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BRIEF REPORT**CONTINGENCY MANAGEMENT FOR TOBACCO SMOKING
IN METHADONE-MAINTAINED OPIATE ADDICTS****STEVEN SHOPTAW, MURRAY E. JARVIK, WALTER LING, and
RICHARD A. RAWSON**

Los Angeles Addiction Treatment and Research Center and West Los Angeles VA Medical Center

Abstract — Seventeen methadone-maintained cigarette smokers received 4 weeks of contingency management (CM) as a stop-smoking intervention. Results indicated that CM patients significantly reduced breath CO levels from baseline to completion of treatment and that 23.4% of patients maintained 1 week or more of continued smoking abstinence. Results indicated a link between smoking abstinence and reduced cocaine use, although not reduced opiate use, which raised questions about possible shared biological and psychological mechanisms for tobacco and cocaine use.

I N T R O D U C T I O N

Most opiate addicts smoke cigarettes despite the strong, long-term link between cigarette smoking and mortality (Hser, McCarthy, & Anglin, 1994). Evaluations of stop-smoking interventions for methadone-maintained patients have typically reported poor outcomes (Jarvik & Schneider, 1992; Story & Stark, 1991), clearly illustrating the need for more effective smoking-cessation strategies. Long-term follow-up of opiate addicts also suggests that nonsmokers have lower rates of cocaine and other illicit drug use (Hser et al., 1994). Developing and implementing effective smoking-cessation programs for methadone-maintained patients may lead to future reductions in associated health care costs and immediate reductions in illicit drug use.

This project evaluated the efficacy of contingency management, a behavioral intervention applied effectively in treating opiate (Stitzer, Iguchi, & Felch, 1992) and cocaine dependence (Higgins et al., 1991; Higgins, Budney, & Bickel, 1994) as a stop-smoking intervention among methadone-maintained opiate addicts. We also explored whether reductions in tobacco smoking corresponded with reductions in other drug use.

M E T H O D

Subjects

Seventeen patients (4 females, 13 males) receiving methadone-maintenance treatment at the Los Angeles Matrix Institute on Addictions participated in this pilot study. The average patient was 43.76 years old, smoked at least one-half pack of cigarettes per day, and was in satisfactory clinic standing. Patients averaged 8.22

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Requests for reprints should be sent to Steven Shoptaw, PhD, Matrix Center and Los Angeles Addiction Treatment Research Center, 10350 Santa Monica Blvd., Suite 340, Los Angeles, CA 90025.

years ($SD = 0.36$) of prior methadone maintenance and 23.94 ($SD = 10.69$) years of prior cigarette smoking.

Prior use of illicit drugs was estimated by analyzing required monthly urine samples collected for 1 year before the stop-smoking treatment. The predominant pattern of detected drug use was regular illicit opiate use and infrequent or no cocaine use (41.2%; $n = 7$). Equal percentages of patients (17.6%; $n = 3$) either had all samples positive for illicit opiates and cocaine or had all samples negative for illicit opiates and cocaine. Samples from three patients (17.6%) indicated infrequent use of illicit opiates and cocaine. The remaining patient was a regular user of cocaine only.

Procedure

Patients were recruited through flyers placed in the clinic lobby announcing a new stop-smoking treatment and by counselors approaching appropriate candidates directly. Interested potential candidates were informed of the nature of the contingency management (CM) treatment and of the data collection requirements. After providing written consent, patients began treatment.

Contingency management

Data collection requirements consisted of thrice weekly breath and urine samples. The 4-week CM treatment involved providing incentives, delivered in the form of vouchers, that could be exchanged for merchandise at the end of treatment for "no smoking" breath samples (CO levels ≤ 4 ppm). The first "no smoking" breath sample was worth \$2.50. Vouchers increased \$0.50 in value for each consecutive "no smoking" breath sample. Patients who provided breath samples that indicated recent smoking received no voucher for that day. Patients who completed the 4-week treatment without smoking could earn \$73.00. Although urine samples were analyzed for drugs of abuse, vouchers were contingent on smoking abstinence only.

Data analysis

Breath carbon monoxide (CO) was analyzed using a portable Bedfont Micro Smokerlyzer. A criterion of 4 ppm CO or less indicated abstinence from smoking. Urine samples were analyzed for metabolites of heroin (morphine) and cocaine (benzoylecgonine) by a local toxicology laboratory using HP/LC methods. CM efficacy was measured by the number of consecutive "no smoking" CO breath samples (maximum length of smoking cessation). Urine toxicology results were compiled using the Treatment Effectiveness Score (TES) (Ling, Shoptaw, Rawson, & Klett, 1995), a measure that assigns one point for each scheduled urine sample negative for metabolites of illicit drugs. "Dirty" and missing samples receive no points. In this 4-week trial, patients' TES could range from 0 (all 12 samples positive for drug metabolite) to 12 (all 12 samples negative for drug metabolite). The TES values were calculated separately for heroin and for cocaine; TES distributions were nonnormal and comparisons used nonparametric statistics, although mean values were reported for descriptive purposes. Scores were compared using the "any," "minor," and "major" smoking-cessation response variables described below to evaluate associations between smoking cessation and illicit drug use.

R E S U L T S

Question 1: How effective was CM as a smoking intervention in this group of substance abusers?

All patients resumed smoking by the end of the trial, although smoking levels were

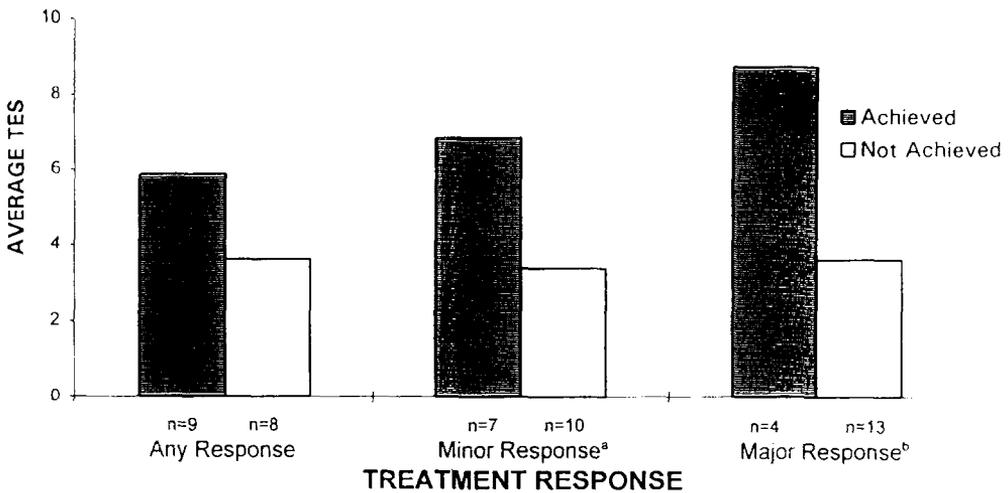


Fig. 1. Cocaine TES by Tobacco Smoking Cessation Response. ^a Significant difference between patients able to achieve "minor" response compared to patients unable to achieve "minor" response (Mann-Whitney $U = 56.6$, $df = 1$, $p < .05$). ^b Significant difference between patients able to achieve "major" response compared to patients unable to achieve "major" response (Mann-Whitney $U = 3.00$, $df = 1$, $p < .01$).

significantly reduced at treatment completion compared to baseline. Over three-quarters (76.5%) ended their treatments with lower CO levels compared to baseline. The CO reductions averaged from 16.06 ppm at baseline ($SD = 7.7$) to 10.35 ppm ($SD = 6.6$) by the end of the trial, a statistically significant and clinically relevant outcome (matched $t = 3.04$, $df = 16$, $p < .01$). Overall, 9 patients (52.9%) provided at least one "no smoking" breath sample (defined as "any response"); 7 (40.8%) produced at least two consecutive "no smoking" samples (defined as "minor response"; 3 days of smoking abstinence); and 4 patients (23.4%) produced three or more consecutive "no smoking" samples (defined as "major response" — a week of continual smoking abstinence). Post hoc analyses indicated patients with less nicotine dependence at baseline (lower than average baseline CO) were significantly more likely to use CM to achieve smoking abstinence than were patients with greater nicotine dependence.

Question 2: Did smoking cessation correspond to reductions in illicit drug use?

Urine toxicology results indicated that 94% ($n = 16$) of patients used illicit opiates at least once during the trial, and 65% ($n = 10$) used cocaine at least once. Analyses of cocaine TES indicated that patients able to maintain smoking abstinence were significantly less likely to use cocaine (see Fig. 1). Further, the length of smoking abstinence corresponded in a stepwise fashion with lowered rates of cocaine use. No statistically significant associations were found between smoking cessation and illicit opiate use.

DISCUSSION

Results from this pilot study indicate that methadone-maintained opiate addicts find CM to be an acceptable smoking-cessation treatment, and that they can use CM treatment to achieve modest smoking reductions. Questions remain whether different

reinforcement schedules or whether integration with other smoking-cessation interventions (e.g., nicotine transdermal patch) would increase efficacy of CM with this population.

Of interest was the finding that smoking outcomes were significantly associated with cocaine use in a stepwise fashion such that patients able to achieve longer periods of smoking cessation were less likely to use cocaine, although there was no association between smoking outcomes and illicit opiate use. The underlying mechanisms for these results could range from common brain pathways (e.g., nicotine and cocaine share neurotransmitter systems, and smoking could "prime" cocaine use or the reverse), to psychosocial mechanisms (e.g., smoking cessation generalizes to avoidance of cocaine use). However, these results provide initial support for the linkage between reductions in tobacco smoking and corresponding reductions in cocaine use in this population.

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