In another smoking cessation study, Bowen, Spring, and Fox (submitted for publication) randomly assigned 31 participants to either a high- or low-carbohydrate diet. Subjects in the high-carbohydrate group were given specific dietary advice encouraging the use of carbohydrates; they were also given tryptophan as a dietary supplement. In the low-carbohydrate group, dietary advice focused on consumption of foods low in carbohydrates. Each group attended four 2-hour meetings per week. Sessions for both groups stressed information about the effects of tobacco, self-management strategies, rapid smoking, and relapse prevention techniques.

The rationale for this treatment approach is based on a 1982 report that smoking cessation is accompanied by an increase in preference for sweet-tasting high carbohydrate foods (Grunberg 1982a). Grunberg (1986) suggested that carbohydrates act through serotonergic mechanisms to attenuate withdrawal. Tryptophan is thought to increase the production of serotonin in the brain. At the end of treatment, 13 of 16 subjects (81 percent) in the high-carbohydrate group were abstinent (confirmed by CO assessments) compared with 9 of 15 subjects (60 percent) in the low-carbohydrate group. This difference was in the hypothesized direction but was not statistically significant. Also consistent with the hypothesis, nonabstainers in the high-carbohydrate group were smoking significantly fewer cigarettes than nonabstainers in the low-carbohydrate group. In both groups subjects gained weight after quitting smoking. No significant differences were observed between experimental groups in the number of subjects who gained weight or in the average amount of weight gain per subject.

Of the three investigations that have evaluated the impact of a weight-control program on weight gain and cessation (Bowen, Spring, Fox, submitted for publication; Grinstead 1981; Mermelstein 1987), none were successful in preventing weight gain and only one (Mermelstein 1987) reported a significant between-groups difference in the amount of weight gain. None of the smoking-plus-weight-control programs were clearly successful in significantly enhancing cessation rates.

At least three investigations have indicated that individuals can stop smoking without significant weight gain. However, these studies have been limited to subjects typically at high risk of CVD who participated in multicomponent CVD risk factor reduction trials. In a study involving MRFIT participants at the upper 10 to 15 percent on a measure of CVD risk, Schoenenberger (1982) reported that continuing smokers had lost an average of 4.6 pounds at a 3-year followup, but that those who quit smoking had gained less than 1 pound. All subjects participated in several treatments that focused on stopping smoking and improving diet. In a 6-year followup of these participants, quitters had gained 4.7 pounds compared with a 1.3-pound weight loss among nonquitters overall (Gerace et al., in press). However, weight gained after cessation varied as a function of baseline daily cigarette consumption. For those who had smoked 1 to 19 cigarettes per day, quitters averaged a 0.5-pound weight gain compared with a 2.4-pound weight loss among continuing smokers . For those who had smoked 20 to 39 cigarettes per day, quitters averaged a 4.6-pound weight gain compared with a 1.4-pound weight loss among continuing smokers. For those who had smoked more than 40 cigarettes per day, quitters averaged a 7.2-pound weight gain compared with a 1.0-pound weight loss among continuing smokers. Thus, weight gain after smoking

cessation was positively related to daily cigarette consumption before quitting (Gerace et al., in press).

Hickey and Mulcahy (1973) reported on 124 male smokers who survived a myocardial infarction and participated in a lifestyle modification program. At 2-year followup, these investigators found an average weight gain of 1.6 pounds (change not significant) among the 60 individuals (48 percent) who quit smoking. Those individuals who continued to smoke averaged a small, but nonsignificant weight loss (0.8 pound). In a study of CVD risk factor assessment in Paris, Ducimetière and colleagues (1978) randomly assigned 271 smokers to either a cessation-plus-diet advice group or a smoking cessation-only group. The two groups did not differ in weight at pretest, but at 2-year followup, subjects in the cessation-plus-diet group had significantly lower weights than subjects in the cessation-only group. However, the two groups did not differ in smoking cessation, and the large degree of attrition in the cessation-plus-diet group must be noted when evaluating treatment outcome.

Thus, it appears that for individuals at high risk for CVD participating in intensive, multicomponent risk factor trials, smoking cessation can occur without significant increases in body weight. Future research needs to focus on whether similar results can be obtained with the typical smoker in a more cost-effective intervention.

Pharmacologic Methods for Reducing Postcessation Weight Gain

Three pharmacologic approaches have been evaluated as potential treatments for reducing postcessation weight gain: nicotine polacrilex gum, d-fenfluramine, and phenylpropanolamine (PPA). The available information on pharmacologic interventions for reducing postcessation weight gain is summarized below.

There is substantial evidence that nicotine is the agent in tobacco that causes changes in body weight (US DHHS 1988a) Therefore, the most obvious pharmacologic approach that may prove useful in reducing postcessation weight gain is nicotine replacement. The least hazardous vehicle currently available to deliver nicotine is nicotine polacrilex gum. As literature documenting the use of the gum to aid in quitting smoking has grown (Schwartz 1987; US DHHS 1988a), several correlational studies have reported that use of the gum reduces postcessation weight gain (Emont and Cummings 1987; Fagerström 1987; Hajek, Jackson, Belcher 1988), although this effect is not observed uniformly (Hjalmarson 1984; Tonnesen et al. 1988). In one study, Fagerström (1987) conducted a followup of 28 patients who were still abstinent at 6-month posttreatment after attending a smoking cessation clinic. These subjects received 2 mg of nicotine gum. Subjects were divided at the median (263) number of pieces of gum chewed. Six months after treatment, less frequent gum users had gained an average of 6.8 pounds, whereas the body weight of more frequent gum users had increased by 2.0 pounds. Fagerström (1987) hypothesized that higher nicotine polacrilex gum use was necessary to produce blood nicotine levels approaching the effective dosages achieved by smoking.

Emont and Cummings (1987) also found that nicotine polacrilex gum use reduced postcessation weight gain and that this effect was related to the amount of gum chewed. These investigators studied 104 participants of a 2.5-week stop-smoking clinic. Of the

subjects who were either abstinent at 1 month or had smoked fewer than 5 cigarettes in the month since treatment, 20 had used nicotine polacrilex gum in their attempts to quit. Use of nicotine polacrilex gum in general was not significantly related to weight gain. However, when number of pieces of gum chewed per day was considered, there was a significant inverse correlation ($r=-0.37$) between nicotine polacrilex gum use and increase in body weight. When broken down by initial daily cigarette consumption, the relationship between nicotine polacrilex gum use and weight gain held only for individuals who had smoked more than 26 cigarettes per day. Neither the Fagerström (1987) nor the Emont and Cummings (1987) studies biochemically verified smoking status or measured blood nicotine levels.

In the only controlled investigation of this kind, Gross, Stitzer, and Maldonado (1989) examined the relationship between nicotine polacrilex gum use and body weight. Subjects were randomly assigned in a double-blind study either to a nicotine polacrilex gum or a placebo condition. Smoking and nicotine polacrilex gum use were verified with CO, thiocyanate, and cotinine measurements. Of the original 127 subjects, 40 completed the 10-week abstinence trial. In this period, abstinent subjects in the placebo group gained an average of 7.8 pounds, 4.0 pounds more than the abstinent nicotine polacrilex gum users. There was also evidence for a nicotine dose effect on weight gain. Users of fewer than 6.5 pieces of gum per day gained 5.0 pounds over the 10 weeks, whereas more frequent nicotine polacrilex gum users gained 1.5 pounds. Gross, Stitzer, and Maldonado (1989) present strong support for nicotine polacrilex gum's suppression of postcessation weight gain in this rigorous study. Once nicotine polacrilex gum use was discontinued, weight gain in both active gum and placebo conditions was comparable (6.8 vs. 8.7 pounds at 6-month followup). Thus, in this study, nicotine replacement delayed rather than prevented postcessation weight gain.

A recent controlled study (Spring et al., in press) evaluated the effects of d-fenfluramine on postcessation changes in food intake and weight gain. D-fenfluramine, which releases and blocks re-uptake of serotonin, is a prescription drug that has anorectic qualities without stimulating the central nervous system (CNS). For this study, 31 overweight female smokers were placed either on placebo or 30 mg d-fenfluramine per day in a double-blind assignment. Subjects then quit smoking and were observed for 4 weeks. Although the numbers of subjects remaining abstinent were small (five in the placebo group and eight in the d-fenfluramine group), significant differences in food intake between the two groups were observed over time. By 48 hours after discontinuing smoking, placebo-treated subjects consumed approximately 300 cal more per day than during the baseline measurement period. This increase resulted largely from increased consumption of carbohydrate-rich meal and snack foods. The difference in weight gain between the two groups was significant, with the placebo-treated subjects gaining an average of 3.5 pounds and the d-fenfluramine-treated subjects losing an average of 1.8 pounds. No significant differences in smoking cessation were observed, although statistical power to detect a difference was low.

A recently completed, placebo-controlled investigation evaluated the effects of phenylpropanolamine (PPA), which is an over-the-counter sympathomimetic agent that has weak CNS effects and more pronounced peripheral effects, on weight gain associated with smoking cessation (Klesges, Klesges et al. 1990). It is used both as an

anorectic agent and as a decongestant. Subjects were 57 adult female cigarette smokers who were randomly assigned, in a double-blind procedure, to either gum with PPA, (25 mg tid), placebo gum, or no gum. After a baseline assessment, subjects were paid to quit smoking for 2 weeks. Smoking cessation was verified by weekly as well as by random (spot), CO assessments. Of the 57 subjects enrolled in the study, 41 (72 percent) were successful in quitting smoking. Of subjects receiving PPA, 94 percent quit smoking, whereas 63 percent of the two control groups quit smoking. Of those subjects remaining continuously abstinent over the 2 weeks, dietary intake decreased 630 kcal on average in the PPA group, whereas intake in the other two groups remained unchanged. Decreases in intake of all major nutrients (carbohydrate, fat, and protein) were observed in the PPA group. Abstinent subjects receiving PPA gained significantly less weight (mean change=0.09 pounds) compared with either the placebo gum group (mean change=1.59 pounds) or the no gum group (mean change=1.94 pounds).

To summarize this Section, additional minor weight control modifications to smoking cessation programs do not generally yield beneficial effects in terms of reducing postcessation weight gain or increasing cessation rates. However, aggressive weight control programs, perhaps offered after individuals have quit smoking (Wittsten 1988), may be able to produce smoking cessation without unwanted weight gain. Nicotine polacrilex gum, d-fenfluramine, and PPA all have promise as adjuncts for reducing postcessation weight gain, but research to date is extremely preliminary.

Focus needs to be on more effective behavioral methods for reducing unwanted postcessation weight gain and on combination therapies that include behavioral and pharmacologic strategies. High priority must be given to the development and evaluation of effective programs that can be offered in a cost-effective manner. Given the probable role of metabolic rate on postcessation weight gain, weight programs may need to focus on reduction of dietary intake rather than dietary maintenance. Additionally, aggressive weight management programs may not be necessary, or even wanted, for many subjects who quit smoking (Gritz, Klesges, Meyers 1989). Future investigations need to determine, of those who quit smoking, the individuals best suited for weight management programs without compromising smoking cessation.

Studies on the effects of nicotine polacrilex gum, d-fenfluramine, and PPA on postcessation weight gain yield some cautious optimism. However, longer followup periods and larger, more heterogeneous samples must be utilized in future investigations. It also appears, at least with nicotine polacrilex gum (Gross, Stitzer, Maldonado 1989), that weight gain can occur rapidly after gum use is discontinued. This delayed weight gain , and its possible role on post-drug relapse ,needs to be investigated. Future research also needs to focus on specifying the influence of moderator variables, such as initial daily cigarette consumption, age, gender, and level of drug use on the effectiveness of these pharmacologic agents in preventing weight gain. Finally, the relative efficacy of these agents needs to be evaluated, and comparisons between pharmacologic and behavioral approaches to postcessation weight gain should be considered.

CONCLUSIONS

1. Average weight gain after smoking cessation is only about 5 pounds (2.3 kg). This weight gain poses a minimal health risk.

2. Approximately 80 percent of smokers who quit gain weight after cessation, but only about 3.5 percent of those who quit smoking gain more than 20 pounds.

3. Increases in food intake and decreases in resting energy expenditure are largely responsible for postcessation weight gain.

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CHAPTER 11
PSYCHOLOGICAL AND BEHAVIORAL CONSEQUENCES AND CORRELATES OF SMOKING CESSATION

CONTENTS

INTRODUCTION

Former smokers often describe quitting smoking as a turning point in their lives. For many individuals, cessation leads to an improved sense of well-being and often serves as a catalyst for other positive health-related lifestyle changes (Finnegan and Suler 1985; Knudsen et al. 1984; Suedfeld and Best 1977). These improvements in psychosocial functioning and health-related lifestyle behaviors may contribute to and reinforce continued abstinence. However, some smokers may hesitate to try to quit because they fear negative changes in mood and well-being (Gritz 1980; Hall 1984; Tamerin 1972). In addition, relapsers often attribute their return to smoking to unwanted changes in mood or to a strong desire for a cigarette (Baer 1985; Chapman, Smith, Layden 1971; Marlatt and Gordon 1980; Russell 1970; Shiffman 1982).

This Chapter reviews findings on short-term withdrawal effects and the longer term psychological and behavioral effects related to abstinence from smoking. Short-term withdrawal effects are described in the 1988 Report of the Surgeon General on nicotine addiction (US DHHS 1988). The first Section of this Chapter updates this review by examining recent studies in six areas: craving as a withdrawal symptom, changes in alcohol and caffeine use, withdrawal relief versus enhancement models of the effects of abstinence on performance, variability in withdrawal, timecourse of withdrawal, and nicotine withdrawal as a cause of relapse. The second Section reviews longer term changes, such as changes in the use of alcohol, illicit drugs, and other tobacco products as well as increases in other health-related practices and preventive health behaviors, including participation in cardiovascular and cancer screening. A major portion of this Section reviews the relationship of long-term abstinence to psychological factors such as mood, coping with stress, self-efficacy, and locus of control. Because the long-term psychological and behavioral effects of smoking abstinence have never been summarized, this Section will include a more indepth review of studies than will be provided in the Section on short-term effects.

Providing smokers with information on transient adverse withdrawal effects and the distinction between these and the longer term psychological and behavioral benefits of abstinence may allay fears and help remove barriers to quitting or to maintaining abstinence. This information may also help to develop more effective programs that help the smoker plan and cope with the effects of cigarette abstinence. For example, education about the signs and symptoms of withdrawal from tranquilizers appears to help long-term users stop using tranquilizers (Lader and Higgitt 1986).

SHORT-TERM EFFECTS OF SMOKING CESSATION: NICOTINE WITHDRAWAL

Brief Review of Previous Work

Over the last decade, several reviews have been published on nicotine withdrawal (Hatsukami, Hughes, Pickens 1985; Henningfield 1984; Hughes, Higgins, Hatsukami 1990; Murray and Lawrence 1984; Shiffman 1979; US DHHS 1988; West 1984). Perhaps the most widely-accepted description of nicotine withdrawal is that which

appears in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-III-R, American Psychiatric Association 1987) (Table 1). In addition to the signs and symptoms listed in DSM-III-R, depression, disrupted sleep, impatience, and perhaps increased pleasantness of sweets are common and valid indicators of nicotine withdrawal (Hughes, Higgins, Hatsukami 1990). However, an especially important effect not included in DSM-III-R is impaired performance, particularly on vigilance and rapid information processing tasks (Snyder, Davis, Henningfield 1989; Wesnes and Warburton 1983). Other consequences of withdrawal, which may not be clinically evident, include slowing of the electroencephalogram, changes in rapid eye movement during sleep, decreased levels of catecholamines, decreased thyroid function, increased levels of medications, decreased orthostatis, and increased skin temperature (American Psychiatric Association 1987; Hughes, Higgins, Hatsukami 1990).

TABLE 1.—Diagnostic categorization and criteria for nicotine withdrawal—nicotine-induced organic mental disorder

The essential feature of this disorder is a characteristic withdrawal syndrome due to the abrupt cessation of or reduction in the use of nicotine-containing substances (e.g., cigarettes, cigars, pipes, chewing tobacco, or nicotine gum) and that has been at least moderate in duration and amount.

Among many heavy cigarette smokers, changes in mood and performance that are related to withdrawal can be detected within 2 hr after the last tobacco use. The sense of craving appears to reach a peak within the first 24 hr after cessation of tobacco use and gradually declines thereafter over a few days to several weeks. In any given case it is difficult to distinguish a withdrawal effect from the emergence of psychological traits that are suppressed, controlled, or altered by the effects of nicotine or from a behavioral reaction (e.g., frustration) to the loss of a reinforcer.

Mild symptoms of withdrawal may occur after switching to low-tar (nicotine) cigarettes and after stopping the use of smokeless (chewing) tobacco or nicotine polacrilex gum.

Diagnostic criteria for nicotine withdrawal:

A. Daily use of nicotine for at least several weeks.

B. Abrupt cessation of nicotine use or reduction in the amount of nicotine used followed within 24 hr by at least four of the following signs:

(1) craving for nicotine
(2) irritability, frustration, or anger
(3) anxiety
(4) difficulty concentrating
(5) restlessness
(6) decreased heart rate
(7) increased appetite or weight gain

SOURCE: Condensed from the American Psychiatric Association (1987).

The signs and symptoms of nicotine withdrawal are observable; they are often of clinically significant magnitude and occur in self-quitters as well as those who attend smoking cessation clinics (Hughes, Higgins, Hatsukami 1990). Most withdrawal

symptoms are opposite to those produced by administration of nicotine, occur for a specified period of time, and with continued abstinence, return to levels similar to those experienced by a smoker. Relief of withdrawal by use of nicotine polacrilex gum, occurrence of withdrawal upon cessation of nicotine polacrilex gum, and occurrence of withdrawal upon switching to low-nicotine cigarettes indicate that a lack of nicotine is responsible for most withdrawal effects (Hughes, Higgins Hatsukami 1990; West 1984).

Craving as a Withdrawal Symptom

Recent articles have attempted to clarify the role of craving in cigarette smoking (Kozlowski and Wilkinson 1987a; West and Kranzler 1990; West and Schneider 1987). The term "craving" has been used loosely and interchangeably by both smokers and investigators to indicate a strong desire or urge to smoke. The problems associated with this terminology and the advantages to using the term "strong desire" have been outlined (Hughes 1986a; Kozlowski and Wilkinson 1987a; Kozlowski and Wilkinson 1987b; Marlatt 1987; Rankin 1987; Shiffman 1987; Stockwell 1987; West 1987; West and Schneider 1987; Kozlowski, Mann et al. 1989). Although an increased desire for a cigarette is a common consequence of abstinence, part of the craving may result from the desire to relieve other withdrawal symptoms by having a cigarette. For example, a review by West and Schneider (1987) demonstrated that withdrawal effects, such as irritability and restlessness, are positively associated with craving. They noted that drugs such as clonidine may alleviate craving because these agents reduce the other symptoms. Thus, craving might be alleviated by reducing other withdrawal symptoms (Kozlowski and Wilkinson 1987a).

An urge to smoke may be due to several factors, such as response to environmental stimuli associated with cigarette smoking or deprivation, onset of withdrawal symptoms, and protracted withdrawal. That such effects are physiologically, behaviorally, or cognitively mediated has been debated widely (Kozlowski and Wilkinson 1987a, b; West and Kranzler 1990; West and Schneider 1987).

The desire to smoke as indicative of nicotine withdrawal has been a subject of some controversy for five reasons. First, the referent for the terms craving and desire is unclear. In 1955, the World Health Organization (WHO) stated, "a term such as 'craving' with its everyday connotations should not be used in the scientific literature . . . if confusion is to be avoided" (WHO 1955, p. 63). On the other hand, craving for a cigarette is the most commonly reported postcessation symptom (Hughes, Higgins, Hatsukami 1990); and therefore, it is difficult to ignore these self-reports.

Second, craving readily occurs even when smokers are not trying to abstain (Hughes, Higgins, Hatsukami 1990; Hughes and Hatsukami 1986). However, many other withdrawal symptoms, such as irritability, are also experienced by smokers (Hughes, Higgins, Hatsukami 1990).

Third, several factors other than abstinence, such as sensory cues associated with smoking (Rose 1988), the "behavior" of smoking (Hajek et al. 1989), and expectancy (Hughes et al. 1989; Gottlieb et al. 1987), can influence craving. However, these factors can also affect other withdrawal symptoms (Francis and Nelson 1984). In addition,

demonstrating that a symptom is influenced by a nonabstinence variable does not mean that the symptom cannot be induced by abstinence; it simply suggests nonspecificity; that is, abstinence is only one of many causes.

Fourth, nicotine polacrilex gum does not predictably reduce the desire for a cigarette (Hughes 1986b; West 1984; West and Schneider 1987). However, one possibility is that more cigarette-like (i.e., more bolus-like) routes of administration of nicotine, such as aerosols, nasal sprays, and vapors, would decrease desire to smoke (Pomerleau et al. 1988).

Fifth, managing craving may be critical to cessation of smoking. Recent prospective studies have indicated that postcessation self-reports of craving are predictive of later relapse (Gritz, Carr, Marcus 1990; West, Hajek, Belcher 1989; Killen et al. 1990). Also, the ubiquity of smoking cues and the availability of cigarettes may make craving especially prevalent and difficult to resist.

Recent research contradicts the commonly held notion that the desire for cigarettes is less than that for prototypic drugs of abuse (Kozlowski, Wilkinson et al. 1989). Persons presenting for treatment of alcohol and drug problems compared the strongest urge they had for cigarettes with their strongest urge for the alcohol or drug for which they were seeking treatment. Among alcohol-dependent persons, 50 percent reported that their strongest urges for cigarettes were greater than their strongest alcohol urges, 32 percent reported that the strongest urges were about the same for both cigarettes and alcohol, and 18 percent reported that their strongest urges for alcohol were greater than for cigarettes. Among drug-dependent persons, 25 percent said their strongest urges were for cigarettes, 27 percent said their strongest urges were about the same, and 48 percent said their strongest urges were for their drug of choice.

In the treatment of drug dependencies, such as alcohol, use of the term craving has been historically associated with theories of loss of control (Ludwig and Wikler 1974). Typically, tobacco researchers are not implying loss of control over smoking when they use the term craving (Kozlowski and Wilkinson 1987a). Smokers may or may not be implying loss of control when they use the term.

In summary, although the desire to smoke may have a more complex origin than other withdrawal symptoms, it is a predictable and important withdrawal effect. The occurrence of craving after cessation has several implications. It suggests that nicotine delivered in a cigarette-like system may be the best method to relieve the desire to smoke because the delivery would mimic some of the sensory cues associated with smoking (Rose 1988; Hajek et al. 1989). Also, it suggests that for smokers who wish to avoid medication, behavioral strategies could be used to combat even pharmacologically mediated desires to smoke.

Changes in Alcohol and Caffeine Use

Initial short-term changes in alcohol and caffeine intake upon smoking abstinence are of increasing interest. It is unclear that smoking cessation impedes abstinence or prompts relapse back to drinking among those with alcohol dependence (Kozlowski, Ferrence, Corbit 1990). Such changes in alcohol and caffeine use were not reviewed extensively in the 1988 Surgeon General's Report on nicotine addiction (US DHHS

1988). Long-term effects of abstinence on alcohol intake are reviewed later in this Chapter.

Two prospective studies found that among smokers trying to stop smoking permanently, alcohol use significantly decreased, by about 75 percent per drink per day in one study, during the first week after abstinence (Hughes and Hatsukami 1986; Puddey et al. 1985). A third study reported that subjects who had a larger decrease in the number of cigarettes smoked postcessation had a larger decrease in alcohol use (Olbrisch and Oades-Souther 1986). However, a recent study suggested the opposite; that is, alcohol use increased among females who stopped smoking temporarily for 1 week for the duration of an experiment (Perkins, Epstein, Pastor 1990). This discrepancy across experiments may be due to gender or motivational differences in the populations. In the latter case, an increase in alcohol consumption may occur when smokers in an experiment do not try to control their alcohol intake during temporary smoking abstinence; however, when smokers are trying to stop permanently they may decrease alcohol use voluntarily as an aid to smoking cessation.

Abstinence does not appear to change short-term caffeine intake (Benowitz, Hall, Modin 1989; Hughes and Hatsukami 1986; Hughes 1990; Hughes et al. 1990; Kozlowski 1976; Puddey et al. 1985; Rodin 1987). Smoking increases the elimination of caffeine, probably through non-nicotine-related mechanisms (Benowitz 1988); thus, when smokers stop, their rates of elimination of caffeine decrease (Benowitz, Hall, Modin 1989; Brown et al. 1988). With no change in caffeine intake, blood levels of caffeine increase 2.5-fold (Brown et al. 1988). Because several of the symptoms of caffeine intoxication are similar to those of nicotine withdrawal (e.g., anxiety, restlessness, and irritability), it has been suggested that these increased levels of caffeine may mimic or potentiate symptoms attributed to tobacco withdrawal (Sachs and Benowitz 1990).

Withdrawal Relief Versus Enhancement Models of the Effects of Smoking on Performance

The effects of abstinence on performance were reviewed in the Surgeon General's Report on nicotine addiction (US DHHS 1988). This review and others (Hughes, Higgins, Hatsukami 1990) have concluded that abstinence impairs performance on attention tasks, especially those labeled as rapid information processing, selective attention, sustained attention, or vigilance tasks. This impairment may persist for at least 7 to 10 days (Snyder, Davis, Henningfield 1989) and is reversed by nicotine replacement (Snyder and Henningfield 1989). However, it is not clear that abstinence impairs learning, memory, performance on more complex tasks, problem solving, or reaction time.

In the prototypic procedure for studying the effects of smoking on performance, smokers abstain overnight; performance is then measured before and after smoking a cigarette. A possible result would be that performance on a vigilance task was better after smoking than before smoking. Some researchers might interpret this difference as an indication that smoking enhances performance (Wesnes, Warburton, Matz 1983). However, another interpretation is that the presmoking performance level was poor