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Achieving smoking abstinence is associated with decreased cocaine use in cocaine-dependent patients receiving smoking-cessation treatment

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Abstract

Background—Past research suggests that a significant relationship exists between cigarette smoking and illicit-stimulant abuse. The present study evaluated the association between achieving smoking abstinence in response to smoking-cessation treatment (SCT) and illicit-stimulant abstinence in cocaine- and/or methamphetamine-dependent participants.

Methods—Secondary analysis of a randomized, 10-week trial conducted at 12 substance use disorder (SUD) treatment programs. Two hundred and sixty seven adults, meeting DSM-IV-TR criteria for cocaine and/or methamphetamine-dependence and interested in quitting smoking were randomized to SUD treatment as usual plus SCT consisting of weekly individual smoking cessation counseling, extended-release (XL) bupropion (300 mg/day), nicotine inhaler, and contingency management for smoking abstinence. Illicit-stimulant-abstinence was measured by self-report and urine drug screens. Smoking abstinence was assessed via self-report and carbon monoxide levels.

Results—A significant effect was found for the cocaine-dependent subsample (N=147) in which participants who stopped smoking were abstinent for illicit stimulants an average of 78.2% of the post-smoking-quit weeks (weeks 4-10) relative to 63.6% in participants who continued smoking ($X^2(1)=8.55$, $p<.01$, $d=0.36$). No significant effects were found for the sample as a whole (N=249) or for the methamphetamine-dependent subsample (N=102).

Conclusions—The present results suggest that cocaine-dependent patients achieving smoking abstinence in response to SCT might evidence not only improved smoking outcomes but improved cocaine-use outcomes as well. Future research to replicate this finding appears warranted.

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Contributors

Dr. Winhusen conceptualized the research hypotheses, contributed to the analysis and interpretation of the data, and led the drafting of the manuscript. Mr. Lewis conducted analyses and critically reviewed the manuscript. Ms. Kropp and Mr. Theobald contributed to the interpretation of the findings and critically revised the manuscript for important intellectual content. All authors have approved the final manuscript.

Conflict of Interest

The authors have no potential conflicts of interest to report.

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Keywords

cocaine; smoking cessation; methamphetamine

1. INTRODUCTION

A link between cigarette smoking and cocaine abuse has been established in pre-clinical, human laboratory, and clinical studies. In mice, exposure to nicotine prior to cocaine increases behaviors associated with cocaine and results in synaptic changes in the striatum and amygdala that are not associated with nicotine or cocaine alone (Huang et al., 2013; Levine et al., 2011). Pre-treatment of rhesus monkeys with nicotine prior to cocaine enhances the preference for cocaine (Mello and Newman, 2011) while the combination of nicotine and cocaine results in greater drug administration than either substance alone (Mello et al., 2013; Mello and Newman, 2011). Human laboratory studies have found that cocaine administration increases the rate of cigarette smoking (Nemeth-Coslett et al., 1986; Roll et al., 1997) and that mecamylamine, a nicotine antagonist, reduces cue-induced cocaine craving (Reid et al., 1999) while nicotine increases it (Reid et al., 1998).

Clinically, the rates of cocaine dependence are significantly higher in cocaine users who initiated cocaine after having started smoking compared to both non-smokers and those who initiated cocaine before smoking (Levine et al., 2011). The rate of smoking in cocaine abusers is 75-80% (Budney et al., 1993; Gorelick et al., 1997; Sees and Clark, 1993) and smoking cigarettes is associated with more severe cocaine addiction, including more frequent cocaine use, a greater likelihood of injecting or smoking cocaine, and more severe employment and legal difficulties (Budney et al., 1993; Roll et al., 1997). In addition, smoking status is a significant predictor of cocaine use (Frosch et al., 2000; Patkar et al., 2003; Roll et al., 1996) and cocaine users who smoke concurrently smoke more cigarettes (Brewer et al., 2013; Roll et al., 1996) and experience greater cravings for both nicotine and cocaine (Brewer et al., 2013).

A smaller body of research also suggests that there is a connection between methamphetamine use and cigarette smoking. In rats, co-administration of nicotine and methamphetamine results in expression of different genes in the brain than those expressed when either drug is administered alone (Saint-Preux et al., 2013). It has been suggested that nicotine and methamphetamine may substitute for each other, to some extent (Gatch et al., 2008; Hiranita, et al., 2006). In nicotine-naïve rats, nicotine priming alone reinstates methamphetamine-seeking, though to a lesser degree than methamphetamine-priming and cue-induction (Hiranita et al., 2006). In contrast, Neugebauer and colleagues (2010) found that nicotine priming reinstated methamphetamine-seeking only in rats that had been previously treated with nicotine rather than saline. In humans, a laboratory study found that *d*-amphetamine dose-dependently increases cigarette smoking (Tidey et al., 2000). A secondary analysis of a clinical trial of bupropion for methamphetamine dependence revealed a positive association between number of cigarettes smoked and methamphetamine use, but only in the placebo group (Brensilver et al., 2013). The high prevalence of smoking in methamphetamine abusers, estimated to be over 87% (Grant et al., 2007; Weinberger and Sofuoglu, 2009), also suggests a link between cigarette smoking and methamphetamine use.

Given the link between illicit stimulant use and cigarette smoking, we have hypothesized that better illicit stimulant-use outcomes would be obtained for stimulant-dependent patients who achieved smoking cessation abstinence in response to smoking-cessation treatment (Winhusen et al., 2012a). The data to test this hypothesis came from a recent multi-site trial conducted by the National Institute on Drug Abuse (NIDA) National Drug Abuse Treatment

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Clinical Trials Network (CTN) which evaluated the impact of concurrent substance use disorder (SUD) and nicotine dependence treatment for cocaine and/or methamphetamine-dependent patients who were also nicotine dependent. The primary analyses from the trial revealed that there were no significant treatment effects on stimulant-use outcomes (Winhusen et al., in press). The present analyses evaluated the association between achieving smoking abstinence and illicit-stimulant abstinence in the post-smoking-quit phase of the study for participants randomized to the smoking-cessation condition. Because there are important differences between cocaine and methamphetamine (Newton et al., 2005; Winhusen et al., 2013), we evaluated the association for the entire sample as well as separately for the cocaine-dependent and methamphetamine-dependent participants.

2. METHODS

2.1 Study Design

Details of the trial are provided elsewhere (Winhusen et al., 2012a). Briefly, the study was a 10-week, intent-to-treat, 2-group randomized controlled trial with follow-up visits at 3 and 6 months post-smoking quit date conducted at 12 SUD outpatient treatment programs. Treatment programs which did not provide smoking-cessation treatment were eligible to participate. Eligible participants were randomized to treatment as usual (TAU) or TAU with smoking-cessation treatment (TAU+SCT). During the 10-week treatment phase, participants were scheduled to attend two research visits per week for efficacy and safety assessments. There were single follow-up visits at 3-months and 6-months post-quit date. Participants randomized to the TAU+SCT arm participated in the SUD treatment as typically provided by the study site and also received SCT consisting of extended-release (XL) bupropion 300 mg/day, nicotine inhaler, individual 10 minute smoking-cessation counseling weekly for 10 weeks, and prize-based contingency management for smoking abstinence (Carbon Monoxide (CO)< 4ppm) during the post-quit phase.

2.2 Participants

Recruitment was primarily from clinic patients entering substance use disorder (SUD) treatment at a participating site. Eligible participants were adults enrolled in outpatient SUD treatment, and interested in quitting smoking. The main inclusion criteria were: meeting DSM-IV-TR criteria for cocaine- and/or methamphetamine-dependence, smoking at least 7 cigarettes daily and a CO level ≥ 8 ppm, and being in good physical health as determined by medical history, vital signs, and electrocardiogram. The decision to require 7 CPD was based on a prior trial completed by our group in which the more standard ≥10 CPD criterion was a primary reason for excluding African-American, but not Caucasian, smokers. Exclusion criteria included a medical or psychiatric condition potentially making study participation unsafe, current treatment for nicotine dependence; for women, pregnancy, breastfeeding, or unwillingness to use adequate birth control. The present study evaluated the association between smoking abstinence and illicit-stimulant abstinence in the cocaine-dependent (n=147), methamphetamine-dependent (n=102), and pooled (n=249) samples.

2.3 Measures

Abstinence from cocaine/methamphetamine was defined by stimulant-negative urine drug screens (UDS) and self-report of no stimulant use at each research visit. A rapid UDS system that screened for drugs of abuse including cocaine, methamphetamine, amphetamine, opioids, benzodiazepines, and marijuana was used to analyze the urine samples (Branan Medical Corporation). To avoid falsification, urine samples were collected using temperature monitoring and the validity of urine samples was checked with the use of a commercially available adulterant test. Self-report of substance use was assessed using the Timeline Follow-back (TLFB) method (Sobell and Sobell, 1992; Fals-Stewart et al., 2000),

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which is widely employed and well-validated. Achievement of smoking cessation was defined as achieving smoking-point prevalence abstinence (PPA) during the final treatment week (week 10). PPA is a standard measure and is defined as self-report of not smoking in the previous seven days, confirmed by a CO level <8 ppm (Hurt et al., 2003). In accordance with National Institutes of Health policy, participants self-reported their race and ethnicity based on the race/ethnicity classifications used in the 2000 United States Census.

2.4. Data analysis

All analyses were completed using SAS, Version 9. 3 (SAS Institute, 2010). Illicit-stimulant abstinence weeks during the post-smoking-quit phase (e.g., study weeks 4-10) were regressed using a mixed model regression including smoking-cessation status (e.g., continued smoking vs. smoking abstinent), and study week randomizing on participant. To determine whether any differences in stimulant abstinence were specific to smoking-cessation status, participant demographic and baseline characteristics on which the groups significantly differed were evaluated, via the corrected Akaike Information Criteria (AICC), for inclusion as covariates in the regressions. This approach allowed an evaluation of the association of illicit-stimulant abstinence with smoking-cessation status when other variables related to smoking-cessation status were controlled. It has been recommended that effect sizes be reported along with p-values to provide information about the clinical significance of an effect in addition to its statistical significance (Nakagawa, 2004). Consistent with this recommendation, we calculated the Cohen's *d* (Cohen, 1988), using mean and standard deviation estimates generated from the raw data, for statistically significant effects.

3. RESULTS

Demographic and baseline characteristics for the participants are provided in Table 1. The association between smoking abstinence and illicit stimulant abstinence was evaluated for the pooled sample and for the subsets of cocaine-dependent and methamphetamine-dependent participants. As can be seen in Table 2, the results revealed a significant difference only for the cocaine-dependent subsample in which participants who stopped smoking were abstinent for illicit stimulants an average of 78.2% of the post-smoking-quit weeks (weeks 4-10) relative to 63.6% in participants who continued smoking; the effect size for this difference is d=0.36.

4. DISCUSSION

The present study evaluated the association between smoking abstinence and illicit-stimulant abstinence in cocaine- and/or methamphetamine-dependent participants receiving smoking-cessation treatment. The results revealed a significant association for the cocaine-dependent participants with no significant differences for the sample as a whole or for the methamphetamine-dependent participants.

The present results are consistent with a fairly substantial literature finding a significant relationship between cigarette smoking and cocaine abuse. They are also consistent with the finding that methadone-maintained participants receiving smoking-cessation treatment provided significantly more cocaine-free urines during weeks in which they were smoking abstinent (Shoptaw et al., 2002). In the present study, a significant association between smoking cessation and illicit-stimulant abstinence was not found for the methamphetamine-dependent patients and, thus, the effect in the cocaine-dependent patients appears to be more than a matter of patients who are able to stop one substance (cigarettes) being better able to stop using substances in general (e.g., illicit stimulants). The effect observed in the cocaine-

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dependent patients had a Cohen's d (Cohen, 1988) of .36, which is in the range of a clinically-meaningful effect.

A potential explanation for the observed significant association between smoking cessation and illicit-stimulant use in cocaine- but not methamphetamine-dependent participants comes from a secondary analysis exploring the relationship between menthol cigarettes and illicit stimulant abuse in these participants (Winhusen et al., 2013). Specifically, the results revealed that 67% of the cocaine-dependent participants smoked menthol cigarettes and that menthol cigarettes were associated with self-reported prolongation of the cocaine high and with greater illicit stimulant use, associations not seen in the methamphetamine-dependent participants. Thus, for a majority of cocaine-dependent participants, achieving smoking abstinence could directly impact the reinforcing effects of cocaine by removing a substance that prolongs the illicit-stimulant high whereas methamphetamine-dependent participants achieving smoking abstinence would not obtain a similar benefit.

The present finding is of interest for several reasons. First, because the prevalence of smoking in cocaine-dependent patients is 75-80% (Budney et al., 1993; Gorelick et al., 1997; Sees and Clark, 1993), the proposed treatment approach would be applicable for the majority of cocaine-dependent patients. Second, cigarette smoking is deadly, accounting for 443,000 deaths annually in the United States (Centers for Disease Control and Prevention, 2008). Thus, using smoking-cessation treatment to intervene with both cocaine use and cigarette smoking would impact two important public health issues.

The present study has several strengths and a few limitations. First, this trial was conducted at 12 sites, which enhances the generalizability of the results, and included a large sample of stimulant-dependent participants. Another study strength is that it was conducted with individuals seeking treatment at SUD treatment programs and, thus, the results are likely generalizable to individuals in treatment for stimulant-dependence disorders (Winhusen et al., 2012b). A limitation is that the findings are correlational in nature and, thus, cause and effect determinations cannot be made. Second, this study included a treatment-seeking population of outpatients who were interested in quitting smoking, so the findings may not be generalizable to individuals not seeking treatment. Finally, bupropion may decrease cocaine use in some cocaine-dependent patients (Margolin et al., 1995; Poling et al., 2006; Stoops et al., 2012) and, thus, the observed connection between smoking cessation and illicit stimulant use may reflect a bupropion-induced global decline in addiction in some participants.

In conclusion, the present results suggest that cocaine-dependent patients achieving smoking cessation in response to smoking-cessation treatment might evidence not only improved smoking outcomes but improved cocaine-use outcomes as well. Future research to replicate this finding appears warranted.

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Table 1

Participant demographic and baseline characteristics as a function of dependence diagnosis and smoking-cessation abstinence

	Pooled Sample TAU+SCT (N=249)			Cocaine-Dependent TAU+SCT (N=147)			Methamphetamine-Dependent TAU+SCT (N=102)		
	Continued Smoking N=174	Smoking Abstinent N=75	Smoking Analysis[a]	Continued Smoking N=107	Smoking Abstinent N=40	Smoking Analysis[a]	Continued Smoking N=67	Smoking Abstinent N=35	Smoking Analysis[a]
Age, mean (SD), y	37.7 (9.9)	36.2 (10.1)	W=1.1	41.4 (9.1)	40.4 (9.9)	W=0.3	31.8 (8.0)	31.5 (8.2)	W=0.4
Sex, male, n (%)	89 (51.1)	47 (62.7)	$X^2(1)=2.8$	57 (53.3)	29 (72.5)	$X^2(1)=4.4^*$	32 (47.8)	18 (51.4)	$X^2(1)=0.1$
Race, n (%)			$X^2(2)=0.9$			F=0.017			$X^2(1)=0.1$
African-American	56 (32.4)	27 (36.0)		56 (52.8)	27 (67.5)		0 (0)	0 (0)	
Caucasian	105 (60.7)	41 (54.7)		48 (45.3)	12 (30.0)		57 (85.1)	29 (82.9)	
Other/mixed	12 (6.9)	7 (9.3)		2 (1.9)	1 (2.5)		10 (14.9)	6 (17.1)	
Ethnicity, Hispanic, n (%)	20 (11.6)	11 (14.7)	$X^2(1)=0.5$	10 (9.4)	1 (2.5)	F=0.117	10 (14.9)	10 (28.6)	$X^2(1)=2.7$
Baseline use (prior 28 days)									
Days/stimulant use	2.3 (5.2)	1.3 (4.1)	T(174.0)=1.5	2.7 (5.7)	2.2 (5.5)	W=1.4	1.5 (4.1)	0.3 (0.9)	$T(76.6)=2.3^*$
Stimulant-free, n (%)	118 (67.8)	61 (81.3)	$X^2(1)=4.7^*$	68 (63.6)	31 (77.5)	$X^2(1)=2.6$	50 (74.6)	30 (85.7)	$X^2(1)=1.7$
Days/drug use	5.1 (8.7)	2.1 (5.4)	$T(218.4)=3.3^{**}$	5.7 (9.3)	3.5 (6.9)	T(93.1)=1.6	4.1 (7.8)	0.5 (1.6)	$T(76.3)=3.6^{***}$
Drug-free, n (%)	90 (51.7)	55 (73.3)	$X^2(1)=10.1^{**}$	51 (47.7)	26 (65.0)	$X^2(1)=3.5$	39 (58.2)	29 (82.9)	$X^2(1)=6.3^*$
Smoking history									
No. of Smoking years	21.1 (9.5)	19.4 (9.7)	W=1.4	24.2 (9.0)	22.4 (9.8)	W=1.0	16.1 (8.1)	16.0 (8.4)	W=0.2
No. of cigarettes/day	17.1 (8.6)	14.8 (7.6)	$W=2.1^*$	18.1 (8.9)	16.1 (7.5)	W=1.4	15.5 (7.7)	13.3 (7.5)	W=1.3

Note.

[a] W= Wilcoxon rank-sum test; X^2(df)= Pearson chi-square test; T(df)= Student's t-test; F= Fisher's exact test.

* p<.05

** p<.01

*** p<.001. Where not specifically indicated, numbers represent means (standard deviations).

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Table 2

Stimulant-abstinence during the post-smoking-quit phase as a function of dependence diagnosis and smoking-cessation abstinence

	n	% Weeks abstinent	Smoking Analysis (X^2)
Pooled sample (N=249)			
Continued smoking	174	71.0 (39.4)	2.97
Smoking abstinent	75	83.3 (31.4)	
Cocaine-dependent (N=147)			
Continued smoking	107	63.6 (41.8)	8.55**
Smoking abstinent	40	78.2 (35.7)	
Methamphetamine-dependent (N=102)			
Continued smoking	67	83.3 (31.7)	0.04
Smoking abstinent	35	89.2 (24.7)	

Note. $X^2(1)$= Type III Wald chi-square.

** p<.01.