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Predicting Relapse Back to Smoking: Contrasting Affective and Physical Models of Dependence

Susan L. Kenford
Xavier University

Stevens S. Smith
University of Wisconsin Medical School

David W. Wetter
University of Texas at Houston

Douglas E. Jorenby and Michael C. Fiore
University of Wisconsin Medical School

Timothy B. Baker
University of Wisconsin—Madison

Traditional models of *physical* dependence suggest that nicotine dependence should be reflected by the extent of drug exposure (e.g., smoking rate) and by evidence of physiological adaptation (e.g., withdrawal severity). An *affective* model suggests that nicotine dependence should be related to an individual's tendency to experience negative affect and expectations that nicotine use would ameliorate such affect. This research investigated the ability of these 2 models to predict relapse back to smoking at 6 months postquit. Logistic regression models were developed and tested in 505 heavy smokers participating in nicotine patch clinical trials. Results supported both models, but the most potent predictor of outcome was postquit negative affect, which accounted for much of the predictive validity of traditional measures of nicotine dependence. Affective reactivity appears to be a core constituent of dependence.

Each year over 17 million smokers undertake a serious attempt to quit smoking, but only about 1.3 million succeed and sustain abstinence (Hatziandreu et al., 1990). Many explanations have been offered to account for smokers' failure to attain and sustain abstinence, including insufficient motivation, low self-efficacy, inadequate skills, high nicotine dependence, inadequate social support, and so on. Indeed, numerous variables have been correlated with cessation outcomes (e.g., U.S. Department of Health and Human Services, 1996). Unfortunately, the identification of diverse outcome correlates has not increased markedly our understanding of the determinants of cessation failure, nor has it elucidated the nature of nicotine dependence.

One limitation of much prior research is that predictor variables are not selected on the basis of well-defined theories or models. Consequently, variables are typically tested in isolation or in a hit-or-miss manner. Because of this, investigators may have ignored important dimensions of theoretically relevant domains, and results do not lead programmatically to increased understanding of relapse or dependence. This strategy does not yield a cohesive theoretical foundation and may result in important domains or constructs not being assessed. Moreover, even when formal prediction models are created, they are rarely directly compared with one another. Therefore, it is impossible to ascertain which constructs or models are superior and whether two constructs or models yield additive or redundant information. In such cases, identification of significant predictors may reflect statistical power more than discriminative validity. Additionally, in the rare instances when specified predictive models are evaluated, they typically are not created through formal model-building procedures, procedures that allow for standardization of model development and evaluation.

The present research was designed to address some of these limitations. First, two contrasting theoretical models of nicotine dependence were specified a priori—the *physical dependence* model and *affect regulation* model—and relevant indices were selected. Next, measures of the core dimensions of the two models were incorporated into a set of double-blind, placebo-controlled nicotine patch clinical trials that consisted of 632 smokers in total. Only variables derived from the two theoretical models were used

Susan L. Kenford, Department of Psychology, Xavier University; Stevens S. Smith, Douglas E. Jorenby, and Michael C. Fiore, Department of Medicine and Center for Tobacco Research and Intervention, University of Wisconsin Medical School; David W. Wetter, M. D. Anderson Cancer Center, University of Texas at Houston; Timothy B. Baker, Department of Psychology, University of Wisconsin—Madison.

Preliminary analyses using these data were reported by Susan L. Kenford in her dissertation and in Piasecki, Kenford, Smith, Fiore, and Baker (1997). That research used a split sample and not the full sample as was used in the present research. In addition, the present investigation contains additional analyses and validity checks. Other publications from the data set include Fiore et al. (1994), Hurt et al. (1994), Kenford et al. (1994), Wetter et al. (1994), and Wetter et al. (1999).

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Correspondence concerning this article should be addressed to Susan L. Kenford, Department of Psychology, Xavier University, Elet Hall, 3800 Victory Parkway, Cincinnati, Ohio 45207-6511. E-mail: kenford@xavier.xu.edu

in model development (other than control variables). Formal model-building procedures proposed by Hosmer and Lemeshow (1989) were used to create the models. Two types of models for each theoretical category were built: (a) full multivariate models containing all theoretically specified predictors and (b) parsimonious models, which were the best-fitting models, containing only significant elements. As a final analytical step, variables from each model were systematically incorporated into the alternative model to determine the uniqueness or redundancy of the two models. The resultant models consist of predictors that clinicians can use to predict relapse vulnerability.

Physical Dependence Model

"Dependence" is often invoked to explain motivation to use drug and the difficulty in sustaining abstinence (Fagerstrom, 1978; Hughes, Gust, Skoog, Keenan, & Fenwick, 1991; Stitzer & Gross, 1988; West, Hajek, & Belcher, 1989). However, there is little agreement as to its definition, its cardinal manifestations, or its underlying processes. Despite variation in the definition of dependence, there is general agreement that an inability to achieve sustained abstinence from drug use is a central feature.

Models of physical dependence derive from traditional pharmacologic theories that hold that repeated drug administration leads to neuroadaptation, which leads to both tolerance and increased, or compulsive, self-administration (e.g., Goldstein, 1972; Mello, 1978). Models of physical dependence typically posit that dependent organisms take large amounts of drug not only because of tolerance but also to avoid or escape aversive withdrawal symptoms. This is a recursive process because high levels of drug self-administration result in more severe withdrawal (e.g., Cannon, Baker, Berman, & Atkinson, 1974; Goldstein, 1972). Therefore, models of physical dependence assert that the amount of drug exposure reflects susceptibility to the drug withdrawal syndrome and that both the amount of drug self-administration and severity of withdrawal index drug dependence.

Models of physical dependence have implicitly guided attempts to assess individual differences in level of drug motivation and smoking relapse vulnerability. Such "modal models" of dependence have been used to set, or investigate, nicotine replacement dosing regimens (e.g., Dale et al., 1995; Niaura, Goldstein, & Abrams, 1994) and to predict relapse risk (Killen, Fortmann, Kraemer, Varady, & Newman, 1992). This research has generated a core set of dependence measures that we have classified into three different dependence domains: *withdrawal severity, drug exposure* (amount and level of drug self-administration), and *pattern of compulsive use*.

A relatively small group of measures has been used to assess the core elements of nicotine dependence. Withdrawal severity has been assessed through well-validated scales that tap subjectively appraised withdrawal symptoms (e.g., Hughes & Hatsukami, 1986; Jorenby, Hatsukami, Smith, & Fiore, 1996). Drug exposure has been assessed through self-report of smoking rate and pattern and through biochemical indices of nicotine exposure, for example, blood nicotine/cotinine levels and expiratory (breath) carbon monoxide (CO; see Henningfield, 1995). Compulsive use has been assessed by means of self-report with items from measures such as the Fagerstorm Tolerance Questionnaire (FTQ) or the Fagerstrom Test of Nicotine Dependence (FTND; e.g., Heatherton, Kozlowski, Frecker, & Fagerstrom, 1991; Kozlowski, Porter, Orleans, Pope, & Heatherton, 1994).

Numerous studies have shown that the above dependence measures can predict cessation success (e.g., Gritz, Carr, & Marcus, 1991; Hughes et al., 1991; Killen et al., 1992; West et al., 1989; West et al., 1984). In addition, such measures appear to identify those smokers requiring high-dose nicotine gum pharmacotherapy (Fiore, Jorenby, & Baker, 1997; Henningfield, 1995). Some studies have failed to confirm the predictive validity of these widely used dependence measures (Hurt et al., 1994; Kenford et al., 1994; Kozlowski et al., 1994; Nides et al., 1995). However, the supportive evidence is sufficiently strong so that the measures are frequently recommended as dependence indicators or as predictors of cessation success (Fiore et al., 1997; Foulds, 1996; Kozlowski et al., 1994; Lichtensein & Glasgow, 1992). Moreover, because of their reputed validity, the use of some of these measures, such as the FTQ, have become de rigueur in smoking cessation research (Kinnunen, Doherty, Militell, & Garvey, 1996; Lerman et al., 1996; Rabios & Haaga, 1997). Finally, professional and scientific panels have strongly endorsed these measures. The American Psychiatric Association (1996) Work Group on Nicotine Dependence concluded that the FTQ/FTND is a reliable and valid predictor of cessation success. Moreover, this group equated nicotine dependence with exceeding a threshold score on the FTQ. The Agency for Health Care Policy and Research smoking cessation clinical practice guideline (U.S. Department of Health and Human Services, 1996) also endorses these measures as valid indices of nicotine dependence and predictors of cessation success.

Despite the general consensus that measures of drug exposure or compulsive use are valid measures of dependence and accurately predict success, vital questions remain about their status and meaning. For instance, it is unclear that the measures reflect a unitary phenomenon or that the measures cohere. In addition, because the various measures are typically not used or tested en masse (e.g., Abelin et al., 1989; Fagerstrom & Schneider, 1989; Pomerleau, Fertig, & Shanahan, 1983; Sutherland et al., 1992), little is known about whether the different dependence measures make unique contributions to the prediction of outcome, what their relative validities are, and whether they yield interactive effects. Finally, because prediction models based on the concept of physical dependence have not been contrasted with distinct alternative models, it is unknown whether physical dependence constructs provide any special or unique information regarding quitting success.

The physical dependence model evaluated in the present research was designed to address major elements of the modal model of dependence. Nicotine exposure was assessed using self-reported cigarette consumption and three biochemical indices: serum nicotine level, cotinine level, and breath CO. Withdrawal severity was assessed with the well-validated Minnesota Withdrawal Scale (Hughes & Hatsukami, 1986), and compulsive use was assessed with the FTQ.

Affect Regulation Model

Various models of drug motivation hold that affective states serve as vital control stimuli for drug self-administration and that drug motivation is indexed by activity in affective processing systems (Baker, Morse, & Sherman, 1987; Niaura et al., 1988). Although theoretical models implicate affect in drug motivation,

they do not suggest a particular affective model of relapse vulnerability. However, a variety of findings suggest promising model elements. Epidemiologic studies investigating the role of negative affect in smoking and nicotine dependence have found a consistent relationship between depression and smoking. Several epidemiologic studies (Anda et al., 1990; Glassman et al., 1990) have found that smokers with a history of depression are more likely to fail in any given quit attempt. Others (Breslau, Peterson, Schultz, Chilcoat, & Andreski, 1998) have shown that individuals with depression are more likely to progress over time to daily smoking. Moreover, antidepressant drugs such as bupropion and nortryptilene are effective smoking cessation aids (Hall et al., 1998; Jorenby et al., 1999).

Recent causal modeling research and theory implicate particular affective person factors in substance abuse severity. Implicated variables include (a) history of experiencing severe negative affect, in particular, depression (Anda et al., 1990; Breslau et al., 1998; Covey, Glassman, & Statner, 1997; Glassman et al., 1990); (b) high levels of stress (e.g., Cooper, Russell, & George, 1988, Cooper, Russell, Skinner, Frone, & Mudar, 1992; Wills, DuHammel, & Vaccaro, 1995); (c) coping style (Cooper et al., 1988; Cooper et al., 1992; Rabios & Haaga, 1997); and (d) expectations about the positive effects of drug use (e.g., Brandon & Baker, 1991; Brown, Goldman, Inn, & Anderson, 1980; Wetter et al., 1994). In addition to these variables, negative affect per se has been found to predict severity of drug use and the motivation to use drug (e.g., Baker et al., 1987; Brandon, Tiffany, Obremski, & Baker, 1990; Payne, Schare, Levis, & Colletti, 1991; Shiffman, 1986; Stapleton et al., 1995; Zinser, Baker, Sherman, & Cannon, 1992).

The aforementioned dimensions of negative affect were incorporated into a relapse model in which postcessation negative affect constitutes the primary response channel reflecting drug dependence (Baker et al., 1987). According to this model, negative affect reflects the loss of reinforcement when drug becomes unavailable, just as negative affect or "mourning" reflects the loss of reinforcement when a person becomes unavailable (i.e., negative affect is a "read-out" of withdrawal processing). In theory, postcessation negative affect should predict likelihood of relapse to drug use because it reflects precessation drug reinforcement and because its occurrence or severity signals the availability of negative reinforcement available through drug relapse (i.e., reinforcement due to avoidance or escape of negative affect). Either factor could account for an association between negative affect and relapse.

The criteria for inclusion of a variable in the affect model then are whether, in theory, the variable (a) measures or predicts severe negative affect upon cessation or (b) increases the likelihood that negative affect, if it occurs, will lead to relapse. In the present study, the affect model includes the following constructs and measures. *Negative affect* was assessed with the negative affect items of the Positive and Negative Affect Scale (PANAS; Watson, Clark, & Tellegen, 1988); *coping style* was assessed with the Three Factor Coping Scale (Rohde, Lewinsohn, Tilson, & Seeley, 1990); *stress* was assessed with the Perceived Stress Scale (PSS; Cohen, Kamarck, & Mermelstein, 1983), *depression history* was assessed with a single item shown to predict relapse in prior research (Brandon et al., 1990); and *expectations that smoking would reduce negative affect* were assessed with the Negative Reinforcement Scale of the Smoking Consequences Questionnaire (Copeland, Brandon, & Quinn, 1995; Wetter et al., 1994). In theory, the likelihood that a smoker would experience negative affect after cessation would be predicted by measures of negative affect, inadequate coping style, high levels of stress, and a positive history of depression. Both inadequate coping style and negative reinforcement expectations would predict drug relapse in response to negative affect.

Goals of the Research

The present study was designed to test theoretically based competing models of nicotine dependence as indexed by relapse to smoking. Neither the physical dependence model nor the affect regulation model contains variables that assess situational or contextual factors. The intent was to create and test models of *individual differences.* Certainly some contextual factors such as presence of smokers in the home or social support predict outcome (e.g., Clark, Kviz, Crittenden, & Warnecke, 1998; Gunn, 1983; Nides et al., 1995; Willemsen, De Heis, Van Breukelen, & Oldenburg, 1996). However, the goal of this research was not to predict outcome, per se, but rather to index theoretically derived individual differences associated with outcome. Thus, both models really constitute dependence models: one reflects conventional wisdom and comprises variables suggested by physical dependence models; the other is based on affective models of dependence and drug motivation and comprises affective processing variables. By testing two carefully derived models within the same data set, we can gain information about the relative strength and weaknesses of each. Additionally, by creating a third, integrated model comprising predictors from both classes, we can determine not only how the models perform in isolation but also if integration of concepts across the two classes improves our prediction and understanding of dependence.

Method

Data Set

The data set used in this study was collected as part of three nicotine patch clinical trials reported elsewhere (Fiore et al., 1994; Hurt et al., 1994; Kenford et al., 1994; Piasecki, Kenford, Smith, Fiore, & Baker, 1997; Wetter et al., 1994; Wetter et al., 1999). The total sample ($N = 632$) comprised 280 participants from four sites who took part in the first trial, 112 participants from a single site who took part in a second trial, and 240 participants from three sites who took part in a third trial. All trials were randomized, double-blind, placebo-controlled trials. The first trial was conducted at four university sites and consisted of 8 weeks of 22-mg nicotine patch therapy (or placebo patches) and weekly group counseling meetings. The second trial was conducted at a single university site and consisted of 4 weeks of 22-mg nicotine patch therapy (or placebo) followed by 2 weeks of 11-mg nicotine patch therapy (or placebo) along with 8 weeks of brief individual counseling. The third trial was conducted through the Mayo Clinic at three sites (Minnesota, Arizona, and Florida) and consisted of 8 weeks of 22-mg nicotine patch (or placebo) therapy along with 8 weeks of brief individual counseling.

Recruitment and Inclusion Criteria

Participants were recruited through media announcements concerning participation in a clinical trial of a transdermal nicotine patch. Inclusion criteria consisted of being between 21 and 65 years of age, having a history of smoking a minimum of 15 cigarettes per day for the past year, having

an expired CO level > 10 parts per million (ppm), and being motivated to quit smoking. Exclusion criteria consisted of presence of cardiovascular disease, pregnancy or lactation, regular use of psychotropic drugs, alcohol or drug abuse, chronic dermatologic disorders, use of any experimental medication within the past 30 days, and current symptomatic psychiatric disorder. Inclusion and exclusion criteria were assessed during a screening visit, and qualified individuals were scheduled for an electrocardiogram and brief medical examination.

Counseling Interventions

Group smoking cessation counseling. Groups consisted of 8–12 participants who were heterogeneous in regards to patch status. Groups met once per week for 60 ± 10 min and were led by experienced facilitators (psychologists or advanced psychology graduate students). In each session, participants reported their smoking status and any problems or concerns from the previous week. The facilitators, working from a standardized treatment manual, conveyed relevant information and introduced topics in the context of discussions, providing didactic as well as supportive treatment. Facilitators encouraged group members to anticipate urges and problem situations and to then generate appropriate coping strategies or plans.

Individual smoking cessation counseling. Individual counseling was delivered by registered nurses, psychologists, or advanced psychology graduate students meeting with individual participants for 15–20 min once per week. At each meeting, patch use was assessed, and standardized topics were introduced and discussed. This treatment was intended to resemble the type of adjuvant treatment that a smoker might receive from a health care clinician following the National Cancer Institute's "How to Help Your Patient Quit Bmoking" program.

Outcome Measure: Six-Month Abstinence

Abstinence was assessed at weekly intervals throughout the active study period and at 3- and 6-month follow-up points. At all points, abstinence was a point-prevalence measure defined as no smoking at all during the previous 7-day interval. All analyses were conducted on an intent-to-treat basis; participants without biochemical verification of abstinence (expired CO of < 10 ppm), study dropouts, and those lost to follow-up were classified as smokers.

Implicit Dependence Model Predictors

Predictor measures were selected a priori on a theoretical basis during the design of the clinical trials to address psychological aspects of relapse.

Nicotine exposure variables. The nicotine exposure variables consist of the following:

1. The number of precessation cigarettes smoked per day was selected because it provides a direct self-report index of self-administration. Traditional models of dependence posit increased levels of drug self-administration as a function of tolerance.

2. Prequit expired CO is a noninvasive biochemical assessment of smoke inhalation. Expired CO readings for the average nonsmoker are between 1 and 5 ppm. Smokers typically produce breath samples that contain over 10 ppm CO (Cocores, 1993).

3. Prequit blood nicotine level is a second biochemical marker of self-administration. The level of nicotine that is found within smokers' blood is a reflection of their unique administration patterns and metabolic rates (Henningfield & Woodson, 1989). Smokers vary not only in the number of cigarettes smoked but also in the amount of nicotine obtained from any given cigarette. Therefore, examination of blood nicotine level provides a more precise estimate of nicotine self-administration among heavy, regular smokers than do more gross measures such as smoking rate. Nicotine is known to have a half-life of approximately 2 hr (range = 1–4 hr).

4. Prequit blood cotinine level is a third biochemical marker of self-administration. Cotinine is the primary metabolite of nicotine. Cotinine has a half-life of approximately 16–20 hr and is therefore more stable than nicotine. Additionally, cotinine reflects the rate and efficiency of nicotine metabolism by the individual (Jacob, Benowitz, & Shulgin, 1988).

Multiple biochemical markers of dependence were included to sample adequately the range of individual differences in self-administration. While interrelated, these biological markers have different sensitivities as a function of different half-lives and differential reflections of metabolism, and they reflect both the behavior of self-administration and the dosing consequences of self-administration.

Compulsive use. The FTQ (Fagerstrom, 1978) is an eight-item, paper-and-pencil measure designed to assess self-administration, compulsive use, and perceived need for nicotine. This measure (including the FTND) is the single most widely used measure of nicotine dependence.

Withdrawal severity. The Minnesota Withdrawal Index (Hughes & Hatsukami, 1986) is a checklist format, paper-and-pencil measure that is completed daily for 1 week prequit and 8 weeks postquit as a measure of withdrawal severity. This measure contains hallmark symptoms of nicotine withdrawal (i.e., craving, anger, difficulty concentrating, anxiety, depression, irritability, increased hunger, and insomnia) that are rated on a scale of 0 (*not present*) to 4 (*severe*). These daily scores are summed and averaged to create a composite weekly score for each symptom. This measure has been shown to be valid and reliable (Hughes & Hatsukami, 1986; Hughes et al., 1984).

Affect Regulation Model Predictors

Predictor measures were selected a priori on a theoretical basis during the design of the clinical trials to address psychological aspects of relapse.

1. The negative affect items of the PANAS scale were used to assess negative affect (Watson et al., 1988). This self-report, paper-and-pencil, adjective checklist assesses both positive and negative affective states. Participants rate each adjective on a scale of 1–5 for how true it was for them over a specified time period (i.e., past week, past month). Scores are then summed and averaged to create the scale scores. The negative affect scale (N-PANAS) assesses general dimensions of negativity and provides a broader index of negative affect than would a measure of depression or anxiety per se. This measure is short and simple and has been shown to have good psychometric properties (Watson et al., 1988). Participants completed a "week" version of this measure prequit and weekly for the first 8 weeks postquit.

2. Subjective cumulative current stress was assessed using the PSS (Cohen et al., 1983). This is a self-report, paper-and-pencil, state measure of the degree and intensity of subjectively rated stress. Participants rate 14 statements on a scale of 1–5 on how they apply to the past week. Items are summed and averaged to create the scale score. This measure has been shown to be a valid index of subjective stress and to have adequate psychometric properties (Cohen et al., 1983). Participants completed the PSS prequit and weekly for the first 8 weeks postquit.

3. History of depression was assessed with an item asking "Have you ever had a period of time where you felt down every day, most of the day, for at least 2 weeks?" This was asked during study screening. Presence of current major depression was an exclusion criterion, but history of depression was not. Single items assessing depression history have been shown to be valid predictors of relapse likelihood in previous research (Brandon et al., 1990).

4. Smoking expectancies were assessed using the Smoking Consequences Questionnaire (Brandon & Baker, 1991). Items reflecting potential consequences of smoking are rated on a scale of 0–9 for likelihood of occurrence. This is a self-report, paper-and-pencil scale that has been found to have four major factors: Positive Reinforcement, Negative Reinforce-

ment, Appetite/Weight Control, and Negative Consequences. Of interest to this study is the Negative Reinforcement scale. Within the affect regulation model, the extent to which drug use is viewed as a means of diminishing a dysphoric mood state is of key importance. The entire measure and constituent factors have been shown to have good psychometric properties and to be related to postcessation affect, withdrawal, and relapse (Wetter et al., 1994).

5. Coping style was assessed using the Three Factor Coping Scale (Rohde et al., 1990), a self-report, paper-and-pencil measure that elicits rating of coping strategies on a scale of 0–7 with respect to likelihood of strategy use; these ratings are summed and averaged to create a scale score. This measure has three major factors: Solace Seeking, Active Cognitive, and Ineffective Escapism. The scale has been shown to have adequate psychometric properties (Rohde et al., 1990).

Many of the predictors (e.g., smoking rate, expired CO, blood nicotine, blood cotinine, FTQ, depression history, PANAS, Three Factor Coping Scale, Smoking Consequences Questionnaire, and baseline withdrawal) were collected prequit at a medical screening visit that occurred within a 2-week window of the participant's designated quit date. Additional repeated assessments were collected at each clinic visit as indicated earlier.

Analyses

Participants' attrition at each site was examined to evaluate the poolability of the data sets. Two of the eight sites were dropped because of attrition that exceeded 50% over the treatment period. These exclusions decreased sample size from 632 to 505. Other analyses comparing demographic and smoking variables across sites revealed isolated site differences. However, subsequent correlational analyses revealed that despite differences in levels of these predictors across some sites, predictor–criterion relations were similar across sites. Therefore, data were pooled across the remaining six sites. In addition, subsequent to developing the competing models for each theoretical approach, site was included in the final models, and no significant site effects were found.

Predictor variables were examined for outliers, nonnormalcy, and multicollinearity. Outliers (3 *SD* above or below a variable's mean) were recoded to the most extreme value above or below the mean that fell within 3 *SD* of the mean (Tabachnick & Fidell, 1989). Because logistic regression was used for model testing, linearity of the logit for continuous predictors was assessed (Hosmer & Lemeshow, 1989). Two predictors, negative affect at Week 1 postquit and negative reinforcement expectancies, were not linear in the logit and, as a result, were dichotomized. Analyses revealed multicollinearity (Menard, 1995) within the following pairs of predictors: withdrawal at Week 0 and Week 1, perceived stress at Week 0 and Week 1, blood nicotine level, and blood cotinine level. As a result, only withdrawal at Week 1, blood nicotine level, and perceived stress at Week 1 were retained as predictors. These predictors were retained because of their higher point-biserial relations with the criterion. For purposes of model testing, only participants with complete data ($n = 386$) were used to allow for direct comparison across models. No missing data were replaced.

Because of the dichotomous nature of the dependent measure (smoking status at 6-month follow-up), logistic regression was used to test models. For each theoretical approach, full multivariate models that included all the predictors specific to either the physical dependence model or the affect regulation model were tested. All models included patch status (active or placebo) and gender as control variables. All possible two-way interactions between all predictor and control variables were tested, and significant interactions were retained. The Hosmer and Lemeshow (1989) goodness-of-fit ($\hat{C}$) statistic was computed to assess model fit; a conservative value of $p > .20$ indicated adequate fit. In addition, integrated models were tested that included entry of all the predictor variables from both theories. First, all control variables were entered, then the entire set of variables from each model was entered by turn in separate analyses (i.e., all the physical dependence variables followed by the affect regulation variables; then, in a subsequent analysis, all the affect regulation variables followed by the physical dependence variables). Evaluation of the increment in model chi-square was used to indicate whether improved model fit was achieved when the predictors from the alternative theory were included. Next, parsimonious best-fitting models within each theoretical category were constructed using Hosmer and Lemeshow's (1989) model-building techniques. After control variables were entered, each predictor was examined for its relation to outcome and then sequentially removed until only significant predictors remained in the model. Finally, integrated parsimonious models were developed using the same strategy as with the full models. Integrated models were built that incorporated the full complement of predictors from the competing model. Again, increment in model chi-square was examined to determine if improved model fit was attained with the addition of predictors from the competing model.

Results

Theoretical Full Multivariate Models

Physical dependence model. Tests indicated no significant interactions between predictor variables (including control variables). The full main effects model obtained a $\hat{C}$ value of 6.72, with 8 degrees of freedom ($p = .5666$), suggesting an acceptable fit. Review of the model indicated that only two model elements, blood nicotine and Week 1 withdrawal, significantly predicted 6-month outcome. Additionally, the control variable, gender, predicted abstinence rates (see Table 1).

Affect regulation model. For the affect regulation model, there was one significant interaction: Negative Reinforcement × Ineffective Escapist Coping. In addition, significant main effects included postcessation negative affect (N-PANAS), depression history, and negative reinforcement expectancies from smoking, as assessed with the Smoking Consequences Questionnaire. The full main effects plus interaction model obtained a $\hat{C}$ value of 3.07, with 8 degrees of freedom ($p = .9293$), indicating good model fit (see Table 2).

Integrated models. Two series of logistic regression analyses were conducted to assess integrated models. In the first analysis, the affect regulation set of variables was added to the logistic model after variables belonging to the physical dependence model. Results from this analysis indicated that the physical dependence variables yielded $\Delta\chi^2(5, N = 386) = 14.24$, $p < .015$. When the affect regulation variables were added to the physical dependence

Table 1
Full Multivariate Implicit Dependence Model Predicting 6-Month Abstinence Data

Variable	*B*	*SE*	Wald	*p*	Odds ratio
Patch status	0.501	.291	2.958	.085	1.65
Gender	0.989	.279	12.581	.000	2.69
Smoking rate	0.002	.016	0.008	.927	1.00
Expired CO	−0.004	.012	0.097	.756	1.00
Baseline nicotine	0.031	.088	0.190	.068	1.03
Fagerstrom score	0.039	.088	0.190	.663	1.04
Withdrawal 1	0.568	.198	8.220	.004	1.76

Note. Baseline nicotine = prequit serum cotinine levels; CO = carbon monoxide; Withdrawal 1 = withdrawal ratings for the first postwithdrawal week.

Table 2
Full Multivariate Affect Regulation Model Predicting 6-Month Abstinence Data

Variable	*B*	*SE*	Wald	*p*	Odds ratio
Patch status	0.493	.305	2.605	.107	1.640
Gender	0.815	.305	7.136	.007	2.260
History of depression	1.110	.475	5.479	.019	3.038
Negative Affect 0	−0.229	.309	0.871	.351	0.749
Negative Affect 1	1.320	.316	17.350	.000	3.740
Perceived Stress 1	0.363	.272	1.781	.182	0.144
Negative reinforcement	2.690	.872	9.529	.002	14.760
Coping 1	−0.183	.220	0.692	.406	0.833
Coping 2	0.247	.244	1.023	.312	1.280
Coping 3	0.235	.168	1.967	.161	1.265
Coping 2 × Neg Ref	−0.882	.368	5.741	.017	0.414

Note. Negative Affect 0 = negative affect at baseline; Negative Affect 1 = Negative affect 1 week postquit; Perceived Stress 1 = perceived stress 1 week postquit; Negative reinforcement = expectations of negative reinforcement; Coping 1 = coping through cognitive control; Coping 2 = coping through ineffective escapism; Coping 3 = coping through solace seeking; Coping 2 × Neg Ref = interaction of ineffective coping and negative reinforcement expectations.

variables, they yielded $\Delta\chi^2(9, N = 386) = 46.16, p < .000$. In the second analysis, the physical dependence variables were added after the affect regulation variables results indicated that the affect regulation variables yielded $\Delta\chi^2(9, N = 386) = 55.85, p < .000$, and the physical dependence variables obtained $\Delta\chi^2(5, N = 386) = 4.55, p = .473$. These results indicate that the affect regulation model accounts for unique predictive validity beyond that identified by the physical dependence model, whereas the predictive validity of the physical dependence model is comprised by the predictive validity of the affect regulation model (see Table 3).

Parsimonious Models

Parsimonious models, eliminating all nonsignificant predictors, were built within each category.

Physical dependence model. For physical dependence, the resulting model comprised nicotine at Week 0 and withdrawal index at Week 1 (see Table 4). This model obtained a $\hat{C}$ of 9.77, with 8 degrees of freedom ($p = .281$), indicating adequate fit.

Affect regulation model. The final parsimonious affect regulation model comprised history of depression, negative affect Week 1, negative reinforcement expectancies, ineffective escapist coping, and an interaction of Ineffective Escapist Coping × Negative Reinforcement Expectancies (see Table 5). The model obtained a $\hat{C}$ of 1.595, with 8 degrees of freedom ($p = .991$), indicating good fit.

Table 3
Comparison of Two Full Models

Step	Variables entered	*G*	*p*
1	Control variables (patch status; gender)	20.41	.000
2	Implicit dependence variables	14.25	.0141
3	Affect regulation variables	46.16	.000
1	Control variables (patch status; gender)	20.41	.000
2	Affect regulation variables	55.85	.000
3	Implicit dependence variables	4.55	.473

Table 4
Parsimonious Implicit Dependence Model

Variable	*B*	*SE*	Wald	*p*	Odds ratio
Patch status	0.495	.290	2.93	.088	1.64
Gender	0.985	.275	12.82	.000	2.68
Baseline nicotine	0.030	.015	3.82	.051	1.03
Withdrawal 1	0.585	.195	9.01	.003	1.80

Note. Baseline nicotine = prequit serum cotinine levels; Withdrawal 1 = withdrawal ratings for the first postwithdrawal week.

Integrated parsimonious models. Once the two most parsimonious models were identified, analyses were conducted to build an integrated parsimonious model. The same procedure was used as with the full models. Results indicated that when entered first, the physical dependence predictors accounted for $\Delta\chi^2(2, N = 386) = 13.93, p < .001$, and when entered second the affect regulation predictors accounted for $\Delta\chi^2(5, N = 386) = 41.78, p < .000$. When the affect regulation predictors were entered first, they obtained $\Delta\chi^2(5, N = 386) = 51.84, p < .000$, and when the physical dependence variables were entered second, they accounted for $\Delta\chi^2(2, N = 386) = 3.88, p = .144$. As in the full model, the affect regulation variables accounted for predictive validity above and beyond that identified by the physical dependence model, whereas the physical dependence model did not possess incremental validity once affect regulation predictors were entered (see Table 6).

Three-Month Outcomes

The parsimonious models within each theoretical class were then tested using abstinence at 3 months as the outcome variable. This was done to determine if similar results would be obtained across different posttreatment intervals.

Physical dependence model. The physical dependence model obtained a $\hat{C}$ value of 9.918, with 8 degrees of freedom ($p = .271$), indicating adequate model fit. Only withdrawal at Week 1 remained a significant predictor of relapse, obtaining Wald $\chi^2 = 4.97, p < .027$.

Table 5
Parsimonious Affect Regulation Model

Variable	*B*	*SE*	Wald	*p*	Odds ratio
Patch status	0.471	.300	2.480	.116	1.60
Gender	0.918	.293	9.815	.002	2.50
History of depression	1.210	.463	6.867	.009	3.37
Negative Affect 1	1.250	.306	16.780	.000	3.50
Negative reinforcement	2.620	.845	9.599	.002	13.71
Coping 2	0.298	.232	1.650	.199	1.35
Coping 2 × Neg Ref	−0.868	.364	5.700	.017	1.35

Note. Negative Affect 1 = negative affect 1 week postquit; Negative reinforcement = expectations of negative reinforcement; Coping 2 = coping through ineffective escapism; Coping 2 × Neg Ref = interaction of ineffective coping and negative reinforcement expectations.

Table 6
Comparison of Parsimonious Models

Step	Variables entered	*G*	*p*
1	Control variables (patch status; gender)	20.41	.000
2	Implicit dependence variables	13.93	.001
3	Affect regulation variables	41.78	.000
1	Control variables (patch status; gender)	20.41	.000
2	Affect regulation variables	51.84	.000
3	Implicit dependence variables	3.86	.473

Affect regulation model. The affect regulation model obtained a $\hat{C}$ value of 6.597, with 8 degrees of freedom ($p = .581$), indicating adequate model fit. Negative affect Week 1 (Wald $\chi^2 = 16.07$) and negative reinforcement expectancies (Wald $\chi^2 = 7.382$) remained significant predictors of relapse.

Integrated models. As with 6-month data, an integrated model was tested. Results revealed that when entered first, the physical dependence predictors accounted for $\Delta\chi^2(2, N = 386) = 8.217$, $p < .017$, and when entered second, the affect regulation predictors accounted for $\Delta\chi^2(5, N = 386) = 37.04$, $p < .000$. When the affect regulation predictors were entered first, they obtained $\Delta\chi^2(5, N = 386) = 41.23$, $p < .000$, and when the physical dependence predictors were entered second, they accounted for $\Delta\chi^2(2, N = 386) = 3.94$, $p = .139$. As with 6-month outcome, the affect regulation variables accounted for predictive validity above and beyond that identified by the physical dependence model, whereas the physical dependence model did not possess incremental validity once affect regulation predictors were entered.

Model Classification Accuracy

To address the issue of the classification or predictive accuracy of the two parsimonious models, we computed receiver operating characteristic (ROC) analyses that yielded measures of sensitivity (true-positive rate), 1-specificity (false-positive rate), and area under the ROC curve (AUC) for each model. Models with good predictive accuracy are characterized by a high true-positive rate and a low false-positive rate.

Table 7 provides a comparison of the two models' false-positive rates for sensitivity values ranging from .1 to .9. As shown in Table 7, false-positive rates are consistently higher for the physical dependence model as compared with the values for the affect regulation model.

Table 7
Sensitivity and 1-Specificity for the Negative Affect and Nicotine Dependence Models

	1-specificity (false-positive rate)	
Sensitivity	Negative affect model	Nicotine dependence model
.1	.01	.03
.2	.01	.05
.3	.04	.08
.4	.07	.23
.5	.11	.27
.6	.20	.34
.7	.28	.41
.8	.38	.50
.9	.54	.62

ROC curves, representing the plot of sensitivity and 1 − specificity values, are shown in Figure 1 for the affect regulation model and for the physical dependence model. The steeper rise in the curve for the negative affect model compared with the curve for the physical dependence model is indicative of better discrimination or ability to classify correctly participants who are abstinent and those who will relapse. This result is also reflected in the AUC values for each model. AUC values range from 0 to 1, with higher values indicating better predictive power. The affect regulation model yielded an AUC of .79, whereas the physical dependence model yielded a value of .70. Thus, it appears that the affect regulation model has better ability to classify individuals in terms of long-term smoking status.

Discussion

This study is unique in that it compared theoretically derived alternative models of relapse vulnerability within the same data set and evaluated the two models with respect to model fit and their relative and incremental validities.

Basic Findings

Among the traditional measures of physical dependence, withdrawal severity was the best predictor of abstinence at 6 months. Other indices of dependence (e.g., FTQ, smoking rate, blood nicotine, and blood cotinine) are much more widely used in nicotine research as outcome predictors and as indicators of dependence (e.g., Abelin et al., 1989; Blondal, 1989; Foulds, 1996; Stapleton et al., 1995) and are more frequently recommended as valid dependence indicators (e.g., Foulds, 1996; Killen et al., 1992). However, the widely used FTQ was poorly related to outcome, and none of the other dependence measures performed well.

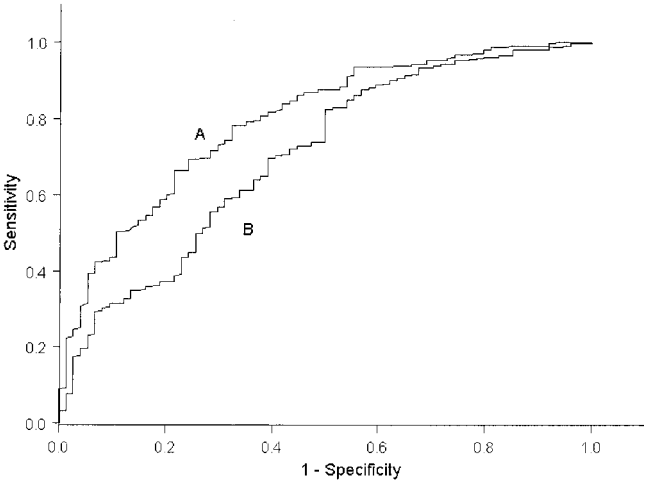

Figure 1. Receiver operating characteristic curves for the two models: Curve A = affect regulation model; Curve B = physical dependence model.

Tests of the affect regulation model supported the predictive validity of two affect variables: history of depression and negative affect experienced within the first week postquit. These variables had large odds ratios and significantly incremented model chi-square values. Moreover, goodness-of-fit statistics showed that these variables yielded good predictability of outcome across deciles of risk.

Significant components of each model were entered into logistic regression equations with variables from the alternative model. These analyses showed that the best component of the physical dependence model, withdrawal severity, failed to predict relapse if affective variables were in the model. Thus, within the constraints of the present prediction analyses, traditional markers of dependence did not increment the predictive validity of a model comprising two affective variables. Additionally, analyses examining each model's ability to classify long-term smoking status correctly indicated that the affect regulation model provided greater classification accuracy. Another finding of note was that numerous prequit affective measures (e.g., perceived stress, coping style, affect, and expectancies) provided no unique or incremental predictive information once history of depression and severity of postcessation negative affect were entered in the prediction model. In a serendipitous finding not targeted by this study, female smokers showed significantly greater risk of cessation failure than did male smokers (Perkins, 1996; Wetter et al., 1999). Moreover, the present results suggest that the gender differences in relapse cannot be explained directly by differences in physical dependence or affect, at least as they were assessed in this research.

Implications

Dependence. Countless recent studies have used measures of nicotine self-administration, self-reports of chronic compulsive nicotine use, or measures of withdrawal severity to assess nicotine dependence (e.g., Fagerstrom & Schneider, 1989; Foulds, 1996; U.S. Department of Health and Human Services, 1996). Indeed, these measures have been recommended as valid indicators of nicotine dependence (American Psychiatric Association, 1996; The Smoking Cessation Clinical Practice Guideline Panel and Staff, 1996). However, the present findings, as well as other recent data (Kenford et al., 1994; Taylor et al., 1996), argue for a reappraisal of the dependence model that has implicitly or explicitly guided much recent research.

First, none of the dependence indicators performed well in predicting cessation outcomes. This is damaging because one hallmark of dependence is the tendency to resume drug use despite desire, intention, and effort to quit. The present results suggest that the dependence indicator–construct complex is not performing in a manner consistent with theory.

As indicated earlier, different investigators claim that "dependence" predicts or explains cessation outcomes; yet these claims rest on studies that index dependence with such diverse measures as smoking rate, CO level, the FTQ, and so on. The present findings suggest that multiple factors may account for the predictive relations attributed to dependence. In the present context of modeling cessation outcomes, it was seen that the contribution of physical dependence variables was not unique. Rather, their capacity to order cessation outcomes was redundant with information contained in affective variables. Indeed, it appears that the extent that withdrawal symptomatology predicts relapse is largely due to the ability of withdrawal measures to tap affect (Piasecki et al., 2000). Further, many dependence indicators reflect drug self-administration patterns but are not theoretically or conceptually linked to explanatory mechanisms that could account for an impact on cessation outcome. An attractive hypothesis is that high levels of self-administration per se lead to severe withdrawal symptomatology. Unfortunately, much evidence regarding this assumption is negative (Fagerstrom & Schnieder, 1989; but see Piasecki, Fiore, & Baker, 1998). Therefore, dependence is currently used in a tautological manner and, thus, provides little or no insight into mechanisms responsible for important endpoints.

There is no doubt that dependence indicators such as precessation blood nicotine level and the FTQ can predict cessation outcomes (e.g., Fagerstrom & Schneider, 1989). Indeed, the present study found that blood nicotine predicted outcome. Such findings may be misleading, however, in that dependence measures have rarely been pitted against alternative, formal prediction models. Thus, data have not been available on the relative validity and uniqueness of predictive information.

One could argue that dependence measures would have performed better had the population under study comprised participants with a broader range of smoking histories. The present study involved smokers volunteering for a formal smoking cessation program. These smokers may be especially dependent on tobacco, and this might have restricted variance in the dependence measures and constrained relations with outcome. The relative worth of different motivational models can be determined with certainty only after research has been conducted across a wide range of populations. However, the nature of dependence, and its assessment, would require reevaluation if it were discovered that the construct could not account for important addiction outcomes among chronic, heavy users, especially if other constructs were able to do so.

The present research does not prove that traditional dependence measures lack utility. For example, some evidence suggests that some physical dependence measures can be useful for determining dose of nicotine replacement (e.g., Blondal, 1989; Tonnesen, Norregaard, Sawe, & Simonsen, 1993). In addition, the measures can sensitively reflect amount of smoke intake, which, no doubt, makes them useful in identifying the "smoker taxon." However, the present results along with other studies show that dependence measures are inconsistently related to the intransigent nature of smoking, they appear to have heterogeneous causal determinants, and they have not been strongly linked to motivational mechanisms. Thus, although these indicators identify the smoker taxon, neither theory nor empirical work has tied them tightly to mechanisms that can account for the inability to discontinue drug use or smoking.

Affect regulation. This research provides information about the role of affect in determining cessation outcomes. For instance, the results clearly suggest that it is negative affect *occasioned by abstinence* that especially predicts cessation outcomes, not necessarily chronic psychological distress (see Covey, Glassman, & Stetner, 1990). Also, the results suggest that affective variables powerfully predict outcomes across a wide range of smokers. It has been tempting to speculate that affective variables predict addiction outcomes only for subpopulations of smokers with comorbid affective illness (e.g., Glassman & Covey, 1996). However, post-

cessation negative affect predicted outcomes across the total sample of smokers, and its predictive validity was not dependent on moderation by another variable. Moreover, negative affect predicted outcomes in a linear manner across the measured range of affect. These observations suggest that negative affect resides at the core of the motivationally potent withdrawal syndrome.

The lack of incremental validity of the withdrawal measures calls into question whether the withdrawal and negative affect measures are, indeed, distinct. It is certainly the case that the underlying constructs are distinct. There are elements of the nicotine withdrawal syndrome that would not be incorporated into a typical measure of negative affect (e.g., bradycardia, hunger, and increased sleep fragmentation). However, the well-accepted withdrawal measure in this research may inadequately sample nonaffective variance in the nicotine withdrawal syndrome, and therefore, its use may have preordained a lack of incremental validity. More research is needed to determine whether nonaffective variance in withdrawal is meaningfully related to relapse.

Our view is that the negative affect and withdrawal measures are distinct (indeed, the measures were correlated only at .49 at postquit Week 1), largely because the withdrawal measure comprises a small number of nonaffective items. The lack of incremental validity of the withdrawal measure can be attributed, we believe, to the fact that only the affective constituents of the syndrome have motivational significance (e.g., Piasecki et al., 1997). In simple terms, a bradycardic, overweight, hyposomniacal smoker will not return to smoking if she or he is happy. The N-PANAS outpredicted Minnesota Withdrawal Scale because it (the N-PANAS) is a superior measure of negative affect. Therefore, our results do not cast doubts on the psychometric status of the withdrawal measure. A great deal of research shows that both affective and nonaffective responses sensitively index drug deprivation (Baker et al., 1987; Hughes et al., 1991; Hughes & Hatsukami, 1986). The present research merely joins a growing body of evidence that it is the affective constituents of withdrawal that motivate self-administration (e.g., Baker et al., 1987; Piasecki et al., 2000; Zinser et al., 1992).

Our results are silent with respect to the specific mechanisms that yield abstinence-induced negative affect. Such negative affect may reflect diverse processes ranging from homeostatic rebounds of second-messenger and protein phosphorylation mechanisms in neurophysiologic systems that mediate affect (e.g., Nestler, Guitart, & Beitner-Johnson, 1993) to psychological processes instigated by reinforcer loss.

An Intoxication–Abstinence Dissociation

One striking finding of this research is that no measure of current functioning, collected during the precessation period, predicted outcomes accurately. Thus, outcomes were unrelated, or only modestly related, to precessation smoking rate, blood nicotine level, CO level, current perceived need for nicotine, blood cotinine, perceived stress, negative affect, and coping style. Among precessation measures, expectations that smoking would ameliorate negative affect predicted outcomes to a significant degree. Precessation factors certainly can predict outcomes (e.g., Fagerstrom & Schneider, 1989; Stapleton et al., 1995), but they appear to be relatively weak predictors when compared with suitable postcessation events (e.g., Kenford et al., 1994). This suggests that factors that influence ongoing self-administration may be quite distinct from those that determine cessation success. This observation is incongruent with motivational models that suggest that a single mechanism (e.g., habit, positive reinforcement, or smoking "drive") accounts for both sustained administration and reinitiation of drug use.

If it is indeed true that distinct (but perhaps loosely coupled) motivational substratums exist (one influencing either the abstinent or drug-intoxicated states), and the present results can be generalized to other addictions, it suggests explanations for several phenomena characteristic of addictions. For instance, addicts find it difficult to assess accurately their ability to cease drug use when they make this prediction while using the drug (Brandon et al., 1990). This may be because the phenomenology and the motivational influences on the addict make it difficult to predict or anticipate the motivational impact of a change in drug state (Jellinek, 1960; Marlatt, 1979; Williams, 1966; Zweben & Barrett, 1993). In effect, drug addiction resembles a dissociative disorder in the sense that changes in drug use status can profoundly affect the addict's phenomenology, cognitive processing, affective status, attitudes, behavior, and vulnerability to psychopathology (Glassman & Covey, 1996; Martin, Van Loon, Iwamoto, & Davis, 1987; Zinser et al., 1992). An inability to anticipate the impact of incipient intoxication or abstinence may undermine attempts to control drug use.

Caveats and Limitations

Important caveats should be observed when interpreting or generalizing from the present findings. For instance, the population studied consists of heavy smokers seeking treatment. This population is perhaps unrepresentative of the majority of smokers who do not seek formal treatment (Fiore et al., 1990). Also, the sample size of the present study may have produced idiosyncratic results.

The present results were shaped by the particular model-building procedures used and the measures selected for analysis. For instance, the use of structural equation modeling with latent variable analysis might result in a different pattern of findings. Moreover, the use of different control variables such as self-efficacy or social support indices (Gulliver, Hughes, Soloman, & Dey, 1995) might have altered some of our findings. Also, different findings might obtain if one predicts short-term cessation success rather than relapse per se.

Another potential limitation of the present work is the measures used to assess the constructs of interest. For example, a single-item depression history measure was used. This may have been a less sensitive indicator of the importance of depression history in relapse prediction than a more extensive measure (i.e., structured interview of depression). In addition, the relation between withdrawal or affective symptoms and relapse might have been stronger if individual differences in symptom trajectory were assessed (Piasecki et al., 2000).

Overall, the present results suggest that affect and affect regulation are sensitive indicators of dependence and relapse. Although traditional measures of physical dependence may predict outcome well over a full range of smokers, they possess only modest validity within a population of inveterate smokers, and they do not

increment prediction when measures of affective status are available.

References

Abelin, T., Ehrsam, R., Buhler-Reichert, A., Imhof, P. R., Muller, P., Tommen, A., & Vesanen, K. (1989). Effectiveness of a transdermal nicotine system in smoking cessation studies. *Methods and Findings in Experimental Clinical Pharmacology, 11,* 205–214.

American Psychiatric Association. (1996). Practice guidelines for the treatment of patients with nicotine dependence. *American Journal of Psychiatry, 153*(Suppl.), 1–31.

Anda, R. F., Williamson, D. F., Escobedo, L. G., Mast, E. E., Giovino, G. A., & Remington, P. L. (1990). Depression and the dynamics of smoking. *Journal of the American Medical Association, 264,* 1541–1545.

Baker, T. B., Morse, E., & Sherman, J. E. (1987). The motivation to use drugs: A psychobiological analysis of urges. In C. Rivers (Ed.), *The Nebraska Symposium on Motivation: Alcohol use and abuse* (pp. 257–323). Lincoln: University of Nebraska Press.

Blondal, T. (1989). Controlled trial of nicotine polacrilex gum with supportive measures. *Archives of Internal Medicine, 149,* 1818–1821.

Brandon, T. H., & Baker, T. B. (1991). The Smoking Consequences Questionnaire: The subjective expected utility of smoking in college students. *Psychological Assessment: A Journal of Consulting and Clinical Psychology, 3,* 484–491.

Brandon, T. H., Tiffany, S. T., Obremski, K. I., & Baker, T. B. (1990). Postcessation cigarette use: The process of relapse. *Addictive Behaviors, 15,* 105–114.

Breslau, N., Peterson, E. L., Schultz, L. R., Chilcoat, H. D., & Andreski, P. (1998). Major depression and stages of smoking. *Archives of General Psychiatry, 55,* 161–166.

Brown, S. A., Goldman, M. S., Inn, A., & Anderson, L. (1980). Expectations of reinforcement from alcohol: Their domain and relation to drinking patterns. *Journal of Consulting and Clinical Psychology, 48,* 419–426.

Cannon, D. S., Baker, T. B., Berman, R. F., & Atkinson, C. A. (1974). A rapid technique for producing ethanol dependence in the rat. *Pharmacology, Biochemistry & Behavior, 2,* 831–834.

Clark, M. A., Kviz, F. J., Crittenden, K. S., & Warnecke, R. B. (1998). Psychosocial factors and smoking cessation behaviors among smokers who have and have not tried to quit. *Health Education Research, 13,* 145–153.

Cocores, J. (1993). Nicotine dependence: Diagnosis and treatment. *Recent Advances in Addictive Disorders, 16,* 49–60.

Cohen, S., Kamarck, T., & Mermelstein, R. (1983). A global measure of perceived stress. *Journal of Health and Social Behavior, 24,* 385–396.

Cooper, M. L., Russell, M., & George, W. H. (1988). Coping, expectancies, and alcohol abuse: A test of social learning formulations. *Journal of Abnormal Psychology, 97,* 218–230.

Cooper, M. L., Russell, M., Skinner, J. B., Frone, M. R., & Mudar, P. (1992). Stress and alcohol use: Moderating effects of gender, coping, and alcohol expectancies. *Journal of Abnormal Psychology, 101,* 139–152.

Copeland, A. L., Brandon, T. H., & Quinn, E. P. (1995). The Smoking Consequences Questionnaire—Adult: Measurement of smoking outcome expectancies of experienced smokers. *Psychological Assessment, 7,* 484–494.

Covey, L. S., Glassman, A. H., & Stetner, F. (1990). Depression and depressive symptoms in smoking cessation. *Comprehensive Psychiatry, 31,* 350–354.

Covey, L. S., Glassman, A. H., & Stetner, F. (1997). Major depression following smoking cessation. *American Journal of Psychiatry, 154,* 263–265.

Dale, L. C., Hurt, R. D., Offord, K. P., Lawson, G. M., Croghan, I. T., & Schroeder, D. R. (1995). High-dose nicotine patch therapy. Percentage of replacement and smoking cessation. *Journal of the American Medical Association, 274,* 1353–1358.

Fagerstrom, K. O. (1978). Measuring degree of physical dependence to tobacco smoking with reference to individualization of treatment. *Addictive Behaviors, 3,* 235–241.

Fagerstrom, K. O., & Schneider, N. G. (1989). Measuring nicotine dependence: A review of the Fagerstrom Tolerance Questionnaire. *Journal of Behavioral Medicine, 12,* 159–182.

Fiore, M. C., Jorenby, D. E., & Baker, T. B . (1997). Smoking cessation: Principles and practice based on the AHCPR Guideline, 1996. *Annals of Behavioral Medicine, 19,* 213–219.

Fiore, M. C., Kenford, S. L., Jorenby, D. E., Wetter, D. W., Smith, S. S., & Baker, T. B. (1994). Two studies of the clinical effectiveness of the nicotine patch with different counseling treatment. *Chest, 105,* 524–533.

Fiore, M. C., Novotny, T. E., Pierce, J. P., Giovino, G. A., Hatziandreu, E. J., Newcomb, P. A., Surawicz, T. S., & Davis, R. M. (1990). Methods used to quit smoking in the United States. Do cessation programs help? *Journal of the American Medical Association, 263,* 2760–2765.

Foulds, J. (1996). Strategies for smoking cessation. *British Medical Bulletin, 52,* 157–173.

Glassman, A. H., & Covey, L. S. (1996). Smoking and affective disorder. *American Journal of Health Behavior, 20,* 279–285.

Glassman, A. H., Helzer, J. E., Covey, L. S., Cottler, L. B, Stetner, F., Tipp, J. E., & Johnson, J. (1990). Smoking, smoking cessation and major depression. *Journal of the American Medical Association, 264,* 1546–1549.

Goldstein, D. H. (1972). Relationship of alcohol dose to intensity of withdrawal signs in mice. *Journal of Pharmacology and Experimental Therapeutics, 180,* 203–215.

Gritz, E. R., Carr, C. R., & Marcus, A. C. (1991). The tobacco withdrawal syndrome in unaided quitters. *British Journal of Addiction, 86,* 57–69.

Gulliver, S. B., Hughes, J. R., Soloman, L. J., & Dey, A. N. (1995). An investigation of self-efficacy, partner support and daily stresses as predictors of relapse to smoking in self-quitters. *Addiction, 56,* 767–772.

Gunn, R. C. (1983). Smoking clinic failures and recent life stress. *Addictive Behaviors, 8,* 83–87.

Hall, S. M., Rues, V. I., Munoz, R. F., Sees, K. L., Humfleet, G., Hartz, D. T., Frederick, S., & Triffleman, E. (1998). Nortriptyline and cognitive behavioral therapy in the treatment of cigarette smoking. *Archives of General Psychiatry, 55,* 683–690.

Hatziandreu, E. J., Pierce, J. P., Lefkopoulo, M., Fiore, M. C., Mills, S. L., Novotny, T. E., Giovino, G. A., & Davis, R. M. (1990). Quitting smoking in the United States in 1986. *Journal of the National Cancer Institute, 82,* 1402–1406.

Heatherton, T. F., Kozlowski, L. T., Frecker, R. C., & Fagerstrom, K. O. (1991). The Fagerstrom test for nicotine dependence: A revision of the Fagerstrom Tolerance Questionnaire. *British Journal of Addiction, 86,* 1119–1127.

Henningfield, J. E. (1995). Nicotine medications for smoking cessation. *New England Journal of Medicine, 333,* 1196–1203.

Henningfield, J. E., & Woodson, P. P. (1989). Dose-related actions of nicotine on behavior and physiology: Review and implications for replacement therapy for nicotine dependence. *Journal of Substance Abuse, 1,* 301–317.

Hosmer, D. W., & Lemeshow, S. (1989). *Applied logistic regression*. New York: Wiley.

Hughes, J. R., Gust, S. W., Skoog, K., Keenan, R. M., & Fenwick, J. W. (1991). Symptoms of tobacco withdrawal: A replication and extension. *Archives of General Psychiatry, 48,* 52–59.

Hughes, J. R., & Hatsukami, D. K. (1986). Signs and symptoms of tobacco withdrawal. *Archives of General Psychiatry, 43,* 289–294.

Hughes, J. R., Hatsukami, D. K., Pickens, R. W., Krahn, D., Malin, S., &

Luknic, A. (1984). Effect of nicotine on the tobacco withdrawal syndrome. *Psychopharmacology, 83,* 82–87.

Hurt, R. D., Dale, L. C., Fredrickson, P. A., Caldwell, C. C., Lee, G. A., Offord, K. P., Lauger, G. G., Marusic, Z., Neese, L. W., & Lundberg, T. G. (1994). Nicotine patch therapy for smoking cessation combined with physician advice and nurse follow-up. *Journal of the American Medical Association, 271,* 595–600.

Jacob, P., Benowitz, N. L., & Shulgin, A. T. (1988). Recent studies of nicotine metabolism in humans. *Pharmacology, Biochemistry & Behavior, 30,* 249–253.

Jellinek, E. M. (1960). *The disease concept of alcoholism*. New Brunswick, NJ: Hillhouse Press.

Jorenby, D. E., Hatsukami, D. K., Smith, S. S., & Fiore, M. C. (1996). Characterization of tobacco withdrawal symptoms: Transdermal nicotine reduces hunger and weight gain. *Psychopharmacology, 128,* 130–138.

Jorenby, D. E., Leischow, S. J., Nides, M. A., Rennard, S. I., Johnston, J. A., Hughes, A. R., Smith, S. S., Muramato, M. L., Daughton, D. M., Doan, K., Fiore, M. C., & Baker, T. B. (1999). A controlled trial of sustained-release bupropion, a nicotine patch, or both for smoking cessation. *New England Journal of Medicine, 340,* 685–691.

Kenford, S. L., Fiore, M. C., Jorenby, D. E., Smith, S. S., Wetter, D. W., & Baker, T. B. (1994). Predicting smoking cessation: Who will quit with and without the nicotine patch. *Journal of the American Medical Association, 271,* 589–594.

Killen, J. D., Fortmann, S. P., Kraemer, H. C., Varady, A., & Newman, B. (1992). Who will relapse? Symptoms of nicotine dependence predict long-term relapse after smoking cessation. *Journal of Consulting and Clinical Psychology, 60,* 797–801.

Kinnunen, T., Doherty, K., Militell, F. F., & Garvey, A. J. (1996). Depression and smoking cessation: Characteristics of depressed smokers and effects of nicotine replacement. *Journal of Consulting and Clinical Psychology, 64,* 791–798.

Kozlowski, L. T., Porter, C. Q., Orleans, T. C., Pope, M. A., & Heatherton, T. F. (1994). Predicting smoking cessation with self-reported measures of nicotine dependence: FTQ, FTND, and HSI. *Drug and Alcohol Dependence, 34,* 211–216.

Lerman, C., Audrain, J., Orleans, C. T., Boyd, R., Gold, K., Main, D., & Caporaso, N. (1996). Investigation of mechanisms linking depressed mood to nicotine dependence. *Addictive Behaviors, 21,* 9–19.

Lichtenstein, E., & Glasgow, R. W. (1992). Smoking cessation: What have we learned over the past decade? *Clinical Psychology, 60,* 518–527.

Marlatt, G. A. (1979). Alcohol use and problem drinking: A cognitive–behavioral analysis. In P. C. Kendall & S. D. Hollon (Eds.), *Cognitive behavioral intervention: Theory, research and procedure* (pp. 319–355). New York: Academic Press.

Martin, W. R., Van Loon, G. R., Iwamoto, E. T., & Davis, L. (1987). Tobacco and health overview: A neurobiologic approach. *Advances in Behavioral Biology, 31,* 1–13.

Mello, N. K. (1978). Alcoholism and behavioral pharmacology of alcohol: 1967–1977. In M. A. Lipton, A. DiMascio, & K. F. Killiam (Eds.), *Psychopharmacology: A generation of progress* (pp. 1619–1637). New York: Raven Press.

Menard, S. (1995). *Applied logistic regression analysis*. Thousand Oaks, CA: Sage.

Nestler, E. J., Guitart, X., & Beitner-Johnson, D. (1993). In S. G. Korenman & J. D. Barchas (Eds.), *Biological basis of substance abuse* (pp. 49–70). New York: Oxford University Press.

Niaura, R., Goldstein, M. G., & Abrams, D. B. (1994). Matching high- and low-dependence smokers to self-help treatment with or without nicotine replacement. *Preventive Medicine, 23,* 70–77.

Niaura, R. S., Rohsenow, D. J., Binkoff, J. A., Monti, P. M., Pedraza, M., & Abrams, D. B. (1988). Relevance of cue reactivity to understanding alcohol and smoking relapse. *Journal of Abnormal Psychology, 97,* 133–152.

Nides, M. A., Rakos, R. F., Gonzales, D., Murray, R. P., Tashkin, D. P., Bjornson, B. W. M., Lindgren, P., & Connett, J. E. (1995). Predictors of initial smoking cessation and relapse through the first 2 years of the Lung Health Study. *Journal of Consulting and Clinical Psychology, 63,* 60–69.

Payne, T. J., Schare, M. L., Levis, D. J., & Colletti, G. (1991). Exposure to smoking-relevant cues: Effects on desire to smoke and topographical components of smoking behavior. *Addictive Behaviors, 16,* 467–479.

Perkins, K. A. (1996). Sex differences in nicotine versus nonnicotine reinforcement as determinants of tobacco smoking. *Experimental and Clinical Psychopharmacology, 4,* 166–177.

Piasecki, T. M., Fiore, M. C., & Baker, T. B. (1998). Profiles in discouragement: Two studies of variability in the time course of smoking withdrawal symptoms. *Journal of Abnormal Psychology, 107,* 238–251.

Piasecki, T. M., Kenford, S. L., Smith, S. S., Fiore, M. C., & Baker, T. B. (1997). Listening to nicotine: Negative affect and the smoking withdrawal conundrum. *Psychological Sciences, 8,* 184–189.

Piasecki, T. M., Niaura, R., Shadel, W. G., Abrams, D., Goldstein, M., Fiore, M. C., & Baker, T. B. (2000). Smoking withdrawal dynamics in unaided quitters. *Journal of Abnormal Psychology, 109,* 74–86.

Pomerleau, O. F., Fertig, J. B., & Shanahan, S. O. (1983). Nicotine dependence in cigarette smoking: An empirically-based, multivariate model. *Pharmacology, Biochemistry & Behavior, 19,* 291–299.

Rabios, D., & Haaga, D. A. (1997). Cognitive coping, history of depression and cigarette smoking. *Addictive Behaviors, 22,* 789–796.

Rohde, P., Lewinsohn, P. M., Tilson, M., & Seeley, J. R. (1990). Dimensionality of coping and its relation to depression. *Journal of Personality and Social Psychology, 58,* 499–511.

Shiffman, S. (1986). A cluster-analytic classification of smoking relapse episodes. *Addictive Behaviors, 11,* 295–307.

Stapleton, J. A., Russell, M. A. H., Feyerabend, C., Wiseman, S. M., Gustavsson, G., Sawe, U., & Wiseman, D. (1995). Dose effects and predictors of outcome in a randomized trial of transdermal nicotine patches in general practice. *Addiction, 90,* 31–42.

Stitzer, M. L. & Gross, J. (1988). Smoking relapse: The role of pharmacological and behavioral factors. *Progress in Clinical and Biological Research, 261,* 31–42.

Sutherland, D., Stapleton, J. A., Russell, M. A., Jarvis, M. J., Hajek, P., Belcher, M., & Feyerabend, C. (1992). Randomized controlled trial of nasal nicotine spray in smoking cessation. *The Lancet, 340,* 324–329.

Tabachnick, B. G., & Fidell, L. S. (1989). *Using multivariate statistics*. New York: HarperCollins.

Taylor, C. B., Miller, N. H., Herman, S., Smith, P. M., Sobel, D., Fisher, L., & DeBusk, R. F. (1996). A nurse-managed smoking cessation program for hospitalized smokers. *American Journal of Public Health, 86,* 1557–1560.

Tonnesen, P., Norregaard, J., Sawe, U., & Simonsen, K. (1993). Recycling with nicotine patches and smoking cessation. *Addiction, 88,* 533–539.

U.S. Department of Health and Human Services. (1996). *Smoking cessation: Clinical practice guideline* (No. 18, DHHS Publication No. AHCPR 96-0892). Washington, DC: U.S. Department of Health and Human Services, Public Health Service, Agency for Health Care Policy and Research.

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology, 54,* 324–333.

West, R. J., Hajek, P., & Belcher, M. (1989). Severity of withdrawal symptoms as a predictor of outcome of an attempt to quit smoking. *Psychological Medicine, 19,* 981–985.

West, R. J., Jarvis, M. J., Russell, M. A. H., Carruthers, M. E., Path, M. R. C., & Feyerabend, C. (1984). Effect of nicotine replacement on the

cigarette withdrawal syndrome. *British Journal of Addiction*, *79*, 215–219.

Wetter, D. W., Kenford, S. L., Smith, S. S., Fiore, M. C., Jorenby, D. E., & Baker, T. B. (1999). Gender differences in smoking cessation. *Journal of Consulting and Clinical Psychology*, *67*, 555–562.

Wetter, D. W., Smith, S. S., Kenford, S. L., Jorenby. D. E., Fiore, M. C., Hurt, R. D., Offord, K. P., & Baker, T. B. (1994). Smoking outcome expectancies: Factor structure, predictive validity, and discriminant validity. *Journal of Abnormal Psychology*, *103*, 801–811.

Willemsen, M. C., De Heis, H., Van Breukelen, G., & Oldenburg, B. (1996). Determinants of intention to quit smoking among Dutch employees: The influence of social environment. *Preventive Medicine*, *25*, 195–202.

Williams, A. F. (1966). Social drinking, anxiety and depression. *Journal of Personality and Social Psychology*, *3*, 689–693.

Wills, T. A., DuHammel, K., & Vaccaro, D. (1995). Activity and mood temperament as predictors of adolescent substance use: Test of a self-regulation mediational model. *Journal of Personality and Social Psychology*, *68*, 901–916.

Zinser, M. C., Baker, T. B., Sherman, J. E., & Cannon, D. (1992). Relation between self-reported affect and drug urges and cravings in continuing and withdrawing smokers. *Journal of Abnormal Psychology*, *101*, 617–629.

Zweben, A., & Barret, D. (1993). *Treating alcohol problems: Marital and family interventions.* New York: Guilford Press.

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