CHAPTER VI

EFFECTS OF NICOTINE THAT MAY PROMOTE TOBACCO USE

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Despite the well-known health hazards associated with cigarette smoking and tobacco use, more than 50 million Americans continue to use these products. (See Chapter I for a brief review of health hazards and Appendix A for prevalence of use data.) Chapter IV presents evidence that tobacco use is an orderly form of drug-seeking behavior that involves nicotine self-administration. It is clear from Chapter IV that tobacco use involves several biobehavioral processes of drug dependence, including nicotine reinforcement and withdrawal. The initiation and maintenance of this dependence process may be promoted by other actions of nicotine. For example, some cigarette smokers report that smoking helps them to think better, to cope with stress, and to keep body weight under control. The fact that people believe that tobacco use has these effects may contribute to initiation, maintenance, and relapse.

This Chapter examines the evidence on the following three effects of nicotine:

- enhancement of human performance
- control of stress responses
- control of body weight.

These particular topics are presented because there is scientific literature relevant to each topic and because nicotine has been suggested to be central to each of these effects.

The three topics are discussed separately in this Chapter because the substantive material and relevant data are distinctly different for each topic. Also, the research on each topic is at a markedly different evidentiary stage at this time. Whereas studies on nicotine and performance are intriguing, there are some serious methodological concerns that force caution in the interpretation of the available experimental investigations. In contrast, the relationship between stress and smoking (i.e., that stress increases smoking) is well documented by self-report data, and several investigators have offered detailed theoretical explanations and mechanisms to account for this phenomenon. However, much of this speculation has preceded experimental investigations. In still another stage of investigation, extensive data have been gathered on the relationship between cigarette smoking and body weight, and laboratory studies have carefully assessed the role of nicotine. Explanations for the relationship between nicotine and body weight are based on investigations that were designed to test specific variables involved in this relationship. All three topics are currently receiving research attention and are considered to be important areas for more extensive investigation. This Chapter is meant to complement the information presented in Chapter IV to provide a more complete understanding of tobacco use. Most of the studies discussed in this chapter have examined effects of cigarette smoking. Some studies present data on effects of nicotine alone. The similarity in findings of

these two types of studies supports the conclusion that nicotine is responsible for the effects of cigarette smoking.

Tobacco Use, Nicotine, and Human Performance

Some cigarette smokers believe and report that smoking helps them to think and to concentrate (Russell, Peto, Patel 1974). These possibilities have been studied in the laboratory using several different tasks. Unfortunately, this research literature has methodological limitations. Most of the published studies compare smokers smoking with smokers not smoking. Few studies have included nonsmokers not smoking as a control group. When smokers smoking perform better than smokers not smoking, it is impossible to know if smoking actually improved performance, if abstinence from smoking impaired performance, or both. In addition, most studies allowing smoking and evaluating performance did not measure nicotine levels in the subjects. Therefore, the role of nicotine generally is inferred but not directly assessed. A few studies administered nicotine by oral tablets to smokers and to nonsmokers. This Section examines the effects of cigarette smoking and nicotine on attention, learning and memory, problem solving, and the control of motor function. Implications of these effects for tobacco use are discussed.

Attention

Effects of cigarette smoking on attention have been examined in the laboratory using sustained attention tasks, selective attention tests, and perceptual intrusion or distraction measures. The results using each measure are reviewed separately.

Sustained Attention

Vigilance tasks are the fundamental paradigm in the laboratory for defining sustained attention. Attention is directed to one or more sources of input for long periods of time. The subject is required to detect and respond to small, infrequent changes in the input. Performance in vigilance situations is often assessed in terms of the detection rate, i.e., the proportion of signals correctly detected, and the false-alarm rate, i.e., the number of occasions on which a signal is reported when one has not been presented. Measures of stimulus sensitivity and response criterion can be derived from the detection rate and the false alarm rate using the Theory of Signal Detectability (Green and Swets 1966) in order to assess performance. During a typical vigilance session, the detection rate decreases (the vigilance decrement), but it is also important to know if there is a decrease in false alarms, which would mean a criterion shift. If the rate at which a subject detects the stimuli falls, but there are no changes in false alarms, then there is a reduction in stimulus sensitivity.

In a study of smoking and visual vigilance, the Mackworth Clock (Mackworth 1950) was used because it produces a reliable vigilance decrement. Cigarette smokers who were allowed to smoke at 20-min intervals throughout the 80-min vigilance task maintained their stimulus sensitivity to experimental targets (Wesnes and Warburton 1978). In contrast, sensitivity was reported to drop for a group of nonsmokers and for a group of smokers who were not allowed to smoke. This finding suggests that smoking helped to maintain vigilance, but it could be that abstinence from smoking contributed to the performance decrement for smokers who were not allowed to smoke.

Tong and coworkers (1977) studied the performance of nonsmokers, smokers not smoking, and smokers smoking on a 60-min auditory vigilance task. While nonsmokers and smokers not smoking detected fewer signals as the test progressed, smokers smoking increased their number of detections. Again, it seems that smoking improved vigilance. However, this conclusion is tempered by the fact that the nonsmokers generally performed better than did the smokers on this task. Wesnes and Warburton (1978) reported that smokers maintained their initial level of stimulus sensitivity to auditory targets over an 80-min vigilance session when they smoked cigarettes at 20-min intervals. When they performed the task while smoking nicotine-free cigarettes, their sensitivity decreased over time. A similar study with a higher target density found a similar result: smoking was accompanied by maintained stimulus sensitivity (Mangan 1982). Whether smoking increased vigilance or whether abstinence decreased vigilance is not clear.

To determine whether nicotine was responsible for these effects of cigarette smoking on attention, Wesnes, Warburton, and Matz (1983) gave subjects nicotine tablets under the tongue and examined visual vigilance. The tablets consisted of nicotine placed on an alkaline matrix material to permit buccal absorption. Nicotine helped reduce the vigilance decrement by maintaining stimulus sensitivity. The nicotine tablets produced the same effects in nonsmokers, light smokers, and heavy smokers (Wesnes, Warburton, Matz 1983). Wesnes and Warburton (1978) found a similar effect of nicotine tablets on smokers but found no effect on performance by nonsmokers. Wesnes and Warburton (1984b) reported a small improvement in performance by nonsmokers given 1.5-mg-nicotine tablets; l.0-mg- and 0.5-mg-nicotine tablets did not improve performance.

The effects of smoking on sustained reaction time performance, which has a vigilance component, were studied by Frankenhaeuser and others (1971). The experimental sessions lasted 80 min during which subjects continually performed a simple visual reaction time test. In the nonsmoking condition, the speed of reaction decreased over time; in the smoking condition, there was little change over the

session. Subjects abstained from smoking the night before participating in this study. Therefore, the smokers in the nonsmoking condition were deprived for many hours.

Wittenborn (1943) factor analyzed attention tests and found that picking out various sequences of numbers or letters from an array was most heavily loaded on what he called an "attention" or "mental concentration" factor. Williams (1980) assessed the effects of smoking by smokers on a test of this sort that involved crossing out each letter "E" found in sheets of randomly ordered letters arranged in lines of 30 letters. Smoking cigarettes produced significant improvement in performance of the letter cancellation task compared to sham smoking an unlit cigarette (Williams 1980). Because the subjects had abstained from smoking overnight before the experiment, it is not clear whether smoking improved performance or whether deprivation caused a decrease in performance.

A computer version of the letter crossing test is the Bakan task (Bakan 1959), in which a series of digits is presented at the rate of l/sec from which subjects are required to detect certain specified three-digit sequences. Measures of both the speed and the accuracy of detection rate are made: Performance on this rapid visual information processing task after smoking was improved in both speed and accuracy above baseline levels, whereas either not smoking or smoking nicotine-free cigarettes resulted in a decline in speed and accuracy below baseline levels (Wesnes and Warburton 1983). The improvement in both speed and accuracy indicates that there is no speed and accuracy tradeoff. Higher-yield cigarettes improved performance more than low-yield ones, suggesting that nicotine is involved in these effects (Wesnes and Warburton 1984a). This interpretation is supported by studies with cigarettes with similar nicotine content but varying levels of tar and carbon monoxide (CO); cigarettes with the same nicotine content have the same effect on speed and accuracy (Warburton, in press). However, these conclusions must remain tentative until nicotine levels in the body are measured.

Analyses of performance during cigarette smoking indicate a 15-percent increase in speed and accuracy (Wesnes 1987) and improvement puff by puff (Warburton, in press). Rapid visual information processing has been studied during cigarette smoking puff by puff. Even with one puff, the probability of correct detections in the smoking conditions was higher than in the nonsmoking condition, and a single puff produced a change in reaction time (Warburton, in press). These findings suggest that smoking improves performance. However, these within-subject analyses need to be replicated and compared to nonsmoker control groups.

Selective Attention

Selective-attention tasks involve either focused or divided attention. Focused-attention tasks require subjects to attend to one source of information to the exclusion of others. Divided-attention tasks require subjects to divide their monitoring between two or more sources of information.

One study of selective attention (Tarriere and Hartemann 1964) combined central guiding with peripheral visual monitoring. The task lasted for 2.5 hr, and the measure of performance reported was the percentage of the peripheral visual signals that were missed during the session. Monitoring performance was maintained by smoking, in contrast to the large increase in the percentage of signal omissions when the subjects (all of whom were smokers) were not smoking.

In a study of divided attention, a test was based on the rapid visual information task (Warburton and Walters, in press; Wesnes and Warburton 1984a). Subjects were presented with digits at a rate of 50/min in both the visual and auditory modalities, with a different sequence for each modality. The detection of sequences in both parts of the divided attention task improved significantly after the smoking of one cigarette in comparison with not smoking. Smoking a cigarette also prevented the increase in reaction times that occurred in the control condition (smokers not smoking).

These studies show that smokers who smoke before selective attention tasks perform better than smokers who abstain from smoking before these tasks. Both the sustained and selective attention data indicate that smoking helps the smoker to perform.

Distraction

The Stroop test has been used in smoking research to examine distraction effects. The Stroop test uses three sets of displays: a list of color words printed in black, a set of color patches, and a list of color words with the words printed in incongruent colors (e.g., the word "Green" printed in blue). Subjects' word reading is faster than color naming, while naming the incongruently printed color words takes much longer than naming the patches. The time difference between naming the colors in the two conditions is the Stroop effect. This score indicates the subject's ability to focus attention on a relevant stimulus dimension of print color and to ignore an irrelevant semantic one.

The effects of nicotine on the Stroop performance of smokers and nonsmokers have been studied (Wesnes and Warburton 1978; Wesnes and Revell 1984). Wesnes and Warburton (1978) reported that nicotine reduced the size of the Stroop effect and that there were no differences between smokers and nonsmokers in the amount

of improvement produced by nicotine. This finding supports the argument that the effects of nicotine on attention are similar in smokers and nonsmokers. However, only six smokers and six nonsmokers participated in this study. Also, the performance by nonsmokers was not improved by nicotine tablets in the Wesnes and Revell (1984) study. Therefore, conclusions must be tentative until the findings of Wesnes and Warburton (1978) are replicated.

Evidence from the few distraction studies that have been reported is consistent with the results for sustained and selective attention. It may be that smoking and nicotine improve a general attentional processing capacity including improved attention to relevant stimuli (sustained and selective attention data) and ability to disregard irrelevant stimuli (distraction data). However, until studies include nonsmoker control groups and measure nicotine levels in the body, the conclusion that smoking improves attention remains plausible but equivocal. It is reasonable to conclude that the attention of smokers is better after smoking than after deprivation from cigarettes.

Learning and Memory

Numerous animal studies have demonstrated that nicotine improves learning and memory when it is administered pretrial and posttrial (Bättig 1970; Bovet-Nitti 1965; Castellano 1976; Erickson 1971; Evangelista, Gattoni, Izquierdo 1970; Stripling and Alpern 1974; Szekely, Borsy, Kiraly 1974). The effects of smoking and nicotine on human learning and memory are surprisingly complex in comparison with the effects described in reports of animal studies. Some studies of the effects of smoking on human learning and memory have shown that smoking improves this aspect of mental ability (Mangan 1983; Mangan and Golding 1978; Warburton et al. 1986). Studies of the effects of pure nicotine on human learning and memory have shown that nicotine improves memory just as smoking does (Warburton et al. 1986). However, Hull (1924) found evidence of impairment in auditory memory and in the efficiency of rote learning immediately after smoking, and later studies also have found that smoking can interfere with learning and memory, especially immediate memory (Gonzales and Harris 1980). The effects of smoking and nicotine on learning, immediate memory, delayed recall, and state-dependent memory are addressed separately.

Learning

There is no evidence for improved acquisition of information (i.e., general learning) after smoking. For example, Carter (1974) reported a higher number of correct responses from 10 smoking subjects than

from 10 nonsmoking subjects on a letter-digit substitution task for the second of 2 10-trial blocks given in the first 2 sessions (7 days apart). However, there was no difference between groups in savings (number of trials) for serial learning of a letter-digit substitution task.

Kleinman, Vaughn, and Christ (1973) had nonsmokers, 24-hr deprived smokers, and nondeprived smokers do paired-associate learning of a low- or high-meaningful list of nonsense syllables. There was no difference in learning among the groups on both trial and errors to a criterion. However, deprived smokers performed better on the high-meaningful list and worse on the low-meaningful list than did either of the other two groups.

The effects of nicotine on learning also have been investigated. Andersson and Post (1974) compared the effects of nicotine cigarettes with those of nicotine-free cigarettes in subjects learning a nonsense syllable list. Significant increases in heart rate indicated that nicotine was absorbed from the nicotine cigarettes. The first cigarette was given after the first 10 trials of learning the list, and a second cigarette, of the same kind, was given after 20 trials. The learning curves were identical for the two conditions prior to smoking. After nicotine, the number correct decreased and remained below the scores in the nicotine-free condition, but the learning curves were parallel. Thus, the rate of learning was not changed by smoking. After the second nicotine cigarette, the number of correct syllables increased significantly to the same level of acquisition performance as in the nicotine-free cigarette condition. Relative to the previous performance, nicotine had improved recall of the syllables. The difficulty in interpreting the effects of nicotine in this study is that learning and recall occurred over a 20-min period, while plasma and brain levels of nicotine would be expected to fall well below their peak levels. These data give no evidence of nicotine impairing acquisition, because the learning curves are parallel after the nicotine cigarette. However, it appeared that after the first nicotine cigarette, the information stored in the non-nicotine state was less available in the nicotine state, a phenomenon known as state-dependent learning. (See “State-Dependent Memory” below for a fuller discussion of this phenomenon.)

In another study, Andersson (1975) examined the effects of smoking on verbal rote learning using a similar procedure. Ten smokers were tested on two occasions during which they were initially given 10 successive trials followed by an 8-min break. In one condition, the subjects smoked a 2.1-mg-nicotine-delivery cigarette during this period, and in the other they simply rested. Then, another 10 trials took place, after which a 45-min break was given, followed by a final learning trial. As in the previous study, recall was significantly lower immediately after smoking. This lowered recall

tended to recover on successive trials. After the 45-min break, the recall in the two conditions was again identical.

Immediate Memory

In a study of immediate memory (Williams 1980), subjects were tested within 15 min after smoking one cigarette. They were given lists of numbers to memorize and then were immediately asked to recall them in the correct sequence (constrained recall). No main effects were significant. Controlling for presmoking performance, the number of errors increased with strength of cigarettes smoked.

Houston, Schneider, and Jarvik (1978) had 23 heavy smokers, deprived of cigarettes for 3 hr, read a list of words. The subjects were matched on a free-recall test prior to smoking. Each member of one group smoked a 1.5-mg-nicotine cigarette, and each member of the other group smoked a non-nicotine cigarette. The subjects were given three lists with free recall tests after each one. The immediate recall scores showed that the nicotine group had significantly less recall than the placebo group did. When testing was given once just after the input, however, facilitation was seen (Warburton et al. 1986). After smoking a l.4-mg-nicotine cigarette, each of these subjects was shown a list of nouns and immediately asked to write down as many as possible. Measures of immediate recall were improved in smokers after smoking compared with not smoking.

Comparison of Immediate and Delayed Recall

Gonzales and Harris (1980) assessed the effects of smoking or abstinence on immediate and delayed memory of new and old (previously presented) words, as well as category clustering. Smokers smoking showed significantly poorer immediate and delayed recall of old words and less clustering of words into categories on the delayed recall test as compared with smokers who were not allowed to smoke before the tasks.

Mangan (1983) examined the effects of smoking a low- (0.7 mg) and a middle- (1.3 mg) nicotine-yield cigarette on paired-associate and serial learning and retention. Conditions included high and low intralist interference. Cigarettes improved retention in paired-associate learning, with task difficulty apparently having little relevance. Smoking impeded learning under low-interference conditions, but facilitated learning of high-interference sets.

Mangan and Golding (1983) studied the effects on memory of smoking deprivation and of smoking a single cigarette immediately after acquisition of a paired-associate learning task. Subjects were retested for retention of the memorized material at intervals of 30 min, 1 day, 1 week, and 1 month. At 30-min retest, nonsmokers showed superior recall compared with all smokers. After 1 month,

subjects who each smoked a low- and medium-nicotine cigarette were better than those who smoked high-nicotine cigarettes. They also achieved superior recall compared with nonsmokers.

Peeke and Peeke (1984) tested the effects of smoking one cigarette on verbal memory and attention in four experiments. In one study, subjects were allowed to smoke before the test ("pretrial smoking"), after the test ("posttrial smoking") or not at all ("no smoking"). Recall of a 50-word list was tested immediately after intervals of 10 and 45 min. Pretrial smoking resulted in improved recall 10 and 45 min after learning, but not immediately. Posttrial smoking was ineffectual. Tests at 1, 5, and 30 min after presentation of a 20-word list were compared with results from pretrial smoking. Improved recall occurred for pretrial smoking. The high-nicotine cigarette produced improved recall on both immediate- and delayed-recall tests. The low-nicotine cigarette was less effective. Light and heavy smokers did not differ in the effect of smoking on recall.

Andersson and Hockey (1977) presented words in different positions on a computer screen to smokers allowed to smoke or not allowed to smoke. In one condition, subjects had to remember the words in presentation order. In the second condition, subjects were asked to remember words, word order, and location. There were no differences between the smoking and no-smoking conditions in the percentage of words that were recalled in the correct order or for the percentage of words that were recalled correctly, regardless of word order. However, recall of position on the screen was poorer for the smoking group. When the subjects were asked to attend to all three aspects of the material, the groups did not differ significantly in their recall, although there was a trend for location to be recalled better after nicotine use than after deprivation. This study suggests that nicotine can enhance storage of information only if the subjects perceive that the information is relevant.

State-Dependent Memory

In a state-dependent design, one group of subjects learns after a dose of drug while a second group learns after a placebo or nothing. For the recall test both groups are divided: half of each group is tested with the agent presented during learning and half is switched to the other condition. If the recall scores are better for those groups that learned in the same chemical state, then state-dependent learning is said to have occurred. Numerous animal studies have provided evidence of state dependency with cholinergic drugs (Warburton 1977). The possibility that nicotine produces state-dependent learning in human subjects has been investigated in several studies.

Kunsendorf and Wigner (1985) examined state-dependent recall on text material. Subjects spent 15 min studying a 550-word article on

education and answered 6 factual questions based on the article after a 10-min break. The treatment conditions were smoking versus no smoking during the study period and during testing. When studying and testing were conducted for the same subject state (either smoking or no smoking), memory was better than when study and testing were conducted for different states.

Other investigators also have found evidence for state-dependent learning with smoking. Peters and McGee (1982) used the state-dependent design to test smoking's effect on recall and recognition memory. After smoking a l.4-mg-nicotine cigarette, each subject was shown a list of nouns and immediately asked to write down as many as possible. There was no evidence of any difference in immediate recall, a finding in agreement with Andersson and Hockey (1977) and Houston, Schneider, and Jarvik (1978). However, on the following day, there was a state-dependent effect on the recognition test but no difference between the same-state groups.

In another recognition study (Warburton et al. 1986), smokers who were deprived of cigarettes for more than 10 hr were each given a 1.4-mg-nicotine cigarette or nothing immediately before serial presentation of a set of Chinese characters. Subjects were divided into four equal groups: Those who did not smoke prior to learning or recall; those who did not smoke prior to learning, but had a cigarette prior to recall; those who had a cigarette prior to both learning and recall; and those who had a cigarette prior to learning, but none prior to recall. Subjects who smoked prior to learning had significantly better recognition scores than the subjects who did not smoke in the first part of the experiment. There was no effect of smoking on recall performance. A significant interaction term indicated that changing the chemical state interfered with recognition.

Warburton and colleagues (1980) used nicotine tablets in the state-dependent design. After ingesting the tablet, each subject listened to words and then performed successive subtractions for 1 min to prevent rehearsal. Immediate free recall was improved. One hour later, the subjects were given either nicotine or placebo tablets. They were asked to recall as many of the words as they could in another 10-min free recall test. Long-term recall was significantly better when subjects had taken nicotine prior to learning, but was not when taken prior to recall. A significant interaction term gave evidence for a state-dependent effect of nicotine and showed that nicotine was facilitating the input of information to storage, but had no direct effect on storage or retrieval.

These findings suggest that there is a state-dependent effect of smoking on cognitive performance. The seeming impairment of immediate memory, however, complicates any simple generalizations about smoking and memory or nicotine and memory. As with the attention literature, studies need to include nonsmokers as

controls to determine whether smoking or abstinence from smoking affects learning or memory. In addition, task characteristics and individual differences among subjects must be considered in future investigations. Based on the available evidence, there are no clear effects of smoking on learning or memory.

Problem Solving

Human problem-solving capabilities involve both attention and memory. Attention is important because distraction from the task will cause a deterioration in problem-solving performance. Memory also plays a critical role in thought, both guiding the operations of the thought processes and limiting their power. Problems can be broadly categorized as well defined and ill defined. A well-defined problem has a clearly stated goal with a clear method to ascertain if the problem solving will lead to the correct solution. A well-defined problem can be solved by convergent thinking that produces logically correct answers. A simple example of a well-defined problem is addition. Ill-defined problems are solved by divergent thinking that leads to inventive solutions.

Hull (1924) found that smoking increased the rate of complex mental addition, but had no measurable effect on the accuracy of addition. Kucek (1975) found that the reduced efficiency of mental addition that was produced by doing a tracking task was ameliorated by smoking. The improvement was especially manifested in the most neurotic subjects. One interpretation of this improvement is that the attentional effects of nicotine enabled the filtering out of the distracted thoughts that interfered with performance.

A task that has elements of both convergent and divergent thinking is the Luchins Jar test (Luchins 1942), in which subjects are asked to solve a number of "numerical problems" involving the measurement of a quantity of water by means of a set of measuring jars. For the first six trials, exactly the same solution can be used, but after trial six, both the old formula and a new, easier formula are appropriate. A measure of convergent thinking is performance on the first six trials, while divergent thinking is assessed from the time taken to discover the new, easier solution. Smokers who were allowed to smoke performed better on the first half of the test in which subjects used the same solution repeatedly (convergent thinking), but were slower to change to a simpler solution when it was available, divergent thinking (Warburton 1987). While it could be argued that nicotine had impaired divergent thinking, it has been argued that it is more efficient for a subject to use a known strategy, no matter how clumsy It might be, than to attempt to invent a new one, i.e., to maintain attention (Norman 1980).

Motor Control

The effects of smoking on motor control were investigated in the early laboratory study of Hull (1924). He found a marked increase in hand tremor, a slight increase in resistance to muscular fatigue and in speed of reading reaction time, and no measurable effect on the rate of tapping or on the rate or accuracy of eye-hand reaction. These reports have received support from more recent studies (Lyon et al. 1975; Smith, Tong, Leigh 1977). West and Jarvis (1986) reported that nasal administration of nicotine increases tapping rate in nonsmokers.

Tremor

Lippold, Williams, and Wilson (1980) recorded finger tremor during a control period, sham smoking, or cigarette smoking with a strain gauge and an accelerometer. Smoking increased tremor amplitude at least twofold.

Simple Reaction Time

Cotten, Thomas, and Stewart (1971) investigated the immediate effects of smoking one cigarette on simple reaction time after each subject smoked a cigarette with a 1.5-mg nicotine yield. The mean reaction times immediately following and 5 min after smoking were significantly slower than for all other test intervals. Reaction times for the 40- and 55-min intervals were significantly faster than the reaction time before smoking.

Morgan and Pickens (1982) examined whether reaction time performance after smoking varied as a function of cigarette smoking. Twelve regular smokers were tested on a reaction time task immediately after smoking on three different occasions. In each session, they were allowed ad libitum smoking of their own cigarette, or ad libitum smoking of a standard cigarette, or they had to smoke a standard cigarette with a prescribed puff pattern. Reaction time performance was significantly faster after smoking under the latter two of the three conditions. Mean reaction times were significantly shorter for the smokers smoking than for the smokers not smoking.

Choice Reaction Time

Myrsten and Andersson (1978) compared the effects of smoking for both simple and complex reaction time tasks. In the simple reaction time testing periods, smoking prevented the significant increase in reaction time that occurred over time in the nonsmoking condition. In the complex reaction time periods, smoking significantly reduced reaction time, whereas reaction time increases were not significant in the nonsmoking condition.

Decision time and motor time scores on a choice reaction time task were measured after smoking (Lyon et al. 1975; Smith, Tong, Leigh 1977). Decision time scores were significantly decreased by smoking, and the high-nicotine cigarette had the greatest effect. Motor time scores were not improved, and hand steadiness was significantly impaired by smoking.

Smokers, deprived smokers, and nonsmokers performed a compensatory tracking task while simultaneously performing a cross-adaptive loading task (Schori and Jones 1975). With the cross-adaptive technique, the size of the subject's total work load (tracking and loading tasks combined) was individually tailored to use each subject's entire attentional capacity. No differences were detected as a function of smoking either in tracking or in loading task performance.

Smokers, deprived smokers, and nonsmokers performed a complex motor task, consisting of five subtasks, for an extended period of time at two levels of task complexity (Schori and Jones 1974). On only one subtask, on one of the two performance measures obtained, were differences as a function of smoking condition evident. Specifically, response latencies for nonsmokers were shorter than those for smokers and deprived smokers at the high level of task complexity, but were longer at the lower level. Because the performance differences were small, Schori and Jones (1974) concluded that for all practical purposes, smoking had no effect on performance.

Implications for Tobacco Use

Some cigarette smokers report that smoking helps them to think and perform. Laboratory studies of attention and state-dependent learning are generally consistent with this perception, but studies of memory and learning do not support this perception. Data on problem solving are too limited to allow clear conclusions. The improvement in attention, state-dependent learning, and some motor performance tasks are, in most cases, superior in smokers who are allowed to smoke compared with a smoking abstinence condition. Therefore, these effects may, in part, reflect reversal of the deleterious effects of smoking abstinence. In contrast to this cautious interpretation, however, it should be noted that the experiments that administer nicotine and report similar improvements in nonsmokers and smokers are consistent with the interpretation that smoking improves some cognitive performance. In light of these data, smokers' self-reports and perceptions may be correct that smoking helps them to attend, think, and perform. However, until more careful investigations are reported, conclusions concerning the effects of smoking and nicotine on human performance must remain tentative. Future studies should include nonsmokers as controls and should measure nicotine levels after smoking or abstinence.

Current methods in cognitive psychology indicate that different paradigms for evaluating memory and performance (e.g., data-dependent versus context-dependent memory measures) produce opposite effects in many cognitive tasks (Richardson-Klavehn and Bjork 1988). The effects of smoking and nicotine on these different types of tasks need to be evaluated. A recent presentation on smoking and performance, for example, reported that smoking improved performance on simple reaction tasks but impaired performance on more complex comprehension and motor performance tasks (Spilich 1987). Tasks requiring different levels of demand must be examined. Moreover, future research should evaluate performance over time to determine whether any short-term effects of smoking or nicotine on performance persist or are reversed later on. Nonetheless, the fact that smokers smoking generally perform better on some cognitive tasks (especially attention tasks) than do smokers not smoking may encourage smokers to continue smoking and may encourage relapse.

Tobacco Use, Nicotine, Stress, and Mood Regulation

Cigarette smokers commonly report that they smoke in response to stressful situations and that smoking calms them. In addition, many smokers report that smoking helps to regulate dysphoric mood or affect. Reports of a relationship between stress and smoking generally have been regarded as puzzling in light of the sympathomimetic effects (i.e., sympathetic nervous system (SNS) activating actions) of nicotine, but the consistency of these claims has brought research attention to these topics. The possibility that smoking may help to regulate dysphoric moods that involve low arousal states is easier to understand. This Section reviews the relevant research literature and presents current thinking to help explain these phenomena.

Subjective Well-Being, Stress, and Mood Regulation

The state of subjective well-being is construed as one in which positive affect (pleasure, happiness) is high and negative affect (frustration, anger, tension) is low (Watson and Tellegen 1985). Departures from an optimal state may occur because of internally generated affect (worry, anxiety) or through environmental events that strain the coping ability of the individual (Dohrenwend and Dohrenwend 1981). A state of subjective stress is postulated to be a joint function of the current environmental demands and the current coping abilities of the individual (Lazarus and Launier 1978; Lazarus and Folkman 1984). When demands exceed coping ability, a state of subjective stress may arise that manifests at the psychological level as symptoms of psychological distress and at the physiological level as changes in (SNS) arousal, changes in endocrine systems,

and decrements in specific task performance (Baum, Grunberg, Singer 1982; Cohen, Kamarck, Mermelstein 1983). In natural settings, stress may occur because of discrete events that cause a transient peak in subjective distress or in conditions that persist over considerable periods of time and thus present sources of chronic strain to affected individuals (Pearlin and Schooler 1978; Pearlin et al. 1981).

Overall mood states are related to independent contributions by dimensions of positive affect and negative affect: well-being is determined by low negative affect and by high positive affect (Diener 1984). Studies of mood states in natural settings over intermediate time periods of 1 day to 1 week show that the dimensions of negative and positive mood are independent, that is, they both occur on a regular basis in daily life and both contribute to overall mood states (Stone, Helder, Schneider 1987). Mood may be regulated both by reduction of negative affect and by increase of positive affect (Tomkins 1962, 1963; Wills and Shiffman 1985).

Subjective well-being could be improved through reducing the perceived environmental demands, through physiologically influencing stress-related arousal states, through reducing perception of unpleasant physical states, or through altering the balance of positive/negative affect in daily life. These mechanisms are relevant to understanding the relationship between stress and cigarette smoking (Tomkins 1965).

Perceived Functions of Smoking

A number of epidemiological studies have examined the perceived functions that smoking provides for users by employing large samples that are usually representative of communities; in some cases, representative national samples have been obtained. These studies ask respondents about various functions that smoking is perceived to provide for them, and the researchers aim to determine basic functional dimensions through factor analysis or cluster analysis of the motive reports. The questionnaire items used to elicit smoking functions vary considerably, including items that elicit agreement/disagreement with statements about smoking, items that elicit the frequency or likelihood of smoking in defined situations, or items that ask about a desire to smoke in certain settings. Although the methodology and sampling procedures have varied considerably across studies, there is consistency in the results. One higher order domain of intercorrelated motive dimensions indicates that smoking is perceived to provide *negative affect reduction;* another domain indicates that smoking is perceived to provide *positive affect enhancement.* Findings from the relevant studies, classified in terms of these higher-order domains, are presented in Table 1. (Survey studies also indicate that many smokers report that smoking keeps

weight down and that weight control is one of their major concerns (Charlton 1984a,b; Feldman, Hodgson, Corber 1985; Page 1983). However, for purposes of expositional clarity, this Section focuses on affect regulation and stress. Smoking and body weight are discussed in the next Section of this Chapter.)

A typical study of perceived functions was conducted in the United States by Ikard, Green, and Horn (1969) with a representative national sample of 2,094 adult respondents. In this study, subjects were presented with a list of 23 statements about smoking, representing various combinations of situation and emotion and were asked to indicate their agreement or disagreement about whether the statement was true for them. Orthogonal factor analysis of the items indicated that six basic motives were represented in the data. A factor termed "Reduction of Negative Affect" was loaded by items such as "When I feel upset about something, I light up a cigarette" and "Few things help better than cigarettes when I'm feeling upset." The domain of positive affect enhancement was represented by a factor dimension termed "Pleasurable Relaxation," which included items such as "Smoking cigarettes is pleasant and relaxing." This factor was not correlated with any of the other five factors found in the study, indicating that it is an independent functional dimension. A factor concerning addictive smoking, which included items reporting a strong desire or craving for cigarettes, was substantially correlated with the negative affect factor and for that reason is included under the domain of negative affect reduction.

Other studies of smoking motives have replicated the two domains of negative- and positive-affect regulation. Under the general domain of negative affect reduction, McKennell (1970) surveyed a representative national sample of 1,140 adolescents and adults in Great Britain and found that three factors termed "Nervous Irritation Smoking," "Smoking Alone," and "Food Substitution" were strongly intercorrelated, all representing an increased probability of smoking during unpleasant states. Coan (1973) and Leventhal and Avis (1976), in studies with college students, found almost identical factors termed "Negative Affect Reduction" and "Anxiety Reduction," which in each case were substantially correlated with another factor representing addictive smoking. Additionally, Coan (1973) found a factor termed "Distraction," which included items suggesting that smoking was sometimes used as a means of diverting attention from disturbing stimuli. (This self-report is consistent with the discussion of distraction studies presented in the first Section of this Chapter.) Best and Hakstian (1978) surveyed a sample of 331 adult commuters with an inventory about the relative strength of their urge to smoke in each of 63 situations. Intercorrelated dimensions termed "Nervous Tension," "Frustration," "Embarrassment," "Discomfort,"

TABLE l.--Summary of studies of perceived functions of smoking

Domain/Factors	Ikard et al. (1969)	McKennell (1970)	Coan (1973)	Leventhal and Avis (1976)	Best and Hakstian (1978)	Baumann and Chenoweth (1964)
Negative affect reduction	Negative affect reduction	Nervous irritation	Negative affect reduction	Anxiety reduction	Nervous tension	n.a.[1]
	Addictive smoking	Food substitution	Addiction	Addiction	Frustration	
		Smoking alone	Distraction		Discomfort	
			Agitated state		Anger/Impatience	
					Restlessness	
Positive affect enhancement	Pleasurable relaxation	Relaxation	Pleasurable relaxation	Pleasure/Taste	Relaxation	Pleasure
		Social confidence smoking	Dependence on mental state			
			Sensorimotor pleasure			
Other functions	Habitual smoking		Habitual action	Habit	Automatic smoking	Habit
	Stimulation	Activity accompaniment	Stimulation	Stimulation	Sensory stimulation	
	Sensorimotor manipulation		Concentration	Fiddling	Concentration	
		Social smoking		Social reward	Social smoking	Positive peer relationships
			Unpleasant habit		Inactivity/Boredom	
					Time structuring	

NOTE: Factors of comparable content are on the same line.
1n.a.=factors not available because relevant items not in study.

"Restlessness," and "Anger/Impatience" all indicated elevated rates of smoking in different types of negative affect situations.

Under the domain of positive-affect enhancement, findings are less consistent because studies typically included relatively few items on pleasurable aspects of smoking. Despite this methodological limitation, each of the studies contains one or two factors that represent a function of smoking to produce positive affect. A factor termed "Pleasurable Relaxation" found by Coan (1973) indicated smoking in circumstances that were relaxed and comfortable, and comparable factors termed "Pleasure" were found among adults (Leventhal and Avis 1976) and adolescents (Baumann and Chenoweth 1984). In each case, these dimensions were uncorrelated with negative affect factors or with other dimensions found in the study. Factors that were termed "Relaxation" by two investigators (Best and Hakstian 1978; McKennell 1970) represent smoking in conditions where one is alone or wants to cheer up.

The studies have indicated some additional functional dimensions not included within the two affective domains. Some dimensions represent habitual or automatic smoking that occurs without conscious attention. These self-reported dimensions are consistent with the data presented in Chapter IV that address compulsive drug-seeking properties of nicotine and tobacco use. Another common dimension represents smoking to increase stimulation, typically in conditions of inactivity or boredom; sometimes another dimension is included, indicating that smokers report that smoking helps improve concentration (Best and Hakstian 1978; Coan 1973; Leventhal and Avis 1976). This latter perceived effect is discussed in detail in the first Section of this Chapter. Dimensions representing smoking in social situations indicate that smoking occurs primarily at parties or social gatherings, and these factors typically are uncorrelated with affective dimensions.

With regard to individual differences in motives for smoking, there are some consistencies across studies. Amount of smoking tends to be greater for persons scoring high on negative affect reduction (Ikard, Green, Horn 1969; McKennell 1970), although persons scoring high on habitual smoking may have a greater frequency of smoking (Ikard, Green, Horn 1969; Leventhal and Avis 1976). Sex differences are sometimes found in functional dimensions, with females scoring higher on negative-affect reduction (Frith 1971; Ikard, Green, Horn 1969; Ikard and Tomkins 1973), whereas males score higher on habitual, relaxation, or stimulation smoking (Frith 1971; Ikard; Green, Horn 1969; McKennell 1970). Findings on external correlates of motive dimensions indicate that adolescents who score high on the Pleasure dimension are more likely to initiate or increase smoking over time (Baumann and Chenoweth 1984), and adult smokers who score high on Negative Affect reduction are more

likely to relapse after smoking cessation treatment (Pomerleau, Adkins, Perstchuk 1978).

McKennell (1970) found 65 to 75 percent of adults reporting that they perceived smoking to reduce nervous irritation, and comparable levels of endorsement were found for other dimensions of negative- and positive-affect regulation factors. Some data indicate that endorsement rates for habitual, stimulation, sensorimotor manipulation, and social confidence smoking are low in absolute terms (Ikard, Green, Horn 1969; McKennell 1970). A study of young children (Eiser, Walsh, Eiser 1986) found that mood regulation effects of smoking were clearly perceived by subjects in the 7- to 8- and 10- to 11-year-old age ranges; this suggests that perceived functions of smoking may be learned partly by observation rather than through direct experience.

The conclusion from this literature is that in the general population, persons perceive that smoking has functions that are relevant for mood regulation. Persons report that they smoke more in situations involving negative mood, and they perceive that smoking helps them to feel better in such situations. Additionally, smoking is perceived to increase positive mood in some situations. These data do not necessarily indicate that the various functions characterize different types of smokers; rather, they suggest that most functions are salient to an individual but are operative at different times or in different situations. Similar to the discussion of smoking and performance in the first Section of this Chapter, self-reports by smokers that they smoke under stress may indicate direct effects of smoking and nicotine or may reflect effects of smoking deprivation that are relieved by smoking. Whichever interpretation is correct, individuals certainly report that stress is associated with smoking.

Stress and Smoking

There is evidence that stress can increase the likelihood of initiation of smoking if cigarettes are available. Further, considerable evidence exists to link negative-affect states to smoking behavior. The database includes studies of stress as a risk factor for smoking initiation during adolescence and studies on stress and rates of smoking among adults.

Stress and Smoking Initiation

Several studies have shown stress to be related to the onset of smoking in early adolescence. Studies of smoking initiation typically survey a large sample of adolescents beginning at approximately 12 years of age, because the onset of cigarette smoking is greatest during the junior high school period (Fishburne, Abelson, Cisin 1980; Green 1979). Measures of psychosocial risk factors are obtained from

questionnaire scales, and indices of smoking status are usually obtained from self-report by respondents, sometimes accompanied by a biochemical index of smoking. There is evidence indicating that self-reports of smoking by adolescents are generally accurate, although the accuracy of self-report data may be increased by administration of biochemical measures (Murray et al. 1987). Convergent results from cross-sectional and prospective studies show that stress is antecedent to substance use onset and is not a consequence of the initiation of smoking (Gorsuch and Butler 1976; Kandel 1978; Kandel, Kessler, Margulies 1978; Kaplan et al. 1986).

The most direct evidence linking smoking to negative mood states is based on measures of subjective stress. A cross-sectional study by Mitic, McGuire, and Neumann (1985) surveyed a random sample of 1,684 school students in grades 7 through 12 in a medium-sized Canadian community and obtained measures indexing whether students felt nervous, anxious, or worried as a result of 12 potential problem areas. Analyses for the total sample indicated that regular and heavy smokers scored higher on perceived stress, compared with nonsmokers. A related study by Hirschman, Leventhal, and Glynn (1984) employed as the criterion variable a retrospective report of smoking experiences during the previous 2 years. Data were obtained from a stratified sample of 386 students in grades 2 through 10 in a midwestern community. Analyses of data on smoking transitions indicated that a measure of affective distress was related to rapid transitions from experimental to regular smoking. These results were obtained in multivariate analyses with control for other variables including age, peer and parental smoking, and risk-taking tendency.

Comparable findings occurred in a prospective study by Wills (1985, 1986) of a population sample of 675 students in the 7th grade in a New York City school district. Analyses for a 14-item scale of subjective stress reactions showed that high stress was related to increased levels of smoking over a 2-year period. Additional data from this cohort and a replication cohort of 901 students were obtained with measures of everyday negative events and major life events. Multivariate analyses of these data indicated that all three measures of stress were related to smoking, with major negative events being the statistically strongest predictor. These analyses indicated that the effect of stress on smoking was not attributable to other variables including sex, race, locus of control, self-esteem, social activity, and assertiveness. These findings are consistent with laboratory data indicating that females under stress are more willing to try additional cigarettes after an initial smoking experience (Silverstein et al. 1982).

It should be noted that adoption of cigarette smoking has been shown to be a risk factor for subsequent adoptions of other types of

substance use. Although many adolescents who smoke do not become regular users of other drugs, there are typically a concurrent correlation between smoking and other types of drug use (Hays, Stacy, DiMatteo 1984; Single, Kandel, Faust 1974; Revell, Warburton, Wesnes 1986) and a statistical relationship between early cigarette smoking and subsequent use of hard liquor and marijuana (Kandel 1975; Donovan and Jessor 1983). There is no direct evidence linking multiple drug use to mood regulation effects, but it has been shown that negative life events are a risk factor not only for cigarette smoking, but also for several types of other drug use (Bruns and Geist 1984; Kellam, Brown, Fleming 1982; Newcomb and Harlow 1986).

For interpretation of data on stress and smoking in adolescents, the primary methodological issue concerns a possible third confounding variable. It may be that high levels of subjective stress are most prevalent among adolescents who have difficulty adjusting to school and family because of underlying psychopathology (Depue and Monroe 1986) and who identify with the values of a deviant lifestyle that includes substance use and delinquent behavior (Jessor and Jessor 1977). The current evidence argues against this interpretation; some data show that stress-smoking correlations remain significant with control for variables such as risk-taking, perceived control, and self-esteem (Hirschman, Leventhal, Glynn 1984; Newcomb and Harlow 1986; Wills 1985), and it has been shown that negative life events that could not be self-caused by adolescents show an independent predictive relationship to smoking (Wills 1986). The current evidence, however, is minimal and does not clearly rule out the alternative interpretation. At present it can be concluded that subjective stress may be a risk factor for adolescent smoking.

Stress and Cigarette Consumption

In considering evidence on affective factors and cigarette consumption among regular users, both epidemiological and laboratory data are available. Designs in the epidemiological studies are relatively weak because studies are largely cross-sectional, making causal interpretation difficult. When longitudinal data are available, the followup periods are rather short (approximately 1 year) in relation to the probable time course of stress-smoking relationships in adult populations. The following section presents the epidemiological evidence and laboratory studies of stress and smoking.

A large body of personality research has linked measures in the category of "neuroticism" to cigarette smoking among adult populations (Kozlowski 1979). These measures, which include scales of nervousness, emotionality, and anxiety, are conceptually similar to the concept of negative affectivity as defined by Watson and Clark (1984); that is, the tendency to perceive and experience negative

affect. Theoretically, this is the most relevant construct for examining links between affective factors and smoking. Of the 50 studies reviewed by Kozlowski (1979), half showed a significant relationship between neuroticism and smoking. Three studies in this literature showed the relationship between neuroticism and smoking to be more characteristic of females than males (Cherry and Kiernan 1976; Clausen 1968; Waters 1971). These studies were mostly cross-sectional, making inferences of causality problematic because of the possibility that smoking caused feelings of anxiety and depression. Also, Cherry and Kiernan (1976) analyzed longitudinal data and found that neuroticism predicted initiation of smoking by women but neuroticism predicted decreased likelihood of quitting by men. One prospective study (Seltzer and Oechsli 1985) related personality measures obtained at age 10 to smoking status at age 16 in a sample of 1,127 subjects from health maintenance organizations in the Oakland, California, area. The prospective analyses showed that measures of anger, restless sleep, and Type A personality were significantly related to onset of smoking. These analyses were performed with control for parental socioeconomic status and smoking. Measures of neuroticism and anxiety did not discriminate smokers in these analyses.

In the laboratory, smokers tend to smoke more during stressful situations (Epstein and Collins 1977; Rose, Ananda, Jarvik 1983; Schachter et al. 1977). Individuals attempting to quit smoking tend to experience relapses into a state of continued smoking during stressful situations (Shiffman 1986). Such findings are consistent with the self-reported claims of smokers that they smoke in order to reduce stress-induced negative affect. However, there is no convincing research evidence to indicate whether smoking actually reduces stress. It may be that smoking reduces stress relative to smoking deprivation or that smoking increases during stress without attenuating it.

It has been suggested that smokers smoke as a technique to deal with stress (Wills 1985). If smoking is indeed used as a coping mechanism, individuals with poor coping skills and/or with high degrees of chronic stress would be expected to have a higher prevalence of smoking. Three prospective studies have found associations between anxious, aggressive, and generally neurotic personality traits in childhood and the tendency toward smoking later in life (Cherry and Kiernan 1976; Lerner and Vicary 1984; Seltzer and Oechsli 1985). Cross-sectional surveys have repeatedly supported these findings, showing that neurotic, depressed, angry, and rebellious individuals are more likely to smoke compared with more emotionally stable individuals (Spielberger 1986). Ninety percent or more of alcoholics smoke (Istvan and Matarazzo 1984) compared with about 30 percent of the general adult non-alcoholic population in the

United States. Individuals who commit suicide are much more likely to be smokers (Cederlof, Friberg, Lundman 1977; Doll and Peto 1976). It has been argued that individuals with personality disturbances and related psychological problems may, in some cases, be using nicotine as a form of self-medication (Brown 1973; Warburton, Wesnes, Revell 1983). It has also been noted that the symptoms of nicotine withdrawal syndrome are very similar to those of clinical depression (Gilbert and Welser, in press). Emotional and psychological disorders with high incidences of tobacco consumption are characterized by high degrees of negative affect, and it seems likely that, like other tobacco consumers, individuals with such disorders use tobacco as a means of coping with negative affect and stress.

Recent studies have used measures more directly linked to the experience of stress. In a survey of a sample of 505 Navy men on amphibious assault ships, Burr (1984) employed a 19-item measure indexing perceived stress from the domains of job, organization, and family and related the stress scales to a single item about smoking status. Results showed that two scales from the stress measure, indexing Role Conflict and Family Strain, were significant discriminators of smokers and nonsmokers in this sample. These results are cross-sectional, but were obtained in a multivariate analysis that included a measure of locus of control. Similar results were found in a cross-sectional study by Tagliacozzo and Vaughn (1982) in a sample of 448 hospital nurses, using a 26-item inventory of job-related stress. In this study, the stress-smoking relationship was found primarily among respondents who were younger (<28 years) and single. Billings and Moos (1983) studied a community sample of 608 adult respondents in the San Francisco area and found that heavy smokers differed from nonsmokers in showing higher levels of anxiety/depression symptoms and negative life events (during the previous year) in the areas of work strain and family illness. Correlations between stressors and amount of smoking were found primarily for heavy smokers, not for light smokers in this population. These data are consistent with findings from a community sample of 938 adults in New Haven (Lindenthal, Myers, Pepper 1972). This study found that a high level of negative events (during the previous year) was related to increased rates of smoking, with some data suggesting that this effect occurred primarily among persons scoring high on psychological impairment as measured by the Gurin Index. In this study the relationship between stress and smoking held with control for sex, race, age, marital status, and social class.

Only two studies have examined smoking and stress at more than one time point. Conway and associates (1981) studied a sample of 34 Navy officers in a training setting. Data were obtained on stressors and smoking for 14 study days over an 8-month period. The days were categorized by independent raters for stress level; additionally,

subjects made a daily rating on an eight-item scale of mood and subjective stress. Results showed that rates of smoking were significantly correlated with both the daily subjective stress measures and with the objective categorization of days for stress level. Items on perceived stress, anger, fatigue, and fear were significantly related to smoking in the overall sample, but an item on depression was not significantly correlated with smoking. Within-subject analyses of stress-smoking relationships indicated that the significant overall correlations were apparently due to a small number of individuals, but there were no data presented to discriminate these more reactive individuals from other members of the sample. A prospective study by Aneshensel and Huba (1983) was based on longitudinal data from four time periods with a community sample of 742 adult respondents in the Los Angeles area. Data on cigarette smoking, scored on a l-to-5 scale, were obtained at baseline and at a l-year followup interval. Results showed that a baseline measure of depression was not related to smoking either concurrently or over the l-year interval.

The field studies are, for the most part, ambiguous with respect to causal interpretation. This difficulty is alleviated in laboratory studies in which subjects are randomly assigned to conditions and predictor variables are experimentally manipulated. Several studies of stress and smoking in laboratory settings have consistently found that stress increases rates of smoking. The stressors manipulated include threat of electric shock (Schachter et al. 1977), noise (Cherek 1985; Golding and Mangan 1982), and performance anxiety (Rose, Ananda, Jarvik 1983). These latter researchers also employed a concentration task and found that smoking increased in both the anxiety and concentration conditions, compared with a control condition. One study, using a public speaking manipulation, failed to find a significant effect of stress on smoking (Glad and Adesso 1976).

Based on epidemiological and laboratory research, it can be concluded that stress increases the rate of smoking among regular smokers. The convergence of results from cross-sectional, retrospective, and repeated-measures studies, in combination with findings from laboratory research, supports the interpretation of a causal relationship. There is some evidence suggesting that life stress has a greater impact among heavy smokers and among persons scoring high on negative-affect measures, but evidence on individual differences in this literature is minimal. The psychological mechanisms linking stress to increased smoking have not been clearly demonstrated (Leventhal and Cleary 1980; Schachter, Silverstein, Perlick 1977; Pomerleau and Pomerleau 1984). It may be that smoking attenuates stress (e.g., by regulating mood), that smoking increases during stress but does not attenuate it, or that smoking during stress is experienced as less stressful only when compared with smoking

deprivation during stress. Some laboratory studies and substantial theoretical speculations have addressed these issues and are discussed below.

Do Smoking and Nicotine Reduce Stress and Improve Mood?

There is evidence that smoking is perceived as helpful for coping with stress and dysphoric mood. A further question is whether smoking actually reduces stress or improves mood. In epidemiological studies, this question has not been directly addressed, a major limitation in the literature. There are some laboratory studies that bear on this question. This Section summarizes experimental findings concerning the effects of smoking and nicotine on stress and affect modulation.

Self-Reported Stress Reduction and Affect Modulation

Smoking-deprived smokers usually report more negative affect than do smokers who are allowed to smoke if the setting is one which tends to produce mild-to-moderate negative affect. Compared with those deprived for an hour or more, individuals allowed to smoke report less anxiety (Gilbert and Spielberger 1987; Heimstra 1973; Pomerleau, Turk, Fertig 1984; Jarvik et al., in press) as well as less anger and irritation (Cetta 1977; Heimstra 1973; Neetz 1979) during performance of a variety of slightly stressful tasks. Tobacco deprivation is also associated with self-reports of decreased alertness, lessened mental efficiency, and increased boredom during a variety of cognitive tasks (Frankenhaeuser et al. 1971; Heimstra 1973).

Experimental research suggests that nicotine is the most important, and possibly the essential, component of the affect-modulating properties of tobacco use (Gilbert and Welser, in press; Pomerleau and Pomerleau 1984). For example, studies comparing the effects of nicotine-containing gum with no-nicotine placebo gum report that nicotine reduces negative affect in nicotine-deprived habitual smokers (Hughes et al. 1984; Jarvis et al. 1982; West et al. 1984). In addition, habitual smokers assigned to smoke cigarettes of normal nicotine yield report less negative affect than those who smoke very-low-nicotine-yield cigarettes (Gilbert 1985; Perlick 1977).

However, a number of studies have not observed reduced negative affect due to smoking high- versus low-nicotine-yield cigarettes (Bowen 1969; Dubren 1975; Fleming and Lombardo 1987; Gilbert and Hagen 1980; Gilbert 1985; Hatch, Bierner, Fisher 1983). Gilbert and Welser (in press) suggest that these studies included inadequate periods of tobacco deprivation and excessively rapid smoking of multiple cigarettes (probably producing nicotine toxicity). Degree and type of stress to which subjects are exposed may also influence outcomes. There is evidence suggesting that nicotine has stress-

attenuating effects when stressor stimuli are mild or moderate, distal (anticipatory), and ambiguous, but fails to have such effects when stressors are brief, proximal, and/or intense (Gilbert and Welser, in press). More research is needed to evaluate these possibilities.

Behavioral Indices of Stress Reduction and Affect Modulation

A small number of studies that used behavioral indices of affect support the hypothesis that nicotine can reduce negative affect. Several studies report that smoking, or smoking a high-nicotine relative to a low-nicotine cigarette, is associated with reduced aggression (Cherek 1981; Schechter and Rand 1974). However, Jones and Leiser (1976) found no such effects on aggressive behavior by using similar procedures. In addition, without nonsmokers as controls, it is impossible to know whether the differences that were reported between conditions resulted from nicotine administration or nicotine deprivation.

Hughes and colleagues (1984) asked spouses to provide daily ratings of the subjects' behavioral indications of mood. These subjects had abruptly quit smoking and were randomly assigned to chew placebo gum or gum containing nicotine. Subjects who chewed the placebo gum were rated by their spouses as exhibiting significantly more anger and tension after quitting smoking, while those who chewed nicotine polacrilex gum showed little change in these emotional states. Thus, it appears that the nicotine provided by the gum replaced the nicotine previously obtained by smoking, so that there was little change in mood. However, it also appears that nicotine deprivation resulted in the tension and anger and that nicotine did not reduce these variables below baseline values.

Several studies have used pain thresholds as dependent variables in assessing the effects of smoking and nicotine on anxiety. Two studies that tested the effects of smoking cigarettes of different nicotine yield on electric shock endurance report elevated endurance thresholds in subjects who smoked relative to those who did not and in the high-nicotine-cigarette conditions relative to the low-nicotine-cigarette conditions (Nesbitt 1969; Silverstein 1982). The increased willingness to endure electric shock by individuals in the smoking and high-nicotine conditions was interpreted by these investigators and others (Schachter 1973) as indicating that nicotine reduces the anxiety associated with the electric shock. Other studies used the length of time that individuals are willing to endure pain associated with immersion of a hand or foot in ice water (the cold-pressor test) as an indicator of anxiety. These studies also showed that smoking and another means of nicotine administration (snuff) increase endurance in this test. However, the anxiolytic interpretation of increased pain thresholds has been questioned (Gilbert 1979),

because of the observation that in some situations nicotine has been reported to increase detection thresholds for tactile (including electrical) stimuli. It may be that nicotine reduces sensitivity to pain directly, rather than via reduction of anxiety. Several studies have failed to find increased shock endurance thresholds associated with smoking (Jarvik et al., in press; Milgrom-Friedman, Penman, Meares 1983; Shiffman and Jarvik 1984). In addition, it is unclear whether smoking and nicotine reduced these operational estimates of stress or whether smoking deprivation increased them.

Studies of the effects of acute doses of nicotine on behavioral measures of activity in animals indicate that nicotine may reduce negative affect in a number of different species (Bell, Warburton, Brown 1985; Emley and Hutchinson 1983). However, close inspection of the procedures used in these studies reveals that doses that suppress behavioral indices of emotion also may produce nicotine toxicity. Such high doses may decrease a large variety of behavioral indices due to the induction of physical distress. However, Silverman (1971), using doses of nicotine comparable to smoking doses, reported nicotine-induced reductions of aggression. Careful evaluation of studies of the effects of nicotine on indices of emotion in nonhuman subjects indicates that while these studies generally support the view that nicotine has inherent negative-affect-reducing properties independent of withdrawal effects, most have administered such high doses of nicotine as to make their relevance to habitual nicotine use in humans questionable.

Overall, evidence from experimental studies supports survey findings suggesting that tobacco use and nicotine consumption are associated with decreases in negative affect in habitual tobacco users. As was true for the learning and performance literature, caution must be exercised in generalizing about smoking and nicotine's effects on stress and mood because most laboratory studies compare smokers smoking with smokers not smoking. Few studies include the important control group of nonsmokers not smoking to allow unequivocal determinations of whether smoking and nicotine are stress reducing or whether smoking abstinence and nicotine deprivation are stress increasing. Certainly, it seems that smoking by smokers is stress reducing compared with smokers not smoking. The experimental literature suggests that smoking and nicotine may reduce negative affect most effectively in situations involving mild or moderate distal (anticipatory) anxiety and/or ambiguous stressors. The roles that individual differences in personality, temperament, and psychopathology may play in determining the nature or degree of the stress-reducing effects of nicotine are yet to be determined.

Suggested Mechanisms Underlying Nicotine's Effects on Stress and Mood

Based on the extant epidemiological literature linking stress and smoking and the laboratory studies indicating that stress increases smoking, several investigators have offered mechanisms to explain these relationships. These theoretical positions are varied and none has yet received unequivocal support to the exclusion of the other proposed mechanisms. Perhaps several or all of these mechanisms are operating. The major positions are reviewed below.

An Emphasis on Nicotine Withdrawal Symptoms

Schachter (1979) suggested that nicotine reduces negative affect in smokers simply by reducing symptoms of nicotine withdrawal. Increased irritability, anxiety, and depression are the most common symptoms of smoking withdrawal (Murray and Lawrence 1984), and these are the very emotions that appear to be most consistently reduced by acute doses of nicotine in nicotine-deprived smokers (Gilbert and Welser, in press). Thus, alleviation of withdrawal symptoms may account for the capacity of nicotine to reduce negative affect in nicotine-deprived smokers.

The degree to which an individual is physically dependent on nicotine may account for the variable effects observed. Perlick (1977) found that normal-nicotine-delivery cigarettes alleviated annoyance in heavy but not light smokers. On the other hand, the reduction in negative affect following nicotine administration may not be simply and solely a consequence of withdrawal symptom relief, because several investigations showing such effects used minimally deprived individuals who had not developed withdrawal symptoms (Pomerleau 1981).

A variant of this proposed mechanism suggests that smoking increases under stress and in dysphoric mood states because biological and psychological effects of stress and dysphoric moods are similar to the experience of nicotine withdrawal. From past experience, smokers learn that smoking alleviates these unpleasant states. Therefore, stressors and dysphoric moods come to elicit smoking because of conditioned responses or because of misattribution of the unpleasant experiences to nicotine withdrawal (Barefoot and Girodo 1972; Grunberg and Baum 1985). This misattribution model has some empirical support but requires careful examination.

Neurochemical Models

Evidence has been offered in support of the hypothesis that nicotine-induced release of glucocorticoids and other neuromodulators, such as the endogenous opioid beta-endorphin, may account for nicotine's capacity to reduce stress and negative affect (Gilbert 1979;

Pomerleau and Pomerleau 1984). While high doses of nicotine and rapid smoking of cigarettes after a period of smoking deprivation cause reliable increases in plasma concentrations of such neuromodulators (Seyler et al. 1986), it is not clear whether normal smoking during nonstressful conditions causes increases in these neuromodulators (Gilbert and Welser, in press). However, normal smoking in combination with mild-to-moderate stress may result in such increases. In addition, even if such neurochemical changes occur, it is not clear whether they act to modulate stress or dysphoric moods.

Biphasic Action on the Sympathetic Nervous System

Studies of human performance show that performance on simple tasks is improved by higher arousal, but performance on complex tasks is impaired by a high arousal level (Levine, Kramer, Levine 1975). In coping with the varying demands of daily life, at times it may be advantageous to vary the level of sympathetic nervous system (SNS) arousal. The ability to regulate arousal in this fashion would enable individuals to appraise stressful situations as less threatening and could result in improved performance in various conditions. There is some evidence suggesting that nicotine may have biphasic effects on SNS responses, producing either stimulatory effects or dampening effects under different conditions. Under conditions of low environmental demand, the effect of nicotine is generally to produce stimulatory or SNS arousal effects, including increases in heart rate and blood pressure (Grunberg and Baum 1985; MacDougall et al. 1983, 1986). This effect may be responsible for the perceived functions of "stimulation" or coping with "inactivity/boredom" (Best and Hakstian 1978; Coan 1973; Ikard, Green, Horn 1969; Leventhal and Avis 1976), and there is evidence indicating that smoking improves performance on simple tasks (Suraway and Cox 1986; Wesnes and Warburton 1983). At high levels of arousal, however, there is some evidence that nicotine produces central nervous system (CNS) tranquilization effects or reduces reactivity to stressful stimulation (Armitage, Hall, Sellers 1969; Ashton et al. 1974; Golding and Mangan 1982; Woodson et al. 1986). Evidence suggests that nicotine can restore high brain activation to moderate levels. In low-arousal situations, such as vigilance tasks, nicotine produces cortical activation and increased alertness (Edwards et al. 1985). Increased cortical activation could increase hedonic tone directly or indirectly by allowing the individual to perform more effectively on desired tasks and thus to experience indirect rewards such as the perception of increased self-efficacy. In contrast, nicotine has been associated with decreased cortical activation and reduced anxiety in stressful conditions (Gilbert 1985; Golding and Mangan 1982). Nicotine administration by smoking and

other means may allow individuals to achieve a hedonically more desirable level of cortical activation (Eysenck 1972).

At present, there is no direct evidence linking these physiological effects to perceived stress reduction or improved performance under stressful conditions. This position is also consistent with the findings reported in the first Section of this Chapter.

Altered Body Activity

Several mechanisms based on altered body activity may account for nicotine's stress-reducing effects. First, based on evidence that nicotine may in some situations increase the threshold for electric shock (Mendenhall 1925) and on the observation that nicotine-induced increases in cardiovascular activity typically do not produce corresponding increases in perceived heart activity (Gilbert and Hagen 1980), nicotine may reduce the intensity of emotional experiences by increasing perceptual thresholds for emotion-related feelings of bodily arousal (Gilbert 1979). The small number of studies evaluating this hypothesis have provided mixed results (Sult and Moss 1986), possibly because some have not been carried out under conditions of high stress. This elevated perceptual threshold model is consistent with the CNS arousal modulation model and with the neuromodulator model in predicting that under conditions of heightened stress, nicotine should elevate perceptual and pain-endurance thresholds.

A related possibility is that smoking reduces sensitivity to painful stimuli and sensitivity to internal proprioceptive cues that produce discomfort. Antinociceptive action (i.e., reducing perception of pain stimuli) has been documented in several animal studies (Friedman, Horvath, Meares 1974; Sahley and Berntson 1979; Tripathi, Martin, Aceto 1982). Evidence from humans is mixed, with several studies showing that smoking increases tolerance to painful stimuli (Pomerleau, Turk, Fertig 1984; Nesbitt 1973; Silverstein 1982), and the effect is attributable specifically to nicotine intake rather than to the physical act of smoking (Fertig, Pomerleau, Sanders 1986). Several studies have failed to find effects of smoking on pain thresholds (Shiffman and Jarvik 1984; Sult and Moss 1986; Waller et al. 1983). These null results may be attributable to methodological details such as gender differences or differences in current nicotine level.

Another possibility is that nicotine produces a state of tranquillity or relaxation by reducing the level of tonic and/or phasic muscular activity (Gilbert 1979). Experimental evidence strongly supports the view that nicotine depresses certain muscular reflexes (Domino 1979; Hutchinson and Emley 1973). Ginzel and Eldred (1972) and Ginzel (1987) have shown that nicotine produces muscle relaxation in the cat. Epstein and coworkers (1984) have reported that smoking by humans reduces sensitivity to perception of muscle tension.

Schachter (1973) suggests that nicotine reduces emotional experience by reducing emotion-induced phasic increases in autonomic nervous system (ANS) activity. Because nicotine typically increases activation of the ANS, this increase in tonic ANS activation should produce a ceiling effect such that the additional arousal increase associated with the onset of emotional stimulation is less than the emotion-induced arousal that occurs without nicotine. This third hypothesis assumes that phasic, rather than tonic, activation of the ANS is an important contributor to the subjective experience of emotion. Consistent with this possibility, nicotine increases tonic heart rate, but reduces phasic heart rate responses to stressors (Schachter 1973; Woodson et al. 1986).

Hedonic Systems Model

Nicotine-induced modulation of one or more systems in the brain associated with pain and pleasure may account for the capacity of nicotine to reduce negative affect and increase feeling of well-being (Eysenck 1973; Jarvik 1973). Eysenck (1973) suggests that feelings of well-being produced by nicotine and other means can be increased by influencing three hedonic systems: the primary reward, the primary aversion, and the secondary reward systems. Activating the primary system is thought to produce pleasure directly, while activating the secondary reward system produces rewarding effects indirectly, by inhibiting the aversion system. Eysenck suggests that nicotine administered during highly stressful situations may improve mood by means of the secondary system, while nicotine administered during low-arousal conditions may directly stimulate primary reward systems. Any primary rewarding effect of nicotine appears to be very subtle; many smokers and a smaller percentage of nonsmokers report pleasurable stimulant effects following the administration of nicotine (Jones, Farrell, Herning 1978). However, the subjective effects of nicotine appear to depend greatly upon expectations (Hughes et al. 1985); individuals who are not habitual tobacco users typically report that nicotine administered in any form produces unpleasant effects (Nyberg et al. 1982). In addition, the biochemical representation of affective states is not well understood (McNeal and Cimbolic 1986), and these states are a joint function of physiological and psychological factors (Reisenzein 1983; Schachter and Singer 1962). Experimental studies of stressful situations have shown that smoking produces reduction in subjective ratings of anxiety (Jarvik et al., in press; Pomerleau, Turk, Fertig 1984), but several studies have failed to find effects of smoking for subjective anxiety (Fleming and Lombardo 1987; Shiffman and Jarvik 1984) or emotional behavior (Hatch, Bierner, Fisher 1983). It appears that anxiety-reduction effects are observed primarily when smoking occurs before, rather than during, the stressful situation (Gilbert, in press).

Therefore, the anxiety reduction may result from cognitive appraisal rather than from direct reduction of negative affect, but it should be noted that comparable patterns of findings are commonly observed for most anxiolytic medications (Janke 1983).

Regarding positive affect, it has been suggested that effects of nicotine on endogenous opioid systems may relate to experienced pleasure (Pomerleau and Pomerleau 1984). There is some evidence that effects of cigarette smoke on the upper and lower respiratory airways contribute to pleasurable functions of smoking (Rose et al. 1985), but direct evidence of an influence on positive affect has not been demonstrated.

Lateralized Affective Processors Model

The capacity of nicotine to decrease negative affect may stem from its capacity to increase activation of the left cerebral hemisphere compared with the right hemisphere (Gilbert 1985). Lateralized effects on electrocortical (Elbert and Birbaumer 1987; Gilbert 1985; Gilbert, in press) and electrodermal (Boyd and Maltzman 1984) activity have been reported. These electrophysiological studies along with behavioral studies (Gilbert and Welser, in press) suggest that during stressful/high-arousal conditions, nicotine reduces right-hemisphere more than left-hemisphere parietal activation, while during low-stress situations it may activate the right hemisphere more than the left. Activation of the right hemisphere appears to be more related to the experience of negative affect (Davidson 1984), while the left hemisphere is more the biological seat of logical sequential and verbal information processing (Tucker and Williamson 1984). Thus, nicotine-induced reductions of right-hemisphere activation are associated with reductions in negative affect. Consistent with this finding, simultaneous reductions in right-hemisphere EEG activation and in negative affect have been reported while subjects viewed a stressful movie (Gilbert 1985). These lateralized effects may occur as a result of nicotine's influence on one or more relatively lateralized neurotransmitter systems (Gilbert and Hagen 1980). The lateralized effect model suggests a common biological basis for a diverse set of psychological and physiological effects of nicotine.

Hypothalamic Consummatory Drive Model

Both exposure to nicotine and the activity of the hypothalamus are linked to hunger and body weight, as well as to affective, cognitive, and perceptual processes. Stimulation of the ventromedial hypothalamus or deactivation of the dorsolateral hypothalamus produces effects similar to those produced by the administration of nicotine: decreased emotionality, decreased sensitivity to distracting stimuli,

heightened activity level, low taste responsivity, and weight loss (Nisbett 1972). Nicotine withdrawal, as well as lesions of the ventromedial hypothalamus or stimulation of the dorsolateral hypothalamus (Nisbett 1972), leads to the opposite effects: increased emotionality, increased distraction by external stimuli, decreased activity level, increased taste responsivity, and weight gain (Grunberg and Baum 1985; Perlick 1977). There are a number of commonalities between nicotine and food consumption (Grunberg and Baum 1985). Food consumption, like nicotine, reduces anxiety (Schachter 1971), and many individuals smoke (Rose, Ananda, Jarvik 1983) and/or eat more (Morley, Levine, Rowland 1983) when anxious. Nicotine may reduce aspects of the hunger drive (Grunberg and Baum 1985) and may be reinforcing for this reason. The hypothalamic consummatory drive model suggests that consummatory drive reduction by nicotine should reduce the agitation and irritability associated with a high drive state.

Indirect Models: Psychological Enhancement and Sensory Gratification

Nicotine may reduce negative affect indirectly by enhancing cognitive functioning and associated task performance (Ashton and Stepney 1982; Wesnes and Warburton 1978). The effects of smoking and nicotine on performance (reviewed earlier in this Chapter) are consistent with this interpretation. Nicotine may improve affect both directly, by one or several of the mechanisms discussed above, and indirectly, by enhancing certain psychological processes. Moreover, there is evidence that smoking improves visual sensory processing while blunting auditory distracters in humans (Friedman and Meares 1980).

Sensory experiences related to tobacco consumption may contribute to the motivation for its use and its affect and stress-related effects. Some smokers report smoking because they like handling cigarettes, watching smoke, and/or the sensory experience of smoke in the throat and lungs (Russell, Peto, Patel 1974). Experimental studies, although limited in number, have supported the view that sensory factors are important contributors to the satisfaction and craving-reduction associated with smoking (Rose et al. 1985). The strong sensory impact associated with all forms of common tobacco use may also reduce negative affect by providing distraction from negative thoughts and stimulation that relieves boredom (Gilbert and Welser, in press).

Implications for Tobacco Use

Stress is a risk factor for smoking initiation and increases cigarette smoking (e.g., puffs per cigarette) among regular users.

Smoking is stress reducing for many smokers, and nicotine appears to be involved in this effect. It is likely that the effects of nicotine on stress and on mood involve several mechanisms including alleviation of withdrawal symptoms, peripheral muscle relaxation, central neurochemical changes and electrocortical arousal, interaction with consummatory reward systems, and indirect effects such as psychological enhancement and sensory gratification. Future research needs to address and compare the possible mechanisms. Regardless of which mechanisms are operating, the relationship between stress and smoking undoubtedly reinforces habitual tobacco use and may contribute to initiation and relapse.

Tobacco Use, Nicotine, and Body Weight

Cigarette smokers weigh less than comparably aged nonsmokers, and many smokers who quit smoking gain weight (Grunberg 1986a; Rodin and Wack 1984; Wack and Rodin 1982). It has been suggested that some people smoke to prevent weight gain as the result of smoking cessation (Birch 1975; Charlton 1984b; Grunberg 1986a). Therefore, methods to control weight gain following cessation have been recommended (Birch 1975; Ducimetiere et al. 1978; Grinstead 1981; Grunberg and Bowen 1985a). How much weight gain actually occurs following smoking cessation (Albanes et al. 1987; Bosse, Garvey, Costa 1980; Rabkin 1984; Wack and Rodin 1982), the specific mechanisms (i.e., changes in dietary intake, physical activity, and/or changes in resting metabolic rate) responsible for this weight gain (Grunberg 1986b; Hofstetter et al. 1986), and whether weight gain (or fear of weight gain) affects either cessation or relapse efforts (Hall, Ginsberg, Jones 1986; Klesges and Klesges, in press; Kramer 1982) remain controversial. This Section reviews data relevant to the smoking/body weight relationship.

The Relationship Between Smoking and Body Weight

The relationship between smoking and body weight has been extensively examined and reported for more than 100 years (Kitchen 1889; Otis 1884). Human studies can be summarized into two broad areas: (1) cross-sectional evaluations that have compared the weights of smokers, nonsmokers, and in some cases, ex-smokers; and (2) longitudinal, within-subject evaluations that have measured weight changes in smokers, ex-smokers, and nonsmokers over time. The cross-sectional evaluations reported since 1970 are tabulated in Table 2, and the longitudinal studies reported since 1970 are summarized in Table 3. Both tables present the reference and year, a brief description of the sample design, major findings, observed moderator variables (e.g., gender, number of cigarettes per day) for weight, and major limitations of the study. Only studies published

since 1970 are summarized in this Report because there are so many studies and because reviews of earlier investigations (Bosse, Garvey, Costa 1980; Grunberg 1986a) indicate that the results are completely consistent with the studies presented in Tables 2 and 3.

Cross-Sectional Evaluations of Smoking and Body Weight

Of the 28 cross-sectional evaluations presented in Table 2, 25 (89 percent) reported that smokers weigh less than nonsmokers. An additional study (Sutherland et al. 1980) found this relationship for women but not for men and another study (Hjermann et al. 1976) found this relationship for older (45 to 49 years) but not younger (40 to 44 years) men. Only one study did not report an inverse relationship between smoking and body weight, and that study examined visitors to a "health exhibit," a population that may be health conscious and predisposed to making positive health changes (Waller and Brooks 1972). This one discrepant study included a high percentage of cigar and pipe smokers (many of whom do not inhale). While it is difficult to summarize the cross-sectional studies because of differences in reporting techniques, it was found that smokers overall weighed an average of 7.13 lb (range: 2.36 to 14.99) less than nonsmokers.

Because smoking and alcohol consumption are correlated, one study (Williamson et al. 1987) examined, through multivariate methods, the effects of smoking and alcohol consumption on body weight. This study reported that alcohol consumption accounted for approximately 44 percent of the reduction in body weight in women who smoked compared with women who did not smoke. For men, statistical adjustment for alcohol consumption did not alter the weight-lowering effect of smoking.

Cigarette consumption, age, and gender have been adequately evaluated to reach some conclusions regarding their impact on the relationship between smoking and body weight. The effect of cigarette consumption has been parametrically evaluated in eight studies. Six (Albanes et al. 1987; Hjermann 1976; Holcomb and Meigs 1972; Jacobs and Gottenborg 1981; Khosla and Lowe 1971; Lincoln 1970; Stephens and Pederson 1983) of the eight investigations (75 percent) reported a nonlinear relationship. In all of these reports, nonsmokers had the greatest body weights; moderate smokers (typically 10 to 20 cigarettes/day) had the lowest body weights; and some heavy smokers (typically > 20 cigarettes/day) had body weights approaching that of nonsmokers. Two studies (Bjelke 1971; Kopczynski 1972) reported no relationship between level of smoking and weight.

The effect of age on the smoking/body weight relationship was examined in six investigations. Five of six studies (86 percent) (Albanes et al. 1987; Bjelke 1971; Hjermann et al. 1976; Jacobs and

TABLE 2.—Cross-sectional evaluations of smoking and body weight

Study	Design and sample	Major results	Moderator variables	Limitations
Albanes et al. (1987)	12,103 men and women, NHANES II Survey	Smokers weighed 5.95 lb less than nonsmokers, controlled for age, sex; smokers taller and leaner than nonsmokers, based on skinfold	Age: current smokers gained less after age 25 than either nonsmokers or ex-smokers Smoking duration: body mass index decreased with smoking duration increase Smoking rate: moderate smokers leaner than low or high rate smokers	Smoking self-report
Andrews and McGarry (1972)	All 18,631 pregnant women, Cardiff, Wales, 1965–1968	Across all heights, smoking mothers lighter than nonsmokers		Pregnant women only; birth survey record data; actual weight changes not presented
Biener (1981)	274 (174 men, 100 women) ex-smokers, worksite setting	49% women, 39% men gained weight following cessation; quitter approximate average gain: women 11 lb, men 15 lb		Retrospective postcessation gain self-report; no nonsmoker control group
Blair et al. (1980)	183 white male, 284 white female insurance company employees; average age 34	Smokers 2.64–7.5 lb lighter than nonsmokers, 0.88–15.21 lb lighter than ex-smokers; smaller skinfolds for smokers of both sexes than nonsmokers		Small sample size; white office workers only

TABLE 2.—Continued

Study	Design and sample	Major results	Moderator variables	Limitations
Bjelke (1971)	8,638 male, 10,331 female respondents, mail survey, Norway general population "systematic sample"	Used "bulk index" (weight/height^2); both sexes current smokers less bulky than quitters and never smokers	Smoking rate: not related to weight Age: older respondents greater smoker/nonsmoker bulk differences Sex: women greater smoker/nonsmoker bulk differences	Self-report by mail; no weights, no statistical analyses presented
Fehily et al. (1984)	211 nonsmoking, 282 smoking men, aged 45–59, heart disease study	Smokers weighed 7.5–10.3 lb less than nonsmokers, 6.6–9.4 lb less than ex-smokers; pipe/cigar smokers weighed 2.4 lb more than nonsmokers; weight/height^2 index results similar		Small, all white, restricted sample; smoking self-report
Fisher and Gordon (1985)	15% random sample, 10 U.S., Canadian clinics; 2,269 male, 2,105 female whites, aged 20–59, LRC Prevalence Study	Men: smoking nondrinkers weighed 6.6 lb less than nonsmoking nondrinkers; smoking drinkers weighed 2.2 lb less than nonsmoking drinkers Women: smoking nondrinkers weighed 2.2 lb less than nonsmoking nondrinkers; smoking drinkers weighed 4.4 lb less than nonsmoking drinkers		All white population; smoking self-report
Friedman et al. (1981)	38 smoking-discordant monozygotic twin pairs, average age 40 years	Smokers weighed 5.07 lb less than nonsmokers		Self-report by mail; small restricted sample

TABLE 2.—Continued

Study	Design and sample	Major results	Moderator variables	Limitations
Garn et al. (1978b)	17,649 pregnant women, national health survey	Smoking mothers prepregnancy weight less than nonsmoking mothers; difference: whites 2.43 lb, blacks 3.53 lb	SES and race: no smoking/weight relationship influence	Pregnant women only; self-reports
Garrison et al. (1983)	Framingham study participants; assessed 1949–1952	Nonsmokers 55% of highest weight group; smokers 80% of lowest weight group		Sample size, weights not given; no statistical evaluation
Goldbourt and Medalie (1977)	10,059 male government workers, aged 40–65	Current smokers 1/4 inch taller, 2.36 lb less than nonsmokers; ex-smokers in between; leaner skinfolds for smokers than ex-smokers and nonsmokers		Limited age range, employment group; smoking self-report
Gyntelberg and Meyer (1974)	5,249 employed men, aged 40–59, Denmark	Nondrinking smokers 1.5 percentile points lighter than nondrinking nonsmokers; light drinking smokers 2.9 percentile points lighter; heavy drinking smokers 5.9 percentile points lighter than drinking nonsmokers		All-male sample, one city; smoking self-report
Hjermann et al. (1976)	Approximately 18,000 male participants, aged 40–49, coronary risk factor screening, Oslo	Aged 45–49 smokers body weight 3.09 lb less than nonsmokers; aged 40–44 difference not significant; no group weight/height^2 index differences	Smoking rate: heavy smoker (>20/day) body weights higher than lighter smoker Age: older smokers (45–49) weighed less than nonsmokers; younger smokers (40–44) no effect	Smoking self-report; limited age range; one city; all men

TABLE 2.—Continued

Study	Design and sample	Major results	Moderator variables	Limitations
Holcomb and Meigs (1972)	226 manufacturing company male hourly employees, aged 55–59	Mild to moderate smokers 14 lb lighter than never smokers, ex-smokers, and heavy smokers	Smoking rate: heavy smokers (>1 pack/day) heavier than lighter smokers, equivalent to nonsmokers	Smoking self-report; limited age, incomes; all men
Huston and Stenson (1974)	184 men, British Field Regiment	≤10 mm subscapular skinfold men averaged 22 cigarettes/day; ≥15 mm subscapular skinfold men averaged 12 cigarettes/day		Limited male sample; smoking self-report; no separate smoker/ nonsmoker data
Jacobs and Gottenborg (1981)	3,291 white men and women, aged 20–59, no cardiovascular disease or elevated risk factors; randomly selected middle-class suburb census tract blacks	Smokers lighter than never smokers and quitters	Smoking rate: male moderate smokers (14–29 cigarettes/day) 6.39 lb lighter than nonsmokers, 2.65–9.93 lb lighter than light and heavy smokers; female moderate smokers 5.07 lb lighter than never smokers, 1.54–8.38 lb lighter than heavy smokers Age: moderate/never smoker weight difference increased with age	Smoking self-report; restricted population
Khosla and Lowe (1971)	10,482 male steel workers, Wales	Per weight/height^2 index, smokers lighter than nonsmokers	Smoking rate: heavy smokers (>35 cigarettes/day) heavier than moderate smokers (15–34) Age: group weight differences increased after age 35	Smoking self-report; restricted population
Kittel et al. (1978)	8,284 male factory workers, Belgium	Relative weights significantly lower for cigarette smokers than never smokers, ex-smokers, and pipe/cigar smokers		Limited population, risk factor Rx program

TABLE 2.—Continued

Study	Design and sample	Major results	Moderator variables	Limitations
Kopczynski (1972)	3,059 random selectees, pulmonary disease study, Poland	Nonsmokers heavier than smokers, except 20-year-old men	Sex, age, smoking rate: no smoking/weight relationship influence	Smoking self-report; weights not reported
Lincoln (1970)	3,220 male household heads, aged 41–70, across United States	Smokers weighed 3–14 lb less than nonsmokers	SES: smoker/nonsmoker weight difference increased as income decreased Smoking rate: heavy smokers (≥21 cigarettes/day) weighed 4 lb more, moderate smokers (11–20 cigarettes/day) 4 lb less than all-smoker average	Restricted population; men
Matsuya (1982)	90 telephone employees, Japan	Ex-smokers weighed 5.29 lb more than nonsmokers; light smokers 2.87 lb less, heavy smokers 0.44 lb less than ex-smokers		Small, nonrepresentative sample; data self-report
Nemery et al. (1983)	210 steelworkers, aged 45–55, ≥10 years' service, Belgium	Smokers weighed 12.13 lb less than never smokers, 14.33 lb less than ex-smokers		Restricted population; smoking self-report
Stamford et al. (1984a)	164 (56 smokers, 108 nonsmokers) premenopausal women; smokers: ≥20 cigarettes/day, ≥5 years, inhale	Smokers weighed 11.96 lb less, had lower average Quetelet Index than nonsmokers		Small sample size; premenopausal women only; data self-report
Stamford et al. (1984b)	269 adult men, fitness center screened; smokers: ≥20 cigarettes/day, ≥5 years, inhale	Smokers weighed 14.99 lb less, had 12% less body fat than nonsmokers		Select sample, exercising men; smoking self-report; heavy smokers

TABLE 2.—Continued

Study	Design and sample	Major results	Moderator variables	Limitations
Stephens and Pederson (1983)	15,518 persons aged >10; questionnaire, anthropometry	Smokers weighed less than nonsmokers; female smokers weighed 1.32 lb more to 5.73 lb less than female nonsmokers; men weighed 3.09–7.7 lb less; smokers averaged 3.445 lb less than nonsmokers		White women self-report, smoking self-report; no statistical significance tests
Sutherland et al. (1980)	Random sample, 175 men and women, rural town, New Zealand	Weight/height^2 index and skinfolds significantly higher in nonsmoking than smoking women; higher for nonsmoking men, but not significant	Sex: male smokers not significantly leaner than nonsmokers; smoking women lighter than nonsmoking women	Smoking self-report; small sample size
Waller and Brooks (1972)	2,169 health exhibit visitors	"Little weight difference" among current smokers, nonsmokers, and ex-smokers		Smoking self-report; bathroom scale weight; health-conscious population; high % cigar/pipe smokers; no statistical evaluations
Zeiner-Henriksen (1976)	Approximately 15,000 randomly selected Norwegians	Current smokers average and relative weight lower than nonsmokers or ex-smokers		Smoking and weight self-report, questionnaire

TABLE 3.—Longitudinal evaluations of smoking and body weight

Study	Design and sample	Major results	Moderator variables	Limitations
Blitzer et al. (1977)	57,032 women, aged 20–59, self-help weight loss groups	Quitters gained 7.0–10.2 lb more than continuing smokers	Smoking rate: weight gain/previous smoking rate proportional	Smoking and weight self-reports; all women trying to lose weight
Bosse et al. (1980)	1,749 adult men, Normative Aging Study, assessed over 5 years	Average 5-year gains: never smokers 1.81 lb; former smokers 1.87 lb; current smokers 2.00 lb; ex-smokers who quit 6.34 lb	Age: younger quitters gained more Adiposity: fatter quitters gained more Tar rate: higher pretest tar rate smokers gained most Anxiety: high related to higher gain	Smoking self-reports; all men; actual weights not presented
Burse et al. (1982)	4 paid volunteers; 11-day baseline, 21-day quit period, 20-day resumption period	3 of 4 gained weight; 1.98 lb increase during cessation; 1.76 lb loss on resumption		Very small sample, paid volunteers; short-term evaluation
Cambien et al. (1981)	1,097 Paris civil servants, aged 25–35, screened, randomly assigned, cardiovascular risk factor reduction intervention or control groups; 2-year followup evaluation	Treatment group quitters gained 4.85 lb, control group quitters 7.50 lb; nonsmokers and no-change smokers gained 1.54 lb in treatment group, 2.2 lb in control		Smoking self-report; risk factor reduction program participants
Carney and Goldberg (1984)	13 women, 5 men, aged 28–67, smoked ≥ 20 cigarettes/day, ≥ 5 years; 12 male controls; 15 smokers abstained 2 weeks	Quitters weight change range: −3.09 to +9.0 lb	Smoking rate/duration: no weight change relationship Biological variables: weight gain positively related to lipoprotein lipase activity in adipose tissue	Smoking self-report; controls weight changes not reported; short-term evaluation

TABLE 3.—Continued

Study	Design and sample	Major results	Moderator variables	Limitations
Coates and Li (1983)	373 male asbestos-exposed smokers, aged ≥42; 87% white, mean education 12.8 years; 12 months assessment after cessation effort	Continuous quitters gained 5.15 lb; continuous smokers gained 0.35 lb		Smoking self-report; all male, nonrandom sample
Comstock and Stone (1972)	502 male telephone workers, aged 40–59, mostly white; 2 assessments 5 years apart	5-year followup average gains: never smokers 2.43 lb, ex-smokers 5.07 lb, continuing smokers 2.42 lb; quitters 11.24 lb and showed greatest skinfold increases	Smoking rate: increasing quitter weight gain with heavier prequit smoking	Smoking self-report; men only
Dallosso and James (1984)	16 (8 men, 8 women) antismoking clinic participants; mean age, men 47.1, women 35.4; assessed before and 6 weeks after clinic	10 quitters gained 3.00 lb; 5 continuing smokers lost 0.99 lb		Small sample size; smoking self-report; limited followup
Emont and Cummings (1987)	125 stop-smoking clinic participants; pretreatment and 1-month followup assessments	76% quitters and slippers (≤5 cigarettes/day) averaged 5.8 lb gain	Nicotine gum: gain/gum use reliable negative correlation for heavy smokers: gain not related to age, sex, marital status, baseline body weight	Weight gain, smoking self-report, confounded by gum use; limited followup; incomplete data
Fagerström (1987)	28 nicotine gum users; abstinent at 6 months	Infrequent gum users gained 6.83 lb, frequent users 1.98 lb	Nicotine gum: frequent users gained less weight	Small sample size; measures unclear
Friedman and Siegelaub (1980)	Multiphasic health checkup patients; smoked, then quit 12–18 months later (N=3,825) or continued (N=9,392)	Quitters gained 2–3 lb more than continuing smokers	Smoking rate: higher initial smoking rate related to greater weight gain after cessation	Smoking self-report; whites only data

TABLE 3.—Continued

Study	Design and sample	Major results	Moderator variables	Limitations
Garn et al. (1978b)	6,979 women followed through ≥2 pregnancies	Higher prepregnancy weights for habitual nonsmokers than habitual smokers: whites 3.4 lb, blacks 4.1 lb; lower habitual smoker gains between pregnancies for both races	Race: no weight/smoking relationship influence	Smoking self-reports; restricted population
Garvey et al. (1974)	870 white male veterans, aging study, assessed 4–7 years after initial assessment	Smoking/weight change significantly related; recent quitters (≤5 years) gained 4.19 lb more than smokers, nonsmokers, former smokers	Age: 40–54 quitter weight increase greatest	Smoking self-report; exact quit date unknown
Glauser et al. (1970)	7 male smokers, cessation program; assessed preprogram, 1 month postprogram	At 1-month followup, participants gained 6.4 lb		Smoking self-report; exact quit date unknown
Gordon et al. (1975)	4,798 Framingham study participants: 1,498 male smokers, 492 male nonsmokers, 1,634 female nonsmokers, 1,174 female smokers; examined short-term changes after biennial exam 1, long-term effects between biennial exams 4, 10	At entry, male smokers weighed 8.0 lb less than nonsmokers; short-term male quitters gained 3.8 lb, nonsmokers 0.5 lb, continuing smokers 0.3 lb; new smokers lost 9 lb; too few female quitters to evaluate		Smoking self-report; change analysis, men only
Gormican et al. (1980)	301 pregnancy obstetrics records, women, aged 17–35	Smoker, nonsmoker prepregnancy weight similar; no last 2 trimester weight gain difference (nonsmokers 24.6 lb, smokers 22.6 lb)		Clinic record data; pregnancy weight gain data only

TABLE 3.—Continued

Study	Design and sample	Major results	Moderator variables	Limitations
Grinstead (1981)	45 subjects (38 women, 7 men), average age 40; evaluated 6 months after cessation treatment; saliva thiocyanate verification	During program, 63% subjects averaged 2.88 lb increase, 34% averaged 2.46 lb decrease; at followup, 37% averaged 6.97 lb gain, 43% averaged 3.27 lb loss		Questionnaire, phone interview data
Gritz et al. (in press)	554 self-quitters (245 men, 309 women), mean age 41.4, 85% Caucasian, 9% black, 4% Asian, 1% Asian-American, 1% Native American; 1-year followup	35% previous quitters gained, 3% lost; at 1 year, abstainers averaged 6.1 lb gain; relapsers gained 2.71 lb while abstinent, lost 1.3 lb upon relapse; continuous smokers gained 0.3 lb		Questionnaire, phone interview data
Grossarth-Maticek et al. (1983)	1,353 subjects, Yugoslavian village of 14,000; every 2d household oldest member; evaluated 1965–1966, 1969	Smoking reduction/weight increase relationship (regression coefficient -0.30)		Smoking self-report; weights, weight changes not reported
Gunn and Shapiro (1985)	89 cessation clinic participants; all quit at initial evaluation; 3-month followup assessment	43 of 54 (80%) quitters gained 2–30 lb		Smoking, height, weight self-report; inadequate statistical evaluation
Hall et al. (1986)	255 smoker participants (122 men, 133 women), 2 smoking treatment trials; 6-, 12-month followups; biochemical verification	Abstainers gained more than smokers at 1 year	Smoking rate: pretest smoking level/postcessation weight gain positively related Chronic dieting: chronic diet subjects gained most	Multiple Rx (e.g., nicotine gum) participant data included
Hatsukami et al. (1984)	27 smokers hospitalized 7 days; 20 subjects smoked 3 days, then quit 4 days; 7 control group subjects smoked throughout	Quitters gained 1.76 lb in 4 days		Small sample size; inpatient environment

TABLE 3.—Continued

Study	Design and sample	Major results	Moderator variables	Limitations
Haworth et al. (1980)	536 women (234 nonsmokers, 302 smokers) interviewed last prenatal visit (18%) or within day after delivery (82%)	No smoker/nonsmoker pregnancy weight gain difference		Smoking self-report; pregnancy weight gain data only
Hickey and Mulcahy (1973)	150 men (124 smokers); 6-month, 2-year followups after myocardial infarction	Quitter, reducer, continuing smoker differences not significant		Smoking self-report; postmyocardial infarction may motivate healthy behavior
Holme et al. (1985)	16,202 Oslo men, aged 40–49, screening program; 1,232 (elevated cholesterol or upper quartile coronary risk score) randomly assigned diet/smoking intervention or control; 5-year followup	17% controls, 24% intervention quit; 1- to 2-year-quitter weight increased more than controls, then decreased to below prequit level		Smoking self-report; confounded by high cardiovascular disease risk health intervention; weights not reported
Howell (1971)	Retrospective, 1,121 men, aged 40–54; 15- to 20-year weight gain examinations	Light smokers (<20 cigarettes/day) gained 1.9 lb less than heavy smokers, 3.1 lb less than ex-smokers, 3.6 lb less than never smokers	Smoking rate: lower rate related to less weight gain	Retrospective report
Hughes and Hutchinson (1983)	37 smokers and 19 ex-smokers with pulmonary emphysema followed ≥3 years	Smokers lost 0.32 lb/yr, ex-smokers gained 1.17 lb/yr; significant difference		Smoking self-report; pulmonary emphysema population

TABLE 3.—Continued

Study	Design and sample	Major results	Moderator variables	Limitations
Jenkins et al. (1973)	2,318 men (546 never smokers, 359 previous quitters, 547 light smokers, 866 heavy smokers), aged 39–49, 11 California corporations in Western Collaborative Group Study; changes assessed since age 25; 1960–1969 study	Weight loss more likely for light and heavy smokers than never smokers and quitters		Smoking self-report; weights not presented
Kramer (1982)	175 subjects, commercial cessation program (41 nonparticipants or nonlocated, 59 quitters, 75 continuing smokers) ≥1-year followup	76% nonsmokers, 56% smokers gained weight; these smokers mean gain 1.7 lb, these nonsmokers mean gain 3.0 lb		All data self-report; high attrition, data loss; presentation incomplete
Lund-Larsen and Tretli (1982)	12,329 men and women, aged 20–49, cardiovascular disease project; 2 screenings 3 years apart	Smokers mean and relative weight less than nonsmokers; female quitters gained 5.95 lb, male quitters 7.84 lb; smoking-starter men lost 1.98 lb, women 5.5 lb; smokers and nonsmokers little/no change	Sex: men, women weight change/smoking cessation and initiation similar	Self-report
Manley and Boland (1983)	39 male, 55 female smokers, cessation program; randomly assigned, 1 of 3 4-week treatments or attention placebo control; 3-month followup; CO verification	31% abstinent at followup: abstainers averaged 10.93 lb gain, relapsers 6.92 lb		Relapser definition unclear

TABLE 3.—Continued

Study	Design and sample	Major results	Moderator variables	Limitations
Noppa and Bengtsson (1980)	1,302 Swedish women, aged 38–60	Current smokers leaner than nonsmokers; At 6 years, quitters gained 7.72 lb; smoking-starters lost 1.54 lb, nonchangers gained 3.09 lb		Smoking self-report
Pincherle (1971)	222 upper-class male quitters; followup $\geq$1 year after first visit	28% gained weight; 22% lost		Smoking self-report; limited population; incomplete report; no weights presented
Powell and McCann (1981)	29 women, 22 men, 5-day cessation project; 2- and 6-month followup	At 2 months, 54% gained weight, range 3–20 lb, mean 8.96 lb; all subjects mean 4.69 lb		Smoking self-report; no separate abstainer, smoker data; small sample size
Puddey et al. (1985)	66 cessation program volunteers, pair-matched by age, sex, body mass index; randomly assigned experimental, control groups; 2-week baseline, 6-week treatment, 6-week followup; thiocyanate, CO verification	14 quitters gained 3.97 lb; controls 0.44 lb		Small sample size
Rabkin (1984)	40 male, 67 female smokers, assigned to 3 cessation groups; followup 3 weeks post-completion; biochemical verification	67.3% gained weight, average 1.76 lb; skinfold increase 6.6 mm	No age, age at smoking start, rate, relative weight, anxiety correlation to male or female weight change	Small sample size; weight self-report

TABLE 3.—Continued

Study	Design and sample	Major results	Moderator variables	Limitations
Rantakallio and Hartikainen-Sorrin (1981)	12,068 pregnant women, n. Finland, 1966; 15% smokers (smoked after 2 months pregnant); nonsmoking controls matched for age, parity, place of residence, marital status	No smoking/nonsmoking pregnancy weight gain difference		Pregnant women only; smoking self-report; pregnancy weight gain data only
Rush (1974)	162 low-income urban pregnant women, no known medical problems, <140 lb preconception weight; had borne low birthweight infant; randomized controlled nutritional supplementation trial	Mean pregnancy weight gain lower for smokers (0.73 lb/wk) than nonsmokers (0.90 lb/wk)	Smoking rate: higher rate related to lower pregnancy weight gain	Pregnant women only; smoking self-report; pregnancy weight gain data only
Schoenenberger (1982)	4,421 male MRFIT volunteers, aged 35–57, good health but upper 10–15% coronary risk factor score; randomly assigned to intervention or control groups; followup 3 annual visits	With MRFIT intervention, significant body weight decrease in smokers (mean 4.6 lb), nonsmokers (mean 5.8 lb), reducers (mean 3.75 lb); quitters average weight change minimal (mean 0.55 lb)		Smoking self-report; confounded by risk factor reduction program participation; restricted population
Seltzer (1974)	794 adult white male veterans, average age 45; Normative Aging Study; screened for "high" health level, geographic stability; 214 screened at 5 years	At admission, ex-smokers 5.9 lb heavier than nonsmokers, 8.1 lb heavier than current smokers; at 5 years, quitters gained 8.0 lb, continuing smokers 3.5 lb		White veterans; smoking self-report

TABLE 3.—Continued

Study	Design and sample	Major results	Moderator variables	Limitations
Stamford et al. (1986)	13 sedentary women, 48-day successful quitters; 1-year followup	At 48 days, weight increased 4.85 lb; at 1 year, quitters increased 18.07 lb; 3 relapsers reduced weight to baseline levels; per hydrostatic weighing, gain was 96% fat		Small female sample; smoking self-report
Tuomilehto et al. (1985)	10,940 cardiovascular disease prevention program participants, aged 25–59, random sample, e. Finland; selectees with high blood pressure or hypertensive medicine assessed 5 years apart; smoking data from 2,264	Quitters body mass increased 2.31 lb/m^2; starting smokers decreased 1.46 lb/m^2		Smoking self-report; hypertensives
Vandenbroucke et al. (1984)	3,091 Netherlands civil servants, spouses (1,583 men, 1,508 women), aged 40–65, general health exam; 25-year followup	76.6% lean, 65.1% obese men smoked; 22.1% lean, 11.3% obese women smoked		Smoking self-report; restricted population

Gottenborg 1981; Khosla and Lowe 1971) documented increasing weight differences between smokers and nonsmokers with advancing age. Typically, aging smokers failed to gain as much weight as aging nonsmokers.

Three evaluations systematically compared males with females (Bjelke 1971; Kopczynski 1972; Sutherland et al. 1980). Two of the three (Bjelke 1971; Sutherland et al. 1980) reported the differences in body weight between smokers and nonsmokers to be greater in females than in males.

Longitudinal Evaluations of Smoking and Body Weight

Table 3 presents the results of 43 longitudinal evaluations of the effects of smoking on body weight. Consistent with the cross-sectional evaluations, the overwhelming majority (86 percent, 37 of 43) present evidence that smokers who quit smoking gain weight, that people who quit smoking gain more weight than nonsmokers, and that people who initiate smoking lose weight relative to nonsmokers. Of the six studies that did not find these relationships, three limited their examination to smoking and weight changes in pregnant women (Gormican, Valentine, Satter 1980; Haworth et al. 1980; Rantakallio and Hartikainen-Sorri 1981), two relied on participants making broad cardiovascular risk factor reduction efforts in subjects at high risk for cardiovascular disease (Hickey and Mulcahy 1973; Holme et al. 1985), and the remaining study supplied incomplete reports of the data (Kramer 1982). Of those studies on the effects of smoking cessation on weight, the length of followup ranged from 4 days to 7 years. According to these investigations, those who quit smoking gained an average of 6.16 lb (range: 1.76 to 18.07) during the year after cessation.

Daily cigarette consumption was the only moderator variable that received sufficient attention in this group of studies reaching specific conclusions. Seven of nine studies (78 percent) (Blitzer, Rimm, Giefer 1977; Bosse, Garvey, Costa 1980; Comstock and Stone 1972; Friedman and Siegelaub 1980; Hall, Ginsberg, Jones 1986; Howell 1971; Rush 1974) reported a positive relationship between cigarette consumption and weight change; that is, as pretest cigarette consumption increased, postcessation weight gains also increased. Two studies (Carney and Goldberg 1984; Rabkin 1984) did not find a relationship between cigarette consumption and postcessation weight gain.

In summary, there is substantial evidence of an inverse relationship between cigarette smoking and body weight. Of 71 studies reported since 1970, 62 (87 percent) collectively indicate that smokers weigh less than nonsmokers and that people who quit smoking gain weight. Older smokers, females, and those smoking approximately one pack of cigarettes/day may experience the

largest weight control effects of cigarette smoking. Smokers who smoke heavily tend to gain the most weight following smoking cessation. These generalizations are consistent with reviews based on other studies reported since 1880 (Grunberg 1986a). Not all smokers who quit smoking gain weight. Further, for ex-smokers who do gain weight, the amount of weight infrequently poses a serious health risk.

The Role of Nicotine

Animal studies indicate that nicotine administration results in weight loss or decreased weight gains and that cessation of nicotine results in body weight gains greater than those of controls (Bowen, Eury, Grunberg 1986; Grunberg 1982, 1985, 1986b; Grunberg, Bowen, Morse 1984; Grunberg, Bowen, Winders 1986; Grunberg, Winders, Popp 1987; McNair and Bryson 1983; Morgan and Ellison 1987; Schechter and Cook 1976; Wager-Srdar et al. 1984; Wellman et al. 1986). Most of these studies report inverse dose-response relationships between nicotine and body weight.

Recent research on nicotine polacrilex gum with humans corroborates the role of nicotine in body weight effects. Fagerstrom (1987) reported that subjects who quit smoking were much less likely to gain weight when they consistently used nicotine polacrilex gum. Abstinent subjects who regularly used the gum gained less than 2 lb at a 6-month followup. In contrast, the infrequent gum users gained almost 7 lb ($p<0.05$). Emont and Cummings (1987) reported a significant negative relationship ($r=-0.37$) between the number of pieces of nicotine polacrilex gum chewed per day and weight gain for heavy smokers (>26 cigarettes/day). No such relationship between gum use and weight gain was observed for lighter smokers (<26 cigarettes/day).

Mechanisms Underlying The Relationship Between Smoking and Body Weight

The inverse relationship between smoking and body weight may result from changes in energy intake, changes in energy expenditure, or both. Energy intake involves dietary intake. Energy expenditure is affected by behavioral factors (physical activity) and biological factors (e.g., metabolism). These potential mechanisms are examined below.

Dietary Intake

Several prospective investigations have evaluated dietary intake changes following smoking cessation in humans. Hatsukami and coworkers (1984) hospitalized 27 smokers for a 7-day period. After a 3-day baseline, 20 of the subjects were deprived of smoking for 4 days

while the remaining 7 served as a control group. During this 4-day period of abstinence, caloric intake increased significantly (from 1,397 to 1,651 kcal), which corresponded with a significant 1.76-lb increase in weight. In the most comprehensive study to date, Stamford and coworkers (1986) evaluated changes in dietary intake, physical activity, and resting metabolic rate in 13 sedentary females who quit smoking for a 48-day period. Following smoking cessation, mean daily caloric consumption increased by 227 kcal, which accounted for 69 percent of the variance in postcessation weight gain (4.85 lb). Robinson and York (1986) followed 11 smokers who quit for 7 days. Mean dietary intake significantly increased, but changes in resting metabolic rate were not observed. Dallosso and James (1984) followed 10 subjects for 6 weeks after they participated in a stop-smoking clinic. There was a 4-percent drop in resting metabolic rate in smokers who quit, a drop which was reliable when the data were expressed per kilogram of body weight. The average dietary intake increased, by 6.5 percent, but this difference did not reach statistical significance.

Preliminary results of a recent investigation indicate gender differences in the effects of short-term smoking cessation on body weight and food intake (Klesges, Meyers et al. 1987). Female smokers who quit for 1 week increased their body weight and dietary intake significantly more than male smokers who quit. This sex difference is consistent with animal studies (Grunberg, Bowen, Winders 1986; Grunberg, Winders, Popp 1987). Given females' marked concerns regarding postcessation weight gain (Klesges and Klesges, in press), future studies will need to investigate possible gender differences in response to smoking cessation.

Several studies indicate that smokers may differ from nonsmokers in their intake of sweet-tasting simple carbohydrates (sugar) in particular. In a human laboratory study, Grunberg (1982) observed that smokers who were allowed to smoke ate less sweet food than smokers who were not allowed to smoke or nonsmokers. Smokers not allowed to smoke also reported the greatest preference for sweet foods. There were no differences among the three subject groups in consumption of other types of foods. Rodin (1987) conducted a prospective study in which food intake after smoking cessation was carefully evaluated. Smokers who gained weight after quitting smoking increased their sugar consumption in particular. Further, smokers increase consumption of sweet snack foods when they are deprived of cigarette smoking (Duffy and Hall, in press; Perlick 1977). On the other hand, two early investigations (Bennett, Doll, Howell 1970; Richardson 1972) found *higher* sugar consumption in smokers relative to nonsmokers. However, Richardson (1972) found that this difference was because of low-sugar intake in ex-smokers, while Bennett, Doll, and Howell (1970) argued that the differences

were largely due to increased added sugar intake because of hot beverage consumption. These two studies, which are inconsistent with the more recent studies, did not carefully measure all food intake and did not assess intentional changes in food intake to control body weight.

Several animal experiments have documented that food intake decreases during nicotine administration and increases after administration has ceased and that these changes in food intake correspond with changes in body weight (Bowen, Eury, Grunberg 1986; Grunberg 1982; Grunberg, Bowen, Winders 1986; Levin et al. 1987; McNair and Bryson 1983; Wager-Srdar et al. 1984). Consumption of sweet foods by male rats is particulary affected by nicotine (Grunberg 1982; Grunberg et al. 1985). However, nicotine also reduces bland food intake in female rats and has a greater effect on body weight of female rats than of male rats (Grunberg, Winders, Popp 1987; Grunberg, Bowen, Winders 1986; Levin et al. 1987).

Several investigations have reported that changes in body weight in animals also occur without observing decreases in food intake as the result of nicotine administration (Grunberg, Bowen, Morse 1984; Schechter and Cook 1976; Wellman et al. 1986). In one investigation, chronic exposure to cigarette smoke reduced body weight and food intake in rats; however, hamsters exposed to cigarette smoke decreased body weight without reducing food intake (Wager-Srdar et al. 1984). Several methodological factors complicate these results, including the use of different strains of animals, different routes of administration and dosages of nicotine, and whether acute versus chronic effects of nicotine were reported. However, these results indicate that more than the mechanism of food intake was involved in producing nicotine- and smoking-related weight changes.

Data from short-term human studies and several animal experiments indicate that dietary intake is involved with smoking-related energy imbalance. Based on self-reported cross-sectional surveys, it has been reported that smokers' dietary intake is the same as (Albanes et al. 1987; Fehily, Phillips, Yarnell 1984; Fisher and Gordon 1985; Matsuya 1982) or significantly higher than (Picone et al. 1982; Stamford et al. 1984a,b) that of nonsmokers while the smokers simultaneously maintained a lower body weight. Assuming that smokers are not consistently biased in their reports of dietary intake, it appears that either differences in physical activity or metabolic rate are maintaining the body weight differences between smokers and nonsmokers.

Physical Activity

The data available from cross-sectional investigations, short-term prospective studies, and animal investigations seem to indicate that changes in physical activity do not play a role in either differences in

body weight between smokers and nonsmokers or the weight gain associated with smoking cessation. Some cross-sectional investigations have found that smokers have *lower* levels of physical activity compared with nonsmokers (Kannas 1981). Others have not found differences in physical activity and physical fitness between smokers and nonsmokers (Gyntelberg and Meyer 1974; Stamford et al. 1984b; Stephens and Pederson 1983). A recent review (Blair, Jacobs, Powell 1985) that addressed the relationships among exercise, physical activity, and smoking concluded that smoking and physical activity are negatively associated; however, the relationship was extremely weak and variable.

Animal studies on the relationship between nicotine and physical activity have generally found that physical activity plays a small role or fails to correspond to decreases in weight during nicotine administration (Bowen, Eury, Grunberg 1986; Cronan, Conrad, Bryson 1985; Grunberg and Bowen 1985b). One study found that decreases in physical activity after cessation of nicotine appeared to contribute to postdrug body weight increases (Grunberg and Bowen 1985b), but this effect was quite small and occurred only in males.

A few prospective human investigations have evaluated physical activity changes following smoking cessation (Hatsukami et al. 1984; Hofstetter et al. 1986; Klesges, Brown et al. 1987; Rodin 1987; Stamford et al. 1986). These investigations found no changes in physical activity as a result of smoking cessation.

Metabolic Rate

Metabolic rate is an important consideration in energy imbalances associated with smoking cessation because approximately 75 percent of total energy expenditure is in the form of metabolism (Bernstein et al. 1983; Ravussin et al. 1982). Metabolism increases as the result of acute nicotine administration and immediate effects of smoking (Ghanem 1973; Ilebekk, Miller, Mjos 1975; Robinson and York 1986; Schievelbein et al. 1978; Wennmalm 1982). The major question, however, is whether these effects persist long enough to have a direct impact on body weight. Given that (1) smokers do not have higher levels of physical activity compared with nonsmokers (Blair, Jacobs, Powell 1985), (2) some studies report smokers' dietary intakes are the same as or higher than those of nonsmokers (Picone et al. 1982; Stamford et al. 1984a,b), and (3) smokers maintain lower body weights than nonsmokers, it is reasonable to postulate that changes in metabolism contribute to the relationship between smoking and body weight. Additionally, there are several reports in the literature on animals that have documented nicotine-induced reductions in body weight without a concomitant reduction in food intake (Grunberg, Bowen, Morse 1984; Schechter and Cook 1976; Wellman et al. 1986).

Direct evidence supporting a chronic metabolic mechanism that modulates the smoking/body weight relationship is beginning to emerge. Metabolic rate was chronically measured in a study of rat and hamster exposure to cigarette smoke (Wager-Srdar et al. 1984). Higher resting metabolic rates were observed on only one of the test days compared with the pretest in the rat investigation, while no significant differences were observed in the hamster study. Another recent investigation (Wellman et al. 1986) evaluated brown adipose tissue (BAT) thermogenesis at different levels of nicotine and caffeine injections. No differences in BAT thermogenesis were observed in response to either nicotine or caffeine. The group that received a combination of caffeine and nicotine showed a 63 percent increase in BAT thermogenesis.

The few studies that have evaluated metabolic rate changes in response to smoking cessation in humans have produced inconclusive results. Three investigations found metabolic changes after cessation in human smokers. An early report (Glauser et al. 1970) found decreases in oxygen consumption for seven male subjects who quit smoking for 1 month (neither food intake nor physical activity was monitored). A more recent investigation found a 4-percent drop in metabolic rate (reliable when data were expressed per kilogram of body weight) and no significant increase in dietary intake for 10 subjects who quit smoking for 6 weeks (Dallosso and James 1984). In the only study that used a respiration chamber, Hofstetter and others (1986) reported that total energy expenditure was 10 percent higher during a 24-hr period of smoking versus a 24-hr period of abstinence in eight smokers. No changes were observed in physical activity or mean basal (sleeping) metabolic rate (dietary intake was held constant). However, this difference in energy expenditure disappeared after 24 hr.

Three investigations did not find a change in metabolic rate as the result of smoking cessation. Burse and associates (1982, 1975) did not observe changes in resting metabolism in a sample of four smokers who quit for 3 weeks. This investigation did find reliable increases in desire for food, however. In another study, 11 smokers were studied after a 7-day period of smoking abstinence (Robinson and York 1986). Total energy expenditure following a meal did not change during the cessation period. Stamford and colleagues (1986) failed to find changes in oxygen consumption in 13 subjects who quit smoking for 48 days. This investigation did find marked dietary intake changes that accounted for 69 percent of the variance of postcessation weight gain.

There are several possible explanations for the inconsistency observed in the literature on metabolic rate. Different investigators have used different criteria (e.g., resting oxygen consumption, BAT thermogenesis) for operationalizing metabolism. It is possible that

previous dieting history (Brownell et al, 1986) and the use of nicotine polacrilex gum (Fagerstrom 1987) may directly impact the metabolic response to smoking cessation. It is not clear what the metabolic response to nicotine with added agents is likely to be. For example, one study found that while neither nicotine nor caffeine alone produced a change in BAT thermogenesis, the two combined increased thermogenesis by 63 percent (Wellman et al. 1986). This finding is particularly interesting given that smokers may be more likely to drink caffeinated beverages than nonsmokers (Blair et al. 1980). Finally, the available literature on human studies used very small subject groups, making it impossible to detect subtle but potentially meaningful changes in resting metabolic rate. The small sample sizes do not allow for an evaluation of variables that may potentially moderate the metabolic response to smoking cessation.

Summary of Mechanisms Literature

Changes in dietary intake appear to be involved in weight gains after cessation of smoking or cessation of nicotine administration. Physical activity plays little or no role in the relationship between smoking and body weight. The data on metabolic contributions to postcessation weight gain are suggestive, but further research is needed. Unfortunately, much of the relevant human research literature is characterized by small sample sizes, short followup evaluations, and inadequate evaluations of energy balance following smoking cessation. To date, only one investigation has comprehensively evaluated (i.e., simultaneous assessment of dietary intake, physical activity, and metabolic rate) energy balance changes as the result of smoking cessation. This was a sample of 13 sedentary females followed for 48 days (Stamford et al. 1986). Comprehensive, prospective evaluations of energy balance changes in response to smoking cessation are needed. Additionally, no study has evaluated possible long-term changes in dietary intake, physical activity, and metabolic rate as a result of smoking cessation. The longest followup period reported in the literature to date is 2 months (Dallosso and James 1984). Finally, evaluation of potential moderator variables of dietary intake, physical activity, and metabolic rate as the result of cessation is needed. Gender (Grunberg, Winders, Popp 1987; Klesges, Meyers et al. 1987), previous dieting history (Brownell et al. 1986; Hall, Ginsberg, Jones 1986), pretest levels of lipoprotein lipase (Carney and Goldberg 1984), and the use of nicotine polacrilex gum (Fagerstrom 1987) appear to be important variables influencing weight gain and need further investigation.

Does the Relationship Between Smoking and Weight Promote Either the Initiation or Maintenance of Smoking Behavior?

Some research attention has been given to body weight as a potential moderator of smoking initiation, maintenance, and cessation. Unfortunately, many investigations do not report weight-related issues (Borkon, Baird, Siff 1983; Eiser et al. 1985; Pederson and Lefcoe 1976; Perri, Richards, Schultheis 1977). The investigations that have evaluated these issues consistently report relationships between body weight and smoking initiation (Charlton 1984a) and maintenance (Klesges and Klesges, in press).

A survey of 16,000 school children (Charlton 1984a) in England found that the heaviest regular smokers were the most likely to agree that smoking controls weight (42.2 percent) compared with those students who never smoked (16.6 percent). Agreement increased with increased levels of smoking. More girls than boys agreed with this statement, and girls were also more likely to be regular smokers. Charlton (1984b) also reported that among the perceived effects of smoking, smokers viewed "calming the nerves" as the most popular reason (72 percent) followed by "smoking keeps your weight down" (39 percent).

Other investigations are consistent with the Charlton (1984a,b) report. In a recent study of 1,000 adolescents in Canada (Feldman, Hodgson, Corber 1985), significantly more girls than boys were concerned about becoming overweight (36 vs. 14 percent, $p<0.001$). In girls 18 years or older, 52.6 percent of smokers reported worrying about their weight, whereas only 31 percent of nonsmokers reported weight-related concerns ($p<0.05$). In a study of smoking intentions among 400 U.S. high school males, Tucker (1983) reported that overweight boys scored much higher on smoking intent than either normal weight or underweight boys ($p<0.005$). Another survey evaluated gender differences in a sample of 221 college cigarette-smoking intenders and nonintenders (Page 1983). Results indicated that females were much more likely to intend to smoke than males. Females were also more likely to believe that smoking maintains body weight, and smoking intenders were also more likely to believe that smoking controls weight. Finally, in a retrospective survey of more than 1,000 young adults (Klesges and Klesges, in press), overweight females reported that they were much more likely (20 percent) to start smoking for weight-related reasons compared with normal-weight females (2 percent). No differences between overweight versus normal-weight males (8 vs. 6 percent) were observed.

Several surveys on smoking maintenance have shown that individuals report that weight control is a powerful motivator to continue to smoke. Physicians who smoked were much more likely than those who had quit (46 vs. 22 percent) to believe that smoking cessation

increases appetite and weight (Fletcher and Doll 1969). Nurses who failed to quit smoking listed (in order) loss of determination, stress, and weight gain as the major reasons for failure (Knobf and Morra 1983). Beliefs regarding the weight-control effects of smoking and quitting differentiate smokers and nonsmokers (Hill and Gray 1984; Loken 1982; Shor et al. 1981). Females are particularly worried about postcessation weight gains (Klesges and Klesges, in press; Sorensen and Pechacek 1987). They are more likely to endorse smoking as an active weight-loss strategy (39 vs. 25 percent) and are more likely to report relapse for weight-related reasons (20 vs. 7 percent) (Klesges and Klesges, in press).

The research cited above is based on self-reports of the weight-control effects of smoking and, as such, could be viewed as an excuse for smoking. Two recent worksite-based investigations evaluated whether pretest concerns regarding smoking and weight-related issues prospectively predicted cessation. Maheu (1985) evaluated 49 subjects who either received a competition-based (n=32) or a no-competition condition (n=17). In the competition-based condition, participants were told that they would be rewarded if those at their worksite lost more weight than those at a neighboring worksite. At a 3-month followup, 78 percent of the subjects in the competition and 76 percent of the subjects in the no-competition condition were reportedly abstinent. Regression analysis at followup indicated that the best pretest predictors of smoking cessation (in order) were negative responses to the questions: (1) “Do you think smoking helps control your weight ?”; (2) “Did one of your parents smoke when you were young?”; and (3) “If you have tried to quit before, did you suffer any withdrawal symptoms?” Klesges, Brown, and associates (1987) found that the best predictors of cessation at posttest were pretest cotinine levels and anticipated weight gain as the result of smoking cessation. The best predictors of cessation at followup were the number of coworkers who smoked followed by anticipated cessation-related weight gain.

A recent community survey evaluated predictors of current and former smoking status in a sample of 611 nonsmokers, ex-smokers, smokers who had tried to quit smoking, and smokers who had not attempted cessation (Klesges, Somes et al. 1987). The best predictors of smokers who had never attempted cessation versus those with a history of cessation efforts were a greater concern related to weight control, followed by knowledge of the health consequences of smoking. Smokers who had not attempted cessation were significantly more likely to cite weight-control issues compared with smokers who had made active attempts at smoking cessation. Collectively, these investigations indicate that weight-related concerns may not only predict successful smoking cessation, but also attempted smoking cessation.

Weight gain following smoking cessation as a predictor of smoking relapse has been evaluated in two recent investigations. Hall, Ginsberg, and Jones (1986) found a relationship between smoking status at a l-year followup and weight gain at 6 months; greater weight gain during the first 6 months predicted continued abstinence. This finding was contrary to expectations. In another investigation, Gritz, Carr, and Marcus (in press) found that continuous abstainers had gained an average of 6.1 lb, relapsers had gained 2.7 lb and subsequently lost half the gain (1.3 lb), and never quitters had gained only 0.3 lb. While it was expected that postcessation weight gain would be predictive of relapse, one would expect that those who have been abstinent from cigarettes would have gained more weight than those who either failed to quit or those who relapsed, because these latter groups have regained the weight reducing effects of smoking. Additional research will need to evaluate the impact of weight gain on relapsers at the point of relapse compared with the impact on abstainers at a comparable point in time. Further, it is clear that actual weight may have little relationship with subjects' perceptions of their weight status. For example, overweight males consistently view themselves as normal weight, while underweight and normal-weight females consistently view themselves as overweight (Klesges 1983). Very small weight gains in some subjects (e.g., normal-weight females) may be much more predictive of relapse than very large weight fluctuations in others (e.g., overweight males) (Klesges 1983). Future research should evaluate potential variables (e.g., gender, obesity) that may moderate the relationship between weight gain and smoking relapse.

In summary, weight-related issues may be important in the maintenance and cessation of smoking. Weight-reducing effects of smoking may encourage smoking initiation by some people, but the data on this point are currently unconvincing. Future research should focus on who (e.g., males versus females, those with a history of chronic dieting) is most at risk to smoke because of weight-related concerns. In particular, prospective studies on weight-related issues as they predict smoking initiation, cessation, and relapse are needed.

Implications for Tobacco Use

Cigarette smokers weigh less than comparably aged nonsmokers, and many smokers who quit smoking gain weight. This inverse relationship between smoking and body weight is well established, and the role of food intake and energy expenditure as mechanisms for this relationship is currently receiving research attention. The postsmoking weight gains are frequently undesired by the ex-smoker. People are quite aware of the relationship between smoking and body weight, and this relationship may encourage some people to initiate smoking and to keep smoking. However, other people may

modify food intake and avoid weight gains after cessation of smoking.

Summary and Conclusions

1. After smoking cigarettes or receiving nicotine, smokers perform better on some cognitive tasks (including sustained attention and selective attention) than they do when deprived of cigarettes or nicotine. However, smoking and nicotine do not improve general learning.
2. Stress increases cigarette consumption among smokers. Further, stress has been identified as a risk factor for initiation of smoking in adolescence.
3. In general, cigarette smokers weigh less (approximately 7 lb less on average) than nonsmokers. Many smokers who quit smoking gain weight.
4. Food intake and probably metabolic factors are involved in the inverse relationship between smoking and body weight. There is evidence that nicotine plays an important role in the relationship between smoking and body weight.

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