ENVIRONMENTAL TOBACCO SMOKE:
1998 - 2005 UPDATE
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INTRODUCTION

During the past seven years, an overwhelmingly large volume of new studies has emerged, expanding the already existing solid knowledge base on environmental tobacco smoke, ETS. These studies - only a limited selection of which can be addressed in this Update - not only confirm and strengthen previous findings but also add significant new data that make the need for strict public health measures for the protection from exposure to ETS of nonsmokers, especially children and workers in the hospitality industry, ever more urgent.

Anne Morrow Donley, to whom I owe the inspiration to undertake this survey, has incorporated the new 1998-2005 information which follows below into the original ETS FACT SHEET of 1998 to provide a comprehensive resource on ETS readily accessible at her webpage http://www.gasp.org/ETS2005.html.

Credit is also due to Julie Andersen and J.R. (Pinky) Few of the Tobacco-Free Marion County Coalition in Arkansas who took it upon themselves to convert this Update into a PDF Version, ETS 1998 - 2005 Update.pdf. A .pdf document can be opened by different types of computers without loss of its formatting.

ENVIRONMENTAL TOBACCO SMOKE IS A CAUSE OF CANCER

The list of agencies and organizations that classify ETS as a human carcinogen has grown in recent years and comprise now:

- Surgeon General's Report on the Health Consequences of Involuntary Smoking, USDHHS 1986.

- National Research Council of the National Academy of Sciences, Report on Environmental Tobacco Smoke, 1986.

- U.S. EPA, 1992, endorsed and reprinted by the National Cancer Institute, USDHHS, as Smoking and Tobacco Control Monograph 4, 1993.

- California EPA, 1997, endorsed and reprinted by the National Cancer Institute, USDHHS, as Smoking and Tobacco Control Monograph 10, 1999.

- National Toxicology Program at the National Institute of Environmental Sciences, USDHHS 2000, 2002, 2004.

- World Health Organization, International Agency for Research on Cancer, IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans, 2002, representing the first worldwide recognition of ETS as a human carcinogen.

- California EPA Update, 2004 Draft Report, the most comprehensive and the most rigorous coverage of the damaging effects of ETS to date. It can be accessed at : http://www.arb.ca.gov/toxics/ets/dreport/dreport.htm (1).

The TABLE below compares the findings for various cancers of Cal EPA 1997 with those of Cal EPA 2004.

EVIDENCE FOR CAUSAL ASSOCIATIONS BETWEEN ETS AND CANCER				
Outcome	# of studies 1997	# of Additional Studies in Update	Findings Cal EPA 1997	Findings Cal EPA 2004 Update
All Cancers Adult	5	0	Suggestive	Suggestive
Childhood (Mother smoker)	7	5	Inconclusive	Inconclusive
Childhood (Father smoker)	1	5	Inconclusive	Suggestive
Lung	19	17	Conclusive	Conclusive (strengthened)
Breast	4	13	Inconclusive	Conclusive
Nasal sinus	3	0	Conclusive	Conclusive
Nasopharyngeal	0	4	(no studies)	Suggestive
Cervical	4	0	Suggestive	Suggestive
Bladder	2	0	Inconclusive	Inconclusive
Stomach	1	2	Inconclusive	Inconclusive
Brain Adult	3	0	Inconclusive	Inconclusive
Brain Children	10	12	Inconclusive	Suggestive*
Leukemia Childhood	8	6	Inconclusive	Inconclusive
Lymphomas	6	4	Inconclusive	Suggestive (in children)

* May reflect an association with paternal pre-conception smoking rather than ETS exposure

In updating and summarizing the carcinogenic effects of ETS exposure (see Table), the 2004 Cal EPA Report strengthens the association with lung cancer and projects an annual mortality in the U.S. of 3,400 lung cancer deaths. But lung cancer is no longer the only type of cancer that incriminates ETS as a cause.

The 2004 Report presents the first conclusive evidence that ETS exposure is also causally related to BREAST CANCER with an overall increase in risk of approximately 30% (the breast cancer risk increase for smokers can be several hundred percent). This finding will be alarming news for many. Earlier studies on breast cancer had been inconclusive because of the complexity of variables that needed to be controlled before statistical significance could be achieved. These variables are menopausal status, age of exposure, and probably the same factors that are responsible for breast cancer in active smokers, such as genetic susceptibility and the hormone receptor status of the tumor.

The new Report also provides suggestive evidence that ETS is a cause of cervical cancer, nasopharyngeal cancer, and of brain cancer and lymphomas in children.

How reliable are EPA's risk estimates? A recent study from the Johns Hopkins Bloomberg School of Public Health states that the current EPA Assessment System for Population Exposure Nationwide (ASPEN), which is being used nationally to assess the public health impact of ambient air toxins, UNDERESTIMATES their cancer risk by a factor of as high as three, when compared to directly measuring indoor, outdoor and personal exposures (2).

LUNG CANCER

ETS and lung cancer continue to take center stage probably because the 1992 U.S. EPA's classification of ETS as a Group A Human Carcinogen was challenged in a lawsuit by the tobacco industry and subsequently struck down by Federal Middle District Court Judge William Osteen's 1998 ruling in favor

of the industry. As a private attorney in 1974, William Osteen worked for tobacco growers as a lobbyist (3). The EPA appealed Judge Osteen's ruling, and not only did the Fourth Circuit Court of Appeals dismiss Judge Osteen's ruling, but it threw out the case against the EPA altogether. The tobacco companies had the option to appeal to the U.S. Supreme Court but failed to do so. Obviously they knew they would lose, because too many credible scientific studies have since reaffirmed that ETS causes lung cancer. Regrettably, the general public still appears to be largely ignorant about the details of this story and often expresses doubt about the now scientifically documented fact that ETS is a cause of lung cancer.

Among the new studies on ETS and lung cancer is one large multinational study, conducted between 1985 and 1994 by Brennan and 17 co-authors from prestigious institutions, comprising 1,263 lung cancer patients who never smoked and 2,740 control subjects from metropolitan areas in Germany, Italy, Sweden, France, Spain, Portugal, United Kingdom, and USA (4). The study found several statistically significant cause-effect associations and clear dose-response relationships consistent with a causal association between exposure to ETS from spousal, workplace, and social sources, and the development of lung cancer among never-smokers. Sensitivity analysis for the effects of misclassification (both positive and negative) indicated that the observed risks are likely to - again - UNDERESTIMATE the true risk.

The epidemiological data are supported by experimental findings. Apart from the nicotine metabolite, cotinine, the most consistently elevated biomarkers in people exposed to ETS are the tobacco-specific nitrosamine NNK and its metabolic products (5). NNK is a potent lung carcinogen which can be formed in the body from nicotine (6). NNK and its metabolites are present in the first urine of infants born to smoking mothers!! (7). NNK has also been identified in elementary school children (8). Again, widespread ignorance prevails as to the fact that tobacco-specific nitrosamines, such as NNK, originate from nicotine which, in public comments, is often deemed relatively benign except for its potent addictiveness.

A statement by Farone reveals tobacco industry's early knowledge about NNK:

> "In the 1970s research paid for by the Cigarette Manufacturing Industry uncovered the fact that tobacco specific nitrosamines were the most significant risk in lung cancer both among smokers and in non-smokers subjected to sidestream smoke. [This information] was withheld, not only from the public, but also from other researchers within the industry and even within the company that obtained the results." (9).

ENVIRONMENTAL TOBACCO SMOKE AND THE CARDIOVASCULAR SYSTEM

Based on National Vital Statistics of 2000, the Cal EPA 2004 Report finds a causal association between ETS exposure from spousal smoking and death from coronary heart disease (CHD) in nonsmokers. The causal association found in the 2004 Report is stronger than the one arrived at in the1997 Cal EPA Report.

Compared to nonsmokers not exposed to ETS, there is a 30% increase in risk (relative risk of 1.3). The latest estimate of the annual death toll from ETS-related CHD in the U.S. ranges from 22,700 to 69,600 (10). Underlying conditions such as diabetes, hypertension, or vascular disease worsen the risk.

ETS may also contribute to stroke due to atherosclerosis of the carotid artery and the large arteries of the brain as well as the degeneration of intracerebral arteries. An extensive cohort study in China in over

60,000 women found a statistically significant relationship between ETS exposure by husbands' smoking and the occurrence of stroke (11).

The mechanisms by which ETS affects the heart and blood vessels comprise ❶arterial wall thickening, ❷lesion formation, ❸decrease in aortic distensibility and reactivity, and ❹endothelial dysfunction, which has emerged as a major factor in CHD.

ETS- induced endothelial dysfunction, i.e., a functional impairment of the linings of blood vessels, may finally explain why ETS can cause much greater damage to the cardiovascular system than one would have expected simply by comparing the quantities of cigarette smoke to which active and passive smokers are exposed. A recent landmark experiment in healthy young nonsmokers revealed that a mere thirty minute exposure to ETS causes changes in coronary blood flow, specifically a substantial reduction in the coronary flow velocity reserve, that are indistinguishable from those of habitual smokers (12). These changes are caused by nicotine which inhibits the self-regulatory widening of the coronary blood vessels (vasodilatation) in response to nitric oxide, an endogenous agent released by endothelial cells. ▶▶*Since this effect of nicotine reaches its maximum already in the small amounts present in ETS, the difference between passive and active smoking as to their effects on blood vessels is greatly narrowed. By the same token, smoking of only 1 to 3 cigarettes per day can cause heart disease by this mechanism*◀◀ (13).

Other factors contributing to heart disease and stroke are the adverse effect of ETS on blood lipids: increase in "bad" cholesterol, LDL, decrease in "good" cholesterol, HDL, as well as platelet activation and elevated fibrinogen levels, all of which are associated with endothelial damage and plaque formation leading to atherosclerosis that, in turn, predisposes to coronary heart disease and stroke {for more detailed information and references to pertinent publications consult the Cal EPA 2004 Report (1)}.

Alarmed by these new findings, the AMERICAN HEART ASSOCIATION issued in 2002 tough NEW HEALTH GUIDELINES for preventing heart disease and stroke, urging "complete cessation" for smokers and "no exposure to environmental tobacco smoke" for everyone. According to an AHA spokesperson, ▶▶*"The evidence on the dangers of secondhand smoke has been accumulating to the point where we should be saying that all exposure should be avoided"* ◀◀ (14).

The need for protection from ETS exposure was further corroborated by a 2004 report of the American Family Physician (http://www.aafp.org/afp/20041115/tips/14.html), that regular exposure to ETS significantly increased white blood cell counts, C-reactive protein, homocysteine, fibrinogen, and total and LDL cholesterol, all of which have been identified as "inflammation markers" instrumental in the development of atherosclerotic disease. Since several of these markers are often included in the battery of laboratory tests ordered for routine physical check-ups, physicians and patients must be made aware of the potential impact that ETS exposure - which is still quite pervasive - can have on these lab values and cardiovascular morbidity.

ENVIRONMENTAL TOBACCO SMOKE AND THE RESPIRATORY SYSTEM

Again, the Cal EPA 2004 Report (1) covered numerous new studies, corroborating and expanding the findings of earlier reports.

Children suffer the gravest consequences of ETS exposure, manifesting in 💣impaired lung development, 💣decreased lung function, 💣sudden infant death syndrome (SIDS), 💣acute and chronic

respiratory illnesses (including otitis media), and foremost the induction and exacerbation of asthma. Their causal associations have been found to be conclusive. In adults too, asthma induction as well as exacerbation of existing asthma are causally related with ETS exposure.

Normally, people are not sufficiently aware of their breathing, a largely automatic process, to pay attention to what they breathe or how much they breathe. For an adult male, the average daily intake of food is 1.5 kg, that of water 2.5 kg, but the amount of air exchanged by the lungs is as high as 15.0 kg.

Neither are people cognizant of the fact that the surface area of the lungs, consisting of millions of tiny air sacs (alveoli) open to the environment, is as large as a tennis court. This large surface area, constituting the interface between the inhaled air and a rich network of fine blood vessels surrounding the alveoli, guarantees an adequate uptake of oxygen to supply every cell in the body as well as the necessary removal of the toxic combustion product, carbon dioxide.

It may, therefore, be quite sobering to realize that, according to an estimate from 1981, nationally each year an estimated 2.25 million metric tons of gaseous and inhalable particulate matter of ETS are discharged into our personal air space (15).

Emission estimates from 2002 for three major components of ETS in tons/year in the U.S. are (1):

Nicotine	647
Carbon Monoxide	30,200
Respirable Suspended Particulates (RSP's)	5,860

- Nicotine - a potent poison in its own right - is the precursor of the lung carcinogen NNK;
- Carbon monoxide, known for its high toxicity, disables the oxygen-carrying capacity of the hemoglobin in red blood cells;
- RSPs, constituting the visible smoke, which is only between 5 and 10%(!) of the total effluent from a burning cigarette, carry the bulk of the approximately 60 carcinogens.

If a single one of these carcinogens occurred in a manufacturing process unrelated to tobacco, the Occupational Safety and Health Administration (OSHA) would require the worker to wear protective clothing and a face mask. But no federal law specifically protects nonsmokers (or smokers) from the carcinogenic burden of ETS - let alone smokers from inhaling mainstream smoke....

Particulate matter in outside air is regulated under the U.S. EPA's Clean Air Act. The National Ambient Air Quality Annual Standard has been 50 micrograms (ug) per cubic meter (m3) for particles 10 micron in diameter (PM10), 1/7 the width of a human hair. Particle concentrations from 150 to 350 ug/m3 are designated as "unhealthful" and those over 420 as "hazardous." Because the above standard proved inadequate, EPA recently issued a new ambient standard of 15 ug/m3 for fine particulates of less than 2.5 micron in diameter (PM2.5) which penetrate deep into the lungs (16). Tobacco smoke particles being less than one micron in diameter fall in this category and can cause lung cancer and cardiopulmonary mortality (17). But again, the federal law stops at the entrances to indoor airspaces, leaving it to local authorities to protect the public who spend 90% of their time indoors.

If we were to apply the enforceable outdoor standard to indoor air, it would be violated wherever smoking is allowed. Especially bars with particulate concentrations that can be as high as 1,300 ug/m3 (18), thus exceeding the original standard by 26 times and the revised standard by almost 90 times, rank high up in the "hazardous" category (19). Prior to the N.Y. City smoking ban, air quality tests in several Manhattan smoking bars revealed that the air was 50 times worse there than at the entrance to the

Holland Tunnel. In a home environment where only one cigarette is being smoked RSPs are about 300 ug/m3 (1).

Contrary to the public perception that outside air pollution is worse than indoor tobacco smoke, fine particulate matter pollution from cigarettes is ten times higher than that from diesel car exhaust (20). Particulate matter from smoking three cigarettes equals the exhaust emitted by a diesel car running for 30 minutes in a garage (20).

New research in Sweden has discovered that smoke-polluted air contains more than a hundred times the concentrations of endotoxins that are present in average smokefree indoor air (21). These endotoxins which arise from bacteria can induce serious inflammatory reactions and lead to bronchitis and asthma.

PRE- AND POSTNATAL EXPOSURE OF CHILDREN TO ETS

Child abuse can start even before the child is born. Smoking and ETS-exposed mothers harm the fetus. ETS exposure in pregnancy is responsible for a reduction in birth weight which is associated with respiratory problems and perinatal mortality (1). Nicotine, which impacts the brain during critical stages of its intrauterine development in experimental animals, is a likely cause for the deficits in learning and memory, and the emotional and behavioral problems seen in childhood and later in life (22). Prenatal nicotine also primes the adolescent brain for addiction. Carcinogens reaching the fetus via the placental circulation can cause cancer later in life. Experimental findings in mice suggest that prenatal ETS exposure may also promote development of adult cardiovascular disease (23).

Children suffer great physical and psychological distress from ETS exposure, asthma being probably the most dreaded immediate consequence. A new study comparing various potential causes of asthma in children, aged 4 to 6 years in 235 schools in the UK identified ETS as the prominent risk factor (24).

In a study of 4,399 children aged 6 to 16, even the lowest exposure, as monitored by the levels of cotinine, a metabolite of nicotine, in blood, urine, saliva and hair, was found to significantly impair, in a dose-related manner, the children's reading, math and reasoning scores (25). Forty percent of U.S. children are exposed to ETS in their homes and more than 21.9 million children are at risk for ETS-related reading deficits. No safe level of exposure to ETS could be found.

The 2004 Report of the National Toxicology Program, NTP, states "Despite the fact that exposure is known to cause cancer and cancer deaths in nonsmokers, approximately 43% of U.S. children aged 2 months to 11 years lived in a home with at least one smoker. In addition, 37% of non-smoking adults reported exposure to environmental tobacco smoke at home or at work" and "...it is estimated that more than half of U.S. youth are still exposed to environmental tobacco smoke and approximately 9 to 12 million children, aged six and younger, are exposed to environmental tobacco smoke in their homes" (26).

The seriousness of children's exposure to ETS is further corroborated and re-emphasized by a seven year study in more than 100,000 ETS-exposed nonsmokers (27), prompting BBC to air a Cancer Alert For Smoking Parents (28).

Apart from the physical damage caused by ETS, smoking in a child's daily environment often induces children to try out cigarettes and become smokers themselves.

OTHER ADVERSE HEALTH EFFECTS OF ENVIRONMENTAL TOBACCO SMOKE

Essentially the same diseases and injuries inflicted by smoking upon the organism - and the fetus in pregnancy - can manifest in nonsmokers breathing the smoke escaping from lit cigarettes, cigars and pipes. Their prevalence and intensity may be much less, depending on the amount of exposure and individual susceptibility, except for coronary heart disease whose occurrence in ETS-exposed nonsmokers is disproportionally higher than to be expected from a quantitative comparison of exposure to active and passive smoke (12).

In a major study in over 4,800 never-smokers from Hong Kong, significant dose dependent associations were reported between passive smoking and mortality from lung cancer, chronic obstructive pulmonary disease, stroke, ischemic heart disease, and from all cancers, all respiratory and circulatory diseases, and all causes, with no differences between males and females (29).

More information on ETS-related health effects can be found in the comprehensive Cal EPA 2004 Report (1).

A review of 106 articles asking whether passive smoking causes harm, found that 63% concluded it was harmful and 37% that it was harmless. The only factor that could be correlated with the conclusion was whether or not the author was affiliated with the tobacco industry (30).

SMOKEFREE LEGISLATION

The adverse health effects of ETS have reached such a critical mass that continued exposure of children and adults would violate the basic rights of citizens to health and happiness as decreed by the Constitution. Although smokefree public places have been slow in coming, impeded by misinformed business owners and fraudulent tobacco industry propaganda, the process has recently accelerated. The fact that "What is good for health is also good for business" is gradually taking hold in the public mind.

Workplaces across the nation could be made smokefree by a single action, if - in the case of tobacco smoke - OSHA would honor "The General Duty Clause" of the Occupational Safety and Health Act of 1970 which states in Section 5 (a): "Each employer shall furnish to each of his employees employment and a place of employment which are free from recognized hazards that are causing or are likely to cause death or serious physical harm to his employees." But OSHA, apparently under pressure from external, though government condoned, forces, has resisted to protect employees from ETS exposure.

A research team at Health Canada found that working in bars and restaurants can triple lung cancer risk. They also observed a dose-response relation between the degree of exposure and lung cancer risk. They saw this as the most compelling scientific evidence yet for a total ban on workplace smoking, including bars and restaurants (31). However, the heavy hand of the tobacco industry in this country has impeded progress toward this goal.

Cities, counties and states are becoming smokefree only through the laborious effort of individual initiatives. The American Nonsmokers' Rights Foundation published a graph and table of local and state 100% smokefree indoor air laws introduced from 1990 through 2004 (32). As yet, California, Connecticut, Delaware, Maine, New York, Massachusetts, and Rhode Island (by 2006) enacted statewide smoke-free workplace laws that cover restaurants and bars.

Worldwide, entire countries have recently become smokefree: Ireland, Norway, Sweden, New Zealand, Malta, Uganda, Bhutan, India, and Italy have enacted smokefree workplace legislation for all workers, including restaurant and bar workers.

One of the most informative governmental reports on ETS in the workplace, based on worldwide research, came from Ireland in 2002 (33). It concluded that ▶▶ *the weight of evidence for lung cancer, cardiovascular and respiratory disease, and for adverse effects on reproduction calls for legislative measures to protect employees from exposure to ETS at work*◀◀. Ireland was among the first countries to become totally smokefree.

BENEFITS OF SMOKEFREE LEGISLATION

Smokefree laws benefit the public in three ways: they help smokers quit, they reduce the incidence of smoking-related disease, and they substantially reduce the cost of health care and business.

The city of Helena, Montana, offers a prime example. The number of heart attacks in Helena decreased substantially after the city banned indoor smoking, then rose quickly to its former level after the law was struck down in court six months later (34). This event prompted the Centers of Disease Control and Prevention (CDC) for the first time to warn people at risk of heart disease to avoid all buildings and gathering places that allow indoor smoking (35).

In Western New York State, air pollution of RSPs (PM2.5) in a sample consisting of seven bars, six bar/restaurants, five restaurants, two bowling alleys, a pool hall, and a bingo hall dropped by 84% after implementation of the Clean Indoor Air Law in 2003 (36).

New York City exemplifies the success of ETS bans. "The city's bar and restaurant industry is thriving and its workers are breathing cleaner, safer air," said a report issued by the Economic Development Corporation and the Departments of Finance, Health & Mental Hygiene and Small Business Services (37). Mike O'Neal, who served as president of the N.Y. State Restaurant Association for 17 years, supported legislation for a comprehensive smoking ban. "I feel strongly," he said "that it is pro-business and pro-health to eliminate smoking in all workplaces. We owe our workers a safe, healthy, work environment."

The message of smoking bans in public places to parents goes a long way toward telling them not to smoke in their own homes, as California statistics have convincingly shown. When smokefree workplaces increased from 35% in 1990 to 93% in 1999, smokefree residences rose from 8% in 1992 to 74% in 1999 (38).

The strict smoking bans in California have also led to a substantial decline in smoking prevalence as well as the state's heart disease, lung cancer, and chronic obstructive pulmonary disease (COPD) rates, falling well below the national average(39, 40). New figures show that smoking among California youth has decreased to record lows. According to the 2004 California Student Tobacco Survey released by the California Department of Health Services (CDHS), 13.2 percent of high school students in California smoked last year, compared with 16.0 percent in 2002 and 21.6 in 2000. Among middle school students in grades six through eight, 3.9 percent reported smoking last year, compared with 4.4 percent in 2002 and 6.7 percent in 2000 (41).

One out of every six deaths from cardiovascular disease is caused by smoking, the leading preventable risk factor. If all American workplaces were made smokefree, a computer simulation based on survey data from 1999 and 2000 projected that, over a period of seven years, 2,420 deaths from heart disease could be avoided and $280 million saved. Nonsmokers accounted for 60% of the reduction in heart attacks because workplace exposure to ETS is still extensive (42).

Americans for Nonsmokers' Rights, ANR, conducted an extensive survey showing that smokefree ordinances do substantially reduce smoking (43).

VENTILATION CANNOT PROTECT FROM ETS EXPOSURE

At a workshop sponsored by OSHA and the American Conference of Governmental Industrial Hygienists (ACGIH) in June 2000, a panel of ventilation experts concluded that neither dilution ventilation, the type of ventilation used in virtually all mechanically ventilated buildings, nor air cleaning, nor displacement ventilation technology can reduce ETS risk to minimum levels for workers or patrons in the hospitality industry without impractical increases in ventilation (see 33).

NEW VOICES FROM THE TOBACCO INDUSTRY

Philip Morris (PM), the most successful U.S. based multinational cigarette maker, has undertaken an extraordinary sea change in confronting the rising tide of devastating health news about smoking (active as well as passive). After more than half a century of blatant denial, PM is suddenly coming out to fully embrace the death and destruction its products inflict (44). "Our consumers have told us [that] as a responsible manufacturer of a risky product, they expect us as a company to be open about the risks of smoking and to be communicating that information voluntarily above and beyond the law." (45) The situation appears to be totally grotesque, bordering on the obscene, in that there is a manufacturer who admits its merchandise harms and kills yet continues to advertise and sell it at home and abroad, and to harvest the world's children as customers to secure future profits. The fact that PM was able to do this with impunity, and by preserving the status quo of conducting "business as usual" without even the least objection by government or society, reflects the depth of social morass and the moral abyss of disintegrating values into which this civilization has plunged.

ETS UPDATE 1998-2005 - MAIN POINTS

- A tobacco industry lawsuit challenging the U.S. EPA's 1992 classification of ETS as a Class A Human Carcinogen was dismissed by the Fourth Circuit Court of Appeals.

- Additional studies reaffirmed and strengthened the causal association between ETS exposure and lung cancer.

- Children exposed to ETS are at risk of lung cancer later in life.

- The World Health Organization's International Agency for Research on Cancer formally endorsed ETS as a cause of lung cancer.

- The weight of additional studies prompted the association between ETS and breast cancer to be changed from "inconclusive" to "conclusive."

- The causal association between ETS and nasopharyngeal cancer, and brain cancer and lymphomas in children is "suggestive." Additional studies also affirmed the causal association with cervical cancer as "suggestive."

- The metabolites of the potent nicotine-derived lung carcinogen, NNK, were identified in the body of nonsmokers exposed to ETS, including infants born to smoking mothers and elementary school children.

- In a study of 4,399 children aged 6 to 16, nicotine exposure as determined by its metabolite, cotinine, in blood, urine, saliva and hair, was found to significantly impair, in a dose-related manner, the children's reading, math and reasoning scores.

- A mere thirty minute exposure of healthy, young nonsmokers to ETS causes a substantial reduction in the coronary flow velocity reserve indistinguishable from that seen in habitual smokers. This may explain why ETS causes much greater damage to the cardiovascular system than one would expect on a quantitative basis.

- Smokefree legislation significantly reduces smoking and smoking-related disease.

- In an unprecedented turn, Philip Morris agrees that active and passive smoking cause lung cancer, cardiovascular and respiratory disease, etc. PM also admits that "There is no safe cigarette." Notwithstanding these admissions, Philip Morris continues to market its potentially lethal merchandise across the world.

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35) CDC Advisory on Heart Disease from ETS , Washington Post, April 23, 2004.

36) Indoor Air Quality in Hospitality Venues Before and After Implementation of a Clean Indoor Air Law in Western New York, 2003. Office on Smoking and Health, MMWR 53 (44), November 12, 2004.

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38) E.A. Gilpin, et al. Clean indoor air: Advances in California, 1990-1999. American Journal of Public Health 92: 785-791, 2002.

39) C. Fichtenberg and S. A. Glantz. Association of the California Tobacco Control Program with declines in cigarette consumption and mortality from heart disease. New England Journal of Medicine 343: 1772-1777, 2000.

40) UC DAVIS Study: Fewer Californians dying of lung cancer, pulmonary disease as smoking decreases. EurekaAlert May 21, 2001.

41) Joe Cherner. Smokefree California - 15 years later. Joe (at) smokefree.org 1/29/05.

42) M.Ong. Health benefits of snuffing out workplace smoking. Business Wire, June 22, 2004 and News-Medical in Medical Study News, June 24, 2004.

43) ANR (http://www.no-smoke.org). Effect of Smokefree Air Ordinances on Smoking Prevalence and Cessation, Nov. 2004. FS-07)_110504.doc.

44) From the Philip Morris website: www.philipmorris.com

Cigarette Smoking and Disease in Smokers

Philip Morris USA agrees with the overwhelming medical and scientific consensus that cigarette smoking causes lung cancer, heart disease, emphysema and other serious diseases in smokers. Smokers are far more likely to develop serious diseases, like lung cancer, than non-smokers. There is no safe cigarette. These are and have been the messages of the U.S. Surgeon General and public health authorities worldwide. Smokers and potential smokers should rely on these messages in making all smoking-related decisions.

Secondhand Smoke

Secondhand smoke, also known as environmental tobacco smoke or ETS, is a combination of the smoke coming from the lit end of a cigarette plus the smoke exhaled by a person smoking. Public health officials have concluded that secondhand smoke from cigarettes causes disease, including lung cancer and heart disease, in non-smoking adults, as well as causes conditions in children such as asthma, respiratory infections, cough, wheeze, otitis media (middle ear infection) and Sudden Infant Death Syndrome. In addition, public health officials have concluded that secondhand smoke can exacerbate adult asthma and cause eye, throat and nasal irritation. Philip Morris USA believes that the public should be guided by the conclusions of public health officials regarding the health effects of secondhand smoke in deciding whether to be in places where secondhand smoke is present, or if they are smokers, when and where to smoke around others. Particular care should be exercised where children are concerned, and adults should avoid smoking around them.

We also believe that the conclusions of public health officials concerning environmental tobacco smoke are sufficient to warrant measures that regulate smoking in public places. We also believe that where smoking is permitted, the government should require the posting of warning notices that communicate public health officials' conclusions that secondhand smoke causes disease in non-smokers.

45) Andrew White, Philip Morris Asia Pacific vice-president of corporate affairs, quoted by The Standard, Sing Tao Newspaper Group and Global China Group, Nov. 16, 2004, at http://www.thestandard.com.hk/stdn/std/Metro/FK16Ak03.html .