. Original Article

Effects of passive smoking on children health

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Smoking is the single greatest avoidable cause of disease and death. According to the World Health Organization (WHO), 1.1 billion people worldwide regularly smoke tobacco products and smoking accounts for 10,000 deaths per day due to the effect of smoke on the body. Non-smokers are at risk because of inhalation of passive smoke, also known as environmental tobacco smoke (ETS). Exposure to passive smoking (ETS) is harmful to the health of the general population and particularly hazardous to children. Passive smoking exposure causes disease and premature death in children and in adults who do not smoke. Researchers found significant dose-dependent associations between passive smoking and chronic obstructive pulmonary disease, stroke, ischemic heart disease, and all forms of cancer, and all respiratory and circulatory diseases.[1] The home is the place where children are most exposed to second hand smoke; where they spend much of their early life in the presence of their parents. Every day more than 15 million children are exposed to smoke in their homes. Children are especially sensitive to the effects, due to the fact that their bodies are still growing and developing and they breathe faster than adults and therefore may inhale more smoke. Children who live in homes where parents smoke have high levels (more than five times) of cotinine (a biological marker of secondhand smoke exposure) than children who have nonsmoking parents[2]. Also children with childhood ETS exposure were more likely to become smokers in adolescent or in adulthood.[3,4]

Keywords: Tobacco, cancer, toxicology.

Objective

The aim of this paper is to throw light on the harmful effect of passive smoking on children.

Method

The paper is a review of available literature, which was collected from statistical data of WHO atlas and published scientific papers that deal of ETS effects.

Findings

There is widespread scientific consensus that exposure to Environmental Tobacco Smoke (ETS) is harmful to the population and is linked to a number of adverse health outcomes. The link between ETS and health risks is accepted by all major medical and scientific organizations,

ETS prevalence and Negative Health Effect:

Report of the Surgeon General in 1979 noted several adverse respiratory outcomes in children and adults, as well as some acute cardiovascular effects associated with involuntary exposure to tobacco smoke[5].

Later in 1999 responding to the 1997 declaration on children's environmental health, the environment leaders of the eight (G8), World Health Organization (WHO) convened an international consultation on Environmental Tobacco Smoke (ETS) and child health[6].The consultation concluded that ETS is a real and substantial threat to child health, causing death and suffering throughout the world.

In 2004 study by the International Agency for Research on Cancer (IARC) of the World Health Organization (WHO)[7] concluded that nonsmokers are exposed to the same carcinogens as active smokers. More than 4,000 chemicals have been identified in secondhand tobacco smoke[8,9]. The National Toxicology Program estimates that at least 250 are known to be harmful, and over 50 of them are known to cause cancer. The U.S. Environmental Protection Agency (EPA), the

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U.S. National Toxicology Program (NTP), the U.S. Surgeon General, and the International Agency for Research on Cancer (IARC) have classified secondhand smoke as a known human carcinogen. The Surgeon General[9] reported that the concentrations of many cancer-causing and toxic chemicals found in tobacco smoke are potentially higher in secondhand smoke than in the smoke inhaled by smokers. The US Surgeon General, in his 2006 report, estimated that living or working in a place where smoking is permitted increases the non-smokers' risk of developing heart disease by 25–30% and lung cancer by 20–30%. Frequent exposure to environmental tobacco smoke during childhood was associated with lung cancer in adulthood[10] especially that 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK),a highly carcinogenic tobacco-specific nitrosamine, was detected in infants exposed to ETS support the concept that persistent ETS exposure in childhood could be related to cancer later in life[11].

Sudan is one of the developing Arab countries which relatively contain high prevalence of smoker 23.5%, as estimated in 1999[12]. There are no published studies about the prevalence of ETS and its effect. In some Arab countries such as Egypt[13], Lebanon[14], Jordan[15] and Syria[16,17] the negative effects of ETS on adults and children found attention by some researchers. In Lebanon Hala Tamim et al[14] reported that the prevalence of household smoking (either cigarette and/or narghile) in a sample of 1057 of pre- school children was 53.3%. This is markedly higher than that reported by the National Health Interview Survey in the United States in 2000, which estimates that children's passive smoking exposure at homes declined from 36% to 25% between 1992 and 2000. In Jordan E. Badran et al[15] made a study to estimate exposure to ETS during the first year of life. 220 infants attending the outpatient pediatric clinic of the University of Jordan for routine visits with their mothers were recruited to the study. A total of 60.0% of infants were reported to be exposed to passive smoking at home and 36.4% had detectable levels of urine cotinine. Detectable saliva cotinine levels in 8/20 mothers of neonates (1–2 days old) suggested in utero exposure. In 1999 the first research was done in Syria by Maziak[17] et al to study the effect of household ETS on children. 1859 children under 12 years from both urban and rural areas were studied. The study found a clear association between Sudden Infant Death Syndrome (SIDS) and childhood respiratory morbidity with parental smoking or other household smoker. Exposure to ETS does not only cause health problems among those who are exposed but also leads to increased health care utilization and cost for the respiratory problems that the developing countries suffer from and in most probability these problems do not find enough health care and may end by death. The American Society of Heating, Refrigerating and Air-Conditioning Engineers (ASHRAE), the pre eminent U.S. standard-setting body on ventilation issues, has concluded that ventilation technology cannot be relied on to completely control health risks from secondhand smoke exposure[8,9]. Secondhand smoke contains a number of poisonous gases and chemicals, including hydrogen cyanide (used in chemical weapons), carbon monoxide (found in car exhaust), butane (used in lighter fluid), ammonia (used in household cleaners), and toluene (found in paint thinners).

The health problems can start before birth, as active and passive smoking in pregnancy also have adverse health effects in the offspring of smoking mothers as quoted by Hawamdeh et al[18]. ETS is associated with a 10% increase in prenatal mortality and an increase in the incidence of premature labour and placental abruption. Smoking may also cause congenital damage to the developing respiratory system of the fetus, either to the bronchial tree or to the developing lung as also shown in animal models[19,20]. Other well-documented health effects include low birth weight, spontaneous abortion and numerous other causes of morbidity and neonatal mortality[21,22]. Sudden infant death syndrome (SIDS) has been directly linked to ETS exposure in numerous studies[23-25].

Furthermore ETS exposure was found to cause acute otitis media, recurrent otitis media, middle ear effusion, and inflammation of adenoid and/or tonsils[26,27], and there is strong relation between parental smoke and snoring in children[28,29].

ETS and Immunity:

Smoking has marked impact on the immune system, compromising the host's ability to mount appropriate immune and inflammatory responses and contributing to smoking-related pathologies. In a recent study by Marseglia et al[30] to investigate relation between ETS and recurrent respiratory infections in a cohort of children representing with both recurrent respiratory infections with a history of ETS exposure these factors were related to lower local production of interferon- γ (IFN-γ) when compared with a similar non- exposed population. Children exposed to passive smoke suffered from a significantly greater number of respiratory infections and have a lower percentage of IFN-γ producing CD-8+ cell in adenoids than non-exposed children.

ETS and Serious Bacterial Infection:

In the late 1960s Green and Carolin quoted by Philip[21] et al showed the depressant effect of tobacco smoke on the in vitro antibacterial activity of alveolar macrophages. As shown by several experimental animal studies, cigarette smoke depresses phagocytosis, impairs mucociliary clearance, enhances bacterial adherence, disrupts the respiratory epithelium, and decreases the serum Ig levels by ~10% to 20% lower than those of nonsmokers.

ETS and Asthma:

The Environmental Protection Agency (EPA)[31] reported that ETS exposure is causally associated with additional episodes and increased frequency and severity of symptoms in children with asthma. The report estimated that 200,000 to 1,000,000 asthmatic children have their condition worsened by exposure to ETS. Exposure to ETS during childhood is associated with an increased prevalence of asthma among adult who are never-smokers, especially in non-atopic subjects[4,32].

Other negative affects ETS:

ETS significantly reduces lung function[33-36]. Salivary cotinine levels, which are a reasonable marker for passive smoking, correlate inversely with small airway function. Also it may induce early lung damage in healthy male adolescents[37], adult cardiovascular disease[38] and also lead to lower High Density Lipoprotein (HDL) Cholesterol level in children with household smoke exposure than in those without household smoke exposure[39,40]. ETS may worsen the risk profile for later atherosclerosis among high-risk young persons. During the past decade, ETS exposure is being increasingly associated with behavioral and cognitive problems in children[41,42]. Also exposure of children to ETS leads to significant alterations in serum ascorbic acid levels[43].

Smoke & ETS as Predisposing factors to Carcinoma:

The risk pertains to all major types of lung cancer, particularly squamous and small cell types[44]. However, nonsmokers who live with smokers absorb and metabolize carcinogens that have been linked to an increased incidence of lung cancer. Researchers have estimated that ETS is responsible for approximately 3,000 lung cancer deaths per year among nonsmokers in the U.S[10].

Eleven compounds in tobacco smoke (2-naphthylamine, 4aminobiphenyl, benzene, vinyl chloride, ethylene oxide, arsenic, beryllium, nickel compounds, chromium, cadmium and polonium-210) have been identified by the International Agency for Research on Cancer as Group 1 carcinogens.

The first conclusive evidence on the danger of ETS came from Takeshi Hirayama's[45] study in 1981 on lung cancer in non-smoking Japanese women married to men who smoked. Further studies confirmed this finding[46,47]. Children exposed to passive smoking on a daily basis face more than triple the risk of lung cancer in later life compared with youngsters who live in smoke-free environments. The massive study, involving more than 303,000 non-smokers across Europe, found that living in a smoky

environment as a child significantly increased the danger of lung cancer in adulthood[48]. Hemminki K, Chen B[49] has reported that passive smoking during childhood is associated with an increased risk of nasal cancer. The same study also suggests that bladder and kidney cancers may be increased through breast feeding and in utero exposure. They used the Swedish family cancer database from 1958 to 2002 to determine whether children of parents with lung cancer were at risk for various cancers. This database included 17,693 mothers and 41,838 fathers with lung cancer. They found 173,715 cancers in the children of parents with lung cancer. Offspring of mothers with lung cancer had an increased risk of upper aero digestive (SIR 1.45), nasal (SIR 2.93), lung (SIR 1.71), kidney (6.41) and bladder cancer (1.52).

Further more there is emerging evidence that in-utero exposure to active or passive tobacco products by mothers has also been associated with an increased incidence of brain tumors in childhood[50]. Also exposure to paternal preconception smoking alone or in combination with postnatal passive smoking may be an important factor in the risk of childhood leukemia[51].

Conclusion and Recommendations

In Sudan we need to pay attention to the effect of environmental tobacco smoke (ETS) exposure. Both governmental and non-governmental agencies should work together to make this clear to the community.

Parents and caretakers of children need to be aware of negative effect of ETS and need to eliminate tobacco smoke exposure in environment and homes.

The prevalence of environmental tobacco smoke (ETS) exposure among children in Sudan is unknown. We need to asses it by prepared questionnaires. However, objective validation by assessing biomarkers in body fluids is often necessary to determine the true extent of the exposure.

In Sudan research is needed to assess the negative clinical effects of ETS.

We advise the government to pass laws prohibiting smoking in public facilities.

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