which signaled the value of the upcoming trials. Specifically, both healthy and depressed youth similarly increased recruitment for high-stakes cues, suggesting that the stakes context representation was intact. However, while the MDD group did express value-selective coding in the ventral striatum for the stakes cue when anticipating a forthcoming block of high-stakes trials, this value signal did not translate into value-guided speeding during high-stakes responses.

The findings that both groups demonstrated value-coding in the ventral striatum during the stakes cue, but only the healthy control group expressed magnitude-tracking in the dorsal striatum during reward outcome suggests that aberrant valuation processes in youth with depression may be specific to value-based outcome representation. Further, these neurocognitive dissociations may reflect distinct functional contributions of ventral and dorsal sub-regions within striatum. These local anatomical distinctions in group effects within the ventral and dorsal striatum are of particular interest given that striatal subdivisions are thought to be functionally dissociable (Haber & Knutson, 2010). Striatal subregions are thought to subserve distinct cognitive processes, as the ventral and dorsal striatum interact with different cortical regions via looped connections within distributed corticostriatal circuits (Haber & Knutson, 2010). The ventral striatum has been shown to process reward valuation, whereas the dorsal striatum has been implicated in motivated action selection (Burton, Nakamura, & Roesch, 2015; Haber & Knutson, 2010). Ventral regions are thought to communicate value-related information to dorsal regions via indirect loops to coordinate goal-directed behavior (Haber, Fudge, & McFarland, 2000). Here, we found that individuals with depression exhibited blunted reward magnitude tracking in the dorsal striatum, and dorsal striatal activity has been shown to subserve the translation of motivational value into motoric approach responses (Burton et al., 2015; Wang, Miura, & Uchida, 2013). Notably, we also demonstrate that for the healthy control group, highrelative to low-magnitude increases in dorsal striatal activity were associated with enhanced high-stakes speeding, highlighting the connection between value-selective dorsal striatal function and motivated motor approach behavior. Future work should examine how distinct striatal subdivisions contribute to specific valuation differences in depression, and how aberrant striatal function may impact value-guided action selection.

Together, these findings suggest that youth with depression may not incorporate reward outcome history and outcome-related striatal signals to dynamically update their behavior. One limitation of the current study is that participants were not required to adaptively update and speed up responses to receive beneficial outcomes. Therefore, the present research cannot delineate whether the differences observed in the MDD group reflect effortful updating computations (Treadway et al., 2012) or conditioned response associations, and future work should assess how shifting cognitive demands may influence dynamic changes in goal-directed behavior. In addition, the current paradigm included only two reward levels, which precluded an examination of more complex and dynamic value coding patterns in the striatum; this is an important direction for future research on context-dependent valuation.

The current results suggest that aberrant reward magnitude tracking may be a stateindependent feature given that this functional profile was detected in youth with current and pastyear MDD, independent of depression severity and recency, diagnostic comorbidity, or psychiatric medication use. In contrast, reward reactivity analyses revealed that youth with current or past-year depression exhibited comparable reward reactivity (rewards>losses) to healthy youth. However, this effect was modified by current depression severity. Specifically, among youth with a history of MDD, current depression symptom severity was inversely associated with reward reactivity in the striatum, such that those with more severe depression exhibited less striatal reward reactivity. These results converge with earlier studies indicating that depression symptom severity correlates with blunted reward reactivity in youth (e.g., Forbes et al., 2010) and adults (e.g., Pizzagalli, Holmes, et al., 2009).

Given that reward reactivity varied according to current depression symptom severity, whereas reward magnitude tracking was consistent across a heterogeneous sample of youth with depression, these distinct valuation components may capture temporally distinct aspects of depression (i.e., current depression symptom severity vs. past-year depression diagnosis). Although we were able to examine reward dysfunction based on depression severity and recency, the current study, due to the cross-sectional design, cannot elucidate whether aberrant reward magnitude tracking precedes the onset of depression or predicts the time course of depressive symptoms. The precise temporal development of reward magnitude tracking as it relates to depression during adolescence is an important area for future research. In addition, it is important to note potential limits of generalizability given that our sample focused on females only within a specific age range (15-20 years old). Future research is needed in males as well as wider age ranges to examine these reward-related processes across development.

In sum, these findings suggest that youth with depression exhibit aberrant reward magnitude tracking regardless of depression recency and severity, whereas blunted reward reactivity varies according to current depression severity. Aberrant reward magnitude tracking may contribute to the motivational deficits observed in depression, as reward magnitude tracking is important for prioritizing high-value outcomes and strategically approaching optimal situations (Diederen et al., 2016). In other words, when faced with varying magnitudes of rewards, youth with depression may not titrate performance according to the value of the outcome and may have difficulty orienting attention and allocating resources towards high-value goals. Moreover, disruptions in adaptive reward functioning during adolescence could be particularly pernicious given how important this developmental period is for the refinement of adaptive reward learning (Hartley & Somerville, 2015). Moving beyond prior research focused on blunted reward reactivity in depression, the current findings highlight the need to isolate specific reward valuation deficits underlying reward dysfunction in youth with depression.

Footnote

¹ The same measures were administered to all participants in the MDD sample to ensure consistency and allow for continuous comparison across the full sample.

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Table 1

	Healthy Control (n=26)	Major Depressive Disorder (n=56)	Statistical Test ^a	р	ES ^b
Demographics					
Age: $M(SD)$	17.23 (1.73)	17.61 (1.17)	t(36.05)=1.01	.32	<i>d</i> =.26
Handedness: % right handed	92.3	87.5	$\chi^2(1, N=82)=0.42$.52	φ=.07
Race/Ethnicity: %			$\chi^2(1, N=82)=11.84$.02	φ=.38
White/Caucasian	30.8	62.5			
Black/African American	19.2	8.9			
Asian	30.8	7.1			
Hispanic	7.7	7.1			
Other/Multiracial	11.5	14.3			
WASI IQ: M (SD) ^c	105.15	104.89	t(45.49)=0.09	.93	<i>d</i> =.02
	(12.97)	(11.98)			
Socioeconomic status ^d					
Highest level education–Mother: %	<i>n</i> =12	<i>n</i> =49	$\chi^2(1, N=61)=2.74$.43	φ=.21
Less than high school	0	2.0			
High school diploma or GED	0	16.3			
Some college	25.0	16.3			
4-year college or more	75.0	65.3			
Highest level education–Father: %	<i>n</i> =11	<i>n</i> =48	$\chi^2(1, N=59)=.70$.87	φ=.11
Less than high school	0	4.2			
High school diploma or GED	18.2	22.9			
Some college	9.1	6.3			
4-year college or more	72.7	66.7			
Psychiatric diagnoses, symptoms, o	and medication				
Major diagnoses: $n(\%)^{e}$			N/A		
Depressive Disorder	0	56 (100%)			
Anxiety Disorder	0	45 (80.4%)			
ADHD	0	2 (3.6%)			
Behavior Disorder	0	6 (10.7%)			
Bulimia Nervosa	0	5 (8.9%)			
OCD	0	5 (8.9%)			
PTSD	0	8 (14.3%)			
SUD	0	20 (35.7%)			
Number of major diagnostic	0	2.64 (1.09)	N/A		

Major sociodemographic and psychiatric characteristics of the Healthy Control and Major Depressive Disorder groups (all female).

categories: M (SD)				
Depressive symptoms (CDI): M	N/A	8.39 (4.85)	N/A	
(SD)				
Psychiatric medications (any):	0	38 (67.9%)	N/A	
$n(\%)^{\mathrm{f}}$				
Antidepressants	0	34 (60.7%)		
Anxiolytics	0	6 (10.7%)		
Anticonvulsants	0	6 (10.7%)		
Antipsychotics	0	3 (5.3%)		
Stimulants	0	4 (7.1%)		

CDI=Children's Depression Inventory; ES=effect size

^a Welch's *t* tests were computed to account for uneven sample sizes, resulting in adjusted degrees of freedom.

^b Effect sizes (ES) reported as either Cohen's *d* for *t* tests or phi coefficient (φ) for chi-square analyses.

^c Two subtests (matrix reasoning and similarities) of the Wechsler Abbreviated Scale of Intelligence–Second Edition (WASI-II) were used to approximate full scale IQ (Wechsler, 2011).

^d Socioeconomic status (SES) was approximated with parental education. Sample sizes are listed for SES data as not all information was available for all participants.

^e Anxiety Disorder includes current panic disorder, agoraphobia, social phobia, specific phobia, or generalized anxiety disorder; ADHD=Attention-Deficit Hyperactivity Disorder; Behavior Disorder includes current conduct disorder or oppositional defiant disorder; Depressive Disorder includes current major depressive disorder or dysthymia; OCD=Obsessive Compulsive Disorder; PTSD=Posttraumatic Stress Disorder; SUD=Substance Use Disorder includes current alcohol or substance abuse/dependence.

^f Specific medication information is available in Supplementary Table 7.

Figure Captions

Figure 1. Task overview. Participants viewed a low-stakes or high-stakes cue followed by four guess trials (card with ?), when participants guessed whether the number on the back of a card was higher or lower than five. Each guess was followed by win or loss feedback. Low-stakes correct guesses received a low-magnitude reward of $+20\phi$; incorrect guesses and missed responses incurred a low-magnitude loss of -10ϕ . High-stakes correct guesses received a high-magnitude reward of +\$1.00; incorrect guesses and missed responses incurred a high-magnitude loss of -50ϕ .

Figure 2. Reaction time by group. Z-scored guess reaction time was calculated to compare response vigor on low- and high-stakes blocks between Healthy Control and Major Depressive Disorder groups (y-axis depicts averaged z-scores). (*Note*. As participants did not know during the guess phase whether a trial would result in reward or loss, reaction times were examined for all trials regardless of whether they led to reward or loss.)

Figure 3. Magnitude tracking: High-magnitude versus low-magnitude reward responses.

(A) Representative slices from whole-brain corrected statistical maps (FWE p<0.05) illustrating enhanced functional recruitment for high-magnitude relative to low-magnitude reward outcomes (+\$1 vs. +20¢). The map for the Healthy Control (HC) group is displayed on the left, and the map for the Major Depressive Disorder (MDD) group is displayed on the right (no striatal activity present). (B) Representative slices from whole-brain corrected group comparison map illustrating regions exhibiting larger high-magnitude>low-magnitude reward differences in HC compared to MDD. (C) Extracted parameter estimates for the high->low-magnitude reward response from a striatum region of interest (4mm sphere in right putamen at 22,10,-10) are displayed to illustrate descriptive trends. (Plot for visualization purposes only and not intended for statistical inference.)

Figure 4. Reward reactivity: Reward versus loss responses. (A) Representative slices from whole brain corrected statistical maps for the Healthy Control group illustrating enhanced functional recruitment for rewards (+\$1 and +\$0.20) versus losses (-\$0.50 and -\$0.10). (B) Reward versus Loss contrast for the Major Depressive Disorder group. There were no group differences for this contrast. (C) Extracted parameter estimates for the Reward>Loss response from a striatum region of interest (4mm sphere in right ventral striatum at 12,10,-4) are displayed to illustrate descriptive trends. (Plot for visualization purposes only and not intended for statistical inference.)

Figure 5. Reward reactivity by outcome magnitude responses. Extracted parameter estimates for the high-magnitude>baseline and low-magnitude>baseline reward and loss responses from the ventral striatum (4mm at X=12, Y=10, Z=-4 defined by peak in reward>loss contrast across groups). Plot is displayed for visualization purposes to illustrate descriptive trends.

Figure	1



Figure 2





Reward Reactivity: Reward vs. Loss Outcome (\$1 & 20¢) > (-50¢ & -10¢)



