

Changes in Tobacco Smoking Following Treatment for Cocaine Dependence

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Abstract: Incorporation of smoking cessation into cocaine treatment programs remains a challenge. A major concern is that cocaine abusers may tend to substitute one drug for the other. If this is true, successful treatment of cocaine abuse should lead to an increase in tobacco smoking. We compared tobacco smoking at admission, end of treatment and 9-month follow up for 168 crack cocaine dependent patients entering a 12-week outpatient treatment program for substance abuse. Smoking cessation was not a part of treatment. As expected cocaine patients improved with treatment and showed significant reduction in scores on the Addiction Severity Index (ASI). There were no significant changes in number of cigarettes smoked per day or scores on the Fagerstrom Test for Nicotine dependence (FTND) from baseline to end of treatment or follow-up. Also, there were no differences in the proportions of nonsmokers and smokers who changed their smoking habits over the treatment and follow up period. At follow up subjects who were abstinent as well as those using cocaine showed no changes in tobacco smoking. There is no evidence that reduction in crack cocaine smoking

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following treatment is accompanied by an increase in tobacco smoking. It appears that concerns over tobacco being substituted for cocaine may be unfounded in this population.

Keywords: Tobacco, smoking, cocaine, substance abuse

INTRODUCTION

In contrast to the noticeable decrease in smoking among the general adult population in the United States, little or no decline in tobacco use has been observed among those who abuse alcohol and illicit drugs (1). The prevalence of tobacco smoking in substance abusers is found to be 4–5 times higher than the general population with smoking rates of 75–85% reported among cocaine abusers (2–5). The health hazards of tobacco use among substance abusers, including premature death, have been well-documented. For example, Hurt et al. (6) found that individuals treated for substance dependence continued to have a high risk of developing tobacco-related diseases and were also more likely to die from tobacco-related causes than from illnesses related to their primary substance of abuse.

Despite substantial evidence on the medical consequences of smoking and the addictive properties of nicotine most chemical dependency programs have failed to incorporate smoking cessation as a part of overall treatment of substance abusers (7). A striking example of this practice is a nationwide survey that found that availability of smoking cessation treatments in substance abuse programs in 2000 was essentially the same as 15 years ago (8). Such resistance has largely resulted from beliefs that substance abusers do not want to stop smoking at the time they receive drug abuse treatment; or those who quit smoking are at greater risk of relapsing to other illicit drugs, or dropping out of treatment.

Concerns about crack cocaine abusers relapsing to drug use after smoking cessation have not been supported by most studies. For example, Campbell et al. (9) found that substance-abusing patients who were either successful or unsuccessful at smoking cessation relapsed to other drugs at the same rate. Similarly, Hurt (10) reported no difference in abstinence rates from nonnicotine drugs between those who had received smoking cessation treatment one year earlier and those who had not. There is also evidence that tobacco use predicts poor outcome for cocaine-dependent patients (11) and that smoking cessation might actually improve abstinence from other drugs and alcohol (12, 13).

These studies, however, did not address the parallel question of whether a reduction in crack cocaine might increase tobacco consumption. This notion is based on several similarities shared by crack cocaine and

tobacco smoking. These include common forms and routes of administration; and similar effects on mesolimbic dopamine pathways that mediate cue-induced craving (14) leading to the belief that tobacco smoking may be a possible trigger for crack use and vice versa (15). In this context, there are reports that cocaine dependent patients can relapse after seeing their favorite cigarette lighter (16). There is little research on this topic and the data are mixed. Wiseman and McMillan (17) have presented perhaps the most thorough investigation of the relationship between cocaine treatment and smoking cessation. They noted a significant decrease in cigarette smoking after successful cocaine treatment in 30 of 43 outpatients. Apart from the small sample size, the study included only cocaine abstinent subjects, and there was no follow up. Given the paucity of data in this area, we prospectively studied whether cocaine abusers receiving drug abuse treatment changed their smoking habits at the end of treatment and at follow up.

METHODS

Subjects

One hundred sixty-eight cocaine-dependent subjects were recruited from a publicly funded, university-affiliated, intensive outpatient cocaine treatment program that serves a predominantly inner-city population in Philadelphia. The study was approved by the Institutional Review Board of Thomas Jefferson University and written informed consent was obtained from all subjects. Over 90% of patients in our treatment program were African American, and over 95% smoked crack cocaine; therefore, only African American patients smoking crack cocaine were included in the study. The Structured Clinical Interview (SCID) for DSM-IV Axis I Disorder (18) then was administered to individuals who volunteered for the study. Subjects were included if they were diagnosed with primary cocaine dependence in accordance with DSM-IV criteria. Patients who used other illicit drugs or alcohol were included only if their drug of choice was cocaine. Patients with past or present comorbid schizophrenia or bipolar disorder were excluded.

Assessments at Admission to Treatment Program

Tobacco Use

At admission, all cocaine dependent patients were assessed to determine whether they smoked tobacco and fulfilled DSM-IV criteria for nicotine

dependence. For smokers, numbers of cigarettes smoked per day were recorded. Patients who had not smoked in the previous 12 months were considered former smokers while individuals who had smoked less than 100 cigarettes in their lifetime were considered nonsmokers. At admission, subjects also completed the Fagerstrom Test for Nicotine Dependence (FTND), a widely used and validated 6-item questionnaire to assess severity of smoking that can be completed in 2 minutes (19). It is a revised version of the Fagerstrom Tolerance Questionnaire (20). The 6 questions on the FTND pertain to: smoking within 30 minutes of waking up, difficulty in stopping smoking in places where it is forbidden, difficulty in giving up morning cigarettes, number of cigarettes smoked per day, smoking during early part of the day, and smoking despite being ill. The total scores on the FTND range from 0 to 10. Cigarette smoking was not an eligibility criterion for the study and the sample included both nonsmokers and tobacco smokers.

Drug Use and Psychopathology

Subjects received a standard set of assessments as part of the intake process. This included demographic data, addiction and medical history, urine drug screens and physical examination. Severity of substance use was assessed using the Addiction Severity Index (ASI) a 40-minute structured interview assessing the severity and patterns of drug and alcohol use and impairment in employment, medical, legal, family/social and psychiatric domains of functioning (21). For each problem area it provides a subjective severity rating and a more objective composite score. Scores range from 0 (minimum) to 1 (maximum) in each domain. It has been widely employed in clinical and research settings. Depressive symptoms were rated using the Beck Depression Inventory (BDI), a 21-item, self-report questionnaire that assesses depressive symptomatology and requires about 10 minutes to complete (22). Urine drug screens were obtained for all subjects at admission using Accutests (Jant Pharmacal Corporation, California). The AccutestMulti Drug Screen is a one-step immunoassay for the detection of cocaine metabolites, opiates, amphetamines, cannabinoids, barbiturates and phencyclidine.

Treatment Program

Treatment for cocaine dependence was provided in a 12-week outpatient group therapy format with group sessions 3 times a week, each session lasting for 3 hours. In addition, patients participated in one 45-minute individual therapy session per month over the 12-week period. The treatment

approach was problem oriented and focused on attaining well-defined objectives. Techniques appropriate to particular problem areas were drawn from different treatment models including relapse prevention strategies (23). Such a multimodal approach is fairly typical of many outpatient treatment programs in the United States. Our program does not currently incorporate smoking cessation as a part of overall treatment for cocaine dependence; however data from the current study was expected to contribute toward plans to address tobacco use in our patient population. The ASI and FTND assessments were repeated at 6 week and at end of treatment (12 weeks or earlier if patient prematurely terminated treatment).

Follow-Up

Follow-up interviews for cocaine dependent subjects were performed at 9 months after admission treatment by research assistants and included administration of an abbreviated form of the ASI, the FTND and urine drug screens. The follow-up personnel were blind to the treatment condition or PRL measurements. Ninety-nine (58.9%) of the subjects were followed up.

Outcome Measures

In-treatment, end-of-treatment, and follow-up assessments were performed to provide objective estimates of abstinence from cocaine, retention, and participation in treatment, and attrition from treatment. These were as follows: A) *Number of negative urines*: Urine Drug Screens (UDS) similar to the admission UDS were obtained for subjects each week for the 12-week period. A urine sample was considered dirty if it was positive for any substance. The number of UDS negative for all tested substances was used as a measure of substance use during treatment. B) *Days in treatment*: This reflects treatment retention and was recorded as the number of days between the first and last counseling session. C) *Number of group and individual counseling sessions attended*: The total number of group and individual sessions attended by each patient offers another estimate of treatment retention and also reflects participation in the treatment process. D) *Dropouts*: These were defined as individuals who attended no more than 2 treatment sessions during the 12-week program. E) *Symptom reduction during treatment and at follow-up*: The baseline (admission) composite score in each of the 7 problem areas of the ASI minus the last composite score obtained during the 12-week treatment period was employed as a measure of problem reduction during treatment. Similarly, the follow-up composite scores on the ASI were

subtracted from the baseline ASI to compute problem reduction at follow-up.

Data Analysis

Comparisons of ASI and FTND scores at admission with those at end of treatment were performed using paired t tests (two-tailed). Similar comparisons were also performed between admission and follow up assessments. The last observation carried forward (LOCF) analysis was used for patients who did not complete treatment, but had FTND and ASI assessments performed on at least 2 occasions. The proportion of subjects who changed their smoking habits over time was examined using the McNemar test (24). This test calculates the significance of change for proportions of paired samples. Pearson correlations were employed to examine relationship between ASI and FTND scores. Demographic variables were compared using chi square or t tests as appropriate.

RESULTS

Subjects

Out of 258 crack cocaine dependent patients screened, a total of 168 subjects met the inclusion criteria and were enrolled in the study. Excluded subjects were: 8 patients with current major psychiatric disorder, 73 patients who also met criteria for alcohol/other illicit drug dependence, 9 patients who were intermittent tobacco smokers and/or primarily smoked cigars or chewed tobacco. The sample included 114 (67.8%) men and 54 (32.2%) women; 142 (84.5%) were African American, 20 (11.9%) were Caucasian, and 6 (3.6%) were Hispanic.

One hundred thirty-nine (82.7%) of patient were tobacco smokers. The mean FTND score at admission was 4.3 ± 2.6 . Cocaine patients smoked 16.6 ± 8.3 cigarettes smoked per day at admission to the treatment program. There were no gender or ethnic differences in FTND scores or number of cigarettes smoked among cocaine abusers.

Change in Cigarettes Smoked and Level of Nicotine Dependence During Cocaine Treatment and Follow-Up

First we examined the change in smoking for the entire cohort from baseline to end of treatment and follow-up by comparing cigarettes smoked per day and FTND scores at baseline, end of treatment and follow up in the total sample. The results are summarized in Table 1. There were

Table 1. Change in nicotine dependence scores (FTND) and number of cigarettes smoked during treatment and follow up of cocaine abusers

Smoking status	Number of subjects	Mean (SD)	Paired t-test (Significance)
Cigarettes/day			
At Baseline compared to end of 12-week treatment	108	Baseline	
		16.49 (9.7)	-1.321
At Baseline compared to follow-up	99	End of 12-weeks	(0.189)
		17.03 (10.0)	
At Baseline compared to follow-up	99	Baseline	
		17.17 (9.6)	-1.773
At Baseline compared to follow-up	99	At Follow-up	(0.079)
		18.02 (9.8)	
Fagerstrom Test for Nicotine Dependence			
At Baseline compared to end of 12-week treatment	108	Baseline	
		4.32 (2.66)	-0.815
At Baseline compared to end of 12-week treatment	108	End of 12-weeks	(0.417)
		4.38 (2.58)	
At baseline compared to follow-up	99	Baseline	
		4.37 (2.63)	-1.290
At baseline compared to follow-up	99	At Follow-up	(.200)
		4.46 (2.54)	

no significant differences in the number of cigarettes smoked per day or level of nicotine dependence over the treatment and follow up period. Next, we investigated whether cocaine dependent patients changed their smoking status during treatment and follow-up. For this purpose we analyzed the proportion of smokers and nonsmokers who maintained or changed their smoking habits from baseline to follow-up. The results are summarized in Table 2.

The majority of cocaine patients maintained their smoking status from baseline to end of treatment and follow up. For example 85% and 75% of nonsmokers at baseline continued to be nonsmokers at end of treatment and at follow-up respectively. Similarly, of the patients who smoked 11–25 cigarettes at baseline, 79% and 76% continued to smoke at the same level at the end of treatment and at follow-up, respectively. There were no significant increases or decreases in level of smoking across the various categories.

Change in Smoking Status in Cocaine Using and Abstinent Subjects at Follow-Up

We also examined the possibility that subgroups of subjects, in particular those who were abstinent from cocaine may show an increase

Table 2. Proportion of cocaine abusers who changed smoking status at 12-weeks and at follow-up

Smoking status at baseline	Smoking status after 12-weeks of treatment*				
	Non smokers N (%)	1–10/day N (%)	11–25/day N (%)	>25/day N (%)	Total N = 108 (%)
Non smokers	16 (85)	2 (10)	1 (5)	0 (0)	19 (100)
1–10/day	1 (14)	5 (72)	1 (14)	0 (0)	7 (100)
11–25/day	1 (2)	2 (3)	46 (79)	9 (16)	58 (100)
>25/day	0 (0)	1 (4)	0 (0)	23 (96)	24 (100)

Smoking status at baseline	Smoking Status at Follow-up**				
	Non smokers N (%)	1–10/day N (%)	11–25/day N (%)	>25/day N (%)	Total N = 99 (%)
Non smokers	12 (75)	3 (19)	1 (6)	0 (0)	16 (100)
1–10/day	1 (20)	3 (60)	1 (20)	0 (0)	5 (100)
11–25/day	1 (2)	2 (4)	41 (76)	10 (18)	54 (100)
>25/day	0 (0)	1 (4)	0 (0)	23 (96)	24 (100)

* $\chi^2 = 177.47$ (df = 9) M^cNemar p = 0.096 (Exact test)

** $\chi^2 = 137.40$ (df = 9) M^cNemar, p = 0.07 (Exact test)

Note: Data on dropouts were excluded from analyses.

in smoking. For this purpose we compared the change in cigarettes smoked per day from baseline to follow-up separately in subjects who were abstinent from cocaine (had negative urines) and those who were using cocaine (had positive urines). The results are summarized in Table 3.

As seen in Table 3, both groups of subjects—those who were abstinent from cocaine as well as those who were using cocaine—at follow-up showed no changes in their smoking status from baseline to follow-up. For example, among cocaine-abstinent subjects, 80% of those who smoked between 11–25 cigarettes per day at baseline remained at the same level at follow-up. In the cocaine using group, the comparable proportion was 87%. We also compared the proportion of nonsmokers and smokers at follow up between subjects with cocaine positive and cocaine negative urines. The results showed that the proportion of nonsmokers and smokers at follow up did not differ between the two groups ($\chi^2 = 1.46$, $p > .05$). Also patients with cocaine positive and cocaine negative urines at follow-up did not differ in the number of cigarettes smoked ($t = 1.34$, $df = 1$, $p = 0.47$, two-tailed).

Table 3. Smoking status at follow up among abstinent and actively using cocaine abusers

Urines at follow-up	Baseline Smoking	Smoking status at follow-up				Total
		Non Smoker	1-10/day	11-25/day	> 25/day	
Negative Urine N = 46	Non-Smoker	6 (75 %)	1 (13 %)	1 (13 %)	0 (0 %)	8 (100 %)
	1-10/day	2 (33 %)	4 (67 %)	0 (0 %)	0 (0 %)	6 (100 %)
	11-25/day	1 (5 %)	1 (5 %)	16 (80 %)	2 (10 %)	20 (100 %)
	>25/day	0 (0 %)	0 (0 %)	0 (0 %)	12 (100 %)	12 (100 %)
Positive Urine N = 39	Non-Smoker	6 (100 %)	0 (0 %)	0 (0 %)	0 (0 %)	6 (100 %)
	1-10/day	0 (0 %)	2 (50 %)	2 (50 %)	0 (0 %)	4 (100 %)
	11-25/day	0 (0 %)	0 (0 %)	14 (88 %)	2 (13 %)	16 (100 %)
	>25/day	0 (0 %)	0 (0 %)	1 (8 %)	12 (92 %)	13 (100 %)

Note: Urine samples not obtained on 14 subjects. All $\chi^2 < 1.84$, $p > .05$ in each case.

Relationship Between Changes in Drug Use and Smoking

Finally, we explored whether subjects who showed either an increase or decrease in measures of drug severity showed changes in smoking. We first examined whether change in symptoms occurred in the sample during treatment and at follow-up by comparing the mean pretreatment and end-of-treatment ASI composite scores and the mean pretreatment and follow-up ASI scores. As expected, cocaine abusers showed a reduction in their drug use with treatment. At the end-of-treatment program, statistically significant reduction in ASI composite scores had occurred in the 5 of the 7 areas functioning during treatment: drug ($t=3.24$, $p<.01$), alcohol ($t=2.98$, $p<.01$), medical ($t=2.62$, $p < .05$), family ($t=2.79$, $p<.05$) and psychiatric ($t=3.71$, $p<.01$). No differences were observed in ASI composite scores for employment ($t=.07$) and legal ($t=.10$) domains. At 6-month follow up, the symptom reduction on ASI was maintained in 4 domains: drug ($t=2.91$, $p<.01$), alcohol ($t=2.72$, $p<.01$), medical ($t=2.48$, $p<.05$), and psychiatric ($t=2.93$, $p<.01$).

We then determined the correlation between ASI scores in each domain and number of cigarettes smoked at beginning of treatment, at end of treatment (12 weeks) and at follow-up. There were no significant correlations between ASI scores in each domain and cigarettes smoked at the 3 time points (r ranged from -0.03 to 0.11 , $p > .05$ in each case). We also examined the association between changes in ASI scores from beginning to end of treatment and follow-up with changes in number

of cigarettes smoked over the corresponding period. There were no significant correlations between changes in ASI scores on each domain with changes in cigarettes smoked from beginning to end-of-treatment (r ranged from -0.03 to 0.04 , $p > .05$ in each case) or from beginning of treatment to follow-up (r ranged from -0.02 to 0.03 , $p > .05$ in each case). These results show that there were no relationships between changes in drug use and smoking over the treatment and follow-up period.

Because age or mood symptoms could have affected tobacco or drug use, we reanalyzed the data to determine whether age and BDI scores were correlated with FTND scores. No significant correlation was observed of FTND scores with age ($r = -.08$) or BDI scores ($r = .10$) at baseline, end of treatment (all r values $< .11$) or follow up (all $r < .10$). Therefore, it is unlikely that age or depression could have confounded the results. We also examined whether there were gender differences in our findings. Men and women did not significantly differ in FTND scores ($t = 1.26$) or number of cigarettes smoked ($t = 1.13$) at baseline. There were no differences in proportion of smokers and nonsmokers at baseline, end of treatment and at follow up among men and women ($\chi^2 < 1.91$, $p > .05$ in each case).

DISCUSSION

Changes in Smoking During Cocaine Treatment

The principal goal of this study was to explore the disturbing possibility that those who reduced or stopped their use of crack cocaine might increase their consumption of tobacco as a substitute. The results did not support this hypothesis. The smoking habits of cocaine users mostly remained the same over the treatment and follow-up period. Over 85% of cocaine abusers who did not smoke at baseline continued to be nonsmokers at end of the 12-week treatment. Similarly 70–95% of smokers at baseline continued to smoke at the same levels at end of treatment. At 9-month follow-up, 75% of nonsmokers and 60–95% of smokers had not changed their smoking status. Also, there were no overall differences in the number of cigarettes smoked or level of nicotine dependence over the study period.

It is not surprising that cocaine abusers did not report much change in their smoking habits during treatment and follow-up because the treatment did not address tobacco use. Our findings are consistent with findings from a recent study on 649 substance abusers entering treatment found that 13% of baseline smokers reported quitting at 12-month follow-up (13). In this context, 2 studies have found little or no change

in tobacco smoking after beginning cocaine use (25, 26). However discrepant findings have also been reported. Wiseman and McMillan (17) found a significant decrease in cigarette smoking after successful cocaine treatment in 30 of 43 patients in an outpatient setting. It must be noted though that the subject pool included subjects who were abstinent from cocaine for an average of 13 months at the time of the interview, and the reduction in smoking was restricted to former heavy tobacco smokers (25–140 cigarettes per day when using cocaine). The majority of medium to light smokers reported smoking the same amount or more following cocaine cessation.

Smoking Cessation in Cocaine Abusers

Consistent with literature (2) we found a very high rate of smoking (82.5%) in crack-cocaine abusers. Studies have shown that substance abusers who start or continue to smoke during substance abuse treatment may be at higher risk for relapse (13, 27). It also appears that spontaneous quit rates among substance abusers in treatment are low (28). Despite this evidence smoking cessation treatment is often not a part of drug treatment programs including the university-affiliated program where the present study was conducted. Our findings show that successful treatment of cocaine abuse does not lead to increase in tobacco use; furthermore, cocaine abusers are unlikely to quit smoking by themselves. This highlights the importance of incorporating smoking cessation interventions as a part of substance abuse treatment programs. Our findings also suggest that concurrent smoking cessation with drug treatment may not lead to adverse outcomes, however, more definitive data regarding this issue may be forthcoming from the randomized controlled studies such as the ongoing Timing of Alcohol and Smoking Cessation Trial (29).

Limitations

A potential confounder could be that smokers did not increase their smoking because of a ceiling effect, that is, further increases in smoking were limited by preexisting heavy smoking by cocaine abusers. For this purpose we analyzed the data separately for light (1–10 cigarettes per day), moderate (11–25 cigarettes per day) and heavy (more than 25 cigarettes per day) smokers. Nearly two-thirds of smokers were light or moderate smokers. Only 15–20% of light and moderate smokers showed an increase in their smoking over the study period suggesting that the results were unlikely to be biased by ceiling effect. Another confounder was the possibility that the increase in smoking in patients who stopped

using cocaine could be masked by reduction in smoking in those who continued to use cocaine. This was not supported by data that showed subsets of abstinent as well as drug using patients at follow up did not report substantial change in their smoking habits (Table 3). Moreover, there was no association between changes in drug use as measured by ASI and number of cigarettes smoked over the treatment period. Certain other limitations of the study need to be acknowledged. These include a measure of smoking by self-reports, a mixed population of cocaine and other substance abusers found in a “real world”, clinical setting rather than a “pure” cocaine abusing group, a 30% dropout rate, and an exclusively African American population. Therefore, our findings deserve replication using objective measures of tobacco use such as quantitative cotinine levels, and across other ethnic groups.

CONCLUSIONS

We found no evidence that reduction in crack-cocaine smoking following treatment is accompanied by an increase in tobacco smoking. It appears that concerns over tobacco being substituted for cocaine may be unfounded in this population. Longitudinal studies that include objective measures of tobacco use are needed to confirm our findings. Nevertheless, the data do not support exclusion of smoking cessation from overall treatment of cocaine abusers.

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