

Cigarette Smoking, Anxiety, and Depression

Holly E. R. Morrell¹ and Lee M. Cohen^{1,2}

Published online: 18 May 2006

This review provides a detailed analysis of anxiety and depression as they relate to each stage of the cigarette smoking cycle: initiation, maintenance, and cessation with an emphasis on nicotine withdrawal. An analysis of the literature confirms that cigarette smoking is highly comorbid with anxiety disorders and clinical depression, and that this relationship appears to be moderated by factors such as age of the smoker, type of disorder, and level of nicotine dependence. Studies also offer evidence to suggest a relationship between smoking and both subclinical anxiety and depression. Research findings have not revealed whether common factors influence the development of anxiety, depression, and smoking, whether anxiety and depression lead to smoking, or whether the reverse is true. Nevertheless, a current understanding of the links among smoking, anxiety, and depression confirms current prevention and cessation techniques, as well as suggests new directions for research and clinical practice.

KEY WORDS: anxiety; depression; nicotine; smoking; smoking cessation.

Cigarette smoking is the leading cause of preventable death in the United States. Nearly a quarter of the population smokes, and this habit has been linked to approximately 430,000 deaths per year (Centers for Disease Control and Prevention (CDC), 2001). Smoking is associated with asthma, chronic bronchitis, lung cancer, coronary heart disease, and emphysema (National Institute on Drug Abuse, 2001). Studies indicate that medical expenses resulting from health problems associated with tobacco use range from \$53 to \$73 billion per year (CDC, 2001). The harsh personal and economic costs related to smoking have prompted a significant amount of research over the past 30 years focused on understanding the addictive nature of cigarette smoking, with the hope that new information might help health care professionals to improve smoking prevention and cessation programs.

Although approximately 35% of smokers attempt to quit smoking each year, less than 5% are successful (American Psychiatric Association (APA), 2000). The majority of researchers agree that one of the primary ob-

stacles to successful smoking cessation is nicotine withdrawal (Kassel, Stroud, & Paronis, 2003; Niaura et al., 1999; Robinson et al., 1995; West, 1984). The advent of nicotine replacement therapy (e.g., nicotine gum, the nicotine patch, etc.) has helped many people manage symptoms of withdrawal, and cigarette smoking has steadily decreased in the general population (Hughes, 1993; Office on Smoking and Health, 2002). However, smoking rates remain high among individuals with particularly compelling needs for nicotine, such as those individuals who desire regulation of negative affective states like anxiety and depression (Cohen, McCarthy, Brown, & Myers, 2002; Gilbert & Wesler, 1989; Hall, Muñoz, Reus, & Sees, 1993; Russell, Peto, & Patel, 1974; Warburton, Revell, & Thompson, 1991). Numerous studies suggest that anxiety and depression may play a role in the initiation, maintenance, and cessation of smoking behavior (e.g., Breslau, Kilbey, & Andreski, 1991; Glassman et al., 1990; Hall et al., 1993). This in turn implies that a thorough understanding of the mechanisms underlying the connection between smoking and various types of negative affect may aid researchers and practitioners in developing more successful smoking prevention and cessation programs. Given the potentially valuable clinical implications of understanding how anxiety and depression relate to smoking

¹Department of Psychology, Texas Tech University, Lubbock, Texas.

²To whom correspondence should be addressed at Department of Psychology, Texas Tech University, Box 42051, Lubbock, Texas 79406-2051; e-mail: lee.cohen@ttu.edu.

behavior, the following review addresses each in the context of the stages of the cigarette smoking cycle: initiation, maintenance, and cessation (Flay, 1993).

CIGARETTE SMOKING AND ANXIETY-ANXIOUS MOOD

A number of studies implicate anxiety as an integral component of the nicotine withdrawal syndrome (Gilbert et al., 1998a,b; Hughes, 1992; Hughes, Gust, Skoog, Keenan, & Fenwick, 1991; Hughes & Hatsukami, 1986). The DSM-IV-TR (APA, 2000) lists anxiety as a symptom of the nicotine withdrawal syndrome, but does not operationally define the construct to distinguish between clinical anxiety and anxious mood. One may assume that the DSM is referring to some type of anxious mood that does not necessarily meet criteria for a specific anxiety disorder. Similarly, many of the smoking studies that include anxiety as a variable assess what can be more correctly termed “anxious mood.” Often, researchers operationalize anxiety as a score on some measure of anxious mood (e.g., the State-Trait Anxiety Inventory; STAI; Spielberger, Gorsuch, & Lushene, 1970) that is not diagnostic of a clinical anxiety disorder as defined by the DSM. Studies that focus on the relationship between anxiety disorders and smoking behavior usually evaluate participants according to DSM diagnostic criteria for anxiety disorders, sometimes in addition to administering measures of subjective anxious mood.

Comorbidity estimates for anxiety and smoking vary according to the type of anxiety disorder, the research design (cross-sectional versus longitudinal), and the population under investigation (adolescents versus adults). Estimates may also vary as a function of whether smoking is defined in terms of simple smoking status (smoker versus non-smoker) or nicotine dependence. Adolescent comorbidity studies have produced variable results, but adult studies have generated more consistent findings. Typically, comorbidity estimates between smoking and any type of anxiety disorder range from 9% to 62%, with nicotine-dependent smokers exhibiting higher levels of comorbidity (see Breslau et al., 1991; Degenhardt, Hall, & Lynsky, 2001). A recent study conducted in a nationally representative sample found the prevalence of mood disorders and anxiety disorders to be 21.1 and 22%, respectively, among individuals diagnosed with nicotine dependence (Grant, Hasin, Chou, Stinson, & Dawson, 2004a). These prevalence estimates are much higher than those in the general population (9.2% for mood disorder and 11.1% for anxiety disorder; Grant et al., 2004a,b). Nicotine-dependent individuals were 3.3–3.9 times as

likely to have an anxiety disorder, and 2.6–4.6 times more likely to have a mood disorder (Grant et al., 2004a).

Comorbidity studies in adolescents suggest a weak or non-existent relationship between tobacco use and anxiety disorders. Brown, Lewinsohn, Seeley, and Wagner (1996) examined the relationship between smoking and various psychiatric disorders in a sample of 1,507 adolescents using a combined cross-sectional and longitudinal design (i.e., measurements were taken at two points in time approximately 14 months apart). As there was not a high incidence of any specific anxiety disorder, the authors collapsed across anxiety disorders in their analyses. In the cross-sectional analyses, smokers did not exhibit a higher rate of anxiety disorder than non-smokers (9.2 and 8.3%, respectively). Costello, Erkanli, Federman, and Angold (1999) found that 12.8% of girls and 16.1% of boys aged 9–13 with an anxiety disorder also qualified for nicotine use or dependence, after statistically controlling for other forms of psychiatric comorbidity; these percentages were not statistically significant. Dierker, Avenevoli, Merikangas, Flaherty, and Stolar (2001) observed that only adolescents who qualified for a diagnosis of nicotine dependence were at a significantly elevated risk for having a comorbid anxiety disorder. Although the results from these studies indicate little or no relationship between tobacco use and anxiety disorders in adolescents, they may be qualified by the distinction between smoking status and nicotine dependence, as dependent adolescents appear more likely to have a comorbid anxiety disorder.

The relationship between anxiety and smoking appears more tenable in adults. Hughes, Hatsukami, Mitchell, and Dahlgren (1986) found that 47% of psychiatric outpatients with an anxiety disorder also smoked, which was significantly higher than in a population-based control group. Himle, Thyer, and Fischer (1988) attempted to replicate Hughes et al.’s (1986) findings, in addition to observing comorbidity estimates for each specific anxiety disorder. They found the following comorbidity rates: simple phobia, 47%; social phobia, 27%; agoraphobia, 57%; panic disorder, 47%; generalized anxiety, 29%, and obsessive compulsive disorder, 9%. Post-traumatic stress disorder (PTSD) was not included in their study because of the small sample size of individuals meeting diagnostic criteria for PTSD. Himle et al. demonstrated that smoking rates might differ among the anxiety disorders, and that epidemiological researchers might be well advised to avoid collapsing across anxiety disorders in their analyses when possible, as this analytic strategy might obscure true comorbidity effects.

Breslau et al. (1991) found that comorbidity levels depended on severity of nicotine dependence, as defined by DSM-III-R (APA, 1987) diagnostic criteria, in a

cross-sectional sample of 1007 young adults aged 21–30 (cf. Dierker et al., 2001). The lifetime prevalence for any anxiety disorder among smokers with mild nicotine dependence (mild) was 36.8%, and the lifetime prevalence for those with moderate nicotine dependence (moderate) was 62.3%. Data for individuals with severe nicotine dependence were not available because very few participants fell into this category. Comorbidity estimates also varied according to type of anxiety disorder, with percentages in the moderate group still generally greater than those in the mild group. Logistic regression analyses indicated that moderately dependent smokers were four times more likely to have an anxiety disorder than non-smokers, even after statistically controlling for age and other drug dependencies.

A more recent study of German adults revealed significant comorbidity between being a daily smoker and having an anxiety disorder (John, Meyer, Rumpf, & Hapke, 2004). Findings indicated that former daily smokers and current daily smokers were 1.4 and 1.6 times more likely, respectively, than never smokers to have an anxiety disorder. Individuals reporting three or more dependence symptoms were 3.7 times more likely than those with no dependence symptoms to have an anxiety disorder, and individuals reporting one to two dependence symptoms were 1.6 times more likely to have an anxiety disorder. These findings further support the hypothesis that higher levels of nicotine dependence are associated with anxiety disorders.

The few studies that have examined comorbidity between smoking behavior and anxiety disorders suggest that smokers are more likely to have an anxiety disorder than non-smokers, even though this relationship is less clear in adolescents than adults. There are several explanations for the fact that results from adolescent and adult comorbidity studies have yielded conflicting findings. First, smoking status and nicotine dependence are not necessarily identical constructs (i.e., just because one smokes does not mean that one is dependent upon nicotine), a fact that limits the ability to compare results across studies that employ different dependent measures. Second, base rates of anxiety disorders may be different in the two populations, which is likely to influence the results of cross-sectional studies. Differing base rates may reflect the developmental course of anxiety disorders, a possibility that highlights the importance of longitudinal research in this area. Smoking rates also differ depending on the type of anxiety disorder, and these estimates vary additionally by population. Another trend in the literature is that greater nicotine dependence is associated with a greater likelihood of having a comorbid anxiety disorder, and adults may be more likely to be dependent on nicot-

tine because they are more likely to have smoked longer than adolescents. Finally, adults may have greater access to cigarettes because they can legally purchase them, are more likely to have a steady income, and are less likely to be required to hide their habit from others.

Smokers often report that they smoke to relieve anxiety (Schneider & Houston, 1970), and studies show that smokers smoke more in stressful and anxiety-provoking situations (e.g., Rose, Ananda, & Jarvik, 1983). Smokers also exhibit higher baseline levels of anxiety than non-smokers (McCrae, Costa, & Bosse, 1978; Schneider & Houston, 1970; Williams, Hudson, & Redd, 1982). These facts have led researchers to hypothesize that elevated anxiety, whether in the form of an anxiety disorder or anxious mood, may be a predisposing factor in smoking initiation. Research in this area, however, has generated mixed results.

Sonntag, Wittchen, Hofler, Kessler, and Stein (2000) investigated the relationship between nicotine dependence and social phobia according to DSM-IV criteria in a sample of German individuals aged 14–24. They assessed participants at baseline in 1995 and at follow-up in 1996–1997 and 1998–1999. Nearly 32% of individuals meeting the criteria for social phobia also met full criteria for nicotine dependence, even after controlling for comorbid depression and dysthymia at baseline. General social fears, but not social phobia, predicted later nicotine dependence. The authors speculated that they might have had insufficient power (i.e., only 7.2% of their sample met the criteria for social phobia) to detect a true prospective relationship between social phobia and nicotine dependence. In another longitudinal study of adults, Breslau, Novak, and Kessler (2004) found that social phobia did predict the onset of daily smoking, as did PTSD. This relationship was only significant when the disorders were considered active (i.e., when the individual was currently experiencing symptoms).

Several studies indicate that there is no relationship between anxiety disorders and smoking initiation. Brown et al. (1996) conducted longitudinal analyses that revealed no relationship between any psychiatric disorder (except depression) and subsequent smoking initiation in adolescents. Similarly, Dierker et al. (2001; described in the previous section) found no significant links between the presence of any anxiety disorder and later nicotine dependence. Rohde, Kahler, Lewinsohn, and Brown (2004) also observed that the presence of any anxiety disorder did not predict later progression to daily smoking in individuals whose mean age was 16.6 years at baseline and 24 years at final evaluation.

Research in both adolescent and adult populations shows that smoking may actually predispose an individual

to developing an anxiety disorder. Pohl, Yeragani, Balon, Lycaki, and McBride (1992) observed that panic disorder preceded smoking initiation in adults, but these data were purely retrospective. Breslau and Klein (1999) investigated the relationship between smoking and panic attacks (and panic disorder) in more than 1000 adults over a period of 5 years, and results demonstrated that smoking initiation increased the risk for later panic attacks. Since these data were prospective, they offer stronger support than Pohl et al.'s findings for the hypothesis that smoking status actually predicts the onset of an anxiety disorder.

Johnson et al. (2000) studied smoking status and anxiety disorders among 688 youths over a period of 6–7 years (mean age = 16 years at baseline). Anxiety in adolescence was not significantly associated with smoking in young adulthood, but smoking in adolescence was predictive of anxiety disorders in young adulthood. Thirty-one percent of adolescents who smoked more than 20 cigarettes per day developed anxiety disorders in young adulthood, as compared with only 9% of adolescent non-smokers. Heavy and chronic smokers during adolescence were at greater risk for generalized anxiety disorder, agoraphobia, and panic disorder during young adulthood, even after controlling for numerous covariates such as depression. There were no associations among smoking status, obsessive compulsive disorder, and social anxiety disorder. Of those individuals who had an anxiety disorder at baseline, 42% reported smoking before being diagnosed with the anxiety disorder (only 19% reported the reverse).

Because the directionality of the relationship between anxiety/anxious mood and smoking initiation remains unclear at this time, one can only speculate about implications for preventing smoking initiation. If more rigorous studies indicate that certain anxiety disorders predispose individuals to start smoking, perhaps standard treatments for these anxiety disorders can include an educational component addressing smoking prevention. This strategy may be more effective when combined with coping skills training, such that individuals with an anxiety disorder will become proficient in managing their anxiety without smoking. Providing both psychological and pharmacological relief for anxiety may also reduce the likelihood of smoking initiation. Efforts at anxiety reduction may aid in breaking the smoking cycle among individuals who have already begun to smoke.

Whereas there are a number of studies addressing the connection between anxiety disorders and smoking initiation, there is little research on the connection between subclinical anxious mood and smoking initiation. Much of the research on anxious mood tends to be applicable to the maintenance and relapse phases of the smoking cycle. The next question therefore concerns how anxiety

disorders and anxious mood might influence the maintenance of smoking behavior. One explanation is based on the affect regulation model of drug use, which states that individuals smoke to regulate their affective state. With regard to anxiety, this theory states that the anxiolytic effects of smoking negatively reinforce smoking behavior. The anxiety may arise from internal sources (e.g., trait anxiety), environmental sources (e.g., interpersonal stressors or fear), or as part of the nicotine withdrawal syndrome (i.e., smoking may be maintained by the desire to avoid the nicotine withdrawal syndrome).

A number of studies has examined whether smoking relieves anxiety. While reviewing such studies, it is important to remember that researchers define anxiety (and attempt to produce it) in a variety of ways. It is also not always easy to distinguish between the physiological anxiety-reducing effects of nicotine or the actual smoking behavior. Furthermore, even though many smokers report smoking to relieve anxiety, this behavior actually produces symptoms consistent with physiological arousal (Gilbert, 1979; Robinson et al., 1995). To determine whether individuals smoke to relieve anxiety, it is vital to know if smoking reduces anxiety levels, and if individuals smoke more during anxiety-provoking situations. Only one study in this domain has focused on anxiety reduction in participants with an existing anxiety disorder, while the remainder assesses changes in anxious mood or stress level. Fleming and Lombardo (1987) tested the anxiolytic effects of smoking among 20 female smokers and 20 female non-smokers with a self-reported phobia for rats. The authors instructed participants to approach a large, white rat and rate their anxiety levels at 5-foot intervals. Then participants completed the State Form of the State-Trait Anxiety Inventory at the end of the experiment. Although approaching the rat produced significant increases in anxiety, neither participant smoking status (smoker versus non-smoker) nor being allowed to smoke during the experiment influenced anxiety levels. The authors posited that level of anxiety moderates the anxiolytic effects of smoking, such that smoking is not anxiolytic under conditions of extreme anxiety. However reasonable this hypothesis may seem, its applicability to Fleming and Lombardo's study is based primarily on the assumption that their manipulation did in fact produce unusually high levels of anxiety.

Hatch, Bierner, and Fisher (1982) also found that smoking does not relieve anxious mood. They examined the anxiolytic properties of nicotine during a stressful speech task. Participants were randomly assigned to one of three groups: No-smoking, high-nicotine, and low-nicotine. All participants were then given 10 min to mentally prepare a 3-min speech on abortion, after which they

delivered the speech. Measures indicated that anxiety did increase during the preparation and speech phases of the experiment, but that smoking did not alleviate this anxiety.

In contrast to Fleming and Lombardo's (1987) and Hatch et al.'s (1982) results, several studies demonstrate that smoking relieves anxious mood in a variety of contexts, including during difficult problem-solving tasks. Pomerleau, Turk, and Fertig (1984) found that smoking significantly relieved anxious mood, as measured by the STAI, in six male participants during an unsolvable anagram task. Jarvik, Caskey, Rose, Herskovic, and Sadehpour (1989) sought to replicate and extend Pomerleau et al.'s results by investigating the anxiolytic effects of smoking during an anagram task, a cold pressor task, a white noise condition, and an auditory vigilance task, all of which have been shown to induce anxiety. Smoking relieved anxious mood in the anagram task and the cold pressor task, although the latter effect was only marginally significant. Smoking did not relieve anxious mood in response to white noise or the auditory vigilance task, but insufficient power may have contributed to these findings (i.e., there were only eight participants per condition).

The connection between smoking and anxiety relief has also been tested in stressful social interactions. Gilbert and Spielberger (1987) manipulated smoking status (allowed to smoke versus not allowed to smoke) among pairs of individuals who were instructed to discuss a topic on which they disagreed. Results demonstrated that, in spite of overall increases in physiological arousal (e.g., heart rate), participants reported less anxiety when allowed to smoke. These results might relate to the fact that smokers often report smoking more in social situations, although more research in this area is needed. Given the high degree of comorbidity between social fears, social phobia, and smoking (Breslau et al., 2004; Sonntag et al., 2000), it may be reasonable to hypothesize that a certain segment of the smoking population experiences elevated levels of social fear, and therefore smokes as a way of coping with that fear.

Gilbert, Robinson, Chamberlin, and Spielberger (1989) moved beyond the realm of social interaction and examined the anxiety-reducing properties of smoking in response to a stressful movie. They instructed 40 smokers and 40 non-smokers to watch a movie about industrial accidents, which was intended to induce anxiety. Before the movie, half of the smokers smoked a high-nicotine cigarette and half smoked a low-nicotine cigarette. Non-smokers puffed on a soda straw to control for possible effects of smoking behavior alone. Individuals who smoked high-nicotine cigarettes reported significantly smaller increases in anxiety during the movie than individuals in

the low-nicotine group or the control group. Britt, Cohen, Collins, and Cohen (2001) examined the anxiolytic properties of smoking and chewing gum in response to a stressful speech task. Smokers were told that they must prepare a speech about their body and physical appearance. Measurements of craving, withdrawal, and anxiety were taken at baseline, after speech preparation but before smoking or chewing gum, after smoking or chewing gum, after the speech itself, and after a 10-min rest period. Analyses revealed that individuals in the smoking group experienced a significant decrease in anxiety over time relative to the chewing gum group and control group, therefore supporting the hypothesis that smoking can be anxiolytic.

More recent research suggests that attentional processes may mediate the relationship between smoking and anxiolysis. Kassel and Shiffman (1997) postulated that smoking "constrains smokers' attention to the most immediate and salient stimuli in their environment—when such stimuli are available" (p. 360). As a result, smokers are more likely to focus on immediate and distracting stimuli than more distal anxiogenic stimuli, thus reducing anxious mood. Two studies have confirmed the hypothesis that smoking relieves anxious mood in minimally deprived smokers in the presence of a distractor, and that this effect cannot be attributed to the behavioral aspects of smoking or the nicotine withdrawal syndrome (Kassel & Shiffman, 1997; Kassel & Unrod, 2000). In contrast, Herbert, Foulds, and Fife-Schaw (2001) demonstrated that attentional processes in non-deprived smokers did not mediate the anxiolytic effects of nicotine. These findings suggest that attentional mediation may only occur under certain conditions, such as when smokers are minimally deprived. It is also important to keep in mind that these studies induced anxious mood in different ways, which may make it difficult to compare results. Kassel and colleagues instructed participants to give a speech about what they liked and disliked about their body, but Herbert et al. informed participants that their behavior and facial expressions would be monitored during a rapid visual information processing task.

Research also demonstrates that smokers do smoke more when experiencing anxious mood. For instance, Rose et al. (1983) found that individuals smoked more in terms of both volume and puffing frequency during an induced stage fright task and a monotonous concentration task compared to a relaxation condition (cf. Epstein, Ossip, Coleman, Hughes, & Whist, 1981). It may be reasonable to infer from such studies that anxious mood is an important factor in maintaining the smoking cycle.

A majority of studies have shown that anxious mood increases upon smoking cessation. Only one study (West & Hajek, 1997) to date has not confirmed this

relationship; however, the researchers failed to measure anxiety levels during the peak of the nicotine withdrawal syndrome (Wiseman, 1999). Furthermore, a preponderance of smokers report that they smoke in response to experiencing negative affect, including anxiety and stress. Several studies also appear to validate the anxiolytic properties of smoking and the observation that smokers smoke more when anxious or stressed. Such evidence suggests that anxiety, either in the form of an anxiety disorder or general anxious mood, may hinder smoking cessation efforts (Covey, 1999). Unfortunately, there is little direct empirical evidence supporting this possibility, as most studies of affect regulation and smoking cessation have focused on major depressive disorder (MDD), depressed mood, and undifferentiated measures of negative affect rather than on anxiety or anxious mood.

Two recent studies have evaluated panic disorder and panic attacks as predictors of cessation outcomes. Amering et al. (1999) retrospectively examined the relationship between a DSM-III-R diagnosis of panic disorder and smoking behavior in a sample of 102 adult Austrian outpatients. They found that 72% of participants reported smoking before the onset of panic disorder. Over half of these participants (55%) reported that they had reduced smoking, and 26% reported that they had stopped smoking as a result of their panic disorder. Nineteen percent reported that they smoked more after developing panic disorder. This research suggests that the presence of panic disorder might actually increase an individual's likelihood of reducing or quitting smoking. However, the results of this study cannot be generalized to persons with other anxiety disorders, and they are limited by the fact that the data were purely retrospective. Zvolensky, Lejuez, Kahler, and Brown (2004) retrospectively observed the opposite pattern among 40 adult smokers, half of whom reported a history of panic attacks, but not panic disorder. Results indicated that smokers with panic attacks reported significantly shorter quit attempts and more severe withdrawal symptoms (*viz.*, anxiety, difficulty concentrating, restlessness, and irritability) than those without panic attacks. The most likely reason for the discrepancies between Amering et al.'s (1999) findings and Zvolensky et al.'s (2004) findings is the fact that they sampled different populations (*e.g.*, individuals with diagnosable panic disorder versus individuals who merely reported a history of panic attacks).

Two recent studies investigated the link between trait anxiety and smoking cessation. Takemura, Akanuma, Kikuchi, and Inaba (1999) examined whether trait anxiety influenced smoking cessation in over 2000 male Japanese government employees using a Japanese translation of the STAI. They reported that high trait anxiety did not increase

the risk for smoking initiation but did predict unsuccessful plans to quit smoking. Despite the high level of statistical power in this study because of the large sample size, results might be limited by retrospective bias and by the cross-sectional design. Additionally, findings may only generalize to Japanese male adults. Becoña, Vázquez, and Míguez (2002) assessed trait anxiety as a correlate of smoking cessation in 214 Spanish adults who volunteered to complete a 6-week cessation program. A comparison of smokers who remained abstinent, as well as smokers who resumed smoking within the 12 months following treatment, revealed that those in the latter group experienced significantly more state anxiety at post-treatment (but not pre-treatment). The authors did not include a control group, nor did they use their data to predict prospectively smoking cessation from state anxiety scores; therefore, inferences about a causal relationship between state anxiety and smoking cessation outcome cannot be made based on their findings.

Some indirect evidence concerning the impact of anxiety on smoking cessation can be gleaned from smoking cessation treatment studies (Covey, 1999). Cinciripini et al. (1995) studied the effects of buspirone (Buspar), a medication with anxiolytic properties, on smoking cessation. They discovered that smokers high in self-reported anxiety were not as likely to maintain abstinence compared to smokers low in self-reported anxiety. Studies of bupropion (Wellbutrin), an antidepressant approved by the FDA for smoking cessation, indicate that this medication relieves a host of withdrawal symptoms (including anxiety) and that quitters who take bupropion are more likely to remain abstinent. David et al. (2003) showed that quitters taking bupropion reported significantly less anxiety than quitters in a placebo control group. Individuals in the former group were also more likely to remain abstinent 2 weeks post-cessation. Despite the apparent link between certain pharmacological treatments for nicotine withdrawal and anxiety reduction, the assumption that anxiolysis is significantly responsible for improved abstinence rates remains speculative.

Results of the studies discussed above suggest that there may be a connection between anxiety and smoking cessation outcomes, but more research is needed to answer the following four critical questions: (1) Does the presence of an anxiety disorder (in addition to panic disorder) predict relapse following cessation; (2) do measures of anxious mood predict relapse; (3) do anxiety-provoking situations produce greater urges to smoke in quitters; and (4) what variables mediate and moderate the relationship between anxiety and relapse? As there is little direct evidence of a predictive relationship between anxiety and smoking cessation, the effectiveness of specific

anxiolytic smoking cessation strategies can only be conjectured. Even though anxiety-reducing treatments have been shown to increase the likelihood of abstinence from smoking, it is unclear what proportion of their overall effectiveness can be attributed to anxiolytic properties. Nevertheless, it appears that such treatments (especially pharmacological ones) can be considered important aids to smoking cessation, and as such, cessation programs might be well advised to include both psychological and pharmacological anxiety-reduction strategies. These components might be particularly important as part of a multimodal treatment program for heavy smokers who have failed at repeated attempts to quit smoking.

CIGARETTE SMOKING AND DEPRESSION—DEPRESSED MOOD

Studies investigating smoking and affect have typically focused on clinical depression and depressed mood. In the smoking literature, clinical depression is often synonymous with major depressive disorder, as defined in the DSM. Very few studies have assessed the relationship between smoking behavior and other mood disorders, such as Dysthymic Disorder or Bipolar I Disorder; therefore, these disorders are excluded from the present review. Depressed mood is generally operationalized as a score on a non-diagnostic measure of dysphoria, such as the Depression-Dejection scale of the Profile of Mood States (POMS; McNair, Lorr, & Droppleman, 1971). Terminology is vague in this area of research, but for present purposes, a specific structure is imposed to provide clarity. Major depressive disorder is referred to precisely as such, and subclinical depressed mood (i.e., depressed mood in the absence of a clinical diagnosis) is labeled *depressed mood*. The term *depression* is used to refer to the overarching construct that includes both MDD and depressed mood, and *negative affect* is used to denote a blend of depressive and anxious symptomatology. A more precise definition is provided where appropriate.

Most research confirms that there is a high degree of comorbidity between smoking behavior and MDD (Breslau et al., 1991, 1995, 2004; Breslau, Kilbey, & Andreski, 1994; Breslau, Peterson, Schultz, Chilcoat, & Andreski, 1998; Degenhardt et al., 2001; Dierker et al., 2001; Fergusson, Lynsky, & Horwood, 1996; Grant et al., 2004a; John et al., 2004; Kandel, Huang, & Davies, 2001; Pérez-Stable, Marín, Marín, & Katz, 1990). Evidence regarding the comorbidity between smoking and MDD is generally stronger and more consistent than the evidence regarding the comorbidity between smoking and anxiety. This relationship is apparent regardless of population,

research design, and definition of smoking behavior. Perhaps one of the reasons for a consistently high degree of comorbidity between MDD and smoking behavior is that MDD is typically operationalized as a single, multifaceted construct rather than an entire class of related disorders (cf. anxiety disorders).

Comorbidity estimates between MDD and smoking behavior or nicotine dependence range from 20 to 65%, with nicotine dependence being a stronger correlate of MDD than smoking status (Breslau et al., 1991, 1994, 1998, 2004; Degenhardt et al., 2001; Dierker et al., 2001; Fergusson et al., 1996; Kandel et al., 2001). Breslau et al. (1991) found that 39% of young adult smokers with moderate levels of nicotine dependence qualified for a diagnosis of comorbid MDD, as compared to 19.2% of mildly dependent smokers and 10.1% of non-dependent smokers. A recent study of German adults revealed significant comorbidity between being a daily smoker and having a mood disorder (John et al., 2004). Findings indicated that current daily smokers were 1.8 times more likely than never smokers to have a mood disorder. Furthermore, individuals reporting three or more dependence symptoms were 3.6 times more likely than those with no dependence symptoms to have a mood disorder, and individuals reporting one to two dependence symptoms were 1.7 times more likely to have a mood disorder.

Comorbidity rates are also generally higher among smokers than non-smokers regardless of dependence status. Kendler et al. (1993) observed that 24.9% of non-smokers and 51.7% of smokers also met criteria for MDD. Among a community sample of adolescents, Brown et al. (1996) found that 17.1% of non-smokers and 32% of smokers had MDD. High comorbidity between smoking behavior and MDD has also been confirmed among Latinos, despite nationally lower smoking rates in this population (Pérez-Stable et al., 1990). Pérez-Stable and colleagues found that, on average, 30.7% of Latino smokers were also diagnosed with MDD, as compared to 18.4% of former smokers and 15.2% of never smokers.

The consistently high levels of comorbidity between smoking behavior and MDD have led to multiple hypotheses about the nature of the relationship between depression and smoking initiation. The four hypotheses most commonly addressed in the literature are: (1) Premorbid MDD or depressive symptomatology predisposes individuals to initiate smoking; (2) smoking status or nicotine dependence is a precursor to the development of MDD or depressive symptoms; (3) there is a reciprocal relationship between depression and smoking; or (4) there is a non-causal relationship between smoking and depression, such that common factors (e.g., genetics or shared environment) influence the development of both conditions. Each

of these plausible rival hypotheses can be conceptualized as a correlate of the affect regulation theory of smoking, which states that individuals smoke to regulate affective state.

Studies that have examined MDD as a predictor of smoking and vice versa have generally observed highly significant relationships in both directions, whether the design is cross-sectional and retrospective or longitudinal and prospective (Breslau et al., 1998, 2004; Brown et al., 1996; Costello et al., 1999; Dierker et al., 2001; Kandel & Davies, 1986; Killen et al., 1996; Rohde, Lewinsohn, Brown, Gau, & Kahler, 2003). Most of these studies have been conducted with adolescents, given that risk for smoking initiation is greatest during adolescence (Johnston, O'Malley, & Bachman, 1998). Brown et al. (1996) assessed 1709 adolescents, aged 14–18, at two time-points approximately 1 year apart. Results revealed that adolescent smokers were nearly twice as likely as their non-smoking counterparts to have experienced their first major depressive episode within 1 year of the baseline assessment. Conversely, lifetime prevalence of MDD significantly predicted smoking initiation during the following year (adjusted OR = 2.04). These results were significant even after controlling for the presence of other psychiatric disorders.

In another study, Breslau et al. (1998) conducted a 5-year longitudinal examination of 1000 young adults and found that history of MDD predicted progression to daily smoking, and history of daily smoking increased the risk for MDD. Breslau and colleagues also observed that individuals with active MDD were more likely to progress to daily smoking compared to individuals with MDD in remission (Breslau et al., 2004). Among adolescents, data indicate that individuals with clinically significant levels of depression are more likely to initiate smoking if they are also highly receptive to tobacco advertisements (Tercyak, Goldman, Smith, & Audrain, 2002). In fact, there appears to be a strong reciprocal relationship between MDD and nicotine dependence at all stages of the smoking cycle (Breslau et al., 1998; Fergusson et al., 1996).

Research investigating the relationship between sub-clinical depressed mood and smoking behavior has produced similar results to those seen in studies evaluating MDD. Again, a preponderance of studies in this area focus on adolescents. Choi, Patten, Gillin, Kaplan, and Pierce (1997) found that smoking status was the single most important predictor of subsequent depressive symptoms in a sample of nearly 7000 U.S. adolescents, aged 12–18; however, they did not examine depressive symptoms as predictors of smoking initiation. Escobedo, Reddy, and Giovino (1998) addressed this issue using the same sample and found that adolescents with self-reported depressive

symptoms were 1.3 times more likely to initiate smoking compared to adolescents without depressive symptoms. Wu and Anthony (1999) sampled a younger group of adolescents (aged 8–14) and found that, while smoking predicted later depressed mood, depressed mood did not predict later smoking initiation.

In sum, research indicates that depression predicts smoking initiation and smoking behavior predicts depression. At first glance, these results might be construed as supporting two mutually exclusive rival hypotheses, but this apparent contradiction can be reconciled by reciprocal or noncausal hypotheses (e.g., Wang, Fitzhugh, & Green, 1996). Orlando, Ellickson, and Jinnett (2001) used structural equation modeling procedures to analyze data from a 5-year longitudinal study of adolescents in the 10th grade, 12th grade, and 5 years post-graduation. They examined the relationship between smoking status and emotional distress, as defined by a measure designed to assess depression, anxiety, and lack of positive affect. They found that initial smoking directly predicted subsequent smoking, and initial emotional distress directly predicted subsequent emotional distress. However, distress at Time 1 predicted smoking at Time 2, and smoking at Time 2 predicted distress at Time 3. In other words, initial distress predicted later smoking behavior, which in turn predicted subsequent distress. Although such results are interpreted with caution because Orlando et al. did not measure MDD or pure depressive symptoms, they lend support to the hypothesis that there is a reciprocal relationship between smoking status and depressed mood.

Support for a model of bidirectional influence between smoking behavior and depression does not negate the possibility that common factors may affect both outcomes. To examine the latter possibility, Windle and Windle (2001) tested the influence of multiple psychosocial factors that may theoretically lead to increases in both smoking and depressive symptoms (e.g., parental smoking, social support, peer substance use, self substance use, and activity level). They examined 1218 adolescents every 6 months for 2 years, starting when the participants were in either 10th or 11th grade. After controlling for a number of possible confounding variables, Windle and Windle still found strong evidence to support a reciprocal relationship between smoking and depression. These results can be generalized to smoking initiation with caution, however, because Windle and Windle were more concerned with depression as a predictor of increases in smoking behavior rather than initiation. In an earlier study, Kendler et al. (1993) assessed genetics as a shared causal indicator for smoking and MDD by examining a large ($N = 1566$), cross-sectional sample of female twins. They obtained clear evidence against a bidirectional

model of influence, and instead found that genetics alone appeared to influence both smoking and MDD.

Despite the strong findings from Kendler et al. (1993), empirical evidence is generally supportive of the reciprocal hypothesis of the link between MDD and smoking behavior. However, it is likely that any model of smoking initiation must be more complex than any of these models for several reasons. First, the presence of any given risk factor may be highly dependent on the environment of the individual (e.g., suburb versus inner city). Second, predictors of smoking initiation may change relative to the developmental stage of the individual. Third, researchers may have overlooked potential mediator and moderator variables in the quest for direct predictors. Finally, intraindividual variation may account for a significant amount of the variance in either smoking initiation or depression.

Affect regulation models of smoking posit that individuals not only begin smoking, but also maintain smoking behavior, to regulate negative affect; therefore, explanations of smoking initiation and maintenance are inextricably linked. The picture becomes more complex when the issues of smoking cessation and relapse are considered together. Another assumption of affect regulation models is that factors maintaining smoking behavior are also likely to influence smoking cessation (e.g., factors such as depression and anxiety may make quitting more difficult), and thus mechanisms underlying cessation appear inseparable from those underlying maintenance. Little research directly addresses the issue of maintenance and depression, and instead, most of the relevant evidence comes from smoking cessation studies.

Smoking behavior and nicotine may regulate negative affect through various biological and psychological mechanisms. It is likely that several mechanisms operate simultaneously to influence depression. It is therefore particularly difficult to distinguish among biological mechanisms of action, because nicotine produces a wide variety of neurochemical effects, and because environmental and psychological factors may also influence neurological substrates (Brandon, 1994). Most of the existing biological theories postulate that nicotine either initiates arousal associated with positive affect or inhibits arousal that can be attributed to negative affective states (Carmody, 1992). Consistent with these theories, research indicates that nicotine is associated with increased levels of neurotransmitters that stimulate reinforcement centers of the brain and reduce pain, such as β -endorphin, norepinephrine, epinephrine, acetylcholine, and dopamine (Brandon, 1994).

Neurochemical effects of nicotine may maintain smoking behavior in several ways. Leventhal and Cleary (1980) proposed that reductions in plasma nicotine level

produce dysphoria, and that dysphoria becomes conditioned to low plasma nicotine levels over time. Therefore, smokers learn that they must increase plasma nicotine (i.e., smoke) to relieve dysphoria, and their motivation to do so ultimately maintains smoking behavior. Pomerleau and Pomerleau (1984, 1988) developed a biobehavioral theory of smoking, which states that smoking becomes conditioned over time to internal and external cues relating to negative affect because affective stress has been relieved in the past by nicotine-induced increases in β -endorphin and cholinergic activity. Epping-Jordan, Watkins, Koob, and Markou (1998) have found evidence to suggest that nicotine lowers brain reward thresholds, such that it is easier to stimulate brain reward function, the opposite of which is observed during nicotine withdrawal. Withdrawal-induced decreases in brain reward function may be associated with increases in negative affect, which smokers may be motivated to relieve by smoking.

There are several potential psychological mechanisms that appear complementary to biological mechanisms and may assist in explaining smoking maintenance. Strictly behavioral theories focus on classical and operant conditioning, whereas cognitive theories include the influence of expectations and appraisals (Hughes, Higgins, & Hatsukami, 1990; Kassel et al., 2003). Most proposed behavioral mechanisms are based on the underlying assumption that negative affective states become conditioned over time to trigger craving for a cigarette (Carmody, 1992). As a result, smoking behavior becomes a conditioned response to experiencing negative affect.

Social cognitive theory emphasizes cognitive reactions to dysphoric states, such that an individual's expectations regarding nicotine's impact on their dysphoria influence motivation to continue smoking (Carmody, 1992). For example, if an individual expects that smoking will help him or her feel better, then he or she may be more likely to smoke to relieve negative affect. Over time, this may become a conditioned association (Carmody, 1992). According to social learning theory, smoking gradually becomes a conditioned response to negative affect, alternative coping skills are inhibited in favor of smoking, and the individual learns that negative affect can only be controlled by smoking (see Brandon, 1994).

It is unlikely that any one mechanism in isolation can fully explain how negative affect serves to maintain smoking behavior. It seems likely that biological, psychological, and social mechanisms interact to reinforce smoking behavior in response to negative affective states such as depression. For instance, increases in dopamine levels in the brain during smoking may teach smokers that cigarettes "make them feel good." Expectations about mood improvement may in turn lead smokers to try

smoking as a means of relieving dysphoria. As dysphoria and smoking are paired with each other over time, actual mood enhancement and strongly reinforced expectations regarding affective relief may aid in conditioning smoking behavior to dysphoria. Gradually, internal and external cues that coincide with negative affect are likely to prompt smoking behavior, which may be further reinforced by a lack of alternative coping strategies.

Until recently, most research on the relationship between depression and relapse following smoking cessation has been descriptive in nature. Studies in this area have focused on detecting the presence of a link between depression and relapse rather than how that link is established and maintained. The vast majority of these studies showed that smokers with a history of depression or smokers who experience depressive symptoms during a quit attempt are more likely to relapse than smokers without these risk factors (Anda et al., 1990; Brandon, 1994; Carmody, 1992; Covey, 1999; Glassman, 1993; Glassman et al., 1990; Hall et al., 1993; Kassel et al., 2003; Rohde et al., 2004). A few studies have nevertheless produced contradictory results, suggesting that there is no relationship between these two constructs (Breslau et al., 2004; Hall, Muñoz, & Reus, 1994; Niaura et al., 1999). To resolve the ambiguity in this area, Hitsman, Borrelli, McChargue, Spring, and Niaura (2003) conducted a meta-analysis of the existing literature. They included studies that used valid measures of history of depression and smoking cessation, and that assessed abstinence as the dependent variable. A meta-analysis of the 15 studies that met these criteria revealed that history of depression did not significantly predict smoking cessation outcome. Furthermore, variables such as gender, sample composition, smoking status, treatment modality, type of depression assessment, and time interval since last depressive episode did not moderate the effect of depression history on cessation outcome. While these results might appear discouraging, it is worth noting that only a history of a single major depressive episode was examined as a predictor of abstinence. The impact of recurrent depressive episodes, MDD during abstinence, or subclinical depression during abstinence, all of which have been verified as predictors of cessation outcome (Hitsman et al., 2003), were not considered.

There is a trend in the literature to explain the mechanisms through which depression influences smoking cessation. Shiffman and colleagues proposed that there is distinction between depression as a background factor or a precipitating factor for relapse (Shiffman & Waters, 2004). They described background factors as those that affect relapse, but that change slowly with time, so that they cannot be used to explain why a relapse occurs at

a specific point in time (e.g., ongoing stress or an “imbalanced lifestyle”). They defined precipitating factors as immediate and pivotal in producing relapse. For example, a fight with a significant other may cause symptoms of depression, which in turn may trigger a smoking relapse within a matter of hours. Shiffman and Waters (2004) used daily diary methodology to compare the predictive power of negative affect (a construct that included symptoms of depression, anxiety, and withdrawal) as either a background or precipitating factor influencing relapse. Results demonstrated that negative affect was not a background factor, but was a precipitating factor, because increases in negative affect 6 hr prior to relapse were strongly predictive of later relapse. Clinically, this suggests that quitters might benefit from identifying idiosyncratic precipitating factors and learning alternate ways to cope with them.

Other researchers have posited that smoking cessation may actually precipitate a major depressive episode, as defined by DSM diagnostic criteria, which in turn may cause a relapse as a means of affect regulation. This risk for a major depressive episode may be elevated in individuals with a previous history of MDD. A few studies have generated evidence for this hypothesis. Glassman (1993) observed that 18% of smokers with a history of MDD experienced post-cessation depressive symptoms, as opposed to 2% of smokers without a similar history of MDD. However, it is unclear whether these depressive symptoms were clinically significant. Glassman (1993) reported the results of another smoking cessation trial in which nine of 300 smokers experienced “deterioration in their psychological status” so severe that they were advised to return to smoking: Six of these individuals had a reported history of major depression. Covey, Glassman, and Stetner (1997) found a 2% incidence of post-cessation “serious depressive episodes” in individuals without a history of major depression, a 16% incidence in individuals with a history of one major depressive episode, and a 30% incidence in those who had reported multiple major depressive episodes in the past. Taken together, results suggest that smoking cessation may provoke a major depressive episode in some quitters with a history of MDD. Whether such a major depressive episode consistently guarantees relapse remains unclear.

Perhaps the time course of depression, rather than mere presence, predicts relapse. Burgess et al. (2002) explored the possibility of common temporal depression profiles in a sample of smokers with a history of MDD across 8 weeks of abstinence. They found that depressive symptomatology usually followed one of five time courses: rapid increase, delayed increase, brief reactivity (an immediate increase in depressive symptoms after cessation, followed by a decline), delayed decrease, and

rapid decrease. Furthermore, profile membership significantly predicted relapse: participants who experienced an increase in depression were more likely to relapse within 1 year compared to those who showed a decrease in depression. Rapid increases were also characterized by an earlier age of smoking onset and higher rates of recurrent depression, which suggests that a history of depression may predict an individual's temporal depression profile following smoking cessation. Brief reactors demonstrated high levels of abstinence within the first 6 weeks of abstinence, but high levels of relapse in the ensuing months. It is suggested that individuals who experience an immediate decline in depressive symptoms might feel rewarded by their elevated mood, and therefore more motivated to remain abstinent. By contrast, a marked increase in depression might discourage quitters and compromise their coping skills.

Cinciripini et al. (2003) evaluated coping skills, self-efficacy, urges to smoke, and withdrawal severity as potential mediators and moderators of the relationship between pre-cessation depressed mood, measured by the POMS, and relapse. They randomly assigned 121 heavy smokers to one of two treatment conditions: behavioral therapy and behavioral therapy plus the transdermal nicotine patch. Participants were assessed at baseline, at 1, 2, 4, and 8 weeks post-cessation, and at follow-up (6 months post-cessation). Cinciripini et al. (2003) found that absolute level of self-efficacy at 2, 4, and 8 weeks post-cessation was the only significant predictor of abstinence rates 6 months later. These results indicate that low self-efficacy is one of the mechanisms by which higher levels of pre-cessation depressed mood correspond with higher relapse rates. It appears that low self-efficacy following cessation is most important, perhaps because levels of self-efficacy are influenced by negative quit-related events that occur within the first week or two of abstinence (Cinciripini et al., 2003). Cinciripini et al.'s findings are consistent with research demonstrating that low context-specific self-efficacy is associated with lapses in abstinence (Gwaltney et al., 2002).

Few studies have directly tested the predictive efficacy of affect regulation models against competing models of relapse. Recognizing this, Kenford et al. (2002) compared a general affect regulation model with a physical dependence model of relapse in a sample of 505 adult heavy smokers. Their affect regulation model included predictors such as post-cessation negative affect, current stress level, history of depression, smoking expectancies, and coping style. Their physical dependence model included number of cigarettes smoked per day, carbon monoxide (CO) level, blood nicotine level, blood cotinine level, level of dependence, and withdrawal sever-

ity. Across several time periods, Kenford et al. consistently found that the affect regulation model significantly predicted relapse over and above any contributions from the physical dependence model. Within the affect regulation model, history of depression, greater post-cessation negative affect, and higher negative reinforcement expectancies were the strongest predictors of relapse. Withdrawal severity was the best predictor of abstinence in the dependence model, but this effect disappeared when negative affect was included in the model.

In sum, the affect regulation model of smoking appears to be a valid explanation of relapse following smoking cessation, although more varied and stringent tests are required. The specific mechanisms by which depression actually leads to relapse are less clear. Research implies that history of a single depressive episode does not predict relapse (Hitsman et al., 2003), but that immediate depressed mood may be a factor that precipitates relapse (Shiffman & Waters, 2004). It is even possible that smoking cessation can provoke a major depressive episode. Level of post-cessation self-efficacy may mediate the relationship between depression and relapse, such that lower levels of self-efficacy in response to depression predict higher relapse rates. Levels of post-cessation depression may also follow one of several different temporal trajectories. Type of trajectory, such as an immediate and significant increase in depressed mood following cessation, may significantly increase the likelihood of relapse.

Empirical evidence supports the theory that depressed smokers are more likely to relapse after a cessation attempt. It follows that smokers who experience depression should receive more intense and specialized treatment for smoking cessation. At this point, determining who to assign to more intensive treatment becomes more complicated, because research also demonstrates that history of a single major depressive episode does not necessarily predict who is more likely to relapse (Haas, Muñoz, Humfleet, Reus, & Hall, 2004; Hitsman et al., 2003). Instead, history of recurrent episodes of major depression may be a better predictor, along with level of depression immediately prior to cessation. It is therefore important to assess an individual's complete history of depression and current mood state before he or she begins treatment.

Smokers who report elevated levels of depression prior to smoking cessation may be more likely to relapse. In such cases, integrating well-validated psychological (e.g., cognitive-behavioral techniques) and pharmacological treatments for depression into the cessation protocol may improve abstinence rates. Discrete increases in negative affect during smoking cessation may also predict a smoking relapse within 6 hr (Shiffman & Waters,

2004). Thus, an intensive cessation program includes an assessment of potential factors and situations that typically induce negative affect in the individual prior to treatment. An alternate method may involve having patients self-monitor during their cessation attempt to identify variables or incidents that trigger a relapse. Once such triggers are identified, the clinician can teach the patient relevant coping strategies. In situations where time and resources are limited, patients may be given a pamphlet explaining how to self-monitor and describing general coping strategies. Some research suggests that depressed smokers do not typically respond well to coping skills training alone, but respond better to mood management training (see Brown et al., 2001). An intensive treatment program might therefore benefit from combining coping skills training and supportive therapy.

Quitters who experience rapid, delayed, or even brief initial *increases* in depressed mood during cessation may be more likely to relapse than quitters who experience a rapid or delayed *decrease* in depressed mood (Burgess et al., 2002). Assessing depression at baseline and at designated intervals (e.g., every week) may help the clinician to determine what trajectory the patient's depression is likely to follow. As a result, the clinician might be able to identify who is at greatest risk for relapse and respond by providing more intensive therapy sessions (or even booster sessions during the rough times), prescribing higher doses of antidepressant medications, or both. The clinician could also explore possible reasons for the increase in depression and address them accordingly. Reasons might include simple withdrawal-related increases in depression, a negative event, negative beliefs about one's ability to quit smoking, and so forth.

Another issue in preventing relapse is self-efficacy: some studies indicate that individuals with lower self-efficacy are less likely to remain abstinent (Gwaltney et al., 2002), and individuals who experience depression during a cessation attempt are more likely to suffer from low self-efficacy (Cinciripini et al., 2003). These studies suggest that it may be useful to assess self-efficacy before and periodically throughout treatment and to include brief self-efficacy training in smoking cessation programs (Abrams et al., 2003). However, more empirical evidence supporting a causal connection between self-efficacy and relapse is needed before widespread implementation of such suggestions.

No intervention is complete without long-term relapse-prevention strategies. Depressed ex-smokers might be most concerned with learning to avoid smoking in response to dysphoria. General relapse prevention strategies usually involve three main components: (1) The individual is educated about the nature and time course of

withdrawal; (2) he or she is taught to recognize, avoid, and cope with relapse triggers; and (3) he or she is encouraged to view lapses as learning experiences rather than failures (Abrams et al., 2003). With regard to depression, quitters could be educated about the nature and time course of depression as it relates to smoking cessation, taught to recognize and deal with affective cues that might trigger a relapse, and counseled to avoid letting brief lapses damage their sense of self-efficacy.

GENERAL DISCUSSION

Cigarette smoking is associated with high health and economic costs to the individual, the health care system, and the workforce. Thus, there has been a strong emphasis on smoking prevention and cessation in the U.S. over the past 20–30 years. Researchers have spent considerable effort studying what factors promote smoking initiation and hinder smoking cessation in the hopes that a greater understanding of these variables might improve the efficacy of current prevention and cessation programs. Two factors that are often perceived to influence smoking behavior strongly are anxiety and depression.

Anxiety can be identified as either the presence of a diagnosable anxiety disorder or simply anxious mood. Anxiety disorders appear to be comorbid with smoking behavior, although comorbidity estimates vary with the sample under investigation (adolescents versus adults), the experimental methodology employed (cross-sectional versus longitudinal), the definition of smoking behavior (nicotine dependence versus smoking status), and the type of anxiety disorder (e.g., social anxiety versus simple phobia). Two consistent trends are observed in the literature: comorbidity estimates are more stable in adult populations and are higher with greater levels of nicotine dependence.

Research to date has been unable to clarify the relationship between anxiety and smoking initiation. Some studies suggest that anxiety predicts smoking initiation, others predict the opposite, and still others fail to find any relationship between the two conditions. The literature does implicate anxiety in the maintenance of smoking behavior. Many smokers cite anxiety relief as their primary reason for smoking, they tend to smoke more in anxiety-provoking situations, and smoking appears to relieve anxiety in a variety of contexts. Attention may mediate this relationship between smoking and anxiety, such that smoking truly demonstrates anxiolytic properties in the presence of distracting stimuli. Anxiety may also precipitate relapse among quitters, although the research in this area remains somewhat sparse and inconclusive.

Epidemiological studies consistently show high comorbidity rates between depression (MDD) and smoking behavior, regardless of sample characteristics, research design, and operationalization of smoking status. There is also evidence to suggest a reciprocal relationship between the onset of MDD and smoking initiation. However, the possibility that MDD and smoking are caused by common factors that precipitate the development of both conditions cannot be ignored. The exact nature of these potential shared risk factors is not yet known, but one study has strongly implicated genetic influences (Kendler et al., 1993).

Depression may be an important predictor of relapse in smokers attempting to quit. Studies have shown that individuals experiencing escalating levels of depression and lower levels of self-efficacy immediately following cessation are more likely to relapse. Discrete incidents that induce negative affect may also precipitate a relapse within a few hours. Several mechanisms have been proposed to explain how depression maintains smoking behavior, and therefore contributes to relapse. For example, the neurochemical effects of nicotine might alleviate depression by increasing positive affect or decreasing negative affect. Individuals may learn over time to regulate affect with nicotine, such that they lose their ability to cope with depression-inducing stimuli.

How anxiety and depression contribute to smoking initiation is not completely understood, and therefore more research is needed in this area. With regard to treatment for smoking cessation, a review of the literature stresses the importance of a thorough assessment prior to cessation that covers previous quit attempt history, past and present anxiety, past and present depression, and level of self-efficacy. It also suggests that assessment in all these domains is useful when conducted periodically throughout the cessation attempt to monitor patient progress and identify potential problems. Intensive therapy might include education, well-validated brief treatments for anxiety and depression, supportive counseling, coping skills training, self-efficacy training, and long-term relapse prevention. While standardized measures and treatment techniques are useful, treatment programs are best tailored to the needs of each individual.

ACKNOWLEDGMENTS

The authors wish to thank Dr. Stephanie Harter and Dr. Steven Richards for their comments and suggestions on this manuscript.

REFERENCES

- Abrams, D. B., Niaura, R., Brown, R. A., Emmons, K. M., Goldstein, M. G., & Monti, P. M. (2003). *The tobacco dependence treatment handbook*. New York: Guilford Press.
- American Psychiatric Association (1987). *The diagnostic and statistical manual of mental disorders* (3rd ed., revised). Washington, DC: Author.
- American Psychiatric Association (2000). *The diagnostic and statistical manual of mental disorders* (4th ed., text revision). Washington, DC: Author.
- Amering, M., Bankier, B., Berger, P., Griengl, H., Windhaber, J., & Katschnig, H. (1999). Panic disorder and cigarette smoking behavior. *Comprehensive Psychiatry*, *40*(1), 35–38.
- Anda, R. F., Williamson, D. F., Escobedo, L. G., Mast, E. E., Giovino, G. A., & Remington, P. L. (1990). Depression and the dynamics of smoking. *Journal of the American Medical Association*, *264*, 1541–1545.
- Becoña, E., Vázquez, F. L., & Míguez, M. (2002). Smoking cessation and anxiety in a clinical sample. *Personality and Individual Differences*, *32*, 489–494.
- Brandon, T. H. (1994). Negative affect as motivation to smoke. *Current Directions in Psychological Science*, *3*, 33–37.
- Breslau, N. (1995). Psychiatric comorbidity of smoking and nicotine dependence. *Behavior Genetics*, *25*, 95–101.
- Breslau, N., Kilbey, M. M., & Andreski, P. (1991). Nicotine dependence, major depression, and anxiety in young adults. *Archives of General Psychiatry*, *48*, 1069–1074.
- Breslau, N., Kilbey, M. M., & Andreski, P. (1994). DSM-III-R nicotine dependence in young adults: Prevalence, correlates, and associated psychiatric disorders. *Addiction*, *89*, 743–754.
- Breslau, N., & Klein, D. F. (1999). Smoking and panic attacks: An epidemiologic investigation. *Archives of General Psychiatry*, *56*, 1141–1147.
- Breslau, N., Novak, S. P., & Kessler, R. C. (2004). Psychiatric disorders and stages of smoking. *Biological Psychiatry*, *55*(1), 69–76.
- Breslau, N., Peterson, E. L., Schultz, L. R., Chilcoat, H. D., & Andreski, P. (1998). Major depression and stages of smoking: A longitudinal investigation. *Archives of General Psychiatry*, *55*, 161–166.
- Britt, D. M., Cohen, L. M., Collins, F. L., & Cohen, M. L. (2001). Cigarette smoking and chewing gum: Response to a laboratory-induced stressor. *Health Psychology*, *20*(5), 361–368.
- Brown, R. A., Kahler, C. W., Niaura, R. A., Abrams, D. B., Sales, S. D., Ramsey, S. E., et al. (2001). Cognitive-behavioral treatment for depression in smoking cessation. *Journal of Consulting and Clinical Psychology*, *69*, 471–480.
- Brown, R. A., Lewinsohn, P. M., Seeley, J. R., & Wagner, E. F. (1996). Cigarette smoking, major depression, and other psychiatric disorders among adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry*, *35*, 1602–1610.
- Burgess, E. S., Brown, R. A., Kahler, C. W., Niaura, R., Abrams, D. B., Goldstein, M. G., et al. (2002). Pattern of change in depressive symptoms during smoking cessation: Who's at risk for relapse? *Journal of Consulting and Clinical Psychology*, *70*(2), 356–361.
- Carmody, T. P. (1992). Affect regulation, nicotine addiction, and smoking cessation. *Journal of Psychoactive Drugs*, *24*(2), 111–122.
- Centers for Disease Control and Prevention (2001). Youth tobacco surveillance: United States, 2000. *MMWR Morbidity and Mortality Weekly Report*, *50*(4), 1–84.
- Choi, W. S., Patten, C. A., Gillin, J. C., Kaplan, R. M., & Pierce, J. P. (1997). Cigarette smoking predicts development of depressive symptoms among US adolescents. *Annals of Behavioral Medicine*, *19*, 42–50.
- Cinciripini, P. J., Lapitzky, L., Seay, S., Wallfisch, A., Meyer, W. J., & Van Vunakis, H. (1995). A placebo-controlled evaluation of the effects of buspirone on smoking cessation: Differences between high- and low-anxiety smokers. *Journal of Clinical Psychopharmacology*, *15*, 182–191.

- Cinciripini, P. M., Wetter, D. W., Fouladi, R. T., Blalock, J. A., Carter, B. L., Cinciripini, L. G., et al. (2003). The effects of depressed mood on smoking cessation: Mediation by postcessation self-efficacy. *Journal of Consulting and Clinical Psychology, 71*(2), 292–301.
- Cohen, L. M., McCarthy, D. M., Brown, S. A., & Myers, M. G. (2002). Negative affect combines with smoking outcome expectancies to predict smoking behavior over time. *Psychology of Addictive Behaviors, 16*(2), 91–97.
- Costello, E. J., Erkanli, A., Federman, E., & Angold, A. (1999). Development of psychiatric comorbidity with substance abuse in adolescents: Effects of timing and sex. *Journal of Clinical Child Psychology, 28*, 298–311.
- Covey, L. S. (1999). Nicotine dependence and its associations with psychiatric disorders: Research evidence and treatment implications. In D. F. Seidman & L. S. Covey (Eds.), *Helping the hard-core smoker: A clinician's guide* (pp. 23–50). Mahwah, NJ: Lawrence Erlbaum Associates.
- Covey, L. S., Glassman, A. H., & Stetner, F. (1997). Depression and depressive symptoms in smoking cessation. *Comprehensive Psychiatry, 30*, 350–354.
- David, S. P., Niaura, R., Papandonatos, G. D., Shadel, W. G., Burkholder, G. J., Britt, D. M., et al. (2003). Does the DRD2-Taq1 A polymorphism influence treatment response to bupropion hydrochloride for reduction of the nicotine withdrawal syndrome? *Nicotine and Tobacco Research, 5*(6), 935–942.
- Degenhardt, L., Hall, W., & Lynskey, M. (2001). Alcohol, cannabis and tobacco use among Australians: A comparison of their associations with other drug use and use disorders, affective and anxiety disorders, and psychosis. *Addiction, 96*(11), 1603–1614.
- Dierker, L. C., Avenevoli, S., Merikangas, K. R., Flaherty, B. T., & Stolar, M. (2001). Association between psychiatric disorders and the progression of tobacco use behaviors. *Journal of the American Academy of Child and Adolescent Psychiatry, 40*, 1159–1167.
- Epping-Jordan, M. P., Watkins, S. S., Koob, G. F., & Markou, A. (1998). Dramatic decreases in brain reward function during nicotine withdrawal. *Nature, 393*, 76–79.
- Epstein, L. H., Ossip, D. J., Coleman, D., Hughes, J., & Whist, W. (1981). Measurement of smoking topography during withdrawal or deprivation. *Behavior Therapy, 12*, 507–519.
- Escobedo, L. G., Reddy, M., & Giovino, G. A. (1998). The relationship between depressive symptoms and cigarette smoking in US adolescents. *Addiction, 93*, 433–440.
- Fergusson, D. M., Lynskey, M. T., & Horwood, J. (1996). Comorbidity between depressive disorders and nicotine dependence in a cohort of 16-year-olds. *Archives of General Psychiatry, 53*, 1043–1047.
- Flay, B. R. (1993). Youth tobacco use: Risks, patterns, and control. In J. Slade & C. T. Orleans (Eds.), *Nicotine addiction: Principles and management* (pp. 365–384). New York: Oxford University Press.
- Fleming, S. E., & Lombardo, T. W. (1987). Effects of cigarette smoking on phobic anxiety. *Addictive Behaviors, 12*, 195–198.
- Gilbert, D. G. (1979). Paradoxical tranquilizing and emotion-reducing effects of nicotine. *Psychological Bulletin, 86*, 643–661.
- Gilbert, D. G., McClernon, J., Rabinovich, N. E., Plath, L. C., Jensen, R. A., & Meliska, C. J. (1998a). Effects of smoking abstinence on mood and craving in men: Influences of negative-affect-related personality traits, habitual nicotine intake and repeated measurements. *Personality and Individual Differences, 25*, 399–423.
- Gilbert, D. G., McClernon, J., Rabinovich, N. E., Plath, L. C., Masson, C. L., Anderson, A. E., et al. (1998b). Mood disturbance fails to resolve across 31 days of cigarette abstinence in women. *Journal of Consulting and Clinical Psychology, 70*(1), 142–152.
- Gilbert, D. G., Robinson, J. H., Chamberlin, C. L., & Spielberger, D. C. (1989). Effects of smoking/nicotine on anxiety, heart rate, and lateralization of EEG during a stressful movie. *Psychophysiology, 26*, 311–320.
- Gilbert, D. G., & Spielberger, C. D. (1987). Effects of smoking on heart rate, anxiety, and feelings of success during social interaction. *Journal of Behavioral Medicine, 10*, 629–638.
- Gilbert, D. G., & Wesler, R. (1989). Emotion, anxiety, and smoking. In T. Ney & A. Gale (Eds.), *Smoking and human behavior* (pp. 171–196). New York: Wiley.
- Glassman, A. H. (1993). Cigarette smoking: Implications for psychiatric illness. *American Journal of Psychiatry, 150*, 546–553.
- Glassman, A. H., Helzer, J. E., Covey, L. S., Cottler, L. B., Stetner, R., Tipp, J. E., et al. (1990). Smoking, smoking cessation and major depression. *Journal of the American Medical Association, 264*, 1546–1549.
- Grant, B. F., Hasin, D. S., Chou, S. P., Stinson, F. S., & Dawson, D. A. (2004a). 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.
- Grant, B. F., Stinson, F. S., Dawson, D. A., Chou, S. P., Dufour, M. C., Compton, W., et al. (2004b). Prevalence and co-occurrence of substance use disorders and independent mood and anxiety disorders: Results from the National Epidemiologic Survey on Alcohol and Related Conditions. *Archives of General Psychiatry, 61*, 807–816.
- Gwaltney, C. J., Shiffman, S., Paty, J. A., Liu, K. S., Kassel, J. D., Gnys, M., et al. (2002). Using self-efficacy judgments to predict characteristics of lapses to smoking. *Journal of Consulting and Clinical Psychology, 70*(5), 1140–1149.
- Haas, A. L., Muñoz, R. F., Humfleet, G. L., Reus, V. I., & Hall, S. M. (2004). Influences of mood, depression history, and treatment modality on outcomes in smoking cessation. *Journal of Consulting and Clinical Psychology, 72*(2), 563–570.
- Hall, S. M., Muñoz, R. F., & Reus, V. I. (1994). Cognitive-behavioral intervention increases abstinence rates for depressive-history smokers. *Journal of Consulting and Clinical Psychology, 62*, 141–146.
- Hall, S. M., Muñoz, R. F., Reus, V. I., & Sees, K. I. (1993). Nicotine, negative affect, and depression. *Journal of Consulting and Clinical Psychology, 61*, 761–767.
- Hatch, J. P., Bierner, S. M., & Fisher, J. G. (1982). The effects of smoking and cigarette nicotine content on smokers' preparation and performance of a psychologically stressful task. *Journal of Behavioral Medicine, 6*(2), 207–216.
- Herbert, M., Foulds, J., & Fife-Schaw, C. (2001). No effect of cigarette smoking on attention or mood in non-depressed smokers. *Addiction, 96*, 1349–1356.
- Himle, J., Thyer, B. A., & Fischer, D. J. (1988). Prevalence of smoking among anxious outpatients. *Phobia Practice and Research Journal, 1*, 25–31.
- Hitsman, B., Borrelli, B., McChargue, D. E., Spring, B., & Niaura, R. (2003). History of depression and smoking cessation outcome: A meta-analysis. *Journal of Consulting and Clinical Psychology, 71*(4), 657–663.
- Hughes, J. R. (1992). Tobacco withdrawal in self-quitters. *Journal of Consulting and Clinical Psychology, 60*(5), 689–697.
- Hughes, J. R. (1993). Pharmacotherapy for smoking cessation: Unvalidated assumptions, anomalies, and suggestions for future research. *Journal of Consulting and Clinical Psychology, 61*(5), 751–760.
- Hughes, J. R., Gust, S. W., Skoog, K., Keenan, R. M., & Fenwick, J. W. (1991). Symptoms of tobacco withdrawal. *Archives of General Psychiatry, 48*, 52–59.
- Hughes, J. R., & Hatsukami, D. K. (1986). Signs and symptoms of tobacco withdrawal. *Archives of General Psychiatry, 43*, 289–294.
- Hughes, J. R., Hatsukami, D. K., Mitchell, J. E., & Dahlgren, L. A. (1986). Prevalence of smoking among psychiatric outpatients. *American Journal of Psychiatry, 143*, 993–997.
- Hughes, J. R., Higgins, S. T., & Hatsukami, D. K. (1990). Effects of abstinence from tobacco: A critical review. In L. T. Kozlowski, H. M. Annis, H. D. Cappell, F. B. Glaser, M. S. Goodstat, Y. Israel, et al. (Eds.), *Research advances in alcohol and drug problems* (Vol. 10, pp. 317–398). New York: Plenum.
- Jarvik, M. E., Caskey, N. H., Rose, J. E., Herskovic, J. E., & Sadehghpour, M. (1989). Anxiolytic effects of smoking associated with four stressors. *Addictive Behaviors, 14*, 379–386.

- John, U., Meyer, C., Rumpf, H.-J., & Hapke, U. (2004). Smoking, nicotine dependence, and psychiatric comorbidity—A population-based study including smoking cessation after three years. *Drug and Alcohol Dependence, 76*, 287–295.
- Johnson, J. G., Cohen, P., Pine, D. S., Klein, D. F., Kasen, S., & Brook, J. S. (2000). Association between cigarette smoking and anxiety disorders during adolescence and early adulthood. *Journal of the American Medical Association, 284*, 2348–2351.
- Johnston, L. D., O'Malley, P. M., & Bachman, J. G. (1998). *Smoking among American teens declines some*. Ann Arbor, MI: University of Michigan News and Information Services [online]. Retrieved from <http://www.monitoringthefuture.org/press.html>
- Kandel, D. B., & Davies, M. (1986). Adult sequelae of adolescent depressive symptoms. *Archives of General Psychiatry, 43*, 255–262.
- Kandel, D. B., Huang, F.-Y., & Davies, M. (2001). Comorbidity between patterns of substance use dependence and psychiatric syndromes. *Drug and Alcohol Dependence, 64*(2), 233–241.
- Kassel, J. D., & Shiffman, S. (1997). Attentional mediation of cigarette smoking's effect on anxiety. *Health Psychology, 16*, 359–368.
- Kassel, J. D., Stroud, L. R., & Paronis, C. A. (2003). Smoking, stress, and negative affect: Correlation, causation, and context across stages of smoking. *Psychological Bulletin, 129*(2), 270–304.
- Kassel, D., & Unrod, M. (2000). Smoking, anxiety, and attention: Support for the role of nicotine in attentionally mediated anxiety. *Journal of Abnormal Psychology, 109*, 161–166.
- Kendler, K. S., Neale, M. C., MacLean, C. J., Heath, A. C., Eaves, L. J., & Kessler, R. C. (1993). Smoking and major depression: A causal analysis. *Archives of General Psychiatry, 4*, 231–244.
- Kenford, S. L., Smith, S. S., Wetter, D. W., Jorenby, D. E., Fiore, M. C., & Baker, T. B. (2002). Predicting relapse back to smoking: Contrasting affective and physical models of dependence. *Journal of Consulting and Clinical Psychology, 70*(1), 216–227.
- Killen, J. D., Fortmann, S. P., Kraemer, H. C., Varady, A. N., Davis, L., & Newman, B. (1996). Interactive effects of depression symptoms, nicotine dependence, and weight change on late smoking relapse. *Journal of Consulting and Clinical Psychology, 64*, 1060–1067.
- Leventhal, H., & Cleary, P. D. (1980). The smoking problem: A review of research and theory in behavioral risk modification. *Psychological Bulletin, 88*, 370–405.
- McCrae, R. R., Costa, P. T., & Bosse, R. (1978). Anxiety, extraversion, and smoking. *British Journal of Social and Clinical Psychology, 17*, 269–273.
- McNair, D., Lorr, M., & Droppleman, L. (1971). *Manual for the profile of mood states*. San Diego, CA: Educational and Industrial Testing Service.
- National Institute on Drug Abuse (2001). *Research report: Nicotine addiction* (NIH Publication No. 01-4342, pp. 1–8). Washington, DC: U.S. Department of Health and Human Services.
- Niaura, R., Britt, D. M., Borrelli, B., Shadel, W. M., Abrams, D. B., & Goldstein, M. G. (1999). History and symptoms of depression among smokers during a self-initiated quit attempt. *Nicotine and Tobacco Research, 1*, 251–257.
- Office on Smoking and Health (2002). *Smoking prevalence among U.S. adults*. Retrieved September 7, 2004, from http://www.cdc.gov/tobacco/research_data/adults_prev/prevali.htm
- Orlando, M., Ellickson, P. L., & Jinnett, K. (2001). The temporal relationship between emotional distress and cigarette smoking during adolescence and young adulthood. *Journal of Consulting and Clinical Psychology, 69*(6), 959–970.
- Pérez-Stable, E. J., Marín, G., Marín, B. V., & Katz, M. H. (1990). Depressive symptoms and cigarette smoking among Latinos in San Francisco. *American Journal of Public Health, 80*, 1500–1502.
- Pohl, R., Yeragani, V. K., Balon, R., Lycaki, H., & McBride, R. (1992). Smoking in patients with panic disorder. *Psychiatry Research, 43*, 253–262.
- Pomerleau, O. F., & Pomerleau, C. S. (1984). Neuroregulators and the reinforcement of smoking: Toward a biobehavioral explanation. *Neuroscience and Biobehavioral Reviews, 8*, 503–513.
- Pomerleau, O. F., & Pomerleau, C. S. (1988). *Nicotine replacement: A critical evaluation*. New York: Alan R. Liss.
- Pomerleau, O. F., Turk, D. C., & Fertig, J. B. (1984). The effects of cigarette smoking on pain and anxiety. *Addictive Behaviors, 9*, 265–271.
- Robinson, M. D., Anastasio, G. D., Little, J. M., Sigmon, J. L., Menscer, D., Pettice, Y. J., et al. (1995). Ritalin for nicotine withdrawal: Nesbitt's Paradox revisited. *Addictive Behaviors, 20*(4), 481–490.
- Rohde, P., Kahler, C. W., Lewinsohn, P. M., & Brown, R. A. (2004). Psychiatric disorders, familial factors, and cigarette smoking: III. Associations with cessation by young adulthood among daily smokers. *Nicotine and Tobacco Research, 6*, 509–522.
- Rohde, P., Lewinsohn, P. M., Brown, R. A., Gau, J. M., & Kahler, C. W. (2003). Psychiatric disorders, familial factors, and cigarette smoking: I. Associations with smoking initiation. *Nicotine and Tobacco Research, 5*, 85–98.
- Rose, J. E., Ananda, S., & Jarvik, M. E. (1983). Cigarette smoking during anxiety-provoking and monotonous tasks. *Addictive Behaviors, 8*, 353–359.
- Russell, M. A. H., Peto, J., & Patel, V. A. (1974). The classification of smoking by a factorial of motives. *Journal of the Royal Statistical Society, 137*, 313–346.
- Schneider, N. G., & Houston, J. P. (1970). Smoking and anxiety. *Psychological Reports, 26*, 941–942.
- Shiffman, S. M., & Waters, A. J. (2004). Negative affect and smoking lapses: A prospective analysis. *Journal of Consulting and Clinical Psychology, 72*(2), 192–201.
- Sonntag, H., Wittchen, H. U., Hofler, M., Kessler, R. C., & Stein, M. B. (2000). Are social fears and DSM-IV social anxiety disorder associated with smoking and nicotine dependence in adolescents and young adults? *European Psychiatry, 15*, 67–74.
- Spielberger, C., Gorsuch, R., & Lushene, N. (1970). *Manual for the state-trait anxiety inventory*. Palo Alto, CA: Consulting Psychologists Press.
- Takemura, Y., Akanuma, M., Kikuchi, S., & Inaba, Y. (1999). Cross-sectional study on the relationship between smoking or smoking cessation and trait anxiety. *Preventive Medicine, 9*, 496–500.
- Tercyak, K. P., Goldman, P., Smith, A., & Audrain, J. (2002). Interacting effects of depression and tobacco advertising receptivity on adolescent smoking. *Journal of Pediatric Psychology, 27*(27), 145–154.
- Wang, M. Q., Fitzhugh, E. C., & Green, B. L. (1996). Smoking trends among African-American and White adolescents in the United States. *Journal of Health Education, 26*, 61–62.
- Warburton, D. M., Revell, A., & Thompson, D. H. (1991). Smokers of the future. *British Journal of Addiction, 86*, 621–625.
- West, R. J. (1984). Psychology and pharmacology in cigarette withdrawal. *Journal of Psychosomatic Research, 28*(5), 379–386.
- West, R., & Hajek, P. (1997). What happens to anxiety levels on giving up smoking? *American Journal of Psychiatry, 154*, 1589–1592.
- Williams, S. G., Hudson, A., & Redd, C. (1982). Cigarette smoking, manifest anxiety and somatic symptoms. *Addictive Behaviors, 7*, 427–428.
- Windle, M., & Windle, R. (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.
- Wiseman, E. J. (1999). Smoking cessation and anxiety [Letter to the editor]. *American Journal of Psychiatry, 156*(2), 336.
- Wu, L. T., & Anthony, J. C. (1999). Tobacco smoking and depressed mood in late childhood and early adolescence. *American Journal of Public Health, 89*, 1837–1840.
- Zvolensky, M. J., Lejuez, C. W., Kahler, C. W., & Brown, R. A. (2004). Nonclinical panic attack history and smoking cessation: An initial examination. *Addictive Behaviors, 29*, 825–830.