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CHAPTER 4

Other Conditions and Areas of Research

CONTENTS

SMOKING AND PEPTIC ULCER

Since the publication of the Surgeon General's 1964 Report, three of the continuing prospective mortality * studies (*2, 3, 6, 7*) have provided additional information which confirms the association between cigarette smoking and mortality from peptic ulcer, especially gastric ulcer. The mortality ratios increase with increases in amounts smoked. The tables presented below illustrate the relationships involved. Although Hammond's (*6*) study contained a large number of females, insufficient deaths from peptic ulcer have occurred in cigarette smoking females to provide statistically reliable data. A trend is observable among cigar and/or pipe smokers with regard to increased mortality from gastric ulcer, but the number of deaths is too small for significant conclusions to be drawn.

TABLE 1.—*Death rates and mortality ratios for gastric and duodenal ulcers by specific age groups of male cigarette smokers*

Site	Age 45–64		Age 65–79	
	Death rate	Mortality ratios	Death rate	Mortality ratios
Gastric ulcer	[1] (2) 7	2.95	(7) 26	4.06
Duodenal ulcer	(3) 7	2.86	(21) 31	1.50

[1] Number in parentheses indicates death rate for persons who never smoked regularly.

SOURCE: Hammond, E. C. [table 24(*6*)].

TABLE 2.—*Male death rates and mortality ratios for gastric and duodenal ulcers by specific age groups for current and ex-smokers of cigarettes only*

Site	Current cigarette smokers only				Ex-cigarette smokers only
	Death rate			Mortality ratio (total)	Mortality ratio (total)
	55–64	65–74	75–84		
Gastric ulcer	[1] (2) 7	(5) 17	(—)26	4.13	2.74
Duodenal ulcer	(4) 8	(10) 29	(37) 122	2.98	2.13

[1] Number in parentheses indicates death rate for persons who have never smoked.

SOURCE: U.S. veterans study [app. table A (*7*)].

* All death rates throughout this chapter are per 100,000 population, unless otherwise indicated.

TABLE 3.—*Peptic ulcer death rates by type and amount smoked, in males* [1]

Nonsmokers	All smokers	Cigarette smokers				Given up cigarette smoking	Mixed smokers	Pipe or cigar
		All amounts	1–14	15–24	25+			
0	13	13	2	18	19	12	12	10

[1] Includes gastric and/or duodenal ulcers.

SOURCE: Study of British physicians [tables 23 and 24 (*3*)].

A recent survey (*13*), based on a national sample of 42,000 household interviews, shows that the prevalence of peptic ulcer is almost 100 percent greater in male cigarette smokers and over 50 percent higher in females who smoke cigarettes as compared to those males and females who had never smoked. Hammond's data (*5*) shows twice the number of cigarette smokers reporting the occurrence of peptic ulcer over a 2-year follow-up period as contrasted to nonsmokers. This also increases with increases in the amount smoked.

Several small retrospective clinical studies (*4*, *8*, *12*) have shown significantly more smokers and less nonsmokers in their peptic ulcer patients as compared to control groups. Doll (*1*) reviewed various prospective studies on gastric ulcer therapy regimes, such as: diet—bland, normal, high and low fat; milk drips with alkali; drugs; and advice to stop smoking. The best results were obtained in patients who stopped or cut down on their smoking habits. The Surgeon General's 1964 Report (*14*) points out the conflicting literature concerning the effects of smoking on gastric secretion and motility. Lee (*10*), in a small series of peptic ulcer patients and controls, showed that after smoking, 74 percent of patients and 58 percent of controls had a significant rise in free gastric acidity. Those subjects with initially normal or hyperacidity had the greatest response, whereas, of those with initial hypoacidity only 28 percent had an increase in gastric acidity. Five of nine controls, smoking a non-nicotine cigarette preparation, also had a rise in gastric acidity, perhaps due to factors in smoke other than nicotine or to oral stimulation.

RÉSUMÉ

Cigarette smoking is shown to be associated with peptic ulcer. This relationship is greater for gastric than duodenal ulcer and is proportional to the amount smoked. The etiology of the peptic ulcer diathesis is still unknown. Smoking is a definite risk factor in peptic ulcer mortality. It may also be a factor in the delay in healing of a gastric ulcer. More research is needed on the physiological effect of smoking on the gastrointestinal tract.

Tobacco Amblyopia

Recent evidence points to the tobacco and/or alcohol amblyopias as being manifestations of nutritional amblyopia (*4, 7, 8, 17, 25, 26*). Various deficiencies in factors of the vitamin B complex have been implicated (*4, 6, 7, 10, 17, 25, 26*).

A new theory that chronic low vitamin B_{12} levels potentiate the toxic effects of cyanide in tobacco has recently been expounded (*8, 10, 22, 23*).

The anatomical lesion in amblyopia seems to be a demyelinization of the optic pathways, particularly in the papillomacular bundle (*10, 17, 25*).

In view of the fact that cyanide is neurotoxic, more research is needed in this area to further elucidate its association with this disease entity.

Other Diseases

Several studies have hypothesized that Leber's optic atrophy, which also is attributed to a demyelinization process in optic pathways, may be associated with a defect in cyanide detoxification, which is aggravated by the cyanide in tobacco smoke (*1, 27*).

Visual Acuity

The Surgeon General's 1964 Report, and others, cite evidence of increased levels of carboxyhemoglobin in smokers (*20, 24*), due to the carbon monoxide content in tobacco smoke. It has been suggested that a decrease in nighttime visual discrimination in smokers is related to this increase in carboxyhemoglobin levels (*9, 15, 16, 19*). It may also possibly be due to the relative anoxia produced by the carbon monoxide inhalation from tobacco smoke. A value of only 5 percent carboxyhemoglobin saturation, not uncommon in smokers, creates a physiological state of anoxia equivalent to being at an elevation of 8,000 feet, with an arterial O_2 saturation of only 91 percent (*15, 16*).

Résumé

It is suggested that tobacco amblyopia is but a manifestation of nutritional amblyopia, which is aggravated by tobacco smoking. More research is needed on the toxicity of tobacco smoke, with special concern for the cyanide component. Experiments have shown a visual discrimination deficit, possibly related to the carbon monoxide content of tobacco smoke. Further work is needed in this area in order to ascertain any clinical consequences.

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SMOKING AND CIRRHOSIS OF THE LIVER

Increased mortality of smokers from cirrhosis of the liver is found in the prospective studies. This has generally been thought to be largely secondary to an association between smoking and heavy alcohol consumption. Published data are inadequate to test this interpretation.

The three prospective studies (*1*, *2*, *3*, *4*) all show increased death rates and mortality ratios from cirrhosis of the liver in cigarette smokers.

TABLE 1.—*Mortality ratios and death rates for liver cirrhosis by sex and specific age groups*

	45–64		65–79	
	Female	Male	Female	Male
Mortality ratio	2.16	2.06	[1] 1.40	1.97
Death rate	[2](5)10	[2](9)19	(10)14	(16)31

[1] Calculated from app. table 19 (*3*).
[2] Numbers in parentheses indicate death rates for persons who have never smoked regularly.

SOURCE: Hammond, E. C. [tables 24, 26, and app. table 19 (*3*)].

TABLE 2.—*Male mortality ratios and death rates for liver cirrhosis by age and amount smoked, in U.S. veterans*

	Current smokers of cigarettes only				
	0	1–9	10–20	21–39	40+
Mortality ratio (total)	1.00	2.72	3.15	3.61	5.50
Death rate:					
Age 45 to 54	9	--------	7	7	162
Age 55 to 64	15	12	35	44	46
Age 65 to 74	16	74	57	57	87
Age 75 to 84	53	--------	--------	--------	--------

SOURCE: U.S. veterans study [app. table A (*4*)].

Doll and Hill present their data with respect to cirrhosis of the liver and alcoholism combined. See table 3.

TABLE 3.—*Male death rates for liver cirrhosis by type and amount smoked, in British physicians*

	Non-smokers	All smokers	Cigarette smokers	Number of cigarettes				Given up smoking	Mixed smokers	Pipe or cigar
				1–14	15–24	25+	All amounts			
Cirrhosis of liver and alcoholism	0	11	12	5	8	43	15	3	11	5

SOURCE: Study of British physicians, [tables 21 and 22 (*1*)].

The Surgeon General's 1964 Report points out the association between heavy smoking and excessive alcohol intake. In view of the fact that "The increased death rate from cirrhosis among smokers may reflect the consumption of alcohol and associated nutritional deficiencies rather than the effect of cigarette smoking" (*5*), further research is needed to elucidate the association between smoking and cirrhosis of the liver.

EFFECTS OF SMOKING DURING PREGNANCY

The current new literature on pregnancy and smoking supports the Surgeon General's findings that there are a greater number of "low birth weight" babies and premature babies as defined by weight alone (2,500 g.) in those women who smoke during their pregnancy (*6*, *9*, *18*, *20*, *21*, *24–26*, *28*, *30–32*, *36*). Furthermore, this decreased weight has been shown to be consistent in each trimester (*28*, *31*, *32*, *34*, *36*) and is proportional to the amount smoked during pregnancy (*5*, *24–26*, *30–32*, *34*, *36*).

There are many factors which affect the outcome of pregnancy. These include constitutional, pathobiological and psychological factors. Multiple-regression analyses of these various factors have shown smoking to be a significant negative independent variable with respect to birth weight (*1*, *5*, *27*).

Smoking has been linked to increased incidence of abortions and/or stillbirths (*6*, *25*, *26*, *28*, *30*, *36*), premature rupture of membrances (*30–32*) and decreased male/female birth ratios (*11*, *25*, *26*); however, other studies do not support these findings (*7*, *9*, *24*, *30–32*).

The significance of low birth weight and prematurity in regard to increased fetal and infant mortality, has not been clearly demonstrated. Most studies show no increased mortality (*9*, *31*, *32*). However, Yerushalmy (*34*) and Underwood (*32*), point out that although the overall mortality is the same between infants of smoking versus nonsmoking mothers, premature babies (as defined by birth weight of less than 2,500 g.) of smoking mothers have decreased mortality. Other studies show a slight but significant increase in fetal mortality for mothers who smoke (*6*, *8*). MacMahon (*19*) shows that rather than increasing the proportion of low birth weight babies, smoking actually causes a shift to the left in the entire weight distribution (fig. 1).

Jansson (*15*) in his study states: "Thus, in the absence of other complications, smoking mothers seem to make a proportionally greater contribution to infants in the weight group just below 2,500 g. where the prognosis is better."

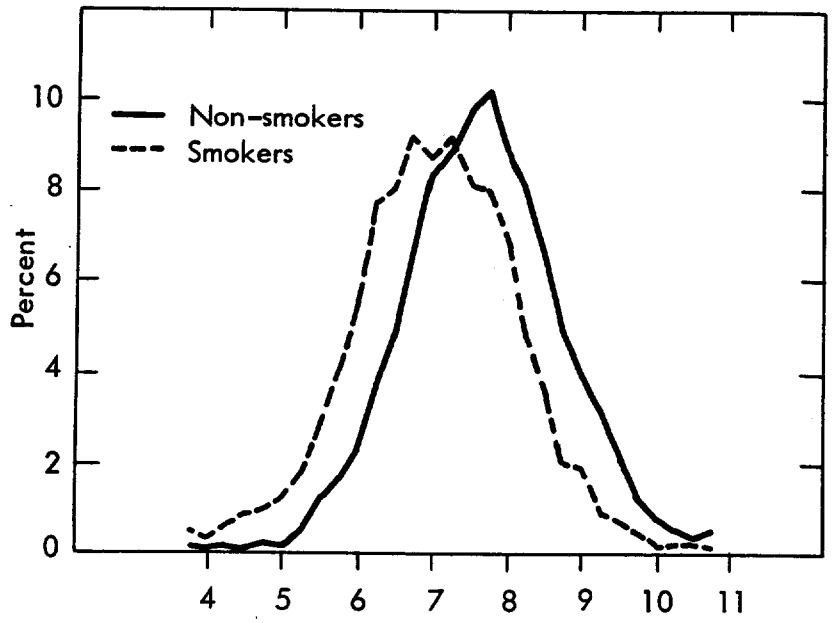

FIGURE 1.—Percentage distribution by birth weight [1] of infants of mothers who did not smoke during pregnancy and of those who smoked one pack or more per day.

Source: MacMahon, B., et al. [fig. 1 (*19*)].

Steele (*29*) suggests that smoking is associated with sudden unexpected deaths in infancy. The relationship of smoking in mothers to increased fetal morbidity, either perinatally or after long-term follow-up, has not been adequately probed and is a major area for future research.

Some studies show a relationship between smoking and decreased gestational age (*6*, *8*, *32*); others do not (*1*, *27*).

Gestational age probably is a better indicator of fetal prematurity than birth weight. Therefore, it may better reflect perinatal risk. Yet even in the studies showing a correlation to smoking, this relationship is less marked than that to birth weight (*6*, *8*, *24*, *32*). This may be due to the relative difficulty in determining the last menstrual period accurately and therefore the true gestational age.

The mechanisms by which smoking affects pregnancy have not been elucidated. Events influencing decreased fetal birth weight have been attributed to several factors:

1. Placental vasoconstriction due to nicotine. No direct evidence of this exists at the present time. The effects of smoking on uterine blood flow are being conducted in animal experimentation (*2*).

[1] Birth weight (scale in pounds; intervals of 4 ozs.)

2. Increased carbon monoxide in cord blood (*12*).

3. Decreased carbonic anhydrase activity due to increase in cord carbon monoxide (*20*).

4. A postulated direct effect on the fetus of some toxic agent in cigarette smoke.

5. Decreased caloric intake due to decrease in appetite of smokers (*10*). Several studies have shown that there is no difference in weight gain during pregnancy between smokers and nonsmokers (*28, 30, 36*). Since smoking in general, does decrease appetite, it might be well to consider a difference in the type and/or distribution of caloric intake between smokers and nonsmokers.

Kumar (*17*) has shown an increase in human uterine activity after smoking, both in frequency and magnitude of contractions. However, these findings were not observable in every patient. There was no significant effect of nicotine on myometrial strips *in vitro* from pregnant human uteri.

King and Becker (*4, 16*) have done experimental work with nicotine on pregnant rats. High concentrations of nicotine had greater toxic effects on pregnant rats than controls. The offspring were lighter in weight and survived less well than controls.

Résumé

Clearly, more research is needed to elucidate the significance of the relationship of smoking in pregnancy and low birth weight. Additional long-range morbidity studies are needed, as well as studies on the effect of smoking on uterine activity and placental blood flow.

Smoking does have an effect on the outcome of pregnancy. However, it is not known whether this effect is deleterious or not.

Until such evidence is presented so as to clearly define the role of smoking in pregnancy, it is more prudent at this time to advise pregnant women to stop or decrease their cigarette-smoking practices.

SMOKING AND ACCIDENTS

The most obvious contribution of smoking to accidents is as a cause of fires. Estimates of the proportion of fire loss due to "smoking and matches" (includes fires attributed to careless smoking and the careless use of matches and lighters by smokers; does not include misuse of matches by children) vary from 19 percent to 25 percent. The

National Fire Protection Association gives "smoking and matches" as a reported cause of fire in various buildings for 1965 as follows (*3*):

	Percent
Apartments	26
Boarding and rooming houses	30
Dormitories, etc	24
Dwellings (1- and 2-family)	19
Hospitals	21
Hotels, seasonal	24
Hotels, year round	35
Motels	20
Nursing	25

In 1965 there were 163,900 fires linked to smoking or the matches used in smoking with a concomitant property loss of $80,400,000—in 1964, there were 159,400 fires and a property loss of $79,500,000 (*5*). The statistics on the number of deaths attributed to those fires are not available, but it is estimated that 1,800 people per year will die due to fires caused by smoking and matches (*7*).

Smoking has been shown to cause decreased visual discrimination especially under conditions of low illumination (*4*). This could have serious implications with respect to night-time driving.

Several studies (*1*, *2*, *6*) have indicated an association between smoking and traffic and industrial accidents, but the evidence is insufficient at this time to draw any significant conclusions. More research is needed in this area.

PSYCHOSOCIAL ASPECTS OF SMOKING

There has been a sharp increase in the attention devoted to behavioral research since the Surgeon General's 1964 Report. A number of new concepts have been developed and more sophisticated multivariate approaches are being used. However, because of the recency of these studies, very little in the way of findings has been published on which firm conclusions may be based.

One of the byproducts of the Surgeon General's 1964 Report has been its stimulation of more research in all areas of smoking, including the psychosocial. Much research will soon be completed but has not yet been reported in the literature.

Three behavioral science conferences have been held since the Surgeon General's 1964 Report. The content of these conferences are either in print (*9*) or will shortly be published (*11*, *21*). These conferences dealt with many different studies and research findings, theories, methodological criticisms, and discussions on a number of important issues. Among the primary purposes was the development and speeding up of communications among those doing work in the field, pre-

venting duplication and wasted effort, developing better measuring instruments, and providing assistance in conceptualizing new theoretical models or further developing approaches already proposed.

Much prior research in the psychosocial aspects related to smoking, while yielding valuable data and suggestive theory, has been concerned largely with discrete variables or attributes and has looked for gross differences between smokers and nonsmokers. Since it is unlikely that such research will discover that either group possesses an attribute that is unique to it other than the behavior of smoking, the ability of any single attribute to differentiate between these two populations is bound to be limited. It is because of this that a number of investigators have turned toward trying to distinguish subgroups of smokers, as well as toward developing more unifying concepts. These efforts are part of the attempt to obtain greater insight into the dynamics of smoking and develop more powerful predictive instruments.

One area that shows conceptual and methodological maturation is that of the study of smoking and personality. Much prior research studied smoking in relation to such concepts as extroversion, introversion, neuroticism, emotional stability, orality, femininity, masculinity, hypochondriasis, psychosomatic symptoms, risk taking and chance orientation, psychopathic tendencies, achievement needs, social approval, relationships to authority, independence, aggression, and the like.

At the 1966 behavior research conference, it was pointed out that a better understanding of the total personality structure must be achieved in order to increase understanding of some of the psychological correlates of smoking. Factor and hierarchical models have much to contribute to this approach (*1*). At the 1967 conference this and other points pertinent to personality research related to smoking were discussed, and a reminder of the utility of multivariate techniques was repeated (*20*). Toward this end these investigators are now studying university students, seeking factors in the realm of personality integration such as experience of control, scope of awareness, reality contact, self-insight, temporal perspective, independence, anxiety, and the like. After these factors have been identified they can be used as independent variables in testing hypotheses suggested by other developments, such as the recently developed typology of smokers, illustrating the potential yield from a cross-fertilization of unifying concepts.

Theories which emphasize the role of anxiety in the development of personality and in the understanding of personality dynamics (*7*) provide a unifying frame of reference which, when combined with an understanding of the gratifications derived from smoking, may lead to useful explanations and investigations into smoking behavior. They may also provide some cohesiveness to research on such concepts as

guilt, self-punishment, need to fail, and risk-taking behavior as they relate to the initiation, continuation, or inability to discontinue smoking. Concepts from depth psychology, and ego psychology in particular may additionally illuminate the source and function of some of the apparent inconsistencies among attitudes, beliefs, and behavior noted by various investigators beyond that provided by dissonance theory (*2*).

Another area showing some growth is represented by attempt to distinguish between the different levels of dosage to which smokers expose themselves beyond that indicated by the average number of cigarettes smoked daily. More sophisticated dosage measurements (*18*) obviously have application in epidemiological research. They may also prove useful in psychosocial research. There is the possibility that an interplay exists between the degree and kind of exposure, physiological and psychological processes, and the dynamics, mechanisms, or degree of difficulty involved in achieving long-term cessation of smoking.

Another conceptual development was contained in the proposal, reported at the first of the national behavioral conferences (*16*) and later refined (*15*), of a new way to define smokers—in terms of the smoker's use of cigarettes to help manage affect, i.e., emotions. From the types of smoking identified (habitual smoking, smoking to increase positive affect, to reduce negative affect, and psychologically addictive smoking) and from a theoretical discussion of the dynamics involved in their formation, possibilities exist for the development, testing, and application of theories and techniques for producing cessation either in a clinic or a natural setting. By identifying differences between smokers in the psychological use of cigarettes, the typology makes it possible to develop theories and techniques to reinforce behavior change and to expand knowledge of the dynamics of smoking behavior.

These concepts are undergoing empirical identification and verification at both the national level and in a variety of clinical settings studying behavior change.

In one study (*13*), for example, which compared three methods of aid to people who were trying to give up smoking, efforts were made to assess the subjects' progress, the nature of the change process, and the social-psychological factors which influence the ability to give up smoking and resist resuming. The investigators are analyzing their data from the conceptual base of smoking types as well as from other points of view in an examination of cessation processes.

The smoking typology is also being applied in an analysis of a survey of adults' and adolescents' smoking habits and attitudes in the United Kingdom (*10*). At the recent 1967 behavioral conference results were presented showing the relation between these smoker types and nervous irritation and relaxation smoking scales, wishing and trying to give up smoking, and addiction indicators for both adolescents and adults.

There were other kinds of analyses described in this research which provide the stimulus for further development and testing of theory.

In this country, a parallel set of surveys has been going on which utilized many questions from the above-mentioned survey just as that survey also borrowed from it. Cross-cultural comparisons are thus possible.

The national surveys of adult smoking behavior, beliefs, and attitudes in this country stimulated, and were also based upon, an organizing framework which discussed some dimensions of a model for behavior change (*6*). This framework incorporated the concepts related to the typology of smokers previously mentioned and also leaned heavily on a behavior model developed originally to provide a theoretical base underlying participation in a mass X-ray screening program (*3*, *4*, *12*). Four dimensions of the framework are discussed and postulated as being essential in considering whether smoking behavior change will or will not take place. They are: The motivations for change (e.g., the exemplar role, economics, esthetics, mastery, and others beside the health threat); the perception of the threat (e.g., the awareness of the threat, the acceptance of the importance of the threat, the personal relevance of the threat, and beliefs about the susceptibility of the threat to intervention); the reasons for smoking in terms of affect management, and the potential development and use of alternative psychological mechanisms; and factors supporting or inhibiting continuing reinforcement (e.g., the role of social forces, interpersonal influences, the mass media, the behavior and attitudes of certain key groups, and the general level of acceptability of the behavior).

Backed by the longitudinal data at the national level and subject to multivariate analysis, this conceptual framework can potentially be developed to the point whereby the parts may be related quantitatively and qualitatively to each other and thus afford a more dynamic interpretation of the behavior change under consideration. The possibility also exists for the development of an instrument for the prediction of change, as well as an opportunity for the verification of some prospective findings reported earlier (*14*). These constructs have also since been extended to considerations of the process of either taking up smoking or remaining a nonsmoker (*5*).

In another area of investigation, one project (*19*) is concerned not so much with individual differences but with cultural differences in values, attitudes, and behavior related to smoking among various ethnic groups in the Southwest and has as its main assumption the probable existence of a common core of psychosocial factors operating to produce different motivation patterns among young people socialized in a particular cultural environment.

Another kind of research—that of the controlled experiment manipulating one variable at a time with a number of small samples—

has advantages which were discussed at the 1967 behavior research conference (*8*). The use of such laboratory methods and controls has been shown to be particularly useful in communications research, including the study of factors that affect a person's acceptance and use of health information. More systematic efforts are needed which will relate the content of the message, the form of the message, the kind of medium used, and the characteristics of the communicator to changes in smoking behavior which are also related to the psychosocial nature of the target audience. In particular, emotion-provoking communications need to be studied in relation to various factors that are known to maintain actions, such as public commitment and conformity to group norms.

As in the case with epidemiological investigations, however, it is probable that more prospective research studies combining social-psychological, sociological, and anthropological concepts must be carried out before a better understanding of smoking behavior initiation (or non-initiation), continuation, or change can be achieved.

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