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Addressing tobacco use disorder in smokers in early remission from alcohol dependence: The case for integrating smoking cessation services in substance use disorder treatment programs

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ABSTRACT

Despite the declining overall rate of cigarette smoking in the general population in the United States, the prevalence of smoking is estimated to be as high as 80% among treatment-seeking alcoholics. The serious adverse health effects of tobacco and heavy alcohol use are synergistic and recent evidence suggests that smoking slows the process of cognitive recovery following alcohol abstinence. In addition, substantial evidence shows that treatment for tobacco dependence does not jeopardize alcohol abstinence. In this paper, we focus on the impact and treatment implications of tobacco dependence among treatment-seeking alcoholics through a review of five areas of research. We begin with brief reviews of two areas of research: studies investigating the genetic and neurobiological vulnerability of comorbid tobacco and alcohol dependence and studies investigating the consequences of comorbid dependence on neurobiological and cognitive functioning. We then review literature on the effects of smoking cessation on drinking urges and alcohol use and the effectiveness of smoking cessation interventions with alcoholic smokers. Finally, we offer recommendations for research with an emphasis on clinical research for enhancing smoking cessation outcomes in this population.

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1. Introduction

The adverse health effects of tobacco use, most notably heart disease, cancers of the lung, throat and mouth, and chronic pulmonary obstructive disease, have been extensively documented in the literature (Surgeon General's Report, 2004). Approximately 435,000 smokers die each year in the United States as a result of smoking (Mokdad, Marks, Stroup, Gerberding, 2004). Evidence also has shown that the adverse health effects of chronic alcohol and tobacco use are synergistic (Castellsague et al., 1999; Pelucchi, Gallus, Garavello, Bosetti, & LaVecchia, 2006). Yet, while the overall rate of smoking in the United States has declined, the rates remain significantly elevated among both treatment-seeking and community-dwelling alcoholics. In a national epidemiological study, Grant, Hasin, Chou, Stinson, and Dawson (2004) found that the prevalence of nicotine dependence among people with alcohol dependence was over two times higher (45%) than in the general population. The prevalence of smoking in clinical populations of alcoholics is estimated to be as high as 80% (Hughes, 1995; Kalman, Morissette, & George, 2005).

Fortunately, and contrary to conventional wisdom, many alcohol and other substance dependent persons in early remission from a substance use disorder (SUD) are interested in smoking cessation treatment. For example, Orleans and Hutchinson (1993) found that 46% of substance dependent persons in treatment reported quitting smoking for 24 h or more in the past year and, in a survey of 108 substance dependent inpatients, Irving, Seidner, Burling, Thomas, and Brenner (1994) found that 49% were "very certain" they wanted to quit and 28% were "somewhat certain;" only 12% said they did not want to quit. However, many prefer to consider quitting smoking after resolving their drinking problem (Ellingstad, Sobell, Sobell, Cleland, & Agrawal, 1999). In addition, the preponderance of studies of concurrent smoking and alcohol treatment indicates that concurrent treatment does not jeopardize abstinence from alcohol and other non-nicotine drugs (Prochaska, Delucchi, & Hall, 2004).

Several studies suggest that people in alcohol and other drug recovery who have achieved long-term abstinence from non-nicotine drugs may not differ from other smokers in their ability to quit smoking (e.g., Hughes & Kalman, 2006; Kalman, Kahler, Garvey, & Monti, 2006; Prochaska et al., 2004; Sobell, Sobell, & Agrawal, 2002). By contrast, results from other studies suggest that it may be particularly difficult for persons who have achieved short-term abstinence from alcohol and other drugs to quit smoking. In a meta-analysis of eight clinical trials of smokers in treatment for a SUD, the mean quit rate at follow up for both intervention and control conditions was 7% (Prochaska et al., 2004). In our treatment study of smokers in alcohol recovery, smoking cessation outcome was related to length of sobriety at time of enrollment (Kalman et al., 2006). The quit rates of participants with greater than 12 months of sobriety vs. 12 or fewer months were 30% and 10%, respectively, at 36-week follow up (see also Joseph, Willenbring, Nugent, & Nelson, 2004). Taken together, these data suggest both that the alcohol treatment setting provides an important opportunity to address tobacco dependence and that innovative approaches are needed to enhance smoking cessation outcomes in this population.

Clinicians and program administrators are often unsure about whether and how to treat tobacco dependence in persons receiving treatment for comorbid alcohol dependence. The purpose of this review is to provide these professionals with clinically relevant and scientifically grounded information about the treatment of tobacco dependence in smokers with comorbid alcohol dependence. For many years, the scientific evidence was too scant to provide any guidance. However, a growing body of research has begun to answer these basic and important questions.

This review is divided into 5 sections. First, we provide a brief review of some of the genetic and neurobiological factors that appear to create a vulnerability to comorbid tobacco and alcohol dependence. Second, we briefly review studies on the adverse health effects of comorbid heavy drinking and tobacco use and the emerging literature on the consequences of comorbid dependence on neurobiological and cognitive functioning. Third, we update literature on the effects of smoking cessation on drinking urges and alcohol use, first reviewed by the first author of this paper over ten years ago (Kalman, 1998). Fourth, we discuss innovative approaches for improving the effectiveness of smoking cessation interventions with smokers with a recent (past year) history of alcohol problems. We conclude with directions for future research with an emphasis on recommendations for clinical research to enhance smoking cessation outcomes in this population.

2. The genetics and neurobiology of the comorbidity of tobacco and alcohol dependence

Twin studies have demonstrated that common genetic factors exert an important influence on the co-occurring use of tobacco and alcohol, and the role of genetic factors appears to be particularly strong among smokers with a history of alcohol dependence (Heath, Slutske, & Madden, 1997; Hopfer, Stallings, & Hewitt, 2002; Kozlowski et al., 1993; True et al., 1999; see also review by Tyndale, 2003). For example, in their study of male twins, True et al. (1999) found a substantial genetic correlation ($r = 0.68$) between lifetime nicotine and alcohol dependence and that 26% of the total variance in genetic risk for alcohol dependence overlapped with the genetic risk for nicotine dependence. Although attenuated, a significant association remains after controlling for potentially confounding variables such as general psychopathology and personality (Madden, Bucholz, Martin, & Heath, 2000).

Human studies suggest that nicotine intake primes alcohol consumption (Barrett, Tichauer, Leyton, & Pihl, 2006; Rose et al., 2004) and alcohol intake acutely increases smoking behavior and nicotine reward (McKee, Krishnan-Sarin, Shi, Mase, & O'Malley, 2006; Mitchell, DeWitt, & Zacny, 1995; Rose et al., 2002). Animal studies suggest that such "cross sensitivity" has a genetic component (see review in Balogh, Owns, Butt, Wehner, & Collins, 2002). Rat and mice lines that are selectively bred to be high in alcohol sensitivity are also more sensitive to some of the effects of nicotine, including its anxiolytic and locomotor depressant effects and nicotine-induced hypothermia (e.g., Blomqvist, Ericson, Johnson, Engel, & Soderpalm, 1996; Cao et al., 1993; de Fiebre & Collins, 1991; de Fiebre et al., 2002; Gordon, Meehan, & Schechter, 1993). Le et al. (2006) demonstrated cross-sensitivity among offspring in an animal study.

Evidence has also been found in animal and human studies for cross tolerance between nicotine and alcohol (Balogh et al., 2002). In a study with human subjects, there was near complete overlap in women between genetic influences on risk of cigarette smoking and decreased sensitivity to alcohol intoxication following a challenge dose of alcohol; the genetic correlation for men was nonsignficant, however (Madden, Health, & Martin, 1997). Some have speculated that cross sensitivity is to the rewarding effects and cross tolerance is to the aversive effects of these substances (Collins & Marks, 1995; Perkins, 1997; Pomerleau, 1995).

The Collaborative Study on the Genetics of Alcoholism (COGA) has investigated the genetic basis of the alcohol/tobacco dependence phenotype (see reviews in Bierut, Schuckit, Hesselbrock, & Reich, 2000 and Grucza & Bierut, 2006).[1] Findings reflect the fact that multiple potential genetic pathways are likely to be involved in comorbid tobacco and alcohol dependence. For example, Ye, Zhong, and Zhang (2005) identified eighteen single nucleotide polymorphisms (SNPs) in specific chromosomal regions located on eight genes that may predispose to vulnerability to the use of both substances. As

[1] Note that the COGA assessed for "habitual smoking" (i.e., daily smoking) but did not conduct a diagnostic assessment for tobacco dependence.

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Li, Volkow, Baler, and Egli (2007) suggest, these genetic pathways, moreover, are likely to involve "multiple genes that interact with one another and with the environment in ways that are strongly influenced by developmental processes" (p. 2). It is also important to emphasize that the risk conferred by some of these pathways may be common to substance use or abuse in general and not unique to the co-use or abuse of alcohol and tobacco (Young, Lawford, Nutting, & Noble, 2004). Finally, the importance of environmental factors is suggested by studies demonstrating that a protective environment moderates the inherited risk across a range of psychopathologies, including substance misuse (e.g., Miles, Silberg, Pickens, & Eaves, 2005).

Advances are also being made in identifying and understanding the neurobiological mechanisms that mediate genetic risk for comorbid alcohol and tobacco dependence. For example, Owens et al. (2003) found strong evidence that sensitivity to the effects of both nicotine and alcohol on acoustic startle in mice is mediated by polymorphisms in genes that code for nicotinic acetylcholine receptors (nAChRs). An association between polymorphisms in these genes and sensitivity to both alcohol and cigarettes has also been found in a human study (Ehringer et al., 2007). Polymorphisms in other receptor systems, including the dopaminergic, gamma-aminobutyric acid, and opioid systems, may also account for individual differences in sensitivity to alcohol and nicotine (Agrawal et al., 2008; Connor et al., 2007; Ray et al., 2006).

3. Health consequences of comorbid tobacco and alcohol dependence

Both alcohol and tobacco use increase the risk of cancers of the upper respiratory and digestive tracts, including cancer of the mouth, throat, larynx and esophagus (Bagnardi, Blangiardo, LaVecchia, & Corrao, 2001; Talamini et al., 1998). Their combined use multiplies the risk (see also Pelucchi, Gallus, Garavello, Bosetti, & LaVecchia, 2006). For example, at the highest level of joint consumption of these substances, Castellsague et al. (1999) found that compared to men who neither smoked nor drank, the odds ratio for esophageal cancer was 6.84 for men who never drank but smoked heavily, 14.13 for men who drank heavily but never smoked, and 50.85 for men who both drank and smoked heavily. Another study found that the risk of mouth and throat cancer among people who drank heavily and smoked was 300 times higher than people who neither smoked nor drank (Zheng et al., 2004). In addition, a twenty year retrospective study found that alcoholic smokers in alcohol dependence treatment were more likely to die from the effects of tobacco (all causes) than alcohol (Hurt et al., 1996).

Recent studies also demonstrate that chronic cigarette smoking compounds both structural and functional alcohol-induced brain impairment. Compared to their nonsmoking counterparts, alcoholic smokers have smaller temporal, cortical and total gray matter volumes, larger frontal white matter volumes and poorer cerebral perfusion (Durazzo, Cardenas, Studholme, Weiner, & Meyerhoff, 2007; Durazzo, Gazdzinski, Banys, & Meyerhoff, 2004; Gazdzinski et al., 2006; Gazdzinski et al., 2005; Mon, Durazzo, Gazdzinski, & Meyerhoff, 2009). Durazzo et al. (2004) also found lower concentrations of the metabolite, *N*-acetylaspartate in frontal white matter and midbrain and lower concentrations of choline in the midbrain. Lower levels of *N*-acetylaspartate are believed to contribute to neuronal atrophy and loss (Schuff et al., 2001), and lower concentrations of choline compromise the integrity of cell membranes (e.g., synthesis and turnover; Miller et al., 1996).

Consistent with these findings, alcoholic smokers have poorer cognitive functioning relative to their nonsmoking counterparts across a broad range of measures, including processing speed, auditory–verbal learning and auditory–verbal memory (Durazzo, Rothlind, Gazdzinkski, Banys, & Meyerhoff, 2006; Friend, Malloy, & Sindelar, 2005; Glass et al., 2006). Smoking severity (but not severity of alcohol use) was inversely correlated with measures of cognitive functioning among current heavy drinkers and alcoholics following one month of alcohol abstinence (Durazzo, Rothlind, Gazdzinski, & Meyerhoff, 2008; Durazzo et al., 2006). Interestingly, Durazzo et al. (2008) did not find comparable effects of medical or psychiatric/other drug use comorbidities on cognitive functioning in this population.

4. The effect of smoking cessation on drinking urges and alcohol use

Recent studies have investigated the importance of smoking as a strategy to cope with urges to drink, the effect of smoking deprivation on drinking urges, and the effect of smoking cessation on risk of alcohol relapse (see Kalman, 1998, for a previous review and discussion of related theory). These studies, which are reviewed below (see also see Table 1), are directly relevant to a belief which causes some clinicians to question the wisdom of encouraging their clients to quit smoking, i.e., that smoking may be an important strategy for coping with urges to drink and, therefore, smokers in this population who quit smoking may undermine their sobriety. The studies reviewed below provide very limited support for this belief.

4.1. Smoking as a strategy to cope with urges to drink

Several studies have investigated the role of smoking as a strategy to cope with urges to drink. In their study of 116 smokers in alcohol treatment, Monti, Rohsenow, Colby, and Abrams (1995) reported that only 20% said they believed smoking decreases their urge to drink. Similarly, in their sample of 130 smokers enrolled in a smoking cessation trial, Kalman et al. (2001) reported that only 29% of participants said that smoking would help them cope with an urge drink during periods of sobriety; among these participants, only 8% said it would help them to cope "a lot". In their study of smokers in alcohol treatment, Asher, Martin, Rohsenow, Traficante, and Monti (2003) reported that only 13% said that their urges to drink would be too strong to resist if they quit smoking. In an ongoing trial of smoking cessation treatment, one week after their quit day, Kalman et al. (unpublished) are asking participants to report on the effect of their smoking quit attempt on their ability to stay sober. Although about 50% have said that trying to quit increased their stress level, only 5% said that it made trying to stay sober a "little more difficult;" none have said it made trying to stay sober a lot more difficult and 45% said it made it either a little or a lot easier to stay sober. Finally, Rohsenow, Colby, Martin, and Monti (2005) reported that smoking to cope did not predict substance use status three months after the start of treatment (the effect size was zero). These findings suggest that only a small minority of smokers in alcohol recovery consider smoking to be an important strategy for coping with urges to drink. There is little evidence from these studies that smoking, in fact, decreases the risk of alcohol relapse.

4.2. The effect of smoking deprivation on urges to drink

Cooney and colleagues conducted a cue reactivity study and found that smoking deprivation does not increase urge to drink in early sobriety (Cooney, Cooney, Pilkey, Kranzler, & Onken, 2003). These investigators recruited 40 alcohol-dependent, heavy smokers in alcohol treatment; mean number of days of abstinence was 16.8. When participants were exposed to alcohol cues following 34 h of smoking deprivation, they did not report any increase in urge to drink compared to the effect of exposure to a neutral (water) cue on drinking urge (see also Monti et al., 1995). Colby et al. (2004) replicated these findings in a sample of primarily college-age moderate to heavy drinking smokers. In addition, in their study, smoking deprivation did not influence psychophysiological reactions to alcohol cues (i.e., salivation, heart rate) or the

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Table 1
Studies of the effect of smoking cessation on drinking urges and alcohol use.

Study	Participants and design	Findings
Studies of smoking as a strategy to cope with urges to drink		
Monti et al. (1995)	A questionnaire study of 116 smokers in residential SUD treatment for alcohol problems.	58% of subjects reoported that they have smoked to cope with drinking urges, but only 20% reported that smoking decreases their urge to drink.
Asher et al. (2003)	A questionnaire study of 96 smokers in residential treatment for alcohol problems. Subjects were enrolled in a smoking intervention clinical trial but were not required to quit.	41% of subjects said quitting smoking during AOD treatment would make it harder to stay sober; however, only 13% said that their urges to drink would be too strong to resist if they quit smoking.
Rohsenow et al. (2005)	A questionnaire study of 160 smokers in residential SUD treatment for alcohol problems. Subjects were enrolled in a smoking intervention clinical trial but were not required to quit.	30% of subjects said quitting smoking during alcohol treatment would make it harder to stay sober; smoking to cope with AOD urges did not predict AOD use (i.e., relapse) three months after the start of SUD treatment.
Kalman et al. (2001)	A questionnaire study of 80 smokers in residential SUD treatment for alcohol problems. Subjects were enrolled in a smoking cessation clinical trial.	29% of subjects said that smoking would help them cope with an urge to drink during periods of sobriety; among these participants, only 8% said it would help them to cope "a lot".
Kalman et al. (unpublished)	A questionnaire study of 130 smokers in residential SUD treatment for alcohol problems. Subjects were enrolled in a smoking cessation clinical trial. One week following their quit day, subjects were asked to report on the effect of quitting smoking on their effort to abstain from alcohol.	5% of subjects said that quitting smoking made trying to abstain from alcohol a "little more difficult;" none said it made trying to abstain "a lot more difficult;" The reamining subjects said either that quitting made abstaining from alcohol either a little or a lot easier (45%) or that it had no effect (50%).
Studies of the effect of smoking deprivation on urges to drink		
Cooney et al. (2003)	40 alcohol-dependent, heavy smokers in SUD treatment for alcohol problems (mean number of days of abstinence was 16.8). Subjects participated in two laboratory sessions: one following 34 h of smoking deprivation.and one following ad libitum smoking. In both sessions, subjects were exposed to alcohol and neutral (water) cues.	Urge to drink was not affected by nicotine deprivation during alcohol cue exposure.
Colby et al. (2004)	College-age moderate to heavy drinking smokers participated in two laboratory sessions: one following 5 h of smoking deprivation.and one following ad libitum smoking. In both sessions, subjects were exposed to alcohol and neutral (water) cues.	Subjects did not report any increase in urge to drink or psycho-physiological reactions (i.e., salivation, heart rate) during alcohol cue exposure vs. exposure to a neutral (water) cue. Smoking deprivation did not influence the amount of alcohol consumed immediately following the cue reactivity procedure.
Cooney et al. (2007)	102 subjects participated in smoking cessation treatment in a SUD program and provided EMA data for 14 days following discharge from the program. Subjects recorded their urge to drink immediately prior to smoking, 5 min after the onset of smoking, and at random prompts.	Frequency of drinking urges among smoking abstinent participants did not differ from those who returned to smoking following concurrent alcohol and tobacco treatment; smoking modestly *increased* urge to drink
Palfai et al. (2000)	In a 2 × 2 factorial design, 56 nontreatment-seeking moderate to heavy drinking smokers participated in one of four conditions: (1) exposure to smoking cues following 6 h of smoking deprivation; (2) exposure to smoking cues following ad libitum smoking; (3) exposure to neutral cues following 6 h of smoking deprivation; (4) exposure to neutral cues following 6 h of smoking deprivation.	Smoking deprivation significantly increased urge to drink and alcohol consumption. Alcohol expectancies partially mediated the relationship between smoking deprivation and alcohol consumption. Cue exposure condition did not affect results.

Note. AOD = alcohol and other drug use. EMA = ecological momentary assessment. SUD = substance use disorder.

amount of alcohol consumed immediately following the cue reactivity procedure. In a study using ecological momentary assessment, Cooney et al. (2007) reported similar findings: frequency of drinking urges among smoking abstinent participants did not differ from those who returned to smoking following concurrent alcohol and tobacco treatment, and smoking modestly *increased* urge to drink. However, an earlier cue reactivity study of nontreatment-seeking moderate to heavy drinking smokers found that smoking deprivation significantly increased urge to drink and alcohol consumption (Palfai, Monti, Ostafin, & Hutchinson, 2000). Alcohol expectancies partially mediated the relationship between smoking deprivation and alcohol consumption (i.e., smoking deprivation activated alcohol-related cognitive schema); other potential cognitive and affective variables (smoking withdrawal-induced negative affect or processing of alcohol-relevant information) were not significant mediators.[2]

4.3. The effect of smoking cessation treatment on alcohol and other drug outcomes

The preponderance of studies of concurrent smoking and alcohol treatment indicates that concurrent tobacco dependence treatment does not jeopardize alcohol and other non-nicotine drug outcomes. In their meta-analysis of twelve clinical trials of concurrent tobacco and AOD treatment, Prochaska et al. (2004) found that participants in the concurrent intervention vs. alcohol treatment only condition were significantly more likely to be abstinent from alcohol and other drugs: 37% and 31%, respectively, in the intervention and comparison conditions. A subsequent study provided further support for this finding (Friend & Pagano, 2005). However, in the largest study specifically designed to investigate this issue, Joseph et al. (2004) found that alcohol use outcomes for participants in the concurrent condition were significantly poorer than for participants in the condition in which smoking cessation treatment was provided six months following an alcohol treatment episode (the "delayed" condition): at 6-month follow up, 41% of participants in the concurrent condition vs. 56% of participants in the delayed tobacco dependence treatment condition had achieved prolonged alcohol abstinence (see also Grant et al. (2003)). Among alcohol relapsers, an inverse relationship has been found between number of cigarettes smoked following relapse and drinking frequency, suggesting that smoking may be serving a coping function (Gulliver et al. 2000; but see Friend & Pagano, 2005). Consistent with most clinical trials, however, a large-scale naturalistic study found that smoking cessation was associated with better AOD outcomes (Kohn, Tsoh, & Weisner, 2003).

5. Improving smoking cessation treatment for alcoholic smokers in early remission

There are many reasons to provide concurrent tobacco and alcohol dependence treatment. Many of these reasons have already been

[2] The period of deprivation does not appear to explain the discrepancy: smokers in two studies were deprived for 5 h (Colby et al., 2004) and 34 h (Cooney et al., 2003), and smokers in the third study were deprived 6 h (Palfai et al., 2000). Nor was sample size related to result. And while there were differences between the three studies in drinking and smoking histories of participants, the differences were greater between the two studies showing no effect of smoking deprivation (Colby et al., 2004; Cooney et al., 2003) than between one of the studies showing no effect and the one positive study (Colby et al., 2004; Palfai et al., 2000).

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discussed in this review: the serious health effects of smoking, the synergistic adverse health effects of comorbid tobacco and alcohol use, the adverse effects of smoking on neurobiological and cognitive recovery from alcoholism, the fact that the majority of these smokers are concerned about their smoking and do not believe that quitting would threaten their sobriety, and that the majority of studies indicate that concurrent treatment does not compromise and even seems to enhance alcohol and other drug outcomes. In addition, alcohol consumption appears to potentiate the rewarding value of smoking (Rose et al., 2002). If the positive effects of smoking are diminished during alcohol abstinence, as this finding suggests, these smokers may be more receptive to motivational and cessation interventions at this time. In this section, we will review studies related to (1) interventions for alcoholic smokers who do not express a readiness to quit, (2) cessation interventions for alcoholic smokers who do express a readiness to quit, and (3) interventions designed to fully integrate tobacco dependence treatment into SUD programs.

5.1. Interventions for alcoholic smokers who are not ready to quit

While many alcoholic smokers are concerned about their smoking, a minority are motivated to make a quit attempt during alcohol treatment (e.g., Flach & Diener, 2004). While these investigators and others (Monti et al., 1995) also reported that interest in quitting smoking increases with greater alcohol abstinence, these smokers are less likely to present for smoking cessation treatment services following discharge (Joseph et al., 2004; Kalman et al., 2001). Alcoholic smokers also make fewer quit attempts in their lifetimes (Hughes & Kalman, 2006). Taken together, the alcohol treatment setting would appear to provide an important opportunity to intervene for the purpose of enhancing motivation to quit smoking.

Two studies have investigated the efficacy of an intervention to enhance motivation to quit smoking among alcoholic smokers (see Table 2A). Bobo, McIlvain, Lando, Walker, and Leed-Kelly (1998) randomly assigned 12 residential drug treatment centers to an intervention or control condition. The intervention condition, which was based on the stages of change model, consisted of four 10–15 minute individual counseling sessions. Only the first counseling session was delivered during a participant's residential stay, however; the remaining three were delivered eight, twelve and sixteen weeks after discharge. At one-, six-, and 12-month follow up, there was no difference in the percentage of smokers who reported quitting for at least 24 h. There were also no differences in 7-day point prevalence abstinence: at the twelve-month follow up the rates for the intervention and control conditions were 9% and 7%, respectively. As the authors stated, the absence of a significant difference may reflect, in part, the low intensity of the planned intervention. In addition, only 31% completed all sessions and 30% completed a single session. Rohsenow, Monti, Colby, and Martin (2002) randomly assigned 126 alcoholic smokers to one of four conditions in the first week of their stay in a 30-day SUD treatment program. Participants received either (1) a single session of brief advice; (2) three sessions of brief advice; (3) a single session of motivational enhancement; (4) three sessions of motivational enhancement. The motivational enhancement intervention consisted of exploring the pros and cons of smoking, imagining life without cigarettes, providing personalized feedback and collaborative goal-setting. Contrary to their hypothesis, smoking abstinent rates were higher in the brief advice conditions at one- and six-month follow up; at six months, the rates were 13% for brief advice and 2% for motivational enhancement ($p < .08$). Thus, a more directive message was associated with greater efficacy. Indeed, 13% in a study of a brief intervention with smokers not necessarily ready to quit at time of recruitment is notable. Booster sessions did not significantly increased cessation rates compared to the single session conditions.

Research is greatly needed to investigate the efficacy of sustained, higher-intensity interventions. These interventions should address known barriers to quitting in this population and be fully integrated into the SUD programs, not "stand alone" initiatives (Asher et al., 2003; Ziedonis, Guydish, Williams, Steinberg, & Foulds, 2006; see also below, Integrating tobacco treatment into SUD treatment). In other words, they should be accorded the same seriousness, priority and intensity given to interventions designed to enhance motivation to abstain from alcohol or any other drug. Few would argue that "one shot," low-intensity interventions would be appropriate for comorbid cocaine or heroin or marijuana use among alcoholics. The health toll that tobacco use incurs on alcoholic smokers merits the same attention. Research is needed to evaluate the efficacy of comparable interventions on motivation to abstain from tobacco use, and SUD programs provide an ideal setting for these investigations

5.2. Cessation interventions for alcoholic smokers who are ready to quit

Successful cessation is particularly difficult for smokers with a recent history of alcohol problems. As discussed earlier, in a meta-analysis of eight clinical trials of concurrent smoking and alcohol treatment, the mean quit rate at follow up for both intervention and control conditions was 7% (see Table 1 in Prochaska et al., 2004, for a description of each study). In our treatment study of smokers in alcohol recovery, smoking cessation outcome was significantly related to length of sobriety at time of enrollment (Kalman et al., 2006): participants with greater than 12 months of alcohol abstinence at the time of enrollment had a significantly higher quit rate than participants less than a year of sobriety (the 7-day point prevalence quit rates at 24-week follow up were 30% and 10%, respectively). In the largest clinical trial to date of smoking cessation treatment with smokers in treatment for alcohol dependence, Joseph et al. (2004) randomly assigned participants to receive smoking cessation treatment either during alcohol treatment (the concurrent treatment condition) or six months later (the delayed treatment condition). At follow up, the smoking abstinence rates were 12% and 14%, respectively, in the concurrent and delayed conditions.

There are several reasons why smokers with a recent history of alcohol problems have difficulty achieving long-term tobacco abstinence. First, relapse to tobacco use may be precipitated by a return to alcohol use. For example, in a clinical trial of concurrent tobacco and alcohol treatment by Burling, Burling, and Latini (2001), smoking cessation rates at one-year follow up were between 29% and 50% for alcohol and other drug abstinent participants and between 0% and 3% for nonabstinent participants (see also McKee et al., 2006; Shiffman et al., 1997). Second, compared to smokers without a recent AOD history, alcoholic smokers tend to be highly nicotine dependent, experience more craving and more severe withdrawal (Currie, Hodgins, El-Guebaly, & Campbell, 2001; Gulliver et al., 1995; Hertling et al., 2005; Marks, Hill, Pomerleau, Mudd, & Blow, 1997); more severe withdrawal is observed even after controlling for nicotine dependence (Marks et al., 1997). Withdrawal effects appear to be most strongly mood related. In their retrospective study, the most significant difference between alcoholic smokers and nonalcoholic smokers was found for the effects of cessation on mood: 31% of alcoholic smokers vs. 5% of nonalcoholic smokers reported feeling depressed following a smoking quit attempt; other between-group differences included irritability or anger, nervousness, restlessness and trouble concentrating. (Marks et al., 1997). Third, as discussed earlier, in a minority of smokers with a recent history of alcohol problems, smoking may be used as a resource for coping with drinking urges or, alternatively, the self-control strength required to cope with drinking urges may deplete a smoker's ability to cope with smoking urges. Fourth, additional comorbidities (e.g., depression) may interfere with cessation among smokers with alcohol dependence (Ait-Daoud et al., 2006; Kodl et al., 2008). Fifth, smoking may attenuate the severity of alcohol withdrawal symptoms by reducing the up-regulation of $GABA_A$ receptors following alcohol abstinence (Mason,

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Table 2
Selected studies of innovative approaches to smoking cessation treatment with relevance to smokers in alcohol recovery.

Study	Participants and design	Intervention	Findings
A. Studies of interventions to enhance motivation to quit smoking among alcoholic smokers			
Bobo et al. (1998)	12 SUD residential treatment programs were randomized to either a tobacco cessation or control condition. 575 smokers participated.	Four 10–15 minute counseling sessions tailored to motivational readiness to quit. The first session occurred during subject's residential stay. Remaining sessions occurred 8, 12 and 16 weeks after discharge.	No significant effects of intervention on tobacco quit attempts or tobacco abstinence at one-, six-, or twelve-month follow up. At 12-month follow up, the quit attempt rate was 54% vs. 49% and the tobacco abstinence rate was 9% and 7% in the intervention and control groups, respectively. Only 31% of subjects in the intervention condition received all four counseling sessions.
Rohsenow et al. (2005)	126 subjects in SUD residential treatment were randomized to (1) one session of brief advice (10 min); (2) three sessions of brief advice; (3) one session of motivational enhancement (50 min); (4) three sessions of motivational enhancement. In conditions two and four, the additional sessions (15 min each) were scheduled one and four weeks after the initial session.	Brief advice consisted of direct advice to quit smoking with referral for treatment. Motivational enhancement consisted of exploring pros and cons of smoking, imagining life without smoking, providing personalized feedback and setting stage-specific goals; referral for treatment was also offered.	Smoking abstinent rates were higher in the brief advice conditions at one- and six-month follow up. At six months, the rates for subjects in conditions one and three were 13% and 2% ($p < .08$), respectively. Only 49% of subjects in the conditions two and four received all three sessions.
B. Pharmacological studies of smoking cessation treatment			
Kalman et al. (2006)	130 smokers in residential SUD treatment (103 with between two and twelve months of alcohol abstinence at enrollment) participated in a randomized, double-blind, placebo-controlled clinical trial.	Subjects received either 21-mg or 42-mg transdermal nicotine. Treatment was provided for eight weeks. All subjects also received counseling.	Among the subgroup of subjects with two to twelve months of abstinence, smoking abstinence rates at 36-week follow up in the 21-mg and 42-mg conditions were 11% and 9%, respectively (difference not significant).
Johnson et al. (2005)	94 smokers with alcohol problems participated in a randomized, double-blind, placebo-controlled clinical trial. Note that the study was part of a larger investigation of the efficacy of topiramate for alcohol dependence and subjects did not have to express a readiness to quit smoking to enroll.	Subjects received either placebo or an escalating dose of topiramate (maximum dose = 300 mg per day). Treatment was provided for twelve weeks.	Smoking cessation rates in the topiramate and placebo conditions were 17% vs. 7%, respectively, at the end of 12 weeks ($p = .04$).
Ebbert et al. (2009)	239 smokers participated in a quasi-experimental study of smoking cessation treatment Study did not enroll smokers with recent alcohol problems.	Subjects received either nicotine replacement (historical controls) or 21-mgs of nicotine replacement plus varenicline (experimental group).	At six-month follow up, smoking cessation rates for subjects in the nicotine replacement plus varenicline and nicotine replacement only condition were 54% and 59%, respectively (difference not significant).
O'Malley et al. (2006)	400 smokers who smoked at least 20 cigarettes per day participated in a randomized, double-blind, placebo-controlled clinical trial. Study did not enroll smokers with recent alcohol problems.	Subjects received either 21-mg transdermal nicotine plus either 0, 25, 50 or 100 mg per day of naltrexone. Treatment was provided for six weeks. All subjects also received counseling.	There was a trend favoring subjects assigned to the 100-mg naltrexone vs. placebo condition at the end of treatment (52% vs. 39%, respectively) in the intent-to-treat analysis. Among treatment completers, the quit rates among smokers in the 100-mg vs. placebo conditions were 72% and 48%, respectively ($p = .004$). Lower naltrexone doses had little effect compared to placebo.
Byars et al. (2005)	44 female smokers participated in a randomized, double-blind, placebo-controlled clinical trial. Study did not enroll smokers with recent alcohol problems.	Subjects received 21 mg transdermal nicotine plus either 0 or 50 mg per day of naltrexone for 12 weeks. All subjects also received counseling.	Analyses were reported for treatment completers only ($n = 12$ in each condition). Continuous abstinence rates at end of treatment were 92% and 50% in the naltrexone and placebo conditions, respectively ($p = .029$).
Krishnan-Sarin et al. (2003)	32 smokers who smoked 20–30 cigarettes per day participated in a randomized, double-blind, placebo-controlled clinical trial. Study did not enroll smokers with recent alcohol problems.	Subjects received either 21-mg transdermal nicotine plus either 0- or 50-mg per day of naltrexone. Treatment was provided for four weeks.	Continuous abstinence rates during the final two weeks of the study were 56% and 31%, respectively, in the naltrexone and placebo conditions (significance level not reported because of small sample).
Lerman et al. (2004)	216 smokers participated in a randomized, double-blind, placebo-controlled clinical trial. Study investigated the moderating role of a functional variant of the mu-opioid receptor on the efficacy of trandermal nicotine vs. nicotine nasal spray. Study did not enroll smokers with recent alcohol problems.	Subjects received either transdermal nicotine or nicotine nasal spray.	There was a significant effect of genotype on abstinence at the end of treatment in the transdermal nicotine group (52% vs. 33%; $p = .02$) but no significant effect in the nasal spray group (29% vs. 30%). The effect of genotype on abstinence at follow up was not significant.
Biberman et al. (2003)	109 smokers participated in a randomized, double-blind, placebo-controlled clinical trial. Study did not enroll smokers with recent alcohol problems.	Subjects received either 10-mg selegiline plus nicotine patch or placebo plus nicotine patch. Selegiline and placebo were administered for 26 weeks. The nicotine patch was administered for 8 weeks.	Continuous abstinence rates at one-year follow up in the selegiline and placebo groups were 25% vs. 11%, respectively ($p = .08$).
Cornuz et al. (2008)	229 smokers participated in a randomized, double-blind, placebo-controlled clinical trial. Study did not enroll smokers with recent alcohol problems.	Subjects received five monthly injections of a nicotine vaccine or placebo.	At 6-month follow up, continuous abstinence rates in the nicotine and placebo groups were 40.3% and 31.3% (not significant). A significant difference in abstinence rates was found for subjects in the active medication group with the highest antibody levels vs. subjects in the placebo group (57% vs. 31%, respectively; $p = .004$).

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Table 2 (*continued*)

Study	Participants and design	Intervention	Findings
C. Studies of behavioral interventions for smoking cessation with smokers in SUD programs			
Burling et al (2001)	150 smokers in residential SUD treatment participated in a randomized clinical trial.	Subjects received either (1) a multicomponent smoking treatment (MST) focused exclusively on smoking cessation; (2) a multicomponent treatment plus generalization (MST + G) that used the smoking cessation experience as an opportunity for "generalization training" from cigarettes to alcohol, i.e., participants examined the similarities between successfully quitting smoking and AOD use; (3) a no treatment control (residents who refused smoking cessation treatment). The smoking cessation intervention in conditions one and two occurred several times per week for nine weeks.	At one-month follow up, the smoking cessation rates in the MST and MST + G conditions were 40% and 27%, respectively. At 12-month follow up, the rates in the MST and MST + G conditions were 19% and 13%, respectively.
Joseph et al. (2004)	499 smokers in residential and day SUD treatment programs participated in a randomized clinical trial.	Subjects were randomly assigned to receive smoking cessation treatment during alcohol treatment (concurrent condition) or six months following treatment (delayed condition)	At 18-month follow up, smoking cessation rates in the concurrent and delayed treatment conditions were 12% and 14%, respectively (difference not significant).
D. Organizational change study of smoking cessation treatment in SUD programs			
Guydish et al. (2009)	Assessments of an organizational change model (ATTOC) designed to promote the integration of tobacco dependence treatment into substance abuse treatment were conducted prior to and following its implementation in 3 residential SUD treatment programs.	The intervention consisted of several consultations over a period of six months which are designed to help administrative and clinical staff in SUD programs to develop tobacco use policies and clinical practices consistent with the principle that tobacco dependence treatment is central to the mission of SUD treatment	Statistically significant pre- to post-test changes were found on several measures, including staff beliefs about providing smoking cessation services improved ($p = .0002$), counselor self-efficacy in addressing tobacco dependence with clients ($p = .0004$), and smoking-related practices used by counselors ($p = .0033$). Residents also reported significant increases in the amount of nicotine dependence services received ($p < .0001$), and more favorable attitudes about smoking cessation during addiction treatment ($p < .0001$).

Note. Several but not all studies in this table investigated innovative approaches to smoking cessation treatment. Study samples are described.
Under "Participants and Design." ATTOC = Addressing tobacco treatment through organziation change. SUD = substance use disorder.
Cotinine is a metabolite of nicotine and has a longer half-life than nicotine.

2005; Staley et al., 2005). Finally, chronic alcohol use may alter the molecular mechanisms of nicotine reinforcement, including nAChRs. Alteration of mechanisms that mediate the reinforcing value of nicotine may also alter the efficacy of medications targeting these receptors for the purpose of treating tobacco dependence (see Littleton, Barron, Prendergast, & Nixon, 2007).

5.3. Pharmacological treatment

Several studies have investigated the efficacy of innovative pharmacological interventions (see Table 2B). Some of these studies have recruited smokers in alcohol recovery (e.g., Kalman et al., 2006); other studies, which have investigated pharmacotherapies in unselected samples, may be promising as approaches with smokers in recovery (O'Malley et al., 2006). The fact that standard combinations of behavioral and pharmacological treatment (e.g., weekly counseling plus 21-mg patch for 8–12 weeks) have produced disappointing results in alcoholic smokers suggests that smokers with a recent history of alcohol problems may benefit from more intensive treatment. In an investigation of high-dose nicotine patch therapy, the quit rates of smokers receiving 42-mg vs. 21-mg of transdermal nicotine were not significantly different at 6-month follow up (Kalman et al., 2006; see also Hurt et al., 2005). The first author of this paper is currently conducting a study of combination pharmacotherapy for smokers with one to twelve months of sobriety. Participants are assigned to nicotine patch plus bupropion or patch plus placebo. Fiore et al. (2008) recommend this combination on the basis of their meta-analysis of three trials. However, to our knowledge, this is the only study to date of combination pharmacotherapy with alcoholic smokers.

Other combinations of pharmacotherapies have shown promise in unselected smokers warrant investigation with alcoholic smokers (see review in Fiore et al., 2008). For example, in a dose-ranging study of naltrexone plus transdermal nicotine, O'Malley et al. (2006) reported some evidence for the efficacy of 100-mg naltrexone in unselected smokers. In an intent-to-treat analysis, there was a trend favoring participants assigned to the 100-mg naltrexone vs. placebo condition at the end of treatment; among treatment completers, the quit rate among these smokers was significantly higher (odds ratio = 2.73; $p = .004$). Significant differences between these two groups were also found for withdrawal symptoms. Long-term quit rates were not significantly different, however (see also Byars, Frost-Pineda, Jacobs, & Gold, 2005; Krishnan-Sarin, Meandzija, & O'Malley, 2003). In a study of treatment for alcohol dependence, Oslin et al. (2003) found that naltrexone response was associated with a polymorphism in the mu-opioid receptor gene, OPRMI. Ray et al. (2006) reported some evidence that this polymorphism is associated with response to both nicotine and alcohol. Consistent with studies demonstrating that nicotine increases release of β-endorphin (Boyadjieva, Reddy, & Sarkar, 1997), Lerman et al. (2004) found that an allele in the same opioid receptor gene moderated end of treatment response to transdermal nicotine but not response to nicotine nasal spray; the moderating effect of the allele was not significant at follow up for either nicotine replacement therapies. Greater treatment response for transdermal nicotine was expected due to the effect of higher and more stable levels of nicotine replacement via transdermal delivery on beta endorphin levels and the potential for greater mu receptor occupancy in smokers with the allele. In a study using historical controls, no difference was found between nicotine replacement vs. nicotine replacement plus varenicline (Ebbert, Croghan, Sood, Schroeder, Hays, & Hurt, 2009). However, in a study of selegiline plus nicotine patch vs. placebo plus nicotine patch, the rates of continuous abstinence at one-year follow up were 25% vs. 11% (Biberman, Neumann, Katzir, & Gerber, 2003). Additional studies of these medications with smokers in alcohol recovery are clearly needed. However, as naltrexone is a first-line medication for the treatment of alcohol dependence, its potential as a treatment for tobacco dependence in combination with other smoking cessation medications is of particular interest.

Topiramate is currently being investigated as a treatment for both alcohol and tobacco dependence. Topiramate, which has several

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mechanisms of action, including enhancement of $GABA_A$ activity and antagonism of glutamate activity, may antagonize the reinforcing effects of both alcohol and nicotine by inhibiting the extracellular release of dopamine in the cortico-mesolimbic system (Johnson, 2004). It may also reduce withdrawal symptoms through its effect on calcium channels and glutamate receptors. In a placebo-controlled study of topiramate for the treatment of alcohol dependence, Johnson, Ait-Daud, Akhtar, and Javors (2005) found that participants assigned to active medication were significantly more likely to be smoking abstinent at follow up. Rates for the topiramate and placebo groups were 17% and 7%, respectively, at the 12-week follow up (odds ratio = 4.97; $p = .04$). The quit rate for the topiramate group is particularly noteworthy because participants were not expressing an intention to quit smoking at study entry.

Rimonabant, a selective type 1 cannabinoid (CB1) receptor antagonist, has also been investigated as a treatment for both alcohol and tobacco dependence (Cahill & Ussher, 2007; Litten, Fertig, Mattson, & Egli, 2005). In a meta-analysis of three placebo-controlled smoking cessation clinical trials, a pooled odds ratio of 1.61 (95% confidence interval: 1.12 – 2.30) was found in with rimonabant (Cahill & Ussher, 2007). However, concerns about psychiatric side effects, including depression, anxiety and suicidal behavior, have led the United States Food and Drug Administration to withhold its approval of the drug (Rumsfeld & Nallamothu, 2008; Stapleton, 2009).

Finally, a number of other medications are currently under development for smoking cessation, including nAChR partial agonists (in addition to varenicline which is already approved by the US Food and Drug Administration) and a nicotine vaccine (Cornuz et al., 2008; Rollema et al., 2007). As chronic alcohol use may alter the molecular mechanisms of nicotine reinforcement (e.g., nAChRs and downstream effects on dopamine release), which in turn, may also alter the efficacy of medications targeting these receptors, clinical trials will be necessary to evaluate the efficacy of these medications with alcoholic smokers.

5.4. Behavioral treatment

While most trials of smoking cessation treatment include standard behavioral therapy with demonstrated efficacy in unselected smokers, researchers have suggested that interventions that are tailored to "the needs of alcoholics using language and symbols compatible with alcohol treatment may enhance overall outcome" (Hurt & Patten, 2003, p. 340; Hughes, 2002). In a study of smokers with a past history of alcohol dependence (mean alcohol abstinence at time of enrollment = of 4.2 years), Martin et al. (1997) report favorable outcomes (26% smoking abstinence at one year) with an intervention that incorporated 12-step principles. To our knowledge only one study has investigated this question with smokers with a recent history of alcohol problems. Burling et al. (2001) randomly assigned alcoholic smokers in residential SUD treatment to one of three smoking cessation interventions: a multicomponent smoking treatment (MST) that focused exclusively on smoking cessation, a multicomponent treatment that used the smoking cessation experience as an opportunity for "generalization training" from cigarettes to alcohol (MST + G; i.e., participants examined the similarities between successfully quitting smoking and AOD use) or a "no treatment" control condition (see Table 2C). The smoking abstinence rates in the two treatment conditions at follow up were significantly higher than that in the control condition. Differences in abstinence rates between the two treatment conditions were not significant although they consistently favored the treatment that exclusively focused on smoking cessation. At one-month post discharge, the rates in the MST vs. MST + G conditions were 40% and 27%, respectively; at twelve-month post quit, the rates were 19% and 13%, respectively. Although the smoking abstinent rates were lower than those typically achieved in trials with unselected smokers, the rate for the MST group in this study is one of the highest obtained in a clinical trial of concurrent smoking and alcohol treatment. In addition, participants in the MST vs. MST + G condition had significantly or near significantly higher AOD abstinence rates at all follow-up assessments; rates were 77% vs. 59% at one-month follow up and 61% vs. 39% at the 12-month follow up, respectively. AOD abstinence rates in the control condition were not significantly different from the rates in the two experimental conditions.

Notably, the treatment provided in the trial by Burling et al. (2001) was especially intensive. The treatment included daily cognitive-behaviorally oriented one-to-one counseling sessions during a five-week prequit phase and the first two postquit weeks of treatment and then biweekly counseling for two weeks. Nicotine patch therapy was also used. The results, which are particularly noteworthy given the fact that many of the participants were homeless and severely alcohol/other drug dependent provide compelling evidence for the importance of highly intensive smoking cessation treatment for smokers who are also in alcohol treatment (see also Hays et al., 2001). The findings of this study, which was conducted in a residential treatment program that routinely addressed tobacco dependence, also provide strong evidence for the efficacy of smoking cessation treatment that is fully integrated into SUD programs (see below for further discussion).

As many alcoholics have a history of major depression (Regier et al., 1990) and negative mood and depression are associated with relapse to drinking (Hodgins, El-Guebaly, & Armstrong, 1995; Joseph et al., 2004), an additional issue of critical importance concerns the effect of a smoking cessation attempt and abstinence on mood among alcoholic smokers. Joseph et al. (2004) did not report data bearing on this issue. However, in a study by Prochaska and colleagues, there was no evidence that smoking cessation worsened depressive symptoms (Prochaska, Hall, Tsoh, Eisendrath, & Rossi, 2008). Indeed, depressive symptoms diminished regardless of smoking status at follow up (see also Munoz, Marin, Posner, & Perez-Stable, 1997 and Thorsteinsson et al., 2001). These findings are promising. However, as Hughes (2007) concludes in his review of whether smoking cessation increases the risk of depression, studies to date are unable to definitively answer this question because of significant methodological limitations. At the same time, the evidence reviewed suggests that close monitoring of smokers with histories of depression is warranted.

Finally, while the effect of smoking cessation on depressive symptoms in smokers with histories of depression is somewhat unclear, two small studies with smokers with past histories of alcohol problems suggest that cessation outcomes may be enhanced when smoking cessation treatment also targets depressive symptoms (Patten, Drews, Meyers, Martin, & Wolter, 2002; Patten, Martin, Myers, Calfas, & Williams, 1998). Patten, Drews, Meyers, Martin, & Wolter (2002) found that behavioral counseling plus mood management therapy vs. behavioral counseling only enhanced successful cessation among smokers with elevated current depressive symptoms and past history of alcohol dependence, although differences were significant only at short-term follow up; smokers with current depressive disorder were excluded from this study.

5.5. Integrating tobacco treatment into SUD programs: the need for organizational change

Alcohol treatment programs now recognize the importance of treating polysubstance disorders. The notable exception in many of these programs, however, continues to be the treatment of tobacco dependence. Hoffman and Slade (1993) have provided one of the most detailed discussions of the steps needed for the successful implementation of tobacco dependence treatment in addiction treatment settings. However, even after 15 years since their seminal work, tobacco dependence treatment is still not integrated into the majority of these treatment programs.

Smoking is often overlooked in these programs due to a variety of barriers, including attitudes of treatment staff (e.g., residents should

avoid major life changes including smoking cessation during their first year of recovery, that stopping smoking may jeopardize drug/alcohol recovery), lack of knowledge about the treatment of nicotine dependence, and a treatment culture amenable to smoking (e.g. "smoke-breaks" structured into the treatment day). Nicotine dependence may also be viewed as a low priority, when compared to more immediate harms of alcohol and illegal drug use, and drug treatment staff may believe their patients are not interested in quitting smoking (see reviews by Hall & Prochaska, 2009; Ziedonis et al., 2006 and Guydish, Passalacqua, Tajima, & Manser, 2007). However, as already discussed, most alcoholics in treatment are concerned about their smoking (e.g., Rohsenow et al., 2005) and the preponderance of evidence indicates that trying to quit during SUD treatment does not interfere with sobriety and, in fact, appears to be associated with better AOD outcomes (Prochaska et al., 2004). In addition, the clinical trial that produced the highest smoking abstinence rate to date was also fully integrated into the SUD treatment program (Burling et al., 2001).

Clearly, there exist many barriers to simultaneous treatment of tobacco dependence and other SUDs. Some are present at the patient and staff levels and others at the organizational level (Asher et al. 2003; Bobo & Davis 1993; Hurt, Croghan, Offord, Eberman, & Morse, 1995; Williams et al. 2005; Ziedonis et al., 2007; Ziedonis et al., 2006). Short staff trainings to address barriers to treating nicotine dependence have had limited effect. Bobo, Anderson, and Bowman (1997) found that a half-day skills-building workshop had no effect on the nicotine-related counseling practices of outpatient staff. In a cross-sectional design, three clinics were assigned to receive the workshop and three clinics were assigned to a non-intervention control condition. In the intervention clinics, clients who received treatment after the workshop took place were no more likely to be counseled for nicotine dependence than clients who received treatment before the workshop took place. The authors conclude: "If the majority of… practitioners in a facility are in the precontemplation stage, more intensive multi-faceted efforts… may be needed to move *staff* through contemplation and into action" (Bobo et al, 1997 , p.28).

Ziedonis and colleagues developed a more intensive, manual-based approach, called "Addressing Tobacco through Organizational Change" (ATTOC), designed to facilitate and support the full integration of tobacco treatment into SUD programs (Guydish et al., 2009; Williams et al., 2005; Ziedonis et al., 2006; see Table 2D). The approach expands on the seminal work of John Slade and draws on models of organizational change which have identified critical factors influencing adoption of innovations in SUD treatment, particularly the importance of recognizing organizational resistance, the specific forms it can take, and the ability to effectively address it (Backer, 1995; Hoffman & Slade, 1993; Rogers et al., 1995). Key elements of the ATTOC model include developing strong support from key administrators and creating a leadership group empowered by administration and comprised of members who will champion the process of integration. A key task of the leadership group is to write a strategic plan which clearly spells out the implementation process, including addressing sources of resistance, and the methods for monitoring this process. Typically, this plan needs to be revised as the process of implementation unfolds.

The strategic plan is guided by clear and measurable goals at multiple levels of the organization. For example, at the patient level, the ATTOC intervention includes integrating focused interventions both for smokers who are highly motivated to quit and for those who express little or no motivation. At the staff level, the model includes tobacco dependence treatment training and supervision and, very importantly, both the expectation that staff is nonsmoking and assistance for smokers to achieve this goal. Staff is trained in the assessment of tobacco dependence and the conduct of brief motivational interventions. They are also trained to lead "wellness and recovery" groups that focus on health promotion in recovery, "preparation" groups that build on the work of the motivational interventions, and smoking cessation groups for smokers ready to quit. Nicotine Anonymous groups are also introduced and in programs that follow a 12-step model are particularly important to the goal of integrating treatment for tobacco dependence. More generally, staff is taught skills for addressing tobacco dependence in all aspects of the SUD treatment program and is expected to document these interventions in the medical record.

Ultimately, the model is designed to help organizations create a tobacco-free environment in which state-of-the-art treatment is provided. However, the model emphasizes the importance of *gradually* integrating tobacco treatment and uses "transitional" goals to promote incremental change. Changes that are likely to encounter the least resistance (e.g., identifying smokers in the clinical chart, relabeling "smoke breaks" to just "breaks") are implemented first; changes that are likely to encounter greater resistance (e.g., the implementation of both an indoor and outdoor smoking ban) are implemented at a later time. SUD programs will vary considerably in the time needed to achieve integrated treatment for tobacco dependence. However, the overarching goal of the ATTOC model is to assist these programs in creating a *self-sustaining treatment culture* where tobacco dependence is treated like any other drug dependence.

Research evaluating the ATTOC model, including reports on model programs and demonstration projects, support its success. Sharp, Schwartz, Nightingale, and Novak (2003) reported on three programs that successfully incorporated nicotine dependence treatment into clinical practice. All of the programs followed the ATTOC model, and all instituted nicotine dependence treatment and a "zero-tolerance" tobacco-free policy. Sharp et al. (2003) contrasts this finding with Rustin (1998) who reported that programs not following the ATTOC model failed in attempts to integrate nicotine dependence treatment. The most rigorous implementation evaluation of the model to date is currently being undertaken. Preliminary analyses indicate that the intervention achieved many of its objectives (Guydish et al., 2009).

6. Directions for future research

The Public Health Service Practice Guidelines for Smoking Cessation (Fiore et al., 2008), the Practice Guidelines of the American Psychiatric Association (2006) and a National Institutes of Health State-of-Science Conference Statement on Tobacco Use (2006) recommend and encourage SUD programs to address tobacco dependence with their clients. If this policy-level recommendation is to become a reality, strong collaborations will need to be developed between SUD program staff and researchers involved in translational research. The model developed by Ziedonis and colleagues for integrating tobacco services into SUD programs provides a science-based vehicle for forging these collaborations and the limited research to date is promising (Foulds et al., 2006; Ziedonis, 2004).

Continued research is clearly needed to investigate the efficacy of integrated models on tobacco and other drug use outcomes. As noted above, findings from Burling et al. (2001) suggest that higher smoking abstinence rates are achieved when smoking cessation treatment is provided in an SUD program that has integrated this service. However, a clinical trial is needed to determine whether a sustained, higher-intensity intervention in the context of integrated treatment produces significantly better outcomes (e.g., more people attempting to quit smoking concurrently with alcohol treatment, higher smoking abstinence rates) than the unintegrated, lower-intensity treatment that have often characterized clinical trials to date with this population.

Research should also investigate the efficacy of a chronic care model of treatment in this population. According to the chronic disease model, for many smokers, and especially highly dependent smokers, tobacco dependence is a chronic medical condition that is best treated in the same manner as other long-term, chronically relapsing conditions such as alcohol dependence, depression and diabetes (Steinberg, Schmelzer, Richardson, & Foulds, 2008). These

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smokers should be offered long-term treatment, including extended use of pharmacotherapy, rather than episodic treatment. Hall, Humfleet, Reus, Munoz, and Cullen (2004) provided evidence that extended treatments that combine medication and psychological interventions can produce abstinence rates that are substantially higher than those in the literature. In their study, one-year abstinence rates for participants who were assigned to extended treatment (which consisted of 52 weeks of medication use and 14 concurrent counseling sessions) vs. eight weeks of treatment were 50% and 30%, respectively (see also Hays et al., 2001). Support for long-term care models of treatment has also been found for other addictive disorders (McKay, 2005) and, notably, are consistent with the twelve-step approach to the treatment of substance use disorders. We are currently recruiting community-dwelling smokers for a study designed to replicate and extend the findings by Hall and colleagues. A similar study with smokers with recent alcohol dependence is also warranted.

Investigations of step-care treatments with this population are also needed. In step-care treatment, interventions are adjusted according to treatment response (McKay, 2005). Most critically for smokers trying to quit, adjustments are considered to prevent an impending lapse or immediately following a lapse to prevent a relapse. In either case, medication dosage may be increased or a different medication added; frequency of counseling may also be adjusted. We could find no studies of the efficacy of lapse prevention and only two studies of step-care interventions for relapse prevention with smokers (Juliano, Houtsmuller, & Stitzer, 2006; Smith, Meyers, & Miller 2001). While these studies found no effects for step-care treatment, they had significant limitations, including a step-care intervention that was not lapse responsive (it was introduced to lapsers 14 days after their quit day regardless of when they lapsed) (Smith et al., 2001) and a step-care intervention (i.e., rapid smoking) that was not well tolerated (Juliano et al., 2006). Investigations of step-care approaches for lapse and relapse prevention for smoking and other addictive disorders are in their infancy. Research is clearly needed to investigate their efficacy for enhancing abstinence, including tobacco abstinence for smokers with alcohol histories.

Another important direction for future research is to identify individual difference variables that either increase or decrease the risk of relapse to alcohol with concurrent smoking and alcohol treatment. For example, it would be useful to identify individuals who are more vulnerable to experiencing a breakdown in self-control strength following a smoking cessation attempt (see Muraven & Baumeister, 2000). An assessment of self-control strength conducted prior to initiation of smoking cessation treatment could be used to predict ability to maintain alcohol and other drug abstinence during a cessation attempt. This information could also be used to help prepare smokers who may be at risk for relapse to alcohol use following a cessation attempt.

At the same time, future research should identify individual difference factors that may decrease the risk of relapse to alcohol with concurrent smoking and alcohol treatment. For example, there may be individuals for who continued smoking during sobriety presents an important cue-based risk factor for a return to drinking, i.e., individuals with a high degree of cross-cue reactivity (Drobes, 2002). Indirect evidence for this is suggested by studies investigating endophenotypes that mediate the relationship between a genotype and phenotype. For example, Hutchinson and colleagues have reported that a polymorphism in the D_4 dopamine receptor, which has also been implicated in the development of incentive salience, is associated with individual differences in craving in cue reactivity studies with both smoking and alcohol cues (Hutchinson, LaChance, Niaura, Bryan, & Smolen, 2002; Hutchinson, McGeary, Smolen, Bran, & Swift, 2002; see also Robinson & Berridge, 2000). These findings suggest that some alcoholic smokers who continue smoking during alcohol abstinence may be especially vulnerable to a return to drinking.

7. Conclusion

As a result of a strong and sustained commitment on the part of the National Institutes of Health over the past twenty-plus years to fund projects across a broad range of disciplines, we have made significant progress both in understanding the causes of tobacco and alcohol comorbidity, its effects and its treatment. From a clinical perspective, we have learned that many smokers in AOD treatment are concerned about their smoking and many have attempted to quit in the year prior to admission. We have also learned that smoking cessation is unlikely to threaten alcohol or other drug abstinence and, in fact, is associated with somewhat enhanced AOD outcome. At the same time, we have learned that it is particularly difficult for these smokers to maintain tobacco abstinence following a quit attempt. Standard treatment is better than no treatment; however, given the low quit rates produced by standard treatments in this population, trials are needed to investigate the efficacy of sustained and intensive treatments. In addition, as new medications are developed, investigations are needed to determine their efficacy in smokers with recent histories of alcohol dependence. Finally, advances in our understanding of the genetic and neurobiology vulnerabilities to tobacco dependence will lead to the development of more efficacious medications for this population.

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