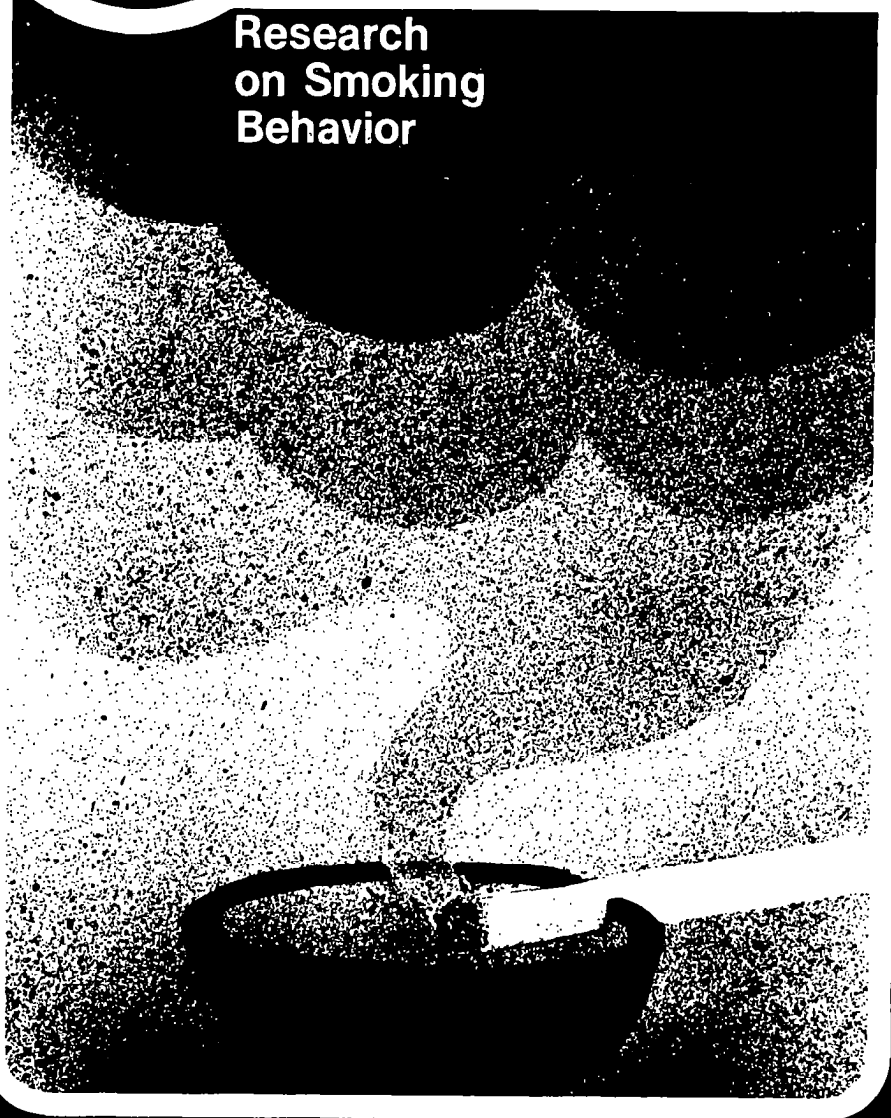
National Institute on Drug Abuse

Research 17

MONOGRAPH SERIES

Research on Smoking Behavior



U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE • Public Health Service • Alcohol, Drug Abuse, and Mental Health Administration

Research on Smoking Behavior

Editors:

Murray E. Jarvik, M.D., Ph.D.
Joseph W. Cullen, Ph.D.
Ellen R. Gritz, Ph.D.
Thomas M. Vogt, M.D., M.P.H.
Louis Jolyon West, M.D.

NIDA Research Monograph 17

December 1977

DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
Public Health Service
Alcohol, Drug Abuse, and Mental Health Administration

National Institute on Drug Abuse
Division of Research
5600 Fishers Lane
Rockville, Maryland 20857

For sale by the Superintendent of Documents, U.S. Government Printing Office
Washington, D.C. 20402

Stock Number 017-024-00694-7

The NIDA Research Monograph series is prepared by the Division of Research of the National Institute on Drug Abuse. Its primary objective is to provide critical re views of research problem areas and techniques, the content of state-of-the-art conferences, integrative research reviews and significant original research. Its dual publication emphasis is rapid and targeted dissemination to the scientific and professional community.

Editorial Advisory Board

NIDA Research Monograph series

Robert DuPont, M.D.
DIRECTOR, NIDA
William Pollin, M.D.
DIRECTOR, DIVISION OF RESEARCH, NIDA
Robert C. Petersen, Ph.D.
EDITOR-IN-CHIEF
Eunice L. Corfman, MA.
EDITOR
Eleanor W. Waldrop
MANAGING EDITOR

Parklawn Building, 5600 Fishers Lane, Rockville, Maryland 20857

Research on Smoking Behavior

ACKNOWLEDGEMENT

This monograph is derived from papers presented at a conference on smoking behavior convened at the University of California at Los Angeles, June 24 and 25, 1977.
(An indication of their impact is that the editor of the NIDA Research Monograph series has stopped smoking .)
Ms. Toby-Ann Cronin played a critical role in the coordination of the conference and in the development of this publication, under NINA contract #271-77-3422 with UCLA.

Library of Congress catalog number 77-090890

DHEW Publication No. (ADM) 78-581
Printed 1978

NIDA Research Monographs are indexed in the *Index Medicus.* They are selectively included in the coverage of Biosciences *Information Service, Chemical Abstracts, Psychological Abstracts,* and *Psychopharmacalogy Abstracts.*

Foreword

In the 14 years since the 1964 Advisory Committee's Report to the Surgeon General on smoking, awareness of the important effect of this widespread behavior on the nation's health has moved in opposite and paradoxical directions. On the one hand, the Report triggered significant changes. A distinct drop in cigarette consumption occurred in that year, and since then consumption has decreased for adult males from 52 per cent to 39 per cent and for adult females from 32 per cent to 29 per cent. A 1975 study shows the number of physicians still smoking has decreased from 30 per cent in 1967 to 21 per cent, dentists from 34 per cent to 23 per cent, and pharmacists from 35 per cent to 28 per cent. On the other hand, the knowledge has become trite and the magnitude of the damage lost sight of. Fifty million Americans still smoke. Ominously, smoking by girls between 12 and 18 nearly doubled between 1968 and 1974, eliminating the difference in smoking behavior between the two sexes. The age at which many children begin regular smoking is down to 11 to 12 years. Not only is early onset of a drug habit often predictive of heaviness of use and difficulty of cessation, but cigarette smoking is often a precursor or gateway substance to use of stronger drugs.

Most people, including health officials, are startled when the figures on smoking damage are put into perspective. For example, the number of people who annually die prematurely from smoking is estimated at 300,000. For comparison, annual automobile fatalities are estimated at about 55,000, overdose deaths attributed to barbiturates are estimated at about 1,400, and to heroin at about 1,750. Over 37 million people (one of every six Americans alive today) will die from cigarette smoking years before they otherwise would. If tobacco-related deaths were eliminated, there would be:

- 300,000 Americans each year who would not die prematurely
- 1/3 fewer male deaths from 35 to 59
- 85 per cent fewer deaths from bronchitis or emphysema
- 1/3 fewer deaths from arteriosclerosis
- 1/3 fewer deaths from heart disease
- 90 per cent fewer deaths from cancer of the trachea and lungs
- 50 per cent fewer deaths from cancer of the bladder

Given the extent of the problem, a consensus is growing that the national effort to cope with it has been defective. Not qualitatively, since good people have done substantial and important work, and many lives have been saved. But quantitatively the effort has

been too little and its priority insufficiently urgent. Cigarette smoking is the largest preventable cause of premature death, illness, and disability we have. These smoking damage figures are so large because of heavy promotion, governmental protection and subsidy,. a health industry largely preoccupied with other things, and an entrenched and overlearned addictive behavior that has proven extraordinarily hard to reduce or extinguish, But what should we think of ourselves, individually or collectively as a nation, if we concede, therefore, that we are helpless to change this toll and must learn to tolerate it?

The NIDA Division of Research has given increased priority to this issue during the past few years for several reasons: the increasing identification of smoking as a prototypic addiction, the status of smoking as a gateway drug to use of stronger or illicit drugs, and our focus on substance abuse as a generic phenomenon that includes tobacco. The Royal College of Physicians 1977 report on Smoking and Health says of the habit, " . tobacco smoking is a form of drug dependence different from but no less strong than that on other drugs of addiction.. " The current International Classification of Diseases (ICD) now lists tobacco smoking disorder as a drug problem, and, as Dr. Jerome Jaffe tellingly recounts in this monograph, so, at last, does the new psychiatric Diagnostic and Statistical Manual (DsM)III draft revision.

Public health policy on smoking should probably recognize that extinction of the habit is an unrealistic goal and even undesirable, given the adjunctive coercion such a goal would require. But we should take care to devote a degree of concern and excellence to understanding and dealing with the problem that is' commensurate with its size. The idea of control appears central, whether this is exercised in brain and CNS responses, by genetic or psychological predisposition, through learning or reinforcing factors in the cultural and institutional environment. Cur research agenda needs to address all these levels.

Correspondingly, one measure of the usefulness of the papers in this monograph is their scope, from the basic opponent process theory formulated by Dr. Joseph Ternes to the activist prevention policy presented by Dr. Ellen Gritz; from the differently impressive synoptic views of Drs. Russell and Van Lancker to the valuable particular cost figures calculated by Drs. Lute and Schweitzer. To have put between two covers a set of papers of such high caliber, timeliness, and utility is a welcome achievement.

William Pollin, M.D.
Director
Division of Research
National Institute on Drug Abuse

Preface

The National Institute on Drug Abuse has been given the lead role in the Federal Government to carry out and support research on tobacco smoking behavior. Unlike other Federal efforts in this area of public health, NIDA will focus its effort on the dependence process associated with tobacco smoking. Tobacco smoking can be viewed as a prototype dependence 'process which has a significant impact on the public health. The morbidity and premature mortality figures associated with this habitual behavior ark high. It is estimated, for example, that some 250,000-300,000 premature deaths can be directly *or* indirectly related to tobacco smoking. While much research has been devoted to the biomedical and pathological consequences of smoking (early onset of cardiovascular and pulmonary disease and lung cancer), relatively little Federal research support has been provided for understanding the biological, behavioral, psychological, and societal factors which may be substantial in the etiology and maintenance of this habitual behavior. Little, if any, research has been focused on elucidating withdrawal phenomena associated with cessation of smoking or factors leading to relapse and recidivism. Other areas of research which are ripe for development are innovative treatment procedures for teaching people how to stop smoking and pharmaco-therapeutic techniques for maintaining abstinence. Development of biological assays to detect tobacco, nicotine, and its metabolites is essential. Such technology would afford researchers a way to validate self report data in follow-up studies and epidemiological surveys of smokers.

NIDA is currently developing an extra-mural funding program targeted for research on tobacco smoking as a dependence process. Part of our effort in this area of smoking research is exemplified by this conference.

Norman A. Krasnegor, Ph.D.
Division of Research
National Institute on Drug Abuse

Monograph Contributors

Ransom J. Arthur, M.D.
Professor and Executive Vice Chairman, Department of Psychiatry and Biobehavioral Sciences;
Associate Dean for Curricular and Student Affairs
UCLA School of Medicine
760 Westwood Plaza
Los Angeles, California 90024

Joseph W. Cullen, Ph.D.
Deputy Director
-UCLA Cancer Center
Center for the Health Sciences
Los Angeles, California 90024

Emerson Foote
Life Member, Board of Directors
American Cancer Society
Gipsy Trail,
Camel, New York 10512

Bernard H. Fox, Ph.D.
Manager, Social Science Office of Field Studies and Statistics
National Cancer Institute
National Institutes of Health
Bethesda, Maryland 20014

Dorothy E. Green, Ph.D.
Consulting Research Psychologist
3509 N. Dickerson Street
Arlington, Virginia 22207

Ellen R. Gritz, Ph.D.
Assistant Research Psychologist
Department of Psychiatry and Biobehavioral Sciences, UCLA;
Psychologist, Veterans Administration Hospital Brentwood
Los Angeles, California 90073

Jerome H. Jaffe, M.D.
Chief, Psychiatric Research
Department of Biological Psychiatry, New York Psychiatric Institute
722 West 168th Street
New York, New York 10032

Murray E. Jarvik, M.D., Ph.D.
Professor of Psychiatry and Pharmacology, UCLA;
Chief, Psychopharmacology Unit
Veterans Administration Hospital
Brentwood
Los Angeles, California 90073

Robert P. Liberman, M.D.
Research Psychiatrist, UCLA
Camarillo-Neuropsychiatric Institute Research Center, Box A
Camarillo, California 93010

Edward Lichtenstein, Ph.D.
Professor of Psychology
University of Oregon
Eugene, Oregon 97403

Bryan R. Lute, M.S., M.B.A.
Doctoral Candidate, Department of Health Services, School of Public Health, UCLA
Center for the Health Sciences
Los Angeles, California 90024

Leo G. Reeder, Ph.D.
Professor of Public Health
School of Public Health, UCLA
Center for the Health Sciences
Los Angeles, California 90024

Michael A.H. Russell, BM, MRCP? MRCPsych . The Institute of Psychiatry, The Maudsley Hospital Addiction Research Unit
101 Denmark Hill
London, England

Leonard H. Schuman, M.S., M.D.
Professor and Director, Division of Epidemiology
School of Public Health
University of Minnesota
1360 May Memorial Bldg.
420 Delware Street: S.E.
Minneapolis, Minnesota 55455

Jerome L. Schwartz, Dr. P.H.
Chief, Health Care Research
Office of Planning and Program Analysis, Department of Health, State of California
714 P Street
Sacramento, California 95814

Stuart 0. Schweitzer, Ph.D.
Associate Director of Health Economics, School of Public Health, UCLA
Center for the Health Sciences
Los Angeles, California 90024

Kenneth I. Shine, M.D.
Chief, Division of Cardiology
Associate Professor of Medicine, UCLA
Center for the Health Sciences
Los Angeles, California 90024

Joseph W. Ternes, Ph.D.
Clinical Psychologist
Drug Dependence Treatment and Research Center
Philadelphia V.A. Hospital
University & Woodland Avenues
Philadelphia, Pennsylvania 19104

Phoebus N. Tongas, Ph.D.
Chief Psychologist
Department of Psychiatry
Kaiser-Permanente Medical Center
1515 N. Vermont Avenue
Los Angeles, California 90027

Julien L. Van Lancker, M.D.
Professor and Chairman
Department of Pathology, UCLA Center for the Health Sciences
Los Angeles, California 90024

Thomas M. Vogt, M.D., M.P.H.
Assistant Professor of Epidemiology
School of Public Health, UCLA
Center for the Health Sciences
Los Angeles, California 90024

Louis Jolyon West, M.D.
Professor and Chairman
Department of Psychiatry and Biobehavioral Sciences
UCLA School of Medicine
Director, The Neuropsychiatric Institute, 760 Westwood Plaza
Los Angeles, California 90024

Ernst L. Wynder, M.D.
President. American Health Foundation
1370 Avenue of the Americas
New York, New York 10019

Contents

Introduction

Murray E. Jarvik, M.D., Ph.D.

Cigarette smoking, as it exists in the world today, is a most remarkable phenomenon. It is a habit of the most widespread proportions, and a product of the twentieth century. Today, cigarette smoking has ramifications in almost every area of knowledge -- in politics, economics, psychiatry, psychology, sociology, anthropology, pharmacology and pathology. It is strange that people should go to such lengths to burn and then inhale some vegetable matter. We must find out what is rewarding about it. Furthermore, how rewarding can it be that it overcomes the clearly demonstrated punishment meted out by this tenacious habit? In an attempt to obtain some answers to these questions, the symposium was organized and this volume produced.

In order to understand the habit, we analyzed it from four different aspects: epidemiology, etiology, consequences and treatment. Thus, we want to know something about the distribution of cigarette smoking in the world today; we would like to understand why people smoke; we want to see in the light of present day knowledge just what is known about the dangers of cigarette smoking; and we want to find out how people who desire to stop smoking can succeed at this difficult task.

Leonard Schuman has reviewed the epidemiological data concerning patterns of smoking in the United States. Similar patterns seem to exist in other countries of the world, although the level of smoking seems to be the highest in our country. The three events which have had the greatest impact on smoking in this century are World Wars I and II and the Surgeon General's Report of 1963. The former increased and the latter decreased smoking. There does appear to be a general overall decline in smoking today, but only of modest proportion, clearly not a serious threat to the cigarette industry, nor a great boost to the health of the country. A major problem is women -- both teenage girls, who continue to show a rise in smoking, and adult women, who fail to show the cessation rates of men. The reason for this sex difference is not evident.

Male smokers, of course, continue to predominate over female smokers, though the gap is narrowing. There is a marked preponderance of smoking among divorced or separated persons, as compared to married

or single individuals of either sex. Smoking also seems to be more common among persons of lower socioeconomic level and lower educational achievement, except that more females in higher income groups smoke.

Ernst Wynder has considered some of the public health aspects of cigarette smoking, and has discussed measures that can or should be taken to reduce the adverse impact of smoking upon health. He has discussed the interaction of cigarette smoking with other risk factors such as alcohol, occupational hazards and hypercholesterolemia and hypertension. He feels it is particularly important for individuals at high risk for cancer or coronary disease to eliminate smoking. Smoking itself does not appear to be a causative factor in coronary disease when other risk factors are absent. Air pollution seems to have relatively little impact on health, and the evidence linking it to disease is thus far unsatisfactory. On the other hand, smoking has a major health impact. The majority of smokers want to stop, but it is evident that efforts should be made to convince the minority who wish to continue, that this is inimical to their health. Even among the group that wants to stop, most-of them will not succeed. The development of group therapies seems to be the most cost effective method, and research must be done into the prevention of relapse after termination. Parallel to these efforts is the development of less harmful cigarettes for those who cannot or will not stop smoking. Dr. Wynder suggests that greater efforts are needed and more enthusiasm should be generated from the medical and scientific professions towards the elimination of disease.

In my paper, I have tried to examine the evidence that nicotine plays a central role in cigarette smoking. There are many studies that support the view that nicotine is necessary for smoking. However, there is considerable controversy over whether a pharmacological agent is sufficient to maintain the smoking habit. We know that most of the acute biological effects (good and bad) of smoking can be attributed to nicotine. The chronic effects seem to be due to a combination of nicotine, carbon monoxide and tar. We know that on occasion, people will smoke cigarettes which have a very low nicotine content or indeed which have none at all; however, they do not like them. In many ways this behavior is similar to the drinking of decaffeinated coffee or near beer.

Inhalation of nicotine ought to mimic smoking, but no study has investigated the reinforcing effects of nicotine given by this route through physiological effects resembling smoking (Herxheimer et al. 1967). One would guess that people are not too keen to take injections of nicotine, although there are really relatively few studies which have investigated this route of administration. The fact that nicotine injections do not seem to be pleasurable, but that nicotine given in forms other than tobacco is not particularly desirable, poses a serious problem for the nicotine hypothesis. I have therefore tentatively proposed that perhaps nicotine is optimally reinforcing when it is combined with some other constituents

of tobacco. Since tar and nicotine seem to covary, I would guess that some constituent of tar may potentiate the reinforcing effects of nicotine, but this point requires further investigation with cigarettes varying independently in tar and nicotine content. We have some evidence that nicotine has greater control over smoking than tar (Stolenman et. al. 1974).

Dorothy Green has described some of the findings which were obtained in the unique surveys of smoking carried out by the National Clearinghouse for Smoking and Health in 1964, 1966, 1970 and 1975. These surveys revealed four factors to be dominant as motivations for quitting: health, example, aesthetics and mastery. Except for aesthetics, these factors might reasonably be expected to play a role in other substance abuse habits such as narcotics addiction, alcoholism and over-eating. The remarkable thing is that people go on smoking despite the wide advertising of health hazards of smoking. Hochbaum's model, with its five factors, were examined in these surveys. The factors were: Knowledge of the threat, importance of the threat, personal relevance, capability of doing something about it, and value of doing something about it. It is evident that these factors are extremely important in the public health control of smoking behavior, just as they are with the other public health hazards. From a practical point of view, the real problem is to design an effective method of dealing with each of these exceedingly challenging factors.

Silvan Tomkins' brilliant insight into motivation resulted in a theory with four smoking types: positive affect smokers, negative affect smokers, addictive smokers, and "pure habit" smokers (Tomkins 1966). Green and her co-workers found six factors which were parallel to the Tomkins typology. It is evident that the National Clearinghouse for Smoking and Health had access to material which was very unique. The problems of the survey were those of all verbal questionnaires, that the meaning of the questions had to be accepted at their face value.

Thomas Vogt presents a very scholarly paper in which he describes a method of measuring exposure to cigarette smoke by measuring carbon monoxide in expired air and thiocyanate in blood plasma. He has correlated questionnaire estimates of smoking with these chemical measures and has discussed the relative merits of each method of estimating smoking. It is interesting that the proportion of thiocyanate-carbon monoxide variance explained by questionnaire items is greater for age in which smoking was started than cigarettes per day. This, perhaps, reflects the accuracy with which subjects were able to answer these questions (a measure of validity). Dr. Bernard Fox provides a very incisive discussion of the papers in this section with particular attention to Dr. Vogt's presentation.

Dr. Ternes has presented us with an interesting application of Richard Solomon's opponent process theory to smoking. Solomon

demonstrated that in dogs conditioned to punishing electric shock, they not only developed tolerance to the situation but they developed a strong positive reaction to cessation of the negative stimulus which persisted for a long time. This was a type of non-pharmacological abstinence syndrome. In smoking, as with other forms of dependence, a difficult question to answer is why extinction is so difficult and relapse so common. The opponent process theory assumes that each form of reinforcement (a) is accompanied by an opposing reinforcement (b) which outlasts the termination of the initial reinforcement. The (b) process contributes to the abstinence syndrome seen upon abrupt termination of many forms of drug addiction. The success and usefulness of this attractive theory depends a great deal upon our ability to identify and characterize the (a) and (b) processes for each habit. There is obviously great variability in the way in which smokers react to cessation of their habit but the great majority suffer some type of deprivation symptoms which must play a great role in relapse. More research is needed to clarify and identify these processes.

Leo Reeder has examined some sociocultural factors and their relationship to smoking. First, nature has divided human beings into obvious visibly different groups on the basis of sex and age. And these different groups do show different smoking behaviors. Since smoking is an acquired habit, it is evident that teenagers smoke less than individuals over 20 years of age. But smoking is declining among adults, whereas it is increasing in teenagers. Most of the decline is in adult men, with relatively little decline in adult women. The percentage of teenage boys who smoke has remained constant over the past 20 years, whereas it has increased tremendously in teenage girls, until today they equal teenage boys in the incidence of smoking. It is evident that girls are taking up smoking more readily, and women are giving it up with much greater difficulty than boys or men. trend continues, female smokers will outnumber male smokers. It is interesting to speculate on why women are so susceptible to smoking today. Is it a consequence of the "women's liberation movement"? Or could it be that women have become aware of the fact that they are less at risk from cardiovascular dangers of smoking than men are?

Among the sociological factors that are easily measured, it can be seen that smoking rates are highest among divorced or separated individuals. Again, we have the problem of deciding which is cause and effect. Getting divorced may make individuals smoke more, perhaps because of the stresses and strains associated with such a change in marital status. It seems unreasonable to assume that smoking per se will drive people into divorce, although a non-smoking spouse may not find it comfortable to live with a smoking partner. Perhaps there is a third factor of emotional stability which leads people both to smoke and to get divorced. This is clearly a phenomenon of great social importance and worth investigating.

The role of socioeconomic status in smoking is complicated. There is an interaction with sex and educational level. Poorly educated women are less apt to smoke, whereas poorly educated men are more apt to smoke. The converse seems to be true of the upper end of the educational level. It is of some significance that smoking prevalence is one of the exceedingly few behaviors in which the sexes fill opposite trends on the socioeconomic continuum. More research is needed to find out whether poorly educated women and well educated men desist from smoking for the same reasons. One might guess that the educated men don't smoke because they are strongly influenced by the health hazards. Poorly educated women, on the other hand, take their lower class sex roles very seriously and have been prohibited from smoking, not for health reasons but because religious and cultural mores prohibit them from doing so. Dr. Reeder doesn't give the figures, but we might guess that incidence of smoking is much closer between well educated men and women than it is between poorly educated men and women.

Most psychiatrists have noted that there is a higher frequency of smoking in their patients than in a general population. Reeder corroborates this impression with results from the Midtown study, where mental health was inversely related to smoking in men.

Other sociocultural factors identified in smoking are important and should be explored further. Social pressure makes individuals conform to the behavior of parents, sibs and peers, and smoking behavior of course is influenced in the expected direction. It is evident that smoking, like all other forms of substance dependencies, is molded by the influence of people surrounding the smoker. Jerome Jaffe discussed the conditions under which tobacco use could be considered a psychiatric disorder. It is certainly an anomaly that cigarette smoking has been viewed in contemporary society by a very different light from traditional addictions such as opiate, alcohol, and barbiturate addictions. It is obvious that in many individuals dependence upon tobacco is every bit as strong as dependence upon these other drugs. Dr. Jaffe has made a very careful review of the conditions under which tobacco use should be considered a psychiatric disorder to be listed in the third edition of the American Psychiatric Association's Diagnostic and Statistical Manual (DSM-III). The inclusion of Tobacco Use Disorder in this manual should have a profound effect upon the reputation of this behavior in the community and may hopefully result in the application of third party payments for the treatment of the disorder.

Bryan R. Luce and Stuart 0. Schweitzer present a very provocative discussion of the economic costs of smoking-induced illness. Their analysis is of necessity based upon somewhat limited data and, therefore, entailed a good deal of extrapolation. But their final estimate that smoking costs us about 42 billion dollars a year or 2½% of the Gross National Product is really a frighteningly large

figure. It is evident that economic considerations alone should prompt the government to do something to correct this situation.

Julien van Lancker has given a very comprehensive survey of the danger of smoking, as perceived from its earliest discovery to the present times. He points out quite properly that even though it was known that tobacco ought not to be smoked from earliest times, attempts at suppressing it invariably failed. Of course, it may be that in. the old days the negative reinforcement for smoking came mainly from the threats and punishment meted out by man, whereas today the approach is more rational and emphasizes the disease causing properties of the habit. I would expect that this scholarly chapter will become an important reference work for those interested in the pathological changes wrought by smoking.

After considering the composition of tobacco, Dr. Van Lancker discusses the toxicology of tobacco components. He discusses the myriad of compounds which can cause harmful effects, including nicotine, carbon monoxide, methyl alcohol, lead and arsenic. Then he discusses the effects of smoking on the cardiovascular and respiratory systems. Smoking influences pregnancy deleteriously. Fully a third of this paper is devoted to the relationship between smoking and cancer. He discusses possible mechanisms in some detail and carefully documents each point. Despite the massive amount of evidence linking cancer and smoking, Dr. Van Lancker concludes that "a clear-cut causal relationship between cigarette smoking and cancer has not been demonstrated." Some experts would disagree with this conclusion and would argue that no stronger causal relationship has been shown for any other disease and a presumed etiological agent.

The last section of this symposium deals with the practical steps that can be taken to reduce cigarette smoking in our population. In smoking, as in all other habits, "an ounce of prevention is worth a pound of cure". Ellen Gritz discusses the various methods which have been used, and which might be further used to prevent the onset of smoking. School programs in San Diego and Los Angeles, California, and in Houston, Texasproduce a sizable reduction in smoking in the most susceptible groups of teenagers. As the proportion of smokers in the general population decreases, it should become progressively easier to convince school board members to institute such programs in the city schools. The other medium to which teenage children are exposed a great deal is television. Since the anti-smoking spots have disappeared, television has probably had less impact on the smoking habits of teenage children. Twenty years ago, movie and television heroes. and heroines smoked, but nowadays by and large, they do not. They are role models for susceptible audiences and ought to help in diminishing smoking.

Although the vast majority of smokers begin the habit in their mid or early teens, there is a small number of individuals who

begin smoking at later ages, even into the sixties and seventies. The later an individual begins to smoke, the less of a problem smoking is because there is obviously less cumulative effect. Furthermore, the ease of stopping is probably inversely related to the age of starting. Until twenty years ago, there was little reason for intelligent people to stop smoking. To be sure, there were always aesthetic and religious reasons, but the one major factor which today deters and convinces people that they should not smoke was lacking. This was the official pronouncement by the United States Government that smoking is harmful (Surgeon General Report 1964). In the middle of this century, the majority of physicians smoked and gave their stamp of approval to their patients who smoked. Today, the picture is quite different since only a fifth of physicians continue to smoke, and each year the number of physicians who smoke is diminishing. The major reason that people stop smoking is because they recognize some type of personal danger from the habit. This is a highly abstract intellectualized reason in most instances since the health consequences of smoking are so long delayed. Thus individuals stop smoking either through forma-programs of some type or else - spontaneously on their own. The Graham and Gibson study (1971) showed the importance of an intellectual grasp of the health . hazards of smoking. They also showed that actual serious illness had a great impact upon smokers and caused them to stop smoking. Of course in addition to their societal pressures from friends and family, media messages also played a role. Dr. Gritz has pointed out that in dealing with smoking, the psychoanalytic defense mechanisms are all employed by the smoker to deny or repress the deleterious effects of smoking.

Jerome Schwartz has presented a comprehensive report on the various methods used to induce smoking cessation, and then he describes what procedures are employed in different countries.

Dr. Schwartz has done a commendable job in attempting to make order out of chaos. It appears that smoking control methods have improved in the last decade, though there is no clearly definable reason that one could attribute such success to. Obviously, long-term results are more important than short-ten results, and progress is being made in finding prognostic indicators of long-term success. The most general finding which requires further investigation is that women are more difficult to cure than men, both on a short-term and on a long-term basis. The most important contribution of Schwartz' paper is that it allows one to compare effectiveness of different methods.

As one can see from the tables, success in keeping clients abstinent for a year varies considerably for different programs. Some, who must be quite honest in reporting, report a low of zero per cent (Pederson 1975; Kreutzer 1967). Others report phenomenal success with a high of 88% (Kline 1970) or 85% (Quarter 1972). Of course, these results should be scrutinized

much more closely to see what they really mean and just how replicable they are likely to be.

However, there is such tremendous variability in results within each category of treatment that it is evident that something other than the treatment method contributes to success. It may be the setting, the personality of the therapist, or some other individual factor in the patient. During the next few years, it will be important to pin down the factors which are prognostically important so that a more rational approach to therapy can be evolved.

Emerson Foote has provided us with an eloquent plea for the elimination of cigarette advertising and the substitution of a publicly subsidized educational advertising campaign against cigarettes. There is no question that the barrage of information regarding cigarettes, which reaches the public is one-sided. An exception must be made for members of the medical profession and highly educated groups who have ready access to health information. Although I agree with Mr. Foote in principle, it is evident that putting his plan into action would be quite difficult. Common sense tells one that the mere tolerance of cigarette advertising in the community seems to justify it and give the habit tacit approval . And yet it is hard to obtain a quantitative measure of just how much influence advertising has upon smoking. In countries with a collective economy such as the Soviet Union or Communist China, there is no cigarette advertising. And yet, the incidence of smoking is extremely high. To be sure, it might be even higher if cigarette advertising were allowed. My own feeling is that although I am sympathetic with Mr. Foote's aims, I feel that from a practical point of view, more would be accomplished if we ignored cigarette advertising by the cigarette industry, but attempted to mount a more vigorous campaign of anti-cigarette propaganda. I do believe, even though it is difficult or impossible to prove, that when the equal time arrangement existed on television and there were ads against cigarette smoking sponsored by various health agencies, that these did have a direct effect in reducing smoking.

In recent years, Senator Gregorio of the California State Senate has attempted several times to get a bill passed whereby the state of California would subsidize anti-smoking advertisements. Needless to say, the cigarette industry has been very vigorous in their opposition to such a bill, and indeed the bill has not yet succeeded in passing the legislature. It is conceivable that if it were subject to referendum, it would be passed since anti-smoking sentiment now is more prevalent than pro-smoking sentiment in California citizens.

Controversy has always surrounded attempts by the government to regulate or even influence personal habits, and yet in a sense that is what government is for. As Luce and Schweitzer pointed out in this volume, allowing people to harm themselves in great numbers

has a significant economic impact upon the rest of us. Where does one draw the line in civil rights? Because of the disastrous results in alcohol prohibition, few people would recommend an outright prohibition ofcigarette sales. A strong argument can be made for legalizing vice (alcohol, drugs) since it can be better regulated than when it is left in control of the underworld. Outlawing something which is widely desired has never solved a problem. The answer is to find out why a habit like smoking is so strongly reinforcing and then determine whether the harmful components can be eliminated and the pleasurable components retained.

The political implications of smoking analysis are rather complex. The tobacco industry and the Department of Agriculture support smoking, whereas the Department of Health, Education and Welfare opposes it. Although the financial advantage appears to be on the side of the smoking forces, the movement nevertheless seems to be occurring in the direction of less smoking. Ironically, it doesn't matter whether a government is free and democratic as our own, or a dictatorship such as exists in the Soviet Union. Smoking seems to be tolerated as long as it seems to bring in revenue to the treasury of the country. King James I, who started out as an implacable foe of tobacco, became much more tolerant of smoking when large revenues from tobacco began to flow into his treasury. When those in charge of the economics of the country can be made to realize that the cost of smoking is greater than the income it produces, only then will there be official governmental policy against this pleasurable habit.

Edward Lichtenstein has given a brief overview of recent developments in social learning approaches to smoking. Rapid or forced smoking, a procedure popularized by Lichtenstein, was found to be one of the most effective methods of helping people to stop smoking. There is still some concern about possible cardiovascular risks to some individuals. The most popular programs are multicomponent in nature, and as can be expected, did give the most divergent results with one year follow-ups of success ranging from 20% to 50%. Controlled smoking, like controlled drinking, is a controversial area. Advocates of the "cold turkey" method of cessation do not feel that it is profitable for individuals to attempt to merely cut down. Lichtenstein feels now that external stimulus control alone might not be sufficient to persuade all smokers to stop, and that information on physiological processes, particularly nicotine, may play an important role. Tension reduction by other methods such as relaxation procedures ought to substitute for some of the reinforcing effects of smoking, but thus far results have been mixed. Another area which has been relatively neglected is the investigation of relapse episodes and what causes them. The dynamics of relapse needs careful study and explication.

It may be better to have a therapy which only cures a very small fraction of the population, but which can be utilized widely rather than a therapy which results in a high cure rate which is

very expensive and available to only very few individuals. Obviously, treatments which require individual therapists are the most expensive , but it is not clear whether they are also the least cost effective. One of the most important problems of smoking research is that it is carried on by graduate students who have only a short time to study patients, perhaps three or four months. This is insufficient to determine the true effectiveness of any given method. It will be necessary to subsidize smoking clinics so that long-term follow-ups will be feasible.

Phoebus Tongas' paper addresses some of the problems raised by Dr. Lichtenstein. His smoking clinic is embedded in a long-term prepaid health maintenance group. In this setting, he has been able to compare several different methods of achieving non-smoking behavior.

These have included aversive conditioning (rapid smoking), covert conditioning, behavioral group therapy, and a combined condition. The best procedure was the combined one which involved multiple therapies. At the end of one year, this group had 77% complete abstinence, and at the end of two years 64% abstinence. This study is based upon 72 subjects. Tongas points out that the study of long-term maintenance of non-smoking behavior is the major area which must be explored in the future. Like Lichtenstein, he points out that reinforcement of Ph.D. candidates for studies of this type is inadequte because the payoff is low and the risk is high. Consequently, such research is neglected. I might add that research into the long-term efficacy of any type of psychotherapy is largely lacking. Tongas echoes the complaint of Lichtenstein that long-term follow-up research is needed, but is not well supported nor is the delayed reinforcement desired either by Ph.D. candidates or researchers. The only answer is a special governmental program designed specifically to support this type of research. The study of smoking cessation procedures may be taken as a model for psychotherapy. First, smoking, unlike other forms of drug abuse, is a legal habit which can be studied with few constraints. Secondly, unlike most forms of psychopathological behavior, there is a clearly definable endpoint, namely non-smoking. All the difficulties inherent in most forms of psychotherapy are also present in the therapy of smoking. Tongas suggests that we concur that future research on the therapy of smoking behavior might focus upon respondent, cognitive, and operant behavior. We've only scratched the surface in reviewing the factors that may be useful in this type of therapy.

Dr. West discussed the use of hypnosis in the treatment of the smoking habit. There is apparently considerable variability in the success of this method. The good hypnotic subject can often be induced to stop smoking permanently with a single hypnotic treatment. Many such cases have been reported. At the same time, there are some subjects who soon resume smoking no matter how expertly they are hypnotized. Spiegel (1970)) Kroger (1976), Hall and Crasilneck (1974) and others have reported various approaches

to hypnotherapy in treatment of smoking, with results ranging from 20% to 90%. There are several advantages to hypnosis over other treatments for smoking. 'It requires little or no equipment. In favorable cases, the time demands upon both therapist and patient can be relatively small. However, it is often necessary to couple it with other forms of therapy and with aggressive follow-up procedures to make sure that cessation is permanent.

REFERENCES

Graham, S. and Gibson, R. W.. Cessation of patterned behavior : withdrawal from smoking. *Social Science and Medicine,* 5:319-337, 1971.

Hall, J. S., and Crasilneck, H. B. An evaluation of current methods of modifying smoking behavior. *J. Clin. Psychol.* 30(4):431-438, 1974

Herxheimer, A., Griffiths, R. L., Hamilton, B., and Wakefield, M. Circulatory effects of nicotine aerosol inhalations and cigarette smoking in man. *Lancet, 2:754-755,* 1967.

Hochbaum, G. Behavior in response to health threats. *Annual Meeting, American Psyohological Association,* Chicago, Illinois, September 1960.

Kline, M. V. The use of extended group hypnotherapy sessions in controlling cigarette smoking. *Int. J. Clin. Exp. Hypnosis,* 18:270-281, October 1970.

(Editorial note: References to Kreutzer, 1967, Pederson, 1975, and Quarter, 1972, studies can be obtained directly from Dr. Jerome L. Schwartz)

Kroger, W. S. and Fezler, W. D.. Excessive Smoking. In: *Hypnosis and Behavior Modification: Imagery Conditioning.* Philadelphia: J. B. Lippincott Co., 1976

Spiegel, H.. A single-treatment method to stop smoking using ancillary self-hypnosis. *Int. J. Clin. Exp. Hypnosis,* 18(4):235-250, 1970.

Stolerman, I. P., Bunker, P., and Jarvik, M. E.. Nicotine tolerance in rats: role of dose and dose interval. *Psychopharmacological,* 34:317-324, *1974.*

Tomkins, S. S.. Psychological model for smoking behavior. *Am. J. Public Health and the Nation's Health,* 56(12): 17-20, 1966.

AUTHOR

Murray E. Jarvik, M.D., Ph.D.
Professor of Psychiatry and Pharmacology
Neuropsychiatric Institute
University of California
760 Westwood Plaza
Los Angeles, California 90024

Chief, Psychopharmacology Unit
Veterans Administration Hospital Brentwood
Wilshire and Sawtelle Boulevards
Los Angeles, California 90073

Smoking Problems: An Overview

Michael A.H. Russell, M.B., MRCP, MRCPsych.

> "This is the ghost of normal everyday assumptions which declares that the ultimate purpose of life, which is to keep alive, is impossible, but that this is the ultimate purpose of life anyway, so that great minds struggle to cure diseases so that people may live longer, but only madmen ask why. One lives longer in or&r that he may live longer. There is no other purpose. That is what the ghost says."
>
> Robert M. Pirsig, *Zen and the Art of Motorcycle Maintenance,* 1974.

At times I feel more akin to Pirsig's "madmen" than to the "great minds" and although I have certainly devoted more tine to struggling with the problems of preventing smoking related disease than to the art of motorcycle maintenance, I still sometimes question which is more important. For someone in this position there is clearly a bias to seek a solution to the smoking problem by pursuing the goal of safer smoking rather than that of abstinence and no-smoking.

DEFINING THE PROBLEM

In Britain, three out of four smokers either wish to or have tried to stop smoking; yet it is doubtful whether more than one in four of them succeeds in stopping permanently before the age of sixty (Royal College of Physicians, l977). The position is probably similar in the U.S.A. Thus, most people seem to smoke not because they want to but because they cannot easily stop and for them smoking is obviously a compulsive activity rightly classed among the addictive behaviors, though possibly not, as Jerry Jaffe would have it, elsewhere in this monograph, as a psychiatric disorder. Certainly it is a problem for them.

A problem can be defined as the situation that exists when one's path to a particular goal is thwarted, and it is dispersed when one either finds a way through and solves it or decides that the goal is not

worth pursuing anyway. Smoking is a problem for two reasons: because it is addictive and because it is harmful. Addiction is not a problem per se. It only becomes a problem when one strives to overcome it, and the main reason for wishing to overcome smoking is because it is harmful . If it were not harmful there would be no need to stop, and hence no problem. On the other hand, it it were not so addictive it would pose little problem for it would then be far less difficult to stop. It is the concurrence of both harmfulness and addiction that accentuates the problem.

Part of our failure to resolve the problem of smoking results from lack of clarity about our goals. For example, "How to stop people from smoking" is really composed of two quite separate problems: a) How to help people who want to stop; b) How to convince those who do not want to stop that they ought to stop. One requires treatment and guidance to overcome dependence, the other requires motivational techniques. In our discussions we have not always kept this distinction in mind. However, our major muddle is about *the* ultimate goal. At times it has seemed that the ultimate goal is to prevent and stop people from smoking. Yet this is merely secondary. The primary goal is surely to reduce and prevent smoking-related disease. If smoking were not so harmful there would be no need to stop and prevent it. At this conference, we seem to have paid little more than lip service to the goal of safer smoking as a practical and realistic approach to the solution of the primary problem of how to reduce and prevent smoking-related disease.

CHANGE IN SOCIAL CLIMATE

It is now 28 years since Wynder and Graham reported in the pages of the Journal of the American Medical Association (Wynder and Graham, 1950) that tobacco smoking, especially cigarettes, "seems to be an important factor in the induction of bronchiogenic carcinoma". In so doing they turned a pleasurable pastime into a major problem.

Since the 1950's, there have been all manner of anti-smoking campaigns, in the press, on radio, on television, in schools-and at places of work. Health warnings have been put on cigarette packets. Withdrawal clinics have been opened (and closed). There are frequent reports in psychological journals of attempts to change smoking attitudes and behavior, and almost every issue of medical journals contains an article on some aspect of the harmful effects of smoking. Restrictions have been imposed on smoking in certain public places. Advertising of cigarettes has been banned on television in some countries, and banned altogether in others.

What has all this achieved? It has certainly changed the social climate from one of approval to a general belief that people should not smoke, so that now most smokers do at least want to stop even though relatively few succeed. There has been, as Leonard Schuman and Leo Reeder point out, a modest decline in smoking among men of middle-age and high socio-economic status. But women and children smoke as much as ever. Some 20 million Britons and almost 60 million Americans still smoke. Yet one in three of them will die as a result of it - or so we are told by the Royal College of Physicians in their latest

report published in June of this year (1977).

So we have shown in this monograph that smoking is harmful (see Wynder, Van Lancker), we have told smokers so and most of them accept it (see Dorothy Green). Why then do they go on smoking? - due to the Dependency Factor. It is this block that creates the problem. Thus, while substantial progress has been made in public education and motivational approaches, we have got virtually nowhere towards our prime goal, because we cannot yet do much to help smokers overcome their dependency.

THE DEPENDENCY FACTOR

Tobacco smoking is a form of drug dependence and the modem cigarette is a highly efficient device for self-administering the drug nicotine (Russell, 1976b). By inhaling, the smoker can get nicotine to his brain more rapidly than the heroin addict can get a "buzz" when he shoots heroin into a vein. It takes only 7 seconds for nicotine in the lungs to reach the brain compared with the 14 seconds it takes for blood to flow from arm to brain. Furthermore, the smoker gets a "shot" of nicotine after each inhaled puff. At 10 puffs per cigarette, the pack-a-day smoker gets more than 70,000 nicotine shots. to his brain in a year. It is hardly surprising that cigarette smoking is so addictive.

Once in the body, nicotine is potent and varied in its effects. By its sedative action it can literally "calm the nerves" and reduce muscle tension especially in those who are anxious and worried. But it is also a stimulant, helping to allay boredom and fatigue and in some cases to improve thinking and concentration and the ability to cope with stress. Above all smoking is a source of enormous pleasure; though it is not clear how much this is due to some subtle action of nicotine on the brain or to other factors such as the oral aspects of its involvement in social rituals.

To assess nicotine intake simply in terms of the numbers of cigarettes smoked, their nicotine yield, or even the number of puffs and butt length at which the last puff is taken, is for present-day standards far too crude. By regulating puff-rate, puff-size and the amount of inhalation, a smoker is able to exercise an accurate and almost instantaneous control over his nicotine dosage. Blood nicotine levels vary markedly between different smokers and range from below 10 to over 50 nanograms per millilitre of blood. However, any individual smoker obtains a fairly consistent level after each cigarette, whether it is smoked in the morning or afternoon from one day or week to the next (Russell, 1976b).

The role of nicotine and other factors is discussed in the etiology section of this monograph. The degree to which smokers modify their smoking pattern to regulate nicotine intake is uncertain (Russell, 1977 in press; 1978 in press). Murray Jarvik's paper expresses doubt that tar alone could be rewarding at all and suggests his co-factor hypothesis to explain this. However, it is not clear why he, should object to interpreting any reinforcing value of tar as due to secondary or conditioned reinforcement arising from the frequent

and close association with nicotine.

People take up smoking, usually in adolescence, for a variety of psychosocial reasons - to look "tough" or "grown-up" or because "most of their friends smoke". Indeed at this stage the effect of nicotine is usually unpleasant rather than pleasant. But tolerance soon develops to the unpleasant side-effects and, as this threshold is passed, increasing amounts of nicotine get inhaled so that the smoker progresses almost inevitably to a stage of dependence. Just how easily this occurs is shown by the startling statistic that of those teenagers who smoke more than one or two casual cigarettes only 15% will avoid escalating to regular dependent smoking (McKeMell and Thomas, 1967).

Once a smoker - always a smoker! This is only a slight exaggeration. As mentioned already, it is unlikely that more than one in four smokers succeeds in giving up for good before the age of sixty. But this is not through lack of trying. Three *out* of four smokers either wish to or have tried to stop smoking, and continue simply because they cannot easily stop. In other words, they smoke because they have become addicted. It is only a small minority - 2% according to one study (ibid.) - for whom smoking is a take-it-or-leave-it affair, and who limit themselves to intermittent or occasional smoking, once or twice a week or less.

In essence the term "dependence" or "addiction" refers to a state in which the urge or need for something is so strong that the individual suffers or has great difficulty in doing without it, and in extreme cases cannot voluntarily stop using it when it is available. Tobacco smoking clearly falls into this category, and few other forms of drug-taking are as addictive as the puff-by-puff shots of nicotine obtained by smoking cigarettes. Not with alcohol, cannabis and possibly even heroin is the addiction so easily acquired. For most people, to smoke cigarettes at all is to become dependent. Cigarette smoking is clearly a drug addiction problem. Until this is understood we can make little headway towards a solution.

WHY CIGARETTE SMOKING IS SO ADDICTIVE

In view of the practical implications it is worth digressing to consider some of the possible reasons why cigarette smoking is so *addictive*, but in this difficult area one cannot do more than make suggestions . The opponent process theory presented so clearly by Joseph Ternes in these pages is ingenious but I must confess to my failure so far to study it sufficiently closely to assess its application to cigarette smoking. The suggestions here are based on a more traditional and straightforward learning theory approach.

This is not the place to discuss the nature of dependence or addiction, nor to go into a semantic and conceptual clarification of physical versus psychological dependence. More detailed consideration of these issues can be found elsewhere (Russell, 1976b; Russell, 1976c). I have used the terms "dependence" and "addiction" interchangeably to refer the urge or need for an object or activity. How high a degree of dependence is required before it is labeled as a "dependence order" or "addiction" is somewhat arbitrary. Furthermore,

pharmacological rewards both primary (e.g., stimulant, euphoriant, anxiety-reducing actions) 'and acquired (relief or avoidance 'of physical withdrawal effects) are really no more than one class of reinforcer, just as psychological rewards or social pressures are other classes. The degree of dependence on a particular object or activity is governed by its power as a positive reinforcer rather than the class of reinforcement it provides. Thus, strong psychological or social rewards will make for a higher degree of dependence than weak pharmacological ones. If the term addiction is used to denote strong dependence, it need not be restricted to refer only to strong pharmacological needs but could equally apply to strong psychological needs, as in the case of addiction to gambling or television viewing.

Reference has already been made to the very rapid and very numerous pharmacological reinforcements afforded by the puff-by-puff nicotine-bolus form of intake from inhaled cigarette smoking. This is unmatched by any other form of drug-taking, and is further enhanced by the rapid clearance and metabolism of nicotine. Its short half-life in blood and brain allows repeated and frequent use without loss of effect. It also produces a sharp "let-down" from those effects which depend on a direct action.

The wide variety of reinforcements is again unmatched by other forms of drug-taking. As Jarvik has mentioned, smoking doses of nicotine produce a whole array of effects both centrally and peripherally which could be highly rewarding. Added to these are the various psychological and social rewards which operate mainly in adolescence to determine the onset of smoking (Royal College, 1977; Russell et al., 1974).

Perhaps the most important reason for the high addictive potential of cigarette smoking is that it does not impair performance. Unlike alcohol and many other drugs of dependence, nicotine enhances rather than impairs the capacity of normal people to work and socialize. There are, therefore, no immediate negative consequences. All the uncomfortable health consequences are, for most smokers, extremely remote in time and are therefore of weaker influence. Indeed, most smokers do not stop until the motives to stop are strengthened by being experienced in the here-and-now as occurs, for example, with a current health problem, or financial crisis, or when in a non-smoking environment .

Another reason is the relative social acceptability of smoking. Although the social climate has changed, cigarette smoking is still, in most social circles, far more acceptable than use of other drugs, with the exception perhaps of tea and coffee, or sleeping pills (taken at night but not by day), or tranquilizers which are medically prescribed. It is, for instance, more acceptable to smoke than to drink in the morning. Smoking is acceptable in most social settings and this enables the conditioning of numerous environmental cues to smoke.

The wider social acceptability is obviously partly linked to the fact that performance is not impaired.

Another factor is its availability. In modem societies, cigarettes must be. one of the most 'readily available of all 'commodities - second only to water or the air we breathe. The availability is also linked to low financial cost. Two packs a day is a good deal cheaper than a bottle of gin.

Finally, cigarette smoking combines a pharmacological effect with a sensorimotor ritual. The ritual involving virtually all the senses provides an elaborate network of sensory and motor stimuli to act as substrates for secondary conditioning. The intimate involvement of the mouth no doubt also contributes strongly, for few other areas even approach the mouth as a locus of pleasurable self-indulgence.

PREVENTION OF RECRUITMENT TO SMOKING

The analysis of the smoking problem discussed so far has suggested that the motivational approaches employed by anti-smoking educational campaigns have succeeded in changing the social climate to a general belief that people should not smoke, so that most smokers would indeed like *to* stop but are prevented by their dependence on nicotine. This is difficult to reconcile with the continuing high incidence of recruitment of new smokers among teenagers at school and soon after leaving school. They are not yet affected by the dependency factor but are, nevertheless, taking up smoking at a younger and younger age. In Britain, some children begin to smoke at 5 years of age, and it has been found that about one third of adult smokers began before they were 9 (Royal College, 1977). Why have anti-smoking campaigns been successful in motivating (though not enabling) adults not to smoke, but failed with children?

There must be some very powerful psychosocial motives to smoke which operate in children but not in adults. Part of the answer may be as follows. During the adolescent stage of personality development two important processes operate to determine life-style and behavior. One is modeling and identification with adults; the other is rebelliousness against adults to assert one's own self vis a vis the kind of person adults want one to be. Now, smoking can be used symbolically to serve either of these major needs, both of which are of far more immediate concern to the teenager than possible negative health consequences many years later. One implication of this interpretation is that as long as there are adult models who smoke, children will continue to take up the habit.

TREATMENT FOR SMOKERS

Those who have worked in a withdrawal clinic or had anything to do with helping people to stop smoking know how hard a task it can be. Attention has been drawn to the striking similarity in the relapse rates after treatment for smoking, alcoholism and addiction to heroin (Hunt et al.. 1971). At the Smokers Clinic at the Maudsley Hospital we have had direct-experience with more than 500 clients (Russell, 1977). We have tried hypnosis, lobeline tablets, electric aversion, tranquilizers, beta blockers, covert sensitization, nicotine and lobeline aerosols, satiation procedures, nicotine chewing gum, rapid smoking, and cue exposure. We have treated people individually or

in small groups. We have oscillated from behavioral methods to drugs and back again. We have not had a break-through.

Our results have been similar to those of other workers as outlined by Jerry Schwartz elsewhere in this monograph. About 60-80% of participants have stopped by the end of treatment, but the success rate at one year follow-up dwindles to 20-30% depending on the base. We have, however, learned that if a smoker &es not stop or almost stop within one to two weeks, it is not worth carrying on with the treatment (Russell et al., 1976). No treatment method has, in our hands, had a strong specific effect with much advantage over placebo and simple support plus record-keeping. But this is not as discouraging as it sounds, for the attention-placebo effect itself can be quite strong, which suggests that it might be worth developing and deliberately enhancing it rather than discounting it. It is after all the only positive smoking treatment effect which has been universally and unequivocally demonstrated.

Schwartz's review focuses mainly on comparisons between different studies and different forms of treatment. He believes that the results are beginning to show an improvement. However, this does not necessarily mean that treatment methods are improving; it could equally well be due to the selection of greater numbers-of better motivated and less dependent subjects. In my view, most of the variation between studies is due to differences in the subjects treated rather than the effectiveness of the treatment methods used. Indeed, it is virtually impossible to make valid comparisons between treatment methods which are not based on random assignment of subjects after identical sampling and selection procedures. In other words, straight comparisons between different studies done at different centers using different types of subjects and different selection procedures are virtually meaningless.

At our clinic, for example, all subjects go through quite a protracted assessment involving questionnaires, a clinical interview and at least a week of base-line record-keeping before being taken into a treatment trial. About half of them drop out during assessment and do not even start treatment. Our results have been based on those who start treatment and would be only half as good (or twice as poor) if based on those who attended the initial assessment interview. Our approach is geared for testing and comparing treatment methods within our own samples, but not for direct comparison of success rates with those of other workers. Besides attracting and using different kinds of subjects, different centers use different selection procedures and different bases for their success rates. For example, walk-in clinics are at a disadvantage if they use all first attenders as their base,. On the other hand, some studies base their results only on those who complete treatment.

Another problem regarding the assessment of treatment efficacy is frequently overlooked. While many studies use attention-placebo controls, very few include no-treatment controls, possibly for ethical reasons. As mentioned above, we have not yet found a treatment method which produces better long-term results than attention-placebo controls, but our attention-placebo controls have always done substantially better than no-treatment controls whose success rate among

our subjects is about 5%. It is only by comparison with no-treatment controls that any valid assessment of treatment efficacy can be obtained. When this is done the true success rate at one year follow-up seldom exceeds 15-25% at best, and virtually all of this effect is due to attention-placebo response.

Though one might view 15-25% as a worthwhile success rate, the lack a specific treatment effect after some twenty years of research effort is a sad reflection on behaviorist and pharmacological skills. Apart from the limitations of the treatment procedures, the poor results are probably largely due to the fact that it is usually the most difficult cases who seek treatment. Anti-smoking procedures have been tested mainly on two kinds of subjects. On the one hand are the extreme cases who attend withdrawal clinics. They tend to be neurotic, highly dependent and beset by other problems. The other main source of subjects are groups of first year social science students who may not be so dependent but have little motivation for permanent abstinence .

The situation is well illustrated by the Allen and Fackler Study of parents of Philadelphia school children (1967). Questionnaires were sent to 30.796 parents: 21.553 were returned: 11.477 of these were smokers of 'whom 4,775 expressed a desire to stop-smoking. These were offered treatment but only 150 attended the withdrawal clinic. Sixty-four of them stopped smoking but at one year follow-up only thirty-five were still abstinent. Only thirty-five ex-smokers out of more than 30,000 contacts; and who is to say that these thirty-five would not have stopped on their own without the intervention? However, the main lesson of this study is the small and probably highly biased sample of 150 (3%) who attended the clinic, out of 4,775 smokers who wanted to stop.

SOME NEW APPROACHES TO TREATMENT

The traditional clinic approach typically involves the application of intensive, relatively costly and largely ineffective treatments to highly selected, small and rather unpromising groups of smokers. In my opinion, this is for practical purposes a waste of time. Its only justification is as a research tool to seek better methods and to increase understanding of the problems involved. A change of strategy is required. *We* are beginning to explore the following four approaches :

(i) Because of the large numbers, literally millions, of smokers who require help to overcome their dependence, for treatment to be effective at a national level it must be simple to administer and economical of therapists' time. Ideally, the focus should be on the development of self-treatment packages and methods suitable for use on the mass media. A success rate of only 10% achieved by these methods would be more useful than a 100% success from electric aversion requiring fourteen 45 minute individual sessions. The development of brief simple methods which could be used by family doctors, occupational health nurses, etc., during the course of their everyday practice would also be fairly cost-effective.

(ii) To gain access to more "normal" smokers with better prospects of success. This approach obviously dovetails with the' first approach, since mass media, family doctors, etc., enable contact to be made with a wide range of smokers. Active intervention at the place of work as suggested by Wynder in his paper is another way to achieve this.

(iii) To focus on preventing relapse as much as on achieving initial abstinence.

(iv) To tailor the treatment to the individual smokers particular problem: e.g., pharmacological vs. psychological, lack of motivation vs. high dependence.

DEPENDENCE AND MOTIVATION TO STOP

To clarify the problem of stopping people smoking, it is helpful to have in one's mind a simple two-dimensional model. It seems that there are two main dimensions of relevance - Dependence and Motivation to stop. Some people do not stop simply because they are not motivated. Others are motivated but fail because they are highly dependent. There is not much point offering treatment to someone low on motivation because he is unlikely to come and get it. Neither is there much point merely trying to motivate someone who is already highly motivated but continues smoking only because he is highly dependent.

Yet this is precisely what happens at many withdrawal clinics. Their clients are usually highly motivated already, but they are nevertheless shown films and given leaflets on the dangers of smoking;-they are told endlessly how much their smoking is costing them financially and how much better they would feel if they stopped. For good measure, pictures or even a specimen of cancerous lung in a bottle is sometimes handed around. It is not surprising that they fail, for they are simply being told things they already know and which have already motivated them to take the. trouble to go to a clinic. They are already motivated and what they need is not to hear what they already know but to receive help and guidance with overcoming their dependence. Paradoxically, the other commonly used target group for testing anti-smoking procedures, young psychology students, usually receive behavioral methods designed to overcome dependence, yet their principal problem is low motivation rather than high dependence.

It is suggested that an attempt is made to assess a smoker's degree of dependence and motivation to stop and to then apply the appropriate techniques for that smoker's problem, as demonstrated in Figure 1. It should, however, be emphasized that this two-dimensional model has not yet been fully validated, nor have satisfactory scales been developed for the measurement of Dependence and Motivation to stop. Furthermore, the two dimensions are not orthogonal or independent as shown in the figure but tend to be positively correlated.

MOTIVATIONAL APPROACHES

Motivational techniques are Only appropriate for those who require motivation. This is not the place, nor am I the person, to outline

FIGURE 1

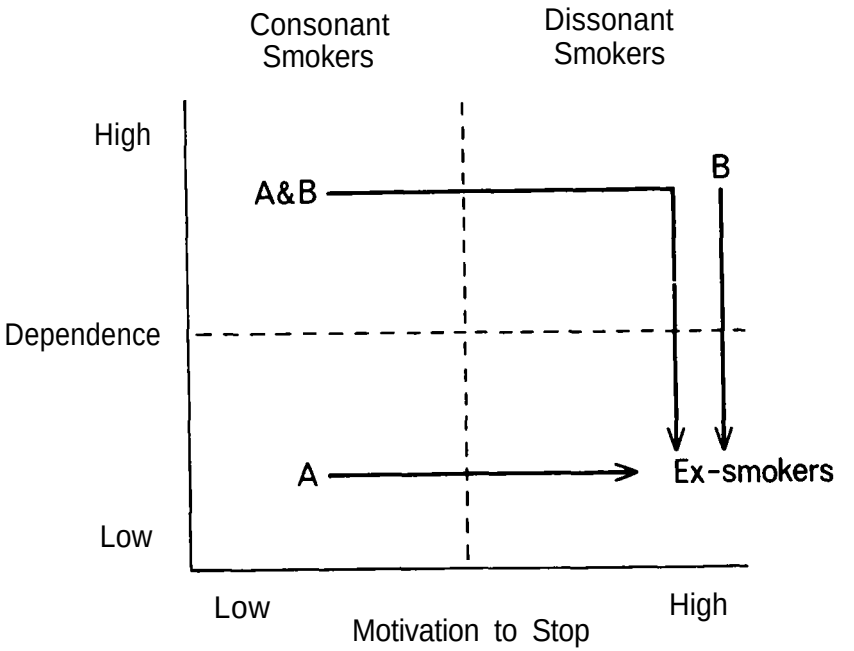


Figure 1. Two-dimensional model showing motivational (A) and treatment (B) approaches to smoking cessation. Consonant smokers are those who are quite happy about their smoking and have no wish to Stop. Dissonant smokers are the ones who would like to stop and may have tried but who continue smoking because they are dependent on it. The two term were first used to categorize Smokers by McKennell and Thomas (1967). About 75% of *cigarette smokers in Britain are dissonant smokers. According to the model, Smokers who are low, on dependence and also low on motivation to stop require only a motivational approach (A); once motivated they are able to stop without much difficulty and without requiring treatment. Highly dependent smokers who are also motivated require a treatment approach (B) to overcome their dependency. Highly dependent Smokers who are not motivated to stop require both approaches, A and B. Logically, anyone who falls well into the lower right corner of the* figure should *stop smoking.*

the complexities. and problems involved in developing more effective techniques of persuasion. But these approaches to the problem of smoking deserve high priority because of their potential to be effective on a large scale and even at a national level. They have this great potential for two reasons. First, they can be used not only on individuals and small groups, but also on a large scale via the mass media such as television or even by legislative action as in raising the price of cigarettes. Because of the large numbers of smokers involved a relatively small effect becomes worthwhile in terms of overall numbers. A second reason why motivational approaches are potentially so useful is that they can be applied to the mildly dependent, poorly motivated (consonant) smokers who have better chances of success though they would never go near a withdrawal clinic.

Figure 2 shows how smokers can be effected through their purses and their pockets. There is a strong inverse relation between changes in the price of cigarettes and their rate of consumption. When the price rises consumption falls and vice versa. For every 1% increase in price, consumption falls by about 0.6% and the negative correlation between the two is as high as 0.9. Price changes accounted for about 80% of the changes in cigarette consumption by men in Britain over the 25 years between 1946 and 1971 (Russell, 1973). So far, this powerful tool has been used to raise revenue rather than to promote health.

Another way to make contact with large numbers and all kinds of smokers is through their family doctors. Over 90% of the population in Britain visit their General Practitioners (Family Doctors) at least once over a five year period so that Britain's GP's are collectively able to make contact with some 18 million of the 20 million smokers in Britain. We are, at present, analyzing the effect of simple but firm advice to stop smoking given on a single occasion by GP's in their own individual style, to all patients who smoke cigarettes over one or two minutes' of a routine consultation. At one year follow-up 19% stopped smoking compared to 11% of controls who received no advice. The effect may not be large, but the intervention was minimal and could be applied to large numbers. If all the 20,000 plus GP's in Britain were to persuade even one patient a week to stop smoking, the yield would be more than one million ex-smokers a year. To equal this it would be necessary to set up 10,000 withdrawal clinics each having a 33% success rate with 300 subjects a year. The effect of GP's could be greatly enhanced if they could be persuaded to supplement the motivational technique of advice-giving with some form of cost-effective treatment approach such as support or nicotine chewing gum; similarly a motivational program on television would probably be much more successful if followed by some guidance on self-treatment.

To summarize so far, motivational approaches have changed the social climate but smokers' attempts to stop smoking have been blocked by the dependency factor. Traditional treatment approaches have failed partly through lack of an effective method with a clear-cut specific (as opposed to attention-placebo) effect, but mainly because they have been applied to the most difficult cases. It is suggested that

FIGURE 2

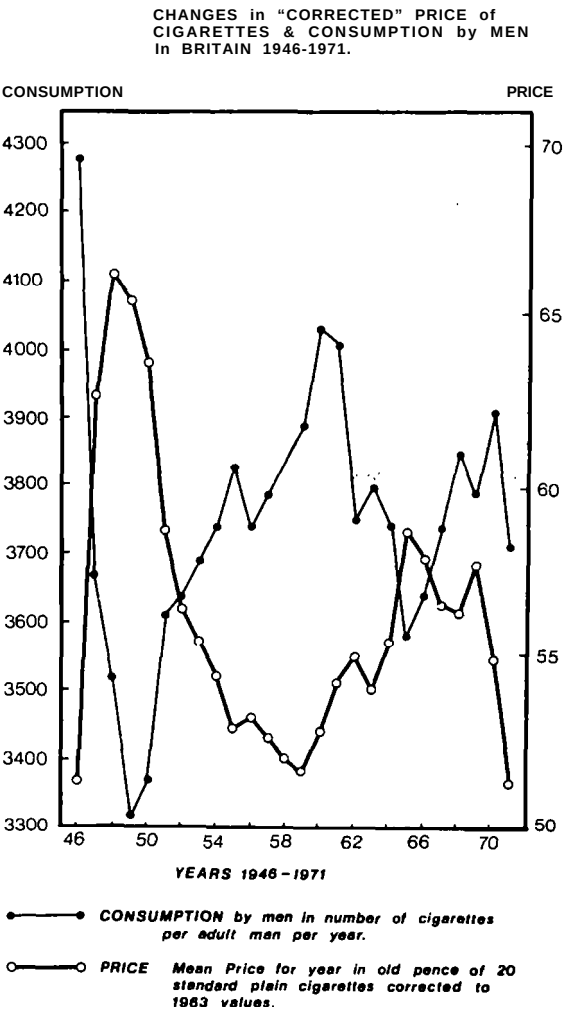


Figure 2. Changes in "corrected" price of cigarettes and consumption by men in Britain, 1946-1971: ●——● number of cigarettes smoked per adult man per year; o——o mean price for year in old pence of 20 standard plain cigarettes corrected to 1963 values (see reference 13). Reports by the Royal College of Physicians in 1962 and 1971 were surrounded by unusually concentrated anti-smoking publicity.

more smokers could be persuaded and helped to stop smoking if the motivational and treatment approaches were dovetailed by the use of mass media and other approaches to bring relatively simple but more cost-effective treatment procedures, as well as motivational techniques, to bear on large numbers and all kinds of smokers.

SAFER SMOKING

There are four ways to reduce smoking-related illness and premature death in a population: (i) reduce the number of people who smoke; (ii) identify individuals at high risk of developing illness from smoking so that they may stop or reduce their intake; (iii) reduce the dose of smoke taken in by those who do smoke; (iv) identify and reduce as far as possible the harmful components of tobacco smoke.

The first approach has so far clearly failed and will, in my view, continue to fail over the foreseeable future, certainly on any worthwhile scale. The satisfaction of smoking and the difficulty of stopping are simply too great, and for most smokers outweigh the health risks. It has been impossible to eradicate tobacco use in any free society throughout four centuries. Its use is, therefore, likely to continue at least until there is some other drug substitute for nicotine or some major socio-cultural or religious change outlaws self-gratification.

The second approach is also limited. Although there is some hope that it may one day be possible to identify smokers who are especially liable to get lung cancer, bronchitis and emphysema, there is no guarantee that such individuals would then give up smoking. It is already known that people with hypertension, diabetes or high blood lipids have an increased risk of complications if they smoke, yet such knowledge does not always deter them. In one study of smokers who had been hospitalized for myocardial infarction only 62% were persuaded to stop smoking despite the intensive efforts of the consultant cardiologist and his team which included involvement of the patients' families and regular home visits for as long as a year (Burt et al., 1974).

The third approach is quite complex and depends more on the inhalation pattern than crude consumption in terms of the number of cigarettes smoked. Cigarette smokers appear to have almost as much difficulty reducing consumption as they do in stopping altogether. To bring about a major reduction in the number of cigarettes smoked per smoker in a substantial proportion of the total smoking population would be a tremendous undertaking involving all the motivational approaches such as mass communications and price increases which have been outlined as measures to stop people smoking. Furthermore, it would not be very helpful if people smoked fewer cigarettes but then inhaled them more deeply and smoked them to a shorter butt length. The reduction in the numbers of cigarettes smoked per smoker does not, therefore, appear a very fruitful, goal for the foreseeable future. Pipe and cigar smoking have been shown on epidemiological evidence to be safer than inhaled cigarette smoking. This suggests that it might be possible to engineer a reduction of inhalation of cigarette smoke by raising the pH and the yield of relatively harmless irritants. However, it is doubtful whether a population hooked on puff-by-puff

inhaled nicotine boli would be sufficiently satisfied by the slower absorption of nicotine through the mouth and nose to refrain adequately from inhalation.

The fourth way to reduce smoking-related illness is to reduce the harmfulness of tobacco smoke and this applies particularly to the development of safer cigarettes.

SAFER CIGARETTES

The obvious approach to safer cigarettes would seem to be to identify and then reduce the harmful products in the mainstream smoke, but it is not quite so straightforward. Of the many harmful components of cigarette smoke, the tar is probably most lethal and is generally held to be responsible for cigarette-induced lung cancer and bronchitis (Royal College, 1977). A case is beginning to emerge for attributing to carbon monoxide (Co) the increased risk of coronary heart disease among cigarette smokers (Ibid.). The amount of damage caused by other toxic components is less clear, though a number would seem to warrant attention. These include hydrogen cyanide, phenols, aldehydes, acrolein, oxides of nitrogen and sulphur, ammonia, hydrogen sulphide, nitrosamines, and toxic metals. Few would argue that cigarettes would be less harmful if the yields of all these poisons were substantially reduced.

What about nicotine? There is no firm evidence that it is harmful in smoking doses, though it has not been cleared of contributing to cardiovascular pathology. Owing to this doubt it is certainly desirable that smokers take in as little nicotine as possible. Rut, as has been discussed above, there is some evidence that nicotine is the primary addictive component of tobacco. If this is so, it is not really feasible to lower the nicotine yields of cigarettes beyond the minimal requirements of smokers. In theory, as long as sufficient nicotine is present, reduction of all the other harmful constituents to very low levels would be tolerated by smokers. In practice, some adjustment may be necessary to changes in and loss of flavor contained in the tar. The key to safer cigarettes, therefore, lies with nicotine; and two crucial questions remain unanswered. These are a) how much it controls the smoking habit, and b) how harmful it is.

THE LOW-TAR, LOW-NICOTINE APPROACH

The tobacco industry has made considerable progress in reducing the harmful substances in cigarette smoke. This has been achieved in a variety of ways including use of selected strains of tobacco plant, changes in agricultural and curing procedures, use of reconstituted sheets, incorporation of tobacco stalks, reduction of the amount of tobacco needed to fill a cigarette by expanding it (like puffed wheat) to increase its “filling power”, and by the use of filters and high-porosity wrapping papers. By such means tar and nicotine yields of cigarettes have been substantially reduced over the past 10-15 years and more importantly the carcinogenicity of the tar per unit weight has also been decreased. Wynder, in his paper, presents some epidemiological evidence which suggests that the risk of lung cancer may be lower as a result of these changes, but this conclusion is,

at present, only tentative, as the smokers who changed their cigarettes were self-selected.

Somewhat in the wake of the tobacco industry, health authorities began to take an interest in lowering the tar and nicotine yields of cigarettes. At the Second World Conference on Smoking and Health (London, 1971) Wynder chaired a workshop on less harmful ways of smoking. In his summary recommendations to the Conference, he declared - "The manufacturer should be encouraged to produce cigarettes with increasingly lower tar and nicotine yields "(Richardson. 1972). In a contribution to that workshop Gori stated that "a cigarette can be called less hazardous if it delivers the least tar and nicotine per cigarette" (Gori, 1972). I can recall making a cautionary comment from the floor of that conference that excessive lowering of the nicotine yield might lead to a counterproductive compensatory increase in the degree of inhalation (Richardson, 1972, p. 52).

Experimental work since this time, some of it outlined by Jarvik in his paper, has shown that just as a drinker tends to drink a larger volume of beer than of wine or spirits, so many smokers tend to modify their smoking pattern inversely according to the strength of the cigarette being smoked. In contrast to the standardized puffing of the smoking machines on which the tar and nicotine yields are based, when the smoker switches to a low-tar, low nicotine cigarette, he smokes more cigarettes, takes more puffs and inhales more deeply. Conversely, when smoking a high-tar, high-nicotine cigarette there is a tendency to smoker and inhale less. We cannot, therefore, be sure that the reduction in tar and nicotine yields of cigarettes from sales-weighted averages of around 30mg and 2mg respectively in the 1960's to current average levels of about 18mg and 1.2mg have been matched by a proportionate reduction of intake into smokers' lungs. Indeed, average consumption per smoker has *increased* over this period and this is but one rather crude index of smoke intake. It is possible that the reduced hazards suggested by epidemiological studies are due to the reduced carcinogenicity of cigarette smoke rather than the lowering of tar and nicotine yields.

Adjustment to the reduction in tar and nicotine yields of cigarettes to present-day levels has been relatively easy for smokers. Little persuasion was needed for smokers to switch from plain to filter-tipped cigarettes. Over the past two or three years, however, the rate of decline seems to have slowed, despite intensification of the campaign to encourage smokers to switch to low-tar cigarettes. In Britain, most of the reduction of tar yields was already achieved by April 1973 when the official tar and nicotine tables were first published (Figure 3). It happened before the public was really aware of any differences between brands. We now seem to have encountered a kind of "acceptability barrier" at yields of around 10-14mg of tar and 0.8 - l.0mg of nicotine, and it looks as though it is going to be a difficult task to persuade a majority of smokers to get very much below these levels. Tobacco manufacturers can and indeed do produce cigarettes with tar and nicotine yields which are so low as to be negligible. The trouble is that hardly anyone smokes them, and those who do are probably non-inhalers anyway so that it matters little what they smoke.

FIGURE 3

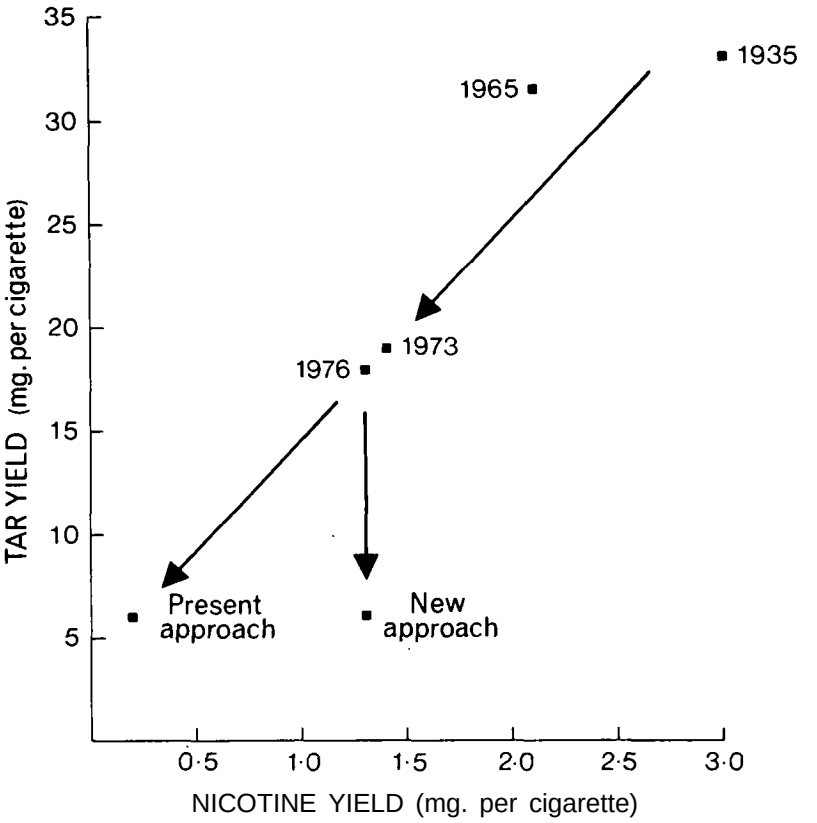


Figure 3. Changes in average tar and nicotine yields of British cigarettes, 1935-76, and projected changes for reducing tar intake to 33% of 1976 level by following the present low-tar, low-nicotine approach, or by adopting a new low-tar, medium-nicotine approach. Relatively little change has occurred since 1973 when Government tar and nicotine tables were first' published. (see reference 19).

I believe that the reason why it has been relatively easy to bring smokers down to: this barrier is' because' it ha's' not really required them to reduce their tar and nicotine intake. They have simply adjusted by taking larger puffs, one or two more puffs, a few more cigarettes and inhaling more deeply. All this has been relatively painless, and largely unconscious. But there are limits to the number of puffs a cigarette will give and it becomes uncomfortable and awkward to increase the puff volume above about 60ml.. Thus, the "acceptability barrier" may be set by the capacity of smokers to adjust their smoke intake in these ways. After this a different form of adjustment is demanded. This involves the smoker adjusting to a lower dose of smoke, and this is a far more difficult task. It brings the smokers once again into confrontation with the Dependency Factor which, as discussed above, has blocked substantial progress after two decades of campaigning to get people to stop smoking.

To simply pursue the low-tar, low-nicotine approach to safer smoking by ever-increasing exhortation will, in my view, prove as frustrating as the campaign to stop people smoking. At the Third World Conference on Smoking and Health, in New York in 1975, an enthusiastic workshop chaired by Wynder was devoted to less hazardous cigarettes. Low-tar, low-nicotine cigarettes were strongly advocated. Possibly because no social scientists were invited to this workshop, in the 553 page report (Wynder et al., 1976)) "consumer acceptability" is paid little more than lip-service in the form of one or two passing allusions. Certainly no systematic attention was given as to how the Dependency Factor is to be overcome or by-passed to enable use of safer cigarettes to become a reality.

THE LOW-TAR, MEDIUM-NICOTINE, APPROACH

Tar and nicotine yields of present-day commercial cigarettes correlate highly, 0.9 or more. We cannot, therefore, be sure that the changes in smoking pattern discussed above are induced by a need to regulate nicotine intake rather than tar intake. Though it is by no means proven, it is probable that nicotine is the primary addictive agent. If this is so, to expect people who cannot stop smoking to smoke cigarettes with hardly any nicotine is illogical. Owing to the high correlation of tar and nicotine yields in present-day cigarettes, a smoker cannot switch to a cigarette with a very low tar yield without having to put up with a very low nicotine yield, and this he simply will not do. I have suggested (Russell et al., 1973b; Russell, 1976a) that we should aim at lowering the yields of tar, CO, and all the other harmful components of cigarette smoke, but that the nicotine yield should be kept medium or even high. This approach requires that emphasis be placed on the ratio of tar to nicotine yields as well as on the absolute yields.

The difference between the present low-tar, low-nicotine approach and the new low-tar, medium-nicotine approach which I am suggesting is shown in Figure 3. The figure illustrates the situation in Britain and shows how the two approaches would operate to achieve a reduction of the national sales-weighted average tar yield of cigarettes to a hypothetical target of one third of the present level. To achieve this by following the' traditional low-tar, low-nicotine approach

with its highly correlated tar and nicotine yields would require a concurrent reduction of average nicotine yields to around 0.4mg. In my opinion, this is so far below the present acceptability barrier of most smokers that it would involve a long drawn-out, painful and frustrating campaign covering at least ten to twenty years. On the other hand, with the new low-tar, medium-nicotine approach the preservation of adequate nicotine levels would enable smokers to make a rapid and relatively painless adjustment which could no doubt be facilitated by the incorporation of tar-free flavors and other additives . The technology is available to do this and by these means the target might be achieved within five to ten years.

It has surprised me to find how resistant other workers in the Smoking and Health field can be to this concept. A frequent immediate response is to say that nicotine may not be completely safe as though this negates the whole thesis. Apart from those in the tobacco industry, very few have shown immediate recognition of the potential import of this approach. These few include G. F. Todd, former Director of the Tobacco Research Council in London, Edward Brecher, a lay writer (1976), Stanley Schachter (1977 in press), and Gio Tori, whose recent prescription for a safer cigarette is highly pertinent (1976). My own reservations about this approach are (i) that it depends above all *on* the importance of nicotine as the main determinant of dependent smoking and this is still uncertain; (ii) that we do not know whether or not nicotine is harmful in smoking doses taken with minimal amounts of Co; (iii) that the safer it becomes to smoke, the less incentive there will be for smokers to stop, so that a successful safer cigarette might have a counterproductive effect on the prevalence of smoking. The first two problems can be elucidated by research but the third could be more difficult.

Because so many people seem to find it difficult to accept the low-tar medium-nicotine approach, it might be helpful to illustrate it with the following analogy. Supposing that some common disease like arthritis were found to be strongly associated with alcohol intake, and that this were due to the presence in alcoholic drinks of excessive quantities of some trace metal like nickel. Would the solution be to advocate another "Prohibition"? Certainly not. We would seek, surely, to remove the offending metal from alcoholic drinks. There would be no problem about implementing this because people drink for the effect of alcohol, not for the metal; so that the disease caused by the metal could be eliminated, or at least greatly reduced, almost at a stroke. Would it be reasonable for us to hold back simply because preventing the disease by removing the metal would not at the same time reduce the diseases and social ill consequences caused by the alcohol itself? Again, certainly not. To extend the analogy further, supposing in this hypothetical situation, a high correlation were found between the alcohol content and metal content of drinks. Would we waste time by exhorting people to drink beer rather than spirits or, even more ludicrously, expect them to switch to, quarter strength shandy (a mixture of beer and lemonade usually mixed SO/SD) and to take it in small amounts, by small sips, from small glasses, as if they were still drinking spirits? This might seem ridiculous, yet it is precisely what we have been doing about smoking. The current approaches to the

smoking problem are either to take the path of no-smoking or prohibition, or, on the other hand, to adopt, the low-tar, low-nicotine approach which is analogous to the quarter-strength shandy approach.

TABLE I

Approaches to Reduce National Tar Intake by Two-Thirds

1) Reduce the prevalence of smoking by 66%

2) Reduce the consumption of smokers by 66%

3) Reduce Tar/Nicotine yields by 66% from the present mean of 18/1.2 to 6/0.4
(yields are in mg per cigarette)

4) Reduce the Tar/Nicotine ratio by 66% from the present mean of 15. to 5. This could be achieved by the following combinations of Tar/Nicotine yields: 6/1.2, 18/3.6, 12/2.4, 9/1.8, 4/0.8.

The four possible approaches to reduce smoking-related disease are shown in the table above, using as an illustration the target of securing a two-thirds reduction in national tar intake. The same principles would apply to other harmful components, except for nicotine which would not be amenable to the fourth approach. I believe that the fourth approach is the most feasible and, furthermore, in carrying it out would not be necessary to discontinue the first two approaches.

SUMMARY AND CONCLUSIONS

1) The problem of stopping people smoking is presented in the context of two main dimensions - their degree of motivation to stop, and the strength of their dependency on nicotine.

2) Anti-smoking campaigns using motivational approaches have succeeded in changing the social climate to a general belief that people should not smoke. However, smokers attempts to stop have been blocked by the dependency factor.

3) No cost-effective way has yet been found for helping smokers to overcome their dependence. No treatment method has been shown to have a specific effect on reducing smoking in the long-ten, and all the positive effects of treatment are due to its attention-placebo element.

4) One reason why treatment fails is that it is usually applied to selected groups which include either the most highly dependent (withdrawal clinic) or poorly motivated (social science students) smokers.

5) It is suggested that motivational and the more cost-effective treatment procedures should be synchronized and applied,-via mass-media and other approaches such as family physicians, to large populations of “normal” smokers. The emphasis should be on seeking a

modest success rate among large samples using cost-effective methods,-rather than high success rates from elaborate methods in small samples.

6) Since it is almost inevitable that tobacco use will continue at a substantial level whatever is done to stop or reduce it, research into safer forms of smoking should receive the highest priority.

7) People smoke for nicotine but die from tar., CO and other toxins. The most logical approach to safer cigarettes is to seek to identify and then reduce the yields of all harmful components, but to maintain an adequate nicotine yield to be acceptable to the smoker and to keep down his smoke intake.

REFERENCES

Allen, W. A. and Fackler, W. A. An exploratory survey and smoking control program conducted among parents of Philadelphia school children. In: Zagona, S. V., ed. *Studies and Issues in Smoking* Behavior, Tucson: University of Arizona Press, pp. 63-67, 1967.

Brecher, E. M. Less tar, less nicotine: is that good? *Consumer Reports,* pp. 274-279, May, 1976.

Burt, A., Illingworth, D., Shaw, T. R. D., Thomley, P., White, P., and Turner, R. Stopping smoking after myocardial infarction. *Lancet,* I: 304-306, 1974.

Gori, G. B. Research in smoking and health at the National Cancer Institute. *J Natl Cancer Institute, 48:* 1759-1762, 1972.

Gori, G. B. Low-risk cigarettes: a prescription. *Science,* 194: 1243-1246, 1976.

Hunt, W. A., Barnett, L. W., and Branch, L. G. Relapse rates in addiction programs. *J Clin Psycho1* 27: 455-456, 1971.

McKennell, A. C. and Thomas, R. K. Adults' and adolescents' smoking habits and attitudes. *HMSO,* London, 1967.

Richardson, R. G., ed. *Proceedings of the Second World Conference on Smoking and Health.* London: Pitman Medical, 1972.

Royal College of Physicians, Smoking or Health. London: Pitman Medical, 1977.

Russell, M. A. H. Changes in cigarette price and consumption by men in Britain, 1946-71: A Preliminary Analysis. *Brit J Prev Soc Med,* 27: 1-7, 1973.'

Russell, M. A. H., Wilson, C., Patel, U; A., Cole, P. V., and Feyerabend, C. Comparison of the effect of 'tobacco consumption and carbon monoxide absorption of changing to high and low nicotine cigarettes. *Brit Med J,* 4: 512-516, 1973b.

REFERENCES (Cont)

Russell, M. A. H., Peto, J., and Patel, U. A. The Classification of Smoking by Factorial Structure of Motives. *J Roy Statist Soc A,* 137: 313-333, 1974.

Russell, M. A. H. Low-tar, medium-nicotine cigarettes: A new approach to safer smoking. *Brit Med J,* 1: 1430-1433, 1976a. (Russell, 1976a).

Russell, M. A. H., Tobacco smoking and nicotine dependence. In: Gibbons, R. J., et al., eds. *Research Advances in Alcohol and Drug Problems,* Volume 3. New York: Wiley and Sons, pp. 1-47, 1976b.

Russell, M. A. H. What is dependence? In: Edwards, G., Russell, M. A. H., Hawks, D., and MacCafferty, M., eds., *Drugs and Drug Dependence.* Farnborough, England: Saxon House, pp. 182-187, 1976c.

Russell, M. A. H., Armstrong, E., and Patel, U. A. Temporal contiguity in electric aversion therapy for cigarette smoking. *Behav Res & Ther,* 14: 103-123, 1976.

Russell, M. A. H. Smokers Clinic. In: *The Bethlem Royal Hospital and the Maudsley Hospital Report 1970-1975, Part 1,* 1977, pp. 68-70. Cawley, R. and Myers, M., eds., 1977.

Russell, M. A. H. Self-regulation of nicotine intake by smokers. *Proceedings of International Workshop on Behavioural Effects of Nicotine,* Zurich: 1976. In press, 1977.

Russell, M. A. H. How important is nicotine in tobacco smoking? *Proceedings of Conference on Commonalities in Substance Abuse and Habitual Behaviour,* Warrenton, Virginia, March 1977. In press, 1978.

Schachter, S. Pharmacological and psychological determinants of smoking. *Annals* of *Internal Medicine.* In press, 1977.

Wynder, E. L. and Graham, E. A. Tobacco smoking as a possible etiologic factor in bronchiogenic carcinoma: A study of 684 proved cases. *J Amer Med Assoc,* 143: 329-336, 1950.

Wynder, E. L:, Hoffman, D., and Gori, G. B., eds. Modifying the risk for the smoker. *Proceedings of the Third World Conference on Smoking and Health,* Volume 1, U.S. Dept. of Health Education and Welfare, 1976.

AUTHOR

M. A. H. Russell, BM, MRCP, MRCPsych.
Institute of Psychiatry
Addiction Research Unit
The Maudsley Hospital
101 Denmark Hill
London S.E.5. 8AF
England

SECTION I: EPIDEMIOLOGY

Patterns of Smoking Behavior

Leonard M. Schuman, M.D.

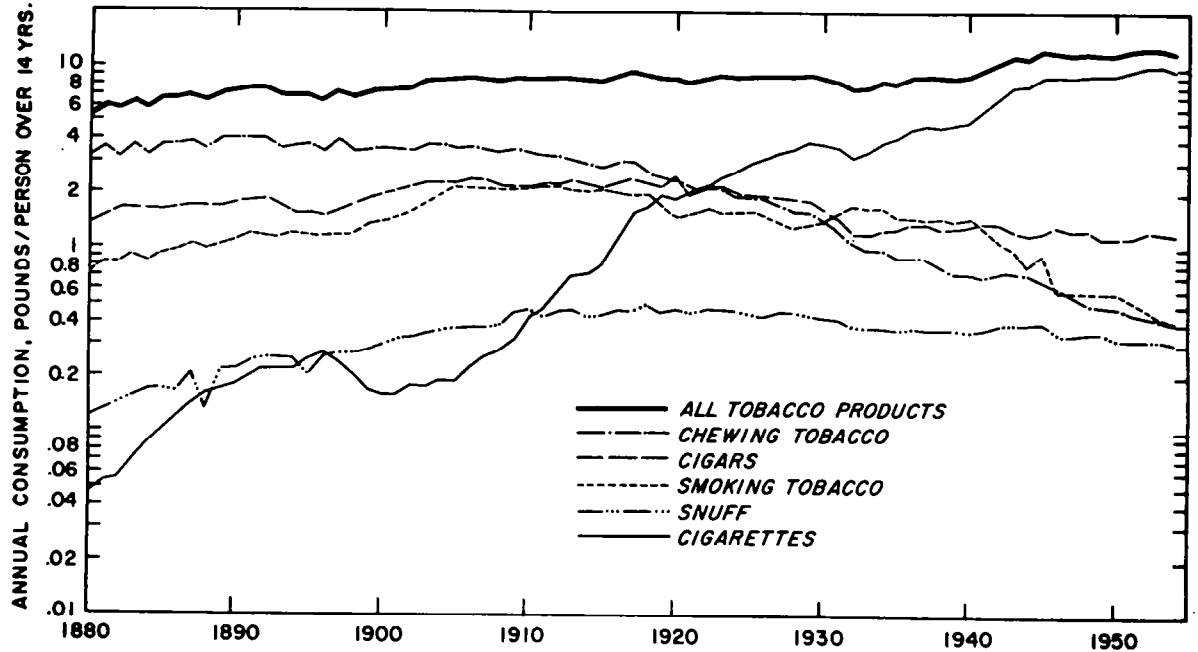
INTRODUCTION-HISTORICAL

In the long anthropologic history of man the wholesale adoption by Western European culture of the habit patterns in the use of tobacco is relatively quite recent. As is well known, the flowering of the Renaissance and its stimulus on the powerful urge for exploration and discovery led, within about 100 years, to the opening of the New World, the discovery of the use of Nicotiana tabacum by the American Indian and its introduction into Europe by 1558. Thus the habit pattern which European man adopted is but 400 years old. Less than 60 years later tobacco had became, in turn, a staple agricultural community in Virginia and its principal currency. With the burgeoning of the migrant population to the Americas tobacco culture expanded rapidly both societally and agronomically.

Relatively reliable historical data on tobacco products in the United States are available through the records of the U.S. Department of Agriculture which, along with the records of production, compiled by the Internal Revenue Service for tax purposes, provide estimates of per capita consumption of such products. These data are available in this form only from 1880 but will suffice for our purposes. Figure 1 presents the trend in annual tobacco products consumption in pounds of tobacco (unstemmed-processing weight) per person over 14 years of age (Milmore and, Conover, 1956). The relative stability of consumption of all tobacco products combined as compared to some of the individual products is quite notable, even though the overall consumption was actually 2.2 tin-es as great in 1954 as in 1880. The individual form of use of tobacco have varied considerably over time, however.

It will be seen that, whereas prior to the end of World War I chewing of tobacco was the principal modality of use, cigarettes, and particularly pre-fabricated cigarettes, became the principal form of tobacco use from the decade of the 1920's onward. Thus the wide scale adoption of cigarettes smoking is an even more recently acquired habit.

Figure 1. Tobacco products, unstemmed-processing weight: Consumption per person over 14 years of age, United States, 1880-1954



Source: P.H. Monograph 45, pg. 109

The consumption curve for cigarettes reveals several interesting slope trends. The precipitous rise in consumption from 1910 to 1930 was followed by a virtual slowing of the annual increases until World War II when a precipitous increase ensued once more. There is good evidence from increases in use of "smoking tobacco" in that period that pipes were not the only modality of tobacco consumption in that category but that "roll-your-own-cigarettes" accounted for 46% of this category, particularly during the Great Depression of 1933-40 (Milmore, 1956). Economic necessity was the "mother of invention" but had little if any impact on the overall tobacco consumption trend (see Figure 1).

The second precipitous increase from 1940 to 1945 was stimulated by World War II with large scale consumption by troops overseas and by the broadening of the consuming base as more and more women adopted the habit in this period. Subsequent surveys, to be described later, have substantiated this increase in this facet Of risk-taking behavior among females at that time.

Cigar and snuff consumption have shown far more stable rates although both categories of tobacco use show gradually achieved peaks and plateaus followed by declines with the plateau of use of cigars occurring between 1905 and 1920 and returning to approximately the 1880 rate in 1954. Consumption of snuff increased to a plateau between 1910 and 1930 but with only a slight decline since then. However, snuff consumption at its peak never exceeded 5% of the total tobacco use by weight.

The category of "smoking tobacco" which, for the most part, had represented pipe smoking with increasing proportions for "roll-your-own" cigarettes to a peak for the latter during the Depression, experienced a precipitous drop with the onset of World War II so that by 1954 total consumption in this category was but" 20% of its peak in 1911 and but 60% of its level in 1880.

In an overview of the latter half of the period 1880-1954, i.e., from the United States' entry into World War I in 1917, it may justifiably be stated that the 28% increase in the total tobacco consumption in this time span is primarily the result of the almost 6-fold[1] increase in cigarette consumption offset by a 70% decline in other forms of tobacco use. Expressed in another way, of the more than 8 pound increase in cigarette consumption per person in this period, 41% of the increase may be considered as additional tobacco consumption and the remaining 59% as a shift from other forms of tobacco (Milnore & Conover, 1956). Thus this period saw a tremendous shift to cigarette use from other forms of tobacco.

[1]Calculated from data provided in the Milnore and Conover paper of 1956.

RECENT TRENDS

I have deliberately divided this discussion of trends in tobacco use patterns into two periods of time, that prior to 1954 and the period from 1955 to the present, for two reasons. First, until the Milmore and Conover study of 1955 there were no critical analyses of existing consumption data, however inadequate such data might be for systematic epidemiologic inquiries. Secondly, there is a need to study the immediate and long term impact on patterns of tobacco use of information which began-to reach- the consuming public in the early 1950's and again in the early 1960's. I am. of course. alluding-to the unfolding of the epidemiologic studies on the relationship of tobacco use, particularly cigarette smoking, to health and disease and to the Advisory Committee's Report to the Surgeon General in January of 1964 (U.S. DHEW, 1964).

In Table 1 there are presented the tobacco consumption data for the period 1900 to 1960 in 10 year periods and for 1961 to 1975 in annual periods (USDA! 1976). It is- to be noted that the base denominators are decidedly different for the period 1962-75 and an improvement over the data prior to that when all the population over a given age was utilized as the base, irrespective of whether the particular use was practiced by one or both sexes. Furthermore, caution should be exercised in comparing the data in the periods before and after 1962 because of the discontinuity which exists not only in the sex of the respective population bases but also the elevation of the minimum age from 15 to 18 years. Whereas the former change would tend to reflect the real situation with respect to the specific tobacco use population by sex, the age change in the base would tend, on the other hand, to attribute to older persons in the population greater use, at least of cigarettes, than is actually the case, for the size of the smoking teenage population under 18 years has increased markedly as will be seen later. Their omission not only explains the seemingly marked increase between years 1961 and 1962 in cigarette consumption but also obscures the true reductions which have actually occurred in the adult population.

Momentarily accepting the relative value of these data, it will be noted that declines in the consumption of cigars has continued steadily as has the use of pipe tobacco and snuff. A deviation from this trend is noted for one year, 1964, the year of the Report to the Surgeon General, when along with an abrupt decline in cigarette consumption there was noted an abrupt rise in consumption of cigars and pipe tobacco. Following this the declines resumed. The picture is not quite as salutary for cigarette consumption Not overlooking the reservations in the data alluded to immediately above and the discontinuities imposed, the period of the reports of the initial studies on the relationship of smoking to health seems superficially at least to have been followed by a plateauing of the increase in cigarette consumption and in 1964, the year of the Report of the Advisory Committee, by a distinct drop in consumption. Since then some rises and declines in consumption seem to have

Table 1. Consumption of tobacco products in the United States
1900-1975

Year	Per capita consumption, all persons aged 15 years and over				
	Cigarettes No.	Cigars No.	Pipe Tobacco lbs.	Chewing tobacco lbs.	Snuff lbs.
1900	49	111	1.63	4.10	0.32
1910	138	113	2.58	3.99	0.50
1920	611	117	1.96	3.06	0.50
1930	1365	72	1.87	1.90	0.46
1940	1828	56	2.05	1.00	0.38
1950	3322	50	0.94	0.78	0.36
1960	3888	57	0.59	0.51	0.29
1961	3986	56	0.59	0.51	0.27
1962	3958	55	0.56	0.50	0.26

Table 1. (Continued)

Year	Per capita consumption, persons aged 18 and over as designated				
	Cigarettes (M+F) No.	Large Cigars and Cigarillos (M only) No.	Pipe tobacco (M only) No.	Chewing tobacco (M only) lbs.	Snuff (M+F) lbs.
1962	4265	121.9	1.24	1.10	0.28
1963	4345	124.6	1.22	1.11	0.27
1964	4195	154.4	1.42	1.11	0.26
1965	4259	143.9	1.19	1.07	0.24
1966	4287	136.1	1.13	1.05	0.23
1967	4280	130.7	1.08	1.04	0.23
1968	4186	126.5	1.11	1.05	0.21
1969	3993	125.0	1.08	1.09	0.20
1970	3985	125.3	1.15	1.06	0.19
1971	4037	119.2	1.06	1.09	0.19
1972	4043	108.9	1.00	1.08	0.18
1973	4148	102.4	0.88	1.10	0.18
1974	4141	91.9	0.87	1.13	0.18
1975	4121	82.4	0.76	1.15	0.17
1976 (Est)	4110	74.1	0.75	1.17	0.17

Source: U.S. Agricultural Marketing Service: The Tobacco Situation, 1977

occurred. In general they point to possibly dramatic changes that may be masked by missing date, namely the trends of use by males and females. This data cannot be derived from total population per capita consumption and without knowledge of the proportions of smokers among subsets of the population, including males vs. females at respective ages.

TOBACCO SURVEYS

Until 1955 there had been no reliable surveys of tobacco use among consumers published in the literature. The few to which allusions were made in some quarters were commercially derived, kept as trade secrets and were not published. Although case-control studies in respectable numbers had already been completed and published by this time and several promising prospective studies had been initiated, from the former there could not be derived proportions of the general population who smoked and the latter were usually special populations selected in one way or another and except for one, the now famous veterans study by Dorn (Kahn, 1966), were not necessarily representative of the U.S. population, albeit of males. With the emergence of the epidemiologic studies on the relationship between smoking and several diseases, it became imperative to ascertain not only the proportions of smokers in the general population but in the various subsets of that population by age, sex, occupation, residence, race, type of smoking and degree of smoking by several measures, in order that the actual observed morbidity and mortality of a selected associated disease could be shown to be consistent with the estimates of the excess risks among smokers and the population of smokers affected. It was for such a reason among several others that Haenszel et al. (1956) mounted the first study of tobacco use patterns in the general population of the United States utilizing the Bureau of the Census' Current Population Survey approach in February of 1955.

Subsequent surveys of marked import were conducted by the National Clearinghouse for Smoking and Health set up shortly after the Advisory Committee's Report. The survey in 1964, less than one year after the Report to the Surgeon General's Advisory Committee, one in 1966, and others in 1970 and 1975, were conducted for the Clearinghouse by private opinion corporations, while an additional survey in June 1966 was conducted by the Bureau of the Census for the Division of Health Interview Statistics of the National Center for Health Statistics (Ahmed and Gleeson, 1970), once more utilizing the Current Population Survey approach and questions on smoking habits phrased similarly to those used in 1955. Figure 2 presents the data for the two Current Population Surveys whereas Figures 3 and 4 present the findings of the opinion surveys which, in addition to smoking behavior questions, also probed for attitudes and reactions to the smoking and health problem.

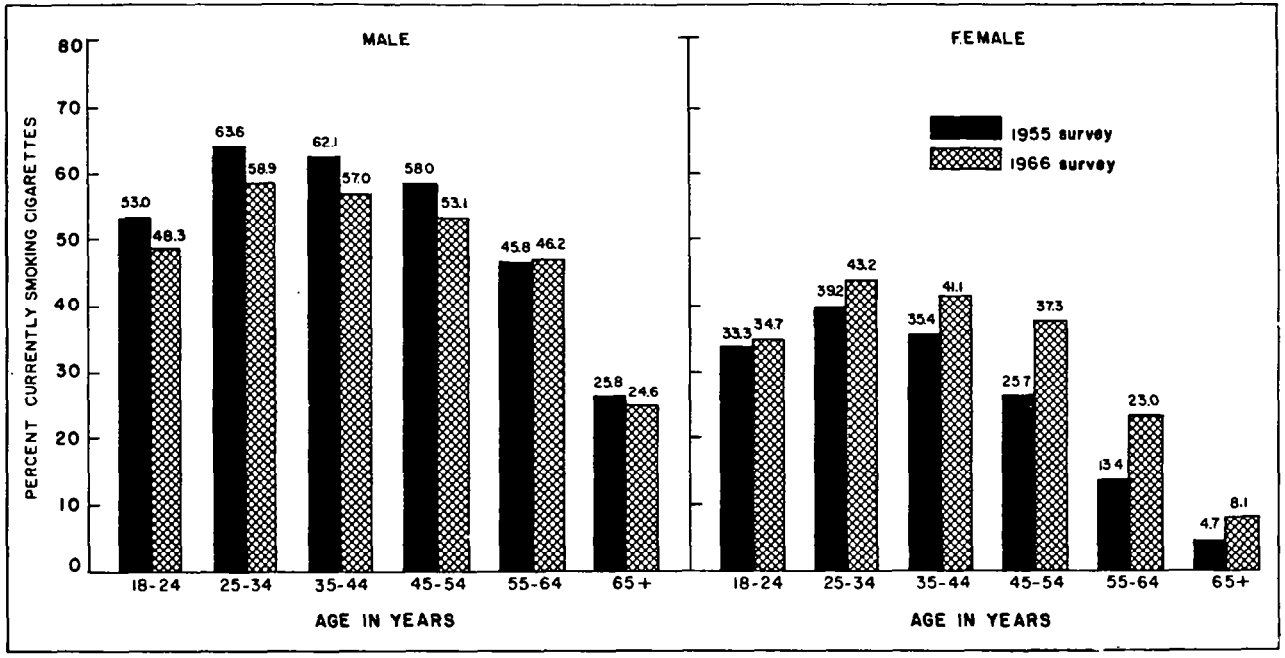
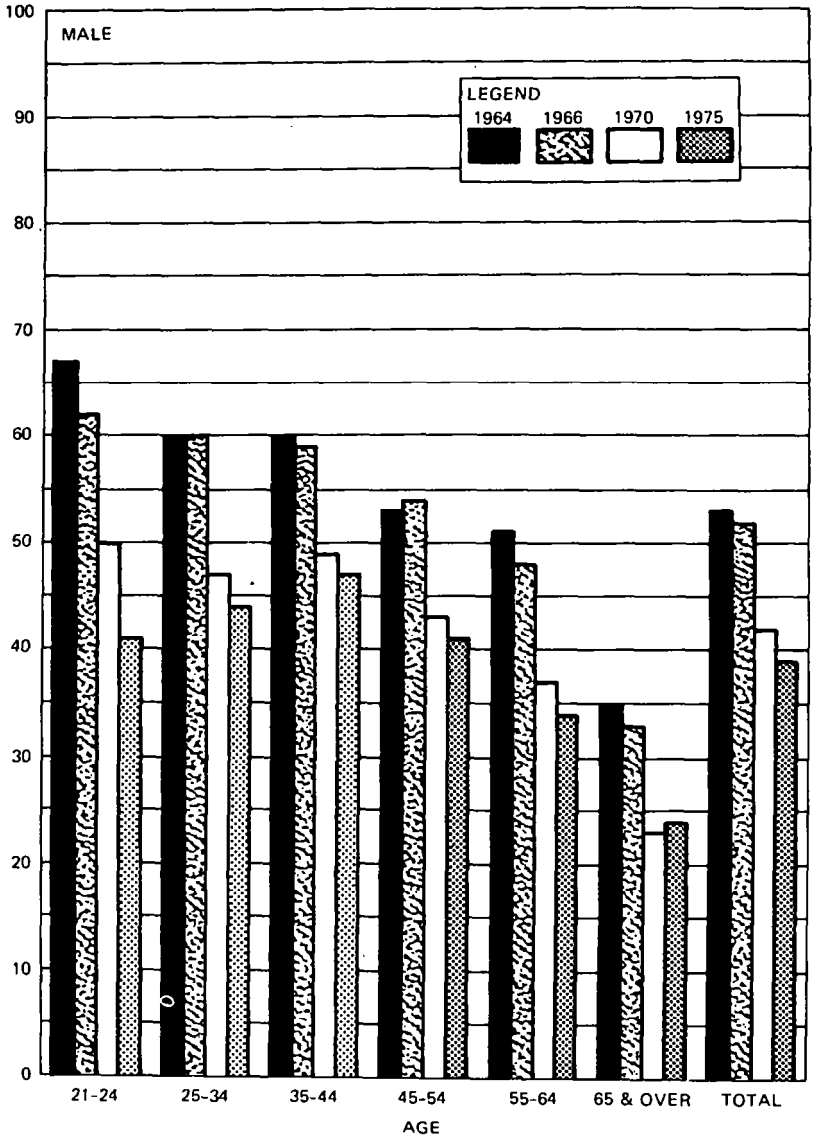


Figure 2. Percent of population currently smoking cigarettes regularly or occasionally, by age at time Of survey and sex.

Source: PHS public No. 1000-Series 10, No. 59, pg. 7

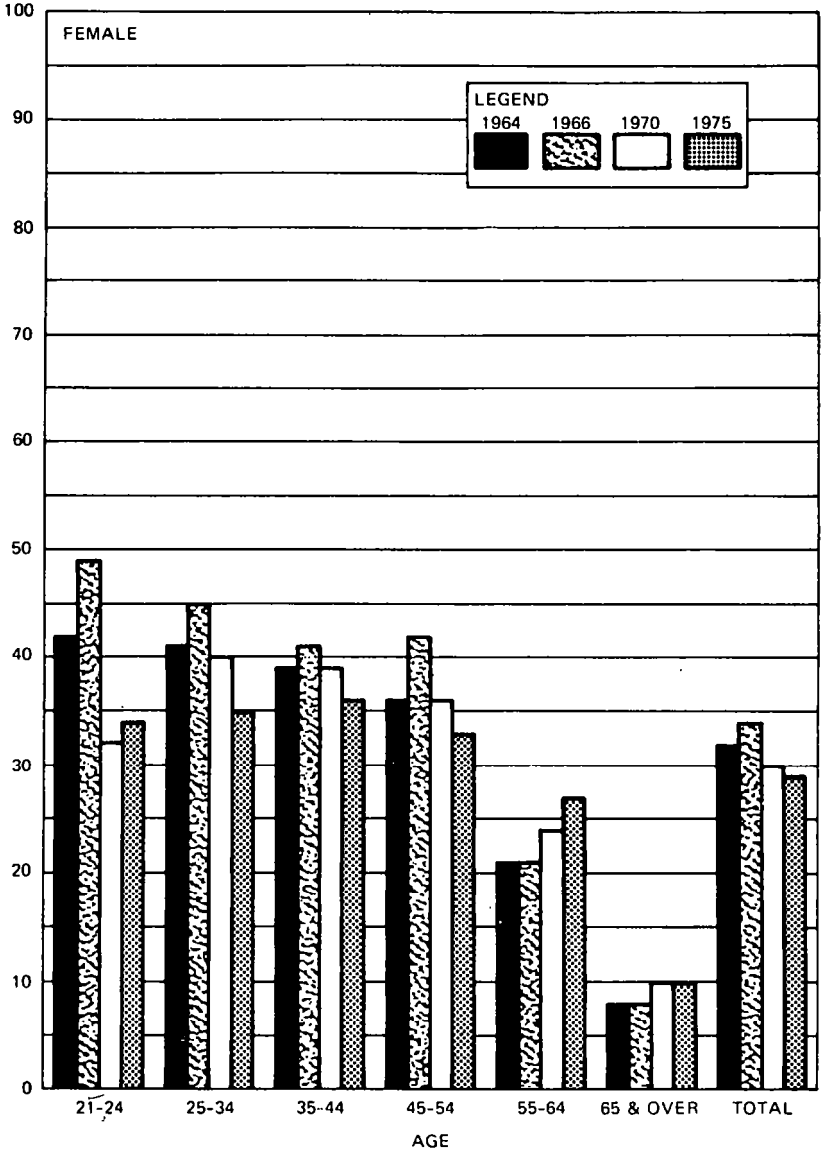
Figure 3

PROPORTION OF SMOKERS IN ADULT POPULATION
1964-1975



Source: NCSH, CDC, DHEW

Figure 4
PROPORTION OF SMOKERS IN ADULT POPULATION
1964-1975 - Continued



Source: NCSH, CDC, DHEW

For the most part the two sources of survey data for the years 1955 and 1966 and for 1964 and 1966 respectively tend to corroborate the downward trend in cigarette smoking, among males in all age groups except the 55-64 year group where a slight increase occurred and the initially upward trend for females for all of the age groups. In the survey years 1970 and 1975 percentages of male smokers among the several age groups continued their decline with but one exception in the age group 65 and over where an increase is noted for 1975. This may be an extension of the opposite trend noted in this age cohort 10 years before. Females surveyed in 1970 and 1975 showed a reversal of the initial increases of the 1966 survey in all the age groups except for those 55 and older and in the 21-24 year age group where an increase in 1975 was observed, although the percentage was still smaller than those observed in corresponding age groups in 1964 and 1966. Of further importance is the observation that reductions in smoking among adult females have been proportionately far less than reductions in the males. There is sane evidence that the female and even the female health professional believes herself to be at far less risk from the hazards of smoking than she actually is (Schuman, 1972). The female is thus, obviously, an important target for smoking behavior change.

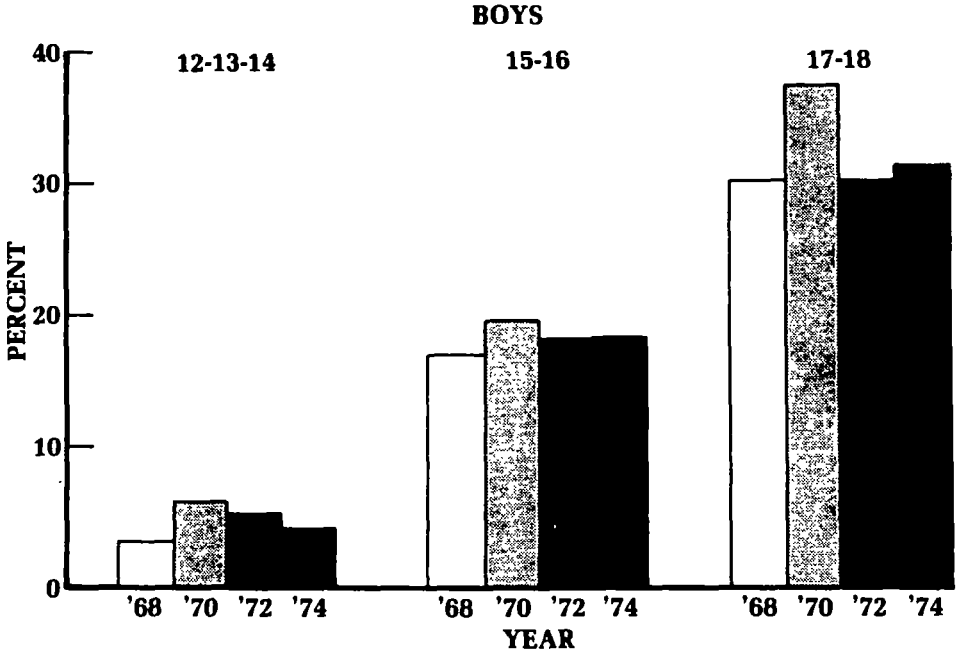
Thus far most of our discussion has dealt with adult populations. Teen-age smoking has begun to present serious problems particularly from the standpoint of primary prevention of initiation of the smoking habit. Four national sample surveys have been conducted in the United States among persons aged 12 through 18 years in 1968, 1970, 1972, and 1974. (DHEW, 1972 and DHEW, 1976) The findings are best presented graphically in Figures 5 and 6. An increase in smoking in the 6-year period was noted for the teen-age group as a whole. However, except for increases in 1970 with subsequent reductions among boys, the group increases have been contributed to virtually entirely by the steady increases among girls for every age group. By 1974 virtually no difference existed between the proportions of strokers among boys and girls.

AGE AT INITIATION OF SMOKING

Relevant to the problem of teen-age stroking and the continuance of the habit into and through adult life is the trend in the age at which smoking is started. Data are available from the Current Population Surveys of 1955 and 1966. The proportion of male self-respondents in the 1966 survey was disappointly low. Furthermore, persons in the military service were excluded from both surveys. Since a high proportion of males 18-24 years of age were in the Armed Forces in 1966 in contrast to 1955, this too was deemed to be a shortcoming, so that no analyses of trend in age at initiation of smoking was executed for males.

However, the female patterns among the several age groups in the surveys are presented in Figure 7. Between 1955 and 1966 a decided shift to younger age groups was noted in the smoking initiation

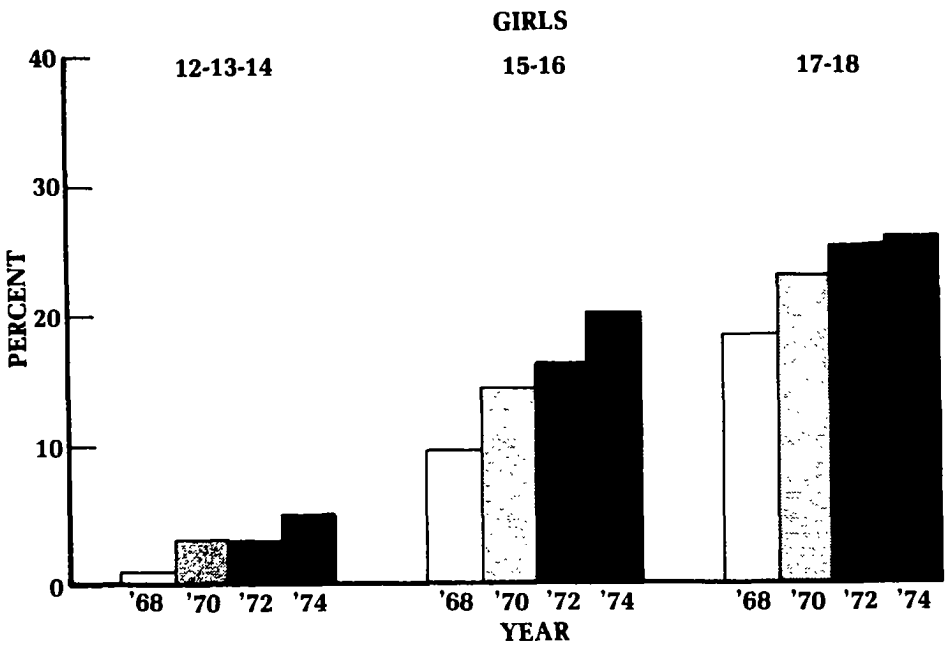
PERCENT
CURRENT REGULAR SMOKERS-TEENAGE,
1968-1974



Source: DHEW public No. (NIH) 76-931

Figure 5

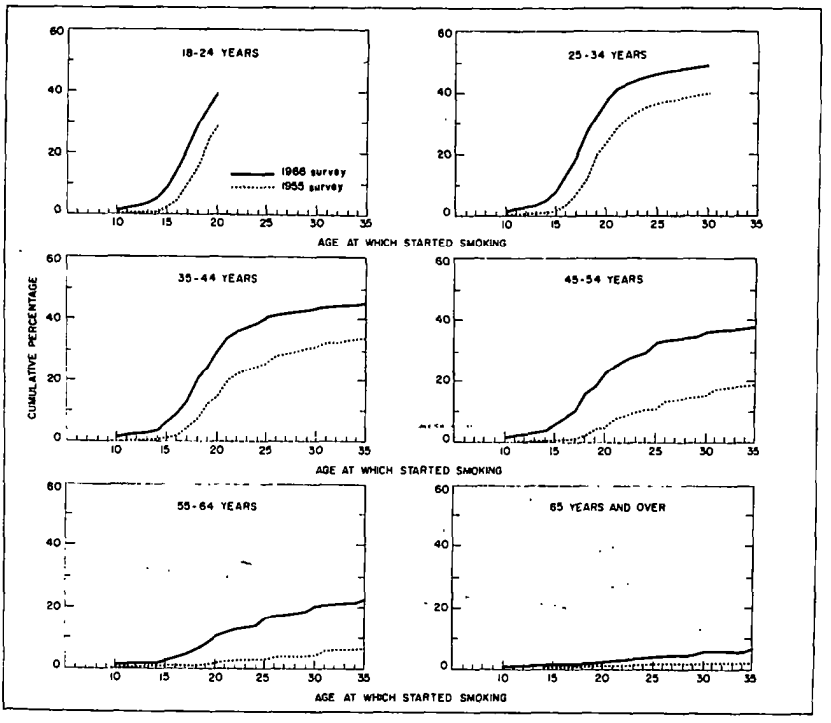
PERCENT
CURRENT REGULAR SMOKERS-TEENAGE,
1968-1974



Source: DHEW Public No. (NIH) 76-931

Figure 6

Figure 7. Cumulative percentage of females becoming regular cigarette smokers prior to age specified by age at time of survey.



Source: PHS Public No. 1000-Series 10, No. 59, pg. 10

age among women through all age groups. This was most marked for the age group 45-54 years in which the greatest making gains had occurred in the 11-year period between the surveys. Preliminary analyses of the 1975 survey data (DHEW/CDC/NCI, 1976) reveal a similar trend among males towards earlier initiation of smoking than that observed in the earlier surveys (DHEW, 1964, 1968 and 1970).

AMOUNT SMOKED PER DAY

Just as age at initiation of smoking would imply an overall exposure experience for the population at a point in time for any given age group, so the amount of cigarettes (or cigars or pipes) smoked per day constitutes a relevant component of the habit pattern with respect to exposure to the hazard. The complexities of trend analyses of habit components whose values vary not only by sex, but by birth cohort are well known to all. So, in our limited time such trends will be examined for the total survey samples by sex irrespective of the age compositions of the samples which, though comparable, have had varying proportions of smokers, abstainers and discontinuing smokers for the several periods and age groups.

In Table 2 there are presented the average number of cigarettes smoked daily by the population of cigarette strokers as calculated from the data available in the Current Population Survey of 1955 and the surveys by the Clearing house in 1964. 1966. 1970 and 1975. The cigarette smoking male's daily consumption after 1964 seems to have plateaued with virtually little change in 1975. Female smokers, however, after an apparent plateau between 1964 and 1970, resumed their increase and have now almost equalled the male in daily consumption.

INHALATION PRACTICES

Another component of smoking behavior highly relevant to selected health hazards is the inhalation practices of the smoker. The numerous studies, both case-control and cohort, on the relationship of smoking to selected diseases have demonstrated a dose-response gradient not only with the amount smoked and duration of smoking but also with the degree of inhalation. A considerable amount of attention in our efforts at behavior modification has been directed toward changes in the way people smoke. Trends in this component of smoking behavior are presented in Table 3 in which practices have been presented with regard to depth of inhalation by such responses as "deeply into the chest," "only partly into the chest" and "into the chest but I don't know how far." This table also includes data on the proportions of smokers who "inhale almost every puff" of each cigarette. A distinct optimistic trend in depth of inhalation practices is noticeable in this eleven-year period for both males and females. It is, of course, difficult to assess the relative roles which numerous factors may have played in this decline, particularly in the face of increases in smoking among some female age-groups.

Table 2. Average number of cigarettes currently smoked per day by males and females in the U.S.
(CPS - 1955 and NCSH - 1964, 1966, 1970, 1975)

sex	Average No. of Cigarettes Daily				
	1955 (CPS)	1964 (NCSH)	1966 (NCSH)	1970 (NCSH)	1975 (NCSH)
Male	17.7	21.7	21.9	22.3	22.4
Female	13.0	17.2	17.1	17.4	18.8

Sources: Haenszel (1956); DHEW/NCSH (1964, 66, 70, 75).

Table 3. Percentages of Current Male and Female Cigarette Smokers Inhaling Smoke and Inhaling Virtually Every Puff of Each Cigarette in the U.S. NCSH Surveys of 1964, 1966, 1970 and 1975.

Sex	Percentages Inhaling				
	Degree	1964	1966	1970	1975
Male	Deeply	36.5	31.8	34.3	30.3
	Partly	39.9	44.5	31.6	32.7
	Into chest, DK how far	-	-	2.0	2.2
Total		76.4	76.3	65.9	65.2
Female	Deeply	22.5	15.5	17.5	16.4
	Partly	39.7	40.8	24.9	25.7
	Into chest, DK how far	-	-	3.1	2.0
Total		62.2	56.3	45.5	44.1
	Percentages Inhaling Almost Every Puff				
Male		63.1	63.0	60.5	58.5
Female		54.8	52.1	47.2	50.7

Sources: DHEW/NCSH (1964, 1966, 1970 and 1975)

TYPE OF CIGARETTES SMOKED

Our attention is being directed primarily to cigarette smoking and this is as it should be inasmuch as this habit constitutes the vast bulk of the tobacco hazard to human health. Without wishing to engage in polemics on the relative merits of cessation of smoking vis-a-vis reduction in numbers consumed, abstention from inhalation and use of filters (a first step toward the creation of a so-called "safe" cigarette), since these will undoubtedly be discussed in depth later in this conference when the problems of behavior modification through motivational techniques will be entertained, and although I am one of those who feel greater efforts must be expended toward total cessation, nevertheless it must be recognized that lowering the dose of a deleterious agent should lower the risk of untoward effects. Thus filtration of smoke, if effective, should lead to a lessening of the hazard. Such filtration might reduce particulates and condense some distillates but usually is ineffective against gases. Nevertheless the production of filter-tip cigarettes began to rise in 1950. From an estimate of 0.5 percent of the cigarettes produced in 1950, production of such cigarettes rose to 1.3 percent in 1952; 27.6 percent in 1956; 45.3 percent in 1958 to 54.6 percent in 1962. By 1975, production was up to 87.7 percent and the preliminary figures for 1976 is 88.5 percent.

This trend coincides well with the increasing demand for filter-tip cigarettes as is evidenced by the survey data noted in Table 4. The bulk of filter-tip cigarettes consumed are king-size. Increases in their use have been, for both sexes, primarily at the expense of the non-filter types. It will be noted that women adopted the king-size-filter-tip cigarettes earlier and more rapidly than did the men. When the 100 m.m. cigarette came along women adopted these quickly as well. By 1975 (DHEW/NCI, NCSH 1976) 35-percent of women smokers compared to 17 percent of the men utilized the 100 m.m. brands. This was double the proportions smoked by each sex 5 years earlier. Predictions of the effect of greater amounts of tobacco smoked per cigarette, albeit with filters, would be sheer speculation..,.Whereas, in 1975, somewhat more men than women (60.4% vs. 53.646) smoked king-sized-filter tip cigarettes, more women than men smoked filter tip cigarettes of any size (90.6% vs. 79.3%) thus maintaining their lead in the use of filters.

Time does not permit detailed discussion of trends in the tar and nicotine content (but not other toxic materials) of cigarettes and their use other than that the smoker is turning to cigarettes with lower tar and nicotine levels. It is my assumption that my colleague Dr. Wynder will have some significant remarks to make in this regard. The tar and nicotine "derby" initiated by the Federal Trade Commission's requirement of labeling cigarette packages with the levels of these substances is a matter of fact. Its effect remains to be evaluated.

Table 4. Percentages of Current Male and Female Smokers Using the Several Available Types of Cigarettes by Size and Presence of filter. NCSH Surveys of 1964, 1966, 1970 and 1975.

Type of Cigarette		Percentages Smoking			
Length Filter	Sex	1964	1966	1970	1975
King-size - No Filter	M	18.6	17.8	13.5	9.2
	F	15.2	14.5	11.3	5.6
King-size - Filter	M	49.7	55.2	58.5	60.7
	F	70.1	76.6	62.2	54.0
loo mm - Filter	M	–	–	8.8	16.7
	F	–	–	18.2	34.6
Regular size - No filter	M	26.4	24.9	15.3	10.6
	F	7.8	5.1	4.6	2.9
Regular size - Filter		4.3	1.8	1.1	1.9
	F	6.5	3.8	2.9	2.0
Totals		99.0	99.7	97.2	99.1
	F	99.6	100.0	99.2	99.1

Sources: DHEW/NCSH (1964, 1966, 1970 and 1975)

OTHER HOST CHARACTERISTICS AND DEMOGRAPHIC VARIABLES

One would be derelict in discussing patterns of making behavior. not to mention several other selected characteristics of smokers which, for the epidemiologist and social scientist, help to define populations at risk and provide the targets for remedial action. Without belaboring the voluminous data collected on several of these variables in all four surveys of smoking behavior in the Clearinghouse series, a brief summary will suffice. In all the surveys, except for declines in current smoking percentages, the same relative relationships between smoking habits and certain demographic characteristics have maintained themselves. In fact the same rank order has prevailed since the first large scale survey in 1955 (Haenszel, 1956).

Marital Status

In Table 5 there are presented the prevalences of cigarette smoking among both men and women by marital status. Both men and women who are divorced or separated have the highest current smoking rates (60% and 50% respectively) as compared to married men (38%) and women (28%). The 1975 rate for the divorced or separated males is a decrease from the rates which prevailed in the earlier surveys, but that for divorced or separated females is a continuing increase over the 1970 rate of 44.1% after a low of 41.7% in 1966.

Thirty-six percent of widowed men were current cigarette smokers in-1975. This is approximately the same rate as in 1970 but significantly lower than the rate in 1964. The widowed male rate is actually slightly lower than the rate for married men. The widowed female, however, has a current smoking rate of 19%, the lowest of all the marital classes. However comparison with the rate in 1964 shows no difference though the rates were lower in the intermediate survey years.

Single male current cigarette smoking rates have shown some marked fluctuations in the 10-year period but in 1975 the rate is now even lower than in 1966. The single female smoking rates have been more stable with the 1975 rate of 31% lower than in any of the other survey years with the exception of the remarkably low 27.6% achieved in 1966.

Educational Achievement and Income

An interesting and not readily explainable pattern of smoking behavior evolves from an analysis of the educational level of the survey samples. Table 6 presents the prevalences of current cigarette smoking only among the several levels of educational achievement for men and women separately, A pattern of an inverse relationship between educational level achieved and the prevalence of cigarette smoking is immediately apparent. However the gradient is not perfect for either sex since the groups in the

Table 5. Distribution of Prevalence of Current Cigarette Smoking by Marital Status and Sex. NCSH Surveys

Year of Survey	Percentage Current Cigarette Smokers							
	Married		Widowed		Divorced/Separated		Single	
	M	F	M	F	M	F	M	F
1964	52.7	31.5	49.4	19.4	62.7	55.3	52.2	38.9
1966	52.9	38:0	28.0	15.2	59.2	41.7	49.6	27.6
1970	39.8	31.7	35.2	16.7	65.0	44.1	56.3	36.1
1975	38.3	28.3	35.7	19.3	60.1	50.0	37.5	30.6

Sources : DHEW/NCSH (1964, 1966, 1970 and 1975)

Table 6. Distribution of Prevalence of Current Cigarette Smoking by Educational Level and Sex. NCSH Surveys.

Year of Survey	Percentage Current Cigarette Smokers											
	Grade School or less		Some High School		High School Graduate		Some College		College Graduate		Post-graduate Degree	
	M	F	M	F	M	F	M	F	M	F	M	F
1964	49.5	18.2	62.0	36.5	56.8	35.4	50.4	36.1	42.3	36.5	42.7	30.1
1966	49.9	18.2	60.4	39.8	55.1	43.2	53.4	35.9	38.6	29.1	33.9	24.6
1970	39.2	19.7	51.0	34.4	47.7	32.2	37.3	36.2	31.8	25.5	29.1	27.2
1975	37.4	18.2	47.8	33.2	45.6	31.9	36.1	32.2	28.2	21.8	27.9	20.2

Sources: DHEW/NCSH (1964, 1966, 1976 and 1975)

surveys who did not go beyond grade school had consistently lower prevalences than did the groups who attended high school. It has, been proposed that since educational level is related to income and since there may be a further relationship between income and the relative incapability of purchasing cigarettes by the lowest income groups this factor would account for the abrupt inflection of the prevalence curve below the high school level.

In an analysis of income data this explanation would be plausible for the female, but not for the male as can be seen from Table 7 which presents smoking status at the several levels of income. The income distribution patterns for smokers are virtually identical among all four surveys and persist through the overall secular trends as wall. It can be noted that a direct relationship between income and prevalence of cigarette smoking exists for the female which would tend to support the "purchasing-power" hypothesis, but this relationship does not appear for the male who tends to show a plateauing of consumption in the middle income groups and lower consumption both in the very low and high income groups . In effect, then, in the highest income groups, the prevalence of current cigarette smoking is almost the same for both sexes. An all-en-bracing reason for these disparities does not suggest itself. It would not be amiss to suggest that several differing factors may be operating in the two sexes.

Occupation

Analyses of data on occupation of respondents in each of the surveys reveals a pattern which has changed very little qualitatively since the earliest comprehensive smoking survey in 1955. Professional and technical workers have continued to have the lowest cigarette smoking rates whereas laborers, craftsman and other 'blue collar" workers, the highest. Over the years of the several surveys these contrasts have prevailed. Disparities between the sexes are however apparent.

The male in "blue-collar" occupations (including farm laborers and foremen) although declining in current cigarette smoking from 6% in 1966 to 51% in 1970 to 4% in 1975 consumes more cigarettes than man in "white-collar" occupations (including farmers and farm managers), which included 48%, 37% and 36% cigarette smokers respectively in the last three surveys.

The female on the other hand shows an opposite relationship. Employed "white collar" female workers smoke somewhat more. commonly (34%) than do women in 'blue-collar" types of occupation (3%). In the 1975 survey, 40% of the women in the sample worked outside of the home and of these 3% were current cigarette smokers as compared to 27% among housewives.

Table 7. Distribution of Prevalence of Current Cigarette Smoking by Income and Sex. NCSH Surveys.

Income	Percentage Current Cigarette Smokers							
	Male				Female			
	1964	1966	1970	1975	1964	1966	1970	1975
Under $3,000	47.5	46.1	40.1	41.1	19.4	18.5	17.2	23.7
3,ooo-4,999	48.5	54.3	45.2	43.8	31.9	25.9	32.6	26.3
$5,000-7,499	58.9	56.4	44.1	41.1	33.5	40.2	27.6	27.0
$7,500-9,999	54.3	53.3	42.9	46.4	37.7	40.6	34.3	31.1
$lO,OOO-14,999	51.8	45.5	43.0	38.3	38.4	46.0	36.8	30.1
$15,000 & over	44.7	50.0	39.6	37.4	38.0	45.6	39.1	33.6

Sources: DHEW/NCSH (1964, 1966, 1970 and 1975)

DISCONTINUANCE OF SMOKING

Earlier in this presentation the declines in the proportions of cigarette smokers among adults 21 years of age and over have been noted for both males and females and over all age groups. This latter portion of the statement is acceptable if we consider that the virtual rise of consumption in the 55 year and over age group of females is in truth a cohort effect and reflects the residual of the real increase in the 45-54 year group in the 1966 survey. With the decline in smoking among adults, the discontinuance patterns would not only be of general interest but could provide requisite information for the behavioral scientist in planning habit modification approaches.

Cross sectional surveys, if conducted identically, can provide more than a static estimate of the proportions of current and former makers. A crude estimate of the degree of entry into the current smoking category and departure into the ranks of the former smoker category is possible provided that information on the category of individuals who have never smoked is also available. Given a constant level of never smokers as prevailed among males in the surveys of 1964, 1966 and 1970 (Table 8), a constant stream of initiated smokers would have entered the current smoking category. However, despite their constant initiation, an increasing proportion of then discontinued their smoking thus depleting the ranks of the current male smokers as can be seen from Table 8. In 1975 a change in the dynamics had occurred in which the ranks of the former smoker were smaller, but because the formerly static level of "never smokers" had now increased since fewer persons had begun to smoke, the decline in the current smoker category continued. This pattern, however, was not followed by the female. In the first three surveys her initiation of smoking actually increased so that despite increases in discontinuances virtually proportionate to initiations, her current smoking category remained relatively constant. In 1975 with virtually no change in the proportion discontinuing, but with a slight decline in initiation, the current smoking proportion among females declined slightly.

To the behavioral scientist the characterization of current and former smokers with respect to certain process characteristics including recidivism is important not only for design of approaches to behavior modification but for evaluation of the methods utilized in encouraging cessation. The surveys of the Clearinghouse have provided a wealth of data on opinions, attitudes and beliefs, many of then relevant to the problem of prevention of smoking or interceding for cessation. A troublesome problem which many of us have experienced is that of recidivism Data on the number of attempts at cessation of smoking do provide a reasonable measure of recidivism, particularly for the current smoker who has tried and failed.

Table 8. Distribution of Classes of Cigarette Smokers Aged 21 Years and Over According to Sex. Four Surveys of the NCSH.

Year of Survey	Percentages by Class of Smoker and Sex							
	Never		Ever		Former		Current	
	M	F	M	F		F	M	F
1964	24.9	61.1	75.1	38.9	22.2	7.4	52.9	31.5
1966	24.5	56.8	75.5	43.2	23.6	9.5	51.9	33.7
1970	25.1	54.7	74.9	45.3	32.6	14.8	42.3	30.5
1975	31.5	56.6	68.5	43.4	29.2	14.5	39.3	28.9

Sources: DHEW/NCSH (1964, 1966, 1970 and 1975).

In Table 9 there are presented the experiences of both current and former smokers in attempts at discontinuing their cigarette smoking habit. Throughout the three surveys depicted, the currently continuing smoker, both male and female, has made more attempts in the 4-year period prior to each respective survey to discontinue smoking than has the former smoker. The order of magnitude of the frequency of attempts seems to have been approximately equal for males and females. It is obvious that former smokers have found it easier to quit, since a smaller number of them required many attempts to cessation and in the 1970 survey a fewer number of attempts by the presently former smokers led to cessation within the 4-year period.

The factors in recidivism are numerous and complex and will undoubtedly be discussed by behavioral scientists during this conference. Of interest are some of the other characteristics related to discontinuance of smoking. Evidence exists that at least for male smokers, current or continuing smokers had begun smoking at a younger age than discontinued smokers. Men and women continuing smokers have on the average smoked 5 to 6 years longer than discontinued smokers. A greater proportion of smokers of cigarettes who had higher education have become former smokers. A greater proportion of white collar workers who have smoked cigarettes have become ex-smokers than have those smokers among blue-collar workers. The greatest increases in smoking cessation occurred among the older age-groups in men but in the 25-34 year age group in women.

SUMMARY

In summary, cigarette smoking has largely replaced all other forms of tobacco use in the United States. World War I and World War II were the settings for the rapid expansion of cigarette use by men and women respectively. A plateau was reached and, currently, a small decrease experienced in per capita cigarette tobacco consumption following the release of the Report of the Advisory Committee on Smoking and Health to the Surgeon General in 1964.

Several smoking surveys have revealed a decline in current cigarette smoking among adults far more prominent among men than women. After an initial rise among teen-age boys a decline in cigarette smoking has occurred. This has not been the case with teen-age girls, who show a continuous increase in proportional smoking. Both males and females in the U.S. population are initiating their smoking habit at ever earlier ages. Among adults, male consumption of cigarettes per day has plateaued during the past 10 years, but some increases are noted for females. Declines in inhalation practices with regard to depth of inhalation have been demonstrated, but little change has been noted in percentage inhaling every puff.

Table 9. Distribution of Current and Former Cigarette smokers Aged 26 Years and Over According to Attempts at Quitting Smoking by Sex. Three Surveys of the NCSH.

Year of Survey	Present Smoking Status	Percentage Trying to Quit					
		Tried at least once		Tried once only		Tried 2 or more times	
		M	F	M	F	M	F
1964	Current	55.8	50.6	18.5	18.2	37.3	32.4
	Former	48.6	35.3	13.0	9.9	35.6	25.4
1966	Current	54.8	57.1	17.7	16.6	37.1	40.5
	Former	49.2	41.7	12.1	7.6	37.1	34.1
1970	Current	48.6	49.8	19.2	16.6	29.4	33.2
	Former	42.1	49.5	22.9	25.3	19.2	24.2

Sources: DHEW/NCSH (1964, 1966, 1970).

N.B. Question on quitting in 1975 survey was in a different and hence non-comparable form.

The market has been preponderately converted to king-size-filter cigarettes and females primarily use this type of cigarette.

Cigarette smoking is found more commonly among males than females, though increases in proportion of smokers have been noted in the older age groups of women; more commonly among divorced or separated persons of either sex than married or single persons; more male blue-collar workers than white-collar; more female white-collar than blue-collar workers; more among those with lower educational achievement; and more females in higher income groups.

Current smokers continue to have problem discontinuing smoking and have made more futile attempts than ex-smokers in the latter's precessation period.

It is difficult to assign specific causes to the modest gains that have been made in cessation of smoking or nodification of smoking behavior. What impact such motivational factors as dissemination of information on the health hazards of smoking, labeling of cigarettes, information on modification of smoking behavior, smoking cessation clinics, the prohibition of television advertisement of cigarettes, and the exemplar role of health professionals' personal cessation of smoking may have had on this modest modification of behavior is entirely conjectural. At least the relative roles which these and other factors have played have barely begun to be tested.

The challenge for behavioral scientists, health educators and clinicians is great and the conquest of the problem would, in my opinion, provide the greatest forward thrust in the prevention of the greatest amount of early morbidity and mortality from disease than any other single measure of intervention.

REFERENCES

Ahmed, P.I. and Gleeson, G.A. Changes in Cigarette Smoking Habits Between 1955 and 1966. P.H.S. Publication No. 100-Series 10, No. 59, Rockville, Md. 1970.

Haenszel , W. , Shinkin, M.B. and Miller, H.P.. Tobacco Smoking Patterns in the United States. U.S. DHEW Public Health Monograph No. 45, Gov't Printing Office, Washington, D.C. 1956.

Kahn, H.A. The Dorn study of smoking and mortality among U.S. veterans. In: Haenszel, W., editor. *Epidemiological Approaches to the Study* of *Cancer and Other Diseases* Bethesda, U.S.P.H.S. Nat'l. Cancer Inst.. Monograph No. 19, January 1966.

Milmore, B.K. and Conover, A.G. Tobacco Consumption in the United. States, 1880 to 1955. *Agric. Econ. Res.*, 8:9-13, 1956.

Schuman, L.M. Relationship of Membership Responses to Health Related Facts in the APHA Membership Smoking Survey. Presented at the 100th Annual Meeting of the American Public Health, Atlanta City, N.J., Nov. 13, 1972.

U.S. Dept. of Agriculture Economic Research Service, The Tobacco Situation Ts-159, Washington, D.C. 1977.

U.S. Dept. of Health, Education and Welfare, Public Health Service. Smoking and Health, Report of the Advisory Committee to the Surgeon General of the Public Health Service, P.H.S. Publication No. 1103, U.S. Gov't Print. Off. 1964.

U.S. Dept. of Health, Education and Welfare, Public Health Service. Use of Tobacco-Practices, Attitudes, Knowledges and Beliefs, U.S. Fall 1964 and Spring 1966. NCSH, July 1969.

U.S. Dept. of Health, Education and Welfare, Public Health Service. Teen-age Smoking: National Patterns of Cigarette smoking, Ages 12 through 18, in 1968 and 1970. DHEW Publication No. HSM72-7508, Washington, D.C. 1972.

U.S. Dept. of Health Education and Welfare, Public Health Service. Adult Use of Tobacco-1970, NCSH, DHEW Publication No. (HSM) 73-8727, 1973.

U.S. Dept. of Health Education and Welfare, Public Health Service. Teen-age Smoking: National Patterns of Cigarette Stoking, Ages 12 through 18, in 1972 and 1974. DHEW Publication No. (NIH) 76-931, Washington, D.C. 1976.

U.S. Dept. of Health, Education and Welfare, Public Health Service. Adult Use of Tobacco-1975, Center for Disease Control and National Cancer Institute, June 1976.

AUTHOR

Leonard M. Schuman, M.D.
Professor and Director; Division of Epidemiology
School of Public Health
1360 Mayo Memorial Building
420 Delaware St. S.E.
University of Minnesota
Minneapolis, Minnesota 55455

DISCUSSION OF DR. SCHUMAN'S PAPER

Data presented by Dr. Schuman indicated that the age at which smoking started was related to success or failure of smoking cessation at a later time. The earlier one starts smoking the less likely the individual will stop smoking. Since the onset age of smoking is now decreasing, it will be more difficult in the future to help people stop. This apparent or implied cause-effect relationship is consonant with most learning theory principles that earlier formed habit patterns are more easily acquired and more resistant to extinction both in animals and humans. This phenomenon would be in keeping with the nicotine dependence data so well researched by Dr. Russell.

Dr. Schuman's data showed that successful ex-smokers made fewer attempts to quit smoking prior to doing so, than those who did not succeed in quitting. In other words, people who have continued to smoke, continue to attempt cessation, but continue to fail. This is significant but gloomy. Failure rates were not the same for males and females and that deserves study in itself. While fewer attempts for successful quitters supports a biochemical explanation, the phenomenon can also be explained behaviorally. If success in quitting (expectation) is not achieved early, the individual gradually develops a defeatist attitude. He/she may enter new programs but will expect to fail and this will be reinforced over and over.

One of Dr. Schuman's slides revealed that per-capita cigarette con*sumption* went down in 1969-1970. Since this period was coincident with the demise of cigarette advertising in television, a meaningful tactic might be to suppress advertising in the written media. In response to this, Dr. Schuman argued that during this same period teenagers were smoking more. Therefore, the drop in percapita consumption had not included their share. To attribute this drop to the manipulation of the media, however, is still speculative and only a case controlled study would clarify the issue.

It was also argued that the American Cancer Society has been very successful in adding to the decline in cigarette consumption [reported by independent small scale studies) through a series of public television programs showing the dire effects of long term smoking behavior on actual people, including some television stars. Likewise then, the effect of the "moratorium" agreement between the Advertising Council, the Tobacco Industries and the American Cancer Society must be considered in this reduction of cigarette consumption.

Joseph W. Cullen, Ph.D.

Interrelationship of Smoking to Other Variables and Preventive Approaches

Ernst L. Wynder, M.D.

INTRODUCTION

It has been well documented that cigarette smoking alone and in synergy with various other factors enhances man's risk for a number of specific diseases (U.S. Public Health Service, 1964, 1971; Hamnond, 1966). Although evidence exists that smoking represents a pharmacological dependence in sanecases, cigarette smoking is also seen to be related to a variety of social and/or cultural factors (Russell, 1971a, 197lb). This communication will present a brief discussion of both of these aspects of the use of cigarettes and on the preventive approaches to cigarette smoking.

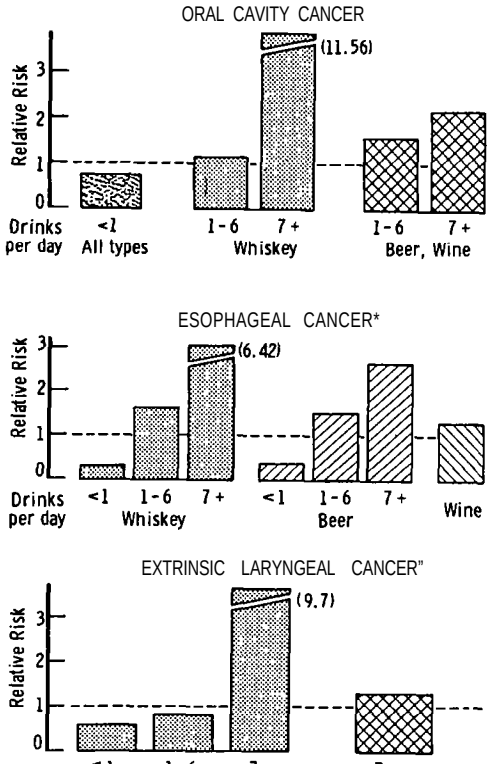
CIGARETTE SMOKING AND OTHER RISK FACTORS

Alcohol

The risk of developing cancer of the upper alimentary tract has been shown to be significantly increased by a combination of tobacco usage and heavy alcohol consumption (Wynder et al., 1976; Moore. 1971; 1965) (Figure 1). The data indicate that tabacco consumption; whether in the form of cigarette, cigar or pipe smoking or the chewing of tobacco, enhances the risk of cancer of the mouth, larynx, and esophagus. Heavy alcohol intake, however, will not by itself produce a significant increase of such cancers (Figure 2). Thus, alcohol principally acts as a tumor promoter in tobacco carcinogenesis. While experimental data have shown that alcohol can act as a solvent for carcinogens present in tobacco smoke by enhancing the effect of such components as benzopyrene in the esophagus of mice, the major effect of alcohol appears to relate to nutritional deficiences (Wynder and Klein, 1965). Ap-

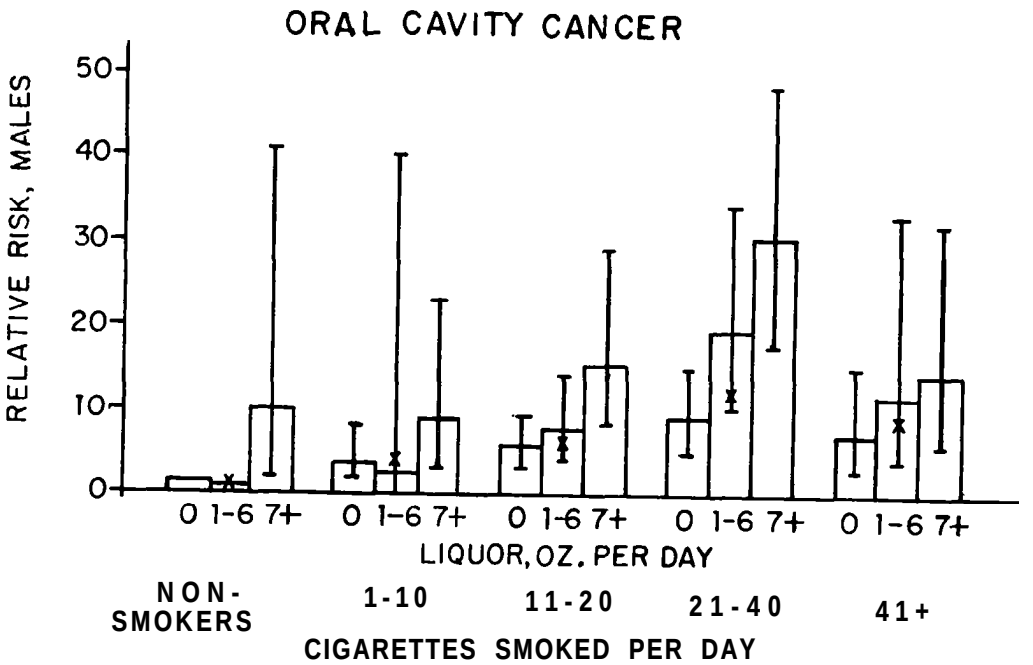
FIGURE 1

RELATIVE RISKS OF DEVELOPING CANCERS OF THE ORAL CAVITY, ESOPHAGUS, AND LARYNX BY ALCOHOL CONSUMPTION



- Relative risk standardized for tobacco consumption.
- * Relative risk compared with those smoking 16 - 34 cigarettes/day.

FIGURE 2


ORAL CAVITY CANCER
RELATIVE RISK, MALES
50
40
30
20
10
0
0 1-6 7+ 0 1-6 7+ 0 1-6 7+ 0 1-6 7+ 0 1-6 7+
LIQUOR, OZ. PER DAY
NON-SMOKERS
1-10
11-20
21-40
41+
CIGARETTES SMOKED PER DAY

proximately 90% of alcoholics are smokers (Dreher and Fraser, 1968) and these people are known to commonly Suffer from Various nutritional deficiences, especially in terms of a deficiency in the intake of vitamin B (Wallgren, 1971). Such deficiencies have been suggested to be responsible for the increased risk among heavy drinking smokers for cancer of the upper alimentary tract and larynx, both glottic and supraglottic type (Wynder et al., 1956, 1976). Finding an increased risk of glottic cancer among alcoholics indicates that direct contact with alcohol is not required to induce such a risk.

Occupations

Experimental and epidemiologic data suggest a causal relationship between exposure to a variety of industrial agents and the development of a number of cancers, particularly those of the respiratory tract. An interaction has also been noted between smoking and exposure to selective occupational conditions. Data indicate that, among smokers to asbestos amd/or uranium ores acts as a promotor for tobacco carcinogens (Hoffman and Wynder, 1976). The risk, of lung cancer has been shown to be increased particularly for smokers who work with asbestos but, in the absence of tobacco usage, the asbestos exposure is less powerful (Hammond and Selikoff, 1973; Selikoff et al., 1968). This interrelationship does not apply for mesotheliana -- a condition known to be directly related to asbestos exposure. Any discussion of the risk associated with occupational exposures and the development of cancer --whether cancer of the lung, bladder or any other tobacco-related cancer - must also include the influence of smoking in the etiolcgy of the disease. It is important, therefore, that we collect both detailed occupational data and reliable smoking histories. Such data will permit the determination of the relative importance of each risk factor on the developnent of a specific cancer. Such epidemiologic effort is also necessary because, as will be shown subsequently, cigarette smoking habits differwidely among different occupational groups.

Hypercholesteremia and Hypertension

Cigarette smoking plays a particularly important role in premature sudden death from coronary disease (Hammond, 1966; Doyle et al., 1976). Epideniologic data obtained from Crete, Yugoslavia, Japan and other countries suggest that this effect of cigarette smoking occurs principally in populations where arteriosclerosis is prevalent -- a condition associated with hyperlipidema (Keys, 1970). The underlying cause of arteriosclerosis, appears to be hyperlipidemia, especially in terms of hypercholesteremia, with cigarette smoking exerting a pronounced secondary effect. It has been reasoned that nicotine in cigarette smoke, perhaps secondary to the production of catecholamines, leads to amythmias and/or increased sensitivity for thrombus formation (Ball, 1974; Rose, 1973). The extent to which carbon monxide contributes to the

pathogenesis of arteriosclerosis continues to be a question of considerable scientific debate.

The risk for heart attacks is further increased when hypercholesteremia and cigarette smoking are prevalent in an individual. with hypertension (Truett et al., 1967). In this instance, there are three major risk factors that have to be carefully and individually examined to determine the specific contribution each factor makes to the risk of coronary attacks. It needs to be re-emphasized, however, that hypercholestermia is the underlying risk factor, because both hypertension and cigarette smoking have a relatively small effect on coronary death in countries where atherosclerosis has a low prevalence rate.

Air Pollution

Although sporadic and intense episodes of air pollution have been associated with deaths from acute pulmonary disease, there is no conclusive evidence that air pollution, per se, directly contributes to the incidence of lung cancer. The geograpic distribution of lung and other neoplasm suggests the presence of an "urban factor" affecting the development of these diseases (Wynder and Harmond, 1962). However, when the data are standardized for comparability of reporting, for variation in smoking habits and in occupational categories between urban and rural areas, the differences in lung cancer death rates largely disappear (Hoffmann and Wynder, 1976). Perhaps such negative findings should be expected Men we consider the relative concentration or exposure to carcinogens in polluted air compared to cigarette smoke. Since air is inhaled in relatively mall doses through the nose and cigarette smoke is highly concentrated and inhaled directly into the lungs, tobacco smoke represents a much more intense exposure to the respiratory tract than air pollution. As a point of reference, one cubic centimeter of heavily polluted air includes up to 100,000 particles per cubic centimeter whereas inhaled cigarette smoke includes up to 5 billion particles per cubic centimeter (Hoffman and wynder, 1970).

In summary then, as we look at the risk among cigarette smokers in reference to different diseases, we must examine a number of other environmental factors in order to evaluate the degree and the nature of the role played by each.

FACTORS INFLUENCE SMOKING HABITS

The fact that there was once a time when cigarettes were not part of man's culture and that even today there are large segments of populations that do not use tobacco, clearly indicates that cigarette smoking is not a necessary component of man's existence. This does not deny that once a personhas begun the habit, he may find it satisfying and wish to continue it throughout his life. Such dependence occurs despite knowledge that this habit is asso-

ciated with a high degree of risk for specific diseases (U.S. DHEW, 1976a).

The Beginning of Smoking

Children have a natural tendency to imitate adult behavior not only because of a desire to be a member of the grown-up world but also in an effort to identify with the parents. Thus, parents and other adults who smoke provide behavior patterns that seem appealing and mature to the young.

Children view these behaviors as socially acceptable and tempting despite parental and medical admonitions against the initiation of the habit. It is not surprising, therefore, that even with the knowledge that cigarette smoking is a potentially harmful habit, a sizeable proportion of teenagers smoke. During the past decade, the proportion of boys between 15 and 16 who have become regular smokers has remained fairly stable (around 19% of the population) whereas a steady increase in smoking is evident among girls this age (U.S. DHEM, 1976a); the proportion of teenage girls (15-16) who are current smokers has more than doubled since 1968 (Figure 3).

These results clearly indicate an inherent failure of current healtheducation techniques in our school system and underscore the necessity of developing and implemanting new methods to discourage children from engaging in cigarette smoking.

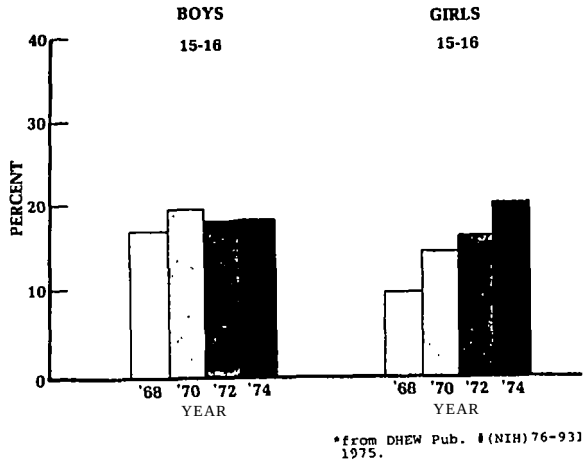
Smoking and Adults

Efforts to educate the public about the harmful effects of smoking have been more successful with adults than children. Our data, in addition to those of others have shown an increased ability to stop smoking among the more educated males -a finding which is less applicable to females partially because fewer women than man comprise the smoking population. (Figures 4 and 5). It is possible that the ability to quit smoking is more related to the fact that smoking is becoming a socially undesirable habit than it is related to an increased awareness, of health risks associated with cigarette consumption If we can promote the feeling that smoking is socially unacceptable then it is possible that in the future cigarette smoking will disappear, at least from certain social groups, just as the spittoon has disappeared from the Waldorf Astoria.

The cigarette smoking habit has also been shown to have a cultural component in that the habit varies by religious, racial and ethnic group. Because of religious restrictions, Seventh Day Adventists and Mormons do not use tobacco and this abstinence is directly reflected in the lower mortality rates from cigarette related diseases among these special groups (Lyon, et al., 1976;

FIGURE 3

PERCENT
CURRENT REGULAR SMOKERS-TEENAGE,
1968-1974 *



*from DHEW Pub. #(NIH)76-931, 1975.

FIGURE 4

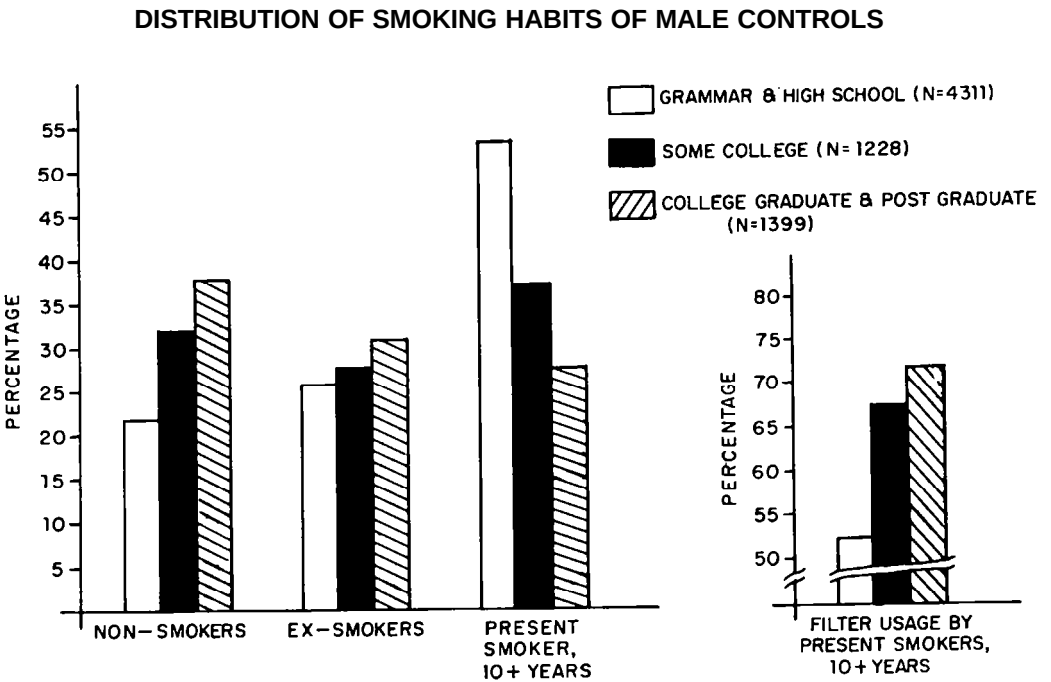
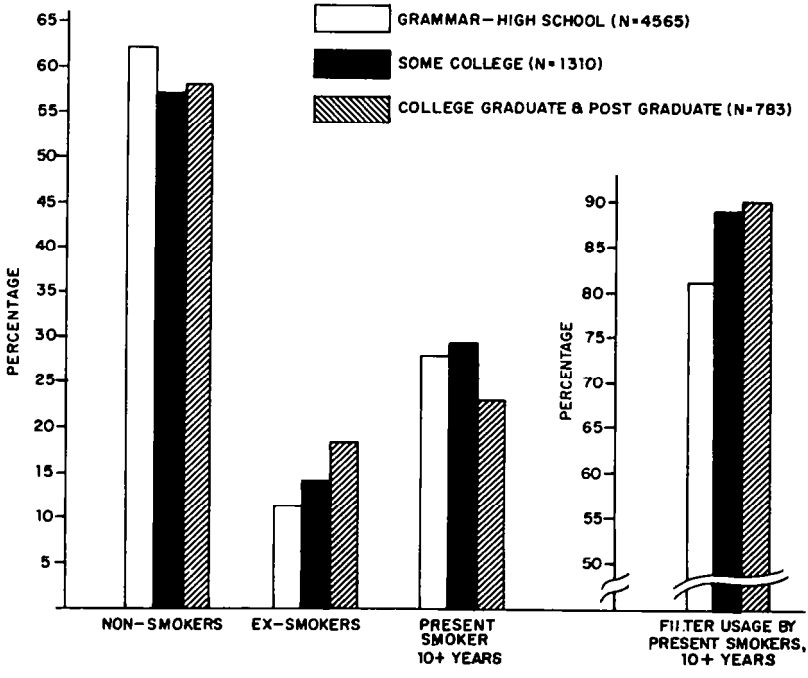

DISTRIBUTION OF SMOKING HABITS OF MALE CONTROLS
GRAMMAR & HIGH SCHOOL (N=4311)
SOME COLLEGE (N=1228)
COLLEGE GRADUATE & POST GRADUATE (N=1399)
PERCENTAGE
55
50
45
40
35
30
25
20
15
10
5
NON-SMOKERS
EX-SMOKERS
PRESENT SMOKER, 10+ YEARS
PERCENTAGE
80
75
70
65
60
55
50
FILTER USAGE BY PRESENT SMOKERS, 10+ YEARS

FIGURE 5

DISTRIBUTION OF SMOKING HABITS OF FEMALE CONTROLS



Phillips, 1975; Wynder et al., 1959). In addition to these groups, our data show Jewish males have less exposure to cigarettes than non-Jewish males, independent of their educational background: a finding which is consistent with the lower rates of lung cancer in these men (Figure 6).

In contrast to this are the Blacks who consistently are underrepresented in the non-smoking and exsmoking categories and who report using non-filter cigarettes more regularly than their White counterparts. This finding also obtains regardless of educational back-

Our data further indicate that among those groups who have proportionately fewer smokers, i.e., Jews, White males with postgraduate education, and women, more of the makers tend to use filter cigarettes. Thus, we are aware of population groups with varying levels of exposure to tobacco smoke condensate. We must continue to monitor the use of tobacco by these groups in an attempt to understand the demographic factors which relate and lead to these differences in smoking habits. Attitudes towards smoking within specific cultural settings are key factors influencing present and future smoking habits of various populations.

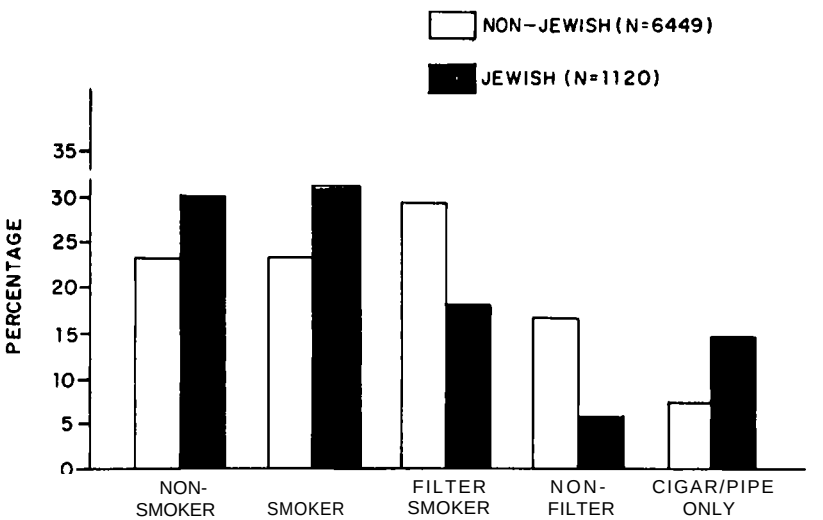
Relationship to Other Habits

Not surprisingly, cigarette smoking is closely associated with a number of other habits. For instance, cigarette smoking is closely related to alcohol consumption as well as to coffee drinking (Figure 8). As indicated previously, epidemiologists need to standardize data for each variable in order to isolate the effects of the different interrelated factors. Such standardization would permit attributing appropriate etiologic, significance to each factor. For instance, no association between increased risk of lung cancer and alcohol consumption is found when the data are standardized for cigarette smoking. However, if the data were not so standardized, a lung cancer patient would be found to consume significant more alcohol than the matched control patient. This standardization does not mask or hide any real association between alcohol and disease, as is apparent when cancer of the upper alimentary tract is studied. when smoking is standardized for this cancer, an increased risk among smokers is found for alcohol consumption (Figure 9).

This close association between smoking and drinking must be carefully considered by those involved in smoking cessation activities. In order to be successful in efforts to help people give up smoking the therapist must also attempt to monitor and/or temper the person's drinking habits. The same applies to coffee consumption. The first cigarette in the morning together with a cup of coffee is a ritual for many people, and this pairing

FIGURE 6

DISTRIBUTION OF SMOKING STATUS OF MALE CONTROLS



AHF, 1976

FIGURE 7

DISTRIBUTION OF SMOKING STATUS OF MALE CONTROLS BY RACE

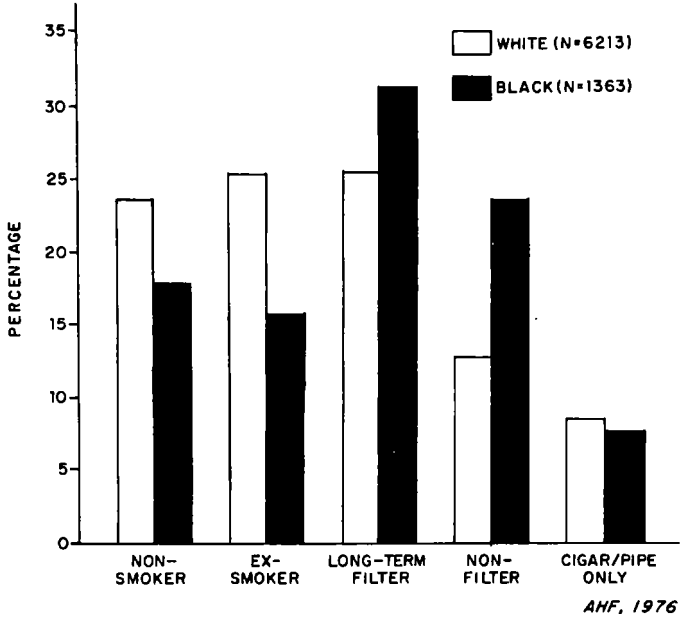


FIGURE 8

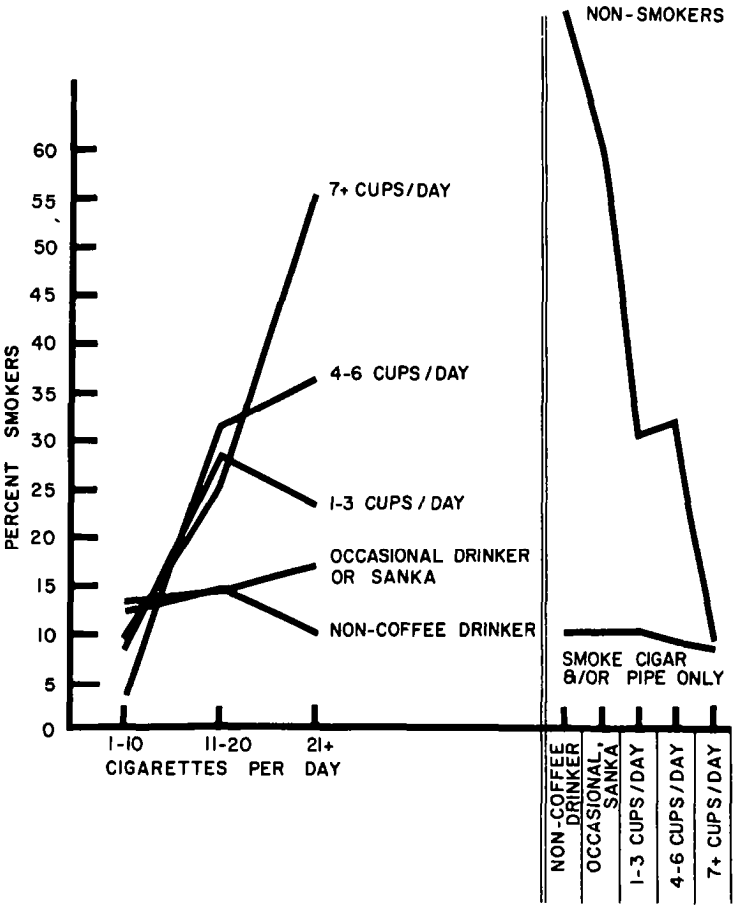
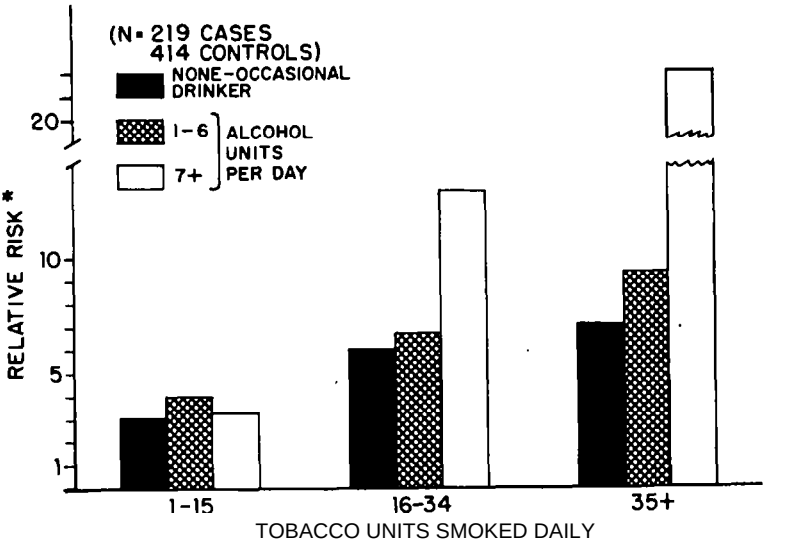

NON-SMOKERS
7+ CUPS/DAY
4-6 CUPS/DAY
1-3 CUPS/DAY
OCCASIONAL DRINKER OR SANKA
NON-COFFEE DRINKER
SMOKE CIGAR &/OR PIPE ONLY
PERCENT SMOKERS
60
55
50
45
40
35
30
25
20
15
10
5
0
1-10
11-20
21+
CIGARETTES PER DAY
NON-COFFEE DRINKER
OCCASIONAL, SANKA
1-3 CUPS/DAY
4-6 CUPS/DAY
7+ CUPS/DAY

FIGURE 9

RELATIVE RISK OF LARYNGEAL CANCER FOR MALE SMOKERS BY AMOUNT SMOKED AND ALCOHOL CONSUMED.



* RELATIVE TO THE RISK OF 1.0 FOR NONSMOKERS WHO NEVER OR ONLY OCCASIONALLY DRANK ALCOHOL.

AHC, 1975

frequently continues throughout the day.

when considering risks associated with bladder cancer, coffee consumption disappears as an independent fact or when the data are standardized for tobacco usage (Wynder and Goldsmith, 1977). The same is true for heart disease. That is, once the effects of cigarette smoking are controlled for, the increased risk previously associated with coffee drinking and heart disease disappears (Dawber, et al., 1974, 1975; Hennekins, et al., 1976; Paul, 1968). Again, it is imperative to disassociate the two habits.

There are a number of instances where the epidemiology of interrelated variables becomes even more complex. A case in point is the purported association between sugar consumption and coronary death (Yudkin and Roddy, 1964). When the data are standardized for coffee intake - a habit which is closely associated with sugar intake -- and in turn standardized for level of cigarette consumption, the reported effect of sugar as a risk factor disappears (Jick, et al., 1973; Klatsky, et al., 1973).

Occupations and Smoking

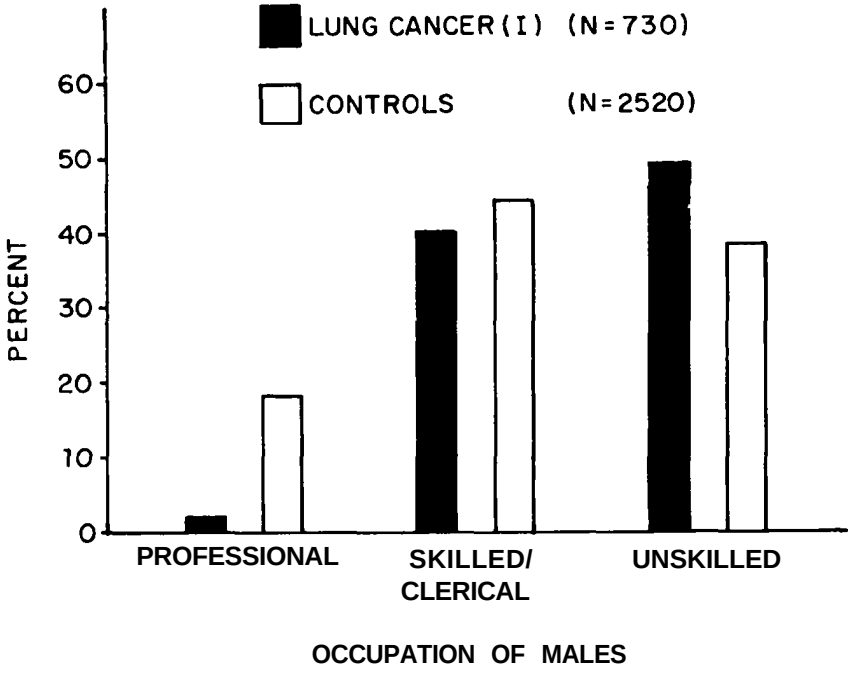
Occupations have a significant influence on smoking habits of the workers both because of the level of education required for various positions, and because of on-the-job smoking restrictions frequently associated with a specific job. When considering occupations ranging from professional to unskilled positions it is easy to see the dramatic effect exerted by the educational component of the particular job on the employees' smoking habits. It is again clear that one must standardize for cigarette smoking before claiming that a given occupational exposure, per se, increases the risk for cancer or other diseases. Our present finding that lung cancer occurs more frequenlty in unskilled workers than in professional workers, is more a reflection of the differences in smoking habits than an effect of different occupational exposures (Figure 10).

It was the purpose of this discussion to show the interrelationship of cigarette smoking to other risk factors and to socio/cultural backgrounds -- knowledge we feel is as important both from an epidemiological point of view as it is for those who are concerned with how to reduce cigarette smoking in our general population.

PREVENTIVE MEASURES

Because tobacco usage is interwoven with a variety of reinforcing personal habits and since it is responsible for an estimated 40% Of male cancer deaths in the.U.S., it is imperative that efforts be directed towards developing more effective preventive programs. We have seen that traditional public health educational efforts of

FIGURE 10


LUNG CANCER (I) (N = 730)
CONTROLS (N = 2520)
PERCENT
60
50
40
30
20
10
0
PROFESSIONAL
SKILLED/ CLERICAL
UNSKILLED
OCCUPATION OF MALES

the past have not been fully effective because a sizeable proportion of both the young and old continue to smoke. This is true even though the health hazards of smoking are known to these people, as indicated in a recent national survey in which two-thirds of smokers indicated concern over the health consequences of their smoking (U.S. DHEW, 1976b). Since it is unlikely that increasing efforts in the area of health education will contribute substantially to producing ex-smokers., additional approaches must be taken.

For Children

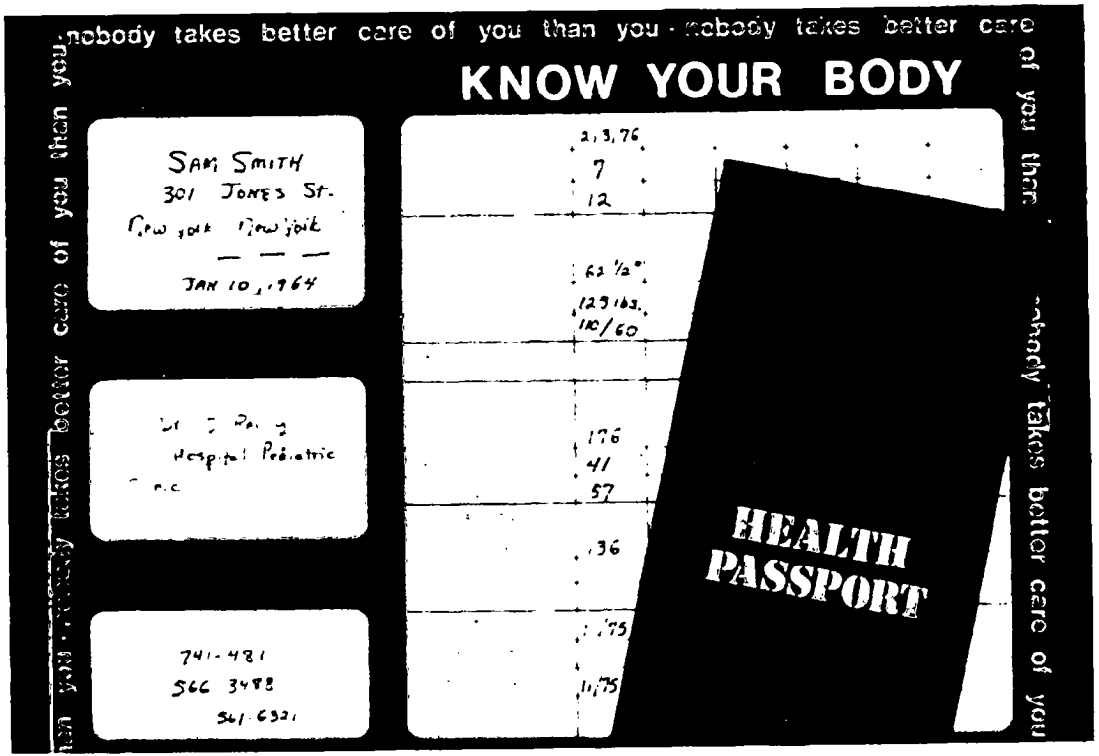
The program for children obviously must be directed towards preventing the initiationof the smoking habit. Parental didactic teaching program and those in schools are apparently not sufficient to curtail the smoking habit of boys ard girls. The American Health Foundation has developed a Know Your body (KYB) program for school children that has at its core the determination of specific risk factors such as cigarette stoking, serum cholesterol, blood pressure, height/weight, etc. Parental involvement in risk factor identification and modification is encouraged and, above all, the child's own involvement in this process is stressed. The risk factors are explained and recorded in a Health Passport for each child (Figure 11).

Clearly what needs to be done is to promote the health conscious. non-smoking child as a status symbol for other children to mimic. We feel that the active and continued involvement of children in these programs throughout the school years is the key to future success, particularly if begun early in the elementary school years and continued through High School. Students must be made aware of the pressures society p1aces on them to take up smoking - Pressures from parents (by example), friends, advertisements (showing beautiful-people smoking). When they understand the pressures, they can be shown how to cope and resist. We need to recognize that health education is more than informing; health education must be practiced. An early education into health and risk factor identification is as important to the future well-being of our children as are learning reading, writing and aritbnetic skills.

For Adults

With regard to adults, we must accelerate efforts in respect to smoking cessation programs. Data from the National Clearinghouse for Smoking and Health and from our own studies indicate that a significant percentage of adults have stopped smoking on their own (National Clearinghouse for Smoking and Health, 1973; Wynder and Mushinski, 1977). As we have previously indicated, the more educated individual is more likely to give up smoking, but we feel this action relates more to peer pressure than to better health knowledge vis-a-vis the less educated groups. It appears that, especially among educated males, the smoker is no longer

FIGURE 11


nobody takes better care of you than you · nobody takes better care
KNOW YOUR BODY
SAM SMITH
301 JONES ST.
NEW YORK NEW YORK
JAN 10, 1964
2,3,76
7
12
63 ½"
129 lbs.
110/60
176
41
57
136
175
11,75
741-481
566 3488
561-6321
HEALTH PASSPORT
nobody takes better care of you than
nobody takes better care of you

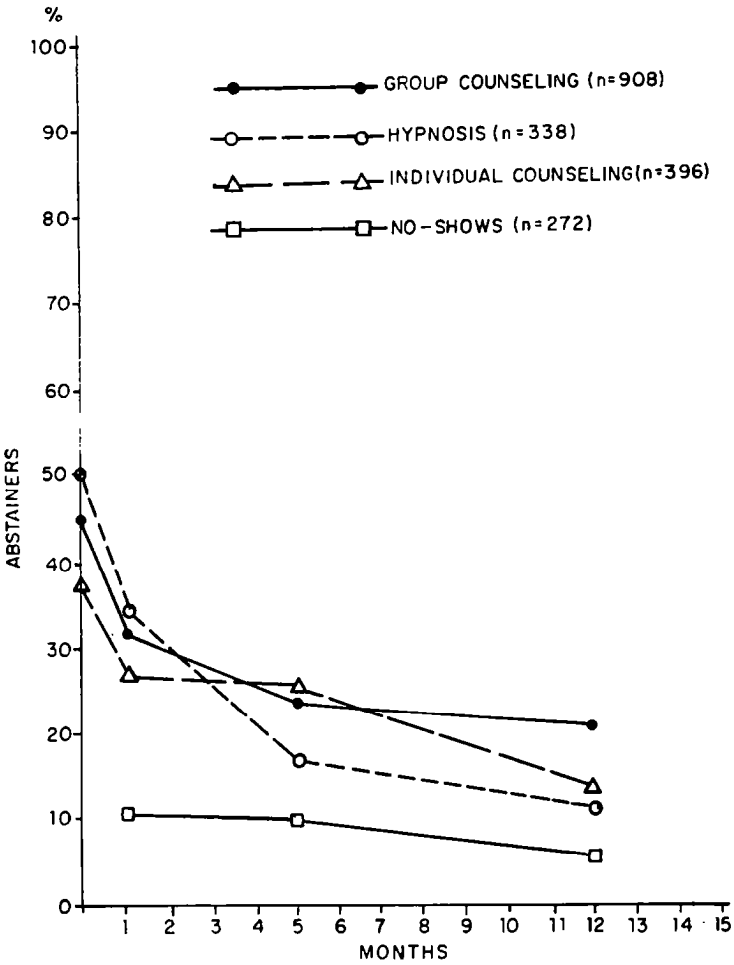
regarded as the "insider." The principal thrust of our educational efforts, therefore, should be directed towards theunattractiveness and/or unacceptability of the smoking habit rather than the continued emphasis on its adverse health effects.

Naturally, there are smokers who require assistance in their efforts to give up smoking. There are marry smokers who want to give up the habit but are incapable of doing so by themselves. Such a situation was evident in a recent Gallup Poll which showed that approximately one-third of smokers who want to quit would be willing to attend a smoking clinic (Gallup, 1974). This is an area where the medical and scientific professions have failed the general public. Whether for econonic reasons, or ah inability or disinterest in behavior modification techniques, the medical profession has not been of assistance to the individual who desires to give up smoking but requires help in doing so. It is evident that well-conducted and well-monitored smoke cessation clinics have proved to be quite a cost-effective means of aiding these individuals (Kristein, 1977). We, and others, have shown that such programs can have a 25% success rate in terms of creating non-smokers out of heavy smokers after one year - a one-year time period of non-smoking generally guarantees that an individual will not return to smoking (Figure 12.) (Shewchuk et al., 1977). Recent evidence indicates that such success rates can be doubled, however, with appropriate maintenance after the termination of therapy.

Comparing individual therapy, hypnosis and group therapy, we have found the latter to be the most cost-effective method. More recently, our clinic procedures have benefitted from the research efforts of behavior scientists interest& in behavior change. Smoking cessation treatment packages are now becoming more sophisticated, structured, and effective as they incorporate new strategies derived from behavior modification and learning-theory perspsctives. Examples of some of these strategies include such diverse techniques as stimulus control, self-regulated punishment, and aversive procedures. These strategies are directed at reducing the rewarding aspects of smoking behavior. In addition, strategies aimed at rewarding non-smoking include such notions as positive reinforcement training, contingency contracting, and non-smoking practice.

The specific role of these techniques will be to help us develop better treatment packages acceptable to the large number of smokers seeking help. Smoking cessation efforts need to be directed particularly to the heavy smoker and to those Smokers with the identified risk factors previously described. As we progress, we should also reach a stage where we can get almost any motivated smoker to achieve initial success off cigarettes. The key to improving these results will be in the development of methods to help smokers stay off cigarettes. A complex overlearned habit such as smoking

FIGURE 12



RESULTS OF INTENSIVE INTERVENTION AHF SMOKING CLINIC PROGRAMS

doubt requires some degree of relearning to overcome. As we begin to achieve greater success in this area, smoking clinics will develop more credibility. The less-motivated two-thirds of smokers who want to quit will also be tempted to give it a try.

We feel that smoking cessation clinics should become an integral part of the entire medical care delivery systemand should receive at least partial reimbursement from health insurance carriers. What type of medical insurance system do we have that pays for all costs related to lung cancer and other tobacco-related diseases but does not reimburse the medical establishment for attempts to reduce the causes of these diseases? Working populations should be able to attend smoking clinics as part of the work schedule and the time could be counted as part of the permitted sick days. It must be stressed, however, that as more and more smoking cessation clinics begin to operate, they must, in order to be accredited, follow specific guidelines (Shewchuk et al., 1977). If properly conducted, smoking cessation clinics should and could make important roads in reducing cigarette smoking in our adult population.

The Less Harmful Cigarette

As long as we as a society, however, permit and condone cigarette smoking (and it is obvious that we are unwilling and/or unable to prohibit this habit), many young people will begin and my adults will continue smoking. It is, therefore, incumbent upon us not only to continue the efforts outlined above but also to continue working towards the development of the "less harmful cigarette" -- a cigarette that is less harmful with respect to cancer as well as to cardiovascular and chronic obstructive pulmonary diseases. During the past thirty years, a significant reduction in the tar and nicotine levels of all cigarettes has taken place in the United States and in other countries (Figure 13). More recently, much effort has been given to the production of cigarettes with tar yields of 10 mg and less (Figure 14). Epidemiologic evidence relative to tobacco-related cancers in addition to data recently reported for coronary heart disease, have indicated a reduced risk among filter smokers for these diseases (Figure 15) (Hammond et al., 1976). Since all of these individuals began their smoking habit with the old high tar, high nicotine cigarette, it can be safely assumed that the risk of tobacco-related diseases among lifelong users of low tar cigarettes would be lower than among those who switch to lower tar cigarettes late in life.

In terms of carcinogenesis, the cigarette must be low in tar as well as specifically low in tumorigenic agents. This area of investigation has been and continues to be researched by Hoffmann and his colleagues (Hoffmann et al,, 1976). Research into the chemical and biological nature of the less harmful cigarette and the epidemiologic monitoring of the health hazards of such cigarettes need to be continued. While the tobacco industry is expected to continue its work in the reduction of tar and nicotine in

FIGURE 13

ANNUAL CONSUMPTION OF TAR NICOTINE PER PERSON
15 YEARS OR OVER, U.S. 1950-1975

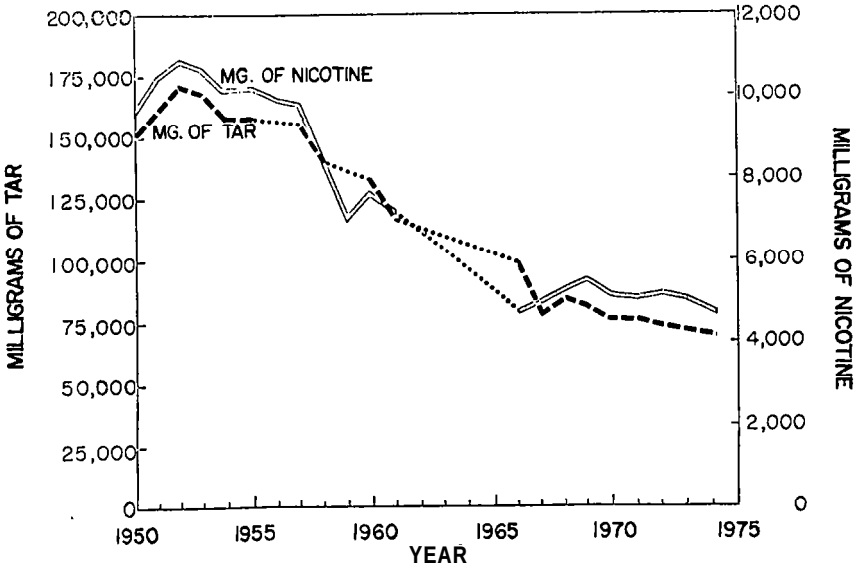


FIGURE 14

MARKET SHARE FOR LOW-TAR (1 - 15MG) CIGARETTES, 1967- 76

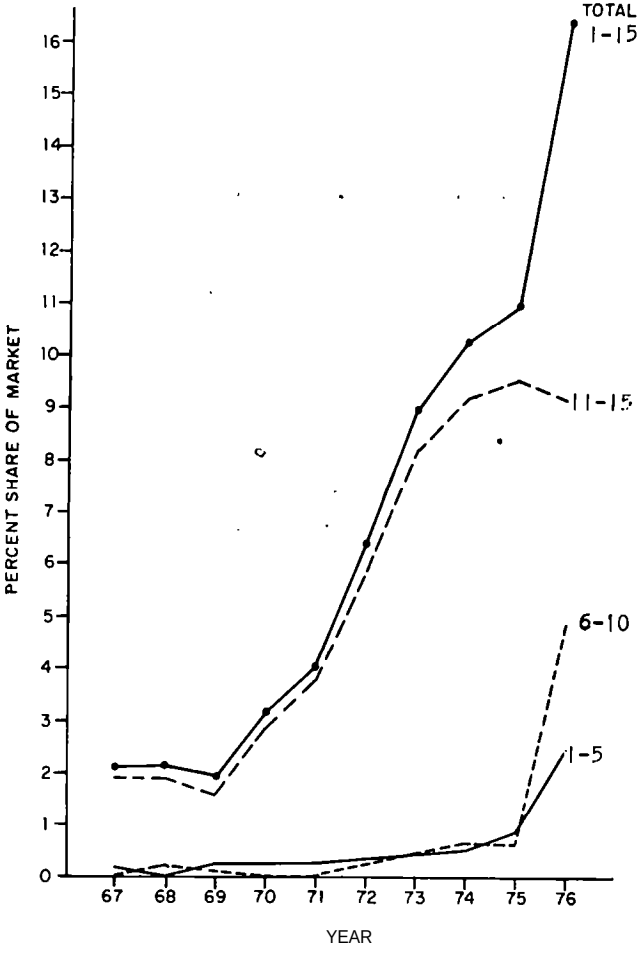
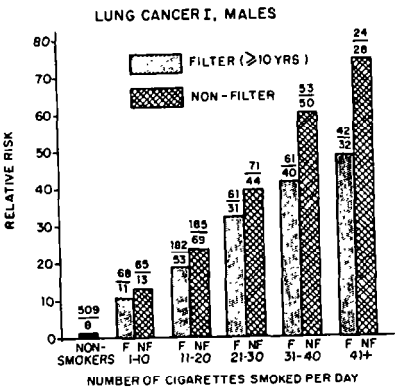
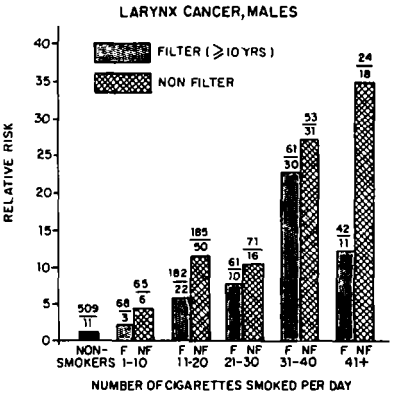
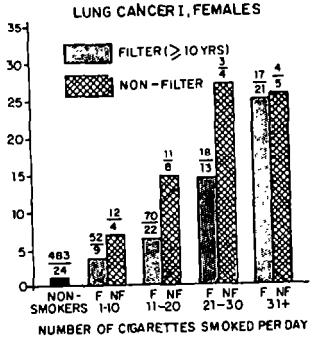
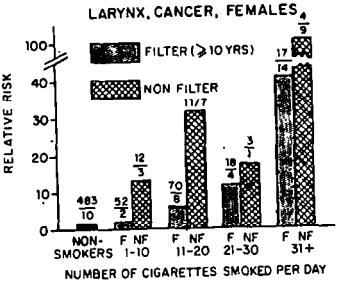


FIGURE 15


LUNG CANCER I, MALES
FILTER (≥10 YRS)
NON-FILTER
RELATIVE RISK
NON-SMOKERS
F NF 1-10
F NF 11-20
F NF 21-30
F NF 31-40
F NF 41+
NUMBER OF CIGARETTES SMOKED PER DAY


LARYNX CANCER, MALES
FILTER (≥10 YRS)
NON FILTER
RELATIVE RISK
NON-SMOKERS
F NF 1-10
F NF 11-20
F NF 21-30
F NF 31-40
F NF 41+
NUMBER OF CIGARETTES SMOKED PER DAY


LUNG CANCER I, FEMALES
FILTER (≥10 YRS)
NON-FILTER
NON-SMOKERS
F NF 1-10
F NF 11-20
F NF 21-30
F NF 31+
NUMBER OF CIGARETTES SMOKED PER DAY


LARYNX, CANCER, FEMALES
FILTER (≥10 YRS)
NON FILTER
RELATIVE RISK
NON-SMOKERS
F NF 1-10
F NF 11-20
F NF 21-30
F NF 31+
NUMBER OF CIGARETTES SMOKED PER DAY

cigarettes, it is also necessary for the non-industrial, scientific Community to contribute to this field. We believe that this field will increasingly contribute to the reduction of tobacco-related diseases.

At present, the funding for tobacco andhealth related programs, whether for education, smoking cessation programs or the develop ment of a less harmful tobacco product, is minimal in contrast to themagnitude of thediseases and medical costs createdby smoking. In view of the fact that tobacco-related cancers account for approximately one-third of the total cancer deaths in males and an increasing number of suchdeaths are being evidenced in females, the total funds available for smoking related preventive programs as part of the National Cancer effort, appears trivial, at best. Inaddition to greater funding, it is necessary for various government and voluntary health agencies to coordinate specific chemical, biological, clinical and educational research programs in an effort to combat these diseases.

Summary

What needs to be done with respect to reducing tobacco-related illnesses is to coordinate and support a broad innovative program involving health education for our children and for adults; to develop and practice better smoking cessation programs for smokers; and to further the development of an increasingly less harmful cigarette. These program need to have the financial and scientific support of governmental agencies, voluntary health organizations, the medical establishment, various scientific professions, and society, in general. There appears to bean insufficient sensitivity in these groups towards this issue, particularly when compared with other public health issues concerning environmental carcinogens. Excessive tobaccousage has been well demonstrated to have a far greater impact on ill health than most other environmental factors. The question that we continue to ask ourselves as a society is why we do not act differently. One great disappointment of those engaged in the smoking and health area for much of their lives is not so much the action of vested interests that could be expected, but rather the general apathy of the madical and scientific professions towards tobacco-related issues.

It has been said that those who do not learn from history are condemned to relive it; one of history's lessons is that most major triumphs in medicine have resulted from preventive measures. When the final chapter on tobacco-related diseases is written, history will once again have repeated itself. The question remains whether we as a society have the ability to learn this lesson from history and act upon it accordingly.

Ball, K.: Smoking and the Heart; The Basis for Action. *The Lancet* 11:822-826. 1974.

Dawber, T.R., Kannel, W.B. and Gordon, T.: Coffee and Cardiovascular Disease. New England *J. Med.,* 291:871-874. 1974.

Dawber, T.R., Kannel, W.B. and Gordon, T.: statistics of coffee and Myocardial Infarction. *New England J. Med.,* 292:266-271. 1975.

Doyle, J.T., Kannel, W.B., McNamra, D.M., Quickenton, P. and Gordon, T.: Factors Related to Suddenness of Death from coronary Disease; Combined Albany-Framingham Studies. *Am. J. Cardiol.,* 37: 1073-1078. 1976.

Dreher, K.F. and Fraser, J.G.: Smoking Habits of Alcoholic Outpatients. *Internl. J. Addictions,* 3:65-80. 1968.

Gallup Opinion Index: Report Number 108:20-21. June 1974

Hammond, E.C., Smoking in Relation to the Death Rates of 1 Million Men an Women. *Natl. Cancer Inst. Monograph Number* 19 :12-204. 1966.

Hammnd, E.C., Garfinkel, L., Seidmn, H. and Lew, E.A.: Tar and Nicotine Content of Cigarette smoke in Relation to Death Rates. *Environ. Res.,* 12:263-274. 1976.

Hammond, E.C. and Selikoff, I.J.: Relation of Cigarette Smoking to Risk of Death of Asbestosis Disease Among Insulation Workers in the U.S. *Intern. Agency Res. Cancer Sci. Publ.* 8:312-317. 1973.

Hennekins, C., Drolette, M.E., Jesse, M.J., Davies, J.E. and Hutchison, G.B.: Coffee Drinking and Death due to Coronary Heart Disease. *New England J. Med.,* 12:633-636. 1976.

Hoffmann, D., Schmeltz, I., Hecht, S.S. and Wynder, E.L.: Chemical Studies cm Tobacco Stroke. XXXIX; On the Identification of Carcinogens, Promoters, and Cocarcinogens. Pages 123-146 in *Smoking and Health. I; Modifying the Risk for the Smoker:* DHEW Publ. No. (NIH) 76-122, 1976.

Hoffmann, D. and Wynder. E.L.: Chamber Development and Aerosol Dispersion. Pages 173-191 in *Inhalation Carcinogenesis:* Hanna, M.G., Jr. (ed.). Washington, D.C.: U.S.A.E.C.. 1970.

Hoffmann, D. and Wynder, E.L.: Environmental Respiratory Carcinogenesis. Pages 324-365 in *Chemical Carcinogens:* Searle, C.E. (ed.). New York: Acs Monograph Series 173. 1976.

Hoffmann D. and Wynder, E.L.: Smoking and Occupational Cancers. *Prev. Med.,* 5:245-261. 1976.

Jick, H., Miettinen, O.S., Neff, R.K., Shapiro, S., Heinomn, O.P. and Slone, D.: Coffee and Myocardidal Infarction. *New England J. Med.,* 289:63-67. 1973.

Keys, A. (ed.): Coronary Heart Disease in Seven Countries. *Circulation,* 41 (Supplement 1): 211 pages. 1970.

Klatsky, A.L., Friedman, G.D. and Siegelaub, A.B.: Coffee Drinking Prior *to* Acute Myocardial Infarction. *J.A.M.A.* 226-540-543. 1973.

Kristein, M.: Econmic Issues in Prevention. *Prev. Med.,* 6:252-264. 1977.

Lyon, J.L., Klauber, M.R., Gardner, J.W. and smart, C.R.: Cancer Incidence in Mormons and Non-Mormons in Utah, 1966-1970. *New England J. Med.,* 294:129-133. 1976.

Moore, c.: Cigarette Smoking and Cancer of the Month, Pharynx, and Larynx. *J.A.M.A.* 218:553-558. 1971.

Moore, c.: Smoking and Cancer of the Mouth, Pharynx, and Larynx. *J.A.M.A.* 191:104-110. 1965.

National *Clearinghouse* for Smoking and Health: *Adult Use of Tobacco; 1970* Washington, D.C.: U.S. Depart. of Health, Education, and Welfare. 1973.

Paul, O.: Stimulants and Coronaries. *Postgrad. Med.,* 44:196. 1968.

Phillips, R.L.: Role of Life-style and Dietary Habits in Risk of Cancer Among Seventh-Day Adventists. *Cancer Research,* 35:3513-3522. 1975.

Rose, G.: *Smoking and* Cardiovascular Disease. *Am. Heart J.,* 85:838-840. 1973.

Russell, M.A.H.: Cigarette Dependence. I. Nature and Classification. *Brit. Med. J.,* May: 330-331. 1971 (a).

Russell, M.A.H.: Cigarette Smoking. Natural History of a Dependence Disorder. *Brit. J. Med. Psychol.,* 44:1-16. 1971 (b).

Selikoff, I.J., Hammond, E.C. and Churg, J.: Asbestos Exposure; smoking and Neoplasia. *J.A.M.A.*. 204:106-110. 1968.

Shewchuk, L.A., Dubren, R., Burton, D. et al.: Preliminary Observations on an Intervention Program for Heavy smokers. *Intern. J. Additions.* 12: 1977.

Shewchuk, L.A. and Wynder, E.L.: Guidelines on Smoking Cessation Clinics. *Prev. Med.,* 6:130-133. 1977.

Truett, J., cornfield, J., and Kannel, W.B.: A Multivariate Analysis of the Risk of Coronary Heart Disease in Framingharm. *J. Chron. Dis.,* 20:511-524. 1967.

U.S. Department of Health, Education, and Welfare: Teenage Smoking. *National Patterns of Cigarette Smoking,. Ages 12 through 18 in 1972 and 1974* Atlanta, Georgia: Center for Disease Control. 1976 (a).

U.S. Department of Health, Education, and Welfare: *Adult Use of Tobacco,* 1975 Atlanta, Georgia: Center for Disease Control. 1976 (b).

U.S. Public Health Service: *Smoking and Health. Report of the Advisory Committee to the Surgeon General of the Public Health Service.* Public Health Service Bulletin 1103. 1964.

U.S. Public Health Service: The Health Consequences of Smoking. A Report to the Surgeon General, 1971. U.S. Public Health Service Publication No. 71-7513, Washington, D.C. 1971.

Wallgren, H.: Effect of Ethanol on Intra-cellular Respiratory and Cerebral Function. Pages 103-159 in *The Biology of Alcoholism: Vol. I. Biochemistry Kissen,* B. and Begleiter, H.- (eds.): New York: Plenum. 1971.

Wynder, E-L., Brass, I.D.J. and Day, E.: A Study of Environmental Factors in Cancer of the Larynx. *Cancer,* 9:86-110. 1956.

Wynder, E.L., Covey, L.S., Mabuchi K. & Mushinski, M.H.: Environmental Factors in Cancer of the Larynx: A Second Look. *Cancer,* 38:1591-1601. 1976.

Wynder, E.L. and Goldsmith, R.: The Epidmiolcgy of Bladder Cancer: A Second Look. *Cancer,* in press.

Wynder, E.L. and Hammond, E.C.: A Study of Air Pollution Carcinogenesis. I: Analysis of Epidemiologic Evidence. *Cancer,* 15:79-92. 1962.

Wynder, E.L. and Klein, U.E.: The Possible Pole of Riboflavin Deficiency in Epithelial Neoplasia. I: Bpithelial Changes in Mice in Simple Deficiency. *Cancer,* 18:167-180. 1965.

Wynder, E.L., Isron, F.R., and Bross, I.J.: Cancer and Coronary Artery Disease Among Seventh-Day Adventists. *Cancer,* 12:1016-1028.

Wynder, E.L. and Mushinski, M-H.: Epideniology of Tobacco Usage Among a Large Hospital-based Control population. in preparation.

Yudkin, J. and Roddy, J.: Levels of Dietary Sucrose in Patients with Occlusive Atherosclerotic Disease. *The Lancet,* II:1-8. 1964.

AUTHOR

Ernst L. Wynder, M.D.

The Naylor Dana Institute for Disease Prevention and the Health Maintenenace Institute, The American Health Foundation, 1370 Avenue of the Americas, New York, N.Y. 10019.

Supported in part by NCI Contract Number SHP-74-106 and CA 012376-05.

DISCUSSION OF DR. WYNDER'S PAPER

The first question directed to Dr. Wynder concerned specific agents in cigarette smoking and their relationship to specific disorders, one implication being the impact of these relationships for the development of a safer cigarette. While stressing the complexity of the question, Dr. Wynder stated that cancers of the mouth, larynx and esophagus are probably solely related to the contact carcinogens in the particulate phase; that cancers of the bladder and pancreas could conceivably be related to volatile nitrosamines in a gas phase; that in the area of cardiovascular disease, nicotine and carbon monoxide have been implicated with arteriosclerosis and sudden death although there are differing opinions; and that we know very little about the role of specific cigarette components and emphysema. Is the causal agent in the particulate or gas phase? Are the gaseous components that relate to the celiatoxic effects or to the effects of degrading mucous important? There are few human studies done; yet it is very important to determine, for example, whether a specific reduction of celiatoxic components would reduce the incidence of emphysema.

Dr. Wynder was asked about a paper in his recently edited book from the World Health Conference in which Dr. Ward, from England, states that coronary mortality decreases substantially if the smoker exercises, concluding that this may be due to the reduction in the half life of carboxyhemoglobin. His response was that the epidemiological problem in such studies is that exercise is so closely interlinked with other risk factors that causal attribution is impoysible . These studies would have to be expanded significantly for further definition.

Questioned about the different success characteristics of various cessation programs, particularly considering the success of the "MR. FIT' Program (Multiple Risk Factor Intervention Trial), Dr. Wynder stressed the importance of commitment to any program by staff, including zealous follow-up in the form of calling people at home, getting their family involved and being persistent. While this costs more, he feels the cost effectiveness warrants the initial monetary outlay. Cost is a major consideration for any program, so much so in his own efforts at the American Health Foundation (AHF), that he has established a Division of Health Economics. Dr. Wynder stressed that a prevention program should not be directed solely to smoking, since cost effectiveness is maximized when blood pressure, alcohol consumption and weight control are also considered. Indeed, he claims that these behaviors are synergistic. For example, AHF is finding that success in reducing someone's alcohol consumption by preventive intervention increases that person's performance in a smoking. cessation program.

Dr. Wynder's Health Passport strategy was praised for the implication that children using it would become more interested in their own health care and at an early age. The question was raised whether this approach included an experimental control. In the case of making, for example, could it be stated with any certainty that these children are or will smoke less than a matched control group? Without hard evidence such as control group differences it would be difficult to

convince a large city school system, such as Los Angeles, to foster preventive health programs for cancer or heart disease. The typical response from these school systems is that the problems they are concerned with are here and now - drug abuse, venereal disease and crime - not problems which won't reveal themselves potentially for years. Dr. Wynder replied that the AHP, through its program in a predominantly black New York school. is finding once again the synergistic effect. When students become interested in their health using the Health Passport, the principal notes increased compliance to school regulations. There is 'a ripple effect; it is anticipated that the ripple will touch on other health and sociological behaviors.

It is difficult to document all this scientifically. The end points for success will take years to measure and, of course, measuring will cost money. But the theoretical principles underlying the Health Passport intervention appear sound. In the AHP study design for this program some schools are only screening and no intervention occurs. At other schools interventions will be carried out in varying degrees. In that way, the study can show to what extent intervention is successful.

Joseph W. Cullen, Ph.D.

Smoking Behavioral Factors as Predictors of Risks

Thomas M. Vogt, M.D., M.P.H.

The ability to detect an epidemiologic association is related to the accuracy with which the dependent and independent variables can be assessed. Literally hundreds of papers have been written over the past three decades, in which cigarette smoking has been associated with various disease outcomes. The vast majority of these papers have measured exposure by interview and/or by questionnaire. Very little has been written concerning the accuracy of these smoking assessment techniques. Since the way in which a smoker responds to questions about his smoking behavior is itself a smoking related behavioral variable, it would be very useful to have other methods of validating these responses.

In recent years a technology has been developed which permits a biochemical measure of tobacco exposure through the analysis of blood or urinary nicotine (Russell and Feyerabend, 1975), plasma thiocyanate (Bark and Higson, 1963; Butts et al. 1374; Denson et al. 1967; Vogt et al. 1977), blood carboxyheroglobin (Ringold et al. 1962; Russell et al. 1973) or expired air carbon nonoxide levels (Aronow et al. 1971; Robinson et al. 1975; Russell et al. 1973, Vogt et al. 1977). Although each of these tests has its own sources of error, they differ from questionnaire errors in being less subject to conscious or unconscious manipulation by the individual.

In this report an extensive smoking questionnaire-interview is compared with two of these biochemical measures of exposure. This is a combination of material presented at the AHA Council on Epidemiology meeting in New Orleans in February 1976 and of additional data presented at the Seattle meeting of the Society for Epidemiologic Research in June 1977. Plasma thiocyanate is elevated in smokers as a consequence of the trace amounts of cyanide found in tobacco. The biologic half-life of thiocyanate is 10-14 days (Pettigrew and Fell, 1972). Expired air carbon monoxide is elevated in smokers because of the high concentrations of carbon monoxide in cigarette smoke which are converted to carboxyhemoglobin after inhalation. Carboxyhemoglobin may be directly analyzed in blood samples, but it is technically simpler and less expensive to analyze expired air carbon

monoxide Which is directly proportional to the blood carboxyhemoglobin levels (Cohen et al. 1971; Rea et al. 1973; Ringold et al. 1962). Half-life of the carboxyhemoglobin is 3-4 hours. Because the findings have important implications for the study of smoking behavior it was elected to include them in this monograph.

The subjects in this study were males aged 35-57 enrolled in the San Francisco clinic of the Multiple Risk Factor Intervention Trial. One hundred and forty-two persons received both expired air carbon nonoxide and plasma thiocyanate determinations at the time of entry into the study. Ninety-eight of these were snokers and 44 Were non-stokers by questionnaire. Persons Who Smoked only pipes, cigars or cigarillos are excluded from this analysis.

Each man filled out a smoking questionnaire in an interview with a clinic staff member. This questionnaire consisted of many items, 17 of Which are discussed here. In addition, a second smoking questionnaire was completed a short time later during the same clinic visit by the staff member administering the expired air carbon monoxide test. Results from this second questionnaire are included only to the extent of comparing response to the question:

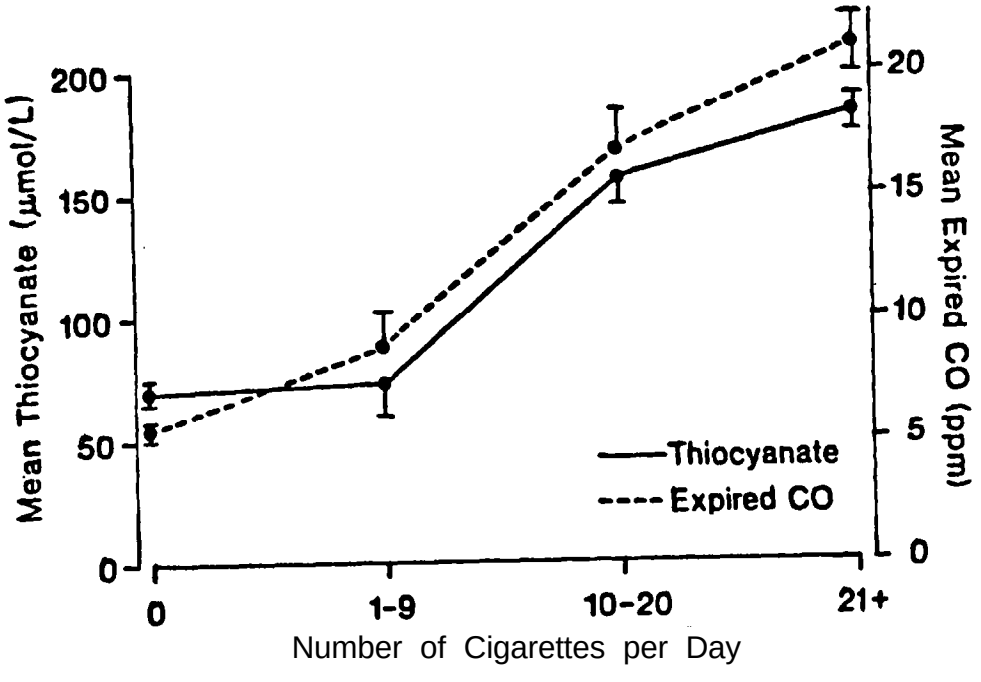
How many cigarettes do you usually smoke per day?

When it is asked of current smokers on two different occasions. Twenty-two percent of persons gave different answers on the two questionnaires given about an hour apart. Three persons denied smoking on one of these questionnaires and admitted it on the other. One person said he smoked 50 cigarettes per day on one questionnaire and 2 cigarettes per day on the other. The digit bias seen in questionnaire responses is evident from the fact that half the smokers report smoking 20, 30 or 40 cigarettes per day. This information confirms the fact that the questionnaires suffer problems of reliability and repeatability. Initially we used the data from these tests to separate smokers from non-smokers.

Figure 1 sumnarizes these results. We found that plasma thiocyanate and expired air carbon monoxide levels exhibited a dose-response relationship With reported number of cigarettes smoked.

In addition, we found that most stokers can be classified by either biochemical test alone into smoking and non-smoking groups, and that the two tests together provide almost 99% agreement With the questionnaire in classification of non-marginal smokers. We identified a category of marginal smokers

FIGURE 1


Mean Thiocyanate (μmol/L)
200
150
100
50
0
Mean Expired CO (ppm)
20
15
10
5
0
Thiocyanate
Expired CO
0
1-9
10-20
21+
Number of Cigarettes per Day

THIOCYANATE vs EXPIRED CO IN NON SMOKERS AND ALL CIGARETTE SMOKERS

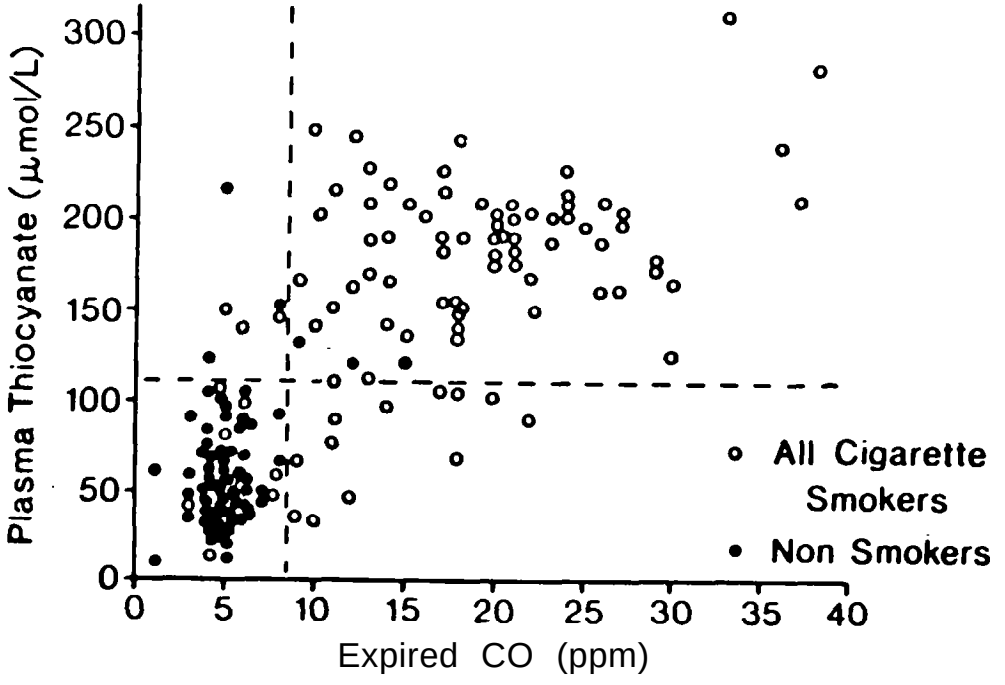


FIGURE 2

THIOCYANATE vs EXPIRED CO IN NON SMOKERS
AND "TYPICAL" CIGARETTE SMOKERS

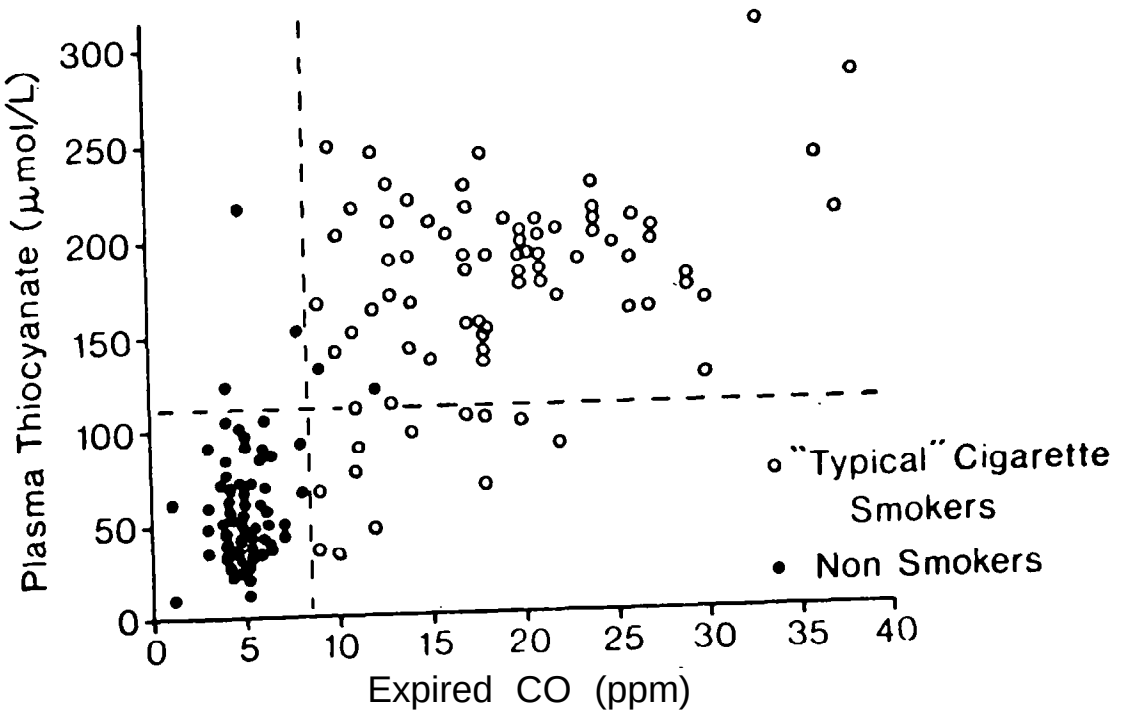


FIGURE 3

THIOCYANATE vs CO IN "ATYPICAL" CIGARETTE SMOKERS

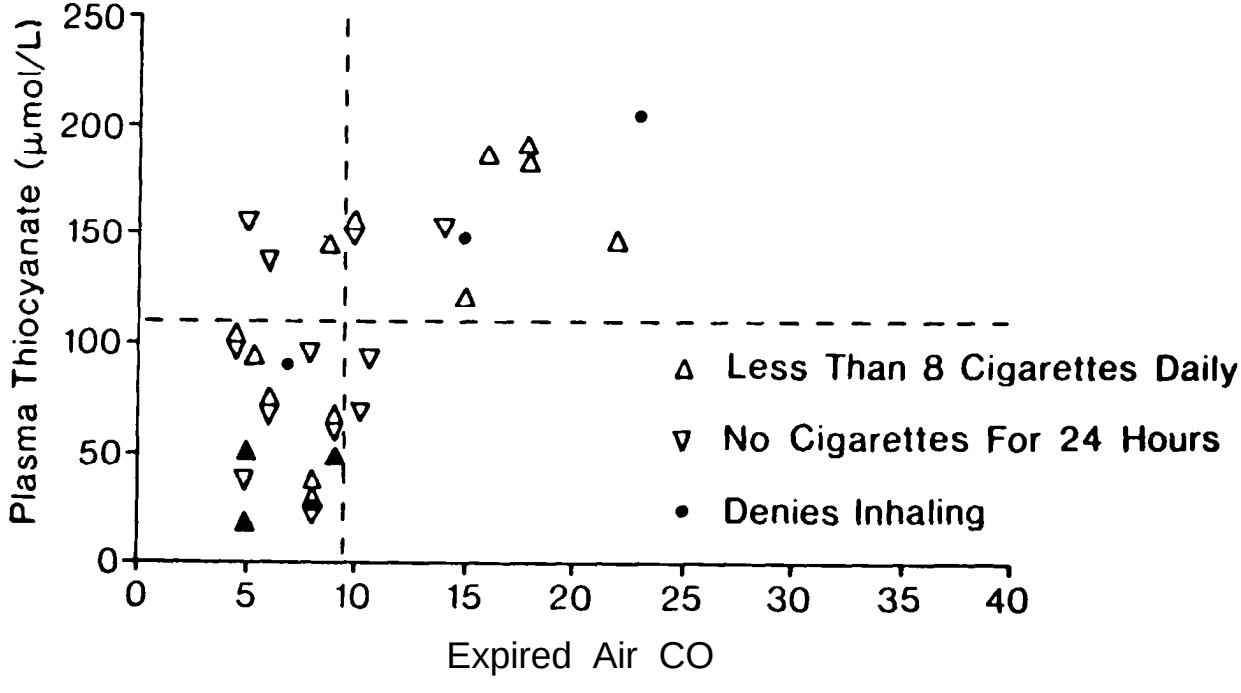


FIGURE 4

TABLE 1

Product-moment correlation matrix for SCN, CO and 5 questionnaire variables for the 98 smokers

	4% Started Smoking	Cig/Day	Highest No. Cig/Day	Time Since Last Cig	SCN	co	SCN+CO
Age of Smoker	-.051	-.130	-.068	.200	-.198	-.196	-.220
Age Started Smoking		-.163	-.095	.038	-.197	-.052	-.121
Cigarettes Per Day			.732	-.294	,483	.491	.551
Highest No. Cigarettes Per Day				-.293	.395	.355	.392
Time Since Last Cigarette					-.244	-.405	-.372
SCN						.573	.880
co							.893

TABLE 2

Stepwise multiple regression for all 17 questionnaire variables with the SCN+CO index as the dependent variable. Smokers only. Questionnaire items are listed by order of entry into the regression equation. R^2 = the proportion of the SCN+CO index variance explained by all questionnaire items entered up to and including a given variable.

	Questionnaire Variable	R^2
1.	Cigarettes per day	.244
2.	Time elapsed since last smoking	.308
3.	Longest time smoker ever stopped smoking	.370
4.	Frequency of inhalation	.397
5.	Average amount of each cigarette left unsmoked	.427
6.	Use of cigarillos plus cigarettes	.451
7.	Use of pipes plus cigarettes	.461
8.	Time since a smoker last quit smoking	.471
9.	Nicotine content of usual brand	.477
10.	Tar content of usual brand	.484
11.	Age of smoker	.490
12.	Use of cigars plus cigarettes	.495
13.	Depth of inhalation	.501
14.	Highest number of cigarettes smoked per day in the past	.503
15.	Amount of each cigarette left unburned	.505
16.	Use of filter cigarettes	.505
17.	Age at which smoking was started	.505

on the basis of their questionnaire responses (persons who deny inhaling and/or who smoke less than 8 cigarettes per day by questionnaire), and found that their biochemical indices were highly scattered. Sane had CO and SCN levels as high as heavy smokers, others had profiles similar to those of non-smokers.

Figure 2 shows the relation between SCN and expired air CO for all persons tested. The dichotanization is good, but there are false negatives - persons who say they smoke, but who fall in the non-smoking for both tests. Removing the marginal smokers, (Figure 3) however, eliminates all of these false negatives.

In Figure 4 these marginal smokers are viewed separately.

Their levels of exposure are highly variable, but the CO and SCN tests consistently agree on their relative exposures. These data indicate that the group of persons who think they don't inhale and/or who smoke < 10 cigarettes per day is really a composite of persons whose exposure is minimal and a second group whose exposure is similar to that of heavier smokers. Our experience suggests that in general, those with less exposure have always smoked marginally, while those with higher exposure were heavier smokers who have cut down.

Nearly all "errors" in categorization involved persons with marginal ("atypical") smoking histories. If this group is removed from the analysis, the remaining smokers are identified by the two tests with an accuracy of 98%. The "atypical" smokers had unpredictable CD and SCN results ranging from non-smoking to heavy exposure levels. It is inferred that while many "atypical" smokers may be at little or no increased risk of disease, some individuals (identifiable only by biochemical testing) may have the same risk as conventional heavy smokers. The discovery of a group of persons whose tobacco exposure is not adequately categorized by smoking history is an important outcome of CO and SCN testing.

Following this analysis multiple regression and analysis of covariance were used to explore the relationship between the individual questionnaire items and the results of the bio-chemical measures of exposure.

Table 1 is a matrix of product-moment correlation coefficients among the five questionnaire variables for the 98 subjects who reported that they smoke cigarettes. The SCN+CO index is a single variable calculated in the following way: The values of SCN and COD were normalized by subtracting their mean values and dividing by the standard deviation to form the first principal component. This summary variable explains 80% of the total variation in SCN and CD measurements and reflects in one number the joint effects of the two variables. SCN and CO levels are more highly correlated with each other (r = .57) than with the reported number of cigarettes per day, (r = .48 and .49 respectively)

and the SCN+OO index is more highly correlated with reported smoking frequency than is either test alone. SCN levels are directly correlated with the highest number of cigarettes smoked per day in the past and inversely related to the age of the smoker, the age at which smoking was started and the time since last smoking. Expired CO) is also directly related to the highest number of cigarettes smoked per day in the past and inversely correlated with time since last smoking and with age.

In the multiple regression analysis the time elapsed since last smoking contributes to the co regression because the biologic half-life of the carboxyhenoglobin is only 3-4 hours. The multiple regression of SCN with its half-life of 10 to 14 days is not appreciably affected by the time since last smoking, and the relatively strong correlation of -.244 is presumably due to the association between time since last smoking and the number of cigarettes smoked per day. The overall multiple R for oo is as great as that for SCN suggesting that the time since last smoking does not substantially confound the oo data. Neither SCN and oo nor the combined SCN+CO index are correlated appreciably with the tar or nicotine content of the usual brand smoked.

Table 2 presents the results of the multiple regression analysis used to evaluate the separate contribution of each questionnaire variable in predicting the SCN+CO index. The overall squared multiple correlation coefficients demonstrate that all questionnaire variables combined account for 42% of the variation in SCN and co and for 50% of the variability of the SCN+co index. The reported number of cigarettes smoked per day accounts for more than half of the multiple R . Only two other variables add significant information to the regression - the time elapsed since last smoking (6%) and the longest time the smoker has ever quit smoking (6%). The remaining 14 variables together add another 1%. With a larger sample size the contribution of some of these might become statistically significant, but it is not likely their biologic importance would be great. The contribution of cigars (n = 16), pipes (n = 7) and cigarillos (n = 7) is not interpretable from these data due to the fact that so few persons used them in addition to their cigarettes. It is inferred that measures of dosage such as reported depth of inhalation may be related to disease chiefly through their association with smoking frequency. The validity of this inference, however, must be tempered by the absence of empirical evidence to date linking SCN and CD to tobacco-associated diseases, although it has been well demonstrated that these tests do measure acute exposure. Prospective studies are needed to validate their use of dosage indicators.

An analysis of covariance was performed to explore the relationship that each of the variables showed with the biochemical measures of exposure. CD and SCN were designated as dependent variables measured at different levels of an independent variable, in this case the longest time a smoker has quit in the past.

Adjustment for reported number of cigarettes per day and time. since last smoking was accomplished by treating them as concomitant variables. Present smokers who have never stopped smoking have mean CO levels after adjustment for reported smoking frequency that are 40% higher than those of smokers of the same amount who have quit for more than a year in the past. This finding is intriguing and suggests that the degree of success in past efforts to 'quit -- 'a measure of the smoker's bond with his cigarette -- may reflect the dosage of tobacco exposure from each cigarette. An alternative, but less likely explanation, is that persons who have never quit smoking in the past are more likely to under-report the number of cigarettes they smoke. The conclusion is the same in either event -- that most information concerning habitual tobacco exposure is contained in two questions: 1) How many cigarettes do you smoke per day? and, 2) What is the longest period you ever quit smoking? In contrast, reported depth of inhalation is significantly associated with CO and SCN levels before adjustment. However, after adjusting for number of cigarettes per day and the time since last smoking the statistical significance of the relationship is lost. This suggests that reports of an association between questionnaire assessed depth of inhalation and disease may be spurious and due to the fact that persons who smoke more cigarettes tend to report deeper inhalation. This does not necessarily deny an effect of depth of inhalation so much as it suggests that individuals are not very good at estimating their own actual inhalation patterns. Similar analyses for other questionnaire variables were performed. No significant associations were found between any of these questions and the biochemical measures of exposure after adjustment for smoking frequency and recency, confirming the multiple regression findings.

The argument presented thus far suffers from a circular reasoning problem; the questionnaire responses that have been used to validate the biochemical indices may themselves be suspect. The validity of the biochemical tests as measures of tobacco exposure is supported by the nearly perfect way that the tests separate smokers from non-smokers.

In addition, the 17 questionnaire variables explain only half of the variability in SCN and CO levels. The remaining variation may result from measurement error, non-tobacco sources of exposure to SCN and CO, and/or biologic variance. Measurement error is small for both tests; Co results are repeatable with a correlation coefficient of .97 and the SCN levels in pooled human sera in our laboratory have a standard deviation equal to only .83umol/l. The environment contains other sources of SCN (Bark and Higson, 1963; Denson et al. 1967) and CO (Goldsmith, 1968) but the clear separation of non-smokers from smokers suggests that ambient sources of exposure do not substantially distort the SCN and CO levels in the population sample tested.

The CO and Sa determinations are inexpensive and objective. The SCN determinations can be carried out unobtrusively in any study for which serum or plasma is being obtained. The CO reading is obtained non-invasively with a portable instrument that provides immediate results. The findings of this study suggest that future work with these techniques might considerably refine our understanding of the relation between tobacco exposure and disease by allowing the identification of specific individuals whose risk of tobacco-associated disease is much higher than that of other smokers due to the high degree of exposure they receive from their tobacco use. It also suggests that smokers who cannot or will not quit might be trained to smoke in less harmful ways by using the CO analyzer as a feedback device. At any rate, these data indicate that some of our basic assumptions about tobacco and disease - such as the relation between reported depth of inhalation and disease outcome - are deserving of conscientious re- examination.

REFERENCES

Aronow, W.S., Dendinger,,B.S. and Rokaw, S.N.: Heart Rate and Carbon Monoxide Level After Smoking High-, Low-, and Non-nicotine Cigarettes. A Study in Male Patients with Angina Pectoris. *Ann. Int. Med.,* 74:697-702, 1971.

Bark, L.S. and Higson, H.G.: A Review of Methods Available for the Detection and Determination of Small Amounts of Cyanide. *Analyst 88:751-760,* 1963.

Butts, W.S., Kuehneman, M. and Widdowson, G. : Automated Method for Determining Serum Thiocyanate, to Distinguish Smokers from Non-smokers. *Clin. Chem.,* 20:1344-1348, 1974.

Cohen, S.I., Perkins, N.M., Ury, H.K. and Goldsmith, J.R.: Carbon Monoxide Uptake in Cigarette Smoking. *Arch. Environ. Health,* 22:55-60, 1971.

Denson, P.M., Davidow, B., Bass, H. and Jones, E.: A (Chemical Test for Smoking Exposure. *Arch. Environ. Health,* 14:865-874, 1967.

Goldsmith, J.R.. and Landaw, S.A.: Carbon Monoxide and Human Health. *Science,* 16:1352-1359, 1968.

Pettigrew, A.R. and Fell, G.S.: Simplified Colormetric Determination of Thiocyanate in Biological Fluids, and Its Application to Investigation of the Toxic Amblyopias. *Clin. Chem.,* 18:996-1000, 1972.

Rea, J.N., Tyrer, P.J., Kasap, H.S. and Beresford, S.A.A.: Expired Air Carbon Monoxide, Smoking, and Other Variables. *Br. J. Prev. Soc. Med.,* 27:114-120, 1973.

Ringold, A., Goldsmith, J.R., Helwig, H.I., Finn, R. and Schuette, F. Estimating Recent Carbon Monoxide ‘Exposures. *Arch. Environ. Health,* 5:308-318, 1962.

REFERENCES (Cont)

Robinson, J.C. and Forbes, W.F.: The Role of Carbon Monoxide in Cigarette Smoking. I. Carbon Monoxide Yield from Cigarettes. *Arch. Environ. Health,* 30:425-434, 1975.

Russell, M.A.H., Cole, P.V. and Brown, E.: Absorption by Non-smokers of Carbon Monoxide from Room Air Polluted by Tobacco Smoke. *Lancet,* 1:576-579, 1973.

Russell, M.A.H. and Feyerabend, C.: Blood and Urinary Nicotine in Non-smokers. *Lancet,* 1:175-181, 1975.

Vogt, T.M., Selvin, S., Widdowson, G. and Hulley, S.B.: Expired Air Carbon Monoxide and Serum Thiocyanate as Objective Measures of Cigarette Exposure. *Amer. J. Public Health,* 67:545-549, 1977.

ACKNOWLEDGMENT

The author would like to acknowledge the extensive effort put into this research by his co-investigators Stephen B. Hulley, M.D., M.P.H. and Steve Selvin, Ph.D.

AUTHOR

Thomas M. Vogt, M.D., M.P.H.
Assistant Professor of Epidemiology
School of Public Health
University of California, Los Angeles

DISCUSSION OF DR. VOGT'S PAPER

Dr. Vogt's data showed that smokers maintain stable levels of carbon monoxide and thiocyanate while decreasing the numbers of cigarettes smoked per day. The question was asked whether they could be titrating, that is, inhaling more deeply or switching to cigarettes with less tar, some of which have higher associated outputs of carbon monoxide. Dr. Vogt's response was that while these data are preliminary, he thinks that is what is happening.

A question arose about the wash-out of the degree of inhalation obtained when the amount of cigarettes smoked was adjusted. Could it not be, even in a non-inhaler, and specifically with thiocyanate, that mucousal absorption is a factor? Dr. Vogt supported the need to study titration as a factor. He asserted, however, that the dose-response curves, looking at mean thiocyanate levels, are reliable and that several other investigators have reported similar curves. People who smoke very little have low thiocyanate levels. Resides, in the unadjusted data, the expected association between the depth of inhalation and thiocyanate levels did obtain. He is not aware of any study where there has been an adjustment for cigarettes per day in determining exposure levels. He could only conclude that the data suggested to him that this adjustment washes out the association.

Another possibility is exploring the product of the amount of inhalation times the number of cigarettes. This recommendation led to speculation that saliva contains significantly higher thiocyanate levels than blood or tissue and that it is rapidly absorbed through mucousal tissue. Consequently, people who don't inhale may still get substantial systemic levels of thiocyanate. Dr. Vogt ad&d that the same effect of depth inhalation also washed out for carbon monoxide with covariance analysis.

Final topics related first to side stream smoke then to the amount of inhaled carbon monoxide. To what extent do people who say they do not inhale keep their cigarette close to their nose, thereby achieving the same or at least a partial effect of inhalation? Dr. Vogt acknowledged the importance of the question but did not have data to add any clarification. Finally, he was asked about other factors that may influence thiocyanate levels such as alcoholism, since it has relevant effects on oral enzyme systems. Dr. Vogt referred to recent data collected by his group indicating no discernible association between carbon monoxide or thiocyanate and alcohol consumption. Studies examining whether thiocyanate levels consequent to consumption of cyanide-containing foods (e.g., turnips, cabbages, almonds, etc.) reveal only negligible increases (a few micromoles/liter).

Joseph W. Cullen, Ph.D.

DISCUSSION

Bernard H. Fox, Ph.D.

I have been asked to discuss the preceding presentations formally. My comments will deal briefly with Drs. Schuman's and Wynder's presentations, review some facets of Dr. Vogt's paper: and close with *a* few considerations generally relevant to the conference topic.

In view of Dr. Schuman's usual thoroughness and diligence, it only remains to suggest that his synthesis of data on social phenomena and smoking should and will become an important source for *over*-view or baseline purposes. Such a collection in one place is very valuable. One comment might be in order, however. He remarked that age at start of smoking has declined over the years. He also declared that those who stopped smoking started later than those who continue to smoke. If this still holds true after controlling for amount smoked, we will probably find it much harder in the future to be successful in cessation programs, assuming that there is a cause-effect relationship between age at starting and success in stopping, rather than an association by happenstance. I propose that habit patterns of lower organisms that are learned early are acquired more easily and last longer than those learned later in life. I believe it to be true of humans too. This proposed phenomenon would ride on top of nicotine-dependence phenomena so well researched by Dr. Russell. Two corollary hypotheses are that filter users will turn out to be late starters because, nicotine demand will probably be high in early starters; and early starters will be heaviest smokers. That is why a test of the first proposal should involve controlling for amount smoked.

Dr. Wynder's summary of the possible sources of the smoking habit and of the contribution of smoking to the epidemiology of various diseases leads him to recommend several courses of action. That he is a member of a scant and lonely chorus does not change their intrinsic good sense; and the fact that we and the world at large have been slow to follow up on them only reinforces his logic in making those recommendations here.

Dr. Vogt's paper, presenting new data, draws attention of a different kind, and is perhaps more vulnerable to minor quibbles than the other speakers' summary material, as is the case with most new work.

First, he should be complimented on using the breath-holding technique for collecting carbon monoxide samples. For many years I was involved in measurement of breath alcohol, and in the process of attempting to reduce the variability of the instrument, carried out a test of short and long breath-holding techniques (Fox et al. 1969). Dr. Vogt's breath-holding time corresponds to one of the ones I tested. The diffusion coefficient of CO being larger than that of alcohol, his breath-holding procedure would yield a value even closer to equilibrated gas concentration than I found for alcohol.

Second, his noting of digit bias was valuable. It may be worth estimating the measure of such bias. A similar end-digit preference was found in readers of the normally distributed blood pressure of sane 40,000 gravidae over their pregnancy terms (Friedman et al. 1976). In a distribution of a sample of some 1400 readings, the mean diastolic pressure was close to 70 mm Hg, with a standard deviation of about 10. If the class interval is 10, beginning with the decade, the reading of 70 is overloaded. I calculated that there were 20.5% fewer entries in the frequency of the 60-69 class interval than there would be if the distribution were continuous, and normal. A shift of one unit in the class interval to 61-70 would underload the interval 71-80 by about the same amount. In Dr. Vogt's data about 30 cigarettes per day were smoked on average, with S.D. g 17. While a distribution with class interval of 10 and S.D. of 17 would seem to lead to smaller error of frequency. than one with S.D. of 10, the proportion of digits ending in zero within an interval is greater here than in the blood pressure case (70%, 75%, and 80% respectively in the intervals below, containing, and above the mean) and the distribution is skewed to the right and leptokurtic, with 64% of the cases being contained in those three intervals. Such conditions tend to restore the magnitude of expected error to a value, I estimate, close to the same 20% in the central intervals. The impact of such error could be considerably reduced if analysis were done on intervals whose bounds ended in the digits 4, 5, . The blood pressure data were, in fact, dealt with in this way, thus avoiding some biases that are associated with clustering at the digit zero. I calculated the error of interval frequency where the zero stood in the middle of the interval to be about 4%, which is tolerable. The same should hold here.

Dr. Vogt has used multiple regression and covariance analysis to arrive at estimates of relationship between number of cigarettes smoked per day (cig./day) and levels of carbon monoxide (CO) and thiocyanate (SCN). In these procedures the zero-order regressions used were all linear. However, in using these statistics there

is considerable danger of underestimating the relationship for one set of reasons and overestimating it for another. First, data are available (Hawkins et al., 1976;-Coburn et al., 1965; and Wald et al., 1975) to show that the relation of CO level and cig./day is curvilinear, taking the form of a negatively accelerated exponential rising to an asymptote. The curve tends to flatten out quickly at low CO levels for-active workers and to continue rising to a high CO asymotote for sedentary workers (Hawkins et al.. 1976). We infer' that a coefficient of linear correlation between cig./day and CO level will considerably underestimate the true relationship in the case of active people, where the curve is severe, and where high levels of smoking yield little increase in CO over moderate levels. In the case of sedentary people the curve is closer to linear over a larger range of CO level and cig./day, and a linear correlation will suffer a much smaller underestimation. Since the asymptote depends strongly on the rate and depth of breathing, which are in part functions of activity level, I hypothesize that idiosyncratic differences in rate and depth among people having equal activity -- say, sedentary -- should likewise lead to differences in curvilinearity . In future studies it would be very valuable to record these variables. In any case, because of the exponential form of the curve, a log transform would lead *to* almost linearized data, and the analysis would proceed as before, with smaller underestimation of the multiple regression due to curvilinearity.

I hypothesize the same theoretical rationale for SCN. Since the half-life of SCN is 12-14 days and cyanide uptake with consequent development of SCN proceeds at a relatively constant rate during the day, it is reasonable to suppose that the SCN level does not achieve unlimited growth, and that an asymptotic relationship to cig./day similar to that of CO obtains (Butts et al., 1974). We do not, however, have independent estimates of the levels of this asymptote for many populations or the relationship among factors determining level of SCN such as amount of SCN absorbed, amount reabsorbed-in the gut from $saliva_i$ and the like. Some estimates exist. however (Boyland et al.. 1974). On examining Dr. Vogt's SCN data I found a clear asymptote emerging in the region of 200 umol/l. This is not far from the asymptote derivable from the data of Butts et al., 1974. Moreover, for Vogt's subjects, the level of SCN approached an asymptote faster than the CO data, which were close to linear at levels up to 50 cig./day. The combination (CC + SCN index) has a mild curvature at these levels, so that while a log transform would improve the relation of the index to cig./' day, only a modest amount would be added to the total explained variance for cig./day levels up to 50. In the present sample from the MR FIT program, the mean cig./day were high compared to the average sample of smokers, and from the C0 curve, this sample seems to be made up mostly of low activity people (or they were made less active by the experimental situation). Since the sample contains three smokers whose daily reported consumptions were 60

and one each of 70, 74 and 97, if these four data points showed relatively small CO rise, as I suspect, then they would affect the relationship inordinately, and the log transform might, in fact, improve things more than a little, even for the CO curve, thus making it more important to apply such an adjustment..

Thus far we have pointed to the probable underestimation of multiple correlation values because linear regressions of raw rather than log data were used in the analysis. On the other hand, overestimation is almost certain for multiple correlation values, since the least-squares estimate capitalizes on chance correspondences. Whatever the population correlation p, R is Inflated over p. Formulas for estimating an unbiassed R^2 exist (Olkln et al. 1958). In this case the increased explained variance associated with the addition of 14 variables over a basic three differs little from that which will accrue if all 14 were independent of the outcome. This fact is consistent with Dr. Vogt's suspicion that these variables do not add much to the basic ones. Nevertheless, I think that if certain other information were available some of them might be predictive; e.g., depth of inhalation or frequency of inhalation. A propos of the exponential curve, Fig. 1 for Vogt's sample seems to show considerable departure from the Hawkins curves. Correspondence with those curves becomes substantially greater if some adjustments are made in the figure. First, the zero point is 5 units too far from the center of the l-9 interval. For the CO curve an adjustment leads to almost a linear rise from no smoking to 10-20 cig./day. While the class intervals are irregular (1-9 followed by 10-20), the curve is not affected by the digit bias, which begins beyond 10 cig./day. More Important, however, grouping the remainder of the data values in a category 21+ leads to too steep a rise from 10-20 to that point, which is placed as far from 10-20 as the latter is from l-9. A weighted centroid would place the point at about 43, and the last rise In both curves would be rather flat, if one decided to display the data of 58 out of 98 data values at one data point, a procedure which might be questioned.

Dr. Vogt suggests the CO analyzer as a feedback device for those trying to reduce smoking. I applaud heartily. The need for additional reinforcement to people trying to stop smoking is very Important. But we must be cautious in such an effort, as will be seen from the issues to be raised now.

I would like to address a related matter. Several people have said there is some doubt about the truthfulness of those claiming to have quit in smoking cessation classes. Delarue in Canada (Delarue et al. 1971) showed this doubt to have some grounding, in that 22 out of 107 claimed ex-smokers had carboxyhaemoglobln (COHb) levels above 2%. While there may be found sane nonsmokers with such levels (Russell et al. 1976), Delarue et al. tallied their group. At what point would one say there is little doubt: 3% COHb, 4%? Delarue et al. found 7 cases between 2% and 3%.

Are all of these to be deemed nonsmokers? At this point an SCN test would be valuable, since its half-life is longer. Of the 22 cases, 8 admitted to lying. A similar experience was described by Ohlti et al. 1976. They found 35 out of 109 with raised COHb values. Of 32 contacted, 13 admitted smoking. Half of the 35 had levels above 3% COHb. Of the 19 remaining, 7 refused to come In for a test. The levels of 11 of the remaining 12 were normal on retesting with advance knowledge.

The point is that while we can use a blood, breath or saliva test, knowledge of that test may result In a subject not smoking for the test. Evans showed that some children did not smoke after seeing a saliva analysis on film and having their own saliva sampled. (Evans et al. 1977). OhlLn took blood when the subjects didn't expect It!

But if you are to carry out 6, 12, 1.8 and 24 month follow-ups, the jig is up on the first test at 6 months, when you make the appointment. It would be possible to deceive the subject and say you are looking for something else, but then the Committee on Experimentation on Human Subjects may look askance at the deceit. How shall one manage It? Perhaps the best way is to use a test of a long-lasting residual such as SCN or alkaline phosphatase and tell the subject truthfully that he will be tested. It will be the rare subject who will abstain as much as two weeks in anticipation of the test. He will probably think that a day Is enough. If he asks the duration of a blood sign, one ought to tell him the truth. But then, one or two such who do deceive will not destroy the experiment. As many as 20 or 30 percent deceiving, however, might.

A larger, scale test than Ohlin's with better ascertainment should be carried out; I hope to be able to do this.

Dr. Vogt's circular reasoning problem may not be as severe as he thinks, but there may be a more fundamental Issue to consider. In his first paragraph he suggests biochemical tests to validate questionnaire responses. In such a case the latter are being used as a convenient predictor or measure of the more fundamental criterion, biochemical tests. If so, then the measure cannot validate the criterion, as suggested in the circular reasoning argument. On the other hand, if the criterion is whether a person smokes, then the first paragraph is Inconsistent with such a criterion. Predicting the criterion of separating smokers and nonsmokers is an objective in one part of the study -- Figs. 2 and 3; and predicting the criterion of levels of CO and SCN from questionnaire responses is an objective in another -- the multiple regression and analyses of covariance. If these objectives are identified and addressed as complementary problems, with awareness in each case of predictor and criterion, there is no circularity, provided they are separated. Perhaps one should ask whether there are even more fundamental criteria. They would be identified if we answered the question, what use will be made of a given criterion? For

example, are we interested in CO for its own sake or because It may predict heart disease risk? If so, CO is then an intermediate criterion. Do we want to how how much tar is Inhaled, for cancer related studies? Then number of cigarettes may be Important, and Inhalation becomes a critical variable. Are we interested In heart disease in general? Then CO level or cig./day may not be enough. We may need to add nicotine level, or perhaps substitute It for cig./day.

In any case, Dr. Vogt has demonstrated that answers to his questionnaire do not predict well the levels of CO or SCN, but that he can identify self-reported smokers and nonsmokers rather well from CO and SCN levels in the special situation of a MRFIT sample.

One last item. I followed with interest Dr. Russell's published discussions and his opinion that we need a high nicotine, low CO, low tar cigarette because this will lead to the lowest rate of harm from smoking. I would like to know if a cost-benefit study could be done on this topic (using lives or years lost as the cost factor) and address- the known rates of excess heart disease mortality as well as cancer mortality. This cigarette would be compared with a moderately lower nicotine cigarette having corresponding levels of tar and CO. Here, some people would accommodate over time. The net yield of lives or years saved (lost) would be evaluated in both cases and compared.

REFERENCES

Boyland, E. and Walker, S.A.: Effect of Thiocyanate on Nitrosation of Amines. *Nature*, 248: 601-602, 1974.

Butts, W.C., Kuehnemann, M. and Widdowson, G;M;: Automated Method for Determining Serum Thiocyanate, to Distinguish Smokers from Nonsmokers. *Clin. Chem.*, 20: 1344-1348, 1974.

Coburn, R.F., Forster, R.E. and Kane, P.B.: Considerations of the Physiological Variables that Determine the Blood Carboxyhemoglobin Concentration in Man. *J, Clin. Invest.*, 44: 1899-1910, 1965.

Delarue, N.C. and Moss, G.: *The Toronto Smoking Withdrawl Centre: A Report Prepared for the Department of National Health and Welfare-A Smoking and Health Research Report.* Ottawa, 1971.

Evans, R.I., Rozelle, R.M., Mittelmark, M.B., Hansen, W.B., Bane, A.L. and Havis, J.G.: Deterring the Onset of Smoking in Children: Knowledge of Immediate Physiological Effects and Coping with Peer Pressure, Media Pressure, and Parent Modeling. *J. Appl. Soc. Psychol.*, In press, 1977.

Fox, B.H. and Pelch, A.: Breath-holding, Full-time, and Half-time Exhalation in Breath Alcohol Testing. *Proc., 5th International Conference on Alcohol and* Traffic *Safety,* University of Freiburg, Freiburg, Germany, Part II, 45-51, 1969.

Friedman, E.A. and Fox, B.H.: Evaluation of Criteria. (In) *Progress in Clinical and Biological Research, Vol. 7. Blood Pressure, Edema and Proteinuria in Pregnancy. Friedman,* E.A. et al. (Eds.), Alan R. Liss Inc., New York, 1976.

Hawkins, L.H., Cole, P.V. and Harris, J.R.W.: Smoking Habits and Blood Carbon Monoxide Levels. *Environmental Res.,* 11: 310-318, 1976.

Ohlin, P., Lundh, B. and Westling, H.: Carbon Monoxide Levels and Reported Cessation of Smoking. *Psychopharmacology,* 49: 263-265, 1976.

Olkin, I. and Pratt, J.W.: Unbiased Estimation of Certain Correlation Coefficients. *Ann. Math. Stat.,* 29: 201-211, 1958.

Russell, M.A.H. and Cole, P.V.: Confirmation of Abstinence from smoking. *Br. Med. J. :* 755-756, 25 Sept., 1976.

Wald, N., Howard, S., Smith, P.G. and Bailey, A.: Use of Carboxyhaemoglobin Levels to Predict the Development of Diseases Associated with Cigarette Smoking. *Thorax,* 30: 133-140, 1975.

AUTHOR

Bernard H. Fox, Ph.D.
National Cancer Institute
National Institutes of Health
Bethesda, Maryland 20014

Epidemiology: Session Overview

Joseph W. Cullen, Ph.D.

Many smokers rationalize their habit by pointing to the late demise of some close relative who smoked excessively but lived to be 96. Is there epidemiologic evidence to account for this discrepancy? The answer offered was that there is such evidence which may be explained by some genetic protection. But the fact is that smokers of longer-lived parents have been shown to die earlier than non-smokers. It was also argued, that in all these studies, including the case-control variety, we always contend with interview bias. For example, someone may say his father lived to be very old in spite of his excessive smoking. It is not always possible to determine whether that is true. Or in another instance, one person's interview is colored by the fact that he/she just spit up blood while a second person has been asymptomatic even though he/she has the same disease and is working and coping. These factors may color the interview. While there is a well developed biostatistical technology and it is used effectively, the data being analyzed are only as valid as the validity of the interview. This in turn depends on the validity of self-report. Whether one is involved in ascertaining a smoking history, a history of alcohol consumption, venereal disease, coffee and saccharine intake, etc., there is always this limitation of the classical case control study. This deficiency is another reason to applaud the use of biochemical indicators. In fact, a triangular approach of interview-biochemistry-behavioral observation may be the best approach.

An exchange between Drs. Vogt and Fox relating to the latter's comments on Dr. Vogt 's paper pointed to the variance in the biochemical indices not explained by the questionnaire. Perhaps Dr. FOX'S suggestion will increase the correlations, but due to the questionnaire's inaccuracy, it did not seem conceivable that they could ever get as high as .8 or .9. Even if the indices were precise measures of exposure, which they are not, you still would not see 100% correlation with the questionnaire because it is not a 100% predictor of exposure. It is still not known what percent of the unexplained variation represents error in either analysis or the biochemical measure and what represents truth in the biochemical measure which is not put into the questionnaire. Dr. Fox acknowledged this and reasserted that a perfect correlation is not what is expected nor what he implied.

However, he suggested that a high relationship was not found, partly because a number of things have not been measured: such as, intervals between the last cigarette and carbon monoxide determination, rate of occupational and other activity, and rate of breathing. If these three items are joined with the questionnaire items, a better prediction might be found since they have been found to be critical in controlling levels of carbon monoxide. The choice of criterion and predictors is arbitrary. In one part of the study you are setting up carbon monoxide and thiocyanate levels as outcome criteria. Just why should they be the important ones? Why not nicotine level? cotinine level? nicotine-l'-N-oxide level ? The number of cigarettes should not necessarily be discarded simply because they don't reflect wholly the carbon monoxide level. This is important because in a smoking cessation program one is not directing the procedures to the inside of the man; one is directing them to his smoking behavior. If so, then cigarettes- smoked. is one reasonable criterion for this prediction situation. In other words, the' best choice of variables of measurement techniques, and of analysis depends on the objective.

The question arose again about what motivates individuals coming to smoking clinics versus those, for example, entering the "MR. FIT*' program. Since the latter program involves much more than smoking (or perhaps not smoking at all since clients may be motivated to enter the program for the heart risk benefits, etc.) are the programs comparable?' Comparability is needed to make cost comparisons. Dr. Wynder addressed this question by describing the various types of individuals he has encountered in the New York City area. One type of individual he sees coming to the AHF is part of the labor union force and an expressed motivation of these individuals was merely to get several days off per year regardless of the program. In the case of 'MR. FIT", even after advertising in a popular New York magazine, virtually no one would come in, even though a free preventive health program was offered. By advertising in a different magazine, one which is replete with advertisements of free offers, thereby attracting a different population of readers, some people came in. They were more educated; most were pressured by their wives to come. The point is that motivation is the key. Although the AHF is now getting 70% participation in its programs, that is not enough. The answer is not in more esoteric research on motivation or another aspect of this problem, but rather getting into the field like the barefoot Chinese physicians and 'beating the bushes". There is too much emphasis on statistical manipulation and "ivory tower" conceptualization. Not enough emphasis is given to going to the work place and impressing the worker with direct contact.

It was pointed out in response to the differences between people, that the people coming to the regular smoking clinics are hard core smokers. more desperate, and having more difficulties quitting smoking. Moreover, within the 'MR. FIT" study there are basically two groups of people: one is an intervention group, and the other is the group that goes back to their own doctor. the latter are the "regular care people". They will have higher quitting rates and they are not in the study.

SECTION II: ETIOLOGY

Biological Factors Underlying the Smoking Habit

Murray E. Jarvik, M.D., Ph.D.

INTRODUCTION

Cigarette smoking is one of the most popular and persistent habits of our modem age. Yet, despite the fact that hundreds of millions of people have chewed, sniffed and inhaled tobacco products for the past 500 years, remarkably little is known about the true reasons why people pursue the substance with such fervor. We how a great deal more about the reinforcing properties of other substances which are used and abused in society than we do about tobacco. For example, opium, coca and wine have been studied extensively and we have a pretty good idea of why people take them. Users are seeking optimal levels of a single chemical substance in each of these vegetable products, namely: morphine, cocaine and ethanol. The wonders of modem chemistry have made synthetic products such as amphetamine and barbiturates available. Most important of all, animal models have been devised to demonstrate that these chemicals are self-administered when animals have the option of taking them intravenously (Kumar and Stolemmn, 1977; Thompson and Pickens, 1971). When an animal self-administers a drug it enables us to explore the brain and determine the mechanism of reinforcement.

By contrast, the picture is not so clear for tobacco. It has not been possible to develop reliable animal models of self-administration of tobacco products, and this limits our psychobiological investigations. We don't even know for sure what the principal reinforcing ingredient in tobacco is, though many of us have expressed strong faith that it is nicotine. In this paper we will examine some of the evidence for and against the view that nicotine is the prime incentive for smoking.

EFFECT OF CIGARETTE SMOKING ON ORGAN SYSTEMS

Let us first briefly review some of the many physiological effects produced by smoking cigarettes (Health Consequences of Smoking, 1964; Larson and Silvette, 1975) and then see if we can tell which

of these are related to the pleasure produced by this habit. The first stimuli that the smoker perceives from a cigarette are its visual characteristics, and upon lighting it he can see the smoke, feel the warmth and smell the aroma. Upon inhaling the smoke he feels it in his mouth and some of it is deposited on his buccal and nasal mucous membranes where the nicotine is rapidly absorbed (though less so if the pH is acid than alkaline) (Armitage, 1974). Tars which may have been deposited upon his fingers are also deposited on his teeth and mucous membranes. Taste and smell are stimulated and so are pain receptors in the pharynx, larynx, trachea and bronchii.

As the smoker inhales the smoke into his lungs the respiratory epithelium is coated with fine particles and droplets from the smoke. The nicotine and carbon monoxide paralyze the cilia (Rylander, 1972) but increase the secretion of mucous, and coughing is likely to occur. All of these stimuli, so unpleasant to the novice, come to be reinforcing to the habitual smoker and are incorporated as part of the "feel" of smoking.

The inhaling smoker takes a deep breath of smoke to enable the smoke particles to reach his alveoli and he may hold this breath for several seconds. The duration of inspiration determines how much of the nicotine and other constituents will be absorbed into the lungs and vascular circulation during each breath. Then he exhales the remaining smoke, some of it through his nose, some of it through his mouth. In the meanwhile, his face and head are surrounded by a halo of smoke. The main purpose of all this activity appears to be to get nicotine into the blood and particularly into the brain as 'quickly as possible.

Of course, the entry of smoke into the lungs is only the beginning of the effects that smokers will experience either acutely or chronically. The acute actions of inhaling the smoke from a cigarette are largely attributable to the alkaloid, nicotine. The direct effects of this drug are to stimulate nicotinic cholinergic receptors, and its indirect effects are largely due to release of catecholamines. Its peripheral effects are largely due to the release of epinephrine from the adrenal medulla and-may be prevented by adrenergic blocking agents. (Cryer et al. 1976).

The chronic effects of smoking on the skin, mucous membranes, respiratory system and other systems are all undesirable, but their onset is so subtle and insidious that the smoker is not usually aware of their existence or he has habituated to them (for example to his cough or hoarseness). If they were more acute in onset they would be more punishing and therefore tend to inhibit his smoking. But their slow onset does not favor the establishment of a conditioned aversive association with smoking. The punishment produced by the onset of insidious disease such as emphysema or thromboangiitis obliterans does not seem to diminish the response of smoking. From my own observations an acute myocardial infarct usually results in prompt cessation of smoking. There should be more research on the influence of medical catastrophes upon smoking behavior.

An example of an undesirable insidious effect of smoking is skin wrinkling. Daniel1 (197l) demonstrated that smokers have significantly more wrinkling of the skin than non-smokers. Although other factors such as actinic rays and genetic susceptibility play a role, some as yet unidentified substance in cigarette smoke either attacks the skin directly from the air or produces its 'effect via the blood stream. If young smokers could be threatened effectively with the association between smoking and wrinkling it might help to deter them from pursuing this habit.

The teenage smoker, in contrast to the middle aged chronic smoker, experiences primarily the pleasant effects of smoking. In fact, impairment of pulmonary function can be demonstrated in teenage smokers but only by rather subtle tests (Seely, 1971). By middle age and older these differences are more pronounced (Editorial, Br. Med. J., 1975). The pathological changes induced by smoking in various parts of the body slowly accumulate over time measured in years and decades and if death occurs in middle age or later these can be fairly easily identified by the pathologist. Every child should know that smokers' lungs are darker than non-smokers' because smoke residues accumulate there.

Atherosclerotic changes are found more frequently in the cardiovascular system of smokers than of non-smokers. We have already heard that cigarette smoking is an important risk factor in cardiovascular disease, but the exact mechanism whereby it produces its deleterious actions has not been identified. It seems to be an interaction between a number of factors including an increase in platelet adhesiveness (Mustard, 1972), anoxemia following carboxyhemoglobin formation by carbon monoxide, and the effects of catecholamine release by nicotine.

The gastrointestinal system is adversely affected by smoking. Smoking seems to play a role in the genesis of periodontal disease, peptic ulcer (Alp, 1970), and impairment of intestinal secretion (Bynum, 1972). Smoking can cause difficulties with pregnancy and is related to fetal mortality and impairment of fetal growth (Health Consequences of Smoking, 1973).

The mortality ratio is raised in smokers for a great variety of diseases including cancer of the lung, larynx, oral cavity, esophagus, bladder, kidney, stomach and prostate. This should not be too surprising since the carcinogens from cigarette smoke are blood borne, though they might be expected to concentrate in the oral cavity and respiratory system and in urine. The mortality ratio is increased in smokers for bronchitis and emphysema, stomach and duodenal ulcers, cirrhosis of the liver, coronary artery disease, hypertensive heart disease, general arteriosclerosis, cerebrovascular disease, influenza, pneumonia, and nephritis. Even deaths from accidents, suicides and other forms of violence were higher in smokers than non-smokers. of course, smoking may not be the cause of the fatal condition but may be fortuitously related to it. But the association with increased

risk certainly bears looking into. (Smoking and its effects on health, 1975).

It is well known that weight gain frequently accompanies cessation of smoking though it is generally small in magnitude (WHO. 1975). Smokers tend to be lighter than non-smoke& (Khosla and Lowe. 1971: Gordon et al. 1975) and it has been proposed that smoking impairs utilization of 'food (WHO, 1975). The slimming effect of smoking may be considered a virtue by those seeking a svelte appearance but the cost seems disproportionately high for the slight benefit.

SOURCES OF REINFORCEMENT IN CIGARETTE SMOKING

Although it seems difficult for a psychopharmacologist to ignore the possibility, indeed the probability or certainty that the chemical composition of cigarette smoke is of vital importance in explaining smoking behavior, there are behavioral scientists who totally ignore chemistry. They focus instead upon the fact that smoking is initiated by peer pressure and some have expressed the view that oral and manual satisfaction is all that is necessary to maintain the habit. The symbolic significance of the cigarette may become quite complex in psychoanalytic terms. It is probably wrong to go to the opposite extreme and deny the importance of psychological factors in the maintenance of the smoking habit, but there is much direct evidence that cigarette smoking necessarily involves tobacco. Cigarettes made of non-tobacco materials such as lettuce or Cubebs are not popular. The evidence that nicotine is a vital ingredient is somewhat more circumstantial; it can be seen in Fig. that the most popular brands deliver a substantial amount of nicotine.

A pack a day smoker takes more than 50,000 puffs per year and each puff delivers a rich assortment of chemicals into the lungs and bloodstream. Each puff stamps in the habit a little more, and accompanying this effect is the establishment of considerable secondary reinforcement including the sight and smell of cigarettes, the lighting procedure, and the milieu and context of smoking. Smokers tell us that they enjoy a cigarette at the end of a meal, with a cup of coffee or with a cocktail. It would be surprising if chemical factors were not involved in these pleasurable experiences. Also it is not surprising that such an overlearned habit surrounded by secondary reinforcers is difficult to extinguish.

The possible candidates for reinforcing pharmacological agents are shown in Tables I, and II (Schmeltz and Hoffman, 1976). Although nicotine is the most popular suspect for the reinforcing agent in tobacco there are other possibilities. Tar and carbon monoxide are the two most likely contenders so let us consider each of these and then return to nicotine.

TABLE I

Cigarette Smoke: Gas Phase Components (ug/Cigarette*) (35)

Carbon monoxide	13,400
Carbon dioxide	50,600
Ammonia	
Hydrogen cyanide (hydrocyanic acid)	240
Isoprene (2-Me-l, 3 butadiene)	582
Acetaldehyde	770
Acrolein (2-propenal)	84
Toluene	108
N-Nitrosodimethylamine	0.08
N-Nitrosomethylethylamine	0.03
Hydrazine	0.03
Nitrcanethane	0.5
Nitroethane	1.1
Nitrobenzene	
Acetone	578
Benzene	67

* 85 mm non-filter, blended cigarette (U.S.)
** Gas phase portion only (74 pg/Cig. in particulate phase)

TABLE II

Cigarette Smoke: Particulate Phase Components (vg/Cigarette) (35)

(TPM *, wet	31,500
dry	27,900
FTC **	26,100
Nicotine	1,800
Phenol	86.4
o-Creso1	20.4
m- and p-Cresol	49.5
2,4 Dimethylphen	9.0
p-Ethylphenol	18.2
B-Naphthylamine	0.028
N-Nitrosonomicotine	0.14
Carbazole	
N-Methylcarbazole	0.23
Indole	14.
N-Methylindole	0.42
Benz(a)anthracene	0.044
Benzo(a)pyrene	0.025
Fluorene	0.42
Fluoranthene	0.26
Chrysene	0.04
DDD	1.75
DDT	0.77
4,4'-Dichlorostilbene	1.73

* U.S. cigarette, 85 mm, without filter tip, 1968
** TPM=FTC = TPM-H_20-nicotine

Carbon Monoxide

After nicotine the substance in cigarette smoke with the most pronounced acute pharmacological action is carbon monoxide. The CO content of cigarette smoke is considerably higher than Los Angeles smog on the worst day. Cigarette smoke contains more than 2% CO, or 20,000 parts per million (ppm). Los Angeles air on a smoggy day contains 50 or more ppm of CO (South Coast AQMD, 1977). It can be seen in Table III that the-expired breath of a heavy smoker can contain more than 30 ppm of CO and the level of carboxyhemoglobin can be as high as 7% (Goldsmith and Landaw, 1968). Carbon monoxide impairs the oxygen carrying capacity of the blood and may impair functioning of the nervous system. It appears to pose a serious threat both acutely and chronically to the functioning of the cardiovascular system. Indeed it is thought by some (Strong et al. 1969) that the carbon monoxide in cigarette smoke is the culprit responsible for the increased risk of myocardial infarction and stroke in cigarette smokers. The combination of nicotine with its catecholamine releasing properties and carbon monoxide in the blood of smokers constitutes an obvious cardiovascular risk.

TABLE III

Proportion of smokers and median concentrations of expired Co in a population of longshoremen (N-3311). (California State Department of Public Health)

Category	Median concentration (parts per million) of CO measured in expired air	Median percentage of carboxyhemoglobin estimated from regression
Never smoked (23.1)	3.2	1.2
Ex-smoker (12.1)	3.9	1.4
Pipe and/or cigar smoker only (13.4)	5.4	1.7
Cigarette smoker		
Light smoker (half pack or less) (13.0)		
Inhaler	17.1	3.8
Noninhaler	9.0	2.3
Moderate smoker (more than half pack or less than 2 packs (31.3)	27.5	5.9
Noninhaler	14.4	3.6
Heavy smoker (2 packs or more) (7.0)		
Inhaler	32.4	6.8
Noninhaler	25.2	5.6

* Values in parentheses are percentages of study population by smoking pattern.

It is highly unlikely that carbon monoxide is the reinforcing agent in smoking although it may interact with nicotine. Other 'forms of tobacco (snuff and chewing tobacco) have been used through the ages and do not produce carbon monoxide. Furthermore, cigarettes which are low in nicotine (and also in tar) yield amounts of carbon monoxide similar to those of nicotine cigarettes but are not popular. It must be admitted that there has been an increase in the popularity of filter cigarettes over recent years which do yield higher quantities of carbon monoxide. However, no one has shown that carbon monoxide intoxication is pleasurable.

Tar

The other substance in cigarette smoke of extreme importance is the tar. It is well known that this portion of the smoke contains numerous carcinogenic compounds,. Lately there has been interest in an enzyme arylhydrocarbon hydroxylase (AHH) which is isolated from pulmonary macrophages and- from- lymphocytes (Health Conseouences of Smoking, 1975). This enzyme metabolizes carcinogenic polycyclic hydrocarbons and its activity is increased by exposure to cigarette smoke. There are individuals who have a genetic lack of this enzyme and they appear to be at greater risk for developing lung cancer if they smoke. Workers exposed to uranium or asbestos are also at greater risk for developing lung cancer if they smoke. Incidentally, another genetic factor/ increasing the risk to smokers is congenital lack of alpha-l-antitrypsin which predisposes the individual to emphysema. Individuals with a genetic predispositionfor 'damage from smoking should receive clear warning of their exceptional danger.

The possibility that tar may be reinforcing is not so easily disproven because the tar and nicotine content of cigarettes tend to One study was done in our laboratory with cigarettes in tar and nicotine were dissociated and varied (Goldfarb et al. 1976) (Fig. 1). The number of cigarettes- smoked was related to the nicotine content but not to the tar. There may be an interaction between tar and nicotine. Nicotine strongly influences strength ratings in the expected direction. High tar cigarettes actually were perceived as milder than low tar. The results were consistent with the hypothesis that people 'smoke to obtain nicotine, but it would be important to extend and confirm these findings with a wider range of tar and nicotine content.

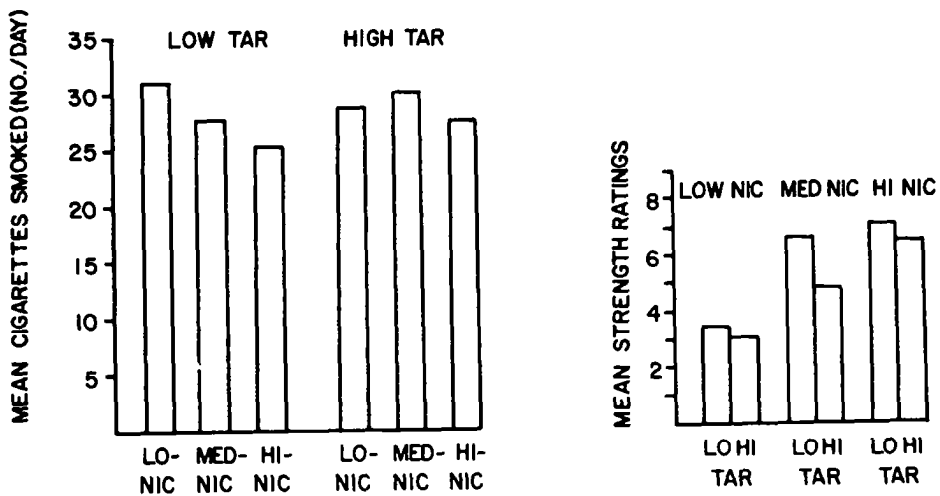


Figure 1
Number of cigarettes smoked and ratings of strength as a function of nicotine and tar

Nicotine

Nicotine (Fig. 2) has frequently been proposed as the primary incentive in smoking (Jarvik, 1972). Whether it is the only

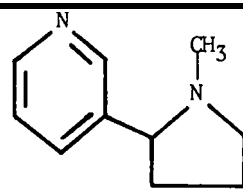


Figure 2
Nicotine Formula

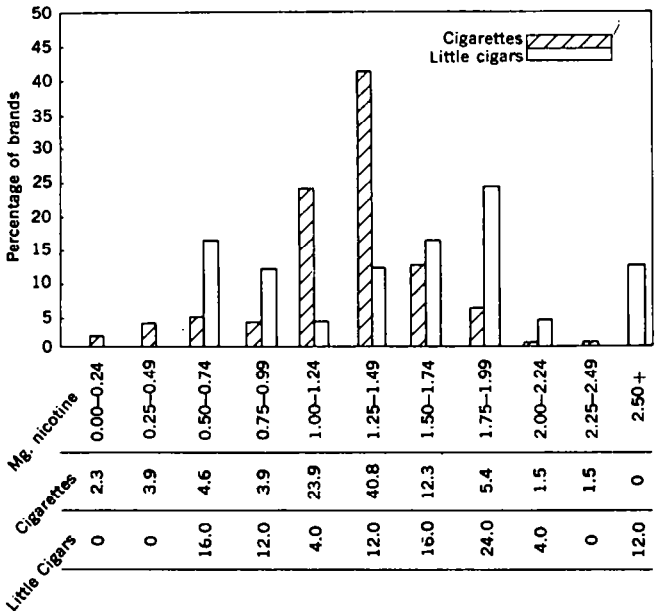
reinforcing agent or not, it is still the most powerful pharmacological agent in cigarette smoke. It is rapidly extracted by the alveolar capillaries and enters the pulmonary circulation and is pumped to the aorta where it stimulates the aortic and carotid chemoreceptors and may produce reflex stimulation of the respiratory and cardiovascular centers in the brain stem. Within one circulation time one fourth of the nicotine which was inhaled passes through the brain capillaries and, since it is highly permeable to the blood brain barrier (Oldendorf 1977), passes promptly into the brain.

Once in the brain nicotine stimulates nicotinic cholinergic synapses which are widespread. It also releases various biogenic amines including the catecholamines and possibly 5-hydroxytryptamine. It stimulates the emetic chemoreceptor trigger zone in the medulla and in novices or in large doses it causes nausea and vomiting. A variety of hypothalamic and pituitary hormones are stimulated by nicotine (Volle and Koelle, 1975). The effects that nicotine has on associative centers in the brain are still unexplored but may be of extreme importance in explaining its use and desirability. Studies from a number of laboratories indicate that nicotine can have a facilitating effect upon learning and memory in animals (McGaugh, 1973) and possibly in humans (Andersson and Hockey, 1977).

The other three-fourths of the nicotine is delivered via the aorta to the rest of the body and it acts wherever there are nicotinic sites. Thus it stimulates autonomic ganglia with, for example, activation of the gastrointestinal tract. By the same mechanism it releases epinephrine from the adrenal gland with all the "flight and fight" reactions (Cannon, 1932) that this hormone can produce. These include mydriasis, tachycardia, vasoconstriction, bronchiolar dilitation, decrease in gastrointestinal motility (however, this is generally successfully overcome by nicotinic ganglionic stimulation), and glycogenolysis. Among these are a rise in free fatty acids in the blood. It can also release catecholamines such as norepinephrine from nerve endings -and chromaffin cells through the body.

Much of the evidence for the role of nicotine as the primary reinforcer in cigarette smoke is circumstantial. Smokers clearly prefer cigarettes with nicotine than without (Goldfarb, 1970) but they will smoke nicotine free cigarettes grudgingly. In Fig. 3 it can be seen that the most popular cigarettes today have a nicotine content between 1.25 and 1.49 mg per cigarette. (US Dept. HEW, 1973).

Figure 3 Percent distribution of 130 brands of cigarettes and 25 brands of little cigars by nicotine content.



Mg. nicotine	0.00–0.24	0.25–0.49	0.50–0.74	0.75–0.99	1.00–1.24	1.25–1.49	1.50–1.74	1.75–1.99	2.00–2.24	2.25–2.49	2.50+
Cigarettes	2.3	3.9	4.6	3.9	23.9	40.8	12.3	5.4	1.5	1.5	0
Little Cigars	0	0	16.0	12.0	4.0	12.0	16.0	24.0	4.0	0	12.0

Cigarettes with a nicotine content less than 0.3 mg/cig do not do well on the market. Generally these are smoked by individuals who are trying to cut down or somehow diminish the harmful effects of smoking. Tobacco free cigarettes are doomed to oblivion almost from the start unless they are made of marihuana. Lettuce cigarettes had a brief vogue in the United States but the two companies producing the two different brands which were on the market went bankrupt.

It is important to note that low or no nicotine cigarettes allow their smokers to go through all the motions of smoking: lighting, handling and puffing can be the same as with usual cigarettes. So all the opportunity for visual, olfactory and oral

gratification is present. But it is the rare smoker who continues to smoke cigarettes lacking nicotine for any length of time when the more popular high nicotine cigarettes are available. The most likely explanation for this preference is that nicotine is reinforcing. Nicotine alone will partially suppress smoking but what little evidence exists in this area is conflicting.

There are very few studies in which nicotine alone has, been administered to man in an attempt to produce reinforcement. (Johnston, 1942; Jarvik, et al. 1970; Lucchesi et al. 1967; and Kumar et al. 1977). Johnston injected himself and other volunteers with nicotine and obtained clear evidence of reinforcement . However, these unique studies were uncontrolled for suggestion. There were three studies in which nicotine was given either by ingestion or intravenously, and in all three it was incapable of completely suppressing smoking, though it usually had some suppressant effect. Indeed in the experiment by Kumar et al. (1977) there was no discernible effect of a rapid intravenous infusion of 1.17 mg of nicotine (Fig. 4). Subjects went on puffing on their cigarettes just as they did with an equivalent injection of distilled water alone, and there was no delay in latency to the first puff.

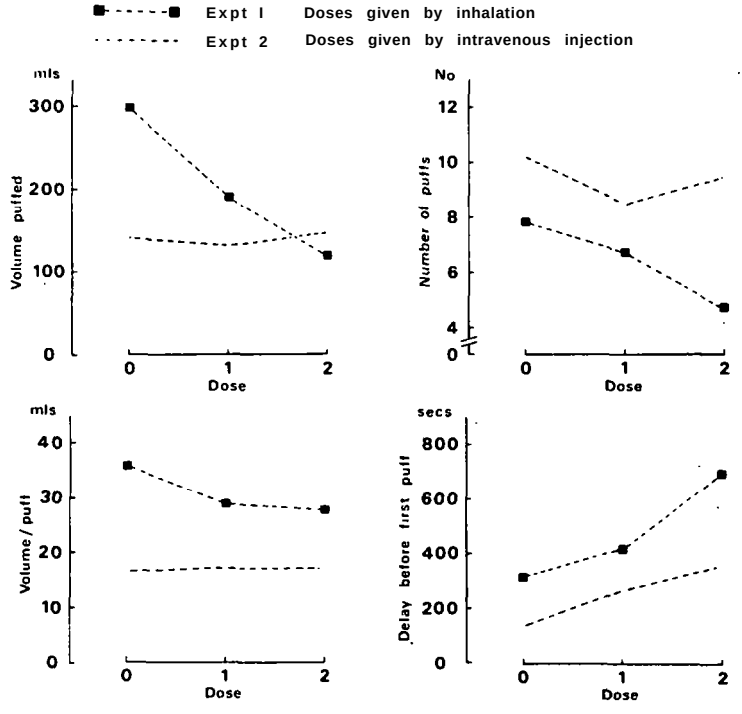


Figure 4 Effects of inhaled smoke and intravenous nicotine upon smoking. (Kumar et al. 1977)

These results are certainly disturbing to me and my colleagues who are proponents of the nicotine hypothesis of smoking. It is clear that the intravenous infusions had no effect on the subsequent puffing of cigarettes, whereas the cigarettes smoked immediately preceding the test session had a marked effect both on latency to the first puff and on the rate and volume of puffing. Is there any possible explanation of this discrepancy? Perhaps the nicotine delivered to the blood and brain were not equivalent in the two conditions. Perhaps the intravenous dose should have been higher, and furthermore, it might have been swamped by the fact that ad lib smoking was allowed during the intravenous administration of nicotine.

Clearly it would be important to take blood levels of nicotine during and following intravenous infusion and from the other arm, and compare it with blood levels during and following smoking. If the level of nicotine in the blood following cigarettes were greater than that from the infusion it might account for the results. Possibly the nicotine reacts differently when it mixes with venous blood and is carried through the right heart to the lesser circulation than it does when it passes through the pulmonary alveoli and into the left heart and greater circulation. Perhaps the heart rate changes which were used as indices of nicotine were elicited with a lesser dose when given intravenously than when nicotine was inhaled. In any event, this is clearly a critical experiment and needs to be repeated and analyzed more carefully.

On the other hand, the other half of the same experiment did show a dose response effect attributable to nicotine. Preloading by having subjects smoke before testing did reduce subsequent puff-

But could it have been something other than the nicotine in the smoke that was responsible for this effect? Kozlowski, working with us (Kozlowski, 1975) found similar results of preloading (Fig.5), a fast evanescent effect with cigarettes and a slower, persistent effect with nicotine gum.

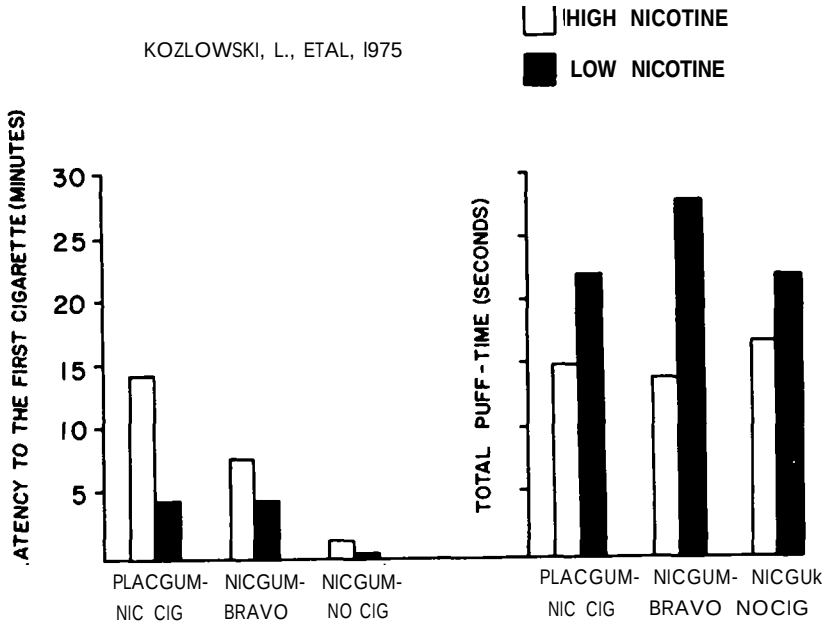


Figure 5. Effects of preloading with chewing gum, tobacco cigarettes or lettuce cigarettes upon latency to first cigarette and total puff time. Note that nicotine level of gum and cigarettes influenced latency and puff time.

One way to study this alkaloid would be to use cigarettes in which everything but nicotine content was held constant. We did such an experiment. When we allowed subjects to smoke experimental tobacco cigarettes with a nicotine content of 0.2 mg per cigarette compared with 2.0 mg per cigarette we found that the subjects smoked more of the low than the high nicotine content cigarettes (Fig. 6a, below). Figures 6b and 6c follow.

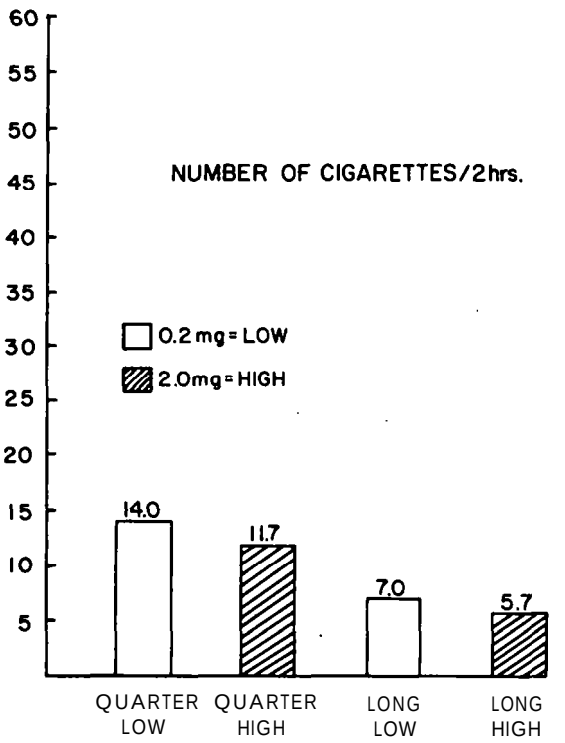
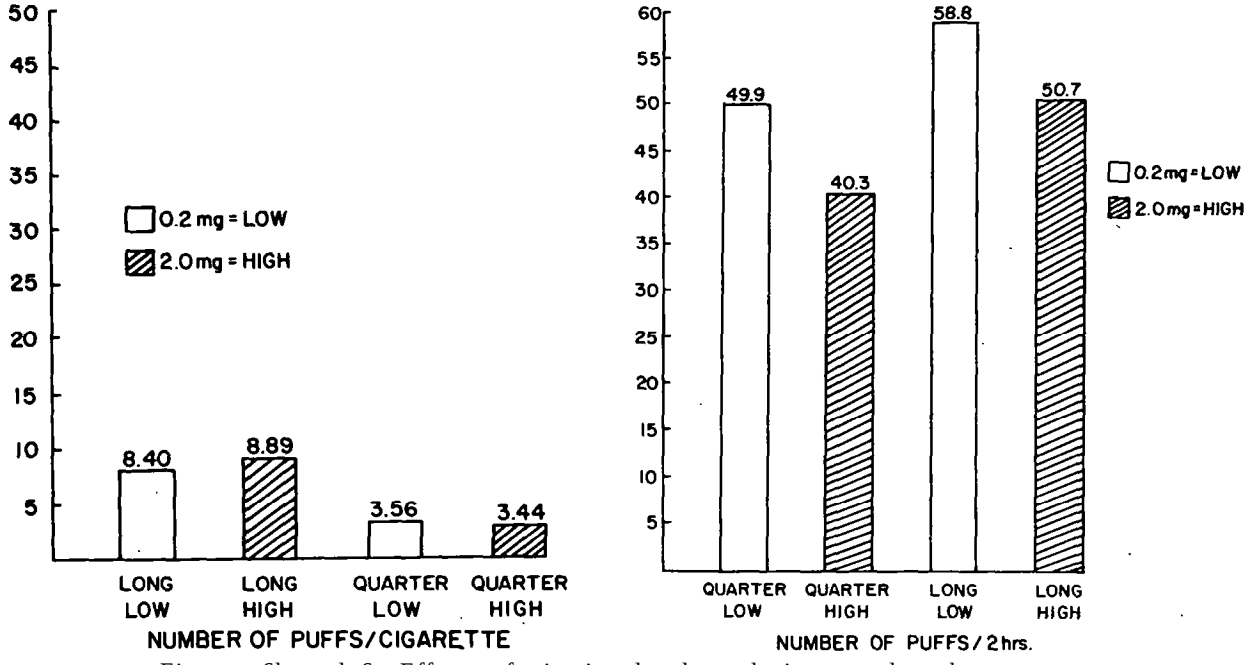


Figure 6a



Figures 6b and 6c Effects of nicotine levels and cigarette length upon number of cigarettes, number of puffs and puffs per cigarette in two hour session. Both nicotine content and length influence smoking behavior in the expected direction.

These results are similar to those found by Russell (1975). It can be seen that the total number of cigarettes as well as the total number of puffs was greater for the low than for the high nicotine cigarettes. The number of puffs per cigarette was not significantly greater. The cigarettes were lit at a rate controlled by the subjects to regulate the nicotine intake. But the puffing rate appears to have been invariant, triggered or released by the first puff and then followed by a behaviorally stereotyped pattern uninfluenced by the nicotine level. Perhaps if Kumar et al. had used cigarette lighting instead of puffing their results would have resembled those of Lucchesi et al. (1967).

TOLERANCE AND DEPENDENCE IN SMOKING

One of the more spectacular manifestations of drug dependence upon heroin or alcohol is the abstinence syndrome. Whenever either of these drugs is stopped there is a fairly rapid appearance of unpleasant signs and symptoms which become sufficiently severe in a matter of days to make the individual seek out these drugs to relieve these illnesses. As can be seen in Fig. 7, addicts stabilized on morphine showed slightly abnormal but fairly steady levels of function on respiration, blood pressure, temperature, sleep, eating, weight and subjective symptomatology . Within two to three days of withdrawal these functions became markedly abnormal and then gradually returned toward normal in about two weeks. As Goldstein et al. (1974) put it, “Tolerance to the narcotics is invariably accompanied by physical dependence”.

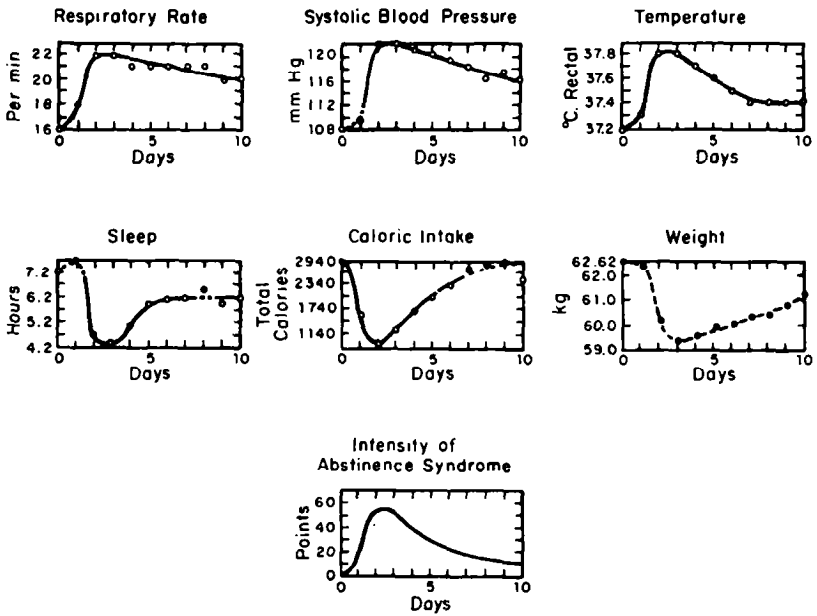


Figure 7 Time course of the abstinence syndrome for several variables following abrupt morphine withdrawal. Note the overshoot. (Goldstein et al. 1974; adapted from Kolb and Himmelsbach)

Although there appears to be evidence of some tolerance to some effects of smoking it is not at all clear how much occurs to what effects. Also, there is considerable controversy over whether a physical abstinence syndrome actually occurs, and if so, how strong it is. Our own studies indicate that with abrupt cessation of smoking there are indeed both subjective and objective changes. These appear to be mild in nature and almost never do subjects complain of being sick as they do on sedative or narcotic withdrawal.

Tolerance to nicotine has clearly been demonstrated in animals (Stolerman, 1974) but may not be representative of what occurs in man. Since non-smokers are unable to inhale cigarette smoke, it is difficult to compare them to smokers. Since in humans the ability to inhale smoke is itself an indication of tolerance it is difficult to use inhalation tests to compare non-smokers to smokers for nicotine tolerance. In our own studies we have concentrated on the effects of smoking upon heart rate (Fig. 8). It can be seen that there is a drop in heart rate following cessation of smoking in heavy smokers. Conversely, there is a rapid rise in heart rate with the first cigarette following a period of abstinence.

If the fall in heart rate following cessation produced an overshoot or rebound, then we would be able to say that there was evidence of a physical abstinence syndrome. One sees the overshoot phenomenon with some measures when heroin or alcohol is abruptly stopped in addicts. It would be important to follow acutely abstinent smokers for several days to determine evidence for overshoot and recovery in physiological functions such as heart rate.

Even if overshoot did not occur upon withdrawal it is conceivable that discomfort could accompany the effects of acute abstinence. Indeed some individuals complain bitterly while others do not seem to miss their cigarettes. The presence of craving and discomfort alone may be sufficient to indicate that a withdrawal syndrome is being manifested (Shiffman and Jarvik, 1976).

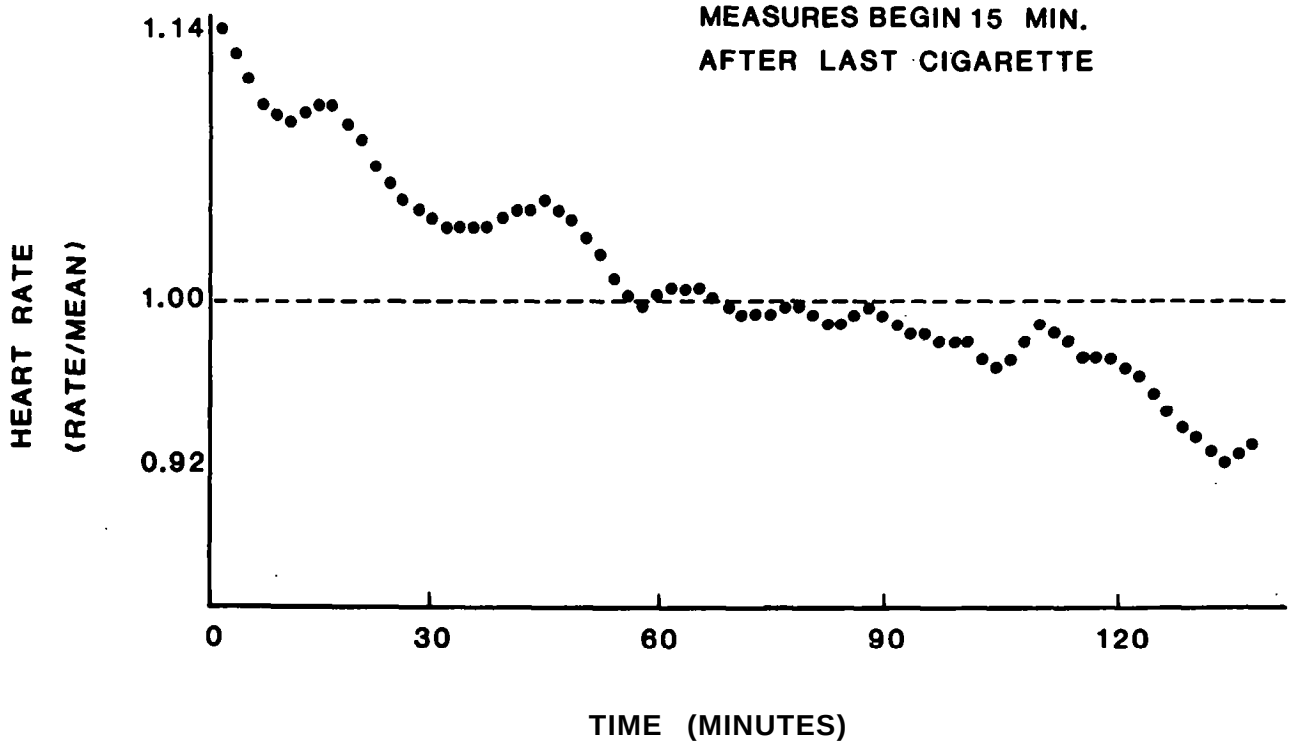


Figure 8 Heart rate decrease during two hours of abstinence from cigarette smoking, average of 31 subjects. Note gradual decline with lack of rebound or overshoot.

NICOTINE, A CO-FACTOR IN REINFORCEMENT OF SMOKING?

The evidence gathered so far indicates that nicotine plays an important role but not an exclusive one in the control of smoking. It might almost be said that nicotine is necessary but not sufficient for smoking behavior to occur, and to be sustained. Our older experiments with lettuce cigarettes (Goldfarb, 1970) and more recent ones with nicotine free tobacco (Jarvik et al. in preparation), show that people will smoke these cigarettes but their satisfaction is low and given the opportunity they would certainly choose a regular cigarette. The fact that lettuce cigarettes reinforced with nicotine were not accepted more readily than non-nicotine cigarettes has been a cause for concern and seriously undermined our support of the pure nicotine hypothesis. The relative inability of intravenous nicotine to suppress smoking (Lucchesi et al. 1967; Kumar et al. 1977) is a similar source of consternation.

In most of the studies where nicotine has been shown to influence smoking (Jarvik et al. in preparation; Russell, 1975) the smoker has had access to tobacco smoke. These effects could be explained more easily if in order to produce its optimal reinforcing effects nicotine had to interact with some other substance in tobacco.

Biology is full of examples of co-factors influencing various processes. For example, many of the vitamins such as thiamine and riboflavin function as coenzymes in metabolic reactions (Goodman and Gilman, 1975). Even cigarettes appear to operate as a co-factor in the genesis of myocardial infarcts. The Framingham Study (Kannel and Castelli, 1976) indicates that cigarettes operate as a risk factor in the presence of hypertension, high cholesterol and cardiac enlargement but not alone. Experimental studies indicate that nicotine alone will produce heart damage in animals only in the presence of vascular damage including that produced by carbon monoxide (Strong, 1969).

We propose therefore that the actions of nicotine in producing pleasure from smoking are potentiated by something else in the tobacco smoke and quite possibly this unknown substance is found in the tar.

Although nicotine makes up 93% of the alkaloid fraction of cigarette smoke, there are 13 other alkaloids present which have a variety of pharmacologic effects. Battig (1970) has shown a differential effect of nicotine and tobacco Smoke alkaloids upon swimming endurance in the rat. Nicotine improved, and the total alkaloids impaired, performance.

The alternative to a nicotine co-factor is the possibility that smoking is maintained by secondary reinforcers, assuming that nicotine is the primary reinforcer. However, if nicotine were the primary reinforcer then it should always be capable of producing reinforcement alone. Except for Johnston's somewhat

anecdotal study (1942), there is no evidence that nicotine alone is pleasurable or sought either by smokers or non-smokers. To be sure, the conditions under which it becomes reinforcing may be very special, but this too would imply that there are co-factors necessary for its actions. The distinction between a reinforcing co-factor and a secondary reinforcer is that the former is active in full force the first time it is presented to the subjects. A secondary reinforcer develops its potency gradually with repeated pairings with the primary reinforcer, and it extinguishes if it is not paired with the primary reinforcer.

To be sure, it would be a remarkable coincidence that two interacting reinforcers should be present in the same exogenous substance, but experimental evidence leads us to this hypothesis. One way to isolate the second chemical would be to compare chewing tobacco with nicotine chewing gum for reinforcing value. If there is a difference, then the chewing tobacco would have to be analyzed and its ingredients tested singly and in combination for reinforcement. Nicotine alone does not seem capable of totally substituting for cigarette smoke in the smoking habit, and yet nicotine must play a vital role. Finding the key to this riddle is an important challenge to those of us working in the field of psychopharmacology.

REFERENCES

Alp, M.H., Hislop, I.G., and Grant, A.K.: Gastric ulcer in South Australia, 1954-63. 1. Epidemiological factors. *Med. J. Aust.,* 2(24):1128-1132, Dec. 12, 1970.

Andersson, K. , and Hockey, G.R.J.: Effects of cigarette smoking on incidental memory. *Psychopharmacology,* 52(No.3):223-226, 1977.

Annitage, A.K., Dollery, C.T., George, C.F., Houseman, T.H., Lewis, P.J., and Turner, D.M.: Absorption and metabolism of nicotine by man during cigarette smoking. *Br. J. Clin. Pharmacol.,* 1:180P, 1974.

Battig, K. : Differential effects of nicotine and tobacco smoke alkaloids on swimming endurance in the rat. *Psychopharmacologia,* 18(No.3):300-304, 1970.

British Medical Journal: Smoking and lung ageing (Editorial). *Br. Med. J.,* 4(5991):247, November 1, 1975.

Bynum, T.E., Solomon, T.E., Johnson, L.R., and Jacobson, E.D.: Inhibition of pancreatic secretion in man by cigarette smoking. *Gut,* 13(5)361-365, May, 1972.

Cannon, *W.B.:.The Wisdom of the Body.* New York: Norton, 1932.

Cryer, P.E., Haymond, M.W., Santiago, J.V., and Shah, S.D.: Norepinephrine and epinephrine release and adrenergic mediation of smoking-associated hemodynamic and metabolic events. *N. Engl. J. Med.,* 295:573-577, September 9, 1976.

Daniell, H.W.: Smoker's Wrinkles: a study in the epidemiology of "crow's feet ." *Ann Intern Med.,* 75:873-880, 1971.

Goldfarb, T., Gritz, E.R., Jarvik, M.E., and Stolerman, I.P.: Reactions to cigarettes as a function of nicotine and "tar". *Clin. Pharmacol. Ther.,* 19(No.6):767-772, June, 1976.

Goldfarb, T.L., Jarvik, M.E., and Glick, S.D.: Cigarette nicotine content as a determinant of human smoking behavior. *Psychopharmacologia (Berl.),* 17:89-93, 1970.

Goldsmith, J.R., and Landaw, S.A.: Carbon monoxide and human health. *Science,* 162:1352-1359, 1968.

Goldstein, A., Aronow, L., and Kalman, S.M.: *Principles of Drug Action: The Basis of Phamracology,* New York: John Wiley & Sons, 1974. Second edition.

Goodman, L.S., and Gilman, A.: *The Pharmacalogical Basis of Therapeutics,* New York: Macmillan Publishing Co., Inc., 1975. Fifth edition.

Gordon, T., and Kannel, W.B., Dawber, T.R., and McGee, D.: Changes associated with quitting cigarette smoking: The Framingham Study, *Am. Heart J.,* 90(No.3):322-328, 1975.

Jarvik, M.E.: Further observations on nicotine as the reinforcing agent in smoking. *Smoking Behavior: Motives and Incentives.* A Special Project of the Council For Tobacco Research. St. Martin Island, French-Netherlands Antilles, January 12-15, 1972. Washington: Winston & Sons, 1973.

Jarvik, M.E., Glick, S.D., and Nakamura, R.: Inhibition of cigarette smoking by orally administered nicotine. *clin. Pharmacol. Ther.,* 11.:574-576, 1970.

Johnston, L.M.: Tobacco smoking and nicotine. *Lancet,* 742, 1942.

Kannel, W.B., and Castelli, W.P;: Significance of nicotine, carbon monoxide and other smoke components in the development of cardiovascular disease. In: hynder, E.L., Hoffman, D., and Gori, G.B. (Editors): Modifying the Risk for the Smoker, Volume 1. Proceedings of the Third World Conference on Smoking and Health, New York, June 2-5, 1975. *DHEW Publication No. (NIH)76-1221:* 369-381, 1976.

Khosla, T., and Lowe, C.R.: Obesity and smoking habits. *Br. Med. J.*, 4:10-13, 1971

Kozlowski, L.T., Jarvik, M.E., and Gritz, E.R.: Nicotine regulation and cigarette smoking. *Clin. Pharmacol. Ther.*, 17(No. 1):93-97, 1975.

Kumar, R., and Stolerman, I.P.: Experimentalandclinical aspects of drug dependence. In: Iversen, L.L., Iversen, S.D., and Snyder, S.H. (Editors): *Handbook of Psychopharmacology*, Volume 7. New York: Plenum Press, 1977.

Kumar, R., Cooke, E.C., Lader, M.H., and Russell, M.A.H.: Is nicotine important in tobacco smoking? *Clin. Pharmacol. Ther.*, 21 (No. 5) :520-529, May, 1977.

Larson, P.S., and Silvette, H.: *Tobacco : Experimental and Clinical Studies.* Supplement III. Baltimore: The Williams & Wilkens Company, 1975.

Lucchesi, B.R., Schuster, C.R., and 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. *Clin. Pharmacol. Ther.*, 8:789-796, 1967.

McGaugh, J.L.: Drug facilitation of learning and memory. In: Elliot, H.W., Okun, R., and George, R. (Editors): *Annu. Rev. Pharmacol.*, 13:229-241, 1973.

Mustard, J.F.: Platelets and thrombosis in acute myocardial infarction. *Hosp. Practice*, 7(1)115-128, January, 1972.

Oldendorf, W.H.: Distribution of drugs to the brain. In: Jarvik, M.E. (Editor): *Psychopharmacology in the Practice of Medicine,* New York: Appleton-Century-Crofts, 1977.

Russell, M.A.H., Wilson, C:, Patel, U.A., Feyerabend, C., and Cole, P.V.: Plasma nicotine levels after smoking cigarettes with high, medium and low nicotine yields. *Br. Med. J.*, 2:414-416, 1975.

Rylander, R.: Tobacco smoke toxicity: the experimental evaluation. *Rev. Environ. Health*, 1(1):55-74, 1972.

Schmeltz, I. and Hoffman, D. Chemical studies on tobacco smoke. XXXVIII. The physiochemical nature of cigarette smoke. In: Wynder, E.L., Hoffman, D., and Gori, G.B. (Editors): Modifying the Risk for the Smoker, Volume I. Proceedings of the 3rd World Conference on Smoking and Health, New York, June 2-5, 1975. *DHEW Publication No. (NIH)* 76-1221:13-34, 1976.

Seely, J.E., Zuskin, E., and Bouhuys, A.: Cigarette smoking: objective evidence for lung damage in teen-agers. *Science,* 172:741-743, 1971.

Shiffman, S.M., and Jarvik, M.E., Smoking withdrawal symptoms in two weeks of abstinence. *Psychopharmacology,* 50:35-39, 1976.

South Coast Air Quality Management District, California Air Resources Board, State of California. *Rule 703, Episode Criteria, Regulation VII, Emergencies,* p. 5, 1977.

Stolerman, I.P., Bunker, P., and Jarvik, M.E.: Nicotine tolerance in rats: role of dose and dose interval. *Psychopharmacologia,* 34: 317-324, 1974.

Strong, J.P., Richards, M.L., McGill, H.C., Jr., Eggen, D.A., and McMurry, M.T.: On the association of cigarette smoking with coronary and aortic atherosclerosis. *J. Atherosclerosis Res.,* 10:303-317, 1969.

Thompson, T., and Pickens, R. (Editors): *Stimulus Properties of Drugs.* New York: Appleton-Century-Crofts, 1971.

U.S. Public Health Service: Smoking and Health. Report of the Advisory Committee to the Surgeon General-of the Public Health Service: 1964. U.S. Department of Health, Education and Welfare. PHS Publication 1103.

U.S Public Health Service: The Health Consequences of Smoking. A Report of the Surgeon General: 1973. U.S. Department of Health, Education and Welfare. *DHEW Publication No. (HSM) 73-8704.*

U.S Public Health Service: The Health Consequences of Smoking. A Report to the Surgeon General: 1975. U.S. Department of Health, Education and Welfare. *DHEW Publication No. (CDC) 76-8704.*

Volle, R.L. and Koelle, G.B. Ganglionic Stimulating and Blocking Agents in L.S. Goodman and A. Gilmari (Eds.) *The Pharmacological Basis of Therapeutics.* New York: MacMillan Publishing Co., Inc., 1975. Fifth Edition pp. 465-474.

REFERENCES (Cont)

World Health Organization: Smoking and Its Effects on Health: Report of a WHO Expert Committee. *World Health Organization Technical Report Series No. 568.* Geneva: World Health Organization, 1975.

AUTHOR

Murray E. Jarvik, M.D., Ph.D.
Professor of Psychiatry and Pharmacology
Neumpsychiatric Institute
University of California
760 Westwood Plaza
Los Angeles, California 90024

Chief, Psychopharmacology Unit
Veterans Administration Hospital Brentwood
Wilshire and Sawtelle Boulevards
Los Angeles, California 90073

DISCUSSION

Dr. Vogt raised the question of whether lettuce cigarettes with added nicotine were not equivalent to ordinary tobacco cigarettes. I replied that the lettuce tar may not be equivalent to tobacco tar, and furthermore, the alkaloids which are present might be different. I cited the study by Battig, in which he compared the effect of nicotine alone upon rat behavior, with all the other alkaloids. He found that nicotine facilitated performance, whereas the combination of alkaloids impaired performance. This study indicates that at least under some circumstances there seems to be an interaction between nicotine and other substances.

Dr. West brought up the comparison between nicotine and cocaine, and the effects of repeated reinforcement. I pointed out that there was immense repetition in reinforcement with cigarettes, with more than 50,000 puffs a year for a pack-a-day smoker. This, of course, results in a very strong resistance to extinction. An annual model of nicotine dependence would ve very useful. Unfortunately, animals will not self-inject nicotine or do so only indifferently whereas they will self-inject cocaine and amphetamine very readily. The fact that animals self-administer nicotine desultorily makes me think that if nicotine were combined with some other substance, perhaps a constituent of tar, they would self-inject much more strongly. On the other hand, I was unable to train monkeys to puff in a reliable manner approximately the human rate on real cigarettes. Either my training procedure was inadequate or cigarette smoking is a peculiarly human form of behavior.

It was remarked that intravenous injections of nicotine may not provide the same panoply of cues that inhalation of tobacco smoke into the mouth does. Dr. Russell pointed out the paradox that intravenous nicotine did not inhibit puffing behavior, whereas forced smoking did. Obviously, there was something different, and it must have been more than just the nicotine to account for these results. Dr. Jaffe pointed out that there are differences between intravenous and oral routes of administration of drugs. He used as an example, Talwin, (pentazocine). Opioid addicts will take this intravenously,

but do not seem to like to take it orally; this may be due to its metabolism by the liver. He also points out that self-administration of 'nicotine intravenously by humans would perhaps yield a better picture of how reinforcing it was. He then remarked that the negative results obtained with Russell's intravenous nicotine, as well as the negative results with nicotine enriched lettuce cigarettes, and the small or absent reinforcing effect seen with nicotine chewing gum all present a very important paradox which must be explained if we are to understand the role of nicotine in smoking.

Murray E. Jarvik, M.D., W.D.

Psychological Factors in Smoking

Dorothy E. Green, Ph.D.

BACKGROUND

Dr. Schuman mentioned and reported on some of the results from the surveys carried out by the National Clearinghouse for Smoking and Health in 1964, 1966, 1970, and 1975. He pointed out that while he was reporting on data primarily concerned with the proportion of smokers, ex-smokers, and never-smokers in the population, these surveys measured other variables. I will report on some of those variables which concern the dynamics of smoking.

The first survey, conducted in 1964 just a few months after the Task Force report to the Surgeon General came out, was done when little was known about the whole problem of cigarette smoking. Therefore, the material in the earlier surveys was based on psychological knowledge about human behavior in general and about health behavior in particular.

I would like to trace what has happened since in the studies of the dynamics of smoking. The 1964 and 1966 surveys--the two were about eighteen months apart--tested many hypotheses about smoking, the continuation of smoking, and the giving up of smoking. From the results of these two surveys we were able to develop measures of some of the concepts which were explored. In fact, nineteen measures of psychological aspects of cigarette smoking were refined and retested. These measures were included in the 1970 and again in the 1975 survey. I will report here on some of the findings from these two surveys, indicating the kinds of changes that took place between 1970 and 1975.

FACTORS MOTIVATING QUITTING

Four factors related to motivation for quitting were identified. As one would expect, many people reported that they want to quit smoking to protect their health. People scoring high on this factor agree with statements such as "Cigarette smoking might give me a serious illness." Second, people realize that their cigarette smoking is an example to others; they are aware that others may be influenced

to take up or continue smoking. Third, aesthetics may play a role in a decision to stop smoking. Smokers agree that smoking is a "messy kind of habit" that "causes damage to clothing and personal property." A fourth reason for wanting-to quit is mastery: Many resent a habit which, they feel, prevents them from completely controlling their lives. They agree that, "quitting smoking would show that I have willpower."

There are undoubtedly many common motivations for discontinuing the smoking habit other than the four described above. One of the factors that we searched for in the late 1960's but were unable to find in this country was an "economics" factor. We tried to determine if the money spent on cigarettes was a deterrent to smoking. It was not a common one. Subjects' attitudes seemed to be, "I'll spend anything to get my cigarettes!" We tried isolating the economics factor in terms of the holes burned in clothes and furniture, but this was related to the aesthetics factor. Similarly, cost of illness caused by smoking became more appropriately related to the health factor. We were never able to define an economic factor. It might be possible to do so now that taxes have raised cigarette pack prices very significantly.

In comparing the data from the 1970 to 1975 surveys, we find little difference in mean scores (Table 1). The reasons for wanting to quit that were important in 1970 were still important in 1975.

Table 1

MOTIVATIONS FOR WANTING TO QUIT SMOKING

	Mean Scores	
	1970	1975
Health Example	9.8	9.9
Aesthetics	9:2	9:3
Mastery	9.7	9.5

In view of the changing attitudes toward cigarette smoking, however, we can speculate about some factors probably not present a few years ago that-might be identified now. One, for example, is the smoker's feeling that he is an unwelcome nuisance. We hear smokers say. "I'd like to quit smoking because I feel so terrible when I light a cigarette in someone else's house;" "I am bothering the people around me;" "I know other people don't want to breathe my smoke." And they are right--more and more non-smokers are saying that it is annoying to be around people who are smoking. This is a strong motivation that we would not have found earlier.

Another reason for wanting to quit is related to the changing image of the smoker. At one time, the smoker was pictured as sophisticated, glamourous, and romantic. This image has changed a great deal over the years. In motion pictures, for example, we are less likely to see two lovers sharing a cigarette than we are to see the tough

hoodlum smoking. With such a changing image, the smoker may be likely to want to quit to avoid the modem stereotype.

FACTORS IN CHANGING HEALTH BEHAVIOR

The next set of factors we identified deals with health and were based on Godfrey Hochbaum's model of health behavior. Five necessary conditions for changing health behavior are posited: knowledge of the threat; importance of the threat; personal relevance; capability doing something about it; and value of doing something

Before the late 1960's we were not able to identify knowledge of the threat as a separate factor. Since the 1960's, widespread awareness of the health threat posed by smoking has brought about the separate identification of the first factor. The importance of the threat is indicated by such statements as, "Cigarette smoking is enough of a health hazard for something to be done about it."

While recognizing that cigarette smoking is, in general, an important health threat, a smoker may still deny its personal relevance. S/He my say, "I don't smoke enough to get any of the diseases cigarette smoking is supposed to cause," or, "I haven't smoked long enough to worry about the diseases cigarette smoking is supposed to cause." As long as he holds the "It can't happen to me" attitude, he will not act on his knowledge of the threat.

Given acceptance of the threat and its personal relevance? the smoker still has to believe there is some value for him in stopping smoking. Those who contend that "If a person has already smoked for many years, it probably won't do him much good to stop," will be easily deterred from any effort to quit.

Lastly, before he is willing to attempt a difficult change in behavior, a person must believe that he can succeed. No one likes to fail, and the person who thinks failure likely will almost certainly try to avoid such a situation. Thus, optimism is virtually necessary for making the attempt.

The mean scores on four factors are reported in Table 2. Practically no change occurred in the scores on Importance or Value of Stopping. Scores on Personal Relevance decreased slightly: People were slightly more inclined to think, "It will not happen to me." There was an increase in the mean score on Capability. This probably reflects a tendency, over the five-year period, for those who found it fairly easy to quit have done so, so that those still smoking in 1975 were those pessimistic about their success in quitting. Many had probably tried and failed.

FACTORS CONTRIBUTING TO CIGARETTE USE

The next set of factors was based on Silvan Tomkins' "management of affect" theory. What do people use the cigarette for? How do they use the cigarette to manage their feelings? We found three

Table 2

PERCEPTION OF THE HEALTH THREAT

	Mean Scores	
	1970	1975
Importance	8.5	8.8
Personal Relevance	9.1	7.6
Value of Stopping	9.1	9.5
Capability for Stopping*	6.3	*8.5*

*A high score indicates perceived difficulty in stopping smoking.

positive uses. The most prevalent of these has been called Pleasurable Relaxation. It includes smoking a cigarette when you are already feeling good in order to enhance your enjoyment. The feeling of relaxing after a good meal with a cup of coffee and a cigarette is an example. The cigarette makes something that was already good, better. At least some of the time, most smokers use the cigarette to enhance an already-existing sense of well-being.

Some people, not nearly as many, use the cigarette for stimulation--to pick them up. Some use it for the pleasure of handling the cigarette. This is much more typical of the pipe smoker. For example, a pipe smoker who spends an hour to fill his pipe and five minutes to smoke it or the smoker who taps the cigarette and fiddles around with it before he lights it. These behaviors all make positive use of the cigarette.

At the same time, there are many people who use the cigarette to reduce negative feelings. When they are angry, upset, or nervous they light a cigarette. The cigarette becomes a *catch*. It is the converse of smoking a cigarette when you are feeling good to make you feel better. Those who smoke a cigarette when they are feeling bad do so to keep from feeling quite so bad.

A very prevalent phenamenon is Psychological Addiction. The typical addict experiences an increasing need for another fix of whatever he is addicted to as soon as the effects of the first wear off. Similarly, the psychologically addicted smoker feels the need for the next cigarette build up from the time he puts out the cigarette he has been smoking. He is the smoker who cannot bear having no cigarettes in the house. He will go out in the middle of the night to get them because he fears a situation in which a cigarette will *not* be available the minute he wants one.

The last of these factors is Habit. In this case the smoker uses the cigarette not to manage affect at all, but simply from habit. He lights a cigarette when he already has one burning in the ashtray.

These are the factors identified in the use of cigarettes. I am sure there are more, since people are so various. The heavy smoker may use the cigarette for nearly every factor cited. A few people--the

fast-moving, "salesman" type--tend to use it for stimulation, to keep than going. The housewife--when the children get on her nerves and are driving her crazy--has to reduce tension somehow. She finds respite when she sits down with a cup of coffee and a cigarette. Although the factors which enter into a smoker's use of cigarettes are widely varied, we have determined a moderate correlation between psychological addiction and reduction of negative affect.

The mean scores for 1970 and 1975, reported in Table 3, show no change during the five-year period.

Table 3

USES TO WHICH THE CIGARETTE IS PUT

	Mean Scores	
	1970	1975
Stimulation	6.2	6.0
Handling	5.7	5.8
Pleasurable Relaxation	11.3	11.2
Tension Reduction	10.3	10.3
Craving: Psychological Addiction	9.4	9.4
Habit	6.3	6.3

FACTOR ANALYSIS OF TEENAGE SMOKING

Experience with identifying factors associated with the dynamics of adult smoking provided help in studying the taking up of smoking by teenagers. The National Clearinghouse for Smoking and Health contracted with the research firm Education and Public Affairs to carry out the study. After a comprehensive review of existing literature, depth interviews with teenagers were conducted. Group and individual interviews were held in four kinds of communities: inner-city, suburban, blue collar, and rural. These open-ended inter-views provided draft statements, in the teenagers' own words, related to attitudes toward smoking. Through an iterative interview process, the statements were revised to increase clarity, readability, and understandability.

The revised instrument was administered to about 2,600 teenagers in grades 7 through 12, drawn from a national probability sample of school districts. The data were analyzed to eliminate questions that were difficult to answer and those that showed little variance in response. A factor analysis of 108 items was performed, and the questionnaire was reduced to 83 items. The 83-item instrument, along with a number of demographic questions, was then administered to approximately 5,200 pupils, again drawn from a national probability sample of school districts. Factor analysis of the data from this administration resulted in eight factors, which are described briefly below.

The first is a health factor--the effect of cigarette smoking on health. Second, in addition to seeing smoking as detrimental to personal health, the teenager sees it as detrimental to the environment. These two factors are viewed as "costs". The third describes the benefits of smoking: "It makes you feel good. It's pleasurable ." Since many teenagers know that smoking is harmful to health and also cite positive aspects of smoking, cognitive dissonance results which must be reduced in some way. *Thus* we find the fourth factor to be a rationalization factor. It describes the teenager who says, "I'm not going to smoke long enough for it to hurt me" or, "I'm going to smoke for a few years and quit." The fifth factor reflects a common stereotype of teenage smoking, the attitude that peer pressure is the most compelling influence in taking up smoking. The sixth factor concerns another stereotype of the teenage smoker, here internalized by teenagers themselves. The smoker is described as a "bad boy," e.g. , "Smokers are more likely to get into trouble," and, "Smokers don't make as good grades."

The two final factors on the surface are not related to cigarette smoking. One describes feelings toward authority. Teenagers evidence a great ambivalence: They would like to be able to turn to their parents whenever they can; they would also like to be rid of them forever. This factor has to do with feelings toward authority. Factoring teenagers' attitudes toward authority showed smokers are more likely than non-smokers to feel that "a teenager should be able to do whatever he wants to do whenever he wants to do it." The last factor deals with the attitude that "what happens to me in my life is very important to me, and I can do something about it.!' We have recently identified this factor in the adult surveys, also. If a subject feels that whatever happens to his body is something he cannot control, he may be apathetic towards changing health behavior. But if he feels that he is the one in charge, he can control his health, then he is more likely to develop those habits regarded as leading to a healthier life.

CONCLUSION

There is much that still needs to be learned about giving up smoking, continuing to smoke in the face of health threats, and taking up smoking. We must look to future research in the behavioral aspects of smoking for the answers.

REFERENCES

Hochbaum, Godfrey. Behavior in response to health threats. *Annual Meeting, Amer. Psych. Assoc.,* Chicago, Illinois, September 1960.

Tompkins, S.S. Psychological model for smoking behavior. *Amer. J. Pub. Health,* 56:17-20, 1966.

Tables: Data drawn from Unpublished data, U.S. Public Health Service, National Clearinghouse for Smoking and Health, 1975 Adult Survey.

AUTHOR

Dorothy E. Green, Ph.D.
Consulting Research Psychologist
3509 N. Dickerson Street
Arlington, Virginia 22207

DISCUSSION

Dr. Green was asked whether or not people smoke for a variety of reasons such as a substitute for food or a way to express anger. She stated that there is no data to justify the assumption that people smoke to avoid eating. However, there is data that seems to indicate that smokers *either* eat less, or gain less weight, and that hunger and over-eating become an important withdrawal symptom following abstinence. (Khosla and Lowe, 1971). Dr. Russell wanted to know why motives for continuing to smoke seem so much stronger than motives for stopping. Dr. Green replied that man is simply not rational enough to appreciate the danger smoking poses to health, which would have. to be the major motive for quitting. When smokers stop smoking, they realize in retrospect that they suffered discomforts which they would not admit while they were smoking, such as scratchy throat and coated tongue. Dr. Green also brought up the point that smoking might be a type of sub-intentional suicide, where the threat of death is entertained with equanimity. In teenagers, it appears that social pressure is an extremely important factor in determining if one will smoke. If parents and an older sibling smoke, the teenager is four times as likely to smoke than if none of them smoke. High school students in a college preparatory course are less likely to smoke than those in other courses. Children of college educated parents are less likely to smoke. Those who participate in more activities in high school are less likely to smoke. On the other hand, those who engage more in alcohol-related activities and sex are more likely to smoke. It seems that social factors are exceedingly important in determining smoking behavior in teenagers. This gives us a handle on controlling such smoking.

Murray E. Jarvik, M.D., Ph.D.

An Opponent Process Theory of Habitual Behavior With Special Reference to Smoking

Joseph W. Temes, Ph.D.

HABITUAL BEHAVIOR AND SMOKING

An individual may smoke at times because he seeks to reduce the tensions of life, while on other occasions he may desire a mild stimulant. Depending on the speed and depth of smoke inhalation, the smoker may produce these opposite effects due to nicotine's action on the nervous system. Regardless of the reasons which lead many individuals in our culture to experiment with cigarette smoking, there is general agreement that, for the vast majority of those who choose to smoke, cigarettes are habit forming. However, smoking is only one of many types of acquired motivations. Drug addiction and alcoholism are some other examples of acquired motivaation, so too is the type of over-eating that leads to obesity. Together they belong to a generic class which may be labeled habitual behavior.

An opponent-process analysis of the various habitual behaviors suggests that the common element is an aversive state of craving which is engendered by the termination of a pleasurable stimulus. Instrumental escape and avoidance responses are energized by aversive states. Therefore, all forms of habitual behavior are seen as escape or avoidance responses. This analysis implies that simple means exist for the prevention of the development of such habits. However, it also implies that once established such habitual behaviors, due to the nature of the underlying mechanisms, should be very difficult to modify (rehabilitate) and highly susceptible to relapse. This is because once acquired these operants are overdetermined by the normal functioning of the opponent-process system which regulates affect and hedonic tone. Thus, we assume that the development, maintenance, and, in the case of attempted abstinence, the relapse to habitual behavior, are evidence of a normally functioning homeostatic system. Regardless of whether one views the outcome as pathology or not, the rubric of habitual behavior implies a common explanatory mechanism. In this regard, the opponent-process model holds great appeal in that it appears to account for these commonalities in a reasonably parsimonious manner, and provides

detailed predictions regarding temporal parameters, ease of conditioning and generalization of affective states which motivate habitual behavior. The main difference from prior conceptions of acquired motivation is that the opponent-process model proposes a primarily non-associative mechanism to explain the development of habitial behavior while maintenance and relapse are attributed to conditioning and generalization.

NON-ASSOCIATIVE EFFECTS OF UNCONDITIONED STIMULI AND REINFORCERS

Assume, as Pavlov did, that if the nervous system is put out of balance by external stimulation, it strives to return to a resting state. Obviously the balance referred to by Pavlov is a homeostatic mechanism. Stimuli capable of producing an imbalance are typically identified by psychologists as unconditioned stimuli (UCSs) or as reinforcements. The primary affective process typically elicited by an UCS is called an emotional or affective unconditioned response (UCR) . In the terminology of the opponent-process theory of acquired motivation (Solomon and Corbit 1973, 1974; Hoffman and Solomon 1974; Solomon 1976) the UCS is an affect-arousing stimulus capable of eliciting primary affective processes (UCRs) known as a-processes.

Although the theory is still somewhat informal and lacking empirical validation, a description of the model of the affective dynamics of acquired motivation is possible. The standard pattern of affective dynamics (Figure 1 Panel A) for intense UCSs is biphasic, involving two affective states and a third hedonic neutral or baseline condition. Two dynamic opponent processes, a positive excursion from baseline and a negative one are assumed to underly these affective states. The positive excursion from baseline always follows the onset of any intense affect-arousing stimulus. This process is labeled the a-process. The negative excursion from baseline follows the onset of the a-process albeit in a more sluggish or dampened fashion. This is-the b-process.

The a-process closely tracks the input variable, is phasic and does not show much habituation during any individual stimulus presentation. An a-process may reflect either pleasant or unpleasant affect. Its character is determined by the nature of the stimulus which elicits it. Thus a painful stimulus, such as intense electric shock, should elicit an unpleasant affective state and a pleasant stimulus, such as a sweet taste, should elicit a pleasant affective state.

The primary affective process is postulated to elicit a secondary affective process, the b-process. This is the postulated nervous mechanism for the restoration of homeostasis (neutral affect) which the a-process puts out of balance. The action of the b-process is to oppose or suppress the affective or hedonic impact of the affect arousing stimulus. However, in addition to being an opponent process the b-process has the following characteristics:

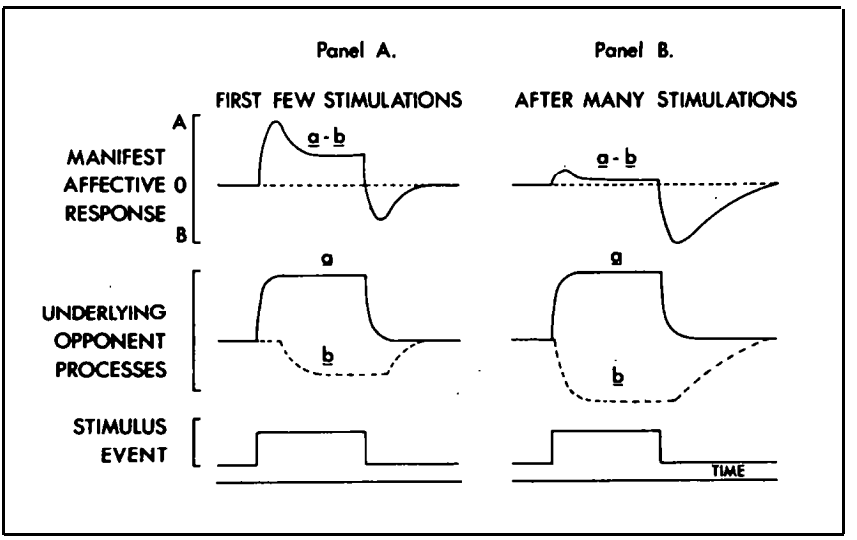


Figure 1. The consequences of subtracting the b-process from the a-process when the b-process is weak and when it is strengthened by repeated use. The Resultant A-state is small after the b-process is strengthened, but the B-state is more intense and longer lasting. Notice the a-process precipitates the b-process (dotted line, second tier) thus modulating the A-state (solid line, first tier). On termination of the stimulus event the a-process rapidly returns to baseline and the b-process perseverates, thus unmasking the B-state (solid line,-first tier). Reproduced from Solomon, R.L. and Corbit, J.D. An Opponent Process Theory of Motivation. *Psychological Review,* 1974, 81, 119-145. Copyright 1974 by the American Psychological Association. Reprinted by permission.

it is more sluggish than a-processes, having a delayed latency; thus, it is slower to peak and slower to decay or return to baseline following the termination of the affect arousing stimulus.

The processing system proposed here involves three components; (See Figure 2). Incoming information, the perceptual signal, is handled categorically and its side effect is the arousal of the primary affective process, the a-process. This is the first component of the processing system, The elicitation of the a-process triggers the b-process or opponent affective reaction which is the second component of the processing system. The b-process is negatively signed relative to the a-process and the algebraic summation of these opponents is the third processing component. The result, following a square have input of an affect-arousing stimulus, is a dynamic sequence of events having five distinct features (See Figure 3). when the stimulus onsets the a-process very rapidly reaches its peak amplitude, (feature l). It is followed by an adaptation phase (feature 2) or adjustment which reduces the intensity of the a-process to a lower level. The adjusted level (feature 3) is relatively more steady and is maintained until stimulus termination. Since the-a-process closely tracks the input variable, it is abruptly terminated as the stimulus off-sets. Then another process, heretofore masked, the b-process is revealed. It is characterized by a peak amplitude immediately after stimulus off-set (feature 4) and a slow decay to baseline (feature 5). These five features taken together as a sequence of events describe what is known as the standard pattern of affective dynamics in the opponent-process model.

The affective state or hedonic condition of the organism at any moment is postulated to be the algebraic sum of the intensities of the a- and b-processes where b is always assumed to reduce a, i.e., b has a negative sign. Thus, whenever a>b, the organism experiences an affective state dictated by the nature of the a-process. This condition is known as the A state. When, however, a<b the organism experiences a consequent affective state which has the same hedonic tone as the b-process. This condition is known as the B state. If A is a positively reinforcing state then, axiomatically, B is negatively reinforcing and vice versa.

One can best observe the quality and intensity of the primary affective reaction of the stimulus (the A state) immediately following its onset, i.e., at the peak of the a-process. Likewise, one can observe the quality and intensity of the secondary affective reaction (the B state) directly following the termination of the stimulus, i_ie., at the peak of the b-process. Thus the opponent of the reaction aroused in the presence of the UCS is similar to an affective negative after-image which becomes manifest when a strongly reinforcing stimulus is suddenly terminated. This affective negative after-reaction, the B state, is a new condition which is only revealed after the termination of the UCS. It would not have occurred if the UCS had not been presented. Thus, we have defined three qualitatively distinct conditions: the baseline or homeostatic neutral, the A state or primary affective condition, and the B state or secondary affective condition.

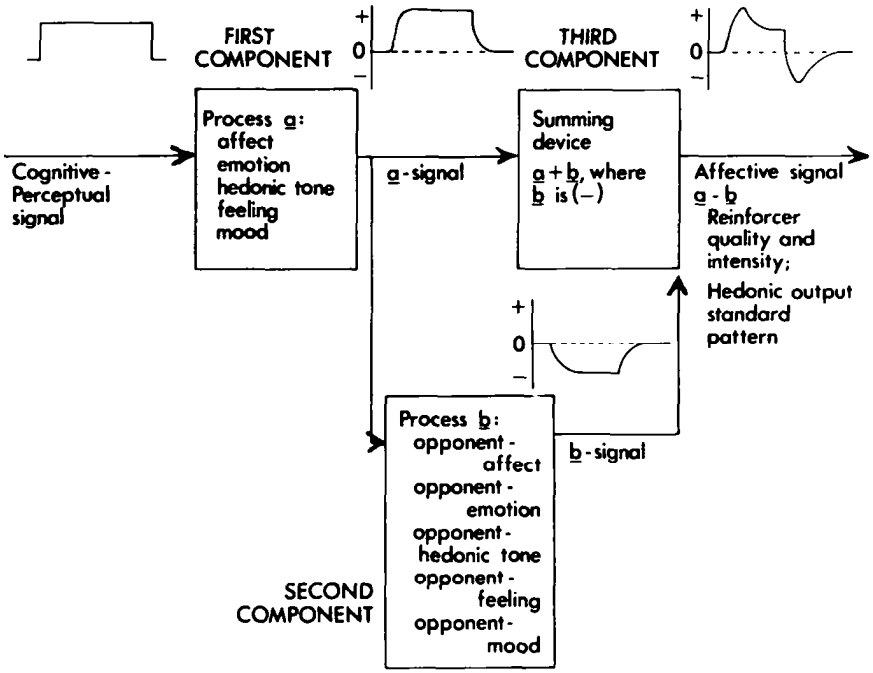
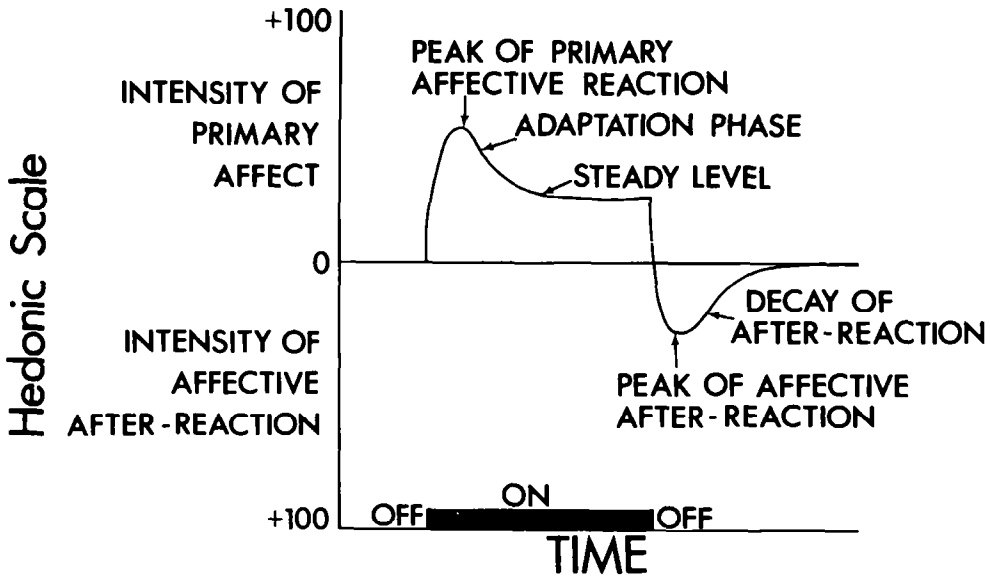


Figure 2. A box-flow diagram of the interaction between a-process and b-processes. The b-processes are activated whenever a-processes are activated, and the resultant a-b is determined by a summator. Reproduced from Solomon, R.L. and Corbit, J.D. An Opponent Process Theory of Motivation. *Psychological Review*, 1974, 81, 119-145. Copyright 1974 by the American Psychological Association. Reprinted by permission.



Standard Pattern of Affective Dynamics

Figure 3. The standard pattern of affective dynamics, showing the five distinctive features of affect resulting from a typical, square-wave input. The B-state becomes manifest after the A-state.is..terminated. Reproduced from Solomon, R.L. and Corbit, J.D. An Opponent Process Theory of Motivation. *Psychological Review,* 1974, 81, 119-145.

Based on experimental observations and his own clinical impressions of motivational events, Richard Solomon has used the following example of a dog habituated to a long series of painful electric shocks to illustrate the dynamics of affective behavior. When the dog is initially placed into a Pavlovian harness and 10 seconds of electric shock is delivered to its paws, several responses occur. When the shock first comes on, the dog shrieks, struggles, strains its head back, its eyes bulge and it urinates and defecates. Additional autonomic changes include pupillary dilation, piloerection, tachycardia and increased respiration and blood pressure. The heart rate increases to a peak and then declines while the shock is still present (See Figure 4). After 10 seconds, when the shock is terminated, there is a decelerated heart rate which reaches a minimum and then slowly returns (increases) to baseline rate. Observable behavior such as vocalizations appears correlated with heart rate, i.e., vocalization and struggling are most intense right after shock onset and decrease while the shock is still on. This is also true of the pupillary and piloerectile responses. After the shock goes off, a new state of relief reveals itself. That is to say, termination of the painful stimulus does not merely result in a rapid return to baseline. Rather, a new affective state is almost immediately manifested. This new state is hedonically quite different from both the baseline state and the primary state produced by the aversive stimulation. The baseline state was somewhat neutral, the state produced by the aversive shock was an unpleasant condition but the state which follows the termination of the shock is a new and pleasant affective state.

Another example from my own research on drug abuse will serve to show the same pattern of affective dynamics when the stimulus is pleasant rather than aversive. (All habitual behaviors are of this type, although termination of pleasurable affect is not the only condition which may engender an aversive craving state.) Initial opiate experiences, although partially aversive, are usually *also* partially pleasurable. Human heroin addicts report a surge of intense pleasure, the rush, which accompanies the drug administration followed by a period of less intense euphoria. However, with repeated use, the pleasurable aspects of the drug effects are progressively reduced such that the rush is less intense and the euphoria is greatly attenuated. Concomitant with the reduction of positive effects, a set of aversive somatic symptoms begins to occur when the drug effect "'wears off." This syndrome will grow in strength when the drug dose is-repeated. 'During periods of abstinence between doses, the phenomenon of drug hunger and craving is increasingly present. According to an opponent-process analysis of these data, heroin is an affect arousing stimulus and the rush and euphoria, the A state, are manifestations of the underlying primary affective process, summed with the opponent b-process. The aversive somatic after-reaction, the B state, occurs when heroin is withheld. It is hedonically the opposite of the pleasurable A state. It occurs as a function of the growth of the opponent b-process. The b-process actively suppresses or counteracts the a-process resulting

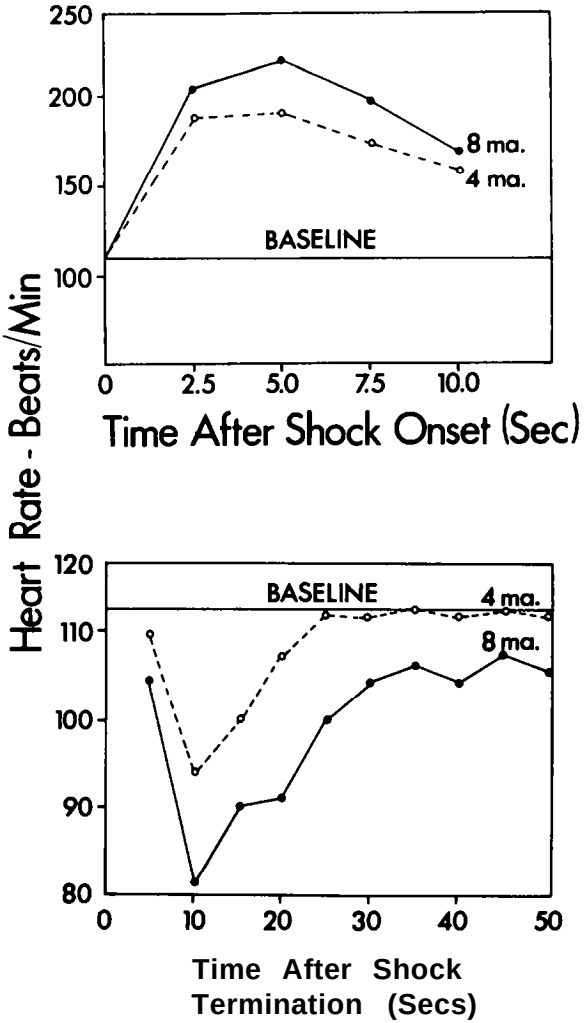


Figure 4. Typical course of heart-rate reaction to intense foot shocks in the dog. While ten-second shock is on, heart rate increases and then decreases. When shock is terminated, heart rate falls significantly below the baseline or resting level. Then it slowly returns to baseline, but more slowly after 8ma. shock than after 4ma. shock. Reproduced from Church, R.M., LoLordo, V.M., Overmier, J.B., Solomon, R.I. and Turner, L.H. Cardiac Responses to Shock in Curarized Bogs: Effects of Shock Intensity and Duration, Warning Signal and Prior Experience with Shock. *J of Comp and Physiol Psych,* 1966, 62, 1-7. Copyright 1966 by the American Psychological Association. Reprinted by permission.

in the reduction and attentuation of the rush and euphoria. The B state energizes operants which attempt to terminate or avoid the aversive somatic symptoms. The performance of an appropriate operant such as drug seeking (coping) is selectively reinforced by the abrupt relief from the somatic symptoms and by the pleasurable components of the A state, i.e., the rush and euphoria.

Conditions for the Growth of the b-process

It is the thesis of this presentation that the standard pattern of affective dynamics as proposed by the opponent-process theory of acquired motivation accounts for the data of the various forms of habitual behavior. Thus, a specification of the assumptions which account for the dynamics of acquired motivation is tantamount to a general theory of habitual behavior. Our task then is to describe. the conditions which lead to the strengthening of the b-process, for it is the growth of these affective negative after-reactions which provides the motivation for the development and maintenance of what are more commonly known as bad habits or vices. This is because "getting used to" an intensely reinforcing stimulus over prolonged or repeated presentations of that stimulus is assumed to reflect an intensification of the b-process. Thus, as the b-process grows in strength, it serves to reduce the magnitude of the-affective reaction of the UCS. Hence, changes in the pattern of affective dynamics that result from the growth of the b-process include a reduction in the intensity of the A state and an increase in the intensity and duration of the B state (See Figure 1, panel B). "Getting used to" a UCS also involves becoming increasingly reactive to the termination of the UCS. The inference which follows from this postulate is that manifest A states decrease in intensity with repeated elicitation while their consequent B states increase in intensity and duration. Thus, habituation or tolerance is seen as the natural result of the repeated use of an intensely arousing affective stimulus.

Parameters which affect the growth of the b-process include: the intensity of the UCS, the duration of UCS, the interval between UCS presentations, and the frequency of UCS presentation. Thus the opponent-process theory postulates that the b-process will be strengthened through use and weakened through disuse. In other words, repetition of an affect-arousing UCS produces an orderly growth in the negative after-reaction to that stimulus. Likewise, the intensity and duration of the UCS are positively correlated with the intensity and duration of the b-process which it engenders. Thus, Starr (1976) found a high positive correlation between the length of exposure to an imprinting stimulus (the UCS) and distress calling after stimulus removal (the B state) in *ducklings. This* implies that exercising the b-process through continuous exposure to the UCS results in 0ptimaT recruitment of the b-process.

Another condition for strengthening the b-process is an interval between UCS presentations short enough to prevent the complete decay

of the b-process. Thus, the opponent-process theory assumes that for any-particular affect-arousing stimulus, intensity, and duration there exists a critical interval or duration of decay. If stimulus presentations are scheduled at intervals longer than this critical duration no growth in the b-process is predicted and the a-process, being relatively unopposed, will continue to engender a strong A state. However, if UCS presentations are scheduled within this critical decay duration, a summation process leads to the growth of the b-process. The opponent-process theory also assumes that increases in the intensity and duration of the UCS will cause the critical decay duration to increase.

Motivational Attributes of A and B States

The opponent-process theory assumes that organisms will act vigorously and purposefully only in the presence of an aversive state. In other words we believe that only an aversive state can energize operant behavior. Although pleasure seeking behaviors do appear to occur, actually these are operant behaviors which are effective in removing or decreasing an aversive condition; which conditions may be either an A state or a B state. Thus, an organism will be behaviorally amused whenever an aversive A state is precipitated by the onset and maintenance of an aversive UCS. Also, instrumental behavior will be energized by the termination of a positively reinforcing UCS which results in an aversive B state. Although they do not energize operant behavior, pleasurable A and B states may serve as positive reinforcers. Their onset and maintenance have a selective function in that they reinforce operants upon which they are contingent and thus determine which operant will be energized when a specific aversive A or B state again occurs. In the same manner, sudden termination of an aversive A state will reinforce the operant which just preceded its termination. Thus, in the typical escape learning situation, the cue for instrumental escape *responses* is an aversive A state. Whereas in the drug dependent individual, the cue for drug seeking behavior is the aversive B state or drug craving. What this amounts to is a simple motivational system that can both amuse and select appropriate behaviors. By definition, an appropriate behavior is a response that has proven effective in preventing or terminating an aversive state. Pleasurable A and B states serve to reinforce behavior but at least initially are unable to energize behavior.

For purposes of illustration I shall describe in detail some experiments in which a pattern of habitual opiate seeking behavior was engendered in selected individual monkeys living in normal social groups under semi-natural conditions in Puerto Rico (Termes 1974). *Three* groups (12-14 animals in each) of Rhesus monkeys living in large open corrals were studied over the course of three years. A few individuals of known social rank were selected to be made dependent on an opiate drug. When an animal (e.g., an alpha male) was separated from his group and subjected to a series of gradually increasing doses of passively administered morphine (Ternes and

Colon 1976) or methadone (Jemail and Temes 1974)) it showed the following behaviors: Initial injections caused the animal to be mildly ill as evidenced by huddling, piloerection, gagging or vomiting and pupillary constriction (the A. state). Most of these somatic symptoms rapidly dropped out after a few repetitions of the drug. However, after several repetitions, a new set of signs occurred. Several hours prior to the injection on any particular day, the animal became restive, would not eat, had rhinorrhea, yawned frequently, showed piloerection, at times was hyper-reactive to external stimulation and was generally irritable, sometimes threatening and attacking other members of the group (state B). These are known withdrawal signs in monkeys. Immediately after the injection was administered, these symptoms disappeared and the animal suddenly became quite normal (e.g., the animals began to eat or to groom or be groomed by other animals).

The injection procedure entailed entering a small squeeze cage which was affixed to the corral fence and had a guillotine door which was manually closed by the experimenter after the monkey entered it. The experimenter then restrained the monkey by drawing the side of the cage toward him (the squeeze apparatus) until the animal's movement was restricted and the morphine injection was administered intramuscularly into the femoral muscle. Although initially this routine was intensely aversive to the monkey, he rapidly adapted to it. After a few trials he voluntarily entered the cage and squeezing was usually unnecessary. Frequently the animal attempted to facilitate the injection by presenting his hind parts and remaining motionless until the injection was administered. The injection procedure was systematically paired with an auditory stimulus, tape recorded music. Playing the music prior to injection caused the animal to become restless and to enter the restraining device voluntarily.

Usually the injection was given around mid-day. However, if the music was played at midnight the animal would voluntarily enter the cage even though there was no artifical light (note that rhesus monkeys are not a nocturnal species and that their night vision is no better than man's). If the music was presented by a strange experimenter the monkey would still voluntarily, although somewhat more reluctantly, enter the squeeze cage. After the monkey entered the injection apparatus, if the drug was not administered, the animal would become highly agitated and would frequently approach and attempt to re-enter the squeeze apparatus several times. If only part of the injection procedure was executed, e.g., music without opening the squeeze cage and no injection, he would show some abstinence signs, i.e., partial withdrawal such as piloerection and yawning. While being regularly maintained on the opiate, the monkey functioned normally and maintained his social rank and privileges within the group, if necessary by means of combat. However, at times he appeared to overreact to minor irritations by severely attacking other members of the group. Alternatively, he sometimes reacted to threatening or disquieting stimuli by entering the injection apparatus. After the animals had been weaned from the drug

and maintained drug-free for several months, the experimenter again played the tape recorded music and the animal showed the following signs: he became restless, had piloerection, yawned, became diuretic, showed rhinorrhea, and again sought out the drug injection.

In the latter example, morphine was the affect-arousing stimulus. We assume that the monkey experienced some pleasurable affective state after each injection. However, the primary datum of interest was the animal's rapid acquisition of highly aberrant behavior patterns. For a free-living rhesus monkey, voluntarily approaching and entering a confining squeeze apparatus, suffering to be mechanically squeezed, allowing a human to touch and to inject him with a needle, are all quite aversive and highly unlikely behaviors unless strong motivational forces are at work. These forces must certainly involve aversive control of the drug seeking response and this motivational control could only obtain from an aversive affective state (the drug craving) which is greater in magnitude than the aversiveness inherent in the sequence of instrumental behaviors which constitute injection seeking. Thus, we assume that the habitual drug-seeking behavior in this monkey is evidence of an aversive B state. The non-associative procedure of repeated morphine injections appears to have provided sufficient motivation for the acquisition of a new and different type of behavior. There are, however, other aspects of the training procedure which illustrate the operation of associative and generalization processes. We shall have occasion to return to these aspects later in the discussion.

Considering cigarette smoking and heroin abuse as forms of habitual behavior suggests that one is as addictive as the other. Although it may entail more trials to establish the habit of smoking, tolerance to tobacco, at least to its aversive qualities, appears to develop very rapidly. The initial experience with any affect-amusing stimulus is often a blend of both aversive and pleasurable feelings. Thus, the novice smoker may experience a fall in blood pressure, a slowing of the pulse, nausea, sialosis, cold sweat, pallor, and occasional nausea and vomiting. For some individuals these aversive symptoms are so traumatic that they outweigh the positive aspects of the drug effect. These people usually do not become smokers. Similar anecdotal results (e.g., Brown, 1965), have been reported for opiates. In these instances, the first or early self-administration of heroin or other psychotropic drugs, although noticeably changing the affective state of the user, also induces severe aversive symptoms such as nausea, headache and vomiting. Additionally, some few individuals find a state of altered perception to be aversive. These individuals have a very low addiction liability. According to an opponent-process analysis, the motivation to avoid these aversive components is stronger than the motivation to redose for the pleasurable components. Those individuals, however, who adapt rapidly to the aversive aspects of the stimulus, i.e., those who develop rapid tolerance to aversive symptoms are prime candidates for becoming habitual users since the pleasurable aspects of the A state will selectively reinforce the

operants which produce it. Hence, cigarette smoking will be reinforced by the pleasurable affect produced by tobacco smoke.

The pattern of affective dynamics for habitual behavior fits the data of cigarette smoking. The affect-arousing stimulus, cigarette smoke, is primarily a pleasurable UCS which engenders a positively reinforcing affective state, A, and an aversive b-process which produces a craving or cigarette hunger state, B, when the addicting elements wear off or are withheld. Initial encounters with smoking are attributable to the positive reinforcement of smoking operants by the pleasurable A state. However, with repetition of these operants, the pleasurable aspect of smoking decreases, while the aversive aspect of abstinence increases in both intensity and duration. A gradual transition takes place in which control of the smoking operant by the pleasant state A is reduced while the aversive state B gains control over the behavior. Thus, smoking changes from an appetitive response to either an escape or avoidance response. Once the metamorphosis is complete, the individual may be described as an addict since his smoking behavior is primarily determined by the presence of a strong craving or hunger for smoke, or by cues which signal the imminent onset of such a state.

THE ROLE OF ASSOCIATIVE PROCESSES

The opponent-process theory assumes that b-processes are strengthened through non-associative means and thus, at least initially, do not depend on learning or conditioning mechanisms. However, it is highly probable that conditioning will occur whenever an effective UCS appears to be contingent upon the occurrence of neutral stimuli. Such neutral stimuli are called CSs in Pavlovian terms; in opponent-process terminology they are called CSAs (for a-process).

Many types of stimuli may be conditioned to produce cigarette craving due to their inadvertant pairing with the UCS, cigarette smoke, throughout the development of the cigarette habit. During the initial exposures, the A state which is pleasurable can be associated with a variety of salient environmental cues which happen to be repeatedly paired with the pleasure of smoking. For example! satiety cues after a meal may be paired with smoking, or the stimulating affects of coffee or alcohol may be repeatedly paired with the enjoyable cigarette A state. These stimuli are CSAs. Other examples might be any boring or mildly tedious task which can be accomplished while smoking for pleasure, e.g., driving, studying, manual labor, etc.

A CS repeatedly associated with a B state will be procedurally identical to a CS in a Pavlovian backward conditioning paradigm. Such a CS functions as an inhibitor of the initial reaction to the UCS. In opponent-process parlance, such an inhibitory backward conditioning CS is called a CS_B because its occurrence is temporally contiguous with the peak of the b-process. As the smoker becomes tolerant to nicotine and the pleasurable aspects of smoking are reduced, the aversive B state will be repeatedly paired with several different

kinds of stimuli and situations. For example, an empty purse or pocket where you usually carry your cigarettes, the last cigarette in a package, places where smoking is prohibited (church, bus, theater), people that don't smoke, etc., are CS_Bs which are repeatedly paired with intense cigarette craving, state B. Both types of CSs, CS and CSB can be expectedto gain in associative strength but at different phases in the addictive cycle. However, their CPs should, in both instances, reinforce smoking operants.

Recovery from any single presentation of CSA should be biphasic while the response to any single CS_B presentation, i.e., CR_B, should be monotonic. This suggests that CS_A elicits a conditioned A state (CPA), the a-process which underlies this state will engage the b-process. Termination of CSA should be followed by a B state which will then slowly dissipate. However, the elicitation of a CR_B by the CS_B will not amuse the a-process and its termination will be followed only by the slow decay of the b-process to some baseline state.

In my monkey experiments (Temes 1974) the music, the apparatus and the injection itself are Pavlovian CSs for the pleasurable affective state elicited by the morphine UCS. However, if the CSs were presented and the drug was not administered, the animal appeared to suffer an enhancement of withdrawal. This is because the CR elicited by the CS_A is biphasic, the first phase CR which is not followed by the UCS leading rapidly to an increase in drug craving. state B. This increased-craving-then energized an increase in the rate and intensity of the drug-seeking *operant, in* this case trying to reenter the squeeze cage where morphine injections were usually administered.

During initial or early exposure to a new or unfamiliar UCS the a-process is strong relative to the strength of the b-process. The state A is quite intense at this time while the state B is rather less intense. Pairing of a CSA with UCS onset (peak of state A) should result in the rapid growth of the excitatory properties of CS and pairing CS_B with UCS offset (peak of the B state) should result in the relatively slower growth of the opponent properties of CSB. However, after the subject has become experienced with the UCS, the conditioning increments induced by pairing CS_A with the UCS will be relatively small because the A state is less intense as a consequence of the suppressive effects of the strengthened b-process. In contrast, the increments in conditioning induced by-pairing the CS_B with the peak of the b-process should be relatively larger when B is stronger. Following line of reasoning then, pre-exposure to the UCS prior to Pavlovian differential conditioning should impair conditioning of a CS_A and facilitate conditioning of a CS_B. This is a unique prediction of the opponent-process theory. It suggests that-the-degree of prior exposure to, the UCS determines, at least partially. whether the excitatory conditioning of a CS+ proceeds more rapidly than the inhibitory- conditioning-of CS-. -
Wikler (1973) pointed out that certain symptoms which accompany the

unconditioned effects of centrally acting drugs are adaptive responses to the direct effects of the drug. With repeated drug administrations, the individual is capable of developing new successive adaptations to the initial actions of the drug. Such counteradaptations may be intimately involved in the development of tolerance. According to Wikler's model, a CS which is paired with direct pharmacological reinforcement will come to elicit such a counteradaptation as a CR. This CR will be opposite in direction to the agonistic effects of the drug. These feedback mechanisms will counteract the effects of the drug and it is possible that with repeated dosing such counteradaptive responses may become intensified. These data fit the opponent-process interpretation of affective or hedonic arousal.

Conditioned Craving

Clinicians working in methadone treatment programs have often noticed that patients report "sickness" despite high maintenance levels of methadone. Similarly, it is not unusual to observe withdrawal signs, e.g., tearing, yawning and runny nose, during group therapy with detoxified addicts when drugs are discussed. Wikler (1948) noted what he thought was a conditioned withdrawal syndrome in rats. After repeated pairing of narcotic withdrawal with environmental stimuli, the environment itself appeared to acquire the power to elicit the withdrawal symptoms and signs. He proposed that in detoxified addicts (who are no longer physically dependent) relief of conditioned withdrawal symptoms may be a major motivating factor for the resumption of drug taking behavior.

Our group (O'Brien et al. 1977) has demonstrated conditioned withdrawal syndrome when sounds and smells were paired with injections of a narcotic antagonist in methadone maintained addicts. Thus, conditioned withdrawal syndrome appears to be an instance of direct condition of the B state, CR_B. However, the same result may also follow the presentation of a CSA. This may be what was observed in the morphine dependent rhesus monkey. When the morphine injections were suspended in the study described above, the animal appeared to experience abstinence agony for a few days but later he appeared to get well. His behavior as the dominant animal of the group appeared normal in every way. He no longer approached the injection environment except in a random fashion (i.e., he no longer attempted to enter the squeeze cage and sit near the guillotine door). However, after three months, when the experimenter again played the tape recorded music and opened the squeeze cage door, the animal became restless and withdrawal signs such as piloerection, yawning, and gagging were observed. Eventually the monkey, given up to 15 minutes of exposure to these stimuli, would voluntarily enter the injection apparatus. Sometimes, however, although the animal was obviously experiencing aversive symptoms, he would not enter during the 15 minute trial. If, however, an additional opportunity was given after a 30 minute period, the animal would respond by entering the injection apparatus immediately, usually, by running at full speed.

Pharmacologically there was no reason for the animal to respond in this fashion. This seems to be another demonstration of conditioned withdrawal syndrome. However, in this instance the external stimuli, which in opponent-process terms would be a compound CS_A, elicited only an attenuated CRA which was followed by a withdrawal response. The fact that the animal did not respond at first but that he almost always responded after termination of the CSA is suggestive of a biphasic CR of the type predicted by the opponent-process model.

Conditioned withdrawal probably takes on several different quantitative values. Under certain conditions it may be sufficient to energize operants which lead to redosing. At other times it may only involve conditioned craving. This craving state may not, by itself, provide sufficient motivation to reactivate the habit. However, it is conceivable that a conditioned craving response may summate with some other aversive affective state and energize a drug-seeking operant through the process of response generalization.

GENERALIZATION A AND B STATES

The common feature of all habitual behaviors is the intense craving which characterizes the B state. This craving or hunger may not be entirely specific. Although some individuals may discriminate the precise qualitative aspects of the craving, it is probable that a generalization gradient for aversive states exists. Thus, intense aversive affective states conceivably could be mistaken, substituted or generalized to the cigarette craving B state. We know that the prediction for an aversive B state is escape or avoidance operants. If an aversive state (e.g., test anxiety) is mistakenly labeled cigarette craving, this state should also energize smoking operants. Or consider the possibility that another aversive state may *potentiate* or interact with the cigarette craving b-process. This could shorten the interval between cigarettes and reduce the satisfaction which cigarettes provide. The possibility for generalization in the other direction also exists. Thus, for example, a mild cigarette craving might heighten test anxiety. Most probably there is a continual trade off in such an interaction, the direction of the generalization being determined by the relative intensities of the two states and by the array of effective operants which are available for escape. For example, as the date of an important exam draws near, the anxiety it produces increases as a function of the slope of the individual's anxiety gradient. At some point, this test anxiety may generalize to cigarette craving. If the opportunity to smoke is not available, the student's test anxiety may surge upward and energize studying behavior. If the opportunity to smoke is available, however, it is possible that smoking will occur while studying will be deferred. If however, the student has neither cigarettes nor text book, he may suffer a "nicotine fit" or very intense craving for cigarettes which will energize buying or borrowing operants. Alternatively, if he has both cigarettes and textbook, he may smoke and study concurrently.

The problem of generalization of affective states also includes response generalization. Thus, it is possible that although the student recognizes that his anxiety is primarily due to the impending exam, he nonetheless attempts to find solace in operants for anxiety reduction that have been previously reinforced, i.e., he will perform responses which have frequently terminated or avoided an aversive B state, such as smoking. Thus, the student who has not studied may smoke to reduce his anxiety during the exam since the more appropriate studying operant is no longer an available alternative. On the other hand, when smoking is not an available operant, i.e., when the smoker is "trying to quit," other types of operants which have a prior history of relieving tension, anxiety and craving may be substituted for cigarette smoking.

The naive assumption of most smokers trying to quit is that hunger for food increases when one stops smoking. Although it is true that food may begin to taste better due to a renewal of taste sensitivity, it is more likely that the increased hunger is a vivid example of generalization from state B_1, cigarette-craving, to state B, food hunger. It is also true that in some individuals eating functions as an operant for anxiety reduction and thus response generalization to eating in the abstinent smoker is highly predictable. It should be pointed out that generalization of CS_As and CS_Bs may also occur. We are unfortunately, almost totally ignorant of the laws governing the generalization of aversive A and B states. : Future research in this area could be very important to the development of a workable therapeutic regimen for- the- amelioration of habitual behavior. For example. it could be highly beneficial to engineer response generalization during critical periods of detoxification, "quitting," so that rather than smoking, some other activity which could at least partially reduce craving could occur. Such an alternative is eating. Perhaps other alternative operants such as hobbies $(\text{tennis})_1$ could be developed prior to the period of total abstinence.

The opponent-process model predicts that certain affect-amusing stimuli would be more effective than others in producing generalizations and that their relative effectiveness will depend on the time at which they occur. Thus, early in the withdrawal period we know that the B state, craving for cigarettes, is intense while the A state, pleasure for smoking, is weak. During this early withdrawal state, it should be easy to replace the positive reinforcing affect of smoking, A with another positive reinforcer, A such as highly palatable food.. The strategy is to replace the smoking-operant with the eating-operant. On the other hand, the opponent-process model predicts that at this same time, an attempt to inhibit the smoking operant by aversive control (threat of punishment), an aversive A_2, or the occurrence of an aversive B_2 (e.g., loneliness), could be counterproductive because B_1 is very strong: It will probably generalize in the wrong direction, and it is possible the state B will interact with A_2 or B_2 to produce B_11, an enhanced withdrawal reaction. Later in the withdrawal sequence, our theory predicts

that the A state, cigarette enjoyment, will increase while the B state, craving, will be weakened. At this time a viable strategy might be to attempt to substitute an aversive state such as punishment, A_2, for B_1 or tennis craving, B_2, for B_1. Thus, the abstinent smoker could perform operants to obtain relief from these conditions and perhaps as a secondary gain be relieved from cigarette craving. It is conceivable that an aversive b-process could be confused, i.e., generalized or substituted, with an aversive a-process as well as with other aversive b-processes. The possible Combinations of these substitutions are as follows:

1. An aversive a-process (a_2) could generalize to an aversive b-process (bl), e.g., if b_1 is nicotine craving and a2 is the ex-wife. Relapse in an abstinent smoker might then be potentiated by the sight or thought of the ex-wife. Another example would be nicotine craving, b_1, and test anxiety, a2. As the test draws nearer, as well as during the exam, craving for cigarettes could be heightened.

2. Aversive b_1 generalizes to the aversive a_2. For example, nicotine craving bl and headaches a_2 generalize such that abstinence from cigarettes potentiates the aversiveness of the headache and leads to aspirin taking.

3. Aversive b_1 generalizes to aversive b_2, For example, bl is taste craving and b is nicotine craving. The abstinence from smoking will probably increase the desire to eat more food. Similarly, if b_1 is loneliness it might also be intensified by cigarette abstinence and vice versa.

In any event, response generalization will probably occur especially in the situations where there is no instrumental response available to directly alleviate or terminate the aversive condition. For example, when the loneliness (B_1) state has been elicited by the death of a loved one, no direct means of terminating the B state exists and this may lead the subject to generalization to other response categories which have been selectively reinforced by termination of aversive states. Such a response is smoking. Thus one might try to protect the abstinent smoker from as many intense affective influences as possible. This actually includes two categories, aversive a-processes engendered by aversive UCSs and also intensely pleasant UCSs which elicit pleasurable a-processes and engender equally aversive b-processes upon their Termination. For example, sexual encounters-could also trap the unsuspecting cigarette habitue into smoking. This is not to suggest that one must totally withdraw from life to give up smoking but rather to indicate how very fraught with difficulty the mad to abstinence actually is.

Perhaps the most dangerous possibility, relative to relapse to smoking, is the situation in which the a_2 state is similar in quality and intensity to the aversive bl state. The best example is the generalization from coffee or alcohol effects (mild nervous excitement or tension) and cigarette craving. Smokers who drink

alcohol and/or coffee learn to compensate reciprocally from moment to moment for the stimulating effects of alcohol or caffeine on the one hand and the relaxing effects of nicotine on the other by alternately dosing first with one agent and then with the other. A smoker who wishes to give up the cigarette habit would do well to refrain from coffee and/or alcohol during the critical period of withdrawal, i.e., during the critical decay duration. A rule of thumb for the newly abstinent smoker might be to avoid or limit exposure to intense affect-amusing stimuli both pleasant and aversive.

The discussion of generalization would not be complete without mentioning the pharmacological concept of cross-tolerance and its implications for therapy. Pharmacotherapy in the treatment of habitual behavior is essentially an instance of replacement therapy where a drug or chemical compound serves as the affect-amusing stimulus which is used to terminate the aversive B state. In order to be effective in this manner, the drug must be capable of pmducing an affective resultant which can be generalized to the original affect-amusing stimulus. For example, barbiturates can be substituted for alcohol because their patterns of affect are very similar. Another requirement is that they must be capable of reducing or blocking the affective state which energizes the offending operant behavior, for example, methadone is used therapeutically to block opiate hunger. In the former instance, (alcohol - barbiturate) there is really very little if any therapeutic advantage to substituting one affect-amusing stimulus with another if they both engender similar habitual behaviors. In the latter instance (methadone for heroin), there may be some justifiable therapeutic advantage to using methadone to block heroin withdrawal. Therapeutic advantages reside in the differences as well as in the similarities between the two stimuli. Thus. in as much as the methadone a-process generalizes to the heroin a-process, it can block hemin withdrawal agony B_1,. the motivation for drug seeking behavior. At the same time, its temporal parameters such as the" critical decay duration and duration of action are such that dosages can be more easily reduced, or faded out. However, the methadone assisted detoxification strategy is an imperfect one because at therapeutic doses, which block heroin withdrawal syndrome but do not produce a "high" or euphoria, the opportunity exists for drug taking to be positively reinforced by the pleasurable A state of an opiate. Individuals presently in methadone maintenance therapy frequently take advantage of this fact. At higher doses of methadone which will block heroin high, a methadone high is produced, witnessed by the traffic in illegal methadone, and no therapeutic gain is made in terms of rehabilitation. Perhaps what is necessary is two drugs, one to block the A state and the second to block the B state. Even if these drugs were available, the risk of conditioned abstinence syndrome or craving still exists. Experience with patients in methadone maintenance attests to this fact. It is not uncommon for patients who are being maintained on relatively high doses of methadone (i.e.. high relative to their street heroin habit) to suffer withdrawal and craving reactions. Most of the craving or withdrawal

episodes are probably CRs (conditioned abstinence or conditioned craving) to CS_Bs. At present we are only beginning to understand how the various psychological and physiological components of withdrawal syndrome fit together.

Another pharmacotherapeutic approach would be to detoxify and then block the pleasurable A state pharmacologically. The naltrexone maintenance strategy is an example. Naltrexone at sufficient levels in the blood makes it impossible for a detoxified addict to get high. Detoxified addicts have no pharmacological reason to suffer craving or withdrawal agony. Thus if they take drugs it must be because of a conditioned abstinence reaction. However, should the addict attempt to get high by shooting up heroin, the whole sequence of events which normally leads to reinforcement, e.g., friends, places, "coping," cooking up, syringes, and shooting up, etc., should be subjected to experimental extinction. This type of natural extinction procedure may be the only effective extinction strategy.

At the present time the only form of pharmacotherapy which might be used for treatment of the smoking habit would be self-dosing with nicotine. At least one report (Johnson 1942) of substituting the pleasant affect of nicotine was successful. However, although the author came to prefer the repeated self-injection of nicotine to smoking, the prospect of nicotine self-injection is not a very attractive alternative for most chronic smokers. Recently, Kumar et al. (1977) reported that intravenous doses of nicotine failed to produce an immediate reduction in ongoing smoking behavior, a result that comes as no surprise to those of us who are familiar with methadone maintenance. Thus, for the time being, pharmacotherapeutic approaches to cigarette smoking do not appear promising.

It should be reiterated that the difficulties of becoming abstinent, i.e., enduring the critical decay duration without redosing, are merely part of the problem. Difficult though it may be, the critical decay duration can be breached given that an adequate degree of motivation to "quit" exists. However, data from the various subclasses of habitual behavior indicates that although the obese occasionally go on diets, although the alcoholic and heroin addict can be detoxified, and the smoker can "quit," a high degree of recidivism occurs. The relapse statistics in all cases suggest that other potent determinants of habitual behavior, most likely the generalization and associative processes, continue to exert their influence by energizing habitual operant behaviors. Thus, it is the associative and generalization processes which are primarily responsible for relapse in the addictive cycle.

THERAPEUTIC STRATEGIES SUGGESTED BY THE OPPONENT-PROCESS MODEL

A number of behavioral strategies have been indicated above which appear to follow both explicitly and implicitly from the theoretical model. In closing this discussion, it would probably be helpful to specify a list of these strategies for the treatment of the chronic

cigarette habit. Functionally they may be divided into two separate categories: pre-detoxification or strategies for "quitting' and post-detoxification or strategies for remaining abstinent.

Strategies for quitting:

1. Try cold turkey. The opponent-process model indicates that the optimal approach to becoming abstinent is to completely stop presenting the affect-amusing stimulus. In drug parlance, this is the "cold turkey" strategy, so named for the goose flesh which accompanies abstinence agony when a heroin addict undergoes unassisted withdrawal. This approach dictates that the critical decay duration and accompanying abstinence agony is endured without any additional pharmacological aid. According to the opponent-process model this is the most rapid and efficient, if not painless, method of detoxification. If one is a chronic smoker, then the fastest method of "kicking the habit" should be to just stop smoking. Since the opponent-process model indicates that the b-process is weakened through disuse, and since the b-process,is-the substrate of cigarette craving, not exercising the b-process for a period longer than the critical decay duration should dissipate the desire to smoke.

2. Try fading procedure or "cutting down." Fading is a procedure which leads to a less painful albeit longer detoxification procedure. Gradually reducing the intensity of the affect-amusing stimulus should lead to a reduction of the strength of the b-process. In a sense, the aversiveness of the withdrawal procedure is under the control of the smoker who may administer small doses of nicotine whenever craving, the B state, becomes too aversive. Although less painful in terms of intensity of aversive stimulation at any given moment, the withdrawal is prolonged and of course the risk of relapse is high since-the procedure continues to exercise the b-process. It also continues the rather questionable practice of presenting both CS_As and CS_Bs such as cigarettes, matches, buying, lighting, smelling smoke, etc., whose CRs should produce craving and also should engender the restrengthing or growth of the b-process.

Try to avoid CS_Bs which can only produce an increase in craving, CR_BS. Thus, for example, if an empty pocket or purse is a cue for cigarette borrowing, "bumming a smoke," try carrying a full unopened package in that pocket. Also try to avoid places where you have frequently experienced cigarette craving in the past such as theaters or buses. Such an environment could only be expected to intensify cigarette craving by eliciting CRBs.

4. Try to avoid intense affect-arousing stimuli both pleasurable and aversive. The process of generalization from one aversive B state to another, e.g., from loneliness to cigarette craving, could intensify or potentiate the occurrence of a smoking operant. Likewise, an aversive A state such as a headache could similarly generalize to cigarette craving and energize the smoking operant. Other such aversive states that could probably generalize to the cigarette craving

B state are emotions which are known to occur during nicotine withdrawal, for example, irritability, tension, anxiety, depression, etc. Thus, stimuli known to arouse these states should also be avoided.

5. Try to engineer response generalizations which reduce craving by making operants which lead to pleasurable A states available. For example, provide the opportunity to snack on something tasty. We how that at the start of abstinence, since the b-process is strong, the A state is weak. Thus, mildly pleasurable affect-amusing stimuli, e.g., peanuts, may outweigh the pleasurable aspects of smoking. Therefore, hopefully, the peanut craving will energize eating, not smoking, and the operant will reduce craving in general.

6. Take a vacation when you want to quit smoking. The logic of the strategy is that the CSAs and CSBs which have been conditioned during the years of smoking, and perhaps quitting, i.e., during the addictive cycle, will either not be present at all or will be significantly reduced when you are on vacation. Vacations are relaxing, or should be. However, if you usually smoke while driving, don't go on an automobile tour. Likewise, avoid shocking new experiences such as sky diving or other thrilling new encounters. Don't come back until the craving is gone, but don't stay in the withdrawal environment after it is gone. In other words, the vacation site will be the setting for experiencing the most intense cigarette craving and should therefore become conditioned as a CSB. So don't stay there too long.

Strategies for staying abstinent.

1. If possible, plan your quitting to coincide with a change of jobs or houses or cities. Take a vacation and quit, and then go to the new environment which is at least partially free of CsAs and CSBS .

2. Engineer response generalizations of the following types: Type (1), generalize aversive b_1, (e.g., cigarette craving) to aversive a_2 (e.g., test anxiety) where the most available relief-producing operant is a slow but mildly pleasant task, such as reading a book of short stories to reduce anxiety about a literature exam. Type (2)) generalize aversive b (cigarette craving) to aversive b_3 (craving to play tennis) where an available operant for the relief of the craving is an enjoyable hobby or sport.

3. Submit CS_AS and CS_Bs to experimental extinction. Recognize that the risk of relapse remains high until the power of these CSs. has been reduced or extinguished. This procedure should be employed with great caution. 'Experimental extinction is the procedure of CS presentation not followed by a reinforcement or UCS. During early extinction trials, an intensification in the rate and intensity of the CR commonly occurs. In this instance, placing an unlighted

cigarette in your mouth, drinking coffee, drinking alcohol, eating a heavy meal, etc., all constitute experimental extinction trials. The increased craving which accompanies these early trials, if it is not terminated by smoking, should dissipate on later trials. However, the danger inherent in this procedure lies in the fact that if the elevated craving energizes the smoking operant, the reacquisition of the smoking habit will occur quite rapidly. This is known as the phenomenon of savings in Pavlovian conditioning. It means that the slope of the reacquisition curve is steeper than the original acquisition curve. Another danger lies in the fact that after abstaining from smoking for a time, the A state will be very intense and this means that the first few cigarettes after relapse will be intensely enjoyable.

Thus, it seems that there are pitfalls at every turn. The smoker trying to "kick the habit" does not have an easy task. His behavior seems to be over-determined by the normal function of his homeostatic mechanisms. However, knowing what to expect and planning for it will be helpful. The opponent-process model provides a guide.

GLOSSARY

Aversive control. The use of an aversive stimulus to manage behavior in escape or avoidance training.

Backward conditioning A conditioning paradigm in which the temporal relationship between the CS and UCS is reversed such that the UCS precedes the CS.

Conditioned response (CR). A learned response to a CS that did not originally elicit the response.

Conditioned stimulus (CS). A previously neutral stimulus that has acquired the capability of eliciting a CR after having been paired with the UCS.

Conditioning An associative process by which responses are In Pavlovian conditioning, an originally ineffective stimulus comes to elicit a particular response.

Escape training. A procedure in which the performance of a response terminates the aversive stimulus.

Experimental extinction. The gradual disappearance of a conditioned response when the stimulating situation is repeated but the response is not reinforced.

Motivation. A concept used to explain the initiation, maintenance and direction of goal oriented behavior.

Operant behavior. A conditioned response that manipulates the environment in order to gain reinforcement.

GLOSSARY (Cont)

Opponent-process theory. A theory of unlearned and acquired motivation based on the assumption that animals and human beings possess affective homeostatic mechanisms, whose function is to reduce the affective response to intense emotion arousing stimuli.

Reinforcement. An agent or process that strengthens a response.

Response. Any reaction by an organism to a stimulus.

Response generalization. The tendency to make responses similar to the learned response.

Stimulus generalization. The tendency for stimuli which are similar to a training stimulus to evoke a response.

Stimulus generalization gradient. A function relating response probability or response intensity to stimuli of increasing difference from the CS.

Unconditioned response VCR). The response that is elicited by the UCS and which after training becomes associated, wholly or partially, with the CS.

Unconditioned stimulus (UCS). A stimulus that reliably elicits the response to be conditioned without any prior training.

REFERENCES

Brown, C.: *Manchild in the Promised Land,* MacMillan, N.Y., 1965.

Church, R.M., LoLordo, V.M., Overmier, J.B., Solomon, R.L. and Turner, L.H. *Journal of Comp. and Physiological Psychology,* 1966, 62, pp. 2-3.

Hoffman, H.S. & Solomon, R.L.: An opponent-process theory of motivation: III. Affective dynamics in imprinting. *Learning and Motivation,* 5:149-164, 1974.

Jemail, J.A. & Temes, J.W.: Effects of methadone dependence on the mother-infant affectional system of the rhesus monkey. *Bulletin of the Psychonomic Society,* 4:264, 1974 (Abstract)

Johnson, L.M.: Tobacco smoking and nicotine. *Lancet,* 2:742, 1942.

Kumar, R., Cooke, C., Lader, M.A. & Russell, B.M.: Is nicotine important in tobacco smoking? *Clinical Pharmacology and Therapeutics,* 1977, in press.

O'Brien, C.P., Testa, T., and O'Brien, T.J., Brady, J.P., & Wells, B.: Conditioned narcotic withdrawal in human. Science, 175:1000-1002, 1977.

REFERENCES (Cont)

Solomon, R.L. : An opponent-process theory of motivation: IV. The affective dynamics of drug addiction. In: Maser and Seligman (eds.) *Psychopathology: Laboratory Models.* Freeman and Co., 1977, in press.

Solomon, R.L. & Corbit, J.D.: An opponent-process theory of motivation: II. Cigarette addiction. *Journal of Abnormal Psychology*, 81:158-171, 1973.

Solomon, R.L. & Corbit, J.D.: A opponent-process theory of motivation: I. Temporal dynamics of affect. *Psychological Review*, 8:119-145, 1974.

Starr, M.: Imprinting: Factors Affecting the Development of Separation-Induced Distress Reactions in Newly-Hatched Ducklings. Doctoral Dissertation, University of Pennsylvania, 1976.

Temes, J.W.: Maintenance of morphine dependent rhesus monkeys living in normal social groups. *Bulletin of the Psychonomic Society*, 4 : 263, 1974 (Abstract) .

Temes, J.W. & Colon, D.: Morphine addiction of an alpha male as a leveling factor in intragroup aggression in the rhesus monkey. *Bulletin of the Psychonomic Society*, 8:245, 1976 (Abstract).

Wikler, A. : Recent progress in research on the neurophysiological basis of morphine addiction. *American Journal of Psychiatry*, 105:328, 1948.

Wikler, A.: Dynamics of drug dependence: Implications of a conditioning theory for research and treatment. *Archives of General Psychiatry*, 28:611-616, 1973.

Wikler, A.: Conditioning of successive adaptive responses to the initial effects of drugs. *Conditional Reflex*, 8:193-210, 1973.

ACKNOWLEDGMENT

I am grateful to Dr. Richard Solomon for his thorough criticism of an earlier draft of this article.

The helpful criticism of Dr. Jay Ann Jemail and Steven Seaman is also gratefully acknowledged.

AUTHOR

Joseph W. Temes, Ph.D.
Research psychologist
Drug Dependence Treatment and Research Center
Philadelphia Veterans Administration Hospital

Assistant Professor of Psychology
Department of Psychiatry
University of Pennsylvania
School of Medicine

DISCUSSION

This theory does not require that the affective arousing stimulus produce either a positive or negative affective reaction. If the A state is pleasurable, the B state will be aversive. If the A state is aversive, the B state will be pleasurable. In animals, a pleasurable induction can lead to abstinence agony when a conditioned stimulus is withheld.

One of the problems with smoking is to identify and characterize the nature of the A state. Several types of smokers have been characterized including the positive and negative affect smokers. .Some smokers claim they receive no pleasure from smoking, but that they can't live without it. That implies a very strong B state and a small A state. A period of abstinence probably increases the pleasure, though, so that even if smoking was not very enjoyable, after a day or two of not smoking, the first few cigarettes might be highly reinforcing. Unfortunately, there is a Pavlovian concept of savings involved, here such that the pleasurable aspect drops out rapidly when smoking is reinitiated.

Although the diagrams of the sequence of A and B processes were sequential, the conditioning process is not sequential, The onset of the unconditioned stimulus (A process) immediately engenders the B process, although the B process may not be seen until the stimulus is terminated. In the early stages B is weak, although it is engendered immediately by the A process. B's existence can be demonstrated by terminating A. Even when no affective state is produced by an unconditioned stimulus, the B state might still be produced. For example, drugs in very small doses may not be perceived, but with continued use a strong B state (withdrawal) can be produced despite the fact that no identifiable A state ever occurred. The theory might be modified to try to separate the affective action and reaction from what may be activity at other levels.

This theory is set up to handle acquired motivation assuming there is some affect following the unconditioned stimulus. If a stimulus does not produce an affective state it is not an affective stimulus: S. Siegel has demonstrated that rats given multiple low doses of morphine in one environment develop tolerance in that environment but

that they will have an analgesic response to the same dose when switched to a new environment. When a drug is removed from an animal that has been very gradually habituated, a physiological deprivation is produced which leads to an initial A process. There is nothing about physiological deprivation that is inconsistent with the theory because that may be the A process itself.

If this point seems unclear, it may be because disagreement was expressed among discussants as to whether or not it is possible to produce an A state without an affective process. When an organism is conditioned only motor responses are usually observed. However, if one also examines autonomic functioning it is seen that the heart rate of a dog, for example, conditions much sooner than the motor responses. Further, the heart rate conditioned response never disappears or extinguishes. This schizokinesis, as it is called, has been documented many times. Thus if an affective state is not seen it may be because all of the possible affective states were not examined. In other words, the concept of affective state must be broadened to include any organismic reaction to an introduced substance. This is not to say that the unconscious organism is having an affective state, but that any kind of response to a given stimulus is part of that response.

The issue of what constitutes an affective state was discussed at length. Some discussants felt that when trying to explain habitual behavior that involves pharmacological substances it is necessary to discuss rate functions at the receptors. It is possible to occupy receptors so slowly that you get nothing that any human could perceive . Then, by gradually increasing the doses, physiological withdrawal may be produced without ever having induced an affective state. A response to this point was that if you don't perceive it, no affective state may be there.

In cigarette smoking, there is no clearly defined withdrawal syndrome. The effects of smoking cessation are individual in perception. Dr. Ternes, in responding to these comments pointed out that physiology and behavior are not clearly defined when discussing smoking or other drugs either. There is an enormous variability in the reactions of addicts to self -injected opiates as opposed to injections from others, especially when they are aware that the injection might contain a placebo but are uncertain about it. The affective value of cigarette ,smoking is a central issue since it may have either a positive or a negative affect according to this theory. Some people have said that the only way to get ,smokers to quit is to produce a negative affect in association with smoking. Other experience, however, suggests that creating a feeling of relief may be very effective. This probably depends on where the smoker is. During early withdrawal, substituting a positive affect stimulus might outweigh the pleasurable aspects of smoking. Later, after a period of smoking cessation when the B state has became weak, it ought to be easier to substitute other types of mildly aversive stimuli.

Thomas M. Vogt, M.D., M.P.H.

REFERENCES

Siegel, S.: Evidence from rats that morphine tolerance is a learned response. *Journal of Comparative and Physiological Psychology*, 89:498-506, 1975.

Siegel, S.: Morphine tolerance as an associative process. *Journal of Experimental Psychology: Animal Behavior Procesees*. 3:1-13, 1977.

Sociocultural Factors in the Etiology of Smoking Behavior: An Assessment

Leo G. Reeder, Ph.D.

INTRODUCTION

Ever since the publication of the Surgeon General's Report on the dangers of smoking in 1964, there has been heightened interest in the health effects of smoking and a large body of epidemiologic data has documented an association between cigarette smoking and a variety of diseases including coronary heart disease, cancer, chronic bronchitis, ulcers, etc. (CDC, 1975). Indeed, it has been demonstrated by Enterline (1960)) Preston (1970a, 1970b), and ketherford (1972) that cigarette smoking is the most likely candidate to account for the major part of the widening mortality gap between the sexes. The contribution of cigarette smoking to excess widowhood in the USA has been estimated (Grannis, 1970) to be substantial. It is the most important preventable cause of premature death and illness in this country. In addition, there is a vast literature on the psychological or personality correlates of smoking behavior, sociological and social psychological factors related to smoking behavior, and, of course, attempts to control smoking behavior through a variety of behavior modification approaches. In this present paper we shall assess the state of the art of research in the etiology of smoking behavior. Thus, the focus is upon those factors related to initiation of smoking behavior rather than cessation of smoking. Problems associated with getting people to stop smoking are quite different from those involved with etiology.

Although a vast literature exists on smoking, including its health consequences, behavior modification and other intervention strategies to have people cease smoking, psychological and personality correlates, and other aspects, very little is known about the sociocultural etiology of smoking. Examination of the literature directly relevant to this paper suggests that smoking behavior is, in fact, entwined in a most complicated set of social and psychological processes. First, we shall present the major distributional features of the characteristics of this behavior by socio-demographic

background factors. Next, we shall present what, in our opinion, are the most salient findings on social and social psychological processes associated with 'smoking behavior'. Finally, we shall suggest some areas for future scientific research on the social epidemiology of smoking.

Consumption of cigarettes has increased nearly threefold since 1930 in the USA and Western European countries (with notable reductions recently occurring in certain countries). Until very recently males had been indulging in the habit with considerably greater frequency than females.

Despite anti-smoking campaigns and warnings that cigarettes are potential health hazards, the amount smoked, according to the Department of Agriculture, increased 2.1 percent in 1976 over 1975. There is a trend, however, toward the use of low-tar, low nicotine cigarettes.

SOCIO-DEMOGRAPHIC BACKGROUND CHARACTERISTICS

Age-sex trends--The data in Table I indicates that cigarette smoking the United States was less prevalent among persons 20 years of age or older in 1975 than it was in the comparable age group 10 years previously. The ten-year period between 1965 and 1975 shows a decline of 8 percent or nearly a one-fifth reduction in the proportion of smokers in this age group. these data, based upon a national telephone survey and in-person survey conducted for the National Clearinghouse on Smoking and Health, are significant in a number of ways. For although there has been a drop in the proportion of adult smokers, there has been an increase in teen-age smokers; the increase of 6 percent between 1965 and 1975 occurred during a time of decline in the rate of growth of the 13-19 year old population. In raw numbers, the increase in teen-age smokers is cause for concern. We shall return to this topic later.

Despite an increase in the adult population from 118 million to 139 million persons during the 20 year period under study, the total number of cigarette smokers declined from 49.7 million to 46.9 million (see Table II). Declines were noted for both sexes, when compared with 1965 although the drop for males is substantially greater than for females. In fact, among women, each successive generation appears to have adopted smoking at levels approaching those of men; this was particularly true for younger members of the sex with the beginning of World War II. This trend has resulted in equality of smoking rates for those who are now 21 years of age.

The National Clearinghouse on Smoking and Health has sponsored a series of national surveys that provide longitudinal data on the smoking habits of the American public, beginning with 1955. These data are summarized in Table III and Chart 1. Examination of these data may help clarify some of the confusion created by the paradox of "more cigarettes being sold than ever before" as well as more people giving up the habit of smoking than ever before, and other variations in the smoking patterns of segments of American society.

TABLE I. Estimated cigarette smokers, by age, United States, 1955, 1965, and 1975 [1]

Age Group (years)	Year	Total Population* (millions)	Cigarette Smokers** (millions)	Smokers
13-19	1955	16.0	2.2	14
	1965	24.4		14
	1975	29.5	6.0	20
20 and over	1955	104.8	39.6	38
	1965	118.0	49.7	42
	1975	138.8	46.9	34

[1]Center for Disease Control, PHS, Morbidity and Mortality Weekly Report, Vol. 26, No. 19, May 13, 1977.

* U.S. Department of Commerce, Bureau of the Census, Current Population Report. Estimates of the Population of the United States, by Age, Sex, and Race: 1970 to 1975. Series P-25.

** Based on national surveys in 1955, 1965, and 1975, sponsored by National Clearinghouse on Smoking and Health, Bureau of Health Education, Center for Disease Control. Atlanta.

TABLE II. Estimated cigarette smokers, by sex in persons 20 years of age or older in the United States, 1955, 1965, and 1975.[1]

Sex	Year	Total Population* (millions)	Cigarette Smokers** (millions)	% Smokers
Male	1955	50.9	26.5	52
	1965	65.8	30.0	53
	1975	66.1	25.9	39
Female	1955	53.9	13.1	24
	1965	61.2	19.7	32
	1975	72.7	21.0	29

'Center for Disease Control, PHS. Morbidity and Mortality Weekly Report, Vol. 26, No. 19, May 13, 1977.

*U.S. Department of Commerce, Bureau of the Census, Current Population Report. Estimates of the Population of the United States, by Age, Sex, and Race:' 1970 to 1975. Series P-25.

**Based on national surveys in 1955, 1965, and 1975 sponsored by National Clearinghouse on Smoking and Health, Bureau of Health Education, Center for Disease Control, Atlanta.

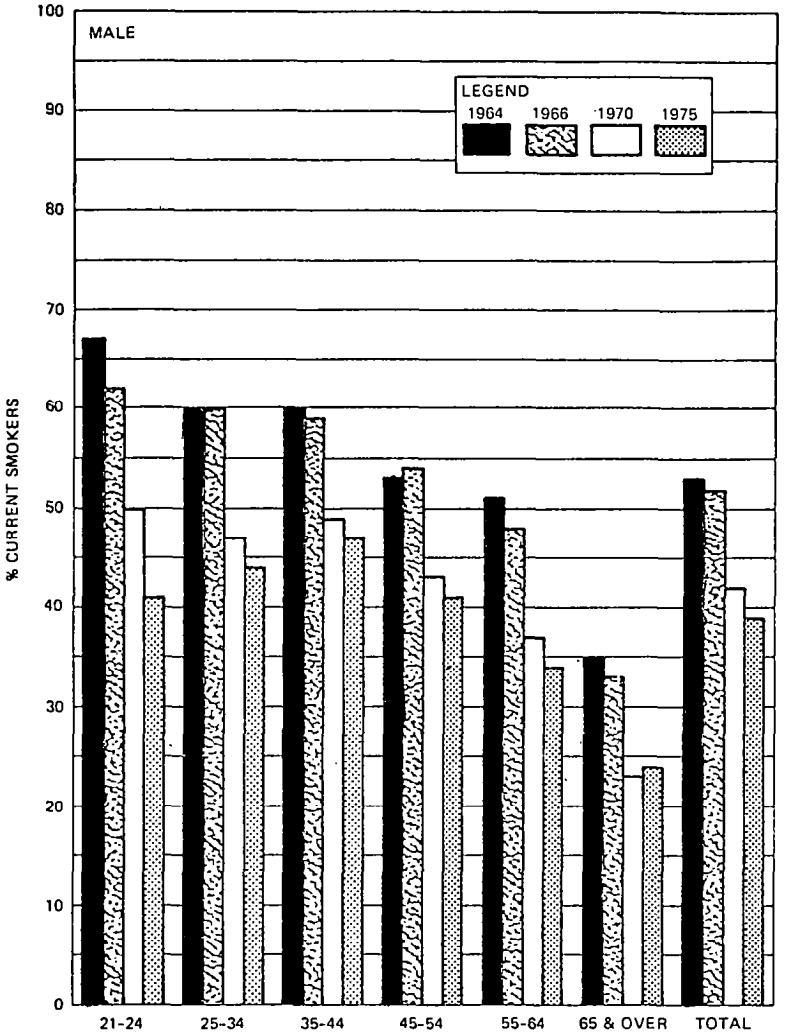
TABLE III

Estimates of Cigarette Smokers in the United States in 1955, 1965, and 1975 Among Teenagers (Ages 13-19) and Adults (Ages 20 and Over) and Separately for Males and Females'

		Total Population	Current Cigarette Smokers	Former Smokers	Never Smoked	% Smokers	Quit Rate
		Both Sexes Combined: Numbers in Millions					
Teenagers 13-19	1955	16.0	2.2	0.2	13.6	14	a%
	1965	24.4	3.5	1.4	19.5	14	29%
	1975	29.5	6.0	3.1	20.4	20	34%
Adult; 20 b Over	1955	104.8	39.6	7.5	57.6	38	16%
	1965	118.0	49.7	17.8	50.5	42	26%
	1975	138.6	46.9	29.5	62.4'	34	39%
All Persons Aged 13 *b* over	1955	120.7	41.8	7.7	71.2	35	16%
	1965	142.4	53.2	19.2	70.0	37	27%
	1975	168.3	52.9	32.6	82.8	31	38%
			MALES				
Teenagers (Boys) 13-19	1955	7.6	1.5	0.1	6.0	20	6%
	1965	12.4	2:3	0.9	9.1	19	29%
	1975	15.0	3.1	1.6	10.3	21	34%
Adults (Men) 20 b Over	1955	50.9	26.5	5.5	18.9	52	17%
	1965	56.8	30.0	12.7	14.2	53	30%
	1975	-66.1	25.9	19.0	21.2	39	42%
All Hales Aged 13 & over	1955	58.5	28.0	5.6	24.9	40	17%
	1965	69.2	32.3	13.6	23.3	47	30%
	1975	81.1	29.0	20.6	31.5	36	42%
			FEMALES				
Teenagers (Girls) 13-19	1955	8.0	0.7	0.1	7.2	9	10%
	1965	12.0	1.2	0.5	10.4	10	28%
	1975	14:5	2.9	1.5	10.1	20	35%
Adults (Women) 20 b Over	1955	53.9	13.1	2.0'	38.7	24	13%
	1965	61.2	19.7	5.1	36.3	32	21%
	1975	72.7	21.0.	10.5	41:2	29	33%
Females Aged 13 b Over	1955	61.8	13.8	2;1	45.9	22	13%
	1965	73.2	20.9	5.6	46.7	29	21%
	1975	87.2	23.9	lZ.0	51.3	27	33%

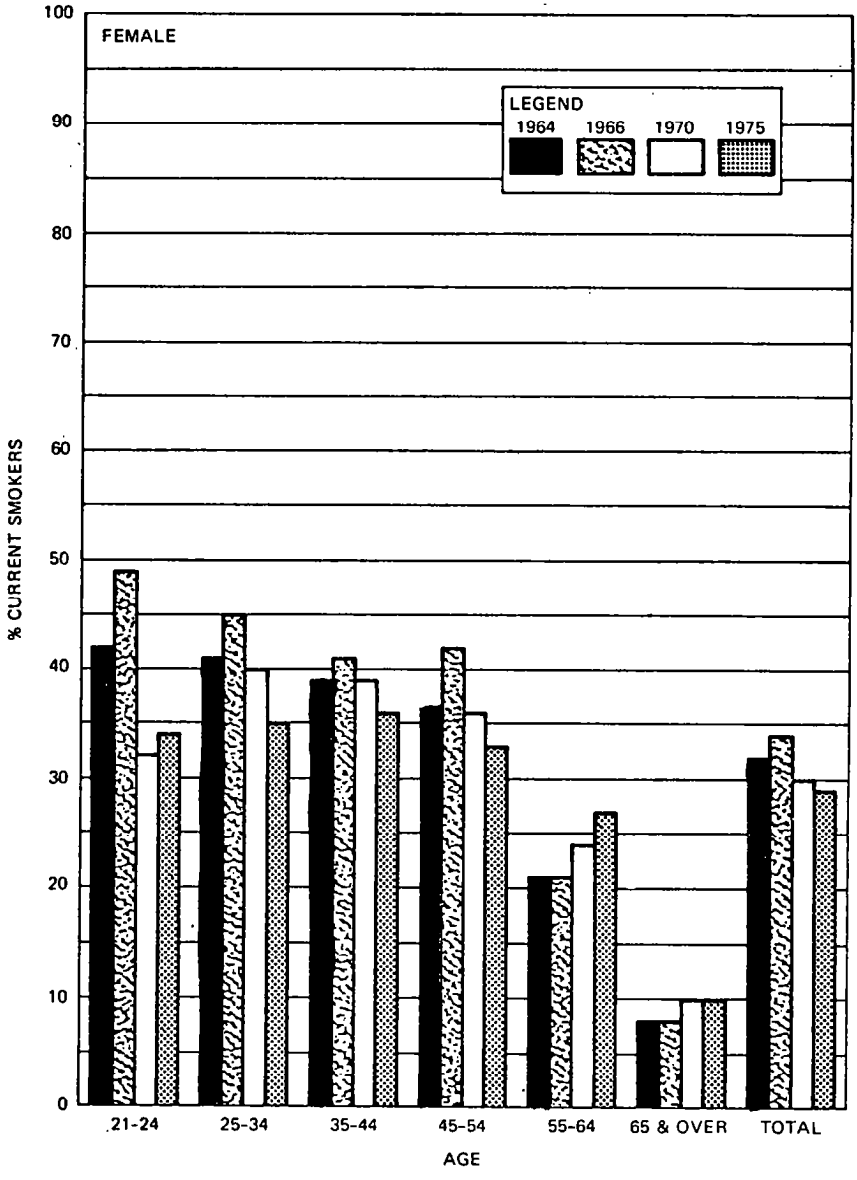
1. These data. provided by Dr. Daniel Horn, Director. National Clearinghouse for Smoking and Health, Center for Disease Control, Public Health Service, were included in a statement to the Commission on Smoking' Policy of the American Cancer Society, Los Angeles. California, Marth 22, 1977.

CHART 1
PROPORTION OF SMOKERS IN ADULT POPULATION
1964-1975



1. Source: U.S. Dept. of HEW, PHS, Center ;for Disease Control, Bureau of Health Education and National Cancer Institute, NIH. Adult Use of' Tobacco, 1975, June 1976.

CHART 1
PROPORTION OF SMOKERS IN ADULT POPULATION
1964-1975 - Continued



The picture that emerges from Table III and Chart 1 is that the adoption of the smoking habit appears to have been slowed considerably; if not halted. Among men, decreases in the proportion of smokers may be observed in every age group except the oldest. There was virtually no change in the proportion of men aged 65 and over who were cigarette smokers, probably a function of the length of time smoked It may be noted that there was a small increase in the number of smokers in the youngest age group of women in Chart 1; the smoking habits of the 21- 25 age group are, almost identical with those of men of the same age.

As we noted earlier, the decade of the 1940's witnessed a surge in adoption of cigarette smoking, especially among men. The extreme right-hand column of Table 3 shows that a large proportion of these men, now in their 50's, have given up smoking. This has had the effect of bringing down both the proportion smoking and the numbers smoking. Note especially the high proportion of former smokers. Smoking patterns of teenagers continues to present a contrary picture, especially for girls (see Table 3). Their smoking habits approximate those of teenage boys but the adoption rate has diminished. Demographically, this age group will decline in the immediate years ahead because of the declining birth rate. Thus, if there is no change in the current level of teenage smoking (one out of five), there are likely to be fewer smokers in this age group.

Marital Status--There appear to be no differences in smoking behavior between males and females that are married. The highest smoking rates are among those who are divorced or separated. This finding had been corroborated. earlier by Schwartz and Dubitsky (1968) in a study of Kaiser Health Plan members. Again, the proportion of smokers has been declining among males but increasing among females.

Socioeconomic Status (SES)--Among both males and females there is a gradient, in an inverse direction, between level of educational achievement and smoking behavior. The better *educated groups* have lower proportions of smokers; but this gradient is less sharp for women. Substantial differences are found in the most recent study of the National Clearinghouse between males and females with respect to occupation and smoking habits. For example, among males, white collar workers are much less likely to be current smokers than are those in all other occupations. This is consistent with the relationship for educational status. On the other hand, among employed women, white collar workers are more likely to smoke than are those in other occupations. Moreover, there is a greater prevalence of smoking among women employed outside of the home as compared to housewives. Again, with respect to family income, there is a disparity in smoking behavior between males and females. Men in the upper middle income categories are less likely to smoke while women in this group are more likely to be current smokers. Thus, on these two 'dimensions of SES we find that there is a topsy- turvy relationship between smoking rates and sex--inversely among men, directly among women (i.e., the higher the SES the larger the female smoker frequencies). This finding was also reported by Srole and Fischer (1973) in an analysis of the famous Midtown study of mental disorders. As these investigators noted, "in the huge corpus of

sociological research on SES, there are exceedingly few behaviors in which the' sexes show contrary trends on the socioeconomic continuum...smoking prevalence joins these rare exceptions."

ihese data lead us to speculate on the relationship between smoking behavior in females and changing sex roles. A brief review of these changes reveals the following trends: women have entered the labor force in greater numbers (Department of Labor figures show there were nearly 36 million women in the labor force in 1974, representing 46% of all women 16 and over); women have attended college and entered the professions with greater frequency; there is a general trend toward equality in virtually all domains of social and economic life. Concomitantly, the pattern of smoking behavior has also undergone changes toward equality. However, in the case of socioeconomic status the pattern is delayed, so that the smoking behavior may be perceived as in some way an indicator of increased socialpower and/or independence.

SOCIAL AND SOCIAL PSYCHOLOGICAL PROCESSES

Social Mobility--Two distinguished investigators have reported a relationship between social mobility and smoking behavior. Clausen (1968) found that men who were upwardly mobile in SES, over their parents' SES, were less likely to smoke, while men who moved down in the social order ten&d to be heavy smokers. Srole and Fischer (1973) reported on their Midtown in mgs that, "SES-mobility among Midtown men holds a discrete relationship to smoking, with upward mobility depressing the rate, so to speak, and downward mobility elevating it." The data with regard to women in the Midtown study is not clear. It is difficult to interpret these data further but they suggest directions for further research which we shall discuss presently. It appears that SES-mobility between adolescence and adulthood may partially account for the lower smoking frequencies for men found in the higher current or own-SES positions, from an analysis of the data of the two studies.

Social Alienation--One of the most powerful predictor variables in social psychological research involves the concept of alienation. It is generally recognized that there are several dimensions to this concept and in this discussion we shall not elaborate on these dimensions, except to say that we include measures of anomia (Srole, 1956), locus of control (Rotter, 1966), and powerlessness (Seeman, 1959). Essentially individuals are asked a series of "social perception" questions in each of these constructs and are given a score. Thus, in the case of anomia, it is postulated that "the more fully the person feels himself integrated in his social space the lower will be his anomia score. Conversely, the more alienated the individual is in his or her social work, the higher will be the anemia score." (Srole and Fischer, 1973). locus of control refers to the extent to which one believes that the outcome of events is contingent on his own actions or dependent on his behavior (reflecting an internal orientation) versus one who believes that the locus of control of rewards and punishments in his world are independent of his behavior (reflecting external orientation).

The data to date suggest that there is alink between anemia and smoking. Thus, the Midtown study indicates that for men on all SES-origin strata, smokers as a group have larger mean anomia scores than do abstainers. There is a reversal again for women; smokers have smaller anomia scores than do abstainers. Clearly, more research is needed to explicate these puzzling results. Parenthetically, the Midtown investigators found that those men classified as 'Well" in terms of mental health status, tended to have the smallest smoker frequencies and those who were classified as "Impaired" in mental health status had the largest frequencies, and this was' true in all SES groups. No consistent mental health status correlation was found for women and smoking. It must be noted that these are based on data obtained in the early and mid-fifties and thus must be interpreted with some caution in light of the changing trends noted earlier. Moreover, among the Midtown men large anomia scores are strongly linked to impaired mental health, to downward SES-mobility as well as to large smoker rates. Since anomia reflects a perspective of the individual, a perception that the milieu is not supportive or fulfilling of his needs and aspirations. and thus. is alienated from it. The link of smoking to the mental health, downward mobility, and anomia for women is confounded by the inconsistencies mentioned earlier.

With respect to the locus of control variable, there is no logical reason to expect a relationship between whether one initiates smoking behavior and degree of externality (Foss, 1973). .Rather, the locus of control variable might have significance with smoking cessation rates among smokers. A smoker who is externally focuse would be less likely to quit since he feels that his actions do not significantly affect his rewards and punishments. But this is not relevant as an initiation factor. Nor has powerlessness been studied. in relation to initiation of smoking behavior.

Up to this point, the discussion has focused primarily on adult smokers. To complete the picture of our current state of Knowledge concerning etiology it is necessary to turn to the data on teenagers.

SOCIAL CORRELATES OF SMOKING BEHAVIOR AMONG TEENAGERS

In this section, we shall first examine the research findings concerning socio-demographic characteristics and then consider social psychological factors related to the smoking behavior of young people. Like other variables considered in this paper, the data on the socio-demographic antecedents of smoking behavior in teenagers is of a univariate or correlational, rather than causal nature. Nevertheless, the data are suggestive for further hypotheses.

Educational Antecedents of Teenage Smoking Behavior--Several studies have now documented that there is a relationship between academic achievement during adolescence and the acquisition of the smoking habit (Matarazzo and Matarazzo, 1968; Lieberman, 1969; Newman, 1968; Bureau of Health Education, 1976). The data suggest that those academically less successful than their peers contain higher proportion of smokers than is found among their more successful classmates. These studies also indicate that smokers more often

are enrolled in vocational or non-college preparatory courses. Thus, the educational aspirations of smoking adolescents were lower than those of non-smokers. This is of special interest in terms of the data on social mobility noted earlier. For example, in his longitudinal study in Oakland, Clausen found that in examining motivation for achievement rather than achievement itself, boys who were non-smokers in 1964 had been notably high in motivation to achieve and need for recognition in 1938-39, at the time they were high school seniors (Clausen, 1968). To explain these results we might refer to the views of some researchers in this field. For example, some have postulated that smoking is a response to low achievement (Salber, 1968) and others that cigarette smoking is a form of compensatory behavior for adolescents who are not succeeding, academically or socially (Newman, 1968), or a method of coping with anxiety (Mausner and Mischler, 1967). But only a causally-oriented study could determine this relationship or uncover some as yet unknown variable leading to both low achievement and cigarette smoking (Williams, 1972).

Familial Factors--One of the most consistent findings of the four national surveys of teenage cigarette smoking conducted for the National Clearinghouse on Smoking and Health is that teenagers who live in single parent homes are much more likely to be cigarette smokers than those in households where both parents live in the home (Teenage Smoking, 1976). Another consistent finding is that parental smoking influences the adoption of cigarette smoking by teenagers; and this pattern is strongest where both parents smoke, weaker when only one parent smokes, and if neither parent Smokes, the situation is even better. Moreover, the pattern is consistent across all four surveys with respect to an older sib smoking. Both boys and girls with older siblings are more likely to smoke if one or more older siblings smokes than if none of them smokes; the difference is on the order of 3:l. Both boys and girls are more likely to start smoking if their mother smokes than if the father smokes and this is somewhat more true for girls than boys. As we might expect, the presence of both a parent and one older sibling who smokes increases the likelihood of a teenager smoking fourfold (Teenage Smoking, 1976).

Smoking Behavior of Friends--There are no studies that dispute the link between a teenager's smoking behavior and that of his friends. In the 1974 survey, among Smokers, 87% indicated that at least one of their four best friends was a regular smoker.

Won-smokers show the opposite pattern. Thus, there is no question that smokers have friends who smoke and non-smokers have friends who do not smoke. Although the reasons for this are not clear we know that' during the adolescent period, peer group relations are particularly important; there is a shift in dependence orientation and the adolescent is changing his significant others from parents to peers. Considering the data concerning the relationship between familial smoking behavior and the important effect of this role modeling on the adoption of smoking behavior by the young boy or girl, it is not surprising that they might more often select peers with similar behavior patterns. Indeed, there are considerable

data. from a variety of studies demonstrating that individuals do select others for friends and associates who are more like than unlike themselves.

Implications for Etiology--The data on parents, sibs, peers and status mobility suggest several factors influencing initiation of smoking in teenagers. First, there is role-modeling of behavior, a fundamental feature of socialization from earliest childhood through adulthood. Second, there is a tendency to express conformity. to. the behavior of significant others, such as parents, older sibs, peers, and older youngsters. A number of investigations have concluded that degree of conformity to school expectations, primarily those of student groups, determines the character reputation of the adolescent. Moreover, behavior of students is functionally related to the general social positions they occupy (including the network of interpersonal relationships) in the social structure of the school. Through the processes of social influence, the groups to which an individual belongs maintains and enforces conformity to norms.

Evidence from studies of social status and status congruence suggests some consequences of occupying lower status positions. When there is some possibility of status improvement, lower status persons will tend to express a liking for high-status ones. When upward social mobility is limited but stimulations to access are frequent, individuals may fantasize or vicariously enjoy the higher position; often, however, they may reject the occupants of higher social status positions.

In this assessment we have tried to distill the most salient findings from a variety of studies. We have deliberately excluded from consideration a large number of reports because of: inadequacies in their study design, sample population or lack of an appropriate study population, and similar reasons. In the next section, we shall take note of needed research on the sociocultural etiology of smoking behavior.

FUTURE ETIOLOGICAL RESEARCH ON SOCIOCULTURAL FACTORS

There are major gaps in our knowledge:

1. There appears to be limited research on social processes associated with familial and peer group socialization as these are related to the development and maintenance of smoking behavior. Studies completed to date suggest the need for dynamic (not static) studies focusing upon populations-at-risk for this behavior pattern, utilizing a variety of research strategies.

2. A major research thrust would systematically explore the apparent link between poor academic achievement and failure to rise in the social order as these relate to the first recommendation and smoking behavior.

3. A major flaw 'in current research literature is the relative absence of theory. With the exception of the personality research there is little of theoretical import that has guided the vast

amount of research. If we are to understand this specific behavior pattern then we must follow the scientific guidelines for all research on human behavior and use established concept and theory, and perhaps build new conceptual frameworks and theory.

4. There are a number of methodological inadequacies in the current literature. For example, with the exception of a few Studies, including those of the National Clearinghouse on Smoking and Health, the samples of populations are often biased by virtue of their being non-random, non-probability "samples." Secondly, measures of smoking behavior are not standardized. The benefits of standardization of measures was recently described by Aday and Anderson (1977) as follows: "The power to test a particular model or theory would be greatly enhanced if uniform methods of measuring the relevant concepts could be developed and data collected on them in a variety of settings. . .over time .'I Moreover, reliability and validity of items can be assessed more readily and there are economic benefits. Clearly, there are occasions, as when studying special population sub-groups with differential cultural and language problems, when standardization must be modified.

Another methodological inadequacy concerns the analytical levels of measurement generally employed in the studies reviewed. With few exceptions, the studies reviewed for this paper have involved single variable comparisons between smoking and various other characteristics of the individual and his environment. Naturally, any endeavor requires preliminary, exploratory research design. However, without adequate controls on the number of plausible rival hypotheses, further progress in smoking research will prove to be elusive. The components of smoking behavior involve a complex multi-dimensional model, including biological, social, and social psychological variables. Smoking research is now at the point where the interaction of these variables must be considered analytically. What is now needed is the measurement of several such variables simultaneously.

Finally, the methodological inadequacy of cross-sectional designs is well-known and with few exceptions (e.g., Clausen, 1968; Srole-Fischer, 1973), the literature is limited to one-shot studies. There may be interaction between smoking behavior and personal and social characteristics, such that measurement of these variables at some point beyond the initiation of smoking behavior is no guarantee that all of these characteristics were present at the initiation of smoking behavior. History and maturation may drastically change with the passage of time (Campbell and Stanley, 1963). Thus, prospective study designs are needed. These prospective designs are difficult to design, manage, and analyze properly. Nevertheless, if the knowledge base is to be developed beyond its descriptive level, this is the prescription for success.

5. Smoking behavior research must be accorded higher status by scientists than at present if we are to uncover the complicated etiological chain affecting smoking behavior. Raised priority, increased funding level and other resources are required to

stimulate the caliber and extent of research required. It would not be inappropriate to use as a model for the effort the program initiated into the social epidemiology of drug and alcohol abuse.

Each of these substances now has its own national research institute . The fact that this Conference is sponsored by the National Institute on Drug Abuse may signal a change in the appropriate direction.

REFERENCES

Aday,, L., and R. Andersen: Standardized Measures of Standard Variables. Paper presented at the Biennial Conference on Health Survey Research Methods, Williamsburg, VA, 1977.

Bureau of Health Education: Adult Use of Tobacco - 1975. U.S. Dept. of Health, Education and Welfare, Public Health Service, Center for Disease Control, June, 1976.

Campbell, D. T., and J. C. Stanley: *Experimental and Quasi-Experimental Designs for Research.* Chicago: Rand McNally College Publishing Company, 1963.

Center for Disease Control: The Health Consequences of Smoking. U.S. Department of Health, Education and Welfare, Public Health Service, 1975.

Clausen, J. A.: Adolescent Antecedents of Cigarette Smoking: Data from the Oakland Growth Study. *Social Science and Medicine,* 1:357-382, 1968.

Enterline, P. E.: *A Study of Factors Associated with Male-Female Differentials in Mortality.* Unpublished Ph.D. dissertation Department of Sociology, American University, Washington, D.C., 1960.

Foss, R.: Personality, Social Influence and Cigarette Smoking. *Journal of Health and Social Behavior,* 14, (September):279-286, 1973.

Grannis, G. F.: Demographic Perturbations Secondary to Cigarette Smoking. *Journal of Gerontology,* 25:55-63, 1970.

Lieberman Research Inc.: The Teenager Looks at smoking. Lieberman Research Inc., conducted for the American Cancer Society, 1969.

Matarazzo, J. D. and R. G. Matarazzo: Smoking. In *International Encyclopedia of Social Science,* 14:335-340, 1968.

Mausner, B. and J. B. Mischler: Cigarette Smoking Among Junior High School Students. *Journal of Special Education,* 1:61-66, 1967.

Newman, I. M.: The Social Dynamics of Cigarette Smoking in a Junior High School. Doctoral dissertation, University of Illinois, 1968.

Preston, S. H.: An International Comparison of Excessive Adult Mortality. *Population Studies,* 24:5-20, 1970a.

REFERENCES (Cont)

_____: *Older Male Mortality and Cigarette Smoking.* Berkeley: University of California Press, 1970b.

Retherford, R. D.: Tobacco Smoking and the Sex Mortality Differential. *Demography,* 9(May):203-216, 1972.

Rotter, J. B.: Generalized Expectancies for Internal Versus External Control of Reinforcement. *Psychological Monographs,* 90:1-28.(Whole number 609), 1966.

Salber, E. J.: Smoking Among Teenagers. *Bulletin of the New York Academy of Medicine,* 44-1521, 1968.

Schwartz, J. L. and M. Dubitsky: *Psycho-social Factors Involved in Cigarette Smoking and Cessation.* Berkeley, CA: The Institute for Health Research, September, 1968.

Seeman, M.: On the Meaning of Alienation. *American Sociological Review,* 24 (December):782-791, 1959.

Srole, L: Social Integration and Certain Corollaries. *American Sociological Review,* 21:709-716, 1956.

Srole, L. and A. K. Fischer: The Social Epidemiology of Smoking Behavior 1953 and 1970: The Midtown Manhattan Study. *Social Science and Medicine,* 7:341-358, 1973.

Teenage Smoking: National Patterns of Cigarette Smoking, Ages 12 through 18, in 1972 and 1974. U.S. Department of Health, Education and Welfare, Public Health Service, National Institutes of Health, 1976.

Williams, T. M.: *Summary and Implications of Review* of *Literature Related* to *Adolescent Smoking,* U.S. Department of Health, Education and Welfare, Health Services and Mental Health Administration, 1972.

ACKNOWLEDGMENT

The author is grateful for the assistance provided by Mr. Selwyn M. Waingrow of the Bureau of Health Education, Center for Disease Control, Atlanta, Georgia and to Mr. Don Gray, graduate research assistant, UCLA.

AUTHOR

Leo G. Reeder, Ph.D.
Professor of Public Health and Sociology
University of California, Los Angeles

DISCUSSION

Dr. Vogt wanted to brow whether one should not put more money into advertising than into prospective studies. Dr. Reeder said both approaches are necessary. Nevertheless, Dr. Jarvik doubts the efficacy of advertising campaigns, even when considering if it makes any difference whether the campaign is aimed primarily at inducing brand changes or at proselytizing new smokers.

Dr. Wynder remarked that the tobacco industry devotes much more time and effort to the smoking problem than does the consuming public. He went on to state that he agreed that advertising probably makes very little difference in the amount of smoking which occurs, referring to the fact that in Communist countries smoking is very prevalent without any advertising. Furthermore, in many of the European countries, such as Italy and France, there is a tobacco monopoly, no advertising, and yet smoking is quite common. However, in the United States the anti-smoking advertising on television seemed to be effective and the loss of that advertising has hurt the anti-smoking forces.

A conference participant asked whether or not the invention of the birth control pill influenced smoking because the pill was viewed as a liberating factor, and smoking has been considered a liberating factor for women. Dr. Green responded that lifestyle was a very important determining variable - that smoking went with one type of lifestyle and not with another.

Murray E. Jarvik, M.D.

Tobacco Use as a Mental Disorder: The Rediscovery of a Medical Problem

Jerome H. Jaffe, M.D.

INTRODUCTION

In this last quarter of the twentieth century, most people are not startled to learn that the excessive habitual use of alcohol and the compulsive use of opiates are listed as diagnostic entities in both the International Classification of Diseases (ICD #8), and in the second edition of the American Psychiatric Association's Diagnostic and Statistical Manual (DSM-II). To find these behaviors or conditions listed suggests that they are viewed, at least by those who put together these compendia of human afflictions, either as serious disorders worthy of note and worthy of professional attention, or at least as disorders which require some designation for purposes of statistical recording. It is, then, a curious anomaly that there is, at present, under the heading which covers drug use and drug dependencies, no listing in either manual for the excessive use of tobacco.

The omission of excessive tobacco use can hardly be attributed to the rarity of the behavior. At one time, the majority of adult American males were regular smokers. In many countries more than 40% of adults are still regular cigarette smokers. It is conceivable that the omission of smoking behavior is based on the assumtion that while the medical consequences of excessive smoking are of great significance, the behavior itself is not appropriately viewed as a drug-using behavior. This might explain its absence from DSM- II. One might suppose, then, that excessive tobacco use, although not grouped with other morbid habits or with drug-using behaviors, might at least be mentioned or listed somewhere in the overall classification of ICD; but such a supposition would be in error. Nowhere in the Tabular List of the International Classification of Diseases adopted for use in the United States in 1969 (ICDA #8) is there any mention of the substance “tobacco”.

To test the assumption that a tobacco use syndrome might have been considered too inconsequential to be included among 'the mental disorders, we need to look at what behaviors were included. We find listed in ICDA #8, under the topic of "drug dependence", dependence on cannabis (bhang, hashish, marihuana). 'as well as dependence on psychostimulants, including not only amphetamine, but also caffeine. Also included in this section (304.8) is dependence on chloroform and ether; mention is also made of glue-sniffing.

If we then check for excessive tobacco use under the category of habits, on the assumption that excessive tobacco use is viewed as a habit rather than a "drug dependence", we find in the section on mental disorders, under the topic of 'habit-like phenomena", listings of eating disturbances (306.5), including both anorexia nervosa and 'perverted appetite", habit spasms, and tics (306.2). Under 'behavior disorders of childhood" we find jealousy, tantrums and truancy (308). But we do not find mention of the possibility that the smoking of tobacco or the excessive use of tobacco is worthy of notation, at least in this part of the world compendium of medical conditions.

Neither was tobacco to be found under "drug intoxications", "poisonings", or under "drug side effects", for which there are separate listings. The section labeled 'Adverse Effects of Chemical Substances" (960-989) is subdivided into acute effects, allergic reactions, chronic effects, internal chemical bums, and late effects resulting either from absorption, injection, inhalation, or ingestion. This category is then further divided into Adverse Effects of Medical Agents and Adverse Effects of Non-medical Agents. There is no mention of either nicotine or tobacco.

One might expect to find some mention of tobacco or nicotine or, perhaps, of "tars" or carbon monoxide under the heading "Toxic Effects of Substances Chiefly Non-medicinal as to Source". This section includes substances as varied as ethyl alcohol, wood alcohol, and a variety of petroleum products, acids, and caustic alkalis. While the listing includes battery acid and starting fluid, drain cleaners. lye and tetraethyl lead, the only mention of the substance "nicotine" is found under pesticides (in section 989.3), along with organonhosphates . There is a section titled "The Toxic Effect of Carbon Monoxide" (986)) but the idea that the most common mechanism by which carbon monoxide is introduced into the body is associated with smoking of tobacco did not seem to occur to the compilers of ICDA #8. There are only four possible sources of carbon monoxide intoxication suggested: coal gas, motor vehicle exhaust gas, stove gas, and utility gas.

As a last and most fascinating observation we might note that, under the heading "Accidental Poisoning by Noxious Foodstuffs and Poisonous Plants", (E868), there is room enough to mention deadly nightshade, hemlock, noxious fungi, mushrooms, poisonous berries, shellfish, and toadstools, but the plant nicotiana tobacum does not appear.

The American Psychiatric Association's Diagnostic and Statistical Manual, second edition, (DSM-II), does mention tobacco. The word is found in an exclusionary phrase which describes drug dependence. In

DSM-II drug dependence is included under "personality disorders and certain other non-psychotic mental disorders", 'and a drug dependent person isdescribed as someone addicted to or "dependent on drugs other than alcohol, tobacco or ordinary caffeine containing beverages." Alcohol is included in a separate section; caffeine and tobacco are not.

Of course, it is possible that such committee-produced documents developed in the mid-1960's might overlook the importance of excessive tobacco use as a disorder in its own right, despite the wide publicity given to high level government reports on the adverse consequences of smoking. One cannot assume merely from the absence of the entity in a diagnostic manual that the problem was totally ignored by psychiatry, or medicine in general, or that it is still being ignored.

One way to test the possibility that psychiatry is more concerned about the problem of smoking than one might infer from an inspection of DSM-II or ICDA is to examine the textbooks and reference books commonly used in teaching psychiatry to medical students and residents. Tobacco use is not mentioned in the indexes nor discussed in the texts of Kolb's widely used *Modern Clinical Psychiatry* (Eighth Edition, 1973), Ewalt and Farnsworth's *Textbook* of *Psychiatry* (1963), nor Batchelor's revision of *Henderson and Gillespie's Textbook of Psychiatry* (1969), nor Redlich and Freedman's *Theory and Practice of Psychiatry* (1966). Other problems of drug use are discussed.-

A rapid inspection of the larger reference texts and handbooks showed that the two volume American *Handbook of Psychiatry* (Arieti 1959) contained a single paragraph about tobacco smoking as a psychosomatic disease of the respiratory tract. There was no mention of tobacco smoking or nicotine in the index of the first edition of *The Comprehensive Textbook* of *Psychiatry* (Freedman and Kaplan 1967); glue-sniffing is discussed in the section on drug abuse. Only in the last two years have tobacco use and smoking begun to appear in the multivolume reference texts. Thus, tobacco is mentioned in a passing comment on drug use in the second edition of *The Comprehensive Textbook* of *Psychiatry* (Freedman et al. 1975). Tobacco is mentioned (briefly) as a drug used for non-medical purposes in the chapter by Shick and Freedman, Research on Non-Narcotic Drug Abuse, in Volume VI of the *American Handbook of Psychiatry* (Hamburg and Brodie 1975), and there are scattered references in several chapters in Volume V (Treatment) of the same series (Freedman and Dyrud 1975)) which indicate that yoga, hypnosis, hypnotherapy, and behavioral therapy have all been used to treat making.

But, the sum and substance of this brief review is that not one of the standard works in psychiatry treats the problem of tobacco use as seriously as the problem of glue-sniffing. This attitude may be changing rapidly, but it may be of some interest to ask how and why psychiatry, which seems to to have retained an interest in other drug using behaviors, maintained such an indifference with respect to tobacco use.

THE HISTORY OF TOBACCO USE AS A DISORDER

It is not certain who first made the observation that some people who begin to use tobacco eventually seem unable to stop using it, or who first drew the conclusion: that tobacco use 'induces some change in the user's ability to control his or her own behavior with respect to the substance. According to an early history of smoking (Corti 1932), one of the first 'Europeans to comment on this aspect of tobacco use was Bishop Bartolome de las Casas, a missionary who accompanied the Spanish to the Americas. In 1527, de las Casas described the way in which the Indians sucked in the smoke from a burning bundle of dried leaves, or "tobacos", and apparently experienced a sense of drowsy intoxication that was accompanied by a decrease in fatigue. He then noted that the use of these "tabacos" had

> been adopted also by the settlers in this region. I have seen many Spaniards in the island of Hispaniola who use them, and who, when reproached for such a disgusting habit, replied that they found it impossible to give it up. I cannot understand what enjoyment or advantage they &rive from it (de las Casas, in Corti 1932, p. 42-43).

It is difficult to know how to interpret a statement to one's Bishop that a behavior of which he disapproves is beyond one's control. However, there is at least the possibility that some of these early smokers exhibited a behavior which we might describe, using present day terminology, as a form of drug dependence or addiction. The similarities between the use of tobacco and the use of alcohol were noted, in fact, soon after the introduction of tobacco into Europe. One of those who wrote about tobacco use soon after its use had become customary in England was King James I, who observed:

> ... many in this kingdome have had such a continual1 use of taking this onsavorie smoke, as now they are not able to forbeare the same, no more than an olde drunkard can abide to be long sober, without falling into an uncurable weakness and evil1 constitution ... *(King* James *I, A Counterblaste to Tobacco,* 1604, quoted in Corti 1932, p. 80).

King James further believed that drunkenness:

> was the root of all the vices and among its evil consequences was an unquenchable &sire for tobacco. Now just as hardly anyone succumbs to the vice of drinking on his first visit to the tavern ... but yields gradually to the lure of intemperance, till, after 'a long course of bestial indulgence. he comes' to rejoice in his servitude, so the smoker at first hesitates between his liking for the reek of tobacco and his natural shrinking from so unnatural a habit, but soon become so obstinately addicted to it that he would sacrifice every pleasure in life rather than give it up ... (king James cited by Corti 1932, p. 80-81).

Writing at about the same time, Sir Francis Bacon said:

> ... In our time the use of tobacco is growing greatly and conquers men with a certain secret pleasure, so that those who have once become accustomed thereto can later hardly be restrained therefrom (Bacon, *Historia vitae et mortis*, quoted in Corti 1932, p. 94).

Tobacco use increased despite such observations, and it is clear from certain writings that the notion of tobacco use as a behavior analogous to alcoholism and opiate use was still to be found among members of the medical orofession more than 200 years later. In 1853 a prize was offered for' the 'best tract" on "The Physical and Moral Effects of the Use of Tobacco as a Luxury", and the three prize winning essays were published as a monograph. One physician respondent wrote:

> Most emphatically does tobacco enslave its votaries . . . It is the uniform testimony of those who have attempted to emancipate themselves from their attachment and bondage to tobacco, that to break the chains in which they are bound, requires the sternest efforts of reason, conscience, and the will (Harris 1853, p. 21).

"The slave of tobacco", wrote another,

> is seldom found reclaimable . . . I Know full well the difficulty of reclaiming the drunkard. But the tobacco drunkard is still less hopeful. I have, indeed, in the course of the last quarter of a century, met with instances of entire emancipation, but they have been few and far between (Alcott 1853, p. 23).

In evaluating the significance of these comments, we need to keep in mind that the prizes were offered by a group within the temperance movement in the United States, and that at the time the essays were written alcoholics were still called "drunkards" and the behavior was seen as being a moral problem more than a medical one.

Perhaps a reaction to such moralistic attacks on smoking was to be expected, and between World War I and World War II, a dramatic change took place. Whether the attitude of medicine played a role in this change is uncertain, but in 1922, A. A. Brill, one of the pioneers of the psychoanalytic movement made the statement that:

> one is justified in looking with suspicion on the abstainer; most of the fanatical opponents of tobacco that I have known were all bad neurotics (Brill, cited by Tamerin and Eisinger, 1972, p. 1224).

Writing in 1924, the German pharmacologist, Louis Lewin (called by some "the father of psychopharmacology"), said of tobacco:

> It must be pointed out that the attraction of tobacco is not exercised with that vigour and inexorable constraint which we have remarked in the case of the narcotic substances . . . If the use of tobacco has to be stopped for medical or other reasons, no suffering of the body or morbid desire for the drug appears. The consumption of tobacco is an enjoyment which man is free to renounce, and when he indulges in it he experiences its benevolent effects on his spiritual life . . . Smoking does not call forth an exaltation of internal well being as does the use of wine, but it adjusts the working condition of the mind and the dispsoition of many mentally active persons to a kind of serenity or "quietism" during which the activity of thought is in no way disturbed . . . (Lewin 1924, p. 310, 1964 edition).
>
> ... It is also well known that inveterate smokers are not exempt from the symptoms of acute intoxication if they overpass the limit of toleration. It is, moreover common knowledge that the use of tobacco for smoking and chewing does not necessitate a progressive increase of the dose as in the case in other toxic substances and that the symptoms due to withdrawal of tobacco, if they occur at all, are easily overcome. These latter consist of an extreme feeling of discomfort and, eventually bad humour and dejection. It is very exceptionally that graver symptoms occur (Ibid, p. 313).

In 1926, Sir Humphrey Rolleston, who chaired the committee which established the British practice of having the medical profession provi& drugs for individuals addicted to opiates, summed up the issue of smoking as an addiction by observing that:

> This question turns on the meaning attached to the word "addiction" and may therefore be a verbal problem. The Ministry of Health's Departmental Committee on Morphine and Heroin Addiction (1926) defined an addict as "a person who, not requiring the continued use of a drug for the relief of the symptoms of organic disease, has acquired, as a result of repeated administration, an overpowering desire for its continuance, and in whom withdrawal of the drug leads to definite symptoms of mental or physical distress or disorder " That smoking produces a craving for more when an attempt is made to, give it up . . . is undoubted, but it can seldom be accurately described as overpowering, and the effects of its withdrawal, though 'there may be definite restlessness and instability; cannot be compared with the physical distress caused by withdrawal

> in morphine addicts. To regard tobacco as a drug of addiction may be all very well in a humorous sense, but it is hardly accurate (Rolleston 1926, p. 963).

One might have assumed that Freud's compulsive cigar smoking would have generated considerable interest among psychiatrists. Instead, there appears to have been a prolonged period of relative disinterest, despite the obvious concern of psychiatry with other forms of excessive drug use. One of the few exceptions was Bergler's (1946) paper on compulsive smoking, in which he tried to separate normal smoking from excessive use and compulsive use. (On the basis of five cases *seen* in psychoanalysis, Bergler concluded that compulsive smokers were "psychic masochists").

It would be inappropriate, however, to conclude that no one was interested in smoking as a disorder. There were, in fact, hundreds of papers on tobacco use and smoking written over this period of time. These are ably summarized in the classic reviews of Larson et al. 1961, and Larson and Silvette 1968, 1971. The point to be noted here, however, is that those who made the decisions about what is and what is not a serious disorder or a problem of drug dependence continued to view smoking, even heavy smoking, as a phenomenon to be sharply separated from excessive use of alcohol or opiates. For example, prior to 1973, the prestigious World Health Organization Expert Committee on Problems of Drug Dependence consistently omitted mention of tobacco, use as a problem in discussions of other forms of drug dependence. It may be that the close articulation between the actions of such groups and the legal restriction on the drugs they discussed led them to believe that the inclusion of tobacco as a substance to which dependence could develop might weaken recommendations for controlling other substances. But it is also possible that they fervently believed that *there was a very sharp distinction* between tobacco use and *other* drug-using behaviors. Thus, the late Dr. Maurice Seevers, one of the giants in the field of pharmacological research on drug dependence, and a major force on the WHO Expert Committee, took the position that "by no stretch of the imagination can either nicotine or caffeine conform to any accepted definition of addiction" (Seevers 1962). It is likely that Seevers was greatly influenced by an incredibly laborious experiment on nicotine dependence which was carried out in his own laboratories, but was never reported in any archival journal. In a discussion at a symposium in 1968, in which he was describing the failure of certain drugs to induce physical dependence, Seevers commented:

> If nicotine is given to the monkey intravenously every hour in a dose of 2 mg per kg for a total of 48 mg per kg intravenously over a period of 24 hours for as long as 2 months, no evidence whatsoever of physical dependence or any evidence of excitability is observed when this drug is withdrawn (Seevers: 1968).

In its landmark 1964 report, *Smoking and Health,* the Advisory Committee to the U.S. Surgeon General leaned heavily on the absence of any findings of physical dependence on tobacco in adopting the position of the WHO Expert Committee on Drug Dependence that tobacco was not

appropriately viewed as a dependence-producing substance. Another issue which may have played a role in perpetuating the position that tobacco use was 'distinct from other drug-using behaviors was the belief expressed by some experts on Smoking that one of the factors which may inhibit people who want to stop smoking from doing so is an overestimation of the difficulty in stopping. From such a viewpoint, to include smoking with other drug dependencies might inhibit some people from starting to smoke, but might also make many smokers hesitant to try stopping.

Just as it is not clear how tobacco use came to be excluded from discussions of drug dependence, it is difficult to point to any one event. or paper, or statement that-led to the effort to reassess its status. . In 1961, Larson, Silvette and Haag published their classic review, *Tobacco.* In 1963, Knapp and his co-workers published a paper in the American Journal of Psychiatry on the addictive aspects of heavy smoking and pointed out several psychological and physiological withdrawal phenomena. In 1965. W. Russell Brain. in writing on drug addiction; expressed the view that tobacco and alcohol were both drugs of dependence. In the third edition of Goodman and Gilman's *The Pharmacological Basis of Therapeutics,* mention of tobacco as a form of compulsive drug use was included in the chapter, "Drug Abuse and Drug Addiction" (Jaffe 1965). In the fourth edition of that text, the absence of public perception of tobacco as an addiction, in the face of everyday examples of the compulsive use of tobacco, was referred to as an illustration of the inconsistency in our use of terms such as "addiction" and "dependence" (Jaffe 1970). Ulett and Itil (1969) published their observations on changes in the electroencephalogram during smoking deprivation. Russell's paper on tobacco use as a form of dependence was published in 1971 and subsequently was introduced to a wide public by Brecher, who quoted from it -liberally in *Licit and Illicit Drugs* (Brecher 1972). By 1972. the .National Commission of Marihuana and Drug Abuse had been obliged to deal with tobacco in its comprehensive report, Drug use in America; and in 1973 and 1974, expert committees of the WHO made the following comments on tobacco, which, while still hesitant, are in striking contrast to the previous views of Dr. Seevers. Tobacco, they state is:

> clearly a dependence-producing substance with a capacity to cause physical harm to the user, and its use is so widespread as to constitute a public health problem (WHO 1973).

However, these committees go on to say that tobacco produces:

> relatively little stimulation 'or depression of the central nervous system, or disturbances in perception, mood, thinking, behaviour, or motor function. Any such psychotoxic effects produced by tobacco, even when it is used in large amounts, are slight compared with those of the types of dependence-producing drugs listed above. It is for this reason that dependence on tobacco - perhaps the most widespread form of drug dependence - is not given specific attention in this report (WHO 1974).

In the absence of sinister forces working against such an eventuality, it was inevitable that the question of tobacco use, per se, as a disorder would arise among those charged with the perio
the ICD and DSM. By 1975, the decision had been made to. include tobacco use in the Ninth Edition of the ICD, in the section on mental disorders. But even in the process of doing so, society's peculiar ambivalence about tobacco has made itself apparent. In ICD #9 draft, 304, "drug dependence" is-briefly defined in the standard manner 'as:

> A state, psychic and sometimes also physical, resulting from taking a drug characterized by behavioural and other responses that always include a compulsion to take a drug on a continuous or periodic basis in order to experience its psychic effects, and sometimes to avoid the discomfort of its absence. Tolerance may or may not be present. A person may be dependent on more than one drug.

Included here are dependence of the morphine type, barbiturate type, cannabis, LSD, absinthe and glue-sniffing. But there is a specific note which states that tobacco dependence is excluded. Tobacco dependence is included under a separate section titled "non-dependent abuse of drugs" (305), for which the following definition is offered:

> [This] Includes cases where a person, for whom no other diagnosis is possible, has come under medical care because of-the maladaptive effect of a drug on which he is not dependent (as defined in 304) and that he has taken-on his own initiative to the detriment of his health or social functioning.

Tobacco dependence is specifically listed as 305.1, with the following, somewhat self-contradictory statement:

> [This includes] cases in which tobacco was used to the detriment of a person's health or social functioning or in which there is tobacco dependence. Dependence is included here rather than under 304 because tobacco differs from other drugs of dependence in its psychotoxic effects.

It seems that the ICD is now willing to include tobacco use as a problem worthy of notation for statistical purposes; it is not clear whether there is a willingness to acknowledge its tendency to produce dependence. It is obvious that a distinct separation is suggested between tobacco and other commonly used drugs which lead to dependence.

By 1975 it had become evident that the forthcoming revision of the American Psychiatric Association's Diagnostic and Statistical Manual, DSM-III, could not continue to ignore excessive or compulsive tobacco

using behavior. Therefore,' the' problem was considered. by the Advisory Committee on Drug Use' Disorders to. the 'Task 'Force on Nomenclature and Statistics of the APA. -The decision to include some forms of tobacco use- in DSM-III was far less difficult than the. problems of where to include them and how to differentiate those varieties of tobacco use that do not represent "mental disorders" from those that do.

After considering the criteria used for defining other drug use disorders, as well as other habits and compulsions, the committee agreed that when a smoker expresses concern and distress about his or her inability to control tobacco-using behavior (usually cigarette smoking), then this, in itself, is a sufficient criterion to establish the diagnosis of a tobacco use disorder. There was considerable controversy, however, about whether even heavy smoking could be properly viewed as a mental disorder if, despite the medical risks, the smoker states that he or she is satisfied to be a smoker, since, unlike the ingestion of alcohol, tobacco use does not produce obvious disability or impairment of mental function. Over the short run, smoking usually causes neither distress nor disability. In this sense, chronic use of tobacco is not equivalent to a state of chronic intoxication. In short, the same issues that concerned Rolleston and Seevers and the World Health Organization Expert Committee on Drug Dependence were still unresolved.

That heavy smoking predisposes to a wide variety of somatic diseases is no longer seriously contested. (The serious adverse effects of smoking on health have been discussed at length elsewhere.) However, the Advisory Committee to DSM-III believed that a behavior that merely predisposes to other medical illnesses is not necessarily, in and of itself, a disease or a disorder. The logic of this position is unassailable. We certainly would not want to consider skiing as a mental disorder, although it clearly raises the likelihood of developing several well-defined orthopedic disorders. Risk taking, per se, is not a mental disorder.

Thus, in the meetings of the Advisory Committee to consider the inclusion of tobacco use in DSM-III it was argued that tobacco, even heavy tobacco use, is a disorder only if and when it meets additional criteria. In this sense, the Americans who worked on DSM-III seemed to agree in part with the compilers of ICD about the appropriateness of separating tobacco use from the use of drugs ,like alcohol or opiates. One such additional criterion was that the user should express concern or distress at the inability to stop. The difficult question was how to view the user who denied such concern.

One line of reasoning suggested that we follow the precedent used for drugs such as alcohol and opiates and consider that whenever tobacco use is associated with physical dependence on tobacco, the behavior leading to this state should be viewed as a disorder. The rationale was that dependence (the need to take the substance to avoid withdrawal) represents an organismic dysfunction. In this sense, it is not necessary for the individual to express concern or dissatisfaction with the state of physical dependence, any more than an alcoholic need express dissatisfaction with physical dependence on alcohol in order to

be viewed as having a behavioral or mental disorder.

However, major problems arise when this proposed criterion is examined further. Conditions orbehaviors are not generally regarded as mental disorders if there is widespread social support for regarding the conditions as "normal". As has been pointed out in other papers, 'physical dependence on nicotine appears to be variable in its intensity, but it is probably present to some degree in all smokers who consume a pack or more of medium to high nicotine cigarettes per day. Because this is so, subclinical withdrawal phenomena, experienced by the smoker as restlessness, increased irritability, decreased capacity to concentrate, and a need for a cigarette, probably have their onset, in many smokers, within an hour or two after the last cigarette, a time course consistent with the very short (20-30 minutes) biological half -life' of nicotine. Society does not at present regard these subclinical withdrawal phenomena as signs of "illness". Furthermore, since tobacco is so readily available, more severe withdrawal phenomena usually occur only if the user decides to stop or because of external circumstances (such as illness) is forced to do so. Even when severe withdrawal phenomena do occur - irritability, inability to concentrate, drowsiness, etc. - society generally has taken the view that such signs and symptoms are "normal" and to be expected under the circumstances. While tremulousness following abrupt withdrawal of alcohol, or autonomic disturbances from withdrawal of opiates are equally to be expected under the circumstances, they are, for some reason, not regarded as equally "normal under the circumstances" and are viewed as representing signs of illness. While it may be true that for the overwhelming majority of smokers tobacco withdrawal is not as disabling as withdrawal from alcohol or opioids. and while there are no known deaths from nicotine withdrawal symptoms, the tendency to define nicotine dependence as essentially inconsequential for all smokers is probably not well grounded in clinical observations. Nevertheless, the prevailing attitude is that smoking can be discontinued without any serious adverse effects. In this instance transient discomfort and distress are simply dismissed.

However, even if society as a whole were to accept the view that the repetitive seeking of tobacco involves some element of physical dependence and the behavior is, therefore, the manifestation of a disorder, it is quite likely that the average smoker who is not experiencing tobacco-related medical problems would simply reject the label that would be thrust 'upon him or her.

In short, the use of strict physiological criteria to separate the occasional use of tobacco from a tobacco use disorder was rejected by the subcommittee of DSM-III because society would reject such a definition, smokers would reject such a definition, and-because the subtler manifestations of physical dependence are too difficult to define. This reasoning also contributed to the decision to use the term "tobacco use disorder", rather than "tobacco dependence", in DSM-III.

All of these considerations went into the decision to develop criteria for tobacco use disorder which differed from those applied to the use of other drugs, such as alcohol or opioids; where the regular use of high doses directly causes behavioral and social disability. Under

the criteria finally developed for DSM-III, Tobacco Use Disorder would exist when:

1) The patient experiences distress at the need to repeatedly use tobacco ; or

2) Both (a) and (b) :
   (a) In the judgment of the diagnostician, the individual manifests a serious medical disorder in which tobacco smoking is a significant etiological or exacerbating factor; and
   (b) There is evidence of current physiological dependence on tobacco or nicotine either by the presence of the tobacco withdrawal syndrome or by the daily intake of nicotine of sufficient magnitude that the diagnostician judges that the withdrawal syndrome would ensue if the intake of tobacco stopped for more than 24 hours.

We recognize that these criteria lead to certain unusual inconsistencies . For example, an individual who is a heavy smoker and who has no tobacco-related medical problems may deny concern about smoking. That individual, at that moment, does not have Tobacco Use Disorder. Should the same individual exhibit concern about an inability to stop smoking, the behavior would meet the criteria for a disorder. Denials of concern by individuals dependent on alcohol or opiates are disregarded in the diagnosis of alcoholism or opiate addiction because of the direct association of high doses or dependence with disability. Indeed, denial is often viewed as a hallmark of drug dependence problems. Under the criteria to be used in DSM-III there will be many heavy smokers who are obviously physiologically dependent on tobacco but who deny concern or interest in cessation and who will not be diagnosed as having Tobacco Use Disorder.

However, on the basis of current medical knowledge, the individual with peripheral arterial disease who is experiencing pain on exercise or other disability, and is seen as being physically dependent on nicotine but who is unable or refuses to follow a directive to cease smoking, will have the Tobacco Use Disorder - a mental disorder - regardless of whether that individual expresses concern about smoking or concedes the connection between the smoking and the disease. There is, of course, a very fine line separating this position from one in which failure to comply with medical advice could constitute a mental disorder. The use of this second criterion is based on the analogy with other forms of drug dependence in which it is-implied that if an individual with a drug related medical problem (e.g., cirrhosis) were not drug dependent, both the drug using behavior and the expressed concern would be different.

The criteria used here do have the advantage of allowing us to deal with the advent of very low tar, low nicotine cigarettes which are believed to pose a much reduced probability of 'inducing medical illnesses (see Gori 1976). The use of such cigarettes will obviously affect both the' user's perception of the importance of, stopping the use of tobacco, and the likelihood that smoking-related diseases will develop. Such individuals may develop mild physical dependence on

nicotine, but may also incur a much reduced risk and may deny any serious concern about their behavior. Under the criteria proposed, such smokers do not have a mental disorder.

This attempt at developing operational criteria for Tobacco Use Disorder encountered some criticisms, some of which indicated unfamiliarity or misperception of the rationale for developing diagnostic categories in the first place. For example, one psychiatrist (who hastened to identify himself as a user of tobacco) assumed that the inclusion of an entity within DSM-III was tantamount to suggesting that the treatment of such an entity ought to be covered by health insurance, or that those who had such a disorder ought to be treated by members of the medical profession and to be hospitalized if necessary (see Proctor .1977). There is, of course, no necessary or invariable relationship between a given diagnostic entity and the determination that the entity justifies some help or intervention, nor who should provide such help, nor how the costs of such help should be handled. To define a mental disorder does not imply the need for a psychiatrist nor that society should assume the costs of intervention.

Although in presenting these criteria for Tobacco Use Disorder we have not used the terms "addiction" or "compulsive drug-using behavior", it is common knowledge that, for some individuals, the involvement with and need for tobacco smoking appears to be every bit as *intense* and difficult to control as other drugs of addiction or dependence. We cannot tell whether the inclusion of Tobacco Use Disorder in DSM-III will have any positive impact on cigarette smokers or society. It may not even have any impact on psychiatrists. But, it is at least some small satisfaction to be able to say that 400 years after Bishop Bartolome de las Casas first wrote about the peculiar behavioral syndrome associated with tobacco use, the medical profession now also acknowledges its existence,

REFERENCES

Alcott, W. A.: *The Physical and Moral Effects of Using Tobacco as Luxury.* Wm. Harmed, New' York, 1853.

American Psychiatric Association Diagnostic and Statistical Manual of Mental Disorders, Second Edition (DSM-II). American Psychiatric Association, Washington D.C., 1968.

American Psychiatric Association Diagnostic and Statistical Manual of *Mental Disorders, Third Edition* (DSM-III). American Psychiatric Association, Washington D.C., in preparation.

Arieti, S., ed.: *American Handbook* of *Psychiatry,* Volumes I and II. Basic Books, Inc., New York, 1959.

Arieti, S. and Brody, E. B., eds.: *American Handbook of Psychiatry,* Second Edition. Basic Books, Inc., New York, 1974.

Batchelor, I. R. C.: *Henderson and Gillespie's Textbook* of *Psychiatry.* Revised by I. R. C. Batchelor, Tenth Edition. Oxford University Press, New York, 1969.

Bergler, E.: Psychopathology of compulsive smoking. *Psychiatric Quart.,* 20:297-321, 1946.

Brain, W. R.: Drug addiction. *Nature,* 208:825-887, 1965.

Brecher, E. M.: *Licit and Illicit Drugs.* Consumers Union, Mt. Vernon, New York, 1972.

Corti, E.: *A History of Smoking.* Harcourt, Brace and Company, New York, 1932.

Ewalt, J. R. and Farnsworth, D. L.: *Textbook of Psychiatry.* McGraw-Hill Book Co., Inc., New York, 1963.

Freedman, A. M. and Kaplan, H. I.: *Comprehensive Textbook of Psychiatry,* First Edition. Williams and Wilkins Company, Baltimore, 1967.

Freedman, A. M., Kaplan, H. I., and Sadock, B. J.: *Comprehensive Textbook* of *Psychiatry,* Second Edition. Williams and Wilkins Company, Baltimore, 1975.

Freedman, D. X. and Dyrud, J. E., eds.: *Treatment.* Volume V, *American Handbook of Psychiatry,* Second Edition (Arieti, S. and Brody, E. B., eds.). Basic Books, Inc., New York, 1975.

Gori, G. B.: Low risk cigarettes: a prescription. *Science,* 194:1243-1246, 1976.

Hamburg, D. and Brodie, H. K., eds.: *New Psychiatric Frontiers.* Volume VI, *American Handbook of Psychiatry,* Second Edition. (Arieti, S. and Brody, E. B., eds.). Basic Books, Inc., New York, 1975.

Harris, E.: *Tobacco: The Effects of its Use AS a Luxury on the Physical and Moral Nature of Man.* Wm. Hamed, New York, 1853.

International Classification of Diseases, Eighth Revision. (Adapted for use in the United States). U.S. Department of Health, Education and Welfare, USPHS Center for Health Statistics, PHS Publ. #1693. U.S. Government Printing Office, Washington, D.C., 1969.

International Classification of diseases, *Ninth Rev.* (In preparation).

Jaffe, J. H.: Drug Addiction and Drug Abuse. In, *The Pharmacological Basis of Therapeutics,* Third Edition [Goodman, L. S. and Gilman, A., eds.). Macmillan, New York, 1965.

Jaffe, J. H.: Drug Addiction and Drug Abuse. In, *The Pharmacological Basis of Therapeutics,* Fourth Edition (Goodman, L. S. and Gilman, A., eds.). Macmillan, New York, 1970.

Knapp, R. P., Bliss, C. M., Well:,, H.: Addictive aspects in heavy cigarette smoking. *Am J of Psychiatry,* Volume 119, pp. 966-972, 1963.

Kolb, L. C.: *Modern Clinical Psychiatry,* Eighth Edition. W. B. Saunders Co., Philadelphia, 1973,

Larson, P. S., Haag, H. B., Silvette, H.: *Tobacco: Experimental and Clinical Studies.* The Williams and Wilkens Co.,, Baltimore, 1961.

Larson, P. S. and Silvette, H.: *Tobacco: Experimental and Clinical Studies, Supplement 1.* The Williams and Wilkens Co., Baltimore, 1968.

Larson, P. S. and Silvette, H.: *Tobacco: Experimental and Clinical Studies, Supplement 2.* The Williams and Wilkens Co., Baltimore, 1971.

Lewin, L.: *Phantastica: Narcotic and Stimulating Drugs.* E. P. Dutton & Co., Inc., New York, 1964. (Published originally in German in 1924.)

National Commission on Marihuana and Drug Abuse. Second Report. *Drug Use in America: Problem in Perspective.* U.S. Government Printing Office, Washington, D.C.; 1973.

Proctor, R. C.: Letter to the editor. *Psychiatric News,* May 20, 1977.

Redlick, F. C. and Freedman, D. X.: *The Theory and Practice of Psychiatry.* Basic Books, Inc., New York, 1966.

Rolleston, H.: Medical Aspects of Tobacco. *The Lancet,* London, 1:961-965, 1926.

Russell, M. A. H.: Cigarette smoking: a natural history of a dependence disorder. *Br. J. Med. Psychol.,* 44:1-16, 1971.

Seevers, M. H.: Medical perspectives on habituation and addiction. *JAMA,* 181:92-9 8, 1962.

Seevers, M. H.: Discussion of a paper. In, *The Addictive States.* (Wikler, A., ed.) The Williams & Wilkins Company, Baltimore, 1968, p. 244.

Smoking and Health. Report of the Advisory Committee to the Surgeon General of the Public Health Service. Washington D.C. Public Health Service Publ. No. 1103, 1964.

Tamerin, J. S. and Eisinger, R. A.: Cigarette smoking and the psychiatrist. *Am. J. Psychiat.,* 128:1224-1229, 1972.

Ulett, J. A., and Itil, T. M.: Quantitative electroencephalogram in smoking and smoking deprivation. *Science,* 164:969-970, 1969.

REFERENCES (Cont)

World Health Organization. *Expert Committee on Drug Dependence. Twentieth Report.* Technical Report No. 551, WHO, Geneva, 1974.

World Health Organization. *Youth and Drugs.* Report of a WHO Study Group. Technical Report No. 516, WHO, Geneva, 1973.

AUTHOR

Jerome H. Jaffe, M.D.
Professor of Psychiatry
College of Physicians Surgeons
of Columbia University
722 West 168th Street
New York, New York 10032

Etiology: Session Overview

Murray E. Jarvik, MD., Ph.D.

Dr. Russell mentioned the recently published Royal College of Physicians ' Third Report, in which tobacco was considered' to produce a high dependency. He quoted evidence that London opiate addicts rated tobacco as their most needed drug. He also cited Lee Robins' study, which showed that Vietnam opioid addicts withdraw from heroin as easily, if not more easily, than tobacco addicts withdrew from tobacco. Dr. Jaffe commented on the fact that tobacco use has not been considered as a psychiatric disorder, and that there are probably not more than a half dozen psychiatric papers written on smoking. Sometimes smokers anthropomorphize cigarettes, attributing to them qualities of human beings, talking to them, saying such things as "I hate to give you up". For these smokers, it may be as difficult to give up smoking as any other love object.

Dr. Green pointed out that in 1967, psychiatrists and pediatricians had the highest incidence of smoking, but in the most recent survey, the differences between specialties seemed to be gone.

It was asked whether psychiatric texts are not often reflections and reinforcements of the values of the status quo. Dr. Jaffe replied that when something is sufficiently deviant, and when there is pressure on people to experience the deviance as ego-syntonic, then they feel guilty and ashamed of it. When you define a syndrome as a disorder, it may actually cause distress. Under such circumstances, people will come in seeking help to get rid of their deviant status.

Dr. Jaffe went on to say that smoking causes a productivity loss in terms of illness and early demise in its users, and that this can have a deleterious effect upon society as a whole. The point was raised concerning the relevance of delayed disability or delayed impact. Dr. Jaffe pointed out that anything more than four years away is considered a delay.

Ransom Arthur wondered whether one drug ever displaces another, or is the effect merely additive. Dr. Jaffe indicated that they are all additive and that alcoholics, for example, are much heavier users of

tobacco than non-alcoholics. Marijuana seems to be an exception in that there now seem to be heavy marijuana smokers who are not tobacco smokers. But for most other illicit drugs, i.e., opiates and alcohol, the incidence of tobacco smoking is extremely high. Dr. Arthur remarked on how the zeitgeist changes: twenty-five or thirty years ago smoking was not considered an important enough problem to even discuss, whereas, today it deserves examination by psychiatrists. If smoking is considered a disorder in the DSM-III, then it may be that third party payments will become available for the treatment of this disorder. Obviously, a good deal of political action will be necessary before such an economic change comes to pass.

SECTION III: CONSEQUENCES

The Economic Costs of Smoking-Induced Illness

Bryan R. Lute, M.S.P.H, MBA, and

Stuart O. Schweitzer, PhD.

INTRODUCTION

Although speculation regarding the effects of smoking dates back to the sixteenth century, when tobacco was introduced to the Old World, clinical and epidemiological investigation has positively linked smoking with specific illness and death only within this century. The economic effects of smoking have been relatively neglected, however. This report is intended to shed additional light on them.

The calculation of the costs of smoking is most important, not only because of the nation's present preoccupation with health care costs in general but also because of political decisions that are being made daily in both public and private sectors concerning the allocation of resources, including specifically government regulation, health education, and the role of health prevention. Anti-Smoking advertising, for example, can be justified only by assuming that the benefits of the campaign outweigh the costs of the operation. But decisions can be made more rationally if we know (1) the actual costs of smoking, and (2) both the costs and the effectiveness of different methods of intervention.

COST OF DISEASE CONSIDERATIONS

Both direct and indirect health care costs are included in the economic impact of smoking. Direct costs are those associated with the prevention, detection, and treatment of illnesses attributable to smoking. Indirect costs are earnings lost through morbidity and mortality; consequently, they measure the value society places on an individual's contribution to the economy. But the less obvious considerations, such as smoking-caused fires with their direct and indirect health care and property costs, operational expenditures of anti-smoking organizations, and cost of purchasing cigarettes, etc., must be taken into account as

well. The sum of these disadvantages must be weighed in turn against the definite benefits, such as tax revenues, and the possible benefits, such as lowered *anxiety* levels in the smoking population and the health implications of associated weight loss.

Although estimating these costs is conceptually simple, most of the relationships have never been adequately defined. In fact, very little information is available concerning health care utilization that is directly attributable to smoking, since most of the research to date has merely linked smoking habits with particular diseases, correlated death with smoking, or isolated causative agents (e.g., carcinogens) within the smoke.

REVIEW OF THE LITERATURE

The literature linking smoking to disease is well known.. It might be helpful, however, to acknowledge some of the more important works. The Surgeon General's report of 1964 made the smoking-health controversy a public issue by correlating the higher death rates by various diseases with differing smoking habits, and by linking smoking with bronchopulmonary disease.

Since 1964, the literature periodically compiled by sources such as the U.S. Public Health Service in its *The Health Consequences of Smoking* (1965) and the Royal College of Physicians' *Smoking and Health Now* (1971) has linked a rather significant roster of conditions to smoking *(Med. J. Australia - Special Supplement 1975):*

1. Cardiovascular disease
2. Chronic obstructive bronchopulmonary disease
3. Cancer
   a. lung
   b. larynx
   c. oral cavity
   d. esophagus
   e. urinary bladder
   f. pancreas
4. Pregnancy complications
   a. decreased fertility
   b. increased spontaneous abortions
   c. increased still-births
5. Peptic ulcer
6. Infancy respiratory disease
7. Oral disease (noncancerous)
8. Accidents
   a. fire
   b. automobiles

Unfortunately,. most of these studies do not lend themselves to generalized economic analysis. Instead of reporting that a specified portion of Disease X is believed to be caused by smoking, the results are usually couched in terms such as: "Males who are heavy smokers and are between the ages of 30 and 45 are Y times more likely to develop Disease X."

Several studies have linked increased health care utilization to smoking status, but the results do not lend themselves to direct economic analysis (Ashford 1973; Cakes et al. 1974). Still other authors have attempted to estimate the economic health costs of smoking, but their figures are in need of updating and refining. Soper (1972) estimates that the total economic health cost of smoking (medical care, lost income, and property loss due to fires) in 1966 was $5.3 billion, but his figures were based on an earlier Canadian study, and were determined by such crude measures as the CNT ratio of Canada to that of the United States,

Williams and Justus (1974) estimate that the 1970 health costs attributable to smoking are $4.23 billion. However, they derive their figure from a 1958 source that states that the total cost of respiratory disease in the United States was $2 billion (Ridker 1967), which they had to inflate to 1970. They then apply it to a "best estimate" from yet another Canadian study which states that 70 percent of chronic bronchitis and emphysema is due to smoking (Bates 1967).

Walker (1974) editorialized: "It has been estimated that $11.5 billion is spent annually in the United States for health care costs resulting from cigarette smoking." His source was a one-paragraph item in *American Medical News* (1974) attributing the statement to a physician addressing the American Lung Association.

Clearly the range of estimates and the imprecise analytical basis *justify a need* for *better* health cost estimates associated with smoking.

METHODOLOGY AND RESULTS

In an authoritative article, Cooper and Rice (1976) have published economic cost of disease data disaggregated into 16 diagnostic categories. Their analysis includes both direct and indirect costs, together with an in-depth explanation as to how these costs were derived. Table I is extracted from this study to present the costs of the major disease categories associated with smoking.

TABLE I[1]

Total economic costs of selected diseases: estimated direct costs indirect costs of morbidity and mortality, with present value of lifetime earnings discounted at 4 percent, by diagnosis, 1972. (in millions)

Diagnostic Category	Total costs	Direct costs	Indirect Costs	
			Morbidity	Mortality
	$17,367	$ 3,872	$ 862	$12,633
	40,060	10,919	6,417	22,724
	16,454	5,931	7,089	3,434

Boden (1976) reports figures from the working papers of the NIH Task Force on Prevention in Environmenta1 Health (1976) that. estimate the percentage of major' disease. categories' due to environmental problems, including smoking. Fortunately, his disease categories parallel those of Cooper and Rice. Table II presents these estimates.

TABLE II[2]

Smoking Factors of Major Health Problems

Disease	'Estimated Smoking Factor
Neoplasms	20.0%
Circulatory System	25.0
Respiratory System	40.0
Accidents (fires only)	1.1[3]

To determine the economic health costs attributable to smoking, the total economic costs (Table I) are multiplied by the corresponding estimated smoking factor (Table II). Table III presents the results, inflated to the 1975 prices, and includes the cost of property fires caused by smoking *(Med. J. Australia - Special Supplement 1975).* The total direct cost of smoking is thereby estimated at $7.5 billion, which is a approximately 7.9 percent of all direct health care costs in the nation.[7] The total (direct and indirect) smoking related economic cost of these diseases is $25.9 billion. This is an even larger proportion (11.3%) of the total cost of all diseases, probably due to extended morbidity and high mortality of the particular diseases considered (cancer, cardiovascular, respiratory).

TABLE III

Total economic costs of smoking : estimated direct costs, indirect costs of morbidity and mortality, with present value of lifetime earnings discounted at 4 percent, by diagnosis, 1975.
(in millions)

Diagnostic Category	Direct Costs[4]	Indirect Costs[5] Morbidity	Mortality	Property Costs	Total
Diseases:					
Neoplasms	$ 983.5	$ 218.9	$ 3,208.8	-----	$ 4,411.2
Circulatory system	3,466.8	2,037.4	7,213.9	-----	12,718.l
Respiratory system	3,012.9	3,601.2	1,744.5	-----	8,358.6
Accidents:					
Fires	43.9	33.3	151.6	$166.86	395.6
Total	$7,507.1	$5,890.8	$12,318.8	$166.8	$25,883.5

A. Other health related costs

As mentioned earlier, there are other costs of smoking that should be considered, such as those associated with diseases of lesser economic significance (e.g., noncancerous oral diseases) or diseases in which cigarette smoking has a relatively small impact (e.g., peptic ulcer). These estimates are not yet available. Nevertheless their aggregate would no doubt be significant, and our estimates are correspondingly understated.

B. Tobacco and GNP costs[8]

Not to be neglected in the economic analysis of smoking is the cost of tobacco and its associated taxes. In 1975, tobacco accounted for 4.6 percent of the value of all crops sold in the United States (U.S. Dept. of Agriculture 1976). Cigarette smokers smoked an incredible total of 607 billion cigarettes annually, which is an average of 4,121 (206 packs) per adult (18 years and older). American smokers paid $15.7 billion for all tobacco products ($14.4 billion for cigarettes) and of this, $5.8 billion was collected as taxes by all levels of government. If the estimated number of smokers were 60 million, as is reported by the National Cancer Institute (1976), then each smoker spent, on the average, $240 a year on 506 packs of cigarettes.

The net GNP effect is the total of (a) all direct and indirect health costs, (b) fire damage, and (c) tobacco sales. Table IV shows this total to be $41.5 billion, which is approximately 2.5 percent of the GNP, an average of $692 per smoker per year.

TABLE IV

Total Costs of Smoking and Tobacco, 1975
(in millions)

Direct health care costs	$ 7,507.1
Fire property damage	166.8
Lost earnings	18,209.6
Cost of tobacco (retail)	15,660.0
Total	$41,543.5

Smoking results in a major drain of the nation's economic resources regarding both direct health care costs and those costs associated with lost earnings due to sickness and death. Other costs that are considered are those associated with smoking-caused building fires and the purchase of tobacco products. In the absence of smoking, these resources would be reallocated to other sectors of the economy, and there would be more healthy individuals to share the respective benefits.

Although the cost of illness computations of Cooper and Rice are generally considered authoritative, the reader is cautioned that direct application of these estimates to calculate the costs of smoking is difficult .because the proportions of each illness which are attributed to smoking (i.e., Table II) have only been roughly estimated by the NIH Task Force on Prevention in Environmental Health, and have not been produced by rigorous analysis of empirical data. Consequently, empirical research is needed to improve these estimates and other research is needed to determine the costs and effectiveness of differing anti-smoking techniques. Once this information is available, it can be related to similar data pertaining to other health problems. Only then can rational decisions be made concerning the distribution of health resources within a program for the prevention of disease. The fact remains, however, that on the basis of methods delineated here, smoking related diseases cost the nation $25.9 billion annually and account for 11.3% of the total economic cost of all diseases.

FOOTNOTES

1. Extracted from Cooper and Rice, p. 31.

2. Extracted from Boden, p. 469.

3. Estimated from the National Fire Protection Association, *Fire Journal,* Nov. 1976.

4. Inflating 1972 figures from Cooper and Rice, p. 31, by the increase of the medical care component of the CPI.

5. Inflating 1972 figures from Cooper and Rice, p. 31, by the increase in GNP implicit price deflator.

6. Data from the National Fire Protection Association estimates.

7. Cooper and Rice exclude some costs such as administration, public health, research, etc., which cannot or should not be allocated to disease categories.

8. Tobacco information, unless otherwise noted, is extracted from *Tobacco Situation,* Dec. 1976, a USDA quarterly publication.

REFERENCES

Ashford, J.R. : Smoking and the Use of the Health Services. *Brit. J. Prev. and Soc. Med.,* 27:8-17. 1973.

Bates, D.V. : Health Costs of Diseases Related to Air Pollution. In *Poverty and Our Environment - Conference Background Papers,* A4-2-4, Vol. 1., Montreal. 1967.

Boden, L.I.: The Economic Impact of Environmental Disease on Health Care Delivery. *J. Occup. Med.,* 18:467-72. 1976.

Cooper, B.S., Rice, D.P.: The Economic Cost of Illness Revisited. *Soc. Sec. Bull.,* 39:31-36; 1976.

Fires and Fire Losses, Classified, 1975. *Fire Journal,* 70:17-19. 1976.

List of Illness and Disabilities Associated with Smoking. *Med. J. of Australia - Special Supplement.* 1975.

National Cancer Institute and Health Program; *Toward Less Hazardous Cigarettes.* DHEW, PHS, NIH, p. iii. 1976.

National Institutesof Health Task Force on Prevention in Environmental Health: Theory, Practice, and Application of Prevention in Environmental Health. In *Preventive Medicine USA,* Prodist. 1976.

Oakes, T.W. et al.: Health Service Utilization by Smokers. *Med. Care,* 12:958-65. 1974.

Ridker, R.G.: *Economic Costs of Air Pollution,* Praeger. 1967.

Royal College of Physicians: *Smoking and Health Now.* 1971.

Soper, F.A.: The Economics of Smoking. *Rehab.* (Bonn), 25:48-50. 1972.

U.S. Department of Agriculture, Economic Research Service: *Tobacco Situation.* 1976.

U.S. Department of Health, Education, and Welfare, Public Health Service: *Health Consequences of Smoking.* Annual reports since 1965.

U.S. Department of Health, Education, and Welfare, Public Health Service: *Smoking and Health.* Report of the Advisory Committee to the Surgeon General of the Public Health Service. 1964.

Walker, W.J.: Government-Subsidized Death and Disability. *JAMA,* 230:11. 1974.

Williams, J.R., Justus, C.G.: Evaluation of Nationwide Health Costs of Air Pollution and Cigarette Smoking. *J. Air Pollut. and Control Assoc.,* 24:1063-66. 1974.

AUTHOR

Bryan R. Luce, M.S., M.B.A.
School of Public Health
University of California
Los Angeles, CA 90024
Human Resources Research Center
University of Southern California
University Park
Los Angeles, CA 90007

Stuart O. Schweitzer, Ph.D.
Associate Director of Health Economics
School of Public Health
University of California
Los Angeles, CA 90024
Human Resources Research Center
University of Southern California
University Park
Los Angeles, CA 90007

DISCUSSION

These figures are all derived from secondary sources. Their accuracy is not certain since they are an update of a previous study done in the Sixties. The cost data were developed for the Commission on Heart Disease, Cancer and Stroke, although they are more refined here, and they were collected by Rice and Cooper.

It would be easy to calculate attributable risk for those cancers that are related to smoking using data from the third national cancer survey. The cost data were based on a rather large sample of the twenty million people surveyed. Cancer incidence was &rived from a 10% direct interview sample, examination of hospital records and from patients' individual receipts for costs. Those tapes are available from N.C.I., and they cover all costs, not just those of hospitalization.

We are spending $140 billion per year on health care now, and by 1980 this figure will top $200 billion. Success in preventive medicine depends on the economics of health care. Tobacco use produces what might be termed negative benefits - immediate benefit to the individual, but long-term negative effects on health and the economy. Phillip Abramson says that when someone else pays for something the costs are infinite. We might reflect on this fact in planning approaches to these problems. It would be useful to do a hospital census and estimate the number of in-patients and out-patients who would not require care if they had never smoked. Possibly such a study could be done, however, priorities don't seem to be in this direction.

Attributable risk estimates are available currently which show substantial risks associated with smoking for a wide variety of disease categories. It is, perhaps, unwise to assume that the entire excess risk of disease in smokers is a result of their smoking. Enidemiologically some of the diseases associated with smoking-have-not been shown to be etiologically related. Others, such as lung cancer, clearly are causally related to smoking. Blanket assumptions that every disease associated with smoking is caused by smoking create a credibility gap because they are subject to successful refutation by

industry sources. For this reason, it would seem important in estimating the dollar costs of tobacco use to separate costs which are clear and definite results of smoking from those which assume causality where it has not been proven to exist.

Another issue worth considering is what would happen to health costs if, say, cancer were eliminated. Probably the effects would not be great. It has been estimated, for example, that mean life span at age 60 or 65 would be increased by about 1.4 years, and that two-thirds of deaths would then be cardiovascular, a condition which is more expensive to care for than cancer. This viewpoint was very controversial, and several discussants rejected it on the grounds that treatment of cardiovascular disease wasn't more expensive than cancer and that this selective statistic focused only on people in their sixties, ignoring the impact of younger persons.

A number of suggestions were made concerning the fact that smokers do not pay the actual costs of tobacco use. Some felt that if health resources were to be reallocated that they should focus not on hospitals or medical care, but on the health insurance system. Persons who smoke should pay for the health costs of smoking.

The aim of a health care system is often forgotton. Many persons die in their sleep in their eighties, never having cost society or themselves much in terms of health care or social support. This is the idea that must be the aim of all health plans.

Thomas M. Vogt, M.D., M.P.H.

Smoking and Disease

Julien L. Van Lancker, M.D.

Historical Sketch of the Discovery and the Spread of Tobacco

Although Pliny the Elder, Herodotus, Pomponius Mela and Colonius reported the smoking of various materials (including cow dung for the treatment of melancholy!!!) , tobacco smoking seems to have originated among the Indians of the New World.

It is said that after a long famine in the land of Hurons, the Indians prayed to the Great Spirit for help. A naked girl appeared, sat on the dry land, and placed her hands on the ground. Corn grew where she placed her left hand, potatoes where she placed her right hand, and tobacco where she sat down (Koskowski 1955; Ochsner 1954).

For the Indians of the New World, tobacco was a most valuable commodity, and they certainly attributed exceptional properties to tobacco. The leaves were used for medicinal purposes in the form of 'poultices and pastes in the treatment of ulcers. Tobacco was smoked at religious and secular occasions. Who is not familiar with the "peace calumet"? Tobacco smoking was believed to give new psychic experiences, with visions of the after world; it stimulated devotees during ritual dances by combatting weariness, pain and hunger.

Hispanola, two of Columbus' sailors, Luis de Torres and Jerez, went on a search for the great Chinese Khan only to find Indian men and women smoking cigars (Ochsner 1954). Soon after the discovery by the Spanish of Hispanola and Cuba, the French of Canada and Florida, the Portugese of Brazil, and the English of Virginia, the Atlantic powers of the Old World adopted tobacco. The modes of consumption of tobacco - cigar, pipe and cigarette smoking, tobacco drinking and snuff taking - were often copied from the Indian nation with whom the explorers had come in contact. As trade between Europe, Africa, Asia and Australia expanded, tobacco was introduced to these various continents.

The sailors that landed in the New World were undoubtedly fascinated by the use of tobacco. It is said that when Rodrigo de Jerez returned to his native town in Spain, he appeared in front of his guests with smoke streaming from his nostrils, ears and mouth. Narrow-minded citizens believed him to be possessed by the Devil and denounced him to the Inquisitors, who dutifully incarcerated him for the proper time. Released from jail, Jerez discovered that tobacco smoking had become an accepted custom, even by the clergy (Bucher 1950; Apperson 1914).

Some of the landmarks in the discovery of tobacco are listed in Table I.

TABLE I

SOME LANDMARKS IN THE DISCOVERY OF TOBACCO

1492	Columbus' sailors observed smoking Indians
1512	Juan Ponce de Leon brings tobacco to Portugal
1556	Andre Thevet returning from Brazil introduced and brought tobacco seeds to France
1558	Tobacco is cultivated in Portugal
1558	Francesco Hemandez of Toledo brings tobacco to Spain
1559	Damien de Goes gives Jean Nicot tobacco plants who describes the "medicinal properties" of the plant
1559	Francisco Hemandez , private physician of Philip II, returned from Mexico and planted tobacco in Spain
1565	Sir John Hawkins brings tobacco seeds to England
1565	Sir Walter Raleigh introduced smoking in England
1565	Konrad Gesner introduces tobacco in Zurich
1573	Tobacco is cultivated in England
1603-1617	Smoking is introduced in Turkey

Among those that claimed medicinal properties for the tobacco plant are Leibault from Paris, Gohory from Paris and Nicholas Nomartes from Sevilla[1] The publications of these physicians contain tobacco-based recipes for external and internal use. Tobacco ointments, pastes and poultices were credited to cure ulcers, wounds, contusions, nolimetangere, scrofula and scabies. According to Nomartes, practically every disease of the gastrointestinal, respiratory and genitourinary tracts could benefit from the prescription of tobacco

TABLE II

SOME OF THE NAMES USED FOR TOBACCO

Herbe d'Ambassadeur: (after Nicot the French Ambassador to Portugal

Herbe du Grand Prieur

Nicotinae : after Nicot

Caterina of Medicia: after Catherine of Medici

Herbae la Reine Mere

Herbe de Sainte Croix: after two cardinals

Varinaes : after Varian in Venezuela

Herbe Saincte or Sacrea

Saine Saincte

Panacee Antartique

Herbe propre a tous maux

Herba di Sancta Croce

Herbe divine

Herba panacea

Sam Sancta Indorum

Sacra Herbea

Sancta Herbea

Indianisch Wunderkraut

Heilkraut

TABLE III

PUBLICATIONS PRAISING THE USE OF TOBACCO

1504-1564	"Agriculture et Maisson Rustique." The work of Charles Estienne.
1511 Sevilla	Nicolar Monardes, "Segunda parte de libro de las causas que se traen de nuestras Indians occidentales que servin al uso de medecina."
1570	Liebault advocated an extract of mortar ground dried tobacco leaves for treatment of all sorts of ulcers and skin diseases.
1572 Paris	Gohory publishes, "Instruction sur l'herbe Petun, ditte en France l'herbe de la Royne au Medicie."
1583 Antwerp	Gilles Everaerts, "De Herba Panacea, Quorum Alii Tabaccum, Alii Petum Aut Nicotianum Vacant."
1585 Rome	Castore Durante, 'Herbario Nuovo. "
1590 Sevilla	Jose de Acosta, 'Historia Natural y Moral de 10s Indias . "
1595 London	Anthony Chute, "Tobaco," "Herbal1 or General."
1597 London	John Gerard, "Historic of Plantes."
1600 London	William Vaughan, "Natural and Artificial Direction for Health."
1610 London	Edmund Gardiner, "The Judgment of Tobacco."
1614 Edinburg	William Barcley, 'Wephentes or the Virtues'of Tobacco. "
1622 Leyde	Johan Neander, "Tobacologia: hoc est tabaci, sen nicotinae description medico - cheiurgico pharmaceutics."
1626 London	Francis Bacon, "Sylva Sylvarum."
1627 London	Raphael Thorius, 'Hymnus Tabaci Sive de Pacto."
1644	"In Tabacuum Chigramua."
1648 Pavia	Jean-Chrysostome Magmen, "Exercitationes de Tobaco ."
1730	Halle Hoffman, "Medecina Rationalis Systematica."

1757 Ratisbonne	Johann Gottlieb Schaeffer, “Der Gebrauk und Nutzen der Tobackrauchsclystiers nebt liner daze bequemem Maschine."
1785 London	Thomas Fowler, ‘Medical Reports of the Effects o Tobacco Principally with Respect to its Diuretic Quality in the Cure of Dropsie and Dysuries.”

TABLE IV

SOME PUBLICATIONS AGAINST THE USE OF-TOBACCO

601 London - Anonymous ‘Work for Chimney Sweepers or a Warning for Tobacconists”

604 London - James I “‘A Counterblaste to Tobacco”

665 Strasbourg - Simon Paulli (from Copenhagen) Yonznentarious de abusa tobaci .”

670 Amsterdam - Thomas Theodor Kerchring “Spicilegium Anatomicum.

preparations. Nomartes claims to have cured headaches, coughs, asthma, gout, stomach pains, constipation, renal stones, flatulence, rheumatism, toothache and hemoptysis with either tobacco syrups, tobacco enemas or, in practically all cases, by inhalation of tobacco smoke. To reduce pains associated with delivery or pregnancy, he recommends that hot tobacco leaves be applied on the navel.

Works of the two Frenchmen and the Spaniard were translated in various languages and spread all over the Old World. In their zeal, the translators expanded the list of diseases that could be cured with tobacco to include syphilis, consumption, epilepsy. Neilander from Leyden even added to the phannacopea a collyra made of tobacco extract which, when applied to the eyes of older humans, would restore intact vision.

It is not likely that the average man and woman who smoked took the curative properties of tobacco seriously. They could hardly have escaped allergies, bronchitis and other complications associated

with smoking. Moreover, even in those days when tobacco was the queen of the phannacopea, some physicians like Van der Meer of Delft, seeded skepticism about the leaf's medicinal power and recommended restraint in its use.

Queens (Catherine the Great, Catherine of Medicis), kings and emperors (Peter the Great, Frederick William I of Prussia, George I, Napoleon[2], Napoleon III), generals (Moltke. Blucher[3]), playrights (Moliere[4]; Cornielle), philosophers(Francis Bacon, Voltaire), diplomats (Metternick. Talleyrand. lord Clarendon and Bismarck) and a bishop (John Fletcher), ali claimed that tobacco, smoked or snuffed, had helped their professional activities in one way or another.

Although most forms of tobacco smoking are destructive of health, cigarette smoking is probably the most harmful. It is believed that cigarettes were first used in Mexico, where chopped tobacco was wrapped in corn husks. In Spain, cigarettes were-mostly smoked by women. Those British officers who survived the tribulations of the Crimean War made the cigarette fashionable in their ancestral clubs. During the hay days of capitalism, James Buchanan puke resorted to that infallible method of private enterprise, advertising, to enlighten an austere America and teach it the joys of smoking cigarettes. In 1867 Duke brought Polish and Russian Jews to the U.S. to-manufacture cigarettes. This smoking material, still a luxury during the Civil War. was consumed at the rate of 1.000 million cigarettes by 1883. The rate of consumption of cigarettes rocketed (Figure 1) from then on.

This new form of addiction was not unique to the U.S.; it flourished practically everywhere in the world, even among the poorest nations.

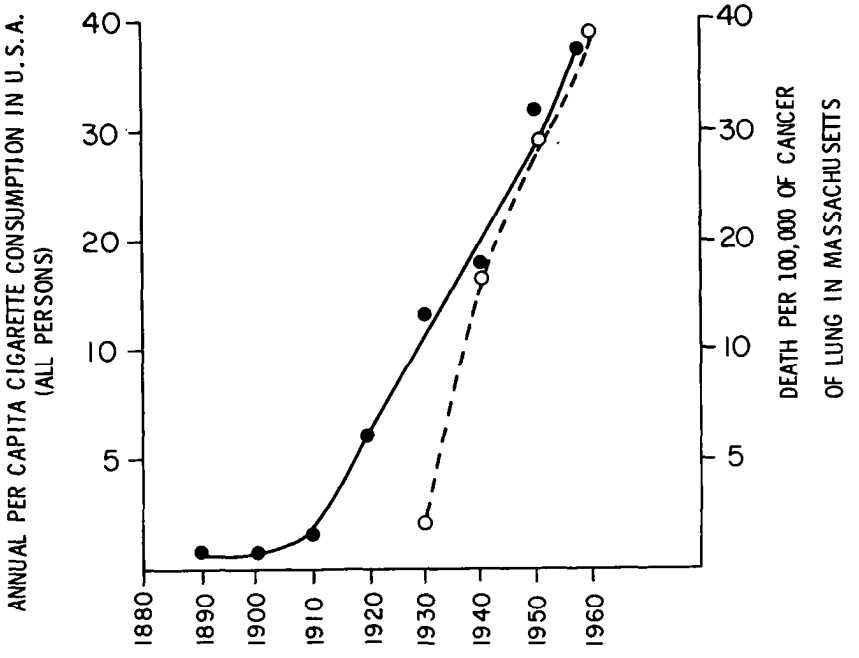
The wisest fool of Christendom, James I of England, led the antagonists of tobacco use. Not only did he state that tobacco has no medicinal properties, but he compared its black, smelly smoke to the horrible vapors that exhale from hell. In 1605 the king organized, in Oxford, the first public debate on the effects of tobacco. Black brains and black viscera, allegedly obtained from the dead bodies of inveterate smokers, were produced for everyone to examine with horror. The definite position of the king did not shake Dr. Cheynell's faith in the miraculous drug; he impudently appeared on the podium with a lighted pipe.

In 1642 Pope Urban VIII, horrified by the disrespectful behavior of his flock in the cathedrals, basilicas and churches of all Christ-' endom, wrote a bull condemning the use of tobacco in general and in holy places in particular and excommunicated all offenders. Apparently the smell of cigarette, cigar and pipe smoke was competing, even during the solemn masses, with the delicate fragrance of incense, and monks were coughing in the midst of their Gregorian chants.

Excommunication may seem bad enough, but a decree of 1634 punished Russian tobacco users by nose slitting, castration, flogging and banishment. These rather drastic punishments were abolished only under Peter the Great, who took to smoking a pipe in his effort to open a window on the West.

FIGURE 1

CIGARETTE SMOKING AND INCIDENCE OF CANCER OF LUNG



In Turkey, one of the ways that the use of tobacco was punished was by suspending the offender from a pipe introduced into a hole pierced through his nose. The rationale for these punishments was that the use of tobacco produced sterility and reduced the fighting qualities of soldiers.

Frederick the Great prevented his mother, the Queen of Prussia, from taking snuff during the coronation ceremony. Voltaire relates that Louis XV banished users of snuff from the court of France. The rebellious king's daughters borrowed pipes from the Swiss guards and organized clandestine smoking parties in their private apartments.

Many physicians claimed that tobacco caused ailments of the intestine (colics and diarrhea, nausea, emesis), of the respiratory system (ulceration of the lungs, asthma, cough), of the cardiovascular system (pain in the heart, apoplexy) and in addition caused undernourishment, impotence and dulling of the brain. In a rather gratuitous but imaginative interpretation of the origin of the word tobacco, Dr. Hodgkin in 1857 claimed that the use of tobacco, by drying the stomach, caused craving for drink and therefore endeared the user to the cult of Bacchus. He concluded that tobacco was derived from 'To Baccho." In an issue of Lancet in 1857 he claimed that tobacco causes dementia.

Many religious sects banished tobacco altogether: Mormons, Seventh t!ay Adventists, Parsees of India, Sikhs of India, monks of middle Korea, Tsai Li sect of China, some Ethopian Christian sects, Wahabi followers in Saudi Arabia, certain Bedouin tribes and followers of Mahdi in Sudan. As history tells us too well, consumption of tobacco survived the tortures by King's policies, the ostracism of church leaders and the warnings of physicians. Therefore. it is likely that if the habitual smoker might have been skeptical about miracle cures advocated by the most enthusiastic devotees of tobacco, they also paid little attention to the threats of their leaders and the advice of their doctors. Most could not resist the novelty and fashionable appeal of the new custom of smoking or snuffing and chewing tobacco. As the nations of the world indulged in these new sources of pleasure, social pressure increased and the custom of using tobacco spread. Soon both government and private enterprise became aware of the profit to be made by selling tobacco. The kings of France established "Le monopole du tabac" which was abolished after the French Revolution and reestablished by Napoleon. Most governments of the world have imposed taxes on tobacco (Table V). After Duke's pioneering efforts, the industrialists of the world followed suit and hastened to satisfy the craving for the weed. Clever advertising expanded the market. As a result, smokers almost all over the world can now enjoy their habit with the tacit approval of their government, vigorous encouragement of the tobacco industry, absolution of their church and the resigned silence of their physicians. Although all the ills said to derive from the use of tobacco do not obtain (their brains and entrails will not be blackened by the inhaled smoke), they will find that insidious and almost inevitably fatal cancers will grow in their lungs, that their arteries and heart will be damaged and that their lungs will be permanently ruined.

TABLE V

SOURCES OF GOVERNMENT REVENUE ON TOBACCO

Taxes on the stored leaves	U.S.
Taxes on the manufactured product	U.S.
Taxes on duties and imports	U.K. and many European countries
Tobacco monopolies	France, Japan

The truce between devotees and antagonists of tobacco use has, however, allowed a more serene evaluation of the role of smoking as a cause of disease. There seems to be little doubt that tobacco smoking contributes to the development of diseases of the cardiovascular and respiratory systems, and it cannot be excluded that it also causes disease of the gastrointestinal and urinary tracts.

Tobacco Growing and Manufacturing

Some elementary notions on the complex subject of tobacco culture and manufacture will help to understand the relationships between tobacco and disease (Bucher 1950; Trifkovic 1945).

The wild ancestors of the cultivated tobacco plant are of the genus Nicotina which contains more than 50 different species. Cytogenetic studies of cultivated plants indicate that the plants are allopolploid; that is, the chromosomes of the parent species are combined in the cultivated plant.

Probably one of the reasons why tobacco was adopted all over the world is that the plant grows vigorously under a wide range of climatic (from the tropics to Finland) and soil conditions. The properties of the final product depend on the climate, soil, types of seeds and types of protection used against disease.

Some tobacco diseases and insect pests are listed in Table VI.

Methods of tobacco disease control include crop rotation, use of resistant strains, sprays and fumigation. How much the sprays and fumigants contribute to the nefarious effects of tobacco has not been accurately evaluated. It is possible that some of the fungi which attack tobacco produce toxins that may be carcinogenic, or that the pesticides themselves are carcinogenic (e.g., arsenical pesticides).

Tobacco is cured in four steps: wilting, yellowing, coloring and drying. leaves are yellowed in humid air. They are alive and subsist on their starch reserves. The yellowing is caused by the breakdown of chlorophyll (Bucher 1950; Ochsner 1954; Koskowski 1955).

TABLE VI

DISEASES OF TOBACCO	INSECTS THAT ATTACK TOBACCO
Black root rot	Plant bed
Fusarium wilt	Green June beetle larvae
Tobacco mosiac	cut worms
Bacterial leaf spot	Flea beetles
Downy mildew	Field
Blank skunk	Flea beetles
	Cut worms
	Bud worms
	Aphids
	Stored leaf
	Tobacco beetle
	Manufactured product
	Cigarette beetle

TABLE VII

TIME REQUIRED FOR CURING

Air curing	One or two months
Fire curing	Ten weeks
Flue curing	Four to six days

The yellow leaves are then dried; as the cellsdie, oxidases come in contact with phenols to yield a brown pigment. Freshly dried tobacco leaves produce a smoke which irritates the throat. The pleasant fragrance and attractive taste develop during the fermentation and autolysis stage.

Fermentation releases $C0_2$, H_20 and heat; as a result the leaves lose a great deal of weight. Autolysis is carried out by the catalytic action of hydrolases on proteins, polysaccharides and glucosides to yield sugars and aromatic substances. Good tobaccos 'are cured in a manner that minimizes fermentation and enhances autolysis.

Various methods of drying are used: sun, air, fire and flue curing. Turkish and some American tobaccos are sun cured. Air curing is done in specially climatized rooms. In colonial days, wooden fires were kindled on the floor of the barn in which the tobacco was hang- . The smoke of the burning wood altered the taste of the tobacco giving it a creosote aroma. In 1825 wood fires were replaced by charcoal fires . After the Civil War, flue curing was introduced. In flue curing a furnace generates the heat which is carried to the room containing the tobacco through metal pipes (Table 7).

Composition of Tobacco

A myriad of compounds can be identified in tobacco leaves and smoke. Some, like nicotine, are of pharmacological importance; others, like carbon monoxide, methyl alcohol, lead and arsenic, are frank toxins.

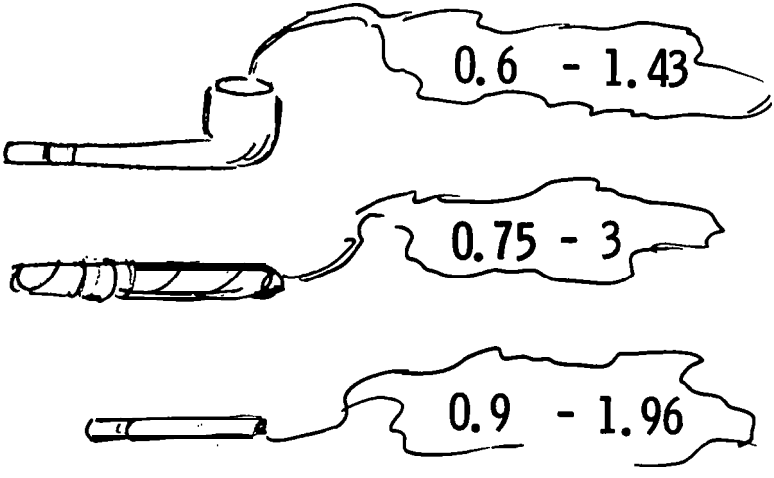
In 1829 Posselt and Reinmann (Apperson 1914), in Heidelberg, discovered and isolated the alkaloid nicotine. It is an a-pyridino-B-tetrahydrol-N-methylpyrrole. Fresh, it is a colorless oil fluid with an unpleasant odor and is very soluble in water. Exposed to air it becomes brown. Nicotine is elaborated by the root of the plant. If a tobacco leaf is grafted to a tomato plant, no nicotine appears in the tobacco leaf. In contrast, if a tomato leaf is grafted to a tobacco plant, the tomato-leaf contains nicotine. The amount of nicotine found in tobacco varies with the species, the nature of the soil, the climate and a number of other factors. It ranges from 0.5 to 8% of the weight of the dried tobacco leaf. Unless 'the tobacco is chewed or snuffed, it is the amount of nicotine that appears in the smoke that is critical (Bailey et al. 1928: Lehmann et al. 1943). Nicotine exists in tobacco as an organic salt. When tobacco is. burned, the salts are converted to a volatile form. The amount of nicotine present in the smoke depends on the brand of tobacco, its humidity and the habits of the smoker, whose rate of inhalation determines the rate of burning and the temperature of the smoke (Bogen 1929). (See Figures 2- and 3) .

Burning one cigarette produces approximately 800 cc of gas. Two major components of the gas are carbon dioxide and carbon monoxide. The faster one smokes, the, greater the volumes of CD_2 and CC produced and inhaled. The overall composition of the smoke also depends on the brand of tobacco. For example, some American tobaccos yield more aldehydes, including furfural, than oriental tobacco. In contrast, oriental tobacco yields more ammonia than American tobaccos.

FIGURE 2

NICOTINE CONTENT IN TOBACCO

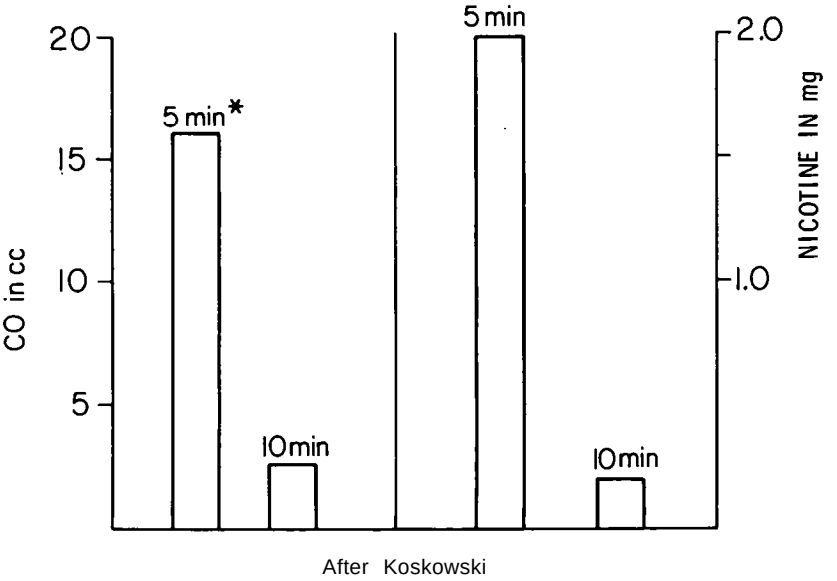
(In percent of weight in fresh tobacco)



after Koskowski

FIGURE 3

NICOTINE AND CO CONTENTS IN THE MAIN STREAM
OF TOBACCO SMOKE IN INTERRUPTED INHALATION



After Koskowski

*Time Used to Smoke a Cigarette

Nicotine is generally believed to cause the acute symptoms that develop after smoking. Seventy nine to 88% of the nicotine present. in the smoke is absorbed in the lungs. when one cigarette is smoked, 2.5 to 3.5 mg of nicotine are absorbed. The intravenous injection of 1 mg.of nicotine produces symptoms similar to those engendered by smoking one cigarette. More nicotine is absorbed from humid than dry tobacco, because when the tobacco is dry, more nicotine is destroyed during combustion (Adler et al. 1906).

Cigarette smoking is usually believed to be the most harmful form of tobacco smoking; pipe smoking being the least noxious. This is probably because cigar and pipe smokers do not usually inhale. The temperature of the smoke is relevant to the effects of tobacco on health and varies with the duration of the smoking of cigarettes (McNally 1932). (See Figure 4) For example, if a cigarette 6.5 cm long is smoked in a period of two minutes the temperature of the smoke may rise to 110° C, but it will only rise to 46° C if it takes eleven minutes to smoke the cigarette. The temperature of burning tobacco in a pipe varies with the type of pipe used and also with the mode of inhalation. Given a brand of tobacco, the temperature reached in a wooden pipe is always lower than that in a clay pipe. In the wooden pipe it might be 535° C, in the clay pipe 590° C. Again for a given brand, the temperature in a wooden pipe may rise from 370° C when inhalation is normal to 480° C when strong inhalation is used. When tobacco is smoked in a clay pipe with strong inhalation, the temperature may reach 700° C (Cooper et al. 1932). Higher temperatures at the stem of the pipe probably generate more carcinogenic substances in the tar. Therefore, high temperatures of burning tobacco might play an important role in the pathogenesis of cancer of the lip, mouth and throat observed in pipe smokers .

Toxicology of Tobacco Components

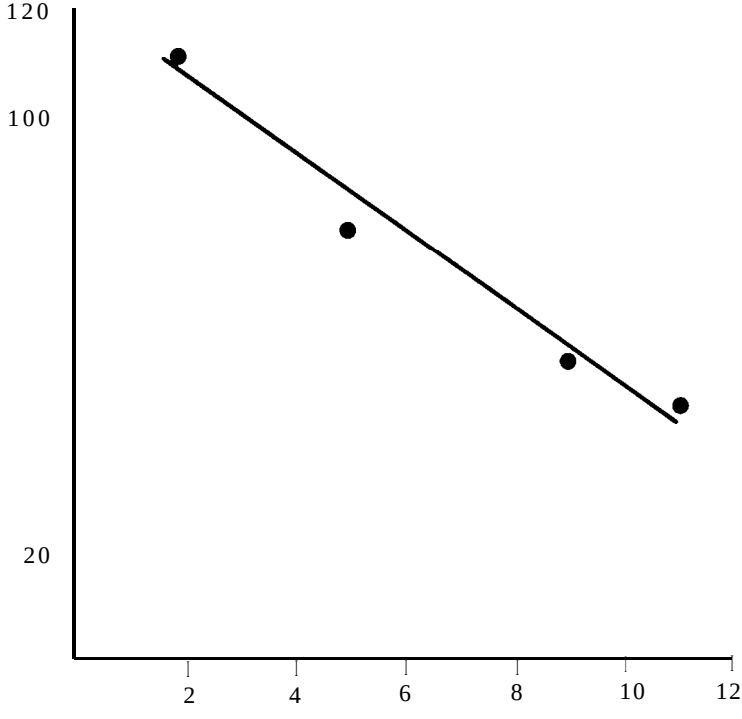
CO (Carbon Monoxide)

Carbon monoxide is a colorless, odorless and nonirritant gas. It is produced by incomplete combustion of organic substances found in coal gas, but not in natural gas. Its toxicity stems from its binding to hemoglobin to form carboxyhemoglobin which is 2.10 times stronger than the binding of oxygen to hemoglobin to form oxyhemoglobin. Thus, repeated exposure to even small amounts of carbon monoxide can markedly reduce the amount of hemoglobin available for combination with oxygen and cause anoxemia of all tissues. The heart and the brain, which are heavily dependent upon aerobic respiration for function, are the first victims of anoxemia. Carbon monoxide binds not only to hemoglobin, but also to many other iron proteins, including the cytochromes, major electron transporters (Goldsmith et al. 1968).

Concentrations of carbon monoxide in cigarette smoke vary from approximately 2.9 to 5.1%. Its affinity for hemoglobin is 200 times that of oxygen. Carbon monoxide causes anoxemia by at least three different mechanisms: formation of carboxyhemoglobin, shift in the affinity of hemoglobin for oxygen and interference with 2,3-diphosphoglycerate regulation of oxygen affinity. Carbon monoxide shifts the oxyhemoglobin dissociation curve, increases the affinity of hemoglobin for oxygen and prevents oxygen release at the tissue level.

FIGURE 4

TEMPERATURE OF SMOKE WHEN A ClGARETTE OF 6.5cm IS SMOKED



After Koskowski

Carbon monoxide interferes with the homeostatic mechanism by which 2,3-diphosphoglycerate controls the affinity of hemoglobin for oxygen (Oski et al. 1970).

The deleterious effects of carbon monoxide may not be restricted to the smoker. Studies by Russell et al. have shown that inhalation of smoke in an unventilated room for 78 minutes is equivalent to the absorption of the amount of carbon monoxide that would emanate from the smoking of one cigarette (Chevalier et al. 1966; Russell et al. 1973).

Intoxication with carbon monoxide is observed among individuals working with blast furnaces, engine drivers, people working in badlv ventilated tunnels and car drivers (especially if wood gas is used) (Gettler et al. 1933; Bonnevie et al. 1948; Astrup et al. 1968). Carbon monoxide is also found in the street air, and policemen regulating traffic may occasionally suffer from headaches, nausea and muscular fatigue as a result of carbon monoxide intoxication. The blood of policemen regulating traffic may contain as much as 30% carboxyhemoglobin (Wilson et al. 1926; Lilienthal 1950). Similarly, taxi drivers have been found to have carbon monoxide content of 1.47 to 4.33 % in the blood, leading to a concentration of carboxyhemoglobin of 8 to 19%. The greatest concentrations of carbon monoxide in the smoke are obtained-during cigar smoking and the concentration in the lungs will be 0.04%. At the end of smoking one cigar, 5% of the blood does not function as an oxygen carrier and Jongbloed has calculated that the smoking of one cigar is equal to the loss of 250 cc of blood (Jonabloed 19391. Such an alteration is of little significance if 'it is not cumulative and takes place in healthy humans, but it may be of great consequence to a person with severe atherosclerosis or suffering from other diseases causing anoxemia. Usually a concentration of 5% of carboxyhemoglobin does not generate any symptoms as shown in Figure 5.

Other Compounds

The fermentation of the polysaccharide pectin, found in the tobacco plant, yields methyl alcohol. It is estimated that 40 mg of methyl alcohol are absorbed after smoking 20 unfiltered cigarettes and 42 mg are absorbed after smoking 10 cigars.

In addition to methyl alcohol, tobacco smoke contains ammonia, formaldehyde, phenols, creosote, anthracen and pyrene. Small amounts of hydrocyanic acids are also formed, but are not believed to be of toxicological significance. An increase in thiocyanate in the blood of inveterate smokers has been observed. The thiocyanates are usually excreted through the saliva (Trasoff et al. 1936). It is estimated that heavy smokers may eliminate as much as 400 mg of thiocyanate in their saliva. Combustion of glycerol generates acroleine which provokes local irritation. Fertilizers and insecticides may add arsenic and lead to the tobacco.' Half of the arsenic in tobacco enters the smoke, the rest remaining in the ash. The amount of arsenic in the inhaled smoke ranges from 3.3 to 10.5 mg per cubic meter of smoke (Thomas et al. 1945). Figure 6 gives the arsenic content in the various components of a burned cigarette given a pattern of smoking (Koskowski 1955). It is difficult to estimate how

FIGURE 5

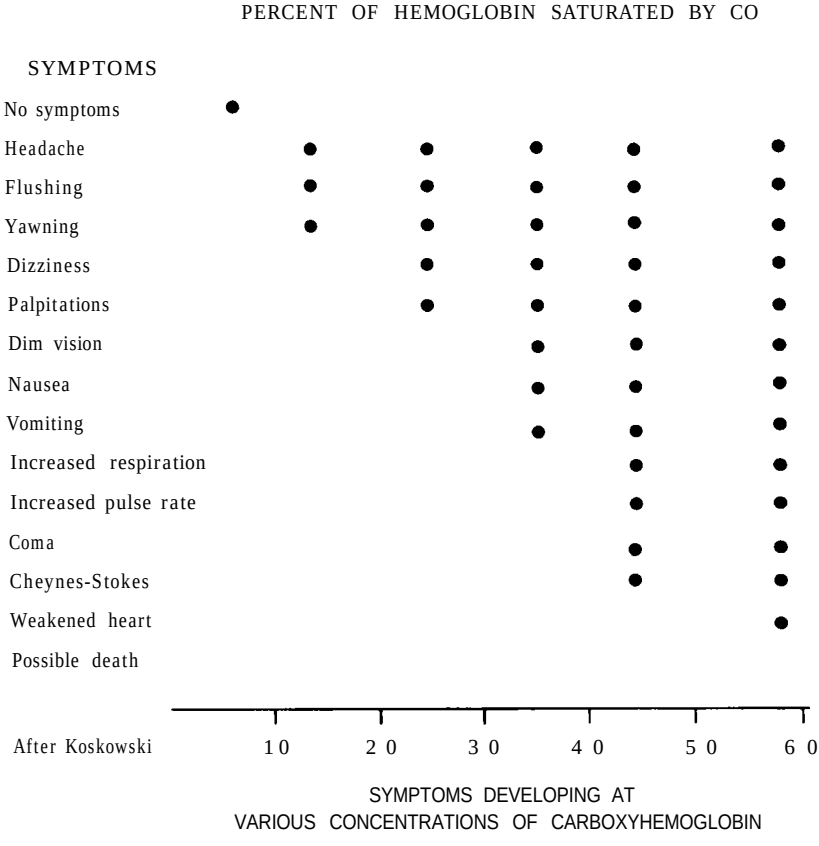
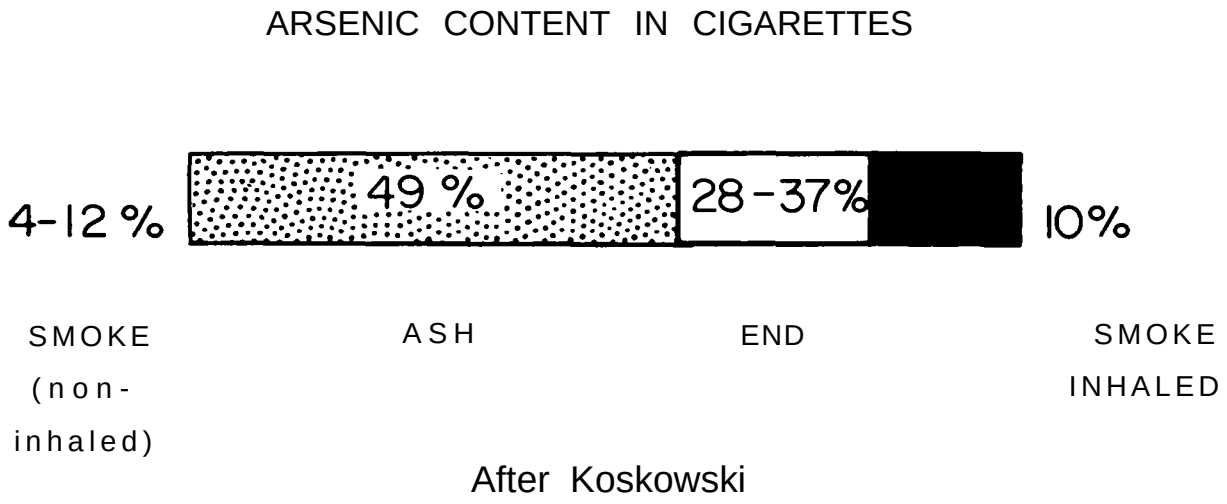

PERCENT OF HEMOGLOBIN SATURATED BY CO
SYMPTOMS
No symptoms
Headache
Flushing
Yawning
Dizziness
Palpitations
Dim vision
Nausea
Vomiting
Increased respiration
Increased pulse rate
Coma
Cheynes-Stokes
Weakened heart
Possible death
After Koskowski
10
20
30
40
50
60
SYMPTOMS DEVELOPING AT
VARIOUS CONCENTRATIONS OF CARBOXYHEMOGLOBIN

FIGURE 6


ARSENIC CONTENT IN CIGARETTES
4-12 %
49 %
28-37%
10%
SMOKE (non-inhaled)
ASH
END
SMOKE INHALED
After Koskowski

much arsenic contributes to ailments of heavy smokers (Holland et al. 1958). The presence of arsenic in smoke is probably more relevant to the dermatitis and eczema observed among tobacco dealers and workers. Similarly, lead may have caused poisoning among tobacco workers (Jordans et al. 1936), but it is not likely to play a toxicological role among heavy smokers.

Effects of Smoking on the Cardiovascular System

Nicotine primarily affects the cardiovascular and respiratory systems (English et al. 1940; Roth 1951; Shepherd 1951; Russek 1955; Friedell et al. 1969; West et al. 1958; Kien et al. 1960; Roth et al. 1960; Thomas et al. 1960; Von Ahn 1960; Leaders et al. 1962; Irving et al. 1963; Doyle et al. 1964; Pentecost et al. 1964; Folle et al. 1966; Mulcahy et al. 1966; Schwartz et al. 1966; Kuhn 1967; Nadeau et al. 1967; Romero et al. 1967; Kannel et al. 1968; Puri et al. 1968; Miyazaki 1969; Strong et al. 1969; Hammond et al. 1969; Ross et al. 1970; Seltzer 1970). Among the changes induced in the cardiovascular system by smoking are increase in heart rate, blood pressure, cardiac output, stroke volume, velocity of contraction, myocardial contraction force and myocardial oxygen consumption; development of arrythmia and alteration of electrocardiographic and ballistocardiographic patterns (Friedell 1953; Bum et al. 1958; Forte et al. 1960; Frankl et al. 1966; Clark et al. 1967). Nicotine is suspected to be responsible for these changes: its parenteral administration reproduces all the changes (Adler et al. 1906).

Nicotine is known to stimulate neurons in sympathetic and parasympathetic ganglia (Dietrich et al. 1939). 'The postganglionic fibers of the sympathetic ganglia that innervate the heart, smooth muscle, blood vessels and some glands, are stimulated by the chemical transmitter, norepinephrine. Therefore, it is believed that epinephrine and norepinephrine might be the mediators of the response to nicotine (Kershbaum et al. 1963; Aviado et al. 1966; Westfall et al. 1966; Kershbaum et al. 1967b; Balazs et al. 1969). Clear-cut evidence that ordinary smoking produces levels of nicotine high enough to act upon the sympathetic ganglia is not available. Sensitive, specific and rapid assay for plasma nicotine are needed.

Nicotine increases the coronary blood flow (Bargeron et al. 1957;. Bellet et al. 1962) when injected directly into the carotid of dogs. However, it is believed that patients with compromised coronary circulation do not respond to the nicotine stimulus to blood flow and that therefore the flow of blood to the heart is not kept in tune with the increased workload brought about by nicotine.

Nicotine inhaled in cigarette smoke is rapidly absorbed from the lung and can be found in brain, adrenal medulla and sympathetic ganglia within 5 minutes. The alkaloid is metabolized in the liver, the kidneys and the lungs, probably by mixed-function oxidases and is excreted in the stomach and the kidney.

Nicotine exhibits hemodynamic effects on the heart through the release of catecholamines. The increased cardiac output is compensated for by a rise in coronary circulation in healthy patients, but in those with severe atherosclerosis, such compensation does not take place. Whether nicotine has an atherogenic effect in humans needs

to be established. In animal experiments, amounts much higher than the nicotine uptake in smokers are needed to produce atherogenic disease.

Nicotine has a triple effect on the cardiovascular system. It causes an hemodynamic response of the heart; it leads to increased circulation of free fatty- acids (Gofman et il. 1955, Wenzel et al. 1958: Page et al. 1959: Wenzel et al. 1959: Acheson et al. 1961: Konttine; 1962; Wilens et al. 1962; Konttinen et al 1963; Auerbach et al. 1965; Kershbaum et al. 1965; Blomstrand et al. 1966; Kedra et al. 1966; Mcahy et al. 1966; Choi 1966; Kershbawn et al. 1967a; Kershbaum et al. 1967b; Van Buchem 1967; Auerbach et al. 1968; Astrup 1969; Brody et al. 1969; Kjeldsen 1969; Stefanovich et al. 1969; Webster et al. 1970; Wherat 1970) and causes an increase in platelet stickiness and aggregation (Blackbum et al. 1959; Mustard et al. 1963; Ambrus et al. 1964; Ashby et al. 1965; 'Kedra et al. 1965; Sogani et al. 1965; Murphy 1968; Pozner et al. 1970).

Increased incidence and severity of atherosclerosis among smokers is observed at autopsies. Nicotine is known to induce necrosis in the arterial walls and when nicotine is associated with cholesterol in the diet, endothelial fibrosis takes place (Figures 7 and 8).

Carbon monoxide also seems to be atherogenic, either by increasing the permeability of the arterial wall to lipoprotein or by inhibition of reoxidation of NACH, an important cofactor for biosynthesis of fatty acids.

Both nicotine and carbon monoxide are believed to increase serum lipid levels.

Thus the combined action of carbon monoxide and nicotine is as follows: carbon monoxide reduces the amount of oxygen to the myocardium while nicotine increases the amount of work of the heart. An increase in incidence of atherosclerosis and thrombosis may well result from the combined action of carbon monoxide and nicotine (Regan et al. 1960; Cohen et al. 1969; Eerkson et al. 1970). A schematic representation of the pathogenesis of heart disease in smokers is presented in Figure 9.

In Great Britain 52,000 people die from smoking every year; half of the deaths are caused by cardiovascular disease (Ball et al. 1974).

It is estimated that a 20% reduction in cigarette consumption by heavy smokers could reduce the number of deaths by cardiovascular disease by 8,000.

Doll and Hill (Doll et al. 1964a; Doll et al. 1964b) studied the relationship between smoking habits among British physicians and concluded that the mortality rate of heavy smokers between 35 and 44 years old is five times that of nonsmokers in the sane age group. As the smokers grew older their relative chances of developing coronary heart diseases decreased. It was only four times as great for smokers than for nonsmokers between ages of 45 and 54. and the difference became insignificant over the age of 55. Similar results were obtained in a study among U.S. veterans (Kahn 1966). Smokers between

FIGURE 7

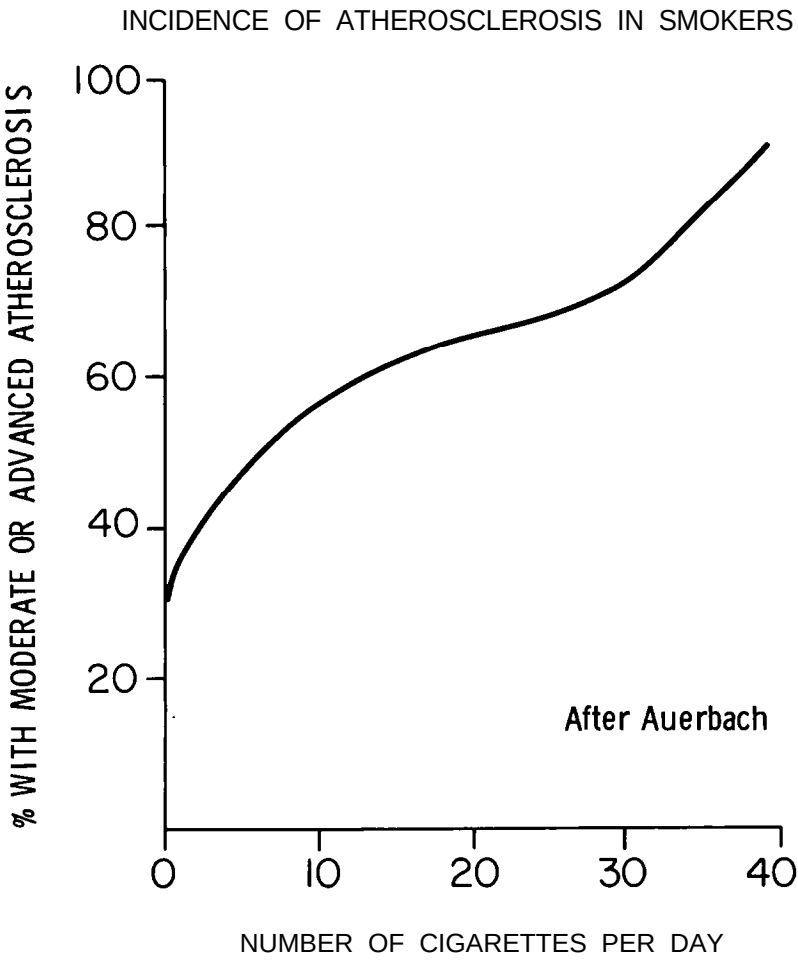
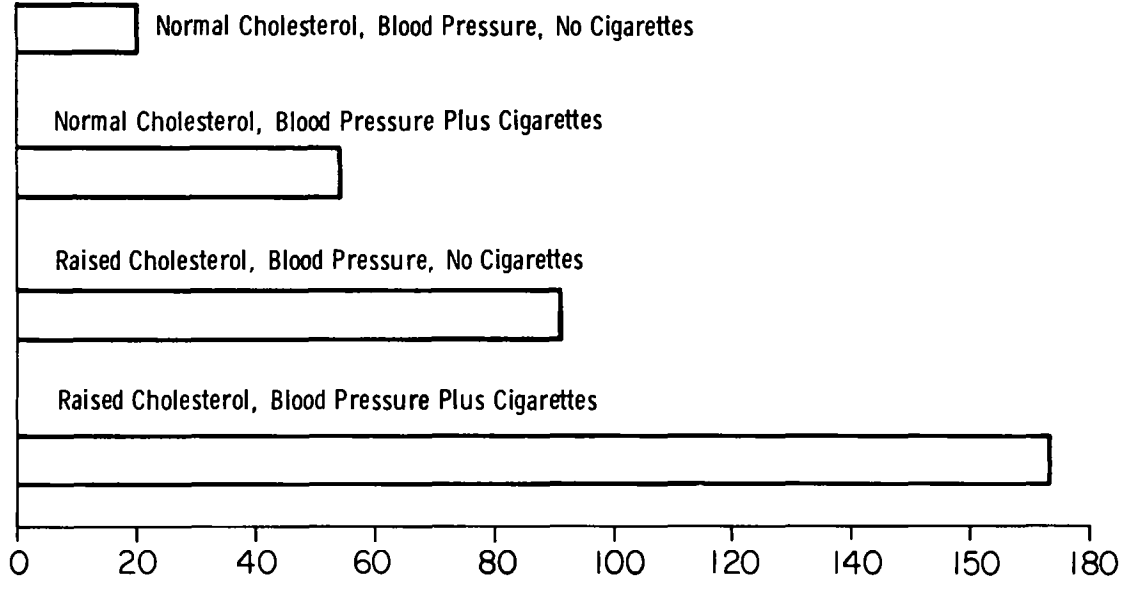

INCIDENCE OF ATHEROSCLEROSIS IN SMOKERS
% WITH MODERATE OR ADVANCED ATHEROSCLEROSIS
100
80
60
40
20
0
10
20
30
40
After Auerbach
NUMBER OF CIGARETTES PER DAY

FIGURE 8

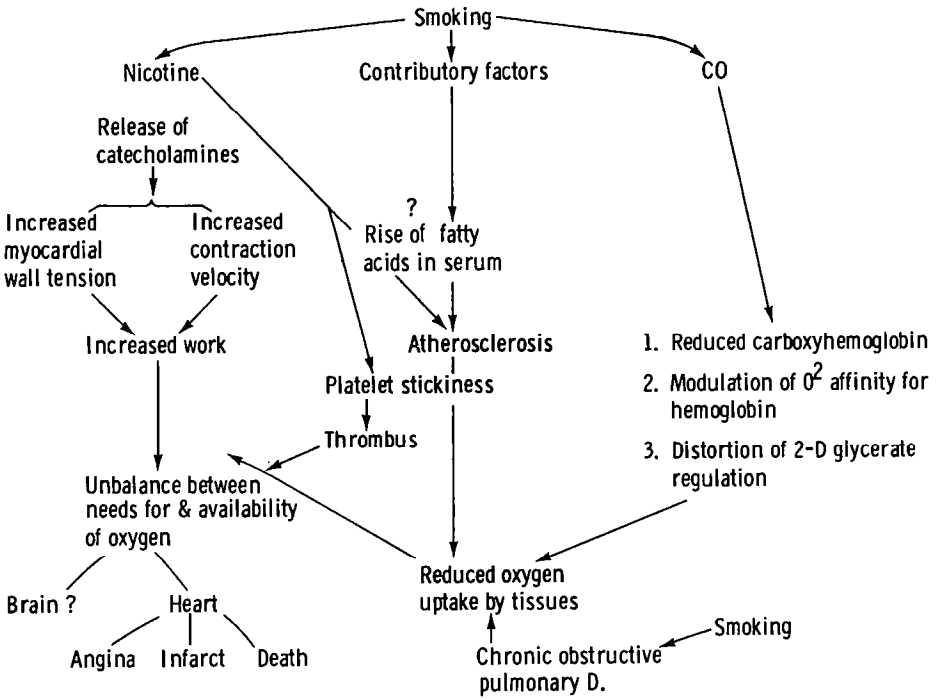
RISK FACTORS IN CORONARY HEART DISEASE



(After the Intersociety Commission for Heart Disease Resources)

FIGURE 9

PATHOGENESIS OF HEART DISEASE IN SMOKERS



35 and 54 had a 5 to 19 fold increase in risk of death from coronary heart disease compared to nonsmokers. All smokers do not develop fatal myocardial disease. However, the chances of developing non-fatal coronary heart disease is much higher among smokers, especially young men, than among nonsmokers. Smoking also increases the incidence of. angina, although true tobacco angina or angina produced by smoking alone is rare.

If the contribution of cigarette smoking to coronary heart disease cannot be ignored (Buechley et al. 1958; Cederlof et al. 1969), neither should its role in the pathogenesis of coronary heart disease be exaggerated. Smoking may be deleterious to individuals with hypertension and cholesteremia, which are frequent among Westerners, but it may not affect individuals with low cholesterol and normal blood pressure, as is the case in Crete, Corfu and parts of Japan and Yugoslavia .

According to Ochsner, King George VI of England, a heavy smoker, suffered from both thromboangitis obliterans, or Buerger's disease, and cancer of the lung. Buerger's disease is an uncommon obstructive vasculitis of the arteries and sometimes the veins of the lower extremities. The disease is aggravated by smoking and cessation of smoking aids in complete or partial remission. Few nonsmokers ever contract the disease (Barnett et al. 1960; Begg 1965; Schwartz et al. 1965; Hass et al. 1966; Brown et al. 1969; Kjeldsen et al. 1969).

Effects of Smoking on the Respiratory System

Three types of diseases are usually classified under the generic term "chronic obstructive bronchopulmonary disease." They are chronic bronchitis, pulmonary emphysema and reversible obstructive lung disease or bronchial asthma. Inasmuch as bronchial asthma is not usually caused by cigarette smoking, it will not be included in this discussion. Chronic bronchitis is characterized by cough and sputum production and should last for periods of at least three months each year for two consecutive years.

In pulmonary emphysema, destructive changes of the alveoli lead to a permanent expansion of the air space beyond the terminal bronchiole. Pulmonary emphysema is characterized by dyspnea. When both chronic bronchitis and amphysema are present, there is cough, sputum excretion and dyspnea.

A majority of patients suffering from chronic obstructive pulmonary disease are cigarette smokers (Bickerman et al. 1954; Flick et al. 1959; Franklin et al. 1961;,Hemandez et al. 1966; Aviado et al. 1967; Crowdy et al. 1975). Chronic obstructive pulmonary disease has been produced experimentally in dogs trained to inhale cigarette smoke through a tracheostomy (Ausrbach et al. 1967a; Auerbach et al. 1970b). Changes in bronchi and lung parenchyma are proportional to the total amount of smoke inhaled. Cigarette smoke inhibits ciliary activity of the bronchial epithelium (Mendenhall et al. 1937; Falk et al. 1959; Ballenger 1960; Wynder et al. 1963; Dalhanm et al. 1964; Dalhamn et al. 1965; Wynder et al. 1965; Dalhanm 1966; Dalhamn et al. 1968; Kaminski et al. 1968; Dalhamn et al. 1970) and the phagocytic activity of the macrophages of the pulmonary alveoli. This results in defective clearance of inhaled foreign material, including viruses and

bacteria, and results in increased incidence of respiratory infection. Whether or not pulmonary surfactants are affected by cigarette smoke remains to be established (Miller *et* al. 1962; Cook et al. 1966; Gianunona 1967; Scarpelli 1968; Pratt et al. 1969). Decreased surfactant production interferes with the proper expansion of the alveolar wall. Ventilator-y functions have been shown to be decreased in smokers compared to nonsmokers (Motley et al. 1958; Higgins 1959; Liebeschuetz 1959; Wilson et al. 1960; Read et al. 1961; Krumholz et al. 1964; Zwi et al. 1964; Krwnholz et al. 1965a; Krumholz et al. 1965b; Peterson et al. 1968; Wilhelmsen et al. 1969; Chiang et al. 1970; James 1970).

Cigarette smoking seems to be the main cause of chronic obstructive pulmonary disease. Although the incidence of these diseases in pipe and cigar smokers is higher than in the general population, it is still lower than among cigarette smokers. Although, in general, the effects of cigarette smoking are much more deleterious than atmospheric pollution, under conditions of severe atmospheric pollution the combination of cigarette smoking and pollution may cause more severe chronic obstructive pulmonary disease. Resultant respiratory infections will be more frequent among cigarette smokers than nonsmokers (Boake 1958; Edwards et al. 1959; McDermott et al. 1965; Pamell et al. 1966; Megahed et al. 1967; Rimington 1969; Lambert et al. 1970).

Pathological changes observed in the tracheobronchial tree and assocciated with smoking include goblet cell distension, alveolar septal rupture, thickened bronchial epithelium and mucus gland hypertrophy (Ide et al. 1959; Leuchtenberger et al. 1960a; leuchtenberger et al. 1960b; Anderson 1963; Anderson et al. 1965; Anderson et al. 1966; Auerbach et al. 1967b; Auerbach et al. 1970a).

Anderson has claimed that centrolobular emphysema is more characteristic of smokers than panlobular emphysema. Smokers and nonsmokers are found in equal numbers in patients with panlobular emphysema, while 98% of patients with centrolobular emphysema are smokers.

The mechanism by which bronchitis develops in cigarette smokers is not clear. In an attempt to elucidate the pathogenesis of chronic bronchitis, Kilbum and MacKenzie (Kilbum et al. 1975) studied the leukocyte recruitment to airways by cigarette smoke, using hamsters forced to breathe fresh cigarette smoke in miniature chambers. The results suggest that cigarette smoke does not recruit polymorphonuclears by the synergetic action of the nonparticulate and the particulate components of the smoke.

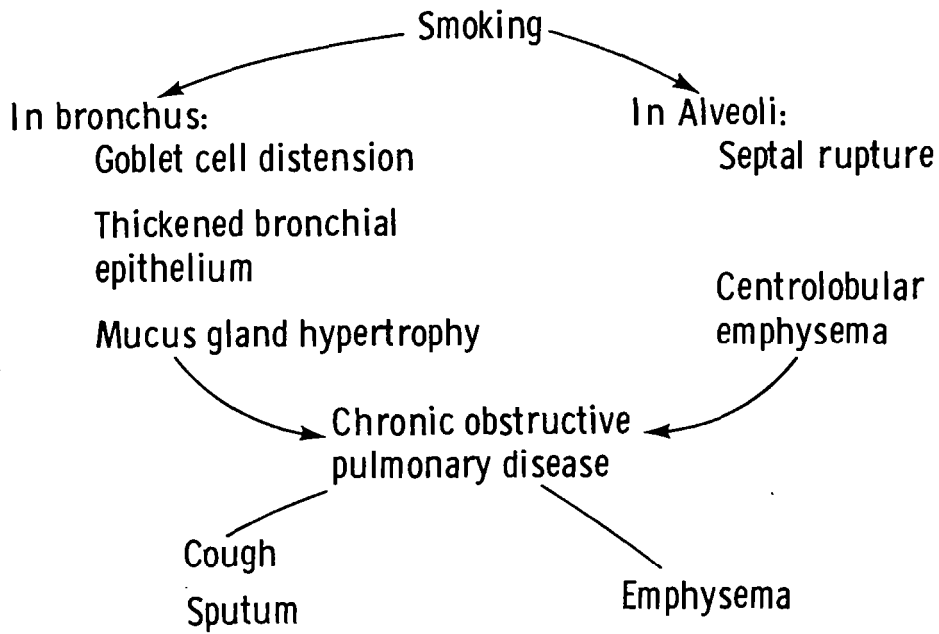
Auerbach has shown that the consumption of half a pack of cigarettes a day causes only minor emphysema, but the consumption of two packs or more leads to very severe emphysema characterized by the development of large holes in the pulmonary parenchyma (Auerbach 1972). A schematic representation of the pathogenesis of chronic obstructive pulmonary disease is presented in Figure 10.

Smoking and Child Development

Children born from mothers who smoked heavily during pregnancy are usually smaller at the time of delivery than children born from nonsmokers. In general, infants born from smoking mothers are from 70

FIGURE 10

PATHOGENESIS OF COPD IN SMOKERS



to 250 gm lighter than children born from nonsmokers. Inasmuch as the chances of optimal physical and intellectual development decrease with the weight of the infant (optimal weight 3,000 gm), several investigators have been concerned with the 'potential effect of smoking on child development and the mechanism by which fetal growth is retarded in smoking women (Lowe 1959; Frazier et al. 1961; Haddon et al. 1961; Heron 1962; Kumar et al. 1963; Murdoch 1963; Young et al. 1963; Mantel1 1964; Peterson et al. 1965; Becker et al. 1966; Downing et al. 1966; Mosier et al. 1967; Becker et al. 1968; Duffus et al. 1968; Russell et al. 1968; Younoszai et al. 1968; Buncher 1969; Younoszai et al. 1969; Hardy et al. 1972). Two mechanisms of retardation have been proposed: one is the anoxemia caused by the higher concentration of carboxyhemoglobin, the other is the food intake. It would seem that smoking mothers gain less weight than nonsmoking mothers. A study by Davies et al. (Davies et al. 1976) suggests that increasing the weight of smoking mothers might prevent the harmful effects of smoking on fetal growth. However, at four and seven years there are no significant differences between physical and intellectual functions of children born from smoking and nonsmoking mothers.

The mortality in babies of smokers is significantly higher than in babies of nonsmokers for both stillbirths and neonatal deaths. For reasons unknown, smoking mothers have a reduced incidence of pre-eclamptic toxemia, as compared to nonsmoking mothers.

Although there is no evidence that the cigarette smoke is teratogenic in vivo, studies in vitro have shown that the cigarette smoke contains mutagenic factors the salmonella microsomal system (Kier et al. 1974).

Smoking and Cancer

Cancers Linked to Tobacco Use

John Hill, a physician who wrote operas, novels and farces, seems to have been the first to attract public attention to the carcinogenic properties of tobacco. He wrote a note entitled, "Caution Against the Immoderate Use of Snuff," in which cancer of the nostrils was attributed *to* the use of snuff.

The use of tobacco in any form has often been suspected to cause cancer at various sites in the body: lips, tongue, tonsils, larynx, lung, stomach, intestine, pancreas and bladder (Levin et al. 1950; Hammond 1975; Rothman 1975; Wynder et al. 1975).

Cancer of the lip, a type of cancer often referred to as cancer of "country folks ," is more prominent among land laborers who smoke short and uncleaned pipes (Ewing 1940; Levin et al. 1950). The incidence of cancer of the lip is high among those who keep a cigar or cigarette in their mouths all the time and who smoke them to the end.

Ewing suspected that tobacco played an etiological role in carcinoma of the mouth and tongue. Syphilis and leukoplakia also contribute to the increased incidence. Pipe and cigarette holder users are more prone to develop cancer of the mouth than cigarette smokers, probably because these smoking devices are usually placed in the same place in the mouth. Tobacco chewers develop cancer of the mouth usually at the sites where the tobacco is tucked: the cheeks and the gums. In

Asia cancer of the cheek and mouth is frequent among Hindus who chew tobacco and betel (Nehta et al. 1961; Atkinson et al. 1964; Hirayama 1966).

During the end of the 19th and beginning of the 20th centuries the incidence of cancer of the lung was very low (Adler 1912). Between 1880 and 1910 two cancers of the lung were discovered at autopsy in Strasbourg. Eighteen cases a year were observed between 1946 and 1950. After 1910 the incidence of cancer of the lung was on the rise. Two major changes were responsible, at least in part for this increase in incidence: extension of average life span and improved diagnosis as a result of the sequential discovery of Roentgen diagnosis, bronchoscopy and cytology. In 1936 Fleckseder found in a - limited study that 94% of the oatients with cancer of the lung were heavy smokers (Fleckseder 1936). The association between tobacco smoking and the incidence of cancer of the lung has since been repeatedly investigated (Wynder et al. 1950: Wynder et al. 1951: Doll et al. 1952; Hammond 1952; Hammond et al.1952; Ochsner et al. 1952; Wynder et al. 1953a; Hammond 1954; Haenzel et al.. 1956; Davies 1959; Abelin et al. 1967; MacMahon 1975).

In almost all countries where the incidence of cancer of the lung has been studied, number of cigarettes smoked rises with the incidence of cancer of the lung.

The incidence of cancer of the lung was low among women until they took up smoking (Haenzel et al. 1958). It remains low in religious groups such as the Seventh Day Adventists and Mormons, who do not approve of smoking. The incidence of cancer of the lung was low in Iceland prior to 1940, but rose rapidly during and after World War II. Cigarette smoking was unpopular on the island until the start of the war (Drogendijk 1964; Drogendijk 1966).

There seems to be little doubt that tobacco smokers are at high risk of developing cancer of the upper gastrointestinal and respiratory tracts. The etiology of cancer of the mouth and pharynx is of particular interest. The risk of developing cancers of the mouth and pharynx is two to six times higher among heavy drinkers (Rothman 1975). In this carcinogenic process tobacco is believed to act as the initiator and alcohol as the promotor.

A similar combined effect of alcohol and smoking has been observed for cancer of the larynx. The risk of developing cancer of the larynx is ten times greater among tobacco smokers who are also heavy drinkers.

Solid statistical evidence establishing a relation between cancer of the lung and cigarette smoking was provided for the first time by Doll and Bradford Hill in 1950 in England and confirmed by Wynder in the United States.

Hammond and Garfinckel have shown that the incidence of cancer of the lung correlates well with the amount of cigarettes smoked, the degree of inhalation and the duration of the smoking habits. The risk for developing cancer of the lung is greater among smokers who start at a young age than among those who start later. Moreover, if one stops

smoking, the deleterious effects of cigarette smoking are reversed. Thus after a certain period of time has passed, the incidence of cancer of the lung in exsmokers is similar to that observed among nonsmokers.

Painstaking double-blind histological studies of large populations of smokers and nonsmokers perfomd by Auerbach strongly indicate that smoking alters the epithelium of the tracheobronchial tube. Lesions consist of basal cell hyperplasia with alterations of the normal epithelial cells into a more atypical cell, sometimes indistinguishable from cancer cells; in fact, these atypical epithelial cells may even present sites of early invasions. Whether these lesions are precancerous remains to be seen. Yet similar lesions have been produced experimentally in smoking dogs, some of which ultimately developed cancer (Auerbach et al. 1967a; Auerbach et al. 1970b).

Drogendijk has summarized the objections to assigning smoking an exclusive role in the incidence of cancer of the lung. (1) All heavy and moderate smokers do not develop cancer of the lung. (2) Cancer of the lung is occasionally observed among nonsmokers. (3) The incidence of lung cancer seems to have increased in dogs as well as in humans. Unless dogs smoke in secret, cigarette smoking can hardly be incriminated. On the basis of such observations, Drogendijk concluded that cigarette smoke is not the sole cause of cancer of the lung, but that other factors, such as air pollution by soot or possibly asbestos or consumption of alcohol, contribute to the etiology of cancer of the lung (Drogendijk 1966).

Cumulative effects of air pollution and cigarette smoke are believed to operate in the etiology of cancer of the lung. Except for very heavy smokers, from whom the incidence of cancer of the lung is the same among those who live in Liverpool and rural Wales, the incidence of cancer of the lung is significantly higher among smokers who live in the city than among those who live in the rural areas (Hitosugi 1968; Pike et al. 1975). Smoking and asbestos also combine their effects to cause cancer of the lung (Cole et al. 1975).

However, there seems to be no influence of previous pulmonary disease on the incidence of cancer of the lung (Dalhanm et al. 1967).

In conclusion, one can argue that factors other than tobacco smoke contribute to the incidence of cancer of the lung and question the mechanism by which cigarette smoke causes cancer of the lung, the evidence that cigarette smoking is a major contributory factor to the development of cancer of the lung is overwhelming.

Tobacco Carcinogenesis

Since cigarette smoke is a capital contributor to cancer of the lung, the disease could possible be prevented, if we knew the molecular' mechanisms by which cigarette smoke causes cancers. Such a pathogenic mechanism can be best understood by taking into account progress made in the knowledge of chemical carcinogenesis. Because the survival advantages of a cancer cell are transferred from one generation of cells to another and because most chemical carcinogens have been found to be mutagenic, it is believed that chemical carcinogens

initiate the alteration of gene expression that is characteristic of cancer by modifying DNA molecules. Some carcinogens, the alkylating agents for example, enter the cell and bind directly to DNA, but most substances that act as carcinogens are metabolically converted before binding to DNA. This is certainly the case for polycyclic hydrocarbons (e. g . benzene-pyrene) and acetylaminofluorene. The metabolic conversion occurs in the cytoplasm and is catalyzed by a group of microsomal enzymes, known as the mixed-function oxidases. The exact role of the mixed-function oxidases in chemical carcinogenesis is still questioned. They certainly generate carcinogenic metabolites, but it is also thought likely that they detoxify the procarcinogen and facilitate elimination. Although there is little doubt that carcinogens bind to DNA, conclusive evidence that such binding causes the distortion of the gene expression associated with cancer is lacking.

Even if we assume that the binding to DNA is causally related to the initiation of cancer, such an interpretation of the molecular events must take into account the existence of DNA repair. A carcinogen bound to a DNA base is susceptible to removal by two repair mechanisms, one operating in absence of DNA replication, the other in presence of DNA replication (Van Lancker 1977).

Attempts were also made to identify promoters in tobacco smoke (Bock 1968: Van Duuren et al. 1968: Wynder et al. 19691. Again a number of chemicals are good candidates for that function. However, definite identification of the promoters in cigarette smoke is far from conclusive. Volatile phenols are among the most likely substances that may act as promoters. Volatile aldehydes and acids and formaldehyde vapors are the other substances suspected to operate as promoters.

An association between cigarette smoking and cancer of the bladder is believed to exist (Lockwood 1961: Staszewski 1966: Kida et al. 1968). Trace amounts of A-naphthylamine; an established carcinogen in human bladder cancer, appear in tobacco smoke as a result of pyrolysis of certain amino acids (Masuda et al. 1967; Miller et al. 1967).

In spite of much investigation on the pathogenesis of cancer of the bladder in animals (Boyland et al. 1956; Brown et al. 1960; Cobb et al. 1965; Deeley et al. 1966; Bryan 1969; Conzelman et al. 1969; Brown et al. 1970), those chemicals found in smoke that contribute to the development of cancer of the bladder in humans have not been identified.

Increased amounts of tryptophan metabolites, 3-hydroxyanthranilic and 3-hydroxykynuzenine (two o-aminophenols) have been found in the urine of smokers (Kerr et al. 1965). Whether or not smoking inhibits o-aminophenol metabolism and whether the latter contribute to the pathogenesis of nonoccupational cancer of the bladder, remains to be established.

In conclusion, there are a number of chemicals in the tobacco smoke which might function as initiators or promoters. Preferred candidates are benzene pyrene for the former and phenols for the latter. conclusive evidence that either of these substances function as such in humans or in animals subjected to smoke is not available.

CONCLUSION

The smoking of tobacco products has expanded enormously in the 350 years following its rediscovery by white men because of craving and socioeconomical pressures. What was first believed to be a cure for many diseases turned out to contribute substantially to the occurrence of cardiovascular disease, chronic pulmonary diseases and cancer at various sites.

On the basis of the analysis of the components of tobacco smoke, it is possible to propose a working hypothesis on the contribution of tobacco smoke to the pathogenesis of disease. Quantitative data on the individual potential contributors are, however, not available primarily because of the multiplicity of potential noxious agents and their multistep mode of action.

The multistep development of cancer further complicates the identification of the cause of cancer at a given site. These sites include transformation, initiation and promotion.

Cells are transformed in vitro by viruses, chemicals and ionizing radiation. Transformation first observed with oncogenic viruses. Dulbecco has defined transformation as the process by which animal cells acquire inheritable properties different from those they had before infection (Van Lancker 19761. Various carcinogens have been used to achieve transformation in vitro, including polycyclic hydrocarbons, alkylating agents and substances. Transformation is accompanied by a number of alterations of the cellular, morphological, biochemical and functional properties. These changes include changes in membrane transport, membrane structure, adhesiveness to other cells and substratum, chromosomal number, growth characteristics, serum requirements and morphological features. The relationship between any of these alterations and the transforming event is not known. There is *clearly* a reprogramming of gene expression, it is not known if the pattern of reprogramming is random or nonrandom, or whether a single mechanism of transformation or a variety of molecular insults all ultimately result in a similar modification of cellular properties.

Transformation consists in modulation of gene expression which is transferred from one generation of cells to the next. The molecular trigger leading to transformation remains unknown. What is certain, however, is. that at least in some cases transformation is reversible.

In vivo experimental carcinogenesis occurs in two stages: initiation and promotion. The demonstration of initiation and promotion was done in the classical experiment of Berenblum in which a single application of methylcholanthrene was followed by repeated applications of croton oil. Such treatment yielded skin tumors. If the croton oil was applied before nmthylcholanthrene, no cancer developed. Croton oil alone is noncarcinogenic. A single dose of methylcholanthrene produces only a few tumors. Subsequent administration of croton oil increases the incidence and reduces the latent period for the appearance of cancer. On the basis of these experiments, it was

concluded that methylcholanthrene acts as an initiator, whereas croton oil acts as a promoter. The dissociation of the mechanism of carcinogenesis into two distinct steps has permitted investigators to determine whether the factors that modulate carcinogenesis act on the latent period or promotion. For example, the reduced incidence of tumors due to low caloric intake or the increased incidence of tumors caused by some hormonal treatments, appear to result from an influence on the promoter stage. We know little of the molecular events' associated with initiation or promotion. The general view is that initiation corresponds to a permanent molecular alteration of the cell, while promotion results in cell proliferation. Little is known of the permanent change that occurs during initiation, but it cannot be excluded that binding of the carcinogen to DNA or other macromolecules may be responsible for this change.

Initiation should not be confused with in vitro transformation. Although transformation also provides the cell with survival advantage through modulation of gene 'expression, these changes are reversible, at least in some cases. Certainly transformed cells, when transplanted into animals, yield neoplasms that invade, metastisize and kill the host. Whether or not such cells when transplanted undergo irreversible initiation is not established, neither is it known if reversible transformation takes place in vivo prior to irreversible initiation.

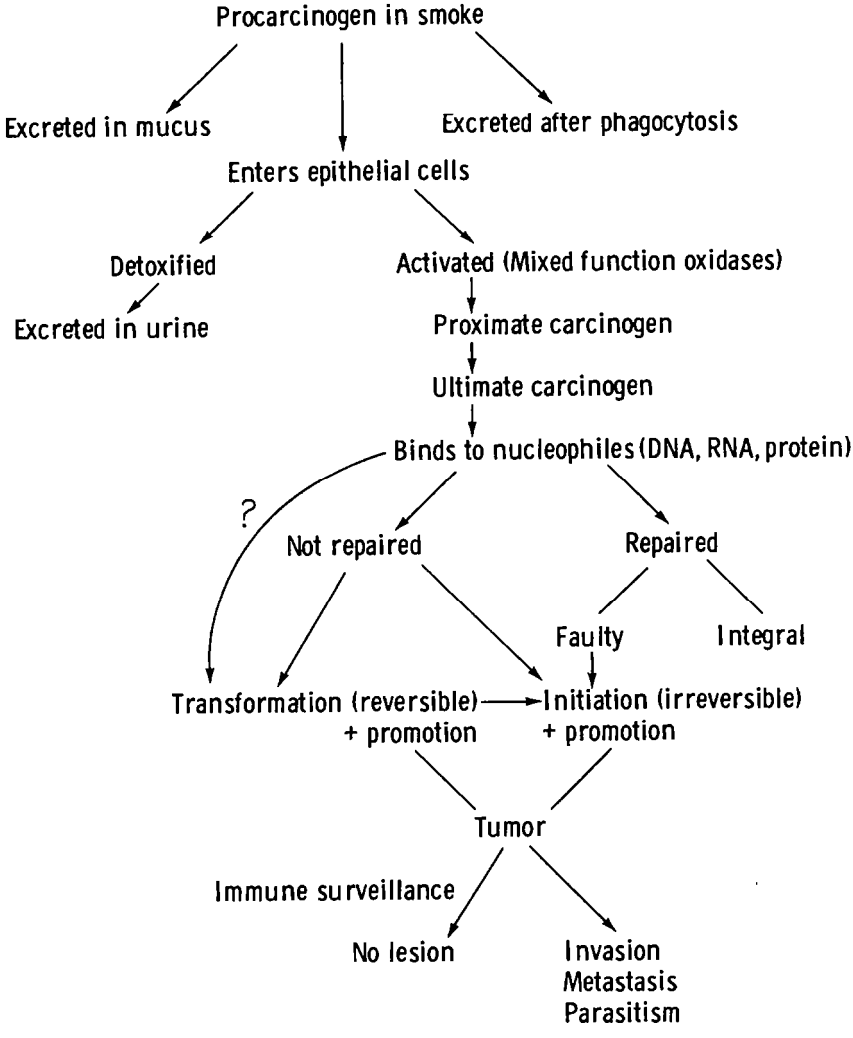
One can therefore hypothetically contemplate three different mechanisms of carcinogenesis in vivo: transformation leading to tumor formation without initiation, transformation leading to initiation followed by promotion, direct irreversible initiation followed by promotion (Figure 11). At present it is impossible to distinguish between these various modes of carcinogenesis.

In any case, the conversion of a normal cell into a cancer cell is a multistep event (Figure 11) involving metabolic conversion of a precarcinogen into a carcinogen, cell transformation, or initiation or both and promotion. The modulation of gene expression responsible for the conversion of a normal into a cancer cell leads to a population of cells with survival advantages capable of proliferation, invasion and metastasis.

With these various mechanisms of carcinogenesis in mind, one can think of ways of preventing tobacco carcinogenesis. Prevention could include: (1) elimination of precarcinogens, carcinogens and promoters by abolishing smoking. Yet, it is unlikely that the Surgeon General. or the Secretary of Health and Welfare would be more successful in stopping the habit of smoking than popes, kings, inquisitors and executors. Heavy taxes might, however, prove more effective than torture. (2) Elimination of precarcinogens, carcinogens or promoters from the smoking material; for example, by growing low tar producing tobacco or by treating the tobacco as to eliminate these substances. (3) In the alternative that tobacco smoke is not the causal agent of cancer and that environmental factors contribute, .in a major manner, to the process of carcinogenesis, elimination of carcinogens, precarcinogens and promoters from the environment might reduce the cancer risk. This situation may obtain for cancer of the. mouth, the esophagus

FIGURE 11

PATHOGENESIS OF LUNG CANCER



and even the larynx. The exclusion of alcohol from the diet might significantly reduce the risk for developing these cancers.

Experimental Tobacco Carcinogenesis

Although epidemiological data has clearly established the existence of a correlation between smoking and cancer, a clear-cut causal relationship between cigarette smoking and cancer has not been demonstrated. Such a causal effect can only be conclusively established in animal experiments. Although the approach to tobacco carcinogenesis is varied (Lorenz et al. 1943; Wynder et al. 1953b; Holsti et al. 1955; Orr et al. 1955; Wynder et al. 1955; Hamer et al. 1956; Guerin et al. 1957; Wynder et al. 1957a; Wynder et al. 1957b; Leuchtenberger et al. 1958; Bock et al. 1959; Bouchard et al. 1960; Leuchtenberger et al. 1960a; Leuchtenberger et al. 1960b; Peacock et al. 1960; Rigdon 1960; Blacklock 1961; Bock et al., 1962; Moore et al. 1962; Rockey et al. 1962; Homburger et al. 1963; Bock et al. 1964; Bock et al. 1965; Rockey et al. 1966; Reddy et al. 1967; Van Duuren 1968; Leuchtenber-, ger et al. 1969; Radford et al. 1969; Saffioti 1969; Saffioti 1970), the extrapolation of results obtained in animal experimentation to humans is difficult for several reasons. First, it is almost mossible to reproduce human smoking habits in animals. Secondly, because of the large number (at least 1200) of compounds and the various properties found in tobacco smoke, the identification of initiators and promoters of cancer is extremely difficult.

Studies in experimental tobacco carcinogenesis have been reviewed (Wynder et al. 1968). These authors have described tobacco smoke as an aerosol composed of gases, organic vapors and particulate matter. The smoke is divided in a side-stream, generated at the burning cone between puffs and a mainstream that travels through the length of the cigarette and is inhaled. The inhaled smoke is first held in the llputh where the hydrophilic volatiles are adsorbed. From there it reaches the lung where up to 90% of the aerosol particles are deposited. The particles may either cling to the mucosa and later be eliminated with the mucus secretion by the movement 'of epithelial ciliae or they may be phagocytized by macrophages which may later die and be expectorated. Consequently in addition to contributing to the pathogenesis of cancer by providing carcinogens, promoters, inhibitors of DNA repair, modulators of mixed-function oxidases, smoke condensates may also contribute inhibitors of ciliary movement and of phagocytosis (Dalhamn 1959;. Ballenger 1960; Carson et al. 1966; Green et al. 1967; Dalhamn et al. 1970).

Polycyclic hydrocarbons, heterocyclic hydrocarbons (Graham et al. 1957; Gellhom 1958; Kuratsune et al. 1965; Roe 1962; Graham et al. 1963; Van Duuren et al. 1966; Carugno et al. 1967; Lasnitzki 1968a; Lasnitzki 1968b: Chan et al. 1969: Cracker et al. 19701. N-nitrosamines, nitroalkanes, aromatic amines and Polonium 210 as among those substances found in tobacco smoke that are suspected to cause initiation. Polynuclear hydrocarbons, among them benzo(x)pyrene, are formed in the burning cone. Pyrolysis yields carbon-hydrogen radicals which through pyrosynthesis combine to form 4 to 6 rings aromatic hydrocarbons. Secondary amines present in smoke react with NO and NO_2 or

with alkaline nitrates to yield nitrosamines. Nitrosamines are carcinogenic in animals (Boyland et al. 1966; Serfontein et al. 1966; Johnson et al. 1968; Davies et al. 1969). Only traces of nitroalkanes, probably formed through the reaction of alkyl radicals with NO_2, are found in tobacco smoke. Again nitro-olefins are carcinogenic in animals. Traces of Polonium 210 are also found in cigarette smoke (Little et al. 1964; Kelley 1965; Gregory 1965; Little et al. 1965; Ferri et al. 1966a; Ferri et al. 1966b; Kilibarda et al. 1966; Little et al. 1967). The concentration of the isotope is greater in the lung, blood and liver of smokers than in the same organs of nonsmokers.

FOOTNOTES

1 Table II lists some of the names, given tobacco and Tables III and IV give a list of some of the most famous publications that appeared during the 16th and 17th centuries on the subject of tobacco and its relationship to medicine.

2 Cavendish tells us in a pamphlet how poison was once added to Napoleon's snuff (Cavendish 1857).

3 His pipe was blown to pieces by a bullet at Waterloo..

4 Quoique puisse dire Aristote et toute la philosophie il n'est rien d'egal au tabac; c'est la passion des honnetes gens et qui vit sans tabac n'est pas digne de vivre non seulement il I;ejouit et purge le cerveau humain mais encore il instruit les ames a la vertue et on apprend avec lui a devenir honnete homme. Moliere, Don Juan.

REFERENCES

Abelin, T. and Gsell, O.T.: Relative risk of pulmonary cancer in cigar and pipe smokers. *Cancer*, 20(8) :1288-1296, 1967.

Acheson, R.M. and Jessop, W.J.E.: Tobacco smoking and serum lipids in old men. *Br. Med. J.*, 2:1108-1111, 1961.

Adler, I.: Primary Malignant Growth of the Lungs and Bronchi. Paul B. Hoeber, New York, 1912.

Adler, I. and Hensel, O.: Intravenous injections of nicotine and their effects upon the aorta of rabbits. *J. Med. Res.*, 15:229-239, 1906.

Anbrus, J.L. and Mink, I.B.: Effect of cigarette smoking on blood . coagulation. *Clin. Pharmacol. Therap.*, 5(4):428-431, 1964.

Anderson, A.E., Jr., Hernandez, J.A., Holmes, W.L. and Foraker, A.G.: Pulmonary emphysema. Prevalence, severity, and anatomical patterns in macrosections, with respect to smoking habits. *Arch. Environ. Health*, 12(5):569-577, 1966.

Anderson, D.O.: Observations on the classification and distribution of pulmonary emphysema in Canada. *Can. Med. Assoc. J.*, 89:709-716, 1963.

Anderson, D.O., Ferris, B.G., Jr. and Zickmantel, R.: The Chilliwack Respiratory Survey, 1963. Part IV. The effect of tobacco smoking on the prevalence of respiratory disease. *Can. Med. Assoc. J.*, 92(20):1066-1076, 1965.

Apperson, G.L.: The Social History of Smoking. London, 1914.

Ashby, P., Dalby, A.M. and Millar, J.H.D.: Smoking and platelet stickiness. Lancet, 2:158-159, 1965.

Astrup, P.: Effects of hypoxia and of carbon monoxide exposures on experimental atherosclerosis. *Ann. Intern. Med.*, 71(2):426-427, 1969.

Astrup, P. and Pauli, H.G. [editors): A comparison of prolonged exposures to carbon monoxide and hypoxia in man. Reports of a joint Danish-Swiss study. *Scan. J. Clin. Lab. Invest.*, 22 (Suppplement 103) : 1-71, 1968.

Atkinson, L., Chester, I.C., Smyth, F.G. and Ten Seldam, R.D.J.: Oral cancer in New Guinea. *Cancer*, 17:1289-1298, 1964.

Auerbach, 0.; Hammond, E.C. and Garfinkel, L.: Smoking in relation to atherosclerosis of the coronary arteries. *N. Engl. J. Med.*, 273(15):775-779, 1965.

Auerbach, O., Hammond, E.C. and Garfinkel, L.: Thickening of walls of arterioles and small arteries in relation to age and smoking habits. *N. Engl. J. Med.*, 278(18):980-984, 1968.

Auerbach, O., Hammond, E.C. and Garfinkel, L.: Relation of smoking and age to emphysema. Whole lung section study. *N. Engl. J. Med.*, 286:853-857, 1972.

Auerbach, O., Hammond, E.C., Kinnan, D. and Garfinkel, L.: Emphysema produced in dogs by cigarette smoking. *J.A.M.A.*, 199(4):241-246, 1967. (a)

Auerbach, O., Hammond, E.C., Kirman, D. and Garfinkel, L.: Emphysema produced in dogs by cigarette smoking. In: Inhalation Carcinogenesis. Proceedings of a Biology Division, Oak Ridge National Laboratory Conference, Gatlinburg, Tennessee, October 8-11, 1969, (Hanna, M.G., Jr., Nettesheim, P. and Gilbert, J.R., editors), pp. 375-387, U.S. Atomic Energy Commission Symposium Series 18, April 1970. (a)

Auerbach, O., Hammond, E.C., Kirman, D. and Garfinkel, L.: Effects of cigarette smoking on dogs. II. Pulmonary neoplasms. *Arch. Environ. Health,* 21(6):754-768, 1970.(b)

Auerbach, O., Hammond, E.C., Kirman, D., Garfinkel, L. and Stout, A.P.: Histologic changes in bronchial tubes of cigarette-smoking dogs. *Cancer,* 20(12):2055-2066, 1967.(b)

Aviado, D.M. and Palecek, F.: Pulmonary effects of tobacco and related substances. I. Pulmonary compliance and resistance in the anesthetized dog. *Arch. Environ. Health,* 15(2):187-193, 1967.

Aviado, D.M., Samanek, M. and Folle, L.E.: Cardiopulmonary effects of tobacco and related substances. I. The release of histamine during inhalation of cigarette smoke and anoxemia in the heart-lung and intact dog preparation. *Arch. Environ. Health,* 12(6): 705-724, 1966.

Bailey, EM., Noland and Matias: Tobacco. *Conn. Agr. Exp. Station,* 295, 1928.

Balazs, T., Ohtake, S., Cummnings, J.R. and Noble, J.F.: Ventricular extrasystoles induced by epinephrine, nicotine, ethanol and vasopressin in dogs with myocardial lesions. *Toxicol. Appl. Pharmacol.,* 15(1):189-205, 1969.

Ball, K. and Turner, R.: Smoking and the heart. The basis for action. *Lancet,* 2:822-826, 1974.

Ballenger, J.J.: Experimental effect of cigarette *smoke on* human respiratory cilia. *N. Engl. J. Med.,* 263(17):832-835, 1960.

Bargeron, L.M., Jr., Ehmke, D., Gonlubol, F., Castellanos, A., Siegel, A. and Bing, R.J.: Effect of cigarette smoking on coronary blood flow and myocardial metabolism. *Circulation,* 15:251-257, 1957.

Bamett, A.J. and Boake, W.C.: Cigarette smoking and occlusive arterial disease of the legs. *Med. J. Aust.,* 1:240-242, 1960.

Becker, R.F. and King, J.E.: Studies on nicotine absorption during pregnancy. II. The effects of acute heavy doses on mother and neonates. *Am. J. Obstet. Gynecol.,* 95(3):515-522, 1966.

Becker, R.F., Little, C.R. and King, J.E.: Experimental studies *on* nicotine absorption in rats during pregnancy. III. Effect of subcutaneous injection of small chronic doses upon mother, fetus, and neonate. *Am. J. Obstet. Gynecol.,* 100(7):957-968, 1968.

Begg, T.B.: Characteristics of men with intermittent claudation. *Practitioner,* 194(2):202-207, 1965.

Bellet, S., West, J.W., Muller, O.F. and Manzoli, U.C.: Effect of nicetine on the coronary blood flow and related circulatory parameters. Correlative study in normal dogs and dogs with coronary insufficiency. *Circ. Res.,* 10(1):27-34, 1962.

Berkson, D.M., Stamler, J., Lindberg, H.A., Miller, W.A., Stevens, E.L., Soyugenic, R., Tokich, T.J. and Stamler, R.: Heart rate: An important risk factor for coronary mortality - ten-year experience of the Peoples Gas Co. Epidemiologic study (1958-68). In: Atherosclerosis. Proceedings of the Second International Symposium (Jones, R.J., ed.), pp. 382-389, Springer-Verlag, New York, 1970.

Bickennan, H.A. and Barach, A.L.: The effect of cigarette smoking on ventilatory function in patients with bronchial asthma and obstructive pulmonary emphysema. *J. Lab. Clin. Med.,* 43(3):455-462, 1954.

Blackburn, H., Jr., Orma, E., Hartel, G. and Punsar, S.: Tobacco smoking and blood coagulation: Acute effects on plasma Stypven time. *Am. J. Med. Sci.,* 238:448-451, 1959.

Blacklock, J.W.S.: An experimental study of the pathological effects of cigarette condensate in the lungs with special reference to carcinogenesis. *Br. J.* Cancer.,15(4):745-762. 1961.
Blomstrand, R. and Lundman, R. : Serum lipids, smoking and heredity. *Acta Med. Stand.*. 180(Supplement 455):51-60. 1966.
Boake, W.C.A. : A study of illness in a 'group of Cleveland families. XVIII. Tobacco smoking and respiratory infections. *N. Engl. J. Med.*. 259(26):1245-1249, 1958.
Bock, F.G. : The nature of tumor-promoting agents in tobacco products. *Cancer Res.,* 28(11) :2363-2368. 1968.
Bock, F.G. and Moore; G.E. : Carcinogenic activity of cigarette-smoke condensate. I. Effect of trauma and remote X irradiation. *J. Natl-Cancer Inst.,* 22(2):401-411, 1959.
Bock, F.G., Moore, G.E. and Clark, P.C.: Carcinogenic activity of cigarette smoke condensates. III. Biological activity of refined tar from several types of cigarettes. *J. Nat'l. Cancer Inst.,* 34(4):481-493, 1965.
Bock, F.G., more, G.E. and Crouch, S.K. : Tumor producing activity of extracts of unburned tobacco. Science, 145 (36841: 831-833. 1964.
Bock, F.G., Moore, G.E., Dowd, J.E. and Clark, P:C. : . Carcinogenic activity of cigarette smoke condensate. Biological activity of refined tar from certain brands of cigarettes. *J.A.M.A.,* 181(8) *:* 668-673, 1962.
Bogen, E.: Composition of cigarets and cigaret smoke. *J.A.M.A.,* 93: 1110-1114, 1929.
Bouchard, J. and May, R.M.: L'effet des goudrons de tabac sur les greffes sous-capsulaires de poumon chez la souris. (The effect of tobacco tars on sub-capsular implants of the lung in mice.) *Anat. Microsc. Morphol. Exp.,* 49(3):307-332, 1960.
Bonnevie and Grut, Rep. IX Cong. Indust. Med., London, September, 1948.
Boyland, E. and Roe, F.J.C.: Carcinogenic nitrosamines which may be present in cigarette smoke. In: Lung Tumors in Animals (Severi, L., ed.), pp. 667-676, University of Perugia, Division of Cancer Research, Perugia, June 1966.
Boyland, E. and Watson, G.: 3-hydrovanthranilic acid, a carcinogen induced by endogenous metabolism. *Nature,* 177(4514) : 837-838, '1956.
Brody, J.S. and Cobum, R.F.: Carbon monoxide-induced arterial hypoxemia . *Science,* 164(3885):1297-1298, 1969.
Brown, H., Sellwood, R.A., Harrison, C.V. and Martin, P. : Thrombo-angiitis obliterans. *Br. J. Surg.,* 56(1):59-63, 1969.
Brown, R.R., Price, J.M., Bumey, S.W. and Friedell, G.H.: Lack of .effect of smoking on the excretion of tryptephan metabolites by man. *Cancer Res.,* 30(3) :611-614, 1970.
Brown, R.R., Price, J.M., Satter, E.J. and Wear, J.B.: The metabolism of tryptophan in patients with bladder cancer. *Acta Unio Int. Contra Cancrum,* 16:299-303, 1960.
Bryan, G.T.: Role of tryptophan metabolites in urinary bladder cancer. *Am. Indust. Hyg. Assoc.,* 30(1):27-34 1969.
Bucher, K.: Les effets de la nicotine. *Rev. ciba* 78:2730-2732, 1950.
Buechley, R.W., Drake, R.M. and Breslow, L. : Relationship of amount of cigarette smoking to coronary heart disease mortality rates in men. *Circulation,* 18(6) *:* 1085 1090, 1958.
Buncher, C. R. : Cigarette smoking and, duration of pregnancy. *Am. J. Obstet. Gynscol;* 103(7) ;942:946, 1969.
Burn, J.H. and Rand, M.J.: Action of nicotine on the heart. *Br. Med. J.* 1:137-139, 1958.

Carson. S.. Goldhamer. R. and Carpenter, R.: Responses of ciliated epithelium to irritants. Mucus-transport in the respiratory tract. *Am. Rev. Respir. Dis.*. 93(3.Part 2):86-92. 1966.
Carugno, N. and Rossi, S:: Evaluation of poiynuclear hydrocarbons in cigarette smoke by glass capillary columns. *J. Gas Chrom.*, 5(2): 103-106, 1967.
Cederlof, R., Friberg, L. and Hrubec, Z.: Cardiovascular and respiratory symptoms in relation to tobacco smoking. A study on American twins. *Arch. Environ. Health,* 18(6):934-940, 1969.
Chan, P.C, Sanders, F.K. and Wynder,.E.L.: Effect of 3,4-benzo(a)-pvrene on mouse lung orimordia in vitro. *Nature,* 228(5208):847-848, 1969.
Chiang; S.T. and Wang, B.C.: Acute effects of cigarette smoking on pulmonary function. *Am. Rev. Respir. Dis.,* 101(6):860-868, 1970.
Chevalier, R.B., Krumholz, R.A. and Ross, J.C.: Reaction of non-smokers to carbon monoxide inhalation. Cardiopulmonary responses at rest and during exercise. *J.A.M.A.,* 198(10):1061-1064, 1966.
Choi, Y.Y.: Effect of nicotine upon cholesterol-induced atherosclerosis in rabbits. *N. Med. J.,* 10(7):685-693, 1966.
Clark, V.A., Chapman, J.M. and Coulson, A.H.: Effects of various factors on systolic and diastolic blood pressure in the Los Angeles Heart Study. *J. Chronic Dis.,* 20:571-578, 1967.
Cobb, B.G. and Anseli, J.S.: Cigarette smoking and cancer of the bladder. *J.A.M.A.,* 193(5):329-332, 1965.
Cohen, S.I., Deane, M. and Goldsmith, J.R.: Carbon monoxide and survival from myocardial infarction. *Arch. Environ. Health,* 19(10): 510-517, 1969.
Cole, P. and Goldman, M.B. Occupation. In: Persons at High Risk of Cancer. An Approach *to* cancer etiology and control (Fraumeni, J.F., Jr., ed.), pp: 167-184, Academic Press, New York, 1975.
Conzelman. G.M.. Jr.. Moulton. J.E.. Flanders. L.E.. III. Springer. K. and Crout, D:W.: Induction of transitional cell carcinomas of the urinary bladder in monkeys fed 2-naphthylamine. *J. Natl. Cancer Inst.,* 42(5):825-836, 1969.
Cook, W.A. and Webb, W.R.: Surfactant in chronic smokers. *Ann. Thorac. Surg.,* 2(3):327-333, 1966.
Cooper, Lamb, Sanders and first: Role of tobacco-smoking in production of cancer. *J. Hygeine,* 32:293-300, 1932.
Cracker, T.T.: Effect of benzo(a)pyrene on hamster, rat, dog, and monkey respiratory epithelia in organ culture. In: Inhalation Carcinogenesis. Proceedings of a Biology Division, Oak Ridge National Laboratory Conference. Gatlinburg. Tennessee. October 8-11, 1969 (Hanna, M:G., Jr., Nettesheim, P. and Gilbert, J.R., editors), pp. 433-443, U.S. Atomic Energy Commission Symposium Series 18, April 1970.
Crowdy, J.P. and Sowden, R.R.: Cigarette smoking and respiratory ill-health in the British army. *Lancet,* 1(7918):1232-1234, 1975.
Dalhanm, T.: The effect of cigarette smoke on ciliary activity in the upper respiratory tract. *A.M.A. Arch. Otolayngol.,* 70:166-168, 1959.
Dalhamn, T.: Effect of cigarette smoke on ciliary activity. *Am. Rev. Respir. Dis.,* 93(3):108-114, 1966.
Dalhanm, T. and Rylander, R.: Ciliastic action of smoke from filter-tipped and non-tipped cigarettes. Nature, 201(4917):401-402, 1964.
Dalhanm, T. and Rylander, R.: Ciliastic action of cigarette smoke. Varying exposure times. *Arch. Ototaryngol.,* 8(4):379-382, 1965.

Dalhamn, T. and Rylander, R.: Tar content and ciliotoxicity of cigarette smoke. *Acta Pharmacol. Toxicol.,* 25(3):369-372, 1967.
Dalhanm, T. and Rylander, R.: Ciliotoxicity of cigarette smoke and its volatile components. *Am. Rev. Respir. Die.,* 98(3):509-511, 1968.
Dalhamn, T. and Rylander, R.: Ciliotoxicity of cigar and cigarette smoke. *Arch. Environ. Health,* 20(2):252-253, 1970.
Davies, D.F.: The relationship between smoking and lung cancer. *Am. Cancer Society,* 1959.
Davies, D.P., Gray, O.P., Ellwood, P.C. and Abernathy, M.: Cigarette smoking in pregnancy: Associations with maternal weight gain and fetal growth. *Lancet,* 1:385-387, 1976.
Davies, R.F. and Day, T.D.: A study of the comparative carcinogenicity of cigarette and cigar smoke condensate on mouse skin. *Br. J. Cancer,* 23(2):363-368, 1969.
Deeley, T.J. and Cohen, S.L.: The relationship between cancer of the bladder and smoking. In: Bladder Cancer. Proceedings of the 5th Inter-American Conference on Toxiciology and Occupational Medicine (Deichmann, W.B. and Lampe, K.L., editors), pp. 163-169, University of Miami, School of Medicine, Coral Gables, 1966.
Dietrich, S; and Schimert, G.: Uber Nicotinwirkungen auf den Kreislauf. *Z. Klin. Med.*. 135:718-732. 1939.
Doll, R. and Hill, A.B.: A study of 'the aetiology of carcinoma of the lung. *Br. Med. J.,* 2:1271-1286, 1952.
Doll, R. and Hill, A.B.: Mortality in relation to smoking: Ten years' observations of British doctors. (Part I) *Br. Med. J.,* 1(5395): 1399-1410, 1964,(a)
Doll, R. and Hill, A.B.: Mortality in relation to smoking: Ten years' observations of British doctors.(Concluded) *Br. Med. J.,* 1(5396): 1460-1467, 1964.(b)
Downing, G.C. and Chapman, W.E.: smoking and pregnancy. A statistical study of 5,659 patients. *Calif. Med.,* 104(3):187, 1966.
Doyle, J.T., Dawber, T.R., Kannel, W.B., Kinch, S.H. and Kahn, H.A.: The relationship of cigarette smoking to coronary heart disease. The second report of the combined experience of the Albany, N.Y. and Framingham, Mass. studies. *J.A.M.A.,* 190(10):886-890, 1964.
Drogendijk, A.C.: Sigaretten roken en zelfmoord. *Ned. T. Geneesk,* 108:2481-2485, 1964.
Drogendijk, A.C.: Smoking and lung cancer. *Triangle,* 7:166-169, 1966.
Duffus, G.M. and MacGillivray, I.: The incidence of pre-eclamptic toxaemia in smokers and nonsmokers. *Lancet, 1(7550):994-995,* 1968.
Edwards, F., McKeown, T. and Whitfield, A.G.W.: Association between smoking and disease in men over sixty. *Lancet,* 1:196-200, 1959.
English, J.P., Willius, F.A. and Berkson, J.: Tobacco and coronary disease. *J.A.M.A.,* 115(16):1327-1329, 1940.
Ewing, J.: Neoplastic Diseases. A Treatise on Tumors. W.B. Saunders and Co., Philadelphia, 1940.
Falk, J.L., Tremer, H.M. and Kotin, P.: Effect of cigarette smoke and its constituants on ciliated mucus-secreting epithelium. *J. Natl. Cancer Inst.*. 23(5):999-1012, 1959.
Ferri, E.S. and Baratta, E.J.: Polonium-210 in tobacco, cigarette smoke and selected human organs. *Public Health Reports,* 81(2): 121-127, 1966.(a)
Ferri, E.S. and Baratta, E.J.: Polonium-210 in tobacco products and human tissues. *Rudiol. Health Data Rep.,* 7(9):485-488, 1966.(b)

Fleckseder, R. : Dber den Bro nchielkrebs und einige seiner Entstehungsbedingungen. *Munch. Med. Wschr.,* 83:1585-1588, 1936.
Flick, A.L. and Paton, R.R.: Obstructive emphysema in cigarette smokers. *A.M.A. Arch. Intern. Med.,* 104:518-526, 1959.
Folle, L.E., Samanek, M. and Aviado, D.M.: Cardiopulmonary effects of tobacco and related substances. II. Coronary vascular effects of cigarette smoke and nicotine. *Arch. Environ. Health,* 12(6):712-716, 1966.
Forte, I.E., Williams, A.J., Potgieter, L., Schmitthenner, J.E., Hafkenschiel. J.H. and Riegel. C.: Coronary blood flow and cardiac oxygen metabolism during nicotine-induced increases in left ventricular work. *Ann. NY Acad. Sci..* 90(1):174-185. 1960.
Frankl, W.S., Friedman, R. and Soloff, L.A.: Cardiac output, blood pressure and free fatty acid responses to smoking in the nonbasal state. *Am. J. Med. Sci.,* 252(1):39-44, 1966.
Franklin, W. and Lowell, F.C.: Unrecognized airway obstruction associated with smoking. A probable forerunner of obstructive pulmonary emphysema. *Ann. Interm. Med.,* 54(3):379-386, 1961.
Frazier. T.M.. Davis. G.H.. Goldstein. H. and Goldberg. I.D.: Cigarette smoking and prematurity: A prospective study. *Am. J. Obstet Gynecol.,* 81(5):988-996, 1961.
Friedell, G.H., Bumey, S.W., Bell, J.R. and Soto, E.: Pathology as related to tryptophan metabolite excretion, occupational history, and smoking habits in patients with bladder cancer. *J. Natl. Cancer Inst.,* 43(1):303-306, 1969.
Friedell, M.T.: Effect of cigarette smoke on the peripheral vascular system. Radioactive iodinated albumin used as indicator of volumetric change. *J.A.M.A.,* 152(10):897-900, 1953.
Gellhom, A.: The cocarcinogenic activity of cigarette tobacco tar. *Cancer Res.,* 18(6):510-517, 1958.
Gettler, A.O. and Mattice, M.R.: "Normal" carbon monoxide content of blood. *J.A.M.A.,* 100:92-97, 1933.
Giammona. S.T.: Effects of cigarette smoke and plant smoke on pulmonary surfactant. *Am. Rev. Respir. Dis.,* 96(3): 539-541, 1967:
Gofman, J.W., Lindgren, F.T., Strisower, B., Delalla, O., Glazier, F. and Tamplin, A.: Cigarette smoking, serum lipoproteins, and coronary heart disease. *Geriatrics,* 10(8):349-354, 1955.
Goldsmith, J.R. and Landaw, S.A.: Carbon monoxide and human health. *Science,* 162(3860):1352-1359, 1968.
Graham, E.A., Croninger, A.B. and Wynder, E.L.: Experimental production of carcinoma with cigarette tar. IV. Successful experiments with rabbits. *Cancer Res.,* 17(11):1058-1066, 1957.
Graham, S., Crouch, S., Levin, M.L. and Bock, F.G.: Variations in amounts of tobacco tar retrieved from selected models of smoking behavior simulated by smoking machines. *Cancer Res.,* 23(7) :1025-1030, 1963.
Green, G.M. and Carolin, D.: The depressant effect of cigarette smoke on the in vitro antibacterial activity of alveolar macrophages. *N. Engl. J. Med.,* 276(8):421-427, 1967.
Gregory, L.P. : Polonium-210 in leaf tobacco from four countries. *Science,* 150:74-76, 1965.
Guerin, M. and Cuzin, J.-L. : Action carcinogene du goudron de fumee de cigarette sur la peau de souris. (Carcinogenic action of tar from cigarette smoke on mouse skin.) *Bull. Assoc. Fran. Etud. Cancer,* 44 (3) : 387-408, 1957.

Haddon, W., Jr., Nesbitt, R.E.L. and Garcia, R.: Smoking and pregnancy: .Carbon monoxide in blood during gestation and at term. *Obstet. Gynecol.,* 18(3):262-267, 1961.
Haenzel, W.. and Shimkin, M.B.: Smoking patterns and epidemiology of lung cancer in the U.S. - Are they compatible? *J. Natl. Cancer Inst.,* 16:1417-1441, 1956.
Haenzel, W. and Shimkin, M.B.: A retrospective study of lung cancer in women. *J. Natl. Cancer Inst.,* 21(5):825-842, 1958.
Hamer, D. and Woodhouse, D.L.: Biological tests for carcinogenic action of tar from cigarette smoke. *Br. J. Cancer,* 10:49-53, 1956.
Hammond, E.C.: The place of tobacco in the etiology of lung cancer. *CA;* May, 1952.
Hammond, E.C.: Smoking in relation to lung cancer - A follow-up study. *Conn. State Med. J.,* 18:3-g, 1954.
Hammond, E.C. Tobacco. In: Persons at High Risk of Cancer. An approach to cancer etiology and control.-(Fraumeni, J.F., Jr., ed.), pp. 131-138. 1975.
Hammond, E.C. and Garfinkel, L.: Coronary heart disease, stroke and aortic aneurysm. Factors in the etiology. *Arch. Environ. Health* 19(2):167-182. 1969.
Hammond; E.C. and Horn, D.: Tobacco and lung cancer. *CA,* 2(3):97-98, 1952.
Hardy, J.B. and Mellits, E.D.: Does maternal smoking during pregnancy have a long-term effect on the child? *Lancet,* 2:1332-1336, 1972.
Hass, G.M., Landerholm, W. and Hemmens, A.: Production of calcific athero-arteriosclerosis and thromboarteritis with nicotin, vitamin D, and dietary cholesterol. *Am. J. Pathol.,* 49(4) :739-771, 1966.
Hemandez, J.A.. Anderson, A.E.. Jr., Holmes, W.L. and Foraker, A.G.: Pulmonary parenchymal defects in dogs following prolonged cigarette smoke exposure. *Am. Rev. Resvir. Die..* 93(1):78-83. 1966.
Heron, H.J.: The effects of smoking during pregnancy: A review with a preview. *N.Z. Med. J.,* 61:545-548, 1962.
Higgins, I.T.T.: Tobacco smoking, respiratory symptoms, and ventilatory capacity. Studies in random samples of the population. *Br. Med. J.,* 1:325-329, 1959.
Hirayama, 'T.: An epidemiological study of oral and pharyngeal cancer in Central and South-east Asia. *Bull. WM. Hlth. Ora..* 34:41-69. 1966.
Hitosugi, M.: Epidemiological study of lung cancer with special reference to the effect of air pollution and smoking habit. *Bull. Inst. Pub. Health,* 17(3):237-256, 1968.
Holland, R.H. et al: A study of arsenic in regular-sized unfiltered and filtered cigarettes. . *Cancer,* 11(6):1115-1118, 1958.
Holsti. L.R. and Ermala. P.: Paoillary carcinoma of the bladder in mice, obtained after peroral administration of tobacco tar. *Cancer,* 8(4):679-682, 1955.
Homburger, F., Treger, A. and Baker, J.R.: Mouse-skin painting with smoke condensates from cigarettes made of pipe, cigar, and cigarette tobaccos. *J. Natl. cancer Inst.,* 31(b)-1445-i459, 1963.-
Ide. G.. Suntzeff. V. and Cowdrv. E.V.A.: A comparison of the histopathology of tracheal and bronchial epithelium of smokers and non-smokers. *Cancer,* 12(3):473-484, 1959.
Irving, D.W., Yamamoto, T.: Cigarette smoking and cardiac output. *Br. Heart J.,* 25:126-132, 1963.
James, R.H.: Prior smoking as a determinant of the distribution of pulmonary ventilation. *Am. Rev. Respir. Dis.,* 101(1):105-107, 1970.

Johnson, D.E., Millar, J.D. and Rhoades, J.W.: Nitrosamines in tobacco smoke. In: Toward a Less Harmful Cigarette (Wynder, E.L. and Hoffman, D., editors), pp. 181-189, U.S. Department of Health, Education, and Welfare, Public Health Service, National Cancer Institute Monograph 28, Bethesda, Md., June 1968.

Jongbloed, J.: Uber den Kohnlenoxydgehalt der Alveolarluft beim Tabakrauchen. *Arch. Int. Pharmacodyn. Therap.,* 63:346-358, 1939.

Jordans, G., Zijilmans, A. and Broos, J.: Lead Poisoning in cigar makers. *Ned. T. Geneeek.,* 80:304-311, 1936.

Kahn, H.A.: The Dorn study of smoking and mortality among U.S. veterans: Report on 84 years of observations. In: Epidemiological Approaches to the Study of Cancer and Other Chronic Diseases. (Haenzel, W., ed.), pp. l-25, U.S. Public Health Service, National Cancer Institute Monograph No. 19, Bethesda, Md., January 1966.

Kaminski, E.J., Fancher, O.E. and Calandra, J.C.: In viva studies of the ciliastic effects of tobacco smoke. Absorption of ciliastic components by wet surfaces. *Arch. Environ. Health,* 16(2):188-193, 1968.

Kannel, W.B., Castelli, W.P. and McNamara, P.M.: Cigarette-smoking and risk of coronary heart disease. Epidemiologic clues to pathogenesis. The Framingham study. In: Toward a Less Harmful Cigarette (Wynder, E.L. and Hoffman, D., editors), pp. 9-20, U.S. Department of Health, Education, and Welfare, Public Health Service, National Cancer Institute Monograph No. 28, Bethesda, MI., June 1968.

Kedra, M. and howski, G.: The influence of tobacco smoking on the development of atherosclerosis and on the composition of blood lipids. *Polish Med. J.,* 5(1):37-43, 1966.

Kedra, M. and Korolko, A.: Tobacco smoking and blood clotting. *Bull. Polish Med. Sci. Hist,* 8:145-148, 1965.

Kelley, T.F.: Polonium-210 content of mainstream cigarette smoke. *Science,* 149(3683):537-538, 1965.

Kerr, W.K., Barkin, M., Levers, P.E., Woo, S.K.C. and Menczyk, Z.: The effect of cigarette smoking on bladder carcinogens in man. *Can. Med. Assoc. J.,* 93:1-7, 1965.

Kershbaum, A., Bellet, S., Hirabayashi, M. and Feinberg, L.J.: Regular filter-tip, and modified cigarettes. Nicotine excretion, free fatty acid mbilization and catecholamine excretion. *J.A.M.A.,* 201(7): 545-546, 1967.(a)

Kershbawn, A., Bellet, S. and Khorsandian, R.: Elevation of serum cholesterol after administration of nicotine. *Am. Heart J.,* 69(2): 206-210, 1965.

Kershbam, A., Khorsandian, R., Caplan, R.F., Bellet; S. and Feinberg, L.J.: The role of catechomaines in the free fatty acid responses of cigarette smoking. *Circulation,* 28(1):52-57, 1963.

Kershbam, A., Osada, H., Scriabine, A., Bellet, S. and Pappajohn, D.J.: Influence of nicotine on the mobilization of free fatty acids from rat adipose tissue in vitro and in the isolated perfused dog limb. *Circulation,* 36(4,Supplement 2):1-20, 1967.(b)

Kida, H., Omoto, T., Sakamoto, K. and Momose, S.: Fukuoka ken hokubu ni okeru boko shuyo no ekigaku to tokei. (Statistics and epidemiology of urinary bladder tumors in northern Fukuoka, Japan.) *Hifu to Hinyo,* 30(5):883-889, 1968.

Kien, G.A. and Sherrod, T.R.: Action of nicotine and smoking on coronary circulation and myocardial oxygen utilization. *Am. NY Acad. Sci.,* 90(1):161-173, 1960.

Kier, L.D., Yamasaki, E. and Ames, B.N.: Detection of mutagenic activity in cigarette sake condensates. *Proc. Nat. Acad Sci. USA* 71(10):4159-4163, 1974.

Kilbum, K.H. and McKenzie, W.: Leukocyte recruitment to airways by cigarette smoke and particla phase in contrast to cytotoxicity of vapor. *Science,* 189:634-637, 1975.

Kilibarda, M., Petrovic, D., Panov, D. and Djuric, D.: Contamination with polonium-210, uranium and radium-226 due to smoking. *Health Phys.,* 12:1808, 1966.

Kjeldsen, K.: Smoking and Atherosclerosis. Investigations on the significance of the carbon monoxide content in tobacco smoke in atherogenesis. Munksgaard, Copenhagen, 1969.

Kjeldsen, K. and Mozes, M.: Buerger's disease in-Israel. Investigations cm carboxyhemoglobin and serum cholesterol levels after smoking. *Acta Chir. Scand..* 135(6):495-498, 1969.

Konttinen, A.: Cigarette smoking and serum lipids in young men. Br. *Med. J.* 1:1115-1116, 1962.

Konttinen, A. and Rajasalmi, M.: Effect of heavy cigarette smoking on post-prandial triglycerides, free fatty acids, and cholesterol. Br. Med. J., 1(5334):850-852, 1963.

Koskowski, W.: The Habit of Tobacco Smoking. Staples Press Limited, London, 1955.

Krumholz, R.A., Chevalier, R.B. and Ross, J.C.: Cardiopulmonary function in young smokers. A comparison of pulmonary function measurements and some cardiopulmonary responses to exercise between a group of young smokers and a comparable group of nonsmokers. *Ann. Intern. Med.,* 60(4):603-610, 1964.

Krumholz, R.A., Chevalier, R.B. and Ross, J.C.: Changes in cardiopulmnary functions related to abstinence from smoking. Studies in 'young cigarette smokers at rest and exercise at 3 and 6 weeks of abstinence. *Ann. Intern. Med.,* 62(2):197-207, 1965.(a)

Krumholz, R.A., Chevalier, R.B; and Ross, J.C.: A comparison of pulmonary compliance in young smokers and nonsmokers. *Am. Rev. Respir. Dis.,* 92(1):102-107, 1965.(b)

Kuhn, R.A.: Mode of action of tobacco smoke inhalation upon the cerebral circulation. *Ann. NY Acad. Sci.,* 142(Article 1):67-71, 1967.

Kumar, D. and Zourlas, P.A.: Studies on human premature births. II. In viva effect of smoking and in vitro effect of nicotine on human uterine contractility. *Am. J. Obstet. Gynecol.,* 87(3):413-417, 1963.

Kuratsune, M., Kohchi, S. and Horie, A.: Carcinogenesis in the esophagus. I. Penetration of benzo(a)pyrene and other hydrocarbons into the esophageal mucosa. *Cann,* 56(2):177-187, 1965.

Lambert, P.M. and Reid, D.D.: Smoking, air pollution, and bronchitis in Britain. *Lancet,* 1(7652):853-857, 1970.

Lasnitzci, I.: The effect of a hydrocarbon-enriched fraction of cigarette smoke condensate on human fetal lung grown in vitro. *Cancer Res.,* 28(3):510-513, 1968.(a)

Lasnitzki. I.: The effect of a hydrocarbon-enriched fraction from cigarette smoke on mouse tracheas growth in vitro. *Br. J. Cancer,* 22(1):105-103, 1968.(b)

Leaders, F.E. and Long, J.P.: Action of nicotine on coronary vascular resistance in dogs. *Am. J. Physiol.,* 203(4):621-65, 1962.

Lehmann, Gunther and Michaelis: Tabakgenuss und Adrenalinspiegal. *Naunyn-Schmiedeberg Arch. Exp. Path. Phamakol.,* 02:627-632, 1943.

Leuchtenberger, C. and Leuchtenberger, R.: Cytologic and cytochemical effects on primary mouse kidney tissue and lung organ cultures after exposure to whole, fresh smoke and its gas phase from unfiltered, charcoal filtered, and cigar tobacco cigarettes. *Cancer Res.,* 29(4) :862, 1969.

Leuchtenberger, C., Leuchtenberger, R; and Doolin, P.F. : A correlated histological, cytological, and cytochemical study of the tracheobronchial tree and lungs of mice exposed to cigarette smoke. I. Bronchitis with atypical epithelial changes in mice exposed to cigarette smoke. *Cancer,* 2(3):490-506, 1958.

Leuchtenberger, C., Leuchtenberger, R., Zebrun, W. and Shaffer, P. : A correlated histological, cytological, and cytochemical study of the tracheobronchial tree and lungs of mice exposed to cigarette smoke. II. Varying responses of major bronchi to cigarette smoke, absence of bromchogenic carcinoma after prolonged exposure, and disappearance of bronchial lesions after cessation of exposure. *Cancer,* 13(4) : 721-732, 1960. (a)

Ieuchtenberger, R., Leuchtenberger, C., Zebrun, W. and Shaffer, P. : A correlated histological, cytological, and cytochemical study of the tracheobronchial tree and lungs of mice exposed to cigarette smoke. III. Unaltered incidence of grossly visible adenomatous lung tars in female CF mice after prolonged exposure to cigarette smoke. *Cancer,* 13(5) :956-958, 1960. (b)

Levin, M. L., Goldstein, H. and Gerhardt, P.R.: Cancer and tobacco smoking. *J.A.M.A.,* 143:336-338, 1950.

Liebeschuetz, H.J.: Respiratory signs and symptoms in young soldiers and their relationship to smoking. *J. R. Army Med. Corps,* 105: 76-81, 1959.

Lilienthal, J.L., Jr.: Carbon monoxide. *Pharmacol. Rev.,* 2:324-354, 1950.

Little, J.B. and Radford, E.P., Jr.: Polonium-210 in bronchial epitheliwn of cigarette smokers. *Science,* 155(3762):606-607, 1967.

Little, J.B., Padford, E.P., Jr., McCombs, H.L. and Hunt, V.R.: Polonim-210 in lungs and soft tissues of cigarette smokers. *Radiat. Res.,* 22: 209, 1964.

Little, J.B., Radford, E.P., Jr., McCoombs, H.L. and Hunt, V.R.: Distribution of polonium-210 in pulmonary tissues of cigarette smokers. *N. Engl. J. Med.,* 273(25) :1343-1351, 1965.

Lockwood. K. : On the etiology of bladder tumors in Kobenhavn-Frederiksberg. An inquiry of 369 patients and 369 controls. *Acta Pathol. Microbiol. Scand.,* 51(Supplementum 145) :l-166, 1961.

Lorenz. E.. Stewart. H.L.. Daniel. J.H. and Nelson. C.V.: The effects of breathing tobacco smoke on strain A mice. *Caner Res.,* 3:123. 1943.

Lowe, C.R. : Effect of mothers' smoking habits on birth weight of their children. *Br. Med. J.*. 2:673-676. 1959.

MacMahon, B.: Overview: Environmental Factors. In: Persons at High Risk of Cancer. An approach to cancer etiology and control (Fraumeni, J.F., Jr., ed.), pp. 285-290, Academic Press, New York, 1975.

Mantell, C.D.: Smoking in pregnancy. The role played by carbonic anhydrase . *N.Z. Med. J.,* 63:601-603, 1964.

Masuda, Y., Moori, K. and Kuratsune, M.: Studies on bladder carcinogens in the human environment. I. Naphthylamines produced by pyrolysis of amino acids. *Int. J. Cancer,* 2:489-493, 1967.

McDermott, M. and Collins, M.M.: Acute effects of smoking on lung airways resistance in normal and bronchitic subjects. *Thorax,* 20:562-569, 1965.
McNally, W.D. : Tar in cigarette smoke and its possible effects. *Am. J. Cancer,* 16:1502-1514, 1932.
Megahed, G.E., Senna, G.A., Eissa, M.H., Saleh, S.Z. and Eissa, H.A.: Smoking versus infection as the aetiology of bronchial mucous gland hypertrophy in chronic bronchitis. *Thorax,* 22(3):271-278, 1967.
Mshta, F.S., Sanjana, M.K., Shroff, B.D. and Doctor, R.H.C. : Incidence of leukoplakia among "pan" (Beta1 leaf) chewers and "bidi" smokers: A study of a *sample-survey. Indian J. Med. Res.,* 49:393-399, 1961.
Mendenhall. W.L. and Shreeve. K.E.: The effect of cigarette smoke on the tracheal cilia. (Abstract) 28th Annual Meeting: April 21-24, 1937, Memphis, Tennessee. The Scientific Proceedings of the American Society for Pharmacology and Experimental Therapeutics, Inc.. *J. Pharmacol. Exp. Ther.,* 60:111-112, 1937.
Miller, C. and Bondurant, S. : Effects of cigarette smoke on the surface characteristics of lung extracts. *Am. Rev. Respir. Dis.,* 85(5):692-696, 1962.
Miller, R.L. and Stedman, R.L. : Essential absence of beta-naphthlamine in cigarette smoke condensate. *Tobacco,* 165 (8) : 32, 1967.
Miyazaki, M.: Circulatory effect of cigarette smoking, with special reference to the effect on cerebral hemodynamics. *Jpn circ. J.,* 33(9) :907-912, 1969.
Moore, C. and Christopher-son, W.M.: The effect of cigarette-smoke condensate on hamster tissues. Exteriorized oral pouch and skin. *A.M.A. Arch. Surg.,* 84(4):425-431, 1962.
Mosier, H.D., Jr. and Armstrong, M.K. : Effect of maternal nicotine intake on fetal weight and length in rats. *Proc. Soc. Exp. Biol. Med.,* 124(4) :1135-1137, 1967.
Motley, H.L. and Kuzman, W.J.: Cigarette smoke. Its effect on pulmonary function measurements. *Calif. Med.,* 88(3):211-220, 1958.
Mulcahy, R. and Hickey,. N.J. : Cigarette smoking habits of patients with coronary heart disease. *Br. Heart J.,* 28:404-408, 1966.
Murdoch, D.E. : Birth weight and smoking. *Nebr. State Med. J.,* 48(11): 604-606, 1963.
Murphy, E.A.: Thrombozyten, Thrombose und Gerinnung (Thrombocytes, thrombosis and clotting.) In: Nikotin: Pharmakologie und Toxikologie des Tabakrauches (Schievelbein, H., ed.), pp. 178-192, Georg Thieme Verlag, Stuttgart (West Germany), 1968.
Mustard, J.F. and Murphy, E.A.: Effect of smoking on blood coagulation and platelet survival in man. *Br. Med. J.,* 1(5334) :846-849, 1963.
Nadeau, R.A. and .James, T.N. : Effects of nicotine on heart rate studied by direct perfusion of sinus node. *Am. J. Physiol.,* 212(4) : 911-916, 1967.
Ochsner, A.: Smoking and Health. Julian Messner, Inc., New York, 1954.
Ochsner, A., DeCamp, P.T., DeBakey, M. and Ray, C.J. : Bronchogenic carcinoma - its frequency, diagnosis and early treatment. *J.A. M.A.,* 148: 691-697, 1952.
Ox-r, J.W., Woodhouse, D.L., Hamer, D., Marchant, J. and Howell, J.S. : Cigarette tobacco tars. *Br. Emp. Cancer Campaign Ann. Rep.,* 33: 233-239, 1955.
Oski, F.A., Gottlieb, A.J., Miller, W.W. and Delivoria-Papadopoulos, M.: The effects of deoxygenation of adult and fetal hemoglobin on the synthesis of red cell 2,3-diphosphoglycerate and its in vivo

cansequences. *J. Clin. Invest.,* 49(20:400-407, 1970.
Page, I.H., Lewis, L.A. and Moinuddin, M.: Effect of cigarette smoking on serum cholesterol and lipoprotein concentrations. *J.A.M.A.,* 171(11):1500-1502, 1959.
Pamell, J.L., Anderson, D.O. and Kinnis, C.: Cigarette smoking and respiratory infections in a class of student nurses. *N. Engl. J. Med.,* 274(8):979-984, 1966.
Peacock, E.E., Jr., Greenburg, B.G. and Brawley, B.W.: The effect of snuff and tobacco on the production of oral carcinoma: An experimental and epidemiological study. *Ann. Surg.,* 151(4):542-550, 1960.
Pentecost, B. and Shillingford, J.: The acute effects of smoking on myocardial performance in patients with coronary arterial disease. *Br. Heart J.,* 26:422-429, 1964.
Peterson, D.I., Lonergan, L.J. and Hardinge, M.G.: Smoking and pulmnary function. *Arch. Environ. Health,* 16(2):215-218, 1968.
Peterson, W.F., Morose, K.N. and Kaltreider, D.F.: Smoking and Prematurity. A preliminary report based on study of 7,740 Caucasians. *Obstet. Gyneool.,* 26(6):775-779, 1965.
Pike, M.C., Gordon, R.J., Henderson, B.E., Menck, H. and SooHoo, J.: Air Pollution. In: Persons at High Risk of Cancer. An approach to cancer etiology and control (Fraumeni, J.F., Jr., ed.), pp. 225-239, Academic Press, New York, 1975.
Pozner, H. and Billimoria, J.D.: Effect of smoking on blood-clotting and lipid and piloprotein levels. *Lancet;* 1(7660):1318-1321, 1970.
Pratt, S.A., Finley, T.N., Smith, M.H. and Ladman, A.J.: A comparison of alveolar macrophages and pulmonary surfactant obtained from the lungs of human smokers and nonsmokers by endobronchial lavage. *Anat. Rec.,* 163(4):497-508, 1969.
Puri, P.S., Alamy, D. and Bing, R.J.: Effect of nicotine on contractility of the intact heart. *J. Clin. Pharmacol.,* 8(5):295-301, 1968.
Radford, E.P., Hunt, V.R. and Little, J.B.: Carcinogenicity of tobacco smoke constituents. *Science,* 165(3890):312-313, 1969.
Read, J. and Selby, T.: Tobacco smoking and ventilator-y function of the lungs. *Br. Med. J.,* 2:1104-1108, 1961.
Reddy, D.G. and Anguli, V.C.: Experimental production of cancer with beta1 nut, tobacco and slaked lime mixture. *J. Indian Med. Assoc.,* 49(7):315-318, 1967.
Regan, T.J., Hellems, H.K. and Bing, R.J.: Effect of cigarette smoking on coronary circulation and cardiac work in patients with arteriosclerotic coronary *disease. Ann. NY Acad. Sci.,* 90(1):186-189, 1960.
Rigdon, R.H.: Effect of tobacco condensate on respiratory tract of white Pekin ducks. *A.M.A. Arch. Pathol.,* 69(1):55-63, 1960.
Rimington, J.: Chronic bronchitis, smoking and social class. A study among working people in the towns of Mid and East Cheshire. *Br. J. Dis. Chest,* 63(4):193-205, 1969.
Rockey, E.E. and Speer, F.D.: The ill effects of cigarette smoking in dogs. *Int. Surg.,* 46(6):520-530, 1966.
Rockey, E.E., Speer, F.D., Thompson, S.A., Ahn, K.J. and Hirose, T.: Experimental study on effect of cigarette smoke condensate on bronchial mucosa. *J.A.M.A.,* 182(11):1094-1098, 1962.
Roe, F.J.C.: Role of 3,4-benzopyrene in carcinogenesis by tobacco smoke condensate. *Nature,* 194(4833):1089-1090, 1962.

Romsro, T. and Talesnik, J.: Influence of nicotine on the coronary circulation of the isolated heart of the cat. *J. Phamacol.,* 19(5) : 322-328, 1967.
Ross, G. and Blesa, M.I.: The effect of nicotine on the coronary circulation of dogs. *Am. Heart J.,* 79(1) :96-102, 1970.
Roth, G.M.: Tobacco and the Cardiovascular System. Charles C. Thomas Publishing Co., Springfield, Ill., 1951.
Roth, G.M. and Schick, R.M.: The effects of smoking on the peripheral circulation. *Dis. Chest,* 37(2) : 203-210, 1960.
Rothman. K.J.: Alcohol. In: Persons at High Risk of Cancer. An approach to cancer etiology and control (Fraumani, J. F., ed.) , pp. 139-150. Academic Press. New York. 1975.
Rusekk, H.I., Zohman, B.L. and Dorset, V:J.: Effects of tobacco and whiskey on the cardiovascular system. *J.A.M.A.,* 157(7):563-568, 1955.
Russell, C.S., Taylor, R. and Law, C.E.: Smoking in pregnancy, maternal blood pressure, pregnancy outcome, baby weight and growth, and other related factors. A prospective study. *Br. J. Prev. Soc. Med.,* 22(3):119-126, 1968.
Russell, M.A.H., Cole, P.V. and Brown, E.: Absorption by non-smokers of carbon monoxide from room air polluted by tobacco smoke. *Lancet.* 1:576-579, 1973.
Saffiotti, U. : Experimental respiratory tract carcinogenesis. *Prog. Exp. Tumor Res.* 11: 302-333. 1969.
Saffibtti, U. : Experimental respiratory tract carcinogenesis and its relation to inhalation exposures. In: Inhalation Carcinogenesis. Proceedings of a Biology Division, Oak Ridge National Laboratory Conference, Gatlinberg, Tennessee, October 8-11, 1969 (Harma, M.G., Jr., Nettesheim, P. and Gilbert, J.R., editors), pp. 27-54, U.S. Atomic Energy Cotission Symposium Series 18, April 1970.
Scarpelli, E.M.: The Surfactant System of the Lung. Lea and Febiger, Philadelphia, 1968.
Schwartz, D., Lellouch, J., Angeura, G., Beaumont, J.L. and Lenegre, J.: Tobacco and other factors in the etiology of eschemic heart disease in man: Results of a retrospective survey. *J. Chronic Dis.,* 19(1):35-55, 1966.
Schwartz, D., Lellouch, J., Angeura, G., Richard, J.L. and Beaumont, J.L. : Etiologie comparee de l'arteriopathis obliterante des msmbres inferieurs et de 1' arteriopathie coronarienne. (Comparative etiology of lower limg obliterative artiopathy and of coronary arteriopathy.) *Arch. Mal. Coeur,* 58(Supplemsnt 3):24-32, 1965.
Seltzer, C.C. : The effect of cigarette smoking on coronary heart *disease. Where do we* stand now? *Arch. Environ. Health,* 20(3): 418-423, 1970.
Serfontein. W.J. and Hurter. P.: Nitrosamines as environmsntal carcinogens. II. Evidence for the presence of nitrosamines in tobacco smoke condensate. *Cancer Res.,* 26(4, Part 1):575-579, 1966.
Shepherd. J.T. : Effect of ciearette smoking on blood flow through the hand. *Br. Med. J.,* 2:7007-1010, 1951.
Sogani, R.K. and Joshi, K.C.: Effect of cigarette and biri smoking and tobacco chewing on blood coagulation and fibrinolytic activity. *Indian Heart J.,* 17: 238-242, 1965.
Staszewski. J.: Smoking and cancer of the urinary bladder in males in Poland. - *Br. J. Cancer,* 20(1):32-35, 1966.
Stefanovich, V., Gore, I., Kajiyama, G. and Iwanaga, Y.: The effect of nicotine on dietary atherogenesis in rabbits. *Exp. Mol. Pathol..* 11(1):71-81, 1969.
Strong, J.P., Richards, M.L., McGill, H.C., Jr., Eggan, D.A. and

McMurray, M.T.: On the association of cigarette smoking with coronary and aortic atherosclerosis. *J. Atherosclerosis Res.,* 10(3):303-317, 1969.
Thomas, C.B. and Murphy, E.A.: Circulatory responses to smoking in healthy young men. Ann. NY Acad. Sci., 90(1):266-276, 1960.
Thomas, M.D. and Collier, T.R.: Concentration of arsenic in tobacco smoke determined by rapid titrimetric method. *J. Hyg. Toxicol.,* 27:201-206, 1945.
Trasoff, A., Abraham, Blumstein, G. and Marks, M.: Immunological aspects of tobacco in thromboangiitis obliterans and coronary artery disease. *Philadelphia J. Allergy,* 7:250-253, 1936.
Trifkovic, B.: Etude Sur les Fermentations Naturelle et Artificielle de Deux Tabacs Cultives en Suisse. These, Ceneve, 1945.
Van Buchem, F.S.P.: Serum lipids, nutrition and atherosclerotic *comp*-lications in man. *Acta Med. Scand.,* 181(4) :403-416, 1967.
Van Duuren, B.L.: Tobacco carcinogenesis. *Cancer Res.,* 28(11):2357-2362, 1968.
Van Duuzen, B.L., Sivak, A., Langseth, L., Goldschmidt, B.M. and Segal. A.: Initiators and promotors in tobacco carcinogenesis. In: Toward a Less Harmful Cigarette (Wynder, E.L. and Hoffman, D., editors), pp. 173-180, U.S. Public Health Service, National Cancer Institute Monograph No. 28, Bethesda, Md., June 1968.
Van Duuren, B.L., Sivak, A., Segal, A., Orris, L. and Langseth, L.: The tumor-promoting agents of tobacco leaf and tobacco smoke condensate. *J. Natl. Cancer Inst..* 37(4):519-526. 1966.
Van Lancker, J.L. Cancer. In: Molecular and Cellular Mechanisms in Disease, vol. 2, pp. 943-1116, Springer-Verlag, Berlin, 1976.
Van Lancker, J.L. DNA injuries, their repair, and carcinogenesis. In: Current Topics in Pathology, vol. 64 (Crundmann, E. and Kirsten, W.H.) editors), pp. 65-127, Springer-Verlag, Berlin, 1977.
Von Ahn. B.: Tobacco smoking. the electrocardioeram. and angina pectoris. *Ann. NY Acad. Sci.,* 90(1):190-198, 1966. .
Webster, W.S., Clarkson, T.B. and Lofland, H.B.: Carbon monoxide-aggravated atherosclerosis in the squirrel monkey. *Exp. Mol. Pathol.,* 13(1):36-50, 1977.
Wenzel, D.G. and Beckloff, G.L.: The effect of nicotine on experimental hypercholesterolemia in the rabbit. *J. Am. Pharm. Assoc.,* Scientific Edition 47(5):338-343, 1958.
Wenzel, D.G., Turner, J.A. and Kissil, D.: Effect of nicotine on cholesterol induced atherosclerosis in the rabbit. *Circ. Res.,* 7:256-261, 1959.
West. J.W.. Guzman. S.V. and Bellet. S.: Cardiac effects of intra-coronary arterial injections of nicotine. *Circ. Res.,* 6:389-395, 1958.
Westfall, T.C., Cippolloni, P.B. and Edmundowicz, A.C. Influence of propranolol on hemodynamic changes and plasma catecholamine levels following cigarette smoking and nicotine. *Proc. Soc. Exp. Biol Med.,* 123:174-179, 1966.
Wherat, A.F.: . Is atherosclerosis a disorder of intranlitochondrial respiration? *Ann. Intern. Med.,* 73(1) :125-127, 1970.
Wilens. S.L. and Plair. C.M.: Cigarette smoking and arteriosclerosis. *Some,* 138:975-977, 1962.
Wilhelnaen. L., Ohra. I. and Tibblin. G.: Decrease in ventilator-y capacitv between aces of SO and 54 in representative sample of. Swedish men *Br. Med. J.,* 3(5670):553-556, 1969.
Wilson, E.D., Gates, I. and Owen. H.: Street risk of carbon monoxide poisoning; *J.A.M.A.,* 37:319,- 1926.
Wilson, R.H., Meador, R.S., Jay, B.E. and Higgins, E.: The pulmonary pathologic physiology of persons who smoke cigarettes. *N. Engl. J. Med.,* 262(19):956-961, 1960.

Wynder, E.L. and Cornfield, J.: Cancer of the lung in physicians. *N. Engl. J. Med.,* 284:441-444, 1953.(a)
Wynder, E.L., Goodman, D.A. and Hoffman, D.: Ciliatoxic components in cigarette stroke. II. Carboxylic acids and aldehydes. *Cancer,* 18(4):505-509, 1965.
Wynder, E.L. and Graham, E.A.: Tobacco smoking as a possible etiologic factor in bronchogenic carcinoma - A study of 684 proved cases. *J.A.M.A.,* 143:329-336, 1950.
Wynder, E.L. and Graham, E.A.: Etiologic factors in bronchogenic carcinoma, with special reference to industrial exposures - Report of 857 proved cases. *A.M.A. Arch. Indust. Hygeine Occup. Med..* 4:221-235, 1951.
Wynder, E.L., Graham, E.A. and Croninger, A.B.: Experimental production of carcinoma with cigarette tar. *Cancer Res.,* 13:855-864, 1953.(b)
Wynder, E.L., Graham, E.A. and Croninger, A.B.: Experimental production of carcinoma with cigarette tar. II. Test with different mouse strains. *Cancer Res.,* 15:445-448, 1955.
Wynder, E.L. and Hoffman, D.: A study of tobacco carcinogenesis. X. Tumor promoting activity. *Cancer,* 24(2):289-301, 1969.
Wynder, E.L. and Hoffman, D.: Experimental tobacco carcinogenesis. *Science,* 162(3856):862-871, 1968.
Wynder, E.L., Hoffman, D., Chan, P. and Reddy, B.: Interdisciplinary and experimental approaches: Metabolic epidemiology. In: Persons at High Risk of Cancer. An approach to cancer etiology and control (Fraumeni, J.F., Jr., ed), pp. 485-501, Academic Press, New York, 1975.
Wynder, E.L., Kaiser, H.E., Goodman, D.A. and Hoffman, D.: A method for determining ciliastic components in cigarette smoke. *Cancer,* 16(9):1222-1225, 1963.
Wynder, E.L., Kopf, P. and Ziegler, H.: A study of tobacco carcinogenesis. II. Dose-response studies. *Cancer,* 10(6):1193-1200, 1957.(a)
Wynder, E.L. and Wright, G.: A study of tobacco carcinogenesis. I. The primary fractions. *Cancer,* 10(2):255-271, 1957.(b)
Young, I.M. and Pugh, L.G.C.E.: The carbon monoxide content of foetal and maternal blood. *J. Obstet. Gynecol. Br. Commonw..* 70(4):681-684, 1963.
Younozai, M.K., Kacic, A. and Haworth, J.C.: Cigarette smoking during pregnancy The effect upon the hematocrit and acid-base balance of the-newborn infant. *Can. Med. Assoc. J., 99(5)* :197-200, 1968.
Younozai, M.K., Peloso, J. and Haworth, J.C.: Fetal growth retardation in rats exposed to cigarette smoke during pregnancy. *Am. J. Obstet. Gynecol.,* 104(8):1207-1213, 1969.
Zwi, S., Goldman, H.I. and Levin, A.: Cigarette smoking and pulmonary function in healthy young adults. *Am. Rev. Respir. Dis.,* 89(1): 73-81, 1964.

ACKNOWLEDGMENTS

I am grateful for the editorial assistance of Donna Maria Roberts. Work done in this laboratory was supported by a USPHS grant, 5 ROl CA 14840-03.

AUTHOR

J. L. Van Lancker, M.D.
Department of Pathology
UCLA Center for Health Sciences
Los Angeles, California 90024

DISCUSSION OF DR. VAN LANCKER'S PAPER

Daniel H. Simmons, M.D., Ph.D., Chief, Division of Pulmonary Disease, UCLA Hospital and Clinics, led the discussion following Dr. Van Lancker's presentation. Dr. Simmons pointed out that the previous speakers, Drs. Van Lancker, Lute and Shine, emphasized lung cancer, circulatory disease and neoplasms, as causes of smoking-related mortality. However, respiratory diseases, such as bronchitis and emphysema, are also important diseases associated with smoking in terms of morbidity. They, too, involve a great amount of disability and cost. This should be kept in mind when discussing the health effects of smoking.

Cigarette smoke accounts for the major portion of inhalation irritants which cause essentially all the lung disease seen by doctors today. Autopsy studies on moderate to heavy smokers show that about 40% develop bronchitis and moderate to severe emphysema. The major defects causing bronchitis and emphysema are not clearly understood. Cigarette smoking affects the lungs' ability to clear foreign material. There is a release of macrophages, neutrophils, and proteases which over a period of time break down the inherent protein tissue of the lung and cause emphysema.

The new low-tar and nicotine cigarettes may not affect the incidence of bronchitis and emphysema although they may reduce the risk for carcinoma of the lung. They do produce more carbon monoxide, a hazard to patients with cardiovascular disease.

Based on his studies of nonsmokers today, Dr. Wynder is convinced that lung cancer is quite rare unless you smoke because epithelial cancers rarely arise in the absence of a specific source of carcinogenic irritation.

It was pointed out that although tobacco smoking is known to be a major cause of disease and premature death, the National Heart, Lung and Blood Institute has funded relatively few smoking research projects.

Data pertaining to smoking and air pollution as contributors to lung disease must be evaluated concomitant with socio-economic factors.

The American Health Foundation is monitoring cigarettes by brand name. They are evaluating the risks involved in smoking and tobacco-related diseases. Lung cancer is a specific endpoint and is fairly easy to evaluate. Coronary disease must be evaluated along with other risk factors such as hyperlipidemia and hypertension. Emphysema and bronchitis are not *easy to evaluate,* partially because pulmonary function tests are inexact.

In discussing the potential reversibility of smoking-related disease, it was noted that cessation of smoking may have immediate effect in coronary disease where supply-demand relationship is a great risk factor contributing to incidence of sudden death. Cessation of smoking *tends* to reduce the supply-demand imbalance.

There is no evidence that smoking by itself causes or reverses development of the atherosclerotic process. Epidemiologists have demonstrated, however, that when risk factors for atherosclerosis, such as hyperlipidemia and hypertension, are present, the addition of smoking substantially increases mortality.

Although emphysema is presently thought to be irreversible, progression might be prevented by cessation of smoking. There is some new information about the formation and degradation of collagen and elastin in the lungs which suggests reversibility or regeneration may be possible.

Clinical bronchitis, including lesions which predate lung cancer, is reversible as has been shown in animal studies.

Possible physiological benefits of cigarette smoking were discussed with the following points being made:

> It is likely that nicotine and smoking are tranquilizers in effect, but are very poor tranquilizers physiologically, with many bad side effects.
>
> In order to do controlled research, stress must be defined biochemically and if so defined, smoking is not a stress reducer. Smoking results in a release of epinephrine and norepinephrine, creating physiological stress rather than reducing it.
>
> Nicotine is a pharmacological agent with unique reinforcement properties. It might be beneficial for some individuals, particularly in the area of anger reduction. It produces some arousal which only amphetamine seems to mimic reasonably well. It also produces some stimulus-barrier effects with benefits of immediate availability, immediate onset and short duration.

Free nicotine might be a useful psychopharmacological agent but the cost-benefit ratio must be dealt with and the cost is not yet known. Some of the reinforcing properties of cigarettes in humans cannot be reproduced in animals. The nicotine effects are clearly only a part of reinforcement from smoking. Nicotine chewing gum is a means of delivering nicotine in a pure form. It is not reinforcing, however. In research, few people who have tried it like it, whether or not they were attempting to stop smoking.

Undesired weight gain is an additional cost-benefit problem related to cessation of cigarette smoking.

Thomas M. Vogt, M.D., M.P.H.

Discussant for Section on Consequences

Kenneth I. Shine, MD.

The costs of cigarette smoking to our society are enormous whether one measures these *costs* in mortality, morbidity or dollars. In terms of mortality, cardiovascular disease is the leading cause of death among adult Americans. Approximately 52% of all deaths in this country. occur as a result of some combination of ischemic heart disease, stroke or the complications of high blood pressure. The role of smoking has been most well documented in ischemic or coronary heart disease. Deaths from this cause can be viewed as a combination of two processes, I.e., the progressive development of atherosclerosis with lipid deposition in coronary blood vessels compromising available blood flow and the acute superimposition of an imbalance between blood availability and cardiac requirements which results in sudden death. Evidence regarding the contribution of cigarettes to the atherosclerotic process is limited. Moreover, epidemiologic data suggests that cigarette smoking alone produces only a small increase in risk of death from coronary disease in the absence of other coronary risk factors. However, the combination of cigarette smoking with other risk factors, such as high blood pressure and hyperlipidemia, produces a more than additive effect suggesting a real synergism between these risk factors. Moreover, among the various risk factors, the most abrupt decrease in mortality associated with cessation of risk has been demonstrable among patients who have stopped cigarette smoking after a myocardial infarction. In such patients, there may be some exacerbation of mortality should their cigarette smoking take place while they are immediately recovering from heart attack, but this is more than compensated by a decrease in the risk of sudden death during intervals up to 12 to 18 months after myocardial infarction. These data suggest a principal effect of cigarette smoking may be a superimposition of further imbalance between cardiac blood supply and demand on long-standing atherosclerotic narrowing of the coronary vessel.

A combination of profound morbidity and mortality is presented by the various cancers which have been associated with cigarette smoking. Cancers of the lung, bladder, mouth, larynx, among others have all been shown to be connected with cigarette smoking with or without the

synergistic effects of other agents such as alcohol.

For morbidity, chronic bronchitis and chronic pulmonary disease including pulmonary emphysema represent particularly important medical and social burdens. The reversibility of these processes upon cessation of smoking probably depends importantly upon the degree to which the process has produced irreversible structural changes. Bronchitis, particularly in its early stages, would appear to be quite reversible, whereas the degree to which pulmonary emphysema can be reversed remains unclear.

In his paper, Dr. Luce has addressed the economic costs of these illnesses in addition to those attributable to fires produced by smoking. His data suggests that direct health costs of $7.5 billion a year, representing 7.8% of total health costs, can be attributed to the smoking factor in these diseases. At least as important is the $18.2 billion which he estimates is lost annually from earnings impaired by the impact of these diseases upon wage earners. When he includes the cost of tobacco itself, the total cost of smoking in our society reaches a staggering $41.5 billion annually.

Dr. Van Lancker reviews the pathophysiology of smoking in relation to the three major causes of disease with which it is associated. Carbon monoxide accumulation in the blood, with or without a change in red blood cell affinity for oxygen, or additional accumulation of carbon monoxide from freeway driving, can clearly impair oxygen delivery to the heart. Nicotine can release catecholamines both from nerve endings and from the adrenal glands so that circulating catecholamines are increased. Some of these effects appear to be involved in an increase in serum glycerol and may contribute to changes in other blood lipids. The relative contribution of carbon monoxide, nicotine or other agents within cigarette smoke producing sudden death or contributing to atherosclerosis remains to be defined. Cigarette smoking clearly produces changes in the epithelial cells within the tracheobroncheal tree, mouth and larynx which progress to malignancy. Dr. Van Lancker outlines the multiple steps which may be involved in this process, emphasizing the likelihood that specific carcinogens are likely converted to other substances before the active carcinogenic substance produces its end result. Although an increased incidence of malignant transformation can be associated with particular substances, the details of the cellular transformations involved remain important challenges to students of carcinogenesis.

In addition to the effects of smoking upon epithelial cells, cigarette smoke produces changes in the goblet cells within the tracheobroncheal tree producing changes in mucous gland secretion and a chronic irritative state which leads to bronchitis and in many cases emphysema.

In-. Van Lancker emphasizes the importance of identifying the specific carcinogens in smoking responsible for each of these actions in the hope that removal of such agents might have protective value in those individuals who cannot give up cigarette smoking.

These presentations by emphasizing the profound economic and physical consequences of cigarette smoking, underline the validity for use of a substantial amount of resources to diminish smoking in our society. Aside from any considerations of human suffering produced by these ailments, smoking in America today simply costs too much.

BIBLIOGRAPHY

Aronow, W. S., et al. Effect of Smoking on Cardiovascular Hernodynamics. *Arch Environ Health* 28:330-332, 1974.

Cryer, P. E., Hammond, M. W. and Santiago, J. V. Norepinephrine and Epinephrine Release and Adrenergic Mediation of Smoking Associated Henkodynamic and Metabolic Events. *NEJM* 295:573-577, 1976.

Doyle, J. T., et al. The Relationship of Cigarette Smoking to Coronary Heart Disease: The Second Report of the Combined Experience of the Albany, N.Y., Framingham, Mass. Studies. *JAMA* 190:886-890, 1964.

Jenkins, C. D., Roseman, R. H. and Zyzanski, S. J. Cigarette Smoking, Its Relationship to Coronary Heart Disease and Related Risk Factors in the Western Collaborative Group Study. *Circulation* 38:1140-1155, 1968.

Kannel, G. T. and McGee, W. B. Death and Coronary Attacks in Men After Giving up Cigarette Smoking. *Lancet* 2:1345-1348, 1974.

Kannel, W. B., et al. Precursors of Sudden Coronary Death: Factors Related to the Incidence of Sudden Death. *Circulation* 51:606-613, 1975.

Vedin, J. A., et al. Sudden Death: Identification of High Risk Groups. *Am Heart J* 86:124-132, 1973.

Wald, N. J. Mortality from Lung Cancer and Coronary Heart Disease in Relation to Changes in Smoking Habits. *Lancet* No. 11, pp. 136-138, 1976.

Wilhelmsson, D., et al. Smoking and Myocardial Infarction. *Lancet* 1:415, 1975.

AUTHOR

Kenneth I. Shine, M.D.
Chief, Division of Cardiology
Associate Professor of Medicine
UCLA School of Medicine
Los Angeles, California 90024

Consequences: Session Overview

Thomas M Vogt, MD, M.P.H

It is a difficult task to disentangle the consequences of smoking on health and the economy from other social and physiological factors which also have an impact in these areas. It is possible, however, to look at the data available and arrive at a few essential conclusions about smoking. First, cigarette smoking is clearly injurious to health. Despite the unsettled questions about the relative influences of toxic substances versus pre-existing personality patterns with respect to many diseases, it is clear that smoking plays an etiologic role in lung cancer, emphysema and chronic bronchitis. It is also certain that these ailments are responsible for a considerable amount of human misery and substantial social and economic expenditures .

Second, it is equally clear that cigarette smoking has a substantial impact on the nation's economy. Whatever the actual figures, there is little doubt that vast sums of money are spent to provide medical care for persons who have illnesses caused by smoking. A great many fires are of cigarette origin. Tobacco subsidies cost real (and quantifiable) taxpayers dollars. Tobacco tax receipts certainly do not cover these costs, so it can be persuasively argued that use of tobacco produces a net social economic deficit.

Two questions arise which every one of us who is interested in the tobacco question must always keep in mind as we proceed to do our work. The first question is: Why do people continue to smoke? To many nonsmokers, the use of cigarettes seems not only senseless, but obnoxious. A corollary of this attitude is that it is easy to quit smoking, and that most smokers could simply stop smoking if they really wanted to. Dr. Temes and other behavioral scientists have done smokers and nonsmokers alike a favor by demonstrating that this simplistic view of tobacco use has little place in addressing the smoking problem. Most long-time smokers don't like smoking and would dearly love to stop. Their failure to do so constitutes testimony to the strength of the habit., Those-who have worked with drug addicts have long known that permanent withdrawal requires not one, but two solutions. The addict must go through physiologic withdrawal, a painful but relatively

short-term process. Next, the addict has to change lifestyles. It is very difficult to give up swimming when you live in the ocean. Smokers face a similar dilermna. Physical reactions to smoking withdrawal are highly varied and individualized. Still, relatively short-term cessation can remove these symptoms. The problem, however, is not short-so much as long-term smoking cessation. The problem in deconditioning smoking behavior might be better appreciated by realizing that a one pack-a-day smoker after ten years has done nothing in his life, save breathing, more often than he or she has smoked a cigarette. In a sense, smoking becomes a part of eating, a part of talking on the phone, a part of work, an inseparable portion of all those daily activities with which it is associated. Try shaving without a mirror (assuming you usually use one) and notice how awkward it feels. Do it for six months and you'll never miss the mirror. Now, imagine you shaved twenty times a day for ten years always with a mirror and then were told that it would be better for you to quit using the mirror.

A consequence of long-term smoking that is seldom discussed is the degree to which the use of tobacco becomes integrated into the daily life. It does not represent a single conditioning process, but dozens of such processes: That is why the heavy smoker finds-it so difficult to quit. The more he/she has smoked. the more difficult it is to find some safe place in life where one can be temporarily free of the craving for a cigarette because so much in life has been psychologically tied to smoking. This is not so much a behavioral manifestation of smoking as a behavioral consequence of it.

Solutions to the smoking problem must involve a thorough understanding of smoking behavior and the translation of that understanding into programsdesigned to assist cessation for current smokers and prevention for those who have not yet started to smoke. Because the economic benefits to be gained from such programs are spread across society rather than concentrated where they would be subject to exploitation by private industry, it will be necessary for the federal government to take a role not only in the research, but in the outreach programs as well.

SECTION IV: BEHAVIORAL CHANGE

Smoking: The Prevention of Onset

Ellen R. Gritz, Ph.D.

INTRODUCTION

I would like to begin this afternoon with a remark by Oscar Wilde, which, although directed at war, is relevant to the problem of smoking. He said, "As long as war is regarded as wicked, it will always have its fascination. When it is looked upon as vulgar, it will cease to be popular". (The Critic as Artist, Part II).

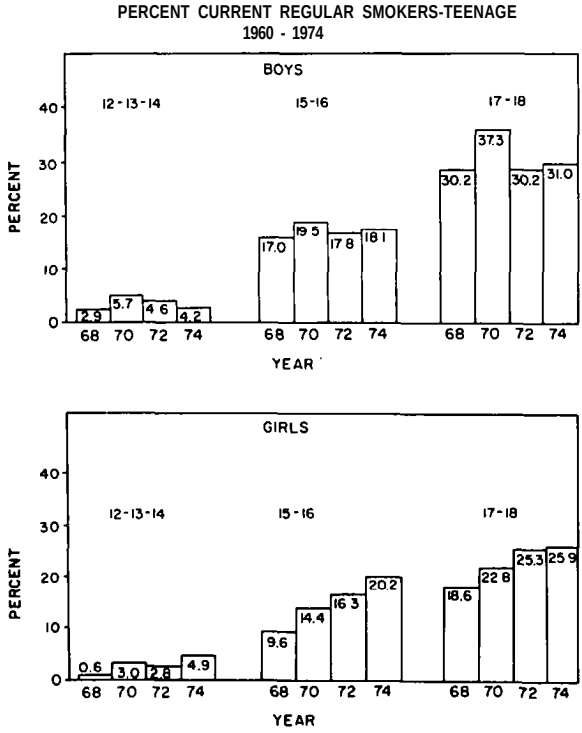
There is little need to underscore to this audience the magnitude of the problem of prevention, nor the difficulties inherent in its implementation. I will concentrate in this presentation upon four areas in which the forces of prevention can be effective: education, psychology, legislation and economic policy. The underlying principle of any approach to smoking prevention is a change in society's attitude toward cigarettes and smoking, from enjoyment, tolerance and acceptance to displeasure, disapproval and rejection. This will not be an easy battle but major changes are already occurring which provide a great deal of encouragement both in this country and abroad.

EDUCATION

With every passing year the tobacco industry refines its manufacturing techniques to produce milder and more flavorful cigarettes, and its advertising techniques to attract wider segments of the population., Before the late 19th 'century, tobacco was primarily a "man's smoke", air-cured, harsh and burning. With the advent of the flue-curing technique, the blending of tobacco became practiced as an art, and the newly invented cigarette machines were used in the genesis of a mammoth industry (Wagner, 1971; Brecher , 1972).

By now it has been well established that the main factors associated with the onset of teenage smoking are peer pressure, parental and family modeling, an image of accelerated maturity, and media messages associated with popularity, sexuality, and social fluency (see Russell, 1971; USDH.EW, 1974a). Although the production of cigarettes in the United States increased exponentially between 1910 and 1960, it was some time before advertising was aimed directly at women; it is certain that children will never be portrayed. But the evidence is undeniable -- the proportion of teenagers who regularly smoke increases eightfold between the ages of 12 and 18, from approximately 4% to 32% (Figure 1).

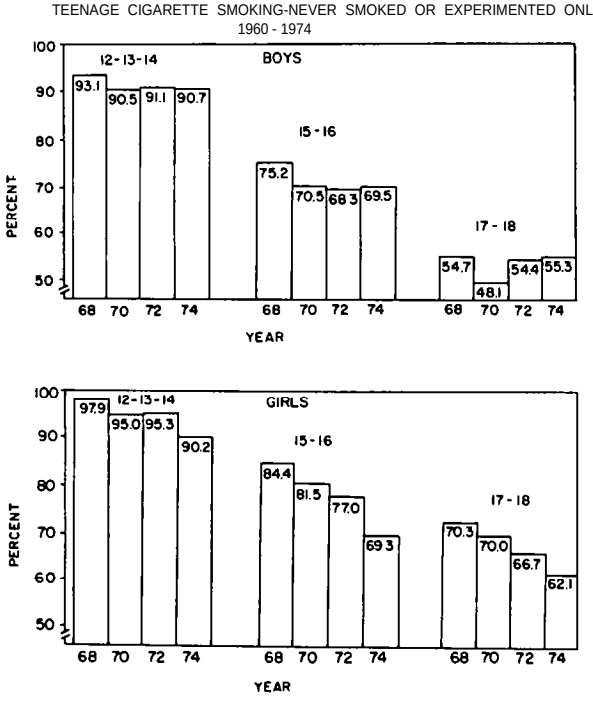
FIGURE 1



(DHEW, 1974b)

National studies of teenage smoking dramatically illustrate the rate at which teenage girls have caught up to their male counterparts (USDHEW, 1974b). Concurrent with this trend is the shrinking percentage of teenagers who remain non-smokers, both across age and time (Figure 2).

FIGURE 2



(DHEW, 1974b)

As in the previous figures, boys exhibited stable smoking patterns between 1968 and 1974, an optimistic finding. At best, the various forces at work encouraging the onset of smoking are not becoming more effective among male youth. Evidence from other nations substantiates the United States data (Bergin and Wake, 1974; Gaedeke and Gehrmann,

1973; Dull. WHO, 1975). It is clear that intervention in the early teenage years, and perhaps even sooner is the single most important project for primary prevention.

The first major effort at school intervention was begun in 1966 as part-of the program of the National Clearinghouse for Smoking and Health (USDHEW. 1976). San Diego County was select& as one of two community "laboratories" in which the effectiveness of a five year program could be tested. One of the aims of the program was to extend "cigarette control" knowledge from the junior high and high school levels, where health education classes were the primary medium, to all grade levels, with appropriately keyed programs. Unique to this program were the development of:

1) A projective coloring book to be used in the lower grades telling a story about forest fire and animals, to which children drew their own responses;

2) Peer presentations at elementary and junior high school levels, by high school student members of Kiwanis Key Clubs;

3) Smoking Sam, a child-sized mannequin used at all grade levels by specially trained young teachers to demonstrate the effects of cigarette smoke on his mason-jar lungs; and

4) Nicoteena, a small doll modeled after Smoking Sam, also visually portraying the effects of tars and other condensates of cigarette smoke in lungs.

The five-year San Diego program was most effective in the schools, (among programs also aimed at health professionals, adult community and mass media). From a survey of over-9000 junior high and senior high school students in 1967 and 1971. it was shown that the percentage of boys and girls smoking decreased, while the national averages. showed increases. Among seventh grade boys, smoking dropped from 16.9% in 1967 to 9.5% in 1971, and among tenth grade boys, from 31.8% to 19.7%. Referring to Figure 1, it can be seen that the San Diego averages for both seventh and tenth grade smoking appear inflated over the national figures; it is conceivable but not obvious that this is because the current smoker estimate in San Diego included both daily and occasional smokers, while the national figures present only regular smokers. In any circumstance, the decline in smoking is impressive, and has spurred the funding of a second intervention study in the Southern California Region under the direction of Community Cancer Control/Los Angeles.

In Los Angeles, the five-year project involves selection of six high schools and their satellite or feeder elementary and junior high schools in neighborhoods reflecting high lung disease rates (CCC/LA, 1976). The choice of high risk neighborhoods is especially important since studies have indicated that children in inner city schools (e.g., Newark, New Jersey) have inadequate health information (Louria, et al., 1976). Peer instructor teams from the high schools will conduct educational programs in grades five through nine, coordinated by a teacher-sponsor in each of the six high schools, Students will be individually followed using pre- and post-testing measures of attitude and behavior change. Accompanying the school intervention program will be a teacher-training program aimed at providing the most current knowledge in smoking and health, to be integrated into all subject matter in the curriculum, not just reserved for health education classes. This five-year program will receive a severe test in the high-risk neighborhoods selected for implementation; it is hoped that the greatest gains will occur in the areas of greatest need.

One other program deserves detailed mention here, a carefully designed study in the Houston (Texas) Independent School District, emanating from the National Heart and Blood Vessel Disease Research and Demonstration Center at the Baylor College of Medicine (Evans, 1976). Approximately 750 seventh-grade students are participating in a short-term pilot study, which will be followed by a four-year longitudinal study involving grades five through twelve. Independent variables are videotape messages, focused discussion, feedback, and behavioral monitoring. Dependent variables measured on pre- and post-tests are smoking information, attitudes, intended behavior, reported behavior and actual behavior, as measured by a nicotine-in-saliva analysis. Non-treatment control as well as "test-only" experimental groups are included in the design.

The focus of the Houston approach is upon the three immediate and major sources of pressure leading children to smoke: peer, parental and media saturation pressures. These influences are described in four short videotapes, presented on successive days. Children of the appropriate age deliver the messages and role-play typical situations. For example, in the "parental influence" videotape, children model parental smoking behavior in the face of pressure both to smoke and not to smoke. Videotape showings are followed by group reinforcement in the form of focused discussions of coping responses which could be used to

resist pressures to smoke. The feedback variable involves the status of group smoking behavior in class.

Perhaps the most interesting twist among the dependent variables is the nicotine-in-saliva determination. which although only utilized as a random check, has been found to increase the accuracy of self report, somewhat the same way as urine tests in methadone maintenance programs and breath carbon monoxide measures in smoking control programs are intended to function (Evans, Hansen and Mittelmark, personal communication). Children seem more susceptible to this behavioral lie detector than adults.

At the conclusion of the ten week pilot investigation, the rate of onset of smoking was 9.6% in the combined experimental groups compared to 18.3% in the control group, a significant difference (Evans, Rozelle, Mittelmark, Hanse, Bare and Havis, in press).

The work of Evans and his collaborators represents a major innovation in educational research in prevention. The careful design and longitudinal involvement promise to contribute much to our knowledge of the effectiveness of using an approach inmediately relevant to children, teaching coping strategies for resisting smoking.

PSYCHOLOGY

While the factors critical to the onset of smoking in children revolve around peer pressure and modeling of parental and societal behavior, the adult model for smoking behavior modification and cessation centers around a rational decision model modified by values, environmental and social factors. Most representative are the Health Belief Model of Berkanovic (1976) and the Personal Choice Health Behavior Model of Horn (1976), both of which couch the intellectual decision for health in terms of psychological utility, a cost-benefit evaluation for change, and the environmental obstacles to achieving behavior modification. Since I am primarily concerned with the prevention of onset of smoking, or in the case of exsmokers, the prevention of the re-onset of smoking, otherwise known as recidivism, I will direct my remarks most specifically to those aspects of the health choice models.

It is not surprising that there has been some confusion over a description of the characteristics of the successful ex-smoker; we are reminded of the vast literature as yet attempting to describe the personality of the smoker. In the 1964 public opinion survey, the motivation for change (the first step in the process of cessation) was comprised of four moderately independent factors: health values, an exemplar role, esthetic considerations and be-

havioral mastery (Horn, 1976). In the 1970 public opinion survey, there was no longer a clear discrimination among the various motivating factors. Rather, a single underlying dimension defined as being either "for" or "against" smoking measured the extent to which one had good reason not to change or good reason to change. The attitude of the public is shaping itself more and more into smoking and anti-smoking factions, which may facilitate the recent quitter's efforts to remain abstinent, both in the form of social support and a vocal media. The factors Horn (1976) identified as important in successful short-term quitting were subsumed under the perception of a health threat, including its importance and relevance, the capacity for behavioral change and the value thereof. These factors proved to have no predictive value for long-term abstinence.

On the other hand, a retrospective analysis of successful abstainers performed by a team of sociologists (Graham and Gibson, 1971) supported the elements of the health belief models. The comprehensive study of 996 white males in a northeastern industrial city revealed certain characteristics associated with successful ex-smokers compared to recidivists, and those who never stopped. The "successes" had superior knowledge of the health hazards of smoking, a more comprehensive set of reasons for stopping, appeared to have reported fewer lingering signs of withdrawal and craving, but greater prior respiratory problems and serious illness. Furthermore, the successes reported more non-smoking behavior in their families (never smoked or ex-smokers) than did the recidivists, who often succumbed to peer pressures to resume. Thus, the personal saliency of health-related issues combined with ease of behavioral change and the presence of a supportive social milieu to produce a successful abstainer.

On one level, long-term cessation is equivalent to never beginning smoking, by resisting the peer pressures, media messages, familial circumstances and all the societal pressures to smoke. Resting beneath this structure is a whole set of very personalized smoking messages -- at work, at play, with meals, under stress, etc. In the critical periods between the end of smoking and the firm establishment of non-smoking lie the problems posed by withdrawal symptoms (Shiffman and Jarvik, 1976; Knapp et al, 1963), the extinction of old behaviors and the initiation of new ones, often in an environment devoid of social supports.

Although this subject will be dealt with in greater depth later in this session, I believe that few individuals can make a successful transition without environmental aid. Indeed, the environment predisposes to recidivism in every move the smoker makes; the stimuli formerly cueing smoking

are ever present. For this reason, the prevention of re-onset of smoking must involve training in resisting the temptations to resume, just as the educational intervention studies are teaching coping strategies never to begin. In addition, new ways to handle stress, anxiety, the "quick" upper or relaxer (smoking's paradoxical twin effects) must be learned and reinforced in smokers. This is difficult: once having tasted of the forbidden fruit, man is loath to forego its pleasures.

On a very long-term basis, the problem of maintaining abstinence will be entirely based on the success of social reinforcers, both from external groups and from within. It is easy to imagine the pyramid effect in longer and longer term abstinence built on pride in accomplishment, change in physical behaviors, economic advantages and reorientation toward non-smoking acquaintances, until the ex-smoker comes to think of himself as a non-smoker.

Thus, the ex-smoker must traverse a long path once accepting the rational decision model to cease smoking. The "truth" or knowledge. of serious health threat may underlie all other reasoning; yet, as Ernest Becker pointed out in the Denial of Death (1973)) man's psychological structure is built on a web of denials, repressions and avoidances of the bloody, gory realizations of his mortality. Separating man from other animals is his ability to isolate his cognitions and mixed motivations from his behaviors, and to regularly engage in self-destructive acts (cf, American Health Foundation, 1976). Lower forms cannot evolutionarily afford such behaviors; one wonders, can we?

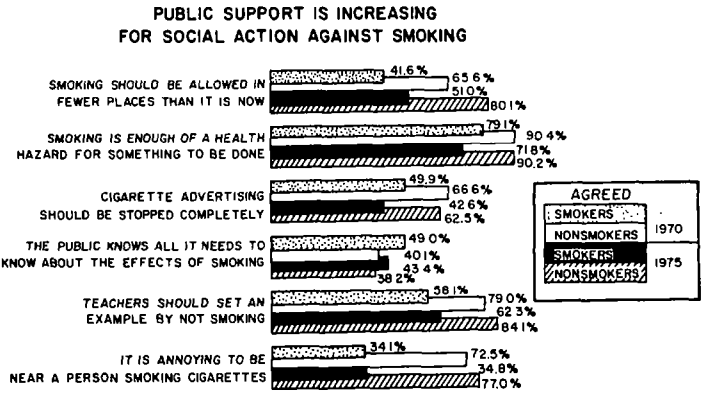
What must occur in the successful abstainer is a reshaping of his behavior "with a lot of help from his friends".

LEGISLATION

With every passing day it is becoming easier for ex-smokers to remain abstinent and for non-smokers to feel the social strength of their forces. The phenomenon of the rise of anti-smoking factions in this country is most gratifying. Non-smokers have become increasingly verbal regarding the need for preventive and protective action (Figure 3).

FIGURE 3

American Cancer Society, Inc. *Task Force on Tobacco and Cancer, Target 5,* 1976.



While it is obvious to the forces of prevention that a consistent national policy regarding smoking is desperately needed, it is equally apparent that in our democratic economy such a policy will have a long evolution.

The existence of conflicting interests and the issue of behavioral freedom pose substantial problems not unique to this country. Consistency of policy cannot always easily be achieved, even in a totalitarian state. Today, in the Soviet Union, the Ministry of Health is attempting to ban smoking in some public places, on internal Aeroflot flights shorter than five hours, and in railroad dining cars, while the Ministry of Light Industry calls for a 16% increase in cigarette output in the current five year plan, and the Ministry of Agriculture for a 50% increase in domestic tobacco production (Los Angeles Times, 5/22/77). At the same time, in the Black Sea resort of Sochi, Russia's first non-smoking city, regulation and enforcement of the ban have slackened considerably in the face of flagrant violation (The London Sunday Telegraph, 3/6/77). From our experience between 1919 and 1933 we may have learned that prohibition is ineffective, but the U.S.S.R. has yet to assimilate that message.

Within our own country, I believe the best stance for smoking prevention is to push strongly for the reduction in the visibility of smoking, by a substantial curtailment of, or total ban on advertising, severe limitation of smoking in public places, and vigorous anti-smoking campaigns publicizing both the dangers of smoking and modes of resisting. We must be constantly working within the legislative process and with those representatives on the national, state and local levels who are actively proposing bills.

Public health legislation, and particularly that involving prevention, winds a slow and tortuous path through the Congress, because the policy areas involved are fragmented and must be considered by a number of different communities (Bauman, in press). In the Senate alone, there are 19 or more committees with jurisdiction over various health-related areas; and legislation outside of a committee or subcommittee's jurisdiction is not likely to be considered. Such a problem was encountered when Senator Kennedy, as Chairman of the Health Subcommittee of Labor and Public Welfare, held hearings on a bill to impose a graduated excise tax on cigarettes according to tar and nicotine content. The committees of Taxation and Agriculture hold dominion over taxes and tobacco.-growing, respectively, and of course, tobacco-state senators testified against the bill.

The position of those working within the Congressional system, such as Ms. Bauman, is to encourage health professionals to form a strong lobby for preventive legislation which could provide input on a variety of bills, and information and communication within the committee system. Included in such a constituency might also be educators, consumer advocates, recreation specialists, those involved in geriatrics in the medical and public domain, and insurance companies. Radical change is not likely to occur in our legislative system, but we must be vocal about initiating it.

I would like to summarize briefly a sample of current proposed and enacted legislation on the national level and within the state of California.

On May 26, 1977, the Food and Drug Administration was petitioned on behalf of former Surgeon Generals Luther Terry and Jesse Steinfeld and numerous other health and anti-smoking organizations led by Action on Smoking and Health (ASH) to regulate the sale of cigarettes as strictly as that of saccharin (Banzhaf, 1977). By considering cigarettes a “device”, they come under the broadest jurisdiction of the F.D.A.; by classifying

nicotine as a drug, cigarettes are regulable under the narrowest interpretation of the F.D.A.'s authority. The petition requests the classification of cigarettes as a prescription drug, to be sold only in pharmacies. In this way the public would be continuously reminded that the sales restriction is connected with the chemical properties of cigarettes, and sales would decrease, especially those to children.

Legislation currently in effect which has had far-reaching impact on encouraging non-smokers to assert their rights include the Interstate Commerce Commission and Civil Aeronautics Board restrictions on seating smokers. Unfortunately, the public remains largely apathetic in supporting these rulings, and the ICC has relaxed its seating restrictions on buses because of the bus company demands. Apparently, the CAB has been considering a total ban on smoking but has received little support in public hearings (Somers, 1977). Eastern Airlines has just agreed to guarantee a no-smoking seat to every passenger, and to reduce the smoking section to 35% of seating, according to the proportion of smokers in the adult population. The agreement was made as the result of a complaint issued by ASH and the Aviation Consumer Action Project; specifically, Eastern was fined $10,000 for denying non-smoking seats on 14 separate occasions (Los Angeles Times, 6/15/77). Consumer actions such as these in support of existing legislation provide another example of an opportunity to build strong preventive constituencies.

On the state and local fronts, proposals banning or severely limiting smoking in establishments open to the public, even if privately owned, are being vigorously introduced. In California, SB 500, introduced by Senator Arlen Gregorio, would ban smoking in a wide variety of facilities open to the public: libraries. halls. auditoriums and courtrooms;' retail establishments; -theaters, movies and sports arenas'; hotels and motels; places of employment ; and all health facilities. Excepted are private rooms in hotels, motels and hospitals, bars, restaurants and certain specified smoking areas in lobbies, lounges, etc. This bill has now passed the California State Senate and has been sent to the Assembly. It is scheduled to take effect on July 1, 1978, if passed (Gregorio, 1977a; Los Angeles Times, 6/17/77).

Within the city of Los Angeles effective ordinances prohibit smoking in elevators, certain areas of retail food marketing establishments, public places of entertainment, businesses and health care facilities except in designated smoking permitted areas. Councilman Marvin Braude, a major anti-smoking advocate, is presently attempting to

have non-smoking areas set aside in restaurants.

This type of legislation is typical of anti-smoking activity throughout the country and represents a major grass roots movement. It is unfortunate that it is not always noticeable at the national level, but as previously mentioned, the standoffs reflect conflicting interest groups from diverse geographic areas and orientations.

ECONOMIC POLICY

I have so far spoken of anti-smoking legislation which is not tied to any specific economic measure. Binding preventive action to economic advantage or disadvantage will most probably accomplish more than coercion. This point has been made tellingly in a paper presented by Dr. Marvin Schneidennan of the National Cancer Institute at the meeting of the American Society of Preventive Oncology (Schneidennan, 1977). He sets forth a number of challenging proposals, just a few of which I wish to discuss, because they represent economically sound examples of the most generalized thinking in this area.

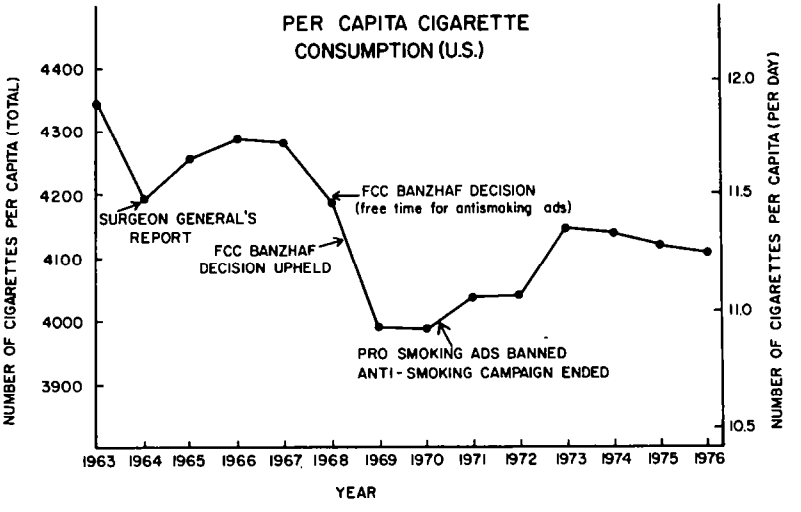
In the first place, we could move towards ending or reducing tobacco subsidies and removing land currently used for tobacco cultivation from that market either by creating a soil bank, as Dr. Schneiderman advocates, which would pay the fanner an equivalent income for not growing tobacco, or by alternate means of providing economic incentives to grow other crops. The price of tobacco would thus be driven up, with consequences of reduced sales and a substantial loss of excise tax revenue, estimated currently at $6 billion a year. However, poor health lowers the wage earning potential of the country, hence lowers the receipts. The increase in productivity and spending of a healthy populace might more than offset a loss in excise tax revenue.

A second major proposal regards cigarette advertising regulation. The tobacco industry currently spends $360 million a year on advertising, approximately half of which is used to promote the 30-odd low tar brands. Cigarette advertising comprises a major source of revenue for magazines: OUI, PLAYBOY and SPORTS derived 38%, 21% and 23% of their revenue from smoking ads and the widely read weeklies TV GUIDE, TIME, NEWSWEEK and SPORTS ILLUSTRATED approximately 15% (Tobacco Reporter, 1977a) . In the next six months alone, $40 million will be spent by one corporation to introduce its latest low-tar and nicotine “naturally flavored cigarette” (New York Times, 5/15/77).

Banning cigarette company promotion of major sports events, a devious association of athletic prowess and health with smoking and even a ban on all cigarette advertising is frequently suggested. It is quite obvious that the latter would substantially deter promotion of new cigarette brands and probably force the corporations to alter marketing tactics. As part of a major anti-smoking drive the British government, with agreement of the tobacco industry, has called for an immediate end to advertising cigarettes in the high tar group and middle to high tar group by the end of 1978 (Tobacco Reporter, 1977b). In Holland, the government is planning to prohibit all tobacco advertising in newspapers and magazines and in Finland a ban on printed cigarette and alcohol advertising will be in force on March 1, 1978 (Tobacco Reporter, 1977c, .d). Strong actions such as these should encourage us with equally forceful measures.

A remarkable lesson was learned from the televised anti-smoking campaigns of 1967-1970. As Figure 4 shows, per

FIGURE 4
Effect of Anti-Smoking Television Campaigns on Per Capita Smoking (U.S.) (USDA, 1977)



capita intake of cigarettes fell noticeably during that time, but increased again with cessation of all television advertising (USDA, 1977). There is a bill in the California State Legislature (SB 189) at this moment proposing to appropriate $2 million from the General Fund for reinstatement of televised anti-smoking ads. The two-year pilot program would evaluate effects on per capita smoking in major population areas in California (Gregorio, 1977b).

It has been suggested that cigarette advertising be taxed, with the proceeds used to contract for commercially developed anti-smoking advertising, with segments aimed specially at women, children and minority groups (Schneiderman, 1977).

Moving into proposals on direct taxation of cigarettes, suggestions have included imposing a nationwide uniform tax-to prevent the bootlegging now occurring in states with high excise taxes, such as New York, graduating taxes according to tar and nicotine content, and relating the amount of the tax to the number of cigarette-related deaths annually (Schneiderman, 1977; Somers, 1977).

Any taxes on advertising and all direct cigarette taxes will raise the price of cigarettes sharply. Reducing the supply of tobacco by taking land out of production will have the same ultimate effect. The end product of all such suggestions are regressive measures which would most directly affect the young and the poor. It is very true that prevention must begin with youth but the effects of such measures on the middle and upper class smoker may not be as great as we hope. Economic measures alone are insufficient smoking deterrents.

Combining the four areas of impact I have discussed today -- education, psychology, legislation and economics -- a variety of comprehensive prevention programs are feasible and very exciting. It only remains for us to continue to build strong constituencies committed to prevention, and to work hand in hand with our legislators to develop vital and workable proposals.

REFERENCES

American Health Foundation. Sympositan: The Illusion of Immortality. *Preventive Medicine,* 5:477-548, 1976.

Banzhaf J.F., III. Citizen Petition. Hearing Clerk, Food and Drug Administration, *Department of Health, Education and Welfare.* May 26, 1977.

Bauman P. and Banta H.D. The Congress and policymaking for prevention. *Preventive Medicine,* in press.

Becker E. *The Denial of Death.* New York: Free Press. 1973.

Bergin J. and Wake E.R. Report to the Department of National Health and Welfare on Canadian research on psycho-social aspects of cigarette smoking (1960-1972), *Canada Department of National Health and Welfare,* 71 pp. 1974.

Berkanovic E. Behavioral science and prevention. *Preventive Medicine,* 5:92-105, 1976.

Brecher E.M. *Licit and Illioit Drugs.* Boston: Little Brown & Co., 1972.

Bulletin of the World Health Organization. Epidemiology of chronic non-specific respiratory diseases. *Bulletin* of *the World Health Organization,* 52:251-260, 1975.

Community Cancer Control/Los Angeles. *A proposal to implement a community based cancer control program in Los Angeles, California,* 108-110, 1976.

Evans R.I. Smoking in children: developing a social psychological strategy of deterrence. *Preventive Medicine,* 2:122-127, 1976.

Evans R.I, Hansen W.B. and Mittelmark M.B.. Increasing the validity of self-reports on behavior in a smoking in children investigation. *Personal communication.*

Evans R.I., Rozelle R.M., Mittelmark M.B., Hansen W.B., Bane A.L. and Havis J. Deterring the onset of smoking in children: knowledge of immediate physiological effects and coping with peer pressure, media pressure, and parent modeling. *Journal of Applied Social Psychology,* in press.

Gaedeke R. and Gehrmann J. (Drug addiction among children and adolescents with particular consideration of habitual sniffing. III. Nicotine. *Klinische Padiatrie (Gr. Lung).* 69:10-16, 1973.

Graham S. and Gibson R.W. Cessation of patterned behavior: withdrawal from smoking. *Social Science and Medicine,* 5:319-337, 1971.

Gregorio A. *Staff analysis of Senate Bill No. 500,* Senate Committee on Health and Welfare, California Legislature, 1977a.

Gregorio A. *Senate Bill No. 189,* Senate Committee on Health and Welfare, California Legislature, 1977b.

Horn D. A model for the study of personal choice health behaviour. *International Journal of Health Education,* 19:89-98, 1976.

Knapp, P., Bliss C.M. and Wells H. Addictive aspects in heavy cigarette smoking. *American Journal of Psychiatry,* 119:966-972, 1963.

London Sunday Telegraph. Better deal for the non-smoker. London, England, March 6, 1977, p. 10.

Los Angeles Times. Russ belatedly start anti-smoking drive. May 22, 1977, Part IV, p. 1.

Los Angeles Tines. Smokers can't sit in 65% of seats. June 15, 1977, Part I, p. 4.

Los Angeles Times. Bill would prohibit smoking in most buildings open to public. June 17, 1977, Part I, p. 17.

Louria D.B., Kidwell A.P., Lavenhar M.A., Thind I.S. and Najem R.G. Primary and secondary prevention among adults: an analysis with comments on screening and health education. *Preventive Medicine,* 5:549-572, 1976.

New York Tines. $40 million for a real smoke. May 15, 1977. Section III, pp. 1, 4, 5.

Russell, M.A.H. Cigarette smoking: natural history of a dependence disorder. *British Journal* of *Medical Psychology,* 44:1-16, 1971.

Schneiderman, M.A. Legislative possibilities to reduce the impact of cancer. *Paper presented to the American Society of Preventive Oncology, First Annual Meeting,* Memorial Sloan Kettering Cancer Center, New York City, February 4-5, 1977.

Shiffman, S.M. and Jarvik, M.E. Smoking withdrawal in two weeks of abstinence. *Psychophartmcology,* 50: 35-39, 1976.

Somers, A.R. Life, liberty and the pursuit of happiness -- and the right to die of lung cancer. *Paper presented to the American Society of Preventive Oncology, First Annual Meeting,* Memorial Sloan Kettering Cancer Center, New York City, February 5, 1977, 1-17.

Tobacco Reporter. U.S. Print ads. 104(5):59-69, 1977a.

Tobacco Reporter. British government declares war on smoking . 104(4):14-18, 1977b.

Tobacco Reporter. Anti-smoke push ready. 104151:61, 1977c.

Tobacco Reporter. Print ban by March 1, 1978. 10415):63, 1977d.

United States Department of Agriculture, Economic Research Service. Consumption per capita of tobacco products in the United States. *Tobacco Situation, 159,* 40 pp., March 1977.

United States Department of Health, Education and Welfare. Public Health Service. Teenage smoking: national patterns of cigarette smoking, ages 12 through 18, in 1972 and 1974. *DHEW Publication No. (NIH)* 76-933, 1974a.

United States Department of Health, Education and Welfare. Center for Disease Control, National Clearinghouse for Smoking and Health. Patterns and prevalence of teen-age cigarette smoking: 1968, 1970, 1972 and 1974. *DHEW Publication No. (CDC) 75-8701,* 1974b.

United States Department of Health, Education and Welfare. Center for Disease Control, National Clearinghouse for Smoking and Health. San Diego smoking research project: five year status. Health Education Project. *DHEW Project No. 200-75-0516:1-7,* Revised 1976.

Wagner, S. *Cigarette Country.* New York: Praeger. 1971.

Wilde, O. The critic as artist, Part II. In: E.M. Beck (Ed), *John Bartlett's Familiar Quotations,* 14th Ed. 839a, Boston: Little Brown E Co., 1968.

AUTHOR

Ellen R. Gritz, Ph.D.
Veterans Administration Hospital Brentwood
Wilshire and Sawtelle Boulevards
Los Angeles, California 90073

Department of Psychiatry
Neuropsychiatric Institute
University of California
760 Westwood Plaza
Los Angeles, California 90024

Smoking Cures: Ways to Kick an Unhealthy Habit

Jerome L. Schwartz, Ph.D.

INTRODUCTION

Michael M. Davis, a social scientist who devoted his career to the field of medical care, stated the following in 1963:

> The striking fact of this Twentieth Century is change, the amount and pace of change. Population has been growing at a rate never before known. In the natural, the biological and the social sciences, the mass of knowledge has expanded in the past 60 years more than in all the previous millennia of human history. Technology--the ways men produce and distribute goods, services and ideas--has moved on the heels of science in dizzying interplay. In the more developed countries the preponderant rural life of the past has given place to urbanism. The political face of the world has been transformed since the First World War (Davis 1963).

This dramatic, recent expansion describes also the growth of the relationship between tobacco and health as it was in this century, when cigarettes became readily available. During World War I the tobacco companies gained mass recruits to smoking. In 1900, the number of cigarettes consumed per adult was 49. By 1920 it was 611, and consumption rose rapidly thereafter from 1,828 in 1940 to 3,888 in 1960 (Gori 1976). The incidence of lung cancer began increasing at the start of this century, and by 1930 lung cancer deaths reached 3,000 per year. It wasn't until 25 years later that organized approaches offering assistance to smokers trying to quit were initiated.

In 1955 public stop-smoking clinics first began in Stockholm, Sweden. These clinics used medications, lectures, pamphlets, and physician counseling in ten-day sessions to help people cure their smoking habit (Schwartz 1969). In the early 1960's, clinics spread to Denmark, Norway, Czechoslovakia, England, Ireland, Scotland,

Germany, Canada, and the United States. During the past several years, some type of cessation program has operated in over two dozen countries, sponsored by voluntary organizations, public health authorities, temperance societies, hospitals, research institutes, commercial organizations, religious groups, schools, health professionals, and lay people. This paper will review the various methods used to help smokers kick their habit.

My purpose here is not to go into a detailed literature review of smoking cessation methods, as I have already done that in my 1969 Evaluation (Schwartz 1969)) my summary paper with Gail Rider at the 1976 Third World Conference on Smoking and Health (Schwartz and Rider 1977), and our forthcoming monograph to be published by the National Clearinghouse on Smoking and Health (Schwartz and Rider 1978); reviews of behavior modification methods have been published by Bernstein and McAlister (1976) and Lichtenstein and Danaher (1977).

In this paper, I will begin by describing briefly the various types of smoking cures, generally called quitting methods. Next, I will review cessation methods used in Europe, Canada, and the United States. I will indicate the range of success achieved by some of the leading investigators, note what we have learned from the control movement, and offer some concluding comments.

SELF-CARE

There are essentially two ways to stop smoking: self-care techniques and organized programs. Self-care consists of three modes: devising one's own way of quitting; utilizing a tool or guide to quitting such as an instructional manual, book, record, cassette, filters or some type of gimmick; or receiving advice on how to quit and then doing it. Self-care methods have enabled the most people to quit smoking. Unfortunately, there are no long-term evaluations of self-care methods.

The Self-Testing Kit and Teenage Self-Test were developed by Dan Horn and the National Clearinghouse for Smoking and Health to help the smoker: gain insight into how s/he feels about smoking, learn the reasons for stopping, perceive the health threat, understand how s/he uses cigarettes and identify factors that inhibit or help quitting. Several million persons have used the kit, but its only evaluation was in connection with a television program. Several voluntary associations have developed self-kits for quitting and the American Cancer Society is packaging a self-quit-kit which will be available for mass distribution within six months.

Various filters are sold as stop-smoking devices. The idea is that filters reduce tar-nicotine levels permitting the smoker to be weaned away from cigarettes. Water Pik ("One Step at a Time") sells four reuseable filters for $10; each filter is used for two weeks. The Venturi System, designed as a four-week method, provides four reuseable filters for $4. The New-Life Stop Smoking Kit, costing $10, provides 44 disposable filters, one to be used each day. There are no available evaluations of the success of filters, but when one quits it is likely that self-care has played a large part in the quitting effort.

The best self-care methods are those devised by the individual smoker. Sometimes an aid is used. Often an illness triggers the quitting attempt. Many people try several times before they succeed. Self-devised methods have contributed to the 13.5 percent reduction in smoking among adult males (1964 to 1975).

ORGANIZED CESSATION METHODS

Most smokers agree with the scientific evidence that smoking is harmful and wish to give up cigarettes, but many cannot do so on their own. A national survey reported that six of ten smokers have seriously attempted to stop; another three of them say they would try to stop if there were an easy way (Bureau of Health Education 1976). Most smokers need help to break the cigarette habit. Over-the-counter remedies are available but many smokers need the encouragement of an organized method or long-term support to sustain their quitting effort.

Over one million Americans are estimated to have quit smoking during the last seven years by using an organized method. Undoubtedly, many more could have been helped to quit if publicity for smoking cessation methods were not so overshadowed by the massive campaign through the printed media that influence, support, and maintain smoking behavior.

For ease of comparison, I will classify organized smoking cessation methods into ten categories: individual counseling, educational programs, groups, medication, hypnosis, aversive conditioning, self-control, mass media, community approaches, and miscellaneous.

During the 1960's, the most popular method of curing the smoking habit was medication. Group methods and the Five-Day Plan were next in importance. In the last seven years, withdrawal clinics utilizing educational and group approaches have become the leading cure. The literature, however, includes numerous reports from behavioral investigators in the United States, who are seeking a more successful cure for smoking.

1. Individual Counseling. In the last six years, many health professionals have devoted time to counseling smokers to give up the habit. Some take part in organized programs, but many practitioners work directly with their patients. Most advice takes the form of information on the harmful effects of smoking and on the benefits of quitting. Pamphlets are generally distributed along with information on how to quit smoking. Physicians often urge patients to quit when they identify respiratory deficiencies or chronic illnesses. The practitioners explain how smoking directly affects the patient's health and emphasize the. long-term benefits of smoking cessation. Some physicians issue a strong warning that the patient must quit smoking to preserve his life. More and more physicians are taking time (or assigning someone in' their office to do so) to advise their patients on how to quit. Follow-up support is offered by keeping in touch with the patient.

2. Educational Programs. Most countries conduct anti-smking educational campaigns in schools. The public is informed about the harmful effects-of-cigarettes through scientific reports, pamphlets, posters, and films produced by heart, cancer, and lung associations or by the government. Newspapers, radio, and television also publicize research findings on lung cancer, heart disease, emphysema, and other diseases.

Smoking cures through educational methods are of two types: those conducted in schools as formal classes, and groups utilizing an educational or lecture approach. The Five-Day Plan, the most popular type of withdrawal clinic, is bringing cessation programs to countries in many parts of the globe. The program, which is mostly lectures, meets for five consecutive days and then for several follow-up meetings held at monthly intervals. The Five-Day Plan advocates a regimen of physical fitness, balanced diet, forced fluids, hot and cold showers, and abstention from coffee, tea, cola drinks, and alcohol. Medical, spiritual, and educational lectures, combined with films and displays of smoke-damaged lung specimens, round out the program. There are many copiers of the Five-Day Plan, most of which combine lectures with techniques aimed at scaring smokers into quitting.

An example of smoking cures through school classes are the programs in New Jersey. The local school district sponsors a series of adult classes in several towns.

3. Groups. The various group control activities are mostly withdrawal clinics sponsored by voluntary associations, health departments, foundations, or commercial organizations. Voluntary associations, such as the American Cancer Society (ACS) or the Lung Association, have produced manuals to guide the volunteer ex-smokers who lead the group. ACS has recently produced a series of short films to trigger discussion. Although different in content, they are similar to the trigger films produced for the National Heart and Blood Institute's Multiple Risk Factor Intervention Trial project. Among the most active group programs are those of the American Cancer Society,.. Lung Association. the American Health Foundation in New York City, the Kaiser Foundation Health Plan in Oakland, and SmokEnders, a commercial agency. The Multiple Risk Factor Intervention Trial project, being run in twenty cities as a risk intervention trial, also uses group methods.

Group programs last from four to twelve weeks. Most of the current programs also offer follow-up maintenance sessions. Keeping records and studying films and materials supplement the group discussions. Some groups start with rapid smoking or behavior modification techniques.

4. Medication. Two general categories of pharmaceutical agents are used to help people quit smoking: agents developed specifically to help smokers overcome the tobacco habit (substitutes and deterrents), and drugs prescribed to help persons trying to quit to overcome withdrawal problems. Lobeline, the most common smoking substitute, takes the place of nicotine. The lobeline base products, such as

Bantron, Nikoban, Lobidan, Tabusine, and lobeline hydrochloride, act as a stimulant on the respiratory system; Tabex, used in Bulgaria, Poland, and Germany, is another nicotine substitute. Swedish investigators are developing a nicotine chewing gum which is being tested in Sweden, England, and at UCLA.

Smoking deterrents are mainly prepared from silver nitrate or potassium permanganate. These preparations irritate the nasal mucosa . Some products in this category are Nosmoke, silver lactate, Omozone, Niperlen, Pastaba, Skopyl, Egazil, Nice-exsin.

Vegetable base products, such as Libbs, Nicocortyl, avena sativa, and Tabazero, have also been used to help people stop smoking. The action of these products is not clear.

5. Hypnosis. Hypnotists vary their approaches but the most common methods are individual sessions of hypnosis, group hypnosis, and self-hypnosis as developed by Herbert Spiegel, M.D. of New York City. Hypnosis is often used along with group therapy or medication.

6. Aversive Conditioning. Psychologists are the leading, but not the only, proponents of aversive conditioning and other behavior modification techniques as cures for the smoking habit. Aversive agents or techniques include electric shock, an unpleasant taste, breath holding, smoke, or imagined stimuli. An electric shock may be delivered to the smoker when the cigarette is puffed or inhaled. Sometimes warm, stale air is the contingent punishment, alternated with clean, mentholated air when the subject is not puffing the cigarette. Blowing smoke at the smoker until the subject is satiated is another aversive technique. The patient may also be asked to chain smoke until s/he can no longer tolerate smoking.

Rapid smoking has been perfected as a cessation technique over the last few years; with this method, the subject is asked to puff the cigarette every six seconds until s/he becomes dizzy or nauseated.

Aversive conditioning can also be imagined stimuli. The subject thinks about the devastating physical effects of smoking or imagines that smoking is causing illness or other unpleasant situations associated with smoking.

7. Self-Control procedures have been used to alter some of the antecedents or consequences of smoking. Cues, pocket timers, metronomes, signal devices, record keeping, and self-monitoring are some of the variations of this approach. Signal devices interfere with the normal smoking response by breaking the connection between environmental cues and the smoking. The subject smokes on a new cue, presented at random times by a portable signaling device. The substitute cue is initially set at the smoker's normal rate and then gradually phased out.

8. Mass Media. Television and radio programs have broadcast withdrawal programs in this country and in several European countries Advance publicity asks listeners to request accompanying kits of material and record cards. The format usually includes the facts about the risks of smoking and ways of curing the habit.' Sometimes

local announcers quit along with the listeners, or sports or public figures present testimonials.

9. Community Approaches involve an entire city or area in a saturation educational campaign. In the Stanford program, three northern California communities participated in a study designed to assess the impact of three methods of public health education and training on the prevention of coronary heart disease. In one community, a random sample of the population received a preventive screening examination. In the second community, a random sample received screening plus an extensive mass media campaign urging individuals to change their smoking and other health related habits. In the third community a random sample received screening and the mass media campaign, and those in the sample identified as having risk factors were offered personal instruction on how to stop smoking. Personal contact and instruction helped 36 percent of the participants to quit, and among high risk men smoking declined 50 percent (McAlister et al. Undated).

10. Miscellaneous Methods. In addition to the principal methods of smoking control, techniques that are either of limited use or of more recent introduction include fear-arousing warnings, role playing, discussion, relaxation, yoga, meditation, acupuncture, telephone messages, and mailings.

ACTIVITY BY COUNTRY

Sweden is still the leader in cessation activity. Public clinics are operated in Stockholm, Goteborg, and Lund. Four treatment methods are presently available in Stockholm: individual support sessions combined with the administration of Skopyl and Egazil, medications which create dryness of the mouth and aversive taste; electric shock; group therapy with hypnosis; and individual hypnosis. Over 5,000 smokers have been treated at the Stockholm Smoking Withdrawal Polyclinic. The Goteborg Withdrawal Clinic treatment offers daily sessions with a physician and drug therapy over a two-week period.

The first multiple risk factor trial was initiated in Sweden. Thirty thousand people were divided equally into an intervention group and two control groups. The intervention groups were treated for smoking, hypercholesterolemia and high blood pressure. Treatment for smoking consisted of health education, positive suggestion, and group meetings.

Sweden also pioneered a nicotine chewing gum (Nicorette), which was developed by the Aktiebolaget Leo Research Laboratories, Helsingborg. In Bulgaria, pharmacists developed Tabex tablets from cytisin to combat smoking; their use is reported in East Germany, Poland and Bulgaria.

Norway's numerous active anti-smoking programs are coordinated by the National Council on Smoking and Health. The Council provides films, broadcasts, and educational government personnel who include field workers to carry out anti-smoking activities. The Self-Testing Kit

developed by the United States National Clearinghouse for Smoking and Health has been translated into Norwegian and distributed widely. The Norwegian Temperance Society-has run clinic programs in twenty communities. A risk factor study is underway in Oslo, and a coronary heart disease prevention program is being conducted in Finnmark, Norway. Infarction and chest clinics also offer smoking cessation assistance. In 1971 and again in 1974, a series of programs on stopping smoking were broadcast on national television.

Withdrawal clinics have been available in Copenhagen since 1958. Initially they used lobeline, restinil, silver acetate, and auto-suggestion exercises, but lately they have tried conversational therapy, hypnosis, breathing and relaxation exercises, and psycho-sedatives that create an unpleasant after-taste to tobacco.

In Finland, clinics have been operated in Helsinki, Turku, and Kuopio . North Karelia County is the intervention community for a multiple risk factor trial. The Five-Bay Plan is in use.

In England and Scotland various cessation programs are supervised by physicians or health educators. Group therapy, health education,. and counseling are the principle methods, although some clinics use hypnotherapy,-aversion,-and chemotherapy. Numerous private physicians in the British Isles report they counsel their patients to quit smoking, and several use psychotherapy, hypnosis, group therapy, and medication in their efforts to help smokers break their habit.

At London's Maudsley Institute of Psychiatry, Russell and his colleagues have tested a number of methods, including electric shock, rapid smoking, satiation, therapeutic support, and medication.

Myocardial infarction and chest clinic programs are available in many Commonwealth cities, including London, Edinburg, and Glascow. The Midspan Health Plan conducted at the University of Glascow assisted- smokers to stop smoking. The multiple risk factor project for London civil servants screened 18.000 peopole for risk of cardio-vascular disease and found that 1,400-of those at risk were identified as cigarette smokers. The project includes an intervention program consisting of advice, counseling and recalls.

The Federal Republic of Germany is the scene of a great deal of smoking withdrawal activity. The Max Planck Institute for Psychiatry, Munich, utilizes a wide variety of approaches: covert sensitization, negative exercise, shock, lobeline, placebos, non-directive psychotherapy, self-control, and treatment by correspondence. Brengelmann has written a manual on smoking cessation therapy (Brengelmann 1975).

The Five-Day Plan has been very active in Germany. Several investigators have tried medication. A cardiology clinic at BadenBaden uses tranquilizers, sedatives, and lobeline sulfate for myocardial infarction patients.

The German Democratic Republic's cessation activities include clinics conducted at factories. Bulgarian Tabex has been used regularly at

clinics along with groups, the Five-Day Plan, and individual counseling.

In Czechoslovakia, a clinic program began in 1959 in Hradic Kralove. Many cities and towns have free cessation clinics. A central training center in Prague instructs clinic workers how to present withdrawal techniques. Psychotherapy and lobeline chewing gum are also available as part of the program. A spa in Pisku treats smokers with psychotherapy along with lobeline inhalation. Myocardial infarction patients are being rehabilitated at the Derer Hospital in Gratislava-Kramare.

Poland is participating in a multirisk factor trial organized by the World Health Organization, as are Belgium, Italy and the United Kingdom. The Polish trial is being conducted among factory workers judged to be at high risk for coronary heart disease. Smoking clinics are available in Warsaw and Poznan, as well as in some factories. A Hungarian cigarette-holder ("superflit") aimed at reducing smoking is used as well as Tabex, lobeline, psychiatric treatment groups and psychodranm.

A multifactor preventive heart disease project in Rome developed out of a coronary heart disease clinic for high risk middle-aged men. Individual advice on how to quit smoking is provided by physicians in repeated sessions. An organized program for persons identified with early pulmonary emphysema is being conducted.

Switzerland also has a multifactor preventive project. Several Swiss clinicians have used medication in their withdrawal work.

Austrian withdrawal clinics started in 1973. A cessation program was conducted by telephone in 1974, and a nationwide cessation program was broadcast on television in 1975.

In France and Belgium clinic methods consist of the Five-Day Plan. In several French cities individual counseling is available.

The University of Toronto and that city's health department provided lectures and group discussion when they operated a smoking program for several years. Now the Metropolitan Toronto Interagency Council directs a clinic program. Besides the Five-Day Plan, several commercial programs are available in Canada. The Tuberculosis and Respiratory Association has an active program in Vancouver, British Columbia. Shock, desensitization, satiation, and other behavior therapy methods are used by university researchers. Hypnosis and counseling are offered by several practitioners and a small scale risk factor project is being sponsored by the Manitoba Heart Association in Winnepeg.

All types of cessation programs are available in the United States. The Five-Day Plan is the most widespread United States program. Sponsored by the Seventh-Day Adventist Church, it is copied by other groups. The Church also sponsors live-in programs which augment the five-day program with lectures by specialists in exercise, counseling, dietetics, physical therapy and pulmonary medicine.

Methods using medication are not as frequent in the United States as in Europe, but many preparations are available in drug stores. Commercial organizations are taking over a larger share of cessation activities, especially on the east and west coasts. SmokEnders and Schick are the most prominent among over a dozen profit-motivated companies. In California. Schick charges $450 for five days of smoke satiation, rapid smoking, and shock treatments followed by eight weeks of educational meetings. SmokEnders charges from $120 to $175 for nine weekly meetings which emphasize changing attitudes and gradual quitting.

Behavior modification techniques are widespread in the United States, although many are experiments conducted by college students as part of their doctoral research. The most consistent long-term group utilizing behavioral methods is Lichtenstein's at the University of Oregon; these researchers have developed rapid smoking techniques and have used hot smoky and fresh air, contingent punishment, negative practice, breath holding, coverant control and attention placebo.

Satiation, self-control, group therapy, rapid smoking, shock treatment, taste aversion, covert sensitization through imagining, systematic desensitization, attention placebo, counseling, and contingent management are the methods which have received the attention of behaviorists.

The extent of systematic counseling by physicians in the United States is not known. Spiegel has pioneered a self-hypnosis technique and numerous other psychiatrists utilize group and individual hypnotherapy to cure the habit. The Multiple Risk Factor Intervention Trial attempts to intervene on three factors. About 12,000 high risk men in twenty cities have completed this group program supplemented by individual counseling.

The Stanford community project was mentioned earlier as achieving success. A public education project involving the entire community of San Diego, which ended in 1975, used a variety of cessation methods

Other methods that have been tried in the United States to help smokers quit include radio and television cessation programs, role playing, psychodrama, relaxation, meditation, adult school classes, and other miscellaneous procedures.

CURE RATES

Frequently smoking trials suffer from poorly done evaluations. In many cases no evaluation is done at all; in others the evaluation is limited to a three to six month follow-up. Psychologists conducting aversive therapy or self-control methods and psychiatrists practicing hypnosis for smoking control used the shortest term follow-ups--generally from one to six months. Often follow-ups are based only on those who complete treatment, those who reply to follow-ups, or those who are cured. Some investigators record the number of cigarettes reduced, rather than the number of people cured entirely of smoking. These studies are of little value as far as the evaluation of smoking cures. Use of controls, valid sampling techniques, and good design would improve evaluation methods.

Table 1 shows the results of 123 smoking cessation trials. Although some methods include several techniques, each effort is listed under its most logical heading. Only those methods which reported a minimum of a five month follow-up were listed; the follow-up period varies up to four years. Slightly over one-half the trials listed in the table had a one year or longer follow-up. The shortest follow-up periods were for the aversive conditioning and hypnosis methods. Only one in four aversive trials and one in three hypnosis trials reported a follow-up of at least one year. Obviously a one or two year result is closer to the true cure rate than a six month report. In some cases, rates were recalculated to include left out subjects; many of the listed results were based only on persons completing the treatment. The number of subjects followed-up is noted, where available.

Thirty percent of the trials listed showed cure rates of at least 33 percent and one-fifth had success rates of at least 40 percent. Almost one-half of the studies had cure rates of 21 percent or less and cure rates for one in six trials were in the zero to 13 percent range. Althouah results varied widely in each method, the best results appear to be in hypnosis, group- counseling, and. rapid smoking; the small number of subjects in some of these trials makes their statistical validity questionable.

Little is known of long-term results from individual physician counseling. English and Scottish doctors have reported from five to 39 percent success, the best results coming with male patients who already have heart or chest problems (Schwartz and Rider 1977).

The Five-Day Plan shows about 70 percent cured on the last night of the course but follow-up reports indicate that recidivism is high. Berglund (1969) found a wide variety of success rates with the Plan: only 10 percent in one Norwegian town, 50 percent in another, and 33 Percent in her Philadelphia evaluation (McFarland et al. 1972). Hirvonen (1972) reported that results in Finland with the Five-Day Plan were 28 percent successful with men but only eight percent successful with women. Hammer (1975) claims excellent results from German Five-Day Plans: 29 percent at one year rising to 43 percent at four years, but this result is an estimate. According to Rice (1973), in-residence treatment at the Seventh Day Adventist Church's facility in St. Helena, California, showed 35 percent cure rates a year afterwards.

Lecture and discussion groups have shown follow-up cure rates in the 20 percent range. One exception is Ball (1970) who achieved 33 percent success after one year with chronic patients. Adult classes in two different areas showed cure rates of 20 and 38 percent (Schwartz and Rider 1978).

Outstanding results with groups are those of Bozetti (1972) in San Diego who reported 85 percent success for men and 57 percent success for women after one year. Ghelov (1975) at the Kaiser Foundation Health Plan in Oakland reports that six month results at the Stop Smoking Clinic, now in its seventh year, have improved from 25 percent, the 1972 result, to 48 percent success in 1974.

Commercial companies in the United States using group methods are claiming good results in their self-conducted evaluations. Three

independent evaluations, however, revealed more modest results: 35 percent of Smoke Watchers' participants were still not smoking a year later (Schwartz 1973)) 39 percent of the "graduates" of SmokEnders (but 27 percent of all participants) were successful after a year (Kanzler, Jaffe, and Zeidenberg 1976), and 21 percent of participants in a Canadian commercial method still did not smoke after six months (Wake, Tyas, and Herrick 1972).

Medication methods (particularly lobeline-type preparations and tranquilizers) have shown very poor results (Schwartz 1969; Jarvik and Gritz 1977). The Bulgarian preparation Tabex, however, shows fair (about 28 percent) success (Scharfenberg et al. 1971). Preliminary reports indicate that the Swedish nicotine chewing gum (Nicorette) offers promise if combined with long-term maintenance support (Branbnark et al. 1973). Russell and his colleagues (1976) at Maudsley found that 70 percent of the subjects who chewed nicotine gum quit smoking and 23 percent were cured after one year.

Some psychiatrists have had success with hypnosis, but others say that hypnosis does not work particularly well with smokers. Follow-up results of 60 percent or better are reported by Nuland and Field (1970), Hall and Crasilneck (1970)) and Kline (1970).

In the past, aversive methods have shown very low success rates (Schwartz 1969; Bernstein and McAlister 1976; Lichtenstein and Danaher 1977). Few evaluations have been made at one year and those extant report little success. Lichtenstein et al. (1973)) Best (1975), and Tongas et al. (1976) have demonstrated that rapid smoking can be effective if combined with follow-up support. Most reports indicate that shock treatment is not effective but several investigators report good results. A variety of behavior modification methods--contingent management, desensitization, covert sensitization--show varied results. The most promising combine self-control methods (Flaxman 1977), but pocket timers and other signal devices show poor results. Brengelmann (Germany 1975) has reported success with some of his methods combining several treatments, particularly self-control with contingency contracting.

Pomerleau and his colleagues (1977) at the University of Pennsylvania are conducting a series of well-done smoking studies which are producing interesting results. They used a multicomponent procedure which combined the following techniques in a single trial: a commitment fee, slow reduction, stimulus control, contingency management, quotas with social reinforcement, covert conditioning, muscle relaxation, exercise, and group support. Subjects were also offered satiation procedures and, as cues, pocket timers. Of 100 subjects followed up, 61 percent quit initially and 32 percent were still off at one year. Subjects undergoing satiation had the same result as those who abstained through slow reduction. Differential effectiveness of procedures was not tested by Pomerleau et al. but they intend to do that in future trials.

It is too early to judge the efficacy of multiple risk factor trials and mass media cessation programs. Risk factors trials in Europe report one-half or more of the participants quit smoking through

these projects. The great number of smokers reached over radio and television suggests the potential of mass media's helping many people to cure their habit. Dubren (1977) reports a 10 percent quit rate for a New York City television cessation program that consisted of nightly 30 second to 90 second messages broadcast over a three week period. A similar program televised in Nashville claimed a 15 percent success rate. Counting their successes accurately is, of course, most difficult.

Techniques based on meditation, yoga, relaxation, and exercise appear to be gaining great interest and promise. Methods aimed at reaching a wider audience, such as treatment by correspondence or telephone, may help people trying to quit smoking who do not wish to attend clinics. Community, office, and factory cessation programs are other ways of reaching people in a familiar environment.

DISCUSSION OF RESULTS

A review of control programs conducted during the last few years demonstrates that certain conditions improve success; these are the use of multiple cessation methods to meet the needs of different types of people or uses of cigarettes; fees, as in the commercial programs which intensify commitment; and the presence of illness or risk factors which enhance the motivation to quit.

A noticeable improvement in cessation programs is the use of several methods to initiate and sustain cessation. Reliance on a single treatment method generally yields poor results because of diverse personality factors. A few investigators are studying personality types and their respective responses to various methods.

Pomerleau analyzed variables and found that four of them could be used to predict long-term treatment outcome: prior smoking rate, years smoking, percent overweight, and compliance with recordkeeping. None of these variables were significant, however, at the end of. treatment. They also found that negative affect smokers eventually showed a higher recidivism rate than non-negative affect smokers, although negative affect did not predict initial treatment outcome. They suggest that participants for whom less favorable prognosis is indicated should be monitored closely throughout treatment and be given more intensive therapy.

The Smoking Control Research Project, in addition to testing cessation methods, compared persons able to stop smoking on a long-ten basis with persons who quit and went back to smoking and with people unable to quit at all (Schwartz and Dubitzky 1968). We also studied the process itself of quitting smoking, factors involved in recidivism, various types of smokers, and approaches to cessation.

We found that any male smoker--regardless of how much or how long he smoked; whatever his personality characteristics, anxiety level, or socioeconomic characteristics or status; or whatever his type of smoking or reasons for smoking--could succeed in giving up the habit temporarily. We discovered differences , however, between long- term and short-term quitters. Men who could quit for good were more satisfied with their lives, their jobs, and their relations with

women; they had lower levels of chronic illness and anxiety; they were less addicted to smoking; and they relied less on cigarettes to alleviate negative affects than persons who returned to smoking.

We learned from the Smoking Control Research Project that many people were psychologically dependent on cigarettes, that tranquilizers did not help, and that group counseling was not superior either to individual counseling or to medication, even though initially, counseling appeared to have higher success rates. We learned that continuing support for at least four months was needed to help people past the common point at which many return to smoking. We also noted that environmental influences had strong impact on those who return to smoking, and that nutritional advice to avoid weight gain is an essential element of a successful control program.

Despite much experimenting, behavioral investigators have not been able to design effective methods of attracting and involving the general public. A few behavioral techniques, such as rapid smoking, can help some hard-core smokers break the smoking habit. Although results are equivocal, rapid smoking and self-control devices present good success for initial cessation. Attention to the multiple environmental, social, and psychological factors involved in smoking, as well as to the addictive element, enhances success rates.

In many countries, two interesting facts about women smokers emerge: first, the per capita cigarette consumption of women has increased, and second, women have lower quitting success rates than men. Attention should be paid to these findings and further study devoted to ascertaining the causes. Methods may be required that focus on the specific needs of women.

If methods are to be replicated, clearer descriptions of the procedures used will have to be provided. Weak methodological design in most smoking control efforts, particularly poor data collection, lack of controls, incomplete follow-ups, and reliance on reduction rather than cessation as a measure of success, has made it difficult to analyze and interpret results. Also needed are careful evaluations of results based on comparisons and long-term follow-up.

COMMENT

Every physician should ask patients about their smoking habits, point out evidence of smoking's harmful effects on the respiratory and cardiovascular systems, present a strong statement about quitting, and if the patient wishes, offer advice on how to quit or referrals to voluntary or commercial methods. Unfortunately, few physicians are willing to take time to counsel patients how to quit or even to make referrals. I have found that long-term support maintenance is necessary even if it is minimal, such as follow-up telephone calls. Most physicians are reluctant to offer any follow-up support, although it could easily be done by mail or bv telephone by the office-nurse or receptionist.

Cessation programs should include efforts to inform young people how difficult it is to stop smoking and to encourage them not to start.

Several countries have initiated wide-scale educational programs. The most notable program is Sweden's 25 year effort whose objective is to make the year group born in 1975 a non-smoking generation (Advisory Committee to the Swedish National Board of Health and Welfare). The program began in 1974 with expectant parents and includes a continuing effort to create a non-smoking environment. The Swedish program offers withdrawal clinics and educational efforts for children, parents, school personnel, health professionals, and general public. Stepwise measures include regulating the tobacco market through price increases, eventually barring cigarette vending machines and advertising, restricting where tobacco products can be. sold, conducting mass media campaigns, and giving government support to anti-smoking organizations. It is this type of total effort, supported by the government and the general public, which is needed to overcome the smoking habit and create a non-smoking environment.

The key to success in smoking cessation methods is a carefully devised system of support for ex-smokers lasting four months after quitting. Once the smoker abstains, a myriad of forces act upon the individual influencing him to return to smoking. These forces include environmental, social, and internal factors, such as mass media, smoking of peers, and stress. It is now widely accepted that cigarettes can cause drug dependence; when the smoker breaks his habit he still has to contend with the effects of his former addiction. It is for these reasons that maintenance takes on added importance. Programs which tailor their follow-up efforts to the individual's situation or special problems will improve their effectiveness.

In comparison with my earlier evaluations of smoking methods, there has been a noticeable improvement in long-term results for programs over the last several years. More sophisticated maintenance methods and more experienced smoking specialists are part of the reasons for the higher cure rates. The environment can be credited with some of the success through the anti-smoking climate that is being created by the public. More smokers are seeking a cure for their smoking and greater numbers of them are succeeding in breaking the habit.

Table 1

REPRESENTATIVE CURE RATES FOR SMOKING CESSATION METHODS

Investigator Location Year	No. of Ss	Type of Program	Cure Rate %	F/Up
INDIVIDUAL COUNSELING				
Cruickshank London, England 1963	32	Physician counseling	30	5 mo
Schwartz & Dubitzky Walnut Creek, Calif 1966	36	Individual counseling and placebo	31	1 yr
	36	Individual counseling and meprobamate	14	1 Yr
Williams England	160	Chest clinic, advice	37	6 mo
Pincherle & Wright London, England 1970	1,493	Anti-smoking message during exam	17-35	1 Yr
Baker, Oscherwitz, et al. San Francisco 1970	50	Pulmonary clinic, physical examinations, counseling, advice	34	6 mo

[Editor's note.] Complete references for the studies listed in this Table may be found in the following publications:

Schwartz, J. L.: A Critical Review and Evaluation of Smoking Control Methods. *Public Health Rep* 84:483-506, June 1969.

Schwartz, J. L. and Rider, G.: Smoking Cessation Methods in the United States and Canada: 1969-1974. In Wynder, E. L., Hoffman, D., and Gori, G. B. (Eds.) *Modifying the Risk for the Smoker,* Vol. II of the Proceedings of the 3rd World Conference on Smoking and Health, DHEW Publication (NIH) 77-1413, 1977.

Schwartz, J. L. and Rider, G.: *Review and Evaluation of Smoking Control Methods: 1976 Update.* National Clearinghouse for Smoking and Health, Bureau of Health Education, Center for Disease Control, U.S. Public Health Service. (Monograph forthcoming 1978).

Investigator Location Year	No. of Ss	Type of Program	Cure Rate %	F/Up
INDIVIDUAL COUNSELING (Cont)				
Lensky Pisku Czech 1971	nr	Psychotherapy, lobeline inhalation	11	15 mo
Porter England, 1972	191	Anti-smoking message during exam	5	6 mo
Han&l England, 1973	45 55	Anti-smoking message during exam	39M 11F	1 Yr 1 Yr
Shewchuck, Dubren, et al. New York City 1975	115 156	Individual counseling American Health Foundation	19 21	l yr 5 mo
Isacsson & Janzon Malmo, Sweden 1976	51	Counseling, anti-smoking Program	33	8 mo
EDUCATIONAL PROGRAMS				
Seventh Day Adventist Church Philadelphia 1964	35	Five-Day Plan	27	15 mo
Mills Hartford, Conn.. 1965	124	Five-Day Plan	26	1 Yr
Guilford Los Angeles 1966	173	Five-Day Plan	16	1 Yr
Thompson & Wilson Pittsburg, Pa. 1966	201	Five-Day Plan	16	10 mo
Seventh Day Adventist Church Eugene, Oregon 1967	30	Five-Day Plan	23	6 mo
Berglund & Green Philadelphia, Pa. 1969	378	Five-Day Plan	33	
Berglund Norway, 1969	195	Five-Day Plan	16	18 mo

nr= not reported

Investigator Location Year	No. of Ss	Type of Program	Cure Bate %	F/Up
EDUCATIONAL PROGRAMS (Cont)				
Porter Memorial Hospital Denver, Colorado 1971	990	Five-Day Plan	40	9mo-5 F
Wake, Tyas, & Herrick Ottawa, Canada 1972	24	Five-Day Plan	21	2yr
Hammer Bad Neuheim, Ger. 1975	nr	Five-Day Plan	43 estimate only	4 yrs
Rice St. Helena, Calif. 1973	188	In residence Five-Day Plan	35	1 yr
Milligan & Suttake Bergen County, N.J. 1973-74	159	Adult classes	20	1 yr
Schwartz Davis, Calif. 1975	8	Adult school class led by nurse	38	2 yr
Law-ton Philadelphia, Pa. 1964	12	Educative	17	15 mo
	11	Nondirective, superficial	20	15 mo
	10	Educative group		15 mo
Ball London, England 1965	75	Group discussion, physician lectures, films, specimens	33	1 yr
Allen & Fachler Philadelphia, Pa. 1965	150	Group discussion	23	1 yr

nr = not reported

Investigator Location Year	No. of Ss	Type of Program	Cure Rate %	F/Up
EDUCATIONAL PROGRAMS (Cont)				
Delarue & Moss Toronto, Canada 1969	472	Physical exams, lectures, discussions	29	1 y-r
Hepper, et al. Rochester, Minn. 1970	107	Lectures, films, panels, discussion, buddy system	13	10 mo
Nemzer Long Island, N.Y. 1973	nr	Amer. Lung Assoc. group, discussion, lectures	20	lyr
Seriff & Finkelstein New York City 1977	78	Lectures, films, questions, answers	15	1 yr
Novak Prague, Czech. 1975	293	Health education, medication, advisory service	32M 26F	2 yr
GROUP COUNSELING				
Lawton Philadelphia 1961	19	Group meetings	18	21 mo
1964	19	Group therapy	11	15 mo
Schwartz & Dubitzky Walnut Creek, Calif. 1966	36	Group counseling and placebo	28	1 yr
	36	Group couns. & meprobamate	19	1 yr
	36	Group counseling	17	lyr
Bozetti San Diego 1972	14	Group psychotherapy	85M 57F	1 yr
Wake, Tyas, & Herrick Ottawa, Canada 1972	nr	Commercial group method	21	6 mo

nr = not reported

Investigator Location Year	ND. of Ss	Type of Program	Cure Rate %	F/Up
GROW COUNSELING (Cont)				
Schwartz Glen Rock, N.J. 1973	16	Smoke Watchers (commercial) group method	38	4-12 mo
Ft. Lauderdale, Fla. 1973	55	Smoke Watchers	25	4-12 mo
Vancouver, B.C. 1973	209	Smoke Watchers	37	4-12 m
Pyszka, et al. Los Angeles, Ca. 1973	354	American Cancer Society Group counseling, insight development	28M 20F	18 mo
Ghelov Oakland, Ca. 1974	134	Group counseling kaiser Foundation Health Plan	48	1Yr
Shewchuck, Dubren, Burton, et al. New York City 1974	104	American Health Foundation groups	21	1 Yr
1975	173		32	5 mo
Qneinder San Diego, Ca. 1974	30	Group, individual sessions, stimulus satiation, self-control	27	1 Yr
Kanzler, Jaffe, & Zeidenberg New York 1975	167	SmokEnders (commercial) group method	27	4 yr
Pederson, Scrimgeour, & Lefcoe London, Ontario 1975	16	Group counseling	0	10 mo
1976	16	Group counseling	19	6 mo

nr = not reported

Investigator Location Year	No. of Ss	Type of Program	Cure Rate %	F/Up
GROUP COUNSELING (Cont)				
Pederson, Scrimgeour, & Lefcoe London, Ontario 1976	21	Group counseling and rapid smoking	38	6 mo
	23	Group counseling, rapid smoking, and hypnosis	13	6 mo
Tongas, Goodkind, & Patterson Los Angeles, Ca. 1976	19	Group therapy	5	2 yrs
MEDICATION				
Ejrup & Wikander Stockholm, Sweden 1957-58	1,012	Injection of lobeline hydrochloride, meprobamate, anticholengeric substances, lectures, counseling	23	6 mo
Rosenberg Copenhagen, Denmark 1958-59	125	Injection of lobeline hydrochloride, restinil, silver acetate, auto-suggestion exercise	9	6 mo
Yllo Stockholm, Sweden 1959		Lobeline, lectures, pamphlets	15	6 mo
Whitehead & Davies Denver, Colorado 1962	16	Methylphenidate	6	16 mo
1963	6	Methylphenidate	0	8 mo
	5	Diazepan	20	8 mo
Ross Buffalo, N.Y. 1963-64	1,472	Lobeline, amphetamine, pentolbarital, methamphetamine, discussion	21M 12F	3-12 mo
Hoffstaedt Newcastle Upon Tyne England 1964	80	Lobeline, hydroxyzine discussion	47	LO mo

nr = not reported

Investigator Location Year	No. of Ss	Type of Program	Cure Pate %	F/Up
MEDICATION (Cont)				
Arvidsson Stockholm, Sweden 1964	54	Lobeline, lectures, pamphlets	31	1 Yr
Ejrup New York City 1965-67	155	Injections of lobeline hydrochloride, amphetamines, counseling	20-26	1 Yr
Schwartz E Dubitzky Walnut Creek, Calif. 1966	36	Prescription-placebo	25	1 Yr
	36	Prescription-meprobamate	8	1 Yr
Arvidsson Stockholm, Sweden 1971	100	Atropine-like substances, groups, aversion therapy	35	1 Yr
	390	Atropine-like substances, suggestion therapy	12F 19M	1 Yr
Westling Sweden 1976	nr	Nicotine chewing gum	26	1 Yr
Russell London, England 1976	43	Nicotine chewing gum	23	l y r
HYPNOSIS				
Moses Jamaica Plains, Mass. 1959-1962	45	Single hypnosis treatment	30M 0F	l-4 yr
Nuland & Field New York City 1970	97	Hypnosis		6 mo
	84	Meditation during hypnosis, self-hypnosis	60	6 mo
Hall & Crasilneck Dallas, Texas 1970	75	Individual hypnosis	73	2 yr
Spiegel New York City 1970	616	Self-hypnosis	20	1 Yr

nr = not reported

Investigator Location Year	No. of Ss	Type of Program	Cure Rate %	F/Up
HYPNOSIS (Cont)				
Kline New York City 1970	60	Group hypnotherapy, relaxation, imagery	88	1 yr
Shewchuck, Dubren, et al. New York City 1975	193	Self -hypnosis, American Health Foundation	17	1 Yr
	113		12	5 mo
Lefcoe London, Ontario 1975	50	Single hypnosis treatment	8	8-12 mo
Pederson, Scrimgeour, & Lefcoe London, Ontario 1975	16	Hypnosis and group counseling	50	10 mo
1976	17	Group hypnosis & counseling	53	6 mo
	16	Hypnotherapy by videotape and counseling	19	6 mo
	16	Relaxation, hypnosis and counseling	13	6 mo
Watkins Missoula, Montana 1976	36	Individual hypnosis, concentration, relaxation	67	6 mo
AVERSIVE CONDITIONING				
Mees Springfield, Oregon 1966	33	Breath holding, electric shock	11	6 mo
Russell London, England 1970	14	Electric shock	43	1 Yr

nr = not reported

Investigator Location Year	No. of Ss	Type of Program	Cure Bate %	F/Up
AVERSIVE CONDITIONING (Cont)				
Chapman, Smith & Layden Seattle, Washington	11	Electric shock, self-management training	55	l yr
1971	12	Electric shock	25	1 Yr
Koenig Palo Alto, Calif. 1966	42	Desensitization, relaxation, counseling	10	6 mo
Keutzer Eugene, Oregon	28	Breath holding	0	6 mo
1967	30	Coverant therapy	20	6 mo
	34	Attention placebo	18	6 mo
	31	Negative practices	10	6 mo
Steffy, Meichenbaum, & Best Waterloo, Ontario, Can. 1970	48	Overt & covert verbalization	17	6 mo
Wagner Columbia, S.C. 1971	27	Systematic &sensitization, satiation, role playing, positive reinforcement	19	6 mo
Lublin & Joslyn 1968	78	Hot smoky air, rapid smoking	19	1 yr
Grimaldi & Lichtenstein Eugene, Oregon 1969	20	Hot smoky air, noncontingent punishment, attention-placebo	0	6 mo
Schmahl, Lichtenstein, E Harris Eugene, Oregon 1972	28	Warm smoky air, warm mentho - lated air, rapid smoking	57	6 mo
Lichtenstein, Harris, et al. Eugene, Oregon 1973	40	Warm smoky air, rapid smoking, individual counseling	52	6 mo

nr = not reported

Investigator Location Year	No. of Ss	Type of Program	cure Pate %	F/Up
AVERSIVE CONDITIONING (Cont)				
Curtis, Simpson, & Cole Ft. Worth, Texas 1973	26	Rapid smoking, group discussion	15	5mo
Morrow, Sachs et al. Sacramento, Calif. 1973	55	Indiv. couns . , stimulus satiation, self -control	46	6 mo
B e s t Vancouver, Canada 1975	20	Satiation	40	6 mo
	20	Rapid smoking	55	6 mo
	20	Satiation & rapid smoking	47	6 mo
	89	Satiation	32	6 mo
Gordon New Brunswick, N.J. 1976	44	Rapid smoking, individ-ualized behavior therapy	16	6 mo
Pederson, Scrimgeour, & Lefcoe London, Ontario 1976	21	Rapid smoking	38	6 mo
Tongas, Patterson, & Goodkind Los Angeles, Ca. 1976	16	Rapid smoking	19	2 yr
	16	Covert condition	19	2 yr
	2l	Rapid smoking, covert condition , group	38	2 yr
Dawley & Sardenga New Orleans, La. 1977	12	Rapid smoking, warm smoky air, handling cigarette litter	17	6 mo
Pomerleau, Adkins, & Pertschuk Philadelphia, Pa. 1977	100	Stimulus control, contin-gency management, covert conditioning, relaxation, group support, pocket timers, satiation	32	1 Yr

nr = not reported

Investigator Location Year	No. of Ss	Type of Program	cure Rate %	F/Up
SELF-CONTROL				
McFall & Hammen Madison, Wise. 1971	36	Self-control & monitoring	5	6 mo
Pomerleau & Ciccone Philadelphia, Pa. 1974	48	Contingency management, self-control	46	l mo
Flaxman Chicago, Ill. 1974	64	Self-control procedures	63	6 mo
MASS MEDIA				
Green Rockville, Md. 1970	207	TV and Self-testing kit	23	1 Yr
COMMUNITY				
McAlister, Meyer & Maccoby Palo Alto, Calif. 1976	56	Screening exam, mass media, counseling	36	2 yr
MISCELLANEOUS				
Elliot & Tighe Hanover, N.H. 1966	14	Threatened monetary loss	38	15 mo
Hall Malmo, Sweden 1975	222	Peer pressure & groups	45	1 Yr
Wetterqvist Lund, Sweden 1973	98 192	Antidotal treatment	20M 11F	1 Yr
Benson & Wallace 1972	886	Transcendental meditation	38	9 mo

nr = not reported

REFERENCES

Advisory Committee to the Swedish National Board of Health and Welfare: *A National Smoking Control Programme.* Stockholm Sweden: National Smoking and Health Association, 1973.

Ball, K.: Cigarette Smoking and the Responsibility of the Hospital Physician. *Brit J Hosp Med* 4:865-866, December 1970.

Best, A. J.: Tailoring Smoking Withdrawal Procedures to Personality and Motivational Differences. *J Consult Clin Psychol* 43:1-8, 1975.

Berglund, E.: *A Follow-up Study of Thirteen Norwegian Tobacco Withdrawal Clinics.* Oslo, Norway: Landsforeningen Not Kreft (The Norwegian Cancer Society), 1969.

Bernstein, D. and Mister, A.: The Modification of Smoking Behavior: Progress and Problems. *Addictive Behav* 1:89-102, 1976.

Bozzetti, L. P.: Group Psychotherapy with Addicted Smokers. *Psychother Psychosom* 20:172-175, 1972.

Brantmark, B., Ohlin, P., and Westling, H.: Nicotine-Containing Chewing Gum as an Anti-Smoking Aid. *Psychopharmacologia* (Berl.) 31:191-200, 1973.

Brengelmann, J. C.: *Manual on Smoking Cessation Therapy.* Cologne, Germany: German Centre for Health Education, 1975.

Bureau of Health Education, Center for Disease Control and National Cancer Institute, *DHEW: Adult Using Tobacco--1975.* National Clearinghouse for Smoking and Health, Bureau of Health Education, Public Health Service, June 1976, 23 pp.

Davis, M. M.: America Challenges Medicine. In First Michael M. Davis Lecture, Graduate School of Business, University of Chicago, May 23, 1963.

Dubren, R.: Evaluation of a Televised Stop-Smoking Clinic. *Public Health Rep* 92:81-84, 1977.

Flaxman, J.: Quitting Smoking Now or Later: Gradual, Abrupt, Immediate and Delayed Quitting. *Behav Ther* 8, 1977 (in press).

Ghelov, T.: Personal Communication, April 23, 1975.

Gori, Gio B.: Approaches to the Reduction of Total Particulate Matter (TFM) in Cigarette Smoke. In Wynder, E. L., Hoffman, D., and Gori, G. B. (Eds .) *Modifying the Risk for the Smoker,* Vol II of the Proceedings of the 3rd World Conference on Smoking and Health, June 1975, DHEW Publication (NIH) 77, 1977 (in press).

Hall, J. A., and Crasilneck, H. B.: Development of a Hypnotic Technique for Treating Chronic Cigarette Smoking. *Intl J Clin Exp Hypn* 18:283-289, October 1970.

Hammer, 0.: The Bad Neuheim Stop-Smoking Therapy and Nonsmoking Training. *Therapiewoche* 25(30):4042-4044, July 23, 1975 (German).

Hirvonen, L.: "Ryhmahoito Tupauoinnistra Vieroittamisessa. (Group Therapy to Break the Smoking Habit.) *Suomen Laakarilehti* 27:3082-3088, 1972 (Finnish).

Jarvik, M. E. and Gritz, E. R.: Nicotine and Tobacco, In, Jarvik, M. E. (Ed.): *Psychopharmacology in the Practice of Medicine.* New York: Appleton-Century Crofts, 1977, pp. 483-498.

Kanzler, M., Jaffe, J. H., and Zeidenberg, P.: Long-and-Short-Ten Effectiveness of a Large-Scale Proprietary Smoking Cessation Program--a Four-Year Follow-up of Smokers Participants. *J Clin psychol* 32,3: 661-674, July 1976.

Kline, M. V.: The Use of Extended Group Hypnotherapy Sessions in Controlling Cigarette Smoking. *Intl J Clin Exp Hypn* 18:270-281, October 1970.

Lichtenstein, E..and Danaher, B. G.: Modification of Smoking Behavior: A Critical Analysis of Theory. Research and Practice. In, Hersen, M., Eisler, R.M. and Miller; P. M. (Eds.), *Progress in Behavior Modification* (Vol. 3). New York: Academic Press, 1977.

Lichtenstein, E., Harris, D. E., Birchler, G. R., Wahl, J. M., and Schmahl, D. P.: Comparison of Rapid Smoking, Warm Smoky Air, and Attention Placebo in the Modification of Smoking Behavior. *J Consult Clin Psycho1* 40:92-98, 1973.

McAlister, A., Meyer, T., Maccoby, N., and Farquhar, J.: Mass Media and Personal Instruction in a Campaign to Reduce Cigarette Smoking. Stanford Heart Disease Prevention Program, Menlo Park, Calif., undated. Mimeographed.

McFarland, J. W., Berglund, E., and Albrecht, C. E.: Present Status of S-Day Plans to Stop Smoking. In, R. E. Richardson (Ed.): *Proceedings of the Second World Conference on Smoking and Health, London, England, Sept. 20-24, 1971.* London: Pitman Medical, 1972, p. 85.

Nuland, W. and Field, P. B.: Smoking and Hypnosis: A Systematic Clinical Approach. *Int J Clin Exp Hypn* 18:290-306, October 1970.

Pomerleau, D., Adkins, D., and Pertschuk, M.: Predictors of Outcome and Recidivism in Smoking-Cessation Treatment. Unpublished paper, Center for Behavioral Medicine, University of Pennsylvania, Philadelphia, 1977.

Rice, A.: Five Day Plans and an Inpatient Stop Smoking Plan. In, J. L. Schwartz (Moderator), Proceedings of the Smoking Cessation Seminar, American Public Health Association, San Francisco, November 8, 1973.

Russell, M. A. H., Wilson, D., Feyerabend, C., and Cole, P. V.: Effect of Nicotine Chewing Gun on Smoking Behavior and as an Aid to Cigarette Withdrawal. *Brit Med J* 6032(2):391-392, August 14, 1976.

Scharfenberg, G., Benndorf, S., and Kempe, G.: Cytisin (Tabex) als Medikamentose Raucherentwohnung-Shilfe. (Cytisine (Tabex) as a Drug Aid in Breaking the Smoking Habit.) *Deutsche Gesundleitswesen* 26:463-465, March 4, 1971 (German).

Schwartz, J. L.: A Critical Review and Evaluation of Smoking Control Methods. *Public Health Rep* 84:483-506, June 1969.

Schwartz, J. L.: Preliminary Report: Smoke Watchers Evaluation. Unpublished, Berkeley, Calif: Institute for Health Research, May 6, 1973.

Schwartz, J. L. and Dubitzky, M.: *Psycho-Social Factors Involved in Cigarette Smoking and Cessation.* Berkeley, Calif.: Institute for Health Research, September 1968, 629 pp.

Schwartz, J. L. and Rider, G.: Smoking Cessation Methods in the United States and Canada: 1969-1974. In Wynder, E. L., Hoffman, D., and Gori; G. B. (Eds .) *Modifying the Risk for the Smoker,* Vol. II of the Proceedings of the 3rd World Conference on Smoking and Health, DHEW Publication (NIH) 77-1413, 1977.

Schwartz, J. L. and Rider, G.; *Review and Evaluation of Smoking Control Methods: 1976 Update.* National Clearinghouse for Smoking and Health, Bureau of Health Education, Center for Disease Control, U.S. Public Health Service. (Monograph forthcoming 1978).

Spiegel, H.: Termination of Smoking by a Single Treatment. *Arch Envir Health* 20:736-742, June 1970.

Tongas, P. N., Patterson, J., and Goodkind, S.: Cessation of Smoking through Behavior Modification Treatment and Maintenance. Paper presented at the Association for Advancement of Behavior Therapy, New York, December 1976.

Wake, F. R., Tyas, J., and Herrick, R.: *Cessation of Smoking Through Clinic Programmes.* Ottawa, Can.: Dept. of National Health and Welfare, 1972.

AUTHOR

Jerome L. Schwartz, Dr. P.H.
Chief, Health Care Research
Office of Planning and Program Analysis
California Department of Health
714 P Street
Sacramento, California 95814

and

Professor of Family Practice
School of Medicine
Department of Family Practice
University of California, Davis

DISCUSSION

Dr. Schwartz's comprehensive review raised several critical issues directed at clinical and research aspects of smoking cures:

> 1) What is the spontaneous rate of quitting in the population and how is it affected by medical reports, the climate of public opinion and environmental influences such as advertising? :
>
> 2) How are the various published reports to be compared with differing methods of calculating success rates, and standardized long-term follow-up?

Although Dr. Schwartz recalculated success rates wherever possible to include all subjects who began treatment, many published reports included only those who completed treatment. This latter calculation produced a systematic bias towards inflated cure rates; an alternate solution would be to publish two "cure" rates, one based on all patients entering treatment and a second on those completing the prescribed therapy. Closely related is the problem of non-standardized follow-up periods; at least one can match cure rates at any given time interval for different studies and treatments.

> 3) How do we evaluate the cost-effectiveness of different treatments, both from the standpoint of the consumer and of society?

Given the stated effectiveness of each general treatment-type, where should the buyer turn, and where should society put its capital? An economic cost-effectiveness analysis would suggest that the expected cost of a cure to the consumer would be the ratio of the expense of the cure in dollars divided by the average probability of cure ($Cost/p(Cure)) . From the standpoint of society. the total number of labor hours (patient and therapist) expended per person who stopped smoking is one possible critical calculation. Such calculations can yield different values to the buyer and to society as a whole: ,a very expensive but effective treatment might be a poorer buy to the consumer, who can purchase many cheaper treatments for the price of one theoretically more effective one; for society, the number of labor hours expended in producing an expensive cure may be less than for a cheaper cure and thus of greater value.

Ellen R. Gritz, Ph.D.

Mr. Emerson Foote has presented us with his observations of the cigarette industry from a unique vantage point. For many years he headed his own large, prominent advertising agency (Foote, Cone & Belding) that handled cigarette advertising. Then he had the courage to break with his past, and actually come out in opposition t0 such advertising. However, it is only in television that cigarette advertising has been effectively curtailed. It still constitutes a large proportion of the advertising which occurs in the press and only a few publications like the Readers Digest are trilling to voluntarily relinquish this enormous source of revenue. Although the conference did not take an official stand on advertising, the subject was discussed with much feeling, and it is clear the government and private health agencies will have to take a stand on this issue. -Ed.

The Time Has Come: Cigarette Advertising Must Be Banned

Emerson Foote

The effort to prevent human suffering from lung cancer and other diseases caused by cigarette smoking has been going on for a long time. You have to go back to Alton Ochsner and Ernst Wynder . It has now been more than twenty-five years since Dr. Wynder made history by scientifically proving the link between cigarette smoking and lung cancer.

Beginning with these pioneers, one of whom honors us with his presence at this conference, one must be awed by the tremendous amount that has been accomplished by scientists working in the field of smoking and disease. But one must be awed even more by how little has been accomplished-despite all we know about tobacco-in slowing down the rate at which Americans continue to kill themselves by their use of cigarettes. This is a strange dichotomy: we know so much about cigarettes, yet we have accomplished so little-comparatively-in controlling their use. Of course millions have stopped smoking. Huge numbers of people are alive today who would not be alive except for the discoveries science has made about cigarettes. Yet, on the other hand, the output of American cigarette manufacturers now exceeds 650 billion units a year. And we must keep in mind that the cigarette industry produces not only cigarettes; it has another product-the annual production of 250,000 dead bodies. From such responsible authorities as the U.S. Public Health Service, The American Cancer Society, and The American Lung Association, 250,000 excess deaths-annually-due to cigarette smoking is a conservative consensus.

You are meeting here in this research conference to seek more effective ways to control smoking behavior and to reduce the toll of death and illness for which cigarettes are responsible. Since I received and accepted Dr. Jarvik's invitation to attend this conference, I have thought long and hard about what I, as a non-scientist, could say to you on the subject of controlling smoking behavior. I do have a few specific suggestions to make. But before making them, I thought I would review with you some of my own experiences in the field of smoking and health; some of my attempts and frustrations, hoping that this section of my talk

will not bore you and that from it you may judge my credibility as one to make suggestions.

You may wonder how I, as an advertising man, ever got involved in anti-cigarette work in the first place. The circumstances are a little unusual-and fortuitous. It began this way. One day in February 1964. I was at work in my office in New York City when my secretary burst in-she didn't-buzz me, she burst in-end said. "Mr. Foote. the White House is on the line." I picked up the phone and a pleasant sounding voice said, "Mr. Foote, this-is Dave Powers. Do you have a private line in your office?" I replied that I did not, and he said,, 'Well, could you go to one and call me back? I have something of importance to say to you which must be discussed privately." One of my colleagues on the same floor did have a private line, so I went to his office and called the White House and asked for Mr. Powers. (I might say that before I made the call back,. I figured that this had a fifty percent chance of being a hoax.) Mr. Powers came on the line and after I identified myself he said, Mr. Foote, I didn't call you, but don't be embarrassed because somebody has called about ten people within the last hour and told them to call us back-people we hid not call. But," he went on, "you're in good company. Governor Rockefeller got one of the calls." Mr. Powers added that they were asking the FBI to try to find who was doing this. I'm sure they never caught the crank.

About a month later, I was working in my office in Chicago one Saturday morning. At that time I maintained an office and a home both in Chicago and New York. I returned home about noon and my wife said. "You've just had a call from the White House. You are to call Mr. Mike Feldman there." My reply was something like this : "Listen, I've been around this track before. It's just another hoax." But my wife said, "How do you know it's a hoax? It might be a real call." So figuring that the odds were about 99 percent against its being genuine, I did call back. Being suspicious, I didn't use the number which had been left/but called Washington information to get the number of the White House-feeling a little foolish doing that. I dialed the number, and asked for Mr. Mike Feldman, who came on the line. I asked him if he had called me and he said yes, he had. He said that President Johnson had decided to establish a commission on heart disease, cancer and stroke and that I was invited to serve on this commission. He explained that a majority of the commission would be medical and scientific people but that some laymen were being invited, of which I was one.

After being assured that the work involved would not be too demanding, I accepted. In a couple of days I received a telegram signed "Lyndon B. Johnson" confirming the appointment.

The first meeting of the Commission was in April 1964 in Washington, under the chairmanship of Dr. Michael DeBakey . The Commission had 28 members, 16 scientists and 12 laymen.

Dr. DeBakey immediately set up eight subcommittees within the Commission to &al with the various phases of its work. I was made chairman of the Subcommittee on Communications. Also, my late great friend, Dr. Sidney Farber, was appointed chairman of the Cancer Subcommittee, and he asked me to serve on that committee in a specia1 capacity-as an adviser on communications.

I already believed-from the Surgeon General's Report which had recently come out, and from other factors-that cigarettes were. extremely deleterious to health. But it was my association with the scientific people on this Presidential Commission which convinced me that smoking really had to be done to combat the health hazard of smoking. I also came rather slowly to the conclusion that somebody in my field -the field of advertising and communications-should play a role in the anti-smoking effort. I did not see myself in this role at first, however. I hoped that somebody else would do it. I had a pretty good job. as chairman of the board of the second largest advertising agency in the world, and I had seven years to go before retirement. I knew that I couldn't do much in an anti-cigarette role without leaving my job because our firm had about twenty million dollars worth of cigarette business in various parts of the world. We didn't have any in the U.S.A. but West Germany, England and Australia were important cigarette markets for us.

Toward the middle of 1964, Dr. Farber asked me if I would make a recommendation to his subcommittee, to be referred later to the full Commission, as to how cigarette packages should be labeled and what should be said in cigarette advertising with reference to the hazard of cigarettes. My recommendation was that a fifteen word phrase be printed on all cigarette packages and included prominently in all cigarette advertising. The phrase was this: "The continued smoking of cigarettes may impair your health and may cause your premature death." At that time I believed, probably naively, that if such a phrase became mandatory in cigarette advertising the cigarette people would quit advertising. Of course they might have. The test never came, because the warning which Congress finally approved in 1965 was such a wishy-washy one that it did nothing to deter cigarette smoking. That was: "Cigarettes may be hazardous to your health."

My recommendation of what to put on cigarette packages and in advertisements was approved by the Cancer Subcommittee of the Commission on Heart Disease, Cancer and Stroke-with one dissenting vote. That dissent came, surprisingly, from the late Dr.. Charles Mayo, who said-and this is almost a verbatim quote-"of course cigarettes are dangerous to health. But if people want to smoke them they ought to be allowed to. I don't believe in this kind of government interference." However, the next day the Executive Committee of the President's Commission on Heart Disease, Cancer and Stroke approved my recommendation unanimously-nine to zero. (Dr. Mayo was not a member of the Executive Committee.) The recommendation still had to be approved by the full Commission, which wasn't to meet for about a month.

After that month-we are now up to August 1964-the Cancer Subcommittee met in Washington on a Sunday evening preceding the meeting of the full Commission the next day. One of the members of the Cancer Subcommittee panel, who had not attended the previous meeting, was the late General David Samoff, head of RCA. General Samoff had apparently not read the minutes of the previous meeting because during the course of our discussion he suddenly came upon the phrase, 'The continued smoking of cigarettes may impair your health and may cause your premature death." He became almost apoplectic, to the extent that Dr. Farber was worried about him. General Samoff said, in effect, 'Why, you can't do this! If I approved such a thing I would look ridiculous to my own company." He went on to say that if we insisted on adopting the phrase, he would go and see President Johnson the next day and get it killed.

I believed then, and I believe now, that he would have and could have accomplished this purpose by a visit with President Johnson. Finally, I withdrew the suggestion because I thought that the work of the entire Commission-dealing with three diseases-was more important than our specific work on cigarettes, as important as that was. The next day, at the meeting of the full Commission, with the bothersome phrase withdrawn, General Samoff was all smiles and said he was glad I had done "the right thing."

As a result of my work with the President's Commission on Heart Disease, Cancer and Stroke, in September 1964 I resigned my position as chairman of the board of my agency. I had decided that I was the only advertising man I knew who was willing to do something actively in the anti-cigarette field.

It may surprise some to know that at the time I left advertising, in September 1964, I had no specific idea of what I was going to do about the cigarette question. For one thing, I had not felt that I could explore this while I was serving my company as chairman. However, only a few days after my resignation was announced I had a call from a man who became a very close and treasured friend. This was Dr. Harold S. Diehl, Senior Vice President of The American Cancer Society for Research and Medical Affairs, and former Dean of the University of Minnesota Medical School. He explained that The American Cancer Society and others in the health field were establishing a new organization to be known as the National Interagency Council on Smoking and Health, and that he was authorized to offer me the chairmanship of this new organization. I accepted and became chairman on December lst, which was the day after my association with my advertising agency ended. (I had agreed to stay on a while after my resignation.)

The first formal meeting of the National Interagency Council on Smoking and Health was held in Washington on January 11, 1965, the first anniversary of the release of the Surgeon General's

Report. It was a lively start. Our meeting was in the auditorium of the NEA Building, at 16th and M Streets, and the place was jammed with media people. Senator Maurine Neuberger and Dr. Luther Terry were our principal speakers and we hit the front page of the New York Times the next day. One reason for this was that the U.S. Public Health Service, at our meeting, for the first time, had released an estimate of the number of premature deaths resulting from cigarette smoking. Their range, on this first release, was a pretty broad one-from 125,000 to 300,000 a year. The New York Times printed these figures in their front page story and as might be expected, the tobacco industry reacted rather violently. At one point they even accused me of making up the figures, though of course I had nothing whatever to do with the calculations.

During the year 1965 I worked almost full-time at my job as chairman of the Council, traveling about the country giving frequent lectures. I must say from a personal standpoint it was quite a change from my job in the ad world. I had one of the better paying jobs on Madison Avenue, and all of a sudden I found myself in a job which paid no salary and where you also had to pay your own expenses-all traveling, and even the salary of my secretary in New York. The Interagency Council may have had some cash budget but I, as chairman, never knew about it. The Public Health Service did give us a small office, and the time of one girl, in the National Library of Medicine. And they took care of postage and things like that. I used to think of the fact that during this same time the Tobacco Institute was rolling in money.

The National Interagency Council, from its inception, has been plagued by lack of money, and it still is. Though one should add that its tiny budget is in no way a measure of either its capacity or its accomplishment. This is because of the invaluable contributions of the many members representing many health organizations who come to its meetings and who do its work. This is not measured in money, but is the vital force in the work of the

I served as chairman of the National Interagency Council for two years, and was followed by Dr. Luther L. Terry, who served for a number of years. Early in 1967, I tried to get the National Interagency Council to endorse a new phrase to go on cigarette packages and in cigarette advertisements. I still hoped that Congress might do something worthwhile in this area. This time I had a shorter phrase. It was this: “Cigarette smoking frequently leads to disease and death.” I did think that might slow things down a bit in cigarette advertisements. At first the Council members, while indicating that they liked the phrase, felt it was not likely they could get it supported by all of their member organizations. Finally I recall saying something like this at the meeting where the matter was being considered: “I would like to depart from formal procedures, and simply ask a question. Will everybody in this room, who believes that the

phrase 'cigarette smoking frequently leads to disease and death' is not true, raise his hand?" Not a single hand went up. And the phrase was then approvedin a formal way. However, it never got outside our Council chambers. It was never publicized. It was never even offered to Congress.

So much for the problem of getting a really tough message on the cigarette package and in the cigarette ads. And this was ten years ago.

Finally, after more than thirteen years of trying to do something to slow down cigarette sales, I find myself holding the same view that I held in 1964: that no major slowdown of cigarette sales. in this country will occur until all promotional effort in support of cigarette sales is abolished.

The amount of cigarette advertising in this country is now in excess of 300 million dollars a year. This sum permits a constant barrage of pro-cigarette promotion which swamps whatever we do in an effort to curb cigarette consumption. This is not to say that present efforts cannot and do not, in some degree, succeed. But they exist in a situation of being engulfed by the. opposition.

I strongly believe that not only should cigarette advertising and promotion be wiped out, but that a very impressive campaign of educational advertising against cigarettes should be mounted-t taxpayer expense. There is no other place to get the money. In my view it would not be inappropriate to spend annually an amount roughly equal to what the cigarette companies are now spending for cigarette promotion. Three hundred million dollars a year for anti-cigarette advertising should be a disturbing figure only if one believes that the lives of one-eighth of the U.S. population are not worth such a sum. 'Iwo hundred and fifty thousand premature deaths annually from the effects of cigarette smoking is one-eighth of all U.S. deaths, which are standing at present at almost exactly two million a year. Unless the death rate is changed, you need only simple arithmetic, not actuarial mathematics, to conclude that roughly one-eighth of the entire American population will die, ultimately, from the effects of cigarette smoking.

Senator Robert F. Kennedy first made this point at the First World Conference on Smoking and Health in 1967, only he said one-seventh of the U.S. population - then twenty-eight million people - would die from cigarette smoking. Cur population has grown and one-eighth of the present U.S. population amounts to twenty-seven million people. Are not the lives of twenty-seven million Americans worth three hundred million dollars a year? This figure is substantially less than one percent of our present Federal budget.

Actually, it might be found that a figure of this size is not necessary because it might be counter-productive to harangue people all the time about not smoking. I simply believe that

whatever the amount is-that can best be used to induce people not to smoke-we should not blink at it. Furthermore, we do know from the results of the counter-cigarette spots which we had during the years of the FCC ruling-even though they ran in the face of a much larger volume of pro-tobacco advertising-that such spots can reduce cigarette consumption.

I have been trying as an individual and with some pretty able and dedicated help, for more than ten years, to get cigarette advertising abolished and to get something going in the form of counter-promotion. And I have really gotten nowhere.

But I believe it can be done-if the members of the scientific community will speak out and make their voices heard on the sub ject .

If the scientists interested in controlling smoking behavior, and reducing cigarette consumption, will take a stand on cigarette advertising I believe something can be done-possibly within a year.

You are scientists and I am an advertising man. But I beg you to believe me that if cigarette advertising is eliminated and counter-educational advertising commenced, then every project you are working on, or may work on in the future to control smoking behavior, will be more successful.

The entire atmosphere has to be changed.

It is worth mentioning in passing, and only in passing, that in recent years the cigarette industry has been artfully maintaining that cigarette advertising has nothing to do with the total sales of cigarettes; and that the only effect of cigarette advertising and promotion is to switch smokers from one brand to the other. Take my word for it-this is utter rot. And they know it is rot. They only advance the argument to try to take the heat off of efforts to restrict cigarette advertising. I can speak with some certainty here because for many years I was in the business of advertising cigarettes-Lucky Strike and Pall Mall and other brands. I spent ten years in this field, from 1938 to 1948. I might add that I have never had any conscious sense of guilt about this, and what I have tried to do in recent years against cigarettes has no connection, in my mind, because when I sold cigarettes there was hardly a scientific voice raised against them. When I joined the American Cancer Society as a director, in 1944, the air was always blue in the rooms where we met, and. I was assured that cigarette smoking was okay.

But that was long ago.

Today, I urge you to take a stand against cigarette advertising. If this conference comes out with a call for the end of cigarette advertising, it will accomplish more than you might think. And

if it could be followed by similar statements from other groups of scientists, and if the members of the scientific community would be willing to speak out even more firmly before Congressional committees, I believe we could see an end to cigarette advertising.

And that would mean, I am utterly sure, much better health for the American people.

AUTHOR

Emerson Foote
Former Chairman of the Board, McCann-Erickson Inc.
Life Member, Board of Directors, American Cancer Society
Gipsy Trail Road
Cannel, New York

Current Approaches

(Panel Discussion)

Social Learning

Edward Lichtenstein, Ph.D.

INTRODUCTION

Since several reviews of social learning' approaches to smoking control have been published recently (Bernstein & McAlister 1976; Lichtenstein & Danaher 1976) there is no point in repeating this material. Instead, I will give a brief overview, comment on some recent developments, and offer some opinions concerning the current state of affairs and future directions.

STATE OF THE FIELD

Recent reviews of the behavioral smoking control literature suggested two promising treatment strategies. Rapid smoking, in a warm, persuasive interpersonal context; and "multi-component interventions" which usually involve both the suppression of smoking and the teaching of nonsmoking and/or self-control skills. Both treatment approaches are complex and unstandardized; there are many procedural variations. Not surprisingly, recent results have been inconsistent. Danaher (1977a) reviewed 22 studies utilizing rapid smoking and found that rapid smoking consistently tended to be more effective than comparison procedures but absolute results were less impressive than in the earlier work with this procedure. Rapid smoking is importantly constrained by the cardiovascular risks imposed and the difficulty in estimating the degree of risk (Lichtenstein & Glasgow, in press).

Multi-component programs are even more diverse and have also yielded inconsistent results. On the positive side, there are several reports of very successful treatment programs with follow-up abstinence rates of 50% or better (Chapman, Smith, & Layden 1971; Pomerleau & Ciccone 1974; Lando 1977; Brengelmann 1973; Delahunt & Curran 1976; Best, Owen & Trentadue 1976). On the negative side, these are selected "successes;" there are many more studies with weak results, and informal communications suggest that investigators find it difficult to replicate their own successes. We don't yet know enough about the factors that make multi-component treatments work or not work.

PROMISING DEVELOPMENTS

Here I touch on recent work which appears to be of interest and importance for the field.

Controlled Smoking

One drink, one drunk; one smoke, one smoker? There are fascinating parallels between theory and research on controlled drinking and controlled smoking. Controlled smoking may be viewed as one approach to safer smoking and, like controlled drinking, is now a plausible treatment goal worthy of a fair trial. Frederiksen and his colleagues at the Jackson Mississippi VA have been exploring controlled smoking (Frederiksen $ Peterson 1976; Frederiksen, Peterson, & Murphy 1976; Frederiksen, Miller, & Peterson 1977). Thus far they have focused on identifying components of smoking behavior that may be amenable to change: e.g., substance, rate, and topography, Topography involves how much smoke is inhaled, number of puffs per cigarette and how much of the cigarette is smoked. These variables have been shown to be amenable to instructional control in small numbers of subjects. It remains to be seen whether smokers can achieve significant and lasting changes in their smoking.

Behavioral Analysis

In principle, social learning treatment of smoking or any behavioral problem requires empirical knowledge of controlling events -- discriminative stimuli and reinforcers. To a large extent, smoking control workers have *assumed* relationships between environmental events and smoking and/or have borrowed treatment strategies and tactics from other areas, especially obesity.

Most social learning workers, including myself, consistently ignore the implications of a large body of research which suggests that nicotine is a primary reinforcer for smoking and that, at least for heavy smokers, there are internal or physiological stimuli that drive the smoking habit (Russell 1976). Schacter has offered one provocative version of this theory (Schacter, Silverstein, Kozlowzki, Perlick, Herman, & Liebling 1971). The challenge for social learning workers is to incorporate this information on physiological processes into treatment programs. At the least, we should probably cease trying to persuade smokers that their habit is entirely or even largely under external stimulus control. Systematic study of environmental controlling stimuli is beginning (Epstein & Collins 1977) and should be pursued.

Another issue for behavioral analysis concerns, the relation of tension or anxiety to smoking. It is widely believed that much smoking occurs in the service of anxiety or tension reduction. Similar assumptions are made concerning alcohol consumption but the evidence in the area of drinking is complex and much of it seems to point away from a tension reduction model. (Nathan 1976). In a well-controlled but highly artificial situation Hutchinson and Emley (1973) have shown that nicotine functions like a minor tranquilizer. If smoking is reinforced by anxiety reduction then our tried and true anxiety management procedures ought to be effective for certain smokers. In

our own laboratory, relaxation procedures have not seemed to contribute to treatment effectiveness (Danaher 1977b; Glasgow 1976) but there are problems in training smokers to become skillful enough in relaxation to use it in their everyday lives. Administering anxiety management procedures according to smokers' measured anxiety levels may be more useful (Pechacek 1977).

A third issue within behavioral analysis concerns relapse or resumption . We know that many smokers, aided or unaided, achieve abstinence for short, or even long, periods of time but then relapse. We know very little about the factors involved in relapse. An empirical analysis of the relapse process would have important implications for improving maintenance. I suggest it will be more fruitful to study relapse episodes and how they are construed than to seek correlations between smoker characteristics and treatment outcome. In our laboratory we are conducting an exploratory study based on structured interviews with persons who have stopped smoking and then relapsed. Marlatt (1977) has performed a similar analysis with alcoholics and his theorizing may provide a framework for construing abstinence violations more generally.

Efficiency

A trend toward making treatment more efficient is observable in a number of recent social learning reports. This usually involves self-administered treatment sessions and especially the use of a manual which the client uses for homework assignments. Such studies, including two in our own laboratory (Kopel 1974; Glasgow 1976)) indicate that weak treatment effects can be just as readily obtained with less therapist time and fewer in-clinic sessions. Social learning methods do have the advantage of being relatively explicit, often simple, and thus amenable to self-administration. Though absolute effectiveness remains relatively weak, there may well be practical payoffs for wider dissemination of social learning methods.

DEVELOPING MORE EFFECTIVE TREATMENT

As the body of principles and methods encompassed by the rubric "social learning" changes and grows, there are corresponding shifts in approaches to smoking control. The complexity of smoking permits a wide variety of social learning strategies and tactics to be applied Unfortunately, choices often seem to depend on current fads or trends rather than flowing from a clinical and empirical analysis of smoking behavior itself. Thus we witnessed a flurry of studies applying Homme's (1965) coverant control[2] procedure when social learning was caught up in a covert conditioning[3] approach to cognition (Mahoney 1974). A more thorough behavioral analysis may help remedy this problem if combined with better integration of research and clinical experience with smokers.

Much of the social learning work is thesis and dissertation research carried out by graduate students who have worked little with smokers prior to the research project. This is because smokers are characteristically not treated in mental health centers, university clinics or counseling centers. Some of the more interesting current work comes from applied settings where social learning trained clinicians are dealing with "real" clients and also conducting research (Pomerleau &

Ciccone 1974; Best et al. 1976).

Partly because of its academic origins, social learning treatments have been more carefully evaluated than other methods. The one flaw in most social learning studies is consistent with the academic setting: an insufficient follow-up period. Graduate students usually cannot wait more than three months before completing their thesis or dissertation.

Graduate students and their professors do know about research design and control groups. Methodological purity is not always a virtue; with smoking it is often a liability. Armchair analyses of smoking control too often lead directly to group designs with control groups which typically find no relative differences among groups and weak absolute effects. Missing is pilot or clinical work with smokers wherein treatment procedures are developed, ineffective procedures discarded and promising once modified for more systematic evaluation. Our medical colleagues may have much to teach us in this regard, I suggest a greater emphasis on a clinical trials research strategy,. Smoking has an absolute zero point and numerous studies, such as those summarized by Hunt and Bespalec (1974) provide a benchmark for purposes of comparison. Rather than employing fancy control groups, it would often be wiser simply to give a reasonable number of smokers the preferred treatment and then collect systemtic follow-up data. Informants in combination with some physiologocal measurement -- either carbon monoxide or thiocyanate -- should be used to check on self-report. I believe we would be more impressed with clinically significant smoking-reduction and abstinence obtained from such a clinical trials study than we would by statistically significant group differences.

In summary, social learning has helped bring methodological rigor to smoking control. Several programs have been effective but the controlling variables are not yet sufficiently understood to permit reliable replication. More research on the variables controlling smoking and relapse and careful clinical trials prior to control groups comparisions are needed in order to develop effective treatments.

FOOTNOTES

1. Social learning is a “liberalized” version of behavior modification which emphasizes the importance of cognitive processes in operant classical and observational learning. The term also highlights the importance of the social-interpersonal environment in the acquisition and maintenance behavior.

2. Covert conditioning is one way of construing cognitive processes within a social learning framework. Mental events (thought) are construed as stimuli and/or responses and then analyzed or modified by means of the traditional classical or operant conditioning paradigms.

3. Coverant control is a procedure derived by Lloyd Homme. Coverant = covert operant; i.e., a thought or mental event that is assumed to function as an operant. Cover-ant control is a procedure for using

reinforcement principles to increase or decrease the frequency of targeted coverants.

REFERENCES

Bernstein, D.A. & McAlister, A. The modification of smoking behavior: Progress and problems. *Addictive Behaviors,* 1976, 71, 418-440.

Best, J.A., Gwen, L.E., & Trentadue, L. Comparison of satiation and rapid smoking in self-managed smoking cessation. Unpublished manuscript, University of British Columbia, 1976.

Brengelmann, J.C. Verbesserte methoden zur behandlung des rauchens . Unpublished manuscript, 1973. (Available from Max-Planck-Institut fur Psychiatric, Dept. of Psychology, Kraepelinstrasse 10, D-8 Munchen 40, West Germany.)

Chapman, R.F., Smith, J.W., & layden, T.A. Elimination of cigarette smoking by punishment and self-management training. *Behavior Research and Therapy,* 1971, 9, 255-264.

Danaher, B.G. Research on rapid smoking: Interim summary and recommendations. *Addictive Behaviors,* 1977a.

Danaher, B.G. Rapid smoking and self-control in the modification of smoking behavior. *Journal of Consulting and Clinical Psychology,* 1977b.

Delahunt, J. & Cur-ran, J.P. Effectiveness of negative practice and self-control techniques in the reduction of smoking behavior. *Journal of Consulting and Clinical Psychology,* 1976, 44, 1002-1007.

Epstein, L.H. & Collins, F.L., Jr. The measurement of situational influences of smoking. *Addictive Behaviors,* 1977, 2, 47-53.

Frederiksen, L.W., Miller, P.M., & Peterson, G.L. Topographical components of smoking behavior. *Addictive Behaviors,* 1977,

Frederiksen, L.W. & Peterson, G.L. Controlled smoking: The case for a new treatment goal. Paper presented at the Association for Advancement of Behavior Therapy, New York, Dec., 1976.

Frederiksen, L.W., Peterson, G.L., & Murphy, W.D. Controlled smoking development and maintenance. *Addictive Behaviors,* 1976, 1, 193-196.

Glasgow, R.E. The effects of a self-control manual and amount of therapist contact in the modification of smoking behavior. Unpublished doctoral dissertation, University of Oregon, 1976.

Hoanne, L.E. Perspectives in psychology, XXIV: Control of cover-ants, the operants of the mind. *Psychological Record,* 1965, 15, 501-511.

Hunt, W.A. & Bespalec, D.A. An evaluation of current methods of modifying smoking behavior. *Journal of Clinical Psychology,* 1974, 30, 431-438.

Hutchinson, R.R. & Emley, G.S. Effects of nicotine on avoidance, conditioned suppression and aggression response measures in animals and man. In W.L. Dunn, Jr. (Ed.), *Smoking Behavior: Motives and incentives.* Washington D.C., Winston & Sons, 1973, pp. 171-196.

Kopel, S.A. The effects of self-control, booster sessions, and cognitive factors on the maintenance of smoking reduction. (Doctoral disseration, University of Oregon, 1974.) *Dissertation Abstracts International,* 1975, 35, 4182B-4183B. (University Microfilms No. 75-3895).

Lando, H.A. Successful treatment of smokers with a broad-spectrum behavioral approach; *Journal of Consulting and Clinical Psychology,* 1977, 45, 361-366.

Lichtenstein, E. & Danaher, B.G. Modification of smoking behavior: A critical analysis of theory, research and practice. In M. Hersen, R.M. Eisler, & P.M. Miller (Eds.), *Progress in behavior modification (Vol. 3).* New York: Academic Press, 1976, pp. 70-132.

Lichtenstein, ,E. & Glasgow, R.E. Rapid smoking: Side effects and safeguards. *Journal of Consulting and Clinical Psychology,* in press.

Marlatt, G.A. Craving for alcohol, loss of control and relapse: A cognitive behavioral analysis. In G. A.. Marlatt and P.E. Nathan. (EdS.) *Experimental and behavioral approaches to alcoholism,* New York: Plenum Publishing Corporation, in press.

Mahoney, J.J. *Cognition and behavior modification.* Boston: Ballinger Press, 1974.

Nathan, P.E. Alcoholism. In H. Leitenberg (Ed.), *Handbook of behavior modification and behavior therapy.* Englewood Cliffs, New Jersey: Prentice-Hall, 1976.

Pechacek, T.F. An evaluation of cessation and maintenance strategies in the modification of smoking behavior. Unpublished doctoral dissertation, University of Texas at Austin, 1977.

Pomerleau, O.F. & Ciccone, P. Preliminary results of a treatment program for smoking cessation using multiple behavior modification techniques. Paper presented at the meeting of the Association for Advancement of Behavior Therapy, Chicago, Nov. 1974.

Russell, M.A.H. Tobacco smoking and nicotine dependence. In R.J. Gibbons, Y. Israel, H. Kalent, R.E. Popham, W. Schmidt, and R.G. Smart (Eds.), *Research advances in alcohol and drug problems (Vol. 3).* New York: Wiley, 1976. pp. l-47.

REFERENCES (Cont)

Schacter, S., Silverstein, B., Kozlowzki, L.T., Perlick, D., Heman, C.P., & Liebling, B. Studies of the interaction of psychological and pharmacological determinants of smoking. *Journal of Experimental Psychology:* General ,1977, 106, 3-40.

AUTHOR

Edward Lichtenstein, Ph.D.
Department of Psychology
University of Oregon
Eugene, Oregon 97403

The Long-Term Maintenance of Nonsmoking Behavior

Phoebus N. Tongas, Ph.D.

The purpose of this paper is twofold: (1) to present the findings of our research on the long-term maintenance of nonsmoking behavior; and (2) to present some thoughts on future directions of research in maintenance work.

OUR STUDY

INTRODUCTION

Approximately two and one half years ago, two very capable colleagues, Mr. Sheldon Goodkind and Mrs. Judi Patterson, joined me in an effort to find an effective, efficient and cost-effective method to help. Kaiser-Permanente members quit smoking. Our organization, as a pre-paid health maintenance group, has a deep commitment not only in treating illness effectively but preventing it. Thus, the maintenance of nonsmoking behavior is not only an intriguing area of research for us but has some very immediate and pragmatic implications with regard to the well-being of our subscribers. Influenced by suggestions made by Bernstein (1970), and Hunt and Matarazzo (1973), concerning the application of multiple rather than single techniques and an emphasis on long-term maintenance of nonsmoking behavior, we set out to investigate the effects of post-treatment maintenance on the cessation of smoking within four behavioral treatment modalities. All of our work has been done with groups to increase cost-effectiveness.

METHOD

SUBJECTS

The subjects were 38 males and 34 females, of whom 40 were physician-referred and 32 self-referred. They ranged from 27 to 73 years, with a mean of 50 and a median of 54. On the average, they smoked 2.5 packs per day for 30 years. They were randomly assigned to four conditions. There were two groups in each condition, one of which was run by a male therapist and one by a female therapist. The conditions were (1) aversive conditioning[1] (rapid smoking); (2) covert conditioning[2]; (3) behavioral group therapy; and (4) a combined condition.

PROCEDURE

An orientation meeting was set up two days before treatment for subjects and their spouses. Orientation lasted for 45 minutes, and included the following: (1) positive social reinforcement for coming to the Program and committing themselves to quitting smoking; (2) presentation of sane facts about the health hazards of smoking, descriptively and photographically; (3) presentation of reactions to be expected from quitting "cold turkey", to minimize unrealistic expectations and anxieties; and (4) information about what to do if they experienced mild withdrawal symptoms. At the end of orientation subjects were given a handout titled 'Yes You Can", which essentially summarized the points made during the orientation lecture. Spouses were given an additional mini-lecture, explaining the kind of treatment their mates would receive, alerting them to various reactions and giving them instructions on how to positively reinforce success, and to not punish, but merely ignore failure. They were also given a handout summarizing the above information titled "You Can Help Too". Contemporaneously, the subject s who were randomly assigned to the four conditions received instructions about the exact treatment procedures and filled out the project questionnaire.

The Aversive Conditioning Group

This group was treated in a room which was especially equipped with a powerful fan capable of evacuating smoke from the room within 10 to 30 seconds. In addition, a Maximyst air apparatus, available for each subject. supplied filtered room air as a re-inforcer for putting out cigarettes after the rapid smoking trials. Rapid smoking consisted of puffing every three seconds or inhaling every six seconds, and various combinations of these. There was a total of five treatment sessions, which were done in five con-secutive days, Maintenance sessions were exactly the same as treatment sessions and were spaced as follows. The first session was four days after the last treatment session. The second was one week later; the third, two weeks later; the fourth, three weeks later; the fifth, four weeks later; and sessions six through fourteen were one month apart. There was a total of 19 treatment and maintenance sessions in the 12 months of the program.

Covert Conditioning

In this condition there were six punishment scenes, six escape scenes, and three positive reinforcement scenes. In addition to the five treatment sessions, subjects were given home assignments and were instructed to do them three times daily. The assignment consisted of five punishment scenes, five escape scenes and five covert positive reinforcement scenes. Everything else in this condition was the same as in the aversive condition.

Behavioral Group Therapy

This was essentially talk therapy with behavioral objectives. The objectives were: (1) to facilitate the expression of feelings with regard to quitting smoking and provide support; (2) to reinforce success, both by therapist as well as group approval; and (3) to ignore failure as it was reported in the session. Subjects received a lecturette on the effects of positive social reinforcement and punishment, and were encouraged to use these principles during group therapy and in contact with each other outside of therapy. In addition, a "Ruddy System", in which each subject was to call two other group members each day just to make contact and find out how they were doing, was installed. If they were doing well, they were socially reinforced, and if they failed, their failure was ignored and the telephone call quickly terminated. In all other respects this condition was the same as the two mentioned above.

The Combined Group

The combined group received a combination of all the three treatments described above. The session was divided into three sections and each treatment occupied approximately one third of the session.

A more detailed description of the methodology may be found in a previous paper (Tongas, Goodkind and Patterson, 1976a).

RESULTS

The results are based on self-reports of all subjects who completed the five day treatment program. Six months after treatment the combined condition yielded 77% complete abstinence; the covert condition, 67%; the aversive condition, 57%; and the group therapy condition, 15% complete abstinence. Twelve months following treatment, the combined condition yielded 77% complete abstinence; the covert condition, 56%; the aversive condition, 36%; and the group therapy, 15%. Physician-referred subjects did not differ in their success rates from self-referred subjects. Results are based strictly oncomplete 100% abstinence from smoking. A 24 month follow-up yielded the following results: combined condition, 62% complete abstinence; covert condition, 33%; aversive condition, 21%; and group therapy, 8% abstinence (Tongas, Patterson and Goodkind 1976b).

DISCUSSION

The non-inclusion of a no-maintenance control group is a methodological weakness. However, since we knew that previous reported success rates ranged between 20% and 30%, we decided to use that as baseline, rather than deprive subjects of a possibly better treatment program, particularly since half of them were urged to stop smoking by their physicians for serious medical problems. A tentative conclusion can be drawn from the above study. It would

appear that a procedure for smoking cessation would have higher probability for long-term success if it consisted of multiple techniques and if it included a long-term maintenance program.

SOME THOUGHTS ON FUTURE DIRECTIONS

Recent despair over the failure of behavioral technology to produce 'nonsmoking behavior (Yates 1975), is unwarranted if the results are evaluated in view of the prevailing research goals. The goals have characteristically been to measure the effects of the brief application of techniques on smoking behavior. The results have been almost uniformly positive. Brief behavioral interventions produce high rates of nonsmoking behavior at the end of treatment. We have thus discovered what Mark Twain knew a long time ago, that it was easy to quit smoking. He had done it a thousand times. Commercial "stop smoking" programs, inspired both by Mark Twain's and psychologists' findings, have been prospering by offering expensive services which guarantee success or money back. Our own research shows better than 90% success as measured by total abstinence at the end of treatment. However, what happens between then and three months later is a disappointing fact well known to all researchers in this area.

Our failure has not been in the development of techniques for short-term cessation of smoking. Our failure has been in developing an effective technology for the long-term maintenance of nonsmoking behavior, which is understandable since this has not been a researchers' goal. The reason for this is quite obvious. Research on the maintenance of nonsmoking behavior does not produce the payoffs that short-term treatment of, smoking behavior does to those who have done most of the work so far. Studies on long-term maintenance would represent punishment rather than reward to Ph.D. candidates who must spend a number of months reviewing the literature, perhaps doing a pilot study, conducting a formal experiment, analyzing the data, writing the final product, and then engineering successful approval of their project by all the metiers of their dissertation committee. Such work has little reinforcement value for academic psychologists as well who are usually under pressure to turn out publications. For obvious reasons, private concerns would have a low interest in the long-term effects of their methods. Thus, we are left with very few sources of support for the type of research goals that would lead to the discovery of effective methods for keeping people away from cigarettes for long periods of time. The federal government is potentially such a source, as are medical organizations, like our own, whose financial rewards are contingent on patients' good health. The problem with the latter is that they have not had a tradition in supporting research with the type of vigor that is needed to arrive at answers which often require substantial investment of money and time.

Our recommendations for research directions in the long-term

maintenance of nonsmoking behavior are of two kinds. One involves a change in research goals, and the other involves changes in the research activity itself.

RESEARCH GOALS

While the goals of discovering techniques for helping people quit smoking for short periods of time have contributed useful information, it is now imperative that we move on to goals which will lead to knowledge that will help people stay nonsmokers. Regardless of whether or not nicotine itself is a physiologically reinforcing substance, the process by which it finds itself in the body is regulated by operant principles. Smoking, especially among heavy users of cigarettes, is an overlearned habit cemented in their repertoire of daily behavior through persistent and long-term practice. It is under the control of such a great number and variety of discriminative stimuli and reinforcers that the task of eliminating it for long periods of time is immensely difficult as every research study has shown. The pursuit of short-term treatment strategies with the hope that somehow a powerful enough technique will be found which will melt away the stubborn persistence of an overlearned habit would be a waste of effort and money. As Hunt and Matarazzo say, "In modifying habit should we not fight overlearning with overlearning, rather than assume that once a person has stopped smoking he is off smoking for good?" (Hunt and Matarazzo, 1973, p. 111).

Perhaps a change in our terms, which often serve as cues to research behavior, might facilitate a transition in goals. Instead of talking about the "therapy of" or the "treatment of" smoking behavior, we propose that we begin to talk about the "maintenance of nonsmoking behavior". We realize, of course, that the cue value of such a phrase would be enhanced if the appropriate behavior it triggers is reinforced by grant money while short-tens treatment types of research earn no such reinforcers. Through the process of discrimination the goals proposed here would soon find faithful advocates.

In addition, we would like to express a bias against the term "smoking control" and the research activity it connotes. While from a research point of view it is desirable to study the parameters that are important in the control of smoking from a strictly pragmatic and functional point of view, such a study would be of little importance. Efforts to control a habit such as overeating makes sense especially since total abstinence from eating, as most people would agree, would be less desirable than obesity. With smokers however, especially with individuals suffering from medical illnesses caused or aggravated by smoking, such control would be of little significance. Total abstinence from smoking, not control of the habit, is the appropriate goal.

RESEARCH STRATEGIES

We propose that future research in the maintenance of nonsmoking behavior focus on the simultaneous manipulation of three major types of human behavior: respondent[3], cognitive[4], and operant[5].

Respondent Behavior

If indeed, as Hutchinson and Emley (1973) have found, nicotine terminates or reduces stressful stimuli, then smoking in humans may reduce emotional behavior produced by aversive stimulation. Internal. visceral cues, may be viewed as triggers of smoking behavior which in turn is reinforced by the soothing effects of nicotine . In a world full of stresses such soothing effects would have to be replaced with equally or more soothing substitutes.

Some of the techniques already used in stress and anxiety reduction such as deep muscle relaxation, bio-feedback, autogenic training, meditation, etc. , deserve a great deal of attention in smoking research. Specifically their systematic use in the long-term maintenance of nonsmoking behavior needs to be explored.

Cognitive Behavior

As with most self-control procedures, covert conditioning (Cautela 1971), including both covert sensitization and covert positive reinforcement, are procedures which facilitate generalization from the so-called “treatment situation” to the “real world”. Research exploring the most efficient and effective use of long-term covert conditioning for the long-term maintenance of nonsmoking might give us a potent as well as easy to use tool. It is possible, for example, that after prolonged covert sensitization work with smokers, especially in their natural environment, the aversion produced will be strong enough to deter smoking for long periods of time. Furthermore, the advantage of covert procedures over overt ones is that they are safe and particularly safer with individuals who suffer from medical problems such as heart conditions, high blood pressure, etc.

Some of the coverant control procedures outlined by Mahoney (1970, 1974), might also prove to be-important tools in the long-term maintenance of nonsmoking behavior. Particularly the strengthening of self-statements incompatible with smoking in combination-with - the covert sensitization procedures mentioned above might be useful in long-term maintenance.

Operant Behavior

We propose the study of two types of operant behavior in maintenance work: (1) that which controls nonsmoking behavior (self-control procedures) ; and (2) behavior incompatible to smoking. They are

both useful in the long-term maintenance of nonsmoking behavior and research designed to develop an efficient and effective technology which would sustain such nonsmoking is of paramount importance.

Self-control strategies must be conceptualized as part of maintenance work because the client is an active participant in the implementation of the goal of nonsmoking. If practiced regularly and consistently for as long as necessary, self-control procedures, in addition to facilitating generalization from the "treatment" situation to the clients' everyday life, might also guarantee long-term behavioral change. Behavioral programming as outlined by Thoresen and Mahoney (1974), would probably be best suited for long-term maintenance work. The self-reward and self-punishment procedures are relatively easy to use but must be so designed that they are supported by clients' natural environment.

The specification and manipulation of behaviors incompatible to smoking are perhaps the areas which will require the most creative and innovative work by researchers. Bandura (1969), aptly expresses the operant view regarding the successful deceleration of undesirable responses when he writes that the optimal application of counter-conditioning involves the deceleration of the target responses with the concurrent acceleration of appropriate substitute behaviors. What are substitute behaviors to smoking? In our research, in a rather unsystematic and global fashion, we encourage various activities from reading a book or performing some other habit to taking a walk when the urge to smoke strikes, as well as at times when there is absence of the urge to smoke. This is the area in which good research is needed in defining as well as programming into the clients' lives very systematically, substitute behaviors which would help break present chains, and weaken the control of many discriminative stimuli in smokers' environment.

In concluding, we would like to suggest that in our bias none of the above proposed directions have a high probability of success without the support of the clients' natural environment. Covert, operant, and respondent manipulation have little chance to contribute to long-term nonsmoking unless they are anchored in and appropriately reinforced by their immediate environment. External backup reinforcing power is needed in order to sustain clients' motivation for long-term homework activity. Thus, family, friends, peers, employers, and colleagues, must begin to be included in long-term smoking research in appropriate ways and as realities in each case allow.

FOOTNOTES

1. Aversive conditioning: The association of an unpleasant stimulus with another stimulus in order for the latter to acquire the properties of the former. In this case, the association of unpleasant rapid smoking with the cigarette, in order to make the cigarette unpleasant.

FOOTNOTES (Cont)

2. Covert conditioning: Conditioning in which the process of association or two stimuli is carried out only in the imagination.

3. Respondent behavior: Autonomic, involuntary behavior.

4. Cognitive behavior: Behavior invoking thinking and imagination.

5. Operant behavior: Voluntary behavior involving striated muscles mediated by the central nervous system.

REFERENCES

Bandura, A. *Principles of Behavior Modification* New York: Holt, Rinehart & Winston, 1969.

Bernstein, D. A. The Modification of Smoking Behavior: An evaluative review. In W. A. Hunt (Ed.) *Learning Mechanisms in Smoking* Chicago: Aldine, 1970.

Cautela, J. R. Covert conditioning. In A. Jacobs & L. B. Sachs (Eds). *The Psychology of Private Events: Perspectives on Covert Response Systems* New York: Academic Press, 1971.

Hunt, W. A. & Matarazzo, J. D. Three years later: Recent developments in the experimental modification of smoking behavior. *Journal of Abnormal Psychology* 1973, 81, 107-114.

Hutchinson, R. R., & Emley, G. S. Effects of nicotine on avoidance, conditioned suppression and aggression response measures in animals and man. In W. L. Dunn, Jr. (Ed.), *Smoking Behavior: Motives and Incentives* Washington, D.C.: Winston & Sons, 1973.

Mahoney, M. J. Toward an Experimental Analysis of Coverant Control *Behavior Therapy* 1970, 1, 510-521.

Mahoney, M. J. & Thoreson, C. E. (Eds.) *Self-Control: Power to the Person* Monterey, Calif.: Brooks/Cole, 1974.

Thoresen, C. E., & Mahoney, M. J. *Behavioral Self-Control* New York: Holt, Rinehart & Winston, 1974.

Tongas, P. N., Goodkind, S. D. & Patterson, J. L. An investigation of the effects of post-treatment maintenance on the cessation of smoking within four behavioral treatment modalities. Paper presented at the meeting of the Western Psychological Association, Los Angeles, Ca., 1976 (a).

Tongas, P. N., Patterson, J. L. & Goodkind, S. D. Cessation of smoking through behavior modification: Treatment and maintenance. Paper presented at the meeting of the Association for Advancement of Behavior Therapy, New York, 1976 (b).

Yates, A. J. *Theory and Practice in Behavior Therapy* New York: Wiley, 1975.

AUTHOR

Phoebus N. Tongas, Ph.D., Chief Psychologist, Department of Psychiatry, Kaiser-Permanente Medical Care Program, Los Angeles, California 90027.

Hypnosis in the Treatment of the Smoking Habit

Louis Jolyon West, M.D.

The influence of one person upon another is a basic aspect of human existence, and the exercise of interpersonal influence has been fundamental to the healing art since its origins in antiquity. To exercise his influence, the healer must attract and hold his patient's attention. In so doing, one possible consequence is that the person may become entranced. Suggestions by healers to patients always have great influence, especially if patients are already receptive to the concepts expressed by their healers. In the state of highly focused attention characteristic of the hypnotic trance, suggestibility is generally increased to a considerable degree.

The use of trances as part of the healing influence of the medical practitioner is described in the most ancient writings. Egyptian physicians in the second century B.C. employed "temple sleep" as a form of treatment. The therapeutics of Greek medicine included the use of suggestion and the induction of trancelike states to facilitate the healing process. While credit for successful cures went to each era's important gods, their physician-priests, and their mysterious medications, the healing influence undoubtedly stemmed primarily from the personality of the physician, the power of his suggestions, and the mental set (or state of expectations) of the patient.

Helmont (1577-1644?) taught that man possesses the power, magnetic in nature, to affect others, particularly those who are sick. In the early part of the eighteenth century Ferdinand Santanelli, who recognized the importance of the imagination in matters of health, also proposed the presence in all material things of a magnetic radiating atmosphere through which disease processes can be affected. From the writings of these men and other more obscure sources, Franz Anton Mesmer (1734-1815)) a student of medicine in Vienna, drew material for a doctoral thesis entitled *De Planatarum Influxu* (On the Influence of the Planets). He postulated that the planets exercised a direct effect upon all tissues of the human body, supporting his view with Galen's statement that the moon influenced epileptics and hysterics and with Tulp's

opinion that renal colic was related to lunar cycles. To provide a vehicle for planetary influence, Mesmer invoked the existence of a mysterious fluid, gravitas animalis (animal gravity), which penetrated everything in the universe.

In 1766 Mesmer undertook the private practice of medicine in Vienna and was an immediate success. In 1771, he encountered a visiting English Jesuit, Maximillian Hell, who was an accomplished astronomer and extremely interested in magnets. Father Hell claimed to have successfully treated some medical disorders by applying powerful magnets to the afflicted parts. This stimulated Mesmer to utilize in clinical practice some of the ideas propounded in his graduation thesis. Mesmerism (animal magnetism, the direct precursor of hypnotism) had begun.

Mesmer was a vital, aggressive, self-confident man. He entered into the practice of magnetism with enthusiasm and with the fervent conviction that he was controlling mysterious cosmic forces which could restore health and harmony to the human body. His cures were numerous, and in a few months his consulting room was crowded with patients from all over Austria.

It did not take Mesmer long to realize that magnets were not essential to his therapy. This led Mesmer to assume that he, himself, was the instrument capable of focusing the magnetic influence, which he now termed "animal magnetism." He published this view in his book, *Schrieben Uber Die Magnetcure,* in 1775.

Perhaps because of his success as much as his unorthodox theories and therapies the tide of medical opinion in Vienna soon turned against, Mesmer. In 1778 he moved to Paris where he became a sensation among the pre-revolutionary French nobility. Despite the opposition he encountered from medical and scientific authorities, he founded a clinic in Montmartre, and for the next five ears he employed magnetism in the successful treatment of hundreds of patients for various ailments. To enhance therapy, he constructed the famous baquet, a large round tub equipped with a double row of bottles containing iron filings in water and with rods to apply to the sufferers' afflicted parts.

Mesmer was convinced of the scientific validity of his method and insisted upon official recognition by the scientific community. In 1784, Louis XVI approved the formation of a Commission of Inquiry composed of nine prominent scientists from the Academy of Sciences and the Academy of Medicine. Members of the Commission included Benjamin Frankiin, Antoine Lavoisier, Antoine de Jussieu (the great botanist). and the memorable Dr. Joseph Guillotine. The Commission investigated Mesmer's clinic with compass and electrometer but could find no evidence of electrical activity in the baquet or its accoutrements. Blindfolded subjects were unable to determine whether or not they had been magnetized. The Commission concluded that Mesmer's results were due mainly to his patients' imaginations, and in a subsequent report noted

that some of his patients had relapsed and others had been rendered worse rather than better. However they failed to account for Mesmer's numerous "cures."

The controversy attendant upon the Commission's report was very disturbing to Mesmer personally, but it did not diminish his popularity in the least. Within a year the Marquis de Puyseguer, one of Mesmer's followers, described the phenomenon of somnambulism, a trance-like condition he had induced while magnetizing a shepherd boy. The phenomenon was quickly reproduced in other subjects and the public's interest in it only added to magnetism's renown. The controversy between Mesmer and the scientific community continued, however, until it became lost in the upheavals of the French Revolution. Mesmer fled to Switzerland and lived to be 82, pursuing to the end his experiments with magnetism and always convinced that he had discovered a great natural healing force that organized medicine must sooner or later accept.

From mesmerism came many offshoots. Some were of a mystical nature and used magnetism to abet clairvoyance or prescience, and to establish communication with the dead. Others attempted to continue with the scientific investigation of trances. The Abbe Faria (1756-1819), recognizing the subjective quality of trance states, demonstrated experimentally that no magnetic- force was required to explain them. Similarly, Jacaues Bertrand (1775-1831) stressed-that the subject's imagination and beliefs were important in the induction of the somnambulistic condition.

After the French Revolution, itinerant mesmerists traveled to other countries giving demonstrations. It was from such a traveler that a New England locksmith named Phineas Quimby learned the technique, and founded a system of "natural healing" called "New Thought ," which taught that all diseases originated in the imagination and thus could be cured by the suggestions of their nonexistence. He produced a spectacular cure in one who was to become his most remarkable patient: Mary Baker Eddy. who developed his method into a system-of her own called Christian Science.

In Manchester, England, in 1841, a surgeon named James Braid (1795-1860) witnessed a demonstration of magnetism by a traveling Frenchman named Lafonatine. He went with intent to scoff, but, after observing the phenomena, he realized their importance. Recognizing that the magnetic explanation of the clinical facts was nonsense, he set about investigating them systematically. He demonstrated their subjective nature, utilized them in his practice, and coined the term "hypnosis," which appeared in his magnum opus, *Neurypnology,* or, *The Rationale of Nervous Sleep.*

The distinguished British physician and medical educator, John Elliotson (1791-1868), became interested in magnetism and forced it upon the attention of the medical profession. One of his students, James Esdaile (1808-1859)) during a sojourn in India

shortly before the introduction of chemical anesthetics in the 1840's, performed more than one thousand operations (including some three hundred major surgical procedures) with hypnotic anesthesia alone. Esdaile cited the absence of flinching, restlessness, rapid pulse, or dilation of pupils during surgery to underscore the "reality" of the phenomenon of anesthesia in the somnambulistic state.

In France, the pursuit of hypnosis was furthered by A. A; Liebeault (1823-1904), a country doctor who used it in his everyday practice. Associated with him was H. Bernheim (1840-1919). a professor of medicine in the Medical School at Nancy, who shed his skepticism to become one of the great experimentalists in the field of hypnosis, introducing a variety of laboratory studies in carrying forward Liebeault's work, in his clinics at the Salpetriere in Paris. Jean Charcot (1825-1893) exercised a great influence in extending hypnosis to the respectable areas of medical practice and to the infant specialty of neurology.

Pierre Janet (1859-1947), who extended this tradition in a modified form, associated the phenomena of hypnosis with the dissociative symptomatology of fugues and somnambulism. Although Janet appreciated the influence of unconscious psychological forces in these phenomena, he refused to follow the lead of Joseph Breuer (l842-1925) and Sigmund Freud (1856-1939) who formulated the cathartic method of hypnotherapy and, in the early days of psychoanalysis, used hypnosis as a means of exploring unconscious mental processes and discharging pent-up emotions.

During the twentieth century, the interest of the health-related professions in hypnosis has been a fluctuating one. In the treatment of war neuroses, psychiatrists frequently found hypnosis to be quick and effective, and many practitioners were introduced to the method under these circumstances. Subsequently, in civilian clinical practice, however, their enthusiasm about hypnotic technique often declined, due in part to occasional embarrassing failures of ordinary hypnotic suggestion and in part to the time-consuming aspects of its use. A few psychiatrists, other physicians and psychologists have continued to explore hypnosis from the experimental point of view. In the past 20 years dentists, anesthesiologists, obstetricians and psychotherapists have probably been the most involved in uses of hypnosis in clinical practice.

Hypnosis, as it is practiced today, is a far cry from Mesmer's baquet . Usually the hypnotist gains his subject's attention by some verbal or nonverbal means, and quickly and easily focuses that attention through interpersonal transactions. Intense rapport can be developed quickly, permitting communications (verbal and nonverbal) from the hypnotist to become highly effective. At the hypnotist's suggestion, parts of the subject's body may move or fail to move due to "involuntary" alterations of motor function. Sensations are easily influenced by the hypnotist,

who usually begins with the common phenomenon of hypesthesia of the skin and follows with other sensory suggestions such as heat or cold.

If the subject is capable of entering a more profound trance, the hypnotist will be able to produce a variety of phenomena, including complete anesthesia of any part of the body, remarkable control of movement, considerable control of many autonomic functions and hallucinations in one or more sensory modalities. Strong emotions and seemingly delusional ideas may be induced. It is also possible to produce "negative hallucinations," in which the hypnotized subject fails to respond to objects actually present in his environment . The subject may appear to relinquish complete control over his actions and behavior, and create the impression of being totally dependent upon the operator. Posthypnotic suggestions of all kinds can be made during the trance by the hypnotist and be accepted by the subject, who acts on than at a designated time after hypnosis has been terminated, as though obeying a spontaneous impulse of his own.

The use of hypnosis to bring about a significant change in behavior (rather than simply to alleviate physical or mental symptoms) was already well developed in clinical practice by the end of the 19th century and up to World War I. A variety of undesirable sexual practices were reported to have been replaced by more acceptable behaviors. Various "bad habits" including alcoholism, drug addiction and the abuse of tobacco yielded to hypnotic suggestion and were reported in the literature of the time as cures. A certain number of slaves to tobacco have continued to seek help from hypnotists, both professional and amateur, to the present day.

Recently there have been a number of literature reviews and several comparative studies on the use of hypnosis in control of smoking behavior (Bernstein 1969; Francisco 1973; Hunt and Bespalec 1974; Johnston and Donoghue 1971; Pederson et al. 1975; Schwartz 1969). In the interest of brevity these Mill not be discussed individually here. However some general remarks on what emerges from such surveys of the effectiveness of hypnosis to break the smoking habit may be in order.

Generally a total cessation of smoking is the goal of hypnotic intervention, and results are likely to appear in sharply defined success or failure classifications rather than in categories that rate degrees of smoking reduction. On the whole, hypnosis rates higher in effectiveness than other single methods in comparable reports. However the range is great: 15% to 90% depending on the sample, the length of follow-up, and other variables. Hypnosis combined with individual counselling or psychotherapy appears to be more effective than hypnosis alone. Multiple doctor-patient contacts, employing hypnotic reinforcements on initial suggestions of abstinence, are more effective than a single session. However Spiegel's success rate with a single-treatment

method was quite respectable (Dengrove 1970; Nuland 1970; Speigel 1970a; Speigel 1970b; Wright 1970). Extended group hypnotherapy sessions as described by Kline produce even better results (Kline 1970). Kroger combines a type of behavior modification with hypnotherapy and self-hypnosis (Kroger and Fezler 1976; Kroger and Libott 1967), apparently to good effect in many cases although his statistics are not reported. Various minor modifications of hypnotic technique in treatment of smoking are described by other competent practitioners in recent articles (Frankel and Orne 1976; Hall and Crasilneck 1970; Nuland and Field 1970; Perry and Mullen 1975).

Two major concerns lie athwart the main road of progress in hypnotherapy of smoking. One is the problems of hypnotizability. The other is apprehension about possible risks involved in the use of hypnosis.

Hypnotizability is a complicated and troublesome subject. Since the review by Deckert and West (1963) there have been a number of worthy contributions to the literature on hypnotizability by such stalwarts as E. Hilgard, M. Orne, A. Weitzenhoffer and a few others. Spiegel et al. recently published an important study that strongly supports the modem view that hypnotizability is a normal characteristic, and is less rather than more likely to accompany psychopathology of various kinds (Spiegel et al. 1975).

What is germane about hypnotizability to the treatment of smoking is that not everyone can be hypnotized; that some who are hypnotized are only lightly affected and thus may be less easily influenced in the desired fashion; and that hypnotizability is a variable not a constant characteristic in any individual, depending on the conditions, the hypnotist, the technique of induction, previous experiences, etc. Taking into account these considerations, and reviewing the experience of others as well as my own, I would venture to set forth the following table as a very rough summary of reasonable expedctations on the effectiveness of direct suggestion in the cessation of tobacco smoking behavior:

		Percentage of Subjects	Percentage of Success (abstinence up to one year)
DEPTH OF HYPNOSIS	NONE	10	5 or less
	LIGHT	50	40
	MODERATE	30	70
	DEEP	10	90

No breakdown by technique would be appropraite because each hypnotherapist will use the methods that he has learned work best for him, and many are inclined to vary their techniques from one case to the next according to the patient's attitude and responses during the preliminary discussion and early in the hypnotic induction procedure.

While there are certain risks in the use of hypnosis (West and Deckert 1965), the professional person using this method to help a patient overcome the tobacco habit should not encounter anything he cannot handle. The rare exception -- probably less than one tenth of one percent of cases -- might be an untoward emotional reaction or a dissociative complication requiring some additional psychiatric intervention. By far the largest problem will prove to be failure of the procedure to produce or sustain the desired result, usually because the smoker finds some way to overcome the effects of therapeutic suggestions given in hypnosis just as he overcomes those given face to face in the physician's consulting room.

A few practical considerations should be added to this discussion. Hypnosis is not difficult to learn, but not everyone is comfortable with its employment. Some professional persons, including psychiatrists, adamantly refuse to use hypnosis because they cannot overcome the feeling that it somehow exploits the therapist's power in relation to the patient. Others are concerned that it may foster excessive dependency in the patient, or otherwise impair the best possible doctor-patient relationship. There are even some psychiatrists and psychologists who still fear that symptom-substitution may result, with the smoking habit (if removed) being replaced by something even worse. This is a most unlikely outcome in contemporary experience; smoking, after all, is very different from conversion hysteria.

There are other difficulties in the general employment of hypnosis in the treatment of smoking. Not all patients are willing to undergo the procedure no matter how far removed the technique may be from the Svengali-Trilby caricature. As noted above. not all of those who do participate will be sufficiently good subjects to produce high success rates. Because the pro-. cedure is time consuming it is, inevitably, expensive. Because it is uncertain it is relatively wasteful as well.

Nevertheless, in closing there are a few positive points I wish to emphasize. Hypnosis is an effective method to help some people to give up smoking. It may well be an effective adjunct to other methods as well. Any modem comprehensive smoking treatment program, using multiple modalities, should include hypnosis as a matter of course. What remains is to train more and better clinical hypnotherapists; to encourage more would-be ex-smokers (let's call them winners rather than quitters) to try hypnosis and, if possible, to repeat it in striving to maintain abstinence; and to undertake more research in order to learn how better to predict who the best subjects for hypnosis will be, and how best to utilize hypnosis in conjunction with other methods within the growing armsmentarium against the smoking habit.

REFERENCES

Bernstein, D.A.: Modification of smoking behavior. *Psychol. Bull.,* 71:418-440, 1969.

Deckert, G.H., and West, L.J.: The problem of hypnotizability: a review. *Int. J. Clin. Exp. Hypn.,* XI:2-5-235, 1963.

Dengrove, E.: A single-treatment method to stop smoking using ancillary self-hypnosis: discussion. *Int. J. Chin. Exp. Hypn..* XVIII:251-256, 1970.

Francisco, J. W.: Modification of smoking behavior: a comparison of three approaches. *Dis. Abstr. Int.,* 33:5511-5512, 1973.

Frankel, F. H., and Orne, M. T.: Hypnotizability and phobic behavior. *Arch. Gen. Psychiat.,* 33:1259-1261, 1976.

Hall, A., and Crasilneck, H. B.: Development of a hypnotic technique for treating chronic cigarette smoking. *Int. J. Clin. Exp. Hypn., XVIII:283-289,* 1970.

Hunt, W. A., and Bespalec, D. A.: An evaluation of current methods of modifying smoking behavior. *J. Clin. Psychol.,* 30:431-438, 1974.

Johnston, E., and Donoghue, J. R.: Hypnosis and smoking: a review of the literature. *Am. J. Clin. Hypn.,* 13:265-272, 1971.

Kline, M. V.: The use of extended group hypnotherapy sessions in controlling cigarette habituation. *Int. J. Chin. Exp. Hypn.,* XVIII:270-282, 1970.

Kroger, W. S., and Fezler, W. D.: *Hypnosis and behavioral modification: imagery conditioning.* Philadelphia: J. B. Lippincott, 1976.

Kroger, W. L., and Libott, R. Y. *Thanks, Doctor, I've Stopped Smoking.* Springfield, Ill.: C. C. Thomas, 1967.

Nuland, W.: A single-treatment method to stop smoking using ancillary self-hypnosis: discussion. *Int. J. Chin. Exp. Hypn..* XVIII:257-260, 1970.

Nuland, W., and Field, P. B.: Smoking and hypnosis: a systematic clinical approach. *Int. J. Clin. Exp. Hypn.,* XVIII:290-306, 1970.

Pederson, L. L., Scrimgeour, W. G., and Lefcoe, N. M.: Comparison of hypnosis plus counseling, counseling alone, and hypnosis alone in a community service smoking withdrawal program. *J. Cunsult. Chin. Psychol.,* 31:498-505, 1975.

Schwartz, J. L.: A critical review and evaluation of smoking control methods. *Public Health Rep.,* 84:483-506, 1969.

Spiegel, H.: A single-treatment method to stop smoking *using* ancillary self-hypnosis. *Int. J. Chin. Exp. Hypn., XVIII:* 235-250, 1970(a).

Spiegel, H.: A single-treatment method to stop smoking using ancillary self-hypnosis: final remarks in response to the discussants. *Int. J. Clin. Exp. Hypn.,* XVIII:268-269, 1970(b).

Spiegel, H., Fleiss, J. L., Bridger, A. A., and Aronson, M.: Hypnotizability and mental health. In: S. Arieti and G. Chrzanowski (Eds.), *New Dimensions in Psychiatry: A World View.* New York: John Wiley & Sons, 1975, 342-356.

West, L. J. and Deckert, G. H.: Dangers of hypnosis. *JAMA,* 192:9-12, 1965.

Wright, M. E.: A single-treatment method to stop smoking using ancillary self-hypnosis: discussion. *Int. J. Chin. Exp. Hypn.,* XVIII:261-267, 1970.

AUTHOR

Louis Jolyon West, M. D.
Professor and Chairman
Department of Psychiatry and Biobehavioral Sciences
Director, The Neuropsychiatric Institute
University of California
760 Westwood Plaza
Los Angeles, California 90024.

Discussant for Section on Behavioral Change

Robert P. Libeman, M.D.

Behavioral Change:

A Stopped Smoker is not a Non-Smoker

The most important outcome of the presentations on Behavioral Change from the UCLA Research Conference on Smoking Behavior is the realization that cessation of smoking is far different than remaining a non-smoker. Mark Twain humorously highlighted this distinction between temporary and permanent discontinuation of smoking when he said, "It's the easiest thing in the world to stop smoking. I should know because I've stopped over 1000 times." Schwartz (1977) and Lichtenstein (1977) have shown how most efforts to get smokers to quit have a very high success rate, if the criterion of success is temporary abstinence. Whether one uses a "psychotherapy robot" (West, 1977) or an attention-placebo treatment, the likelihood of a smoker quitting is very high if the treatment is delivered in a convincing fashion. The challenge to those who design withdrawal treatments for smoking addicts, then, is to develop methods for maintaining abstinence once withdrawal has been accomplished.

One approach to designing treatments with more durable outcomes is to analyze the experiences of smokers who have quit and relapsed and compare their process of relapse with the successful experiences of smokers who quit and remain abstinent. Lichtenstein (1977) recommends a behavioral-empirical analysis of the relapse phenomenon. What factors contribute to relapse? There are a host of possibilities which we can speculate about, including the return of the temporarily abstinent ex-smoker to a family and/or work situation where others still smoke (modeling effects), and the gradually more sparse receipt of social reinforcement from significant others as time passes from the point of withdrawal and people take the ex-smoker's abstinence for granted.

In a recent study of 48 individuals who attempted to stop smoking on their own, a comparison was made between 24 people who were successful in quitting cigarettes and remained abstinent for at least four months and 24 others who were unsuccessful in staying

off cigarettes (Perri et al., 1977). The evaluation was done by interview with a focus on the subjects' experiences during the process of quitting cigarettes. The successful ex-smokers could be distinguished 'from those who failed by their (1) persisting longer in their efforts at quitting; (2) using a greater variety of techniques and employing them more ferquently in their daily life; (3) obtaining more 'social reinforcement for their efforts at abstinence from significant others; and (4) giving themselves more positive self-reinforcement for efforts at abstaining.

The results of this interview study plus the results of studies cited by Schwartz (1977), Lichtenstein (1977), and Tongas (1977) underscore the importance of duration of treatment efforts, multiple techniques, self-control methods, and social support or reinforcement from one's social world in maintaining abstinence. Treatment efforts should target both a decrease and cessation in smoking behaviors and an increase or strengthening of behaviors that can be linked to abstinence. Aversive or satiation methods can be helpful in decreasing the likelihood of smoking and, perhaps, substances such as nicotine chewing gum can be useful in weakening the craving and urge to smoke. With narcotic addicts, similar tendencies to relapse have been successfully reduced by administration of a narcotic antagonist, naltrexone, which blocks the euphoric and other pleasant effects of heroin and reduces the craving for heroin without producing addiction itself (Callahan et al., 1976). By temporarily being freed of urges to use drugs, the addict is accessible and responsive to treatment and rehabilitation efforts aimed at increasing and strengthening his adaptive social and vocational skills. In a like manner, cigarette addicts must be exposed to treatment interventions which not only diminish craving but also provide support and reinforcement for continued abstinence.

The following treatment strategies, taken from the behavior modification literature, can be helpful guidelines for maintaining abstinence in individuals who have stopped smoking:

1. Establish reinforcement contingencies in "real world" settings which will support the desired behavior; or treat only those behaviors that will continue to be reinforced after treatment.

2. Modify the behavior in the natural environment using the significant others in that environment to manage the treatment program.

3. Structure the treatment setting to approximate the "real world" outside and transfer relevant aspects of the treatment setting into the "real world". (Stimulus control.)

4. Use verbal instructions early in the treatment to control the behavior of interest (instructional control).

5. Teach the patients self-management methods, including self-monitoring, self-evaluation, self-instructions, and self-reinforcement procedures in reaching their own behavioral goals.

6. Use intermittant and delayed schedules of reinforcement as the treatment proceeds.

7. Gradually fade out the tangible, material or artificial elements of the treatment, eventually relying solely on the more naturally occurring social and symbolic reinforcers.

8. Pair praise, approval and other social reinforcers with progress In treatment and with the offset of aversive stimuli. In this manner, naturally occurring social reinforcers will be better established to maintain behavioral gains.

9. Use over-training in both the application of aversive consequences to the undesirable behavior and in positive practice of the desired behavior.

10. Involve the patient in setting the goals of treatment and in choosing from alternative treatment methods.

The excellent follow-up results reported by Tongas (1977) In a multiple component treatment program for smoking may, In part, be related to his incorporating some of the above strategies into his approach. Tongas involved spouses In the orientation sessions and found that they did later provide support to the clients for abstaining; used a "buddy system" of mutual support for abstinence which harnessed natural contingencies of reinforcement in the real world; paired praise for progress with the end of the aversion phase of each group session; utilized Instructional control by having an orientation session; and faded the treatment session gradually over a 12 month period.

It is likely that incorporation of the above strategies for facilitating durability of response to treatment will make non-smoking status a clear possibility for even hard-core smokers who have relapsed many times. The same awareness of the differences between methods for acutely changing behavior vs. maintaining behavior change has led workers to develop more effective treatment programs for alcoholics (Nathan), chronically psychotic mental patients (Liberman et al., 1976), and obese individuals (Mahoney and Mahoney, 1976).

Another group of people who need to have their behavioral repertoire strengthened and made durable are those working in education, politics, and the law with the goal of preventing cigarette addiction (Gritz, 1977). There is a need to increase the constituency of individuals trying to bring about prevention of smoking on a larger scale than the clinic permits. Here, also, behavioral principles may have something to offer. There are a number of recently published examples of how social and operant learning interventions have been utilized to increase conservation of energy in large populations, increase the use of public transportation, decrease littering in public parks, widen the participation of poor people in community activities, and influencing citizens to purchase healthier foods in supermarkets (Foxx and Hake, 1977; Burgess et al., 1971; Geller et al., 1973; Everett et al., 1974). These preliminary forays by behavior modifiers into larger scale domains suggests that the anti-smoking constituency can use learning principles and systematic observation to design programs which can have an impact upon the social, legal, political, educational, and economic influences on smoking.

REFERENCES

Burgess, R. L., Clark, R. N. and Hendee, J. C. An experimental analysis of anti-litter procedures. *Journal* of *Applied Behavioral Analysis* 4:71-75, 1971.

Callahan, E., Rawson, R., Glazer, M., McCleave, B., and Arias, R. Comparison of Two Naltrexone Treatment Programs: Naltrexone Alone Vs. Naltrexone Plus Behavior Therapy. In D. Julius and P. Renault (Eds.) *Narcotic Antagonists: Naltrexone Progress Report* NIDA Research Monograph 9. Rockville, MD: Dept. HEW, 150-157, 1976.

Everett, P. B., Hayward, S. C., and Meyers, A. W. The effects of .a token reinforcement procedure on bus ridership. *Journal of Applied Behavior Analysis* 7:1-9, 1974.

Foxx, R. M. and Hake, D. F. Gasoline conservation: A procedure for measuring and reducing the driving of college students. *Journal of Applied Behavior Analysis* 10:61-74, 1977.

Geller, E. S., Farris, J. C., and Post, D. S. Prompting a consumer behavior for pollution control. *Journal of Applied Behavior Analysis* 6:367-376, 1973.

Gritz, E. R. Smoking: the prevention of onset. Presented at the UCLA Research Conference on Smoking Behavior, June 24 and 25, 1977.

Liberman, R. P., McCann, M. J., and Wallace, C. J. Generalization of behavior therapy with psychotics. *British Journal of Psychiatry* 129:490-496, 1976.

Lichtenstein, E. Current status and future directions for behavior modification of smoking behavior. Presented at the UCLA Research Conference on Smoking Behavior, June 24 and 25, 1977.

Mahoney, M. J., and Mahoney, K. *Permanent Weight Control New* York: Norton, 1976.

Nathan, P. E. Alcoholism. In H. Leitenberg (Ed.) *Handbook of Behavior Modification and Behavior Therapy* Englewood Cliffs, N.J. : Prentice-Hall, 3-44, 1976.

Perri, M. G., Richards, C. S., and Schultheis, K. R. Behavioral self-control and smoking reduction: a study of self-initiated attempts to reduce smoking. *Behavior Therapy* 8:360-365, 1977.

Schwartz, J. C. A review of smoking cures. Presented at the UCLA Research Conference on Smoking Behavior, June 24 and 25, 1977.

Tongas, P. N. The effects of maintenance treatment on long-term cessation of smoking. Presented at the UCLA Research Conference on Smoking Behavior, June 24 and 25, 1977.

West, L. J. Hypnosis as a treatment for smoking. Presented at the UCLA Research Conference on Smoking Behavior, June 24 and 25, 1977.

AUTHOR

Robert P. Liberman, M.D.
Camarillo-Neuropsychiatric Institute
(UCLA) Research Center
Box A
Camarillo, California 93010

DISCUSSION

Discussion was initiated by a question concerning the significance of demographic variables such as education and socioeconomic status for smoking prevention and cessation. Dr. Gritz mentioned that in the study cited in her paper (Graham and Gibson, 1971)) ex-smokers and non-smokers were distinguished from recidivists and those who had never stopped smoking by higher professional and educational level. The comment was made that prevention programs have not been successful among the lower socioeconomic classes in general.. Roth the reluctance to delay gratification in the face of constant environmental stress, and attribution of control over one's fate to external sources contribute to a resistance to giving up smoking. The successful ex-smoker experiences a much greater feeling of self-control than those who are unable to quit. Dr. Liberman summed up this point by suggesting that there may be a correlation between social class and educational background and the degree to which the interpersonal surroundings can provide verbal support and reinforcement for abstinence efforts. The educated person may also be more successful at verbal introspection and self-reinforcement,

Another way to approach prevention is to create a new status symbol or "in thing" personified by the non- or ex-smoker, "the winner". Thus, peer pressure and teenage modeling might be used to good advantage in mimicking non-smoking as the desired behavior. Revising the norm from smoking to non-smoking is not an instantaneous process; we are still on the initial segment of a rapidly accelerating curve. Change will be evident when people become ashamed to smoke in public and when the smoker is socially-stigmatized. The work of vocal anti-smoking organizations, such as ASH and GASP, is facilitating the process of change. In addition, the creation of long-term maintenance organizations similar to Weight Watchers and Alcoholics Anonymous could provide 'half-way houses" for ex-smokers until they truly feel themselves to be non-smokers, a process which may take many months. Thus, society can take a much stronger role in prevention by popularizing non-smoking, and psychologically supporting ex-smokers.

Ellen R. Gritz, Ph.D.

Behavioral Change: Session Overview

Ellen R Gritz, PhD.

The achievement of behavioral change is obviously not a goal unique to the smoker; it is shared by those fighting drug-related dependencies such as alcoholism and heroin addiction, and more purely behavioral problems such as obesity. Some collaboration, in terms of sharing conceptual frameworks and treatment methodologies, has occurred among those working in the different areas, yet not in any very systematic fashion. Although the pharmacological basis of smoking has been extensively investigated, no effective chemotherapy has evolved from the nicotine hypothesis. Behavioral treatments have borrowed heavily from operant and social learning theories; in general, resultant success rates are disappointingly low. In the past five years, however, effective procedures for obtaining smoking abstinence have emerged from carefully designed research, and we have some good guesses for long-ten maintenance. The general discussion centered around three key issues: the evaluation of present work; the variables most likely to boost success rates; and suggestions for future directions for treatment and research.

In evaluating the effectiveness of smoking cures, there is a need to decide what constitutes a good result, or the net best effect. Total cost, number of hours of treatment, professional skill of the therapist, and per cent of clients abstinent at the cessation of treatment and over time must all be considered in such a calculation.

For any given treatment, estimates of each variable are not consistent from one study to another. Concentrating on achieved abstinence as the most important variable, it is evident that different investigators purportedly using the same methodology report markedly different results. Can we attribute this to differences among clients, or among treatment personnel? How does the laboratory experience differ from the hospital clinic? Dr. Lichtenstein and his colleagues have reported consistently higher success rates with rapid smoking than other investigators. Dr. Tongas noted that in the replication (presently in progress) of the research reported at this conference, cessation rate is somewhat lower than before, and treatment condition is irrelevant to success. Objective evaluations of hypnotherapy are acutely needed; problems of control groups are particularly severe. Systematic long-term follow-up is also lacking for many of the studies in the literature. These are examples of the state of

the art; the most profitable direction(s) to pursue need to be chosen.

Ultimately, evaluation of treatment success should be performed by an uninvolved party. For the university clinical researcher, controlled designs provide objectivity. Commercial treatment enterprises have been loathe to permit external evaluation while claiming higher success rates than in the literature. The American Cancer Society has recently made a grant to the Center for Health Education in San Francisco to evaluate several private enterprises, if permitted. Such evaluations will ultimately provide an objective source of information for the public and the health professional. Perhaps an organization such as Consumer Reports could then be used as a broadcast medium to reach the smoker in search of a cure.

There are a host of loosely related factors which are capable of boosting success rates. Personality attributes of the smoker have been widely discussed and researched; motivation to quit is primary and must be internal, and not external in origin. Screening devices, such as those selecting for high motivation, or increased probability of treatment effectiveness (e.g., hypnotic suggestibility) can markedly alter success rates. Level of enthusiasm of the project staff is a variable in the treatment situation which might account for the differing success rates among universities or clinics using the same methodology. Factors indirectly involved in treatment are the cooperation of spouses, family members and peers (working environment, best friends). These influences are not often tapped, but have proven important, especially in the maintenance period.

Challenging suggestions exist for future research and treatment. It may be necessary and profitable to develop a whole host of cessation methodologies geared to specific sub-populations ("different strokes for different folks"). There are scattered reports of initially high success rates for almost every type of treatment. Different methodologies available in various places (library, school, office, hospital, community health center) and utilization of the media (newspaper, magazine, radio, television, film) might serve to reach all segments of the population. An interesting example of a new approach being pioneered by the American Health Foundation is a structured intervention program in industry. Workers are approached multiple times with progressively more intense contacts, so that only the hard-core resistant smokers need be treated with the most powerful methods. Methodology should ideally be as simple as possible.

One of the "folks" virtually ignored in the cessation literature is women; their abstention rates have been consistently below that of men in the same treatment groups. This is true across type of treatment. Why is this? Are treatments designed by men for men? Do we need to develop new treatments aimed specifically at women?

The rate of recidivism among ex-smokers is distressingly high. One of the most important directions for future research and treatment is to analyze and lower this rate. Specifically, studies of the

situations in which smoking is resumed, and of the internal stimuli governing relapse are indicated. Hand in hand with this goes the need for work in the area of long-term maintenance. The' paucity of studies has multiple causation: time pressures in dissertation research, a stress on rapid publication, and lack of interest of commercial treatment centers. At present, the federal government and private prepaid health plans have a real investment in supporting and carrying out research and clinical trials in this area.

The pieces of the puzzle are beginning to fit. The most effective treatment strategies and the means to maintain abstinent behavior are. within our grasp. Social forces are rallying to support the rights of non-smokers, to prevent new smokers from entering the ranks, and to make the recent ex-smoker into a permanent success, a "winner".

National Institute on Drug Abuse

Research monograph series

Monographs can be ordered from either the U.S. Government Printing Office (GPO) or from the National Technical Information Service (NTIS) at the addresses below. NTIS prices given are for papercopy and are subject to change. Microfiche copies at $3 are also available from NTIS.

GPO	NTIS
Superintendent of Documents	National Tech. Info. Service
U.S. Government Printing Office	U.S. Department of Commerce
Washington, D.C. 20402	Springfield, Virginia 22161

1 FINDINGS OF DRUG ABUSE RESEARCH. *An annotated bibliography* of *NIMH- and NIDA-supported extramural grant research, 1964-74. Volume 1, 384 pp., Volume 2, 377 pp.* Not available from NTIS
Vol.1: GPO Stock #017-024-0467 $7; Vo1.2: #017-024-0466-9 $5.05

2 OPERATIONAL DEFINITIONS IN SOCIO-BEHAVIORAL DRUG USE RESEARCH 1975. Jack Elinson, Ph.D., and David Nurco, Ph.D., editors. *Task Force articles proposing consensual definitions* of *concepts and terms used in psychosocial research to achieve operational comparability. 167 pp.*
GPO Stock #017-024-0048-4-7 $2.50 NTIS PB #246 338 $6.75

3 AMINERGIC HYPOTHESES OF BEHAVIOR: REALITY OR CLICHE? Bruce J. Bernard, Ph.D., editor. *Articles examining the relation of the brain monoamines to a range of animal and human behaviors. 149 pp.*
GPO Stock #017-024-0048-6-3 $2.25 NTIS PB #246 687 $6.25

4 NARCOTIC ANTAGONISTS: THE SEARCH FOR LONG-ACTING PREPARATIONS. Robert Willette, Ph.D., editor. *Articles reporting current alternative inserted sustained-release or long-acting drug devices.*
GPO Stock #017-024-00488-0 $1.10 NTIS PB #247 096 $4

5 YOUNG MEN AND DRUGS: A NATIONWIDE SURVEY. John A. O'Donnell, Ph.D., et al. *Report* of *a national survey of drug use by men 20-30 years old in 1974-5. 144 pp.*
GPO Stock #017-024-00511-8 22.25 NTIS PB #247 446 46.75

6 EFFECTS OF LABELING THE "DRUG ABUSER": AN INQUIRY. Jay R. Williams, Ph.D. *Analysis and review* of *the literature examining effects* of *drug use apprehension or arrest on the adolescent. 39 PP.*
GPO Stock #017-024-00512-6 $1.05 NTIS PB #249 092 $4

7 CANNABINOID ASSAYS IN HUMANS. Robert Willette, Ph.D., editor. *Articles describing current developments in methods* for *measuring cannabinoid levels in the human body by immunoassay, liquid and dual column chromatography and mass spectroscopy techniques, 120 pp.*
GPO Stock #017-024-00510-0 $1.95 NTIS PB #251 905 $6

8 RX:3 TIMES/WK LAAM - METHADONE ALTERNATIVE. Jack Blaine, M.D., and Pierre Renault, M.D., editors. *Comprehensive summary* of *development of LAAM (Levo-alpha-acetyl methadol), a new drug for treatment of narcotic addiction. 127 pp.*
Not available from GPO NTIS PB #253 763 $6

9 NARCOTIC ANTAGONISTS: NALTREXDNE. Demetrios Julius, M.D., and Pierre Renault, M.D., editors. *Progress report* of *development, pre-clinical and clinical studies of naltrexone, a new drug for treatment of narcotic addiction. 182 pp.*
GPO Stock #017-024-00521-5 $2.55 NTIS PB #255 833 $7.50

10 EPIDFMIOLOGY OF DRUG ABUSE: CURRENT ISSUES. Louise G. Richards, Ph.D., and Louise B. Blevens, editors. *Conference Proceedings. Examination of methodological problems in surveys and data collection. 259 pp.*
GPO Stock #017-024-00571-1 $2.60 NTIS PB #266 691 $9

11 DRUGS AND DRIVING. Robert Willette, Ph.D., editor. *State-of-the art review of current research on the effects of different drugs on performance impairment, particularly on driving. 137 pp.*
GPO Stock #017-024-00567-2 $1.70 NTIS PB #269 602 $6.75

12 PSYCHODYNAMICS OF DRUG DEPENDENCE. Jack D. Blaine, M.D., and Demetrios A. Julius, M.D., editors. *A pioneering collection* of papers to *discover the part played by individual psychodynamics in drug dependence.*
GPO Stock #017-024-00642-4 $2.75 NTIS # to be assigned

13 COCAINE: 1977. Robert C. Petersen, Ph.D., and Richard C. Stillman, M.D., editors. *A series* of *reports developing a picture* of *the extent and limits of current knowledge* of *the drug, its use and misuse. 223 pp.*
GPO Stock #017-024-00592-4 $3 NTIS PB #269 175 "7.75

14 MARIHUANA RESEARCH FINDINGS: 1976. Robert C. Petersen, Ph.D., editor. *Technical papers on epidemiology, chemistry and metabolism, toxicological and pharmacological effects, learned and unlearned behavior, genetic and immune system effects, and therapeutic aspects of marihuana use. 251 pp.*
GPO Stock #017-024-00622-0 $3 NTIS PB # 271 279 $9

15 REVIEW OF INHALANTS: EUPHORIA TO DYSFUNCTION. Charles Wm. Sharp, Ph.D., and Mary Lee Brehm, Ph.D., editors. *A broad review* of *inhalant abuse, including* sociocultural, *behavioral, clinical, pharmacological, and toxicological aspects. Extensive bibliography.* In Press

16 THE EPIDEMIOLOGY OF HEROIN AND OTHER NARCOTICS. Joan Dunne Rittenhouse, Ph.D., editor. *Task Force report on measurement of heroin-narcotic use, gaps in knowledge and how to address them, improved research technologies, and research implications.* In Press

* U.S. GOVERNMENT PRINTING OFFICE: 1978--274-475/1362

DEPARTMENT OF
HEALTH, EDUCATION, AND WELFARE
PUBLIC HEALTH SERVICE
ALCOHOL, DRUG ABUSE, AND
MENTAL HEALTH ADMINISTRATION
5600 FISHERS LANE
ROCKVILLE MARY LAND 20857

OFFICIAL BUSINESS
Penalty for private use, $300

POSTAGE AND FEES PAID
U.S. DEPARTMENT OF H.E.W.
HEW 396

THIRD CLASS
BLK. RATE

NOTICE OF MAILING CHANGE

☐ Check here if you wish to discontinue receiving this type of publication.

☐ Check here if your address has changed and you wish to continue receiving this type of publication. (Be sure to furnish your complete address including zip code.)

Tear off cover with address label still affixed and send to:

Alcohol, Drug Abuse, and Mental Health Administration
Printing and Publications Management Section
5600 Fishers Lane (Rm. 6-105)
Rockville, Maryland 20857

DHEW Publication No. (ADM)78-581
Printed 1978