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Publication Date

2018-09-01

DOI

10.1016/j.addbeh.2018.05.004

Peer reviewed

HHS Public Access

Author manuscript

Addict Behav. Author manuscript; available in PMC 2019 September 01.

Published in final edited form as:

Addict Behav. 2018 September ; 84: 263–270. doi:10.1016/j.addbeh.2018.05.004.

Predictors of reduced smoking quantity among recovering alcohol dependent men in a smoking cessation trial

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Abstract

Introduction: Adults with alcohol dependence (AD) have exceptionally high smoking rates and poor smoking cessation outcomes. Discovery of factors that predict reduced smoking among AD smokers may help improve treatment. This study examined baseline predictors of smoking quantity among AD smokers in a pharmacotherapy trial for smoking cessation.

Methods: The sample includes male, AD smokers ($N = 129$) with 1–32 months of alcohol abstinence who participated in a 12-week trial of medication (topiramate vs. placebo) and adjunct counseling with 6 months of follow-up. Baseline measures of nicotine dependence, AD severity, psychopathology, motivation to quit smoking, and smoking-related cognitions were used to predict smoking quantity (cigarettes per day) at post-treatment and follow-up.

Results: Overall, the sample had statistically significant reductions in smoking quantity. Greater nicotine dependence (Incidence rate ratios (*IRRs*) = 0.82–0.90), motivation to quit (*IRRs* = 0.65–0.85), and intrinsic reasons for quitting (*IRRs* = 0.96–0.98) predicted fewer cigarettes/day. Conversely, greater lifetime AD severity (*IRR* = 1.02), depression severity (*IRRs* = 1.05–1.07), impulsivity (*IRRs* = 1.01–1.03), weight-control expectancies (*IRRs* = 1.10–1.15), and childhood sexual abuse (*IRRs* = 1.03–1.07) predicted more cigarettes/day.

Conclusions: Smokers with AD can achieve large reductions in smoking quantity during treatment, and factors that predict smoking outcomes in the general population also predict greater

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Contributors

Matthew Worley conceptualized the analyses, conducted the analyses, wrote the Results, and edited the full manuscript. Melodie Isgro conceptualized the hypotheses, wrote portions of the manuscript, and reviewed the manuscript. Jaimee Heffner oversaw data collection and reviewed the manuscript. Soo Lee wrote portions of the manuscript. Belinda Daniel assisted with data collection and contributed to portions of the manuscript. Robert M. Anthenelli obtained funding for the study, oversaw data collection, and edited the manuscript. All authors have approved the final version of the manuscript.

Conflict of interest

Dr. Anthenelli has provided consulting and/or advisory board services to Pfizer, Inc., Arena Pharmaceuticals, and Cerecor. The other authors have no disclosures to report.

smoking reductions in AD smokers. Treatment providers can use severity of nicotine dependence and AD, motivation to quit, smoking-related cognitions, and severity of depression to guide treatment and improve outcomes among AD smokers.

Keywords

Smoking cessation; Nicotine; Alcohol dependence; Tobacco

1. Introduction

Cigarette smoking remains a significant and prevalent public health problem contributing to cancer, cardiovascular and respiratory disease, and premature mortality (Services, 2014). Rates of smoking cessation are low (Stead et al., 2012) and smokers with a substance use disorder have even worse rates of cessation (Prochaska, Delucchi, & Hall, 2004). Many alcohol-dependent (AD) smokers continue to smoke during abstinence from drinking, which contributes to chronic, costly health problems (John & Hanke, 2002). Smoking cessation treatments for this population need to be improved. Greater understanding of the factors that contribute to reduced smoking in this population may help advance treatment.

Severity of either nicotine dependence or AD may predict smoking outcomes in this special population. Among smokers without AD, those with less severe nicotine dependence typically have greater quit rates (Caponnetto & Polosa, 2008; Carlson, Taenzer, Koopmans, & Bultz, 2000; Dale et al., 2001; Murray et al., 2000). Severity of AD may also predict smoking outcomes, as lower levels of drinking and alcohol-related problems have predicted better smoking cessation in smokers without AD (Augustson et al., 2008; Hughes & Kalman, 2006; Hyland et al., 2004; Osler, Prescott, Godtfredsen, Hein, & Schnohr, 1999). Among smokers with alcohol or substance use disorders, severity of nicotine dependence has predicted smoking cessation in some studies (Rohsenow et al., 2015) but not others (Rohsenow, Martin, Tidey, Monti, & Colby, 2013), as discussed in a prior review (Heffner, Barrett, Anthenelli, 2007). Prior research has not examined the role of AD severity in predicting reduced smoking within the population of AD smokers.

Other factors that may predict smoking outcomes include measures of co-occurring psychopathology, which tends to be more severe in AD smokers as compared to others (Heffner, Mingione, Blom, & Anthenelli, 2011). Among non-AD smokers, greater severity of depression or anxiety has predicted poorer smoking cessation outcomes (Cinciripini et al., 1995; Weinberger, Mazure, Morlett, & McKee, 2013). Traumatic life experiences, which are prevalent among substance users (Heffernan et al., 2000), also relate to worse smoking outcomes, with sexual abuse associated with propensity for nicotine dependence and lower quit rates (King, Guilbert, Ward, Arwidson, & Noubary, 2006; Smith et al., 2015; Smith, Homish, Saddleson, Kozlowski, & Giovino, 2013). Greater impulsivity (a tendency to act quickly without careful thought) has been linked to both nicotine and alcohol dependence (Chase & Hogarth, 2011; Rubio et al., 2008), and greater impulsivity has predicted poorer smoking treatment outcomes (Loree, Lundahl, & Ledgerwood, 2015). These factors related to impaired psychological functioning are potentially important predictors of smoking outcomes in smokers with AD, as they typically have greater depression, anxiety, and

impulsivity than other smokers (Lawrence, Mitrou, & Zubrick, 2009; VanderVeen, Cohen, & Watson, 2013).

Smoking-related motivations and cognitions have also predicted smoking treatment outcomes in non-AD smokers and could predict reduced smoking in AD smokers. Baseline motivation to quit often predicts smoking cessation (Boardman, Catley, Mayo, & Ahluwalia, 2005; Piñeiro et al., 2016). Higher levels of relative intrinsic (vs. extrinsic) reasons for quitting have also predicted greater rates of smoking ces-sation (Curry, Grothaus, & McBride, 1997). Other predictive cognitive factors include expectancies about the consequences of smoking or quitting smoking (Copeland, Brandon, & Quinn, 1995). Beliefs about use of cigarettes to regulate mood and weight have been associated with smoking intensity or treatment outcomes (Hruska et al., 2014; Weinberger, McKee, & George, 2010). Lower motivation to quit, fewer internal reasons for quitting, and stronger beliefs about the positive aspects of smoking may very well predict poorer smoking outcomes in AD smokers, although some prior studies have not found these effects (Rohsenow, Tidey, Kahler, et al., 2015).

The goal of this study was to identify baseline characteristics that predict smoking outcomes in AD smokers with recent alcohol abstinence who received treatment for smoking cessation. Weekly smoking outcomes were assessed at treatment endpoint (weeks 9–12), middle follow-up (weeks 21–24), and late follow-up (weeks 33–36). To date, no single study of AD smokers has examined a broad range of relevant predictors, including nicotine and alcohol dependence, co-oc-curring psychopathology, and smoking-related motivations and cognitions. Consistent with the typical low rates of cessation in this population (Prochaska et al., 2004), only 9.3% of the sample in this trial quit smoking throughout the final month of treatment (Anthenelli et al., 2017). Therefore, the current study examined reductions in smoking quantity. Reduced smoking is associated with future cessation (Hyland et al., 2005; Klemperer & Hughes, 2015) and although the evidence is mixed, reductions in smoking are also associated with health benefits in some studies (Begh, Lindson-Hawley, & Aveyard, 2015; Gerber, Myers, Goldbourt, 2012). In this population, reduced smoking quantity represents a meaningful intermediate target, with findings having the potential to improve treatment delivery. We hypothesized that lower severity of nicotine dependence, AD, depression, anxiety, impulsivity, smoking reinforcement expectancies, and childhood sexual abuse would predict greater reductions in weekly smoking quantity, while greater motivation to quit smoking would predict greater reductions in smoking at the end of treatment and follow-ups.

2. Materials and methods

2.1. Study design

This secondary analysis study examined data from a randomized, controlled trial of topiramate (100 mg twice daily) vs. placebo for smoking cessation and alcohol relapse prevention, with adjunct brief counseling in both conditions. The study included nicotine-dependent male smokers with alcohol dependence who were currently abstinent from drinking. The decision to include only male smokers was based on our earlier study that found gender-specific effects of topiramate on smoking cessation (Anthenelli, Blom,

McElroy, & Keck Jr., 2008). Study duration was 36 weeks, with 12 weeks of active treatment and 24 weeks of follow-up. With details on primary trial outcomes and study design reported previously in greater detail (Anthenelli et al., 2017), study methods are summarized briefly here.

2.2. Subjects

Study participants were 18–70 years of age, male, had DSM IV-TR current nicotine dependence, smoked an average of at least 10 cigarettes per day in the previous two months, had 1–36 months of abstinence from alcohol, and reported at least moderate motivation to quit smoking (≥6 on a 1–10 scale). Exclusion criteria included recent (past-month) receipt of smoking cessation treatment, alcohol pharmacotherapies, or any investigational drug, as well as a current seizure disorder or history of severe alcohol withdrawal, elevated suicidal risk, history of a psychotic disorder, known hypersensitivity to topiramate, or any clinically significant laboratory abnormalities or medical problems. From the full randomized sample (N = 133) the present study examined a modified intent-to-treat sample (N = 129) restricted to participants from both treatment conditions who took at least one dose of study medication. Due to some missing data on baseline measures, the available sample size ranged from 107 to 126 across the specific analyses. Veterans comprised the majority of the sample (62%), with a substantial proportion of non-white participants (43.7%). Demographic and clinical characteristics of the sample are described in Table 1.

2.3. Measures

2.3.1. Cigarette smoking—The Timeline Follow Back (Gariti, Alterman, Ehrman, & Pettinati, 1998) completed at baseline (past 90 days), weekly during treatment, and monthly during follow-up assessed daily smoking quantity (cigarettes/day), which did not differ significantly between treatment conditions (Isgro et al., 2017). For the smoking outcome variables, daily data were used to obtain a measure average cigarettes/day in the past week for three key periods of study involvement: treatment endpoint (Weeks 9–12), middle follow-up (Weeks 21–24) and late follow-up (Weeks 33–36). Baseline smoking (past-month cigarettes/day) was also used as a covariate in analyses.

2.3.2. Demographics—Demographic characteristics were derived from the Semi-Structured Assessment for the Genetics of Alcoholism (Bucholz et al., 1994) com-pleted at screening.

2.3.3. Substance use severity—Baseline measures of substance use severity included the Fagerström Test for Nicotine Dependence (FTND) for nicotine dependence severity (Heatherton, Kozlowski, Frecker, & Fagerstrom, 1991) and the Michigan Alcoholism Screening Test (MAST) for severity of lifetime alcohol-related problems (Selzer, 1971).

2.3.4. Mental health/psychopathology—Baseline measures in the domain of mental health/psychopathology included the Montgomery-Asberg Depression Rating Scale (MADRS) for severity of depression (Montgomery & Asberg, 1979), the Structured Interview Guide for the Hamilton-Anxiety (SIGH-A) for severity of anxiety (Hamilton, 1959), the Barratt Impulsiveness Scale (BIS) for im-pulsivity (Patton, Stanford, & Barratt,

1995) including the total score and three subscale scores (Attentional, Non-Planning, and Motor Control), and the Childhood Trauma Questionnaire (CTQ) to assess severity of childhood sexual abuse (Bernstein et al., 1994).

2.3.5. Smoking-related motivation and cognitions—Baseline measures in this domain included the Reasons for Quitting scale (RFQ) to assess relative intrinsic reasons for quitting smoking, calculated by the Intrinsic Score – the Extrinsic score (Curry, Wagner, & Grothaus, 1990), and the Smoking Consequences Questionnaire – Adult (SCQ-A) to assess smoking beliefs and consequences (Copeland et al., 1995). The SCQ-A provided four subscale scores including Negative Consequences, Positive Reinforcement, Negative Reinforcement, and Weight Control. A single self-reported item ranging from 1 (“not at all motivated”) to 10 (“highly motivated”) assessed motivation to quit smoking (“On the following 10-point scale, check the corresponding number that best describes your current motivation to quit smoking”). This item was used in our prior work on topiramate (Anthenelli et al., 2008) and is similar to other simple rating scales that had predictive validity in prior studies of smoking (Boudreaux et al., 2012)

2.4. Statistical analyses

Preliminary analyses examined descriptive statistics of predictor variables (see Table 1) and smoking outcomes, along with the overall change in smoking from baseline to each phase. Repeated measures of smoking outcomes were analyzed in multilevel models with random person-level intercepts and the negative-binomial function to account for the skewed distribution of smoking variables. Maximum-likelihood estimation was used to include all available smoking outcome data points under the missing-at-random assumption (Schafer & Graham, 2002), which was supported by preliminary analyses revealing no significant differences between participants with any missing and full data. All predictor variables were mean-centered prior to analyses.

Separate statistical models examined each predictor, with the model predicting smoking during three separate phases: treatment end (Weeks 9–12), middle follow-up (mid-FUP, Weeks 21–24) and late follow-up (late-FUP, Weeks 33–36). The goal of this modeling strategy was to limit the number of separate statistical models while still examining predictor effects separately for these distinct phases of the study, because predictors of sustained reductions in smoking may differ from those that predict reduced smoking during treatment. To separately predict smoking quantity at each phase while controlling for baseline smoking, each model used three categorical “phase” indicators. Independent variables in each model included baseline smoking, the predictor, three phase indicator main effects, the baseline smoking*-phase indicator interactions, and the predictor*phase indicator interactions. The primary effect of interest is the incidence-rate ratio (*IRR*) for the predictor*phase indicator interaction, which estimates the proportional reduction in smoking quantity from baseline to each phase associated with each predictor. Effects of predictors found to be statistically-significant across multiple phases ($p < .05$) were inspected visually by plotting estimated smoking levels at each phase for participants at mean, high (90th percentile), and low (10th percentile) levels of the predictor. Because prior studies found topiramate did not impact smoking cessation or smoking reduction (Anthenelli et al., 2017;

Isgro et al., 2017), treatment condition was not included as a covariate. All analyses were conducted in Stata 15.0 (StataCorp, 2013).

3. Results

3.1. Overall changes in smoking quantity

In prior research, we reported that treatment condition did not impact smoking quantity (Isgro et al., 2017). From baseline to treatment end (weeks 9–12), average smoking across the sample reduced significantly (*IRR* = 0.20, $p < .001$), from a mean of 19.2 cigarettes/day (*SD* = 7.5) to 6.7 cigarettes/day (*SD* = 6.8). Smoking quantity did increase slightly from treatment end to mid-FUP (*M* = 8.4, *SD* = 8.6) and late-FUP (*M* = 8.6, *SD* = 7.9). Despite these slight increases, smoking was still significantly lower at mid-FUP, *IRR* = 0.22, $p < .001$, and late-FUP, *IRR* = 0.23, $p < .001$, as compared to baseline.

3.2. Effects of baseline smoking

As shown by significant baseline smoking*phase interactions for treatment endpoint (*IRR* = 0.96, $p < .001$) and 6-month follow-up (late-FUP *IRR* = 0.97, $p < .05$), smokers with greater baseline smoking had significantly greater reductions in smoking, reflecting their greater room for reduction from baseline. Of note, baseline smoking was significantly and positively correlated with baseline FTND scores ($r = 0.52$, $p < .001$). Results revealed no other significant correlations between baseline smoking and the predictors.

3.3. Predictors of smoking quantity

Among predictors in the domain of substance-related severity, nicotine dependence severity and lifetime alcohol-related problems predicted smoking quantity at all three outcome phases (see Table 2). Greater baseline FTND scores predicted lower cigarettes/day (see Fig. 1, panel A), while greater baseline MAST scores predicted greater cigarettes/day (see Fig. 1, panel B). Therefore, while greater baseline nicotine dependence severity predicted lower smoking quantity, greater lifetime alcohol-related problems predicted greater smoking quantity.

Results revealed several statistically significant predictors in the domain of mental health and psychopathology (see Table 2). Severity of depressive and anxiety symptoms both predicted smoking quantity, with greater MADRS scores predicting greater cigarettes/day at all outcome phases (see Fig. 1, panel C). Greater SIGH-A scores also pre-dicted greater cigarettes/day, but only at treatment end. Among the BIS impulsivity total score and subscales, motor impulsivity most consistently predicted smoking quantity, with greater BIS-Motor scores predicting greater cigarettes/day at all three outcome phases (see Fig. 1, panel D). Other BIS subscales predicted smoking quantity less consistently, with greater BIS-Attention and BIS-Non-planning predicting greater cigarettes/day at treatment end, and greater BIS-Total predicting greater cigarettes/day at treatment end and mid-FUP (see Table 2). Childhood sexual trauma severity predicted smoking quantity during follow-up, with greater baseline CTQ-Sexual Abuse scores predicting greater cigarettes/day at mid-FUP and late-FUP (see Fig. 2, panel D). Overall, results demonstrated that greater severity in depression, anxiety, motor impulsivity, and childhood sexual abuse all predicted greater

smoking quantity, with depression and motor impulsivity predicting smoking quantity at all three phases examined.

Several variables related to smoking motivation and cognitions predicted smoking quantity (see Table 2). Greater motivation to quit predicted lower cigarettes/day at all three outcome phases (see Fig. 2, panel A). The RFQ Intrinsic - Extrinsic difference score, an index of intrinsic vs. extrinsic reasons for quitting, predicted smoking quantity at treatment end and mid-FUP, with greater relative intrinsic reasons predicting lower cigarettes/day at both phases (see Fig. 2, panel B). Smoking quantity was also predicted by one's strength of beliefs in smoking for appetite and weight control, as greater baseline SCQ-Adult Weight Control subscale scores predicted greater cigarettes/day at all three phases (see Fig. 2, panel C). None of the other SCQ subscales (Negative Consequences, Positive Reinforcement, Negative Reinforcement) significantly predicted smoking quantity at any phase. Overall, these results demonstrated that greater motivation and relative intrinsic reasons to quit and lower beliefs in smoking for weight control predicted lower smoking quantity across all phases examined.

4. Conclusions

This study examined baseline predictors of reduced smoking quantity in recovering, AD smokers during and after smoking cessation treatment in a pharmacotherapy trial with adjunct counseling. Smokers with AD are an important subgroup as they have especially low rates of smoking cessation, often continue smoking after remission of AD, and have significant and costly health problems that are uniquely attributable to smoking (John & Hanke, 2002). We focused on predictors of reduced smoking quantity. Even AD smokers who struggle to achieve cessation may be amenable to reducing smoking, and smokers who reduce are more likely to quit in the future than smokers who do not (Begh et al., 2015). By identifying measurable baseline predictors across multiple domains, our study yielded unique findings that could potentially improve treatment of this high-risk population.

In this study AD smokers who had more severe baseline nicotine dependence had greater reductions in smoking. This finding seems counterintuitive since more severe dependence typically predicts worse smoking outcomes (Breslau & Johnson, 2000; Okoli & Khara, 2014), but studies typically examine complete cessation, and some studies have found better outcomes for smokers with more severe initial dependence. For example, African-American smokers who quit smoking had more severe nicotine dependence at baseline than those who only reduced their smoking (Berg et al., 2010). Our study did not group out-comes according to quit status, but directly predicted the quantity of reduction in cigarettes/day, while also controlling for baseline smoking quantity. Therefore, our findings suggest that for a given level of baseline smoking, greater severity of dependence corresponds to greater reductions in smoking during treatment. Dependence severity captures many aspects of smoking behavior including compulsive use and persistence of smoking-related problems. Participants with more severe baseline nicotine dependence may enter treatment with greater awareness of smoking-related problems that contributes to behavioral change.

In this sample of AD smokers, greater severity of lifetime alcohol-related problems predicted greater smoking quantity (i.e., less reduction) across all time points examined. Although the mechanism behind this finding is not clear, research has shown that AD smokers (compared to non-AD smokers) have greater craving to smoke for negative affect relief (Heffner et al., 2011). In our sample where all smokers had lifetime AD, smokers with more severe AD may have experienced stronger such cravings as they attempted to change their smoking. The AD smokers with more severe AD may also carry a general propensity to persist in substance use despite substance-related problems. Behavioral genetics research supports the existence of such general liability factors, such as behavioral disinhibition, that explain increased general risk across addictive behaviors (Derringer et al., 2015). In our study greater impulsivity (particularly motor impulsivity) predicted greater smoking quantity, consistent with prior studies on impulsivity and smoking treatment outcomes in the general smoking population (Doran, Spring, McChargue, Pergadia, & Richmond, 2004). Future research might consider testing interventions that address specific coping and/or impulse-control deficits of AD smokers to potentially improve their smoking treatment outcomes.

We also found that AD smokers with more severe depression or anxiety at baseline smoked in greater quantities at treatment end, with more severe baseline depression also predicting greater smoking throughout follow-up. Although depression and anxiety disorders are associated with propensity for nicotine dependence (Lawrence et al., 2009), the relationship between depression/anxiety severity and smoking treatment outcome is less clear (Berlin & Covey, 2006). One recent review specifically recommends that studies seek to clarify the role of depression severity in smoking treatment outcomes (Weinberger et al., 2013), and our study showed that AD smokers with more severe baseline depression had less reduction in smoking during and after treatment. We did not specifically target depression in treatment or examine changes in negative affect, but research has shown that depression interventions may help improve treatment outcomes for AD smokers making a quit attempt (Patten, Drews, Myers, Martin, & Wolter, 2002). In our sample AD smokers with more severe childhood sexual trauma smoked at greater levels after treatment. Such childhood maltreatment experiences are especially prevalent in smokers with AD or other substance use disorders (Kendler et al., 2000; Wu, Schairer, Dellor, & Grella, 2010). Smoking may serve to modulate trauma-related symptoms (Fu et al., 2007), and it is possible that in our sample of AD smokers, those with more severe prior sexual abuse may have failed to develop alternative methods of coping to support sustained reductions in smoking.

A number of factors related to smoking motivation and cognitions also predicted change in smoking quantity. Greater baseline motivation to quit smoking predicted greater reduction in smoking throughout the study, which is consistent with prior studies demonstrating a relation-ship between pre-treatment motivation and smoking treatment out-comes (Boardman et al., 2005; Piñeiro et al., 2016). Our results add to the literature in finding that AD smokers with greater motivation to quit were more likely to reduce their smoking amount, even if unable to achieve cessation. To address lower levels of motivation, AD smokers might benefit from motivational enhancement interventions, which have previously improved smoking cessation outcomes under certain conditions in some studies (Piñeiro et al., 2016; Rohsenow et al., 2015) but not in others (Rohsenow et al., 2014). Greater intrinsic

reasons for quitting (relative to extrinsic reasons) also predicted greater reduction in smoking at all phases examined, similar to prior research in reasons for quitting (S. J. Curry et al., 1997). Endorsement of weight-control expectancies also consistently predicted smoking quantity. This is consistent with prior findings that greater smoking expectancies were correlated with perceived barriers to quitting (Asher et al., 2003) and lower motivation to quit (Hendricks, Peters, Thorne, Delucchi, & Hall, 2014) among substance-using smokers. This finding on weight-control expectancies was also a notable result in our all-male sample, given that studies on smoking-related weight concerns and applicable interventions have often focused on female smokers (Fulkerson & French, 2003; Sallit, Ciccazzo, & Dixon, 2009). Our study suggests male smokers with greater weight-control expectancies also have greater difficulty in reducing their smoking. Treatment for AD smokers might therefore be improved by including strategies for maintaining weight control while quitting or reducing smoking.

This study had several limitations that may impact interpretation of our findings. This was a secondary analysis of a clinical trial, and we examined hypotheses that were not central to the study design. While the data analyses were conducted selectively and findings were largely consistent with prior literature and theory, these findings should be interpreted with appropriate caution. Due to the exclusion of light smokers and those with low motivation to quit, the results have limited generalizability to AD smokers more broadly. Although the sample contained considerable ethnic diversity which strengthens generalizability, the exclusion of female participants and the mostly veteran sample weakens the generalizability somewhat. Because most predictors were assessed via self-report or interview that are feasible in clinical settings, self-report and/or recall biases likely contributed to measurement error in this study. Finally, for theoretical and practical reasons (i.e., statistical power), this secondary study did not examine smoking cessation which is still considered the “gold-standard” for smoking treatment outcome. Despite these limitations, this study is an important step towards better understanding reduced smoking in a high-risk and understudied population, recovering AD smokers.

In summary, the current study identified characteristics of AD smokers in early recovery from alcohol that predicted greater reductions in smoking quantity during a smoking cessation attempt. Our findings identified several malleable factors that interventions could target to improve treatment outcomes in AD smokers, such as enhancing motivation to quit, reducing symptoms of depression, anxiety, and impulsivity, and facilitating intrinsic reasons to quit smoking. Findings also suggest that addressing weight-control expectancies and symptoms related to childhood trauma may improve outcomes for AD smokers. Because AD smokers have very low rates of smoking cessation and especially severe health problems attributable to continued smoking, future research should build on these findings by examining specific mechanisms and interventions that produce sustained reductions and eventual smoking cessation in this population.

Acknowledgements

The authors would like to acknowledge contributions to the study made by Neal Doran, Esther Wong, Elizabeth Dinh, Jessie Tibbs, Katie Russell, and Chris Wehrle.

Role of funding source

Funding for this study was provided by Department of Veterans Affairs Merit Review#NEUA-003-08S. The writing of this manuscript was further supported by NIAAAU01-AA013641, R01-AA019720, NIDAK23DA039348, and NIDA/VA Cooperative Studies Program grant #s 1031 and 1032. None of the funding sources had any role in the study design, collection, analysis, or interpretation of data, writing the manuscript, or the decision to submit the manuscript for publication.

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HIGHLIGHTS

- Smokers with alcohol dependence have extremely low rates of smoking cessation.
- We examined baseline predictors of reductions in smoking among alcohol-dependent smokers in early sobriety from drinking.
- Less severe alcohol-related problems, depression severity, and impulsivity predicted greater reductions in smoking.
- Greater nicotine dependence, motivation to quit, and intrinsic reasons to quit predicted greater reductions in smoking.
- Predictors of reduced smoking could potentially be used to personalize treatment.

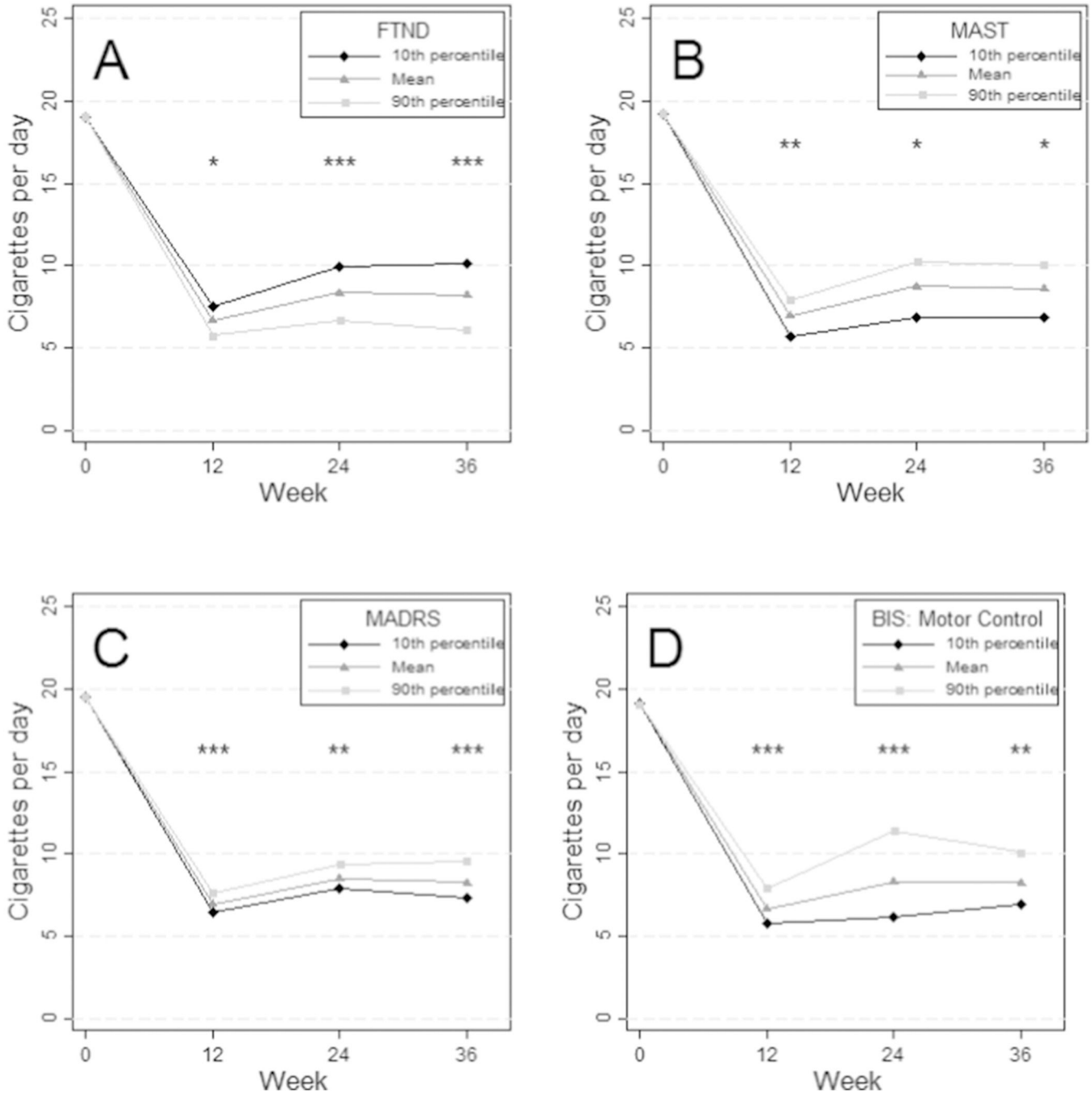

Fig. 1.

Reduction in smoking at treatment endpoint and follow-up, estimated for participants at low, mean, and high levels of baseline Fagerström Test for Nicotine Dependence (FTND), Michigan Alcoholism Screening Test (MAST), Montgomery-Asberg Depression Rating Scale (MADRS), and Barratt Impulsiveness Scale (BIS): Motor Control. Labels in-dicate statistical significance of association between baseline predictor and amount of reduction in smoking at each time point (*$p < .05$, **$p < .01$, ***$p < .001$).

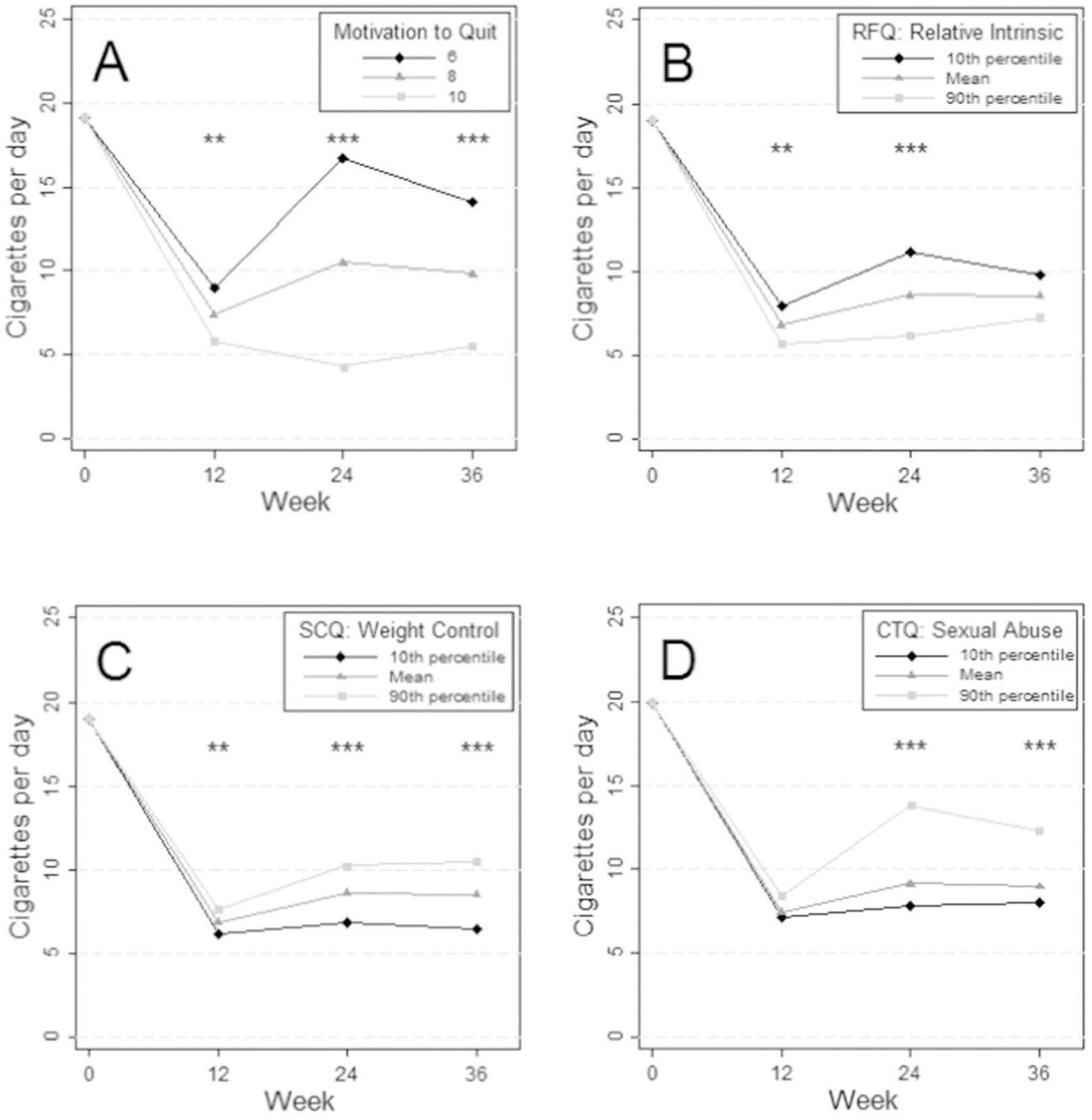

Fig. 2.

Reduction in smoking at treatment endpoint and follow-up, estimated for participants at low, mean, and high levels of baseline motivation to quit smoking, Smoking Consequences Questionnaire (SCQ): Weight Control score, Reasons for Quitting (RFQ): Relative Intrinsic score (Intrinsic-Extrinsic), and Childhood Trauma Questionnaire (CTQ)-Sexual Abuse. Labels indicate statistical significance of association between baseline predictor and amount of reduction in smoking at each time point (*$p < .05$, **$p < .01$, ***$p < .001$).

Table 1

Demographic and baseline characteristics evaluated as predictors of smoking quantity in alcohol-dependent, male smokers.

Demographics	***M* or %**	***SD* or *n***	
Age	47.7	8.9	
Years of education	12.6	1.7	
Race: % (*n*) Caucasian	56.3	72	
Baseline characteristics	*M*	*SD*	Scale Range
Nicotine dependence severity (FTND)	5.2	1.9	1–10
Lifetime alcohol-related problems (MAST)	31.8	11.0	0–53
Depression severity (MADRS)	2.2	4.0	0–20
Anxiety severity (SIGH-A)	1.6	3.0	0–21
BIS: Total	69.0	10.3	30–120
BIS: Attentional	18.0	3.6	8–32
BIS: Non-Planning	29.0	4.8	12–48
BIS: Motor Control	22.1	4.5	10–40
Motivation to quit smoking	8.7	1.2	6–10
RFQ: Intrinsic-Extrinsic	10.6	7.0	−40–40
SCQ-A: Negative Consequences	6.2	1.1	0–9
SCQ-A: Positive Reinforcement	5.4	1.6	0–9
SCQ-A: Negative Reinforcement	6.1	2.0	0–9
SCQ-A: Weight control	3.9	2.2	0–9
Childhood trauma (CTQ): Sexual abuse subscale	7.3	4.8	5–25

Note: FTND = Fagerström Test for Nicotine Dependence; SCQ-A = Smoking Consequences Questionnaire – Adult; RFQ = Reasons for Quitting; MAST = Michigan Alcoholism Screening Test; MADRS = Montgomery-Asberg Depression Rating Scale; SIGH-A = Structured Interview Guide for the Hamilton Anxiety Scale; BIS = Barratt Impulsiveness Scale; CTQ = Childhood Trauma Questionnaire.

Table 2

Statistically-significant predictors of smoking reduction during treatment endpoint and follow-up.

	Treatment Endpoint	3-month Follow-Up	6-month Follow-Up
	***IRR* [95% *CI*]**	***IRR* [95% *CI*]**	***IRR* [95% *CI*]**
Nicotine dependence severity (FTND)	0.90, [0.82,0.98]*	0.82, [0.75,0.90]***	0.84, [0.76,0.92]***
Lifetime alcohol-related problems (MAST)	1.02, [1.00,1.03]**	1.02, [1.00,1.03]*	1.02, [1.00,1.03]*
Depression severity (MADRS)	1.07, [1.03,1.11]***	1.07, [1.03,1.11]**	1.09, [1.05,1.13]***
SIGH-A	1.06*, [1.01, 1.11]	1.04, [0.98, 1.09]	1.05, [1.00, 1.11]
Impulsiveness (BIS): Total	1.03, [1.02, 1.05]***	1.02, [1.01, 1.04]*	1.01, [0.99, 1.03]
Impulsiveness (BIS): Motor	1.06, [1.02,1.09]***	1.07, [1.03,1.10]***	1.05, [1.01,1.09]**
Motivation to quit smoking	0.85, [0.75,0.95]**	0.65, [0.57,0.73]***	0.71, [0.63,0.81]***
Reasons to quit (RFQ): Intrinsic-Extrinsic	0.97, [0.95,0.99]**	0.96, [0.94,0.98]***	0.98, [0.96,1.00]
Smoking consequences (SCQ-A): Weight control	1.10, [1.03,1.18]**	1.13, [1.06,1.22]***	1.15, [1.07,1.24]***
Childhood trauma (CTQ): Sexual Abuse	1.03, [1.00,1.07]	1.07, [1.03,1.11]***	1.07, [1.03,1.11]***

* $p < .05$.

** $p < .01$.

*** $p < .001$.