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Role of Reward Sensitivity and Processing in Major Depressive and Bipolar Spectrum Disorders

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Since Costello's (1972) seminal *Behavior Therapy* article on loss of reinforcers or reinforcer effectiveness in depression, the role of reward sensitivity and processing in both depression and bipolar disorder has become a central area of investigation. In this article, we review the evidence for a model of reward sensitivity in mood disorders, with unipolar depression characterized by reward hyposensitivity and bipolar disorders by reward hypersensitivity. We address whether aberrant reward sensitivity and processing are correlates of, mood-independent traits of, vulnerabilities for, and/or predictors of the course of depression and bipolar spectrum disorders, covering evidence from self-report, behavioral, neurophysiological, and neural levels of analysis. We conclude that substantial evidence documents that blunted reward sensitivity and processing are involved in unipolar depression and heightened reward sensitivity and processing are characteristic of hypomania/mania. We further conclude that aberrant reward sensitivity has a trait component, but more research is needed to clearly demonstrate that reward hyposensitivity and hypersensitivity are vulnerabilities for depression and bipolar disorder, respectively. Moreover, additional research is needed to determine whether bipolar depression is similar to unipolar depression and characterized by reward hyposensitivity, or whether like bipolar hypomania/mania, it involves reward hypersensitivity.

Keywords: reward sensitivity; major depression; bipolar disorder

Costello's Legacy: Loss of Reinforcers or Reinforcer Effectiveness in Depression

In a classic article published in *Behavior Therapy*, Costello (1972) reviewed conceptual arguments and evidence about whether unipolar depression is best characterized as resulting from behavioral extinction due to a loss of reinforcers (rewards) or to a loss of interest in the environment (anhedonia) due to a loss of the effectiveness of rewards. Costello's (1972) review ultimately concluded that anhedonia and a reduction in the effectiveness of rewards were central to an understanding of depression. In the more than 40 years since the publication of Costello's seminal article, research on low reward sensitivity and reward processing in depression has burgeoned, and reward or Behavioral Approach System (BAS; Gray, 1994) hyposensitivity is now one of the prominent models of major depression (Pizzagalli, 2014; Treadway & Zald, 2013). Similarly, the BAS or reward hypersensitivity model has become one of the leading

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biopsychosocial theories of the onset and course of bipolar spectrum disorders (BSDs; Alloy & Abramson, 2010; Alloy, Abramson, Urošević, Bender, & Wagner, 2009; Alloy, Nusslock, & Boland, 2015; Depue & Iacono, 1989; Johnson, 2005; Johnson, Edge, Holmes, & Carver, 2012; Urošević, Abramson, Harmon-Jones, & Alloy, 2008). Consequently, in homage to Costello's recognition of the importance of the reward system in depression, in this article, we provide a model and up-to-date review of the role of reward sensitivity and processing in the onset and course of the full range of mood disorders—both unipolar depression and BSDs.

The Reward System

The reward system, also known as the BAS (Gray, 1994), has been linked to a fronto-striatal neural circuit that responds to stimuli involving the anticipation and receipt of rewards (e.g., Depue & Collins, 1999; Haber & Knutson, 2010). This system regulates goal-directed behavior and approach motivation and is activated by internal (e.g., expectancy of a job promotion) or external (e.g., opportunity to win a prize) goal- or reward-relevant cues or events. Activation of the reward system leads to increased incentive motivation, goal-related cognitions, and motor behavior directed toward attaining rewards, as well as positive goal-striving emotions such as happiness and hope (Depue & Collins, 1999; Gray, 1994), or to anger when goal-striving is frustrated or blocked (Carver, 2004; Harmon-Jones & Sigelman, 2001). Down-regulation or deactivation of the reward system leads to decreased motivation, decreased goal-related cognitions, and increased withdrawal, as well as emotions such as sadness and anhedonia.

Both animal and human research indicate that the ventral striatum (VS) and orbitofrontal cortex (OFC), among other regions, are involved in this fronto-striatal neural circuit (Haber & Knutson, 2010; Kringelbach & Rolls, 2004; Schultz, 2002). The VS appears to play a central role in reward anticipation and is involved in processing both primary (e.g., food) and secondary (e.g., monetary) rewards. The OFC is particularly important for assessing probability of reward receipt and encoding reward value (Haber & Knutson, 2010). Higher self-reported BAS/reward sensitivity has been associated with elevated VS activity during reward anticipation (Caseras, Lawrence, Murphy, Wise, & Phillips, 2013), and individual differences in reward dependence are associated with connectivity between the VS and OFC (Cohen & Ranganath, 2007).

There has been extensive theorizing about adolescent brain development as it relates to changes in reward sensitivity (e.g., Forbes & Dahl, 2012; Olino, in press). Adolescents, relative to children and adults, demonstrate heightened responsivity to rewards across multiple measurement strategies including self-reports of BAS (Pagliaccio et al., in press), reward pursuit behaviors (Anokhin, Golosheykin, & Mulligan, 2015), and neural indices of reward processing (e.g., Forbes, Ryan, et al., 2010). This work has largely relied on cross-sectional studies; thus, additional longitudinal work is needed to further evaluate these trends. Although there are developmental trends in reward processes, our review finds very similar patterns of results of studies of youth and adults, such that development does not appear to be a key moderator of the relationship between reward sensitivity and either unipolar or bipolar mood disorders. However, these developmental patterns in reward sensitivity are important as these changes appear to be synchronous with emergence of mood disorders (Hankin et al., 1998; Lewinsohn, Klein, & Seeley, 1995).

Major Depression and Bipolar Disorder as Opposite Ends of a Reward Sensitivity Dimension

There is a growing recognition of the importance of identifying pathophysiological mechanisms that cut across, or are common to, multiple psychiatric disorders (Insel et al., 2010). An equally important objective, however, is to identify mechanisms and biosignatures that are unique to specific psychiatric disorders. Relevant to these goals, and as summarized here, is evidence that abnormal reward sensitivity is involved across the entire mood disorders spectrum, with blunted reward sensitivity serving as a risk factor for major depression (e.g., Pizzagalli, 2014; Treadway & Zald, 2013), whereas abnormally elevated reward sensitivity is a risk factor for BSDs (e.g., Alloy et al., 2015; Johnson, Edge, et al., 2012). Collectively, this suggests that risk for depression and BSDs are characterized by extreme and opposite profiles of reward sensitivity. For individuals with abnormal reward sensitivity, when they experience reward system deactivating or activating environmental cues or events, their reward systems become too strongly deactivated or activated, leading to depression or hypomania or mania (referred to herein as hypo/mania), respectively (see Figure 1).

There are several important implications of identifying mechanisms of differential risk for depression versus BSDs. First, it can inform our understanding of the pathophysiology of these disorders. We propose that what differentiates risk for BSDs versus depression is vulnerability to hypo/mania. As summarized here, one of the primary risk factors for hypo/mania involves a propensity to experience abnormally