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Dampening Positive Affect and Neural Reward Responding in Healthy Children: Implications for Affective Inflexibility

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Blunted reward processing is evident in and may contribute to the onset of major depressive disorder. However, it is unclear what mechanisms contribute to the development of blunted reward-response prior to depression onset. The current study examined how individual differences in the tendency to dampen positive affect, an affect regulation strategy that decreases positive affect, are associated with reward responding and related brain activation in 39 healthy children (ages 7–10; 51% female; 79% White). To do this, we examined neural responses to winning a reward (candy) within the context of a previous loss, win, or neutral outcome. Whole-brain regression analyses revealed that self-reported tendencies to engage in dampening were associated with blunted striatum and thalamic activation during a winning outcome when following a previous loss outcome, as compared to when following a neutral outcome. This finding was above and beyond the influence of current depressive symptoms. However, tendencies to dampen positive affect were not associated with neural activity during the second of 2 consecutive win outcomes, and thus did not support the notion that dampening is associated with an inability to maintain reward responding. In youth, tendencies to dampen positive affect may be associated with less ability to flexibly upregulate neural reward responding following a loss, possibly leading to the development of affective inflexibility and increased vulnerability to depression. Dampening positive affect may be one mechanism that contributes to aberrant neural reward responding via affective inflexibility and may be a target for prevention in youth.

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INTRODUCTION

The experience of positive affect is associated with many beneficial outcomes, including longevity (Danner, Snowdon, & Friesen, 2001), better physical health (Pressman & Cohen, 2005), better mental health (Fredrickson, 1998, 2001), and greater resiliency (e.g., Fredrickson, Tugade, Waugh, & Larkin, 2003). Conversely, low positive affect and anhedonia, or the reduced experience of pleasure or interest in positive stimuli previously found to be rewarding, is a cardinal symptom of depression (American Psychiatric Association, 2013). Reward-related processing deficits are relatedly a central component of major depressive disorder (for review, see Barch, Pagliaccio, & Luking, 2015), and research has repeatedly demonstrated that diminished neural reward responding in the striatum is characteristic of adults (e.g., Pizzagalli, Iosifescu, Hallett, Ratner, & Fava, 2008) and adolescents with depression (e.g., Forbes et al., 2009) and of children at risk for depression (e.g., Luking, Pagliaccio, Luby, & Barch, 2016a; Sharp et al., 2014). However, we know little about the factors contributing to this reduction in reward-related brain activation. Uncovering these contributing mechanisms might shed light on risk factors for depression that could be targeted for preventive intervention. The current study examined whether a specific affect regulation strategy, namely, the tendency to dampen one's positive affect, is associated with neural reward responding in children who have not yet experienced depression.

Behavioral research posits that positive affective functioning goes awry in depression in two central ways. First, depression is associated with difficulties shifting away from negative emotions toward positive affective experience (Kovacs & Yaroslavsky, 2014; Tomarken & Keener, 1998). Second, depression is associated with an inability to sustain or maintain positive affect during a positive affective experience (e.g., Davidson, 2015). Regarding the first form of positive affect disturbance in depression (i.e., contextual difficulties), theories suggest that difficulties shifting from negative to positive emotional experience may be a component of emotional inertia (a tendency to resist change from the current emotional state; Kuppens, Allen, & Sheeber, 2010; Kuppens et al., 2012) and emotion context insensitivity (reduced reactivity to positive and negative emotional contexts and related affective inflexibility; Rottenberg, Gross, & Gotlib, 2005). Behavioral research supports these complementary theories showing that following a conflict, depressed adolescents and their mothers have difficulty disengaging from negative affective states toward positive states (McMakin et al., 2011) and that an inability to flexibly shift from a negative to a positive emotional interaction is associated with poorer adjustment and less treatment improvement in children (Granic, O'Hara, Pepler, & Lewis, 2007). Moreover, depressed youth demonstrate slower attentional shifting from negative to positively valenced words (Maalouf et al., 2012). Neural findings similarly demonstrate that when instructed to think of a positive memory following a negative mood induction, low-risk youth demonstrated increased prefrontal activation associated with regulation of emotion, whereas youth at high risk for depression show increased activation in the amygdala (Joormann, Cooney, Henry, & Gotlib, 2012). This may indicate difficulty disengaging from the previous negative mood state and fully engaging with the positive memory, especially among the high-risk youth.

Regarding the second form of positive affective disturbance (i.e., temporal difficulties), depression is also characterized by difficulties maintaining positive affective experience. Specifically, depression is associated with a shorter subjective experience of positive affect following positive film clips (McMakin, Santiago, & Shirk, 2009) and shorter positive emotional persistence following a rewarding parent–child task (Fussner, Luebbe, & Bell, 2014). Of interest, in both of these studies, initial positive affective reactivity was not associated with depression, demonstrating that deficits might be more related to difficulty maintaining positive affective responses over time than in initial reactivity. Initial evidence in neuroimaging studies parallels behavioral findings: A shorter duration of ventral striatal response to positive stimuli is associated with depressive symptoms in adults (Heller et al., 2009). In addition, depressed adults who demonstrate the largest improvement in sustained striatal response after treatment also demonstrate the largest improvement in positive affect (Heller et al., 2013). Research with youth demonstrates similar findings. For example, youth with depression exhibit decreased dorsal striatal response to anticipation of reward following a previous win, suggesting difficulties sustaining reward responding across multiple rewarding trials (Olino et al., 2011). Last, research demonstrates that adults who have *increased* abilities to sustain striatal responses demonstrate more adaptive outcomes. Specifically, sustained ventral striatal activity across trials to positive stimuli is associated with increased subjective day-to-day experience of positive affect (Heller et al., 2015), and it predicts higher eudemonic well-being in adults (Heller et al., 2013), suggesting that improving sustained response to reward may be a therapeutic target.

The contextual and temporal components of positive affective functioning in depression are central to the disorder and associated neurobiological functioning. Given that positive affective disturbance associated with mood pathology is characterized by (a) an inability to upregulate positive affect in the context of negative affective states and (b) an inability to sustain positive affect over time, specific tendencies to respond and regulate emotions may be contributing to this aberrant processing. It should be noted that not all affect regulation strategies are adaptive or deliberate, and in fact many strategies are automatic tendencies to either upregulate or downregulate affect (e.g., Gyurak, Gross, & Etkin, 2011; Koole & Rothermund, 2011). Dampening positive

affect is one such strategy that has been implicated in depression. Dampening positive affect is defined as the "tendency to respond to positive mood states with mental strategies to reduce the intensity and duration of the positive mood state" (Feldman, Joormann, & Johnson, 2008, p. 509). Dampening takes a "glass half empty" view of positive affect involving thoughts such as "These feelings won't last" or "I don't deserve this." Dampening is elevated in adult and youth community samples experiencing current depressive symptoms (Feldman et al., 2008; Nelis, Holmes, Palmieri, Bellelli, & Raes, 2015; Raes, Daems, Feldman, Johnson, & Van Gucht, 2009) and in depressed and remitted depressed adult patients (Nelis, Holmes, & Raes, 2015; Werner-Seidler, Banks, Dunn, & Moulds, 2013). However, findings are inconclusive as to whether dampening is a risk factor for depression given mixed findings related to the onset of depression in children and adults (Bijttebier, Raes, Vasey, & Feldman, 2012; Gilbert, Gruber, & Nolen-Hoeksema, 2013; Nelis et al., 2015; Raes, Smets, Nelis, & Schoofs, 2012). It is also currently unknown whether dampening may also be associated with variations in neural responses to reward experiences.

The aim of the current study was to examine whether the tendency to dampen positive affect in healthy children (some of which are at high risk for depression) might be associated with the types of aberrant neural reward responses that are often implicated in depression. As just noted, recent work has emphasized the impaired contextual (e.g., flexibility difficulties in the context of previous negative experiences; McMakin et al., 2011) and temporal (e.g., sustainment of positive affect; Davidson, 2015; Heller et al., 2015; Olino et al., 2011) components of positive affective functioning in depression. Thus, we examined how children's self-reported tendencies to dampen positive affect were associated with neural reward responding depending on differing contextual and temporal features.

Specifically, we examined two hypotheses regarding the relationship between dampening and neural response to reward following either a previous loss or a previous win. First, we hypothesized that a greater tendency to dampen one's positive affect would be associated with blunted striatal responding to wins following losses as compared to wins following neutral outcomes, possibly due to an inability to flexibly shift from a negative to positive experience. Second, we hypothesized that a greater tendency to dampen one's positive affect would be associated with an inability to sustain striatal response to reward after a previous win as compared to wins that followed neutral outcomes. These two noncompeting hypotheses afforded us an examination of two independent ways that positive affective functioning goes awry by examining how dampening tendencies might contribute to these processes. Embedded within these hypotheses was also the notion that higher tendencies to engage in dampening do not necessarily equate an explicit desire to dampen one's positive affect. To the contrary, we

hypothesized that akin to how rumination can become automatic when experiencing negative affect (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008), and akin to how emotion regulation is often engaged in nonconsciously and automatically (Gyurak et al., 2011), we hypothesized that tendencies to dampen positive affect are often automatic and thus would not engage in top-down prefrontal regions. We examined these hypotheses in psychiatrically healthy children who were either at low risk or high risk for future depression by virtue of having a mother with depression in order to examine how these processes may emerge prior to and may confer risk for onset of depression. Children 7–10 years of age were included so as to examine this possible risk factor prior to the onset of puberty and adolescence, when incidence rates of depression drastically increase (Kessler, Chiu, Demler, & Walters, 2005)

METHODS

Participants

One hundred thirty mothers and their 7- to 10-year-old children were recruited for a study examining children at risk for depression based on a maternal history of depression using flyers and brochures distributed in schools in the urban St. Louis, Missouri, community as well as via the Research Participant Registry at Washington University School of Medicine. Mothers first completed a phone screen to determine eligibility. Eligible mothers and children then completed multiple in-person sessions, including a neuroimaging scan. Prior to participation, mothers provided informed written consent and children provided written assent. All procedures were preapproved by the Washington University in St. Louis Institutional Review Board.

Children were eligible if they were psychiatrically healthy as assessed by both mother and child report using the Kiddie-Structured Assessment for Affective Disorders–Present and Lifetime Version (Kaufman et al., 1997). Master's-level clinicians trained to reliability administered all interviews. Exclusion criteria included any current or past psychiatric disorders, current or past psychotropic medication use, older than 7–10 years of age, menarche in female participants, prohibition of candy, gestational age greater than 35 weeks, any learning or major medical disorder, or prenatal exposure to alcohol or illegal drugs (via maternal report).

Mothers were eligible for the larger study if they had no previous history of any Axis I disorder (low-risk status for their child) or they had experienced at least one past depressive episode (high-risk status for their child). Diagnostic history was assessed with the Structured Clinical Interview for *DSM* Disorders (First, Spitzer, Gibbon, & Williams, 2007).

Seventy mother–child pairs (high-risk $n = 26$) met all inclusion criteria and were invited to participate in the neuroimaging session. Of these children, 39 provided sufficient high-quality data across all three runs of the task (high-risk $n = 13$) and are

included in the current analyses. Inclusion for this analysis was more stringent than prior report (Luking et al., 2016a) by requiring complete imaging data on all three runs of the task to ensure a sufficient number of trials in each context condition (gain post gain [GpostG], gain post loss [GpostL], and gain post neutral [GpostN]). There were no risk-group differences on the ratio of children excluded from the current analyses (*p* > .05).

Measures

Dampening Positive Affect

Children's tendency to dampen positive affect was assessed via the child-reported Dampening subscale of the Response to Positive Affect questionnaire (Feldman et al., 2008). The Dampening subscale is an eight-item self-report that asks participants to rate how much they engage in dampening responses (e.g., think *I don't deserve this*) when they are feeling happy, excited, or enthused on a 1 (*almost never*) to 4 (*almost always*) scale. Good internal consistency was obtained in the current sample (α = 0.79).

Depressive Symptoms

Child-reported depressive symptoms were assessed using the Child Depression Inventory–Child Version (CDI-C; Kovacs, 1985). The CDI-C is a 27-item self-report measure that assesses depressive symptoms on a 3-point scale from 0 (e.g., *I have fun in many things*) to 2 (e.g., *nothing is fun at all)*. Age/gender normalized *t* scores were used in current analyses.

Procedure and Task

Diagnostic interviews, demographic information, and self-reported dampening were assessed in a first behavioral session. At the neuroimaging session (*M* = 26.31 days later, *SD* = 35.86), children completed a self-report of depressive symptoms, training and practice trials of the card-guessing game prior to entering the scanner, and the in-scanner task. Children were compensated with a $20 gift card for completing each session (behavioral and neuroimaging session) and candy they won from the card-guessing reward task.

Card-Guessing Game

Each child played a child-friendly card-guessing game, modified from a validated adult version (Figure 1; Delgado, Nystrom, Fissell, Noll, & Fiez, 2000) that has been previously used with adults and children (Barch et al., 2013; Luking & Barch, 2013) and from which behavioral and neuroimaging data from the current sample have been previously published (Luking, Luby, & Barch, 2014; Luking et al., 2016a). In each trial of the game, children viewed a mystery card with a question mark (?) on it and then guessed whether the number on the mystery card was more or less than 5. Candy was won for correct guesses, lost for incorrect guesses, and neither won nor lost if the card number was 5. Mystery card values ranged from 1 to 9, and if no guess was made after 2,000 ms, the card was replaced by a fixation cross for the remaining 2,000 ms of that trial. If a response was made, feedback was displayed immediately for 2,000 ms displaying the number of the card and written feedback stating "Great job!" "Sorry," or "Next Trial." Participants chose to play for Skittles or M&M's, and a 2:1 ratio of gain-to-loss amounts (i.e., either winning four candy pieces or losing two candy pieces) were predetermined to maintain engagement, prevent frustration, and ensure that net candy was won. The task was presented in a fixed pseudorandom order with a rapid event-related design using E-Prime 2.0 (Psychology Software Tools, Pittsburgh, PA) and consisted of three blocks of trials (each lasting approximately 5 min) with 117 trials across blocks (39 of which were gain) and 13 instances of each of the three trial types within each block, jittered with an intertrial interval of 2, 4, or 6 s. Pertinent for the current analyses, no more than three instances of a given feedback type occurred in a row and win feedback followed a win, neutral, and loss feedback with equal frequency, resulting in 13 instances of each of the three context trial types.

fMRI Data Processing and Analyses

Data Processing

The fMRI data were collected using the 3 T Siemens Connectome scanner at Washington University in St. Louis and a 32-channel head coil using sequences developed for the
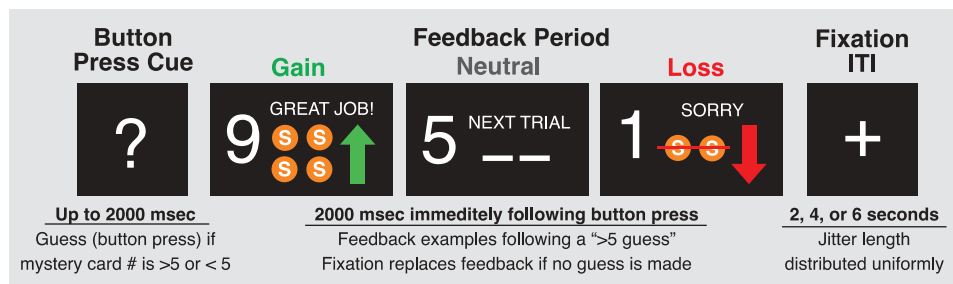

FIGURE 1   Card-guessing game trial examples of gain, neutral, and loss conditions (Luking et al., 2016a). © American Academy of Child and Adolescent Psychiatry. Reproduced by permission of Elsevier and American Academy of Child and Adolescent Psychiatry. Permission to reuse must be obtained from the rightsholder.

Human Connectome Project (Barch et al., 2013). Functional images were collected using a T2*-weighted gradient-echo-planar (EPI) BOLD sequence (repetition time = 775 ms, time to echo = 36.2 ms, flip angle = 52°, matrix = 102 × 102, field of view = 204 × 204 mm, 72 slices, voxel size = 2 × 2 × 2 mm). T1- and T2-weighted images were also collected for registration purposes using a MPRAGE and a 3D SPACE acquisition, respectively. Data were preprocessed using the Human Connectome Project minimal preprocessing pipeline (Glasser et al., 2013), and gradient unwarping, head motion corrections, field map-based EPI distortion correction, brain-boundary-based registration of EPI to structural T1-weighted scan, non-linear (FNIRT) registration into MNI152 space, and grand-mean intensity normalization were applied (Siegel et al., 2014). Previously validated "motion scrubbing" was applied (Siegel et al., 2014) to remove frames that exceeded 0.35 mm frame-wise displacement. General linear models assumed a canonical statistical parametric mapping (SPM) hemodynamic response and were estimated using in-house Washington University software, FIDL.

Data Analyses Investigating Associations With Dampening Positive Affect

Whole-brain regression analyses were conducted to investigate the association between dampening and neural response to winning a reward within specific contexts from previous trials. Given that dysregulation of the time course of positive emotion is implicated in depression (Heller et al., 2009), we examined how dampening influences response to gaining a reward within several contexts. Specifically, we examined the association between dampening and neural response to a win (gain trial) following three contexts: (a) a previous loss (GpostL), (b) a previous win (GpostG), and (c) a previous neutral trial (GpostN). There were 13 instances of each of the three context trial types. These context conditions allowed us to examine how dampening might influence neural response to a win following a previous loss (GpostL), in the context of maintaining that responding following a previous win (GpostG), and whether this differs from a comparison control condition (GpostN). Thus we created two contrasts with these context conditions, (GpostL–GpostN) and (GpostG–GpostN), to examine how dampening influences responding to a gain following a previously emotional (loss or win) above and beyond the association with a GpostN event.

With the two contrast conditions as dependent variables, whole-brain regression analyses controlled for child sex, CDI-C t scores of total depressive symptoms, and risk group (high risk and low risk depending on maternal history of depression). Images were thresholded based on Monte Carlo simulations (3dClustSim; afni.nimh.nih.gov/pub/dist/doc/program_help/3dClustSim.html) using a Gaussian plus mono-exponential spatial autocorrelation function (–acf flag) at $z \geq 2.88$ ($p < .002$) and cluster sizes of 113 contiguous voxels or more to achieve a whole-brain correction of $p = .05$. When significant regions covered multiple brain regions, we split these regions up using peak finding software to identify local maxima within these regions (≥ 10 mm between peaks) for descriptive purposes. After thresholding, mean percentage signal change for each region was extracted for each feedback and context type. Post hoc analyses included decomposing the contrasts to assess whether one condition was driving contrast results. To do this, we computed Pearson's correlations using the individual contrast conditions with dampening in regions showing significant associations in the whole-brain analyses and then using Steiger's Z tests to compare the correlations within in the same sample (Lee & Preacher, 2013). In addition, two follow-up analyses examined the association of dampening (a) when the Risk Group × Dampening interaction was accounted for and (b) in other context conditions post loss. The influence of dampening above and beyond the Risk Group × Dampening interaction was examined by adding the interaction variable to a separate regression predicting activity in regions showing significant associations. The association of dampening in other conditions post loss was examined by dampening (including the same covariates from the original analyses) predicting activation in regions showing significant interactions in a lost post loss (LpostL) condition and a neutral post loss (NpostL) condition.

RESULTS

Demographic Characteristics

As shown in Table 1, high- and low-risk groups did not significantly differ on demographic variables of sex, age, ethnicity, family income, or dampening positive affect, although the high-risk group did endorse elevated total depressive symptoms. Dampening and depressive symptoms were correlated across participants ($r = .39$, $p = .014$). Given this, we controlled for depressive symptoms in our analyses to examine the independent contribution of dampening on reward responding. Children were majority White ($n = 23$, 59%), followed by African American ($n = 8$, 20.5%) and biracial ($n = 8$, 20.5%).

Neuroimaging Results

In the gain following a loss contrast (GpostL–GpostN) analyses, tendencies to dampen significantly predicted reduced activation in response to a win (following a loss) in left ($\beta = .08$), 95% confidence interval [.04, .11], and right ($\beta = .07$), 95% CI [.04, .10], thalamus and right caudate ($\beta = .06$), 95% CI [.03, .09] (see Figure 2 and Table 2), over and above the effects of sex, depressive symptoms, or risk group. The right thalamus cluster (peak = 27, −19, 4; 272 voxels) and the left thalamus cluster (peak = −23, −29, 6; 261 voxels) included multiple brain

Demographics	Total Sample[a]	Low Risk[b]	High Risk[c]
Sex (% Female)	51%	54%	46%
Age in Years	9.14 (1.04)	9.34 (1.02)	8.73 (0.98)
Ethnicity (% White)	59%	62%	54%
Family Income	15.54 (6.31)	16.50 (5.26)	13.62 (7.90)
Dampening	12.87 (4.33)	12.77 (4.59)	13.08 (3.93)
CDI-C	46.49 (12.51)	43.58 (9.82)	52.31 (15.48)*

Note: Family income coded in 21 increments of $5,000 starting with 1 = $1,000–$5,000 and ending with 21 = > $100,000; Dampening = child reported dampening positive affect; CDI-C = Child-Reported Depressive Symptoms total score.
[a]$N = 39$.
[b]$n = 26$.
[c]$n = 13$.
*$p < .05$ for group t test comparing low- and high-risk children.

structures. Thus, we examined peaks within these larger regions of interest (ROIs), as described in the Methods section. For the right thalamus, there were peaks in the putamen/globus pallidus, thalamus (pulvinar and ventral lateral nucleus), and superior temporal gyrus. For the left thalamus, there were peaks in the thalamus (pulvinar), para-hippocampal gyrus and hippocampus (see Table 2). Similar relationships between dampening tendencies and response to the GpostL–GpostN contrast emerged across all subre-gions. Specifically, tendencies to dampening predicted *reduced* activation to a win following a loss versus a win following a neutral outcome (see Figure 3 for relationships with activity in the three original clusters). In the gain following a gain contrast (GpostG–GpostN), dampening did not significantly predict activity in response to a win (following a win), over and above the effects of demo-graphic covariates, depressive symptoms, and risk group. See supplemental text for follow-up analyses of this contrast.

Post hoc correlations decomposing the GpostL–GpostN contrast revealed larger magnitude correlations between dampening and activity in the GpostL condition compared with the GpostN condition. Steiger's Z test for dependent correlations revealed that the associations of the GpostL and GpostN conditions with dampening differed signifi-cantly in all regions (all $Zs > −2.69$, $ps < .003$; see Supplemental Table 1).

Follow-up analyses examined whether dampening ten-dencies remained a significant predictor of regions identified in the whole-brain analyses when the Dampening × Risk Group interaction was added. Regressions including all previously mentioned variables revealed that dampening remained a significant predictor ($ps ≤ .001$) for all three regions when the Dampening × Risk interaction was included. Given the very low power due the covariates and small sample size, especially for the high-risk group

($n = 13$), interpreting the Dampening × Risk interaction is cautioned.[1]

Last, we examined whether dampening findings in the GpostL condition were specific to the gain aspect of the context trial or whether any trial (e.g., neutral or loss) post loss would demonstrate similar deactivations. Regressions (including all original covariates) examined whether dam-pening tendencies were associated with activation in regions identified in the whole-brain analyses for LpostL and NpostL conditions. Dampening was not a significant pre-dictor of activation in any of the LpostL or NpostL regions of interest.

DISCUSSION

The current study examined one potential mechanism by which reduced brain activation in reward-related brain areas related to depression may arise prior to depression onset, namely, dampening positive affect. Previous research has established that neural responses to reward are blunted in both adults (Pizzagalli et al., 2009) and adolescents with depression (Forbes et al., 2009) and in children at risk for depression (Luking et al., 2016a; Sharp et al., 2014). In addition, depression in children and adults has been asso-ciated with higher tendencies to engage in dampening one's positive affect (Bijttebier et al., 2012; Nelis et al., 2015). Findings from the current study bridge this work by provid-ing the first examination of how tendencies to dampen one's positive affect may be associated with aberrant reward responding in never-depressed children. We found that in the context of a previous loss, child-reported dampening tendencies predict reduced activation to reward-related regions including portions of the striatum (caudate and putamen/globus pallidus), the superior temporal gyrus, and bilateral thalamus, all of which show altered activation in adult depression (Zhang, Chang, Guo, Zhang, & Wang, 2013). However, dampening tendencies were not associated with activation to reward in the context of a previous win. To our knowledge, this is the first study examining relation-

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[1] Caution is warranted in interpreting the results of the Risk Group × Dampening interaction due to sample size differences in the two groups, and small within-group, high-risk sample size, however, are provided here for illustrative purposes. In addition to dampening remaining a significant predictor when the interaction was added, the Dampening × Risk interac-tion was also a significant predictor for all three of the GpostL–GpostN contrasts. Regarding the Dampening × Risk interactions, analyses indicated that dampening was more strongly related to activity for the low-risk group ($n = 26$) than for the high-risk group ($n = 13$) in all three regions. Although both the high-risk and low-risk children who reported using more dampen-ing tended to exhibit blunted activation for the GpostL–GpostN, the simple slopes were significant only for low-risk children in the right thalamus/striatum ($b = −0.06$; $SE = 0.11$, $t = −5.62$, $p < .001$), the left thalamus ($b = −0.07$; $SE = 0.12$, $t = −5.70$, $p < .001$), and the right caudate ($b = −0.05$; $SE = 0.01$, $t = −5.65$, $p < .001$).
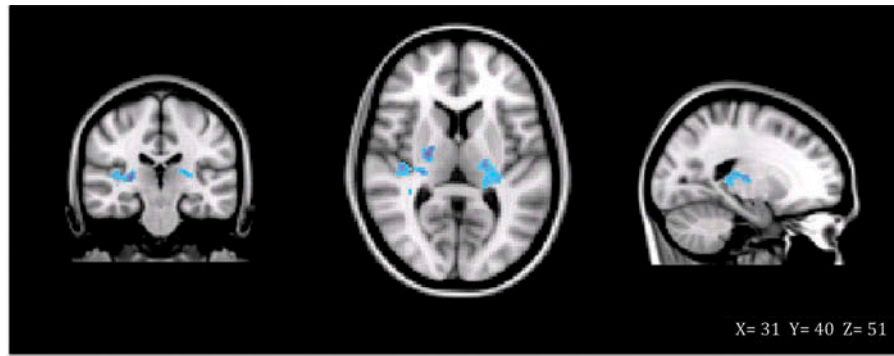
FIGURE 2   Whole-brain regression results of the association of dampening when winning a reward following a loss. *Note*: Whole-brain analysis of GpostL–GpostN contrast ($-2.88 > z > -4.03$). Regions demonstrating negative associations between dampening and activity include bilateral thalamus, right putamen/globus pallidus, right caudate, right superior frontal gyrus, left parahippocampal gyrus, and left hippocampus. See the online article for the color version of this figure.

TABLE 2
Associations of Dampening With Gain Post Loss–Gain Post Neutral Contrast

Region	Side	Peak Coordinates			Cluster Size	B(SE)	Average Z	Average Activity
		X	Y	Z				
Thalamus/Striatum	R	27	−19	4	272	0.07(.02)	4.16	0.08
Putamen/Globus Pallidus	R	24	−15	2	68	0.08(.02)	3.56	0.09
Thalamus/Pulvinar	R	27	−24	6	70	0.08(.02)	3.64	0.002
Ventral lateral nucleus (Thalamus)	R	21	−8	9	62	0.08(.02)	4.00	0.17
Superior temporal gyrus	R	41	−23	6	72	0.06(.02)	3.50	0.05
Caudate	R	28	−40	11	124	0.06(.04)	4.07	0.09
Caudate Tail	R	23	−40	12	52	0.06(.01)	3.84	0.08
Caudate Tail	R	34	−41	14	72	0.06(.01)	3.87	0.09
Thalamus	L	−23	−29	6	261	0.08(.02)	4.03	0.12
Thalamus/Pulvinar	L	−24	−30	9	116	0.08(.02)	3.49	0.12
Thalamus/Pulvinar	L	−20	−21	9	84	0.08(.02)	3.79	0.11
Parahippocampal Gyrus	L	−16	−40	4	28	0.07(.02)	3.20	0.18
Hippocampus	L	−28	−36	01	33	0.06(.01)	3.36	0.05

Note: Three large clusters in the right thalamus/striatum, right caudate, and left thalamus were observed in the whole-brain analysis. Local maxima within these larger clusters were identified and activity was extracted and analyzed from these subregions for clarification purposes. *B(SE)* values = unstandardized regression coefficients and standard errors for dampening scores predicting average activity extracted from each region; Average *Z* = *Z* value for this regression coefficient; Average activity = mean percentage signal change within each extracted region. Coordinates in Montreal Neurological Institute Space.
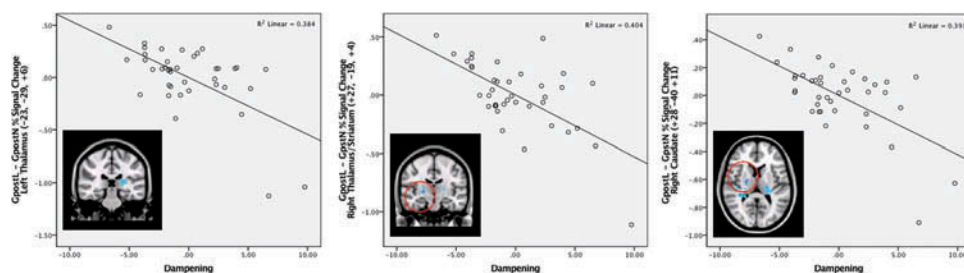

FIGURE 3   Dampening predicting response to gain following a loss (subtracting GpostN). *Note*: Partial regression plots controlling for sex, risk group, and depressive symptoms. Dampening is centered.

ships between tendencies to engage in a positive affect regulation strategy and neural reward responding.

The first aim of the study was to examine reward responding to a win following a loss, so as to examine purported contextual difficulties disengaging from the loss and upregulating subsequent reward responding. Findings indicated that dampening tendencies are associated with less activation in reward-related brain areas, including the caudate, putamen/globus pallidus, thalamus, superior temporal gyrus, and parahippocampal gyrus and hippocampal regions to a win following a loss. These results remained when controlling for the Dampening × Risk Status interaction. Using the same sample, Luking et al. (2016a) demonstrated that in this childhood age range, loss is more salient and is associated with a stronger relationship with risk for depression than blunted response to gains. Given that the current study examined reactivity to a win following a loss, one speculative hypothesis is that the high loss salience contributes to the apparent difficulty disengaging from negative to positive stimuli. Moreover, tendencies to dampen one's positive affect may contribute to this process. Specifically, the negative emotional reaction elicited from the loss may activate cognitive biases such as dampening, and subsequently dampening may contribute to cognitive tempering of the following win. It should be noted, though, that this interpretation is speculative and findings are preliminary, as it is unknown whether children were engaging in dampening (or any regulatory strategy) during the task as dampening was measured with a self-report measure outside of the scanner. However, elevated tendencies to dampen positive affect and associated blunted striatal reward response in these children provides an intriguing first step in understanding how dampening may be a mechanism by which neural reward responding aberrations emerge in depression.

Interesting to note, the regions where dampening tendencies related to GpostL activation are also associated with behavioral and cognitive flexibility in prior studies. The dorsal striatum is implicated in behavioral flexibility (Ragozzino, 2007) and reversal learning (Izquierdo, Brigman, Radke, Rudebeck, & Holmes, 2016), and recently the thalamus has been associated with flexible responding and filtering out distractors during attention tasks (Guo, Ponvert, & Jaramillo, 2016). Taken together, the reduced activation in these regions in the GpostL contrast may demonstrate how dampening contributes to impaired affective flexibility. Affective flexibility is the ability to respond to changing affective contexts, and depressed adolescents demonstrate less affective flexibility, especially when transitioning from negative to positive stimuli (McMakin et al., 2011; Sheeber et al., 2009). Again, although speculative, this blunted activation to the gain in the context of a previous loss may be associated with a decreased ability to fully capitalize on and savor the current reward win. Our follow-up analyses support this conclusion, as dampening was associated with reduced activation only when a gain

followed a loss, not when other outcomes (loss or neutral) followed a loss. Related, these results are also consistent with emotional inertia theories, as a lack of affective or behavioral flexibility may result in maintained negative affective states in the face of shifting contexts. Dampening positive affect may confer risk for depression in youth by operating by means of affective inflexibility. These hypotheses will need to be prospectively studied with designs that allow a more direct evaluation of such mechanisms.

For the second aim examining the neural response to a win following a previous win, we tested the hypothesis that dampening tendencies would be associated with an inability to sustain reward responding. Our lack of significant findings runs counter to work demonstrating that following a previous win, depressed youth exhibit decreased caudate activation during anticipation of a second win (Olino et al., 2011) and depression in adults is characterized by blunted striatal response to multiple blocks of trials (Heller et al., 2009). It should be noted that participants in the current study were not yet depressed and only a subset were at elevated risk for depression, whereas prior research has used depressed adult samples. The inability to sustain positive affect and striatal responding over multiple trials may be a scar of depression rather than a vulnerability factor, emerging only during and after experiencing a major depressive episode. Conversely, the inability to sustain striatal responding may emerge during adolescence, when reward-responding increases in normative development (Galvan, 2013; Luking, Pagliaccio, Luby, & Barch, 2016b). Thus, dampening may not play a role in contributing to this process during childhood.

Although we are hesitant to interpret null effects and it was not originally hypothesized, it should be noted that we did not find evidence of associations between dampening and other brain regions commonly implicated in reward responding, namely, prefrontal regions and another striatal region, the nucleus accumbens (NAcc). Prefrontal regions demonstrate elevated reactivity in response to reward in adults (Knutson, Bhanji, Cooney, Atlas, & Gotlib, 2008) and adolescents (Forbes et al., 2009). The NAcc demonstrates blunted reward responding in adolescent depression (Forbes & Dahl, 2012) and in depressed adults attempting to upregulate positive affect (Heller et al., 2009). It should be noted that a recent meta-analysis did not find reduced activity in prefrontal or NAcc regions during reward processing in adults with depression versus healthy individuals (Zhang et al., 2013). Thus, these regions may not be the most robust indicators of reduced reward responding in depression. Moreover, these regions may not specifically be implicated in reward-related affective inflexibility, which is the purported mechanism evidenced in the current study. Instead, these null findings may speak to the many subprocesses of reward processing that engage different brain regions (Admon & Pizzagalli, 2015b). Another possibility is that a different experimental design might be needed to identify

altered prefrontal or NAcc activation associated with dampening. For example, emerging work examining neural reward responding and positive affect in depression has focused on detailed time course analyses within these regions (Admon & Pizzagalli, 2015a; Heller et al., 2009). Unfortunately, the use of a fast event related design in the current study is not well suited to address this question. Nonetheless, future work examining dampening positive affect in relation to neural decay over time may provide additional insight into how dampening is associated with altered neural response and may reveal stronger associations with prefrontal and NAcc regions.

The current findings validate the neural correlates of tendencies to dampen one's positive affect and have implications for better understanding how positive affect regulation influences aberrant reward response in healthy children. Tendencies to dampen positive affect are associated with blunted reward response that is characteristic of youth depression (Forbes et al., 2009), and dampening may be a pertinent target for preventive intervention. It may be warranted to help children who engage in more dampening to be mindful of and better savor their positive experiences (McMakin, Siegle, & Shirk, 2010), as day-to-day life is associated with multiple "wins" and "losses" that repeatedly occur across many contexts.

Results from the current study should be interpreted within the confines of several limitations. First, the study was cross-sectional, so we are unable to disentangle causality. Thus, we do not know whether dampening contributes to aberrant neural reward processing, or whether aberrant neural reward processing leads to greater use of dampening. Future studies would benefit from using longitudinal samples to identify temporal and causal relations between dampening, neural reward responding, and onset of depression. Second, the study's sample size was small, and results should be considered preliminary. Although the racial diversity of the sample was characteristic of the urban St. Louis area, children were White, African American, or biracial, and results cannot be generalized beyond these racial groups. Related, children were all healthy, and it is unknown how dampening may relate to neural reward responding in the context of experiencing depression. Third, the small sample size also resulted in collapsing across children at both high risk and low risk for depression depending on maternal depression. Fourth, the current reward task did not include an anticipation phase or a reward learning phase, and thus we are unable to determine how tendencies to dampen positive affect may be associated with different neural responding during these other reward-responding contexts. Given that previous research in adults with depression has shown differences in reward responding in early and late blocks of neuroimaging tasks (e.g., Heller et al., 2009), it will be important for future studies with more trials to examine cumulative effects of wins/losses over time. Fifth, dampening was measured as child-reported

tendencies to utilize this strategy, and thus we are unable to determine whether children were actually engaging in dampening (or any other cognitive regulatory strategy) while completing the reward task in the scanner. Although findings provide preliminary evidence that dampening may play a role in aberrant neural reward responding, it will be important for future work to examine how engaging in dampening in the moment influences neural activation.

Despite these limitations, the current study provides initial evidence that above and beyond depressive symptoms, tendencies to dampen positive affect are associated with reduced reward-related neural processing in children who have not yet experienced depression. These findings occurred in response to winning a reward when immediately following a previous loss outcome, demonstrating the importance of context in reward processing. As research continues to identify disruptions in positive affect valence systems in depression, it will be important to identify ways to help children engage in less dampening of their positive affect, possibly attenuating risk for depression in children.

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