EPA STRIVE Programme 2007-2013

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STRIVE Report

Prepared for the Environmental Protection Agency

By

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The EPA STRIVE programme addresses the need for research in Ireland to inform policymakers and other stakeholders on a range of questions in relation to environmental protection. These reports are intended as contributions to the necessary debate on the projection of the environment.

EPA STRIVE PROGRAMME 2007-2013
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<table>
<thead>
<tr>
<th>Symbol</th>
<th>Description</th>
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<tbody>
<tr>
<td>µg/m³</td>
<td>Micrograms per meter cubed</td>
</tr>
<tr>
<td>ALRI</td>
<td>Acute lower respiratory infections</td>
</tr>
<tr>
<td>CAFE</td>
<td>Clean Air for Europe</td>
</tr>
<tr>
<td>CHD</td>
<td>Coronary heart disease</td>
</tr>
<tr>
<td>CI</td>
<td>Confidence Interval</td>
</tr>
<tr>
<td>CO</td>
<td>Carbon monoxide</td>
</tr>
<tr>
<td>CO₂</td>
<td>Carbon dioxide</td>
</tr>
<tr>
<td>COMEAP</td>
<td>Committee on the Medical Effects of Air Pollutants</td>
</tr>
<tr>
<td>COPD</td>
<td>Chronic obstructive pulmonary disease</td>
</tr>
<tr>
<td>CRF</td>
<td>Concentration-Response Functions</td>
</tr>
<tr>
<td>EHIA</td>
<td>Environmental health impact assessment</td>
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<tr>
<td>E-R</td>
<td>Exposure-response</td>
</tr>
<tr>
<td>EPA</td>
<td>Environmental Protection Agency</td>
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<tr>
<td>ERFs</td>
<td>Exposure-response functions</td>
</tr>
<tr>
<td>ETS</td>
<td>Environmental tobacco smoke</td>
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<tr>
<td>EU/m³</td>
<td>Endotoxin unit per meter cubed</td>
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<tr>
<td>ExternE</td>
<td>Externalities of Energy</td>
</tr>
<tr>
<td>GBD</td>
<td>Global burden of disease</td>
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<tr>
<td>GM</td>
<td>Geometric mean</td>
</tr>
<tr>
<td>HEIMTSA</td>
<td>Health and Environment Integrated Methodology and Toolbox for Scenario Assessment</td>
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<tr>
<td>HIA</td>
<td>Health Impact Assessment</td>
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<tr>
<td>IAP</td>
<td>Indoor Air Pollution</td>
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<tr>
<td>IAPAH</td>
<td>Indoor Air Pollution and Health</td>
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<tr>
<td>IAQ</td>
<td>Indoor air quality</td>
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<tr>
<td>IOM</td>
<td>Institute of Occupational Medicine</td>
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<tr>
<td>LAL</td>
<td>Limulus Amebocyte Lysate</td>
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<tr>
<td>INTARESE</td>
<td>Integrated Assessment of Health Risks of Environmental Stressors in Europe</td>
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<tr>
<td>LPG</td>
<td>Liquefied petroleum gas</td>
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<tr>
<td>LRIs</td>
<td>Lower respiratory illnesses</td>
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<td>n</td>
<td>Number</td>
</tr>
<tr>
<td>NO₂</td>
<td>Nitrogen dioxide</td>
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<tr>
<td>OR</td>
<td>Odds Ratio</td>
</tr>
<tr>
<td>PAHs</td>
<td>Polycyclic aromatic hydrocarbons</td>
</tr>
<tr>
<td>PM</td>
<td>Particulate Matter</td>
</tr>
<tr>
<td>PM₁₀</td>
<td>Particulate matter smaller than 10 microns</td>
</tr>
<tr>
<td>PM₂.₅</td>
<td>Particulate matter smaller than 2.5 microns</td>
</tr>
<tr>
<td>ppb</td>
<td>Parts per billion</td>
</tr>
<tr>
<td>ppm</td>
<td>Parts per million</td>
</tr>
<tr>
<td>RR</td>
<td>Relative risk</td>
</tr>
<tr>
<td>SCHER</td>
<td>Scientific Committee on Health and Environmental Risks</td>
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<td>SCOTH</td>
<td>Scientific Committee on Tobacco and Health</td>
</tr>
<tr>
<td>SFU</td>
<td>Solid fuel use</td>
</tr>
<tr>
<td>SHCS</td>
<td>Scottish House Condition Survey</td>
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<tr>
<td>SIDS</td>
<td>Sudden Infant Death Syndrome</td>
</tr>
<tr>
<td>TFC</td>
<td>Total fuel consumption</td>
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<td>WHO</td>
<td>World Health Organisation</td>
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Executive Summary

The objective of the Indoor Air Pollution and Health (IAPAH) research project was to quantify the levels of Indoor Air Pollution (IAP) in Irish and Scottish homes where open combustion takes place, and provide an estimate of the potential health burden due to exposure to combustion derived air pollution in the home. IAP concentrations were measured in 100 homes in Ireland and Scotland. Open combustion was defined as the use of the solid fuels; coal, wood, peat, for heating, gas for cooking or the presence of tobacco smoking. Twenty four hour data on airborne concentrations of particulate matter smaller than 2.5µm (PM$_{2.5}$), carbon monoxide (CO), carbon dioxide (CO$_2$) and endotoxin, together with 2-3 week averaged concentrations of nitrogen dioxide (NO$_2$) were collected. Concentrations of IAP in homes using coal, wood, peat and gas for cooking were low, and mostly well within health based standards, suggesting adequate ventilation, and well maintained combustion systems in the participating homes. PM$_{2.5}$ concentrations in homes using coal, wood and gas cooking using homes were comparable to outdoor ambient concentrations. Peat burning homes had PM$_{2.5}$ concentrations approximately twice that of ambient air, whereas smoker homes had PM$_{2.5}$ concentrations greater than ten times the level measured in coal, wood and gas cooking homes. The average 24-hour PM$_{2.5}$ concentrations in smoker homes are the main cause for concern in terms of IAP from combustion sources in the home.

The average 24-hour PM$_{2.5}$ concentrations was almost six times the World Health Organisation (WHO) 24-hour PM$_{2.5}$ guidance concentration value of 25 µg/m$^3$, and over 4 times the US Environmental Protection Agency (EPA) outdoor Air Quality index ‘unhealthy’ level for sensitive groups of 65 µg/m$^3$ guidance values, and two modified versions of the ‘full chain approach’ to Health Impact Assessment (HIA), the source based approach and the pollutant based approach, were used to estimate the health burden from solid fuel combustion and environmental tobacco smoke (ETS) in the home. The source based approach uses a simple binary exposure metric, which requires information on the proportion of the population exposed to the pollutant source, risk functions for health outcomes associated with the presence of the source, and background rates of disease in the unexposed population for the health endpoints of interest. The pollutant based approach uses a signature pollutant, in this case, PM$_{2.5}$, as a marker of the pollutant source of interest, and requires information on the exposure to PM$_{2.5}$, information on the population exposed, exposure response functions linking exposure with mortality and morbidity, and background rates of morbidity and mortality in the exposed population. Within IAPAH the source based approach was used to estimate the burden on health from exposure to ETS within the home. Two populations were considered, non- or never-smoking children (< 15 yr) and non- or never-smoking adults (< 25 yr) who live in a smoking household. The pollutant based approach was used to estimate the health burden attributable to burning solid fuels, using gas cooking and exposure to ETS in the homes. When estimating the health impact attributable to burning solid fuel and using gas cooking in the home two exposure scenarios were considered, exposure to the source from 6 pm until midnight, or for 24-hours. PM$_{2.5}$ data were adjusted for the contribution of other indoor and outdoor sources. This resulted in the homes using
gas cooking being considered as a control group for the other solid fuel homes. Concentrations of \( \text{PM}_{2.5} \) in coal and wood homes were low and so health impacts were not calculated. Concentrations of \( \text{PM}_{2.5} \) in homes using peat were slightly higher and health impacts were calculated for the exposed population in Ireland only.

Results from the health impact assessment indicate that exposure to ETS represents the greatest impact on health from combustion derived air pollution in the home. Both the source based approach and the pollutant based approach estimate cardiovascular events as the greatest health burden among adults and lower respiratory illness and respiratory symptoms among children who are exposed to ETS at home. Health burden estimates, calculated using the pollutant based approach are higher than those calculated using the source based approach.

The exposure of non-smokers to ETS in the home accounts for a health burden that is broadly comparable to that currently experienced in both countries from road traffic accidents and there is a real need for public health policy and research professionals to develop interventions to address this. We recommend that co-ordinated national campaigns to educate smokers and non-smokers about the health effects of ETS exposure in the home should be developed together with intervention tools to reduce smoking initiation and increase quitting. Research to identify methods that help those who continue to smoke to implement smoke-free homes is also required. In order to be able to evaluate future progress in reducing non-smokers’ exposure to ETS there is a need to have a question to determine population-wide exposure to ETS at home incorporated in existing national health survey campaigns in both countries.
1 Introduction

It is recognized that exposure to air pollutants found in the indoor environment plays a significant role in human health. In the developed world, a significant proportion of our time is spent indoors, (Klepeis et al, 2001), and vulnerable groups such as young children and the elderly can spend up to 100% of their time indoors (Bonnefoy et al, 2004). Exposure concentrations vary and depend on a number of factors including individuals' behaviour and activities, pollutant sources, geographical location.

Previous scientific work on air pollution has mainly focused on quantifying the health effects of outdoor air pollution, and much progress has been made at improving outdoor air quality and regulating sources of outdoor air pollution (European Commission, 2008). While indoor air pollution (IAP) in the workplace and in enclosed public places have been regulated, indoor air quality in domestic settings remains largely unregulated, and there has been little public health activity on targeting sources of IAP in the home. The lack of progress in this important area reflects the relative lack of research on IAP in homes and health.

In 2007 the Scientific Committee on Health and Environmental Risks (European Commission, 2008) identified a number of gaps in the scientific knowledge needed to provide a basis for a health based risk assessment strategy on indoor air quality (IAQ). Many of the gaps relate to the lack of specific information on source pollutant concentrations, exposure patterns and health effects of specific indoor air pollutants. There is no established methodology for Health Impact Assessment (HIA) of pollution from indoor sources. The main stumbling-block is the absence of a recognised set of exposure-response (E-R) relationships linking long-term exposure to indoor combustion sources with mortality and morbidity outcomes.

Exposure to IAP from biomass fuel combustion and environmental tobacco smoke (ETS) has been linked to the development or exacerbation of chronic respiratory illnesses such as asthma, allergies and chronic obstructive pulmonary disease (COPD), and cardiovascular disease (Fullerton et al, 2009; Kurmi et al, 2010). The prevalence of many of these diseases in Western Europe has increased in the past few decades (THADE, 2004). Ireland’s mortality rate from respiratory disease is over twice the EU average (Brennan et al, 2008), while both Ireland and the United Kingdom have particularly high prevalences of childhood allergy and asthma (ISAAC, 2007). While it would be wrong to presume that IAP was a major cause of these changes or differences, the fact of them increases the importance of understanding what role of IAP does play.

Sources of IAP in the home include ingress of outdoor air pollution, cooking emissions (both from fuel and food), tobacco smoke, cleaning and consumer product emissions and heating systems. A great number of studies have examined determinants of indoor air pollutants such as outdoor sources (Monn et al, 1997; Pekey et al, 2010), and smoking (Saraga et al, 2010; Larsson et al, 2004) however, few studies have investigated how the use of fossil fuel for cooking or heating in the home contribute to poor IAQ in European countries. Much work has been published on indoor air pollutants and the burning of solid or biomass fuels for heating and cooking in developing countries (Kurmi et al, 2010; Fullerton et al, 2009) however data from such studies are not easily extrapolated to more economically developed settings because of major differences in housing, ventilation, heating and cooking appliances, and fuels used.
Research on IAP from fuel use in homes in the developed world has tended to focus on homes that use wood (Levesque et al, 2001; Fine et al, 2002; Gustafson et al, 2008) or gas (Garcia-Algar et al, 2004) and few have studied homes using coal (Moriske et al, 1996; Henderson et al, 2006) or peat (Guo et al, 2008). Fuel for heating and cooking in most EU countries tends to be electricity or gas based with efficient stoves and heating devices with flues in most homes. In Ireland, the use of coal and peat as residential energy sources has declined in recent years but there is still a considerable proportion of homes using solid fuels. ‘Fuel poor’ homes are more likely to use solid fuels as opposed to other energy alternatives. Estimates of residential fuel use in Ireland, in 2006 (O’Leary, 2008), indicated that coal and peat accounted for 7.3% and 9.5% of the share of the total fuel consumption (TFC) in the residential sector. The use of natural gas as a residential energy source has increased, and now accounts for 21% of the share of TFC, and electricity and oil accounts for the greatest share of the TFC, 23% and 38% respectively. Although peat is still commonly used in the Highlands and Islands of Scotland, data from Scotland indicate that only about 1% of all homes use solid fuels for heating, and approximately 77% of households use mains gas as their primary heating fuel with a subset of this population having either gas cooking or gas fires in the main living spaces (Amabile at a., 2009). The recent drive for greater use of ‘renewable’ or ‘biomass’ fuels to reduce individuals’ carbon footprints and combat climate change has led to an upsurge in interest in domestic methods of producing power. It is projected that this may lead to an increase in the use of biomass fuels across both countries (O’Leary, 2008). The health consequences of this use are largely unexplored.

Upwards of 900 air pollutants have been identified in the indoor domestic environment. Agents such as nitrogen dioxide (NO₂), particulate matter (PM), carbon monoxide (CO) and polycyclic aromatic hydrocarbons (PAHs) are among some of the priority pollutants known to affect health (WHO, 2010). Studies on IAP from fuel use in the home show that elevated levels of PM, CO, NO₂ and PAHs are associated with the use of fuel or the presence of a smoker in the home. Certain pollutants are more dominant depending on the fuel type used; increased levels of NO₂ have been associated with the use of gas burning appliances (Dennekamp et al, 2001; Garcia-Algar et al, 2004) while elevated concentrations of NO₂ and CO are the principal pollutants associated with the use of wood-burning appliances (Naeher et al, 2007). Studies in smoker homes have shown elevated concentrations of endotoxin and PM₃₂.₅₁ (Larsson et al, 2004). Endotoxin is a biological component of fine PM, derived from the cell wall of gram-negative bacteria. Endotoxin is a potent mediator of airway inflammation and thought to play a role in the development of respiratory disease. Despite this, limited data exist on endotoxin levels in homes using wood, peat and coal (Thorne and Duchaine, 2007).

The complex relationship between human health and IAQ has been prioritized as an area requiring further research by the European Commission and by the World Health Organisation (WHO, 2011) (European Commission, 2011). This study has been carried out to provide data on the levels of IAP in Irish and Scottish homes where burning combustible material takes place and to provide an estimate of the potential health burden generated by the exposure of residents within these homes to these IAP concentrations. (Throughout this report, ‘Ireland’ means Republic of Ireland, unless otherwise stated.)

1.1 Study Details:

₁ Particulate matter smaller than 2.5µm, also referred to as ‘fine’ particles
An Environment and Health research project on Indoor Air Pollution and Health (IAPAH) commenced in December 2008. IAPAH is a collaborative research project with four partners, National University of Ireland, Galway; University of Aberdeen; Institute of Occupational Medicine (IOM), Edinburgh; and the University of Birmingham.

1.2 Research Project Objectives:

This study aims to measure indoor air pollutant levels in homes in Ireland and Scotland, estimate how many people are exposed to different sources and concentrations of key indoor air pollutants and use these data to generate an estimate of the size of health burden that is attributable to air pollution within homes. To achieve this we will draw on published material identifying concentration-response coefficients from outdoor air pollution literature and recent studies examining the relationship between biomass fuel smoke and health in the developing world.

Specific objectives of the IAPAH project include:

1. To provide systematic information on indoor air pollution concentrations in homes in Ireland and Scotland where solid fuels are used for heating (wood, peat, coal) or gas is used for cooking or where tobacco smoking is present;
2. Identify key reviews on long-term exposure to outdoor air pollution and summarise the potential for applying outdoor coefficients to derive indoor coefficients;
3. Determine the number and type of households where people are exposed to elevated IAP levels and the population profile within these homes;
4. Derive estimates of average annual exposures attributable to indoor sources;
5. Provide an estimate of the potential health burden across the population in both countries that arises as a result of poor IAQ from these combustion sources within homes.
2.0 Contribution of solid fuel or gas combustion or Environmental Tobacco Smoke to indoor air pollutant concentrations in Irish and Scottish homes

2.1 Introduction

The first element of the IAPAH project involved measuring a range of IAPs in a sample of Irish and Scottish homes which use solid fuels (coal, peat or wood) for heating, gas for cooking, or had a resident smoker who smoked inside the home. This section outlines the methodologies employed to recruit homes to participate in the project, and to conduct the subsequent air sampling. Summary results and conclusions are also provided. This element of the project has been submitted for publication (Semple et al., 2011).

2.2 Methodology

2.2.1 Recruitment and ethics

Ethical approval for the study was given by the local College Ethics Research Board of the University of Aberdeen, Scotland and by the Research Ethics Committee of the National University of Ireland, Galway. Participants provided informed consent and a consent form was signed by both participant and the researcher in all cases before sampling began.

Recruitment of households took place between October 2009 and March 2010 during the winter period when fuel use would be at a peak and when ventilation levels tend to be minimised. The study was publicised via the local press in Aberdeen, Scotland and Galway, Ireland together with a website (www.nuigalway.ie/iapah). Other participants were recruited via word of mouth and snowballing techniques utilizing those already recruited to the study. Our aim was to recruit 20 households that used peat as heating fuel, 20 households that used coal, 20 that used wood, 20 that used a gas stove to cook and 20 that had at least 1 adult resident smoker (with no other combustion source present e.g. electricity used for heating purposes). Households were to be recruited in and around the city of Aberdeen and Aberdeenshire in North-east Scotland and in and around Galway city in the West-coast of Ireland. Potential participants who expressed an interest in the study were screened for eligibility using a telephone questionnaire which asked questions about solid fuel use and smoking by residents in the home. Households were excluded if they reported burning more than one type of solid fuel/tobacco source within the home.

2.2.2 IAP measurement

Sampling instruments were placed in the main living area of each participating home and generally located in close proximity to each other at a height of about 1.0-1.5m. Where possible, devices were placed at a distance of at least 1.0m from windows, doors and the heating/cooking source under study. A total of five IAPs were measured - PM$_{2.5}$, airborne endotoxin within the total inhalable dust fraction, CO, CO$_2$ and NO$_2$. 
The sampling was performed between 1st October 2009 and 31st March 2010, with a small number of NO₂ tubes collected into April 2010.

TSI SidePak AM510 Personal Aerosol Monitors (TSI Inc., Shoreview, MN, USA) fitted with a PM₂.₅ impactor were used to collect and log real-time data in μg/m³ on airborne PM₂.₅ levels over a 24-hour period. A correction factor for combustion-generated PM₂.₅ of 0.3 was applied to the data derived from the Sidepak device (Repace, 2006). Telaire® 7001i Data loggers (Edinburgh Instruments Ltd, Livingston, UK) were used to log CO₂ levels in ppm with a data logging kit (H08-007-02 Hobo data logger Onset Computer Corporation, Bourne, MA, USA). Assessment of airborne endotoxin was carried out using total inhalable dust sampling following the UK Health and Safety Executive’s ‘Methods for the Determination of Hazardous Substances’ 14/3 (HSE, 2000). After sampling and appropriate storage at 4°C the filters were transported to the Pulmonary Toxicology Facility at the University of Iowa, USA for analysis using the kinetic chromogenic modification of the Limulus Amebocyte Lysate (LAL) assay. Average indoor NO₂ levels were measured over a period of 2-3 weeks using passive diffusion tubes (Gradko International, Winchester, UK). A single sample tube was placed in the main living area of each home away from windows and doors, at 1-1.5 metre height. Tubes were analysed at the Gradko International laboratory (Winchester, England). CO levels were measured and logged every minute over a 24-hour period using Lascar Easylogger EL-USB-CO (Lascar Electronics Ltd, Wiltshire, UK) data loggers.

A sampling box, large enough to accommodate the Sidepak and SKC pump, was constructed from cardboard/wood and padded with insulating material to minimise noise disturbance. The fitted lid was similarly padded. Two holes were cut in the front panel of the box to allow access for the power cables and Tygon tubing. The Sidepak and SKC pump were connected to mains electricity in each home to enable operation for at least 24 hours of sampling. The sampling arrangement is illustrated in Figure 1.

Figure 2.1. Sampling arrangement used in the homes. Photo shows a padded equipment box, the TSI Sidepak AM510 Aerosol monitor, placed inside the box, and its PM₂.₅ impactor head measuring PM attached outside (not visible in photo).

Legend: (from left) the Lascar Easylogger (1) logging CO, an IOM total inhalable dust collector (2), and the Telaire® 7001i Data logger logging CO₂ and relative humidity (%) (3)
2.2.3 Other data collected

Contextual information regarding fuel use, household and occupant activities was systematically collected using diaries and questionnaires. Outdoor temperatures for Scotland were obtained from the UK Met Office for Aberdeen and for Ireland, from Met Eireann for Galway. 24-hour outdoor PM$_{2.5}$ concentrations in Aberdeen and Galway were obtained from the UK Department for Environment and the Mace Head Atmospheric Research Station in County Galway, respectively.

2.2.4 IAP guidance values

Results were compared with WHO (WHO, 1999; WHO, 2010) and US EPA comparison guidelines for outdoor air (US-EPA, 2011). WHO guidelines (WHO, 1999) recommend a CO limit of 6.1 ppm (24-hour average) and a PM$_{2.5}$ limit of 25 µg/m$^3$. US EPA ambient air standards for NO$_2$ are 50 ppb (24-hour average). ASHRAE (1989) indoor air quality guidance suggests that CO$_2$ concentrations above 1000 ppm indicate poor ventilation. There are no standards for household endotoxin other than the Dutch Occupational guidance at 90 EU/m$^3$ (DECOS, 2010))

2.3 Results

2.3.1 Demographics of recruited homes

100 homes using solid fuels (coal, peat, or wood) for heating or gas for cooking, or with at least one resident smoker, were recruited from across Ireland (n=48) and Scotland (n=52) to participate in the study. Homes were located in both urban and rural areas. Table 2.1 shows a summary of the household characteristics of the homes sampled.

Table 2.2 provides summary statistics for the IAP concentrations measured in IAPAH study homes. The overall average PM$_{2.5}$ level found over the 24-hour monitoring period was 37 µg/m$^3$. Lower average levels were found in homes that burned coal (9 µg/m$^3$) or wood (8 µg/m$^3$) and in homes with gas cookers (9 µg/m$^3$). In peat-burning homes the average 24-hour PM$_{2.5}$ level was 16 µg/m$^3$. Much higher particulate concentrations were found in homes with resident smokers (143 µg/m$^3$). Across the 100 homes the average concentrations of CO$_2$ was 713 ppm. The average NO$_2$ concentration was 5 ppb and airborne levels averaged 5.7 EU/m$^3$.

For PM$_{2.5}$ we also calculated a 6-hour evening average from our real-time data. This 6pm-midnight period was derived to better reflect personal exposure of working adults who are likely to spend a proportion of the day outside the home. Over the 100 homes this 6-hour average was 50 µg/m$^3$, 11 µg/m$^3$ for wood, 13 µg/m$^3$ for coal , 12 µg/m$^3$ for gas-cooking homes, 29 µg/m$^3$ for peat and 197 µg/m$^3$ for smoking homes.
Table 2.1: Demographic and housing characteristics of sampled households

<table>
<thead>
<tr>
<th>Demographic/Housing Characteristic</th>
<th>All (n=100)</th>
<th>Scotland (n=52)</th>
<th>Ireland (n=48)</th>
<th>P-value (probability)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coal burning</td>
<td>22 (10)</td>
<td>10 (17)</td>
<td>12 (54%)</td>
<td>NS</td>
</tr>
<tr>
<td>Peat burning</td>
<td>20 (3)</td>
<td>17 (5)</td>
<td>5 (29%)</td>
<td>0.06</td>
</tr>
<tr>
<td>Wood burning</td>
<td>22 (9)</td>
<td>14 (6)</td>
<td>5 (48%)</td>
<td>NS</td>
</tr>
<tr>
<td>Gas cooking</td>
<td>16 (11)</td>
<td>11 (5)</td>
<td>5 (31%)</td>
<td>NS</td>
</tr>
<tr>
<td>Smoking</td>
<td>20 (9)</td>
<td>9 (4%)</td>
<td>11 (56%)</td>
<td>NS</td>
</tr>
<tr>
<td>Age of household giving consent (mean, years)</td>
<td>51</td>
<td>52</td>
<td>50</td>
<td>NS</td>
</tr>
<tr>
<td>Room volume (m³)</td>
<td>57 (93)</td>
<td>56 (88)</td>
<td>58 (98)</td>
<td>NS</td>
</tr>
<tr>
<td>Central heating (%)</td>
<td>93 (84%)</td>
<td>88 (84%)</td>
<td>98 (98%)</td>
<td>0.06</td>
</tr>
<tr>
<td>Type of house (n, %)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Detached</td>
<td>51 (51%)</td>
<td>25 (48%)</td>
<td>26 (54%)</td>
<td>NS</td>
</tr>
<tr>
<td>Semi-detached*</td>
<td>30 (30%)</td>
<td>16 (31%)</td>
<td>14 (29%)</td>
<td>0.09</td>
</tr>
<tr>
<td>Terraced*</td>
<td>8 (8%)</td>
<td>2 (4%)</td>
<td>6 (13%)</td>
<td>NS</td>
</tr>
<tr>
<td>Flat/apartment</td>
<td>11 (11%)</td>
<td>9 (17%)</td>
<td>2 (4%)</td>
<td>NS</td>
</tr>
<tr>
<td>Age of house (n, %)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre early 1980s</td>
<td>59 (59%)</td>
<td>42 (81%)</td>
<td>17 (35%)</td>
<td>NS</td>
</tr>
<tr>
<td>Post early 1980s</td>
<td>41 (41%)</td>
<td>10 (19%)</td>
<td>31 (65%)</td>
<td>0.00</td>
</tr>
<tr>
<td>Pets in household (%)</td>
<td>53%</td>
<td>54%</td>
<td>52%</td>
<td>NS</td>
</tr>
<tr>
<td>Outdoor temp (°C)</td>
<td>6.0 (8.2%)</td>
<td>5.8 (8.1%)</td>
<td>6.2 (8.1%)</td>
<td>NS</td>
</tr>
<tr>
<td>Outdoor PM₂.₅(µg/m³)</td>
<td>8.2 (8.2%)</td>
<td>8.2 (8.1%)</td>
<td>8.1 (8.1%)</td>
<td>NS</td>
</tr>
</tbody>
</table>

NS=Not significant
* Terraced houses= houses in a row of similar houses that share side-walls; Semi-detached houses= consists of pairs of houses built side by side as units sharing a wall.

Table 2.2. Average IAP concentrations measured in IAPAH study homes

<table>
<thead>
<tr>
<th>Pollutant time-weighted average mean values</th>
<th>All (n=100)</th>
<th>Coal (n=22)</th>
<th>Gas Cooking (n=16)</th>
<th>Peat (n=20)</th>
<th>Smoking (n=20)</th>
<th>Wood (n=22)</th>
</tr>
</thead>
<tbody>
<tr>
<td>†PM₂.₅ (µg/m³) (range)</td>
<td>36.8 (8.9)</td>
<td>8.6 (2.28)</td>
<td>15.6 (4.4)</td>
<td>143 (21-463)</td>
<td>7.7 (2-23)</td>
<td></td>
</tr>
<tr>
<td>†PM₂.₅ (µg/m³) (range)</td>
<td>50.2 (13.0)</td>
<td>12.2 (2.57)</td>
<td>29.1 (3.13)</td>
<td>197 (16-539)</td>
<td>10.8 (3-52)</td>
<td></td>
</tr>
<tr>
<td>†CO (ppm) (range)</td>
<td>0.04 (0.01)</td>
<td>0.04 (0.03)</td>
<td>0.04 (0.01)</td>
<td>0.22 (0.14)</td>
<td>0.00 (0.0003)</td>
<td></td>
</tr>
<tr>
<td>†CO₂ (ppm) (range)</td>
<td>713 (642)</td>
<td>687 (450-1171)</td>
<td>713 (490-1097)</td>
<td>818 (469-1290)</td>
<td>708 (520-1540)</td>
<td></td>
</tr>
<tr>
<td>†NO₂ (ppb) (range)</td>
<td>5.12 (4.03)</td>
<td>9.01 (2.11-24.1)</td>
<td>3.99 (1.08-15.8)</td>
<td>6.82 (2.2-13.6)</td>
<td>2.87 (1.05-6.2)</td>
<td></td>
</tr>
<tr>
<td>†Airborne endotoxin (EU/m³) (range)</td>
<td>5.69 (5.78)</td>
<td>3.09 (0.72-6.9)</td>
<td>5.12 (0.12-24.7)</td>
<td>5.38 (0.92-21.7)</td>
<td>7.63 (0.12-16.6)</td>
<td></td>
</tr>
</tbody>
</table>

† 24-hour sampling period
* 6 hour time-weighted average from 6pm-midnight
‡ two week sampling period
Figure 2.2 illustrates the range of 24-hour PM$_{2.5}$ concentrations measured in each fuel-burning or smoking home in both Scotland and Ireland. The horizontal line is the WHO 24-hour guidance value for PM$_{2.5}$ exposure (25 $\mu$g/m$^3$).

**Figure 2.2: 24-hour PM$_{2.5}$ concentrations by fuel type and country.**

### 2.3.2 Real-time data

In each participating household minute-by-minute data over a 24-hour period were collected for PM$_{2.5}$, CO$_2$ and CO concentrations, temperature and relative humidity. Figure 2.3 illustrates the time-course of changing PM$_{2.5}$ levels in one particular smoking household. Peaks represent periods of active smoking within this home with a clear build-up of PM$_{2.5}$ concentrations occurring between approximately 8pm and 1.30am before levels then decrease once the house occupants go to sleep.
2.4 Discussion

2.4.1. Strengths and limitations of the study

Our study characterizes a range of indoor air pollutant concentrations in homes where open combustion takes place. It has a relatively large sample size with 100 homes sampled across two neighbouring countries in Northern Europe. By the nature of our selection process to include homes with a single fuel type we have been able to examine if there are differences in IAP concentrations between different combustion materials. This is a particular strength of this study.

We acknowledge that it is difficult to be certain of the representativeness of the study sample. No data exist on the type and demographics of solid fuel burning or tobacco using homes in Scotland and Ireland so we are unable to compare our recruited group with the overall population from which they were sampled. The lack of a control group is a weakness of the study although, as explained later, homes with gas cooking became in effect a control group for other sources of indoor exposure.

Sampling was not carried out simultaneously in all homes due to the limited amount of equipment we had and so there are likely to have been temporal variations in outdoor pollutant concentrations over the 6-month measurement program. This will have resulted in different contributions to indoor pollutant levels from outdoor pollutants on different days. We believe this effect would have been small and, although we
only have data for outdoor PM$_{2.5}$ levels in the Aberdeen and Galway areas, we note that the inter-quartile range for outdoor PM$_{2.5}$ was $<10 \, \mu g/m^3$ in Aberdeen and $<5 \, \mu g/m^3$ in Galway.

As with all observational studies and exposure measurement it is possible that the act of measurement has influence the parameters under study. The possibility of modification of behaviour in heating, cooking, ventilation and smoking is possible. Our data analysis protocols did remove the first 20 minutes of collected real time data in order to remove the period when the researcher was in the house setting up the instrument and collecting questionnaire data. A similar procedure was used to remove the final 20 minute period of the 24-hour data.

2.4.2. Concentrations found and potential for adverse health effects

The main finding of this part of the study is that homes using solid fuels in open combustion processes have low concentrations of the main IAP measured whereas high concentrations were found in homes with a resident smoker who smoked indoors. Concentrations of CO, NO$_2$ and airborne endotoxin were well within health-based standards in all homes using solid fuels, or gas for cooking, where measurements took place. These generally positive findings for sources other than smoking suggest well-maintained combustion apparatus and generally good control of IAPs in homes burning solid fuel in Scotland and Ireland. PM$_{2.5}$ concentrations were generally similar to outdoor ambient air levels in coal, wood and gas-cooking homes, about twice the outdoor concentration in peat-burning homes and were highest in smoking homes. 24-hour average concentrations were found to exceed the WHO 24-hour guidance level of 25 $\mu g/m^3$ in one-quarter (n=25) of homes although most (n=19) of those homes exceeding this value were smoking homes. Twelve of the smoking homes (60%) had 24-hour PM$_{2.5}$ concentrations that exceeded the 24-hour US EPA 65 $\mu g/m^3$ threshold deemed to be unhealthy. The PM$_{2.5}$ data in particular shows that mean 24-hour average levels in smoking homes was 15 to 20 times higher than those measured in the solid-fuel or gas burning homes. The mean 24-hour level in smoking homes of 143 $\mu g/m^3$ is over 4 times the US EPA outdoor Air Quality Index ‘unhealthy’ level for sensitive groups (35 $\mu g/m^3$) (US EPA, 2011) and approaching six times the WHO 24-hour guidance concentration of 25 $\mu g/m^3$ (WHO, 2010). 24-hour fine particulate matter levels were broadly similar to those found in outside air in coal, wood and gas burning homes. Peat burning homes had particulate concentrations levels that were closer to the WHO annual guidance level for PM$_{2.5}$.

The main cause for concern in terms of IAP from combustion in homes in Scotland and Ireland is from smoking activity. PM$_{2.5}$ concentrations in homes with a resident smoker are, in general, an order of magnitude higher than those found in homes burning coal, wood, peat or gas. The 24-hour PM$_{2.5}$ concentrations in the homes where tobacco smoking took place are considerable and the average value of 143 $\mu g/m^3$ can be compared to similar measurements made in a range of public space environments where smoking takes place. The average of PM$_{2.5}$ measurements in 106 bars across the UK prior to the introduction of smoke-free restrictions was 200 $\mu g/m^3$ (Semple et al., 2010) while a recent study of PM$_{2.5}$ concentrations in 66 US casinos where smoking is permitted reported a geometric mean value of 54 $\mu g/m^3$ (Repace et al., 2011). We acknowledge that the smoking homes included in this study may not be representative of all smoking homes in Scotland and Ireland. They may be unintentionally biased having
poorer ventilation, less household smoking restrictions and higher consumption of cigarettes than average smoking households. Based on other work done by our group (not presented here) we have applied a scaling factor of approximately two thirds to the 143 µg/m³ concentration value and used this in the health impact assessment described in Chapter 3.0.

The percentage of sampling minutes when PM$_{2.5}$ levels exceeded the US EPA ‘unhealthy for sensitive groups’ 35 µg/m³ threshold was typically 60% in smoking homes compared to values of <3% in coal, wood and gas burning homes and 7.3% in peat burning homes. Recent evidence suggests that removing exposure to fine particulate from second-hand tobacco smoke may be associated with a considerable decrease in the risk of cardiovascular and pulmonary events across the population (Mackay et al., 2010a; Mackay et al., 2010b; Oono et al., 2011). The health burden of these particulate matter concentrations is examined in more detail in Chapter 3.0.

Airborne endotoxin concentrations measured in this study are similar to those reported in previous studies in domestic environments. Concentrations were broadly similar in coal (5.78 EU/m³), peat (5.12 EU/m³), and smoking (5.38 EU/m³) homes but were higher in wood burning (7.63 EU/m³) homes. Household data of airborne endotoxin levels indicate that levels are generally less than 10 EU/m³. A large study of the homes of 332 children in Canada (Dales et al., 2006) presented mean concentration of 0.49 EU/m³ while Thorne and Duchaine’s (2007) data describing endotoxin levels in a number of environments indicate geometric mean (GM), inhalable fraction endotoxin in homes of rural asthmatic children of 5.8 EU/m³ (n=326). A further small study measuring airborne endotoxin in 10 homes in northern California (Chen et al., 2009) again suggested mean concentrations of <1 EU/m³. From our data there is little evidence that different fuel types, smoking activity or presence of pets influenced airborne endotoxin levels in the homes we measured although we do not have a control group of homes with no open combustion with which to compare our measurements.

2.4.3. Conclusions

Most of the IAP measured in the homes in this study were generally well controlled and for the purposes of health burden assessment it seems reasonable to focus on concentrations of fine particulate matter from household generated combustion. Coal, wood and gas-cooking homes appear to have PM$_{2.5}$ levels comparable to those found in outdoor ambient air while peat-burning homes and those where tobacco is smoked have higher levels. There is considerable evidence from the scientific literature that small increases in personal exposure to PM$_{2.5}$ may have considerable impacts in cardio-pulmonary health. Part Two of this study looks at the potential health burden to the Irish and Scottish population resulting from exposure to indoor combustion sources and in particular to household combustion-derived PM$_{2.5}$. 
3.0 Burden of disease attributable to indoor air combustion sources - purpose of Health Impact Assessment within IAPAH

One of the main aims of the IAPAH study was to estimate the health impacts of exposure to IAP in the home from exposure to ETS and the combustion of solid fuels (coal, wood and peat) for heating; and gas, for cooking. Within IAPAH, we interpreted this as quantifying the overall burden of disease on the populations of Ireland and Scotland due to the current levels of exposure to indoor air pollutants. In doing this we adopted a simplifying convention that is usual when considering disease burden (see e.g. COMEAP, 2010): the calculations have been done as if the effect of exposure on disease and mortality were immediate; i.e. the effects of current exposure levels were estimated using current population and current background rates of morbidity and mortality, without taking account of any time lag between exposure and increased risk of disease or death.

IAPAH restricted itself to the estimation of current burden of disease; it did not try to estimate (predict) the benefits to public health from introduction of any particular policies and measures which could impact future levels of IAP.

3.1 General methodology for HIA of combustion sources indoors

Working jointly with the EU HEIMTSA project\(^2\), we adapted the ‘full chain’ approach to environmental health impact assessment (www.integratedassessment.eu) developed by EU-funded projects such as ExternE\(^3\), HEIMTSA and INTARESE\(^4\) for application to IAP from combustion sources indoors, Supplementary Report 2 for details (Shafrir et al, 2011a). This general approach tracks the fate of pollutants from their source, through environments within which humans interact with the pollutant, to the specific health impacts caused by those pollutants. This requires considering as an integrated whole, the entire chain or pathway from pollution source through to health outcome, and managing the transitions between steps of the pathway; e.g. the exposure metric used for the estimating exposures must be the same as the exposure metric used for estimating exposure-related risks to health. We did the analysis iteratively, several times, to identify and as far as possible resolve data/evidence gaps and issues of alignment between the component parts of the analysis. Central to the approach is the choice of exposure metric where we considered several approaches and focused on two strategies which we called the source-based approach and the pollutant-based approach.

3.2 The source based approach

The source-based approach uses a very simple exposure metric – exposed, or not exposed, to the source being considered, e.g. to ETS in the home (often understood as living with a smoker), or using gas for

\(^2\) http://www.heimsta.eu
\(^3\) http://www.externoe.info
\(^4\) http://www.intarese.org
cooking, or using solid fuel for heating. This simplicity is its great strength; it implies that a relatively simple set of data is needed for estimating burden. As illustrated in Figure 3.1, these data are (i) the proportion of the population exposed indoors to the combustion source of interest; (ii) risk functions for health outcomes associated with the presence/absence of the exposure and (iii) background rates of disease in the unexposed population, for the selected health endpoints. The main disadvantage of the source-based approach is that it does not take account of intensity of exposure, for example the number of cigarettes a day smoked within the home. The pollutant-based approach is designed to overcome this limitation.

![Diagram](image)

**Figure 3.1** The Source-based approach for calculating the health impacts of exposure to indoor air pollutants

### 3.3 The pollutant-based approach

#### 3.3.1 Description

The pollutant-based approach takes one signature pollutant as a marker of the entire combustion mixture from the source of interest. For solid fuel use and ETS, PM\textsubscript{2.5} was the most relevant signature pollutant; we used it also for cooking with gas.
As outlined in Figure 3.2, assessing the health burden then requires combining information about (i) the relevant population exposed to IAP from combustion sources indoors; (ii) concentrations of relevant pollutants (i.e., for IAPAH, PM$_{2.5}$) within homes with combustion sources of pollution; and (iii) the risk to health of exposure indoors to those levels of PM$_{2.5}$, using exposure-response functions (ERFs) linking PM$_{2.5}$ with mortality and morbidity; and (iv) background rates of morbidity and mortality in the exposed population. (Note: Most of the available ERFs were derived and adapted from outdoor air pollution studies.)

This leads to a more complex model compared to the source-based approach, because of the need to incorporate pollutant concentrations. In IAPAH, we had direct measurements of IAQ, including PM$_{2.5}$, in admittedly a relatively small sample of homes in both Ireland and Scotland and, as indicated in Figure 3.2 and discussed further below, these were used as the principal basis for the pollutant-based assessments.

Pollutant levels were then combined with time-activity patterns (i.e. time spent indoors at home) to determine the annual average exposure to a particular pollutant, e.g. PM$_{2.5}$.

### 3.3.2 Advantages

As noted, one major advantage of the pollutant-based approach is that it takes account of intensity of exposure. Using PM$_{2.5}$ as the signature pollutant in IAPAH also theoretically enables the use of risk functions from outdoor air pollution, which in turn allows quantification of a different and wider set of health outcomes compared to those used in the source-based approach. In particular, it allows inclusion of the effects on mortality of long-term exposure to air pollution represented as PM$_{2.5}$ – and various studies of the burden of disease, or HIA, of outdoor air pollution have shown that this is by far the single most important ‘pathway’ among the many health outcome affected.
Disadvantages and methodological work to understand their importance

Background concentrations and personal exposures: Relationships between outdoor PM and health are based on PM as measured at background concentrations, at distance from source and from most of the population at risk; whereas IAPAH is concerned with PM in the home from indoor combustion sources in the same room or nearby – this is more like PM measured as personal exposures rather than as background concentrations.

To address this, we conducted a literature review of the relationship between annual average PM$_{2.5}$ in outdoor air, as measured at background concentrations, and the annual average of personal exposures to PM$_{2.5}$ attributable to outdoor sources whether experienced outdoors, or indoors, or in traffic; and we derived a conversion or scaling factor, estimated as $(0.7)^1$, by which the concentration-response functions of outdoor air were multiplied to ‘convert’ them to the exposure-response functions needed for IAPAH – see Section 4.0, Supplementary Report 4 (Shafrir et al, 2011b).

Health effects of PM$_{2.5}$ may vary by source (and associated composition) of the pollution mixtures. There are therefore approximations and associated uncertainties in using the effects on health as estimated from studies of PM$_{2.5}$ in outdoor air pollution when quantifying the health effects of other sources of PM$_{2.5}$ which for IAPAH means from IAP due to combustion sources indoors. In outdoor air pollution, the established practice currently, strongly supported by WHO (e.g. WHO, 2007), is to use the same risk functions for different kinds of PM. The solid fuels in IAPAH, i.e. coal, wood and peat, are examples of biomass; and so we reviewed specifically the limited evidence on PM$_{2.5}$ from biomass combustion outdoors (e.g. forest fires); this supported the WHO position of using the same risks (per µg/m$^3$) as in general urban PM$_{2.5}$ (Appendix 1, Supplementary Report 1 (Shafrir et al, 2011c)).

We were initially less convinced that PM$_{2.5}$ could reasonably be used as a marker for ETS indoors, because of the many chemicals including known carcinogens in ETS. However, in 2009 Pope et al. used the metric of inhaled dose of PM$_{2.5}$ to unify risk estimates across studies involving (i) outdoor air pollution; (ii) ETS and (iii) active smoking (see Supplementary Reports 1 (Shafrir et al, 2011c) and 4 Shafrir et al, 2011b)) and this legitimised using the pollutant-based approach for ETS also.

Using gas for cooking is often associated with increases nearby of NO$_2$ rather than of PM$_{2.5}$, and there is a case for using NO$_2$ as the signature pollutant for quantifying health impacts. There are relationships linking NO$_2$ in outdoor air with a wide range of health outcomes, including mortality. However, these are widely understood as reflecting primarily an effect of the complex mixture, including PM, from traffic combustion, rather than an effect of NO$_2$ per se; and so we did not think that they could be transferred with confidence from outdoor to indoor air.

Extrapolation to higher concentrations; non-linearity: The main relationships between outdoor PM$_{2.5}$ and health are based on studies in cities with annual average PM$_{2.5}$ less than about 30 µg/m$^3$, and ETS in homes can gives rise to higher concentrations of PM$_{2.5}$ indoors, making it necessary to extrapolate from the air pollution studies. This was possible using Pope et al. (2009), which took account of non-linearity in extrapolating to the kinds of higher concentrations and exposures implied by ETS indoors.
3.4 The chosen strategy

Supplementary Report 1 (Shafrir et al, 2011c) provides further details on the strategies selected but in summary;

For solid fuel sources, insofar as we quantified, we did so using only using the pollutant-based approach, and PM$_{2.5}$ – we did not use a source-based approach because the evidence of risks came from studies in less developed countries with far higher resultant concentrations of PM$_{2.5}$ indoors than in Ireland and Scotland. Similarly our initial strategy for addressing households where cooking was done with gas was to quantify using PM$_{2.5}$, although in practice (see Section 3.7, below) the attributable concentrations were too small to quantify reliably.

For ETS, however, we used both approaches; and we begin the detailed description of methods and results with the source-based approach to ETS.

3.5 The burden on health of never-smokers attributable to ETS in the home, using living with a smoker as an index of exposure

3.5.1 Population

To link with available risk functions, we aimed to estimate the number of children (<15y) and adult (25y+) never-smokers exposed to ETS inside the home. Sources of relevant information were scarce and different for each country. Estimates of the population of adult never-smokers in Scotland who were exposed to ETS in the home were based on data on never-smokers taken from research studies (Akhtar et al, 2007; Haw and Gruer, 2007). In Ireland this information was not available and estimates for exposed never-smokers were based on data for non-smokers living with a smoker, which in turn had to be derived using complex cross-referencing (see Section 2.0, Supplementary Report 3 (Hurley et al, 2011)) using multiple sources (e.g. Eurobarometer survey$^5$ (European Commission, 2009); research studies). All children aged <15 were assumed to be never-smokers.

Table 3.1 shows the estimated prevalence in each country (the age ranges having been adapted slightly to match our needs):

$^5$ http://www.esds.ac.uk/international/access/eurobarometer.asp
Table 3.1 Estimated prevalence of children and adult non-smokers (Ireland) and never smokers (Scotland) exposed to ETS inside home and percentage of smokers

<table>
<thead>
<tr>
<th>Country</th>
<th>Children (&lt;15)</th>
<th>Adults (25+)</th>
<th>Smokers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ireland</td>
<td>20%</td>
<td>16% (non-smokers)</td>
<td>24%#</td>
</tr>
<tr>
<td>Scotland</td>
<td>27%</td>
<td>12% (never smokers)</td>
<td>26%*</td>
</tr>
</tbody>
</table>

*Office of Tobacco Control (2009) smoking is defined as responding yes to the question “Do you smoke one or more cigarettes each week, whether packaged or roll your own?”

*Scottish Health Survey (2009) smoking is defined as responding yes to the question “Do you smoke cigarettes at all nowadays?”

3.5.2 Health outcomes; risk functions; background rates; impact functions

To identify health outcomes in never-smokers affected by living with a smoker, and associated relative risks (compared with never smokers unexposed to ETS at home), we used reviews by two expert panels: The UK Scientific Committee on Tobacco and Health (SCOTH, 2004) and the US Surgeon General’s report on ‘The Health Consequences of Involuntary Exposure to Tobacco Smoke’ (US Department of Health and Human Services, 2006). Both reports drew on much the same international evidence and came to similar conclusions. We used (see Table 3.2) the 2006 US risk estimates, for health outcomes where the US review concluded that there was sufficient evidence of a causal relationship, provided that suitable background rates could be found. In addition we also used asthma onset in children, which had been identified as another relevant health outcome in a separate review by the California EPA.

We then linked these risk (Fig 3.1) estimates with (estimates of) the background rates of occurrence in Ireland and Scotland of the same health outcomes in children, and in never-smoking adults, unexposed to ETS in the home, and (for lung cancer) taking account also of gender. For adults, we estimated the relevant rates in the general population, irrespective of smoking habit; then (see Section 4 of Supplementary Report 2 (Shafrir et al, 2011a)) adjusted these twice, first to that in the non-smoking population (we were unable to estimate background rates in never-smokers), then to that in non-smokers unexposed to ETS at home. Both adjustments were done using the methodology of the WHO burden of disease study on ETS (Öberg et al., 2010), which takes account of the proportions exposed and the relative risk of exposure. Because the resulting background rates, while markedly lower than those in the general population which includes smokers, apply to non-smokers (i.e. including ex-smokers as well as never-smokers), they may overestimate the background rate in never-smokers.

For most of the health outcomes studied, background rates in the general population in Ireland or in Scotland, in the age ranges needed, were available from national statistics. However, for some health endpoints information was not directly available and ad hoc methods, based on or informed by evidence were used to adjust the available data to give the estimates required. In some instances (e.g. lower respiratory illness in children) estimates from studies in other EU countries were used. Details are given in Supplementary Report 2 (Shafrir et al, 2011a). This information was then combined to give a set of impact functions for both Ireland and Scotland.
**Table 3.2 Risk functions for the health endpoints included in the report**

<table>
<thead>
<tr>
<th>Health Endpoint</th>
<th>Risk Function (95% CI)</th>
<th>Population</th>
<th>ETS exposure</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Lung cancer</strong></td>
<td>1.22 (1.13-1.31)*</td>
<td>25+</td>
<td>F Spouse</td>
</tr>
<tr>
<td><strong>Lung cancer</strong></td>
<td>1.37 (1.05-1.79)*</td>
<td>25+</td>
<td>M Spouse</td>
</tr>
<tr>
<td><strong>Coronary heart disease (CHD)</strong></td>
<td>1.27 (1.19-1.36)*</td>
<td>25+</td>
<td>M, F Spouse</td>
</tr>
<tr>
<td><strong>Sudden Infant Death Syndrome (SIDS)</strong></td>
<td>1.94 (1.55-2.43)**</td>
<td>0-1</td>
<td>M, F Mother (postnatal)</td>
</tr>
<tr>
<td><strong>Lower respiratory illnesses (LRI)</strong></td>
<td>1.56 (1.51-1.62)**</td>
<td>0-4</td>
<td>M, F Mother</td>
</tr>
<tr>
<td><strong>Asthma onset</strong></td>
<td>1.32 (1.24-1.41)**</td>
<td>0-14</td>
<td>M, F Either Mother or Father</td>
</tr>
<tr>
<td><strong>Respiratory symptoms</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wheeze</td>
<td>1.28 (1.21-1.35)**</td>
<td>5-16</td>
<td>M, F Mother</td>
</tr>
<tr>
<td>Cough</td>
<td>1.34 (1.17-1.54)**</td>
<td>5-16</td>
<td>M, F Mother</td>
</tr>
</tbody>
</table>

* Risk function is a relative risk (RR); ** Risk function is an odds ratio (OR) – very similar to RR when the absolute risks are low. M - male; F – female.

### 3.5.3 Results, i.e. estimated health burden

This process, simple in principle (Figure 3.1) but in practice very complicated to implement, resulted in the following estimated annual burden of disease in Ireland and in Scotland (Table 3.3, see overleaf). Results for the two countries are very similar.
Table 3.3  Health effects (cases per year, and 95% CI) attributable to exposure to ETS through never-smokers living with a smoker in Ireland and Scotland

<table>
<thead>
<tr>
<th>Health endpoint</th>
<th>Age Group</th>
<th>Health Effect</th>
<th>(95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Adults</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lung cancer incidence</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Females 25+</td>
<td></td>
<td>3.5 new cases</td>
<td>(2.0 – 5.0)</td>
</tr>
<tr>
<td>Males 25+</td>
<td></td>
<td>4.0 new cases</td>
<td>(0.5 – 8.5)</td>
</tr>
<tr>
<td>Coronary heart disease</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mortality 25+</td>
<td></td>
<td>85 additional deaths</td>
<td>(61 – 110)</td>
</tr>
<tr>
<td>Hospital discharges 25+</td>
<td></td>
<td>310 additional discharges</td>
<td>(210 - 400)</td>
</tr>
<tr>
<td><strong>Children</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SIDS 0-1</td>
<td></td>
<td>3.9 additional deaths</td>
<td>(2.3 – 6.0)</td>
</tr>
<tr>
<td>Lower respiratory illness</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hospital discharges 0-4</td>
<td></td>
<td>500 additional discharges</td>
<td>(460 - 560)</td>
</tr>
<tr>
<td>Symptoms 0-4</td>
<td></td>
<td>270,000 additional symptom days</td>
<td>(250,000 – 300,000)</td>
</tr>
<tr>
<td>Asthma onset 0-14</td>
<td></td>
<td>690 new cases</td>
<td>(520 - 880)</td>
</tr>
<tr>
<td>Respiratory symptoms</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wheeze 5-16</td>
<td></td>
<td>300,000 additional wheeze days</td>
<td>(230,000 – 370,000)</td>
</tr>
<tr>
<td>Cough 5-16</td>
<td></td>
<td>1,800,000 additional cough days</td>
<td>(900,000 – 2,800,000)</td>
</tr>
</tbody>
</table>

3.6 Using PM$_{2.5}$ as an index of exposure, the burden on health attributable to burning solid fuels in the home, or using gas for cooking

3.6.1 Population

As detailed in Section 2.0, Supplementary Report 3 (Hurley et al, 2011), there is very limited information on the number of households using specific solid fuels (as distinct from overall residential solid fuel usage) in Ireland and Scotland. In Ireland we obtained, analysed and summarised data from the Irish Household Budget Survey 2004/2005 (Central Statistics Office, Ireland, 2007), a representative random sample of all private households in Ireland, giving detailed information on household population and the fuel used for heating and cooking, classified as gas, electric, oil and solid fuel, but not by type of solid fuel (coal, peat and wood). The population exposed to peat as primary fuel was estimated by cross-reference with fuel usage data. In Scotland we used data from two or three years of the Scottish House Condition Survey (Amabile et al, 2009), a representative national survey of about 3,000 households annually with separate
information on the use of coal and wood/peat for cooking and heating, and gas for cooking. Estimates of the percentage of the population living in households burning solid fuel for heating, or using gas for cooking, were calculated by the SHCS team. Through these sources we estimated relevant percentages of the population exposed (Table 3.4).

Table 3.4  Percentage of the Irish and Scottish population living in households where solid fuel is used as primary heating fuel (SFU), or gas for cooking. Scottish data for solid fuel use (SFU) aggregate over coal, peat or wood, smokeless fuel, and anthracite.

<table>
<thead>
<tr>
<th>Ireland</th>
<th>&lt; 14 years (%)</th>
<th>14-20 years (%)</th>
<th>Males 21+ (%)</th>
<th>Women 21+ (%)</th>
<th>Households sampled (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>SFU Heating</td>
<td>9.5</td>
<td>11.8</td>
<td>8.5</td>
<td>9.3</td>
<td>8.4</td>
</tr>
<tr>
<td>Gas Cooking</td>
<td>23.7</td>
<td>22.2</td>
<td>26.0</td>
<td>25.3</td>
<td>26.0</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Scotland</th>
<th>&lt; 15 years (%)</th>
<th>15-25 years (%)</th>
<th>Males &gt;25 (%)</th>
<th>Women &gt;25 (%)</th>
<th>Households sampled (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>SFU Heating</td>
<td>1.0</td>
<td>1.5</td>
<td>1.9</td>
<td>1.6</td>
<td>2.5</td>
</tr>
<tr>
<td>Gas Cooking</td>
<td>57.5</td>
<td>53.3</td>
<td>54.9</td>
<td>53.8</td>
<td>49.3</td>
</tr>
</tbody>
</table>

1 The age-ranges used are unusual; we used slightly modified ranges to link with population numbers.

2 Gas cooking in Ireland: either piped gas or Liquefied Petroleum Gas (LPG).

3 Gas cooking in Scotland: i.e. gas cooker; or gas hob and electric oven;

### 3.6.2 Annual average concentrations

We estimated the annual average exposure (in µg/m³) to PM_{2.5} attributable to the indoor source using results from the IAPAH field study (Section 2.0) under two scenarios: as if residents were in the room sampled (i.e. the room most lived in) (i) evenings only – the principal scenario; (ii) all day long – the subsidiary scenario. Neither of these is fully realistic, but together they give a reasonable indication of exposure and associated burden. We used measurements of PM_{2.5} in homes in Ireland and Scotland from the IAPAH field study (Section 2.0) to give estimates of average indoor concentrations of PM_{2.5} in homes using various kinds of solid fuel for heating, or using gas for cooking for evenings (6pm until midnight) and all day. We interpreted these measurements as reflecting the effects of three main components: (i) the indoor combustion source of interest; (ii) the penetration indoors of outdoor air pollution, measured as PM_{2.5}; and (iii) the effect of all other indoor sources that might contribute to measurements of PM_{2.5} indoors, e.g. fine particles from cooking; re-suspended dust; a person’s ‘personal dust cloud’.

Our aim was to estimate the component attributable to the indoor combustion source of interest, by adjusting for the contribution of other sources. We estimated indoor penetration and carried out a literature
review of using gas for cooking, and other indoor sources. The results suggested strongly that the contribution to indoor PM$_{2.5}$ from using gas for cooking (as opposed to the particles generated by cooking food – cooking fume) was so small that it could not reliably be distinguished from background, and that non-zero impacts could not be estimated reliably.

Homes using gas for cooking were taken as a ‘control’ set of homes in the context of the field study, and their PM$_{2.5}$ measurements were compared with field study results from homes using coal, wood and peat for heating. About 30% of SFU homes sampled in field study had the solid fuel as secondary rather than primary heating fuel. For each fuel type the resulting differences in PM$_{2.5}$ concentrations were small and not significant statistically.

Concentrations of PM$_{2.5}$ from homes using coal and wood were very similar to those from homes using gas cooking, i.e. from background; and therefore health impacts for burning coal and wood were not estimated either. However, the field study concentrations for peat were higher than those for background / gas cooking (Section 2.0) and so we decided to estimate health impacts in Ireland, where up to 5% of the population is exposed and most of the field study sampling in peat-burning homes had taken place; but not in Scotland, where the proportion exposed was very small (<0.5%). We assumed exposure to peat as primary fuel for heating for 6 months of the year only.

3.6.3 Health outcomes; Risk functions; Background rates; Impact functions

From the extensive research world-wide linking particulate air pollution outdoors with mortality and morbidity, there is a reasonable consensus internationally on what concentration-response functions (CRFs) to use for HIA in various regions. We based IAPAH on the most important of a set of CRFs used in the HIA of the European Commission’s Clean Air for Europe (CAFE) programme (Hurley et al, 2005). This followed detailed review within the HEIMTSA EU project of the key relationships of mortality with PM$_{2.5}$, using more recent evidence, a review endorsed by an influential WHO working group. Selected functions in PM$_{10}$ were ‘translated’ to PM$_{2.5}$ using a conversion factor of 0.65; and all were converted to exposure-response relationships (i.e. ERFs rather than CRFs) as described in Section 3.4.3, earlier.

We used background mortality rates from Ireland and Scotland but for morbidity we mostly used background rates as used for CAFE HIA. The at-risk population at various ages was then linked with estimated annual average exposures, with the ERFs, and with background rates, to give, separately for Ireland and Scotland, the estimated annual burden of disease attributable to various combustion sources indoors.

3.6.4 Results

We first estimated health impacts under an artificial scenario whereby 10% of the population in Ireland and Scotland were exposed to an attributable concentration of 1 µg/m$^3$. Results for Ireland and for Scotland, based on exposures in winter evenings only (giving an annual average increase of 0.125 µg/m$^3$) are given in Tables 3.5 a and b, respectively. Results for all-day exposure are four times as large. The results can be scaled to give results for specific fuels, if the attributable concentrations and population size can be estimated. We then estimated impacts associated with peat-burning for heating in Ireland. These are given in Table 3.6.
Table 3.5a: Estimated burden on health in Ireland of a 1 µg/m³ increase in attributable concentration of PM$_{2.5}$, exposure winter evenings only

<table>
<thead>
<tr>
<th>Health endpoint</th>
<th>Age group</th>
<th>Total pop. at risk</th>
<th>%exposed</th>
<th>No of cases/days</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chronic bronchitis</td>
<td>18+</td>
<td>3,012,306</td>
<td>10</td>
<td>8</td>
<td>(1-14)</td>
</tr>
<tr>
<td>Cardiovascular hospital admissions</td>
<td>All ages</td>
<td>4,467,854</td>
<td>10</td>
<td>0.5</td>
<td>(0.2-0.7)</td>
</tr>
<tr>
<td>Respiratory hospital admissions</td>
<td>All ages</td>
<td>4,467,854</td>
<td>10</td>
<td>1.2</td>
<td>(1.0-1.4)</td>
</tr>
<tr>
<td>Restricted activity days</td>
<td>18-64</td>
<td>2,841,127</td>
<td>10</td>
<td>5,200</td>
<td>(4,600-5,900)</td>
</tr>
<tr>
<td>Lower respiratory symptom days</td>
<td>5-14</td>
<td>602,919</td>
<td>10</td>
<td>3,700</td>
<td>(1,900-5,700)</td>
</tr>
<tr>
<td>All-cause mortality</td>
<td>30+</td>
<td>2,559,015</td>
<td>10</td>
<td>3</td>
<td>(1-5)</td>
</tr>
</tbody>
</table>

* number of cases; ** number of days

Table 3.5b: Estimated burden on health in Scotland of a 1 µg/m³ increase in attributable concentration of PM$_{2.5}$, exposure winter evenings only

<table>
<thead>
<tr>
<th>Health endpoint</th>
<th>Age group</th>
<th>Total pop. at risk</th>
<th>%exposed</th>
<th>No of cases/days</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chronic bronchitis</td>
<td>18+</td>
<td>3,765,835</td>
<td>10</td>
<td>9</td>
<td>(1-17)</td>
</tr>
<tr>
<td>Cardiovascular hospital admissions</td>
<td>All ages</td>
<td>5,222,100</td>
<td>10</td>
<td>0.6</td>
<td>(0.3-0.9)</td>
</tr>
<tr>
<td>Respiratory hospital admissions</td>
<td>All ages</td>
<td>5,222,100</td>
<td>10</td>
<td>1.5</td>
<td>(1.2-1.7)</td>
</tr>
<tr>
<td>Restricted activity days</td>
<td>18-64</td>
<td>3,304,769</td>
<td>10</td>
<td>6,400</td>
<td>(5,600-7,200)</td>
</tr>
<tr>
<td>Lower respiratory symptom days</td>
<td>5-14</td>
<td>558,101</td>
<td>10</td>
<td>3,300</td>
<td>(1,600-4,900)</td>
</tr>
<tr>
<td>All-cause mortality</td>
<td>30+</td>
<td>3,334,922</td>
<td>10</td>
<td>6</td>
<td>(2-11)</td>
</tr>
</tbody>
</table>

* number of cases; ** number of days
Table 3.6: Estimated burden on health in Ireland of indoor air pollution from burning peat as primary fuel, exposure winter evenings only

<table>
<thead>
<tr>
<th>Health endpoint</th>
<th>Age group</th>
<th>Total pop. at risk</th>
<th>%exposed</th>
<th>No of cases/days</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chronic bronchitis</td>
<td>18+</td>
<td>3,012,306</td>
<td>4.30</td>
<td>55</td>
<td>(5-98)</td>
</tr>
<tr>
<td>Cardiovascular hospital admissions</td>
<td>All ages</td>
<td>4,467,854</td>
<td>4.45</td>
<td>4</td>
<td>(2-5)</td>
</tr>
<tr>
<td>Respiratory hospital admissions</td>
<td>All ages</td>
<td>4,467,854</td>
<td>4.45</td>
<td>9</td>
<td>(7-10)</td>
</tr>
<tr>
<td>Restricted activity days</td>
<td>18-64</td>
<td>2,841,127</td>
<td>4.30</td>
<td>38,000</td>
<td>(33,400-42,600)</td>
</tr>
<tr>
<td>Lower respiratory symptom days</td>
<td>5-14</td>
<td>602,919</td>
<td>4.75</td>
<td>30,100</td>
<td>(15,000-45,400)</td>
</tr>
<tr>
<td>All-cause mortality</td>
<td>30+</td>
<td>2,559,015</td>
<td>4.20</td>
<td>21</td>
<td>(7-38)</td>
</tr>
</tbody>
</table>

* number of cases; ** number of days

3.7 The burden on health of never and non-smokers attributable to ETS in the home, using PM\(_{2.5}\) as an index of exposure

3.7.1 Population

The initial population at risk (children; adult never-smokers living with a smoker) is the same as for the source-based approach to ETS – see Section 3.6.1, above. In addition, we used attributable annual average PM\(_{2.5}\) to estimate the health burden in (i) non-smokers; and (ii) never smokers

3.7.2 Annual average concentrations

The annual average concentrations of PM\(_{2.5}\) attributable to ETS were estimated in a similar way to that for solid fuels (see Section 3.7.2, above), i.e. by using as a control the field study concentrations from homes using gas for cooking (Section 2.0), apart from one major difference. The field study measurements of PM\(_{2.5}\) in homes with ETS were very high compared with results from other studies, and the choice of homes may have contributed to this (Section 2.4.1). Consequently for PM\(_{2.5}\) concentrations in homes with ETS we used not the measurements themselves, but 2/3 of these measurements, before measurements for gas cooking were deducted.

3.7.3 Health outcomes; Risk functions; Background rates; Impact functions

Similarly, the health outcomes, risk functions in PM\(_{2.5}\), and general population background rates used were generally the same as before (see Section 3.7.3, above), but there were two major differences in how they were applied. First, the background rates used were those for non-smokers rather than for the general population as used for solid fuel. Secondly, for the estimates assuming all-day (24-hour) exposures, the annual average exposures were substantially higher than 30 µg/m\(^3\) and so, as indicated in Section 3.4.3, a non-linear relationship based on Pope et al. (2009) was used for mortality. That relationship from Pope et
al. (2009) used cardio-respiratory mortality rather than all-cause mortality, and this was the relationship also used in IAPAH. Using non-linearity led to lower estimated impacts than an estimate based on linear relationships. The ratio of non-linear to linear impacts for cardio-respiratory mortality was then applied to all other estimated impacts, which otherwise would have assumed linearity. Details are given in Supplementary Report 4 (Shafrir et al, 2011b).

### 3.7.4 Results

We estimated impacts associated with ETS exposure in Ireland and Scotland for both non-smokers and never-smokers. Results for never-smokers are given in Table 3.7a and 3.7b. Health burden for non-smokers is approximately 50% higher than for never-smokers.

Table 3.7a: Estimated burden on health of indoor air pollution in never-smokers in Ireland from ETS: winter evening exposure (concentration = 29.82 µg/m³)

<table>
<thead>
<tr>
<th>Health endpoint</th>
<th>Age group</th>
<th>Total population at risk</th>
<th>%exposed</th>
<th>No of cases/days</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chronic bronchitis</td>
<td>18+</td>
<td>1,506,153</td>
<td>16%</td>
<td>846*</td>
<td>(73-1517)</td>
</tr>
<tr>
<td>Lower respiratory symptom days</td>
<td>5-14</td>
<td>602,919</td>
<td>20%</td>
<td>1,293,902**</td>
<td>(643,535-1,951,037)</td>
</tr>
<tr>
<td>Cardiopulmonary mortality</td>
<td>30+</td>
<td>1,279,508</td>
<td>16%</td>
<td>244*</td>
<td>(82-434)</td>
</tr>
</tbody>
</table>

* number of cases; ** number of days

Table 3.7b: Estimated burden on health of indoor air pollution in never-smokers in Scotland from ETS: winter evening exposure (concentration = 29.82 µg/m³)

<table>
<thead>
<tr>
<th>Health endpoint</th>
<th>Age group</th>
<th>Total population at risk</th>
<th>%exposed</th>
<th>No of cases/days</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chronic bronchitis</td>
<td>18+</td>
<td>1,920,576</td>
<td>12%</td>
<td>810*</td>
<td>(70-1,453)</td>
</tr>
<tr>
<td>Lower respiratory symptom days</td>
<td>5-14</td>
<td>558,101</td>
<td>27%</td>
<td>1,542,813**</td>
<td>(767,334-2,326,364)</td>
</tr>
<tr>
<td>Cardiopulmonary mortality</td>
<td>30+</td>
<td>1,700,810</td>
<td>12%</td>
<td>346*</td>
<td>(115-615)</td>
</tr>
</tbody>
</table>

* number of cases; ** number of days
4.0 Conclusions and Recommendations

This program of work has achieved the objectives set out both in terms of characterising exposure to IAPs within domestic environments in Ireland and Scotland and also providing the first detailed estimates of the potential health burden of combustion-generated pollution at home.

4.1 IAPAH field study measurements

The program of measurement in the IAPAH project collected information on IAP concentrations from 100 homes split between Ireland and Scotland. It is encouraging to see that the levels measured of most pollutants in solid-fuel burning homes are generally within available 24-hour guidance limits. This tends to suggest that the homes sampled in both countries have well-maintained solid-fuel heating systems with adequate ventilation and extraction. Concentrations/levels of PM$_{2.5}$ in coal and wood burning homes appeared, on average, to be no higher than in homes using gas for cooking and it is likely that the levels reported in these homes are similar to those in electric cooking/heating homes. Particulate levels in peat burning homes were higher and, on average, about twice the level of gas, wood and coal burning homes and this suggests a non-trivial particulate burden on occupants in these homes.

Measurement of fine particulate (i.e. PM$_{2.5}$) in houses where smoking took place showed much higher concentrations in both countries, and higher also than in other available studies of ETS in homes, perhaps in part because the study selection criteria may unintentionally have tended to include homes with lower levels of air exchange. Averaged over 24 hours the PM$_{2.5}$ levels measured in Ireland and Scotland exceeded 140 µg/m$^3$ and, as such, approach the US EPA outdoor air quality index level that is deemed to be ‘very unhealthy’. It was clear from the field study measurements that among the indoor combustion sources studies, adverse health impacts would be associated primarily with smoking indoors, not with solid fuel use or use of gas for cooking.

4.2 The Health Impact Assessment (HIA) methodology used in IAPAH

The HIA methodology used in IAPAH describes, compares and assesses two fundamentally different approaches to estimating burden of disease from combustion sources indoors. The main difference between them is in how exposures are measured, and the implications of that for the full chain analysis as a whole. The simpler ‘source-based’ approach classifies exposure only by presence or absence of the source. This is the traditional and established approach, partly because it needs much less data to implement, and has been used by WHO in its recent estimates of Global Burden of Disease (GBD) (Smith et al, 2004). The other (pollutant-based) approach is made possible only by the extensive research on PM$_{2.5}$ in outdoor air and the widespread acceptance (e.g. COMEAP, 2009) that this is the best indicator of effects on mortality of the outdoor pollution mixture; together with very recent evidence (Pope et al., 2009) that it is a good indicator also of mortality risks from ETS and from active smoking.

We expect that the pollutant-based approach will become the approach of choice whenever suitable PM$_{2.5}$ data are available, partly because it enables estimates wider than for never-smokers. Because the
approach is new, further methodological development is needed and indeed is under way – we understand (Aaron Cohen, 2011) that the next revision of GBD will include estimates using PM$_{2.5}$. Meantime, for never-smokers, estimates lying somewhere between the main source-based and pollutant-based approaches given here seem a reasonable guide to what’s going on in Ireland and Scotland, and a reasonable basis for development of policy. Results using PM$_{2.5}$ for non-smokers seem reasonable also; those for smokers are much more speculative at this point – they were a supplementary focus for us, and methodological issues to do with non-linearity and background rates have not been addressed fully, and so should be considered as indicative only; nevertheless they do provide some evidence-based guidance on the size of the health burden.

The second point concerns data, and the difficulties of getting what is needed to implement even the simpler source-based approach. In the present study some quite complex processing, linking of data from various sources, was needed to estimate both the population exposed, and the background rates of morbidity in the non-exposed population. We underestimated these difficulties and we encourage others to learn from that, as we also hope to do. We were fortunate that measurements of PM$_{2.5}$ had been designed-in to the IAPAH study – else this aspect also would have needed complex estimation procedures.

All this serves to reinforce that using evidence to inform policy, via HIA and /or Burden of Disease estimates, is a process of controlled approximation rather than an exact science. This is not in any way to denigrate the work or its importance – uncertainties and approximations are unavoidable, and it is very helpful to have them dealt with logically and transparently, as we hope we have done; but it is important to bear in mind that this does not eliminate the uncertainties, it (simply) allows us to work more clearly in and through them.

4.3 The estimated burden on public health

From the HIA part of this study it is evident that, at a population level, the main issue to deal with in terms of combustion-related effects on household air quality is tobacco smoke. Our estimates of the health impact of ETS-derived fine particulate matter suggest that 20% of children in Ireland and 27% of children in Scotland are exposed on a regular basis within their home with over 300,000 never-smoking householders over 30 years of age are exposed to ETS at home in Ireland with a similar number exposed in Scotland. Using a source-based approach to this exposure suggests that 85 cardiovascular deaths may be attributable to ETS exposure in Ireland with 110 deaths in Scotland. Small numbers of deaths due to lung cancer (< 10 per annum) are also likely to occur in both countries. Results of the HIA using the pollutant based approach with PM$_{2.5}$ suggest that the mortality burden for never-smokers may be higher with the figure likely to lie somewhere between 244-340 cardiopulmonary deaths in Ireland and between 346 and 483 deaths in Scotland, depending on the proportion of time that the exposed population spend inside their homes.

The health burden of exposure to combustion-derived particulate at home is considerable and primarily driven by exposure to ETS. In terms of mortality it seems likely that the number of deaths from ETS exposure at home in each country is broadly comparable to those from road traffic accidents (212 in Ireland in 2010; 208 in Scotland in 2010). Morbidity from respiratory illness among children is also likely to be
considerable with ETS exposure causing perhaps upwards of 2 million additional respiratory symptom days per year across both countries.

4.4 Recommendations

The results and conclusion of this study imply that, in considering measures to protect public health from IAP from combustion sources indoors, attention should focus on measures which would reduce the practice of smoking tobacco indoors. The widest health benefits will come from effective programmes to reduce the numbers starting smoking and increase those of smokers quitting. Our results also show that there could be significant health gains for co-residents, usually family members, if those who continue to do so do not smoke indoors at home. Co-ordinated national campaigns aimed at educating smokers about the health effects of ETS exposure at home should be developed as should tools to empower non-smokers to engage with smoking residents about changing behaviours and implementing household smoking restrictions and smoke-free homes.

In support of these policies, and to better estimate their benefits, a programme of further research could usefully focus on the following;

1. Collect annual data on the number of people exposed to ETS at home. A question to gather this information should be inserted in national population surveys in both countries.

2. Greater understanding of household behaviours and the amount of time spent at home by population sub-groups, particularly those with chronic health conditions, older people and the very young.

3. Development of methods to determine the transferability of exposure-response co-efficients from outdoor air pollution to indoor air pollution.

4. Intervention studies to help reduce PM$_{2.5}$ concentrations in homes where smoking takes place.

In order to improve the health of future generations there is a real need for public health policy and research professionals to work together to develop ways of improving air quality in homes as a matter of urgency.
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