Title

Chapter Eight The Role of Depression in the Uptake and Maintenance of Cigarette Smoking

Authors

Audrain-McGovern, Janet
Leventhal, Adam M
Strong, David R

Publication Date

2015

DOI

10.1016/bs.irn.2015.07.004

Peer reviewed

HHS Public Access

Author manuscript

Int Rev Neurobiol. Author manuscript; available in PMC 2020 September 25.

Published in final edited form as:

Int Rev Neurobiol. 2015 ; 124: 209–243. doi:10.1016/bs.irn.2015.07.004.

The Role of Depression in the Uptake and Maintenance of Cigarette Smoking

Janet Audrain-McGovern[*,1], **Adam M. Leventhal**[†], **David R. Strong**[‡]

[*]Department of Psychiatry, Perelman School of Medicine, University of Pennsylvania, Philadelphia, Pennsylvania, USA

[†]Departments of Preventive Medicine and Psychology, Keck School of Medicine, University of Southern California, Los Angeles, California, USA

[‡]Department of Family Medicine and Public Health, University of California, San Diego, California, USA

Abstract

Cigarette smoking and depression both account for significant morbidity, mortality, and economic burden. The prevalence of both smoking and depression increase across mid-to-late adolescence and show high rates of comorbidity. While little is known about why smoking is disproportionately higher among depressed adolescents than adolescents without depression, emerging research has begun to offer some initial insights. The high rates of comorbidity between depression and smoking emphasize the importance of identifying intervention targets to inform smoking prevention efforts for this high-risk group. Interventions during adolescence may lessen the prevalence of depression-prone adult smokers. Depression is over-represented among adult smokers and contributes to lower smoking cessation rates. Negative mood management and pharmacotherapy have been the central focus of smoking cessation interventions for depression-prone populations to date. Converging lines of research highlight novel smoking cessation targets such as the maintenance of positive mood and reward regulation. Smoking cessation research in depression-prone smokers is critical to identify efficacious treatments that will ultimately decrease the excess smoking burden for this population.

1. THE SMOKING AND DEPRESSION PROBLEM

Cigarette smoking is the leading preventable cause of morbidity and mortality in the UnitedStates, accounting for almost 500,000 deaths each year (CDC, 2008). About 20% of adults in the general population smoke cigarettes, exacting an annual economic toll of ~193 billion dollars (CDC, 2008). About 20% of adolescents regularly smoke cigarettes (CDC, 2014), with the percentage of regular smokers doubling from mid-to-late adolescence (CDC, 2014). Of those adults who have ever smoked regularly, the majority began smoking during adolescence (USDHHS, 2012).

[1]Corresponding author: audrain@mail.med.upenn.edu.

Depression is one of the most common mental health disorders among adolescents and adults and accounts for more morbidity, mortality, and economic burden than any other psychiatric diagnosis (Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993; Murray & Lopez, 1996, 1997; Reinherz et al., 1993). About 20% of adolescents have at least one major depressive episode by 18 years of age (USDHHS, 2012). The initial onset of major depression usually occurs during mid-adolescence (Lewinsohn, Rohde, & Seeley, 1998; Lewinsohn, Rohde, Seeley, Klein, & Gotlib, 2000; Williamson, Birmaher, Axelson, Ryan, & Dahl, 2004). Another 20–30% of adolescents experience subthreshold depressive symptoms (depressive episode that does not meet diagnostic criteria for a major depression) and are considered to be at significant risk for the development of major depression, or its recurrence (Audrain-McGovern, Rodriguez, Rodgers, et al., 2012; Fergusson, Horwood, Ridder, & Beautrais, 2005; Georgiades, Lewinsohn, Monroe, & Seeley, 2006; Lewinsohn, Shankman, Gau, & Klein, 2004; Saluja et al., 2004). Research has consistently shown that the prevalence of subthreshold and major depression increase significantly during mid-to-late adolescence (Clarke et al., 2001; Costello et al., 2002; Weller & Weller, 2000).

Cigarette smoking is also over-represented in adult smokers prone to depression. About 40% of individuals with Major Depressive Disorder (MDD) smoke cigarettes (Grant, Hasin, Chou, Stinson, & Dawson, 2004), and up to 60% of smokers seeking to enroll in smoking cessation programs have had at least one lifetime episode of MDD (Brown et al., 2001; Cinciripini et al., 2005; Ginsberg, Hall, Reus, & Muñoz, 1995; Glassman et al., 1988; Hall, Munoz, & Reus, 1994; Hall et al., 1998), compared to 15% of the general population (SAMHSA, 2004). Further, 50% of smokers who enroll in smoking cessation programs have elevated depression symptoms, but do not meet diagnostic criteria for MDD (Lerman et al., 1996; Niaura et al., 2001).

Both smoking and depression increase across mid-to-late adolescence and show high rates of comorbidity. Section 2 summarizes the evidence base regarding how depression influences smoking behavior, how smoking may influence the development of depression, and the possibility that depression and smoking are associated because of common variables. The chapter also describes how individual and environmental mechanisms might shed light on a more complex relationship between adolescent depression and smoking. Parsing depression into its distinct dimensions and accounting for individual differences has the potential to further our understanding of the comorbidity. Finally, we discuss how insights from recent research might be used to develop more effective adolescent smoking prevention and intervention approaches.

Because cigarette smoking is over-represented in adults with past and current MDD, it is critical to evaluate smoking cessation approaches in this population. Section 3 summarizes the difficulties with smoking cessation among this population and the smoking cessation treatment research among adults with depression. By integrating preclinical and clinical research, we highlight potentially novel targets to improve the smoking cessation outcomes among adults with depression.

2. ADOLESCENT SMOKING UPTAKE AND DEPRESSION

Research indicates that depression contributes to adolescent smoking uptake. Major depression is associated with a 19% increase in the average daily smoking rate and a 75% increase in the odds of being nicotine dependent from mid-adolescence to young adulthood (ages 16–21 years old) (Fergusson, Goodwin, & Horwood, 2003). Adolescents with a history of major depression are two times more likely to experiment with cigarettes (Rohde, Lewinsohn, Brown, Gau, & Kahler, 2003), 50% more likely to progress to daily smoking, and significantly less likely to quit by age 25 years old compared to those without a history of major depression (Rohde et al., 2003). Longitudinal research also supports an association between smoking and subthreshold depression. Adolescents with higher depressive symptoms are more likely to initiate experimentation and to progress to regular smoking compared to adolescents with lower depressive symptoms (Escobedo, Reddy, & Giovino, 1998; Killen & Fortmann, 1997; Patton et al., 1998; Wang et al., 1999).

Studies also support the opposite direction of influence, that is, smoking appears to influence the development of subthreshold depression among adolescents. Adolescent heavy smokers are almost four times more likely to develop high depressive symptoms compared to adolescent nonsmokers (Goodman & Capitman, 2000). Similarly, smoking initiation and regular smoking have been shown to predict the development of depressive symptoms (Choi, Pierce, Gilpin, Farkas, & Berry, 1997; Goodman & Capitman, 2000; Steuber & Danner, 2006; Wu & Anthony, 1999).

Further, research has documented bidirectional influences for smoking and depression among adolescents. Smoking predicts the development of subthreshold and major depression and subthreshold and major depression predict the development of regular smoking (Brown, Lewinsohn, Seeley, & Wagner, 1996; Wang, Fitzhugh, Turner, Fu, & Westerfield, 1996; Windle & Windle, 2001). Daily smoking and nicotine dependence predict a history of major depression and a history of major depression predicts the onset of daily smoking and progression to nicotine dependence in young adults (Breslau, Kilbey, & Andreski, 1993; Breslau, Peterson, Schultz, Chilcoat, & Andreski, 1998). A more recent study found a slightly different relationship. Depression symptoms predicted smoking progression across mid-to-late adolescence while smoking uptake mitigated depression symptoms (Audrain-McGovern, Rodriguez, & Kassel, 2009). Although some studies have not found support for a reciprocal relationship (Fergusson et al., 2003; Wang et al., 1999; Wu & Anthony, 1999), a meta-analysis of 15 longitudinal studies of adolescents utilizing various measures of smoking and depression syndrome and symptoms found evidence of a bidirectional relation, with the effect of depression status on smoking likelihood (pooled Odds Ratio =1.73) being larger than the effect of smoking on depression (pooled Odds Ratio =1.41; Chaiton, Cohen, O'Loughlin, & Rehm, 2009).

While the studies noted above have examined whether the nature of the relationship between adolescent depression and smoking is causal (either unidirectional or bidirectional), the relationship may not be causal, but rather due to a common set of genetic or environmental factors that contribute to both depression and smoking. However, the available support for common genetic influences is limited (Dierker, Avenevoli, Stolar, & Merikangas, 2002;

Kendler et al., 1993; Leventhal, Ray, Rhee, & Unger, 2012; Lyons et al., 2008) and controlling for potential confounds is insufficient to explain the association between depression and smoking (Breslau et al., 1993, 1998; Chaiton et al., 2009; Fergusson et al., 2003; Martini, Wagner, & Anthony, 2002; Munafo, Hitsman, Rende, Metcalfe, & Niaura, 2008).

Methodological variability may help to account for some of the disparate findings in this body of research (Dierker et al., 2002; Johnson, Rhee, Chase, & Breslau, 2004; Kassel, Stroud, & Paronis, 2003; Munafo et al., 2008; Windle & Windle, 2001). Some of the methodological issues include different definitions of smoking or stage of smoking (initiation, regular smoking, nicotine dependence), inconsistent measures of depression and depression symptoms, large measurement intervals without repeated assessments of smoking and depression, lack of control for potential confounding variables, and only assessing one directional causal path. More recent research suggests that evaluating indirect or mediated effects between depression and smoking, heterogeneity in the impact of smoking on depression, and parsing depression into its composite dimensions may provide a richer understanding of the comorbidity.

2.1 How Does Adolescent Depression Influence Smoking Uptake?

While the comorbidity between adolescent depression and smoking has been well documented, very little is known about why smoking is disproportionately higher among depressed adolescents compared to adolescents without depression. The mechanisms that account for the comorbidity have received very little attention. While studies have begun to account for why depression influences adolescent smoking uptake, we are aware of only one study that investigated how smoking may influence the development of adolescent depression. Below, we review the available studies which provide a preliminary framework for understanding how depression may foster smoking uptake by considering the challenges associated with depression and the role of cigarette smoking in lessening these challenges. Elucidating mechanisms responsible for the comorbidity may shed light on novel smoking prevention approaches for this at-risk population.

For example, depression may make an adolescent more vulnerable to social influences to smoke, such as peers who smoke, which in turn, promotes smoking uptake. Adolescent depression is often accompanied by interpersonal difficulties including making and retaining friends (Connell & Dishion, 2006; Fergusson et al., 2003). The impact of peer smoking on adolescent smoking acquisition is well documented (Kobus, 2003). Adolescents with higher levels of depression may be more sensitive to peer behavior, more likely to select nonconventional peers, or both (Hoffman, Monge, Chou, & Valente, 2007; Kobus, 2003; Wang, Eddy, & Fitzhugh, 2000; Wills & Cleary, 1999). A greater number of smoking peers provides better access to cigarettes, promotes a normative perception of smoking, and may be a source of peer approval (Chaiton & Zhang, 2007; Ritt-Olson et al., 2005). These issues may be especially salient for youth with higher levels of depression symptoms. A prospective cohort study examined whether peer smoking accounted for the effects of depression on adolescent smoking uptake (Audrain-McGovern, Rodriguez, & Kassel, 2009). The study found that depression symptoms measured at mid-adolescence (age 14) predicted

smoking progression across mid-to-late adolescence (ages 14–18 years old). Peer smoking mediated these developmental influences such that higher depression symptoms predicted an increase in the number of smoking peers, which in turn predicted smoking progression (Audrain-McGovern, Rodriguez, & Kassel, 2009). While most smoking prevention programs address social influences to smoke, considering the unique peer-related issues faced by depressed adolescents may be necessary to protect this vulnerable group.

Converging research offers further insight into reward-related mechanisms that may increase our understanding of the link between smoking and depression among adolescents. Depression, even at the level of elevated symptoms, is often accompanied by withdrawal, less involvement in activities, and diminished reward from usual activities (Jacobson et al., 1996; Lewinsohn et al., 1998; MacPherson et al., 2010). Behavioral theory suggests that this decline in overall reinforcement creates a vulnerability for substance use, especially when substances are readily available (Higgins, Heil, & Lussier, 2004; Jacobson et al., 1996; Rogers et al., 2008). As such, behavioral treatment for depression addresses the frequency of reinforcing activities (Jacobson et al., 1996), given that depressed mood prompts a withdrawal from pleasant activities that then exacerbates depression (Lewinsohn, Youngren, & Grosscup, 1979). Research has documented the clinical relevance of alternative reinforcers (e.g., hobbies, physical activity, special interests) for substance use in general and for smoking specifically (Higgins, Heil, & Lussier, 2004). For example, a lower level of alternative reinforcers has been shown to predict adolescent smoking progression, young adult smoking status, and abstinence from other substances of abuse (Audrain-McGovern, Rodriguez, Epstein, et al., 2009; Audrain-McGovern, Rodriguez, & Kassel, 2009; Audrain-McGovern et al., 2004; Rogers et al., 2008; Van Etten, Higgins, Budney, & Badger, 1998). Based on the literature, it is plausible that depression symptoms contribute to a decline in alternative reinforcers, which in turn, increase the likelihood of smoking uptake in youth.

Within a prospective cohort study, we examined the impact that declines in alternative reinforcers can have on smoking escalation in depressed youth. We observed that higher depression symptoms in late adolescence predicted declines in alternative reinforcers across time, which in turn, predicted increases in smoking uptake and smoking rate in young adulthood (Audrain-McGovern, Rodriguez, Rodgers, & Cuevas, 2011). The results of the study documented that depression is indeed a risk factor for subsequent smoking because of its negative influence on alternative reinforcers. As alternative reinforcers decline in those with heightened depression symptoms, the reinforcing value smoking may increase as smoking may be an easy way to increase the level of pleasurable experiences (Green & Fisher, 2000; Perkins, Hickcox, & Grobe, 2000). In addition to being an easily available reinforcer, nicotine may mitigate reward deficits by enhancing the reward from those alternatives that are available in the environment (Audrain-McGovern, Wileyto, Ashare, Cuevas, & Strasser, 2014; Caggiula et al., 2009; Chaudhri et al., 2006; Spring et al., 2008). Animal models suggest that nicotine potentiates reward from drug and nondrug reinforcers by increasing the sensitivity of brain reward systems (Kenny & Markou, 2006). Promoting greater alternative reinforcers or mitigating declines may prevent smoking uptake and increases in smoking rate.

In an effort to better understand reward-related events that may occur early in the smoking acquisition process for youth with elevated depression symptoms, we examined expectations of rewards from smoking in a subsequent study (Audrain-McGovern, Rodriguez, Rodgers, et al., 2012). As elevated depression symptoms precipitate declines in involvement in other reinforcers or the pleasure derived from those reinforcers, adolescents may develop expectations that other activities, such as smoking confer reward. We hypothesized that that adolescents with elevated depression symptoms develop greater smoking reward expectations that then influence smoking progression. The study revealed that expectations of smoking reward facilitated smoking uptake among depressed adolescents. Higher depression symptoms across mid-to-late adolescence predicted a 17% increase in smoking reward expectations, which in turn predicted a 23% increase in the odds of smoking progression (Audrain-McGovern, Rodriguez, Rodgers, et al., 2012). Similarly, a study in college students found that the cross-sectional association between depression history and smoking initiation was mediated by reward expectations (McChargue, Spring, Cook, & Neumann, 2004b).

Research has shown that expectations that smoking would remove displeasure, such as reducing negative affect, predict smoking escalation in adolescents (Heinz, Kassel, Berbaum, & Mermelstein, 2010). Our study of smoking reward expectations added to this research by showing that expectations that smoking will provide pleasure motivate smoking among depressed youth. Expectations that smoking confers rewards (e.g., enjoyable, something to do when bored, stay thin) increase the likelihood that depressed adolescents will choose to smoke and, as a result, learn that smoking is rewarding. As such, their smoking experience may then serve to validate their expectations. Smoking reward expectations may identify depressed adolescents at risk of smoking. Addressing alternative ways to meet the reward expectations rather than smoking may be an important component to include in adolescent smoking prevention and cessation interventions.

2.2 Which Adolescents Are More Vulnerable?

Most multifactorial models of depression suggest that depression can be parsed into two primary affect dimensions of low positive affect (i.e., diminished levels of happiness, enjoyment, and positive moods) and high negative affect (i.e., sadness, distress) (Clark & Watson, 1991; Shafer, 2006b). Although conceptualizations of the relationship between adolescent smoking and depression have focused on smoking to relieve negative affect, there has been growing interest in the relationship between smoking and positive affect (Forbes, 2009). Adolescent smokers experience increases in positive affect and decreases in negative affect following smoking (Hedeker, Mermelstein, Berbaum, & Campbell, 2009) and greater overall affective changes following smoking predict smoking escalation (Memelstein, Hedeker, Flay, & Shiffman, 2007). Thus, low positive affect and high negative affect reflect empirically distinct processes that may both play a role in adolescent smoking.

Recent studies have shown that negative affect predicted smoking escalation among boys, but only among boys whose smoking motives were to manage affect (Weinstein & Mermelstein, 2013b). Rapid smoking escalation was associated with improved negative affect among boys (Weinstein & Mermelstein, 2013a). However, this effect was not observed

at the 15-month follow-up for boys who had a higher smoking rate and had symptoms of nicotine dependence (Weinstein & Mermelstein, 2013a). Our own findings indicate that adolescent males with higher levels of negative affect were 17% more likely to progress in their smoking across mid-to-late adolescence (Audrain-McGovern, Rodriguez, & Leventhal, 2015). Positive affect had no relation with smoking progression for males.

In contrast to males, this study found that lower positive affect coupled with higher negative affect increased the odds of smoking uptake for adolescent females by 25% (Audrain-McGovern et al., 2015). Recent studies have shown that variability in negative affect predicted smoking escalation among girls (Weinstein & Mermelstein, 2013b), and that rapid smoking escalation was associated with less negative affect variability (Weinstein & Mermelstein, 2013a). In an effort to regulate positive affect, adolescent females with may seek out potent reinforcers like smoking in order to enhance positive affect (Audrain-McGovern, Rodriguez, Leventhal, et al., 2012; Franken, Zijlstra, & Muris, 2006). While cigarettes alone may produce a mild increment in positive emotions, the ability of nicotine to enhance pleasure derived from other reinforcers (Caggiula et al., 2009; Chaudhri et al., 2006; Kenny & Markou, 2006; Spring et al., 2008) may have an important impact on positive affect among girls. These findings emphasize the importance of higher negative affect in smoking uptake for all adolescents, and highlight a unique role for positive affect in smoking uptake for female adolescents.

Hedonic capacity is another variable that may explain individual differences in adolescent smoking uptake among affectively vulnerable adolescents. Hedonic capacity is a stable dispositional ability to experience positive emotion in response to events that are typically considered rewarding (Bogdan & Pizzagalli, 2009; Meehl, 1987, 2001). The capacity to derive pleasure from natural reinforcers is a hallmark characteristic of clinical depression (anhedonia) (Shafer, 2006a; Watson, 2005). As a trait that exists on a continuum, hedonic capacity shows considerable variability among the general population (Fawcett, Clark, Scheftner, & Hedeker, 1983; Harvey, Pruessner, Czechowska, & Lepage, 2007; Meehl, 1975, 1987). At one end of the continuum are individuals who find a broad array of life experiences as rewarding and experience a high degree of pleasure in response to rewards. At the other end of the continuum are individuals who have a blunted response to typical rewarding experiences. Blunted responsiveness to natural reinforcers may lead to declines in those activities and a greater willingness to try pharmacological reinforcers for pleasure, such as cigarettes, to stimulate an under-responsive reward system. Indeed, hedonic capacity has been shown to moderate nicotine’s ability to increase positive affect in adults such that smoking a nicotinized cigarette heightened positive mood in smokers low in hedonic capacity compared to a denicotinized cigarette, but had no effect on mood in smokers higher in hedonic capacity (Cook, Spring, & McChargue, 2007). Those lower in hedonic capacity tend to place higher priority on smoking in comparison to other reinforcers (Leventhal et al., 2014; Leventhal, Waters, Kahler, Ray, & Sussman, 2009).

In a prospective cohort study, we measured hedonic capacity at age 15 years old and examined its impact on adolescent smoking 18 months later (Audrain-McGovern, Rodriguez, Leventhal, et al., 2012). We hypothesized that reduced hedonic capacity might predispose adolescents to smoke and to eventually become regular smokers. Our results

revealed that adolescents low in hedonic capacity were over two and a half times more likely to have smoked a cigarette in the past month at age 15 years old and to show a 90% increase in the rate of smoking escalation across the following 18 months compared to adolescents with high hedonic capacity. In a separate study, we found that 14-year-old never smokers reported greater expectations that smoking produces pleasure and was associated with lifetime smoking (Stone & Leventhal, 2014). Hedonic capacity appears to be a marker for a preexisting vulnerability for the early progression of smoking. Given that nicotine stimulates dopamine neurotransmission and other brain systems associated with reward, it may help adolescents with low hedonic capacity to overcome pleasure deficits by stimulating an under-responsive reward system (Barrett, Boileau, Okker, Pihl, & Dagher, 2004; Brody et al., 2004, 2009; Epping-Jordan, Watkins, Koob, & Markou, 1998). Nicotine may allow adolescents with low hedonic capacity to increase their hedonic tone and derive more pleasure from natural reinforcers in their environment. As such, this trait may be an important variable to identify youth at risk for smoking and an important population to target for smoking prevention and smoking cessation efforts.

2.3 Clinical Implications

Adolescent smoking prevention programs to date have been largely ineffective at preventing adolescent smoking (Skara & Sussman, 2003; Sussman, Lichtman, Ritt, & Pallonen, 1999). While population-based policies have been relatively more successful, almost 20% of adolescents still smoke (CDC, 2012). The adolescent smoking rate, coupled with the limited effectiveness of smoking prevention approaches to date, underscores the need to identify novel factors that drive smoking and their interactions with other variables to inform more effective smoking prevention interventions (Skara & Sussman, 2003; Sussman et al., 1999).

The high rates of comorbidity between depression and smoking emphasize the importance of targeting smoking prevention efforts to this high-risk group. There is an evidence base to support a shift in the long-standing focus of smoking prevention models from social intervention targets to affect-related targets. A shift in prevention targets would be consistent with the over-representation of smoking among affectively vulnerable populations (Audrain-McGovern, Rodriguez, & Kassel, 2009; Audrain-McGovern, Rodriguez, Rodgers, et al., 2012; Windle & Windle, 2001). The observation that affective influences on smoking are already present at age 14 years old highlights mid-adolescence as a critical period for intervention and emphasizes the importance of affect regulation skills to reduce the risk of smoking during this developmental period. Earlier interventions may mitigate the comorbidity that appears to track well into adulthood thereby decreasing the disproportionate smoking attributable morbidity and mortality in a population prone to depression.

Interventions that have depression prevention and management components could have an important impact on smoking uptake as well as subsequent depression. Social influence-based models of smoking prevention or intervention address peer influences to smoke (e.g., cigarette offer refusal skills). However, research suggests that in order to lessen peer influences on smoking behavior among depressed adolescents, it may also be important to address peer issues from the standpoint of adolescent depression (Audrain-McGovern,

Rodriguez, & Kassel, 2009). For example, addressing coping and negative mood management skills, limited social networks, need for peer approval, and accessing nonsmoking peer groups may be especially beneficial for adolescents with elevated depression symptoms.

Moreover, addressing alternative ways to meet the reward expectations of smoking may prove to be an important part of adolescent smoking prevention and cessation interventions for depressed youth (Audrain-McGovern, Rodriguez, Rodgers, et al., 2012). The expectations of smoking include a source of pleasure, something to do, a way to handle problems, a method to stay thin and look mature, and a way to relax. Although relatively less attention has been paid to these features in adolescent smoking prevention programs, counteracting these beliefs may be critical to preventing smoking uptake among youth who have elevated depression symptoms. Likewise, promoting greater alternative reinforcers to smoking or mitigating declines in alternative reinforcers may prevent smoking uptake and increases in smoking rate (Audrain-McGovern et al., 2011), as has been shown in the prevention of alcohol problems in young adults (Murphy, Dennhardt, et al., 2012; Murphy, Skidmore, et al., 2012; Reynolds, Macpherson, Tull, Baruch, & Lejuez, 2011).

For adolescents low in hedonic capacity, finding suitable alternative reinforcers may require greater effort as they will likely have a diminished response to typical alternative rewards (Audrain-McGovern, Rodriguez, Leventhal, et al., 2012). If smoking's role is to permit an adolescent to derive greater reward from natural reinforcers, it may be more effective to help these adolescents acquire skills to enhance pleasure from typical reinforcers rather than identifying reinforcers potent enough to elicit feelings of pleasure. Using Positive Psychology approaches to the treatment of depression, behavioral skills to extract greater enjoyment derived from daily reinforcers—such as mindful savoring of pleasant experiences to extend their duration and intensity—may help ameliorate the pleasure deficit associated with lower hedonic capacity (Lee Duckworth, Steen, & Seligman, 2005; Seligman, Steen, Park, & Peterson, 2005).

Increasing our understanding of the link between affect and adolescent smoking for males and females is needed as the field moves to develop the next generation of smoking prevention interventions. Our research suggests that the mitigation of negative mood for all adolescents, and the maximization of positive mood for female adolescents could prevent the reliance on cigarettes to perform these functions (Audrain-McGovern et al., 2014, 2015). Different strategies may be required to offset the smoking risk carried by low positive affect versus high negative affect (Seligman et al., 2005). Further research of gender differences in how affect influences smoking uptake is meaningful, especially in light of the fact that men and women smoke for different reasons (Perkins, 2009) and because they respond differently to specific smoking cessation pharmacotherapies (Perkins, 2001).

2.4 Conclusions

The prevalence of smoking, depression, and their comorbidity increases across mid-to-late adolescence, highlighting an important period to evaluate the onset, and the inter-relationship between smoking and depression over time. This period of adolescence may determine whether a smoking habit is established and may offer a unique opportunity to

intervene upon smoking behavior as well as depression, at an optimal time, to prevent lifelong health habits associated with morbidity and mortality

Greater understanding of the comorbidity may provide distinct etiological or intervention implications (i.e., depression is a risk factor for smoking or smoking is a risk factor for depression, or they both influence each other). If smoking is a risk factor for depression, prevention programs would need to focus on smoking to effectively reduce risk for both depression and smoking. If depression is a risk factor for smoking, prevention programs would need to focus on depression to effectively reduce the risk for both depression and smoking. Such research will help determine which one (depression or smoking) needs to be the focus of prevention efforts to effectively impact both depression and smoking or if targeting both is important.

While we are beginning to understand the mechanisms that account for the comorbidity between depression and smoking, more research is necessary. Identifying mechanisms will provide targets for smoking and/or depression prevention efforts. Evaluating moderators of the comorbidity between smoking and depression will aid in the identification of vulnerable adolescents to target for intervention. Research examining the conjoint developmental heterogeneity between smoking and depression could yield a refined understanding of the relationship between smoking and depression for specific adolescents (who needs an intervention and what type), who is at risk for adopting and maintaining smoking within the context of depression and who is at risk for developing depression within the context of smoking, when the relationship is established (optimize intervention timing), and what variables characterize at-risk groups (potential intervention content).

3. SMOKING CESSATION AND DEPRESSION

Because persistent cigarette smoking is over-represented in individuals with past and current MDD, it is critical to evaluate smoking cessation approaches in this population. Unfortunately, smokers with current MDD, and a past history of MDD are often excluded from smoking cessation treatment studies, especially those involving pharmacotherapy (Hitsman, Borrelli, McChargue, Spring, & Niaura, 2003). However, research suggests that a significant percentage of these smokers are interested in quitting and MDD severity, duration of MDD history, and recurrent MDD episodes of depression are unrelated to readiness to quit and treatment enrollment (Haug et al., 2005; Prochaska et al., 2004). In fact, depressed smokers will enter into smoking cessation interventions (if permitted) while they are in mental health treatment without adversely affecting their mental health recovery (Hall et al., 2006; Prochaska et al., 2008). Unfortunately, readiness to quit and enrollment in smoking cessation programs among smokers with past or current MDD have not consistently translated to improved smoking cessation outcomes for treatment approaches to date (Hall & Prochaska, 2009).

A history of MDD lowers the odds of short-term and long-term smoking abstinence by about 20% (Hitsman et al., 2013). In addition, pretreatment negative mood and increases in negative mood in the early stages of treatment are predictive of inability to quit and of smoking relapse (Berlin & Covey, 2006; Hall et al., 1994, 1996; Strong et al., 2009). These

mood disturbances tend to be more common among those smokers with a history of MDD (Ginsberg et al., 1995; Hall et al., 1994, 1996; Killen et al., 2004; Levine, Marcus, & Perkins, 2003; Tsoh & Hall, 2004). This is not surprising given that 30–50% of those who remit from an episode of MDD will continue to experience depressive symptomatology (Bothwell & Scott, 1997; Fawcett, 1994; Nierenberg et al., 1999). As such, several studies have focused on treating depression symptoms and negative mood as well as preventing their emergence in smokers with a past history of MDD (single and recurrent) in order to optimize smoking cessation outcomes.

3.1 Smoking Cessation Treatment Outcomes in Smokers with Depression

The hypothesis that smokers with a history of MDD may be less likely to quit smoking successfully because of smoking cessation related exacerbations of negative mood has led to a series of investigations of mood management smoking cessation interventions for this population, with and without pharmacotherapy. These mood management smoking cessation interventions focused on preventing increases in negative mood following cessation or mitigating the impact of negative mood on smoking relapse. These studies are reviewed below.

3.2 Behavioral Therapy for Smoking Cessation: Mood Management

Hall and colleagues were among the first to investigate whether mood management smoking cessation treatment (MM) was more beneficial for smokers with a history of MDD compared to smokers without a history of MDD. The initial study compared 10 sessions of MM to 5 sessions of standard smoking cessation treatment (ST) in 46 smokers with a positive history of MDD compared to 103 smokers with no history of MDD (Hall et al., 1994). All smokers received nicotine gum therapy. Smokers with a history of MDD who received the MM treatment achieved higher rates of continuous abstinence than those in the ST group (34% vs. 18%), although MM did not impact negative mood after quitting. In a follow-up study with 201 smokers (44 with a past history of MDD), where treatment contact time was equated (i.e., 10 sessions for MM and control), the MM treatment was not significantly better than a control group among those smokers with a history of MDD (29% vs. 26% at 6-month follow-up) (Hall et al., 1996). A third study by Hall and colleagues compared the antidepressant nortriptyline to placebo in a group of smokers with (n =65) and without (n =134) a history of MDD who either received 10 sessions of MM or 5 sessions of a health education control condition (Hall et al., 1998). Nortriptyline was not more effective for those with a past history of MDD, but the MM condition was more effective at promoting smoking cessation than the control condition at end of treatment (41% vs. 19%) but not at the 26-week follow-up (18% vs. 13%). The mixed results of these studies suggest that smokers with a history of a single episode of MDD have better smoking cessation outcomes if they receive more behavioral treatment, but the benefits of mood management were more advantageous among smokers with recurrent MDD (Haas, Muñoz, Humfleet, Reus, & Hall, 2004).

Using a sample consisting solely of smokers with a history of MDD (n =179), Brown and colleagues (2001) evaluated whether MM enhanced the effects of ST on smoking cessation rates. Both groups received eight, 2-h sessions over 6 weeks. There were no significant

differences in smoking abstinence at the end of treatment or at any of the posttreatment time points among the treatment groups (e.g., 25% of ST participants and 24% of MM participants were quit at the 6-month follow-up). MM was more effective at promoting smoking cessation among those smokers (n =98) with a history of recurrent MDD in secondary analyses (Brown et al., 2001), but not effective for those with a history of single episode of MD. MM did not influence negative mood, and in fact was associated with increased depressive symptoms, negative mood, risk of subsequent MDD episode, and withdrawal symptoms after smoking cessation (Kahler et al., 2002).

In a subsequent study, Brown and colleagues examined whether the combination of bupropion, an antidepressant, and MM was more effective at promoting cessation among smokers with a history of MDD (N =92) and smokers with elevated depression symptoms (N = ~60) compared to MM and placebo, ST and placebo or ST and bupropion (Brown et al., 2007). Neither MM nor bupropion, alone or in combination, was more effective at promoting smoking cessation for smokers with a history of MDD (single or recurrent) or smokers with elevated depression symptoms compared to smokers with no past history of MDD and smokers with low depression symptoms (N = ~372). Risk of lapse or relapse did not differ among the ST and MM conditions. In a recent meta-analysis (Gierisch, Bastian, Calhoun, McDuffie, & Williams, 2012; van der Meer, Willemsen, Smit, & Cuijpers, 2013), adding mood management to standard behavioral treatments led to significantly improved cessation outcomes among smokers with past depression (13 trials, N =1496, RR 1.41, 95% CI 1.13–1.77). Thus, there is promise for mood management approaches to improve cessation outcomes and a critical need to develop and evaluate specialized behavioral smoking cessation interventions that address the unique needs of smokers with a history of MDD, current MDD, or elevated depression symptoms. Several clinical trials of behavioral interventions that address various affective targets have shown more promising findings (Cinciripini et al., 2010; MacPherson et al., 2010; van der Meer et al., 2013; van der Meer, Willemsen, Smit, Cuijpers, & Schippers, 2010).

3.3 Pharmacotherapy for Smoking Cessation

Smokers with a history of MDD or depression symptoms have been treated with various pharmacological interventions, with a focus on antidepressant therapy. The explicit assumption is that antidepressant medication would offset any increases in negative mood or depression symptoms that may arise following smoking cessation, and therefore promote successful quitting. The implicit assumption is that antidepressants have the same functional role as nicotine, and therefore, may replace nicotine's mood regulation effects (Hall, 2007). Several antidepressants have been evaluated (e.g., sertraline, nortriptyline, bupropion). Overall, these medications have a modest benefit for smoking cessation in smokers with depression (see van der Meer et al., 2013 for a meta-analysis), and none have demonstrated greater efficacy for smokers with a history of MDD compared to smokers without a history of MDD (Catley et al., 2005; Covey, Glassman, Stetner, Rivelli, & Stage, 2002; Hall et al., 1994, 1998, 2002; Hughes, Stead, & Lancaster, 2007; Prochaska et al., 2004). That is, antidepressants do not appear to offset the increased risk of relapse in depressed versus non-depressed smokers. In addition, the effect of antidepressant therapy on negative mood does not necessarily translate to improved smoking cessation outcomes (Brown et al., 2007;

Cinciripini et al., 2005; Evins et al., 2008; Hall et al., 1998); however, see Strong et al. (2009).

Surprisingly, there are few larger-scale smoking cessation investigations that included transdermal nicotine (TN) in smokers with current or past history of MDD (Evins et al., 2008; Hall et al., 2006; Smith et al., 2003). Hall and colleagues recruited 322 smokers with current MDD and randomized these smokers to either receive a brief contact control intervention (i.e., self-help guide, smoking cessation treatment referral list) or a stepped care intervention involving computerized motivational smoking feedback, 6 sessions of psychological counseling which included mood monitoring and management, and TN (Hall et al., 2006). Participants in the staged care condition were only slightly more likely to be smoking abstinent at 3-months (16% vs. 12%) and 6 months (21% vs. 18%) posttreatment compared to participants in the brief contact control intervention.

Nicotine gum use combined with brief therapy has been shown to help smokers with elevated depression symptoms achieve 12-month smoking cessation rates that are comparable to smokers with fewer depressive symptoms (15% vs. 20%) and more than doubled the 3-month quit rates (30% vs. 13%) and the 12-months quit rates (15% vs. 6%) compared to smokers with elevated depression symptoms who received placebo gum (Kinnunen, Doherty, Militello, & Garvey, 1996; Kinnunen, Korhonen, & Garvey, 2008). Nicotine gum use was also associated with a reduction in depression symptoms postcessation among smokers with elevated depression symptoms. It is unknown what proportion of smokers with high depression symptoms had a history of MD, as MDD history was not assessed.

Lastly, a recent study evaluated whether bupropion enhanced smoking cessation rates and reduced abstinence-induced depressive symptoms when added to TN therapy and standard smoking cessation counseling in 199 smokers with current or past MDD (Evins et al., 2008). About 30% of participants were abstinent at the end of 13 weeks of treatment. Bupropion did not improve the efficacy of TN and smoking cessation counseling, nor did bupropion mitigate the abstinence-induced increase in depression symptoms. Importantly, however, TN use was associated with abstinence, such that each patch used increased the likelihood of abstinence by 6% (Evins et al., 2008).

3.4 Summary

Evidence suggests that most negative mood management interventions may not significantly improve smoking cessation rates or mitigate negative mood among smokers with a history of MDD or smokers with higher levels of negative mood (Brown et al., 1996; Haas et al., 2004; Hall et al., 1994; Kahler et al., 2002; Strong et al., 2009). In fact, some negative mood management interventions can exacerbate negative mood, depression symptoms, withdrawal symptoms and increase risk of a major depression episode (Kahler et al., 2002). Antidepressants tend not to offset negative mood or promote smoking cessation among smokers with current or historic MDD (Brown et al., 2007; Cinciripini et al., 2005; Evins et al., 2008; Hall et al., 1998).

It has been assumed that smoking cessation will result in increases in depressive symptoms in all smokers with a history of MDD, but there appears to be significant heterogeneity in depressive symptoms during quitting. About 40% of smokers with a history of MDD have increased depressive symptoms during the 2 weeks following quitting, while other smokers have stable or decreasing depression symptoms (Burgess et al., 2002). The receipt of negative mood management smoking cessation interventions appears to be unrelated to the course of depressive symptoms during smoking cessation (Burgess et al., 2002) and increased depressive symptoms are associated with lower smoking abstinence rates (Burgess et al., 2002). Further, depression-prone smokers experience lower positive affect and higher negative affect on quit day (Strong et al., 2009). As we discuss below, smoking cessation interventions for this vulnerable population may be significantly more effective if positive mood, rather than negative mood is the focus of treatment (Miller et al., 1999; Miller & Miller, 2009; Nutt et al., 2007). In addition, the benefits of nicotine replacement therapy or varenicline have been understudied in this population, but may hold promise in promoting smoking cessation among smokers with past and current MDD, as well as smokers with high levels of depression symptoms (Cook, Spring, McChargue, & Hedeker, 2004; Fiore et al., 2008).

4. TARGETING POSITIVE MOOD MAY INCREASE SMOKING CESSATION TREATMENT EFFECTIVENESS

The relationship between changes in positive mood and the ability to quit smoking and avoid relapse has received far less attention than negative mood. Positive mood is distinct from negative mood and not simply the opposite end of a single mood continuum (Cook et al., 2004; Davidson et al., 2002; Watson, Clark, & Carey, 1988). We are aware of only one mood management smoking cessation treatment study that assessed positive mood as well as negative mood (Strong et al., 2009) in smokers with a past history of MDD. Participants received negative mood management or standard smoking cessation counseling in combination with bupropion or placebo (Brown et al., 2007). Positive mood declined significantly prior to the quit date, irrespective of receiving bupropion or negative mood management smoking cessation treatment. Decreases in positive mood prior to quit day and lower quit day levels of positive mood were significant predictors of smoking lapse, shorter time to relapse, and overall relapse risk (Strong et al., 2009). Increases in negative mood also predicted smoking lapse (Strong et al., 2009). Low positive mood and high negative mood after quitting were found to predict relapse in a study of smokers making an unaided quit attempt (Strong et al., 2011). Although not yet investigated, the dys-regulation of positive mood may exacerbate the relapse risk associated with increased negative mood. Depression treatment research has shown that targeting positive mood increases positive mood and decreases negative mood in outpatients with MDD, although the converse does not appear to be true (Seligman, Rashid, & Parks, 2006; Seligman et al., 2005).

Declines in positive mood may be especially relevant for smokers with current or a past history of MDD. These individuals tend to be less attentive to sources of reinforcement (Forbes et al., 2009; Pizzagalli et al., 2009; Pizzagalli, Iosifescu, Hallett, Ratner, & Fava, 2008), have fewer alternative reinforcers (Audrain-McGovern et al., 2014; Jacobson et al.,

1996; MacPhillamy & Lewinsohn, 1974), and derive less reward from natural reinforcers in their environment that typically increase positive mood (Dunn, Dalgleish, Lawrence, Cusack, & Ogilvie, 2004; Forbes, 2009; Forbes, Shaw, & Dahl, 2007; Pizzagalli et al., 2008; Shankman, Klein, Tenke, & Bruder, 2007; Sloan, Strauss, Quirk, & Sajatovic, 1997; Wichers et al., 2009). As such, positive mood is usually deficient in depression-prone persons (Clark, Watson, & Mineka, 1994; Forbes et al., 2006, 2009; Nutt et al., 2007; Wichers et al., 2009).

Research suggests three mechanisms by which nicotine ameliorates these deficits that characterize depression to increase positive mood (Audrain-McGovern et al., 2014; Perkins et al., 2006; Stein et al., 1998; Warburton & Mancuso, 1998); each of these can be considered a target for a smoking cessation intervention. One, nicotine increases the salience of rewarding stimuli in the environment (Mancuso, Warburton, Melen, Sherwood, & Tirelli, 1999; Powell, Tait, & Lessiter, 2002) and general attention (Evans & Drobes, 2009; Heishman, Kleykamp, & Singleton, 2010). Nicotine withdrawal is associated with declines in general attention (Patterson et al., 2010; Rukstalis, Jepson, Patterson, & Lerman, 2005). Two, smoking is a reinforcer itself and can compensate for limited alternative reinforcers (Audrain-McGovern, Rodriguez, Epstein, et al., 2009; Audrain-McGovern et al., 2011, 2014; Perkins et al., 2000). Depression-prone smokers may find smoking especially reinforcing as evidenced by greater smoking-induced dopamine release compared to smokers not prone to depression (Brody et al., 2009). Three, nicotine enhances reward from available drug and nondrug reinforcers by increasing reward sensitivity or the ability to experience pleasure (Audrain-McGovern et al., 2015; Barr, Pizzagalli, Culhane, Goff, & Evins, 2008; Dawkins, Acaster, & Powell, 2007; Kenny & Markou, 2006; Perkins, Karelitz, Giedgowd, & Conklin, 2013; Spring et al., 2008). Nicotine withdrawal decreases reward sensitivity (Epping-Jordan et al., 1998; Johnson, Hollander, & Kenny, 2008). Thus, DP + smokers may be less able to sustain smoking abstinence because they lose the reward-related effects that were supported by nicotine and experience diminished positive mood. In fact, expectations of positive effects of nicotine have been shown to mediate the relationship between history of depression and smoking behaviors (McChargue, Spring, Cook, & Neumann, 2004a). Declines in positive mood after quitting smoking have been observed in smokers (Cook et al., 2004, 2007; McChargue et al., 2004a) and linked to smoking relapse (al'Absi, Hatsukami, Davis, & Wittmers, 2004; Strong et al., 2009). Furthermore, anhedonia has been shown to predict preference for smoking versus alternative rewards (Cook et al., 2007; Leventhal, Ameringer, Osborn, Zvolensky, & Langdon, 2013; Leventhal, Munafo, et al., 2012; Leventhal et al., 2009, 2014). In addition, anhedonic smokers exhibit greater abstinence-provoked decreases in positive mood (Cook et al., 2007; Leventhal et al., 2013), diminution in reward processing (Leventhal, Ray, et al., 2012), and escalation in urge to smoke for pleasure (Leventhal et al., 2009).

Thus, smoking cessation intervention for depression-prone smokers that target behavioral skills to increase attention to opportunities for reward, identify and engage in reinforcing activities, and maximize enjoyment of those activities to maintain or enhance positive mood may increase the likelihood of quitting success. Such an intervention would integrate the functional role of nicotine (i.e., increases positive mood, greater salience to environmental rewards, increases enjoyment from available rewards) and the phenomenology of depression

(i.e., less positive mood, less attention to opportunities for reward, fewer alternative reinforcers, difficulty deriving enjoyment from rewards). Preliminary research has shown that scheduling pleasant events can facilitate smoking cessation in smokers with elevated depression symptoms (Brown et al., 2001; Hall et al., 1996; MacPherson et al., 2010); however, long-term abstinence may require attention to the quality of the alternative reinforcers selected and the level of enjoyment derived from these alternative reinforcers. Initial research suggests that positive psychology interventions that enhance skills for obtaining positive affect and enhancing the duration and intensity of pleasurable and meaningful experiences may benefit smoking cessation outcomes (Kahler et al., 2014, 2015). Skills to magnify or savor enjoyment derived from alternative reinforcers may help replicate the reward enhancement that accompanies nicotine.

Smoking cessation treatments that incorporate nicotine replacement therapy or varenicline while concurrently addressing the positive mood changes through behavioral counseling may prove especially effective for promoting smoking cessation among smokers with current and a past history of MDD. Behavioral skills to increase attention to opportunities for reward, identify, and engage in alternative reinforcers, and maximize enjoyment derived from those alternative reinforcers may prevent a decline in positive mood and increase likelihood of successful short-term and long-term smoking cessation for smokers with current as well as past history of MD.

5. INDIVIDUAL DIFFERENCES IN CESSATION OUTCOME AND TREATMENT EFFECTIVENESS

In terms of depression as a prognosticative factor of cessation outcome irrespective of treatment type, results from a recent meta-analysis suggest that past depression carries modest risk of elevated cessation failure (Hitsman al., 2013). Evidence that current or recent depression or depressive symptoms predict poor cessation outcomes is more consistent (al'Absi, Hatsukami, & Davis, 2005; al'Absi et al., 2004; Anda et al., 1990; Brown et al., 2001; Catley, Ahluwalia, Resnicow, & Nazir, 2003; Catley et al., 2005; Cinciripini et al., 2003; Japuntich et al., 2007; Leventhal, Japuntich, et al., 2012; Leventhal, Ramsey, Brown, LaChance, & Kahler, 2008; Piper et al., 2010; Schnoll, Leone, & Hitsman, 2013). Furthermore, evidence suggests that variation at subclinical levels of depression, even the distinction between zero versus one symptom (Niaura et al., 2001), predict poorer cessation outcomes (Leventhal et al., 2008) in smokers without current major depression (al'Absi et al., 2004, 2005; Brown et al., 2001; Cinciripini et al., 2003; Ginsberg et al., 1995; Haas et al., 2004; Killen, Fortmann, Davis, Strausberg, & Varady, 1999; Swan et al., 2003). Importantly, there is heterogeneity among smokers with depressive characteristics that explains variance in smoking cessation outcomes, as relapse risk is higher in individuals with recurrent (vs. single episode) forms of depression (Brown et al., 2001, 2007; Haas et al., 2004; Leventhal et al., 2014), assessed using brief depression screeners versus clinical interviews (Hitsman et al., 2013), and with anhedonia versus other types of depressive symptoms (Cook, Spring, McChargue, & Doran, 2010; Leventhal et al., 2008, 2014; Niaura et al., 2001). With regard to the specific symptom types that predict outcome, anhedonia and low positive affect have shown incremental prediction over and above other factors,

including depressed mood, depression proneness, somatic features, personality traits, anxiety disorders, and substance use disorders (Cook et al., 2010; Japuntich et al., 2007; Leventhal, Munafo, et al., 2012; Leventhal et al., 2014). Thus, absence of reward and positive affect may be an important facet of depression that contributes to difficulty quitting smoking.

Individual differences in response to smoking cessation treatments in smokers with current or a past history of MDD have gone largely unexplored. One potential influence on smoking cessation treatment effectiveness in smokers with a history of MDD is hedonic capacity. As noted earlier, hedonic capacity is a stable dispositional ability to experience positive mood in response to things that are typically rewarding (Clark, Fawcett, Salazar-Grueso, & Fawcett, 1984; Cook et al., 2004; Meehl, 1987). Low hedonic capacity, measured by validated questionnaire predicts heightened urge to smoke after smoking abstinence (Cook et al., 2004) and moderates nicotine's ability to increase positive mood (Cook et al., 2007). Research indicates variability in hedonic capacity among depression-prone smokers (Cook et al., 2007; Leventhal, Chasson, Tapia, Miller, & Pettit, 2006), with lower levels predicting inability to maintain abstinence for greater than 24 h (Leventhal et al., 2009).

One could also speculate that aspects of MDD itself may differentially influence response to smoking cessation treatment. There is evidence for only one variable, MDD recurrence appears to modify treatment effects. Mood management smoking cessations interventions appear to be more effective at promoting smoking cessation among smokers with a history of recurrent MDD than those with single episode (Brown et al., 2001; Haas et al., 2004). Severity of MDD or a recent MDD episode at smoking treatment onset does not predict smoking outcomes (Hall et al., 2006).

6. CONCLUSIONS

The comorbidity between smoking and MDD is well established (Grant et al., 2004; Hitsman et al., 2013). Smokers with current or past MDD are often excluded from smoking cessation treatment studies, especially those involving pharmacotherapy. Only a few studies have included samples comprised solely of smokers with current or a past history of MDD. As a result, we lack studies to guide smoking cessation treatment decision making for this under-served population. In order to determine the most effective elements of pharmacological and psychological smoking cessation interventions for smokers with current or past MDD, research needs to focus on this population (Wilhelm, Wedgwood, Niven, & Kay-Lambkin, 2006).

The practice guidelines for smoking cessation (Fiore et al., 2008), recommend that smokers with comorbid psychiatric conditions be provided with the same smoking cessation treatments as smokers in the general population, but acknowledges that psychiatric comorbidity could affect the acceptability, use and effectiveness of smoking cessation treatment. These guidelines also emphasize that current evidence is insufficient to determine whether smokers with psychiatric conditions benefit more from tobacco use treatments focused on unique aspects of their condition or symptoms than from standard smoking cessation treatments (Fiore & Jaen, 2008; Hall, 2007). Negative mood management smoking cessation interventions and antidepressant pharmacotherapy have shown modest effects on

smoking cessation rates, at best and tend not to mitigate negative mood among smokers with a history of MDD or smokers with higher levels of negative mood (Brown et al., 2007; Covey et al., 2002; Evins et al., 2008; Hall et al., 1994; Kahler et al., 2002; Strong et al., 2009).

Thus, although negative mood after quitting smoking has been a hypothesized pathway through which smokers with MDD are unable to sustain smoking abstinence, converging lines of research suggest that smokers with MDD are unable to become or sustain smoking abstinence because they experience a loss in positive mood and reward regulation that was supported by nicotine (Audrain-McGovern et al., 2014). Smoking cessation interventions focused on positive mood and reward enhancement would answer the call to shift the emphasis of addiction treatment from the suppression of negative mood and substance use behavior to the promotion of positive mood and alternative nondrug reinforcers (Miller & Miller, 2009). It is thought that the latter holds greater promise for making substance use a less likely choice among several rewarding alternatives (Miller & Miller, 2009). Such interventions should evaluate mechanisms of treatment effects and explore who may benefit most from such an intervention. Evidence-based treatments will help inform treatment planning by evaluating the effectiveness of standard smoking cessation counseling compared to novel smoking cessation approaches that integrate the role of nicotine and smoking in normalizing symptoms that are associated with depression.

REFERENCES

al'Absi M, Hatsukami D, & Davis GL (2005). Attenuated adrenocorticotropic responses to psychological stress are associated with early smoking relapse. Psychopharmacology, 181, 107–117. [PubMed: 15834539]

al'Absi M, Hatsukami D, Davis GL, & Wittmers LE (2004). Prospective examination of effects of smoking abstinence on cortisol and withdrawal symptoms as predictors of early smoking relapse. Drug and Alcohol Dependence, 73, 267–278. [PubMed: 15036549]

Anda RF, Williamson DF, Escobedo LG, Mast EE, Giovino GA, & Remington PL (1990). Depression and the dynamics of smoking. A national perspective. The Journal of the American Medical Association, 264, 1541–1545. [PubMed: 2395193]

Audrain-McGovern J, Rodriguez D, Epstein LH, Rodgers K, Cuevas J, & Wileyto EP (2009). Young adult smoking: What factors differentiate ex-smokers, smoking cessation treatment seekers and nontreatment seekers? Addictive Behaviors, 34, 1036–1041. [PubMed: 19619948]

Audrain-McGovern J, Rodriguez D, & Kassel JD (2009). Adolescent smoking and depression: Evidence for self-medication and peer smoking mediation. Addiction, 104, 1743–1756. [PubMed: 19549056]

Audrain-McGovern J, Rodriguez D, & Leventhal AM (2015). Gender differences in the relationship between affect and adolescent smoking uptake. Addiction, 110, 519–529. [PubMed: 25393395]

Audrain-McGovern J, Rodriguez D, Leventhal AM, Cuevas J, Rodgers K, & Sass J (2012). Where is the pleasure in that? Low hedonic capacity predicts smoking onset and escalation. Nicotine & Tobacco Research, 14, 1187–1196. [PubMed: 22387990]

Audrain-McGovern J, Rodriguez D, Rodgers K, & Cuevas J (2011). Declining alternative reinforcers link depression to young adult smoking. Addiction, 106, 178–187. [PubMed: 20840206]

Audrain-McGovern J, Rodriguez D, Rodgers K, Cuevas J, Sass J, & Riley T (2012). Reward expectations lead to smoking uptake among depressed adolescents. Drug and Alcohol Dependence, 120, 181–189. [PubMed: 21855235]

Audrain-McGovern J, Rodriguez D, Tercyak KP, Cuevas J, Rodgers K, & Patterson F (2004). Identifying and characterizing adolescent smoking trajectories. Cancer Epidemiology, Biomarkers & Prevention, 13, 2023–2034.

Audrain-McGovern J, Wileyto EP, Ashare R, Cuevas J, & Strasser AA (2014). Reward and affective regulation in depression-prone smokers. Biological Psychiatry, 76, 689–697. [PubMed: 24947541]

Barr RS, Pizzagalli DA, Culhane MA, Goff DC, & Evins AE (2008). A single dose of nicotine enhances reward responsiveness in nonsmokers: Implications for development of dependence. Biological Psychiatry, 63, 1061–1065. [PubMed: 17976537]

Barrett SP, Boileau I, Okker J, Pihl RO, & Dagher A (2004). The hedonic response to cigarette smoking is proportional to dopamine release in the human striatum as measured by positron emission tomography and [11C]raclopride. Synapse, 54, 65–71. [PubMed: 15352131]

Berlin I, & Covey LS (2006). Pre-cessation depressive mood predicts failure to quit smoking: The role of coping and personality traits. Addiction, 101, 1814–1821. [PubMed: 17156181]

Bogdan R, & Pizzagalli DA (2009). The heritability of hedonic capacity and perceived stress: A twin study evaluation of candidate depressive phenotypes. Psychological Medicine, 39, 211–218. [PubMed: 18507876]

Bothwell R, & Scott J (1997). The influence of cognitive variables on recovery in depressed inpatients. Journal of Affective Disorders, 43, 207–212. [PubMed: 9186791]

Breslau N, Kilbey MM, & Andreski P (1993). Nicotine dependence and major depression. New evidence from a prospective investigation. Archives of General Psychiatry, 50, 31–35. [PubMed: 8422219]

Breslau N, Peterson EL, Schultz LR, Chilcoat HD, & Andreski P (1998). Major depression and stages of smoking. A longitudinal investigation. Archives of General Psychiatry, 55, 161–166. [PubMed: 9477930]

Brody AL, Olmstead RE, Abrams AL, Costello MR, Khan A, Kozman D, et al. (2009). Effect of a history of major depressive disorder on smoking-induced dopamine release. Biological Psychiatry, 66, 898–901. [PubMed: 19640507]

Brody AL, Olmstead RE, London ED, Farahi J, Meyer JH, Grossman P, et al. (2004). Smoking-induced ventral striatum dopamine release. The American Journal of Psychiatry, 161, 1211–1218. [PubMed: 15229053]

Brown RA, Kahler CW, Niaura R, Abrams DB, Sales SD, Ramsey SE, et al. (2001). Cognitive-behavioral treatment for depression in smoking cessation. Journal of Consulting and Clinical Psychology, 69, 471–480. [PubMed: 11495176]

Brown RA, Lewinsohn PM, Seeley JR, & Wagner EF (1996). Cigarette smoking, major depression, and other psychiatric disorders among adolescents. Journal of the American Academy of Child & Adolescent Psychiatry, 35, 1602–1610. [PubMed: 8973066]

Brown RA, Niaura R, Lloyd-Richardson EE, Strong DR, Kahler CW, Abrantes AM, et al. (2007). Bupropion and cognitive-behavioral treatment for depression in smoking cessation. Nicotine & Tobacco Research, 9, 721–730. [PubMed: 17577801]

Burgess ES, Brown RA, Kahler CW, Niaura R, Abrams DB, Goldstein MG, et al. (2002). Patterns of change in depressive symptoms during smoking cessation: Who's at risk for relapse? Journal of Consulting and Clinical Psychology, 70, 356–361. [PubMed: 11952193]

Caggiula AR, Donny EC, Palmatier MI, Liu X, Chaudhri N, & Sved AF (2009). The role of nicotine in smoking: A dual-reinforcement model. Nebraska Symposium on Motivation, 55, 91–109. [PubMed: 19013940]

Catley D, Ahluwalia JS, Resnicow K, & Nazir N (2003). Depressive symptoms and smoking cessation among inner-city African Americans using the nicotine patch. Nicotine & Tobacco Research, 5, 61–68. [PubMed: 12745507]

Catley D, Harris KJ, Okuyemi KS, Mayo MS, Pankey E, & Ahluwalia JS (2005). The influence of depressive symptoms on smoking cessation among African Americans in a randomized trial of bupropion. Nicotine & Tobacco Research, 7, 859–870. [PubMed: 16298721]

CDC. (2008). Smoking attributable mortality, years of potential life lost, and productivity losses. United States, 2000–2004. Morbidity and Mortality Weekly Report, 57, 1226–1228. [PubMed: 19008791]

CDC. (2012). Youth risk behavior surveillance—United States, 2011. Morbidity and Mortality Weekly Report. Surveillance Summaries, 61, 1–162.

CDC. (2014). National Youth Tobacco Survey (NYTS). http://www.cdc.gov/TOBACCO/data_statistics/surveys/NYTS/index.htm. accessed 25.02.15.

Chaiton MO, Cohen JE, O'Loughlin J, & Rehm J (2009). A systematic review of longitudinal studies on the association between depression and smoking in adolescents. BMC Public Health, 9, 356. [PubMed: 19772635]

Chaiton MO, & Zhang B (2007). Environment modifies the association between depression symptoms and smoking among adolescents. Psychology of Addictive Behaviors, 21, 420–424. [PubMed: 17874894]

Chaudhri N, Caggiula AR, Donny EC, Palmatier MI, Liu X, & Sved AF (2006). Complex interactions between nicotine and nonpharmacological stimuli reveal multiple roles for nicotine in reinforcement. Psychopharmacology, 184, 353–366. [PubMed: 16240165]

Choi WS, Pierce JP, Gilpin EA, Farkas AJ, & Berry CC (1997). Which adolescent experimenters progress to established smoking in the United States. American Journal of Preventive Medicine, 13, 385–391. [PubMed: 9315272]

Cinciripini PM, Blalock JA, Minnix JA, Robinson JD, Brown VL, Lam C, et al. (2010). Effects of an intensive depression-focused intervention for smoking cessation in pregnancy. Journal of Consulting and Clinical Psychology, 78, 44–54. [PubMed: 20099949]

Cinciripini PM, Tsoh JY, Wetter DW, Lam C, de Moor C, Cinciripini L, et al. (2005). Combined effects of venlafaxine, nicotine replacement, and brief counseling on smoking cessation. Experimental and Clinical Psychopharmacology, 13, 282–292. [PubMed: 16366758]

Cinciripini PM, Wetter DW, Fouladi RT, Blalock JA, Carter BL, Cinciripini LG, et al. (2003). The effects of depressed mood on smoking cessation: Mediation by postcessation self-efficacy. Journal of Consulting and Clinical Psychology, 71, 292–301. [PubMed: 12699023]

Clark DC, Fawcett J, Salazar-Grueso E, & Fawcett E (1984). Seven-month clinical outcome of anhedonic and normally hedonic depressed inpatients. The American Journal of Psychiatry, 141, 1216–1220. [PubMed: 6486255]

Clark LA, & Watson D (1991). Tripartite model of anxiety and depression: Psychometric evidence and taxonomic implications. Journal of Abnormal Psychology, 100, 316–336. [PubMed: 1918611]

Clark LA, Watson D, & Mineka S (1994). Temperament, personality, and the mood and anxiety disorders. Journal of Abnormal Psychology, 103, 103–116. [PubMed: 8040472]

Clarke GN, Hornbrook M, Lynch F, Polen M, Gale J, Beardslee W, et al. (2001). A randomized trial of a group cognitive intervention for preventing depression in adolescent offspring of depressed parents. Archives of General Psychiatry, 58, 1127–1134. [PubMed: 11735841]

Connell AM, & Dishion TJ (2006). The contribution of peers to monthly variation in adolescent depressed mood: A short-term longitudinal study with time-varying predictors. Development and Psychopathology, 18, 139–154. [PubMed: 16478556]

Cook JW, Spring B, & McChargue D (2007). Influence of nicotine on positive affect in anhedonic smokers. Psychopharmacology, 192, 87–95. [PubMed: 17277935]

Cook J, Spring B, McChargue D, & Doran N (2010). Effects of anhedonia on days to relapse among smokers with a history of depression: A brief report. Nicotine & Tobacco Research, 12, 978–982. [PubMed: 20709727]

Cook JW, Spring B, McChargue D, & Hedeker D (2004). Hedonic capacity, cigarette craving, and diminished positive mood. Nicotine & Tobacco Research, 6, 39–47. [PubMed: 14982686]

Costello EJ, Pine DS, Hammen C, March JS, Plotsky PM, Weissman MM, et al. (2002). Development and natural history of mood disorders. Biological Psychiatry, 52, 529–542. [PubMed: 12361667]

Covey LS, Glassman AH, Stetner F, Rivelli S, & Stage K (2002). A randomized trial of sertraline as a cessation aid for smokers with a history of major depression. The American Journal of Psychiatry, 159, 1731–1737. [PubMed: 12359680]

Davidson RJ, Lewis DA, Alloy LB, Amaral DG, Bush G, Cohen JD, et al. (2002). Neural and behavioral substrates of mood and mood regulation. Biological Psychiatry, 52, 478–502. [PubMed: 12361665]

Dawkins L, Acaster S, & Powell JH (2007). The effects of smoking and abstinence on experience of happiness and sadness in response to positively valenced, negatively valenced, and neutral film clips. Addictive Behaviors, 32, 425–431. [PubMed: 16824689]

Dierker LC, Avenevoli S, Stolar M, & Merikangas KR (2002). Smoking and depression: An examination of mechanisms of comorbidity. The American Journal of Psychiatry, 159, 947–953. [PubMed: 12042182]

Dunn BD, Dalgleish T, Lawrence AD, Cusack R, & Ogilvie AD (2004). Categorical and dimensional reports of experienced affect to emotion-inducing pictures in depression. Journal of Abnormal Psychology, 113, 654–660. [PubMed: 15535797]

Epping-Jordan MP, Watkins SS, Koob GF, & Markou A (1998). Dramatic decreases in brain reward function during nicotine withdrawal. Nature, 393, 76–79. [PubMed: 9590692]

Escobedo LG, Reddy M, & Giovino GA (1998). The relationship between depressive symptoms and cigarette smoking in US adolescents. Addiction, 93, 433–440. [PubMed: 10328050]

Evans DE, & Drobes DJ (2009). Nicotine self-medication of cognitive-attentional processing. Addiction Biology, 14, 32–42. [PubMed: 18855804]

Evins AE, Culhane MA, Alpert JE, Pava J, Liese BS, Farabaugh A, et al. (2008). A controlled trial of bupropion added to nicotine patch and behavioral therapy for smoking cessation in adults with unipolar depressive disorders. Journal of Clinical Psychopharmacology, 28, 660–666. [PubMed: 19011435]

Fawcett J (1994). Overview of mood disorders: Diagnosis, classification, and management. Clinical Chemistry, 40, 273–278. [PubMed: 8313608]

Fawcett J, Clark DC, Scheftner WA, & Hedeker D (1983). Differences between anhedonic and normally hedonic depressive states. The American Journal of Psychiatry, 140, 1027–1030. [PubMed: 6869586]

Fergusson DM, Goodwin RD, & Horwood LJ (2003). Major depression and cigarette smoking: Results of a 21-year longitudinal study. Psychological Medicine, 33, 1357–1367. [PubMed: 14672244]

Fergusson DM, Horwood LJ, Ridder EM, & Beautrais AL (2005). Subthreshold depression in adolescence and mental health outcomes in adulthood. Archives of General Psychiatry, 62, 66–72. [PubMed: 15630074]

Fiore MC, & Jaen CR (2008). A clinical blueprint to accelerate the elimination of tobacco use. The Journal of the American Medical Association, 299, 2083–2085. [PubMed: 18460668]

Fiore MC, Jaen CR, Baker TB, Bailey WC, Benowitz N, & Curry SJ (2008). Treating tobacco use and dependence: 2008 update U.S. Public Health Service Clinical Practice Guideline executive summary. Respiratory Care, 53, 1217–1222. [PubMed: 18807274]

Forbes EE (2009). Where's the fun in that? Broadening the focus on reward function in depression. Biological Psychiatry, 66, 199–200. [PubMed: 19577042]

Forbes EE, Christopher May J, Siegle GJ, Ladouceur CD, Ryan ND, Carter CS, et al. (2006). Reward-related decision-making in pediatric major depressive disorder: An fMRI study. The Journal of Child Psychology and Psychiatry, 47, 1031–1040. [PubMed: 17073982]

Forbes EE, Hariri AR, Martin SL, Silk JS, Moyles DL, Fisher PM, et al. (2009). Altered striatal activation predicting real-world positive affect in adolescent major depressive disorder. The American Journal of Psychiatry, 166, 64–73. [PubMed: 19047324]

Forbes EE, Shaw DS, & Dahl RE (2007). Alterations in reward-related decision making in boys with recent and future depression. Biological Psychiatry, 61, 633–639. [PubMed: 16920074]

Franken IH, Zijlstra C, & Muris P (2006). Are nonpharmacological induced rewards related to anhedonia? A study among skydivers. Progress in Neuropsychopharmacology & Biological Psychiatry, 30, 297–300.

Georgiades K, Lewinsohn PM, Monroe SM, & Seeley JR (2006). Major depressive disorder in adolescence: The role of subthreshold symptoms. Journal of the American Academy of Child & Adolescent Psychiatry, 45, 936–944. [PubMed: 16865036]

Gierisch JM, Bastian LA, Calhoun PS, McDuffie JR, & Williams JW Jr. (2012). Smoking cessation interventions for patients with depression: A systematic review and meta-analysis. Journal of General Internal Medicine, 27, 351–360. [PubMed: 22038468]

Ginsberg D, Hall SM, Reus VI, & Muñoz RF (1995). Mood and depression diagnosis in smoking cessation. Experimental and Clinical Psychopharmacology, 3, 389–395.

Glassman AH, Stetner F, Walsh BT, Raizman PS, Fleiss JL, Cooper TB, et al. (1988). Heavy smokers, smoking cessation, and clonidine. Results of a double-blind, randomized trial. The Journal of the American Medical Association, 259, 2863–2866. [PubMed: 3367452]

Goodman E, & Capitman J (2000). Depressive symptoms and cigarette smoking among teens. Pediatrics, 106, 748–755. [PubMed: 11015518]

Grant BF, Hasin DS, Chou SP, Stinson FS, & Dawson DA (2004). Nicotine dependence and psychiatric disorders in the United States: Results from the national epidemiologic survey on alcohol and related conditions. Archives of General Psychiatry, 61, 1107–1115. [PubMed: 15520358]

Green L, & Fisher EB Jr. (2000). Economic substitutability: Some implications for health behavior. In Bickel WK & Vuchinich RE (Eds.), Reframing health behavior change with behavior economics (pp. 115–144). Mahway: Lawrence Erlbaum Associates.

Haas AL, Muñoz RF, Humfleet GL, Reus VI, & Hall SM (2004). Influences of mood, depression history, and treatment modality on outcomes in smoking cessation. Journal of Consulting and Clinical Psychology, 72, 563–570. [PubMed: 15301640]

Hall SM (2007). Nicotine interventions with comorbid populations. American Journal of Preventive Medicine, 33, S406–S413. [PubMed: 18021916]

Hall SM, Humfleet GL, Reus VI, Munoz RF, Hartz DT, & Maude-Griffin R (2002). Psychological intervention and antidepressant treatment in smoking cessation. Archives of General Psychiatry, 59, 930–936. [PubMed: 12365880]

Hall SM, Munoz RF, & Reus VI (1994). Cognitive-behavioral intervention increases abstinence rates for depressive-history smokers. Journal of Consulting and Clinical Psychology, 62, 141–146. [PubMed: 8034816]

Hall SM, Munoz RF, Reus VI, Sees KL, Duncan C, Humfleet GL, et al. (1996). Mood management and nicotine gum in smoking treatment: A therapeutic contact and placebo-controlled study. Journal of Consulting and Clinical Psychology, 64, 1003–1009. [PubMed: 8916629]

Hall SM, & Prochaska JJ (2009). Treatment of smokers with co-occurring disorders: Emphasis on integration in mental health and addiction treatment settings. Annual Review of Clinical Psychology, 5, 409–431.

Hall SM, Reus VI, Munoz RF, Sees KL, Humfleet G, Hartz DT, et al. (1998). Nortriptyline and cognitive-behavioral therapy in the treatment of cigarette smoking. Archives of General Psychiatry, 55, 683–690. [PubMed: 9707377]

Hall SM, Tsoh JY, Prochaska JJ, Eisendrath S, Rossi JS, Redding CA, et al. (2006). Treatment for cigarette smoking among depressed mental health outpatients: A randomized clinical trial. American Journal of Public Health, 96, 1808–1814. [PubMed: 17008577]

Harvey PO, Pruessner J, Czechowska Y, & Lepage M (2007). Individual differences in trait anhedonia: A structural and functional magnetic resonance imaging study in non-clinical subjects. Molecular Psychiatry, 12(703), 767–775.

Haug NA, Hall SM, Prochaska JJ, Rosen AB, Tsoh JY, Humfleet G, et al. (2005). Acceptance of nicotine dependence treatment among currently depressed smokers. Nicotine & Tobacco Research, 7, 217–224. [PubMed: 16036278]

Hedeker D, Mermelstein RJ, Berbaum ML, & Campbell RT (2009). Modeling mood variation associated with smoking: An application of a heterogeneous mixed-effects model for analysis of ecological momentary assessment (EMA) data. Addiction, 104, 297–307. [PubMed: 19149827]

Heinz AJ, Kassel JD, Berbaum M, & Mermelstein R (2010). Adolescents' expectancies for smoking to regulate affect predict smoking behavior and nicotine dependence over time. Drug and Alcohol Dependence, 111, 128–135. [PubMed: 20547013]

Heishman SJ, Kleykamp BA, & Singleton EG (2010). Meta-analysis of the acute effects of nicotine and smoking on human performance. Psychopharmacology, 210, 453–469. [PubMed: 20414766]

Higgins ST, Heil SH, & Lussier JP (2004). Clinical implications of reinforcement as a determinant of substance use disorders. Annual Review of Psychology, 55, 431–461.

Hitsman B, Borrelli B, McChargue DE, Spring B, & Niaura R (2003). History of depression and smoking cessation outcome: A meta-analysis. Journal of Consulting and Clinical Psychology, 71, 657–663. [PubMed: 12924670]

Hitsman B, Papandonatos GD, McChargue DE, DeMott A, Herrera MJ, Spring B, et al. (2013). Past major depression and smoking cessation outcome: A systematic review and meta-analysis update. Addiction, 108, 294–306. [PubMed: 23072580]

Hoffman BR, Monge PR, Chou CP, & Valente TW (2007). Perceived peer influence and peer selection on adolescent smoking. Addictive Behaviors, 32, 1546–1554. [PubMed: 17188818]

Hughes JR, Stead LF, & Lancaster T (2007). Antidepressants for smoking cessation. The Cochrane Database of Systematic Reviews, 1, CD000031.

Jacobson NS, Dobson KS, Truax PA, Addis ME, Koerner K, Gollan JK, et al. (1996). A component analysis of cognitive-behavioral treatment for depression. Journal of Consulting and Clinical Psychology, 64, 295–304. [PubMed: 8871414]

Japuntich SJ, Smith SS, Jorenby DE, Piper ME, Fiore MC, & Baker TB (2007). Depression predicts smoking early but not late in a quit attempt. Nicotine & Tobbacco Research, 9, 677–686.

Johnson PM, Hollander JA, & Kenny PJ (2008). Decreased brain reward function during nicotine withdrawal in C57BL6 mice: Evidence from intracranial self-stimulation (ICSS) studies. Pharmacology, Biochemistry and Behavior, 90, 409–415.

Johnson EO, Rhee SH, Chase GA, & Breslau N (2004). Comorbidity of depression with levels of smoking: An exploration of the shared familial risk hypothesis. Nicotine & Tobacco Research, 6, 1029–1038. [PubMed: 15801576]

Kahler CW, Brown RA, Ramsey SE, Niaura R, Abrams DB, Goldstein MG, et al. (2002). Negative mood, depressive symptoms, and major depression after smoking cessation treatment in smokers with a history of major depressive disorder. Journal of Abnormal Psychology, 111, 670–675. [PubMed: 12428781]

Kahler CW, Spillane NS, Day AM, Cioe PA, Parks A, Leventhal AM, et al. (2015). Positive psychotherapy for smoking cessation: A pilot randomized controlled trial. Nicotine & Tobacco Research. Epub ahead of print.

Kahler CW, Spillane NS, Day A, Clerkin E, Parks A, Leventhal AM, et al. (2014). Positive psychotherapy for smoking cessation: Treatment development, feasibility and preliminary results. The Journal of Positive Psychology, 9, 19–29. [PubMed: 24683417]

Kassel JD, Stroud LR, & Paronis CA (2003). Smoking, stress, and negative affect: Correlation, causation, and context across stages of smoking. Psychological Bulletin, 129, 270–304. [PubMed: 12696841]

Kendler KS, Neale MC, MacLean CJ, Heath AC, Eaves LJ, & Kessler RC (1993). Smoking and major depression. A causal analysis. Archives of General Psychiatry, 50, 36–43. [PubMed: 8422220]

Kenny PJ, & Markou A (2006). Nicotine self-administration acutely activates brain reward systems and induces a long-lasting increase in reward sensitivity. Neuropsychopharmacology, 31, 1203–1211. [PubMed: 16192981]

Killen JD, & Fortmann SP (1997). Craving is associated with smoking relapse: Findings from three prospective studies. Experimental and Clinical Psychopharmacology, 5, 137–142. [PubMed: 9234050]

Killen JD, Fortmann SP, Davis L, Strausberg L, & Varady A (1999). Do heavy smokers benefit from higher dose nicotine patch therapy? Experimental and Clinical Psychopharmacology, 7, 226–233. [PubMed: 10472510]

Killen JD, Robinson TN, Ammerman S, Hayward C, Rogers J, Samuels D, et al. (2004). Major depression among adolescent smokers undergoing treatment for nicotine dependence. Addictive Behaviors, 29, 1517–1526. [PubMed: 15451121]

Kinnunen T, Doherty K, Militello FS, & Garvey AJ (1996). Depression and smoking cessation: Characteristics of depressed smokers and effects of nicotine replacement. Journal of Consulting and Clinical Psychology, 64, 791–798. [PubMed: 8803370]

Kinnunen T, Korhonen T, & Garvey AJ (2008). Role of nicotine gum and pretreatment depressive symptoms in smoking cessation: Twelve-month results of a randomized placebo controlled trial. International Journal of Psychiatry in Medicine, 38, 373–389. [PubMed: 19069579]

Kobus K (2003). Peers and adolescent smoking. Addiction, 98(Suppl. 1), 37–55. [PubMed: 12752361]

Lee Duckworth A, Steen TA, & Seligman ME (2005). Positive psychology in clinical practice. Annual Review of Clinical Psychology, 1, 629–651.

Lerman C, Audrain J, Orleans CT, Boyd R, Gold K, Main D, et al. (1996). Investigation of mechanisms linking depressed mood to nicotine dependence. Addictive Behaviors, 21, 9–19. [PubMed: 8729703]

Leventhal AM, Ameringer KJ, Osborn E, Zvolensky MJ, & Langdon KJ (2013). Anxiety and depressive symptoms and affective patterns of tobacco withdrawal. Drug and Alcohol Dependence, 133, 324–329. [PubMed: 23896304]

Leventhal AM, Chasson GS, Tapia E, Miller EK, & Pettit JW (2006). Measuring hedonic capacity in depression: A psychometric analysis of three anhedonia scales. The Journal of Clinical Psychiatry, 62, 1545–1558.

Leventhal AM, Japuntich SJ, Piper ME, Jorenby DE, Schlam TR, & Baker TB (2012). Isolating the role of psychological dysfunction in smoking cessation: Relations of personality and psychopathology to attaining cessation milestones. Psychology of Addictive Behaviors, 26, 838–849. [PubMed: 22642858]

Leventhal AM, Munafo M, Tidey JW, Sussman S, Monterosso JR, Sun P, et al. (2012). Anhedonia predicts altered processing of happy faces in abstinent cigarette smokers. Psychopharmacology, 222, 343–351. [PubMed: 22311383]

Leventhal AM, Ramsey SE, Brown RA, LaChance HR, & Kahler CW (2008). Dimensions of depressive symptoms and smoking cessation. Nicotine & Tobacco Research, 10, 507–517. [PubMed: 18324570]

Leventhal AM, Ray LA, Rhee SH, & Unger JB (2012). Genetic and environmental influences on the association between depressive symptom dimensions and smoking initiation among Chinese adolescent twins. Nicotine & Tobacco Research, 14, 559–568. [PubMed: 22180578]

Leventhal AM, Trujillo M, Ameringer KJ, Tidey JW, Sussman S, & Kahler CW (2014). Anhedonia and the relative reward value of drug and nondrug reinforcers in cigarette smokers. Journal of Abnormal Psychology, 123, 375–386. [PubMed: 24886011]

Leventhal AM, Waters AJ, Kahler CW, Ray LA, & Sussman S (2009). Relations between anhedonia and smoking motivation. Nicotine & Tobacco Research, 11, 1047–1054. [PubMed: 19571250]

Levine MD, Marcus MD, & Perkins KA (2003). A history of depression and smoking cessation outcomes among women concerned about post-cessation weight gain. Nicotine & Tobacco Research, 5, 69–76. [PubMed: 12745508]

Lewinsohn PM, Hops H, Roberts RE, Seeley JR, & Andrews JA (1993). Adolescent psychopathology: I. Prevalence and incidence of depression and other DSM-III-R disorders in high school students. Journal of Abnormal Psychology, 102, 133–144. [PubMed: 8436689]

Lewinsohn PM, Rohde P, & Seeley JR (1998). Major depressive disorder in older adolescents: Prevalence, risk factors, and clinical implications. Clinical Psychology Review, 18, 765–794. [PubMed: 9827321]

Lewinsohn PM, Rohde P, Seeley JR, Klein DN, & Gotlib IH (2000). Natural course of adolescent major depressive disorder in a community sample: Predictors of recurrence in young adults. The American Journal of Psychiatry, 157, 1584–1591. [PubMed: 11007711]

Lewinsohn PM, Shankman SA, Gau JM, & Klein DN (2004). The prevalence and co-morbidity of subthreshold psychiatric conditions. Psychological Medicine, 34, 613–622. [PubMed: 15099416]

Lewinsohn PM, Youngren MA, & Grosscup SJ (1979). Reinforcement and depression. In Dupue RA (Ed.), The psychobiology of depressive disorders: Implications for the effects of stress (pp. 291–316). New York: Academic Press.

Lyons M, Hitsman B, Xian H, Panizzon MS, Jerskey BA, Santangelo S, et al. (2008). A twin study of smoking, nicotine dependence, and major depression in men. Nicotine & Tobacco Research, 10, 97–108. [PubMed: 18188750]

MacPherson L, Tull MT, Matusiewicz AK, Rodman S, Strong DR, Kahler CW, et al. (2010). Randomized controlled trial of behavioral activation smoking cessation treatment for smokers with elevated depressive symptoms. Journal of Consulting and Clinical Psychology, 78, 55–61. [PubMed: 20099950]

MacPhillamy DJ, & Lewinsohn PM (1974). Depression as a function of levels of desired and obtained pleasure. Journal of Abnormal Psychology, 83, 651–657. [PubMed: 4155711]

Mancuso G, Warburton DM, Melen M, Sherwood N, & Tirelli E (1999). Selective effects of nicotine on attentional processes. Psychopharmacology, 146, 199–204. [PubMed: 10525756]

Martini S, Wagner FA, & Anthony JC (2002). The association of tobacco smoking and depression in adolescence: Evidence from the United States. Substance Use & Misuse, 37, 1853–1867. [PubMed: 12511055]

McChargue DE, Spring B, Cook JW, & Neumann CA (2004a). Reinforcement expectations explain the relationship between depressive history and smoking status in college students. Addictive Behaviors, 29, 991–994. [PubMed: 15219347]

McChargue DE, Spring B, Cook JW, & Neumann CA (2004b). Reinforcement expectations explain the relationship between depressive history and smoking status in college students. Addictive Behavior, 29, 991–994.

Meehl PE (1975). Hedonic capacity: Some conjectures. Bulletin of the Menninger Clinic, 39, 295–307. [PubMed: 1156704]

Meehl PE (1987). “Hedonic capacity” ten years later: Some clarifications. In Clark DC & Fawcett J (Eds.), Anhedonia and affect deficit states (pp. 47–50). New York, NY: PMA Publishing.

Meehl PE (2001). Primary and secondary hypohedonia. Journal of Abnormal Psychology, 110, 188–193. [PubMed: 11261394]

Memelstein RJ, Hedeker D, Flay B, & Shiffman S (2007). Real-time data capture and adolescent cigarette smoking: Moods and smoking. In Stone A, Shiffman S, Atienza A, & Nebeling L (Eds.), The science of real-time data capture: Self-report in health research (pp. 117–135). New York: Oxford University Press.

Miller NL, Markowitz JC, Kocsis JH, Leon AC, Brisco ST, & Garno JL (1999). Cost effectiveness of screening for clinical trials by research assistants versus senior investigators. Journal of Psychiatric Research, 33, 81–85. [PubMed: 10221739]

Miller PG, & Miller WR (2009). What should we be aiming for in the treatment of addiction? Addiction, 104, 685–686. [PubMed: 19413777]

Munafo MR, Hitsman B, Rende R, Metcalfe C, & Niaura R (2008). Effects of progression to cigarette smoking on depressed mood in adolescents: Evidence from the National Longitudinal Study of Adolescent Health. Addiction, 103, 162–171. [PubMed: 18031565]

Murphy JG, Dennhardt AA, Skidmore JR, Borsari B, Barnett NP, Colby SM, et al. (2012). A randomized controlled trial of a behavioral economic supplement to brief motivational interventions for college drinking. Journal of Consulting and Clinical Psychology, 80, 876–886. [PubMed: 22663899]

Murphy JG, Skidmore JR, Dennhardt AA, Martens MP, Borsari B, Barnett NP, et al. (2012). A behavioral economic supplement to brief motivational interventions for college drinking. Addiction Research & Theory, 20, 456–465. [PubMed: 24039620]

Murray CJ, & Lopez AD (1996). Evidence-based health policy—Lessons from the Global Burden of Disease Study. Science, 274, 740–743. [PubMed: 8966556]

Murray CJ, & Lopez AD (1997). Mortality by cause for eight regions of the world: Global Burden of Disease Study. Lancet, 349, 1269–1276. [PubMed: 9142060]

Niaura R, Britt DM, Shadel WG, Goldstein M, Abrams D, & Brown R (2001). Symptoms of depression and survival experience among three samples of smokers trying to quit. Psychology of Addictive Behaviors, 15, 13–17. [PubMed: 11255933]

Nierenberg AA, Keefe BR, Leslie VC, Alpert JE, Pava JA, Worthington JJ 3rd, et al. (1999). Residual symptoms in depressed patients who respond acutely to fluoxetine. The Journal of Clinical Psychiatry, 60, 221–225. [PubMed: 10221281]

Nutt D, Demyttenaere K, Janka Z, Aarre T, Bourin M, Canonico PL, et al. (2007). The other face of depression, reduced positive affect: The role of catecholamines in causation and cure. Journal of Psychopharmacology (Oxford, England), 21, 461–471.

Patterson F, Jepson C, Loughead J, Perkins K, Strasser AA, Siegel S, et al. (2010). Working memory deficits predict short-term smoking resumption following brief abstinence. Drug and Alcohol Dependence, 106, 61–64. [PubMed: 19733449]

Patton GC, Carlin JB, Coffey C, Wolfe R, Hibbert M, & Bowes G (1998). Depression, anxiety, and smoking initiation: A prospective study over 3 years. American Journal of Public Health, 88, 1518–1522. [PubMed: 9772855]

Perkins KA (2001). Smoking cessation in women. Special considerations. CNS Drugs, 15, 391–411. [PubMed: 11475944]

Perkins KA (2009). Sex differences in nicotine reinforcement and reward: Influences on the persistence of tobacco smoking. Nebraska Symposium on Motivation, 55, 143–169. [PubMed: 19013943]

Perkins KA, Doyle T, Ciccocioppo M, Conklin C, Sayette M, & Caggiula A (2006). Sex differences in the influence of nicotine dose instructions on the reinforcing and self-reported rewarding effects of smoking. Psychopharmacology, 184, 600–607. [PubMed: 16075290]

Perkins KA, Hickcox ME, & Grobe JE (2000). Behavioral economics of tobacco smoking. In Bickel WK & Vuchinich RE (Eds.), Reframing health behavior change with behavior economics (pp. 293–311). Mahway: Lawrence Erlbaum Associates, Inc.

Perkins KA, Karelitz JL, Giedgowd GE, & Conklin CA (2013). Negative mood effects on craving to smoke in women versus men. Addictive Behaviors, 38, 1527–1531. [PubMed: 22726579]

Piper ME, Smith SS, Schlam TR, Fleming MF, Bittrich AA, Brown JL, et al. (2010). Psychiatric disorders in smokers seeking treatment for tobacco dependence: Relations with tobacco dependence and cessation. Journal of Consulting and Clinical Psychology, 78, 13–23. [PubMed: 20099946]

Pizzagalli DA, Holmes AJ, Dillon DG, Goetz EL, Birk JL, Bogdan R, et al. (2009). Reduced caudate and nucleus accumbens response to rewards in unmedicated individuals with major depressive disorder. The American Journal of Psychiatry, 166, 702–710. [PubMed: 19411368]

Pizzagalli DA, Iosifescu D, Hallett LA, Ratner KG, & Fava M (2008). Reduced hedonic capacity in major depressive disorder: Evidence from a probabilistic reward task. Journal of Psychiatric Research, 43, 76–87. [PubMed: 18433774]

Powell J, Tait S, & Lessiter J (2002). Cigarette smoking and attention to signals of reward and threat in the Stroop paradigm. Addiction, 97, 1163–1170. [PubMed: 12199832]

Prochaska JJ, Hall SM, Tsoh JY, Eisendrath S, Rossi JS, Redding CA, et al. (2008). Treating tobacco dependence in clinically depressed smokers: Effect of smoking cessation on mental health functioning. American Journal of Public Health, 98, 446–448. [PubMed: 17600251]

Prochaska JJ, Rossi JS, Redding CA, Rosen AB, Tsoh JY, Humfleet GL, et al. (2004). Depressed smokers and stage of change: Implications for treatment interventions. Drug and Alcohol Dependence, 76, 143–151. [PubMed: 15488338]

Reinherz HZ, Giaconia RM, Pakiz B, Silverman AB, Frost AK, & Lefkowitz ES (1993). Psychosocial risks for major depression in late adolescence: A longitudinal community study. Journal of the American Academy of Child & Adolescent Psychiatry, 32, 1155–1163. [PubMed: 8282659]

Reynolds EK, Macpherson L, Tull MT, Baruch DE, & Lejuez CW (2011). Integration of the brief behavioral activation treatment for depression (BATD) into a college orientation program: Depression and alcohol outcomes. Journal of Counseling Psychology, 58, 555–564. [PubMed: 21787070]

Ritt-Olson A, Unger J, Valente T, Nezami E, Chou CP, Trinidad D, et al. (2005). Exploring peers as a mediator of the association between depression and smoking in young adolescents. Substance Use & Misuse, 40, 77–98. [PubMed: 15702650]

Rogers RE, Higgins ST, Silverman K, Thomas CS, Badger GJ, Bigelow G, et al. (2008). Abstinence-contingent reinforcement and engagement in non-drug-related activities among illicit drug abusers. Psychology of Addictive Behaviors, 22, 544–550. [PubMed: 19071979]

Rohde P, Lewinsohn PM, Brown RA, Gau JM, & Kahler CW (2003). Psychiatric disorders, familial factors and cigarette smoking: I. Associations with smoking initiation. Nicotine & Tobacco Research, 5, 85–98. [PubMed: 12745510]

Rukstalis M, Jepson C, Patterson F, & Lerman C (2005). Increases in hyperactive-impulsive symptoms predict relapse among smokers in nicotine replacement therapy. Journal of Substance Abuse Treatment, 28, 297–304. [PubMed: 15925263]

Saluja G, Iachan R, Scheidt PC, Overpeck MD, Sun W, & Giedd JN (2004). Prevalence of and risk factors for depressive symptoms among young adolescents. Archives of Pediatrics & Adolescent Medicine, 158, 760–765. [PubMed: 15289248]

SAMHSA (2004). Office of Applied Studies, National Survey on Drug Use and Health. Subsample. http://www.samhsa.gov/data/nsduh/2k4nsduh/2k4results/2k4results.pdf. accessed 25.02.15.

Schnoll RA, Leone FT, & Hitsman B (2013). Symptoms of depression and smoking behaviors following treatment with transdermal nicotine patch. Journal of Addictive Diseases, 32, 46–52. [PubMed: 23480247]

Seligman ME, Rashid T, & Parks AC (2006). Positive psychotherapy. The American Psychologist, 61, 774–788. [PubMed: 17115810]

Seligman ME, Steen TA, Park N, & Peterson C (2005). Positive psychology progress: Empirical validation of interventions. American Psychologist, 60, 410–421. [PubMed: 16045394]

Shafer AB (2006a). Meta-analysis of the factor structures of four depression questionnaires: Beck, CES-D, Hamilton, and Zung. Journal of Clinical Psychology, 62, 123–146. [PubMed: 16287149]

Shafer AB (2006b). Meta-analysis of the factor structures of four depression questionnaires: Beck, CES-D, Hamilton, and Zung. The Journal of Clinical Psychiatry, 62, 123–146.

Shankman SA, Klein DN, Tenke CE, & Bruder GE (2007). Reward sensitivity in depression: A biobehavioral study. Journal of Abnormal Psychology, 116, 95–104. [PubMed: 17324020]

Skara S, & Sussman S (2003). A review of 25 long-term adolescent tobacco and other drug use prevention program evaluations. Preventive Medicine, 37, 451–474. [PubMed: 14572430]

Sloan DM, Strauss ME, Quirk SW, & Sajatovic M (1997). Subjective and expressive emotional responses in depression. Journal of Affective Disorders, 46, 135–141. [PubMed: 9479617]

Smith SS, Jorenby DE, Leischow SJ, Nides MA, Rennard SI, Johnston JA, et al. (2003). Targeting smokers at increased risk for relapse: Treating women and those with a history of depression. Nicotine & Tobacco Research, 5, 99–109. [PubMed: 12745511]

Spring B, Cook JW, Appelhans B, Maloney A, Richmond M, Vaughn J, et al. (2008). Nicotine effects on affective response in depression-prone smokers. Psychopharmacology, 196, 461–471. [PubMed: 17960366]

Stein EA, Pankiewicz J, Harsch HH, Cho JK, Fuller SA, Hoffmann RG, et al. (1998). Nicotine-induced limbic cortical activation in the human brain: A functional MRI study. The American Journal of Psychiatry, 155, 1009–1015. [PubMed: 9699686]

Steuber TL, & Danner F (2006). Adolescent smoking and depression: Which comes first? Addictive Behaviors, 31, 133–136. [PubMed: 15922514]

Stone MD, & Leventhal AM (2014). Associations between anhedonia and risk factors for smoking initiation among adolescent never smokers. In Poster presented at the 20th annual meeting of the Society for Research on Nicotine and Tobacco, February 2014, Seattle, WA.

Strong DR, Kahler CW, Leventhal AM, Abrantes AM, Lloyd-Richardson E, Niaura R, et al. (2009). Impact of bupropion and cognitive-behavioral treatment for depression on positive affect, negative affect, and urges to smoke during cessation treatment. Nicotine & Tobacco Research, 11, 1142–1153. [PubMed: 19574407]

Strong DR, Leventhal AM, Evatt DP, Haber S, Greenberg BD, Abrams D, et al. (2011). Positive reactions to tobacco predict relapse after cessation. Journal of Abnormal Psychology, 120, 999–1005. [PubMed: 21574668]

Sussman S, Lichtman K, Ritt A, & Pallonen UE (1999). Effects of thirty-four adolescent tobacco use cessation and prevention trials on regular users of tobacco products. Substance Use & Misuse, 34, 1469–1503. [PubMed: 10468104]

Swan GE, Jack LM, Curry S, Chorost M, Javitz H, McAfee T, et al. (2003). Bupropion SR and counseling for smoking cessation in actual practice: Predictors of outcome. Nicotine & Tobacco Research, 5, 911–921. [PubMed: 14668075]

Tsoh JY, & Hall SM (2004). Depression and smoking: From the transtheoretical model of change perspective. Addictive Behaviors, 29, 801–805. [PubMed: 15135563]

USDHHS. (2012). Preventing tobacco use among young people: A report of the Surgeon General. Atlanta: U.S. Department of Health and Human Services, Center for Disease Control and Prevention, Office on Smoking and Health accessed 25.02.15.

van der Meer RM, Willemsen MC, Smit F, & Cuijpers P (2013). Smoking cessation interventions for smokers with current or past depression. The Cochrane Database of Systematic Reviews, 8 CD006102.

van der Meer RM, Willemsen MC, Smit F, Cuijpers P, & Schippers GM (2010). Effectiveness of a mood management component as an adjunct to a telephone counselling smoking cessation intervention for smokers with a past major depression: A pragmatic randomized controlled trial. Addiction, 105, 1991–1999. [PubMed: 20735366]

Van Etten ML, Higgins ST, Budney AJ, & Badger GJ (1998). Comparison of the frequency and enjoyability of pleasant events in cocaine abusers vs. non-abusers using a standardized behavioral inventory. Addiction, 93, 1669–1680. [PubMed: 9926530]

Wang MQ, Eddy JM, & Fitzhugh EC (2000). Smoking acquisition: Peer influence and self-selection. Psychological Reports, 86, 1241–1246. [PubMed: 10932589]

Wang MQ, Fitzhugh EC, Green BL, Turner LW, Eddy JM, & Westerfield RC (1999). Prospective social-psychological factors of adolescent smoking progression. Journal of Adolescent Health, 24, 2–9. [PubMed: 9890358]

Wang MQ, Fitzhugh EC, Turner L, Fu Q, & Westerfield RC (1996). Association of depressive symptoms and school adolescents' smoking: A cross-lagged analysis. Psychological Reports, 79, 127–130. [PubMed: 8873796]

Warburton DM, & Mancuso G (1998). Evaluation of the information processing and mood effects of a transdermal nicotine patch. Psychopharmacology, 135, 305–310. [PubMed: 9498735]

Watson D (2005). Rethinking the mood and anxiety disorders: A quantitative hierarchical model for DSM-V. Journal of Abnormal Psychology, 114, 522–536. [PubMed: 16351375]

Watson D, Clark LA, & Carey G (1988). Positive and negative affectivity and their relation to anxiety and depressive disorders. Journal of Abnormal Psychology, 97, 346–353. [PubMed: 3192830]

Weinstein SM, & Mermelstein RJ (2013a). Dynamic associations of negative mood and smoking across the development of smoking in adolescence. Journal of Clinical Child & Adolescent Psychology, 42, 629–642. [PubMed: 23682640]

Weinstein SM, & Mermelstein RJ (2013b). Influences of mood variability, negative moods, and depression on adolescent cigarette smoking. Psychology of Addictive Behaviors, 27, 1068–1078. [PubMed: 23438244]

Weller EB, & Weller RA (2000). Depression in adolescents growing pains or true morbidity? Journal of Affective Disorders, 61(Suppl. 1), 9–13. [PubMed: 11155967]

Wichers MC, Barge-Schaapveld DQ, Nicolson NA, Peeters F, de Vries M, Mengelers R, et al. (2009). Reduced stress-sensitivity or increased reward experience: The psychological mechanism of response to antidepressant medication. Neuropsychopharmacology, 34, 923–931. [PubMed: 18496519]

Wilhelm K, Wedgwood L, Niven H, & Kay-Lambkin F (2006). Smoking cessation and depression: Current knowledge and future directions. Drug and Alcohol Review, 25, 97–107. [PubMed: 16492582]

Williamson DE, Birmaher B, Axelson DA, Ryan ND, & Dahl RE (2004). First episode of depression in children at low and high familial risk for depression. Journal of the American Academy of Child and Adolescent Psychiatry, 43, 291–297. [PubMed: 15076262]

Wills TA, & Cleary SD (1999). Peer and adolescent substance use among 6th-9th graders: Latent growth analyses of influence versus selection mechanisms. Health Psycholology, 18, 453–463.

Windle M, & Windle RC (2001). Depressive symptoms and cigarette smoking among middle adolescents: Prospective associations and intrapersonal and interpersonal influences. Journal of Consulting and Clinical Psychology, 69, 215–226. [PubMed: 11393599]

Wu LT, & Anthony JC (1999). Tobacco smoking and depressed mood in late childhood and early adolescence. American Journal of Public Health, 89, 1837–1840. [PubMed: 10589312]