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Girls' challenging social experiences in early adolescence predict neural response to rewards and depressive symptoms

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ABSTRACT

Developmental models of psychopathology posit that exposure to social stressors may confer risk for depression in adolescent girls by disrupting neural reward circuitry. The current study tested this hypothesis by examining the relationship between early adolescent social stressors and later neural reward processing and depressive symptoms. Participants were 120 girls from an ongoing longitudinal study of precursors to depression across adolescent development. Low parental warmth, peer victimization, and depressive symptoms were assessed when the girls were 11 and 12 years old, and participants completed a monetary reward guessing fMRI task and assessment of depressive symptoms at age 16. Results indicate that low parental warmth was associated with increased response to potential rewards in the medial prefrontal cortex (mPFC), striatum, and amygdala, whereas peer victimization was associated with decreased response to potential rewards in the mPFC. Furthermore, concurrent depressive symptoms were associated with increased reward anticipation response in mPFC and striatal regions that were also associated with early adolescent psychosocial stressors, with mPFC and striatal response mediating the association between social stressors and depressive symptoms. These findings are consistent with developmental models that emphasize the adverse impact of early psychosocial stressors on neural reward processing and risk for depression in adolescence.

Abbreviations: BA, Brodmann Area; BOLD, blood-oxygen-level-dependent; EPI, echo planar imaging; fMRI, functional magnetic resonance imaging; LN, natural log transformation; mPFC, medial prefrontal cortex; MNI, Montreal Neurological Institute; OFC, orbitofrontal cortex; PGS, Pittsburgh Girls Study; PGS-E, Pittsburgh Girls Study-Emotions Substudy; ROI, region of interest; TE, echo time; TR, repetition time.

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1. Introduction

Depression is a leading cause of global disease burden with a 16.6% lifetime prevalence (Kessler et al., 2005; World Health Organization, 2008). Although the prevalence of depression during childhood is less than 3% (Fleming and Offord, 1990), rates of depression increase sharply during adolescence with the first onset occurring between the ages of 12 and 19 years in 20% of individuals who experience depression during their lifetimes (Kessler et al., 2005). Rates of depression are particularly high in adolescent girls (cumulative prevalence of 20.8%), who are twice as likely to become depressed compared to adolescent boys (Kessler, 1993). Because depression is a recurrent disorder, experiencing depression for the first time in childhood or adolescence, compared with onset later in life, results in greater lifetime depression-related disability (Kovacs, 1997). Thus, studies that examine risk factors for the development of depression in adolescent girls are particularly relevant for prevention and intervention efforts.

Parent and peer relationships are both important to adolescent development, and stressors in either social domain can increase risk for psychopathology. There is a large body of research documenting the impact that parenting behaviors, such as emotional responsiveness and warmth, have on children's emotional development broadly (Eisenberg et al., 1998; Morris et al., 2007), and on depressive symptoms specifically (McLeod et al., 2007). Additional data from longitudinal studies indicate that low parental warmth increases risk for depression in children and adolescents (Ge et al., 1994, 1996; Hipwell et al., 2008) and decreases resilience in adolescents with a high genetic and socioeconomic risk for the disorder (Brennan et al., 2003; Masten et al., 1999). Early adolescence in particular is characterized by decreases in parent–child relationship quality (Loeber et al., 2000; McGue et al., 2005), which could contribute to the higher risk for psychopathology during this developmental period relative to childhood.

Although parents continue to be important sources of social support and play a role in adolescents' mental health, peer relationships become increasingly important as adolescents individuate from parents and form social hierarchies with peers. Stressful interactions with peers, including emotional exclusion and aggression, are particularly difficult experiences for adolescents (Nelson et al., 2005; O'Brien and Bierman, 1988), and approximately 50% of sixth and seventh graders experience these forms of peer victimization (Wang et al., 2009). Peer victimization is also associated with increased risk for depression. A meta-analysis of cross-sectional studies indicated that peer victimization during childhood or adolescence was moderately associated with depression, and had a stronger relationship with depression than other negative psychosocial outcomes such as anxiety (Hawker and Boulton, 2000). Peer victimization also predicted later depressive symptoms in several longitudinal studies of children and adolescents (Keenan et al., 2010b; Sweeting et al., 2006; Vuijk et al., 2007).

Although there is substantial evidence that low parental warmth and peer victimization are both associated with risk for depression, few studies have examined the potential neural mechanisms of these effects. Several developmental models of depression have focused on the interface between adolescent social development and brain development in conceptualizing vulnerability to depression (Davey et al., 2008; Forbes and Dahl, 2005; Nelson et al., 2005). In this view, adolescent development of neural reward circuitry is a key process in the etiology of depression and depressive anhedonia. Furthermore, stressors that occur during adolescence may disrupt the development of reward-related circuitry, such as the medial prefrontal cortex (mPFC) – a region implicated in self-relevant and social processing as well as reward function (Amodio and Frith, 2006; Northoff and Hayes, 2011) – and the ventral striatum – a region implicated in motivation to obtain rewards (Berridge and Robinson, 1998). Consistent with these neurodevelopmental models of depression, neural response during reward anticipation and following rewarding outcomes is disrupted in adolescents and adults with depression (Forbes et al., 2009; Knutson et al., 2008; Pizzagalli et al., 2009; Steele et al., 2007). There is also evidence that exposure to early life stress, such as childhood maltreatment, is associated with reductions in reward-directed behavior (Guyer et al., 2006), and maternal deprivation produces anhedonic behaviors (e.g., decreased sucrose preference) in rodents and non-human primates (Pryce et al., 2005), behaviors that are supported by neural reward circuitry (Berridge and Robinson, 1998).

The aim of the present paper was to examine the relationship between social stressors experienced in early adolescence and neural response to rewards and depressive symptoms in later adolescence. Low parental warmth, peer victimization, and depressive symptoms were assessed at ages 11 and 12 and used to predict neural response during reward anticipation at age 16 in a large sub-sample of adolescent girls from the ongoing Pittsburgh Girls Study (PGS). Depressive symptoms were also assessed at age 16 and used to test associations with neural response to potential rewards in areas that were also associated with early adolescent social stressors. Based on previous studies showing increased mPFC response during reward anticipation in depressed adolescents (Forbes et al., 2009) and adults (Knutson et al., 2001), we expected that low parental warmth, peer victimization, and depressive symptoms would be associated with increased mPFC response during reward anticipation. We also expected that low parental warmth, peer victimization, and depressive symptoms would be associated with decreased ventral striatum response to potential rewards, consistent with other studies that found decreased striatal response to rewards in clinically depressed samples (Forbes et al., 2009; Pizzagalli et al., 2009; Steele et al., 2007). Finally, we hypothesized that neural response to potential rewards in the mPFC and ventral striatum would mediate the association between early adolescent social stress and depressive symptoms at age 16, even after controlling for early adolescent depressive symptoms.