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Passive smoking

Introduction

Key points

- Environmental tobacco smoke is classed as a human carcinogen by the World Health Organization. There are no safe levels of exposure.
- Children exposed to second-hand smoke are at an increased risk of sudden infant death syndrome, acute respiratory infections, ear problems and more severe asthma. Parental smoking causes respiratory symptoms and slows lung growth in children.
- Smoke-free legislation results in significantly reduced respiratory symptoms and improved lung function.

Passive smoking is: 1) exposure to second- or third-hand smoke by breathing ambient air containing toxic substances resulting from the combustion of tobacco products after birth; or 2) exposure *in utero* to maternal blood contaminated with the combustion of tobacco smoking products.

Second-hand smoke (SHS) or environmental tobacco smoke (ETS) is the name given to the mixture of 'mainstream smoke' (smoke exhaled by a cigarette smoker), 'sidestream smoke' (emitted from the smouldering tobacco stick between puffs), contaminants emitted into the air during the puff and contaminants that diffuse through the cigarette paper and mouth-end between puffs. It is a complex mixture of gases and some 4000 particulate chemicals, which are generated during the burning and smoking of tobacco products. Of these, ≥250 are known to be toxic or carcinogenic.

The majority of the compounds present in mainstream smoke are formed during combustion. The constituents of mainstream and sidestream smoke are broadly similar but there are important differences in their rates of emission into the air due to physical and chemical differences in the way cigarettes burn during and between puffs. Mainstream smoke is generated during inhalation at a temperature of approximately 800–900°C, which is higher than that seen in sidestream smoke generation (600°C), due to increased levels of oxygen passing through the cigarette. Mainstream smoke also has a pH of 6.0–6.7, making it more

> *Separating smokers from nonsmokers, cleaning the air and ventilating buildings cannot prevent nonsmokers being exposed to second-hand smoke*

acidic than sidestream smoke (pH 6.7–7.5). The combustion process that generates sidestream smoke also produces smoke with much smaller particulate matter (PM) size and considerably higher concentrations of many carcinogens and toxins. However, dilution, chemical reactions, deposition and other removal processes may decrease the concentration of airborne SHS constituents, alter the size distribution of suspended particles and chemically modify some of the more reactive constituents of SHS (table 1). Third-hand smoke is the name given to substances that are re-emitted from solid surfaces having been initially deposited there during smoking.

Harmful effects of SHS on health

Many US government reports, going back to the 1972 Surgeon General's report 'The Health Consequences of Smoking', have discussed the harmful effects of SHS. The National Research Council and the Environmental Protection Agency (EPA) have also independently assessed the health effects of exposure to ETS (figure 1).

The EPA report, published in 1992, confirmed that the respiratory effects of ETS included:

- Lung cancer in nonsmoking adults. Passive smoking is causally associated with lung cancer in adults, and thus ETS, given the weight of evidence, belongs in the category of compounds classified by the EPA as Group A (known human) carcinogens.
- Non-cancer respiratory diseases and disorders.
  - Exposure of children to ETS from parental smoking is causally associated

Constituent	Emissions in SS per cigarette	Amount in SHS per m^3
Benzene	163–353 µg	4.2–63.7 µg
Benzo[a]pyrene	45–103 ng	0.37–1.7 ng
NNK	201–1440 ng	0.2–29.3 ng
4-Aminobiphenyl	11.4–18.8 ng	
2-Naphthylamine	63.1–128 ng	
1,3-Butadiene	98–205 µg	0.3–40 µg
Formaldehyde	233–485 µg	143 µg

Table 1 – Carcinogens in sidestream smoke (SS) and second-hand smoke (SHS) from cigarettes. NNK: 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone.

with: increased prevalence of respiratory symptoms of irritation (cough, sputum, and wheeze); increased prevalence of middle ear effusion (a sign of middle ear disease); and a small but statistically significant reduction in lung function as tested by objective measures of lung capacity.
  - xposure to ETS is causally associated with additional episodes and increased severity of asthma in children who already have the disease.
  - The epidemiological evidence is suggestive but not conclusive that ETS exposure increases the number of new cases of asthma in children who have not previously shown symptoms. Based on this evidence and the known effects of ETS on the immune system and lungs (*e.g.* atopy and airway hyperresponsiveness) the report concluded that ETS is a risk factor for the induction of asthma in previously asymptomatic children. Data suggest that relatively high levels of exposure are required to induce new cases of asthma in children.
- Subtle but significant effects of passive smoking on the respiratory health of nonsmoking adults, including coughing, phlegm production, chest discomfort and reduced lung function.

"Passive smoking is causally associated with lung cancer in adults"

At the time that the early reports were published, there was some uncertainty about the relationship of SHS to sudden infant death syndrome (SIDS), upper respiratory tract infections and middle ear infections in children. However, by 2006, when the US Surgeon General published 'The Health Consequences of Involuntary Exposure to Tobacco Smoke', it was possible to state clearly that:

- SHS causes premature death and disease in children and in adults who do not smoke.
- Children exposed to SHS are at an increased risk of SIDS, acute respiratory infections, ear problems and more severe asthma. Smoking by parents causes respiratory symptoms and slows lung growth in their children.
- SHS has immediate adverse effects on the cardiovascular system of adults and causes coronary heart disease and lung cancer.
- The scientific evidence indicates that there is no risk-free level of exposure to SHS.
- Many millions of Americans, both children and adults, are still exposed to SHS in their homes and workplaces despite substantial progress in tobacco control.
- Eliminating smoking in indoor spaces fully protects nonsmokers from exposure to SHS. Separating smokers from nonsmokers, cleaning the air and ventilating buildings, however, cannot prevent nonsmokers being exposed to SHS.

Estimates of burden of disease due to SHS

Europe

Two major reports about the burden of disease due to SHS have been published in the past 10 years in Europe: the 'Analysis of the Science and Policy in Europe for the Control of Tobacco' (ASPECT) report in 2005; and 'Lifting the Smokescreen: 10 Reasons for a Smoke Free Europe' by the Smoke Free Partnership (SFP) in 2006. Both dealt with the extent of SHS health effects in European countries and with economic considerations. The SFP report estimated that 79 449 adults died because of SHS exposure in the European Union in 2002 and also estimated mortality for each separate member state. In March 2010, the Royal College of Physicians issued a report entitled 'Passive Smoking and Children'. It focused on the UK but also presented evidence from the world literature on the harmful effects of passive smoking on fetal and reproductive health. The report's authors estimated that in the UK, SHS causes 121 400 new cases of middle ear disease, 20 500 new lower respiratory tract infections and 22 600 new cases of wheeze, as well as 40 cases of SIDS each year.

USA

According to the 1992 EPA report, ETS is estimated to cause around 3000 lung cancer deaths per year among nonsmokers (never-smokers and former smokers) of both sexes in the USA. While there are statistical and modelling uncertainties in this estimate, and the true number may be higher or lower, the overall confidence in this estimate is medium to high, and the assumptions used when calculating it would tend to underestimate the actual population risk.

The ETS exposure of young children and particularly infants from parental (and especially maternal) smoking is causally associated with an increased risk of lower respiratory tract infections (pneumonia, bronchitis, and bronchiolitis). The EPA report estimated, with high confidence, that exposure to ETS in the USA causes 150 000–300 000 of these infections annually in infants and children less than 18 months old,

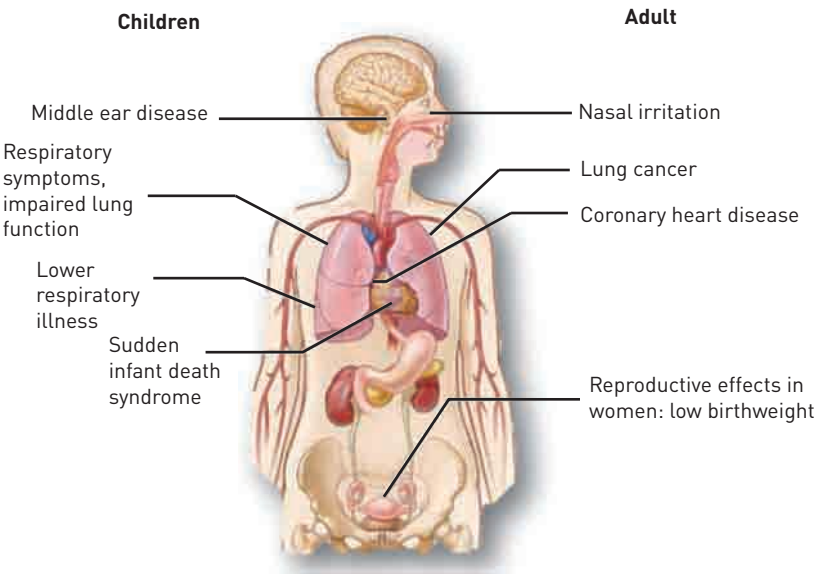

Figure 1. The dangers of second-hand smoke exposure. Reproduced from the US Surgeon General's report 'How Tobacco Smoke Causes Disease', 2010.

resulting in 7500–15 000 hospital admissions. Children up to 3 years of age were at increased risk of lower respiratory tract infections, but no estimated figures were derived for children over 18 months old.

The report also estimated that ETS exposure exacerbates symptoms in approximately 20% of the 2–5 million asthmatic children in the USA, and is a major aggravating factor in approximately 10%.

Measurement of SHS

The most direct and widely used method to measure ETS exposure is personal monitoring of respirable suspended particles and nicotine. Respirable suspended particles and nicotine give a good indication of cumulative exposure over a relatively short period of time. However, they have considerable limitations because of duration of measurements, representativeness of activity and cost.

Particles

Particulate matter, or PM, is the term given to the tiny particles of solid or semi-solid material found in the atmosphere. It consists of varying combinations of dry solid fragments, solid cores with liquid coatings and small droplets of liquid. In the case of airborne particles, the initial sizes of particles produced by cigarette smoking have been quoted to be 0.3–1.0 μm. This figure is not universally accepted, however: sidestream smoke particle size has been said to be typically 0.01–1.0 μm, with mainstream smoke particle size ranging from 0.1–1.0 μm.

Photometers, optical particle counters (OPCs) and condensation particle counters (CPCs) measure airborne particles in real time. Each technology has a unique sensitivity to specific particle characteristics such as size, mass and refractive index.

The instruments most commonly used for PM measurements are the Met One Aerocet 531 aerosol particulate profiler (Met One Instruments) and the TSI SidePak AM510 condensation particle profiler (TSI).

Nicotine

Nicotine vapour can be collected on filters by passive samplers and analysed using gas chromatography/mass spectrometry (GC/MS). Concentrations as low as 0.01 $\mu g \cdot mL^{-1}$ can be detected.

Biomarkers

Biomarkers are also useful for determining exposure to ETS because they enable us to predict potential health risks for exposed individuals, increasing our understanding of tobacco-related cancer mechanisms. Biomarkers offer a way to avoid many sources of bias or inaccurate reporting by study participants.

The most specific markers of exposure to SHS are thiocyanate and nicotine (in saliva, plasma or urine), and cotinine, a nicotine metabolite (in saliva, plasma, urine or hair). Cotinine is currently considered the marker of choice as thiocyanate is also influenced by diet, whereas cotinine appears to be the most specific and sensitive biomarker for smokers and nonsmokers as it reflects exposure to nicotine, which is almost wholly specific to tobacco.

Carboxyhaemoglobin in blood and carbon monoxide in exhaled air are also relatively easy-to-measure markers to quantify tobacco exposure, but they are not specific – road traffic or domestic emissions can affect them. Other substances that can be measured with more difficulty and variable specificity include: adducts of 4-aminobiphenyl to haemoglobin in red blood cells; adducts of benzo[a]pyrene to DNA in white blood cells; adducts of polycyclic aromatic hydrocarbons (PAHs) to plasma albumin; nicotine-derived nitrosamines in urine; hydroxyproline in urine; and *n*-nitrosoproline in urine.

Health benefits of smoke-free legislation

Respiratory health benefits

There is considerable experience and knowledge of the beneficial effects of comprehensive smoke-free legislation. Early studies from the USA showed that there were immediate respiratory health benefits in bar workers. The introduction of the Irish smoke-free legislation, and subsequently the Scottish and other national legislation,

has created an opportunity to measure health benefits at the level of individuals and populations. Various studies have confirmed the US results, showing significantly reduced respiratory symptoms and improved spirometry. In Irish studies, this was accompanied by a reduction in cotinine and exhaled breath carbon monoxide. The Irish research also found a significant improvement in gas exchange in the lung. These significant effects were seen in nonsmokers and ex-smokers but did not reach statistical significance in current smokers. It is also important to note that the subjects were not patients: they were in full-time employment with normal pulmonary function, yet they saw significant health benefits 1 year after the ban.

> *"Although children may benefit from smoke-free legislation in general, the laws do not prevent exposure* in utero, *in the home or in private vehicles"*

Cardiovascular health benefits

Studies on patients in the USA, Italy and Scotland have shown definite reductions in acute myocardial infarctions, varying from 17% in Scotland to 11% in Italy. Similar effects were seen in a small Irish regional study. More recently, an English study has shown a smaller, but nonetheless definite, reduction of 2.5%.

Other benefits

It is expected that the effects of smoke-free policies on lung cancer rates will take some time to be seen and reliably analysed.

There have been several recent reports of the beneficial effects of Irish and Scottish smoke-free legislation in pregnancy and in children.

Protection from exposure to SHS

Exposure to SHS is almost entirely preventable. Through Article 8 of the Framework Convention for Tobacco Control (FCTC) and a Council Recommendation, respectively, the World Health Organization (WHO) and the European Commission have declared that people have the right to be protected from SHS in public and indicated how this can be achieved. They have pointed out that they recognise the harmful effects of SHS, that comprehensive legislation is needed to prevent harm and that since there is no safe level of exposure (SHS is a Group 1 carcinogen according to the WHO), mechanical ventilation solutions are unacceptable. Many countries have now enacted comprehensive legislation since Ireland introduced its smoke-free law in March 2004. These countries include Norway, the UK, Sweden, Finland, Slovenia, Lithuania, Bulgaria and Turkey. Italy and France have laws that, in practice, are almost equivalent to comprehensive smoke-free legislation. Spain, Portugal and Greece initially introduced inadequate laws that did not meet the FCTC standard; Greece (2010) and Spain (2011) have already amended their laws, having shown that partial bans did not work.

Current smoke-free laws protect adults in the workplace and entertainment venues. Although children may benefit from smoke-free legislation in general, the laws do not prevent exposure *in utero*, in the home or in private vehicles. Legislation to protect children is needed and would be feasible in private vehicles, where we know levels of toxins can be very high in the presence of smoking, and that this causes disease in exposed children. Playgrounds in public parks are also increasingly smoke-free in Ireland and in the USA. Targeted smoking cessation services to help pregnant women stop smoking are also needed. Legislation to achieve smoke-free private housing may not be feasible or considered appropriate at present, but education and strong encouragement and advice should be offered. Legislation in municipal housing and in apartment blocks may well become commonplace if voluntary approaches fail.

In conclusion, SHS causes death and disability. It can be prevented, and health benefits due to the introduction of smoke-free legislation in children and adults have been widely reported.

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