



Research paper

Neural reactivity to reward in school-age offspring of depressed mothers

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ABSTRACT

Background: Identifying neural profiles predictive of future psychopathology in at-risk individuals is important to efficiently direct preventive care. Alterations in reward processing may be a risk factor for depression. The current study characterized neural substrates of reward processing in children at low- and high-risk for psychopathology due to maternal depression status.

Methods: Children with ($n=27$) and without ($n=19$) maternal depression (ages 5.9–9.6 years) performed a monetary incentive delay task in which they received rewards, if they successfully hit a target, or no reward regardless of performance, during fMRI acquisition.

Results: Multiple dorsal prefrontal, temporal, and striatal regions showed significant Group (high- vs. low-risk) × Performance (hit vs. miss) × Condition (no reward vs. reward) interactions in a whole-brain analysis. All regions exhibited similar patterns, whereby the high-risk group showed blunted activation differences between trials with vs. without rewards when participants hit the target. Moreover, high-risk children showed activation differences between trials with vs. without rewards in the opposite direction, compared to the low-risk group, when they missed the target.

Limitations: This study had a modest sample size, though larger than existing studies. Children with maternal depression are at elevated risk for future psychopathology, yet not all experience clinically significant symptoms; longitudinal research is necessary to fully track the pathway from risk to disorder.

Conclusion: Children of depressed mothers exhibited attenuated neural activation differences and activation patterns opposite to children without depressed mothers. Our findings may provide targets for hypothesis-driven preventive interventions and lead to earlier identification of individuals at risk.

1. Introduction

Parental, and particularly maternal, depression is one of the best-documented risk factors, increasing risk for depressive disorders and related mood and anxiety symptoms three- to four-fold (Goodman and Gotlib, 1999; Gunlicks et al., 2008; Wiggins et al., 2014), yet the neural mechanisms by which risk is conferred to offspring are largely unknown. Prior studies with adolescents or adults, beyond the age at which depression commonly onsets (i.e., adolescence), who have mothers with history of depression (e.g., Gotlib et al., 2010) and/or who have depression themselves (e.g., Kerestes et al., 2014) suggest that dysfunctional neural responses to reward may be involved in the development of depression. However, in order to fully understand how risk becomes manifested as a depressive illness, it is necessary to

characterize the entire developmental pathway from maternal depression to later offspring depression, including periods prior to the onset of the disorder. As such, the current study focused on children younger than the common age of onset for depression, who are at risk due to maternal depression, in order to identify potential pre-existing neural vulnerability markers for depression.

Prior work on the development of depression has focused on attenuated reward processing as a core clinical characteristic of depressive disorders (American Psychiatric Association, 2013). Indeed, decreased pleasure in depression has been documented through diminished or inappropriate responses to reward: for example, attenuated positive emotional facial expressions in response to reward (Sloan et al., 2001); failure to adapt behavior in response to changing reward conditions (Henriques and Davidson, 2000); and, psychophy-

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biological responses to rewarding stimuli as if they were aversive (Allen et al., 1999). Moreover, an established and growing literature documents that adults and adolescents with depression demonstrate atypical responses in brain regions related to reward and emotion (e.g., prefrontal, limbic, and striatal regions) under various reward conditions (e.g., expecting and/or receiving a reward, not expecting and/or not receiving a reward) (for review, see Kerestes et al., 2014).

In efforts to trace the pathway from risk to manifested disorder, prior work with adult and adolescent offspring of mothers with depression, older than the common age at which depression onsets, demonstrated atypical brain activation including attenuated activation or hyperactivation in prefrontal, limbic, and striatal regions, compared to low-risk individuals without mothers with history of depression (Gotlib et al., 2010; Luking et al., 2016; Morgan et al., 2014; Olino et al., 2014; Sharp et al., 2014) during anticipation and receipt of (or failure to receive) rewards. Moreover, aspects of parenting that are impacted by maternal depression, such as maternal warmth (Morgan et al., 2014) and interpersonal affiliation (Schneider et al., 2012), were linked to attenuated or inappropriately large neural responses to feedback about whether adolescents received a reward. These findings are in line with research suggesting that in addition to genetic factors, maternal depression transmits psychopathology risk through heightened pediatric stress sensitivity, resulting from more hostile parent-child interactions (Dougherty et al., 2013).

However, neural responses to reward in relation to maternal characteristics have been examined primarily during adulthood or adolescence (Gotlib et al., 2010; Morgan et al., 2014; Olino et al., 2014; Sharp et al., 2014), developmental periods when depression is likely to have onset (Merikangas et al., 2010). Little work has attempted to characterize the neural substrates of reward processing in maternal depression offspring prior to adolescence. Characterizing neural profiles associated with risk at younger ages (i.e., middle childhood/preadolescence) is important for elucidating the early stages of the developmental path from risk to manifested disorder, and is crucial if we are to identify targets for preventive interventions.

We addressed this gap in the literature by comparing brain function in preadolescent children with and without maternal depression, while they performed a monetary incentive delay task. We probed responses during both the anticipation and receipt of (or failure to receive) rewards. We expected that children with maternal depression (“high-risk” group) would demonstrate atypical activation, such as attenuated activation between reward conditions (i.e., little difference between reward vs. no reward conditions), compared to children without maternal depression (“low-risk” group) in reward-related regions (i.e., striatal, prefrontal, and limbic), both during anticipation of a potential reward and when receiving feedback about whether they earned the reward.

2. Methods

2.1. Participants

We obtained neuroimaging and clinical datasets from children (ages 5.9–9.6 years) whose mothers had a lifetime history of major depressive disorder and/or dysthymic disorder based on *DSM-IV* diagnostic criteria (high-risk group, $n=27$), and a separate group of children without such maternal depression (low-risk group, $n=19$; see Table 1 for participant characteristics). Participants were recruited from a larger University of Maryland study that aimed to evaluate neuroendocrine risk factors for depression (Dougherty et al., 2013). Recruitment efforts were facilitated by flyers in community locations in the College Park, MD, area and a commercial mailing list; parents with a lifetime history of depression were oversampled. Participants received monetary compensation. Exclusion criteria consisted of a developmental or physical disability in the child, children or parents not speaking English well enough to complete the assessments, and a

Table 1
Sample characteristics.

	Low risk ($n=19$)	High risk ($n=27$)
<i>Child demographics</i>		
Age	7.64 (.84)	7.44 (.73)
Gender (female)	14 (74%)	14 (52%)
Race		
White, Non-hispanic	7 (37%)	12 (44%)
African American	5 (26%)	8 (31%)
Multiracial	2 (11%)	2 (8%)
Other	2 (11%)	2 (8%)
Hispanic ethnicity	2 (11%)	5 (19%)
<i>Child clinical characteristics</i>		
Affective reactivity index	1.61 (1.94)	2.46 (2.67)
PAPA		
Current depressive disorder (MDD, DD, Dep-NOS)	3 (16%)	5 (19%)
Current major depressive episode	1 (5%)	2 (7%)
Current irritability symptoms	1.11 (1.37)	1.96 (1.70)
Current anxiety symptoms*	8.84 (5.30)	15.59 (10.61)
Current ADHD symptoms*	2.89 (3.43)	6.56 (6.38)
Current ODD symptoms	4.05 (2.66)	5.26 (3.23)
<i>Child task performance</i>		
Success rate		
Reward	71% (5.0%)	70% (6.8%)
No reward	69% (7.7%)	66% (8.0%)
Reaction time (ms)		
Reward/Hit	349 (38)	340 (50)
No Reward/Hit	360 (37)	354 (42)
Reward/Miss	531 (108)	503 (101)
No Reward/Miss	548 (105)	580 (129)
<i>Maternal demographics</i>		
Age	39.79 (6.20)	37.48 (6.61)
Marital status (married)	11 (58%)	20 (74%)
Education level (4-year college degree or higher)	14 (74%)	15 (56%)
Income (> \$100,000/year)	8 (42%)	10 (37%)
<i>Maternal clinical characteristics</i>		
Current depressive disorder (past month)	n/a	4 (15%)
Number of MDEs (lifetime)	n/a	2.23 (1.86)
Pediatric exposure to maternal depression	n/a	20 (74%)
Proportion of months exposed	n/a	.26 (.34)
Lifetime anxiety disorder*	1 (5%)	23 (85%)
Lifetime substance use disorder ^a	2 (11%)	4 (15%)

Note: Dimensional characteristics are displayed as M (SD) and categorical characteristics are displayed as N (%); PAPA=Preschool Age Psychiatric Assessment; MDD=Major Depressive Disorder; DD=Dysthymic Disorder; Dep-NOS=Depression, Not Otherwise Specified; ADHD=Attention Deficit/Hyperactive Disorder; ODD=Oppositional Defiance Disorder; MDE=Major Depressive Episode.

* Significant groups differences ($p < .05$).

^a Information is summative across T1 and T2 time points.

lifetime history of psychotic or bipolar disorder in either biological parent. Children and their parental caregivers completed two waves of assessment, first when they were preschool-age (i.e., 3–5 years; Wave 1) and approximately three years later (Wave 2).

Neuroimaging data for the current study were obtained at Wave 2. All children participating in Wave 2 ($N=115$; $M=7.29$ years old, $SD=.92$; 51% female; 39% non-Hispanic White) were invited to complete a neuroimaging session, and 64 volunteered. One child was not scanned due to claustrophobia. Of the 63 remaining participants, 17 were excluded from analyses ($n=10$ exited the scanner early due to discomfort; $n=3$ exhibited excessive head motion [see *fMRI Data Preprocessing*]; $n=2$ completed a different scan protocol; $n=1$ had inadequate neuroanatomical scan coverage; $n=1$ was missing behavioral data), leaving a final sample of $N=46$ usable datasets. One participant had an incomplete imaging run, but available data for that run were included in analyses. The final sample of children whose

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