

# The ironic effects of stigmatizing smoking: combining stereotype threat theory with behavioral pharmacology

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#### **ABSTRACT**

Aims Public service announcements often create media messages intended to stigmatize negative behaviors to reduce and prevent these behaviors. Drawing on social and cognitive psychology, we hypothesize that stigmatizing messages can create stereotype threat would be associated with shorter latency to first cigarette in the laboratory compared to the control condition. **Design** A double-blind, randomized, controlled trial in which participants completed two smoking lapse tasks, one at baseline and one post-intervention/control. **Setting** An experimental psychopharmacology laboratory in the western United States. **Participants** A community sample of non-treatment-seeking daily smokers (n = 77) received either a stereotype threat (n = 39) or neutral/control (n = 38) message. **Intervention** Participants received either a stereotype threat message that stigmatized smoking or a control message. **Measurements** The primary outcome measure was participants' ability to delay smoking during the smoking lapse task in the experimental session **Findings** The difference in delay time during the experimental session at the point where 50% of each group had smoked was 3 minutes. Cox proportional hazard models revealed that participants in the stereotype threat group were significantly less able to delay initiating smoking compared to the control group (hazard ratio = 0.504, P = 0.010, 95% confidence interval = 0.30, 0.85), after controlling for baseline latency to smoke. **Conclusions** Messages that elicit negative stereotypes of smokers operated as 'smoking-promoting messages' in the context of our controlled laboratory investigation.

**Keywords** Human lab, public health, smoking, smoking lapse task, stereotype threat, stigma.

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# INTRODUCTION

Tobacco use is the single most preventable cause of death, disease and disability in the United States [1]. Cigarette consumption is estimated to result in more than \$96 billion a year in medical costs and \$97 billion a year in lost productivity [1]. Furthermore, government-sponsored anti-smoking campaigns tend to cost more than \$50 million a year [2], although the efficacy of these campaigns is unclear. Many anti-smoking campaigns attempt to reduce smoking by stigmatizing smoking [3–7]. For example, in the United States smokers are negatively stereotyped as unattractive, deviant, weak-willed and dirty [8-10] and many anti-smoking campaigns link these stereotypes to tobacco consumption, with the notion that demoralization of smoking is associated with overall smoking reductions [9]. There is some evidence these campaigns work [11], such that advertisements stigmatizing smoking are noticed by

smokers [5] and smoking rates are lower in states where the public feels negatively towards cigarette smoking [12,13]. In addition, smokers who experience unfavorable public reactions are more willing to quit smoking [13,14]. However, there is also some evidence these campaigns do not work. Although these efforts to stigmatize smoking are related to intentions to quit smoking, these quit attempts are rarely successful [5].

To reconcile these inconsistent findings, the present study draws from the social psychological theory of stereotype threat. Stereotype threat is a distracting concern that one will be evaluated through the lens of a negative stereotype [15,16]. Stereotype threat emerges when aspects of the situation make salient relevant negative stereotypes [17,18]. Importantly, it is not necessary for an individual to psychologically identify with their negatively stereotyped group membership for stereotype threat to be experienced [16]. Research consistently shows the negative influence

of stereotype salience on the performance of women and minorities on standardized math and intelligence tests [19,20]. Stereotype threat does not require one to believe the negative stereotypes or feel a strong connection to the negatively stereotyped group but, rather, simply that one is aware of the stereotypes [15,21]. Because stereotype threat is brought about by situational cues to negative stereotypes, it has the potential to occur in any situation in which stereotypes are believed to apply [15,22].

In the United States, stereotypes are often employed in anti-smoking campaigns to reduce tobacco consumption [9]. Given that stereotype threat increases motivation to disprove these negative stereotypes, this suggests that the stigmatization of smoking should yield increased intention and desire to quit smoking. However, this motivation to quit smoking may be undermined by stereotype threat, given that the stereotype threat-driven desire to refute the stereotype creates distracting, anxiety-eliciting concerns that consume executive function and self-control resources which, in turn, are critical to successful smoking cessation [23-25]. Further, anxiety and negative affect tend to increase smoking urges and decrease smoking latency [26,27]. This may result from a stress-and-coping and mood-regulation approach to smoking, whereby smokers tend to believe that smoking can reduce negative affect [28-30].

In this study, we combine a stereotype threat manipulation with a human laboratory smoking lapse task [31,32] to test the effects of stigmatizing smoking in a community sample of non-treatment-seeking daily smokers. Based on the robust stereotype threat literature, we hypothesize that the salience of smoking-related stereotypes will increase the likelihood of smoking in the laboratory.

# **METHODS**

#### Design

This study consisted of a double-blind, randomized, controlled trial in which a community sample of daily smokers completed two smoking lapse tasks, one at baseline and one after receiving either a stereotype threat message that stigmatized smoking (n=39) or a control message (n=38). This design allowed us to test the hypothesis that smokers receiving stigmatizing messages can create stereotype threat would have a shorter latency to smoking the first cigarette in the laboratory, compared to smokers in the control condition.

# **Participants**

Participants were 77 non-treatment seeking daily smokers [29.9% female; mean age = 36.52, standard deviation (SD) = 12.48; 31.2% white, 44.2% African American, 6.5% Latino, 3.9% Asian, 3.9% Middle Eastern, 10.3%

other; 70.1% reported an income of less than \$3 0000]. Participants were recruited from the community using on-line and print advertising. Participants were recruited for this study only and were not a part of a larger study. Inclusion criteria were: (1) aged 18-55 years; (2) smoked ≥ 10 cigarettes per day, verified through cotinine test (≥ 100 ng/ml of cotinine) and expired carbon monoxide (CO) reading level > parts per million (p.p.m.); (3) < 3 months of smoking abstinence in the past year; (4) no recent use of cocaine, methamphetamine, heroin or other illicit drugs (other than marijuana) verified by urine toxicology; (5) negative pregnancy screen (if female); and (6) no life-time history of psychotic disorders. Eligibility was determined via a telephone screening and an inperson screening. Participants completed two separate testing sessions (5–10 days apart): (1) a baseline session and (2) an experimental session. Participants were compensated for each visit.

#### Study sample size

Power calculation for this study is an estimate, as no previous research has combined the proposed methodologies. Sample size estimates were determined to permit analysis of the research questions at an alpha of 0.05 and power level of 0.80. We estimated our effect size as a small-to-medium effect. Estimates of effect size, sample requirement and power follow Cohen (1992) [33]. We used G\*Power version 3 [34]. This analysis suggested the need for 36 participants per experimental condition (total n = 72). Our recruitment efforts slightly exceeded our target enrollment.

### **Procedures**

For both the baseline and experimental sessions, participants came to the laboratory following 12 hours of abstinence from cigarette smoking (biologically verified via carbon monoxide levels). All participants were required to have a breath alcohol concentration (BrAC) of 0.00 g/dl at each visit. All procedures in the baseline and experimental visits were the same.

Participants first completed a battery of demographic and other individual characteristics. To make smoking status salient, participants completed a time-line follow-back in which they were asked to recall how many cigarettes they smoked every day for the last month. Next, participants completed McKee's [31] smoking lapse task, a behavioral measure of ability to refrain from smoking. The smoking lapse task is validated with smokers and is sensitive to real-world predictors of smoking lapse [32,35,36]. In the first part of the task, eight cigarettes of the participants' preferred brand were placed in front of them with a lighter and an ashtray. For 50 minutes participants could begin a cigarette self-administration session at any point. If

participants chose to delay, they were awarded \$0.20 for each 5-minute increment of abstinence, with the potential to earn \$2.00 if they were able to abstain for the entire 50-minute delay period. This part of the task ended either when participants chose to smoke their first cigarette or after participants resisted smoking for the entire 50 minutes.

The second part of the task was a 60-minute cigarette self-administration session in which participants were given \$1.60 and lost \$0.20 for each cigarette that they smoked, with the potential to lose the \$1.60 if they smoked all eight cigarettes in front of them. Thus, if participants resisted smoking for the entire 50-minute delay period as well as the 60-minute self-administration period, they resisted smoking for a full 110 minutes and earned a total of \$3.60 (in addition to the general study compensation). All procedures were standard for the smoking lapse task [31] and the monetary compensation rates were based on comparable samples [37]. Participants were not allowed to keep any unsmoked cigarettes.

#### Conditions

Instructions prior to the smoking lapse task during the experimental session consisted of a double-blind randomized control administration of stereotype threat or control. Participants were urn randomized to either condition. All procedures were carried out on a computer using the program MediaLab. Recorded instructions were used to ensure that the research assistants administering the protocol were blind to experimental condition. The stereotype threat manipulation was administered via computer instructions that were visible only to participants and not to research assistants. The stereotype threat and control inductions were taken from previous stereotype threat research [38,39]. The stereotype threat condition highlighted negative smoking-related stereotypes (taken from previous rehealth campaigns stigmatizing consumption) [9]. Participants were told that the researchers were interested in the differences between smokers and non-smokers in traits such as willpower, laziness, weakness and responsibility, as well as how these traits relate to many important life outcomes. Participants were told the following: 'Your performance on the tasks that you are doing today will be compared to others from across the nation. One specific question is whether nonsmokers are superior across all positive traits or only certain types'. The control condition was a neutral condition and participants were given the following script: 'Today you will work on a number of different tasks'. The only difference between the experimental and control condition was that stereotypes were mentioned.

After the smoking task, all participants completed a thought-listing task to capture participants' affective reactions during the smoking task. Consistent with previous research, we anticipated that stereotype threat would lead to greater negative affect compared to control [40,41]. At the end of the study, participants completed a 10-minute process debriefing including a thorough explanation of the study, stereotype threat and how their behaviors could be influenced by stereotype threat [42].

#### Outcome measures

Latency to smoke

The primary outcome measure was participants' ability to delay smoking during the 110-minute smoking lapse task (delay + self-administration periods) in the experimental visit, controlling for performance during the baseline session. Participants who delayed smoking for the entire 110 minutes of the smoking lapse task were recorded as '110', because the experiment stopped at this predetermined point (i.e. study data were right-censored).

# Thought-listing

Participants completed a retrospective report following this prompt: 'We all have several thoughts that run through our mind at any given time. Please describe everything that you remember thinking about during the last 2 hours' [40,41]. The rest of the page was blank and participants could write anything they wanted.

# Statistical analysis

To analyze the effect of experimental condition (stereotype threat versus control) on latency to smoke during the smoking lapse task, a survival analysis—a series of univariate Cox proportional hazard regressions-will be conducted. This analysis was selected for a number of reasons. First, the present examination focused on time to an event (i.e. smoking). Secondly, there was non-normality of the latency variable. A Cox proportional hazard modeling approach is able to both accurately model this nonnormality as well as include covariates (baseline latency to smoke) in the model [32]. Because one key assumption of a Cox regression model is proportional hazards, we first assess the non-proportionality of hazard functions by generating time-varying covariates (predictor variables × logtime) for each of the predictor variables in our first model. When these time-varying covariates are significant, suggesting non-proportionality, they are retained in the second, final model allowing for a more accurate assessment of the predictor variable of interest [43].

# **RESULTS**

Descriptive statistics on the study outcomes are provided in Table 1. Participants delayed, on average, 23.73 minutes (range = 0–110) before smoking a cigarette. The difference

Table 1 Descriptive summary of study outcomes.

Variable	Control condition $n = 38$		Stereotype threat condition $n = 39$	
	Mean	SD	Mean	SD
Baseline delay in smoking	28.66	32.65	30.77	36.04
Experimental delay in smoking	23.16	34.71	24.28	34.33
Negative emotion words (LIWC)	0.90	1.23	1.44	1.54

SD = standard deviation.

in delay time during the experimental session at the point where 50% of each group had smoked was 3 minutes. The median time to smoke for the whole sample was 13 minutes during baseline and 4 minutes during the experimental session. The proportion of individuals who had smoked at the median time to smoke for the whole sample in the baseline session was 50% (n = 19) in the control group and 54% (n = 21) in the stereotype threat group (P = 0.73). During the experimental session, the proportion of participants who had smoked at the median time to smoke for the whole sample in the was 50% (n = 19) in the control group and 51% (n = 20) in the stereotype threat group (P = 0.93). The distribution of this measure was non-normal, with 42% of participants choosing to smoke immediately and 7.8% abstaining for the entire delay period. Demographic variables (e.g. age, sex, education, race) were not significant predictors of latency to smoke (all Ps > 0.10). Furthermore, the significance of the results did not change when the demographics were included in the model. Thus, demographic variables were removed from the two models in the reported analyses.

A survival analysis using a Cox proportional hazards regression model revealed a significant effect of condition (stereotype threat/control) on latency to smoke [hazard ratio (HR) = 0.504, P = 0.010, 95% confidence interval (CI) = 0.30, 0.85], controlling for latency to smoke during the baseline session and modeling non-proportionality (baseline latency to smoke × log-time: HR = 0.978, P < 0.001); see Table 2 and Fig. 1. This effect indicates that after controlling for baseline, participants randomly

assigned to the stereotype threat condition were significantly less able to delay initiating smoking compared to the control condition.

# Thought-listing

We conducted a text analysis of participants' open-ended thoughts during the smoking session. We used the LIWC2007 Linguistic Inquiry and Word Count text analysis software [44]. The LIWC2007 Dictionary includes 32 word categories tapping psychological constructs (e.g. affect, cognition, biological processes). The subcategory of interest for this study was 'affective processes', which included 'positive emotion' and 'negative emotion'. Under 'negative emotion', there were also the subcategories 'anxiety', 'anger' and 'sadness'. We focused on the category of negative emotion words (e.g. 'awful', 'problems', 'discouraging', 'wrong', 'bad') because we hypothesized that this is where we would see an effect of condition, based on past work on stereotype threat and rumination.

An independent-samples t-test revealed that the difference between stereotype threat and control conditions on the percentage of negative emotion words was not statistically significant,  $(t_{(75)} = 1.706, P = 0.092)$ .

# **DISCUSSION**

Many health campaigns use stigma as a way in which to decrease socially undesirable behavior. In the context of anti-smoking campaigns, for example, these often rely on

 $Table\ 2\ \ \text{Model summary of survival analysis using two Cox proportional hazards regression models}.$ 

	Model 1		Model 2	
Variable	HR (95% CI)	P-value	HR (95% CI)	P-value
Condition	0.409 (0.198-0.847)	0.016	0.504 (0.300-0.847)	0.010
Baseline latency to smoke	1.024 (1.008-1.041)	0.003	1.024 (1.008-1.041)	0.004
Condition $\times$ log (time)	0.887 (0.664-1.186	0.420	_	_
Baseline latency to smoke $\times$ log (time)	0.979 (0.972–0.986)	< 0.001	0.978 (0.972–0.985)	< 0.001

CI = confidence interval; HR = hazard ratio.

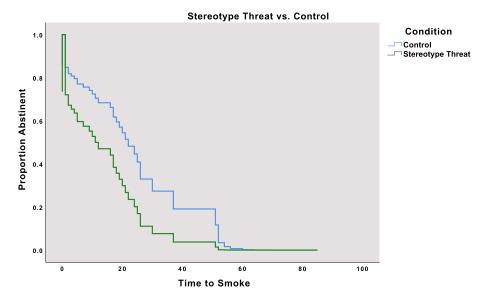


Figure I Cox proportional hazards survival curves depicting latency to smoke (i.e. the proportion of the sample abstaining from smoking as a function of time) during the smoking lapse task as a function of stereotype threat versus control condition, controlling for baseline latency. Non-proportionality was modeled via inclusion of a time-varying covariate (baseline latency to smoke x log-time).

communicating persuasive messages to the public that implicate the negative characteristics associated with smokers [9,13], with the goal of stigmatizing smoking to motivate both prevention and cessation. However, psychological research points to a particularly ironic consequence of stigmatizing smoking for regular smokers: stereotype threat. Stereotype threat, or the concern about confirming negative stereotypes, increases anxiety and depletes selfcontrol resources, the very processes that are central to quit attempts. In the present research, non-treatmentseeking daily smokers assessed in the laboratory smoked more quickly in a smoking self-administration task when presented with a message stigmatizing smoking, compared to when they received no previous message. Thus, our findings suggest that the stigmatization of smoking is not only an unsuccessful intervention tool but that it may backfire. causing regular smokers to smoke more quickly than they otherwise would. Latency to smoke in the lapse task is generally interpreted as an overall indicator of smoking behavior [31,32]. As such, our interpretation of the findings is that messages that elicit negative stereotypes of smokers operate as 'smoking-promoting messages' in the context of our controlled laboratory investigation.

One limitation to the present research is the question of whether stereotype threat is indeed the mechanism that accounts for the increase in smoking in the experimental condition. The present data suggest strongly that this is the case. First, the stereotype threat manipulation used in the present research is a standard manipulation that has been used for more than two decades of stereotype threat research. In contrast, the thought list task revealed no

significant differences between the two groups on negative emotions. A potential alternative hypothesis is reactance in response to the stereotype threat induction [45]. However, this is unlikely, as reactance would predict behavior that is inconsistent with the stereotype [46,47], such as waiting longer to smoke. Lastly, it is important to acknowledge that stereotype threat may operate differently in different domains, as null effects of threat conditions on performance outcomes have also been documented in the literature [48–50].

The present study focused on non-treatment-seeking smokers because this is the target audience for smoking cessation campaigns. Consistent with some research on smoking stigmatization and smoking initiation in nonsmokers [51], stereotype threat theory and research suggests that campaigns that stigmatize smoking may be effective for the prevention of smoking initiation in nonsmokers. That is, because non-smokers cannot yet be categorized as smokers, the stereotypes are not yet applicable to them; thus, these messages should not create stereotype threat [15]. It would also be interesting to examine the effects of campaigns that stigmatize smoking on treatmentseeking smokers. Stereotype threat research suggests that these campaigns would be particularly harmful-stereotype threat is most harmful for those who are most invested in the negatively stereotyped domain [15].

In conclusion, drawing on stereotype threat theory, we predicted and found that stigmatizing smoking in a community sample of adult non-treatment-seeking smokers led these smokers to smoke more quickly in a smoking lapse task compared to a control condition with no salient

stigma. The translational and innovative approach undertaken in this study consists of merging stereotype threat research with behavioral pharmacology. To that end, it is critical to recognize that the behavioral pharmacology paradigm use herein, namely the smoking lapse task, has been widely subjected to testing the effects of smoking interventions, including pharmacotherapies [52,53] and alcohol [54]. Therefore, the ability of a very brief stereotype threat manipulation to alter smoking behavior in the laboratory, over and above baseline performance, is noteworthy and speaks to the robustness of the stereotype threat phenomenon. In conclusion, these findings suggest that a better understanding of the psychology behind persuasive health messages may inform smoking public health efforts.

#### **Declaration of interests**

None.

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