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Reduced reward anticipation in youth at high-risk for unipolar depression: A preliminary study

Thomas M. Olino[a,*], Dana L. McMakin[a], Judith K. Morgan[a], Jennifer S. Silk[a], Boris Birmaher[a], David A. Axelson[a], Douglas E. Williamson[b], Ronald E. Dahl[c], Neal D. Ryan[a], Erika E. Forbes[a]

[a] University of Pittsburgh, United States
[b] University of Texas Health Science Center, United States
[c] University of California-Berkeley, United States

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ABSTRACT

Offspring of depressed parents are at risk for depression and recent evidence suggests that reduced positive affect (PA) may be a marker of risk. We investigated whether self-reports of PA and fMRI-measured striatal response to reward, a neural correlate of PA, are reduced in adolescent youth at high familial risk for depression (HR) relative to youth at low familial risk for depression (LR). Functional magnetic resonance imaging assessments were conducted with 14 HR and 12 LR youth. All youth completed an ecological momentary assessment protocol to measure PA in natural settings and a self-report measure of depression symptomatology. Analyses found that HR youth demonstrated lower striatal response than LR youth during both reward anticipation and outcome. However, after controlling for youth self-reports of depression, HR youth demonstrated lower striatal response than LR youth only during reward anticipation. No significant differences were found between HR and LR youth on subjective ratings of PA or depressive symptoms. Results are consistent with previous findings that reduced reward response is a marker of risk for depression, particularly during reward anticipation, even in the absence of (or accounting for) disrupted subjective mood. Further examinations of prospective associations between reward response and depression onset are needed.

1. Introduction

Offspring of depressed parents are at risk for developing depressive disorders (Lieb et al., 2002; Hammen et al., 2004; Klein et al., 2005; Goodman et al., 2011) and other functional impairments (Beardslee et al., 1998; Lewinsohn et al., 2005). Rich theoretical perspectives outline potential mechanisms of risk, including biological and psychosocial factors (Goodman and Gotlib, 1999, 2002). However, there remains only a modest literature examining specific neural processes through which parental depression is related to offspring depression. One potential mechanism of transmission or vulnerability marker of risk in youth is positive affect (PA) that includes subjective experience and neural functioning.

Positive affect plays a central role in depression as diminished experience of interest and/or pleasure is a cardinal symptom of the disorder and has been linked to risk

* Corresponding author at: Department of Psychology, Temple University, 1701 North 13th Street, Weiss Hall, Philadelphia, PA 19122, United States. Tel.: +1 215 204 1553.
E-mail addresses: thomas.olino@gmail.com, thomas.olino@temple.edu (T.M. Olino).

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for onset, recurrence, and likelihood of remission (Pine et al., 1999; Kasch et al., 2002; McMakin et al., 2012). Studies of PA in the context of depression have relied on indices of personality/temperament (including extraversion and positive emotionality; Compas et al., 2004; Clark, 2005; Kotov et al., 2010), affective experience (including positive affect; Laurent et al., 1999; Joiner and Lonigan, 2000; McMakin et al., 2009), and neural functioning (including response to reward; for a meta-analytic review see Zhang et al., 2013). These affective responses are active when working toward or achieving goals and experiencing PA states. However, most of these investigations have relied on cross-sectional comparisons between depressed and non-depressed participants. Thus, these studies cannot speak to whether altered PA is a predictor of onset, a correlate of the disorder episode, or a consequence (i.e., scar) of experiencing a depressive episode. To determine whether low PA is associated with developing depression, prospective studies are needed. However, it is also important to examine whether low PA is associated with established risk factors for depression, notably family history of depression.

Supportive evidence for reduced PA in youth at familial risk for depression comes from lines of work involving behavioral displays of PA. Offspring of depressed parents demonstrate lower levels of PA than offspring of parents without a history of depression. For example, Durbin et al. (2005) found that three-year old children of mothers with a history of depression demonstrated lower levels of PA, indexed by smiling, laughter, and interest in exploration of stimuli, across a series of structured laboratory tasks. In addition, Olino et al. (2011) examined longitudinal changes in laboratory assessed PA in youth, primarily indexed by smiling and laughter, of depressed and non-depressed mothers spanning late infancy through age 9. The authors found that offspring of depressed mothers demonstrated significantly lower levels of PA across childhood than offspring of mothers without a history of depression. Thus, these studies highlight that behavioral displays of positive affect differentiate between young children at high- and low-risk for depression. However, fewer studies have examined similar questions beyond childhood; thus, it is unclear if similar associations continue to be present in adolescence. Rather than relying on behavioral observations, adolescents can complete reports of affect in their naturally occurring environments that can improve ecological validity of measurement. Further, these results are suggestive that neural mechanisms of PA would also be affected.

At a biological level of analysis, PA is often described as influencing or being influenced by striatal function (among other functions, including avoidance of punishment; Forbes, 2009; Haber and Knutson, 2010). In particular, the ventral striatum (VS), inclusive of the nucleus accumbens, is responsive when pursuing, encountering, and seeing cues of multiple classes of reinforcers, including drugs, food, and money (Berridge and Robinson, 2003; Nestler and Carlezon, 2006; Haber and Knutson, 2010). Indeed, experimental manipulations of the VS, by administering amphetamines, have produced positive affective states in healthy participants (Drevets et al., 2001). In addition, adolescent reports of PA in naturalistic environments have previously been reported to be associated with ventral striatal response during reward anticipation and receipt (Forbes et al., 2009, 2010). Results of studies of reward response across adolescence has provided mixed findings, with some studies finding reduced (e.g., Forbes et al., 2010) and others finding increased (e.g., Galvan et al., 2006) striatal response across development. Despite these differences, however, most researchers interpret the results as indicating that adolescence is a period marked by greater reward responsiveness than during childhood or adulthood.

While much of the work delineating striatal response to reward has focused on healthy populations, a number of recent studies examined the influence of depression on reward function. These studies have typically found that individuals with depression have lower levels of striatal response than individuals without depression (Nestler and Carlezon, 2006; Forbes et al., 2009; Pizzagalli et al., 2009; Smoski et al., 2011; Dichter et al., 2012) and are summarized by a recent meta-analysis (Zhang et al., 2013). However, given the possibility of state influences or scarring effects of depression on striatal function, these studies cannot speak to whether altered reward functioning is a cause, correlate, or consequence of depression. Studies focusing on individuals at-risk for depression are necessary to identify if altered reward-related brain functioning is present in individuals before depressive disorder onsets.

A small number of studies have examined neurobiological response to positively valenced stimuli and rewards in youth at high risk for depression. Monk et al. (2008) found that youth at high-risk for depression demonstrated lower levels of nucleus accumbens response while viewing happy facial expressions than low-risk youth. However, as the task involved passive viewing of faces, it is unclear whether striatal response reflected a motivational tendency toward reward or general response to positive valence. Gotlib et al. (2010) examined response to a monetary incentive task in girls at-risk for depression. The authors reported that high-risk girls demonstrated lower putamen response than low-risk girls during anticipation of reward, but did not find differences during the receipt of rewards. The results reported by Gotlib et al. are suggestive that differences may vary between anticipation and consummatory phases of rewards (Davidson, 1998; Berridge and Robinson, 2003). However, as the high-risk girls in Gotlib et al. had significantly higher (albeit sub-syndromal) levels of depressive symptoms than the low-risk girls, it is possible that current symptoms, rather than high-risk status, may have driven the results.

This seminal work examining differences between youth at high- and low-familial risk for depression has provided support for the hypothesis that reward-system alterations are present before the onset of depression. Indeed, some have hypothesized that low PA, either conceptualized as hypohedonia (Meehl, 1975, 2001) or attenuated reward function (Hasler et al., 2004), are endophenotypes for depression. That is, attenuated PA responses would be present before, during, and following episodes, and are familial, among other considerations for characteristics being endophenotypes (Gottesman and Gould, 2003). However, there are still many questions to