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Environmental Tobacco Smoke: Respiratory and Other Health Effects

Jane Z. Reardon, MSN, RN

Departments of Medicine and Nursing, Hartford Hospital, 80 Seymour Street, Hartford, CT 06102, USA

In general, people are better informed about the risks of active smoking than the dangers of environmental tobacco smoke (ETS). As more information is gathered, however, it has become clear that the dangers of ETS are undeniable. There is no safe level of exposure to ETS. In the 2006 Surgeon General's report on secondhand smoke, Dr. Richard H. Carmona declared, "the debate is over." The data reported since the landmark 1986 Surgeon General's report on the health hazards of secondhand smoke documents beyond any doubt that second hand smoke harms people's health. Millions of Americans still are exposed to ETS in their homes and workplaces despite the considerable progress in tobacco control. Written by 22 scientists, the 2006 Surgeon General's report was reviewed by at least 40 peer reviewers and 30 independent investigators [1]. The damaging effects of ETS reach far beyond the lungs. As discussed in this article, multiple body systems are harmed. The recognized adverse health effects of ETS exposure include chronic obstructive pulmonary disease (COPD); asthma; upper and lower respiratory tract infections; cardiovascular disease; lung, breast, and cervical cancer; and childhood illnesses, such as middle ear disease and sudden infant death syndrome (SIDS). It is reported that ETS exposure is the third leading cause of preventable death in the United States, resulting in more than 50,000 deaths per year [2]. The United States Environmental Protection Agency classifies ETS as a group A carcinogen, given the large number of known or suspected carcinogens in tobacco smoke [3].

Although there is a large body of evidence on the health effects of chronic exposure to ETS, there also is growing evidence regarding its short-term or immediate effects. For example, it has been shown that even very brief exposures to ETS can trigger intense bronchopulmonary responses that could be life threatening for infants, children, or adults who have asthma or highly sensitive respiratory systems. Some scientists, however, warn that the data on acute risks should be disseminated with caution so that media coverage does not distort the evidence and convince the public prematurely that brief, transient exposure is enough to cause chronic health problems [4].

ETS has been targeted as a public health priority for disease prevention. Although legislation has become increasingly effective in reducing ETS in public places, a significant amount of ETS exposure still occurs in the home, affecting particularly vulnerable groups, such as children and the elderly. Although it is impossible to legislate a ban on smoking in the home, educational efforts must be strengthened, and barriers to adopting ETS risk-reducing behaviors must be explored. ETS now is considered an unacceptable and entirely preventable public health hazard.

Definitions

The term, ETS, has been defined as the sum of sidestream smoke (SS) released from the burning tip of a cigarette and mainstream smoke (MS), which is exhaled by smokers. Each type of smoke is comprised of a particulate and a vapor phase. The physical and chemical characteristics of ETS are dynamic and differ significantly between MS and fresh SS. MS contributes 15% of total ETS whereas SS, a product of incomplete combustion,

E-mail address: jreardo@harthosp.org

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constitutes 85% [5]. Particles present in SS have one tenth the particle diameter of MS and, as such, have the potential to reach the most distal alveoli from where they cannot be expelled easily [6].

ETS also is referred to commonly as passive cigarette smoking, involuntary smoking, and secondhand smoking. ETS can be a major component of air pollution in indoor environments.

Composition of environmental tobacco smoke

More than 4000 compounds have been identified in tobacco smoke and of these, at least 60 are known or suspected carcinogens [7,8]. These chemicals comprise approximately 95% of MS weight. This complex mixture of chemical substances also has unique proinflammatory and cytotoxic effects [9]. Inorganic compounds, such as nickel, chromium, cadmium, arsenic, and hydrazine, are related to lung cancer whereas ammonia, nitrogen dioxide, sulfur dioxide, hydrogen cyanide, and acrolein are among the many irritant gases that can contribute to development of airways disease, such as COPD and asthma. In addition, other cancers can be caused by compounds, such as benzene, urethane, vinyl chloride, and aniline [6]. The tobacco-related carcinogens also are associated with a decreased capacity for DNA repair, which is associated with an increased risk for non–small cell lung cancer [10]. Yields per cigarette of some carcinogens have been reported to be greater in SS than with MS. For example, the release of volatile N-nitrosamines and aromatic amines is higher in SS than in MS. A major reason that undiluted SS and MS have different concentrations of toxic and carcinogenic agents is that peak temperatures in the burning cone of a cigarette reach 800°C to 900°C during puffing but only 600°C between puffs, resulting in less complete combustion of tobacco during generation of SS [11]. Table 1 lists many of the toxic and carcinogenic agents identified in SS and MS. Individual chemicals remain relatively constant in different commercial brands, including filter and nonfilter brands of cigarettes [12].

Assessing exposure to environmental tobacco smoke

Overall, the extent of adverse health effects resulting from ETS exposure is related to two major factors: the duration and intensity of exposure and individual susceptibility, believed to be genetically controlled [13].

The determination of an individual's exposure to ETS is a complex task, given the multiple variables involved. The number of cigarettes smoked by people in an environment, the length of time over which smoking occurs, the ventilation properties of a building, and the absorptive qualities of a structure, furniture, carpets, and curtains all contribute to the final exposure assessment [14]. Two approaches can be used to estimate ETS exposure. In the first, data on the smoking habits of the people in an environment in which individuals spend time are collected by questionnaire. This may be data from home, workplace, or other environments in which individuals spend time. Questionnaires can be informative in determining exposure to ETS; however, the lack of standardized, validated tools and the misclassification of exposure (inaccurate recall or inability to estimate accurately) must be considered. The second approach involves quantification of ETS components or their metabolites, either in the environmental air or in individual serum, saliva, urine, or hair. Various biomarkers of ETS (nicotine and its metabolite cotinine) have been developed that can be used to validate questionnaire responses and are useful in assessing recent exposure. Although nicotine has a short half-life (less than 2 hours) cotinine has a 3 to 4 day half-life and can be measured in urine, saliva, blood, and hair [15].

Exposure prevalence

Several studies have estimated the prevalence of ETS exposure [13,16]. Reported prevalence rates of exposure vary between 30% and 80% [17] for adults, with many studies showing the workplace as a major source of exposure. Increased regulation on smoking in the workplace and in most public places in recent years has made the home the leading unregulated source of ETS. This has significant potential impact on preschool-aged children who spend proportionately more time in the home [16]. One source estimates that approximately 43% of children in the United States live with a smoker, whereas in the United Kingdom, 40% to 60% of children are exposed to ETS in the home [18]. Studies of the potential effects of ETS on fetal and child health have multiplied over the past 20 years and an increased risk for multiple disease states has been demonstrated clearly (detailed later). Other areas needing further regulation include several