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## Health Risk of Environmental Tobacco Smoke (ETS)

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### ABSTRACT

Environmental tobacco smoke is a preventable cause of significant morbidity and mortality among non-smokers. Reducing exposure to tobacco smoke is an essential community and public health objective. This report documents the substantial evidence characterizing the health risks caused by exposure to passive smoking. Multiple major reviews of evidence have concluded that environmental tobacco smoke is a known human carcinogen and that exposure to passive smoke causes adverse effects like SIDS, congenital birth defects, lead poisoning, and lung cancer. Regrettably, reductions in exposure have been slower among small children than among adults, as growing workplace restriction protects the majority of adults while the homes remain the most important source of exposure for children. The relationship between passive smoking exposure and potential health hazards to all ages of population were researched. We analyzed and systematically reviewed information from multiple literature sources. We found that everyone from the fetus to the elderly is significantly affected by passive smoking. Like firsthand smoking, secondhand smoking is also hazardous. Health education and regulations regarding secondhand smoking in both the office and home area can improve public health. This paper indicates a critical need for second hand smoke reduction interventions especially among vulnerable populations.

### INTRODUCTION

Passive smoking is also called environmental tobacco smoke (ETS) or second hand smoking (SHS). Adverse health effects result from diluted mixture of mainstream smoke exhaled by smokers and sidestream smoke from the burning end of a cigarette or other tobacco product. Environmental tobacco smoke is chemically similar to smoke inhaled by smokers and contains a complex mix of over 4,000 chemicals, including a number of cancer-causing chemicals and toxic substances such as nicotine, carbon monoxide (CO), and nitrogen. Side stream smoke is the key component of ETS, providing nearly all of the vapor-phase constituents and more than half the particulate substances. Sidestream and mainstream smoke are dissimilar in their temperature of combustion of tobacco, pH, and degree of dilution in air, which happens in combination with a decrease in temperature. All of the five known carcinogens, nine probable human carcinogens, and three animal carcinogens are released at higher levels in sidestream smoke than in mainstream smoke. Additionally, many toxic compounds, such as ammonia and carbon monoxide, are produced at higher levels in sidestream smoke. The risk for cancer and other smoking-associated diseases for nonsmokers are also high. The risk is not lowered by smoking light, mild, or low tar cigarettes. In fact, studies have shown that when smoking these types of cigarettes, smokers often inhale more deeply while smoking.<sup>1</sup>

There are sensitive and positive markers of exposure to passive smoking. Vapor-phase nicotine and respirable suspended particulate matter have been detected as markers for the presence and concentration of ETS in the environment. Cotinine, a metabolite of nicotine and to a lesser degree nicotine itself are extensively used biomarkers of ETS exposure and uptake. The dose-response association between urinary cotinine levels and self reported exposure to tobacco smoke is strong. Biomarker data demonstrate that levels of ETS constituents encountered indoors by smokers are large enough to be absorbed and result in

measurable doses in exposed persons. For example, a huge nationally representative sample of persons age 4 and older indicated that 88% of nontobacco users had measurable levels of serum cotinine, although only 37% of adults and 43% of children were alert that they were exposed to ETS in the home or at work.<sup>2</sup>

### **ENVIRONMENTAL TOBACCO SMOKE AND CHILDREN'S HEALTH**

Most cigarette smokers have children living in the home and 70 percent permit smoking in some or all areas of the home. Urinary cotinine concentrations in infants and young children were linked with the number of smokers reported in the home and the number of cigarettes smoked by the mother during the previous 24 hours.<sup>3</sup> Numerous studies in the early 1970s suggested a connection between ETS exposure in the home and respiratory conditions among children.<sup>4-6</sup> ETS is a major contributor to impaired respiratory health among children, especially young children. U.S. Environmental Protection Agency (EPA) concluded that ETS exposure is causally linked with increased risk of lower respiratory infections (e.g., bronchitis and pneumonia) in children and an approximated 150,000 to 300,000 cases each year among infants and young children up to 18 months of age are related to ETS exposure. The EPA also concluded that ETS exposure is indifferently associated with increased prevalence of middle ear infection, upper respiratory tract irritation, and decreased lung function in children. ETS is usually associated with increased incidents of asthmatic exacerbation in children---an estimated 200,000 to 1,000,000 asthmatic children have their condition worsened by exposure to ETS---and is a risk factor for new cases of asthma among children who have not previously been symptomatic.<sup>7</sup>

Children exposed to ETS have an average 1.87 more days of limited activity, 1.06 more days in bed, and 1.45 more days missing from school than do children not exposed to ETS. Nationwide, children have 18 million days of restricted activity, 10 million days of bed confinement, and 7 million days of school absence attributable to daily ETS exposure.<sup>8</sup>

### **ENVIRONMENTAL TOBACCO SMOKE AND SUDDEN INFANT DEATH SYNDROME**

Multiple research studies have revealed a relationship between infant exposure to ETS and sudden infant death syndrome (SIDS) independent of the effects of maternal smoking during pregnancy.<sup>9-11</sup> This connection has been found for maternal smoking, paternal smoking, and smoking by other relatives or guests in the household. In 1997, California's Environmental Agency reported that there was sufficient evidence to conclude a causal association between ETS and SIDS.<sup>12</sup> How ETS causes SIDS is solely exploratory at present. One analysis is that the toxic chemical in tobacco (eg, nicotine and its metabolites) may actually injure the developing brain, especially the cardiorespiratory center in the midbrain.<sup>13</sup> Another suggestion is that an infectious agent interacting with the chemical products of ETS may cause SIDS.<sup>14</sup> Low levels of nicotine or one of its primary metabolites, cotinine, may actually result in the potentiation of low levels of certain bacterial toxins.<sup>15</sup> This could in part elucidate why the cause of SIDS deaths is not often noticeable on postmortem examination.<sup>16</sup> A third view is that nicotine from ETS may cause SIDS through weakened metabolism of catecholamine's in the brain, resulting in decreased awakening and ventilatory responsiveness to hypoxia. Since comparative growth failure is noted in infants exposed to ETS both prenatally and postnatally, it is rational to deduce that brain growth, including the cardiorespiratory center, is also impaired in these infants.<sup>17</sup>

### **SECONDHAND TOBACCO SMOKE IS A SOURCE OF LEAD EXPOSURE IN US CHILDREN AND ADOLESCENTS**

In the United States, around 1 in 5 children aged 3 to 11 years live with at least 1 individual who smokes. Globally, the burden of SHS exposure during childhood is even higher. Lead is a major neurocognitive and kidney toxicant for children at relatively low levels. Lead is a tobacco constituent that is measured in mainstream smoke (exhaled by the smoker) and sidestream smoke (from the burning cigarette). During the period 1988 to 1994, US children exposed to SHS showed increased blood lead levels.<sup>18</sup> Children are more at risk for lead poisoning because their smaller bodies are in a continuous state of growth and development. Lead is absorbed at a faster rate compared to adults, which causes more physical harm to children than to older people. Furthermore, children, especially as they are learning to crawl and walk, are constantly on the floor and therefore more prone to ingesting and inhaling dust that is contaminated with lead.<sup>19</sup>

The classic signs and symptoms in children are loss of appetite, abdominal pain, vomiting, weight loss, constipation, anemia, kidney failure, irritability, lethargy, learning disabilities, and behavioral problems. Slow development of normal childhood behaviors, such as talking and use of words, and permanent mental retardation are both commonly seen.<sup>20-21</sup>

### **ENVIRONMENTAL TOBACCO SMOKE AND ADULT SYMPTOMS**

The most common complaints are irritation to the eye, nose, and headache. The primary eye symptoms are reddening, itching, and tearing. The chief respiratory tract symptoms are itching, cough, and sore throat. Exposure to ETS precipitates and aggravates asthmatic exacerbation, cough, and hoarseness of voice. ETS causes respiratory tract infection among adults.<sup>22</sup> Exposure to secondhand smoke may increase the probability of cognitive impairment and dementia in adults 50 and over.<sup>23</sup>

Second-hand smoke exposure is linked with deafness in non-smoking adults.<sup>24</sup> Childhood exposure to environmental tobacco smoke is associated with an increased risk of the development of adult-onset atopic dermatitis.<sup>25</sup>

### **ENVIRONMENTAL TOBACCO SMOKE AND LUNG CANCER**

A large number of lung cancer deaths among nonsmokers can be associated with involuntary smoking. Several research studies suggested a positive association between ETS and lung cancer. Twenty four of 30 studies reported a higher risk of lung cancer among never-smokers exposed to ETS than among never-smokers not exposed to ETS. ETS is a human carcinogen, blamed for about 3,000 lung cancer deaths yearly in US nonsmokers.<sup>26</sup> ETS has been categorized as a Group A carcinogen under EPA'S carcinogen guidelines. Multiple studies revealed that spousal smoking is linked with an increased risk of lung cancer among nonsmoking spouses most heavily exposed to ETS.<sup>27</sup>

### **ENVIRONMENTAL TOBACCO SMOKE AND OTHER DISEASES**

Epidemiological evidence of a connection between ETS and cardiovascular disease among nonsmokers has been increasing. A 1994 meta-analysis of 12 studies revealed that there was a 23% higher CHD mortality among never-smokers exposed to ETS than in never-smokers not exposed to ETS.<sup>28</sup> A new cohort study found that ETS exposure increased the progression of atherosclerosis by 20 percent compared to those not exposed to ETS.<sup>29</sup> ETS seems to cause increased platelet aggregation, increased thrombosis, decrease oxygen supply, and increased oxygen demand. The association of ETS with heart diseases results from the carbon monoxide (CO) in smoke. Inhaled CO deprives the body from oxygen; the heart muscle cannot function without oxygen.<sup>30</sup> A cigarette left on fire produces more CO than one actively smoked.<sup>31</sup>

### **FETAL EFFECTS OF MATERNAL SMOKING**

The embryo (product during first trimester of pregnancy) and fetus (product during second and third trimester of pregnancy) develops in their own environment surrounded by the maternal environment. More than 25% of women continue to smoke during their pregnancies. Infants born to women who smoke during pregnancy weigh an average of 200g less than those born to nonsmokers. The incidence of low-birth weight (less than 2,500 g) in infants born to mothers who smoke is twice that in infants born to nonsmokers. The association between maternal smoking and low-birth weight is dose dependent and independent of other factors known to influence birth weight, including race, parity, maternal body weight and height, socioeconomic status, gender of the child, and gestational age. An estimated 17 to 26 percent of low-birth weight could be prevented by eliminating smoking during pregnancy. Women who stop smoking before becoming pregnant have infants of the same birth weight as never-smokers do. Additionally, pregnant smokers who quit in the first 3 to 4 months of pregnancy and remain abstinent through the rest of the pregnancy have normal birth weight infants. Pregnant women who stop smoking before the 30<sup>th</sup> week of gestation have infants with higher birth weight than do continuing-smokers. The effects of smoking are greater on fetuses whose mothers also receive inadequate nutrition. Presumably, there is an additive effect of heavy smoking and poor quality diet.<sup>32</sup>

Preterm delivery is linked with maternal smoking (relative risk of 1.5). An estimated 7 to 10 percent of preterm deliveries could be prevented by eliminating smoking during pregnancy. However, smoking affects birth weight mainly by retarding the fetal growth. The risk for a small for date infant is 3.4 to 4 times elevated among women who smoke during pregnancy than nonsmoking women. In 1985, the center for disease control defined the fetal tobacco syndrome as follows: a)the mother smoked 5 or more cigarettes per day throughout the pregnancy; b)the mother had no evidence of hypertension during pregnancy, specially no pre-eclampsia and documentation of normal blood pressure at least once after the first trimester; c) the newborn infant had symmetrical growth retardation at term, 37 weeks, defined as birth weight less than 2,500g and a ponderal index (weight in grams divided by length) greater than 2.32; and d) there is no obvious cause of intrauterine growth retardation, such as congenital malformation or infection. A number of mechanisms are thought to cause the reduction in fetal growth, including impaired maternal weight gain, and increased cyanide exposure (leading to impaired vitamin B<sub>12</sub> metabolism). The most important mechanism, however, is thought to be intrauterine hypoxia, which is caused by carboxyhemoglobin production from carbon monoxide exposure and vasoconstriction of the umbilical arteries. Even though fetal growth is diminished among maternal smokers, placenta-to-birth weight ratios are larger in comparison with those of maternal nonsmokers, probably because of the larger placental surface is required to provide adequate fetal oxygenation in smokers.<sup>33</sup>

Maternal smoking is associated with higher fetal, neonatal, and infant mortality, independent of sociodemographic factors for such mortality. Some data support an association between smoking and increased risk of spontaneous abortion. Smoking during pregnancy also raises the risk of placenta previa and abruption placentae. One large study showed adjusted infant mortality rates of 15.1 per 1,000 for white nonsmokers and 23.3 per 1,000 for white women who smoked more than one pack per day. Comparable infant mortality rates from black women were 26.0 and 39.9 per 1,000, respectively.<sup>34</sup> A strong risk factor for sudden infant death syndrome (SIDS) is maternal smoking during pregnancy. Studies have shown a 2- to 4- fold increased risk of SIDS among infants whose mothers smoked during pregnancy compared with infants of nonsmoking mothers, even after other risk

factors were controlled.<sup>35-36</sup> Nearly all hypotheses about possible mechanisms center around the effects of maternal smoking on fetal oxygenation and fetal development. One animal study reported that exposure to nicotine leads to reduced tolerance of hypoxic episodes and increased mortality.<sup>37</sup>

A few studies that have examined the long-term consequences of maternal smoking in offspring suggest a slight increase in the incidence of mental retardation, cerebral palsy, epilepsy, hyperactivity, shortened attention span, lower test scores, urinary tract anomalies, and electroencephalographic abnormalities. In a case-control study, there was a modest increase in the incidence of infants with conotruncal heart defects and limb deficiencies associated with both maternal and paternal smoking. Nonetheless, these studies are limited by small numbers and infrequency of events of interest, making any conclusion premature.<sup>38</sup>

### **PUBLIC HEALTH LAWS AND SECOND HAND SMOKING**

Many preemptive laws actually have reduced the level of protections previously afforded by local regulation where the local laws were more severe than state measures. A local debate helps to educate the community about the potential dangers of a particular public health problem (such as debate with a city council about exposure of workers to customers' tobacco smoke in restaurants and other public places). Thus, communities lacking the abilities to participate in local debates over public health decisions may pay less attention to the dangers involved and may be less aware of ways to address the problem. Like Florida, some 19 other states enacted statutes that took the power away from, or preempted, cities or towns that might want to enact local laws with stronger protection for non smokers. Courts will not defer to public health agencies where their actions are not supported by adequate legal authority. Annually, about 1 million young Americans become regular smokers. Nicotine addiction is difficult to remove. Smokers cause about 50,000 deaths each year among nonsmokers exposed to second hand smoke (SHS; also called passive smoking). Policies that discourage tobacco use and protect nonsmokers from exposure to SHS are also key elements of comprehensive tobacco-control program.<sup>39</sup>

Law passed by the state legislatures or regulations promulgated from state agencies may create the authority to provide the funding for local health departments or other entities to act to protect the public health. Usually, local authorities are free to act as long as they do not directly conflict with guidance from state or federal entities. In fact, enforcement of many public health laws is left to local officials. One locality with great success to protect citizens from second hand smoke is New York City, which passed a strict smoke-free air law in 2002 for almost all workplaces, which was later amended when New York State passed a similar law a year later.<sup>40</sup>

### **CONCLUSIONS**

Currently, society has less tolerance for smokers, and smoke free areas are now common. Many American workplaces do not permit smoking indoors and many employers do not hire smokers. Most states restrict smoking in public places such as government buildings. Many states restrict smoking in private workplaces. Nationwide public education campaigns designed to increase awareness of environmental tobacco smoke and local legislature can reduce ETS. On June 22, 2009, President Barack Obama signed the Family Smoking Prevention and Tobacco control Act.<sup>41</sup> This historic legislation provides the Food and Drug Administration (FDA) with the authority to regulate tobacco products, the leading cause of preventable deaths in the United States.

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**KEY TERMS**

Passive Smoking, ETS and Children Health, Lung Cancers, Lead Poisoning and Passive Smoking, SIDS and Congenital Birth Defects