Can Ventilation Control Secondhand Smoke

in the Hospitality Industry?

An Analysis of the Document "Proceedings of the Workshop on Ventilation Engineering Controls for Environmental Tobacco Smoke in the Hospitality Industry", sponsored by the Federal Occupational Safety and Health Administration and the American Conference of Governmental Industrial Hygienists.

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Abstract

A panel of ventilation experts assembled by OSHA and ACGIH concluded that dilution ventilation, used in virtually all mechanically ventilated buildings, will not control secondhand smoke in the hospitality industry (e.g., restaurants, bars, casinos). The panelists asserted that a new and unproved technology, displacement ventilation, offered the *potential* for up to 90% reductions in ETS levels relative to dilution technology. However, this assertion was not substantiated by any supporting data. Air cleaning was judged to be somewhere between dilution and displacement ventilation in efficacy, depending on the level of maintenance. The panel also failed to quantify the ETS exposure or risk for workers or patrons either before or after the application of the new technology. Panelists observed that building ventilation codes are not routinely enforced. They also noted the lack of recognized standards for acceptable ETS exposure as well as the lack of information on typical exposure levels. However, indoor air quality standards for ETS have been proposed in the scientific literature, and reliable mathematical models exist for predicting pollutant concentrations from indoor smoking. These proposed standards and models permit application of an indoor air quality procedure for determining ventilation rates as set forth in ASHRAE Standard 62. Using this procedure, it is clear that dilution ventilation, air cleaning, or displacement ventilation technology even under moderate smoking conditions cannot control ETS risk to *de minimis* levels for workers or patrons in hospitality venues without massively impractical increases in ventilation. Although there is a scientific consensus that ETS is a known cause of cancers, cardiovascular diseases, and respiratory diseases, although ETS contains 5 regulated hazardous air pollutants, 47 regulated hazardous wastes, 60 known or suspected carcinogens, and more than 100 chemical poisons, the tobacco industry denies the risks of exposure, opposes smoking bans, promotes ventilation as a panacea for ETS control, and works for a return to *laissez-faire* concerning smoking in the hospitality industry. Smoking bans remain the only viable control measure to ensure that workers and patrons of the hospitality industry are protected from exposure to the toxic wastes from tobacco combustion.

Table of Contents

Executive Summary

1. OSHA-ACGIH Ventilation Workshop Summary

A panel of 14 experts on ventilation engineering and ventilation practices in the hospitality industry was charged with determining technically and economically feasible engineering controls for ETS in restaurants, bars, and casinos, assuming that total elimination of ETS was not an option. The panel recognized that there was a lack of information on typical ETS exposure levels in such venues, as well as a lack of recognized standards for acceptable exposure. Panelists concluded that well-mixed dilution ventilation, the overwhelming majority of current installations, was unsatisfactory for controlling worker exposure to ETS in hospitality venues. Local area exhaust ventilation, smokeless ashtrays, air cleaning, and displacement ventilation were identified as potentially more effective.   Of these, displacement ventilation was viewed as the most promising, with estimated 90% reductions under the most favorable conditions. These estimates were based on professional judgment rather than on measured data. Moreover, the panel raised several concerns about displacement technology, including lack of familiarity by many ventilation engineers, difficulty with retrofitting existing installations, and potential aesthetic problems.

Ventilated ashtrays as currently available did not appear to be effective, although panelists felt the technology could be made 40% to 50% efficient, provided smokers could be persuaded to use them, a significant potential problem in areas where foreign tourists are frequent customers. These conclusions were professional judgments as opposed to data-based analysis. Although air filters are capable of high capture efficiencies, they also require high airflow to be effective, and needed regular effective maintenance to remain effective. Costs are a major consideration in the restaurant industry, which limits the implementation of high technology solutions such as 100% outside air 1-pass systems. Costs are not a limiting factor in the casino industry for the large casinos, although they are for the small ones. Large fluctuations (e.g., factors of 3) in the smoking population of these venues may occur. A further significant problem is that some building codes do not require that the ventilation system actually be operated, especially in the small non-chain establishments.

In brief, The OSHA/ACGIH workshop concluded that presently available ventilation technology (well-mixed dilution ventilation) was unsatisfactory for controlling worker exposure to ETS. It also concluded that air cleaning was similarly problematic. Of proposed new technology, displacement ventilation was

viewed as having the potential for 90% reductions in ETS levels, although this view was not supported by performance data. Other major problems included the lack of familiarity of most ventilation engineers with the new technology, and the difficulty in retrofitting existing installations. Panelists viewed the lack of enforcement of ventilation rates by local building codes and the use of natural ventilation as further problems. However, it should be noted that in California, Cal-OSHA requires employers to ventilate workspaces during working hours.

2. ETS and Ventilation: Health Risk Assessment Summary

Using U.S. average smoking prevalence, ASHRAE Standard 62-1999 and 62-1989 default occupancy levels, and recommended makeup air supply rates, models show that for dilution ventilation supplied in recommended amounts, estimated ETS RSP levels for hospitality industry venues will be between 100 and 200 $\mu g/m^3$, and air nicotine levels from 10 to 20 $\mu g/m^3$. Predicted levels are significantly lower than observations, suggesting lower ventilation rates or higher smoker densities than expected. This is not surprising since smoker density is not regulated and ventilation rates are not enforced.

Assuming ideal dilution ventilation, i.e., reasonably achievable control technology (RACT), model-estimated ETS risk levels for lung cancer and heart disease combined ranged from 15 to 25 per 1000 workers, which is 15 to 25 times OSHA's significant risk level, and 15,000 to 25,000 times the *de minimis* or "acceptable risk" level for federally regulated hazardous air pollutants. This supports the conclusion of the OSHA/ACGIH ventilation panel that dilution ventilation (better than 99% of current installations) is not a viable control for ETS.

Assuming ideal displacement ventilation, i.e., best achievable control technology (BACT), based on the professional judgment of the OSHA/ACGIH panel, estimated ETS risk levels for lung cancer and heart disease combined would be reduced by 90%. This places estimated ETS risks between 1.5 to 2.5 per 1000 workers, which is 1.5 to 2.5 times OSHA's Significant Risk level, and 1,500 to 2,500 times the *de minimis* or "acceptable risk" level for federally regulated hazardous air pollutants. Even a 90% reduction in ETS exposure yields massively unacceptable risk.

Moreover, the panel's estimates of 90% reductions in ETS concentrations are not supported by measured data. ETS concentrations experienced by workers in smoking areas may actually be increased due to low air flows employed by this technology, and the confinement of smokers to designated smoking areas with a fraction of the volume of the entire building.

All cognizant health and scientific authorities in the U.S., including the US Environmental Protection Agency, the National Institute for Occupational Safety and Health, OSHA, the Surgeon General, the National Academy of Sciences, the National Cancer Institute, the National Toxicology Program and the American Medical Association, have concluded that ETS exposure causes morbidity and mortality. This consensus has been accepted by ASHRAE in ASHRAE Standard 62-1999 and codified in Addendum 62-e.

While indoor pollutants are not regulated under the Clean Air Act, the control technologies utilized are appropriate for the discussion of indoor pollutants such as ETS. Under Section 112 of the federal Clean Air Act, pollutants may be designated as "hazardous air pollutants" (HAPS) if they can cause serious morbidity or mortality, as ETS does. These ETS-like chemicals are regulated by NESHAPS, which are far more stringent than either the "reasonably achievable control technology" (RACT) for existing sources or "best available control technology" (BACT) required for new sources of outdoor air pollution. RACT and BACT are designed to control ordinary non-hazardous air pollutants. NESHAPS regulate HAPS to levels of *de miminis* risk with an adequate margin of safety. ETS actually contains 5 HAPS pollutants, more than 100 poisonous chemicals, and 47 chemicals classified as hazardous waste under RCRA. ETS emitted into the outdoor air from a smokestack industry would qualify for regulation as a HAP mixture, like coke-oven emissions.

While no official ETS indoor air quality (IAQ) standards have been adopted in the U.S., proposed NESHAPS-style ETS IAQ standards have been published, and are based on limiting ETS lung cancer and heart disease risk to *de minimis* levels. Application of these putative standards to restaurants, bars, and casinos shows that tornado-like levels of ventilation would be required to control ETS. Moreover, enforcement of an official ETS-ventilation standard would require establishment of costly new regulatory bureaucracies. Even if official standards for ETS were adopted for lung cancer and heart disease, protecting against the emerging risks of ETS-induced breast cancer, stroke, nasal sinus cancer, respiratory diseases, etc. would remain a formidable obstacle.

The tobacco industry does not concede that ETS poses health risks to nonsmokers. Its goal, as stated on its websites, is to promote ventilation technology as one possible option among many for hospitality business owners, and the industry argues for letting the marketplace decide how to control ETS...

Smoking bans represent the most cost-effective, easiest-to-enforce, and lowest risk alternative for ETS control. They appear profitable for business, and are also the only control measure known which is capable of yielding *zero* risk.

I. The following is a summary of issues raised in the 176 page document *Proceedings of the Workshop on Ventilation Engineering Controls for Environmental Tobacco Smoke in the Hospitality Industry*, sponsored by the U.S. Dept. of Labor, Occupational Safety and Health Administration (OSHA), and the American Conference of Governmental Industrial Hygienists (ACGIH). This discussion includes the available (dilution ventilation and air cleaning) and proposed (displacement ventilation) technology, and summarizes the constrasting views of ventilation engineers present at the workshop.

SUMMARY OF PROCEEDINGS OF THE WORKSHOP ON VENTILATION ENGINEERING CONTROLS FOR ENVIRONMENTAL TOBACCO SMOKE IN THE HOSPITALITY INDUSTRY, JUNE 7-9, 1998, FT. MITCHELL, KY

CO-SPONSORED BY THE
U.S. DEPT. OF LABOR, OCCUPATIONAL SAFETY & HEALTH ADMINISTRATION (OSHA) AND THE AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH)

Summary: In June 1998, OSHA sponsored a Technical Workshop on Ventilation Engineering Controls for Environmental Tobacco Smoke Exposure in the Hospitality Industry. The 3-day workshop, held in Ft. Mitchell, Kentucky, was coordinated by ACGIH. A panel of 14 experts was assembled to provide more information on ETS exposures and to discuss ventilation engineering controls for reducing exposures in restaurants, bars, and casinos. The panelists were either experienced ventilation engineers or facility managers from the hospitality industry.

The workshop was an outgrowth of OSHA's Notice of Proposed Rulemaking on Indoor Air Quality (59 FR 15968) which required control of point sources of pollutants, and specified conditions under which smoking could be allowed in the workplace. Employers were required to establish designated smoking areas, permit smoking only in such areas, and ensure that those areas were enclosed and exhausted directly outdoors, and maintained under negative pressure sufficient to contain tobacco smoke. Employees could not be required to enter the designated smoking areas as part of their normal work. However, while the ETS provisions were feasible for many employers, "it became apparent to OSHA that in businesses where there is substantial contact between customers who smoke and workers (e.g. food, beverage and gaming industries, collectively known as the 'hospitality industry') this provision was not easily applied as

written. During the public hearing on OSHA's proposed standard on indoor air quality, representatives of the hospitality industry supplied very little information on engineering and administrative controls that could be used to protect workers.

The purpose of the 1998 Workshop was "to obtain much needed information on feasible engineering and work practice controls for the hospitality industry (i.e., bars, restaurants and gambling facilities) that could potentially reduce ETS exposure, from the point of view of ventilation engineers and facility management personnel. A Mission Statement was delivered to the panelists by Dr. Steven Guffey, Workshop Chair, and ACGIH Industrial Ventilation Committee Member, University of Washington. Dr. Guffey stated that "the workshop mission was to come up with feasible controls for environmental tobacco smoke (ETS), particularly in the hospitality and restaurant business." He asserted that the workshop's primary aim was to achieve reductions in ETS levels. Dr. Guffey stated that the workshop focus included, but was not limited to, "the unique occupational exposures in the hospitality sector due to the interface between workers and smoking customers. ETS is a contaminant in bars, restaurants and gambling facilities. We will consider engineering controls, such as local source capture ventilation, that control the contaminant at its point of generation; controls that are technically and economically feasible. We can also consider other ventilation engineering controls employed in general industry, such as makeup air islands, and displacement ventilation."

Ventilation was defined (R. Hughes Presentation) as *an application of controlled airflow for the purpose of providing comfort and to provide for contaminant control.* The two basic types of ventilation are local exhaust ventilation and dilution or general ventilation. Local exhaust captures the contaminant right at the source. Local exhaust ventilation can be significant in reducing worker exposure, because the contaminant is captured at or near the source and is prevented from reaching the worker. Local exhaust is primarily for point source contamination. It is very effective for high contaminant levels, and requires low airflow. Dilution ventilation dilutes the contaminant by mixing the large quantities of air with it to lower the concentration level. It does not prevent worker exposure because the contaminant stays in the area. It is usually better for diffuse sources of contamination. Its application is better with low levels of contaminant or low toxicity contaminants. A disadvantage (in addition to the poor exposure control) is that it can require extremely large amounts of airflow.

The major source of information for ventilation design in the commercial or indoor environment is the ASHRAE Handbook of Fundamentals. Information in the ASHRAE Fundamentals focuses primarily on comfort although they do have

information on industrial ventilation. ASHRAE does provide some of the theoretical aspects of ventilation. Industrial ventilation does have applicability for the control of the commercial environment, and while most of the past efforts have been directed to the industrial environment these ventilation techniques are readily adaptable. ACGIH's *Industrial Ventilation* focuses primarily on the industrial environment. It discusses in great detail local and general ventilation, providing information on system components, discussing the construction of exhaust hoods, fans, and duct design.

During the workshop, each panelist presented for 15 minutes on topics including local source capture vs. general dilution ventilation, supply air islands, ventilation performance monitoring, displacement ventilation systems, particulate and gas phase air cleaners, and current practice for designing heating, ventilating, and air conditioning (HVAC) systems. The panel then explored the technological and economic feasibility of applying current prudent practice for application of HVAC controls to the hospitality sector. Finally, the panel made recommendations of the most promising options.

The Executive Summary of the Workshop Proceedings, authored by Dr. Guffey, synopsized the issues involved in "engineering solutions to ETS exposures." Panelists discussed several possible engineering solutions for a variety of ETS exposure conditions in restaurants, bars, and the gaming industry. Displacement ventilation was deemed to have the greatest chance of producing substantial reductions, and could be less costly over time than the dilution methods now in common use. However, a major problem is that displacement ventilation is unfamiliar to most heating, ventilating, and air conditioning (HVAC) engineers, and presents challenges in duct placement, especially in retrofitting existing facilities. Another problem is that displacement ventilation is relatively new and practical applications too recent and sparse to state with confidence that it would apply to larger casinos or to cases where turbulent mixing is not well-controlled. Likewise it may be difficult to use ventilated ashtrays on gaming tables because they would obscure some hand movements, a security issue in casinos. In general, ventilated ashtrays were thought to have less potential to achieve dramatic reductions in exposures, but would reduce the quantity of ETS released into occupied spaces, while using low levels of exhaust air. A drawback is that they would require cooperation of smokers and occupy counter or table space. A combination of displacement ventilation and ventilated ashtrays might be used together, in restaurants and bars.

Although the mission of the group was to develop engineering solutions to ETS exposures, it was recognized that a major complication was **"the lack of a**

recognized standard for acceptable exposure levels, and the lack of important information on typical levels of exposure." It was not clear to Panelists what the typical levels of exposures to workers in restaurants, bars, and gaming establishments would be if current ventilation strategies were well executed. Furthermore, for most ventilation interventions, it was difficult to predict the reduction in exposures that would result because in part efficacy depends on many factors beyond the control of the designer. Factors cited included sources of exposure, mechanisms of exposure, constraints imposed by material handling (e.g., serving of food or drinks or dealing cards), work practices such as standing within arm's reach and avoiding a hurried or unfriendly appearance), competing air motions (e.g. jets from diffusers, convection) and source strength, location, and mobility. Despite these unknowns, the panel believed it could propose measures which "will substantially reduce ETS emissions, and thus exposure to workers." The actual magnitude of reductions would have to be experimentally determined. The sufficiency of the reductions would have to be ascertained when ACGIH or others set a standard of acceptable exposure.

The panel considered such factors as identification of major issues, vital information that is missing or incomplete, smoking locations, sources of smoke, smoker behaviors important to source control, ETS monitoring, important constraints on solutions, general categories of possible solutions, and finally, proposed general control measures for bars, restaurants, and casinos: dilution ventilation, displacement ventilation, and ventilated ashtrays. Estimated percent reductions were made, apparently based on professional judgment rather than data or models. Total elimination of ETS was not an option for consideration.

Panel discussion of major issues:

1. *Vital information missing or incomplete*: missing information on upward velocity of cigarette and cigar smoke (pipes apparently not considered) at different heights above the source, crucial for downdraft control. Panel concluded velocities too great for downdraft to work. Will increasing airflow increase burn rate, discouraging smokers from cooperation in holding cigarettes under small hoods between puffs? Uncertainty about buildup of tars on ducts. Effective filters may require excessive pressures, and may be poorly maintained. Optimal filters and placement -- in the hood or near the fan? Can filtered exhaust air be recirculated or must it be exhausted outdoors? Smokeless ashtray filters are poor on removal efficiency. Restaurant industry panelists complained of the difficulty of adequate maintenance and detrimental effects of increased fan pressures on equipment if filters added to existing

systems. Panelists were unaware of published data on these issues, but thought it could be obtained by future research.

2. *Smoking locations*: Engineering controls need to be discussed in terms of location of activity rather than type of establishment, e.g., tables and booths, bars, gaming tables, slots, and video games, designated smoking lounges where customers are served, stationary workers in service areas, change booths, or cashiers.

3. *Smoking sources*: Exhaled mainstream smoke diffuses over large area, unless the smoker directs it into a receptacle; smokers in motion are a diffuse source of both exhaled mainstream and sidestream smoke. Point source control strategies may not work. It is doubtful that if smokers blow smoke at workers that any kind of ventilation can control it. Velocity and direction is important. Designing systems for mobile source control very difficult. How long does smoker hold cigarette, and how long is it down? Differences between cigars and cigarettes? Pipe smoking was held to be rare, and dismissed as source. ETS generation rates are not well characterized.

4. *Reduction in ETS that must be obtained*? No guidance provided.

5. *Necessary smoker behavior for solution success*: Smoking behaviors differ in restaurants, bars, and casinos. Restaurant smoking is leisurely, casino smoking is intense.

6. *Assumptions about smoker behavior, and likelihood of adoption of requested behavior necessary for substantial reduction*:: Can smoking take place only in designated areas, leaving cigarettes in ashtrays as much as possible, blowing smoke toward ventilated points? In the panelists' experience, compliance with posted rules is high for locations, directional exhaling is possible, especially vertically. Smokers' attitudes toward leaving cigarettes in ashtrays when not in use is unknown, but compliance is judged likely.

7. *Monitoring of ETS*: Best indicators thought to be personal monitoring of airborne nicotine and UV or fluorescent particulate; literature suggests that respirable suspended particles poorly correlated to more specific measures. Body fluid or hair cotinine possible but affected by individual variability. Stationary monitors may be better than personal monitors for short periods due to individual variability.

8. *Important constraints on solutions*:   Acceptable solutions should require minimal effort by smokers and should not make them feel conspicuous or punished. Acceptable solutions <u>must</u> stay within airflow capacity of current equipment except perhaps for large casinos.

9. *Likely attainable ETS reduction for each method*:   Varies among methods. Discussed below.

10. Cost factors and limitations: Cost of additional exhaust ventilation was $1-$2 per cubic foot per minute per year ($1-$2/cfm-y).

General Categories of Proposed Solutions
* Smoking bans
* Limited smoking periods
* Smoking lounges, including self-serve dining areas where employees do not go
* Well-mixed dilution ventilation
* Displacement ventilation
* Local source capture and control using hoods

Since the mission of the workshop was to explore solutions that would allow smoking while "substantially reducing exposure to employees," bans, limitations and non-service smoking lounge options were dismissed. Panelists concluded, furthermore, that while well-mixed dilution ventilation is currently widely used, it appears that it is not a satisfactorily efficient or effective method of controlling ETS exposures to workers in restaurants, bars, and gaming establishments. Especially given the absence of a prescribed quantitative level of acceptable control and measured data demonstrating that control. Thus the workshop focused on the remaining alternatives: displacement ventilation and local exhaust ventilation of ETS sources.

Displacement Ventilation

Displacement ventilation is a dilution design strategy that eschews the turbulence mixing necessary to traditional "well-mixed" designs. Displacement ventilation requires that supply air released in a room be 5 to 10 degrees cooler than the air already in the room. Released at the floor level, it will travel horizontally across open spaces. Since people, mechanical and electrical devices are generally much warmer than this supply air, the convection currents from them carry warm contaminated air to the ceiling area where it can be removed by return air grilles. The rising plume of ETS being warm is helpful, and both sidestream and exhaled mainstream should rise. If the ceiling exceeds 8 feet, then the contaminants near

the ceiling should be well above the breathing zone. This strategy contrasts with well mixed dilution ventilation, which attempts to mix floor and ceiling air using jets from the ceiling diffusers to provide the necessary kinetic energy. To be successful, displacement ventilation requires that there be relatively little disturbance to the air by moving objects (e.g., Casablanca fans), jets of air, etc. (in other words, it is a low-flow technique). It works best when the supply air can be delivered very close to the floor, requiring ducts and supply air grilles to be installed at or near the floor.   If tobacco smoke is exhaled downward, this runs counter to this strategy.   Also, restaurant industry panelists objected to the constraints on layout and esthetics imposed by locating large diffusers near the floor. Experimental verification of efficacy is lacking if diffusers are located in the ceiling near walls and directed downward. The panelists concluded that if conditions are suitable, displacement ventilation has the potential to remove both sidestream and mainstream smoke, and may be used in conjunction with ventilated ashtrays, ventilated booths, and other local exhaust strategies.

Panelists estimated that total ETS reductions were likely to be around 90% or more for good conditions. However, they noted that poor conditions, especially those due to the introduction of turbulence and large eddies, could sharply lower the reductions.

The panelists observed the following concerns:
- Displacement technology is unfamiliar to many HVAC engineers
- Supply air diffusers take up significant wall space
- Ducting of air to floor level can be difficult, especially in existing facilities
- The technology is sensitive to errors in supply air temperature, affecting thermal comfort of patrons
- Low ceilings can lead to stratified temperatures (warm heads, cold feet)
- Concentrations of ETS at ceiling height are dense; workers at elevated stations (as in casinos) could experience increased exposures unless additional measures are taken

Ventilated Ashtrays

Ventilated ashtrays ("smokeless" ashtrays), according to the panelists, in principle could be highly effective in reducing sidestream smoke, but commercial models tested were largely ineffective, although experimental ones built by some panelists have worked much better. In addition, for any ductless unit to remain effective, filters have to extremely well maintained. Panelists felt maintenance would likely be a continuing problem for the hospitality industry.   Operational problems relating to scarcity of space on bar tops and tables and potential problems with

cleaning the units and the surfaces they obstruct may limit their usefulness. Panelists had reservations about whether enclosed ventilated ashtrays would be accepted by restaurants and patrons. Panelists assumed that 50% to 70% of ETS came from sidestream smoke, and assumed that properly maintained devices could collect 95% of the effluent while the cigarette was resident, which they assumed would be 80% of the time, yielding **a net estimated collection efficiency of 38% to 53% of ETS.**

Advantages:
- High potential effectiveness
- Reduce total room ETS burden, including room surfaces
- Low airflow requirements
- Low noise
- Convenient and easily cleaned

Disadvantages:
- Must be ducted to outside unless posessing self-contained filter and fan
- Frequent cleaning of hoods and ducts necessary if not filtered at hood
- Internal hood filters must be frequently cleaned
- For units without internal filters, duct plugging may occur

Canopy Hoods For Tables

Panelists stated reductions in ETS for canopy hoods would depend on airflow levels, but did not estimate likely reductions because minimum airflows were impractically high, in the neighborhood of >300 cfm/hood.

Condensed Workshop Summary:

A panel of 14 experts on ventilation engineering and ventilation practices in the hospitality industry was charged with determining technically and economically feasible engineering controls for ETS in restaurants, bars, and casinos, assuming that total elimination of ETS was not an option. The panel recognized that there was a lack of information on typical ETS exposure levels in such venues, as well as a lack of recognized standards for acceptable exposure. Panelists concluded that well-mixed dilution ventilation, the overwhelming majority of current installations, was unsatisfactory for controlling worker exposure to ETS in hospitality venues. Local area exhaust ventilation, smokeless ashtrays, air cleaning, and displacement ventilation were identified as potentially more effective. Of these, displacement ventilation was viewed as the most

promising, with estimated 90% reductions under the most favorable conditions. Concerns about this technology included lack of familiarity by many ventilation engineers, difficulty with retrofitting existing installations, and potential aesthetic problems.

Ventilated ashtrays as currently available did not appear to be effective, although panelists felt the technology could be made 40% to 50% efficient, provided smokers could be persuaded to use them, a significant potential problem in areas where foreign tourists are frequent customers. Although air filters are capable of high capture efficiencies, they also require high airflow to be effective, and needed regular effective maintenance to remain effective. Costs are a major consideration in the restaurant industry, which limits the implementation of high technology solutions such as 100% outside air 1-pass systems. Costs are not a limiting factor in the casino industry for the large casinos, although they are for the small ones. Large fluctuations (e.g., factors of 3) in the smoking population of these venues may occur. A further significant problem noted by the participants is that some building codes do not require that the ventilation system actually be operated, especially in the small non-chain establishments. However, it should be noted that in California, Cal-OSHA requires employers to ventilated workspaces during working hours, under (CCR Title 8, 5142).

Comment: Despite the wealth of ETS data in the literature compiled in more than 1/2 dozen reports, plus the fact that indoor air quality models have been under development for more than 40 years, the panel did not use either models or data to characterize existing ETS exposures in hospitality venues. The panel did not apply the indoor air quality procedure in ASHRAE 62, section 6.2, which provides a direct solution to the problem by restricting the concentration of ETS to some specified acceptable level. No data were presented to substantiate the panelists' belief that 90% reductions in ETS concentrations were obtainable under either controlled studies or in the field, especially in view of the caveats raised about placement of supply air ducts, turbulent flows, and blowing smoke down or toward the workers (as often happens in casinos). Moreover, in view of OSHA's estimates of more than 13,000 workers' deaths per year from ETS exposure, the panel's attitude that only a 90% reduction is sufficient for ETS control to protect workers seems cavalier. The panels' confidence in displacement ventilation is not well founded.   In addition, the panels' conclusion on ETS-RSP being poorly correlated to more specific measures is not supported (e.g. see EHP, 107, suppl. 2, pp 225-388 (May 1999). Individual variability in cotinine levels does not compromise assessment of ETS dose (Repace et al., 1998).

Environmental tobacco smoke (ETS)

This following demonstrates application of the indoor air quality procedure specified in ASHRAE Standards 62-1981, 62-1989, and 62-1999 to ETS, providing the "direct solution" to the ventilation rates necessary for control..

Hazard Assessment. Environmental tobacco smoke (ETS) is the smoke emitted into the air from the burning end of a cigarette, pipe or cigar, as well as exhaled smoke from the smoker. The breathing of ETS is known as involuntary smoking or passive smoking. A body of evidence on the health risks of ETS has accumulated during the past two decades, connecting exposure to ETS to premature death. The most recent report on ETS from the United Kingdom, the SCOTH Report (1998), concluded that passive smoking is a cause of lung cancer and ischemic heart disease. The SCOTH report concludes that restrictions on smoking in public places and work places are necessary to protect nonsmokers (SCOTH, 1998). The U.S. National Toxicology Program has include ETS on its list of *known human carcinogens* (NTP, 2000), and the Finnish Parliament similarly voted to list tobacco smoke on its national list of carcinogenic substances (CanFin, 1999).

In the USA, in 1997, the Environmental Protection Agency of the State of California (CalEPA, 1997), in a scientific report which considered public comments from individuals from federal, state, and local government agencies, universities, and various research organizations, as well as from the tobacco industry, concluded that in adult nonsmokers, ETS exposure causes lung cancer and nasal sinus cancer, heart disease mortality, acute and chronic coronary heart disease morbidity, and impairs fetal growth in pregnant women as well as inflicting acute eye and nasal irritation. The California EPA(1997) estimated that U.S. ETS exposure caused 3000 lung cancer deaths (LCDs) annually, from 35,000 to 62,000 heart disease deaths (HDDs) from ischemic heart disease per year, and caused an indeterminate number of cases of retardation of fetal growth.

In 1994, The U.S. Occupational Safety and Health Administration (OSHA, 1994), asserted that "employees working in indoor environments face a significant risk of material impairment of their health due to poor indoor air quality." In support of that determination, OSHA cited the risk of heart and lung fatality to nonsmoking U.S. workers from passive smoking, estimated to range as high as 722 annual cases of fatal lung cancer, and 13,000 deaths from heart disease per year, and that these deaths would be avoided by elimination of nonsmokers' exposure to ETS in the workplace. OSHA(1994) proposed a rule to eliminate nonsmokers' ETS exposures in the workplace. In 1992, the U.S. Environmental

Protection Agency (EPA, 1992) declared ETS to be a "known human lung carcinogen," causing conservatively 3000 LCDs annually.

In 1992, the American Heart Association (AHA, 1992) declared ETS to be a "major preventable cause of cardiovascular disease and death," and estimated ETS-related mortality, from heart disease and cancer combined, to approach 50,000 annually, placing passive smoking as the third leading preventable cause of death, after active smoking and alcohol. In 1991, the U.S. National Institute for Occupational Safety and Health (NIOSH, 1991) declared environmental tobacco smoke (ETS) to be a "potential occupational carcinogen," legal terminology for a substance capable of causing human cancer or reducing its latency period. Based upon biological plausibility and epidemiological studies, a number of risk assessments have estimated the lung cancer mortality caused by passive smoking among U.S. nonsmokers to be of the order of 5000 deaths per year (Repace & Lowrey, 1985; 1990). Wigle et al. (1987) estimated that 330 Canadians die of lung cancer from passive smoking annually.

 In 1986 The U.S. Surgeon General concluded that "involuntary smoking is a cause of disease, including lung cancer, in healthy nonsmokers." Also in 1986, The National Research Council (NRC, 1986) of the U.S. National Academy of Sciences, a congressionally chartered private body established to further scientific knowledge and to advise the federal government on scientific issues, stated that "Considering the evidence as a whole, exposure to ETS increases the incidence of lung cancer in nonsmokers."

The body of evidence from spousal smoking studies suggests that the average excess risk of lung cancer from passive smoking is 24% (95% CI: 13% to 36%) [Hackshaw et al., 1997]. However, for nonsmokers exposed to the smoke of a pack of cigarettes per day or more, the risk increase can be considerably greater; the EPA summarized 12 studies that assessed the increase at these higher levels of smoking. For 9 studies in 5 countries, the excess ETS risk in this category ranged from 57% to 220%; 3 other studies in 2 countries reported risks in the 10% to 20% range (U.S. EPA, 1992, Table 5-11). In the U.S. in 1980, the average smoker smoked 32 cigarettes per day (Repace and Lowrey, 1980). Law et al. (1997) reviewed the evidence from 19 published studies of passive smoking and heart disease; they reported that the average excess risk of ischemic heart disease from passive smoking epidemiological studies is 23% (95% CI:14% to 33%), and concluded that platelet aggregation provides a plausible explanation for the mechanism and magnitude of the effect. Kawachi, et al. (1997) studied coronary heart disease (CHD) in 32,000 female U.S. nurses aged 31 to 61 yr., for nonsmoking women exposed only at work, observed a dose-response for passive smoking and CHD. Adjusted relative risks of CHD were 1.00 [for no exposure],

1.58 *(95% CI, 0.93-2.68)* [occasional exposure], and 1.91 *(95% CI, 1.11-3.28)* [regular exposure]. In this study, regular exposure to SHS at work caused a 91% increase in CHD.

Johnson and Repace (in press) observed that the epidemiological studies of passive smoking and disease are flawed where other exposure is common (e.g., in childhood, in social situations, or in the workplace). In such cases lung cancer and other disease risks may be seriously underestimated. Spouses of non-smokers exposed in other circumstances will be misclassified as nonexposed, contaminating the referent group, and attenuating the risk estimate. For example, Hackshaw et al.(1997) estimate that the odds ratio for lung cancer and passive smoking would have been 1.42 (1.21- 1.66) if those with spousal exposure alone were compared with those who were truly unexposed. By comparison, in a recent meta-analysis of risk associated with workplace exposure, Wells (1997) found an estimated relative risk of 1.39 (95% confidence interval 1.15-1.68) for the five studies meeting basic study quality standards. Repace and Lowrey (1985) found that when both workplace exposure and an unexposed referent group were taken into account in the American Cancer Society study of passive smoking and lung cancer, a population relative risk of 1.2 increased to 1.7.

In fact, Repace and Lowrey modeled the risk of workplace exposure, estimating the average relative risk at 2.0 for U.S. office workers in the 1980's.[5] This result is consistent with a value reported by Reynolds et al. (1996). for women with 30 or more years of workplace exposure, i.e. at ages at which lung cancer mortality begins to become significant. Moreover, all of these analyses focus on average risk. Repace et al. estimated that individuals at the 95th percentile (e.g., those experiencing high smoker density and low air exchange) have exposure -- and risk -- as much as four times as high as those at the median. This result is commensurate with observations of dose and risk (Johnson and Repace, in press). In general, the degree of ETS disease risk depends critically upon the average ratio of the smoker density to the air exchange rate in the exposure venues a person frequents during life; e.g., workplace smoker densities are often far higher than in homes, while air exchange rates may be comparable (Repace and Lowrey, 1985; 1993; Repace et al., 1998).

Hazardous Chemicals in ETS
What chemicals in ETS are responsible for these diseases? ETS is a complex mixture of 5000 chemicals (NRC, 1986), many of which remain to be characterized. Listed in Appendix A are 103 chemicals in tobacco smoke which are identified as hazardous. Although OSHA TLVs exist for many of these

chemicals, the effects of exposure to all of them simultaneously, with the multiple possibility of additivity, synergism or antagonism of effect, is not known. There are 60 known or suspected carcinogens in ETS (Repace and Lowrey, 1985).

Markers for ETS: Nicotine and its primary metabolite cotinine are the best indicators of ETS exposure and dose in nonsmokers. Airborne nicotine has been found to be highly correlated to the number of cigarettes smoked in the presence of nonsmokers and to urinary cotinine in those nonsmokers. During passive smoking, nonsmokers inhale nicotine proportionally to the product of concentration, exposure duration, and respiration rate. Inhaled nicotine is absorbed into the bloodstream through the lung, and is rapidly and extensively metabolized with a half-life of the order of 2 hrs by the liver into cotinine and nicotine N-oxide. The intake of nicotine reflects exposure to other constituents of ETS. In nonsmokers, cotinine has a half-life in plasma on the order of 17 hrs and thus is an indicator of the integrated exposure to ETS over the previous 1 to 2 days. Cotinine in body fluids provides a valid quantitative measure of recent integrated ETS nicotine exposure (Repace et al., 1993; 1998; Benowitz, 1999; Samet, et al., 1999). Cotinine appears in all body fluids and on average is excreted in fixed relationships from plasma (i.e., serum) into saliva and urine. Although nicotine is present in trace amounts in certain vegetables, dietary sources are negligible compared to passive smoking as a contribution to body fluid cotinine. Air nicotine can be used to predict ETS-RSP (Leaderer and Hammond, 1991; Repace and Lowrey, 1993; Daisey, 1999). ETS is the major source of exposure of the population to indoor fine particles (Repace and Lowrey, 1980; Wallace, 1996).

The set of equations given in Table 1 permit calculation of one ETS atmospheric or biomarker from another with reasonable accuracy (Repace & Lowrey, 1993; Repace et al., 1998). For example, the estimated daily average population average ETS-RSP exposure during the mid 1980's (U.S. smoking prevalence about 33%) according to Repace and Lowrey (1985) was Q = 1.43 milligrams of ETS-RSP, and at a respiration rate of 24 m^3 per day, corresponds to a daily average ETS-RSP concentration of R_{ave} = 60 μg/m^3. The equations in Table 1 below permit the corresponding mean nicotine and cotinine levels to be calculated: N_{ave} = R/10 = 6 μg/m^3. The corresponding estimated daily average population salivary cotinine level is then S_{ave} = (0.0071)(24)(6) = 1 ng/ml. The estimated daily average population serum cotinine level is then P_{ave} = (1 ng/ml/ 1.16) = 0.88 ng/ml, and the estimated daily average population urinary cotinine level is given by U_{ave} = (6.5)(0.88 ng/ml) • 6 ng/ml. Repace and Lowrey (1980, 1985) estimated that most-exposed nonsmokers had exposures ten times average, yielding maximum exposed individuals with the following: R_{max} = 600 μg/m^3; N_{ave} = 60 μg/m^3; S_{max} = 10 ng/ml; P_{max} = 9 ng/ml, and U_{max} = 60 ng/ml.

The only national probability sample of any ETS marker is that of serum cotinine, performed in the NHANES III study (Pirkle et al., 1996), with data taken between 1988 and 1991 (U.S. smoking prevalence about 29%). NHANES III reported that adults $\geq$ 17 years who reported work exposure only > 3 hr/day had geometric mean serum cotinine levels of 0.6 ng/ml, home exposure only was 0.7 ng/ml, both home and work exposure, 0.9 ng/ml. A bimodal distribution was observed, with a separation between 10 to 15 ng/ml, the region between heavy passive smoking and light active smoking. Despite the uncertainty introduced by comparing geometric means to arithmetic means and the 12% lower smoking prevalence (**see** CalEPA, 1997, fig. 2.6), the Table 1 model estimates are close to NHANES III observations. The Table 1 model predictions can be compared to data reported in the literature, with general agreement as shown in Table 2 below.

Table 1:

Equations for ETS Estimation

[Repace & Lowrey, RISK ANALYSIS, 13:463-475 (1993)].

ETS Marker, Units	Equation
RSP (PM$_{3.5}$), μg/m^3	$R = 10\,N$
Nicotine, μg/m^3	$N = 22\,D_{hs}/C_v$
Salivary Cotinine, ng/ml	$S = 0.0071\,HN$ (H hr daily exposure)
Serum Cotinine, ng/ml	$P = S/1.16$
Urinary Cotinine, ng/ml	$U = 6.5\,P$

[Repace,Jinot, Bayard, et al, RISK ANALYSIS, 18: 71-83 (1998)].

Table 2. Comparison of model with reported measurements of ETS markers

Marker	Modeled Results	Observations	Reference
	(average-to-peak) (see text) Repace et al. model	Various measurements	**CalEPA (1997):**
ETS-RSP	60 - 600 μg/m^3	5 - 500 μg/m^3 (range)	Section 2.3.3
Nicotine	6 - 60 μg/m^3	0.3 - 65 μg/m^3 (range)	Section 2.3.3; Hammond (1999)
Saliva Cotinine	1 - 10 ng/ml	5.6 - 14.2 ng/ml (average-to-peak)	Section 2.4.2
Serum Cotinine	0.9 - 9 ng/ml	2.0 - 13.7 ng/ml (average-to-peak)	table 2.4 and
Urine Cotinine	6 - 60 ng/ml	7.7 - 49.7 ng/ml (average-to-peak)	table 2.5

Analysis:

General dilution ventilation, [which I will characterize as "reasonably achievable control technology," (RACT) on the basis of the panels' statement that it constitutes more than 99% of current HVAC installations], was judged to be inadequate by the panelists for ETS control. RACT, as applied to pollution sources in outdoor air pollution control, is the lowest limit that a particular source is capable of meeting by the application of control technology that is reasonably available considering technological and economic feasibility (EPA, 1983). Displacement ventilation possibly coupled with ventilated ashtrays in some installations (but impractical for all), which I will describe as "best available control technology," or BACT, was judged to be the best potential control measure by the panelists. BACT, again as applied to pollution sources in outdoor air pollution control, refers to the maximum degree of air pollution reduction attainable by a source considering energy, environmental and economic impacts, through the application of available systems, methods and techniques (EPA, 1983). In outdoor air pollution control, BACT does not permit the source to pollute in excess of any requirements imposed by Section 112 of the Clean Air Act, which regulates hazardous air pollutants.

The panelists' conclusions on ETS controls were reached on the basis of professional judgment, which they identified as being hindered by two major problems. The first problem identified by the panelists was the lack of information on existing exposure levels, and the second one was the lack of

recognized standards of acceptable ETS exposure, so that even if displacement technology were to be universally adopted in the hospitality industry, and 90% exposure reductions could be routinely achieved in practice, there is no guarantee that the residual exposure would yield an acceptable risk for hospitality workers. A further problem which emerged in the discussion is that since some building codes do not require operation of the HVAC systems, these codes would have to be changed.   Also, some establishments may have only natural ventilation. Finally, even assuming that recognized standards limiting ETS exposure are adopted an enforcement apparatus would be required to ensure that the standards are being met.

Outdoor air pollution regulation and control has long been guided by atmospheric models for plume dispersion (Turner, 1970). However, it has not generally been recognized that indoor air pollution, particularly from ETS, can be modeled with far greater accuracy than stationary source outdoor air pollution (Wadden and Scheff, 1983; NRC, 1986; Repace, 1987; Ott, 1999).   ETS concentrations predicted by models agree well with measured values in real settings, both on a minute-by-minute basis and for longer time averages, and the models are especially useful for determining the ventilation required to meet suggested indoor air quality standards (e.g., the National Ambient Air Quality Standard for fine particles (currently 15 $\mu g/m^3$ annual ave. $PM_{2.5}$) for given smoking activity levels (Ott, 1999). In particular, the panelists did not apply existing models to estimate current exposure. Further, the U.S. Environmental Protection Agency has declared ETS to be a human carcinogen, a conclusion endorsed by the National Cancer Institute (NCI, 1993) and the National Toxicology Program (1999). Panelists also did not consider whether the residual exposure of workers to ETS after application of BACT would yield an acceptable risk.

Comment: I will now employ published models of ETS exposure and risk to the hospitality workplace to evaluate the current hazard for workers and patrons with dilution ventilation, and estimate the risk under both RACT and BACT. I observe that the panelist's conclusions that RACT will not control ETS and that BACT will achieve a putative 90% reduction in exposure are not supported by data or by models. Accordingly, below I will apply models to estimate current exposure, compare the model results with data for an accepted ETS atmospheric marker, and employ a dose-response relationship to estimate worker risk under RACT and BACT, and compare the estimated risk with established federal regulatory risk criteria.

Modeled ETS Exposure and Risk in Restaurants, Bars, Casinos ...

Introduction

Repace et al.(1998), Repace and Lowrey (1993), Repace (1987), Repace and Lowrey (1985), and Repace (1984) developed models for ETS exposure, dose, and risk which agree well with observations. It is important to note that these ETS models have gained widespread acceptance in the scientific community:

The National Research Council (1986) observed that the most extensive use of the mass-balance equation for assessing ETS in occupied spaces was by Repace and Lowrey (1980), and observed that the model "predicted ETS-Respirable suspended particle (RSP) levels reasonably well over a wide range of values of input parameters." The model was also favorably reviewed in the 1986 Surgeon General's Report on Involuntary Smoking. Ott et al. (1992) derived and validated a general equation for the mean concentration of ETS in an indoor space and concluded that it was structurally equivalent to the model of Repace (1987). The Monte Carlo model of Repace et al. (1998) for predicting ETS exposures was favorably reviewed by Spengler (1999).

Weiss (1986) commented "on the association between passive smoking and lung cancer and the biological and mathematical assumptions underlying Repace and Lowrey's (1985) assessment of risk." Weiss concluded, in part: "Despite the simplifying assumptions of the risk estimates and the flaws in the epidemiologic data from which they are derived, Repace and Lowrey's figures remain the best current estimates of lung cancer deaths from passive smoking." Kawachi et al. (1989) estimated the "relative risk for lung cancer death from exposure to passive smoking in the workplace ... via an exposure response relationship derived by Repace and Lowrey [1985; 1987]." Wigle et al. (1987) used the methods of Repace & Lowrey (1985) to assess lung cancer risk in Canadians. Nagda et al. (1989) assessed the lung cancer risks of passive smoking for flight attendants and passengers on U.S. carriers in part using the risk assessment model of Repace and Lowrey (1985). The U.S. EPA (NCI, 1993) described the risk assessment approach of Repace and Lowrey (1985) for lung cancer as "a novel approach that contributes to the variety of evidence for evaluation [of lung cancer risk] and provides a new perspective on the topic." Tancrede et al. (1987) used the risk assessment model of Repace (1984) to estimate a mean lifetime risk for lung cancer for U.S. nonsmokers from passive smoking of about 5 per thousand, with a 98th percentile of 3.8%. Finally, Samet and Wang (2000) have observed that the calculations made possible by the exposure, dose, and risk models of Repace et al. (1998) for estimating worker risk of lung cancer illustrate that passive smoking

must be considered as an important cause of lung cancer death from a public health perspective, since exposure is involuntary and not subject to control.

Exposure Modeling

Ott (1999) in the OSHA-sponsored Workshop on Environmental Tobacco Smoke Exposure Assessment, observed that much progress has been made over four decades in developing, testing, and evaluating the performance of mathematical models for predicting pollutant concentrations from smoking in indoor settings. Ott (1999) further commented that although largely overlooked by the regulatory community, these models provide regulators and risk assessors with practical tools for the quantitative estimation of ETS exposures. In the same workshop, Spengler (1999) observed that generally the highest ETS exposures are occurring in bars, restaurants, and nightclubs, and using the techniques developed by Repace et al. (1998) reasonable estimates may be made of ETS exposures in offices, restaurants and bars. Repace et al. (1998) have shown that ETS exposure is directly proportional to the smoker density D_{hs}, (in units of habitual smokers per 100 m^3), and inversely proportional to the air exchange rate ϕ_v (in units of air changes per hour: h^{-1}), where a habitual smoker is assumed to smoke at the national average rate of 2 cigarettes per hour, where the smoker density $D_{hs} = 100$ n_{hs}/V, and where n_{hs} is the number of habitual smokers and V is the volume of the space in cubic meters. ASHRAE Standard 62-1989, Ventilation for Acceptable Indoor Air Quality (now supplanted by ASHRAE Standard 62-1999) specifies design ventilation rates based on design occupancy, i.e., 10 L/s per design occupant, and so many occupants per 100 m^2 (100 m^2 is ~1000 ft^2) this becomes a volumetric measure when a ceiling height is assumed. Therefore, for a given smoking prevalence, the design occupancy determines both the smoker density and the air exchange rate.

Repace(1987) derived an equation for the calculation of ETS-RSP levels in units of micrograms per cubic meter ($\mu g/m^3$) for a workplace as a function of the habitual smoker density D_{hs} (units HS/100m^3) in the building and the building's air exchange rate ϕ_v (units hr^{-1}):

$$RSP_{ETS} = 220\frac{D_{hs}}{\phi_v}$$ **(Eq. 1),**

where ϕ_v (fi-vee) is the air exchange rate due to dilution ventilation. The equation incorporates a 20% removal rate for ETS-RSP deposition on surfaces, and assumes an emission rate of 14 mg of ETS-RSP per cigarette and a smoking rate of 2 cigarettes per smoker per hour. If there is additional air cleaning, ϕ_v would be

increased by the air exchange rate due to the air cleaning. ETS nicotine levels may be estimated by dividing Equation 1 by ten (Repace et al., 1993, 1998).

ASHRAE Standard 62-1999 (values were the same for the predecessor Standard 62-1989) specifies the following occupancies, in persons per 100 m^2 of floor area (Table 3) for the given hospitality venues: If a smoking prevalence of 25% is assumed, then the number of expected smokers and the smoker density (in units of habitual smokers per 100 m^3) may be estimated, assuming a 4 meter ceiling height multiplied by the unit space area for the number of occupants. The product of smoking prevalence and occupancy (number of persons per 100 m^2) yields the estimated number of smokers. The corresponding air exchange rate for pollutant removal, in units of air changes per hour (ACH) may be calculated, as follows.

Table 3. Smoker density and Air Exchange Rate (dilution ventilation) at full occupancy for various hospitality venues for a ceiling height of 4 m under ASHRAE Standard 62-1999 per 100 m^2 of floor area, and a smoking prevalence of 25%. (US smoking prevalence in 1993 = 24%.)

Hospitality Venue	Design Occupancy, Persons per 100m^2	Design Ventilation Rate (Lps/occ)	ϕ_v, air changes/hr	n_{hs}, # of habitual smokers (HS) per 100 m^2	D_{hs}, habitual smoker density, HS per 100 m^3
Smoking Lounge	70	30	19	70	17.5
Bar, Cocktail Lounge	100	15	13.5	25	6.25
Dining Room	70	10	6.3	18	4.5
Gambling Casino	120	15	16.2	30	7.5
Bowling Alley	70	13	8	18	4.5

The air exchange rate ACH = (Occupancy, Persons)(Vent Rate Lps/P)(1 m^3/1000L)(3600 s/hr) / (space volume, m^3). For example, for a Dining Room, an occupancy Occ = 70 persons per 100 m^2 of floor area, or per 400 m^3 of space volume, assuming a 4 m ceiling. For a smoking prevalence of 25%, the number of habitual smokers n_{hs} = (0.25)(70) = 18, the habitual smoker density D_{hs} = (.25)(70)/(400) = 4.5 smokers per 100 m^3. The air exchange rate is ϕ_v = (70 occ x 10 Lps/occ x 1 m^3/1000 L)(3600 s/h) / (400 m^3) = 6.25 h^{-1}. [Because there is no enforcement of operational ventilation rates, there is an economic incentive for building owners to supply less.]

Table 4. Estimated RSP and Nicotine Concentrations Based on Equations 1 & 2, for cigarette smoking.

Hospitality Venue	D_{hs}, habitual smoker density, HS per 100 m³	ϕ_v, air changes/hr (design, not enforced)	Estimated RSP level ($\mu g/m^3$)	Estimated Nicotine level ($\mu g/m^3$)	Comment
Smoking Lounge	17.5	19	203	20	Levels will triple if all smoke at once
Bar, Cocktail Lounge	6.25	13.5	102	10	More intensive smoking likely
Dining Room	4.5	6.3	157	16	
Gambling Casino	7.5	16.2	102	10	More intensive smoking likely
Bowling Alley	4.5	8	124	12	

The RSP and nicotine concentrations, estimated in Table 4 for the RACT case of dilution ventilation are liberal in that they assume full occupancy, but are conservative in other respects: (a) since nonsmokers are known to avoid smoky restaurants and bars (Biener et al., 1999), the number of smokers will likely be greater than their prevalence in the population; (b) the air exchange rates are likely to be less than design because to provide design rates of ventilation costs money, and there is no enforcement of operational rates; (c) in bars, nightclubs, and casinos, smoking is likely to be more intensive than the national average of 2 cigarettes per hour (chain smokers smoke up to 6 cigarettes per hour); (d) cigars make more pollution than cigarettes (Repace et al., 1998); (e) if smokers are restricted to designated areas, hospitality workers will work in environments where almost everyone is a smoker, increasing the number of smokers by as much as a factor of 4. For restaurants, cutting back on ventilation might mean air exchange rates closer to 1 air change per hour rather than 6. Nevertheless, Table 4 levels can be compared with the range of observations reported by EPA (1992): for restaurants average RSP values (ch. 3, fig. 3-8) ranged from 40 to 1000 $\mu g/m^3$, and nicotine in restaurants (not necessarily in the same ones) from 6 to 18 $\mu g/m^3$, consistent with the predictions in Table 4, and the caveats in this paragraph.

In table 4, for habitual smoker densities D_{hs} ranging from 4.5 to 6.25 habitual smokers per hundred cubic meters, the estimated nicotine levels range from 10 to 20 $\mu g/m^3$. By comparison, Lockhart (1995) has expressed the nicotine concentration in pubs as a function of active smoker density (the active smoker

density D_{as} is the average number of burning cigarettes per hundred cubic meters ($D_{as} = 1/3\ D_{hs}$) was measured in Canada in 1995. Figure 1 below shows measured levels in ten Vancouver British Columbia (BC) restaurants and pubs with smoking and nonsmoking sections in 1995 (Lockhart, 1995). The smoking prevalence in BC is 23% (Gallup, 1996). It is seen that nicotine levels ranged as high as 40 $\mu g/m^3$ in the smoking sections and as high as 30 $\mu g/m^3$ in the nonsmoking sections, and that the differences between the smoking and nonsmoking sections were slight, due to "well-mixed" dilution ventilation. This corresponds to estimated RSP levels above background of 300 to 400 $\mu g/m^3$, comparable to the levels measured by Repace and Lowrey (1980). Thus the values predicted in table 4 correspond to active smoker densities of from 1.5 to 2.0 active smokers per hundred cubic meters, and to measured nicotine concentrations (interpolating between the smoking and nonsmoking section curves) ranging from about 15 to 30 $\mu g/m^3$, higher than predictions.

Presumably, these restaurants and pubs should have been ventilated according to ASHRAE Standard 62, which (for both the 1989 and 1999 versions) specifies 15 Lps per occupant for pubs and 10 Lps per occupant for restaurant dining rooms. As shown above, this corresponds to design air exchange rates of the order of 15 hr^{-1}, and should have resulted in nicotine concentrations of the order of 10 $\mu g/m^3$ (an active smoker density of 2 burning cigarettes per 100 m^3 corresponds to a habitual smoker density of 6 habitual smokers per 100 m^3). That levels as much as 50% higher than predicted were observed suggests that either the actual ventilation rates were lower than the level mandated by the ASHRAE Standard, or that the smoking rates were higher, or some combination of the two. In either case, this suggests that the estimates in Table 4 are conservative.

Although panelists were sanguine about the prospects of displacement ventilation, I emphasize that no data was presented to support its efficacy on ETS. Its usual application is a one-pass system with 100% outside air introduced into a designated nonsmoking section, with positive air flow directed through an open passageway into a negatively-pressurized smoking section.

ETS, SMOKING VS. NONSMOKING SECTIONS

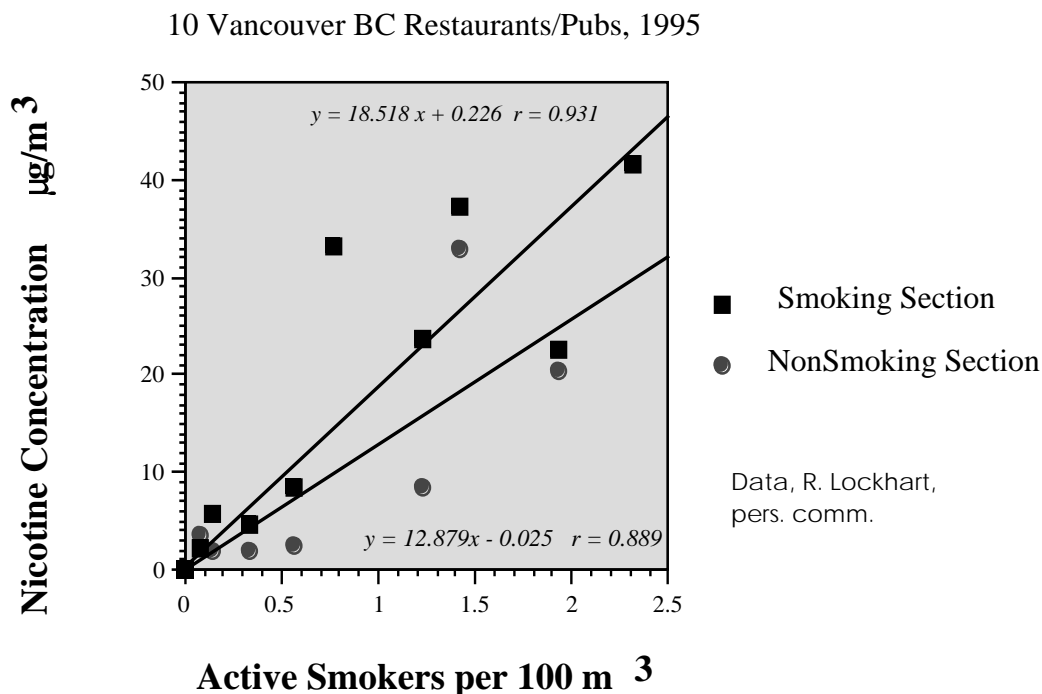10 Vancouver BC Restaurants/Pubs, 1995

Figure 1. Nicotine levels measured in 10 Vancouver, British Columbia Pubs for the Heart and Stroke Foundation of BC and Yukon (Lockhart, 1995). The active smoker density D_s (the average instantaneous density of burning cigarettes) is 1/3 of the habitual smoker density D_{hs} of the habitual smoker model of Repace (1987).

Regulatory Risk Levels

Involuntarily imposed worker risks from ETS can be compared to societal standards for permissible human exposures to environmental carcinogens such as industrial chemical emissions and radionuclides in air and water, and carcinogenic molds and pesticide residues in food. Several U.S. federal regulatory agencies promulgate regulations and standards to protect the public from exposure to environmental carcinogens. It is of interest to inquire as to what levels of population cancer risk typically trigger regulation, what levels are beneath regulatory concern, and how consistently are they applied among various federal agencies. Travis et al.(1990) reviewed the use of cancer risk estimates in prevailing U.S. federal standards and in withdrawn regulatory initiatives, to determine the relationship between risk level and regulatory action in 132 U.S. federal regulatory decisions of record concerning lifetime risk of mortality.

Travis et al. describe two technical risk assessment terms: *de manifestis* risk and *de minimis* risk. A *de manifestis* risk is literally "a risk of obvious or evident

concern," and has its roots in the legal definition of an "obvious risk", i.e., one recognized instantly by a person of ordinary intelligence. *De manifestis* risks are those that are so high that U.S. federal regulatory agencies almost always acted to reduce them, and *de minimis* risks are so low that agencies almost never acted to reduce them. For various reasons, risks falling in between these extremes were regulated in some cases but not in others; however, **residual risks after control are generally *de minimis*.** Travis et al. found when the population at risk was large, as with ETS, *de manifestis* risk corresponded to a lifetime risk of mortality of 3 per ten thousand (3×10^{-4}), and *de minimis* risk was one per million (1×10^{-6}). The U.S. Occupational Safety and Health Administration has defined a working lifetime (45 yr.) risk level of 1 death per 1000 workers at risk as corresponding to a "significant risk of material impairment of health" (U.S. DOL, 1994).

Risk Modeling, Dilution Ventilation (RACT-Case)

ETS risks are estimated based on the ETS-RSP levels from Table 4, using the exposure-response models of Repace and Lowrey (1985b), Repace and Lowrey (1993) and Repace et al. (1998). Under these models, a time-weighted 8-hr average exposure for 260 days/yr over a 40 year working lifetime to an ETS-RSP level of 75 $\mu g/m^3$ corresponds to a working lifetime risk of 1 per 1000 for lung cancer mortality, and 1 per 100 for heart disease mortality. These exposure and risk assessment models may be used to assess the fatal lung cancer and heart disease risk to hospitality workers from ETS exposure at work. This modeling is summarized in Table 5 for five hospitality venues. Under dilution ventilation and occupancy as specified by ASHRAE Standard 62-1999, and with a typical U.S. average smoking prevalence, the combined estimated lung cancer and heart disease mortality risks to hospitality workers range from 15 to 30 per 1000, exceeding all applicable environmental and occupational regulatory levels. A risk of 20 per 1000 is twenty thousand times the *de minimis* risk level. The risks calculated in Table 5 are likely to be underestimated relative to real-world situations, because of two factors: first, since there is no enforcement of operational ventilation rates, and since it costs money to treat outdoor air which is cold or hot and humid, operational rates will be less than design -- it is a simple matter of turning a dial to close down outside air dampers. Second, smoky restaurants, bars, and casinos are likely to have far less nonsmokers and far more smokers than national prevalence figures suggest, because nonsmokers are known to avoid such establishments (Biener et al., 1999); in fact during 1995, based on data provided by Biener et al., the number of Massachusetts nonsmokers who said they avoided smoky restaurants and bars was 80,000 more the total number of Massachusetts smokers.

Table 5. Estimated ETS-RSP concentration and associated[c] lung cancer, heart disease and combined risk for hospitality industry workers using dilution ventilation, assuming a smoking prevalence of 25%, (approx. the U.S. average), and compliance with the ASHRAE Standard 62 1999.

Smoking Area	Estimated ETS-RSP $(PM_{3.5})$, µg/m³		Est. Excess Lung Cancer Mortality per 1000 workers	Est. Excess Heart Disease Mortality per 1000 workers	Est. Total Excess Mortality per 1000 workers
Smoking Lounge[†]	203		2.4	24	26
Bar, Cocktail Lounge	102		1.4	14	15
Dining Room	157		2.0	20	22
Gambling Casino	102		1.4	14	15
Bowling Alley	124		1.7	17	19
Risk Level	LCD[a]	HDD[b]			
de minimis risk	.075	.0075	0.001	0.001	0.001
de manifestis risk	22.5	2.3	0.3	0.3	0.3
OSHA Significant risk	75	7.5	1	1	1

†: assumes workers serve in lounge; a: lung cancer death; b: heart disease death
. c: assumes worker exposure for 8 hours per day, 260 days/yr; 40 yr Working Lifetime (WLT) (NB: Since OSHA assumes a 45-year WLT Sig. Risk occurs at a slightly lower concentration than shown (40/45)(75) or 67 µg/m³ for lung cancer and 6.7 µg/m³ for heart disease.

Based on Table 5, assuming regular patrons have an exposure duration of about 10% of the workers, or 4 hrs per week, the combined lung cancer and heart disease mortality risks to the patrons also exceeds all environmental and occupational regulatory risk levels.

Such increases in RSP levels (over a typical non-ETS background of ~20 µg/m³) would also be expected to result in the denial of access to the workplace and public places of accommodation for both workers and patrons who are asthmatics or who suffer from other cardio-respiratory diseases. Dockery and Pope (1994) found that total daily mortality associated with particulate air pollution shows an approximately 1% increase per 10 µg/m³ daily increase in particulate matter below 10 microns in aerodynamic diameter (PM_{10}). They also found that particulate air pollution is even more strongly associated with cardiovascular mortality, with a dose-response showing a 1.4% increase per 10

$\mu g/m^3$ increase in PM_{10}.   The U.S. National Ambient Air Quality Standard (NAAQS) for $PM_{2.5}$ protects against health effects such as premature death, increased hospital admissions and emergency room visits (primarily the elderly and individuals with cardiopulmonary disease); increased respiratory symptoms and disease (children and individuals with cardiopulmonary disease such as asthma); decreased lung function (particularly in children and individuals with asthma); and against alterations in lung tissue and structure and in respiratory tract defense mechanisms.   The level of 15 $\mu g/m^3$ of the annual standard is an annual average which defines clean air.   The supplemental 24-hr standard of 65 $\mu g/m^3$ is intended to prevent short-term peaks from impacting public health (Fed. Reg., 1997).

In fact, Eisner et al.(1998) studied the association between ETS exposure and respiratory symptoms in a cohort of 53 bartenders before and after California's prohibition on smoking in all bars and taverns in 1998.   74% of the bartenders initially reported respiratory symptoms; of those symptomatic at baseline, 59% no longer had symptoms at follow-up.   77% initially reported sensory irritation symptoms; at follow-up, 78% of these had symptom resolution. After ETS exposure completely ceased, objective measures of pulmonary function showed a marked 5% to 7% improvement after only one month of smoke-free air. Eisner et al. (1998) concluded that establishment of smoke-free bars and taverns was associated with improvement of respiratory health.

As discussed above, Spengler (1999) has observed that ETS exposures in restaurants can be modeled using the techniques of Repace et al. (1998). Samet and Wang (2000) have observed that the risk models of Repace et al. (1998) are useful for estimating worker risk.   Figure 2 combines these models to estimate ETS risk as a function of ventilation rate in a restaurant at a smoking prevalence of 29%, equivalent to 2 smokers per 1000 ft^2 or per ~100 m^2 of floor area.   It is seen that for RACT, or ordinary dilution ventilation to reduce the ETS risk to restaurant workers to *de minimis* levels would require ventilation rates in excess of 100,000 Lps/occ, levels which are impractical by more than 4 orders of magnitude (10,000-fold).   At a smoking prevalence of 25%, as used above, ETS risks are reduced only slightly compared to the risks shown in Fig. 2. If one assumes that BACT, or displacement ventilation, can reduce ETS risks to 1/10, equivalent to a ten-fold increase in ventilation efficiency, the risks still remain unacceptable by three orders of magnitude (1,000-fold).   This is discussed in further detail below.

ETS RISK AND VENTILATION: RESTAURANT WORKERS

29% Smoking Prevalence; 70 Persons per 100 m ^{2}

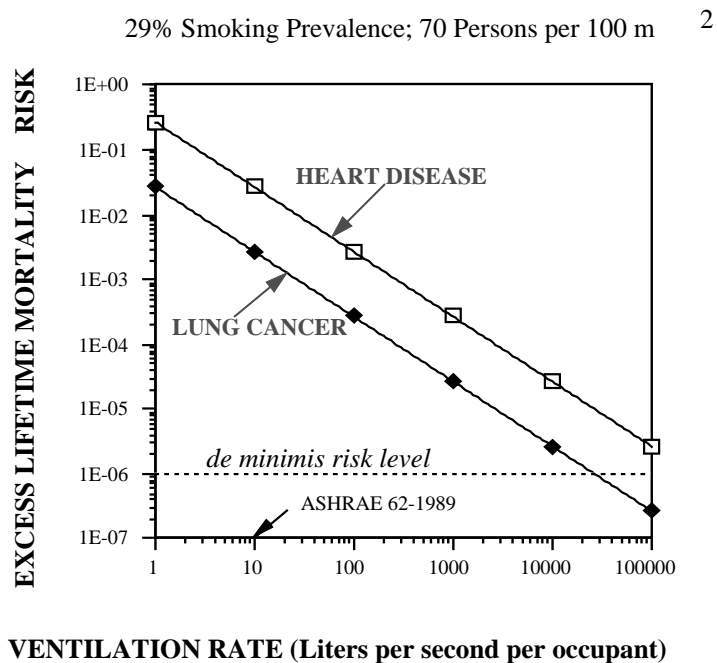**VENTILATION RATE (Liters per second per occupant)**

Figure 2. Estimated excess risks of lung cancer and heart disease for hospitality workers for a smoking prevalence of 29%, a restaurant occupancy of 70 persons per 100 m^2, as a function of ventilation rate supplied per occupant.   The ASHRAE Standard recommendation of 10 Lps/occ (20 cfm/occ) is shown (Risks are estimated based on the models of Repace and Lowrey, 1985; 1993; Repace et al., 1998). Risks to workers in bars and casinos would likely be greater, due to higher actual smoker prevalence and closer proximity of bartenders and casino dealers to smoking. Note that ASHRAE Standard 62-1999 has identical occupancy and ventilation requirements.

Siegel(1993), in a review of the literature, found that restaurant waitresses had a 50% to 100% higher risk of lung cancer compared to the general population. EPA(1992, p. 187) estimated that the annual risk of lung cancer for U.S. nonsmoking women from the general population from all causes was 15 per 100,000, corresponding to a 70 year lifetime risk of 10 per 1000, with 1/3 of that risk from passive smoking, for an estimated lifetime risk from passive smoking at about 3 per 1000 above a non-ETS background of 7 per 1000. By comparison, the estimated excess ETS risk for lung cancer for restaurant workers in Figure 2 from passive smoking in a restaurant workplace in compliance with the ASHRAE Standard is about 3 per 1000, which when added to the general population background, would constitute a 100% increase. Thus, the estimated lung cancer risk from Figure 2 is in good agreement with the results of EPA and Siegel.

Risk Modeling, Displacement Ventilation (BACT)

As discussed above, the OSHA Ventilation Workshop Panelists concluded that displacement ventilation had the potential to achieve 90% reductions in ETS concentrations, although no data on real hospitality facilities taken for actual workers was presented to support this contention. Nevertheless, for the purposes of this analysis, I will presume that this can be accomplished, and that the technology will work as designed and be properly operated and maintained over a working lifetime. Using dilution ventilation, the hospitality venues of Table 4 using perfectly designed and properly operated HVAC systems would have total working lifetime risks for workers of from 15 to 30 per 1000. I will assume that 90% reductions on this ideal level (and not the realistic levels shown in Figure 1) can be achieved using displacement technology or BACT. This would yield estimated combined lifetime risks for workers of from 1.5 to 3 per 1000, which still exceed all environmental and occupational regulatory levels. A risk of 2 per 1000 is two thousand times the *de minimis* risk level. There is a third concept in outdoor air pollution control known as LAER, or lowest achievable emissions reductions (USEPA, 1983). This is the most stringent level of reduction which is contained by any source or category of sources. BACT clearly will not achieve LAER. This level of reduction, however, is easily achieved by smoking bans such as in the State of California. Smoking bans reduce the risk from ETS exposure to zero.

Airborne carcinogens, are not regulated using RACT or BACT. They fall under Section 112 of the Clean Air Act, which governs hazardous air pollutants (HAPS), i.e., pollutants which "may reasonably be anticipated to result in an increase in mortality or an increase in serious irreversible, or incapacitating irreversible, illness" (CAA, 1977). Hazardous air pollutants are regulated under a National Emission Standard for Hazardous Air Pollutants, or NESHAPS. HAPS are regulated after a risk assessment. Severe emissions limitations are imposed HAP sources. The emissions limitations are designed to reduce the aggregate or population risk to *de minimis* levels. This is accomplished by estimating dose-response relationships, estimating population exposure, and requiring reduction of the source emissions to limit the downwind concentration to *de minimis* risk levels. This means less than 1 estimated death per lifetime for the population at risk, irrespective of the costs of containment, since Section 112 is exempt from economics tests.

Table 6 below shows the risks before control for various hazardous air pollutants regulated by the US EPA, compared with ETS. In the case of arsenic, the only copper smelter in the U.S. to emit arsenic (an impurity in the ore) closed down because it could not meet the NESHAPS requirement economically. Note

that with the exception of asbestos, all the remaining HAPS pollutants are themselves also constituents of ETS (Repace and Lowrey, 1985; 1990). Risk assessments have been performed for ETS by the U.S. EPA (1992), by Repace and Lowrey (1985), and by others (Repace and Lowrey, 1990) and average 5000 $\pm$ 2500 LCDs/year. Unlike the other ETS risk assessments which have been performed, Repace and Lowrey (1985) derived a dose-response relationship. Clearly, based on the number of deaths, ETS falls in the category of a hazardous air pollutant. Note that NESHAPS requirements override both BACT and RACT. If regulated under a NESHAPS, ETS deaths would have to be less than 1 death per year, nationally. NESHAPS are also set such that risks to the most-exposed individual are controlled to acceptable levels. Note that unlike ETS, which is a best-estimate risk, the remaining pollutants are generally estimates at the 95% upper confidence interval of a maximum likelihood estimate.
:

Table 6. U.S. EPA-Estimated mortality for Hazardous Outdoor Air Pollutants Regulated under the Clean Air Act compared to those estimated for ETS (US EPA, 1992; Repace and Lowrey, 1990), which is not federally regulated.

Hazardous Air Pollutant	Estimated Annual Cancer Mortality
Environmental Tobacco Smoke	3000
Vinyl Chloride*	<27
Airborne Radionuclides*	17
Outdoor Asbestos Emissions*	15
Coke Oven Emissions*	<15
Benzene*	<8
Arsenic*	<5

*Regulated under Section 112 of the Clean Air Act

ETS itself contains 5 HAPs, vinyl chloride, radionuclides (e.g. Po^{210}), coke-oven like chemicals (e.g. polycyclic aromatic hydrocarbons), benzene, and arsenic. Cigarettes have been manufactured with asbestos filters. In addition, 47 chemicals in ETS have been classified as "hazardous waste" under RCRA (Appendix C). However, alone among well-known toxic and carcinogenic chemicals, ETS is not subject to a NESHAPS, OSHA TLV, or air quality standard. In addition, Congress has exempted regulation of tobacco products under TSCA. Although EPA classified ETS as a "known human carcinogen" in 1992, EPA has no authority to set indoor air quality standards, is explicitly forbidden by Congress from regulating indoor air quality, and EPA's ETS research program was abandoned in 1990. While OSHA proposed (1994) to regulate ETS in workplaces, work on its proposed rule ceased in 1995. In the absence of any <u>official</u> safe level for ETS, it is foolish to make -- or accept -- vague claims that ventilation can

control ETS.   The only prudent approach is a smoking ban. Smoking bans will achieve *de minimis* risk without any engineering controls.

Although smoking bans have been widely opposed by the hospitality industry, their opposition been founded in a misguided belief in business losses that have failed to materialize in any part of the U.S. Although many in the hospitality industry worry about loss of smoking customers, few seem to realize they have already lost a substantial amount of nonsmoking trade. It might be expected that since many nonsmokers avoid smoky places (Biener et al., 1999; Glantz, 1999), and since adult nonsmokers outnumber adult smokers by more than 3:1 nationally, that there would be no economic penalties. In fact, as Figure 3 shows, smoking bans have had no discernible adverse economic impact in California.

California Restaurant and Bar Sales Before and After Smoke-free Laws

First Quarter Taxable Sales Figures for Restaurants & Bars, State of California '92-'99
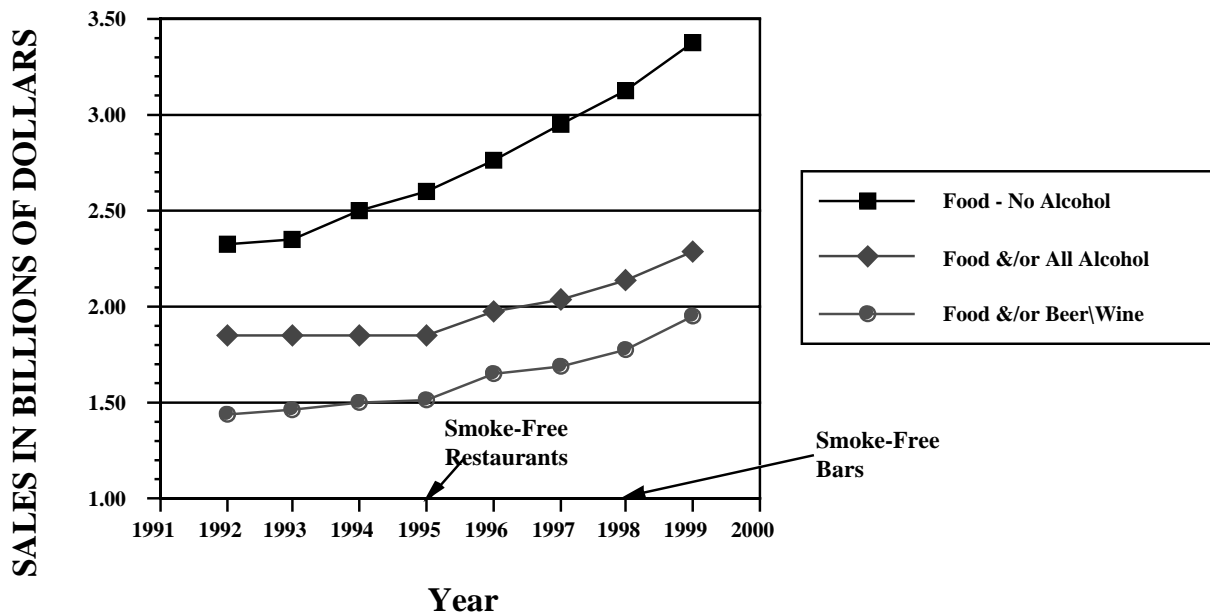*Source: California Dept. of Health; California Board of Equalization*

Figure 3. Data from California food and beverage industry tax receipts shows no economic impact from smoke-free restaurant or bar ordinances.

Conclusions on ETS Risks under RACT and BACT

The best that current dilution engineering technology (RACT) can provide is estimated worker risks of the order of 20 thousand times the *de minimis* level. Similarly, the best that future displacement engineering technology (BACT) can provide is estimated worker risks of the order of 2 thousand times the *de minimis* level. Smoking bans (LAER) provide risks thousands of times lower (actually zero) at no discernible cost to the industry as a whole, while providing obvious significant public and worker health benefits.

The Tobacco Industry and Ventilation

Background. In 1973, ASHRAE Standard 62-73 Section 6.2, specified from 30 cubic feet per minute per occupant (cfm/occ) (15 Lps/occ) to 50 cfm/occ of outdoor makeup ventilation air for bars and cocktail lounges and 10 to 20 cfm/occ for restaurant dining rooms. In 1981, ASHRAE Standard 62-1981, in order to save energy, specified different ventilation rates for smoking and nonsmoking in Section 6, Table 3: smoking restaurants 35 cfm/occ, nonsmoking 7 cfm/occ. Smoking bars and cocktail lounges, 50 cfm/occ, nonsmoking 10 cfm/occ. These rates were recommended by a committee of ventilation engineers from industry in a consensus process. ASHRAE 62-1981 also added a new "indoor air quality procedure" which would bring contaminants to some specified acceptable levels (similar to the procedure I have employed above in Figure 2). It further recommended that "best available control technology be employed for toxic indoor contaminants such as asbestos, radon, and formaldehyde, but stated that for other contaminants such as tobacco smoke, precise quantitative treatment can be difficult."

The tobacco industry's response to these new two-tiered rates, which imposed a penalty on smoking establishments, was to disrupt the committee's functioning using parliamentary maneuvers (Repace, 1991) and ultimately to threaten ASHRAE with litigation. The net result, incorporated into ASHRAE Standard 1989, was abolition of the differential rates for smoking and nonsmoking establishments. The new rates for restaurants were a blanket 20 cfm/occ independent of smoking status, and for bars, 30 cfm/occ. However, in a further capitulation to the tobacco industry, a footnote to the standard stated: "Table 2 prescribes rates of ... outdoor air required for acceptable indoor air quality. These values have been chosen to control CO_2 and other contaminants with an adequate margin of safety and to account for health variations among people, varied activity levels, and a moderate amount of smoking." In the foreword to the Standard, the following opaque disclaimer appeared: "... with respect to tobacco smoke and

other contaminants, this standard does not, and cannot ensure the avoidance of all possible adverse health effects, but it reflects recognized consensus criteria and guidance."

The tobacco industry widely touted ASHRAE 62-1989 in support of its contention that tobacco smoke could be controlled by ventilation, and that smoking bans were not needed. Confidential "draft" tobacco industry strategy documents from a Settlement Agreement Website observed that because ETS was perceived to be a health risk and annoyance, and smoking bans were proliferating. The ASHRAE Standard 62-1989 revision was identified as a major issue: "The proposed revised standard ... would preclude any building where ETS is present from being classified as having acceptable indoor air quality. For new buildings designed to adhere to this standard the result could be the same de facto prohibition of smoking contemplated by the OSHA [Indoor Air Quality] proposal." The strategy document's listed Goal: "Perpetuate the substance of Standard 62-1989, which provides for smoking, as the accepted standard and amend the terms of the revision to accommodate smoking." Litigation options were among the actions considered to further this goal. The hospitality industry was singled out as a major target for "accommodation," with hotels, restaurants, pubs and taverns specifically mentioned. [pmdocs.com, Worldwide Strategy and Plan, pp 2-4, Bates # 2060577486, -87, -88; -502, -522], see appendix D below.

However, despite numerous attempts at amending the standard and several appeals to both ASHRAE and ANSI, the industry failed. After a decade, a new version of the standard was issued which reflected the general medical/scientific consensus on ETS: ASHRAE Standard 62-1999 contained an addendum 62e, which repealed the statement that the ventilation rates in Table 2 "accommodate a moderate amount of smoking." The Foreword to Standard 62-1999 noted: "Since the last publication of this standard in 1989, numerous cognizant authorities have determined that environmental tobacco smoke is harmful to human health. [A list of authorities was given, including the US EPA, WHO, AMA, ALA, NIOSH, NAS, OSHA, and the Surgeon General.]   This addendum does not prohibit smoking or any other activity in buildings, but rather removes the statement that the recommended ventilation rates are intended to accommodate a moderate amount of smoking." The indoor air quality procedure continued to be listed as an alternative performance method to the Ventilation rates prescribed in Table 2.

Current Tobacco Industry Statements on ETS, Ventilation, and the Hospitality Industry

The major tobacco companies, Philip Morris (PM), RJ Reynolds (RJR), and British American Tobacco (BAT) [BAT's U.S. subsidiary is Brown &

Williamson] maintain corporate websites {PhilipMorris.com; RJReynolds.com; BAT.com} which discuss *inter alia*, ETS health and ventilation issues, and the hospitality industry. The relevant documents are given in Appendix D below.

Philip Morris (PM), the largest U.S. tobacco company, maintains the most extensive ETS information (see website headings titled: *Secondhand Smoke; Options Program; Accommodation; Ventilation*:   PM states that while it recognizes that ETS can be annoying to nonsmokers, there are options to "minimize" ETS, and a "sizable segment of the population continues to support 'accommodation' of smoking. PM has an "Accommodation Program" which targets business owners in the hospitality industry by offering access to information on the latest ventilation technology. Ventilation, says PM, plays an important role in accommodation.   However, PM asserts that "owners of restaurants, bars, casinos and other hospitality venues should be permitted to choose what kind of smoking polices to adopt for their establishments. "Designated areas, separate rooms, smoking lounges, and sometimes, no separation at all, are ways that business owners choose to accommodate the 'preferences' of nonsmokers and smokers," says PM. PM cites the Courtesy of Choice program sponsored by the International Hotel and Restaurant Association. The program is supported by local hospitality associations, Philip Morris International, and other tobacco sponsors in some 47 countries and is available in almost 8000 individual hospitality outlets." PM acknowledges that "many scientists and regulators have concluded that ETS poses a health risk to nonsmokers, but that "we do not agree with many of their conclusions." Philip Morris states that "So long as unwanted exposure is minimized, ... concerns regarding ETS can be addressed without banning smoking." (Appendix D).

RJ Reynolds states on its website under "*Secondhand Smoke*," that although "many people find secondhand smoke annoying, and that some ... believe it presents a risk to their health ... There are many ways to allow smokers and nonsmokers to 'peacefully coexist' in public places without resorting to smoking bans: Common courtesy ... -- coupled with adequate ventilation and filtration, and designated smoking areas ... ."   RJR also "does not believe that the scientific evidence concerning secondhand smoke establishes it as a risk factor for lung cancer, heart disease, or any other disease in adult nonsmokers."   "... business owners know best how to satisfy their customers, and they should be allowed to decide whether they want to allow, restrict or ban smoking in their establishments." (Appendix D).

BAT also recognizes {website headings *Environmental tobacco smoke; ETS -- accommodating both smokers and non-smokers*] that ETS "is a significant

annoyance" and that "there have been claims that ETS is a cause of disease ... " however ... we do not believe that exposure to ETS is a risk factor for chronic disease in adults." "We support sensible accommodation of ... smokers and nonsmokers ... through good ventilation." "We also support the Courtesy of Choice campaign run by the International Hotel and Restaurant Association. It aims to help the hospitality industry to accommodate all its customers in restaurants, convention centres, cafes, bars, clubs and hotels, and involves technical analysis of ventilation and owners allocating flexible smoking and non-smoking areas." "... we do not believe that public smoking bans are needed to protect nonsmokers from diseases linked with smoking." (Appendix D).

Summary: Thus, the big three tobacco companies state that they all believe that ETS is just an annoyance -- not a serious health threat, despite all those authoritative government reports to the contrary -- and that ventilation which minimizes smoke is the cure, not smoking bans, especially in the hospitality industry. In other words, the tobacco industry is saying that the hospitality industry should make the final decision on ETS controls: using RACT, BACT, or doing nothing. No mention is made of enforcement, or of acceptable levels of exposure or risk.

Discussion: Mainstream medical and scientific opinion has reached a consensus that passive smoking causes lung cancer and heart disease, as well as many other serious health effects. In addition, it is a major annoyance due to eye, nose, and throat irritation. Although every major medical and scientific group in the U.S. is in unanimous agreement that ETS is hazardous, the tobacco industry refuses to accept this consensus. Instead, the industry promotes "accommodation" of smokers, particularly in the hospitality industry. Accommodation involves using ventilation as a control measure, which leaves workers and nonsmoking patrons exposed to ETS. This promotion of ventilation as a "solution" to passive smoking has several flaws. Ventilation is not tied to risk. Instead, the industry confines itself to stating that "exposures are low."   As proof, the industry cites the Oak Ridge Study (Jenkins and Counts, 1999), which it funded under contract (Glantz et al., 1996). However this study is not representative (Hammond, 1999). Public health authorities cannot accept on faith that the risks will be trivial or non-existent, and promote ventilation to provide comfort for building occupants exposed to ETS. However, even if a limited goal of "comfort" is examined, as Spengler (1999) has observed, the goal of ASHRAE Standard 62-1989 -- providing air of quality that satisfies the comfort of 80% of occupants cannot be met at the current specifications of the standard. In fact ASHRAE Standard 61-1999's Addendum 62-e repeals that goal due to the carcinogenicity of ETS.

Indoor air quality standards for ETS have been proposed by Repace and Lowrey (1985b) based on ETS-RSP and Repace and Lowrey (1993) for nicotine and plasma (serum) and urinary cotinine, and extended to saliva cotinine by Repace et al. (1998). These standards are premised on an exposure-response relationship with the numerator based on lung cancer rate differences between two California cohorts of lifelong nonsmokers-- one presumed to be unexposed to ETS (California Seventh Day Adventists) and the other exposed to ETS (Non-SDAs from the general California population). The denominator of the exposure-response relationship underlying the standard was based on assessing the average population exposure to ETS-RSP (Repace and Lowrey, 1985). Later, ETS-RSP was translated into airborne nicotine and body fluid cotinine equivalents using the equations in Table 1. Estimates of average population exposure to ETS-RSP were validated by predicting serum cotinine levels in good agreement with a national probability sample measured in NHANES III. These atmospheric and body fluid cotinine measures were traced back to the primary determinants of ETS exposure: smoker density and air exchange rate; air exchange rates were those based on ASHRAE Standard 62 (Repace et al., 1998). And the risk model was extended to heart disease mortality (Repace et al., 1998). As Figure 2 shows, contrary to the tobacco industry's vague claims about the efficacy of ventilation, risks cannot be controlled to an acceptable level for both workers and regular restaurant patrons using even the best possible displacement ventilation technology.

Even if a way could be found by some as-yet undiscovered ventilation or air cleaning technology to reduce ETS exposures by 4 orders of magnitude, a regulatory bureaucracy would be required to issue permits for the new technology, which would have to be retrofitted into all existing establishments, and designed into all new establishments. Then an enforcement squad would have to be assembled, trained, and fielded to handle complaints. Measuring either ETS concentrations or ventilation rates is difficult, time-consuming, and expensive. Although ETS-RSP can be measured in real-time, RSP is non-specific for ETS. While ETS nicotine is specific, it cannot be measured in real-time. Ventilation rates also cannot be measured in real-time. Since most ventilation engineers are familiar only with dilution technology, they would have to be trained to install the new technology, and building inspectors would have to be retrained to approve those plans. Because there are tens of thousands of establishments in a State the size of California, this would rapidly become an enforcement nightmare. However, smoking bans will achieve zero risk, and currently appear to be easily enforceable.

A final problem concerns new and emerging ETS risks which have not been quantified and for which no dose-response relationships exist. Other studies have linked ETS to mortality from SIDS, and nasal sinus cancer, and possibly cervical cancer and respiratory disease (CalEPA, 1997). New studies have linked ETS to breast cancer, and stroke. The risk of ETS-induced breast cancer appears to be highly non-linear, as shown in Figure 4, suggesting that developing an ETS-IAQ standard for breast cancer would be problematic. Another largely unrecognized issue is that ETS particles are re-emitted again from room surfaces where they have been deposited, indicating that room surfaces act as secondary sources of ETS particles (Johannson et al., 1993). Gases are also likely to be absorbed on and re-emitted from surfaces. This means that buildings where smoking is permitted become highly contaminated with toxic waste from ETS, massive surface sources of PAHs and other carcinogenic and toxic substances to which nonsmokers can be exposed even when there is no smoking taking place. To appreciate the magnitude of the problem, consider a restaurant with an occupancy of 70 persons per 1000 ft^2, with a smoker prevalence of 29%, for an area smoker occupancy of 2 smokers per 1000 ft^2. Each smoker smokes 2 cigarettes per hour. Assuming smoking occurs in the restaurant for 8 hours daily, and that each cigarette liberates 14 mg of tar, 20% of which deposits on room surfaces. Thus (2.8 mg/cigarette) (2 smokers/1000ft^2)(2 cigarettes/smoker-hour)(8 hours/day)(300 days/year) = 27 grams per year of tobacco tar deposited on room surfaces -- including the HVAC system -- per 1000 ft^2 of floor area. For a 10,000 ft2 restaurant, this is 270 g/year or ~ 1 kg of toxic waste every 4 years.

Active & Passive Smoking and Breast Cancer

Pre: 805 cases, 2438 controls; Post: 1512 cases, 2438 controls

(Johnson, et al ., Cancer Causes & Control, in press)
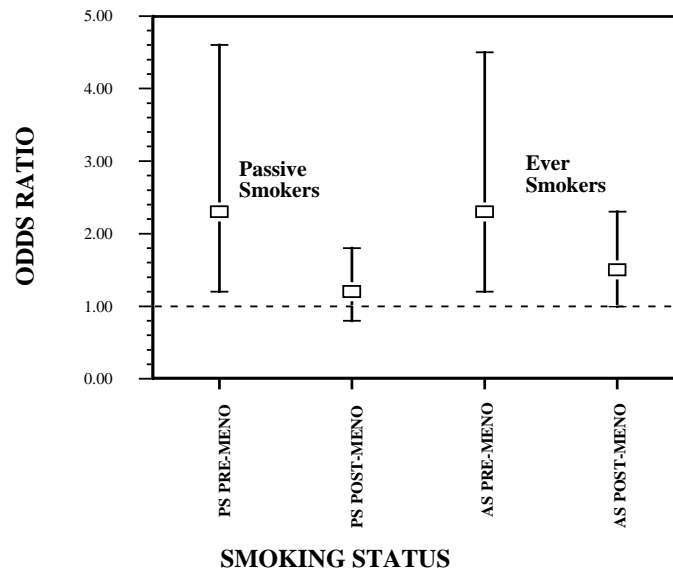
Figure 4. Active and passive smoking and breast cancer in pre-and post-menopausal women [KD Johnson, et al., Health Canada, Cancer Causes and Control, 2000].

Table 7 shows some of these risks (except stroke) as estimated by Wells. Standards would have to be developed for all of them, plus stroke.

Estimated Deaths from ETS

(Wells, AJ., Environment Int. 25:515-519 (1999)

Cause	Deaths in 1998
Lung Cancer	3060.
Heart Disease	47 000.
Breast Cancer	8700.
Cervical Cancer	500.
Nasal Sinus	200.
Brain, Leukemia, & Lymphoma	1000.
Total Deaths in U.S.A.	60 460.

Conclusions

1. The "Proceedings of the Workshop on Ventilation Engineering Controls for Environmental Tobacco Smoke in the Hospitality Industry", sponsored by OSHA and the ACGIH concluded that presently available ventilation technology (well-mixed dilution ventilation) was unsatisfactory for controlling worker exposure to ETS. Air cleaning was similarly viewed as problematic. Of proposed technology, displacement ventilation was viewed as having the potential for 90% reductions in ETS levels, although the lack of performance data, the lack of familiarity of most ventilation engineers with the technology, and the difficulty in retrofitting existing installations poses major problems. Panelists viewed the lack of enforcement of ventilation rates by local building codes and the use of natural ventilation as further problems. Smoking seems to be declining among restaurant patrons.

2. In this report, I model ETS RSP and air nicotine levels for restaurants, bars, smoking lounges, bowling alleys and casinos to estimate hospitality workers' exposure to ETS. ETS RSP has been used as a non-specific tracer for ETS. Air nicotine and body fluid cotinine are the best and most widely used specific tracers for ETS. Using U.S. average smoking prevalence, ASHRAE Standard 62-1999 default occupancy levels, and recommended makeup air supply rates as ideals, shows for this ideal dilution ventilation, estimated ETS RSP levels will be between 100 and 200 $\mu g/m^3$, and air nicotine levels of from 10 to 20 $\mu g/m^3$. These predicted levels appear to be significantly lower than most observations, suggesting lower ventilation rates or higher smoker densities than expected. This is not surprising since neither smoker density nor ventilation rates are regulated.

3. Assuming ideal dilution ventilation, i.e., reasonably achievable control technology (RACT), estimated ETS risk levels for lung cancer and heart disease combined ranged from 15 to 25 per 1000 workers, which is 15 to 25 times OSHA's significant risk level, and 15,000 to 25,000 times the *de minimis* or "acceptable risk" level for federally regulated hazardous pollutants.

4. Assuming ideal displacement ventilation, i.e., best achievable control technology (BACT), estimated ETS risk levels for lung cancer and heart disease combined would be reduced by 90%, ranging from 1.5 to 2.5 per 1000 workers, to 1.5 to 2.5 times OSHA's significant risk level, and 1,500 to 2,500 times the *de minimis* or "acceptable risk" level for federally hazardous pollutants.

5. All cognizant health and scientific authorities in the U.S., including the US Environmental Protection Agency, the National Institute for Occupational Safety and Health, OSHA, the Surgeon General, the National Academy of Sciences, the National Cancer Institute, the National Toxicology Program and

the American Medical Association, have concluded that ETS exposure causes morbidity and mortality. The tobacco industry rejects this consensus.

6. Under Section 112   of the federal Clean Air Act, pollutants may be designated as "hazardous air pollutants" (HAPS) if they can cause serious morbidity or mortality, as ETS does. These ETS-like chemicals are regulated by NESHAPS which are far more stringent than either RACT or BACT. RACT and BACT are designed to control ordinary non-hazardous air pollutants.   NESHAPS regulate HAPS to levels of *de miminis* risk with an adequate margin of safety. ETS contains 5 HAPS pollutants, more than 100 poisonous chemicals, and 47 chemicals classified as hazardous waste under RCRA. Although ETS qualifies, it remains unregulated as a HAP, as a poison, or as hazardous waste.

7. There are currently no official ETS indoor air quality (IAQ) standards in use in the U.S. Proposed NESHAPS-style ETS IAQ standards are based on limiting ETS lung cancer and heart disease risk to *de minimis* levels. Application of these proposed standards to restaurants, bars, and casinos shows that tornado-like levels of ventilation would be required, 4 orders of magnitude (i.e. ten thousand fold) greater than possible by dilution ventilation, and 3 orders of magnitude (i.e., one thousand fold) greater than possible by displacement ventilation, with air cleaning intermediate.

8. Ventilation of buildings is a local government responsibility. Some building codes do not require that ventilation systems be operated after installation. Even under codes that require operation, ventilation standards are not enforced. Enforcement of ventilation standards, although desirable, would require establishment of new regulatory bureaucracies.

9. Enforcement of indoor air quality standards would also require additional new regulatory bureaucracy. Establishment of indoor air quality standards requires a high level of technical expertise, well beyond the capacity of most local government, and would be a years-long process (not including the resultant litigation, based on federal experience. It is doubtful that most jurisdictions would be willing or able to pay for these new regulatory regimes. Even if all the regulatory hurdles involving the setting of IAQ standards for ETS could be surmounted for lung cancer and heart disease, setting standards to protect against risks of ETS-induced breast cancer, stroke, SIDS, nasal sinus cancer, respiratory diseases, etc. would remain.

10. The tobacco industry's open and stated goal, currently available on their websites, is to actively promote ventilation technology as an optional control measure for ETS, at the option of hospitality business owners. The tobacco industry has made the hospitality industry a special target for ventilation technology. None of the "big three" tobacco companies concedes that ETS poses health risks to nonsmokers, and all promote "accommodation," a

vaguely-defined code-word for letting the marketplace decide how to control ETS.

11. It is clear that smoking bans, such as in effect in the State of California represent the most cost-effective, easiest-to-enforce, and lowest risk alternative to ETS control. They appear profitable for business, and are also the only control measure known which is capable of yielding *de miminis* risk.

Appendix A. 103 Poisonous Substances in Tobacco Smoke

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July 19, 2000

This review is based upon the following definition:

poison. def.: a substance (as a drug) that in suitable quantities has properties harmful or fatal to an organism when it is brought into contact with or absorbed by the organism: a substance that through its chemical action usu. kills, injures or impairs an organism <strychnine, carbon monoxide, and other ~s>

Websters Third New International Dictionary, Unabridged. Merriam Webster, Springfield, MA, 1986.

REFERENCE SOURCES for table below

1. NIOSH Pocket Guide to Chemical Hazards, U.S. Dept. Health & Human Services, Centers for Disease Control & Prevention, June 1994.

2. Dangerous Properties of Industrial Materials, NI Sax, 6th Ed., Van Nostrand Reinhold, NY, 1984.

3. The Merck Index - An encyclopedia of chemicals drugs and biologicals. 11th Ed. S. Budavari, MJ O'Neill, A. Smith PE Heckelman Eds. Merck & Co., Rahway, NJ 1989.

4. Reducing the Health Consequences of Smoking, 25 Years of Progress. A Report of the Surgeon General, 1989. USDHHS, Rockville, MD. 1989.

5**.** Smoking and Health, A Report of the Surgeon General, 1979. USDHEW, Washington, DC.

6. Wynder E & Hoffman D, Tobacco and Tobacco Smoke, Academic Press, New York, 1967.

N.B.: The following substances are listed as in tobacco smoke. Although few of them have been actually measured in secondhand smoke, all of them have been measured in mainstream and to a lesser extent, sidestream smoke. Secondhand smoke consists of fresh and aged exhaled mainstream and sidestream smoke, and mainstream smoke is formed in the same burning cone as sidestream. Generally, sidestream and secondhand smoke contain greater total quantities of given chemicals (e.g., more NO_2 and more NNK), and are more toxic than mainstream smoke, which is formed at a higher temperature, and is also filtered by the tobacco rod and the cigarette filter.

Compound(s) Listed in Tables 5,6,7,8 or 9 in Ref. 4 or in Ref.5, Chapter 14.

Poison (Y=yes) Superscripts refer to references sources above

	Compound	Poison
1.	1,1-Dimethylhydrazine†	Y[4]
2.	1-Methylindole	Y[5]
3.	2-Naphthylamine	Y[4]
4.	2-Nitropropane	Y[4]
5.	2-Toluidine	Y[4]
6.	3-Vinylpyridine	Y[4]
7.	4,4-dichlorostilbene	Y[5]
8.	4-(Methylnitrosamino)-1-(3-pyridil)-1-butanone (**NNK**)	Y[4]
9.	4-Aminobiphenyl	Y[4]
10.	5-Methylchrysene	Y[4]
11.	7H-Dibenzo(c,g)carbazole	Y[4]
12.	9-Methylcarbazole	Y[5]
13.	Acetaldehyde	Y[4]
14.	Acetone	Y[4]
15.	Acetonitrile	Y[1]
16.	Acrolein	Y[4]
17.	Acrylonitrile	Y[4]
18.	Alkylcatechols	Y[5]
19.	Ammonia	Y[1]
20.	Anabasine	Y[3]
21.	Aniline	Y[1]
22.	Anthracenes (5)	Y[2]
23.	Antimony	Y[2,5]
24.	Arsenic	Y[4]
25.	Benz(a)anthracene	Y[4]
26.	Benzene	Y[4]
27.	Benzo(a)pyrene	Y[4]

28.	Benzo(b)fluoranthene	Y^4
29.	Benzo(j)fluoranthene	Y^4
30.	Benzo(k)fluoranthene	Y^4
31.	Benzofurans (4)	Y^2
32.	Butadiene	Y^1
33.	Butyrolactone	Y^6
34.	Cadmium	Y^4
35.	Carbon monoxide	Y^4
36.	Carbonyl sulfide	Y^4
37.	Catechol	Y^4
38.	Chromium	Y^4
39.	Chrysene	Y^4
40.	Cresols (all 3 isomers)	Y^5
41.	Crotonaldehyde	Y^4
42.	DDD	$Y^{5,2}$
43.	DDT	$Y^{5,2}$
44.	Dibenz(a,h)acridine	Y^4
45.	Dibenz(a,h)anthrancene	Y^4
46.	Dibenz(a,j)acridine	Y^4
47.	Dibenzo(a,i)pyrene	Y^4
48.	Dibenzo(a,l)pyrene	Y^4
49.	Dimethylamine	$Y^{2,6}$
50.	Endosulfan	Y^5
51.	Endrin	$Y^{5,2}$
52.	Ethylcarbamate	Y^4
53.	Fluoranthenes (5)	Y^2
54.	Fluorenes (7)	Y^2
55.	Formaldehyde	Y^1
56.	Formic acid	Y^1
57.	Furan	Y^2
58.	Hydrazine	Y^4
59.	Hydrogen cyanide	Y^4
60.	Hydrogen sulfide	Y^1
61.	Hydroquinone	$Y^{5,2}$
62.	Indeno(1,2,3-c,d)pyrene	Y^4
63.	Indole	Y^2
64.	Isoprene	Y^2
65.	Lead	Y^4
66.	Lead 210	Y^5

67.	Limonene	Y^2
68.	Manganese	$Y^{5,2}$
69.	Mercury	$Y^{5,2}$
70.	Methanol	Y^1
71.	Methyl formate	Y^1
72.	Methylamine	Y^1
73.	N'-Nitrosoanabasine	Y^4
74.	N'-Nitrosonornicotine	Y^4
75.	N-Nitrosodiethanolamine	Y^4
76.	N-Nitrosodiethylamine	Y^4
77.	N-Nitrosodimethylamine	Y^4
78.	N-Nitrosoethylmethylamine	Y^4
79.	N-Nitrosomorpholine†	Y^4
80.	N-Nitrosopyrrolidine	Y^4
81.	Naphthalene	Y^1
82.	Nickel	Y^4
83.	Nicotine	Y^4
84.	Nitric oxide	Y^4
85.	Nitrogen dioxide (NO_2)	Y^4
86.	NNN	Y^4
87.	Nornicotine	Y^3
88.	o-Toluidine	Y^4
89.	Palmitic acid	Y^2
90.	Parathion	Y^5
91.	Phenol	Y^2
92.	Phenols (volatile)	Y^4
93.	Picolines (3)	Y^3
94.	Polonium-210	Y^4
95.	Propionic acid	Y^1
96.	Pyrenes (6)	Y^2
97.	Pyridine	Y^1
98.	Quinolines (7)	Y^2
99.	Styrene	Y^1
100.	Toluene	Y^1
101.	Toluidine(s)	Y^2
102.	Urethane	$Y^{5,2}$
103.	Vinyl chloride	Y^4

Appendix B.
Equivalency of the Repace (1987) and Ott (1999) models of ETS-RSP

Note on the equivalence of the Repace (1987) (Eq. 1) and Ott(1999) models for ETS-RSP: Ott (1999) gives the following values: For a $V = 500$ m^3 bar with an effective air exchange rate $\phi_p = 6$ hr^{-1} (equivalent to a ventilatory air exchange rate of $\phi_v = \phi_p/1.2 = 5$ hr^{-1}), and an average smoking count $n_{ave} = 2$ cigarettes, the predicted ETS-RSP level is 57 μg/m^3. (The effective air exchange rate for particles was measured to be 1.2 times the air exchange rate due to ventilation alone). Ott's n_{ave} is the same as the number of active smokers n_{as} under the Repace Habitual Smoker model (Repace, 1987), where the number of habitual smokers n_{hs} = 3 n_{as} under the Repace model. Thus n_{hs} = (3 habitual smokers per burning cigarette)(2 burning cigarettes) = 6 habitual smokers, where an habitual smoker is assumed to smoke at a rate of 2 cigarettes per hour. $D_{hs} = 100$ $n_{hs}/V = \{(100)(6$ hs)\} / \{(500$ m$^3)\} = 1.2$ habitual smokers per hundred cubic meters (hs/hcm). Eq. 1 predicts: ETS-RSP = 220 D_{hs}/ϕ_v = (220)(1.2 hs/hcm) / (5 hr^{-1}) = 53 μg/m^3. The slight differences in predictions of the two models are probably due to round-off error. Thus the two models are equivalent. The particle size incorporated into both the Repace and Ott models is PM$_{3.5}$, which is essentially the same as PM$_{2.5}$ (Wallace, 1996). Thus, the Repace (1987) model is understood to be useful under the following conditions: it predicts the time-averaged ETS-RSP (PM$_{3.5}$) concentration assuming that the smokers in the space each smoke identical cigarettes of emissions 14 mg/cigarette at the identical rate of 2 cigarettes per smoker-hour. The model incorporates the ventilatory air exchange rate (essentially that specified by ASHRAE Standard 62), assuming that the effective air exchange rate for ETS particles is 20% higher. Both models are also useful in estimating air exchange rates if the other model parameters are given.

Appendix C
47 Chemicals in ETS are classified as "hazardous waste"
under RCRA (Resource Conservation and Recovery Act)
RCRA Landfill Disposal Regulations from the Code of Federal
Regulations (CFR 40: 268) on the disposal of Hazardous Wastes in
Landfills.

I have identified 47 chemicals in cigarette smoke subject to restrictions by EPA on
land disposal (i.e., being dumped in a landfill), as listed in 40 CFR. In Part 268,
Land Disposal Restrictions (a) the hazardous wastes which are restricted from land
disposal are identified and limited circumstances are defined which permit an
otherwise prohibited waste to be disposed are given. In Subpart A of 40 CFR
section 268.2 (b): "Hazardous constituent or constituents means those constituents
listed in **Appendix VIII to part 261** of this chapter. Below in Table C-1 is a list
of 32 carcinogens in cigarette smoke and also in Appendix VIII to part 261.

Table C-1. Chemical compounds identified in tobacco smoke for which there is "sufficient evidence" of carcinogenicity in humans or animals according to the International Agency for Research on Cancer (1986), and which appear in Appendix VIII, part 261.

acrylonitrile	**dibenzo(a,e)pyrene**	**vinyl chloride**
arsenic	**dibenzo(a,l)pyrene**	**1,1-dimethylhydrazine**
benz(a)anthracene	**dibenzo(a,h)pyrene**	**2-nitropropane**
benzene	**formaldehyde**	**2-napthylamine**
benzo(a)pyrene	**hydrazine**	**4-aminobiphenyl**
benzo(b)fluoranthene	**lead**	**7H-dibenzo(c,g)carbazole**
benzo(k)fluoranthene	**nickel**	
cadmium	**N-nitrosodiethanolamine**	
chromium VI	**N-nitrosodiethylamine**	
DDT	**N'-nitrosodimethylamine**	
dibenz(a,h)acridine	**N'nitrosonornicotine**	
dibenz(a,j)acridine	**N-nitrosopiperidine**	
dibenz(a,h)anthracene	*ortho*-**toluidine**	

In addition, the following 15 compounds listed in Table C-2 are in cigarette smoke (1979 Surgeon General's Report, Ch. 14), and are **also** listed in Appendix VIII to part 261:

Table C-2.

acrolein
chrysene
cresol
cyanogen
DDD
endosulfan
endrin
hydrogen cyanide
maleic hydrazide
mercury
nicotine
parathion
phenol
pyridine
resorcinol

References

Benowitz N. Biomarkers of Environmental tobacco smoke exposure. Env. Health Persp. 107, Suppl 2: 349-356 (1999).

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

Bonita R, et al. Active and passive smoking and risk of stroke. Tobacco Control 8: 156-160 (1999).

Clean Air Act as Amended August 1977, 95th Congress, Washington DC 1977.

Daisey, JM. Tracers for assessing exposure to ETS: What are they tracing? Env Health Persp 107 suppl 2, 1999, 319-327.

Dockery DW, Pope III CA. Acute respiratory effects of particulate air pollution. *Annual Rev of Publ Health* 15:107-132 (1994).

Gallup Poll, volume 56, # 36, Monday, May 13, 1996. Gallup Canada, Inc. (Percent of respondents reported smoking in the past week).

Glantz SA, Slade J, Bero LA, et al., The Cigarette Papers. University of California Press, Berkeley, 1996.

Glantz, SA. Smoke-Free ordinances do not affect restaurant business. Period. J Publ Health Management Practice 5:vi-ix (1999).

Hammond SK. Exposure of US workers to environmental tobacco smoke. Env Health Persp 107 suppl 2, 1999, 329-340.

Jenkins RA, Counts RW. Occupational exposure to environmental tobacco smoke: results of two personal exposure studies. Env Health Persp 107 suppl 2, 1999, 341-348.

Johannson J, Olander L, and Johannsson R. Long term test of the effect of room air cleaners on tobacco smoke. Proc. Indoor Air '93, 6:387-391 (1993).

Johnson KC, Jinfu H, Yang M, et al. Passive and active smoking and breast cancer risk in Canada, 1994-1997. Cancer Causes & Control 11:211-221 (2000).

Johnson KD, Repace JL. Turning over the wrong stone. (letter) British Med J.,

in press.

Kawachi I, et al. "Deaths from lung cancer and ischemic heart disease due to passive smoking in New Zealand." The New Zealand Medical Journal 102: 337-340 (1989).

Lockhart Risk Management Ltd. Vancouver, BC; IAQ Survey Restaurants, Pubs and Casinos done for Heart & Stroke Foundation of BC and Yukon, Oct. 1995, File 477-11.

Nagda et al. Airliner Cabin Environment: Contaminant Measurements, Health Risks, and Mitigation Options. . U.S. Dept. of Transportation Report DOT-P-15-89-5, USDOT, Washington, DC, (1989).

National Cancer Institute. Respiratory health effects of passive smoking: lung cancer and other disorders; The report of the U.S. Environmental Protection Agency. National Cancer Institute Monograph 4, NIH Publication # 93-3605, National Institutes of Health, Bethesda, MD., August 1993.

National Research Council. Environmental tobacco smoke - Measuring exposures and assessing health effects. National Academy Press, Washington, DC, 1986.

National Toxicology Program. 9th Report on Carcinogens 2000. U.S. Dept. of Health & Human Services, National Institute of Evironmental Health Sciences, Research Triangle Park, NC.

Ott WR. Mathematical models for predicting indoor air quality from smoking activity. Env Health Persp 107, suppl 2, 375-381 (1999).

Repace JL, Consistency of Research Data on Passive Smoking and Lung Cancer THE LANCET (ii): 3 March 1984, p. 506.

Repace JL, Lowrey AH. Indoor Air Pollution, Tobacco Smoke, and Public Health, SCIENCE 208: 464 (1980).

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 1998; 18: 71-83.

Repace JL. Indoor concentrations of environmental tobacco smoke: models

dealing with effects of ventilation and room size. Ch. 3, in: *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, Lowrey AH. A Quantitative Estimate of Nonsmokers' Lung Cancer From Passive Smoking", ENVIRONMENT INTERNATIONAL 11: 3-22 (1985).

Repace JL, Lowrey AH. Risk Assessment Methodologies in passive smoking-ind lung cancer. RISK ANALYSIS, 10: 27-37, (1990).

Repace, J.L. Consistency of research data on passive smoking and lung cancer. *The Lancet* 1: 506 (1984).

Repace, JL. Workshop Comments on the dramatic changes in recommended outdoor air rates - what are the rational motives and consequences.. In: *Healthy Buildings '88, vol 4. Conclusions and recommendations for healthier buildings.* B. Berglund, T. Lindvall, Eds. Swedish Council for Building Research, Stockholm, Sweden, June 1991, p. 132-133.

Repace JL, Ott WR, Klepeis NE. Indoor Air Pollution from Cigar Smoke. In: *Smo and Tobacco Control Monograph 9. Cigars - Health Effects and Trends.* National Institutes of Health, National Cancer Institute, Bethesda, MD (1998).

Reynolds P, Fontham ETH, Correa P, Wu A, Buffler PA, and Greenberg RS. Occupational Exposure to Environmental Tobacco Smoke. JAMA 275: 441-442 (1996).

Samet JM, Wang SS. Environmental tobacco smoke. Ch. 10, in: Environmental Toxicants, Human Exposures and their Health Effects. M. Lippmann, Ed. John Wiley & Sons, New York, 2000.

Tancrede M, Wilson R, Zeise L, and Crouch EAC. The carcinogenic risk of some organic vapors indoors: a theoretical survey. Atmospheric Environment 21: 2187-2205 (1987).

Turner, DB. Workbook of atmospheric diffusion estimates. U.S. EPA, 1970.

U.S. Environmental Protection Agency. Glossary for air pollution control of

industrial coating operations, 2nd Ed. EPA-450/3-83-013R, December 1983.

Wadden RA, Scheff PA. Indoor Air Pollution Characterization, prediction, and control. Wiley Interscience, New York, 1983.

Wallace LA. Indoor particles: a review. *J Air Waste Manage Assoc* 46:98-126 (1996).

Weiss, ST. "Passive smoking and lung cancer - What is the risk?" American Review of Respiratory Disease 133: 1-3 (1986).

Wigle DT, Collishaw NE, Kirkbride J, Mao Y. Deaths in Canada from Lung Cancer due to Involuntary Smoking. CMAJ 136: 945-951 (1987).

Appendix D.
Documents on secondhand smoke, accomodation, ventilation, and smoking bans downloaded from tobacco industry websites.

1. Philip Morris <philipmorris.com>
2. British American Tobacco <bat.com>
3. RJ Reynolds <rjr.com>

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