BENEFITS OF SMOKE-FREE REGULATIONS IN OUTDOOR SETTINGS: BEACHES, GOLF COURSES, PARKS, PATIOS, AND IN MOTOR VEHICLES

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Some persons feel that although establishing smoke-free buildings is justified, establishing smoke-free areas outdoors is not. This paper discusses the toxicity of tobacco smoke, the factors determining its concentration, and argues that tobacco smoke in places where people live, work, or congregate, whether indoors or outdoors, poses a nuisance to many, and both an acute and chronic health hazard to some. Thus, local governments are justified in establishing smoke-free zones outdoors.

Tobacco smoke contains at least 172 toxic substances, including 3 regulated outdoor air pollutants, 33 hazardous air pollutants, 47 chemicals restricted as hazardous waste, and 67 known human or animal carcinogens.[1] The law of conservation of

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1. JAMES L. REPACE, EXPOSURE ANALYSIS 203 (Wayne R. Ott et al. eds., 2006).

mass dictates that this must be true whether tobacco smoke is inhaled in the act of smoking, or inhaled by nonsmokers out of the air indoors or outdoors, known as secondhand smoke (SHS).

The concentration of tobacco smoke pollution in buildings and in vehicles is proportional to the density of smokers, and inverse to the ventilation rate.[2] Tobacco smoke pollution outdoors (outdoor tobacco smoke—or OTS), is far more complicated, being determined by the density and distribution of smokers, the wind velocity (direction and speed), and the stability of the atmosphere.[3] High SHS concentrations are produced by high smoker density, low wind velocities, and stable atmospheric conditions. SHS concentrations persist for hours after smoking ceases indoors, while OTS concentrations dissipate rapidly after smoking ceases outdoors.[4] However, during smoking, OTS levels outdoors may be as high as SHS indoors, especially in close proximity to smokers.

I. STATE AND LOCAL OUTDOOR SMOKING BAN POLICIES

Several states have taken steps to restrict smoking in outdoor locations and even in automobiles where children are present. As a result of research conducted by the state, culminating in the listing of OTS as a Toxic Air Contaminant, some of the most restrictive ordinances have been passed in California.

The City Council of Calabasas, California, passed an ordinance that took effect January 1, 2007, "prohibit[ing] smoking in all public places, indoor or outdoor, where anyone might be exposed to secondhand smoke."[5] The outdoor ban "includes outdoor cafes, bus stops, soccer fields, condominium pool decks, parks and sidewalks."[6] "Smoking in one's car is allowed, unless the windows

2. James L. Repace, *Fact Sheet: Outdoor Air Pollution from Secondhand Smoke* (2005), *available at* http://www.repace.com/pdf/OTS_FACT_SHEET.pdf.

3. *Id.*

4. Neil E. Klepeis et al., *Real-Time Measurement of Outdoor Tobacco Smoke Particles*, 57 J. AIR & WASTE MGMT. ASS'N 522, 522 (2007); James L. Repace, Address Before the 13th World Conference on Tobacco OR Health: Abstract of Indoor and Outdoor Carcinogen Pollution on a Cruise Ship in the Presence and Absence of Tobacco Smoking (Oct. 17, 2004) (unpublished working paper, on file with author).

5. John M. Broder, *Smoking Ban Takes Effect, Indoors and Out*, N.Y. TIMES, Mar. 19, 2006, at 1; CALABASAS, CAL., MUN. CODE §§ 8.12.030–.040 (2006), *available at* http://www.bpcnet.com/codes/calabasas.

6. Broder, *supra* note 5, at 1.

are open and someone nearby might be affected."[7] Violators face "warnings, fines of up to $500 for repeat offenses, and misdemeanor charges."[8] The ordinance followed a few "weeks after the California Air Resources Board declared secondhand smoke to be a Toxic Air Contaminant that can lead to respiratory infections, asthma, lung cancer, heart disease and death."[9] "Smoking has been prohibited on most Southern California beaches and piers since 2003."[10] Nationwide, in excess of "700 cities . . . have enacted ordinances placing some limits on outdoor smoking, according to the American Nonsmokers' Rights Foundation."[11] California Governor Arnold Schwarzenegger "signed a bill [making] it an infraction to smoke in a vehicle if someone under age 18 is present."[12] Other California smoking prohibitions "include a ban on smoking in enclosed workplaces and within 25 feet of a playground."[13] Legislation banning smoking in cars with young children present was adopted in Arkansas in 2006, and similar smoking bans with children have been introduced in the states of California, Georgia, Michigan, New Jersey, New York, Pennsylvania, and Vermont.[14] Louisiana has limited smoking in cars when children 13 and younger are in the vehicle.[15]

II. STUDIES OF OUTDOOR TOBACCO SMOKE CONCENTRATIONS

A limited number of controlled experiments and field studies of OTS have been conducted in California, Europe, Maryland, and the Carribean. These studies show that OTS levels outdoors are often as high as SHS levels indoors, although there are differences in the persistence of OTS levels once smoking ceases.

7. *Id.*
8. *Id.*
9. *Id.*
10. *Id.* at 2.
11. *Id.*
12. Steve Lawrence, *State Bans Smoking with Kids in Vehicle*, ASSOCIATED PRESS, Oct. 11, 2007.
13. *Id.*
14. Wayne Ott et al., *Air Change Rates of Motor Vehicles and In-Vehicle Pollutant Concentrations from Secondhand Smoke*, 1–14 J. EXPOSURE SCI. & ENVTL. EPIDEMIOLOGY 1, 13 (2007).
15. Vaughn W. Rees & Gregory N. Connelly, *Measuring Air Quality to Protect Children from Secondhand Smoke in Cars*, 31 AM. J. PREVENTIVE MED. 363, 363 (2006).

A. California

The California Air Resources Board (CARB) study measured OTS nicotine concentrations outside an airport, college, government center, office complex, and amusement park.[16] CARB found that at these typical outdoor locations, Californians may be exposed to OTS levels as high as indoor SHS concentrations.[17] CARB found that OTS was strongly affected by the number of smokers, and moderately affected by the size of the smoking area and the measured wind speed.[18] The CARB study concluded that OTS concentrations are detectable and are sometimes comparable to indoor concentrations. The study also demonstrated that the number of cigarettes being smoked (i.e., total source strength), the position of smokers relative to the receptor, and atmospheric conditions can all lead to substantial variation in average exposures.[19] CARB concluded that OTS is a "Toxic Air Contaminant."[20]

A Stanford University study measured OTS respirable particle concentrations in outdoor patios, on airport and city sidewalks, and in parks.[21] It also conducted controlled experiments of SHS indoors and OTS outdoors.[22] It found that mean SHS particle concentrations outdoors can be comparable to SHS indoors.[23] Within about 2 feet of a smoker, OTS was quite high and comparable to SHS concentrations measured indoors.[24] The study found that levels measured in 2 sidewalk cafés were detectable at distances beyond 13 feet.[25] It further found that, in contrast to SHS, OTS does not accumulate and that OTS peaks are more

16. *See* CAL. ENVTL. PROT. AGENCY: AIR RESOURCES BOARD, PROPOSED IDENTIFICATION OF ENVIRONMENTAL TOBACCO SMOKE AS A TOXIC AIR CONTAMINANT (2005), http://repositories.cdlib.org/tc/surveys/CALEPA2005.

17. *Id.* at 5–12.

18. *Id.* at 23.

19. *Id.* at 82–91.

20. *Id.* at 25.

21. Klepeis et al., *supra* note 4, at 525 (study conducted via "15 on-site field visits to 10 public outdoor locations containing smokers").

22. *Id.* at 525–26.

23. *Id.* at 531.

24. *Id.* at 532 ("Generally, average levels within 0.5 m[eters] from a single cigarette source were quite high and comparable to indoor levels . . . .") (0.5 meters equals approximately 1.64 feet).

25. *Id.* ("[D]uring 2 on-site proximity experiments . . . OTS was still detectable . . . at distances of approximately 3–4 m[eters] from a single cigarette on sidewalk patios.") (4 meters equals approximately 13.12 feet).

sensitive to source-receptor proximity and wind velocity.[26] Thus, long-term averages for OTS concentrations are averaged over a large number of transient peaks, which only occur when smokers are active, whereas indoor concentrations remain high long after smoking has ceased. The total dose to a person indoors from each cigarette will be greater than that received from each cigarette smoked outdoors. The study found upwind OTS concentrations very low and downwind OTS much higher.[27]

B. Denmark

Boffi measured OTS respirable particle pollution in a car park (open space), outdoors in front of a conference center with smokers under a roof (18 smokers during a measurement time of 35 minutes), indoors in the nonsmoking conference center, along the motorway to Copenhagen city centre, and inside a Copenhagen restaurant where smoking was allowed.[28] He found that mean values observed with smokers in front of the conference center were significantly higher than the outdoor parking place, indoor conference center, motorway, and Copenhagen outdoor official data.[29]

C. Finland

Repace and Rupprecht measured OTS respirable particle pollution in 5 outdoor cafés and on city streets in downtown Helsinki.[30] They found that air pollution levels during August 2003 in Helsinki outdoor cafés with many smokers were 5 to 20 times higher than on the sidewalks of busy streets polluted by bus, truck, and auto traffic.[31]

26. *Id.* at 530–32.
27. *Id.* at 532.
28. R. Boffi et al., *A Day at the European Respiratory Society Congress: Passive Smoking Influences Both Outdoor and Indoor Air Quality*, 27 EUR. RESPIRATORY J. 862, 862 (2006).
29. *Id.* at 863.
30. James L. Repace & Ario Alberto Rupprecht, Paper Presented at the 13th World Conference on Tobacco OR Health: Outdoor Air Pollution from Secondhand Smoke (July 14, 2006).
31. *Id.*

D. Maryland

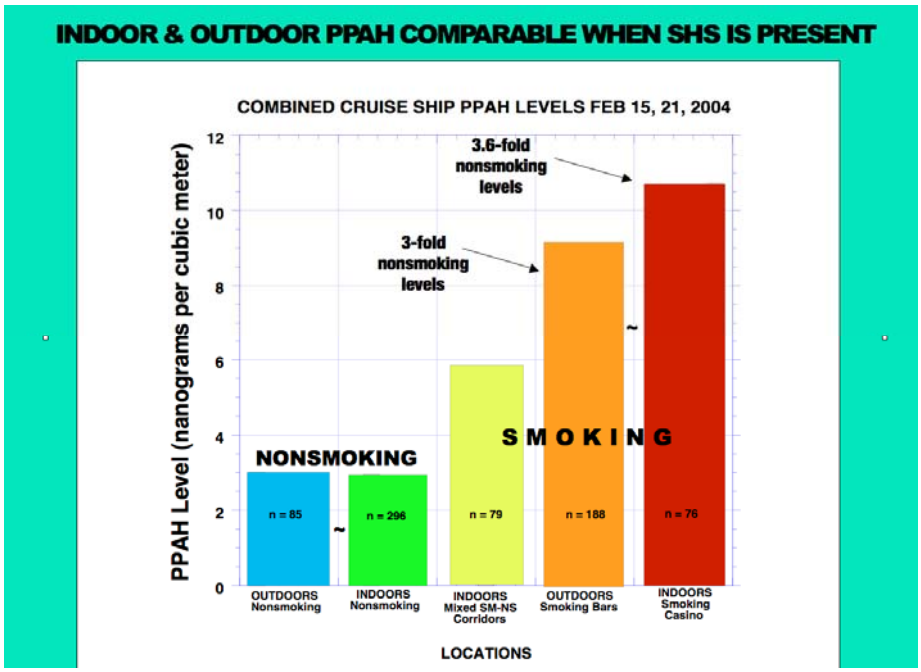
Repace measured outdoor fine particle and carcinogen concentrations from OTS on the campus of the University of Maryland in Baltimore County.[32] Using controlled experiments, Repace found that cigarette smoke respirable particulate (RSP) concentrations decline approximately inversely with distance downwind from the point source, whereas cigarette smoke carcinogen concentrations decline approximately inversely as the square of the distance from source to receptor.[33] The experiments showed that OTS smoke levels did not approach background levels either for fine particles or carcinogens until about 23 feet from the source.[34] Levels of irritation begin as low as 4 micrograms per cubic meter ($\mu g/m^3$) SHS-RSP, and levels of odor detection are as low as 1 $\mu g/m^3$.[35] Thus SHS odor would be detectable in these experiments as far as 7 meters from the source, and levels of irritation would begin at 4 meters from the source.[36]

E. The Caribbean

Experiments conducted on a cruise ship underway at 20 knots at sea in the Caribbean showed that OTS in various smoking-permitted outdoor areas of the ship tripled the level of carcinogens to which nonsmokers were exposed relative to indoor and outdoor areas in which smoking did not occur, despite the strong breezes and unlimited dispersion volume.[37] Moreover, outdoor smoking areas were contaminated with carcinogens to nearly the same extent as a popular casino on board in which smoking was permitted.[38]

32. Repace, *supra* note 2.
33. *Id.* at 9.
34. *Id.* at 10.
35. Martin H. Junker et al., *Acute Sensory Responses of Nonsmokers at Very Low Environmental Tobacco Smoke Concentrations in Controlled Laboratory Settings*, 109 ENVTL. HEALTH PERSP. 1045, 1050–51 (2001).
36. *See id.* at 1049–50.
37. James L. Repace, Address at the 14th Annual Conference of the International Society of Exposure Analysis: Indoor and Outdoor Carcinogen Pollution on a Cruise Ship (Oct. 2004).
38. *Id.*

Figure 1. Indoor and Outdoor Carcinogen Pollution on a Cruise Ship[39]



Outdoor carcinogen levels in the presence of smoking in a ship underway at sea at 20 knots of speed is comparable to indoor levels in the ship's casino, again showing a strong proximity effect despite the open air and strong breezes.[40]

F. Smoking in Cars

Two studies have shown that secondhand smoke in the small volumes of cars leads to very high exposures. Ott, Klepeis, and Switzer measured carbon monoxide (CO) and fine particle ($PM_{2.5}$) from multiple cigarettes smoked inside of 4 motor vehicles under both moving and stationary conditions, and found high particle concentrations inside cars with smokers due to the small volumes of the passenger compartments, and found that the concentrations become extremely high with the low air change rates caused by

39. *Id.*
40. *Id.*

closing windows and air conditioning.[41] They concluded that these extremely high particle concentrations constitute a serious health risk for adults and children who are passengers in a car with a smoker.[42] These findings were echoed by a Harvard School of Public Health report, concluding that SHS in cars can be up to 10 times more of a health risk than SHS in a home.[43] At least 20 states and a number of municipalities have considered limiting smoking in cars where minors are present.[44]

III. DISCUSSION

Individual cigarettes are point sources of air pollution; smokers in groups become an area source of SHS pollution. Outdoor air pollutants from individual point sources are subject to plume rise if the temperature of the smoke plume is hotter than the surrounding air; however if the plume has a small cross-section, as for a cigarette, it will rapidly cool and lose its upward momentum, and then will subside, as the combustion particles and gases are heavier than air.[45] Thus, in the case of no wind, the cigarette plume will rise to a certain height and then descend, and for a group of smokers, for example, sitting in an outdoor café, on a hospital patio, or in stadium seats, their smoke will tend to saturate the local area with SHS.

In the case where there is wind, the amount of thermally-induced plume rise is inversely proportional to the wind velocity—doubling the wind velocity will halve the plume rise.[46] In this case, the cigarette plume will resemble a cone tilted at an angle to the vertical.[47] The width of the cone and its angle with the ground will depend upon the wind velocity: a higher wind will create a more horizontal but wider cone (due to increased turbulence), with uncertain impact on exposure to SHS for downwind nonsmokers.[48] If there are multiple cigarette sources forming an area source of

41. Ott et al., *supra* note 14, at 15.
42. *Id.*
43. Rees & Connelly, *supra* note 15, at 363. The report concludes that levels of RSP measured in private cars were unsafe for children at prolonged rates. *Id.* at 367. *See also* Lawrence, *supra* note 12.
44. Lawrence, *supra* note 12.
45. Repace, *supra* note 2, at 1.
46. *Id. See generally* SAMUEL J. WILLIAMSON, FUNDAMENTALS OF AIR POLLUTION (1973).
47. WILLIAMSON, *supra* note 46; Repace, *supra* note 2, at 1.
48. WILLIAMSON, *supra* note 46; Repace, *supra* note 2, at 1.

SHS, the downwind concentrations will consist of multiple intersecting cones, i.e., overlapping plumes of increased concentration in the volume of overlap, before re-dissipating with increasing distance from the area source.[49] As the wind direction changes, SHS pollution will be spread in various directions, fumigating downwind nonsmokers.

A. Symptomatic Effects

There are a number of studies that show that nonsmokers suffer both illness and irritation from tobacco smoke exposure. SHS contains a large quantity of respirable particles, which can cause breathing difficulty for those with chronic respiratory diseases, or trigger an asthmatic attack in those with disabling asthma.[50] For the remainder of nonsmokers, Junker et al. report eye, nasal, and throat irritation thresholds for 24 healthy young adult females for repeated exposures over the course of 2 hours, corresponding to an SHS-$PM_{2.5}$ concentration of about 4.4 $\mu g/m^3$.[51] As Figure 2 shows, these levels are exceeded even at distances 3 or 4 meters (10 to 13 feet) downwind of a smoker in a sidewalk café, posing an irritation and annoyance problem even for healthy nonsmokers. With larger numbers of smokers, this irritating cloud of pollution would extend to even greater distances. Thus, there is scientific data to support OTS being both a health threat to asthmatic patients and a public nuisance to nonsmokers in general.

49. WILLIAMSON, *supra* note 46.

50. James Repace, Indoor Air Pollution and the Asthma Epidemic 5 (July 1996) (unpublished working paper, on file with author).

51. Junker et al., *supra* note 35, at 1049.

Figure 2. Outdoor Tobacco Smoke (OTS) In a Sidewalk Café and a Backyard Patio[52]

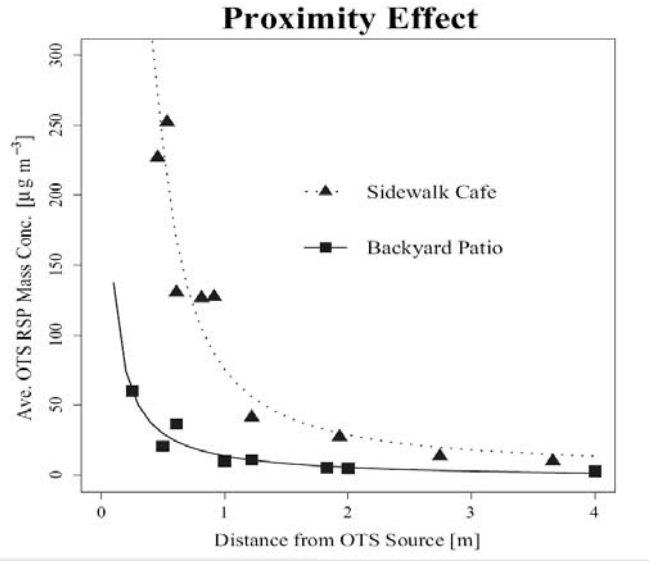


Figure 2. Overall average OTS mass concentrations as a function of proximity to the OTS source measured during experiments on a backyard patio using smoldered cigarettes, and two sidewalk cafés with human-smoked and smoldered cigarettes, for which source proximity was precisely recorded. Background RSP levels were subtracted from all measurements.

Figure 2 illustrates the proximity effect in a sidewalk café: outdoor tobacco smoke was still detectable at distances of approximately 3 to 4 meters from a single cigarette on sidewalk patios. Slightly elevated particle concentrations were detected at a distance of 8 meters from a cluster of burning cigarettes and around the corner of the house during a backyard patio experiment.[53]

Speer investigated subjective reactions of nonsmokers who developed symptoms from passive smoking.[54] Speer divided the nonsmokers into 2 groups: 191 nonsmokers with allergic diseases such as nasal allergy, asthma, and allergic headache, and a control group of 250 non-allergic nonsmokers without such diseases.[55]

52. Klepeis et al., *supra* note 4, at 532, fig. 3.
53. *Id.*
54. *See generally* Frederic Speer, *Tobacco and the Nonsmoker: A Study of Subjective Symptoms*, 16 ARCHIVES ENVTL. HEALTH 443 (1968).
55. *Id.* at 443–44.

Speer concluded that an impressively large number of people complain of symptoms from tobacco smoke, both allergic and non-allergic individuals.[56] The symptoms are summarized in Figure 3 on the following pages.

Figure 3. Known Symptoms of Passive Smoking[57]

Passive Smoking may produce:

- Itching, tearing, burning, reddening, swelling of eyes, blinking—increasing with exposure;
- Sneezing, blocking, running, itching of nose;
- Coughing, wheezing, sore throat—respiratory discomfort might begin within a half hour, persist for 8 to 12 hours;
- Headache, nausea and dizziness;
- Choking sensation;
- Irritation of mucous membranes of nose, throat, lung;
- Respiratory disease exacerbation;
- Respiratory symptoms, depressed pulmonary function.

Passive smoking is the inhalation of secondhand or environmental tobacco smoke (SHS)-polluted air. SHS is the toxic waste of tobacco consumption.

56. *Id.* at 446.

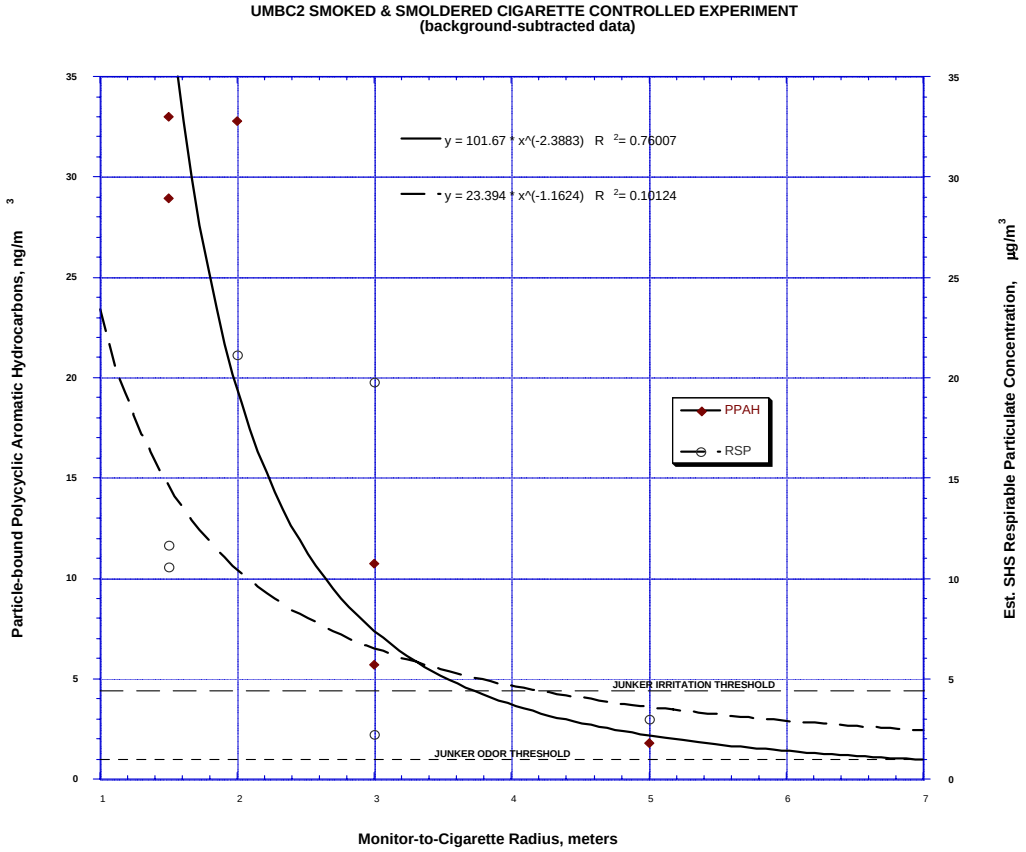
57. *Id.* at 443–46; Herbert Savel, *Clinical Hypersensitivity to Cigarette Smoke*, 21 ARCHIVES ENVTL. HEALTH 146 (1970).

Prevalence of SHS symptoms reported by 10,000 nonsmoking office workers, exposed 8 hours per day[58] • Difficulty working near a smoker (50%) • Forced to move away from desks (36%) • Bothered by SHS (33%) • Eye irritation (48%) • Nasal irritation (35%) • Aggravation of pulmonary disease (25%)	Odor acceptability[59] ~ 1μg/m^3 SHS-RSP; irritation threshold[60]: 4.4 μg/m^3

Savel reported on 8 nonsmokers with clinical hypersensitivity to cigarette smoke; all 8 individuals were allergic nonsmokers, and all developed immediate upper respiratory discomfort after being exposed to cigarette smoke.[61] Savel also reported a number of adverse symptoms, including eye and nose irritation, choking sensation, and both sinus and migraine headaches.[62] Savel concluded that an allergy to cigarette smoke might produce clinically distressing upper respiratory tract symptoms in nonsmokers with allergic backgrounds, exert a depressant effect on the antibacterial defense mechanisms of the lung, exert a toxic effect on lymphocytes, and play a role in the pathogenesis of pulmonary distress.[63]

58. Cary B. Barad, *Smoking on the Job: The Controversy Heats Up*, 48 OCCUPATIONAL HEALTH & SAFETY 21, 21–24 (1979).
59. Junker et al., *supra* note 35, at 1050.
60. *Id.*
61. Savel, *supra* note 57, at 146.
62. *Id.* at 147.
63. *Id.*

Figure 4. Smoked and Smoldered Cigarettes Showing the Cancer-Causing Polycyclic Aromatic Hydrocarbons (PAH) and SHS-RSP Data[64]



The Junker (2001) irritation index shows the median threshold of SHS irritation for healthy nonsmokers.[65] Figure 4 illustrates the proximity effect in an outdoor plaza where students congregated in widely scattered tables on a college campus in Baltimore, Maryland.[66] The proximity effect was studied in a controlled experiment involving 10 college student smokers placed in rings of increasing diameter around 2 air quality monitors so

64. Repace, *supra* note 2.
65. Junker et al., *supra* note 35, at 1045.
66. Repace, *supra* note 2, at 6.

that no matter which way the wind blew, the monitors were always downwind of 1 smoker.[67] Relative to a ring radius of 4 meters (13 feet), where the level is 4 units high, the SHS-RSP exposure concentration at 1.5 meters (5 feet) is 13 units high for particles and 35 units high for PPAH carcinogens, as shown in Figure 4. In this experiment, the proximity effect near a ring-shaped area source increases SHS by a factor of 3 for particles and a factor of nearly 9 for carcinogens.

B. Asthmatic Effects

There is very good evidence that environmental tobacco smoke has direct irritant effects in the case of passive smoking by children under the age of 4; this effect appears to diminish in children aged over 4 years.[68] There is also good evidence that SHS can trigger bronchospasm in some adults with asthma.[69] SHS is associated with wheezing symptoms, medical therapy for wheezing, and wheezing-related emergency department visits by children.[70] A causal association exists between SHS and increased episodes and aggravation of symptoms of children with asthma, affecting 200,000 to 1,000,000 children under the age of 18.[71] More than 14 million Americans reported having asthma in 2000, according to the National Center for Health Statistics.[72] "Asthma is a leading contributor of limited activity and absences from work and school; it also causes 5000 deaths each year in the U.S. The National Heart, Lung, and Blood Institute estimates that the annual direct and indirect costs of asthma were $12.7 billion in 2000."[73] By 2004, 7.1% (20.5 million) of people currently had asthma.[74] Among children under age 18 years, 8.5% (6.2 million) currently had asthma. Among adults 18 years and over, 6.7% (14.4 million) had asthma.[75] According to one report, teenage children exposed to

67. *Id.*
68. Repace, *supra* note 4.
69. *Id.*
70. *Id.*
71. *Id.*
72. Nat'l Heart, Lung, and Blood Inst., Asthma: Frequently Asked Questions, http://www.nhlbi.nih.gov/health/prof/lung/asthma/surveil_faq.htm.
73. Press Release, Nat'l Insts. of Health, NHLBI Funds Centers for Reducing Asthma Disparities (Oct. 30, 2002), *available at* http://www.nhlbi.nih.gov/new/press/02-10-30a.htm.
74. Nat'l Heart, Lung, and Blood Inst., *supra* note 72.
75. *Id.*

tobacco smoke in cars had an even higher risk of persistent wheeze than if they had been exposed at home.[76]

C. Health Risks from Exposure to SHS and OTS

Repeated exposure to a carcinogen, such as air pollution from SHS and OTS, over a lifetime increases the risk of cancer.[77] The U.S. Surgeon General has stated that there is "no risk free exposure to SHS"—chronic risk is proportional to average exposure concentration times duration of exposure times the dose-response relationship.[78] Federal regulatory agencies compute risk over a 70-year standard lifetime (e.g., EPA) or over a working lifetime of 45 years (e.g., OSHA).[79] Typical risks for lung cancer from passive smoking are in the range of 1 to 10 deaths per 1000 persons per lifetime.[80] Typical chronic heart disease risks are 10 times higher.[81] "De minimis" or acceptable risk is typically 1 death per 1,000,000 persons per lifetime.[82] OSHA's "significant risk of material impairment of health" is 1 death or irreversible serious health effect per 1000 workers per 45 year working lifetime.[83] "De manifestis" or obvious risk is 5 deaths or irreversible adverse health effect per 10,000 people at risk.[84] For workers indoors, it would take tornado-like rates of ventilation or air cleaning to reduce risks from chronic workplace exposure to de minimis levels; ergo, there is no risk-free chronic exposure to SHS. This is also likely to be true for waiters in outdoor cafés. Moreover, indoors or outdoors, for persons who have serious asthma, chronic obstructive

76. Peter D. Sly et al., *Exposure to Environmental Tobacco Smoke in Cars Increases the Risk of Persistent Wheeze in Adolescents*, 186 MED. J. AUSTL. 322, 322 (2007).

77. *See* RISK ASSESSMENT FORUM, U.S. ENVTL. PROT. AGENCY, GUIDELINES FOR CARCINOGEN RISK ASSESSMENT 5-1 to -7 (2005) (discussing risk characterization as bringing together hazard, dose-response, and exposure analysis).

78. Americans for Nonsmokers' Rights, *Second Hand Smoke: The Science* 1 (Nov. 2006), *available at* http://www.no-smoke.org/pdf/SHS.pdf.

79. *See* JOHN R. FOWLE III & KERRY L. DEARFIELD, U.S. ENVTL. PROT. AGENCY, RISK CHARACTERIZATION HANDBOOK 154 (2000), *available at* http://www.epa.gov/OSA/spc/pdfs/rchandbk.pdf (EPA); James L. Repace et al., *Air Nicotine and Saliva Cotinine as Indicators of Workplace Passive Smoking Exposure and Risk*, 18 RISK ANALYSIS 71, 78 (1998) (OSHA).

80. *See* James L. Repace et al., *A Quantitative Estimate of Nonsmokers' Lung Cancer Risk from Passive Smoking*, 11 ENV'T INT'L 3, 6–9 (1985).

81. Repace et al., *supra* note 79, at 79.

82. Curtis C. Travis et al., *Cancer Risk Management: A Review of 132 Federal Regulatory Decisions*, 21 ENVTL. SCI. & TECH. 415, 418 (1987).

83. Repace et al., *supra* note 79, at 79.

84. Travis et al., *supra* note 82, at 418.

respiratory disease, or heart disease, even brief exposures to SHS could land them in the emergency room or worse. It is generally these patients who died in the notorious outdoor smog episodes in the Meuse Valley in Belgium in 1930, Donora, Pennsylvania in 1948, and London in 1952, which eventually led to stringent regulation of outdoor air pollution.[85]

Arguments against banning smoking in certain outdoor public venues were advanced by Professor Simon Chapman in his presentation at the Tobacco Control Legal Consortium Symposium on the Limits of Tobacco Control Regulation.

> Our focus in this symposium on whether policy and advocacy for the regulation of SHS might sometimes go "too far." [Where] "going too far" in SHS policy means efforts premised on reducing harm to others, which ban smoking in outdoor settings such as ships' decks, parks, golf courses, beaches, outdoor parking lots, hospital gardens and streets. It is also the introduction of misguided policies allowing employers to refuse to hire smokers, including those who obey proscriptions on smoking indoors while at work. Many people are comforted by the smell of camp and log fires, even seeking out such exposures. But the same people will sometimes become outraged by the occasional, fleeting exposure to tobacco smoke. While nearly identical in terms of their noxious content, both forms of smoke have entirely different *meanings*. If radically different concerns about inhaling essentially the same zoo of noxious particles was all that mattered here, we would have to conclude that many people can be frankly irrational. But outrage about some forms of smoke and open acceptance of others is very explicable to sociologists of risk perception. Among the many key determinants of meaning and outrage are whether a noxious agent is seen as voluntary or coerced; natural or artificial; and whether the risk has been amplified by lots of media attention. We don't read much about the dangers of inhaling campfire smoke, smoke from incense or candles or cooking, but we read a lot about the dangers of secondhand cigarette smoke. I emphasize that I am very supportive of preventing smoking in crowded, confined outdoor

85. WILLIAMSON, *supra* note 46. *See also* STEPHEN T. HOLGATE ET AL., AIR POLLUTION & HEALTH (1999).

> settings such as sports stadia, in most outdoor dining sections of (particularly small) restaurants and in unblocking the entrances to buildings by having smokers move further away.[86]

My response to Professor Chapman's arguments follows: We agree completely on the principle of banning smoking in outdoor cafés and sports stadia. However, I disagree that because campfire smoke and smoke from incense, candles, or cooking have not (yet) received the same level of notoriety that SHS has (largely because they have not been researched until recently), that they do not pose both acute and chronic health hazards resulting from the toxicity of fine particles.[87] In fact, smoke from any source in places where people live, work, or congregate is going to pose a nuisance to many and an acute health hazard to some. Smoke from all of these sources is the product of incomplete combustion and is toxic to humans. As with indoor smoking, if enough persons complain about outdoor smoking, local governments will be moved to protect the public, as they have done for decades with factory smoke and auto exhaust, and are scientifically justified in doing so for OTS on the basis of the exposure analysis discussed herein.

IV. CONCLUSIONS AND POLICY IMPLICATIONS

In 1946, a city ordinance urged by concerned citizens was passed in Pittsburgh, Pennsylvania, despite the absence at that time of any scientific evidence of the health effects of outdoor air pollution levels on the population. Thus, early public air pollution policy was formulated on the basis of intuition. Similarly, a wave of restrictions on outdoor smoking has been passed in several U.S. states, despite the absence of health effects studies on OTS and the paucity of data on OTS concentrations. However, data is accumulating in support of the public's intuitive response to OTS. Recent field studies plus controlled experiments demonstrate that, regardless of which way the wind blows, individuals in an outdoor

86. Simon Chapman, Professor of Public Health at the University of Sydney, Austl., Presentation at the Tobacco Control Legal Consortium Symposium on the Limits of Tobacco Control Regulation at William Mitchell College of Law (Oct. 23, 2007).

87. *See generally* Wayne R. Ott & Hans C. Siegmann, *Using Multiple Continuous Fine Particle Monitors to Characterize Tobacco, Incense, Candle, Cooking, Wood Burning, and Vehicular Sources in Indoor, Outdoor, and In-Transit Settings*, 40 ATMOSPHERIC ENV'T 821 (2006).

café, transiting through a building doorway, on a public street, sidewalk or bus stop, even on the open deck of a cruise ship at sea, or otherwise surrounded by a group of smokers, are always downwind from the source and are thus subject to being enveloped in a cloud of obnoxious, irritating, asthmagenic, carcinogenic, and atherogenic fumes.

These studies also show that under a variety of conditions, levels of OTS can be as high as indoor levels of SHS. Smoking in the small volume of cars leads to much higher levels of tobacco smoke air pollution than in other enclosed environments. Individuals who suffer from asthma, especially children, are at acute risk from OTS. Healthy persons are subject to annoyance and increased risk of developing chronic disease from repeated OTS exposure over a lifetime. This new data confirms public intuition, demonstrating that public demand for smoke-free outdoor spaces is not "going too far," and justifies policies banning smoking in outdoor locations, in vehicles, where people congregate in public, or where workers are placed at risk, such as outdoor cafés.