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Task Force 2: Models of Smoking Relapse

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This report offers frameworks within which research investigations of smoking relapse might be conceptualized. First, variables that have been or should be investigated in relation to relapse are discussed. Next, three broad models of the relapse process are presented. These models present hypothesized relationships between relapse and its predictors over time. Then, several more specific models of the mechanisms of relapse are discussed. These models consider the range of variables that are thought to be important in the causation of relapse. Finally, broad methodological issues and directions for future research on relapse are addressed.

DOMAINS OF VARIABLES RELATED TO RELAPSE

An enormous variety of variables have been studied in relation to relapse, including demographics, personality, environment, smoking history, biological factors, and social factors. Even studies that seem to examine the same variable are methodologically heterogeneous, making most of the data noncomparable. For instance, research on stress and relapse includes studies that (a) characterize ex-smokers according to whether they smoked when stressed (Pomerleau, Adkins, & Pertschuck, 1978), (b) assess the frequency of life events preceding relapse (Ockene, Benfari, Nuttall, Hurwitz, & Ockene, 1982), and (c) ask relapsers whether the situation in which they relapsed was stressful (Shiffman, 1982).

Table 1 outlines the range of variables that have been or could be examined in relation to relapse. The columns in Table 1 refer to the timing and focus of

TABLE 1
Classification of Background and Precipitating Factors

	Factors		
	Background	*Precipitating*	*Stress*
Environmental	Policies (e.g., no smoking areas) Smoking cues Physical	Smoking cues	Noise Crowding Air pollution Disasters
Social	Support Influence (e.g., peer pressure) Family history of smoking Structure Interpersonal interactions	Support Influence (peers) Interpersonal interactions	Role Family
Biological	Pharmacological dependence Genetics Acute effects of smoking Reactivity Biochemical and endocrinologic Withdrawal Weight	Withdrawal Cessation effects Reactivity Conditioning Passive smoking Weight Slips Pharmacological	Physiological sources Biochemical sources
Personal	Personality Coping skill Cognitive orientation Attitudes Health status Other substance use Affect	Coping Cognitive organization Affective Health status Other substance use	Perceived Adjustment
Demographics	Age Sex Occupation Socioeconomic status Education Race Ethnic (cultural) Marital status	Change	Change Co-workers' status
Smoking	Years Brand/Dose Topography Quitting history Reasons for smoking Reasons for quitting Cessation and relapse experiences	Motives/Incentives Stages Slips Other tobacco sources Nicotine gum	

the measurements. The measures are divided into background factors and precipitating factors. Precipitating factors are active at the time of a relapse episode (e.g., an ex-smoker being prompted to smoke by the sight and smell of a cigarette). Assessments of precipitating factors focus on the moments preceding a relapse and are usually retrospective. Background factors heighten the ex-smoker's predisposition or vulnerability to relapse without necessarily specifying the moment at which relapse will occur. These factors may be baseline measurements of stable characteristics (e.g., sex or age) or more fluid measures of slowly changing characteristics (e.g., level of withdrawal discomfort).

The rows in Table 1 represent major domains of variables studied in connection with relapse. (The table entries are meant to be illustrative rather than exhaustive.) Each class of variables may act through either background or precipitating influences, as shown by the examples in the table. Biological factors, for example, include background variables such as the degree of physical dependence on nicotine and precipitating variables such as the ex-smoker's biological response to an initial reexposure to nicotine. Stress is included as a separate column in Table 1 to accommodate the many definitions and measures of stress in use by investigators. The multifaceted nature of the concept is illustrated by its presence in nearly all the rows.

MODELS OF THE RELAPSE PROCESS

Three models of the relapse process are postulated: cumulative, episodic, and interactive. Although these models differ in several important respects, they all share an emphasis on the dynamics of relapse risk over time and are concerned with the fluctuation of relapse proneness (RP) over time.

RP refers to the risk of relapse at any particular time. The determinants of RP are not specified by the process models. Instead, these determinants are specified by the models of relapse mechanisms discussed in the next section of this report. The models of the relapse process allow for any number of predictor variables to combine in either an additive or interactive manner to determine RP.

RP is conceptualized as a continuous variable, and discrete changes in smoking status are held to occur when RP crosses certain threshold values. A transition from abstinence to smoking occurs when RP exceeds a certain value; conversely, abstinence might be restored following a lapse when RP drops below this threshold. One variant posits two thresholds—a lower one marking the boundary between abstinence and a slip and a higher one marking the irrevocable transition from a slip to relapse. This variant implies that the same processes that determine an initial slip determine the transition to

relapse. Although they share this structure, the three different models posit different dynamics for RP.

Cumulative Model

The cumulative model posits that changes in RP occur continuously and smoothly (Figure 1). The focus here is on the background variables that may have a cumulative impact. Therefore, one might posit that relapse occurs when the total amount of stress exceeds a threshold value or when circulating catecholamines (a biochemical index of stress) cross a threshold value. This model is embodied in research that deals exclusively with background variables assessed at baseline. Studies documenting the greater relapse rate of heavier smokers are examples (Graham & Gibson, 1971; Ockene et al., 1982), as are studies in which relapse is predicted from personality variables (Ockene et al., 1982; Smith, 1970). Models centering on the depletion (or production) of a specific biochemical are cumulative. For example, the nicotine regulation model is cumulative.

Episodic Model

The episodic model (Figure 2) posits a more discrete, precipitous process. RP is implicitly treated as stable until an event suddenly precipitates relapse. Relapse is held to occur when a sudden input to the system raises RP above a threshold value. Graphically, relapse episodes represent sudden spikes in RP against a steady background level. This model is embodied in work on relapse

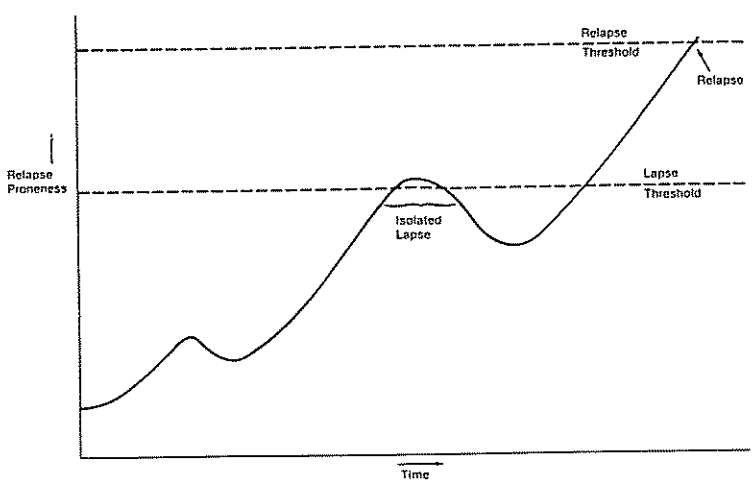

FIGURE 1 The cumulative model of relapse.

episodes (Curry & Marlatt, 1985; Lichtenstein, Antonuccio, & Rainwater, 1977; Lichtenstein & Baer, 1986; Marlatt & Gordon, 1985; Shiffman, 1982, 1984; see also Appendix abstracts by Ossip-Klein and by Shiffman). Although the theoretical postulates of these investigators refer to stable individual differences or to fluctuations in background variables, the empirical data exclusively monitor immediate situational precipitants.

Interactive Model

The interactive model represents a combination of the cumulative and episodic models. Like the episodic model, it postulates that relapse is often precipitated by acute events that raise RP above the threshold level. It emphasizes, however, that such triggering events occur against a background of continuous changes in RP. Graphically, this model is represented by spikes superimposed on a continuously variable background level of RP. The result is that identical spikes or precipitants may or may not trigger relapse, depending on the background level of RP on which they are superimposed (Figure 3).

Unfortunately, we found no examples of empirical work that use this model. It does appear in theoretical discussions such as Marlatt and Gordon's (1985) account of relapse, which posited that the impact of tempting stimuli depends on background variables such as the person's overall level of gratification (the "want–should ratio").

An important aspect of this model is that it allows for complex interactions between background and precipitating variables. The background and

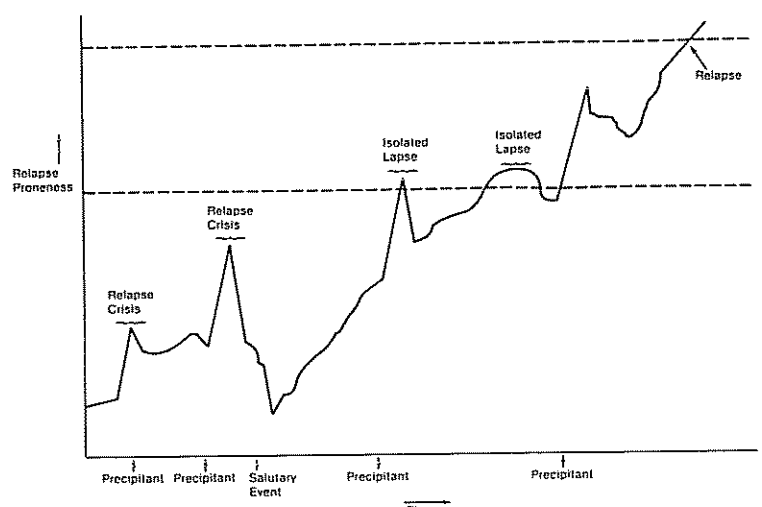

FIGURE 2 The episodic model of relapse.

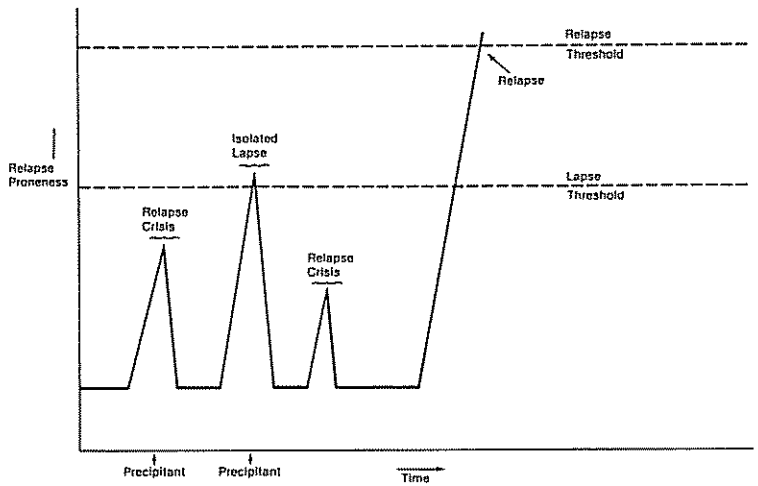

FIGURE 3 The interactive model of relapse.

precipitating variables may be similar, as when a specific stressor occurs against a background of high stress, or they may be quite different, as when a stressor occurs against a background of physiological relapse vulnerability.

Recent findings by Shiffman (1986) highlight the complexity of these interactions. In a preliminary analysis of data from a longitudinal study of ex-smokers, Shiffman found that relapse episodes precipitated by negative affects *and* those precipitated by smoking cues under positive affect were associated with elevated levels of stress. This finding suggests that the background and precipitating determinants of relapse could be opposite.

MECHANISMS OF RELAPSE

Although the process models outlined above specify the dynamics of RP over time, they are generic with respect to the variables controlling RP. In this section, we outline several theoretical models of the relapse process that, in turn, specify the variables that control relapse. We propose three broad models: learning, biobehavioral, and stress-coping. The models are not incompatible; indeed, each incorporates postulates of the others. Each differs from the others in its emphasis on a particular set of variables and mechanisms.

Learning Models

In learning-based models, the smoker's learning history and its relationship to the current environment are the key variables to be considered. To determine relevant learning processes, it is important to consider major types of learning. For example, association (viz., the law of contiguity) of cues with smoking should be examined as a possible reason for relapse. Principles of classical conditioning may influence slips. Also, operant conditioning processes such as reinforcement of smoking (e.g., peer group pressures, biological reinforcement from nicotine) and punishment or nonsmoking (e.g., peer influence, unpleasantness, and impaired performance associated with abstinence) may increase the likelihood of relapse. Social modeling (e.g., adult role models) may increase the likelihood of smoking and relapse.

In the study of relapse, these different learning processes all deserve consideration in combination with biobehavioral factors that are often incorporated into learning explanations of relapse. It will probably prove valuable to determine which learning processes act to influence slips in particular situations (e.g., those in which smoking previously occurred—at parties, after meals, etc.) and in response to particular cues (e.g., alcohol, a cup of coffee, the sight of someone smoking).

From the perspective of learning models, both current environmental stimuli and individual differences in learning should be examined. The presence and frequency of smoking cues in the environment may play a role in the relapse process. Mermelstein, Lichtenstein, and McIntyre (1983) suggested that when both members of a couple smoke, treatment is unlikely to succeed, perhaps because the partner who quits is exposed to a high frequency of smoking cues. Abrams, Monti, and Carey (1986), in a worksite study, reported that the number of friends who smoke is significantly correlated with the 6-month posttreatment rate of smoking. (See also Eisinger, 1972; Goldstein, 1981; Graham & Gibson, 1971; Ockene et al., 1982.) Although it is known that relapse frequently occurs in the presence of smoking cues (Shiffman, 1982), it is not known whether a high frequency of exposure to friends or co-workers who smoke may also have cumulative effects (i.e., may also act as a background factor). One important possibility is that individuals who are close to relapse actively seek out smoking cues and opportunities.

Individuals may also differ widely in their responsiveness to environmental stimuli, either because of biological predispositions (see below) or learning history. In a prospective treatment outcome study, Abrams, Niaura, Monti, and Pinto (1985) found that heart rate arousal during exposure to one's preferred brand of cigarette predicted relapse at 6 months after treatment. This study also suggested that participants' overall reactivity was not the issue, because their reactivity to a social situation did not predict treatment outcome.

Biobehavioral Models

Most likely, both biological and psychological factors operate to influence relapse because they are probably inseparable and operate concurrently.

Nevertheless, in an attempt to achieve some theoretical simplicity and to identify specific empirical questions, this section focuses on models that rely heavily on biological mechanisms.

Factors That Involve Conditioned Biological Effects

Direct conditioned biological effects. There is evidence that environmental cues present during self-administration of drugs become associated with the drug (possibly via classical conditioning) and that these stimuli subsequently come to elicit some of the biological effects of the drug (Grunberg & Baum, 1985; Siegel, 1976). Based on this phenomenon, it may be postulated that situations (e.g., parties, stressful experiences, finishing a meal) and cues (e.g., a cigarette advertisement, an ashtray) that were associated with smoking may elicit the same biological reactions (e.g., increased heart rate, increased blood pressure, cool fingertips) in the ex-smoker that smoking once caused. These familiar feelings of smoking may accentuate craving for a cigarette and the ex-smoker may therefore have an increased tendency to slip.

Counterconditioned biological effects. There is evidence that environmental cues repeatedly associated with drug-taking come to elicit biological effects that are opposite to the direct biological effects of the drug. These data are consistent with opponent-process theory (Solomon & Corbit, 1973). If cues previously associated with cigarette smoking cause biological effects that are experienced as disturbing or unpleasant because of a disruption in physiological homeostasis, then the ex-smoker may have an increased tendency to slip to offset these effects.

Conditioned withdrawal symptoms. Many habitual smokers experience unpleasant symptoms with abstinence from smoking (Shiffman, 1979). It is possible that this unpleasant state, resulting partially from biological effects, may be elicited by environmental cues associated with smoking abstinence. If so, conditioned withdrawal effects may increase the likelihood of a slip (Grunberg & Baum, 1985).

Effects of Abstinence

On abstaining from tobacco use, the former habitual smoker experiences a number of symptoms that result from depletion of availability of components of the tobacco (largely nicotine; Shiffman, 1979). The classic withdrawal symptoms (e.g., headache, irritability, sleeplessness) usually last on the order of days or perhaps weeks (Shiffman & Jarvik, 1976) and generally are consistent with the pharamacokinetics of the metabolism and elimination

of nicotine and its metabolites. During this phase, however, the unpleasant biological effects may increase the likelihood of smoking, thereby offsetting these withdrawal symptoms.

In addition to the classic withdrawal symptoms associated with smoking cessation, there are some consistent effects (e.g., weight gain) that seem to last up to a year or so after cessation (Grunberg, 1982, 1986; Wack & Rodin, 1982). These longer term abstinence effects may also increase the likelihood of slips.

Biobehavioral Effect Factors: General Comments

Although many biological effects of tobacco use and cessation of habitual tobacco use are well known, the role of these factors in relapse has received little research attention. In considering these factors, it is important to use a range of biological measures (e.g., heart rate, blood pressure, catecholamines, cortisol, glucose, insulin, and endogenous opioid peptides). Also, any research on the relative contribution of central versus peripheral physiological effects would be valuable. It might also prove valuable to consider possible individual differences in biological parameters such as dependence or central nervous system reactivity. Recent work combining laboratory and field methodologies (Abrams et al., 1985) found that persons who were more physiologically reactive to stress were more likely to relapse than were their less reactive counterparts. In sum, a variety of biological measures and mechanisms should be considered in relation to relapse.

Stress-Coping Models

Stress-coping models view smoking as a means of coping with stress. In general, stress-coping models hold that when situational demands exceed coping capacity, some people turn to smoking because smoking has been an effective means of dealing with stressful events and their consequences. These models emphasize that smoking is an attempted adaptation to environmental challenges. The key to understanding relapse is the balance between stress and coping. High levels of stress are thought to predispose ex-smokers to relapse, but ex-smokers can be buffered from this effect if they have adequate skills (other than smoking) or resources (e.g., social support) for coping with stress. More specific coping skills can help the ex-smoker prevent or overcome temptations to smoke even if stress is high.

There are two closely related stress-coping models applied to smoking and relapse: (a) affect regulation and (b) cognitive-behavioral. The affect-regulation model suggests that smoking in general and relapse in particular are means of coping with affective responses to stressful events (e.g., Tomkins, 1966). Smoking is used to decrease negative affect to reach a

homeostatic (or more desirable) level. This model leaves open the possibility that smoking can also regulate positive affect, increasing positive affect through physiological effects or through preestablished associations between smoking and positive situations. In a broader conception, this model deals with the regulation of arousal rather than affect. Relapse occurs when there is a substantial shift in arousal or affective state and when other available means of reestablishing comfort are inadequate. Thus, relapse depends on both an affective precipitant and on the absence or ineffectiveness of alternative modes of coping.

A second version of the stress-coping model of relapse is Marlatt and Gordon's (1985) cognitive-behavioral model. They proposed that relapse is promoted when ex-smokers confront potentially stressful situations and feel unprepared to cope with them. The combination of low self-efficacy and positive expectancies about the effects of smoking is thought to make relapse likely. The theory emphasizes cognitive expectancies of drug effects rather than direct pharmacological effects.

A unique feature of the Marlatt and Gordon (1985) model is that it also addresses the transition between an initial slip and relapse. The slip itself results in heightened negative affect, which in turn promotes further smoking. Cognitive changes also follow slips. Self-efficacy is lowered, particularly if the person fails to cope, and other cognitive processes may lead the person to redefine himself or herself as a smoker and terminate maintenance efforts. Marlatt and Gordon's model thus extends the stress-coping model to cover the events following a slip.

Relevant Research

Much of the research on the stress-coping model has focused on factors that precipitate relapse. Marlatt and Gordon (1985) developed a taxonomy of high-risk situations for relapse—"situation(s) that pose a threat to an individual's sense of control" (Marlatt & Gordon, 1985, p. 37). Three types of high-risk situations are most commonly associated with relapse: negative emotional states, interpersonal conflicts, and social pressure. Cluster-analytic work on relapse episodes suggests that there are two major subtypes of relapse situations: those marked by negative affect and those in which smoking-specific cues play a prominent role, accompanied by positive affect and social modeling (Lichtenstein & Baer, 1986; Shiffman, 1986).

The absence or inadequacy of coping in relapse crises has also been consistently identified as a proximal contributor to relapse (Curry & Marlatt, 1985; Shiffman, 1982, 1984). Coping is the single best predictor of continued abstinence in a high-risk situation.

Stress-coping models allow a role for background variables such as elevated stress. High levels of background stress are associated with relapse, both

retrospectively (Gunn, 1983; Ockene et al., 1982) and prospectively (Cohen, Kamarck, & Mermelstein, 1983). Lack of self-efficacy has proved to be a consistent predictor of relapse (Condiotte & Lichtenstein, 1981), although recent work (Baer, Holt, & Lichtenstein, in press) raises questions about the hypothesized mechanisms for this effect. Marlatt and Gordon (1985) also postulated that an imbalanced life style in which stressful or work-oriented activities ("shoulds") predominate over inherently pleasant activities ("wants") is conducive to relapse, but this notion remains to be evaluated empirically.

Stable individual differences also play a role in stress-coping models. The extent to which a person smokes to relieve stress may be an important individual difference variable (Pomerleau et al., 1978). Curry and Marlatt (1985) suggested that individual differences in attitudes about coping may also play important roles: People who expect smoking cessation to require active coping are more successful in maintenance.

Implications and Directions

Additional research is required to clarify a number of questions about the operation of stress-coping models. Understanding the relationship between stress and smoking would help to clarify the role of stress in relapse. Pharmacological evidence primarily suggests that nicotine elevates physiological indicators of arousal and affect; thus (from a physiological perspective), the mechanisms by which smoking would reduce affect are not entirely clear (e.g., Pomerleau, 1981). Future work should include assessments of the influence of smoking on the cognitive, affective, and biological mechanisms associated with stress and the possible interrelationships among them.

Little is known about the determinants of coping skills or coping styles. Furthermore, the relationship between general stress-coping skills and smoking-specific temptation-coping skills in relapse is underresearched (Wills & Shiffman, 1985). Abrams et al. (1986) showed that smoking-specific temptation-coping, especially in negative-affect situations, discriminated quitters from relapsers better than skill in coping with general social situations and social anxiety (stress-coping). Ex-smokers' typical ways of coping with stress may influence relapse in two ways: (a) They may prevent the accumulation of background stress, and (b) they may generalize to specific temptation-coping skills.

There is as yet little consensus about the constituents of coping skills. It may be that the range and flexibility of the coping repertoire, rather than its specific content, determine success (Lazarus & Folkman, 1984). Similarly, the number of coping responses implemented may be more important than the choice of particular coping options. Still, choosing the right coping response for the circumstances may be important. Timing may also be impor-

tant. The decision to avoid tempting or threatening situations, as opposed to confronting them, may have different effects at different times. However, one cannot avoid smoking cues forever, and therefore avoidance may not work as a long-term coping strategy. Eventually individuals must be able to expose themselves to these situations and develop appropriate coping responses. Thus, different coping skills may be required in different phases of maintenance as needed to prevent temptation (anticipatory coping), to deal with it when it arises (immediate coping), and to overcome its aftereffects (restorative coping) (Wills & Shiffman, 1985).

Other aspects of the stress-coping models require further empirical investigation. Marlatt and Gordon (1985) emphasized expectations of short-term positive consequences and difficulty in delaying gratification as potential promoters of relapse. Although much work has been done on cognitive expectations in problem drinking, surprisingly little is known about these factors in smoking. In drinking situations, the mere belief that alcohol has been consumed, rather than the pharamacological content of the beverage, has been related to loss of control over drinking (Marlatt, Demming, & Reid, 1973) and to affect regulation (Abrams & Wilson, 1979; Lang, Goeckner, Adesso, & Marlatt, 1975).

What role do expectancies play in smoking relapse? Studies using placebos and reverse placebos may shed light on the process of relapse (Gottlieb, Killen, Marlatt, & Taylor, 1985; Hughes, Pickens, Spring, & Keenan, 1985). It may be especially important to study the combined effects of expectancy and pharmacology in light of the introduction of nicotine regulation (e.g., Grunberg & Kozlowski, 1986; Henningfield, 1986; Pomerleau, 1984; see also Appendix abstracts by Benowitz and by Pomerleau). Expectancies may also influence the type and timing of coping responses used in different relapse situations.

The interrelationships among the various levels of the stress-coping model require further exploration. To date, much of the work on precipitating variables has been correlational, descriptive, and retrospective. More work is needed to understand how high-risk situations precipitate relapse. For example, might more distal antecedents, such as the stresses of everyday life, accumulate over time and then interact with proximal determinants, resulting in a complex behavior chain that precipitates relapse? Do all environmental antecedent events ultimately result in negative emotional reactions and biochemical-psychological sequelae, forming a "final common pathway" to temptations, slips, and relapse?

METHODOLOGICAL CONSIDERATIONS

Relapse research raises the same methodological considerations as any other field of research with respect to experimental design and appropriate data analyses. However, the current state of research on relapse also raises some specific design and analysis issues that deserve attention.

Methodological Diversity

Research on relapse is likely to profit from a diversity of research methods and approaches. At this point, no method has proved so uniquely valuable as to justify exclusion of other approaches. There is room for approaches ranging from basic laboratory or analogue studies to large-scale epidemiological studies.

Methodological diversity should also rule the selection of variables for study (e.g., the combination of psychosocial and biological measures in one study). Such studies should go beyond merely obtaining biochemical verification of smoking status to achieve an integration of psychosocial and biobehavioral factors in the studies' conceptions, methodologies, analyses, and interpretations (Baum, Grunberg, & Singer, 1982).

Studies should also integrate background and immediate factors in relapse. It would be interesting to examine the relationships to relapse of, for example, stress-related smoking motivation (measured at baseline), increase in daily stress exposure over the follow-up period, and precipitating stressors; sex and social support over the follow-up period; and amount smoked at baseline and severity of withdrawal symptoms. These interactive effects should be examined separately for different stages of the quitting process because many of the effects change over time.

In some cases, multiple measures of a single construct may be necessary. This approach is particularly important for constructs such as stress, social support, and coping, which have multiple and often ambiguous definitions. It is imperative to conceptualize psychosocial variables clearly in terms of specific hypotheses about the mechanisms linking them to relapse. For example, different conceptualizations of social support may operate in very different fashions in facilitating or inhibiting relapse. Specific support for smoking cessation may help maintain motivation or provide specific resources that aid cessation, whereas perceived availability of social support in the face of stress may reduce relapse by encouraging the appraisal of a potentially stressful event as benign (Mermelstein, Cohen, & Lichtenstein, 1983; see Appendix abstract by Cohen).

Clearer conceptualizations of the aim of coping—to relieve stress or to specifically resist temptation to smoke—as well as of the type of coping are needed. The appropriate choice of measures depends on the kind of coping that smoking is thought to replace. For example, an affect-regulation approach suggests that relapse occurs in the face of stress because the person lacks adequate options for emotion-focused coping, whereas a model that focuses on the cognitive aspects of stress appraisal emphasizes the lack of an adequate problem-solving coping repertoire.

Individual Differences and Interactions

Beyond simply including multiple independent variables, relapse research should focus on the interactions among them. Much research has focused on main effects on relapse, but few attempts have been made to identify interactions. The search for a single key variable influencing relapse is appealing because it implies simple interventions to prevent relapse; it is, however, limited. One consequence of focusing on main effects is that variables may be discarded prematurely based on the absence of main effects. Sex is one example. Although several studies have found no main effect of sex on relapse—tempting one to ignore this variable in subsequent analyses—a recent investigation (Swan, Rosenman, Parker, & Denk, 1985) found that predictors of relapse were substantially different for males and females. This finding is particularly noteworthy in light of the report that nicotine affects male and female animals differently (Grunberg, Bowen, & Winders, 1986). Personality variables, largely unproductive as main-effect predictors in smoking research (Smith, 1976), may emerge as more useful in interactive analyses.

Research is needed that emphasizes identification of subgroups vulnerable to relapse under different circumstances. Such research can influence treatment, which is currently applied homogeneously to all smokers. Treatment studies should increasingly focus on developing tailored strategies and on identifying subpopulations most affected by a given procedure, rather than on trying to find a universal "magic bullet."

Data-Analytic Models

Relapse has traditionally been conceptualized as an all-or-none outcome variable the status of which was determined after some finite follow-up interval. Recent data make clear that reality is less simple; for example, some of the successful maintainers will have had one or more slips. Investigators have typically dealt with this complexity by assigning these ambiguous cases to one of several outcome categories (see the Task Force 1 report for a consideration of these issues).

Even when outcome categories take account of multiple possibilities, most data-analytic models do not take account of the dynamic nature of relapse. A single outcome variable, even if multivalued, is still fundamentally static, assuming a single endpoint. Prochaska (1985) argued that the cessation–maintenance–relapse process might best be viewed as cyclical rather than linear. New methods of expressing and analyzing data that retain the dynamic character of the phenomena are needed (see the Task Force 1 report). Conceptualizing the maintenance–relapse process as dynamic makes clear that analyses of relapse have ignored the other aspect of the process: recovery from relapses. A recent study by Swan et al. (1985) suggested that variables

predicting return to abstinence are *not* mirror images of those predicting relapse. Although higher stress predicted relapse, lower stress did not predict return to abstinence.

Design Considerations

Research on relapse may profit from an abbreviation of the interval between observations and an extension of the observation interval. Frequent observations are sometimes necessary to track changes in background variables. An individual's stress level, for example, may change relatively rapidly, with the result that widely spaced observations may miss much of the relevant action. The 3-month, 6-month, and 12-month follow-up intervals that have become standard for outcome studies will often prove inadequate for process studies. Frequent observations may be especially important early in maintenance, when changes may be especially rapid (see Hunt & Matarazzo, 1973). The frequency of observations should be determined by the expected rate of change in the independent variables. Frequent observations are needed also because the *dependent* variable—participant's smoking status—may change rapidly as well.

Longer periods of observation are needed to record long-term changes in smoking status—late relapse (O'Connell, 1985) or resumption of abstinence after a relapse (Prochaska, 1985). Prochaska (1985) showed that relapse is typically followed by reentry into a contemplation phase in which participants continued to struggle actively with their smoking behavior. These data argue for a life-cycle perspective in which the follow-up period encompasses more than one cessation–relapse cycle. Longer observation periods will produce greater opportunities to model relapse and recovery and provide more opportunities to ascertain when equilibrium (if it exists) is attained.