Fact Sheet on Secondhand Smoke *(cont.)*

James. Repace, MSc., Physicist

Ichiro Kawachi, PhD, Associate Professor

Stanton Glantz, PhD., Professor

9/1/99

Technical Appendices A and B

Appendix A.

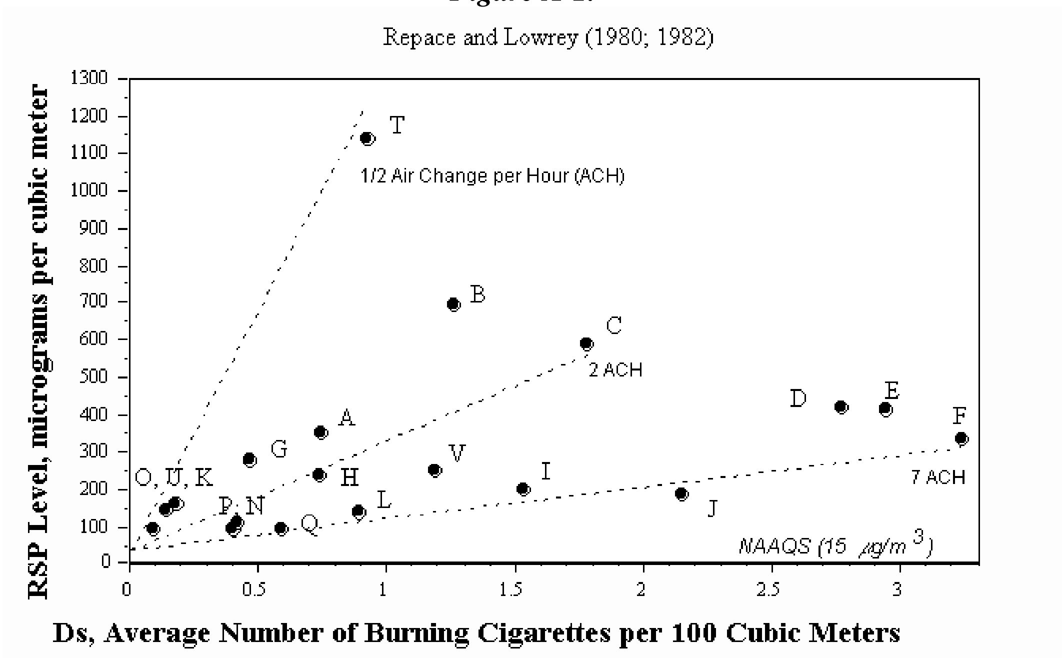
Why Secondhand Smoke Cannot Be Controlled By Ventilation

This is illustrated by Figure A-1 below. The vertical axis refers to air pollution levels measured in restaurants, bars, and other establishments in the Washington DC metropolitan area; the horizontal axis refers to the smoker density. The dashed lines refer to the calculated air exchange rates, which span the range from 1/2 air change per hour in a naturally ventilated bingo game (data point T), to a maximally-ventilated cocktail lounge (data point F) at 7 air changes per hour. Generally, the best ventilated spaces have the highest smoker densities. The number of burning cigarettes per hundred cubic meters multiplied by 3 gives the estimated density of smokers (Repace and Lowrey, 1980, 1982). This means that 1 burning cigarette per hundred cubic meters is equivalent to 3 smokers per hundred cubic meters, assuming the smokers smoke at the U.S. average rate of 2 cigarettes per hour. 1/2 to 7 air changes per hour is the practical range of ventilation in most buildings. The figure illustrates that under all conditions of typical smoking and ventilation, the annual average level of the U.S. National Ambient Air Quality Standard (NAAQS) for fine particles (PM2.5), which defines clean air, is violated. The NAAQS is designed to protect against air-pollution-induced morbidity and mortality.

Modeling Nicotine Concentrations

The major reports on SHS have paid scant attention to the fact that SHS concentrations can be accurately calculated by means of mathematical models. With the ~40 $\mu g/m^3$ background subtracted, the above respirable particle concentrations can be used to estimate nicotine concentrations by dividing by 10 (Hammond, et al., 1987; Repace and Lowrey, 1993; Repace et al., 1998). Repace et al (1998) and Repace and Lowrey (1993) have shown the following expression describes the nicotine concentration as a function of the habitual smoker density and the air exchange rate. The habitual smoker density D_{hs} is three times the active smoker density (i.e., number of burning cigarettes averaged over the observation interval), and assumes that the smokers smoke at the U.S. national average rate of 2 cigarettes per hour per smoker (Repace, 1987).

Figure A-1.



Smoking indoors leads to highly polluted air. Repace and Lowrey (1980;1982) measured fine particle air pollution, i.e., particulate matter 3.5 microns in diameter or less ($PM_{3.5}$) in a variety of establishments (Repace, 1993). Data points E, H, K, L, and N are typical restaurants, B and V are reception halls, J is a hospital waiting room, I is a bowling alley, D, G, and T are bingo games, while O is a sports arena, C and Q are bars, F is a nightclub, U is a dinner theatre, and A is a private home during a party.) All of these establishments are in the Washington, DC metropolitan area. The dashed lines show the estimated air exchange rates. D_s, the number of burning cigarettes per hundred cubic meters, is equal to 1/3 the density of habitual smokers D_{hs}, so that a D_{hs} value of 3 is equal to a D_s value of 1. The U.S. Annual National Ambient Air Quality Standard (NAAQS) for Respirable Particulate Matter 2.5 microns or less ($PM_{2.5}$) is shown for comparison (15 $\mu g/m^3$). Thus, under typical conditions of smoking and ventilation, indoor air becomes massively polluted with fine particle air pollution, jeopardizing human health.

As an example of the use of mathematical models to calculate the nicotine and RSP concentration from secondhand smoke, consider a typical office workplace with D_{hs} = 0.71 habitual smokers per hundred cubic meters (This corresponds to a value of D_s = 0.24 in the figure above). Typical engineering practice recommends a ventilation rate equivalent to C_v = 0.84 air changes per hour (Repace et al., 1998) using the nicotine equation below yields an estimated steady-state nicotine concentration of $N = 22\ D_{hs}/C_v = (22)(0.71)/0.84 = 19$ micrograms per cubic meter ($\mu g/m^3$). Assuming the workers are not present during lunch hour, and allowing for growth and decay of tobacco smoke reduces the time-averaged concentration for an 8 hour workday to 81% of steady state (Repace et al., 1998), or 15 $\mu g/m^3$. By comparison, Hammond et al. (observed an 8 hour time-weighted average nicotine concentration for 9 open office workplaces of 16 μ g/m^3.

Nicotine Concentration Equation

(Repace et al., Risk Analysis 18: 71-83, 1998)

$$N = 22\, D_{hs}/C_v$$

(where D_{hs} = smoker density in habitual smokers per 100 m^3, C_v = air exchange rate in air changes per hour, and N is the equilibrium nicotine concentration in μg/m^3)

As the nicotine concentration equation shows, the concentration will be high whenever the smoker density is high and the air exchange rate is low, irrespective of whether exposure occurs in homes, workplaces, or social settings, contrary to tobacco industry arguments, which assert that workplaces are much less polluted than homes. The relationship between exposure and dose is discussed below.

Appendix B.

Dosimetry of Secondhand Smoke

The major reports on SHS have also paid little attention to the fact that SHS doses in blood, urine, and saliva can be accurately predicted using mathematical models. The model below shows the factors involved in determining dose of the nicotine metabolite cotinine in blood plasma (i.e., serum). What are the factors determining dose, and what do the clinical epidemiological studies of biomarkers show? What is the range of dose? What are the best methods of assessing dose?

Steady-State Plasma Cotinine Model

[Repace and Lowrey, Risk Analysis 13: 463-475 (1993)]

$$P = (\phi \alpha \rho / \tau \delta)\ H\ N \text{ (ng/ml)}$$

ϕ = fraction of nicotine converted to cotinine (0.78)

α = fraction of nicotine absorbed (0.71)

ρ = respiration rate (1 m^3/hr)

δ = plasma clearance rate (64 ml/min)

τ = # minutes/day (1440)

H = exposure duration (hr/day)

N = daily average nicotine concentration ($\mu g/m^3$) x 1000 ng/μg

The above equation shows that plasma cotinine is linear with nicotine concentration. While there may be individual metabolic variability (as there is for all drugs and chemicals) in large numbers of individuals, group doses will reflect group exposures (Benowitz, 1996).

As an example of the use of mathematical models to calculate body-fluid cotinine dose, Repace and Lowrey (1993) estimated that the average U.S. nonsmoker in the 1980's had an average daily nicotine dose from secondhand smoke of 143 μg, averaged over work and home exposure. Assuming a reasonable 7 hr daily exposure, and a respiration rate of 1 m^3/hour, this is equivalent to an estimated nicotine concentration of N = 20 $\mu g/m^3$. In the above plasma cotinine equation, P = [(0.78) (0.71) (1) /(1440)(64)]{7}{20} {1000} = 0.84 ng/ml. By comparison, the U.S. Centers for Disease Control conducted a national probability survey of serum cotinine in the late 1980's and early 1990's; for all nonsmokers with cotinines less than 20 ng/ml, the arithmetic mean serum cotinine was 0.54 ng/ml (D. Mannino, CDC, personal communication). The expected range of serum cotinine from passive smoking appears to be about 0 to 15 ng/ml in nonsmokers (Repace and Lowrey, 1993). Both gas chromatography and radioimmunoassay have been used in measuring body-fluid cotinine (Benowitz, 1996).

References

ASHRAE Standard 62-1989. Ventilation for Acceptable Indoor Air Quality. The

American Society for Heating, Refrigerating, and Ventilating Engineers, Atlanta, GA.

Biener L and Fitzgerald G. Smoky bars and restaurants: who avoids them and why? J Publ Health Manage. & Practice 5: 74-78 (1999).

Bartosch WJ, and Pope GC. The economic effect of smoke-free restaurant policies on restaurant business in Massachusetts. J Publ Health Manage. & Practice 5: 53-62 (1999).

Benowitz N. Cotinine as a biomarker of environmental tobacco smoke exposure. Epidemiologic Reviews 18: 188-204 (1996).

Boffetta P, Agudo A, Ahrens W, Benhamou E, Benhamou S, Darby SC, Ferro G, Fortes C, Gonzalez CA, Jöckel KH, Krauss M, Kreienbrock L, Kreuzer M, Mendes A, Merletti F, Nyberg F, Pershagen G, Pohlabeln H, Riboli E, Schmid G, Simonato L, Trédaniel J, Whitley E, Wichmann HE, Saracci R, et al. Multicenter case-control study of exposure to environmental tobacco smoke and lung cancer in Europe [see comments] *Journal of the National Cancer Institute*. 1998;90:1440-50.

Bonita R, Duncan J, Truelson T, Jackson RT, and Beaglehole R. Passive smoking as well as active smoking increases the risk of acute stroke. Tobacco Control 8:156-160 (1999).

Celermajer DS, Adams MR, Clarkson P, et al. Passive smoking and impaired endothelium-dependent arterial dilatation in healthy young adults. New Engl J Med 1996;334:150-4.

Diez-Roux AV, Nieto FJ, Comstock GW, et al. The relationship of active and passive smoking to carotid atherosclerosis 12-14 years later. Preventive Medicine 1995;24:48-55.

Eisner MD, Smith AK, Blanc PD. Bartenders' respiratory health after establishment of smoke-free bars and taverns. JAMA 280:1909-1914 (1998).

Emmons KM, Abrams DB, Marshall R, Marcus BH, Kane M, Novotny TE, and Etzel RA. An evaluation of the relationship between self-report and biochemical measures of environmental tobacco smoke exposure. Preventive Med 1994; 23:35-39.

Glantz SA and Smith LRA. The effect of smoke-free restaurant ordinances requiring smoke-free restaurants on restaurant sales, American J Publ. Health; 84:1081-1085 (1994).

Glantz SA and Smith LRA. Erratum for "The effect of smoke-free restaurant ordinances requiring smoke-free restaurants on restaurant sales." American J Publ. Health in press (1997).

Glantz SA. Smoke-free restaurant ordinances do not affect restaurant business. Period. J Publ Health Manage. & Practice 5: v-ix (1999).

Glantz SA, Parmley WW. Passive smoking and heart disease; epidemiology, physiology, and biochemistry. Circulation 83: 1-12 (1991).

Glantz SA, Parmley WW. Passive smoking and heart disease; mechanisms and risk. JAMA 273: 1046-1053 (1995).

Hackshaw AK, Law MR, Wald NJ. The accumulated evidence on lung cancer and environmental tobacco smoke. BMJ 315:980-988 (1997).

Hammond, SK; Leaderer, BP; & Roche, A. Collection and analysis of nicotine as a marker for environmental tobacco smoke. Atmos. Env. 21: 457-462 (1987).

Hirayama T. Smoking and Mortality, p.54, in: *Life-style and Mortality, a large-scale census-based cohort study in Japan. Contributions to Epidemiology and Biostatistics, Vol.6, Ed. J. Wahrendorf*, Karger, New York 1990.

Howard G, Burke GL, Szklo M, Tell GS, Eckfeldt J, Evans, Heiss G. Active and passive smoking are associated with increased carotid wall thickness. The Atherosclerosis Risk in Communities Study. Arch Intern Med 1994;154:1277-82.

Howard G, Wagenknecht LE, Burke GL, Diez-Roux A, Evans GW, McGovern P, Nieto J, Tell GS. Cigarette smoking and progression of atherosclerosis. The Atherosclerosis Risk in Communities (ARIC) Study. JAMA 1998;279:119-24.

Hyland A, Cummings KM, and Nauenberg E. Analysis of taxable sales receipts: was New York City's smoke-free air act bad for restaurant business? J Publ Health Manage. & Practice 5: 14-21 (1999).

Imperial Tobacco Ltd. *Overall Market Conditions - F88.* Canadian Court of Appeals Document 39244 AG-214. 1988

IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Tobacco Smoking, Volume 38, International Agency for Research on Cancer, Lyon, France, 1986.

Jaakkola MS, and Jaakkola JJK. Assessment of exposure to environmental tobacco smoke. European Journal of Respiratory Disease 10:2384-2397 (1997).

Kawachi I, Colditz GA, Speizer FE, Manson JE, Stamfer MJ, Willett WC, and Hennekens CH. A prospective study of passive smoking and coronary heart disease. Circulation 95: 2374-2379 (1997).

Kawachi I, Colditz GA, Stampfer MJ, Willett WC, Manson JE, Rosner B, Speizer FE, Hennekens CH. Smoking cessation and time course of decreased risks of coronary heart disease in middle-aged women. Arch Intern Med 1994;154:169-175.

Kawachi I. Passive smoking and coronary heart disease. Cardiologia 1998; 43(7):667-675.

Law MR, Morris JK, Wald NJ. Environmental tobacco smoke exposure and ischaemic heart disease: an evaluation of the evidence. Br Med J 1997;315:973-80.

Novick LF. From the Editor. J Publ Health Manage. & Practice 5: v (1999).

Penn A, Snyder C. 1,3 Butadiene, a vapor phase component of environmental tobacco smoke, accelerates atherosclerotic plaque development. Circulation 1996; 93: 552-57.

Peto R, Lopez AD, Boreham J, Thun M, and Heath Jr C. Mortality from smoking in developed countries 1950 - 2000. Oxford University Press, New York, 1994.

Repace JL, and Lowrey AH. Indoor Air Pollution, Tobacco Smoke, and Public Health. SCIENCE 208: 464-474 (l980).

Repace JL, and Lowrey AH. Tobacco Smoke, Ventilation, and Indoor Air Quality. ASHRAE TRANSACTIONS 88: Part I,895 (l982).

Repace JL, and Lowrey AH. A Quantitative Estimate of Nonsmokers' Lung Cancer Risk From Passive Smoking. Environment International 11: 3-22 (1985).

Repace JL. Indoor concentrations of environmental tobacco smoke: models dealing with effects of ventilation and room size", *Ch. 3, IARC Scientific Publications no.81, Environmental Carcinogens--Selected Methods of Analysis--Volume 9 Passive Smoking;* I.K. O'Neill, K.D. Brunnemann, B. Dodet & D. Hoffmann, International Agency for Research on Cancer, World, Health Organization, United Nations Environment Programme, Lyon, France, (1987).

Repace JL, and Lowrey AH. An enforceable indoor air quality standard for environmental tobacco smoke in the workplace." Risk Analysis, 13:463-475 (1993).

Repace JL, Jinot J, Bayard S, Emmons K, and Hammond SK. Air nicotine and saliva cotinine as indicators of passive smoking exposure and risk. Risk Analysis 18: 71-83 (1998)

Roper Organization. *A Study of Public Attitudes Towards Cigarette Smoking and the Tobacco Industry.* Roper Organization. Prepared for the Tobacco Institute. Washington DC: Roper Organization. May, 1978

SCOTH. Report of the Scientific Committee on Tobacco and Health, UK Department of Health The Stationery Office, 1998.

Steenland K. Passive smoking and risk of heart disease. JAMA 267: 94-99 (1992).

Travis, CC, Richter SA, Crouch EAC, Wilson R, Klema ED. Cancer Risk Management. Environmental Science and Technology. 1990; 21: 415-420.

Tunstall-Pedoe H, Brown CA, Woodward M, Tavendale R. Passive smoking by self-report and serum cotinine and the prevalence of respiratory and coronary heart disease in the Scottish heart study. J Epidemiol and Comm Health 49: 139-143 (1995).

U.S. Dept. of Labor, Occupational Safety & Health Administration. 29 CFR Parts 1910, 1915, 1926, and 1928 Indoor air quality, proposed rule Fed Reg 59 # 65, Tues April 5, 1994, 15968-16039.

U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Office of Research and Development. *Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders*. Report No. EPA/600/6-90/006F. Washington, DC, 1992.

Wells AJ. Passive smoking as a cause of heart disease. JACC 24: 546-554 (1994).

Wells AJ., English PB, Posner SF, Wagenknecht LE, and Perez-Stable EJ. Misclassification rates for current smokers misclassified as nonsmokers. American J Public Health 88: 1503-1509 (1998).

Wells AJ. Re: Breast cancer, cigarette smoking, and passive smoking. American J Epidemiol. 147: 991 (1998).

Wells AJ. Lung cancer from passive smoking at work. American J Public Health 88: 1025-1029 (1998).

Wells AJ. Heart disease from passive smoking in the workplace. J Am Coll Cardiol 1998;31:1-9.

Click a link below to return to any section of the fact sheet:

[Abstract] [Introduction] [Secondhand Smoke and Cardiovascular Disease] [Secondhand Smoke Exposure]
[Smoke-Free Restaurant Laws] [SHS Lessons from California] [Conclusions] **Technical Appendices**