Protecting our children from environmental tobacco smoke: one of our great healthcare challenges

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This editorial refers to ‘Exposure to parental smoking in childhood or adolescence is associated with increased carotid intima-media thickness in young adults: evidence from the Cardiovascular Risk in Young Finns Study and the Childhood Determinants of Adult Health study’†, by S. Gall et al., on page 2484.

This year marks the 50th anniversary of the US Surgeon General’s first report on Smoking and Health. This landmark report in 1964 concluded that cigarette smoking is harmful and causes lung and laryngeal cancer. In 1986, the Surgeon General released a further report on the Health Consequences of Involuntary Smoking, providing conclusive evidence that environmental tobacco smoke (ETS) exposure, or passive smoking, causes lung cancer.

Half a century later and despite major public health initiatives on tobacco control, tobacco use remains a leading cause of preventable death worldwide. It is estimated that about one-third of adults are regularly exposed to ETS globally.1 Within the European Union (EU), ~14% of non-smokers are exposed to ETS at home, and 30% of working adults are potentially exposed to passive smoking at the workplace.2 Astonishingly, as many as 700 million children worldwide are also exposed to ETS, mostly at home.3 Furthermore, those who are most likely to smoke are between the ages of 20 and 45, a period that coincides with parenthood.4 Thus, ETS may represent perhaps the most ubiquitous, hazardous, and potentially preventable adverse environmental exposure for children.

Environmental tobacco smoke is composed of sidestream smoke (emitted from the combusting end of a cigarette) and mainstream smoke exhaled by the smoker, with side stream being the main contributor. At least 4000 chemicals have been identified in sidestream smoke, of which 250 are known to be harmful, including 40–60 carcinogenic compounds.5 Sidestream smoke is qualitatively similar to mainstream smoke but, as it is generated at lower burning temperatures and is unfiltered, the quantity of its chemical constituents is different. Sidestream smoke contains higher quantities of certain carcinogens (e.g. aromatic amines)6 and it is thought to be 3–4 times more toxic than mainstream smoke per gram of particulate matter. Although sidestream smoke is diluted by room air prior to inhalation, there appears to be no safe level of ETS exposure.

In adulthood, an unequivocal causal relationship between ETS and coronary artery disease has been established, conferring a pooled relative risk increase of 25–30% from meta-analyses.7 There is also persuasive (although less conclusive) evidence that ETS is causally linked to other vascular disease such as stroke,8 supported by numerous studies which have consistently demonstrated an association between passive smoking and changes in arterial structure and function, using measurements of carotid intima-media thickness (IMT) and brachial flow-mediated dilatation. Putative mechanisms linking ETS with arterial disease include endothelial dysfunction and injury due to oxidative stress, platelet activation and stimulation of the coagulation cascade, enhancement of inflammation, and activation of matrix metalloproteases in atherosclerotic plaques. In children, ETS exposure is associated with more frequent respiratory tract illnesses, decreased lung function, and middle ear disease.9 In utero, exposure from maternal passive smoking leads to an increased risk of sudden infant death syndrome10 and reduced birth weights.11

It has long been recognized that atherosclerosis begins in childhood, and the study by Gall et al.12 sheds further light that childhood exposure to cardiovascular insults can lead to long-term changes in arterial structure in adulthood. Pooling data from two large and well-conducted longitudinal cohort studies [Cardiovascular Risk in Young Finns Study and Childhood Determinants of Adults Health (CDAH) Study], the authors demonstrate that childhood exposure to smoking by both parents (compared with no exposure) is associated with a thicker carotid IMT in adulthood. Carotid IMT is a well-characterized non-invasive marker of atherosclerosis, correlating with burden of coronary atheroma and predictive of future cardiovascular event rates. This difference in carotid IMT was independent of traditional vascular risk factors and smoking status at follow-up. Interestingly, no difference in carotid IMT was found if only one...
Table 1  World Health Organization MPOWER measures to combat tobacco use

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<tr>
<th>Measure</th>
<th>Description</th>
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<tr>
<td>Monitor tobacco use and prevention policy</td>
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<tr>
<td>Protect people from second-hand smoke</td>
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<tr>
<td>Offer help to quite tobacco use</td>
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<tr>
<td>Warn about the dangers of tobacco</td>
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<tr>
<td>Enforce bans on tobacco advertising, promotion, and sponsorship</td>
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<td>Raise taxes on tobacco</td>
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The strength of this study lies in its relatively large size, long duration of follow-up, and consistent finding of greater carotid IMT in two independent cohorts. However, parental smoking was assessed using a binary questionnaire (yes or no), and objective measures of ETS exposure (such as serum cotinine levels) were not available. Furthermore, ETS exposure during childhood was assessed at a single time point in the CDAH Study, and at two time points (3 years apart) in the Young Finns Study. Thus, the cumulative dose–response effect of ETS exposure during childhood on adult vascular health could not be easily assessed.

The adjusted difference in mean maximum carotid IMT between those exposed to both parents smoking and no exposure was 0.015 mm. What does this effect size mean? The authors calculate that this translates to 3.3 additional years of vascular ageing. Similar effect sizes have been noted in trials concerning vascular risk factor reduction. For example, the Diabetes Controls and Complications Trial (DCCT), a randomized study of intensive vs. conventional glucose control in adult type 1 diabetics, demonstrated a between-group difference of 0.013 mm in carotid IMT at 6 years after the end of the trial, favouring the intensive therapy group. In the METEOR trial, a randomized controlled study of rosuvastatin in patients with a low Framingham risk score but subclinical atherosclerosis, the statin-related change in carotid IMT was 0.014 mm/year better than the placebo group.

So how are we to protect our children from the deleterious effects of ETS exposure? In 2008, the World Health Organization (WHO) introduced MPOWER, an evidence-based intervention outlining six tobacco control measures proven to reduce tobacco use (Table 1). Currently, 2.3 billion people in 92 countries are covered by at least one MPOWER tobacco control measure. Turkey is the only country to implement all MPOWER measures, and this has led to a 13% decline in smoking prevalence in a short period of time between 2008 and 2012. Much more work needs to be done to control the tobacco epidemic and it is up to legislators backed by the support of clinicians, scientists, and advocates to end the tobacco epidemic in the 21st century. Today, this is one of our greatest healthcare priorities.

Conflict of interest: none declared.

References

12. Gall SI, Huynh QL, Magnusson CG, Juonala M, Viikari JSA, Kähönen M, Dwyer TM, Raitakari OT, Venn A. Exposure to parental smoking in childhood or adolescence is associated with increased carotid intima-media thickness in young adults: evidence from the Cardiovascular Risk in Young Finns Study and the Childhood Determinants of Adult Health Study. Eur Heart J 2014; 35:2484–2491.