PEPTIC ULCER DISEASE.

PEPTIC ULCER DISEASE

There is little information dealing specifically with the relationship between smoking and peptic ulcer disease in women. The data which are available suggest the same trend toward higher prevalence of peptic ulcer disease among women who smoke as is observed among men who smoke. Table 1, extracted from the 1979 Surgeon General's Report, shows that the prevalence of "peptic ulcer" in female smokers was higher in two out of three studies of women, which showed a twofold or 1.6 fold higher prevalence (7). The one study which failed to demonstrate an increased prevalence was conducted in rural Poland where very few women smoke (only 7 percent) (6). The median ratio of smoking ulcer patients to nonsmoking ulcer patients has been reported to be 1.7 for men (7). Thus, women smokers seem to show greater susceptibility to ulcer disease than do nonsmokers.

The population of women with ulcers contains a greater proportion of smokers than does the group of women without ulcers. Alp et al. performed a retrospective analysis of 638 patients with gastric ulcer, 230 of whom were women (2). There were 1.9 times as many smokers in the group of women ulcer patients as in an age-matched control group. However, even among the ulcer patients, only 39 percent were smokers. In a smaller series of 31 female patients admitted to hospitals with hemorrhage from, or perforation of, gastric or duodenal ulcers, the prevalence of smoking was 26 percent in both ulcer patients (8/31) and controls (8/31) (1).

In a report examining the effect of smoking on healing rates of gastric and duodenal ulcers, Doll et al. studied 92 women with gastric ulcer and 54 women with duodenal ulcer (3). Smoking was 1.6 times more common in women gastric ulcer patients as in controls matched for age and place of residence ($p < 0.01$). There was no significant excess in the proportion of smokers in the group with duodenal ulcer. The effect of smoking on healing rate was reported for men and women grouped together, so no conclusion regarding specific effects on women is possible.

Although some studies of etiological factors in smoking-induced ulcer disease (gastric acid secretion, pancreatic secretion, etc.) have included women, the number of women has been small, or the data from women have not been presented separately.

In summary, the evidence currently available documents an increased prevalence of peptic ulcer disease in women who smoke. No data are available concerning specific effects of smoking in women on gastric acid secretion, gastric emptying,

TABLE 1.—Prevalence of peptic ulcer in smoking and nonsmoking women (number per 100)

Reference	No. with ulcers	Smokers	Nonsmokers	Ratio*
Higgins, M.W. (1966) (5)	47	2.8	1.4	2.0
Friedman, G.D. (1974) (4)	1092	6.3	3.9	1.6
Jedrychowski, W. (1974) (6)	26	0.8	1.3	0.6

*Ratio = $\frac{\text{Prevalence among smokers}}{\text{Prevalence among nonsmokers}}$

pancreatic secretion, or other processes which might be involved in the pathogenesis of peptic ulcer disease.

Summary

The 1979 Surgeon General's Report included evidence that cigarette smoking in males was significantly associated with the incidence of peptic ulcer diease and increased the risk of dying from peptic ulcer disease by approximately two-fold. The effect of smoking on pancreatic secretion and pyloric reflux demonstrated among men may provide a mechanism by which peptic ulcers develop.

1. Female smokers show a prevalence of peptic ulcer higher than that of nonsmokers by approximately two-fold.
2. The effect of cessation on healing is not known.

References

(1) ALLIBONE, A., FLINT, F. J. Bronchitis, aspirin, smoking and other factors in the aetiology of peptic ulcer. Lancet 2: 179-182, July 26, 1958.

(2) ALP, M. H., HISLOP, I. G., GRANT, A. K. Gastric ulcer in south Australia 1954-1963. 1. Epidemiological factors. Medical Journal of Australia 2: 1128-1132, December 12, 1970.

(3) DOLL, R., JONES, F. A., PYGOTT, F. Effect of smoking on the production and maintenance of gastric and duodenal ulcers. Lancet 1: 657-662, March 29, 1958.

(4) FRIEDMAN, G. D., SIEGELAUR, A. B., SELTZER, C. C. Cigarettes, alcohol, coffee and peptic ulcer. New England Journal of Medicine 290(9): 469-473, February 28, 1974.

(5) HIGGINS, M. W., KJELSBERG, M. Characteristics of smokers in Tecumseh, Michigan. II. The distribution of selected physical measurements and physiological variables and the prevalence of certain

diseases in smokers and nonsmokers. American Journal of Epidemiology 86: 60-77, 1967.

(6) JEDRYCHOWSKI, W., POPIELA, T. Association between the occurrence of peptic ulcers and tobacco smoking. Public Health London 88(4): 195-200, 1974.

(7) U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE. Smoking and Health: a Report of the Surgeon General. Department of Health, Education, and Welfare, Public Health Services, Office of the Assistant Secretary for Health, Office on Smoking and Health. DHEW Publication No. PHS) 79-50066, 1979, 1251 pp.

INTERACTIONS OF SMOKING WITH DRUGS, FOOD CONSTITUENTS, AND RESPONSES TO DIAGNOSTIC TESTS.

INTERACTIONS OF SMOKING WITH DRUGS, FOOD CONSTITUENTS, AND RESPONSES TO DIAGNOSTIC TESTS

Since most published studies investigating the effect of cigarette smoking on measures of health were performed in mixed populations, it is difficult to demonstrate specific factors applicable only to women. Neither the differences between men and women regarding the metabolism and action of drugs nor the pharmacological basis for differences between smokers and nonsmokers is well understood. The same is also true of the observed variations in laboratory values and nutritional needs. Thus, the associations for women between smoking, drugs, variations in clinical laboratory values, and nutritional needs require further study.

Women Smokers and Nonsmokers and Drug Consumption Patterns

The drug consumption pattern of women as compared to men has been studied by a number of investigators using different methodologies. The results consistently show that women are prescribed and take more prescription drugs than men (7,17). In one study where 1-year drug histories were used, the percentage of women using prescription drugs was 29 percent as compared to 13 percent for men (17). Another study which examined only drugs consumed within 48 hours of the interview showed that 60.2 percent of the women had taken medication compared to 41.8 percent of the men (7). The two studies cited are unique in the realm of drug usage studies because they measure actual self-administration of drugs rather than counting physician prescriptions or pharmacy dispensing patterns. Unfortunately, neither of these studies quantified information according to whether the subjects were smokers or nonsmokers.

Other reports show that smokers tend to use more drugs, especially of the psychotherapeutic type and drink more coffee and alcoholic beverages than nonsmokers (18,26). In only one study have women smokers and nonsmokers been compared for use of all drug categories; these data were derived from a self-administered questionnaire asking about drug use for the past year (21). As Table 1 shows, women smokers take more of almost every type of drug than nonsmokers. When the data were organized according to age groups, the 15-to-19-year-old group of women showed a marked elevation in drug use among smokers (Table 2).

Although the data are preliminary, a trend that female smokers consume drugs with greater frequency than female nonsmokers is suggested. It is beyond the scope of this chapter

TABLE 1.—Ratio of percent usage of drug classes, women smoker/nonsmoker status

Drug class	White	Black	Asian
Antihistamine or allergy medicine	0.8	0.9	0.6
Cough medicine	1.7	1.8	0.7
Asthma medicine	0.9	1.0	0.9
Aspirin-containing drugs	1.2	1.2	0.9
Pain medicine	1.2	1.2	1.0
Codeine, morphine, Darvon, Percodan, Demerol	1.5	1.6	1.2
Phenobarbital or other barbiturates	1.3	1.8	1.6
Sleeping pills	1.2	1.3	1.3
Tranquilizers	1.5	1.6	1.8
Anticoagulants	1.3	0.8	0.0
Digitalis or other heart medication	1.0	0.8	0.1
Antihypertensives	0.8	1.1	0.9
Diuretics	1.1	1.0	1.3
Cortisone-type medication	1.0	1.2	1.0
Hormones	1.2	1.3	1.4
Insulin or diabetic pills	0.9	0.8	0.9
Iron or anemia medications	0.9	0.9	0.9
Thyroid medication	1.1	1.3	2.3
Pills to control periods	1.3	1.2	1.5
Contraceptives	1.2	1.1	1.3
Benzedrine or Dexedrine	1.6	1.1	1.1
Weight reduction medication	1.1	0.9	1.3
Penicillin or other antibiotics	1.2	1.2	1.0
Sulfa drugs	1.1	1.2	0.8
Stomach or digestion medicine	1.2	1.2	1.3

SOURCE: Seltzer, C.G. (21).

TABLE 2.—Percentage of positive responses among females in age group 15–19

Question	Smokers	Nonsmokers
Taken phenobarbital or barbiturates?	2.3	1.0
Taken codeine, morphine, etc.?	16.0	6.5
Taken Benzedrine or Dexedrine?	4.9	0.3
Taken penicillin or other antibiotics?	33.0	25.8
Taken pills to prevent pregnancy?	27.0	9.7

SOURCE: Seltzer, C.G. (21).

to differentiate between the behavioral components of this phenomenon or to address the argument that women who smoke are less healthy than nonsmokers. It is beneficial, however, to examine the few reports that address the differences in

drug action between smokers and nonsmokers, regardless of the reasons for drug use.

Altered Clinical Response to Drug Therapy by Smokers Compared to Nonsmokers

The number of studies investigating the differences in the clinical responses to a drug by smokers and nonsmokers are far fewer in number than the studies examining the alterations in metabolism and biochemistry of drugs in smokers. The 1979 Surgeon General's Report included an extensive review of the alterations in drug disposition that occur in smokers (25). That information is useful for clarifying mechanisms by which smoking alters drug metabolism, absorption, excretion, and other functions. The clinical significance of these alterations has not been clarified, however.

The most exhaustive examination of alterations in smokers' clinical response to drugs was done by Jick and his associates in the Boston Collaborative Drug Surveillance Program (BCDSP). Over the past several years, this group has investigated the clinical response of smokers and nonsmokers to six different drugs: propoxyphene (Darvon) (4); diazepam (Valium) (3); chlordiazepoxide (Librium) (3); phenobarbital (3); chlorpromazine (Thorazine) (24); and theophylline tea (19). The differences observed between smokers and nonsmokers were consistent among men and women, except for the theophylline study, in which the toxic effects of therapy were slightly more frequent among women (13.4 percent) than among men (9.19 percent). Only in the chlorpromazine study (24) did the study group (those taking chlorpromazine) contain more women than men, an observation that supports other reports that women use major tranquilizing agents more frequently than men (18).

Since the published BCDSP data is not organized according to groups of women smokers and nonsmokers, any difference in drug use between these groups is not reflected in the data analysis. However, it is important to note that these studies, except as noted in the chlorpromazine study, predominantly involved men. It has been shown that women report more frequent use of the minor tranquilizers such as diazepam and chlordiazepoxide (17). Thus these studies should not be interpreted as reflecting drug response among the general population (17).

The studies on chlorpromazine, diazepam, and chlordiazepoxide showed a lessened frequency of the adverse effect of drowsiness among smokers as compared to nonsmokers (4,24). Conversely, no difference was reported for phenobarbital (3).

The analgesic effect of propoxyphene was reduced in smokers, an effect which was not observed in smokers on aspirin, codeine, acetaminophen, or combinations of these drugs (4).

The evidence for increased theophylline metabolism in smokers is well established and predicts the observed clinical response to theophylline (13). The BCDSP study of theophylline showed that smokers not only required larger doses of theophylline for efficacy, but also were less likely to report adverse effects than nonsmokers, even though they required larger doses.

Theoretically, then, because of a decreased clinical response to a drug, the tendency would be for the smoker to require increased doses to achieve the same therapeutic effect as a nonsmoker.

Therapeutic efficacy and adverse side effects in relationship to gender, smoking history, and drug consumption patterns have not been adequately studied, although the preliminary evidence would indicate an area of potential toxic drug effects and/or therapeutic failures.

Oral Contraceptives and Smoking

Chronic estrogen therapy has a profound interaction with chronic tobacco use. Again, the BCDSP has been most instrumental in assessing the influence of these two factors on the health status of women.

In assessing the relative risk of stroke in women who smoke and take oral contraceptives, the data from the Collaborative Group for the Study of Stroke in Young Women show that smoking alone increased the risk of hemorrhagic stroke (i.e., subarachnoid) from 1.0 for a nonsmoker who did not use oral contraceptives, to 2.6 for a smoker who did not use oral contraceptives. A smoker taking oral contraceptives had a relative risk of 6.1 or 7.6 (depending on the control group) (6). Similar increases in risks do not seem to occur for thrombotic stroke in the smoker taking oral contraceptives, but the risk of a thrombotic stroke for a woman using oral contraceptives alone is about nine times greater than that for a noncontraceptive user (5).

Again using the BCDSP data, the risk of nonfatal myocardial infarction among women under 38 is very low among nonsmokers, whether or not they use oral contraceptives. However, the risk to women who both smoke and use oral contraceptives is substantially higher, ranging from an estimated one per 8,400 annually in women aged 27 to 37 years to one per 250 for women aged 44 to 45 years (16). In a similar study of noncontraceptive estrogens, similar risks were demonstrated for women who both smoke and use estrogens (15). These findings are in agreement

with studies done in Great Britain where oral contraceptives were associated with an overall increase in cardiovascular disease in young women (20).

Another group which has investigated the link between smoking, oral contraception, and myocardial infarction reported that there is a considerable interaction between smoking and contraceptive use. The group found that rate of acute myocardial infarction among female smokers on oral contraceptives is greater than could be accounted for by either smoking or contraceptives alone (22). In earlier studies this same group concluded that there was a dose–response relationship between smoking and myocardial infarction in women, and that among women smoking 35 or more cigarettes per day, the rate of myocardial infarction was estimated to be 20 times higher than among those who never smoked (23).

These data lend themselves to the prediction of risk in only a very general way and provide no particular measures by which a woman—smoker or nonsmoker—can evaluate her own risk of experiencing one of the adverse effects described.

The following section reviews some of the laboratory values that are altered by smoking. Unfortunately, many of the largest studies on the correlation between smoking and alterations in clinical laboratory values have focused on men.

Alterations in Normal Clinical Laboratory Values in Women Smokers

Only a few investigators have studied clinical laboratory values in women smokers and nonsmokers (1,8-12,14,27). Many of these studies show statistically significant differences in a variety of common parameters. The clinical significance of these differences may not be apparent, however, since the actual differences between women smokers and nonsmokers are small. For example, a study of packed red cell volume (PCV) and hemoglobin (Hb) in women smokers and nonsmokers showed the PCV and Hb for nonsmokers to be 41.95 and 13.85 compared to 42.94 and 14.16 for smokers, a difference significant at $p < 0.05$, but a discrimination which physician or patient may find difficult to assess (14).

Small differences in laboratory values between smokers and nonsmokers can be seen in a number of serum chemistry and hematologic tests. One measurement that shows a wide enough variation between smokers and nonsmokers to be recognized clinically is the leukocyte count of a smoker (11,12). It is important to recognize that a WBC of 12,000 per mm^3 is within the normal range for a heavy cigarette smoker, and that the dif-

ferential count remains normal (11). In one study, individuals with chronic bronchitis were excluded from evaluation of leukocyte counts, and the same relative increase in leukocyte count was observed (12).

In several studies of triglyceride and cholesterol values in smoking and nonsmoking women, an elevation of both values, which was not statistically significant, was seen in smokers. The addition of oral contraceptive use to smoking caused a significant elevation over the nonsmoker, noncontraceptive user. The nonsmoker values were 79 ± 6.8 mg/100 ml for triglycerides and 157 ± 7.5 mg/100 ml for cholesterol. In the smoker they were 110 ± 14.8 mg/100 ml and 174.3 ± 8.8 mg/100 ml respectively, whereas the smoker using oral contraceptives had a triglyceride value of 150.0 ± 14.1 mg/100 ml and a cholesterol value of 186.1 ± mg/100 ml. In this same study, there was no significant difference between the levels of vitamins A, E or C in smoking and nonsmoking women (27).

A number of investigators have measured vitamin C levels in smoking and nonsmoking women, with extreme variation in results. Some showed decreased plasma and leukocyte vitamin C levels in smokers, and others showed no differences between smokers and nonsmokers. The discrepancies in these results may in part be related to the amount of dietary vitamin C habitually consumed by the subjects in the various studies (27).

Changes in serum proteins were the subject of another study of women smokers and nonsmokers (26). Significant differences in all serum protein fractions were found in cigarette smokers compared to nonsmokers. In general, the effects increased with the amount smoked. Past smokers showed globulin values that were significantly below those of women who never smoked, but there was no difference observed in the other serum protein fractions between past smokers and those who had never smoked.

The Influence of Smoking on the Nutritional Needs of Women

Outside of a possibly increased need for vitamin C in women who smoke, there is very little information about other nutrient requirements in smokers. In recent years a great deal of time has been spent studying the influence of smoking on fetal development, a subject covered elsewhere in this volume. The special nutritional needs of the nonpregnant smoking woman have not been dealt with in any systematic way.

A recent study involving obese women looked at the influence of smoking cessation on body weight (2). Although the data are innately biased because the study group consisted of women

enrolled in a weight loss program, the results showed that women who smoked less than a half pack of cigarettes a day gained 4 pounds after they quit. Heavy smokers consuming over two packs a day gained an average of 30 pounds over several decades. Moderate smokers gained an intermediate amount. This study does not contradict a commonly held notion that women gain weight when they stop smoking; however, it provides no behavioral or physiological hypothesis for this phenomenon.

Summary

Most published studies investigating the effects of cigarette smoking on drug use have been performed on mixed populations; factors specific for women have not been demonstrated to date. It has, however, been clearly demonstrated that women are prescribed and consume more prescription drugs than men.

1. Studies of selected drugs indicate that smoking may affect clinical responses and alter the dose required for an effective therapeutic result.
2. Smoking interacts with oral contraceptive use to increase the risk of myocardial infarction and subarachnoid hemorrhage.
3. Common clinical laboratory parameters are altered in smokers compared to nonsmokers; the health significance of these changes is unknown.
4. Insufficient information exists for assessment of the impact of smoking on the nutritional needs of women.

References

(1) BILLIMORIA, J. D., POZNER, H., METSELAAR, B., BEST, F. W., JAMES, D. C. D. Effect of cigarette smoking on lipids, lipoproteins, blood coagulation, fibrinolysis and cellular components of human blood. Atherosclerosis 21(1): 61-76, January-February 1975.

(2) BLITZER, P. H., RIMM, A. A., GIFFER, E. E. The effect of cessation of smoking on body weight in 57,032 women: cross sectional and longitudinal analysis. Journal of Chronic Diseases 30(7): 415-429, July 1977.

(3) BOSTON COLLABORATIVE DRUG SURVEILLANCE PROGRAM. Clinical depression of the central nervous system due to diazepam and chlordiazepoxide in relation to cigarette smoking and age. New England Journal of Medicine 288(6): 277-280, February 8, 1973.

(4) BOSTON COLLABORATIVE DRUG SURVEILLANCE PROGRAM. Decreased clinical efficacy of propoxyphene in cigarette smokers. Clinical Pharmacology and Therapeutics 14(2): 259-263, March-April 1973.

(5) COLLABORATIVE GROUP FOR THE STUDY OF STROKE IN YOUNG WOMEN. Oral contraceptives and increased risk of cerebral ischemia or thrombosis. New England Journal of Medicine 288(17): 871-878, April 26, 1973.

(6) COLLABORATIVE GROUP FOR THE STUDY OF STROKE IN

YOUNG WOMEN. Oral contraceptives and stroke in young women. Journal of the American Medical Association 231(7): 718-722, February 17, 1975.

(7) CRAIG, T. L., VANNATTA, P. A. Current medication use and symptoms of depression in a general population. American Journal of Psychiatry 135(9): 1036-1039, September 1978.

(8) DALES, L. G., FRIEDMAN, Q. D., SIEGELAUB, A. B., SELTZER, A. C. Cigarette smoking and serum chemistry tests. Journal of Chronic Diseases 27(6): 293-307, August 1974.

(9) DALES, L. G., FRIEDMAN, Q. D., SEIGELAUB, A. B., SELTZER, A. C., URY, H. K. Cigarette smoking habits and urine characteristics. Nephron 20: 163-170, 1978.

(10) DESMOND, P. V., ROBERTS, R. K., WILKINSON, Q. R., SCHENKER, S. No effect of smoking on metabolism of chlordiazepoxide. New England Journal of Medicine 300(4): 199-200 January 25, 1979.

(11) FRIEDMAN, Q. D., SIEGELAUB, A. B., SELTZER, C. C., FELDMAN, R., COLLEN, M. F. Smoking habits and the leukocyte count. Archives of Environmental Health 26(3): 137-143. March 1973.

(12) HELMAN, N., RUBENSTEIN, L. S. The effects of age, sex, and smoking on erythrocytes and leukocytes. American Journal of Clinical Pathology 63: 35-44, 1975.

(13) HUNT, S. N., JUSKO, W. J., YURCHAK, A. M. Effect of smoking on theophylline disposition. Clinical Pharmacology and Therapeutics 19(5, Part 1): 546-551, May 1976.

(14) ISAGER, H., HAGERUP, L. Relationship between cigarette smoking and high packed cell volume and haemoglobin levels. Scandinavian Journal of Haemotology 8(4): 241-244, 1971.

(15) JICK, H., DINAN, B., ROTHMAN, K. J. Noncontraceptive estrogens and nonfatal myocardial infarction. Journal of the American Medical Association 239(14): 1407-1408, April 3, 1978.

(16) JICK, H., DINAN, B., ROTHMAN, K. J. Oral contraceptives and nonfatal myocardial infarction. Journal of the American Medical Association 239(14): 1403-1406, April 3, 1978.

(17) PARRY, H. F., BALTER, M. B., MELLINGER, Q. D., CISIN, I. H., MANHEIMER, D. I. National patterns of psychotherapeutic drug use. Archives of General Psychiatry 28: 769-783, June 1973.

(18) PARRY, H. F., CISIN, I. H., BALTER, M. B., MELLINGER, Q. D., MANHEIMER, D. I. Increasing alcohol intake as a coping mechanism for psychic stress. In: Cooperstock, R. (Editor). Social Aspects and Medical Use of Psychotropic Drugs. Toronto, Addiction Research Foundation, 1974.

(19) PFEIFER, H. J., GREENBLATT, D. J. Clinical toxicity of theophylline in relation to cigarette smoking. Chest 73(4): 455-459, April 1978.

(20) ROYAL COLLEGE OF GENERAL PRACTITIONERS ORAL CONTRACEPTION STUDY. Mortality among oral contraceptive users. Lancet (4): 727-733, October 8, 1977.

(21) SELTZER, C. G., FRIEDMAN, Q. D., SIEGELAUB, A. B. Smoking and drug consumption in white, black, and oriental men and women. American Journal of Public Health 64(5): 466-473, March 1974.

(22) SHAPIRO, S., SLONE, D., ROSENBERG, L., KAUFMAN, D., STOLLEY, P.D., MIETTINEN, O. S. Oral contraceptive use in relation to myocardial infarction. Lancet (1): 743-747, April 7, 1979.

(23) SLONE, D., SHAPIRO, S., ROSENBERG, L., KAUFMAN, D. W., HARTZ, S. C., ROSSI, A. C., STOLLEY, P. D., MIETTINEN O. S. Relation of cigarette smoking to myocardial infarction in young women.

New England Journal of Medicine 298(23): 1273-1276, 1978.

(24) SWETT, D. Drowsiness due to chlorpromazine in relation to cigarette smoking. Archives of General Psychiatry 31: 211-213, August 1974.

(25) U.S. PUBLIC HEALTH SERVICE. Smoking and Health. A Report of the Surgeon General. U.S. Department of Health, Education, and Welfare, Public Health Service, Office of the Assistant Secretary for Health, Office on Smoking and Health, DHEW Publication No. (PHS) 79-50066, 1979, 1251 pp.

(26) WINGERD, J., SPONZILLI, E. E. Concentrations of serum protein fractions in white women: effects of age, weight, smoking, tonsillectomy and other factors. Clinical Chemistry 23(7): 1310-1317, 1977.

(27) YEUNG, D. L. Relationships between cigarette smoking, oral contraceptives and plasma vitamins A, E, C and plasma triglycerides and cholesterol. American Journal of Clinical Nutrition 29: 1216-1221, 1976.

PART III:

PSYCHOSOCIAL AND BEHAVIORAL ASPECTS OF SMOKING IN WOMEN.

PSYCHOSOCIAL AND BEHAVIORAL ASPECTS OF SMOKING IN WOMEN

Introduction

Currently, women are rapidly approaching men in the rate of initiation and prevalence of cigarette smoking, but seem to have a lower rate for successful cessation of smoking. (See also Part I of this report, Patterns of Cigarette Smoking.) While an increasing percentage of the U.S. population is giving up smoking, nationwide surveys and cessation studies suggest that a smaller proportion of women than men are quitting successfully.

This part discusses tobacco use by women, with comparative reference to men's use wherever appropriate. Special attention is directed to the patterns of initiation, the rise in smoking among girls, and the factors important in the maintenance of smoking behavior, including pharmacological effects, smoking patterns, information dissemination, and stress management. The differences in successful quitting between men and women smokers are discussed with the hope of generating new ideas for research and intervention.

A separate analysis of smoking patterns among women in the health professions is presented. In addition, a section is devoted to the pregnant smoker because the impact of smoking, both on the fetus and on the pregnant woman, makes this a period of particular importance in the life of the women smoker.

Initiation of Smoking in Adolescent Girls

Cigarette smoking, particularly cigarette smoking among young girls, is a changing phenomenon. Shifts in smoking attitudes and behaviors reflect broader social forces, including changes in sex roles and gender differences in responses to public information programs and to social sanctions against smoking.

The trend in adolescent smoking, as in other "adult-like" behaviors such as alcohol use or sexual activity, is toward earlier onset. For example, before the mid-1970s, girls were less likely to start smoking than boys, and when they did, they started later. Neither of these differences holds true any longer.

A number of psychosocial variables correlate highly with adolescent smoking trends. These include the attitudes, perceptions, and behaviors of adolescent girls, their social setting (family, peer groups) and those broad demographic factors (race, education, family income, urbanicity) that help to define an individual's position within the society.

CONCEPTS OF ADOLESCENT BEHAVIOR

Discussions of adolescence with its attendant problems have seldom differentiated between boys and girls, and no theory or model of adolescent behavior has been developed specifically for girls. However, gender differences in development, cognitive processes, sex-role acquisition and achievement have recently been examined and a number of psychological differences have been identified (24,26,51,68,98,211).

The essence of adolescence is growth, transition, and change. The rate of physical growth in adolescence is more rapid than at any other stage of development except the neonatal stage. Adolescent development is a complicated process which involves increasing self-awareness, intellectual and emotional growth, and physiological changes.

What adults characterize as risk taking in adolescence may be exploration of the limits of identity and capability. Adolescents are attempting to resolve the competing and conflicting demands stemming from childhood experience on the one hand and expectations of adulthood on the other: dependency and compliance versus autonomy and independent decision-making; orientation toward family versus orientation toward peers. They face increasing demands for social and cognitive achievement and for developing the self-control required to handle new psychological, physical, and social situations. Inadequate experience with these challenges or failure to meet them may result in low self-esteem and increased anxiety and stress.

Numerous formulations contributing to a general model of adolescent development have emerged. These include life-span theory and cohort change (52,131), adolescent sexuality (32), and differences between early and late adolescence (85).

Douvan and Adelson have identified issues that distinguish adolescence: for girls they are sexuality, interpersonal-intimacy, and identity issues; for boys they are sexuality, autonomy–assertion–independence and identity issues (51). In this study, conducted in the 1950s, girls evidenced conflict between the social roles for which they were preparing (further education and careers) and the future role they desired (marriage–motherhood). La Farge described a similar female adolescent conflict between social rules and individual perceptions (109). Research published in the 1970s shows that young women still have role conflicts different from those of young men (68).

Research on gender–role differentiation in childhood has provided some insight into developmental differences between girls and boys. Maccoby suggests that these differences may

derive from different role models for boys and girls; from the varying responses of significant adults to their behaviors; from biological differences; and from a combination of these (116). Block and Maccoby and Jacklin report that the differences include girls having less confidence in their ability to handle a new task and less sense of control over what happens to them (18,117). Girls also show greater susceptibility to expressed anxiety, greater need for help and reassurance, greater closeness to friends, and more concern for what is socially desirable.

Adolescent behaviors—social or antisocial, adaptive or maladaptive—are a function both of individual choice and of the opportunities for growth and development which a society provides its youth (36). "Not only is the term 'adolescence' a social definition, but what society perceives as an adolescent problem is also socially defined" (52). Similarly, the development of values, motivations, and controls that foster healthy growth and deter the onset of smoking and other undesirable behaviors depends on the opportunities and resources that society makes available to the adolescent.

PREVALENCE AND PATTERNS OF ADOLESCENT CIGARETTE USE

National surveys of adolescent smoking behavior have provided information on gender differences, secular trends, and age subgroupings within the adolescent period. Surveys of smoking patterns, ages 12 to 18, were conducted by the National Clearinghouse for Smoking and Health (NCSH) in 1968, 1970, 1972, and 1974 and by the National Institute of Education (NIE) in 1979 (130,197). Two other periodic surveys, both sponsored by the National Institute on Drug Abuse (NIDA), included cigarette consumption (2,101). A number of studies in specific geographic locales or among specific populations, such as high school students, have also been carried out (198). Differing definitions of a current regular adolescent smoker make comparisons among these studies particularly difficult. In the NCSH and NIE surveys, a regular smoker is defined as one who smokes cigarettes at least weekly. In the NIDA surveys, regular smoking is defined as occurring within the past 30 days.

Prevalence

Table 1 summarizes adolescent cigarette smoking prevalence between 1968 and 1979, by age and gender, as surveyed by NCSH and by NIE. Between 1968 and 1974 there was a significant increase in the percentage of girl smokers in each age category at each point in time, in contrast to the relatively stable

prevalence of current regular smoking among boys. A decline in the average age of smoking initiation for both sexes is suggested by the small but significant increase in smoking prevalence among 12 to 14 year olds. (198). Trends in the data from a national study of high school seniors also support the hypothesis of an earlier age of initiation (101).

In the five years from 1974 to 1979, the proportion of 17 to 18 year old girls who smoked changed little, but the proportion of boys who smoked dropped by a third. It was this difference among 17 to 18 year olds that created the overall higher smoking rate for girls as compared with boys in 1979. However, at ages 15 to 16, the drop from 1974 to 1979 was greater for girls than boys, suggesting that the initiation of smoking is also beginning to decline in those girls born after 1962.

The differences in the within-age-group changes in the smoking prevalence of girls may represent an isolated effect on the cohort of girls born in 1963 and 1964. The change was essentially confined to the 15 to 16 year old subgroups who were born during these years. The precise nature of the interaction of social influences on the development and maturation of this cohort is unclear. However, other data suggest that a marked secular change occurred in cigarette smoking attitudes and behavior which was secondary to an increased awareness of the health risks of smoking.

An alternate hypothesis is that the isolated decline in the 15 to 16 year old subgroup may be an artifact produced by the combined trends of reduced initiation of smoking and the initiation at a younger age. Thus, the decline in prevalence among 15 to 16 year old girls would reflect the decreasing percentage of young women who are taking up smoking, but this trend will be masked in the younger age group by the tendency of those girls who are going to take up smoking to do so at a younger age. The 1979 NIE Survey reports that:

> The increasing prevalence of teenage smoking that was observed in the period between 1968 and 1974 has come to a halt, and a decrease in the smoking rates of both boys and girls has taken place. The decrease in boys' smoking was greater than that of girls, resulting in a higher smoking rate for girls than for boys in 1979. Smoking among boys leveled off in the early 1970s, and then began to decrease. It appears that girls are now following this pattern: the smoking rate has leveled off among 17 and 18 year olds, and probably can be expected to decrease over the next few years (130).

Other surveys (Table 2) support these trends in adolescent girls' smoking behavior. Differences between studies in absolute prevalence rates reported are at least partly due to the

TABLE 1.—Estimates of the percentage of current, regular cigarette smokers, adolescents, aged 12 to 18, United States, 1968–1979

Year	Ages 12–14		Ages 15–16		Ages 17–18		Ages 12–18	
	Male	Female	Male	Female	Male	Female	Male	Female
1968	2.9	0.6	17.0	9.6	30.2	18.6	14.7	8.4
1970	5.7	3.0	19.5	14.4	37.3	22.8	18.5	11.9
1972	4.6	2.8	17.8	16.3	30.2	25.3	15.7	13.3
1974	4.2	4.9	18.1	20.2	31.0	25.9	15.8	15.3
1979	3.2	4.3	13.5	11.8	19.3	26.2	10.7	12.7

NOTE: Current regular smoker includes respondent who smokes cigarettes at least weekly.
SOURCE: National Clearinghouse for Smoking and Health (197), National Institute of Education (130).

difference in the definition of a smoker, and differences in survey technique. The National Institute of Education Survey included as current regular smokers both those who smoke one or more cigarettes per week and those who smoke one or more cigarettes a day. The prevalence rates of Abelson, et al. (2) and Johnston, et al. (101) refer to any cigarette smoking in the past 30 days.

The Abelson, et al. data, which were collected 2 years before that of NIE, show the predicted decline, but to a lesser degree (2,130). The Johnston, et al. data suggest that there was an increase in adolescent girls' smoking as measured in samples of high school seniors between 1975 and 1977 (101). Johnston's figures were retrospectively reported and refer only to youngsters born before and during 1960, and therefore, would not be expected to reflect changes occurring in those cohorts born after 1962 where the decline has occurred. This may explain why the Johnston, et al. 1977 sample did not reflect a downturn, and reports of later cohorts of high school seniors should show a stabilization and then a decline in female smoking rates. Results from a study by the same group in 1978 show the predicted downturn in the smoking habits of high school senior girls (from 39.6 percent in 1977 to 38.1 percent in 1978) as well as boys (from 36.6 percent in 1977 to 34.5 percent in 1978) (103).

Age of Initiation of Smoking

The data in Table 1 show that the prevalence of smoking in girls aged 12-14 increased steadily between 1968 and 1974 to a level equal to or slightly higher than boys of the same age. Between 1974 and 1979 the prevalence of smoking stabilized in