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Effect of Environmental Tobacco Smoke on General and Oral Health

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Abstract

Secondhand smoke is a mixture of the smoke given off by the burning end of a cigarette, pipe, or cigar (sidestream smoke), and the smoke exhaled by smokers (mainstream smoke). Secondhand smoke is also called environmental tobacco smoke (ETS) and exposure to secondhand smoke is sometimes called involuntary or passive smoking. Exposure to environmental smoke has been implicated with serious health risks in children and adults. Nonsmokers exposed to second hand smoke absorb nicotine and other compounds similar to smokers but the smoke is less concentrated than that inhaled by smokers. Many epidemiological studies have shown a causal relationship between environmental tobacco smoke and lung cancer. Environmental tobacco smoke is classified under Group A carcinogen a category reserved only for the most dangerous cancer causing agents in humans. In children ETS exposure increases the risk of respiratory tract infections. In adults it increases the risk of lung cancer and cardiac disease. There is association between ETS exposure and periodontal disease, dental caries. The health risks of ETS have been a major motivation for smoke-free laws in workplaces and indoor public places and these laws have posed a serious economic threat to tobacco industries.

Key Words

Environmental; tobacco; smoke; sidesteam smoke; mainstream smoke

INTRODUCTION

Tobacco use remains the leading cause of premature and preventable death in our society. Worldwide, the tobacco epidemic killed 100 million people in the 20th century and is projected to kill one billion people worldwide in the 21st century. Each year, smoking kills more than five million people around the globe. Developing countries already account for half of all deaths attributable to tobacco (WHO, World Health Report, 2002).[1] This proportion will rise to 7 out of 10 by 2025 because smoking prevalence has been increasing in many low - and middle-income countries even though it is decreasing in high-income countries. The dramatic rise in smoking in the 20th century prompted a prominent historian to refer to that time period as "The Cigarette Century".[2] In the latter part of the 20th century, research on the risks of ETS began to be made public. This public awareness eventually became public policy in 1975 when the U.S. state of Minnesota enacted the Minnesota Clean Indoor Air Act. This made it the first state to ban smoking in most public spaces (with the exception of bars) and as of October 2007, Minnesota enacted a ban on smoking in all restaurants and bars state-wide, called the Freedom to Breathe Act of 2007. In 1990, the US city of San Luis Obispo, California, became the first city in the world to ban indoor smoking in all public places, including bars and restaurants.[3]

ENVIRONMENTAL TOBACCO SMOKE

Environmental tobacco smoke (ETS) or Second Hand Smoke (SHS) is the combination of two forms of smoke from burning tobacco products: Sidestream smoke, or smoke that is emitted between the puffs of a burning cigarette, pipe, or cigar, and Mainstream smoke, or the smoke that is exhaled by the smoker. When a cigarette is smoked, about one-half of the smoke generated is Sidestream smoke. This form of smoke contains essentially all of the same carcinogenic (cancer-causing) and toxic agents that have been identified in the mainstream smoke inhaled by the smoker, but at greater levels [4]. When non-smokers are exposed to SHS it's called involuntary smoking or passive smoking. Non-

smokers who breathe in SHS take in nicotine and toxic chemicals by the same route smokers do.[5] Sidestream smoke has higher concentrations of cancer-causing agents (carcinogens) and is more toxic than mainstream smoke. And, it has smaller particles than mainstream smoke. These smaller particles make their way into the lungs and the body's cells more easily.[5] SHS contains more than 7,000 chemicals, including hundreds that are toxic and about 70 that can cause cancer.[6] Among these are about 60 compounds that are carcinogens, tumor initiators and tumor promoters (substances that can lead to tumor growth once cell changes begin). Some of these compounds are tar, carbon monoxide, hydrogen cyanide, phenols, ammonia, formaldehyde, benzene, nitrosamine, and nicotine. In light of the widespread presence of ETS in both the home and workplace and its absorption by the body, the US Environmental Protection Agency (EPA) released a report in 1992 in which ETS was classified as a Group A carcinogen a category reserved only for the most dangerous cancer-causing agents in humans.[5]

Who is most at risk?[7]

SHS exposure dropped by half from 1 in 2 nonsmokers in 1999-2000 to 1 in 4 nonsmokers in 2011-2012, but exposure remains especially high for certain groups.

- 2 in 5 children (about 15 million) ages 3 to 11 are exposed to SHS.
- Nearly half of black nonsmokers are exposed to SHS, including 7 in 10 black children.
- More than 2 in 5 nonsmokers who live below the poverty level are exposed to SHS.
- More than 1 in 3 nonsmokers who live in rental housing are exposed to SHS.[7]

Health Effects of Secondhand Smoke[8]

Although the smoke to which an involuntary smoker is exposed is less concentrated than that inhaled by smokers, research has demonstrated that the health risk from inhaling smoke is significant.[8]

- There is no risk-free level of secondhand smoke exposure; even brief exposure can be harmful to health.
- Since the 1964 Surgeon General's Report, 2.5 million adults who were nonsmokers died because they breathed secondhand smoke.[8]

Health Effects in Children [8]

In children, secondhand smoke causes the following

- Ear infections
- More frequent and severe asthma attacks
- Respiratory symptoms (for example, coughing, sneezing, and shortness of breath)
- Respiratory infections (bronchitis and pneumonia)
- A greater risk for sudden infant death syndrome (SIDS).[8]

Concentrations of respirable suspended particulate matter (particulates of <2.5 μm) can be two to three times higher in homes with smokers than in homes with no smokers. Children exposed to environmental tobacco smoke have higher rates of lower respiratory illness during their first year of life, higher rates of middle ear effusion, and higher rates of sudden infant death syndrome.[9]

Health Effects in Adults[8]

In adults who have never smoked, secondhand smoke can cause:

- Heart disease[8]

For nonsmokers, breathing secondhand smoke has immediate harmful effects on the heart and blood vessels[8]. Passive smoking has also been reported to alter lipid profiles in adolescents which may shed light on the mechanism of increased risk of coronary heart disease in passive smokers. It is estimated that secondhand smoke caused nearly 34,000 heart disease deaths each year during 2005–2009 among adult nonsmokers in the United States[9].

- Lung cancer[8]

Secondhand smoke exposure caused more than 7,300 lung cancer deaths each year during 2005-2009 among adult nonsmokers in the United States. In 1986, two reports were published on the association between ETS exposure and adverse health effects in nonsmokers: one by the US Surgeon General and the other by the Expert Committee on Passive Smoking, National Academy of Sciences' National Research Council (NAS/NRC). Both of these reports concluded that: ETS can cause lung cancer in healthy adult nonsmokers.[4].

- Stroke[8]

Prenatal and Postnatal Environmental Tobacco Smoke Exposure and Children's Health[10]

A large literature links both prenatal maternal smoking and children's ETS exposure to decreased lung growth and increased rates of respiratory tract infections, otitis media, and childhood asthma, with the severity of these problems increasing with increased exposure. Sudden infant death syndrome, behavioral problems, neurocognitive decrements, and increased rates of adolescent smoking also are associated with such exposures.[10]

EFFECT OF ENVIRONMENTAL TOBACCO SMOKE ON ORAL HEALTH

Passive smoking and dental caries[11,12]

Passive smoking may be a modifiable risk factor for dental caries. ETS is associated with decreased serum vitamin C levels in children and decreased levels of vitamin C are associated with growth of cariogenic bacteria. Also, it is possible that ETS may reduce the protective properties of saliva that operate against caries - saliva acts as a buffering agent when acids are produced, it physically removes debris from tooth surfaces, and it has immunological and bacteriostatic properties.[11] ETS may also encourage the activity of S. mutans in children's mouths by interfering with the normal function of their immune system.[12] Environmental tobacco smoke is known to increase inflammation of the respiratory tract, producing symptoms of various clinical conditions, including allergic rhinitis, which frequently cause mouth breathing and thus result in dry mouth. Thus, ETS could promote dental decay both through a direct effect of nicotine on caries-causing bacterial agents, as well as via other systemic physiological changes in the host. Also, maternal smoking is a principal risk factor for prematurity, low birth weight, and chronic illness in infancy, while these in turn are all associated with generalized enamel hypoplasia in the primary dentition.[11]

Passive smoking and periodontal health[13,14]

There is evidence of a relationship between periodontitis in non-smokers and exposure to environmental tobacco smoke. Non-smokers exposed to ETS absorb approximately one-third the level of nicotine per cigarette absorbed by active smokers. Physiological metabolism of nicotine after exposure yields cotinine (nicotine's metabolite) in saliva, urine and serum. The concentration of cotinine in fluids allows determination of active smoking or environmental exposure, and provides a recent measurement of exposure, as well as an objective biomarker of exposure.[13] The increased risk for periodontitis occurs with the exposure to nicotine which over-stimulates the host response in the oral cavity, complicating the already inflammatory nature of periodontal diseases. ETS is associated with an elevated concentration of inflammatory markers interleukin -1β, albumin and aspartate aminotransferase, in those exposed to passive smoke. In studies by Nishida *et al*., (2006), (2008), significantly higher levels of albumin (an antioxidant) and AST (marker of cell destruction) were observed in individuals exposed to ETS than controls. It is possible that raised salivary albumin were expressed to thwart the detrimental action of free radical and ROS derived from ETS and or inflammatory cells in order to protect oral tissues including those of periodontium.[14] One of the many chemicals that get into the body when a person actively smokes or breathes someone else's smoke is nicotine. Earlier studies have suggested that nicotine in cigarette smoke impairs the immune system and causes blood vessels to constrict, including blood vessels in the tissues around the teeth. This causes a decrease in oxygen in these tissues which, along with an impaired immune system response, creates a favorable environment for bacteria that cause periodontal disease.[15]

Passive smoking and oral cancer[16]

It has also been postulated that chronic exposure to smoke from tobacco products may put nonsmoking individuals at a greater risk of developing neoplasia. In experimental studies, oral tumors have been induced in rodents through exposure of their oral mucosa to carcinogens found in smokeless tobacco. Cats living with smokers may be exposed to the same environmental contaminants as their owners, both through inhalation and through oral ingestion during grooming of particulate matter deposited on the fur. Like humans, cats exposed to household ETS metabolize nicotine into cotinine and demonstrate urinary cotinine levels that increase with exposure dose. Similarly, oral grooming may expose a cat's oral cavity to high levels of other chemicals, such as those contained in flea control products. If cats are exposed to a multitude of potentially toxic agents during grooming, the impact of these agents on their oral health must be considered. This includes their potential role in the development of oral tumors like Squamous cell carcinoma. Studies in human populations have demonstrated the presence of DNA adducts with tobacco-related carcinogens in neoplasms associated with tobacco use, as well as in the oral mucosal cells of cancer-free tobacco users.[16]

Passive smoking and the risk of orofacial clefts[17]

Orofacial clefts are among the most common major birth defects in the United States, with approximately 1 in 870 live births affected by cleft lip with or without cleft palate (CLP), and 1 in 1500 births affected by cleft palate only (CPO). Previous studies have identified a number of factors associated with orofacial clefts, including family history, genetic factors, birth order, occupational

exposures, anticonvulsants, multivitamin intake, alcohol, and smoking. Mothers who smoked heavily in the periconceptional period were about twice as likely to have an infant with any orofacial cleft (1.8; 95% CI 1.1–2.9) than were women who did not smoke during this period. There was a modest association with ETS among infants with CPO with multiple defects (1.7; 1.0-3.0), and a weak association among female infants with CPO. Maternal exposure to cadmium, another component of cigarette smoke, has been associated with cleft palate in animal models.[17]

DISCUSSION

ETS is implicated in a list of diseases that mirrors those caused by firsthand smoke, with a similar mechanism of action. For measurement of environmental exposure, especially in non-smokers, the mechanism of choice is isolation of cotinine in bodily fluids such as serum, saliva and urine.[13] Concerns around second-hand smoke have played a central role in the debate over the harms and regulation of tobacco products. Since the early 1970s, the tobacco industry has viewed public concern over second-hand smoke as a serious threat to its business interests. Harm to bystanders was perceived as a motivator for stricter regulation of tobacco products.[18] Epidemiological evidence has shown that ETS exposure causes an increased risk of cancer of 20-30%, an increased risk of heart disease of 25-30%, an increased risk of strokes of up to 82% and an increased risk of other non-fatal respiratory illnesses.[3] Measures to tackle second-hand smoke pose a serious economic threat to the tobacco industry, having broadened the definition of smoking beyond a personal habit to something with a social impact. In a confidential 1978 report, the tobacco industry described increasing public concerns about second-hand smoke as "the most dangerous development to the viability of the tobacco industry that has yet occurred.[18] Restrictions on tobacco advertisements, governmental health warnings and taxation on tobacco products, have been successful in decreasing smoking prevalence from 42.4% in 1965 to 24.7% in 1998 in the United States. Tobacco control programs have been introduced in various states in the US since the 1980s, funded largely by tax revenue on cigarette sales. These tobacco control programs have included measures such as: television, radio and print media public education campaigns; school-based tobacco prevention programs; smoking cessation material; telephone ‘quitlines’; policy change and enforcement.[3] The potency of the cigarette in violating clean air standards has been greatly underestimated by pro-smoking advocates. This fact needs greater attention and understanding by the public. The potency of the cigarette as a generator of high concentrations of toxic pollutants that can cause cancer and other long and short-term health effects argues strongly for restricting smoking in public places as a matter of prudent public policy.[19]

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