CHAPTER 5

Peptic Ulcer Disease

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Introduction

Previous epidemiological and experimental studies of the relationship between cigarette smoking and peptic ulcer disease were reviewed in the 1971 and 1972 reports on the health consequences of smoking (*17*, *18*) and form the basis of the following summary:

The results of epidemiological studies indicate that cigarette smoking males have an increased prevalence of peptic ulcer disease and a greater mortality from peptic ulcer as compared to nonsmoking males. Among males, the association between cigarette smoking and peptic ulcer disease is stronger for gastric than for duodenal ulcer, but significant for both. For males, cigarette smoking appears to reduce the effectiveness of standard peptic ulcer treatment and to slow the rate of peptic ulcer healing. The relationship between cigarette smoking and the prevalence of and mortality from peptic ulcer disease is less clear for females than for males.

Experimental studies of the effect of cigarette smoking in man, and of the effect of injection and infusion of nicotine in animals, on gastric secretion and motility have produced conflicting results. In dogs, an infusion of nicotine has been found to inhibit pancreatic and hepatic bicarbonate secretion, thus demonstrating a possible link between cigarette smoking and duodenal ulcer.

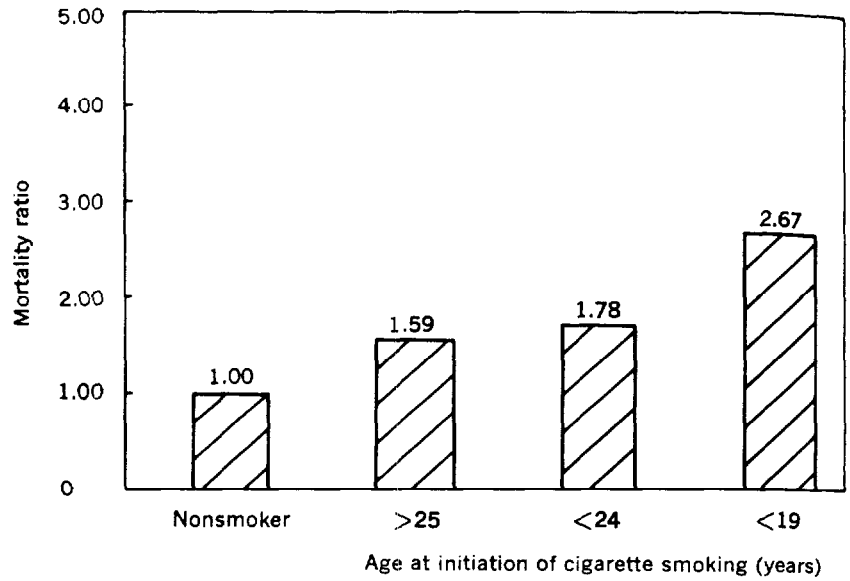
Recently, additional epidemiological, clinical, autopsy, and experimental studies have confirmed the association between cigarette smoking and gastric ulcer mortality and have clarified a mechanism through which cigarette smoking might be linked to duodenal ulcer.

Epidemiological and Clinical Studies

Previous studies of the relationship between peptic ulcer disease and cigarette smoking have been conducted in predominantly white, Western populations. A large prospective epidemiological study is currently being conducted in Japan. From this study, Hirayama (*6*) reported 5-year followup data on 265,118 men and women, aged 40 years and older, representing 91 to 99 percent of the total population in the area of the 29 health districts in which the study was conducted. Both male

and female cigarette smokers experienced higher death rates from gastric ulcer as compared with nonsmokers. The mortality ratio for cigarette smokers was 1.81 for males ($P<0.001$) and 2.15 for females ($P<0.05$). The mortality ratio for smokers (males and females combined) was dose-dependent as measured by age at initiation of smoking (fig. 1). The results of this study, in the context of the genetic and cultural differences between Japanese and Western populations, provide a significant confirmation of the association between cigarette smoking and gastric ulcer mortality.

Figure 1.—Gastric ulcer mortality ratios of Japanese (men and women combined) by age at initiation of cigarette smoking (1966–1970).



SOURCE: Hirayama, T. (6).

Alp, et al. (*1*) conducted a retrospective survey of 638 patients, admitted to two Australian teaching hospitals between 1954 and 1963, with chronic gastric ulcer confirmed by roentgenographic, endoscopic, or surgical examination. The findings in the patients were compared with information available about the South Australian population obtained at census in 1954 and 1961, and with a control group of 233 subjects matched for age and sex with the ulcer patients. Cigarette use, a family history of peptic ulcer, domestic stress, and aspirin and alcohol intake occurred significantly more frequently among ulcer patients. Alp, et al. (*2*) found that after surgical treatment, recurrence of the ulcer was significantly more likely to recur among those patients who continued to smoke, drink, and use aspirin ($P<0.001$).

Fingerland, et al. (*5*) compared the autopsy findings from 765 males with their smoking history. The autopsies were performed without selection during 1965 and 1966 at the University of Hradec Králové, Czechoslovakia. Peptic ulcer was significantly more frequent among male ex-smokers and male lifelong smokers than among male nonsmokers ($P<0.02$). Among males, a dose-response relationship was found between estimated total cigarette consumption and the presence of peptic ulcer at autopsy.

Cooper and Tolins (*4*) reported results from a retrospective study of the relationship between cigarette smoking and postoperative complications among 2,988 males, admitted to 19 Veterans Administration hospitals, for the surgical treatment of duodenal ulcer. Smoking history was obtained for 1,441 of the men, and of these 273 were nonsmokers, 1,018 smoked cigarettes only, and 93 smoked cigarettes plus a pipe and/or cigars. The authors found no evidence of an association between either the number of cigarettes smoked per day, or the number of years of cigarette smoking, and postoperative complications, operative mortality, or length of hospital stay. They emphasized that their results must be viewed with considerable caution and listed several potential sources of bias. In addition, they noted, "* * * that these results apply only to the immediate postoperative findings and do not apply to the long-range effects of smoking upon the patient after surgery for duodenal ulcer disease."

Experimental Studies

Gastric Secretion

Studies in Humans

Morales, et al. (*10, 11*) studied the effect of cigarette smoking on gastric secretion in a group of 312 patients. The patients included 138

with duodenal ulcer, 93 with gastric ulcer, and 81 with other gastrointestinal disorders, who served as controls. Cigarette smoking was significantly more frequent among the patients with peptic ulcer than among the controls.

The chronic effect of smoking on gastric secretion was quite variable. Male smokers among the controls and in the group with duodenal ulcers had a significantly increased baseline acid output as compared with nonsmokers in the same groups ($P<0.05$). After a subcutaneous injection of histamine, only the group of male smokers with gastric ulcers had a significant increase in acid output over the values obtained for nonsmokers in the same group ($P<0.05$). Among the smokers in the control group, the relationship between gastric acid output and the number of cigarettes smoked daily was dose dependent. No such relationship was obtained for either of the two groups with peptic ulcers.

In these experiments, the acute effect of smoking on gastric secretion was slight. In one set of experiments, a group of eight smokers served as its own control. The smoking of two cigarettes prior to collection of gastric juice had no significant effect on acid output as compared to baseline values. After smoking two cigarettes and also receiving a subcutaneous injection of histamine, the patients experienced no significant change in gastric acid output as compared to baseline values; 21 male patients, including members from the groups with ulcers and controls, smoked one cigarette 1 hour after an intravenous infusion of histamine. A transient depression of gastric acid output was noted as compared with the values obtained from nine patients who did not smoke.

Studies in Animals

Konturek, et al. (*8*) studied the effect of intravenous infusion of nicotine on the formation of acute, experimental duodenal ulcers in cats. The authors infused nicotine intravenously in doses comparable to the smoking of four, eight, and 16 cigarettes per hour into cats in whom near maximal gastric acid output had been stimulated with intravenous pentagastrin. The investigators found that nicotine in the two lower doses had no effect upon the gastric acid output stimulated by pentagastrin, but that the highest dose produced a significant decrease in response, due to a fall in both volume and acid concentration. Nicotine alone failed to alter a negligible basal gastric secretion. In control animals (pentagastrin alone), duodenal ulcers were found in eight of 10 animals. Nicotine at the two lower doses, in combination with pentagastrin, produced ulcers in all 26 animals. At the intermediate dose of nicotine, the mean ulcer area was twice that found in

the control group. At the highest dose of nicotine, peptic ulcers appeared in only two of six animals and the area of ulcer was reduced compared to controls.

Shaikh, et al. (*14*) studied the acute and chronic effects of subcutaneously injected nicotine on gastric secretion in rats. Under basal conditions, the volume of gastric secretion was initially depressed, then stimulated, and depressed again as the dose of nicotine was increased. Acid output was decreased over the entire range of nicotine dosage. Pepsin output reflected a similar triphasic response to increasing nicotine doses as did gastric secretory volume. In the absence of nicotine, pentagastrin stimulated gastric volume, acid, and pepsin output. The injection of nicotine, in increasing doses, administered simultaneously with pentagastrin, resulted in a gradual decrease in response for all parameters. Volume of gastric juice, acid output, and pepsin output were all increased significantly by chronic exposure to nicotine alone. Based on an average smoking dose of nicotine, the dose of nicotine employed in the chronic experiments corresponded to the smoking of three to five cigarettes per day.

Thompson, et al. (*16*) extended the study of rats described above by studying the effects of chronic nicotine injections in vagotomized rats and rats with discrete lesions in the hypothalamus. In sham-operated animals, chronic nicotine injections significantly increased baseline volume of gastric juice, acid output, and pepsin output. Following vagotomy, the nicotine response was completely suppressed. Caudal hypothalamic lesions did not influence the response to nicotine in the presence of intact vagus nerves. Anterior hypothalamic lesions, ranging from the anterior hypothalamic area to the ventromedial hypothalamus, blocked the nicotine-induced gastric secretory stimulation in the presence of intact vagi. The authors concluded that chronic nicotine-induced gastric secretory stimulation is mediated via anterior hypothalamic activation and intact vagus nerves. The importance of local effects remained uncertain.

Pancreatic Secretion

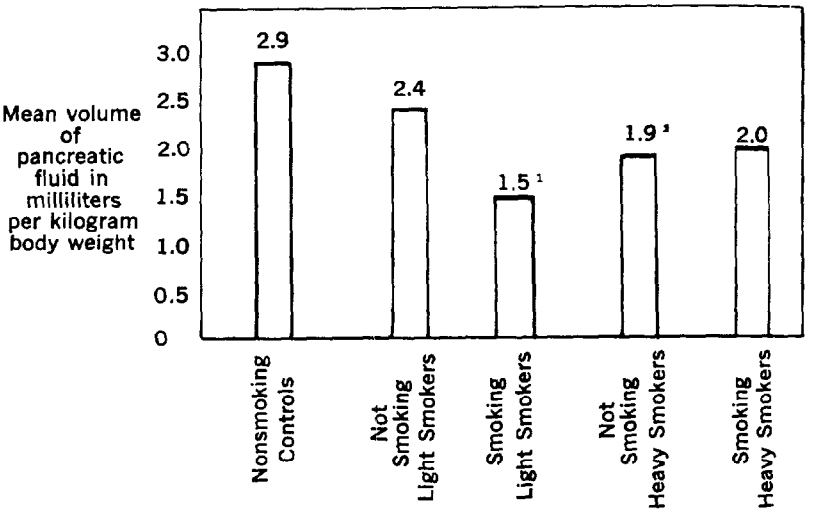
Studies in Humans

Bynum, et al. (*3*) studied the effect of cigarette smoking upon pancreatic secretion in 23 healthy young males and females. Five control male nonsmokers were compared with seven male and two female light smokers (less than one pack of cigarettes per day for less than 3 years) and eight male and one female heavy smokers (more than one pack of

cigarettes per day for more than 3 years). Pancreatic secretion was measured by the double secretin test, using Boots secretin. The experiment was divided into two parts for the smokers: A basal collection period and an experimental period during which the subjects smoked seven nonfiltered cigarettes at the rate of four per hour. Light smokers had basal values for pancreatic secretory volume and bicarbonate output in response to secretin which were not significantly different from controls. After the subjects had smoked, significant depression of both pancreatic volume and bicarbonate output was noted ($P<.001$). Heavy smokers had basal values that were significantly less than in the control subjects ($P<0.01$). Smoking, however, did not further depress the response to secretin (figs. 2 and 3).

Solomon and Jacobsen (*15*) reviewed some possible mechanisms whereby the increased prevalence and mortality from duodenal ulcer among cigarette smokers might be produced. They concluded that evidence from studies in animals, coupled with the findings of Bynum, et al. (*3*), supported the hypothesis that the mechanism active in humans involves impaired neutralization of acid secondary to the inhibition of pancreatic bicarbonate secretion.

Figure 2.—Effect of cigarette smoking on volume of secretin-stimulated pancreatic secretion in humans.

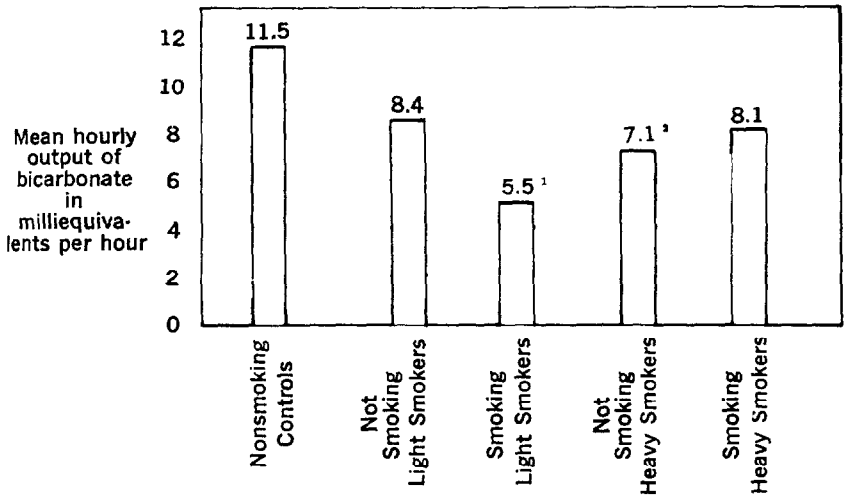


[1] Significantly different from nonsmoking test within group of light smokers ($P < 0.001$).

[2] Significantly different from nonsmoking controls ($P < 0.01$).

SOURCE: Bynum, et al. (*3*).

Figure 3.—Effect of cigarette smoking on secretin-stimulated pancreatic bicarbonate output in humans.



[1] Significantly different from nonsmoking test within group of light smokers ($P < 0.001$).

[2] Significantly different from nonsmoking controls ($P < 0.01$).

SOURCE: Bynum, et al. (3).

Studies in Animals

Konturek, et al. (*7*) extended his research on the mechanism of nicotine-induced inhibition of pancreatic secretion in the dog, using the design previously employed (*9*). Infused secretin alone led to a sustained increase in pancreatic bicarbonate output. Intravenous nicotine, at all four doses of infused secretin, produced a significant inhibition of pancreatic volume and bicarbonate output ($P<0.05$). Infused nicotine appeared to inhibit competitively the effect of secretin on pancreatic secretion of fluid and bicarbonate. Topical (intraduodenal) nicotine failed to affect significantly the response to infused secretin. Stimulation of endogenous secretin by an acid infusion into the duodenum produced the expected pancreatic secretory response. Nicotine either applied to the duodenal mucosa or injected intravenously significantly inhibited the pancreatic secretory response to endogenous secretin. Nicotine had no significant effect on total pancreatic protein output. Nicotine did not alter the cholecystokinin-induced stimulation of pancreatic secretion. The authors concluded that nicotine may inhibit pancreatic secretion of fluid and bicarbonate both

by a direct effect on pancreatic secretory mechanisms, acting as a competitive inhibitor of secretin, and by a secondary effect on the duodenal mucosa, depressing the endogenous release of secretin by acid.

Robert (*12*) studied the potentiation of active duodenal ulcers by nicotine administration in the rat. Subcutaneous infusion of pentagastrin and carbachol resulted in the dose-dependent formation of duodenal ulcers within 24 hours. Nicotine alone produced no ulcers. Increasing doses of subcutaneously infused nicotine, in combination with the other two agents, resulted in a steadily increasing dose-related incidence and severity of the duodenal ulcers. Robert noted that Konturek, et al. (*9*) found that nicotine inhibited pancreatic and biliary bicarbonate secretion in dogs, and that Thompson, et al. (*16*) found that acute doses of nicotine in rats either depressed or did not alter gastric secretion. He concluded that the most probable mechanism by which nicotine potentiated acute duodenal ulcer formation in the rat was via a suppression of pancreatic secretion.

Robert, et al. (*13*) further tested this hypothesis by infusing acid via the esophagus of rats in doses found to cause duodenal ulcers in one-third of the experimental animals. One group of rats also received a subcutaneous infusion of nicotine. Another received nicotine, but only water was infused via the esophagus; 31 percent of the animals receiving acid but no nicotine had duodenal ulcers; 93 percent of the nicotine-acid group had duodenal ulcers, while none of the nicotine-water group had ulcers. The ulcers in the nicotine-acid group were more numerous, extensive, and deeper than those in the animals which received acid alone.

Summary of Recent Peptic Ulcer Disease Findings

In addition to the findings relating cigarette smoking to peptic ulcer disease, summarized in previous reports on the health consequences of smoking (*17, 18*) and cited in the introduction to this chapter, recent studies have contributed further to our understanding of the association:

1. The finding of a significant dose-related excess mortality from gastric ulcers among both male and female Japanese cigarette smokers, in a large prospective study, and in the context of the genetic and cultural differences between the Japanese and previously investigated Western populations, confirms and extends the association between cigarette smoking and gastric ulcer mortality.

2. Data from experiments in several different animal species suggest that nicotine potentiates acute duodenal ulcer formation by means of inhibition of pancreatic bicarbonate output.
3. Cigarette smoking has been demonstrated to inhibit pancreatic bicarbonate secretion in healthy young men and women.

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CHAPTER 6

Pipes and Cigars

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Introduction

This chapter is a review of the epidemiological, pathological, and experimental data on the health consequences of smoking cigars and pipes, alone, together, and in various combinations with cigarettes. Previous reviews on the health consequences of smoking have dealt primarily with cigarette smoking. Although some of the material on pipes and cigars presented in this chapter has been presented in previous reports of the Surgeon General, this is the first attempt to summarize what is known about the health effects of pipe and cigar smoking. Since the use of pipes and cigars is limited almost exclusively to men in the United States, only data on men are included in this review.

The influence of pipe and cigar smoking on health is determined by examining the overall and specific mortality and morbidity experienced by users of these forms of tobacco compared to nonsmokers. Epidemilogical evidence suggests that individuals who limit their smoking to only pipes or cigars have overall mortality rates that are slightly higher than nonsmokers. For certain specific causes of death, however, pipe and cigar smokers experience mortality rates that are as great as or exceed those experienced by cigarette smokers. This analysis becomes more complex when combinations of smoking forms are examined. The overall mortality rates of those who smoke pipes, cigars, or both in combination with cigarettes appear to be intermediate between the high mortality rates of cigarette smokers and the lower rates of those who smoke only pipes or cigars. This might seem to suggest that smoking pipes or cigars in combination with cigarettes diminishes the harmful effects of cigarette smoking. However, an analysis of mortality associated with smoking combinations of cigarettes, pipes, and cigars should be standardized for the level of consumption of each of the products smoked in terms of the amount smoked, duration of smoking, and the depth and degree of inhalation. For example, cigar smokers who also smoke a pack of cigarettes a day might be expected to have mortality rates somewhat higher than those who smoke only cigarettes at the level of a pack a day, assuming that both groups smoke their cigarettes in the same way. Mixed smokers who inhale pipe or cigar smoke in a manner similar to the way they smoke cigarettes might be expected to have higher mortality rates than mixed smokers who do not inhale their cigars and pipes and also

resist inhaling their cigarettes. Unfortunately, little of the published material on mixed cigarette, pipe, and cigar smoking contains these types of analyses or controls.

A paradox seems to exist between the mortality rates of ex-smokers of pipes and cigars and ex-smokers of cigarettes. Ex-cigarette smokers experience a relative decline in overall and certain specific causes of mortality following cessation. This decline is important but indirect evidence that cigarette smoking is a major cause of the elevated mortality rates experienced by current cigarette smokers. In contrast to this finding, several prospective epidemiological investigations, Hammond and Horn (*40*), Best (*9*), Kahn (*50*), and Hammond (*38*), have reported higher death rates for ex-pipe and ex-cigar smokers than for current pipe and cigar smokers. This phenomenon was analyzed by Hammond and Garfinkel (*39*). The development of ill health often results in a cigarette smoker giving up the habit, reducing his daily tobacco consumption, switching to pipes or cigars, or choosing a cigarette low in tar and nicotine. In many instances, a smoking-related disease is the cause of ill health. Thus, the group of ex-smokers includes some people who are ill from smoking-related diseases, and death rates are high among persons in ill health.

As a result, ex-cigarette smokers initially have higher overall and specific mortality rates than continuing cigarette smokers, but because of the relative decrease in mortality that occurs in those who quit smoking for reasons other than ill health, and because of the dwindling number of ill ex-smokers, a relative decrease in mortality is observed (within a few years) following cessation of cigarette smoking. The beneficial effects of cessation would be obvious sooner were it not for the high mortality rates of those who quit smoking for reasons of illness. A similar principle operates for ex-pipe and ex-cigar smokers, but because of the lower initial risk of smoking these forms and therefore the smaller margin of benefit following cessation, the effect produced by the ill ex-smokers creates a larger and more persistent impact on the mortality rates than is seen in cigarette smoking.

For the above reasons a bias is introduced into the mortality rates of current smokers and ex-smokers of pipes and cigars, so that a more accurate picture of mortality might be obtained by combining the ex-smokers with the current smokers and looking at the resultant mortality experience.

Because of a lack of data that would allow a precise analysis of mortality among ex-pipe and ex-cigar smokers, a detailed analysis of these groups could not be undertaken in this review.

For each specific cause of death, tables have been prepared which summarize the mortality and relative risk ratios reported in the major

prospective and retrospective studies which contained information about pipe and cigar smokers. The smoking categories used include: cigar only, pipe only, total pipe and cigar, cigarette only, and mixed. The total pipe and cigar category includes: those who smoke pipes only, cigars only, and pipes and cigars. The mixed category includes: those who smoke cigarettes and cigars; cigarettes and pipes; and cigarettes, pipes, and cigars. Mortality and relative risk ratios were calculated relative to nonsmokers.

The Prevalence of Pipe, Cigar, and Cigarette Usage

The prevalence of pipe, cigar, and cigarette smoking in the United States was estimated by the National Clearinghouse for Smoking and Health from population surveys conducted in 1964, 1966, and 1970 (*98, 99, 100*). In each survey, about 2,500 interviews were conducted on a national probability sample stratified by type of population and geographic area. The use of these products among adults aged 21 and older is summarized in tables 1 and 2. The prevalence of pipe, cigar, and cigarette smoking in Great Britain for the years 1965, 1968, and 1971 is presented in table 3.

TABLE 1.—*Percent distribution of U.S. male smokers aged 21 and older by type of tobacco used for the years 1964, 1966, and 1970*

Forms used	1964 (percent)	1966 (percent)	1970 (percent)
1. Cigar only	6. 8	5. 5	5. 6
2. Pipe only	1. 7	3. 0	3. 6
3. Pipe and cigar	3. 9	4. 9	4. 4
4. Cigarette only	28. 6	31. 2	25. 9
5. Cigarette and cigar	11. 3	9. 9	6. 6
6. Cigarette and pipe	5. 3	4. 9	5. 3
7. Cigarette, pipe, and cigar	7. 7	6. 3	4. 6
8. Nonsmoker	34. 7	34. 3	44. 0
Total	100. 0	100. 0	100. 0
Number of persons in sample	2, 389	2, 679	2, 861
Total pipe users (2+3+6+7)	18. 7	19. 2	17. 9
Total cigar users (1+3+5+7)	29. 9	26. 7	21. 2
Total cigarette users (4+5+6+7)	52. 9	52. 4	42. 3

Source: U.S. Department of Health, Education, and Welfare (*98, 99, 100*).

TABLE 2.—*Percent distribution of U.S. male smokers by type of tobacco used and age for 1970*

Forms used	Age groups				
	21 to 34	35 to 44	45 to 54	55 to 64	65 to 75 +
1. Cigar only	3. 7	6. 5	4. 7	6. 7	9. 3
2. Pipe only	4. 3	3. 5	3. 0	3. 2	3. 6
3. Pipe and cigar	3. 8	3. 3	5. 2	4. 4	6. 9
4. Cigarette only	28. 8	29. 0	27. 1	24. 3	13. 6
5. Cigarette and cigar	6. 8	10. 4	5. 5	5. 2	4. 2
6. Cigarette and pipe	6. 6	4. 4	5. 6	4. 0	3. 8
7. Cigarette, pipe, and cigar	5. 8	4. 8	5. 0	4. 0	1. 4
8 Nonsmoker	40. 2	38. 1	43. 9	48. 2	57. 2
Total	100. 0	100. 0	100. 0	100. 0	100. 0
Number of persons in sample	1, 009	528	523	405	388
Total pipe users	20. 5	16. 0	18. 8	15. 6	15. 7
Total cigar users	20. 1	25. 0	20. 4	20. 3	21. 8
Total cigarette users	48. 1	48. 6	43. 3	37. 5	23. 0

Source: U.S. Department of Health, Education, and Welfare (*100*).

TABLE 3.—*Percent distribution of British male smokers aged 25 and older by type of tobacco used for the years 1965, 1968, and 1971*

Forms used	1965	1968	1971
1. Cigars only	1. 9	2. 8	3. 3
2. Pipe only	5. 1	5. 6	5. 9
3. Cigarettes only	46. 8	45. 7	40. 8
4. Cigarettes and pipe	8. 0	7. 0	6. 1
5. Mixed smokers	7. 5	9. 1	8. 4
6. Nonsmokers	30. 7	29. 9	35. 4
Total	100. 0	100. 0	100. 0
Number of persons in sample	3, 576	3, 566	3, 594
Total pipe users	13. 9	14. 3	13. 3
Total cigar	9. 0	11. 7	11. 3
Total cigarette	67. 6	67. 6	61. 6

Source: Todd, G. F. (*94*).

The Definition and Processing of Cigars, Cigarettes, and Pipe Tobaccos

Cigarettes

The U.S. Government has defined tobacco products for tax purposes. Cigarettes are defined as "(1) Any roll of tobacco wrapped in paper or in any substance not containing tobacco, and (2) any roll of tobacco wrapped in any substance containing tobacco which, because of its appearance, the type of tobacco used in the filler, or its packaging and labeling, is likely to be offered to, or purchased by, consumers as a cigarette described in subparagraph (1)." Cigarettes are further classified by size, but virtually all cigarettes sold in the United States are "small cigarettes" which by definition weigh "not more than 3 pounds per thousand" which is not more than 1.361 grams per cigarette (*96*).

American brands of cigarettes contain blends of different grades of Virginia, Burley, Maryland, and oriental tobaccos. Several varieties of cigarette tobaccos are flue-cured. In this process, tobacco leaves are cured in closed barns where the temperature is progressively raised over a period of several days. This results in "color setting," fixing, and drying of the leaf. The most conspicuous change is the conversion of starch into simpler sugars and suppression of oxidative reactions. Flue-cured tobaccos produce an acidic smoke of light aroma (*35*, *112*).

Cigars

Cigars have been defined for tax purposes as: "Any roll of tobacco wrapped in leaf tobacco or in any substance containing tobacco (other than any roll of tobacco which is a cigarette within the meaning of subparagraph (2) of the definition for cigarette)" (*112*). In order to clarify the meaning of "substance containing tobacco" the Treasury department has stated that, "The wrapper must (1) contain a significant proportion of natural tobacco; (2) be within the range of colors normally found in natural leaf tobacco; (3) have some of the other characteristics of the tobaccos from which produced; e.g., nicotine content, pH, taste, and aroma; and (4) not be so changed in the reconstitution process that it loses all the tobacco characteristics" (*102*). Further, "To be a cigar, the filler must be substantially of tobaccos unlike those in ordinary cigarettes and must not have any added flavoring which would cause the product to have the taste or aroma generally attributed to cigarettes. The fact that a product does