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Silent enemy: Environmental tobacco smoke

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Abstract:

Second-hand smoke, also known as environmental tobacco smoke (ETS), is one of the most harmful indoor pollutants. Exposure to ETS is a worldwide silent cause of mortality and morbidity. Although ETS had been decreased for 20 years, a lot of people who do not smoke still exposed to ETS at home, work, public places, and in vehicles. ETS is a risk factor for many important diseases such as lung cancer, chronic obstructive lung disease, asthma, cardiovascular diseases, upper and lower respiratory tract infections, and sudden infant death. In this article, the harmful effects of ETS and the effects of smoke-free environment regulation on ETS exposure were reviewed.

Keywords:

Environmental exposure, smoke-free environment, tobacco smoke

Introduction

Second-hand smoke, also known as environmental tobacco smoke (ETS), is one of the most harmful indoor pollutants known because it contains 50 known carcinogens. There are two primary sources of second-hand tobacco smoke: sidestream smoke that is released into the air directly from the smoldering end of a cigarette, cigar, or pipe and the mainstream smoke that refers to the smoke inhaled and then exhaled to the environment.^[1] Sidestream cigarette smoke is generated at lower temperatures (approximately 350°C) than mainstream smoke, during the slow and spontaneous smoldering of a cigarette, cigar, or pipe. Importantly, this type of smoke contains four times more harmful compounds than the main strain smoke.^[2] Exposure to ETS causes >41,000 deaths among nonsmoker adults, about 400 deaths in children per year, and loss of production about 5.6 billion dollars per year.^[3,4] Although ETS had been decreased for 20 years, a lot of people who

do not smoke still exposed to ETS at home, work, public places, and in vehicles.^[5] The National Health and Nutrition Examination Survey revealed that the frequency of ETS exposure in the USA was 52.5% in years of 1999–2000, and it decreased to 25.3% in the years of 2011–2012. ETS exposure in 2011–2012 years was found highest among children who were between 3 and 11 ages, non-Hispanic black people, and people who were living under poor conditions.^[6] ETS is a risk factor for many important diseases such as lung cancer, chronic obstructive pulmonary disease (COPD), asthma, cardiovascular diseases, upper and lower respiratory tract infections, and sudden infant death. In this article, the harmful effects of ETS and the effects of smoke-free environment regulation on ETS exposure were reviewed.

Effects of Prenatal Environmental Tobacco Smoke Exposure

ETS exposure may cause preterm birth. A meta-analysis declared that preterm birth may increase risk of the development of asthma in the future life.^[7] Studies showed that hospitalizations due to asthma attacks

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in England, Scotland, and emergency admissions because of asthma in Lexington (USA) decreased after smoke-free air act.^[8,9] A recent meta-analysis notified that hospitalizations due to asthma attacks decreased significantly after smoke-free air act.^[10]

Smoking during pregnancy is related with low birth weight, slowing down in head growth, and sudden infant death. Previous studies showed that prenatal and postnatal tobacco smoke exposure correlated with attention-deficit hyperactivity disorder, cognitive dysfunction, learning disability, behavior, and communication disorder.^[11,12] Evlampidou *et al.* evaluated effects of prenatal ETS exposure on neural development of children by measuring urine cotinine level in a large cohort, including 13,338 mothers and infants. It was found that gross motor function of children that was measured at 18 months was decreased.^[13]

It is not yet clear how does prenatal ETS exposure affect motor and cognitive development of children. Most of the tobacco products can pass placenta, and some of them cause fetal brain hypoxia.^[14] The cerebellum is responsible from general coordinations of movements and equilibrium of the body and contains a large amount of nicotinic receptors. Nicotinic receptors develop during the first trimester. Prenatal ETS exposure may change gene expression and binding of nicotinic receptors.^[15] Recently, Niu *et al.* showed that ETS exposure during pregnancy may increase the risk of low birth weight, and weight of placenta may be effective indirectly on this condition.^[16] A recent case-control study showed that pregnant women with genotypes (CYP1A1 "TC" and GSTT1 "null" or CYP1A1 "AG" and GSTT1 "null") were in higher risk of giving birth to full-term low birth weight when they exposed to ETS during pregnancy.^[17] Size, construction, and function of placenta correlate with transfer of nutrition to the fetus. Toxic contents of tobacco smoke such as nicotine, carbon monoxide, polycyclic aromatic hydrocarbons, cadmium, and thiocyanate may give rise to oxidative stress, vasoconstriction, endothelial dysfunction, and vascular damage. All of these negatively affect the development of placenta and structure and function of vessels.^[18] Toxic chemicals in tobacco smoke causing placental insufficiency prevent transfer of nutrient and oxygen to the fetus and lead to growth retardation or low birth weight.^[19] All of these factors negatively affect development, vascular structure, and function of the placenta.^[18] Table 1 shows prenatal and postterm effects of ETS.

Environmental Tobacco Smoke Exposure and Cardiovascular Diseases

Tobacco smoke also is an important risk factor for cardiovascular diseases. ETS causes 34,000 deaths per

Table 1: Effects of environmental tobacco smoke exposure in prenatal period

Effects in preterm and term period

Vasoconstriction in placenta

Preterm birth

Low birth weight

Postterm effects

Attention deficit and hyperreactivity

Cognitive dysfunction

Gross motor dysfunction

year due to cardiac diseases and 8000 deaths per year related to stroke in the USA. Risk of cardiac diseases and stroke increases, respectively, 25%–30% and 20%–30% among patients exposing ETS at home or work.^[20] Acute changes on vessels following ETS exposure increase the risk of heart attack. ETS exposure has many negative effects on the vascular system. It makes easy formation of thrombus causing platelet activation. It causes endothelial damage directly and indirectly by increasing oxidative stress. It also destroys repairment of the endothelium, decreases level of high-density lipoprotein in blood, and may give rise to endothelial dysfunction and inflammation on the vessel wall. All of these factors enable the development of atherosclerosis.^[21] It was reported that the risk of dementia increased among nonsmokers due to carotid artery stenosis as a result of ETS exposure.^[22]

A recent meta-analysis showed that hospitalization rates related to acute coronary syndrome, ischemic heart disease, sudden cardiac death, and cerebrovascular diseases significantly decreased following smoke-free air act.^[23] The relationship between ETS exposure during childhood and cardiovascular diseases in adulthood was yet not clear. The result of a cohort study by West *et al.* showed after 26 years follow-up that the risk of development of atherosclerotic plaques in adulthood increased in children exposing to ETS.^[24]

Environmental Tobacco Smoke Exposure and Cancer

ETS exposure increases the risk of lung cancer in the rate of 20%–30%. ETS exposure also is a risk factor for gastrointestinal system, genitourinary system, and hematological malignancies.

Chemical content of sidestream smoke is different from mainstream smoke. It contains a lot of carcinogens including aromatic amines, benzene, cadmium, and nickel. Some carcinogens such as aromatic amines are found in sidestream smoke much more amount than mainstream smoke.^[25] A recent study performed in North Europe revealed that the larynx, oral cavity, and lung cancer in waitresses and the pharynx, oral cavity,

and tongue cancers in waiters were more frequently observed than the general population. ETS exposure was defined as one of the risk factors for cancer development in people who were working in this category of occupation.^[26] Patients who were diagnosed as cancer and had a history of ETS exposure were found less successful in quitting smoking.^[27]

Environmental Tobacco Smoke Exposure and Tuberculosis

There is some evidence that ETS exposure increases susceptibility to *Mycobacterium tuberculosis* infection and makes easier progression of the disease by taking a toll of adaptive immune response and natural immune system of lungs.^[28,29] A recent meta-analysis showed that ETS exposure was related with the development of latent tuberculosis infection and active tuberculosis. Sidestream smoke makes a contribution to the development of tuberculosis disease by damaging pulmonary mechanical barriers and immune system. Mucociliary clearance which is the first defense mechanism against *M. tuberculosis* is damaged due to long-term ETS exposure. ETS exposure also increases susceptibility to *M. tuberculosis* infection and development of active disease by decreasing T-cell proliferation and causing dysfunction in alveolar macrophages.^[28]

Frequency of latent tuberculosis infection and active tuberculosis related to ETS exposure shows regional differences. It is not surprising that this ratio is high in Southeast Asia and Sub-Saharan Africa. Frequent ETS exposure among both adult and children in Spanish people makes easier development of the disease despite high level of socioeconomic level.^[30] It was shown recently in a meta-analysis that ETS exposure increased the risk of tuberculosis disease and tuberculosis infection among children.^[31]

Respiratory Tract Infections and Environmental Tobacco Smoke Exposure

ETS exposure is a major risk factor for respiratory tract infections. The relationship between bronchitis, bronchiolitis, otitis media, and other respiratory tract infections is well described. It was estimated that every year respiratory tract infections caused 166,000 deaths and 6.6 million disability-adjusted years among children around the world. It was reported that hospitalizations because of respiratory tract infections decreased after smoke-free environment regulation in England.^[32] Studies performed in adults also showed that smoke-free environment act decreased respiratory tract infections such as bronchitis and pneumonia in this population.^[23]

Environmental Tobacco Smoke and Chronic Obstructive Pulmonary Disease

Smoking is the most important risk factor for COPD. Studies showed that besides active smoking ETS exposure also plays a role in the pathogenesis of COPD and development of COPD attack.

ETS exposure increases the risk of bacterial infections by suppressing immune response of neutrophils and alveolar macrophages and makes easier the development of COPD. In a recent study, it was shown that ETS exposure inhibited phagocytosis of bacteria by disturbing ion channel activity of cystic fibrosis transmembrane regulator gene and facilitated living of *Pseudomonas aeruginosa* in airways. By this way, ETS exposure can play a role in pathogenesis and development of attack of the disease.^[33]

Environmental Tobacco Smoke Exposure and Asthma and Allergic Rhinitis

Asthma is a chronic inflammatory airway disease developing as a result of gene-environment interaction. ETS exposure is one of the risk factors for the development of asthma. There are important evidence that ETS exposure may cause the development of asthma, bronchial hyperresponsiveness, and airway inflammation and causes worse course of asthma.^[34,35] Studies showed that emergency admissions of children due to asthma attack decreased in year following avoidance of ETS exposure.^[36,37] Measurement of urine cotinine level, nicotine level in hair or nail, and carbon monoxide in exhaled air can be used to show ETS exposure. In a study performed in Finland, it was shown that smoke-free environment act might decrease the burden of asthma.^[38] In a recent study, it was found that allergic rhinitis which is a frequent comorbidity of asthma is more prevalent among adults who had ETS exposure.^[39]

Effects of ETS on airways were summarized in Table 2.

Environmental Tobacco Smoke Exposure and Bone

Effects of ETS on the bones are not yet clear but are under investigation. In a recent experimental study, the authors

Table 2: Effects of environmental tobacco smoke on airways

Decrease in mucociliary clearance
Depression in neutrophil response
Depletion in response of alveolar macrophages
Airway inflammation
Bronchial hyperreactivity
Exacerbation of COPD or asthma

COPD: Chronic obstructive pulmonary disease

showed that the second-hand cigarette smoke exposure to rats affected the structure of bones deforming them and making them osteopenic and bone growth was impaired. The effects seem to be related to the duration of exposure.^[40] In the bone tissue, maternal exposure to passive smoking was a determining factor for changes in the bone mineral density and bone mineral content of the offspring.^[41]

Effects of Smoke-free Environment Regulation on Environmental Tobacco Smoke Exposure

Smoke-free environment regulation that was applied in many countries including Turkey is an important attempt in the field of public health. Studies from different countries showed that cotinine levels in the body fluids of nonsmokers decreased after smoke-free environment regulation.^[42,43] In a study performed in our county revealed that symptoms such as nose stiffness, dyspnea, cough, and itching on eyes in workers of restaurants and cafes were decreased after smoke-free environment regulation.^[44]

A recent study performed in England showed that ETS exposure among children decreased in a rate of 79% in 2012 compared to 1998. The decrease was more apparent after smoke-free environment regulation. In this study, salivary cotinine level of children whose parents were smoker founded higher than children who had nonsmoker parents. It was stated that smoke-free environment regulation also should cover houses.^[45] In a study from India, it was shown that banning of smoking at home decreased the second-hand smoking.^[46]

Banning of smoking in closed area is not solely enough to prevent ETS exposure. Urine nitrosamine and salivary cotinine levels were found higher in people who were exposed to the second-hand smoke at open areas of bars and restaurants.^[47] Smoke-free regulation at open areas of bars and restaurants was started in Canada and many states of the USA. A study performed in Canada showed that banning of smoking at open areas of bars and restaurants significantly decreased ETS exposure.^[48] Chaiton *et al.* showed that exposure of smoke at terraces of companies negatively affected quitting success of smokers.^[49]

Conclusion

ETS exposure is a risk factor for many diseases including respiratory and cardiovascular diseases. Smoke-free environment regulation that was implemented in many countries including Turkey decreased frequency of ETS exposure-related diseases. However, prevention of ETS

exposure is not possible solely by banning of smoking in closed areas. Smoke-free environment regulation should enclose private cars, homes, and open areas of public places to be more efficient.

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Conflicts of interest

There are no conflicts of interest.

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