



Published in final edited form as:

Behav Pharmacol. 2018 June ; 29(4): 370–374. doi:10.1097/FBP.0000000000000349.

Decreases in Smoking During Treatment for Methamphetamine Use Disorders: Preliminary Evidence

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Abstract

Despite high rates of smoking (70–90%) and the severely negative impact of smoking on physical and mental health, only 12% of individuals receiving stimulant use disorder treatment also receive smoking cessation treatment. The aim of this investigation was to examine the effect of a contingency management intervention targeting methamphetamine use on cigarette smoking. Sixty-one adults with methamphetamine use disorders who were smokers were assigned to contingency management or standard psychosocial treatment. Rates of smoking-negative breath samples (carbon monoxide <3 ppm) were compared between the two groups while controlling for baseline carbon monoxide level, marijuana use, methamphetamine use, and time. This sub-group of mostly male (59%) participants included 44 participants in the contingency management group and 17 participants in the standard psychosocial treatment. Tobacco smoking participants who received contingency management targeting methamphetamine use were 140% (OR = 2.395; 95% CI: 1.073 – 5.346) more likely to submit a smoking-negative breath sample relative to standard psychosocial treatment during the treatment period, holding constant several other pre-specified

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Conflict of Interest

Drs. McPherson and Roll have received research funding from the Bristol-Myers Squibb Foundation. Drs. McPherson and Layton have received research funding from Ringful Health, LLC. Dr. McPherson has also received research funding from Orthopedic Specialty Institute, and consulted for Consistent Care company. This funding is in no way related to the investigation reported here. None of the other authors have any financial, personal, or other type of relationship that would cause a conflict of interest that would inappropriately impact or influence the research and interpretation of the findings.

covariates. This study provides evidence that a behavioral treatment for methamphetamine use results in reductions in cigarette smoking in adults with methamphetamine use disorder.

Keywords

co-morbid substance use treatment; stimulants and smoking; contingency management; stimulant use; methamphetamine use treatment; human

Introduction

There is a well-documented relationship between the use of stimulants (e.g. methamphetamine, cocaine) and tobacco smoking. Among cocaine users, estimates are 70–80% (Patkar et al., 2006) but among methamphetamine (MA) users this rate is even higher (87–92%) (Grant, Hasin, Chou, Stinson, & Dawson, 2004). There is evidence that these substances upwardly modulate one another's self-administration, which leads to a faster and greater 'cascading' risk of co-addiction (Gatch et al., 2008). This is likely due in part to neurobiologic synergies between dopamine and nicotinic acetylcholine receptors in the central nervous system (see Adinoff, 2004 for a complete review; Williams et al., 2011). However, these effects are still in need of elucidation as there is human laboratory research that has failed to demonstrate a clear modulatory effect of nicotine on stimulants across several studies (Brewer et al., 2013; Sobel et al., 2004).

The mesolimbic dopaminergic and nicotinic cholinergic reward systems interact closely both in the ventral tegmental area and the nucleus accumbens, which provides mechanistic evidence of why the two substances are commonly used in tandem (Gatch et al., 2008). The most common route for stimulant and nicotine reward via dopamine flow is in the nucleus accumbens (Picciotto & Corrigall, 2002). A synaptic dopamine increase from stimulants leads to inhibition of reuptake (e.g. cocaine) or greater facilitated release (e.g., MA). Nicotine stimulates nicotinic acetylcholine receptors in the ventral tegmental area which leads more indirectly to increased dopaminergic activity (Weinberger & Sofuoglu, 2009).

Despite high rates of smoking and the negative impact of smoking on health, only 12% of individuals in stimulant treatment receive smoking cessation treatment (Weinberger & Sofuoglu, 2009). The high rate of cigarette smoking in this population contributes directly to medical illnesses (McClave et al., 2010; premature mortality (Kilbourne et al., 2009) and higher healthcare costs (Hackman et al., 2006). Although some preliminary evidence exists (Reid et al., 2008; Winhusen et al., 2014) the best method of integrating co-addiction treatment remains unclear. This is due, in part perhaps, to the mixed literature around different treatment modalities and their ability to have any kind of crossover effect from the target of stimulant abstinence to the target of smoking cessation (Baker et al., 2005; Patkar et al., 2006; Raczus, Gorelick, & Henningfield, 1998; Weinberger & Sofuoglu, 2009).

Contingency management (CM) is a behavioral intervention based on operant conditioning that provides reinforcement contingent on drug or alcohol abstinence. McDonnell and colleagues observed that among adults with serious mental illness who received CM for stimulant abstinence (primarily cocaine users), smokers assigned to CM also experienced

significant reductions in smoking (McDonnell et al., 2014) While co-use of mutually reinforcing substances may lead to increased use of both substances, decreased use of one of those substances may lead to reduced use of the other. This represents the ‘cascade-up, cascade-down hypothesis’ that we set out to examine in the current investigation, and in particular, the cascade-down component of that hypothesis. We hypothesized that a CM intervention designed to increase MA abstinence among MA dependent participants would also result in decreased tobacco smoking among smokers receiving CM for MA use. Our intention was to build upon the findings by McDonnell and colleagues by examining a similar question but among MA users without serious mental illness.

Methods

Data Source

Data for this study come from a previously published randomized controlled trial that investigated the impact of four different CM duration schedules on MA abstinence among treatment-seeking participants with MA use disorder (Roll et al., 2013) Below we summarize the methodology used in that trial for the purposes of this secondary data analysis (please see Roll et al., 2013 for additional details).

Study Procedures

Participants in this investigation were seeking treatment for MA dependence at a treatment facility located in southern California, USA. Participants randomized into the study met all of the following inclusion criteria: (1) 18–65 years of age, (2) DSM-IV criteria for MA dependence, (3) were willing and able to comply with study procedures, and (4) were willing and able to provide written informed consent. Exclusion criteria included: (1) a medical condition that, in the study PI’s judgment, may have interfered with safe study participation, (2) a recent (past 30 days) history of suicide attempts and/or current serious suicidal intention or plan, (3) a history of violent criminal behavior or current parole status, and (4) any other circumstances that, in the opinion of the PI, would interfere with study participation.

Informed consent was obtained and a baseline interview was conducted in which urine samples were collected and participants completed a battery of questionnaires and interviews. Participants were randomized to one of the four treatment conditions, all of which lasted 16 weeks total: Standard psychosocial treatment only (ST), or ST plus one of three durations of CM which was delivered for 1 month, 2 months, or 4 months. Participants were expected to provide urine samples on a Monday, Wednesday, Friday schedule throughout the course of treatment. ST consisted of a manualized protocol based on the Matrix Model for MA abuse (Rawson et al., 1995).

The CM intervention used the variable magnitude of reinforcement procedure, frequently referred to as the “fishbowl” technique, which is common in CM research (e.g., Roll et al., 1996). This procedure involved making “draws” from a bowl of chips representing different prize magnitudes. The maximum any one person could receive was approximately \$500 and the maximum payout averaged \$250 or less per participant, depending on the percentage of

MA negative urine samples submitted. The number of draws awarded at each urine collection escalated by one chip with consecutive weeks of MA negative urine samples (e.g., one draw in week one, two draws in week two). Missing or MA positive urine samples resulted in a reset to one draw available at the next negative sample submitted. The escalating schedule with a reset contingency has been demonstrated to increase duration of abstinence, but not necessarily decrease probability of relapse.

Outcomes

Urine samples were analyzed for use of cocaine, methamphetamine, amphetamine, marijuana, and opioids, using onsite immunoassays (Integrated E-Z-Split-Key[®] Cup, Innovacon-Inc.). Participants provided breath samples for alcohol (Alco-Pro-Alcosensor-III), and carbon monoxide (CO) analysis (Bedfont-Smokerlyzer Micro-IV). Negative CO tests were defined as CO < 3ppm, a cutoff used previously in our work and that of others. (Javors, Hatch, & Lamb, 2005)

Analytic Strategy

For this analysis, we collapsed the 1 month, 2 month, and 4 month CM conditions into a single CM condition category. The primary outcomes paper reported statistically significant effects across the three different CM duration schedules on MA abstinence. (Roll et al., 2013) We collapsed the three CM conditions for this analysis in order to maximize statistical power and to isolate our hypothesized, indirect effect of CM on smoking. This left two groups; the CM group and the ST group. For this analysis, we included only those participants who submitted a positive CO sample (CO ≥ 3ppm) at baseline, which classified this sub-sample as smokers. The combined sample of CM and ST participants who were classified as smokers was 51% (n=61 out of a possible N=118) of the original RCT.

Smoking-negative breath samples during the 16-week treatment period was the primary outcome, with group assignment (CM versus ST) as the main predictor. We also included the pre-specified covariates of time, time-varying cannabis (smoked cannabis can produce a positive CO result) and MA use, and baseline CO level. Generalized estimating equations (GEE) were used to analyze smoking abstinence over time. We used list-wise deletion, 'positive UA imputation', and multiple imputation (50 imputed datasets, Rubin's rules used to combine parameter estimates) in order to handle missing data. (Rubin, 1996, 2004) List-wise deletion and positive UA imputation are commonly used methods in substance use disorder treatment research. However, multiple imputation is an expert recommended method of handling missing values. (McPherson et al., 2012, 2013, 2015). We use all three methods to demonstrate the robustness of our findings. We also examined total number of smoking-negative breath samples during the 16-week treatment period as a secondary outcome. We used multiple linear regression to analyze this outcome with the same pre-specified covariates.

When analyzing demographics to check for differences in baseline characteristics across the two groups, we used non-parametric statistics (e.g., Kruskal-Wallis), given the imbalance in group sizes, and Fisher's exact test instead of Chi-square tests in cases of low cell counts

(i.e., $n < 5$ per cell). All analyses were performed using Stata 14.2. (StataCorp, College Station, TX).

Results

The average age among both groups was about 32 years of age, and the percentage of females ranged from 35% to 43% (see Table 1). The CM and ST groups were not statistically different on these or any other baseline demographics (e.g., age, baseline CO level).

CM for MA abstinence demonstrated a 187% (OR=2.869, 95% CI: 1.247– 6.603) increase in the odds of submitting a smoking-negative (CO<3ppm) breath sample during treatment, relative to those who received ST for MA abstinence, holding constant the set of pre-specified covariates (see Table 2). Baseline CO was also associated with a 25% (OR=0.762, 95% CI: 0.709– 0.818) decrease in the odds of submitting a smoking-negative breath sample. No other covariates were statistically significant. The same pattern of effects emerged when the missing data were treated with positive UA imputation (165% increase in the odds of smoking-negative breath sample submission in CM versus ST: OR=2.647, 95% CI: 1.157 – 6.54) and multiple imputation (140% increase in the odds of smoking-negative breath sample submission in CM versus ST: OR=2.395, 95% CI: 1.073 – 5.346). Importantly, these effects remained stable regardless of whether methamphetamine use and cannabis use were included (data not shown), but we opted to keep these covariates in the models because of their previously noted relevance.

Our multiple linear regression found that CM (compared to ST) was associated with an increase in the number of smoking-negative breath samples submitted during the treatment period ($B=1.985$, $p=0.050$; 95% CI: 0.001–3.970). A positive MA urine sample at baseline was associated with a decrease in smoking-negative breath samples submitted during the treatment period ($B= -3.156$, $p=0.009$; 95% CI: -5.521 – -0.791). No other covariates demonstrated an association with the outcome of total smoking-negative samples submitted during the treatment period.

Discussion

These preliminary findings demonstrate a reduction in tobacco smoking among smokers assigned to the CM for MA condition, relative to those assigned to the ST condition. The results suggest that smoking behavior can be modified during a MA intervention in adults with a MA use disorder, a population in which smoking is nearly ubiquitous and smoking cessation interventions are not widely available or often well integrated. Perhaps more importantly is that these data provide evidence of a cascading down (i.e., wherein abstinence from one substance could make abstinence from another, behaviorally and neurobiologically related drug, easier) that could be taken advantage of in future co-addiction treatment strategies.

Research in this area has been stalled perhaps in part due to concerns about how providing smoking cessation treatment may decrease the effectiveness of concurrently delivered stimulant addiction treatment.(Prochaska, 2010; Ziedonis et al., 2006) A meta-analysis by

Prochaska and colleagues (Prochaska et al., 2004) that included 19 studies found that provision of smoking cessation treatment may actually produce better addiction treatment outcomes. The way forward is not yet clear, but it is possible that the ‘cascade-up, cascade-down’ hypothesis could be used to effectively leverage the behavioral and neurobiological overlap in several co-morbid addiction varieties, especially if delivered as two high intensity treatments. (Kalman et al., 2010) This report provides important preliminary data that both replicates and extends previous work by McDonnell et al. (2014) in a similar study with primarily cocaine-using seriously mentally ill adults, while our study was with MA abusing non-mentally ill adults.

Future studies should collect concurrent cotinine data, and perhaps additional self-report data (e.g., cigarettes per day), to more accurately assess abstinence. A limitation of our study is that CO breath tests can be sensitive to several other environmental contaminants. However, one of the potential confounds, cannabis use, appears to not have had an impact on the current findings as we controlled for the effect of cannabis use over time.

Overall, our off-target findings of CM are consistent and of moderate size, which is consistent with other reports of off-target effects of CM. (McDonnell et al., 2014; McDonnell et al., 2013; McPherson et al., 2016) Moreover, the tobacco smoking outcomes observed in this preliminary study warrant further investigation. Development of novel behavioral and pharmacotherapeutic treatments that support patients with concurrent addiction to nicotine and MA is key given the high prevalence rates. This report demonstrates that CM may be capable of increasing both MA and smoking abstinence simultaneously, using a relatively low-cost (about \$250 per person) behavioral intervention.

Acknowledgments

Role of Funding Source

This project was supported by a grants to Dr. Donovan and the Clinical Trials Network Pacific Northwest Node (U10 DA013714) and to Dr. John Roll (R01 DA170884) from the National Institute on Drug Abuse. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health.

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Table 1

Participant characteristics among tobacco smokers enrolled into Contingency Management or Standard Treatment for methamphetamine use disorders.

	Standard Treatment (n=17)	Contingency Management (n=44)	p Value
Age ¹	32.88 (10.09)	32.02 (9.19)	0.711
Female ²	35.29 (6)	43.18 (19)	0.574
Race ²			1.000
American Indian/Alaska Native ²	0.00 (0)	4.45 (2)	
White ²	70.59 (12)	63.64 (28)	
Other ²	29.41 (5)	32.56 (14)	
Hispanic ethnicity ²	29.41 (5)	32.56 (14)	0.856
Employment ²			0.656
Employment for pay ²	35.71 (5)	47.06 (16)	
Unemployed for < 1 year ²	42.86 (6)	35.29 (12)	
Unable to work ²	0.00 (0)	14.71 (5)	
Other ²	35.29 (6)	25.00 (11)	
Income (<\$20,000) ²	64.71 (11)	65.91 (29)	0.929
Marital Status ²			0.520
Married ²	11.76 (2)	4.55 (2)	
Divorced ²	29.41 (5)	27.27 (12)	
Separated ²	5.88 (1)	6.82 (3)	
Never married ²	41.88 (7)	56.82 (25)	
Relationship ²	11.76 (2)	4.55 (2)	
Education (>12 th grade) ²	25.00 (4)	36.36 (16)	0.541
Baseline CO Level ¹	14.18 (9.11)	15.45 (8.79)	0.488
Baseline MA+ UA ²	100.00 (17)	97.73 (43)	0.488
Baseline THC+ UA ²	0.00 (0)	2.27 (1)	0.721

Note:

¹ M (SD);

² % (N).

MA = methamphetamine; UA= urine analysis; THC = tetrahydrocannabinol; CO = carbon monoxide

Table 2

Treatment condition and other covariates predicting smoking-negative carbon monoxide breath samples in methamphetamine use disorder patients.

Covariates	Odds Ratio	95% Confidence Interval	p Value
<i>Listwise Deletion for Missing Data</i>			
Group (0=ST; 1=CM)	2.869	1.247 – 6.603	0.013
Baseline CO Level	0.762	0.709 – 0.818	< 0.001
MA UA+	0.446	0.126 – 1.576	0.210
THC UA+	1.872	0.474 – 7.400	0.371
Time (in visits)	1.018	0.998 – 1.039	0.085
<i>Positive UA Imputation for Missing Data</i>			
Group (0=ST; 1=CM)	2.647	1.157 – 6.054	0.021
Baseline CO Level	0.775	0.724 – 0.830	< 0.001
MA UA+	0.460	0.128 – 1.657	0.235
THC UA+	1.947	0.486 – 7.811	0.347
Time (in visits)	1.017	0.998 – 1.038	0.093
<i>Multiple Imputation for Missing Data</i>			
Group (0=ST; 1=CM)	2.395	1.073 – 5.346	0.033
Baseline CO Level	0.773	0.721 – 0.829	< 0.001
MA UA+	0.431	0.119 – 1.562	0.200
THC UA+	1.564	0.371 – 6.600	0.543
Time (in visits)	1.017	0.999 – 1.037	0.099

Note: MA = methamphetamine; UA= urine analysis; THC = tetrahydrocannabinol; CM = contingency management; ST = standard treatment; CO = carbon monoxide.