(*130*) TENOVUO, J., MAEKINEN, K.K. Concentration of thiocyanate and ionizable iodine in saliva of smokers and nonsmokers. Journal of Dental Research 55(4): 661-663, July/August 1976.

(*131*) TRAHAIR, R.C.S. Giving up cigarettes: 222 case studies. Medical Journal of Australia 1: 929-932, May 6, 1967.

(*132*) TROJNAR, R. Badania ukladu adrenergicznego i metabolizmu serotoniny u palaczy papierosow (Studies of adrenergic system and serotonin metabolism in cigarette smokers). Polski Tygodnik Lekarski 31(12): 473-476, March 22, 1976.

(*133*) TROJNAR, R. Wydalanie z moczem niektorych metabolitow katecholamin i serotoniny u bylych palaczy pod wplywem palenia papierosow (Urinary excretion of certain metabolities of serotonin and catecholamines after cigarette smoking in subjects who had stopped smoking). Polski Tygodnik Lekarski 31(13): 521-524, March 29, 1976.

(*134*) TURNER, J.A.M. Confirmation of abstinence from smoking. British Medical Journal 2(6038): 755, September 25, 1976. (Letter)

(*135*) ULETT, J.A., ITIL, T.M. Quantitative electroencephalogram in smoking and smoking deprivation. Science 164(3882): 969-970, May 23, 1969.

(*136*) U.S. PUBLIC HEALTH SERVICE. The Health Consequences of Smoking. A Report to the Surgeon General: 1971. U.S. Department of Health, Education, and Welfare, Public Health Service, Health Services and Mental Health Administration, DHEW Publication No. (HSM) 71-7513, 1971, 458 pp.

(*137*) U.S. PUBLIC HEALTH SERVICE. The Health Consequences of Smoking. A Report to the Surgeon General: 1972. U.S. Department of Health, Education, and Welfare, Public Health Service, Health Services and Mental Health Administration, DHEW Publication No. (HSM) 72-7516, 1972, 158 pp.

(*138*) U.S. PUBLIC HEALTH SERVICE. The Health Consequences of Smoking, 1975. U.S. Department of Health, Education, and Welfare, Public Health Service, Center for Disease Control, HEW Publication No. (CDC) 77-8704, 1977, pp. 87-160.

(*139*) U.S. PUBLIC HEALTH SERVICE. Smoking and Health. Report of the Advisory Committee to the Surgeon General of the Public Health Service. U.S. Department of Health, Education, and Welfare, Public Health Service, Center for Disease Control, PHS Publication No. 1103, 1964, 387 pp.

(*140*) VANUXEM, D., WEILLER, P.J., GRIMAUD, C., GABRIEL, B., MONGIN, M. Polyglobulie du fumeur (Polycythemia of the smoker). Nouvelle Presse Medicale 4(30): 2204, September 20, 1975.

(*141*) VERDY, M., ORSETTI, A., BALDET, L., PUECH-CATHALA, A.-M. Effet de la cigarette sur les taux seriques d'hormone de croissance et de TSH dans le diabete (The effect of cigarettes on serum levels of growth hormone and TSH in diabetics). L'Union Medicale du Canada 104(9): 1356-1359, September 1975.

(*142*) VOGT, T.M., SELVIN, S., WIDDOWSON, G., HULLEY, S.B. Expired air carbon monoxide and serum thiocyanate as objective measures of cigarette exposure. American Journal of Public Health 67(6): 545-549, June 1977.

(*143*) VOLLE, R.L., KOELLE, G.B. Ganglionic stimulating and blocking agents. In: Goodman, L.S., Gilman, A.G., Koelle, G.B. (Editors). The Pharmacological Basis of Therapeutics. Fifth Edition. New York, Macmillan, 1975, pp. 465-474.

(*144*) WENNMALM, A. Nicotine stimulates prostaglandin formation in the rabbit heart. British Journal of Pharmacology 59(1): 95-100, January 1977.

(*145*) WEST, D.W., GRAHAM, S., SWANSON, M., WILKINSON, G. Five-year follow-up of a smoking withdrawal clinic population. American Journal of Public Health 67(6): 536-544, June 1977.

(*146*) WEYBREW, B.B., STARK, J.E. Psychological and physiological changes associated with deprivation from smoking. U.S. Naval Submarine Medical Center, Submarine Medical Research Laboratory, Report No. 490, February 23, 1967, 19 pp.

(147) WIKLER, A. Characteristics of opioid addiction. In: Jarvik, M.E. (Editor). Psychopharmacology in the Practice of Medicine. New York, Appleton-Century-Crofts, 1977, pp. 419-432.

(148) WILHELMSEN, L. One year's experience in an anti-smoking clinic. Scandinavian Journal of Respiratory Diseases 49(4): 251-259, 1968.

(149) WINTERNITZ, W.W., QUILLEN, D. Acute hormonal response to cigarette smoking. Journal of Clinical Pharmacology 17(7): 389-397, July 1977.

(150) WOCIAL, B., CISWICKA-SZNAJDERMAN, M., JANUSZEWICZ, W. Wplyw palenia papierosow na zachowanie sie poziomu katecholamin, wolnych kwasow tluszczowych i kortyzolu w surowicy krwi (Effect of cigarette smoking on serum catecholamines, free fatty acids and cortisol). Kardiologia Polska 19(1): 59-65, 1976.

(151) WOOD, J.E. Effect of smoking on the peripheral circulation in relation to environmental temperature. Annals of the New York Academy of Sciences 90(1): 114-118, September 27, 1960.

(152) WYNDER, E.L., KAUFMAN, P.L., LESSER, R.L. A short-term follow-up study on ex-cigarette smokers, with special emphasis on persistent cough and weight gain. American Review of Respiratory Diseases 96(4): 645-655, October 1967.

(153) YANAGITA, T. Brief review on the use of self-administration techniques for predicting drug dependence potential. In: Thompson, T., Unna, K.R. (Editors). Predicting Dependence Liability of Stimulant and Depressant Drugs. Baltimore, University Park Press, 1976, pp. 231-242.

(154) ZEIDENBERG, P., JAFFE, J.H., KANZLER, M., LEVITT, M.D., LANGONE, J.J., VAN VUNAKIS, H. Nicotine: Cotinine levels in blood during cessation of smoking. Comprehensive Psychiatry 18(1): 93-101, January/February 1977.

16. BEHAVIORAL FACTORS IN THE ESTABLISHMENT, MAINTENANCE, AND CESSATION OF SMOKING.

National Institute on Drug Abuse

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Introduction

Smoking is a behavior—a highly complex act which is accompanied by certain cognitions and hedonic states and based on various biochemical and physiological processes. In that sense, research on smoking behavior is at the interface between psychosocial and biological investigations of smoking. While behavioral research has contributed greatly to the technology of smoking cessation, relatively few behavioral investigations have been carried out to elucidate the mechanisms underlying smoking. Because of this, the present chapter will focus on social learning theory and nicotine regulation as general considerations to provide a context for a behavioral analysis of smoking. An evaluation of the contributions from the experimental analysis of behavior to the treatment of cigarette smoking and recommendations for further research will be made. Behavioral research findings on the establishment, maintenance, and cessation of smoking will be summarized. Emphasis will be on those stages (*16*) of smoking which follow initiation and during which the processes that contribute to the tenacity of the habit and its resistance to change are set in motion.

The Social Learning Model

Social learning theory has functioned less as a formal explanatory model of smoking and more as a methodological approach with an associated intervention technology (*35*). The impetus for using behavior modification techniques has been provided by the belief that research procedures which operationalize definitions, emphasize well-controlled empirical research, and are derived from concepts from the experimental laboratory will provide valuable practical and theoretical knowledge—a belief justified by the previous contributions of the behavioral approach toward the understanding of other difficult problems in human behavior. Behavior modification is derived from basic research on animal learning by Pavlov and Skinner. It emphasizes the control of antecedent and consequent environmental events (stimuli) in determining behavior (*4*). Social learning theory represents an extension of behavior modification to situations which involve interpersonal activity, but it incorporates the added explanatory concept of modeling, based on imitation and social reinforcement.

In brief, a social learning explanation of smoking proceeds along the following general lines (*35*): The habit is acquired under conditions of social reinforcement, typically those of peer pressure. Initially the inhalation of smoke is aversive, but after sufficient practice, habituation (or tolerance) occurs, and the behavior begins to produce sufficient positive reinforcement in its own right to be sustained independently of social reinforcement. Smoking now generalizes to situations other than the one in which it was originally acquired. It is important to note

that, from the perspective of social learning theory, smoking is seen as a learned behavior from the onset.

The analysis continues as follows: Discriminations between situations in which smoking is punished socially and those in which it is either ignored or favorably received are formed, and various circumstances (both external and internal) begin to control smoking. Insofar as they are associated with smoking, some situations, such as an empty cigarette pack or an annoying telephone call, may serve as conditional stimuli (CS's) which elicit covert responses. These responses (i.e., physiological changes or discomfort, perceived as craving) increase the likelihood of smoking. In turn, they can serve as discriminative stimuli (SD's), setting the occasion for the reinforcement provided by smoking. Moreover, stimuli which are preparatory to the act of smoking, such as the sight of a cigarette, can function as secondary reinforcers for behaviors preceding them (for example, purchasing a full cigarette pack). These cues can also serve as discriminative stimuli for behaviors which follow them, such as lighting the cigarette, thus forming a linked chain of responses (a smoking ritual). For successful termination of the overt act of smoking to occur, the extinction of most or all of the conditional stimuli, secondary reinforcers, and discriminative stimuli which make up the habit is required. The way in which these ideas have been put to specific use in therapy will be discussed in some detail later in this chapter.

The number of emotional events which can influence smoking are potentially quite great. If smoking is seen, in part, as an avoidance/escape response to aversive withdrawal states, then, hypothetically, by a process of stimulus generalization, other dysphoric states (for example, anger, tension, boredom) might also serve as discriminative stimuli for smoking. Also, response generalization may occur. In this case, the smoking ritual serves as a temporary escape (coping response) from various aversive situations (that is, smoking as a response which provides relief). Smoking can be seen, therefore, as a generalized primary and secondary reinforcer providing both positive and negative reinforcement over a remarkably wide array of life situations.

From a social learning theory perspective, smoking is difficult to modify because of its ability to provide immediate reinforcement—nicotine from an inhaled cigarette reaches the brain in seven seconds (twice as fast as intravenous administration from the arm). Furthermore, the habit is tremendously overlearned: at ten puffs per cigarette, the pack-a-day smoker gets more than 70,000 nicotine "shots" in a year—a frequency which is unmatched by any other form of drug taking (*40*). While most smokers recognize that sustained smoking can lead to a variety of unpleasant events, ranging from bronchitis to lung cancer, the ultimate aversive consequences of smoking—though potentially of great magnitude—are delayed and therefore have less

influence over ongoing smoking behavior than immediate consequences. This is a situation common to a number of self-management problems (*37*). Unlike alcohol and many other drugs of dependence, there are few immediately noticeable negative consequences (*40*).

To a large extent, behavioral researchers have *assumed* relationships between environmental events and smoking. Treatment practices have been based on general theory rather than on research or a functional analysis of smoking behavior as such. Thus, though part of the promise of social learning theory has been fulfilled, and behavioral concepts may have generated new standards of effectiveness in the treatment of smoking, there has not been a comparable contribution to the understanding of smoking per se.

The Nicotine Addiction Model

A physiologically based model of smoking, emphasizing the key role of nicotine as a reinforcer, has evolved from the work of Schachter (*42, 43*) and others like Jarvik (*19*) and Russell (*40*). The main focus is on explaining the maintenance of the smoking habit following acquisition. Under this formulation, smoking is viewed as an escape/avoidance response to aversive stimulation provided by periodic nicotine withdrawal in the addicted smoker. An internal regulatory mechanism is implied which detects the level of nicotine and maintains it within characteristic upper and lower limits by regulating the frequency of smoking (and possibly other intake parameters).

Much of the evidence in support of smoking as negatively reinforced behavior comes from a series of innovative experiments conducted by Schachter and his associates over a 10-year span. In one study, Nesbitt (*30*) used the amount of shock a subject was willing to tolerate as a behavioral measure of anxiety. They found that heavy smokers tolerated a higher shock intensity (were less "anxious") when allowed to smoke than when not allowed to smoke; nonsmokers tolerated an intermediate shock intensity. The design did not allow a differentiation between the possibility that smokers tolerated higher shock intensity because of a "sedative" effect of smoking (positive reinforcement) or because smoking constituted escape from withdrawal symptoms perceived as "anxiety" (negative reinforcement). To test for this, Silverstein (*46*) varied the amount of nicotine in cigarettes given prior to shock presentation. He found that smokers given a high-nicotine cigarette tolerated more shock than smokers given low-nicotine cigarettes and that there was no significant difference between smokers given low-nicotine cigarettes and deprived smokers. He concluded that the sensory-motor and oral positive reinforcement provided by low-nicotine cigarettes played a negligible role in increasing shock tolerance compared with the negative reinforcement provided by escape from withdrawal symptoms using high-nicotine

cigarettes. Further support came from the observation that nonsmokers exhibited higher endurance thresholds (lower "anxiety") than deprived or low-nicotine smokers. This suggests that "smoking doesn't reduce anxiety or calm the nerves [but rather that] not smoking increases anxiety by throwing the smoker into withdrawal" (*54*). Thus, a nicotine deficit seems to exacerbate the distress induced by aversive shock. Heimstra, et al. (*15*) found the same effect for psychomotor performance on a simulated driving test.

The next problem was to account for why smokers smoke more when stressed. According to Schachter (*42*), the debilitating effects of no or low nicotine are the result of withdrawal, and the effect of stress is to put the smoker into withdrawal by depleting the available supply of nicotine. This hypothesis was strengthened and new leads were generated by biochemical studies showing that, while some nicotine is catabolized (mainly in the liver, at a constant rate determined in part by the duration of the habit), a fraction of the nicotine escapes detoxification and is eliminated directly in the urine. Furthermore, the rate of urinary excretion is rapid, increases linearly with dosage, and increases as the pH of the urine becomes more acid. The hypothesis was confirmed by direct manipulation of urinary acidity through the administration of mild acidifying agents like ascorbic acid or glutamic acid hydrochloride or alkalizers like sodium bicarbonate (*43*). In addition, stressful events associated with heavier smoking increased urinary acidity and nicotine excretion in the expected direction (*42*). To test whether stress or urinary pH or both were the independent variable, Schachter et al. (*43*) independently manipulated stress and pH and reported that smoking seemed to be under the control of urinary acidity rather than stress as such.

Schachter's model posits that nicotine is the primary reinforcer because of its role in reducing tension and distress associated with nicotine deprivation. If this is true, secondary reinforcers should be relatively unimportant. For example, smokers should not smoke nicotine-free cigarettes, and supplying alternative sources of nicotine should eliminate the desire to smoke. According to Jarvik (*19*), much of the evidence for the role of nicotine as the primary reinforcer in cigarette smoke is circumstantial. Smokers evidently prefer cigarettes with, rather than without, nicotine; but they will smoke nicotine-free cigarettes for a while if no others are available. The fact that smoking such cigarettes is not sustained despite the usual cues for smoking suggests that the other variables are secondary reinforcers that extinguish when nicotine—the primary reinforcer—is not present. Attempts to investigate the role of nicotine as the sufficient condition for smoking, however, have produced conflicting results. Preloading nicotine, by having subjects smoke or chew gum containing nicotine before testing, did reduce subsequent puffing (*20*, *21*, *25*). And administration of the drug mecamylamine, which functioned as a

nicotine "antagonist," increased the smoking rate (*52*). But Kumar, et al. (*21*) were unable to demonstrate a dose-response effect on subsequent smoking when nicotine preloading was administered intravenously. The fact that lettuce cigarettes reinforced with nicotine were as unacceptable as non-nicotine cigarettes also seems to undermine the nicotine-only hypothesis (*19*). Jarvik (*19*) concluded that nicotine may be a *necessary but not sufficient* condition for smoking behavior to occur and to be sustained and that more research is clearly needed to settle the issue of whether nicotine functions as the primary reinforcer or as a "reinforcing co-factor."

The nicotine addiction model suggests that the smoker regulates nicotine levels under widely varying conditions. It implies a mechanism which senses nicotine and provides the impetus for directed behavior—possibly a central "nicostat" or the integration of the various peripheral drug effects of nicotine. While the model is plausible and straightforward, critical tests have yet to be performed. Particularly, direct measurements of changes in nicotine titer and of the withdrawal state have not been attempted. Finally, among variables not adequately explained by the model are the role of environmental stimuli in the control of the habit, the nature of individual differences in smoking behavior (for example, light versus heavy smokers and occasional versus chronic smokers), and the mechanism(s) by which relapse occurs following withdrawal (*35*).

A Context for Behavioral Research on Smoking

Clearly, neither social learning theory nor the nicotine addiction model alone can provide a complete understanding of smoking at present. A recent model, the opponent process theory (*47, 48, 49, 53*) does attempt to link psychological and physiological factors involved in the maintenance of smoking in a more comprehensive fashion. The principal features of the opponent process model as it applies to smoking are as follows: (*1*) the reaction to cigarette smoke is biphasic, with a brief pleasurable component (*a* process) followed by a more sustained dysphoric component (*b* process); (*2*) the hedonic tone—pleasurable A state or dysphoric B state—is determined by the algebraic sum of the two opponent processes at a given point in time; and (*3*) stimuli associated with a given state can elicit this state as a conditioned response after repeated pairings.

The opponent process model assumes that cigarettes contain substances which provide pleasure (initiate the *a* process) during early use. While there may be some unpleasant effects on the first few occasions, these should be offset by the drug effect or by other reinforcers such as peer pressure; if not, the act of smoking will not continue. As cigarette smoking becomes established, the opponent

process grows in strength: the pleasurable A state weakens and the withdrawal B state intensifies correspondingly.

Because the *b* process is the opponent of the *a* process, the best way of attenuating the B state is to ingest the substance that produces the A state. As an operant behavior, smoking is both positively reinforced by a pleasurable consequence and negatively reinforced by terminating aversive withdrawal, thus setting up an addictive cycle. As the *b* process is further strengthened, still larger amounts of tobacco have to be smoked to produce a pleasurable A state, resulting in tolerance.

Stimuli associated with smoking (CS_A's), such as a pack of cigarettes or the sight of matches, should elicit a brief conditioned (pleasurable) A state at stimulus onset and a conditioned withdrawal (unpleasant) B state at stimulus offset. Furthermore, stimuli associated with the B state (CS_B's)—such as an empty cigarette pack, empty pockets, no stores, or "no smoking" signs—should elicit conditioned craving or withdrawal. The concept of conditioned A and B state elicitors leads to the important implication that, as the smoking habit becomes well established and the *b* process becomes stronger, CS_A's elicit a brief conditioned state which is pleasant but then is followed by a more extended conditioned craving which intensifies the pre-existing withdrawal B state. Similarly, CS_B's directly elicit conditioned craving, which also adds to the discomfort of the withdrawal state. An additional implication (derived from Pavlovian conditioning theory) is that as CS_B's become stronger, they may become more anticipatory, leading to shorter redosage and restimulation intervals until an asymptote is reached. If the smoker quits, the CS_B's and the *b* process should weaken eventually through disuse, but the CS_A's and the *a* process should intensify correspondingly. Thus, if a cigarette is smoked after a period of abstinence, the pleasurable component has increased to its original level and the resumption of the addictive cycle is facilitated. The smoker is clearly locked into the pattern of smoking and, in that sense, once established, the habit seems to be overdetermined.

The opponent process model has not been tested in formal research on cigarette smoking, though recent experiments in the area of opiate addiction do provide general support (*31, 44, 56*). The demonstration of conditionability, in particular, has important implications for the understanding of smoking recidivism. Wikler (*55*) has observed that environmental stimuli associated with withdrawal may precipitate conditioned craving (or withdrawal) even after an extended abstinence period has ended physical dependence in heroin addicts. The opponent process model predicts a biphasic response by smokers (A state followed by B state) to the presentation and removal of stimuli associated with cigarettes during acquisition. Later on in the addiction process, when tolerance is large, the dominant conditioned effects should be those of craving or withdrawal (B state predominates). The

implication for treatment is that unless conditioned craving is extinguished or modified as a part of therapy, the probability of relapse will remain high.

There are a number of different issues that need to be resolved among the current behavioral formulations of smoking before an adequate understanding is achieved. For example, the nicotine addiction model suggests that the day-to-day regulation of smoking is more under the control of pharmacological variables than of environmental stimuli, though their relative contribution remains to be determined. Moreover, the issue of whether smoking reduces anxiety is not settled. For example, Hutchinson and Emley (*18*) have suggested that nicotine can be classified as a tranquilizer since it decreases aggression as well as the conditioned emotional response (CER). They have speculated that difficulty in training animals to smoke under ordinary conditions may have been because a background of aversive stimulation is needed to provide motivation to use smoking to relieve anxiety. Also, as has been mentioned, the pharmacological primacy of nicotine implied by the nicotine addiction model has yet to be established unequivocally.

The opponent process model encounters similar problems. For example, Wikler (*55*) has argued that certain responses associated with chronic drug use, such as tolerance or conditioned withdrawal, are *counteradaptations*, serving to protect the organism by acting in a direction opposite to the normal drug effect. The opponent process model is stated in sufficiently general terms to incorporate these observations if certain (untested) assumptions are made: Wikler's observations emphasize the dominant drug-negative B state; in opponent process theory, the initial drug-positive *a* process (and thus the pleasurable A state) is still operative but may be so brief and attenuated that it goes undetected. Only closer examination of the time course for the response to drugs at different states of acquisition will settle this issue. An additional complication has been raised by Siegel (*45*), who has shown that the stimuli which constitute the ritual of (repeated) drug injection can elicit conditioned reactions which increase tolerance to the drug; extinction of these conditioned reactions, using a series of saline injections, results in decreased tolerance. Siegel proposes that tolerance is the result of compensatory *associative* processes and is not simply a pharmacological, nonassociative phenomenon. While opponent process theory can be modified to accommodate these findings, by defining them as the manifestations of stimuli which serve as conditioned B state elicitors, the relative contribution of associative and nonassociative factors cannot be specified at present. Furthermore, if tolerance is basically an associative process, the problem of explaining why certain substances, such as nicotine, produce tolerance while others do not will also have to be dealt with (*35*).

The remainder of the present discussion will re-examine some of the phenomena of acquisition, perpetuation, and termination of smoking from the point of view of the three models. Special attention will be given to implications for further research.

The Establishment of Smoking

The establishment of smoking can be seen as the result of initial experimentation with cigarettes repeated sufficiently often for acquisition of a habit and/or for addictive processes to take hold. Among the major variables contributing to initiation are social pressure and imitation of peers or family members who smoke (*1, 11*). The following variables influence the decision to smoke: peer pressure, best friends who are smokers, parents who smoke, adolescent rebellion, imitation of adult behavior, and misconceptions concerning the risks of smoking. A recommendation to conduct longitudinal comprehensive studies on the acquisition of smoking in the natural environment, and to determine the conditions under which smoking does or does not begin, would seem especially appropriate.

Once the smoking habit is acquired, the stage is set for addictive processes to contribute to the maintenance of the habit and to its overdetermination under the influence of the variables alluded to in the several smoking models. Additional physiological variables and explanatory variables from personality theory and typology studies (both types described elsewhere in the present report) are clearly relevant. These two sets of variables suggest a number of possible mechanisms by which acquisition might take place, although, as Leventhal and Cleary (*22*) point out, they are not necessarily the same mechanisms which contribute to onset. The need for careful, directed research in this area is evident to achieve a better understanding of onset and acquisition which may lead to more effective methods for prevention and treatment.

A promising approach to the investigation of physiological and behavioral, as well as psychosocial, factors in acquisition comes from animal research. Some studies have shown that nicotine facilitates conditioned-avoidance behavior as well as positively reinforced behavior in rats (*51*) and that it reduces social or pain-induced aggression in both animals and humans (*18*). Analogues of addiction might also be explored in the laboratory. While the laboratory approach might seem artificial to some, increasing experimental control by restricting extraneous variables has been useful in other difficult areas, such as alcoholism (e.g., Nathan and O'Brien (*29*)) and heroin addiction (e.g., O'Brien, et al. (*32*)). If such explorations are successful, subsequent research could be conducted under increasingly complex and more "natural" conditions. Finally, studies of different methods for deterring smoking in children (e.g., Evans (*7*) and Piper (*34*)) should

increase understanding of the conditions under which smoking begins and allow us to identify those environmental patterns which facilitate the movement from "experimental" smoking to addiction.

The Maintenance of Smoking

Once smoking is established as a habit, a number of factors contribute to its persistence and resistance to change. Each of the formulations described above devotes considerable attention to the phenomenon of maintenance, and a large body of research has been carried out from various points of view. In a sense, maintenance can be seen as a stage of smoking characterized by steady-state behavior. Pattern consistency is provided by environmental influences through stimulus control as well as by underlying physiological processes regulating consumption within characteristic limits. As an acquired motivation, smoking constitutes a behavioral pattern with powerful reinforcing value, overdetermined to a remarkable degree by its generating mechanisms. A better understanding of these processes is needed.

With a few exceptions, the determination of environmental influences on smoking has received relatively little direct attention experimentally, despite the fact that treatment techniques based on social learning theory have been used extensively. Among the better examples of a functional analysis of behavior is a study by Griffiths, et al. (*12*). Following detoxification, alcoholics in a residential laboratory were allowed to consume ethanol at certain times, and the amount of tobacco smoked was measured under various conditions. Cigarette smoking was shown to increase from 26 to 117 percent when the solutions consumed contained ethanol. The effect was robust, was observed in each of the five subjects, and was replicated 15 times employing a within-subject design. Control procedures indicated that the effect did not depend on: (1) the pattern of ethanol ingestion, (2) adjunctive maintenance through social interactions, (3) the pattern of days in which the ethanol or ethanol-free vehicle was scheduled, (4) alterations in the portion of cigarette smoked or the number of puffs taken, or (5) knowledge that a given drink did or did not contain ethanol. The study constitutes a good demonstration of the potential of the experimental analysis of smoking behavior, and the method should be extended to other problems of interest.

Smoking as an avoidance/escape response to withdrawal implies an internal regulatory mechanism by which the levels of nicotine (or other substances) are maintained within limits characteristic for each smoker. To get at these processes in research, measures should be taken of smoking behavior (specifying variables such as puff frequency and duration, depth of inhalation, amount of nicotine drawn from a standard cigarette), of major physiological variables (for example, cardiovascular changes, relevant biochemical activity including cholin-

ergic, catecholamine, and nicotine changes), and of cognitive variables (for example, hedonic states and the subjective desire to smoke at different points in time). As in investigations on the establishment of smoking, a laboratory approach may provide a good initial strategy, if supported by adequately controlled studies in the natural environment.

As a preliminary step, the variables involved in nicotine regulation should be explored directly in habitual smokers by studying the relationships between the act of smoking, subjective desire, and plasma nicotine levels. Also, nicotine excretion rates could be shifted using techniques identified by Schachter, such as drugs or psychological stress, to provide further modulation of physiological, behavioral, and subjective responses, thus replicating and extending previous work in this area. The demonstration of the contribution of nicotine by direct measurement might stimulate further explorations of the relationship between smoking behavior and other important biochemical variables such as catecholamines.

The Cessation of Smoking

Both initiation and cessation can be conceptualized as the result of decisions (evidenced by stated intention or other overt behavior) to start or to stop smoking. Thus, cognitive variables may play a major explanatory role, and the subjective utility of the change under consideration may provide important clues for predicting its outcome or success (*33*). (The cognitive aspects of initiation and quitting are extensively reviewed in a separate context elsewhere in this report.) Once the decision to start or stop smoking has been made, however, behavioral variables and the models described above come into play.

When habitual smokers stop smoking, they may experience a wide variety of unpleasant side effects, including craving for tobacco, irritability, restlessness, dullness, sleep disturbances, gastrointestinal disturbances, anxiety, and impairment of concentration, judgment, and psychomotor performance (*19*). The onset of symptoms may occur within hours or days after quitting and may persist from a few days to several months. Additional objective signs include a decrease in heart rate and blood pressure, increased rapid eye movement (REM) sleep, and slower rhythms in the EEG (*35*). Spontaneous jaw clenching (increased masseter potentials) lasting several weeks has been correlated with verbal reports of irritability (*18*).

After the ex-smoker successfully overcomes withdrawal symptoms, further problems may persist. In terms of the opponent process model, one can construct the following account: Subjectively, the pleasure of smoking in the addicted smoker is masked by the discomfort of craving from not smoking. After abstaining for a few weeks, however, craving decreases. If smoking is resumed, the first few cigarettes seem very strong and are highly pleasurable. Thus, the stage for re-addiction is

set. Moreover, various internal and external stimuli may serve as conditioned elicitors of craving or withdrawal. Particularly troublesome may be events too infrequent to extinguish quickly (e.g., attending a reunion where former classmates smoke) or emotional situations which resemble withdrawal (e.g., anticipation of an unpleasant or challenging social event).

A major contribution of the behavioral approach has been the development of new techniques in smoking cessation—procedures which seem to be more effective than those that preceded them. In most nonbehavioral clinics, fewer than half the smokers quit (e.g., Guilford (*13*)), and of those who quit only 25 to 30 percent are still abstinent 9 to 18 months later (*17*); the estimated long-term abstinence rate in nonbehavioral treatment is about 13 percent (*27*). The three main lines of behavioral treatment have involved punishment and aversive conditioning, stimulus control and contingency management, and controlled smoking procedures. While a thorough review of the modification of smoking is provided elsewhere in this report, the contribution of social learning to therapy is of sufficient importance to warrant a brief review here.

Aversive conditioning techniques are the oldest and most widely utilized behavioral procedures for smoking cessation. Among the aversive stimuli used have been electric shock (e.g., Best and Steffy (*3*)), covert or imagined aversive events, and cigarette smoke (e.g., Resnick (*39*)). The typical procedure has involved contingent punishment for overt smoking behavior in the laboratory or in the natural environment (e.g., Powell and Azrin (*38*)). Some investigators have attempted to punish motoric and cognitive components as well (e.g., Steffy, et al. (*50*)). With the exception of aversive smoking procedures, aversive conditioning techniques have not produced outstanding results (Bernstein and Glasgow (*2*)).

Aversive smoking combines the principles of extinction, negative practice, and aversive conditioning, using stimuli from the cigarettes themselves as the aversive component. The procedure assumes that the positive reinforcing aspects of a stimulus are reduced and become aversive if that stimulus is presented at an artificially elevated frequency or intensity. A further assumption is that aversion based on stimuli intrinsic to the maladaptive behavior is more salient and generalizable than that from artificial sources such as shock (Bernstein and Glasgow (*2*)). The most successful use of aversive smoking can be found in the recent work of Lichtenstein, et al. (*24*), using a technique called rapid smoking. The procedure calls for smoking cigarettes at a rapid rate (inhaling smoke about 6 seconds after each exhalation) until no more can be tolerated. Sessions are repeated on a daily basis until the smoker no longer reports a desire to smoke; booster sessions are provided if the desire returns. In a recent review of several studies using the procedure, the abstinence rate was 54 percent in short-term

follow-up and 36 percent in long-term follow-up (2 to 6 years after treatment). Though the method was a clear improvement over previous approaches, there are a number of problems which may make it less than the optimal procedure for the elimination of smoking. In particular, individuals with cardiopulmonary diseases—those who most need help—are the least likely to tolerate intense exposure to tobacco smoke without ill effect (*35*). Moreover, rapid smoking may be dangerous even to seemingly healthy people (*28*).

Another social learning approach to the modification of smoking behavior is represented by stimulus control tactics. The basic assumption is that smoking is associated with or controlled by environmental cues and that these cues (discriminative or conditional stimuli) contribute to the persistence of the habit (*2*). Treatment involves gradual elimination of smoking through programmed restriction of the range of stimuli that lead to smoking. Typically, self-monitoring is used to increase awareness of smoking along with designated daily quotas to provide targets for reduction (*36*). In general, stimulus control procedures have not been very effective in isolation (e.g., Levinson, et al. (*23*)). When used in combination with contingency contracting, in which deposited money is reimbursed for reaching specified goals (e.g., Elliott and Tighe (*6*)), and with other techniques, however, considerably better results are achieved (Bernstein and Glasgow (*2*)).

Recent research on multicomponent treatment procedures (employing techniques such as stimulus analysis, interference with situational control or environmental stimuli, social and monetary reinforcement of incompatible behavior, group support, and follow-up sessions, presented in an integrated sequence) has produced results as favorable as that reported for rapid smoking, with 61 percent of the first 100 participants quitting smoking after eight sessions of treatment and 32 percent not smoking a year after the onset of treatment (*36*). These data account for all smokers who entered treatment (including the 15 percent of the sample who could not be reached and were classified as smoking) and were based on self-reported smoking status corroborated by urinary nicotine analysis. The recidivism rate of 49 percent also compares favorably with the 70 to 75 percent recidivism reported for nonbehavioral clinics by Hunt and Bespalec (*17*). These positive findings are qualified somewhat by the observation that not all multicomponent treatment combinations are successful (e.g., Danaher (*5*)) and by a controlled multivariate study by Flaxman (*8*) indicating that the variables responsible for a successful outcome are poorly understood.

Smoking practices have changed considerably in recent years as smokers have attempted to reduce health risks on their own (Hammond, et al. (*14*)) by switching to filtered and low tar/nicotine cigarettes (Russell (*41*)). These natural trends provide a context for

recent research by Frederiksen and associates (*9*, *10*), demonstrating that behavioral technology can be used to control not only the rate and strength of cigarettes consumed but also to modify the topography of the habit. Additional impetus for the research comes from the fact that many smokers report difficulty reducing their smoking rate below 10 to 12 cigarettes per day (Levinson, et al. (*23*)). While it has been suggested that the reason for this is that the positive reinforcing value of each cigarette increases when fewer are smoked (Mausner (*26*)), according to opponent process theory there should be a corresponding lessening of the negative reinforcing effect resulting from withdrawal from nicotine over time. Clearly more research is needed to settle this issue. The technology developed by Fredericksen is still in the clinical development stage, and the long-term stability of the changes has yet to be determined. However, because some smokers are motivated to reduce their health risk even though they are unable to quit, controlled smoking technology may provide a useful alternative to the more traditional abstinence-oriented treatment and deserves further exploration.

While recent behavioral treatment seems more effective than previous approaches, 50 percent recidivism and 33 percent long-term abstinence leave considerable room for improvement. What is needed at present is outcome research directed at demonstrating the relative effectiveness of complete treatment packages in long-term randomized clinical trials. Subsequently, when a given procedure is shown to be superior in independent replications, components can be partitioned out and tested in order to produce clinical procedures that are both effective and efficient. Research designs should take into account the fact that recent improvements in outcome statistics for smoking-cessation clinics may reflect changing social attitudes toward smoking and higher levels of motivation rather than better treatment as such (*22*).

In an important sense, current treatment efforts—especially behavioral treatment—have been devoted primarily toward the modification of the overt act of smoking (an operant behavior). Less formal attention has been given to the cognitive and physiological respondents that constitute precursors of smoking (e.g., craving and withdrawal) and that are under the control of both environmental (exteroceptive) and emotional (interoceptive) stimuli. Moreover, the increased success of multicomponent programs may well be the result of more effective handling of these variables, using integrated sequences, than has been possible with unicomponent approaches. The fact that various previously neutral stimuli have been shown to elicit conditioned craving or withdrawal after being paired or associated with these states in various addictions has important implications for smoking treatment.

Treatment can be seen as extinguishing the act of smoking but not necessarily the concomitant conditioned cognitive or physiological respondents. As a result, the ex-smoker may continue to be exposed to various stimuli which have been associated with smoking, and the probability of relapse will remain great (for example, in the "negative affect" smoker (*36*)). Demonstrations that continued autonomic or cognitive reactivity persist after standard smoking-cessation therapy might lead to an entirely new approach to the old problem of relapse. Studies comparing a standard smoking-cessation treatment with "deconditioning" therapy, in which autonomic responses are extinguished in a simulated environment or modified directly using biofeedback, might lead to a demonstrably lower rate of recidivism for those smokers exposed to augmented therapy. The above suggests that basic research which leads to a better understanding of the mechanisms underlying smoking may result in the eventual development of a truly rational and more effective therapy for smoking.

Conclusions

The present chapter makes no claim to be exhaustive. Rather it has surveyed selectively what is known and not known concerning behavior in the establishment, maintenance, and cessation of smoking. The object has been to develop a context for directing research, for improving treatment, and for guiding social policy. In closing, a few specific recommendations seem appropriate.

While it is difficult to pinpoint accurately which of many research possibilities will be most fruitful on an *a priori* basis, certain themes seem particularly important for current behavioral research. They are the phenomenon of withdrawal, the reinforcing effects of nicotine, the role of nicotine antagonists or blockers, and the behavioral pharmacology of cigarette smoking.

1. Withdrawal symptoms of varying severity following cessation are among the principal reasons cited for relapse to smoking. Little scientific information is available on the sequelae to abstinence, however, and at present it is difficult to assess accurately their contribution to recidivism.

2. As discussed at some length, the problem of analyzing the reinforcing effects of nicotine is of great importance in understanding smoking. The role of nicotine as a positive and negative reinforcer should be examined in animals using various routes of administration as well as explored systematically in humans in laboratory and natural settings.

3. A related theme is derived from recent research suggesting that specific CNS receptor sites for nicotine can be blocked in a fashion analogous to the opiate antagonists. This phenomenon has implications

for understanding the effect of nicotine on the body as well as in helping smokers who have stopped to maintain abstinence.

4. The behavioral pharmacology of smoking deserves further emphasis. A more precise definition of smoking behaviors, involving psychometric analyses by puff volume, inter-puff interval, total amount smoked, and rate of smoking may have important implications for the understanding of stimulus control as well as of the relationship between blood nicotine levels and cigarette self-administration. Similarly, the development of objective criteria for validating dependent measures (such as self-reported smoking behavior using various biological assays) seems worthwhile.

In the treatment area, further improvement is clearly needed. Multicomponent procedures have provided sequences for handling different aspects of the smoking-cessation process; and components dealing specifically with problems in measuring baseline smoking, facilitating reduction, inducing abstinence, and managing side effects have been developed. Among the major current deficits for all approaches and programs, however, is maintenance of nonsmoking. Several suggestions have been made from a behavioral point of view. These include: (1) dealing promptly and effectively with the potential side effects of quitting (such as obesity and tension); (2) developing alternative activities to replace smoking (such as regular physical exercise or formal relaxation techniques); (3) providing a cognitive focus on mastery, self-help, and individual responsibility; and (4) adding "booster" sessions and continued interpersonal support in extended follow-up. Much more remains to be done—especially on the utilization of techniques derived from basic research, such as the extinction of conditioned craving described above.

Behavioral research may also make contributions to social policy. For example, the suggestion that nicotine plays a major or dominant role in the self-regulation of smoking raises the issue of the appropriateness of trying to persuade people to smoke low-tar, low-nicotine cigarettes. As Schachter (*42*) puts it, low-tar, high-nicotine cigarettes might be safer because fewer cigarettes would be smoked, thereby minimizing exposure to the products of incomplete combustion known to enhance the atherosclerotic process and to increase the risk of myocardial infarction (*19*). This problem could be investigated further, using a careful description of the number of cigarettes smoked and the number of puffs per cigarette (backed up with quantitative determinations of nicotine, carbon monoxide, tars, and other smoke products), to provide more exact information than is currently available from surveys of smoking in the natural environment. Finally, a greater understanding of the stimulus control of smoking and its limits may be very valuable. From a behavioral perspective, the current growing emphasis on the social unattractiveness of smoking (for example, the nonsmoker's rights movement) is helpful, because it provides a method which

administers more immediate social reinforcement for quitting and staying off cigarettes than has been possible when the focus was strictly on the health consequences of the habit. It should be noted that the effects of these social processes on the decision to quit smoking are still relatively underexplored.

Much work remains to be done in the behavioral research area. Sufficient progress has been made, however, to indicate that the development of a rational therapy for smoking based on a scientific understanding of smoking behavior and its underlying mechanisms constitutes a worthy objective.

Behavioral Factors in the Establishment, Maintenance, and Cessation of Smoking: References

(1) BERGEN, B.J., OLESEN, E. Some evidence for a peer group hypothesis about adolescent smoking. Health Education Journal 21: 113-119, 1963.

(2) BERNSTEIN, D.A., GLASGOW, R.E. The modification of smoking behavior. In: Pomerleau, O.F., Brady, J.P. (Editors). Behavioral Medicine: Theory and Practice. Baltimore, Williams and Wilkins, 1979, 50 pp. (in press)

(3) BEST, J.A., STEFFY, R.A. Smoking modification procedures for internal and external locus of control clients. Canadian Journal of Behavioral Science, 7(2): 155-165, 1975.

(4) BRADY, J.V. Learning and conditioning. In: Pomerleau, O.F. Brady, J.P. (Editors). Behavioral Medicine: Theory and Practice. Baltimore, Williams and Wilkins, 1979, 54 pp. (in press)

(5) DANAHER, B.G. Research on rapid smoking: Interim summary and recommendations. Addictive Behaviors 2(2): 151-156, 1977.

(6) ELLIOTT, R., TIGHE, T. Breaking the cigarette habit: Effects of a technique involving threatened loss of money. Psychological Record 18: 503-513, 1968.

(7) EVANS, R.I. Smoking in children: Developing a social psychological strategy of deterrence. Preventive Medicine 5: 122-127, 1976.

(8) FLAXMAN, J. Quitting smoking now or later: Gradual, abrupt, immediate, and delayed quitting. Behavior Therapy 9(2): 260-270, 1978.

(9) FREDERIKSEN, L.W., MILLER, P.M., PETERSON, G.L. Topographical components of smoking behavior. Addictive Behaviors 2(1): 55-61, 1977.

(10) FREDERIKSEN, L.W., PETERSON, G.L., MURPHY, W.D. Controlled smoking: Development and maintenance. Addictive Behaviors 1(3): 193-196, 1976.

(11) GORSUCH, R.L., BUTLER, M.C. Initial drug abuse: A review of predisposing social psychological factors. Psychological Bulletin 83(1): 120-137, 1976.

(12) GRIFFITHS, R.R., BIGELOW, G.E., LIEBSON, I. Facilitation of human tobacco self-administration by ethanol: A behavior analysis. Journal of the Experimental Analysis of Behavior 25(3): 279-292, 1976.

(13) GUILFORD, J.S. Group Treatment versus individual initiative in the cessation of smoking. Journal of Applied Psychology 56(2): 162-167, 1972.

(14) HAMMOND, E.C., GARFINKEL, L., SEIDMAN, H., LEW, E.A. "Tar" and nicotine content of cigarette smoke in relation to death rates. Environmental Research 12(3): 263-274, December 1976.

(15) HEIMSTRA, N.W., BANCROFT, N.P., DEKOCK, A.R. Effects of smoking upon sustained performance in a simulated driving task. Annals of the New York Academy of Sciences 142(Article 1): 295-307, March 15, 1967.

(16) HORN, D. A model for the study of personal choice health behavior. International Journal of Health Education 19(2): 3-12, April-June 1976.

(17) HUNT, W.A., BESPALEC, D.A. An evaluation of current methods of modifying smoking behavior. Journal of Clinical Psychology 30: 431-438, 1974.

(18) HUTCHINSON, R.R., EMLEY, G.S. Effects of nicotine on avoidance, conditioned suppression and aggression response measures in animals and man. In: Dunn, W.L., Jr. (Editor). Smoking Behavior: Motives and Incentives. Washington, D.C., V.H. Winston and Sons, 1973, pp. 171-196.

(19) JARVIK, M.E. Biological factors underlying the smoking habit. In: Jarvik, M.E., Cullen, J.W., Gritz, E.R., Vogt, T.M., West, L.J. (Editors). Research on Smoking Behavior. NIDA Research Monograph No. 17. U.S. Department of Health, Education, and Welfare, Public Health Service, Alcohol, Drug Abuse, and Mental Health Administration, National Institute on Drug Abuse, DHEW Publication No. (ADM) 78-581, December 1977, pp. 122-148.

(20) KOZLOWSKI, L.T., JARVIK, M.E., GRITZ, E.R. Nicotine regulation and cigarette smoking. Clinical Pharmacology and Therapeutics 17(1): 93-97, 1975.

(21) KUMAR, R., COOKE, E.C., LADER, M.H., RUSSELL, M.A.H. Is nicotine important in tobacco smoking? Clinical Pharmacology and Therapeutics 21(5): 520-529, 1977.

(22) LEVENTHAL, H., CLEARY, P.D. The Smoking Problem: A Review of the Research, Theory and Research Policies in Behavioral Risk Modification. Research and Analytic Report Series. University of Wisconsin, Madison, 1977, 102 pp.

(23) LEVINSON, B.L., SHAPIRO, D., SCHWARTZ, G.E., TURSKY, B. Smoking elimination by gradual reduction. Behavior Therapy 2(4): 477-487, October 1971.

(24) LICHTENSTEIN, E., PENNER, M.R. Long-term effects of rapid smoking treatment for dependent cigarette smokers. Addictive Behaviors 2: 1-12, 1977.

(25) LUCCHESI, B.R., SCHUSTER, C.R., EMLEY, G.S. The role of nicotine as a determinant of cigarette smoking frequency in man with observations of certain cardiovascular effects associated with the tobacco alkaloid. Clinical Pharmacology and Therapeutics 8(6): 789-796, 1967.

(26) MAUSNER, B. Some comments on the failure of behavior therapy as a technique for modifying cigarette smoking. Journal of Consulting and Clinical Psychology 36(2): 167-170, April 1971.

(27) MCFALL, R.M., HAMMEN, C.L. Motivation, structure, and self monitoring: role of nonspecific factors in smoking reduction. Journal of Consulting and Clinical Psychology 37(1): 80-86, 1971.

(28) MILLER, L.C., SCHILLING, A.F., LOGAN, D.L., JOHNSON, R.L. Potential hazards of rapid smoking as a technic for the modification of smoking behavior. New England Journal of Medicine 297(11): 590-592, September 15, 1977.

(29) NATHAN, P.E., O'BRIEN, J.S. An experimental analysis of the behavior of alcoholics and nonalcoholics during prolonged experimental drinking: A necessary precursor of behavior therapy? Behavior Therapy 2: 455-476, 1971.

(30) NESBITT, P.D. Smoking, physiological arousal and emotional response. Journal of Personality and Social Psychology 25(1): 137-144, 1973.

(31) O'BRIEN, C.P., TESTA, T.J., O'BRIEN, T.J. GREENSTEIN, R. Conditioning in human opiate addicts. The Pavlovian Journal of Biological Science 11(4): 195-202, October-December 1976.

(32) O'BRIEN, C.P., TESTA, T., O'BRIEN, T.J., BRADY, J.P., WELLS, B. Conditioned narcotic withdrawal in humans. Science 195: 1000-1002, 1977.

(33) PECHACEK, T.F., DANAHER, B.C. How and why people quit smoking: Cognitive-behavioral implications. In: Kendall, P.C., Hollan, S.D. (Editors). Cognitive-Behavioral Interventions: Theory, Research and Procedures. New York, Academic Press, c 1979. (in press)

(34) PIPER, G.W., JONES, J.A., MATTHEWS, V.L. The Saskatoon smoking study: Results of the first year. Canadian Journal of Public Health 62: 432-441, September/October 1971.

(35) POMERLEAU, O.F. Why people smoke: Current psychobiological models. In: Davidson, P. (Editor). Behavioral Medicine: Changing Health Life Styles. New York, Brunner-Mazel, 1979, 38 pp. (in press)

(36) POMERLEAU, O., ADKINS, D., PERTSCHUK, M. Predictors of outcome and recidivism in smoking-cessation treatment. Addictive Behaviors 3: 65-70, 1978.

(37) POMERLEAU, O., BASS, F., CROWN, V. Role of behavior modification in preventive medicine. New England Journal of Medicine 292: 1277-1282, June 12, 1975.

(38) POWELL, J., AZRIN, N. The effects of shock as a punisher for cigarette smoking. Journal of Applied Behavior Analysis 1(1): 63-71, 1968.

(39) RESNICK, J.H. The control of smoking behavior by stimulus satiation. Behavior Research and Therapy 6: 113-114, 1968.

(40) RUSSELL, M.A.H. Smoking problems: An overview. In: Jarvik, M.E., Cullen, J.W., Gritz, E.R., Vogt, T.M., West, L.J. (Editors). Research on smoking behavior. NIDA Research Monograph No. 17. U.S. Department of Health, Education, and Welfare, Public Health Service, Alcohol, Drug Abuse, and Mental Health Administration, National Institute on Drug Abuse, DHEW Publication No. (ADM) 78-581, December 1977, pp. 13-33.

(41) RUSSELL, M.A.H. Tar, nicotine, and CO yields of cigarettes. British Medical Journal 3(5982): 540, August 30, 1975.

(42) SCHACHTER, S. Pharmacological and psychological determinants of smoking. Annals of Internal Medicine 88(1): 104-114, January 1978.

(43) SCHACHTER, S., SILVERSTEIN, B., KOZLOWSKI, L.T., PERLICK, D., HERMAN, C.P., LIEBLING, B. Studies of the interaction of psychological and pharmacological determinants of smoking. Journal of Experimental Psychology: General 106(1): 3-40, 1977.

(44) SCHUSTER, C.R., THOMPSON, T. Self-administration of and behavioral dependence on drugs. Annual Review of Pharmacology 9: 483-502, 1969.

(45) SIEGEL, S. Morphine tolerance acquisition as an associative process. Journal of Experimental Psychology: Animal Behavior Processes 3(1): 1-13, January 1977.

(46) SILVERSTEIN, B. An addiction explanation of cigarette-induced relaxation. Unpublished doctoral dissertation, Columbia University, 1976, 68 pp.

(47) SOLOMON, R.L. An opponent-process theory of acquired motivation: IV. The affective dynamics of addiction. In: Maser, J.D., Seligman, M.E.P. (Editors). Psychopathology: Experimental Models. San Francisco, W.H. Freeman, 1977, pp 66-103.

(48) SOLOMON, R.L. CORBIT, J.D. An opponent-process theory of motivation: I. Temporal dynamics of affect. Psychological Review 81(2): 119-145, 1974.

(49) SOLOMON, R.L. CORBIT, J.D. An opponent-process theory of motivation: II. Cigarette addiction. Journal of Abnormal Psychology 81(2): 158-171, 1973.

(50) STEFFY, R.A., MEICHENBAUM, D., BEST, J.A. Aversive and cognitive factors in the modification of smoking behavior. Behavior Research and Therapy 8: 115-125, 1970.

(51) STEPHENS, R.M. Psychophysiological variables in cigarette smoking and reinforcing effects of nicotine. Addictive Behaviors 2: 1-7, 1977.

(52) STOLERMAN, I.P., GOLDFARB, T., FINK, R., JARVIK, M.E. Influencing cigarette smoking with nicotine antagonists. Psychopharmacologia 28: 247-259, 1973.

(53) TERNES, J.W. An opponent-process theory of habitual behavior with special reference to smoking. In: Jarvik, M.E., Cullen, J.W., Gritz, E.R., Vogt, T.M., West, L.J. (Editors). Research on Smoking Behavior. NIDA Monograph No. 17. U.S. Department of Health, Education, and Welfare, Public Health Service, Alcohol, Drug Abuse, and Mental Health Administration, National Institute on Drug Abuse, DHEW Publication No. (ADM) 78-581, December 1977, pp. 157-185.

(54) TIME MAGAZINE. The chemistry of smoking. Time Magazine, February 21, 1977, p. 48.

(55) WIKLER, A. Dynamics of drug dependence. Archives of General Psychiatry 28: 611-616, May 1973.

(56) WIKLER, A., PESCOR, F.T. Classical conditioning of a morphine abstinence phenomenon, reinforcement of opioid-drinking behavior and "relapse" in morphine-addicted rats. Psychopharmacologia 10: 255-284, 1967.

17. SMOKING IN CHILDREN AND ADOLESCENTS: PSYCHOSOCIAL DETERMINANTS AND PREVENTION STRATEGIES.

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