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Associations Among Smoking, Anhedonia, and Reward Learning in Depression

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Depression and cigarette smoking co-occur at high rates. However, the etiological mechanisms that contribute to this relationship remain unclear. Anhedonia and associated impairments in reward learning are key features of depression, which also have been linked to the onset and maintenance of cigarette smoking. However, few studies have investigated differences in anhedonia and reward learning among depressed smokers and depressed nonsmokers. The goal of this study was to examine putative differences in anhedonia and reward learning in depressed smokers (n = 36) and depressed nonsmokers (n = 44). To this end, participants completed self-report measures of anhedonia and behavioral activation (BAS reward responsiveness scores) and as well as a probabilistic reward task rooted in signal detection theory, which measures reward learning (Pizzagalli, Jahn, & O'Shea, 2005). When considering self-report measures, depressed smokers reported higher trait anhedonia and reduced BAS reward responsiveness scores compared to depressed nonsmokers. In

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contrast to self-report measures, nicotine-satiated depressed smokers demonstrated greater acquisition of reward-based learning compared to depressed nonsmokers as indexed by the probabilistic reward task. Findings may point to a potential mechanism underlying the frequent co-occurrence of smoking and depression. These results highlight the importance of continued investigation of the role of anhedonia and reward system functioning in the co-occurrence of depression and nicotine abuse. Results also may support the use of treatments targeting reward learning (e.g., behavioral activation) to enhance smoking cessation among individuals with depression.

Keywords: depression; smoking; anhedonia; reward learning; veteran

Cigarette smoking (via its introduction of many chronic medical conditions such as cardiovascular diseases, respiratory diseases, and cancer) is the leading cause of premature death in the United States and constitutes the single most preventable cause of morbidity and mortality worldwide (Centers for Disease Control and Prevention, 2002). Smoking is responsible for enormous health and economic burdens, and is linked to over 440,000 deaths per year in the United States (Centers for Disease Control and Prevention, 2008) and approximately 5 million premature deaths annually worldwide (Warren, 2002). Epidemiological studies indicate that smoking rates are disproportionately high among samples with mental illness (Breslau, 1995), particularly those with depression (Breslau, Novak, & Kessler, 2004; Grant, Hasin, Chou, Stinson, & Dawson, 2004; Lasser et al., 2000). In fact, studies have shown that individuals with major depressive disorder (MDD) are approximately twice as likely to report smoking, compared to individuals without mental illness (35–45% versus 23%, respectively; Lasser et al.).

The association between smoking and depression appears to be bidirectional in nature. Many studies have demonstrated that smoking individuals (compared with nonsmokers) are approximately 2 to 3 times as likely to be currently depressed (Grant et al., 2004; John, Meyer, Rumpf, & Hapke, 2004) and these individuals are also at increased risk for future depression (Brown, Lewinsohn, Seeley, & Wagner, 1996). For example, Breslau, Kilbey, and Andreski (1991) reported that 39% of smokers with moderate nicotine dependence met criteria for MDD, compared to 10% of nondependent smokers. Similarly, rates of depression are higher among smokers (irrespective of dependence status) than nonsmokers (Morrell & Cohen, 2006). Specifically, smokers report higher levels of depressive symptoms (Anda et al., 1990) and experience more frequent depressive episodes (Glassman, 1993) compared to nonsmokers.

Despite the frequent and costly co-occurrence of smoking and depression, the etiological mechanisms that contribute to this relationship remain unknown (Danaei et al., 2009; Mokdad, Marks, Stroup, & Gerberding, 2004; Tsuang, Francis, Minor, Thomas, & Stone, 2012). MDD is a heterogeneous clinical condition marked by both elevations in negative affect and deficits in positive affect (i.e., anhedonia; Brown, Chorpita, & Barlow, 1998). Several studies have demonstrated a relationship between self-reported anhedonia, defined as loss of pleasure or reduced positive emotional reactivity to pleasurable stimuli, and smoking behavior (Carton, Jouvent, & Widlocher, 1994). Specifically, anhedonia has been associated with increased urge and craving to smoke, as well as poor smoking cessation outcomes (Ameringer & Leventhal, 2010; Cook, Spring, McChargue, & Doran, 2010; Leventhal, Ramsey, Brown, LaChance, & Kahler, 2008; Leventhal, Waters, Kahler, Ray, & Sussman, 2009). Thus, preliminary evidence suggests that anhedonia may play an important role in the association between these two conditions.

Dysfunction in the brain's reward system is thought to contribute to reduced hedonic capacity in depression (Dillon et al., 2009; Pizzagalli, Jahn, & O'Shea, 2005). For example, individuals with MDD show weakened responses in striatal regions (caudate, putamen, nucleus accumbens) to rewards and reward-predicting cues (Pizzagalli et al., 2009). In addition, hypoactivity in these regions has been associated with anhedonia in depression and related disorders (Elman et al., 2009; Keedwell, Andrew, Williams, Brammer, & Phillips, 2005). A crucial element of reward system functioning is the capacity to acquire reward-based learning (i.e., the ability to modify behavior in response to positive reinforcement and to learn associations among neutral stimuli and unconditioned rewards). Recent studies suggest that impairments in the ability to adjust behavior as a function of reinforcement may be an important mechanism underlying the experience of anhedonia in mood disorders (Pizzagalli, Goetz, Ostacher, Iosifescu, & Perlis, 2008; Pizzagalli, Iosifescu, Hallett, Ratner, & Fava, 2008).

Phasic signaling in midbrain dopamine neurons has been implicated in reward learning processes. Specifically, dopamine bursts have been linked to both the receipt of unpredicted rewards in early learning phases and the presence of reward-predicting cues in later