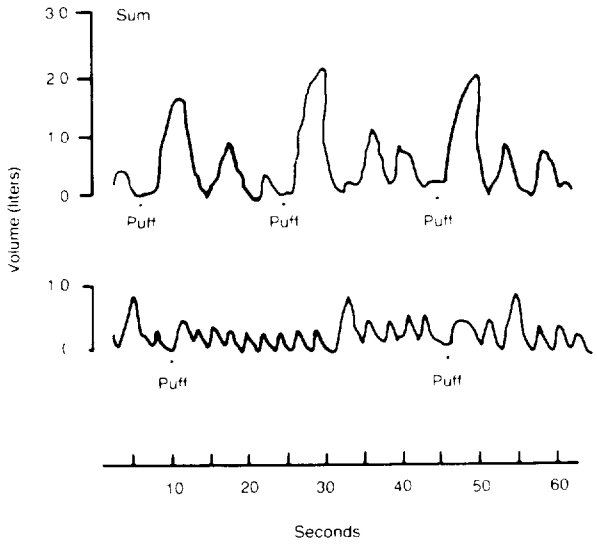

FIGURE 3.—Pattern of inhalation of cigarette smoke mixed with air, in two smokers

SOURCE: Modified from Tobin et al. (1982b).

al. 1980a), 450 to 485 ml (Guillerm and Radziszewski 1978), 389 to 1,136 ml (Adams et al. 1983), 750 to 2,000 ml (Rawbone et al. 1978), and 170 to 1,970 ml (Tobin et al. 1982b). A major factor in the discrepancies between these studies is probably the inaccuracies inherent in some of the methods employed in the measurements, as discussed by Tobin and Sackner (1982). When inhalation volumes are standardized for body size by relating them to vital capacity, marked interindividual variation is still observed (Figure 3), with inhalational volumes ranging from 9 to 47 percent of the vital capacity and a group mean value of 20 percent (Tobin et al. 1982b). Smokers show considerable variation in inhaled volumes while smoking a single cigarette. The volume of inhalation bears no relationship to cigarette consumption in terms of pack-years (Tobin et al. 1982b). Similarly, duration of inhalation shows considerable variation between subjects, with mean individual values ranging from 1.7 to 7.3 seconds (Adams et al. 1983; Tobin et al. 1982b). Repeat measurements at intervals of up to 10 months apart indicate that individual subjects tend to maintain a fairly constant inhalation volume, duration of inhalation, and associated breathhold time (Tobin et al. 1982b; Adams et al. 1983).

The pattern of cigarette smoking shows a wide degree of intersubject variability, including differences in the number of puffs, puff volume, holding pause in the mouth, exhalation of smoke from the mouth before inhalation, partitioning of airflow between the nose and mouth, and volume and duration of inhalation. Given this degree of variation, it is not surprising that smokers might show wide differences in their individual susceptibilities to lung injury. In a study relating inhalation volume—standardized for vital capacity—to the time-volume and flow-volume components of a forced vital capacity maneuver, no significant correlation was observed (Tobin et al. 1982b). Although this lack of a relationship might be interpreted as indicating that the pattern of smoking is unimportant in the development of lung disease, it may also reflect the fact that pulmonary function was normal or near normal in the majority of subjects and that the study was of a cross-sectional design.

Use of Additives in Low Tar and Nicotine Cigarettes

The nominal tar and nicotine yield of cigarettes has continually decreased since the time of the initial reports linking smoking with lung cancer (USDHHS 1981). In 1954, the average tar yield per cigarette was 38 mg, and in 1980 it was less than 14 mg. Initially, tar reduction was achieved by decreasing the cigarette tobacco content or removing tar by smoke filtration, both of which probably resulted in a lower smoke exposure. Since 1971, the reduction in tar yield has exceeded the relative reduction in the weight of tobacco per cigarette; this difference has increased since 1975 (USDHHS 1981). Manufacturing technology has progressed beyond simple reduction in tobacco content: the yield and composition of smoke can be modified by genetic modification of the tobacco leaf (Tso 1972a), changes in its cultivation and processing (Tso 1972b), changes in the porosity of cigarette paper, and alterations in filter design (Kozlowski et al. 1980b).

When initially introduced, lower yield cigarettes lacked palatability and acceptability. Advertisements for the current low tar and nicotine cigarettes emphasize their flavor, presumably achieved by the use of additives in the processing of the tobacco. Additives employed may include artificial tobacco substitutes (Freedman and Fletcher 1976), flavor extracts of tobacco and other plants, exogenous enzymes, powdered cocoa (Gori 1977), and other synthetic flavoring substances. Perhaps more additives are being used in the new lower tar and nicotine cigarettes than in the older brands, and new agents may also be in use. Some of the substances, such as powdered cocoa, have been shown to further increase the carcinogenicity of tar (Gori 1977), and others may result in increased or new and different health risks. The pyrolytic products of these additive agents may

produce novel toxic constituents. A characterization of the chemical composition and adverse biologic potential of these additives is urgently required, but is currently impossible because cigarette companies are not required to reveal what additives they employ in the manufacture of tobacco (USDHHS 1981). No government agency is empowered with supervisory authority in the manufacture of tobacco products. With this lack of basic information and the usually prolonged latent period before manifestation of the adverse effects of smoking, it is likely that a long time period will elapse before we know the hazards of the new cigarettes in current use.

Research Recommendations

1. Longitudinal epidemiologic studies are needed to determine the risk for pulmonary symptoms and dysfunction in smokers of cigarettes with the low tar and nicotine yields found in currently popular brands.
2. Further research is needed to determine the relative potency of high and low tar and nicotine cigarettes in inducing elastase release and producing functional inhibition of α_1-antitrypsin activity.
3. Development of an animal model of cigarette-smoke-induced emphysema would be advantageous in determining the relative risk of lung injury of cigarettes of different composition.
4. More information is required on the smoking behavior of smokers who have voluntarily switched from high to low tar and nicotine cigarettes.
5. The role of cigarette tar, as opposed to nicotine content, in determining smoking behavior needs to be defined.
6. Standard research cigarettes of varying tar and nicotine contents that are palatable and acceptable to smokers need to be developed.
7. The role of variation in smoking behavior in determining susceptibility to lung injury needs to be defined. Studies are required to determine the effect of smoking patterns on the distribution and penetration of the smoke aerosol into the lung.
8. More information is needed on the composition and adverse biologic effects of flavor additives in cigarettes and their pyrolytic products.

Summary and Conclusions

1. The recommendation for those who cannot quit to switch to smoking cigarette brands with low tar and nicotine yields, as determined by a smoking-machine, is based on the assumption that this switch will result in a reduction in the exposure of the

lung to these toxic substances. The design of the cigarette has markedly changed in recent years, and this may have resulted in machine-measured tar and nicotine yields that do not reflect the real dose to the smoker.

2. Smoking-machines that take into account compensatory changes in smoking behavior are needed. The assays could provide both an average and a range of tar and nicotine yields produced by different individual patterns of smoking.
3. Although a reduction in cigarette tar content appears to reduce the risk of cough and mucus hypersecretion, the risk of shortness of breath and airflow obstruction may not be reduced. Evidence is unavailable on the relative risks of developing COLD consequent to smoking cigarettes with the very low tar and nicotine yields of current and recently marketed brands.
4. Smokers who switch from higher to lower yield cigarettes show compensatory changes in smoking behavior: the number of puffs per cigarette is variably increased and puff volume is almost universally increased, although the number of cigarettes smoked per day and inhalation volume are generally unchanged. Full compensation of dose for cigarettes with lower yields is generally not achieved.
5. Nicotine has long been regarded as the primary reinforcer of cigarette smoking, but tar content may also be important in determining smoking behavior.
6. Depth and duration of inhalation are among the most important factors in determining the relative concentration of smoke constituents that reach the lung. Considerable interindividual variation exists between smokers with respect to the volume and duration of inhalation. This variation is likely to be an important factor in determining the varying susceptibility of smokers to the development of lung disease.
7. Production of low tar and nicotine cigarettes has progressed beyond simple reduction in tobacco content. Additives such as artificial tobacco substitutes and flavoring extracts have been used. The identity, chemical composition, and adverse biological potential of these additives are unknown at present.

References

ADAMS, L., LEE, C., RAWBONE, R., GUZ, A. Patterns of smoking: Measurement and variability in asymptomatic smokers. *Clinical Science* 65(4): 383–392, October 1983.

ADAMS, P.I. Changes in personal smoking habits brought about by changes in cigarette smoke yield. In: *Proceedings of the Sixth International Tobacco Scientific Congress*, Tokyo, November 14–20, 1976. Tokyo, The Japan Tobacco and Salt Public Corporation, 1977, pp. 102–108.

ADAMS, P.I. The influence of cigarette smoke yields on smoking habits. In: Thornton, R.E. (Editor). *Smoking Behaviour, Physiological and Psychological Influences.* Edinburgh, Churchill Livingstone, 1978, pp. 349–360.

AMERICAN CANCER SOCIETY. U.S. tar/nicotine levels dropping. *World Smoking and Health* 6(2): 47, Summer 1981.

ASHTON, H., STEPNEY, R., THOMPSON, J.W. Self-titration by cigarette smokers. *British Medical Journal* 2(6186): 357–360, August 11, 1979.

ASHTON, H., WATSON, D.W. Puffing frequency and nicotine intake in cigarette smokers. *British Medical Journal* 3(5724): 679–681, September 19, 1970.

BÄTTIG, K., BUZZI, R., NIL, R. Smoke yield of cigarettes and puffing behavior in men and women. *Psychopharmacology* 76(2): 139–148, February 1982.

BECK, G.J., DOYLE, C.A., SCHACHTER, E.N. Smoking and lung function. *American Review of Respiratory Disease* 123(2): 149–155, February 1981.

BENOWITZ, N.L., HALL, S.M., HERNING, R.I., JACOB, P., III, JONES, R.T., OSMAN, A.-L. Smokers of low-yield cigarettes do not consume less nicotine. *New England Journal of Medicine* 309(3): 139–142, July 21, 1983.

BLUE, M.-L., JANOFF, A. Possible mechanisms of emphysema in cigarette smokers. Release of elastase from human polymorpho-nuclear leukocytes by cigarette smoke condensate in vitro. *American Review of Respiratory Disease* 117(2): 317–325, February 1978.

BOSSE, R., COSTA, P., COHEN, M., PODOLSKY, S. Age, smoking inhalation and pulmonary function. *Archives of Environmental Health* 30(10): 495–498, October 1975.

COHEN, A.B., JAMES, H.L. Reduction of the elastase inhibitory capacity of alpha$_1$-antitrypsin by peroxides in cigarette smoke. An analysis of brands and filters. *American Review of Respiratory Disease* 126(1): 25–30, July 1982.

COHEN, S.I., PERKINS, N.M., URY, H.K., GOLDSMITH, J.R. Carbon monoxide uptake in cigarette smoking. *Archives of Environmental Health* 22(1): 55–60, January 1971.

COMSTOCK, G.W., BROWNLOW, W.J., STONE, R.W., SARTWELL, P.E. Cigarette smoking and changes in respiratory findings. *Archives of Environmental Health* 21(1): 50–57, July 1970.

CREIGHTON, D.E., LEWIS, P.H. The effect of different cigarettes on human smoking patterns. In: Thornton, R.E. (Editor). *Smoking Behaviour, Physiological and Psychological Influences.* Edinburgh, Churchill Livingstone, 1978a, pp. 289–300.

CREIGHTON, D.E., LEWIS, P.H. The effect of smoking pattern on smoke deliveries. In: Thornton, R.E. (Editor). *Smoking Behaviour, Physiological and Psychological Influences.* Edinburgh, Churchill Livingstone, 1978b, pp. 301–314.

DA SILVA, A.M.T., HAMOSH, P. Effect of smoking a single cigarette on the "small airways." *Journal of Applied Physiology* 34(3): 361–365, March 1973.

DA SILVA, A.M.T., HAMOSH, P. Airways response to inhaled tobacco smoke: Time course, dose dependence and effect of volume history. *Respiration* 41(2): 96–105, 1981.

DEAN, G., LEE, P.N., TODD, G.F., WICKEN, A.J., SPARKS, D.N. Factors related to respiratory and cardiovascular symptoms in the United Kingdom. *Journal of Epidemiology and Community Health* 32(2): 86–96, June 1978.

FERRIS, B.G., Jr., CHEN, H., PULEO, S., MURPHY, R.L.H., Jr. Chronic nonspecific respiratory disease in Berlin, New Hampshire, 1967 to 1973. A further follow-up study. *American Review of Respiratory Disease* 113(4): 475–485, April 1976.

FEYERABEND, C., HIGENBOTTAM, T., RUSSELL, M.A.H. Nicotine concentrations in urine and saliva of smokers and non-smokers. *British Medical Journal* 284(6321): 1002–1004, April 3, 1982.

FINNEGAN, J.K., LARSON, P.S., HAAG, H.B. The role of nicotine in the cigarette habit. *Science* 102(2639): 94–96, July 27, 1945.

FLETCHER, C., PETO, R., TINKER, C., SPEIZER, F.E. *The Natural History of Chronic Bronchitis and Emphysema: An Eight-Year Study of Early Chronic Obstructive Lung Disease in Working Men in London.* New York, Oxford University Press, 1976, 272 pp.

FORBES, W.F., ROBINSON, J.C., HANLEY, J.A., COLBURN, H.N. Studies on the nicotine exposure of individual smokers. I. Changes in mouth-level exposure to nicotine on switching to lower nicotine cigarettes. *International Journal of the Addictions* 11(6): 933–950, 1976.

FREEDMAN, S., FLETCHER, C.M. Changes of smoking habits and cough in men smoking cigarettes with 30% NSM tobacco substitute. *British Medical Journal* 1(6023): 1427–1430, June 12, 1976.

FRITH, C.D. The effect of varying the nicotine content of cigarettes on human smoking behaviour. *Psychopharmacologia* 19(2): 188–192, 1971.

GARFINKEL, L. Changes in the cigarette consumption of smokers in relation to changes in tar/nicotine content of cigarettes smoked. *American Journal of Public Health* 69(12): 1274–1276, December 1979.

GOLDFARB, T., GRITZ, E.R., JARVICK, M.E., STOLERMAN, I.P. Reactions to cigarettes as a function of nicotine and "tar." *Clinical Pharmacology and Therapeutics* 19(6): 767–772, June 1976.

GORI, G.B. Low-risk cigarettes: A prescription. Low-toxicity cigarettes hold significant promise in the prevention of diseases related to smoking. *Science* 194(4271): 1243–1246, December 17, 1976.

GORI, G.B. (Editor). *Toward Less Hazardous Cigarettes. The Third Set of Experimental Cigarettes.* U.S. Department of Health, Education, and Welfare, Public Health Service, National Institutes of Health, National Cancer Institute, Smoking and Health Program Report No. 3, DHEW Publication No. (NIH)77–1280, 1977, 152 pp.

GORI, G.B., LYNCH, C.J. Toward less hazardous cigarettes. Current advances. *Journal of the American Medical Association* 240(12): 1255–1259, September 15, 1978.

GRIFFITHS, R.R., HENNINGFIELD, J.E., BIGELOW, G.E. Human cigarette smoking: Manipulation of number of puffs per bout, interbout interval and nicotine dose. *Journal of Pharmacology and Experimental Therapeutics* 220(2): 256–265, 1981.

GUILLERM, R., RADZISZEWSKI, E. Analysis of smoking pattern including intake of carbon monoxide and influences of changes in cigarette design. In: Thornton, R.E. (Editor). *Smoking Behaviour, Physiological and Psychological Influences.* Edinburgh, Churchill Livingstone, 1978, pp. 361–370.

GUYATT, A.R., McBRIDE, M.J., KIRKHAM, A.J.T., CUMMING, G. Smoking and ventilatory response of man to cigarettes of different nicotine content. *Clinical Science* 65(3): 3, September 1983.

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

HAWTHORNE, V.M., FRY, J.S. Smoking and health: The association between smoking behaviour, total mortality, and cardiorespiratory disease in west central Scotland. *Journal of Epidemiology and Community Health* 32(4): 260–266, December 1978.

HEALTH DEPARTMENT OF THE UNITED KINGDOM. *Tar and Nicotine Yield of Cigarettes.* London, Department of Health and Social Security, January 1976.

HENNINGFIELD, J.E., GRIFFITHS, R.R. Effects of ventilated cigarette holders on cigarette smoking by humans. *Psychopharmacology* 68(2): 115–119, May 1980.

HERNING, R.I., JONES, R.T., BACHMAN, J., MINES, A.H. Puff volume increases when low-nicotine cigarettes are smoked. *British Medical Journal* 283(6285): 187–189, July 18, 1981.

HERNING, R.I., JONES, R.T., BENOWITZ, N.L., MINES, A.H. How a cigarette is smoked determines blood nicotine levels. *Clinical Pharmacology and Therapeutics* 33(1): 84–90, January 1983.

HIGENBOTTAM, T., FEYERABEND, C., CLARK, T.J.H. Cigarette smoke inhalation and the acute airway response. *Thorax* 35(4): 246–254, April 1980a.

HIGENBOTTAM, T., SHIPLEY, M.J., CLARK, T.J.H., ROSE, G. Lung function and symptoms of cigarette smokers related to tar yield and number of cigarettes smoked. *Lancet* 1(8165): 409–412, February 23, 1980b.

HILL, P., MARQUARDT, H. Plasma and urine changes after smoking different brands of cigarettes. *Clinical Pharmacology and Therapeutics* 27(5): 652–658, May 1980.

HOFFMANN, D., TSO, T.C., GORI, G.B. The less harmful cigarette. *Preventive Medicine* 9(2): 287–296, March 1980.

JAFFE, J.H., KANZLER, M., FRIEDMAN, L., KAPLAN, T. Money and health messages as incentives for smoking low/tar nicotine cigarettes: Changes in consumption and exhaled carbon monoxide. *British Journal of Addiction* 77(1): 21–34, March 1982.

JAFFE, J.H., KANZLER, M., FRIEDMAN, L., STUNKARD, A.J., VEREBEY, K. Carbon monoxide and thiocyanate levels in low tar/nicotine smokers. *Addictive Behaviors* 6(4): 337–343, 1981.

JANOFF, A., CARP, H., LEE, D.K., DREW, R.T. Cigarette smoke inhalation decreases alpha$_1$-antitrypsin activity in rat lung. *Science* 206(4424): 1313–1314, December 14, 1979.

JARVIK, M.E., POPEK, P., SCHNEIDER, N.G., BAER-WEISS, V., GRITZ, E.R. Can cigarette size and nicotine content influence smoking and puffing rates? *Psychopharmacology* 58(3): 303–306, 1978.

KOZLOWSKI, L.T. Tar and nicotine delivery of cigarettes. *Journal of the American Medical Association* 245(2): 158–159, January 9, 1981.

KOZLOWSKI, L.T. Physical indicators of actual tar and nicotine yields of cigarettes. In: Grabowski, J., Bell, C. (Editors). *Measurement in the Analysis and Treatment of Smoking Behavior.* National Institute on Drug Abuse Research Monograph 48. Department of Health and Human Services, Public Health Service, Alcohol, Drug Abuse, and Mental Health Administration, 1983.

KOZLOWSKI, L.T., FRECKER, R.C., KHOUW, V., POPE, M.A. The misuse of "less-harzardous" cigarettes and its detection: Hole-blocking of ventilated filters. *American Journal of Public Health* 70(11): 1202–1203, November 1980a.

KOZLOWSKI, L.T., FRECKER, R.C., LEI, H. Nicotine yields of cigarettes, plasma nicotine in smokers, and public health. *Preventive Medicine* 11(2): 240–244, March 1982.

KOZLOWSKI, L.T., RICKERT, W.S., ROBINSON, J.C., GRUNBERG, N.E. Have tar and nicotine yields of cigarettes changed? *Science* 209(4464): 1550–1551, September 26, 1980b.

LEE, P.N., GARFINKEL, L. Mortality and type of cigarette smoked. *Journal of Epidemiology and Community Health* 35(1): 16–22, March 1981.

MOROSCO, G.J., GOERINGER, G.C. Pancreatic elastase and serum alpha$_1$-antitrypsin levels in beagle dogs smoking high- and low-nicotine cigarettes: Possible mechanism of pancreatic cancer in cigarette smokers. *Journal of Toxicology and Environmental Health* 5(5): 879–890, September 1979.

NADEL, J.A., COMROE, J.H., Jr. Acute effects of inhalation of cigarette smoke on airway conductance. *Journal of Applied Physiology* 16(4): 713–716, July 1961.

PETO, R., SPEIZER, F.E., COCHRANE, A.L., MOORE, F., FLETCHER, C.M., TINKER, C.M., HIGGINS, I.T.T., GRAY, R.G., RICHARDS, S.M., GILLILAND, J., NORMAN-SMITH, B. The relevance in adults of air-flow obstruction, but not of mucus hypersecretion, to mortality from chronic lung disease. *American Review of Respiratory Disease* 128(3): 491–500, September 1983.

PILLSBURY, H.C., BRIGHT, C.C., O'CONNOR, K.J., IRISH, F.W. Tar and nicotine in cigarette smoke. *Journal of the Association of Official Analytical Chemists* 52(3): 458–462, May 1969.

RAWBONE, R.G., MURPHY, K., TATE, M.E., KANE, S.J. The analysis of smoking parameters: Inhalation and absorption of tobacco smoke in studies of human smoking behaviour. In: Thornton, R.E. (Editor). *Smoking Behaviour, Physiological and Psychological Influences.* Edinburgh, Churchill Livingstone, 1978, pp. 171–194.

REES, P.J., AYRES, J.G., CHOWIENCZYK, P.J., CLARK, T.J.H. Irritant effects of cigarette and cigar smoke. *Lancet* 2(8306): 1015–1017, November 6, 1982.

RICKERT, W.S., ROBINSON, J.C., YOUNG, J.C. Estimating the hazards of "less hazardous" cigarettes. I. Tar, nicotine, carbon monoxide, acrolein, hydrogen cyanide, and total aldehyde deliveries of Canadian cigarettes. *Journal of Toxicology and Environmental Health* 6(2): 351–365, March 1980.

RIMINGTON, J. Phlegm and filters. *British Medical Journal* 2(5808): 262–264, April 29, 1972.

RIMINGTON, J. Cigarette smokers' chronic bronchitis: Inhalers and non-inhalers compared. *British Journal of Diseases of the Chest* 68: 161–166, July 1974.

RIMINGTON, J. The effect of filters on the incidence of lung cancer in cigarette smokers. *Environmental Research* 24(1): 162–166, February 1981.

ROBERTSON, D.G., WARRELL, D.A., NEWTON-HOWES, J.S., FLETCHER, C.M. Bronchial reactivity to cigarette and cigar smoke. *British Medical Journal* 3(5665): 269–271, August 2, 1969.

ROBINSON, J.C., FORBES, W.F. Studies on the nicotine exposure of individual smokers. II. An analysis of smoking habits during a one-week period. *International Journal of the Addictions* 15(6): 889–905, 1980.

RODRIGUEZ, R.J., WHITE, R.R., SENIOR, R.M., LEVINE, E.A. Elastase release from human alveolar macrophages: Comparison between smokers and nonsmokers. *Science* 198(4314): 313–314, October 21, 1977.

RUSSELL, M.A.H. Low tar-medium nicotine cigarettes: A new approach to safer smoking. *British Medical Journal* 2: 1430–1433, 1976.

RUSSELL, M.A.H., JARVIS, M., IYER, R., FEYERABEND, C. Relation of nicotine yield of cigarettes to blood nicotine concentrations in smokers. *British Medical Journal* 280(6219): 972–976, April 5, 1980.

RUSSELL, M.A.H., WILSON, C., PATEL, U.A., COLE, P.V., FEYERABEND, C. Comparison of effect on tobacco consumption and carbon monoxide absorption of changing to high and low nicotine cigarettes. *British Medical Journal* 4(5891): 512–516, December 1, 1973.

RUSSELL, M.A.H., WILSON, C., PATEL, U.A., FEYERABEND, C., COLE, P.V. Plasma nicotine levels after smoking cigarettes with high, medium, and low nicotine yields. *British Medical Journal* 2(5968): 414–416, May 24, 1975.

SCHENKER, M.B., SAMET, J.M., SPEIZER, F.E. Effect of cigarette tar content and smoking habits on respiratory symptoms in women. *American Review of Respiratory Disease* 125(6): 684–690, June 1982.

SCHULZ, W., SEEHOFER, F. Smoking behaviour in Germany—The analysis of cigarette butts (KIPA). In: Thornton, R.E. (Editor). *Smoking Behaviour, Physiological and Psychological Influences.* Edinburgh, Churchill Livingstone, 1978, pp. 259–276.

SPARROW, D., STEFOS, T., BOSSE, R., WEISS, S.T. The relationship of tar content to decline in pulmonary function in cigarette smokers. *American Review of Respiratory Disease* 127(1): 56–58, January 1983.

STEPNEY, R. Would a medium-nicotine, low-tar cigarette be less hazardous to health? *British Medical Journal* 283(6302): 1292–1296, November 14, 1981.

STEPNEY, R. Are smokers' self-reports of inhalation a useful measure of smoke exposure? *Journal of Epidemiology and Community Health* 36(2): 109–112, June 1982.

STERLING, G.M. Mechanism of bronchoconstriction caused by cigarette smoking. *British Medical Journal* 3(5560): 275–277, July 29, 1967.

SUTTON, S.R., FEYERABEND, C., COLE, P.V., RUSSELL, M.A.H. Adjustment of smokers to dilution of tobacco smoke by ventilated cigarette holders. *Clinical Pharmacology and Therapeutics* 24(4): 395–405, October 1978.

SUTTON, S.R., RUSSELL, M.A.H., IYER, R., FEYERABEND, C., SALOOJEE, Y. Relationship between cigarette yields, puffing patterns, and smoke intake: Evidence for tar compensation? *British Medical Journal* 285(6342): 600–603, August 28, 1982.

TASHKIN, D.P., CLARK, V.A., COULSON, A.H., BOURQUE, L.B., SIMMONS, M., REEMS, C., DETELS, R., ROKAW, S. Comparison of lung function in young nonsmokers and smokers before and after initiation of the smoking habit: A prospective study. *American Review of Respiratory Disease* 128(1): 12–16, July 1983.

TOBIN, M.J., JENOURI, G.A., SACKNER, M.A. Subjective and objective measurement of cigarette smoke inhalation. *Chest* 82(6): 695–700, December 1982a.

TOBIN, M.J., SACKNER, M.A. Monitoring smoking patterns of low and high tar cigarettes with inductive plethysmography. *American Review of Respiratory Disease* 126(2): 258–264, August 1982.

TOBIN, M.J., SCHNEIDER, A.W., SACKNER. M.A. Breathing pattern during and after smoking cigarettes. *Clinical Science* 63(5): 473–483, November 1982b.

TRAVIS, J., BEATTY, K., WONG, P.S., MATHESON, N.R. Oxidation of alpha$_1$-proteinase inhibitor as a major, contributing factor in the development of pulmonary emphysema. *Bulletin Europeen de Physiopathologie Respiratoire* 16(Supplement): 341–351, 1980.

TSO, T.C. Manipulation of leaf characteristics through production—Role of agriculture in health-related tobacco research. *Journal of the National Cancer Institute* 48(6): 1811–1119, June 1972a.

TSO, T.C. The potential for producing safer cigarette tobacco. *Agricultural Science Review* 10(3): 1–10, Third Quarter, 1972b.

TURNER, J.A.M., SILLETT, R.W., BALL, K.P. Some effects of changing to low-tar and low-nicotine cigarettes. *Lancet* 2(7883): 737–739, September 28, 1974.

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES. *The Health Consequences of Smoking: The Changing Cigarette: A Report of the Surgeon General.* U.S. Department of Health and Human Services, Public Health Service, Office of the Assistant Secretary for Health, Office on Smoking and Health, DHHS Publication No. (PHS)81-50156, 1981, 269 pp.

U.S. SENATE. *Reviewing Progress Made Toward the Development and Marketing of a Less Hazardous Cigarette.* Hearings Before the Consumer Subcommittee of the Committee on Commerce. Serial No. 90–52, August 23–25, 1967, 329 pp.

WALD, N. Mortality from lung cancer and coronary heart-disease in relation to changes in smoking habits. *Lancet* 1(7951): 136–138, January 17, 1976.

WALD, N., HOWARD, S., SMITH, P.G., BAILEY, A. Use of carboxyhaemoglobin levels to predict the development of diseases associated with cigarette smoking. *Thorax* 30(2): 133–139, April 1975.

WALD, N., HOWARD, S., SMITH, P.G., KJELDSEN, K. Association between atherosclerotic diseases and carboxyhaemoglobin levels in tobacco smokers. *British Medical Journal* 1(5856): 761–765, March 31, 1973.

WALD, N., IDLE, M., BAILEY, A. Carboxyhaemoglobin levels and inhaling habits in cigarette smokers. *Thorax* 33(2): 201–206, April 1978.

WALD, N., IDLE, M., BOREHAM, J., BAILEY, A. Inhaling habits among smokers of different types of cigarettes. *Thorax* 35(12): 925–928, December 1980.

WALD, N., IDLE, M., BOREHAM, J., BAILEY, A. The importance of tar and nicotine in determining cigarette smoking habits. *Journal of Epidemiology and Community Health* 35(1): 23–24, March 1981.

WALD, N., IDLE, M., SMITH, P.G., BAILEY, A. Carboxyhaemoglobin levels in smokers of filter and plain cigarettes. *Lancet* 1(8003): 110–112, January 15, 1977.

WALD, N., SMITH, P.G. Smoking tables for carbon monoxide. *Lancet* 2: 907–908, October 20, 1973.

WILEY, R.M., WICKHAM, J.E. The fabrication and application of a puff-by-puff smoking machine. *Tobacco Science* 18: 69–72, 1974.

WYNDER, E.L., KIYOHIKO, M., BEATTIE, E.J., Jr. The epidemiology of lung cancer. Recent trends. *Journal of the American Medical Association* 213(13): 2221–2228, September 28, 1970.

CHAPTER 7. PASSIVE SMOKING

480-144 O - 85 - 13

CONTENTS

Introduction

This chapter explores recent data that relate involuntary cigarette smoke exposure to the occurrence of physiologic changes, symptoms, and diseases in nonsmoking adults and children. Health effects related to fetal exposure in utero, a subject that has been extensively studied, are not discussed, although instances where such exposure may relate to potential development are pointed out. The interested reader is referred to several excellent recent reviews for a more complete treatment of this issue (USDHEW 1979; USDHHS 1980; Abel 1980; Weinberger and Weiss 1981).

Differences in Composition of Sidestream Smoke and Mainstream Smoke

Involuntary (passive) smoking is defined as the exposure of nonsmokers to tobacco combustion products from the smoking of others. Analysis of the health effects of passive smoking requires not only some knowledge of the constituents of tobacco smoke, but also some quantitation of tobacco smoke exposure. Tobacco smoke in the environment is derived from two sources: mainstream smoke and sidestream smoke. Mainstream smoke emerges into the environment after having first been drawn through the cigarette, which filters some of the active constituents. The smoke is then filtered by the smoker's own lungs, and exhaled. Sidestream smoke arises from the burning end of the cigarette and enters directly into the environment. Differences in the temperature of combustion, the degree of filtration, and the amount of tobacco consumed all lead to marked differences in the concentration of the constituents of mainstream smoke and sidestream smoke (USDHEW 1979; Sterling et al. 1982; Brunneman et al. 1978; National Academy of Sciences 1981; Rylander et al. 1984). Many potentially toxic gas phase constituents are present in higher concentration in sidestream smoke than in mainstream smoke (Brunneman et al. 1978) (Table 1), and nearly 85 percent of the smoke in a room results from sidestream smoke. Smaller amounts of smoke are contributed to the environment from the nonburning end of the cigarette by diffusion through the paper wrapping and by the smoke exhaled by the smoker. Therefore, both active and passive smokers may be similarly exposed to sidestream smoke. Mainstream smoke is inhaled directly into the lungs and is diluted only by the volume of air breathed in by the smoker when he or she inhales. Sidestream smoke is generally diluted in a considerably larger volume of air. Thus, passive smokers are subjected to a quantitatively smaller and qualitatively different smoke exposure than active smokers. The quantification of the exposure of a passive smoker to these sidestream smoke constituents is often difficult. Factors such as the type and number of cigarettes burned, the size of

the room, the ventilation rate, and the smoke residence time are all important variables in determining levels of exposure. Thus, no single variable accurately characterizes exposure to smoke constituents.

Repace and Lowrey (1980, 1982, 1983) have shown that, to a reasonable approximation, exposure to the particulate phase is predicted by the ratio of the smoker density to the effective ventilation rate of the area in which the smokers are located.

Measurement of Exposure

Levels of indoor byproducts of tobacco smoke, with measurements made under realistic exposure conditions, are presented in Table 2. Among the constituents that have been measured, nitrogen oxide, carbon monoxide, nicotine and respirable particulates, nitrosamines, and aldehydes have been shown to be significantly elevated indoors as a result of cigarette smoking. Nitrogen oxide is rapidly oxidized to nitrogen dioxide (NO_2) in air, and reaches equilibrium with outdoor levels of NO_2, provided there are suitable air exchange rates and no other indoor sources, such as a gas stove. The particulate concentration indoors clearly increases with increasing numbers of smokers, although the background level is determined by the outdoor level. The conclusions from the few studies that actually measure ventilation rates during exposure suggest that under "normal" air circulation conditions, carbon monoxide (CO) levels will be relatively low, but still may exceed the ambient air quality standard of 9 ppm (NIOSH 1971). However, even modest reductions in ventilation rates can lead to CO accumulation.

A variety of measures have been utilized to quantify the nonsmoker's exposure to tobacco smoke. No single measure has been uniformly accepted as characterizing the level of smoke. Nicotine is the most tobacco-specific of these measures, but it is relatively complicated and expensive to measure and settles out of the air with the particulate phase, making it a poor measure of gas phase constituents. In addition, nicotine may rapidly deposit on surfaces and subsequently evaporate into the environment (Rylander et al. 1984), making it a poor measure of acute smoke exposure levels. Measurements of total particulate matter are a broader measure of smoke exposure, particularly if the measurements are limited to particles in the respirable range and to environments without other major sources of respirable particles. The smoke particles also settle out of the air and therefore may not reflect the levels of gas phase constituents, and a wide variety of other dusts may contribute particulates to the air, particularly in the occupational setting. A number of authors have measured levels of CO. This measurement is relatively simple and a measure of absorption (carboxyhemoglobin)

TABLE 1.—Ratio of selected constituents in sidestream smoke (SS) to mainstream smoke (MS)

Gas phase constituents	MS	SS/MS ratio	Particulate phase constituents	MS	SS/MS ratio
Carbon dioxide	20–60 mg	8.1	Tar	1–40 mg	1.3
Carbon monoxide	10–20 mg	2.5	Water	1–4 mg	2.4
Methane	1.3 mg	3.1	Toluene	108 μg	5.6
Acetylene	27 μg	0.8	Phenol	20–150 μg	2.6
Ammonia	80 μg	73.0	Methylnaphthalene	2.2 μg	28
Hydrogen cyanide	430 μg	0.25	Pyrene	50–200 μg	3.6
Methylfuran	20 μg	3.4	Benzo[a]pyrene	20–40 μg	3.4
Acetonitrile	120 μg	3.9	Aniline	360 μg	30
Pyridine	32 μg	10.0	Nicotine	1.0–2.5 mg	2.7
Dimethylnitrosamine	10–65 μg	52.0	2-Naphthylamine	2 ng	39

Adapted from U.S. Department of Health, Education, and Welfare (*1979*).

TABLE 2a.—Acrolein measured under realistic conditions

Study	Type of premises	Occupancy	Ventilation	Monitoring conditions	Levels	
					Mean	Range
Badre et al. (*1978*)	Cafes	Varied	Not given	100 mL samples		0.03–0.10 mg/m³
	Room	18 smokers	Not given	100 mL samples	0.185 mg/m³	
	Hospital lobby	12 to 30 smokers	Not given	100 mL samples	0.02 mg/m³	
	2 train compartments	2 to 3 smokers	Not given	100 mL samples		0.02–0.12 mg/m³
	Car	3 smokers	Natural, open	100 mL samples	0.03 mg/m³	
		2 smokers	Natural, closed	100 mL samples	0.30 mg/m³	
Fischer et al. (*1978*) and Weber et al. (*1979*)	Restaurant	50–80/470 m³	Mechanical	27 × 30 min samples	7 ppb	
	Restaurant	60–100/440 m³	Natural	29 × 30 min samples	8 ppb	
	Bar	30–40/50 m³	Natural, open	28 × 30 min samples	10 ppb	
	Cafeteria	80–150/574 m³	11 changes/hr	24 × 30 min samples	6 ppb (5 ppb nonsmoking section)	

TABLE 2b.—Aromatic hydrocarbons measured under realistic conditions

Study	Type of premises	Occupancy	Ventilation	Monitoring conditions	Levels		Nonsmoking controls	
					Mean	Range	Mean	Range
					Benzene (mg/m³)			
Badre et al. (*1978*)	Cafes	Varied	Not given	100 mL samples		0.05–0.15		
	Room	18 smokers	Not given	100 mL samples	0.109			
	Train compartments	2 to 3 smokers	Not given	100 mL samples		0.02–0.10		
	Car	3 smokers	Natural, open	100 mL samples	0.04			
		2 smokers	Natural, closed	100 mL samples	0.15			
					Toulene (mg/m³)			
	Cafes	Varied	Not given	100 mL samples		0.04–1.04		
	Room	18 smokers	Not given	100 mL samples	0.215			
	Train compartments	2 to 3 smokers	Not given	100 mL samples	1.87			
	Car	2 smokers	Natural, closed	100 mL samples	0.50			
					Benzo[a]pyrene (ng/m³)			
Elliott and Rowe (*1975*)	Arena	8,647-10,786 people	Mechanical	Not given	7.1			
		12,000–12,844 people	Mechanical	Not given	9.9			
		13,000–14,277 people	Mechanical	Not given	21.7			
				Separate non-activity days			0.69	
Galuskinova (*1964*)	Restaurant	Not given	Not given	20 days in summer	6.2			
				18 days in the fall		28.2–144		

TABLE 2b.—Continued

Study	Type of premises	Occupancy	Ventilation	Monitoring conditions	Levels		Nonsmoking controls	
					Mean	Range	Mean	Range
Just et al. (*1972*)	Coffee houses	Not given	Not given	6 hr continuous	0.25–10.1		4.0–9.3 (outdoors)	
					Benzo[e]pyrene (ng/m³)			
					3.3–23.4		3.0–5.1 (outdoors)	
					Benzo[ghi]perylene (ng/m³)			
					5.9–10.5		6.9–13.8 (outdoors)	
					Perylene (ng/m³)			
					0.7–1.3		0.1–1.7 (outdoors)	
					Pyrene (ng/m³)			
					4.1–9.4		2.8–7.0 (outdoors)	
					Anthanthrene (ng/m³)			
					0.5–1.9		0.5–1.8 (outdoors)	
					Coronene (ng/m³)			
					0.5–1.2		1.0–2.8	
					Phenols (μ/m³)			
					7.4–11.5			
					Benzo[a]pyrene (ng/m³)			
Perry (*1973*)[a]	14 public places	Not given	Not given	Samples, 5 outdoor locations	<20–760			<20–43

TABLE 2c.—Carbon monoxide measured under realistic conditions

Study	Type of premises	Occupancy	Ventilation	Monitoring conditions	Levels (ppm)		Nonsmoking controls (ppm)	
					Mean	Range	Mean	Range
Badre et al. (*1978*)	6 cafes	Varied	Not given	20 min samples		2–23	(outdoors)	0–15
	Room	18 smokers	Not given	20 min samples	50		0 (outdoors)	
	Hospital lobby	12 to 30 smokers	Not given	20 min samples	5			
	2 train compartments	2 to 3 smokers	Not given	20 min samples		4–5		
	Car	3 smokers	Natural, open	20 min samples	14		0 (outdoors)	
		2 smokers	Natural, closed	20 min samples	20		0 (outdoors)	
Cano et al. (*1970*)	Submarines 66 m³	157 cigarettes per day	Yes		<40 ppm			
		94–103 cigarettes per day	Yes		<40 ppm			
Chappell and Parker (*1977*)	10 offices	Not given	Values not given	17 × 2–3 min samples	2.5 ± 1.0	1.5–4.5	2.5 ± 1.0 (outdoors)	1.5–4.5
	15 restaurants	Not given	Values not given	17 × 2–3 min samples	4.0 ± 2.5	1.0–9.5	2.5 ± 1.5 (outdoors)	1.0–5.0
	14 nightclubs and taverns	Not given	Values not given	19 × 2–3 min samples	13.0 ± 7.0	3.0–29.0	3.0 ± 2.0 (outdoors)	1.0–5.0
	Tavern	Not given	Artificial	16 × 2–3 min samples	8.5			
			None	2 × 2–3 min samples		35 (peak)		
	Office[a]	1440 ft³	Natural, open	2–3 min samples		10.0 (peak)		
				30 min after smoking	1.0			

TABLE 2c.—Continued

Study	Type of premises	Occupancy	Ventilation	Monitoring conditions	Levels (ppm) Mean	Levels (ppm) Range	Nonsmoking controls (ppm) Mean	Nonsmoking controls (ppm) Range
Coburn et al. (*1965*)	Rooms	Not given	Not given	Not given		4.3–9.0		
				Nonsmokers' rooms			2.2 ± 0.98	0.4–4.5
Cuddeback et al. (*1976*)	Tavern 1	10–294 people	6 changes/hr	8 hr continuous	11.5	10–12	2 (outdoors)	
				2 hr after smoking	~1			
	Tavern 2	Not given	1–2 changes/hr	8 hr continuous	17	~3–22	Values not given	
				2 hr after smoking	~12		Values not given	
U.S. Dept. of Transportation (*1971*)[b]	18 military planes	165–219 people	Mechanical	6–7 hr continuous		<2–5		
	8 domestic planes	27–113 people	Mechanical	1¼–2½ hr continuous	≤2			
Elliott and Rowe (*1975*)[c]	Arena 1	11,806 people	Mechanical	Not given	9.0		3.0 (nonactivity day)	
	Arena 2	2,000 people	Natural	Not given	25.0		3.0 (nonactivity day)	
				Nonsmoking arena			9.0	
Fischer et al. (*1978*) and Weber et al. (*1979*)	Restaurant	50–80/470 m³	Mechanical	27 × 30 min samples	5.1	2.1–9.9	4.8 (outdoors)	
	Restaurant	60–100/440 m³	Natural	29 × 30 min samples	2.6	1.4–3.4	1.5 (outdoors)	
	Bar	30–40/50 m³	Natural, open	28 × 30 min samples	4.8	2.4–9.6	1.7 (outdoors)	
	Cafeteria	80–150/574 m³	11 changes/hr	24 × 30 min	1.2	0.7–1.7	0.4 (outdoors)	
				Nonsmoking room			0.5	0.3–0.8
Godin et al. (*1972*)	Ferryboat	Not given	Not given	11 grab samples	18.4 ± 8.7		3.0 ± 2.4 (nonsmoking room)	
	Theater foyer	Not given	Not given	Grab samples	3.4 ± 0.8		1.4 ± 0.8 (auditorium)	

TABLE 2c.—Continued

Study	Type of premises	Occupancy	Ventilation	Monitoring conditions	Levels (ppm)		Nonsmoking controls (ppm)	
					Mean	Range	Mean	Range
Harke (1974a)	Office[d]	~72 m³	236 m³/hr	30 min samples		<2.5–4.6		
	Office[e]	~78 m³	Natural	30 min samples		<2.5–9.0		
Harke and Peters *(1974)*[f]	Car	2 smokers (4 cigs)	Natural	Samples		42 (peak)	(Nonsmoking runs) 13.5 (peak)	
			Mechanical	Samples		32 (peak)	(Nonsmoking runs) 15.0 (peak)	
Harmsen and Effenberger *(1957)*[b]	Train	1–18 smokers	Natural	Not given		0–40		
Perry *(1973)*[b]	14 public places	Not given	Not given	One grab sample	<10			
Portheine *(1971)*[g]	Rooms	Not given	Not given	Not given		5–25		
Sebben et al. *(1977)*	9 nightclubs	Not given	Varied	77 × 1 min samples	13.4	6.5–41.9		
				Outdoors			9.2	3.0–35.0
	14 restaurants	Not given	Not given	Spot checks	9.9 ± 5.5		Values not given	
	45 restaurants	Not given	Not given	Spot checks	8.2 ± 2.2		7.1 ± 1.7 (outdoors)	
	33 stores	Not given	Not given	Spot checks	10.0 ± 4.2		11.5 ± 6.9 (outdoors)	
	3 hospital lobbies	Not given	Not given	Spot checks		4–8	Values not given	

TABLE 2c.—Continued

Study	Type of premises	Occupancy	Ventilation	Monitoring conditions	Levels (ppm)		Nonsmoking controls (ppm)	
					Mean	Range	Mean	Range
Seiff (*1973*)	Intercity bus	Not given	15 changes/hr, 23 cigarettes burning continuously		33 ppm			
			3 cigarettes burning continuously		18 ppm			
Slavin and Hertz (*1975*)	2 conference rooms	Not given	8 changes/hr	Continuous, morning		8 (peak)	1–2 (separate nonsmoking day)	
			6 changes/hr	Continuous, morning		10 (peak)	1–2 (separate nonsmoking day)	
Szadkowski et al. (*1976*)	25 offices	Not given	Not given	Continuous	2.78 ± 1.42		2.59 ± 2.23 (separate nonsmoking offices)	

[a] Three cigarettes and one cigar smoked in 20 minutes.
[b] The Drager tube used is accurate only within ± 25 percent.
[c] The MSA Monitaire Sampler used is accurate only within ± 25 percent.
[d] About 40 cigarettes/day were smoked.
[e] About 70 cigarettes/day were smoked.
[f] Four filter cigarettes were smoked.
[g] No experimental description given.

TABLE 2d.—Nicotine measured under realistic conditions

Study	Type of premises	Occupancy	Ventilation	Monitoring conditions	Levels (μg/m³)		Nonsmoking controls	
					Mean	Range	Mean	Range
Badre et al. (*1978*)	6 cafes	Varied	Not given	50 min sample		25–52		
	Room	18 smokers	Not given	50 min sample	500			
	Hospital lobby	12 to 30 smokers	Not given	50 min sample	37			
	2 train compartments	2 to 3 smokers	Not given	50 min sample		36–50		
	Car	3 smokers	Natural, open	50 min sample	65			
			Natural, closed	50 min sample	1010			
Cano et al. (*1970*)	Submarines 66m[a]	157 cigarettes per day	Yes		32 μg/m³			
		94–103 cigarettes per day	Yes		15–35 μg/m³			
Harmsen and Effenberger (*1957*)	Train	Not given	Natural, closed	30–45 min samples		07.–3.1		
Hinds and First (*1975*)[a]	Train	Not given	Not given	2½ hr samples	4.9		Values not given	
	Bus	Not given	Not given	2½ hr samples	6.3		Values not given	
	Bus waiting room	Not given	Not given	2½ hr samples	1.0		Values not given	
	Airline waiting room	Not given	Not given	2½ hr samples	3.1		Values not given	
	Restaurant	Not given	Not given	2½ hr samples	5.2		Values not given	
	Cocktail lounge	Not given	Not given	2½ hr samples	10.3		Values not given	
	Student lounge	Not given	Not given	2½ hr samples	2.8		Values not given	
Weber and Fischer (*1980*)[b]	44 offices	Varied	Varied	140 × 3 hr samples	0.9 ± 1.9	13.8 (peak)	Values not given	

[a] Background levels have been subtracted.

[b] Control values (unoccupied rooms) have been subtracted.