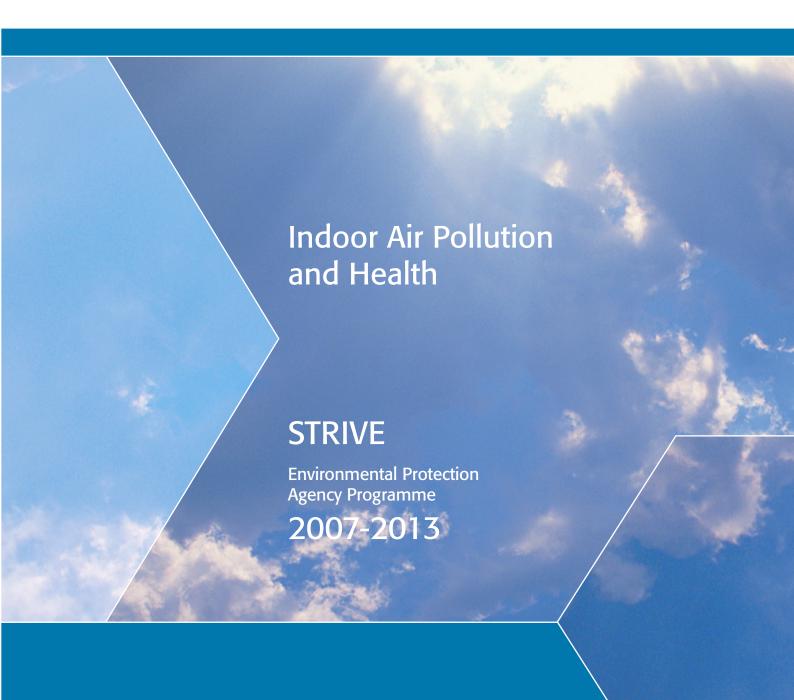


STRIVEReport Series No.104







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EPA STRIVE Programme 2007-2013

Indoor Air Pollution and Health (IAPAH)

(2008-EH-MS-8-S3)

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.

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Executive Summary

The objectives of the Indoor Air Pollution and Health (IAPAH) research project were to quantify the levels of Indoor Air Pollution (IAP) in Irish and Scottish homes from indoor combustion sources, and to provide an estimate of the potential health burden, i.e. the annual damage to health, in Ireland and Scotland, due to exposure to IAP from combustion sources in the home. IAP concentrations were measured in 100 homes in Ireland and Scotland.

Indoor combustion sources was defined as the use of the solid fuels (coal, wood and peat) for heating, gas for cooking or the presence of tobacco smoking. Twenty-four-hour data on airborne concentrations of particulate matter smaller than 2.5 μ m (PM_{2.5}), carbon monoxide (CO), carbon dioxide (CO₂) and endotoxin¹, together with 2-3 week averaged concentrations of nitrogen dioxide (NO₂) were collected.

Concentrations of IAP in homes using solid fuels for heating 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 and wood for heating, and gas for cooking 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 homes using coal, wood and gas for cooking. 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, 2005) 24-hour $PM_{2.5}$ guidance concentration value of 25 μ g/m³, and over four times the US Environmental Protection Agency (EPA) outdoor Air Quality index 'unhealthy' level for sensitive groups of 65 μ g/m³ guidance value. 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 requires:

- Information on the proportion of the population exposed to the pollutant source;
- Risk functions for health outcomes associated with the presence of the pollutant source; and

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¹ Endotoxin is a biological component of fine particulate matter, derived from the cell wall of gram-negative bacteria

 Background rates of disease in the unexposed population for those same health outcomes.

The pollutant-based approach uses a signature pollutant, in this case PM_{2.5}, as a marker of the pollutant source of interest. It 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 health burden 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 burden attributable to burning solid fuels and using gas cooking in the homes, two exposure scenarios were considered:

- Exposure to the source from 6 pm until midnight; and
- Exposure to the source 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 PM_{2.5} in homes using coal and wood for heating were low and so the health burden was not calculated. Concentrations of PM_{2.5} in homes using peat for heating were slightly higher and health burden was calculated, but only for the exposed population in Ireland, the exposed population in Scotland being very small.

Results from the health impact assessment indicate that exposure to ETS represents the greatest health burden from combustion-derived air pollution in the homes. Both the source-based approach and the pollutant-based approach estimate as the greatest health burden cardiovascular events 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 from road traffic accidents in Ireland and Scotland. There is a real need for public health policy and research professionals to address this.

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 guitting.

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 determine population-wide exposure to ETS at home by incorporating this issue in existing national health survey campaigns in in Ireland and Scotland.

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.

A summary of the general methodology, results and conclusions of the HIA, is presented in this report. More detailed project information is provided in four supplementary reports, available on the EPA Safer-data website by clicking here or following the links from (<a href="http://erc.epa.ie/safer/).

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), where vulnerable groups such as young children and the elderly can spend up to 100% of their time (Bonnefoy *et al.*, 2004). Exposure concentrations vary and depend on a number of factors including individuals' behaviour and activities, pollutant sources, and 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 towards improving outdoor air quality and regulating sources of outdoor air pollution (European Commission, 2008). While indoor air pollution (IAP) in the workplace and enclosed public places have been regulated, indoor air quality in domestic settings remains largely unregulated. 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 its health burden.

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 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 exacerbation of chronic respiratory illnesses such as asthma, allergies, chronic obstructive pulmonary disease (COPD), 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 prevalence of childhood allergy and asthma (ISAAC, 2007). While it would be wrong to presume that IAP is a major cause of these higher disease prevalence and mortality rates, these facts highlight the importance of understanding the IAP contribution.

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

emissions from 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 tobacco smoking (Saraga et al., 2010; Larsson et al., 2004). However, few studies have investigated how the use of fossil fuels for cooking or heating in the homes contributes 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 economicallydeveloped settings because 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 only few have studied homes using coal (Moriske et al., 1996; Henderson et al., 2006) or peat (Guo et al., 2008). Fuels 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 account for the greatest share of the TFC, with 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 while 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 et al., 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 increase 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, 2010b). 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 fuels 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., 2010; 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 PM_{2.5}² (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 is 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 for heating (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 (European Commission, 2011; WHO, 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

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;
- To estimate how many people are exposed to different sources and concentrations of key indoor air pollutants; and
- To use these data to generate an estimate of the health burden that is attributable to air pollution within homes.

To achieve this, the research will draw on published materials 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:

- 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;
- Identify key reviews on long-term exposure to outdoor air pollution and summarise the potential for applying outdoor coefficients to derive indoor coefficients;

3

-

¹ Particulate matter smaller than 2.5µm, also referred to as 'fine' particles

- Determine the number and type of households where people are exposed to elevated IAP levels and the population profile within these homes;
- Derive estimates of average annual exposures attributable to indoor sources; and
- 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 Contribution of solid fuel, gas combustion and Environmental Tobacco Smoke to indoor air pollutant concentrations in Irish and Scottish homes

2.1 Introduction to IAPAH field study

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 published in the International Journal Indoor Environment and Health; Indoor Air (Semple et al., 2012).

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 dedicated project website (www.nuigalway.ie/iapah).

Other participants were recruited via word of mouth and snowballing techniques utilising those already recruited for the study. Our aim was to recruit 20 households that used peat as heating fuel, 20 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 questions about solid fuels use and smoking by residents in the home. Households were excluded if they reported burning more than one type of solid fuels/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 sources under study. A total of five IAPs were measured including PM_{2.5}, airborne endotoxin within the total inhalable dust fraction, CO, CO₂ and NO₂. 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 PM_{2.5} impactor, were used to collect and log real-time data in µg/m³ on airborne PM_{2.5} levels over a 24-hour period. A correction factor for combustion-generated PM_{2.5} 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 (Gradko diffusion tubes 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 2.1.



Figure 2.1: Sampling arrangement used in volunteer homes.

Photo shows a padded equipment box, the TSI Sidepak AM510 Aerosol monitor, placed inside the box, and its $PM_{2.5}$ impactor head measuring PM attached outside (not visible in photo). Legend: (1) The Lascar Easylogger logging CO, (2) an IOM total inhalable dust collector, and (3) the Telaire® 7001i Data logger logging CO_2 and relative humidity (%)

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

WHO guidelines for indoor air recommend a 24-hour guideline value of 7 mg/m³ (arithmetic mean); equivalent to 6.1 ppm for CO (WHO, 2010b). For outdoor air, WHO recommends a CO limit of 6.1 ppm (24-hour average) and a 24-hour PM_{2.5} guideline value of 25 μg/m³ (WHO, 2005). US EPA ambient air standards for NO₂ are 50 ppb (24-hour average). ASHRAE (1989) indoor air quality guidance suggests that CO₂ concentrations above 1000 ppm indicate poor ventilation. There are no standards for household endotoxin other than the Dutch Occupational guidance at 90 EU/m³ (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/m³. Lower average levels were found in homes that burned coal (9 µg/m3) or wood (8 µg/m³) and in homes with gas cookers (9) μg/m³). In peat-burning homes, the average 24-hour PM_{2.5} level was 16 μg/m³. Much higher particulate concentrations were found in homes with resident smokers (143 µg/m³). Across the 100 homes the average 24-hour concentration of CO2 was 713 ppm. The average 24-hour NO₂ concentration was 5 ppb and airborne endotoxin levels averaged 5.7 EU/m³.

For PM_{2.5} a 6-hour evening concentration was also calculated from the real-time data. This 6pm to midnight period was derived to better reflect personal exposure indoors at home 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 μ g/m³ with 11 μ g/m³ for wood-burning, 13 μ g/m³ for coal-burning, 12 μ g/m³ for gascooking, 29 μ g/m³ for peat-burning and 197 μ g/m³ for homes with smokers.

Table 2.1: Demographic and housing characteristics of sampled households

Demographic/Housing Characteristic ⁺	All (n=100)	Scotland (n=52)	Ireland (n=48)	P-value (probability)
Coal burning	22	10	12	
Peat burning	20	3	17	
Wood burning	22	17	5	
Gas cooking	16	11	5	
Smoking	20	9	11	
Age of householder giving consent (mean, years)	51	52	50	NS
Room volume (m³) Central heating (%)	57 93	56 88	58 98	NS 0.06
Type of house (n, %) Detached Semi-detached* Terraced** Flat/apartment Age of house (n, %) Pre early 1980s	51 (51%) 30 (30%) 8 (8%) 11 (11%)	25 (48%) 16 (31%) 2 (4%) 9 (17%)	26 (54%) 14 (29%) 6 (13%) 2 (4%)	0.09
Post early 1980s Pets in household (%)	41 (41%)	10 (19%)	31 (65%) 52%	0.00 NS
Outdoor temp (°C) Outdoor PM _{2.5} (µg/m³)	6.0 8.2	5.8 8.2	6.2 8.1	NS NS

Legend:

NS

any house sampled had only one of the five combustion sources listed not significant

* Semi-detached houses
** Terraced houses

consists of pairs of houses built side by side as units sharing a wall houses in a row of similar houses that share side-walls

Table 2.2: Average IAP concentrations measured in IAPAH study homes

Pollutant time-weighted average mean values	AII (n=100)	Coal (n=22)	Gas Cooking (n=16)	Peat (n=20)	Smoking (n=20)	Wood (n=22)
†PM _{2.5} (μg/m ³)	36.8	8.9	8.6	15.6	143	7.7
(range)	50.2	(1-19) 13.0	(2-28) 12.2	(2-44) 29.1	(21-463) 197.2	(2-23) 10.8
*PM _{2.5} (μg/m ³) (range)	30.2	(3-38)	(2-57)	(3-136)	(16-539)	(3-52)
†CO (ppm)	0.05	0.01	0.04	0.01	0.22	0.00
(range)		(0-0.03)	(0-0.31)	(0-0.17)	(0-1.44)	(0-0.003)
†CO ₂ (ppm)	713	642	687	713	818	708
(range)		(480-854)	(450-1171)	(490-1097)	(469-1290)	(520-1540)
‡NO ₂ (ppb)	5.12	4.03	9.01	3.99	6.82	2.87
(range)		(1.48-13.5)	(2.11-24.1)	(1.08-15.8)	(2.2-13.6)	(1.05-6.2)
†Airborne endotoxin (EU/m³)	5.69	5.78 (0.11-25.7)	3.09 (0.72-6.9)	5.12 (0.12-24.7)	5.38 (0.92-21.7)	7.63 (0.12-16.6)
(range)		(5.11 25.7)	(0.12 0.0)	(3.12 21.1)	(5.52 21.1)	(5.12 10.0)

Legend: †24-hour sampling period, *6 hour time-weighted average from 6pm-midnight, ‡two week sampling period

Figure 2.2, below, 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 $\mu g/m^3$) (WHO, 2005).

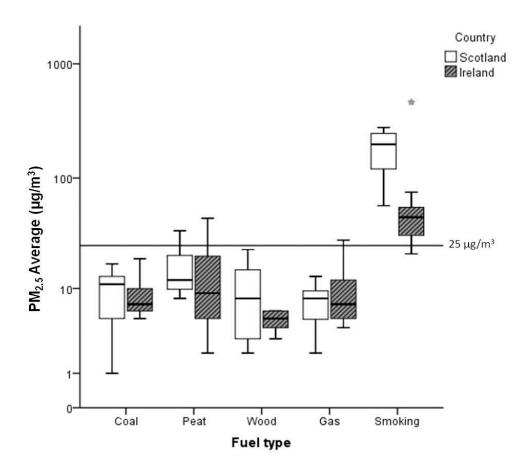


Figure 2.2: 24-hour $PM_{2.5}$ concentrations by fuel type and country. NB: Concentrations are on the log scale.

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 household with ETS. 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.

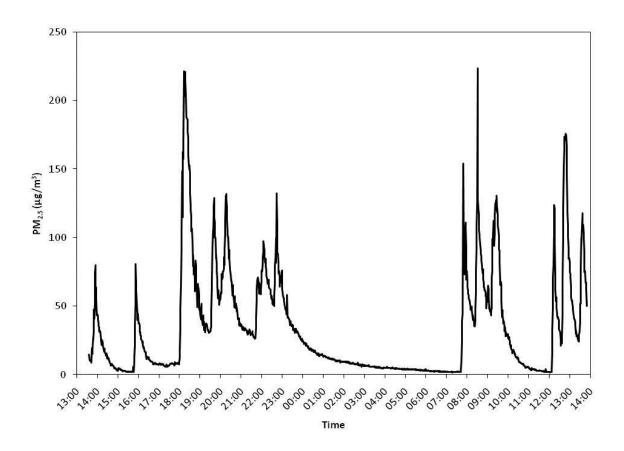


Figure 2.3: 24-hour real-time plot of PM_{2.5} concentrations from one participating household.

2.4 Discussion

2.4.1. Strengths and limitations of the study

The 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 the selection process including homes with a single fuel type, the research has been able to examine if there are differences in IAP concentrations between different combustion materials. This is a particular strength of this study.

It is difficult to ascertain 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. Therefore, the comparison of the recruited group with the overall population from which they were sampled was not possible. 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 available and so there are likely to have been temporal variations in outdoor pollutant concentrations over the 6-month measurement programme. This will have resulted in different contributions to indoor pollutant levels from outdoor pollutants on different days. This effect would have been small and, although only data for outdoor $PM_{2.5}$ levels in the Aberdeen and Galway areas were available, 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 was noted.

As with all observational studies and exposure measurements, it is possible that the act of measurement has influenced the parameters under study. Modification of behaviour in heating, cooking, ventilation and smoking is possible. The 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 smoker resident who smoked indoors. Concentrations of CO, NO2 and airborne endotoxin were well within health-based standards in all homes using solid fuels for for heating, or gas cooking, 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 fuels in Scotland and Ireland. PM_{2.5} concentrations were generally similar to outdoor ambient air levels in homes using coal and wood for heating and gas-cooking homes; about twice the outdoor concentrations in peat-burning homes and were the highest in smoking homes. 24-hour average concentrations were found to exceed the WHO 24-hour guidance level of 25 μ g/m³ (WHO, 2005) in one-quarter (n=25) of homes although most (n=19) of those homes exceeding this value were smoking homes. Twelve of the 20 smoking homes sampled (60%) had 24-hour PM_{2.5} concentrations that exceeded

the 24-hour US EPA 65 µg/m3 threshold deemed to be unhealthy (Semple et al, 2012). The PM_{2.5} data in particular show that the mean 24-hour average levels in smoking homes were 15 to 20 times higher than those measured in the solid fuels-burning or gas-cooking homes. The mean 24-hour level in smoking homes of 143 μg/m³ was over 4 times the US EPA outdoor Air Quality Index 'unhealthy' level for sensitive groups (35 μg/m³) (US EPA, 2011a) and approaching six times the WHO 24-hour guidance concentration of 25 $\mu g/m^3$ (WHO, 2005). 24-hour fine particulate matter levels were broadly similar to those found in outside air in coal- and woodburning and gas-cooking homes. Peat-burning homes had average particulate levels that were closer to the WHO annual guidance level of 25 $\mu g/m^3$ for PM_{2.5} (WHO, 2005).

The main cause for concern in terms of IAP from combustion in homes in Scotland and Ireland is from smoking activity. PM25 concentrations in homes with a smoker resident are, in general, an order of magnitude higher than those found in homes burning coal, wood or peat for heating or using gas for cooking. The 24-hour PM_{2.5} concentrations in the homes where tobacco smoking took place are considerable. The average value of 143 µg/m³ 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 smokefree restrictions was 200 μg/m³ (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 μg/m³ (Repace et al., 2011). Smoking homes included in this study may not be representative of all smoking homes in Scotland and Ireland. Based on other work done by the research group

(not presented here but described in Shafrir *et al.*, 2011b) a scaling factor of approximately two thirds to the 143 μ g/m³ concentration value was applied and used in the health impact assessment.

The percentage of sampling minutes when PM_{2.5} levels exceeded the US EPA 'unhealthy for sensitive groups' 35 μg/m³ threshold (US EPA, 2011) was typically 60% in smoking homes compared to <3% in homes using coal or wood for heating and gas-cooking homes; and 7.3% in peat-burning homes. Recent evidence suggests that removing exposure to fine particulates 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.

Airborne endotoxin concentrations measured in this study are similar to those reported in previous studies in domestic environments (Thorne and Duchaine, 2007). Arithmetic mean concentrations were broadly similar in coalburning (5.78 EU/m³), peat-burning (5.12 EU/m³), and smoking (5.38 EU/m3) homes but were somewhat 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 a mean concentration of 0.49 EU/m3 while Thorne and Duchaine's (2007) data describing endotoxin levels in a number of environments, indicate a geometric mean (GM) of inhalable fraction endotoxin in homes of rural asthmatic children of 5.8 EU/m3 (n=326). Another small study measuring airborne endotoxin in 10 homes in northern California (Chen *et al.*, 2009) again suggested mean concentrations of <1 EU/m³.

From the data, there is little evidence that different fuel types or smoking activity influenced airborne endotoxin levels in the homes that were surveyed although there was no control group of homes with no open combustion with which to compare the measurements.

2.4.3. Conclusions

Most of the IAPs measured in the homes included 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 generated from household combustion. Coaland wood-burning 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.

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 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, this was interpreted as quantifying the overall annual burden of disease on the populations of Ireland and Scotland due to the current levels of exposure to indoor air pollutants. In doing this, a simplifying convention that is usual when considering disease burden (e.g. COMEAP, 2010) was adopted.

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 annual 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 indoor combustion sources

Working jointly with the EU HEIMTSA (Health and Environment Integrated Methodology and

Toolbox for Scenario Assessment) project³, the research team adapted the 'full chain' approach to environmental health impact assessment (www.integratedassessment.eu) developed by EU-funded projects such as ExternE⁴, HEIMTSA and INTARESE⁵ for application to IAP from indoor combustion sources (Shafrir et al., 2011a). This general approach tracks the fate of pollutants from their source, through environments within which humans interact with the pollutants, 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). The analysis was done iteratively, 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 several approaches were considered and this project focused on two strategies referred to as the source-based approach and the pollutant-based approach.

³ http://www.heimsta.eu

⁴ External Costs of Energy: http://www.externe.info

⁵ Integrated Assessment of Health Risks of Environmental Stressors in Europe: http://www.intarese.org

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 cooking, or using solid fuels for heating. This simplicity is its great strength as 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 the intensity of exposure, for example the number of cigarettes smoked per day within the home. The pollutant-based approach is designed to overcome this limitation.

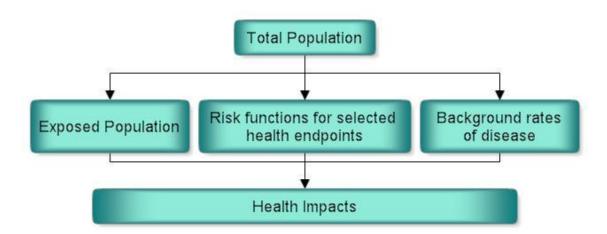


Figure 3.1: The source-based approach for calculating the health impacts of exposure to indoor air pollutants

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 fuels use and ETS, PM_{2.5} was the most relevant signature pollutant. It was used 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 indoor combustion sources;
- (ii) concentrations of relevant pollutants(i.e., for IAPAH, PM_{2.5}) within homeswith combustion sources of pollution;
- (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 (Hurley et al., 2005; WHO, 2006).

This leads to a more complex model compared to the source-based approach, because of the need to incorporate pollutant concentrations. In the IAPAH project, direct measurements of IAQ, including $PM_{2.5}$ were available, in 100 homes in Ireland and Scotland. As indicated in Figure 3.2 and discussed further in this report, 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 estimate the annual average exposure to a particular pollutant, e.g. $PM_{2.5}$.

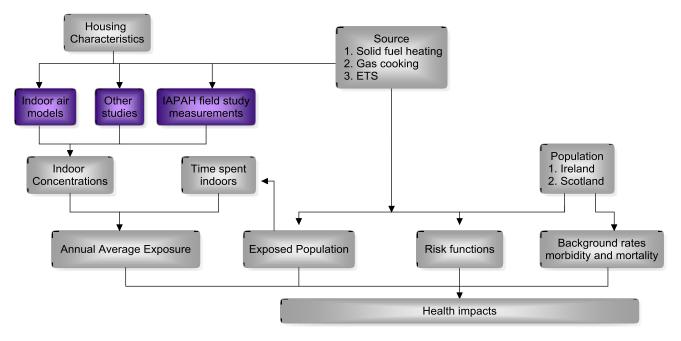


Figure 3.2: Application of the pollutant-based approach within IAPAH (purple boxes are unique to the IAPAH study)

3.3.2 Advantages

As noted, one major advantage of the pollutantbased approach is that it takes account of the 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. This, 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}. 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 outcomes affected (US Environmental Protection Agency, 2011b; Watkiss et al., 2008.

3.3.3 Disadvantages and methodological work to understand their importance

<u>Background concentrations and personal</u> <u>exposures</u>

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, a simple model was constructed of time spent in various micro-environments (indoors; outdoors in traffic; elsewhere outdoors) and associated average concentrations relative to background outdoors. A conversion or scaling factor was estimated as 0.7, by which the CRFs of outdoor air were divided to convert them to the required ERF (Hurley et al., 2011).

Health effects of PM_{2.5} may vary by source (and associated composition) of the pollution mixtures.

There are approximations and 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 indoor combustion sources. 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_{2.5}. The solid fuels in IAPAH, i.e. coal, wood and peat, are examples of biomass; and therefore the limited evidence on health risks of PM_{2.5} from biomass combustion outdoors (e.g. forest fires) was reviewed specifically. This supported the WHO position of using the same risks (per µg/m³) as in general urban PM_{2.5} (Appendix 1, Shafrir et al., 2011c).

The research team was 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 (Shafrir *et al.*, 2011b; Shafrir *et al.*, 2011c). This legitimised using the pollutant-based approach for ETS also.

Using gas for cooking is often associated with nearby increases 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 (Anderson *et al.*, 2007; Nafstad *et al.*, 2004). 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*. Therefore the

research team did not think that these relationships could be transferred with confidence from outdoor to indoor air.

Extrapolation to higher concentrations; nonlinearity

The most influential relationship in PM_{2.5} is that linking long-term exposure to increased risks of mortality. Key relationships from the American Cancer Society study, e.g. Pope *et al.* (2002), Krewski *et al.* (2009), are based on studies in cities with annual average PM_{2.5} less than 30 µg/m³. As noted earlier, ETS in homes can give rise to much higher concentrations of PM_{2.5} indoors, making it necessary to extrapolate from the air pollution studies to effects at higher concentrations. This was possible using Pope *et al.*, (2009), which took account of non-linearity in extrapolating to the higher concentration and exposure levels implied by ETS indoors.

3.4 The chosen strategy

Shafrir *et al.*, (2011c) provides further details on the strategies selected but in summary:

For solid fuel sources, insofar as this project quantified, it was done using only the pollutant-based approach and PM_{2.5.}, A source-based approach was not used because the evidence of risks came from studies in less developed countries with far higher indoors concentrations of PM_{2.5} than in Ireland and Scotland. Similarly, the 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) the attributable concentrations were too small to quantify reliably.

<u>For ETS</u>, however, both approaches were used as detailed in the following sections.

3.5 The burden on health of neversmokers 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, the research team 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 neversmokers 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-smokers⁶ living with a smoker (Shafrir et al., 2011a). This in turn had to be derived using complex cross-referencing (see Section 2.0, Hurley et al., 2011) using multiple sources. All children aged <15 were assumed to be neversmokers.

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

⁶ Non-smokers include both never smokers and exsmokers

Table 3.1: Estimated prevalence of children and adult non-smokers (Ireland) and neversmokers (Scotland) exposed to ETS inside home and percentage of smokers

Country	Children (<15)	Adults (25+)	Smokers
Ireland	20%	16% (non-smokers)	24%#
Scotland	27%	12% (never-smokers)	26%*

Legend:

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), the research team 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. The 2006 US risk estimates, for health outcomes where the US review concluded that there was sufficient evidence of a causal relationship, were used, provided that suitable background rates could be found (see Table 3.2). In addition asthma onset in children, which had been identified as another relevant health outcome in a separate review by the California EPA (California EPA, 2005) was also used.

These risk estimates were then linked (see Figure 3.1) with estimates of the background rates of occurrence in Ireland and Scotland of the same health outcomes in children, and in neversmoking adults, unexposed to ETS in the home, and for lung cancer taking account also of gender. For adults, the research team estimated

the relevant rates in the general population, irrespective of smoking habit; then (see Section 4, Shafrir et al., 2011a)) adjusted these twice, first to that in the non-smoking population (the research team was 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 well as never-smokers), they overestimate the background rate in neversmokers

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 (Shafrir *et al.*, 2011a. However, for some health endpoints (Table 3.2), 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. Details are given in Shafrir *et al.*, 2011a. This information was then combined to give a set of impact functions for both Ireland and Scotland.

[#] 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?"

Table 3.2: Risk functions for the health endpoints included in the report

Health Endpo	int	Risk Function	Population		ETS exposure
		(95% CI)	Age group	Gender	
Lung cancer		1.22 (1.13-1.31)*	25+	F	Spouse
Lung cancer		1.37 (1.05-1.79)*	25+	M	Spouse
Coronary hear	t disease (CHD)	1.27 (1.19-1.36)*	25+	M, F	Spouse
Sudden Infan (SIDS)	t Death Syndrome	1.94 (1.55-2.43)**	0-1	M, F	Mother (postnatal)
Lower respirate	ory illnesses (LRI)	1.56 (1.51-1.62)**	0-4	M, F	Mother
Asthma onset		1.32 (1.24-1.41)**	0-14	M, F	Mother or Father
Respiratory	Wheeze	1.28 (1.21-1.35)**	5-16	M, F	Mother
symptoms	Cough	1.34 (1.17-1.54)**	5-16	M, F	Mother

Legend:

 $^{^{\}star}$ 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 estimated annual burden of disease in Ireland and in Scotland presented in Table 3.3. Results for the two countries were very similar.

Table 3.3 Health effects (cases per year, and 95% CI) attributable to exposure to ETS through neversmokers living with a smoker in Ireland and Scotland $\frac{1}{2}$

Health endpoint	Age Group	He	(95% CI)	
Adults				
Lung cancer incidence				
Females	25+	3.5	new cases	(2.0 – 5.0)
Males	25+	4.0	new cases	(0.5 – 8.5)
Coronary heart disease				
Mortality	25+	85	additional deaths	(61 – 110)
Hospital discharges	25+	310	additional discharges	(210 - 400)
Children				
SIDS	0-1	3.9	additional deaths	(2.3 – 6.0)
Lower respiratory illness				
Hospital discharges	0-4	500	additional discharges	(460 - 560)
Symptoms	0-4	270,000	additional symptom days	(250,000 – 3000,000)
Asthma onset	0-14	690	new cases	(520 - 880)
Respiratory symptoms				
Wheeze	5-16	300,000	additional wheeze days	(230,000 – 370,000)
Cough	5-16	1,800,000	additional cough days	(900,000 – 2,800,000)

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, Hurley *et al.*, (2011), there is very limited information on the *number of households using specific solid fuels* (as distinct from overall residential solid fuels usage) in Ireland and Scotland. For Ireland, the research team 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 fuels, but not by type of solid fuels (coal, peat or wood). The population exposed to peat-burning as primary fuel was estimated by cross-reference with fuel usage data. For Scotland, the research team used data from two or three years of the Scottish House Condition Survey (SHCS) (Amabile et al., 2009), a representative annual national survey of about 3,000 households 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 fuels for heating, or using gas for cooking, were calculated by the SHCS team. Through these sources, relevant percentages of the population exposed were estimated (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, or gas for cooking. Scottish data for solid fuel use aggregate over coal, peat or wood, smokeless fuel, and anthracite

Ireland	< 14 years (%)	14-20 years (%)	Males ¹ 21+ (%)	Women ¹ 21+ (%)	Households sampled (%)
Heating	9.5	11.8	8.5	9.3	8.4
Gas Cooking ²	23.7	22.2	26.0	25.3	26.0

Scotland	< 15 years (%)	15-25 ¹ years (%)	Males ¹ >25 (%)	Women ¹ >25 (%)	Households sampled (%)
Heating	1.0	1.5	1.9	1.6	2.5
Gas Cooking ³	57.5	53.3	54.9	53.8	49.3

Legend:

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

The research team 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) as if residents were in the room sampled (i.e. the room most lived in) under two scenarios: (i) evenings only (6pm until midnight) - the principal scenario; (ii) all day long - the subsidiary scenario: together they give a reasonable indication exposure of associated burden. Measurements of PM2.5 in homes in Ireland and Scotland from the IAPAH field study were used 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. These measurements were interpreted 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; resuspended dust; a person's 'personal dust cloud'.

The aim was to estimate the component attributable to the indoor combustion source of interest, by adjusting for the contribution of other sources. Indoor penetration was estimated and a literature review of using gas for cooking and other indoor sources was carried out. 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 solid fuel use (SFU) homes sampled in field study had the solid fuel as secondary rather than primary heating fuel but this did not result in any significant differences in $PM_{2.5}$ concentrations.

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

From the extensive world-wide research linking particulate air pollution outdoors with mortality and morbidity (WHO, 2006), there is a reasonable consensus internationally on what concentration-response functions (CRFs) to use for HIA in various regions. IAPAH was based on the most important 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, which concluded that no change was needed Selected functions in PM₁₀ 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.

Background mortality rates from Ireland and Scotland were used but for morbidity background rates, as used for CAFE HIA, were mostly used. 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 indoor combustion sources indoors.

3.6.4 Results

The project team estimated health impacts associated with and attributable to peat-burning for heating in Ireland, as given in Table 3.5 below.

Table 3.5: Estimated burden on health in Ireland of indoor air pollution from burning peat as primary fuel (results presented to 2 significant figures)

			Exposure winter evenings (6pm-midnight), concentration = 2.11 µg/m³		Exposure 24-hr concentration = 3.55 μg/m ³		
Health endpoint	Age group	Total pop. at risk(millions)	%exposed	Annual no. cases/days	95% CI	Annual no. cases/days	95% CI
Chronic bronchitis	18+	3.0	4.30	55 [*]	(5-98)	91 [*]	(8-160)
Cardiovascular hospital admissions	All ages	4.5	4.45	4 [*]	(2-5)	6*	(3-9)
Respiratory hospital admissions	All ages	4.5	4.45	9 [*]	(7-10)	15 [*]	(12-17)
Restricted activity days	18-64	2.8	4.30	38,000**	(33,000-43,000)	63,000**	(56,000-71,000)
Lower respiratory symptom days (inc cough)	5-14	0.6	4.75	30,000**	(15,000-45,000)	50,000**	(25,000-76,000)
All-cause mortality	30+	2.6	4.20	21*	(7-38)	34*	(11-63)

Legend: * number of cases, ** number of days

3.7 The burden on health of neverand 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). In addition, attributable annual average PM_{2.5} were used 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), i.e. by using as a control the field study concentrations from homes using gas for cooking (Section 2), 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 the measurements themselves were not used, but 2/3 of these measurements, before measurements for gas cooking were deducted (see Section 4 Shafrir et al., (2011c) for further information).

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), but there were two major differences in how they were applied. First, the background rates used were those for nonsmokers rather than for the general population as used for solid fuels. Secondly, for the estimates assuming all-day (24-hour) exposures, the annual average exposures were substantially higher than 30 µg/m³ 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 cardiorespiratory 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 Shafrir et al., (2011b).

3.7.4 Results

Impacts associated with ETS exposure in Ireland and Scotland were estimated for both non-smokers and never-smokers. Results for never-smokers are given in Table 3.7a and Table 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 (results presented to 2 significant figures): evening exposure (concentration = $29.82 \mu g/m^3$)

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

Legend: * number of cases; ** number of days

Table 3.7b: Estimated burden on health of indoor air pollution in never-smokers in Scotland from ETS (results presented to 2 significant figures): evening exposure (concentration =29.82 μ g/m³)

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

Legend: * number of cases; ** number of days

4 Conclusions and Recommendations

This programme 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 collected information on IAP concentrations from 100 homes split between Ireland and Scotland; most measurements related to monitoring over a full (24-hour) day. It is encouraging to see that the levels measured of most pollutants in homes burning solid fuels are generally within available 24-hour guidance limits. This tends to suggest that the homes sampled in both countries have well-maintained solid-fuels heating systems with adequate ventilation and extraction. Concentrations/levels of PM_{2.5} in coal- and wood-burning homes were, on average, very similar to those 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-cooking and of wood- and coal-burning homes and this

Measurement of fine particulate (i.e. $PM_{2.5}$) in houses where smoking took place showed much higher concentrations in both countries.

suggests a non-trivial particulate burden on

occupants in these homes.

Averaged over 24 hours, the PM_{2,5} levels measured in Ireland and Scotland exceeded 140 μg/m³ and, as such, approach the US EPA outdoor air quality index level that is deemed to be 'very unhealthy'. This is higher also than in other available studies of ETS in homes, and may be in part because the study selection criteria may unintentionally have tended to include homes with lower levels of air exchange. Concentrations such as these nevertheless point to a real problem, and 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 the use of solid fuels for heating or 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 indoor combustion sources. 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 has been 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 PM25 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., 2011) that it is a good indicator also of mortality risks from ETS and from active smoking.

The pollutant-based approach has the great advantage that it enables quantification of the effects of IAP on a much wider range on health outcomes. Because the approach is new, and because there are some uncertainties in applying to pollution from indoor sources a set of risk functions from outdoor air, further methodological development and wider support from established expert groups is needed. This indeed is under way - the project team understands (Aaron Cohen, personal communication, 2011) that the next revision of the GBD will include estimates using PM_{2.5}.7 In the meantime, for neversmokers, estimates between the main sourcebased and pollutant-based approaches given here seem a reasonable guide to what is 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.

Another issue 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. These difficulties were underestimated and others are encouraged to learn from that.

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This is reinforcing 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.

4.3 The estimated burden on public health

No estimates were made of the health burden attributable to the particulate air pollution from combustion of gas for cooking or for the combustion of coal and wood for heating. This should not be interpreted as saying that there are no adverse health effects. It is however reasonable to infer that any associated burden of disease is small, in terms of overall public health in Ireland and in Scotland, and is unlikely to be associated with mass concentrations of fine particulate aerosol.

For the combustion of peat for heating, the estimated population exposed in Scotland was so small that, given that the attributable concentrations of $PM_{2.5}$ were not large, a HIA was not attempted. An assessment of health burden was undertaken for peat burning in Ireland and the resulting estimates show, as expected, some limited impacts on serious health outcomes, including mortality; and more numerous impacts on mild or transient conditions such as lower respiratory symptom days.

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. The project estimates of the health impact on nonsmokers 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 and over 400,000 adult non-smokers are exposed regularly or frequently to ETS at home in Ireland, a similar number in

⁷ This has now been published, Lim SS et al: A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990-2010: a systematic analysis for the Global Burden of Disease Study 2010. *The Lancet* 2012; 380: 2224-2260.

Scotland. Using a source-based approach to this exposure suggests that 85 cardiovascular deaths per annum may be attributable to ETS exposure in Ireland and 110 annual 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 and 340 cardiopulmonary deaths per annum 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 indoor combustion sources, 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

smoke, do not smoke indoors at home. Coordinated 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:

- 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.
- 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.
- Further research is needed to develop methodologies to assess the health burden attributable to indoor air pollution
- Development of methods to determine the transferability of exposure-response coefficients from outdoor air pollution to indoor air pollution.
- 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.

In addition to this summary report, more detailed project information is provided in four supplementary reports, available on the EPA Safer-data website by clicking here or following the links from (http://erc.epa.ie/safer/).

References

Akhtar PC, Currie DB, Currie CE, Haw SJ. (2007). Changes in child exposure to environmental tobacco smoke (CHETS) study after implementation of smoke-free legislation in Scotland: national cross sectional study. British Medical Journal; 335(7619):545. Epub

Amabile, D., Cormack, D., Hinchliffe, S., (2009) Scottish House Condition Survey Key Findings for 2004/5, Scottish Executive

Anderson HR, Atkinson RW, Bremner SA, Carrington J, Peacock J. (2007) Quantitative systematic review of short term associations between ambient air pollution (particulate matter, ozone, 20 nitrogen dioxide, sulphur dioxide and carbon monoxide), and mortality and morbidity. Report to the Department of Health. Available at: https://www.gov.uk/government/uploads/system/uploads/attachment_data/file/147698/dh_121202.pdf.

ASHRAE (1989) American Society of Heating, Refrigerating and Air Conditioning Engineers (ASHRAE). Ventilation for Acceptable Indoor Air Quality (62-1989).

Bonnefoy, X.R., Annesi-Maesona, I., Aznar, L.M., Braubachi, M., Croxford, B., Davidson, M., Ezratty, V., Fredouille, J., Ganzalez-Gross, M., Van Kamp, I., Maschke, C., Mesbah, M., Moisonnier, B., Monolbaev, K., Moore, R., Nicol, S., Niemann, H., Nygren, C., Ormandy, D., Robbel, N., Rudnai, P. (2004) Review of evidence on housing and health, 4th Ministerial Conference on Environment and Health, Budapest, Hungary, 23-25 June 2004).

Brennan, N., McCormack, S., O'Connor, T. (2008) Ireland needs healthier airways and lungs – The Evidence (INHALE) (2nd Edition). The Irish Thoracic Society.

California EPA. (2005). Part B: Health Effects. In: Proposed Identification of Environmental Tobacco Smoke as a Toxic Air Contaminant. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment, Air Toxicology and Epidemiology Branch.

Central Statistics Office Ireland (2007) Irish Household Budget Survey 2004/2005. Available at http://www.cso.ie/newsevents/pr_hbs2004-2005Final.htm. [Accessed August 2011].

Chen Q., Hildemann L.M. (2009) The effects of human activities on exposure to particulate matter and bioaerosols in residential homes. Environmental Science and Technology., 43, 4641-4646.

Cohen, A., The Health Effects Institute Boston Massachusetts, USA, (Personal communication, 2011)

COMEAP. (2010) the mortality effects of long-term exposure to particulate air pollution in the United Kingdom. Committee of the Medical Effects of Air Pollution. Available at http://comeap.org.uk/images/stories/Documents/Reports/comeap%20the%20mortality%20effects%20of%20longterm%20exposure%20to%20particulate%20air%20pollution%20in%20the%20uk%202010.pdf [Accessed August 2011].

COMEAP (Committee on the Medical effects of Air Pollution) (2009). Long-term exposure to air pollution: effect on mortality. COMEAP Secretariat c/o HPA, Didcot, Oxfordshire, UK.

Dales R., Miller D., Ruest K., Guay M., Judek S. (2006) Airborne endotoxin is associated with respiratory illness in the first 2 years of life. Environmental Health Perspectives, 114, 610-614.

DECOS. (2010) Endotoxins: health based recommended exposure limit. A report of the Health Council of the Netherlands. The Netherlands, The Hague, Health Council of the Netherlands. Publication No 2010/040SH.

Dennekamp M., Akram M., Abramson M.J., Tonkin A., Sim M.R., Fridman M., Erbas B. (2010) Outdoor air pollution as a trigger for out-of-hospital cardiac arrests, Epidemiology, 21,494-500.

Dennekamp M, Howarth S, Dick C A J, Cherrie J W, Donaldson K., Seaton A. (2001) Ultrafine particles and nitrogen oxides generated by gas and electric cooking. Occupational and Environmental Medicine, 58(8), 511-516.

European Commission (web site). Framework 7 Environment including climate change (http://cordis.europa.eu/fp7/environment), [Accessed July 2011].

European Commission (2009). Eurobarometer 72.3 Special Eurobarometer 332. Tobacco.

ExternE (web site)–Externalities of Energy. A research project of the European Commission. (http://www.externe.info/, [Accessed August 2011].

European Commission (2008) Scientific Committee on Health and Environmental Risks (SCHER): "Opinion on risk assessment on indoor air quality" DG Health and Consumers of the European Commission.

Fine PM, Cass GR, Simoneit BR. (2002) Chemical characterization of fine particle emissions from the fireplace combustion of woods grown in the Southern United States. Environmental Science Technology,1;36(7):1442-1451.

Fullerton D G, Semple S, Kalambo F, Suseno A, Malamba R, Henderson G, Ayres J G. Gordon S B. (2009) Biomass fuel use and indoor air pollution in homes in Malawi. Occupational and Environmental Medicine, 66, 777-783.

Garcia Algar O, Pichini S, Basagana X, Puig C, Vall O, Torrent M, Harris J, Sunyer J, Cullinan P. (2004) Concentrations and determinants of NO2 in homes of Ashford, UK and Barcelona and Menorca, Spain. Indoor Air. 14, 298-304.

Guo, L, Lewis JO, McLaughlin JP. (2008) Emissions from Irish domestic fireplaces and their impact on indoor air quality when used as a supplementary heating source. Global NEST Journal, ,10(2):209-216.

Gustafson P, Ostman C, Sallsten G. (2008) Indoor levels of polycyclic aromatic hydrocarbons in homes with or without wood burning for heating. Environmental Science and Technology ,15;42(14):5074-5080.

Haw S.J, Gruer L. (2007) Changes in exposure of adult non-smokers to second-hand smoke after implementation of smoke-free legislation in Scotland: national cross sectional survey. British Medical Journal; 335(7619):549. Epub 2007.

HEIMTSA (web site) Health and Environment Integrated Methodology and Toolbox for Scenario Assessment. A research project of the European Commission. (http://www.heimtsa.eu/, [Accessed August 2011].

Henderson KA, Parry S, Matthews IP. (2006) Real-time measurement of short-term peaks in environmental CO concentrations in the homes of the elderly in South Wales. Journal of Exposure Science and Environmental Epidemiology, 16(6):525-530.

HSE. (2000) General methods for sampling and gravimetric analysis of respirable and inhalable dust. UK. Health and Safety Executive MDHS 14/3 2000. Available at: http://www.hse.gov.uk/pubns/mdhs/pdfs/mdhs14-3.pdf [Accessed 22nd July 2011].

Hurley F, Shafrir A, Cowie H, Sánchez Jiménez A, Ayres JG, Coggins M, Semple S, Galea KS. (2011) Indoor Air and Health in Ireland and Scotland (IAPAH) Supplementary Report 3: Estimation of the Health Burden due to Solid Fuel Use and Use of Gas for Cooking. Available from URL: http://erc.epa.ie/safer/iso19115/display?isoID=282#files.

Hurley F, Hunt A, Cowie H, Holland M, Miller B, Pye S, Watkiss P. (2005). Methodology for the Cost-Benefit Analysis for CAFE: Health Impact Assessment. European Commission. (Service Contract for Carrying out Cost-Benefit Analysis of Air Quality Related Issues, in particular in the Clean Air for Europe (CAFE) Programme Volume 2).

INTARESE (web site) Integrated Assessment of Health risks of environmental Stressors in Europe.A research project co-funded by the European Commission. http://www.intarese.org/, [Accessed August 2011].

ISAAC [web site]. International Study of Asthma and Allergies in childhood (http://isaac.auckland.ac.nz/, [Accessed 9th March 2007].

Kleipeis NE, Nelson WC, Ott WR, Robinson JP, Tsang AM, Switzer, P., Behar, J.V., Hern, S.C., Engelmann, W.H., (2001) The National Human Activity Pattern Survey (NHAPS): a resource for assessing exposure to environmental pollutants. Journal of Exposure Analysis and Environmental Epidemiology, 11(3), 231-252.

Kurmi O P, Gaihre S, Semple S. Ayres J. G. (2010) Acute exposure to biomass smoke causes oxygen desaturation in adult women. Thorax, 65, 221-228.

Larsson L, Szponar B. Pehrson C. (2004) Tobacco smoking increases dramatically air concentrations of endotoxin. Indoor Air, 14, 421-424.

Levesque, B., Allaire, S., Gauvin, D., Koutrakis, P., Gingras, S., Rhainds, M., Prud'Homme, H., Duchesne, J.F., (2001) Wood Burning appliances and indoor air quality. The Science of the Total Environment, 281: 47-62.

Mackay D.F, Haw S., Ayres J.G., Fischbacher C. Pell J.P. (2010a) Smoke-free legislation and hospitalizations for childhood asthma. New England Journal Medicine, 363, 1139-1145.

Mackay D.F., Irfan M.O., Haw S. Pell J.P. (2010b) Meta-analysis of the effect of comprehensive smoke-free legislation on acute coronary events, Heart. 96, 1525-1530.

Monn Ch, Fuchs A, Hogger D, Junker M, Kogelschatz D, Roth N. Wanner H.-U. (1997) Particulate matter less than $10\mu m$ (PM₁₀) and fine particles less than 2.5 μm (PM_{2.5}): relationships between indoor, outdoor and personal concentrations. The Science of the Total Environment. 208, 15-21.

Moriske HJ, Drews M, Ebert G, Menk G, Scheller C, Schondube M, Konieczny, L (1996) Indoor air pollution by different heating systems: coal burning, open fireplace and central heating. Toxicology Letters, 88(1-3):349-354.

Naeher L P, Brauer M, Lipsett M. Zelikoff J T, Simpson C, Koenig J Q, Smith, K.R.(2007) Woodsmoke health effects: a review. Journal of Inhalation Toxicology, 19 (1), 1-47.

Nafstad P, Haheim LL, Wisloff T, Gram F, Oftedal B, Holme I, et al. Urban air pollution and mortality in a cohort of Norwegian men. Environmental Health Perspectives, 2004;112:610–615.

O'Leary (2008) Energy in the residential sector, 2008 Report, Sustainable Energy Ireland

Öberg M, Jaakkola MS, Prüss-Üstün A, Schweizer C, Woodward A. (2010). Second-hand smoke: Assessing the burden of disease at national and local levels. Geneva, World Health Organization (WHO Environmental Burden of Disease Series, No. 18).

Office of Tobacco Control (2009). Annual report, Ireland.

Oono I.P., Mackay D.F. Pell J.P. (2011) Meta-analysis of the association between secondhand smoke exposure and stroke. Journal of Public Health, 33(4), 496-502.

Pekey B, Bozkurt Z B, Pekey H, Doğan G, Zararsiz A, Efe N. Tuncel G. (2010) Indoor/outdoor concentrations and elemental composition of $PM_{10}/PM_{2.5}$ in urban-industrial areas of Kocaeli City, Turkey. Indoor Air, 20(2), 112-125.

Pope C.A. III, Burnett, R.T., Turner, M.C., Cohen, A., Krewski, D., Gapstur, S.M., and Thun, M.J., (2011) Lung Cancer and Cardiovascular disease mortality associated with ambient air pollution and cigarette smoke: Shape of the exposure-response relationships, Environmental Health Perspectives, 119(11):1616-1621.

Pope C.A.III, Burnett RT, Krewski D, Jerrett M, Shi Y, Calle EE, Thun MJ. (2009). Cardiovascular mortality and exposure to airborne fine particulate matter and cigarette smoke. Shape of the exposure-response relationship. Circulation, 120: 941-948.

Repace J.L., Jiang R.T., Acevedo-Bolton V., Cheng K.C., Klepeis N.E., Ott W.R. and Hildemann L.M. (2011) Fine particle air pollution and secondhand smoke exposures and risks inside 66 US casinos, Environmental Research, 111, 473-84.

Repace J. Air Pollution in Virginia's hospitality industry. Final report 2006. http://www.repace.com/pdf/VAAQSurvey.pdf [Accessed 22nd June 2011]

Saraga, D.E., Maggos, T., Helmis, C.G., Michopoulos, J., Bartzis, J.G., and Vasilako, C. (2010) PM1 and PM_{2.5} ionic composition and VOCs measurements in two typical apartments in Athens, Greece: investigation of smoking contribution to indoor air concentrations. Environmental Monitoring and Assessment, 167:321–331.

SCOTH. (2004). Update of evidence of health effects of secondhand smoke. London: Department of Health. 3785).

Scottish Health Survey (2009). National Statistics Publication for Scotland. Edinburgh: The Scottish Government.

Semple S., Garden C., Coggins M., Galea K.S, Whelan P., Cowie H., Sánchez-Jiménez A., Thorne P.S., Hurley J.F., Ayres J.G. (2012) Contribution of solid fuel, gas combustion, or tobacco smoke to indoor air pollutant concentrations in Irish and Scottish homes. Indoor Air. 22:212-223

Semple S., van Tongeren M., Galea K.S., MacCalman L., Gee I., Parry O., Naji A, Ayres J.G. (2010) UK smoke-free legislation: changes in PM_{2.5} concentrations in bars in Scotland, England, and Wales, Annals of Occupational Hygiene, 54, 272-80.

Shafrir A, Sánchez Jiménez A, Hurley F, Cowie H, Galea KS. (2011a) Indoor Air and Health in Ireland and Scotland (IAPAH) Supplementary Report 1: Health Impact Assessment: General Methodology and Its Application to the IAPAH study. Available from URL: http://erc.epa.ie/safer/iso19115/display?isoID=282#files.

Shafrir A, Cowie H, Sánchez Jiménez A, Hurley F, Galea KS. (2011b) Indoor Air Pollution and Health in Ireland and Scotland (IAPAH) Supplementary Report 2: Health Impact Assessment: Environmental Tobacco Smoke (ETS) Using Data on Living with a Smoker. Available from URL: http://erc.epa.ie/safer/iso19115/display?isoID=282#files

Shafrir A, Hurley F, Cowie H, Sánchez Jiménez A, Galea KS. (2011c) Indoor Air and Health in Ireland and Scotland (IAPAH) Supplementary Report 4: Health Impact Assessment due to Environmental Tobacco Smoke (ETS) exposure using PM_{2.5}. Available from URL: http://erc.epa.ie/safer/iso19115/display?isoID=282#files

Smith KR, Mehta S, Maeusezahl-Feuz M. (2004). Indoor air pollution from household use of solid fuels. In: Ezzati M, Lopez AD, Rodgers A, Murray CLP, eds. Comparative Quantification of Health Risks. Geneva: World Health Organization: 1435-1493.

THADE report. Towards healthy air in dwellings in Europe 2004. Overview of European data on indoor air pollution in dwellings and related health effects. Available from: http://www.efanet.org/activities/documents/THADEReport.pdf. [Accessed August 2011].

Thorne PS, Duchaine C. (2007) Airborne bacteria and endotoxin. In: Hurst, CJ, Crawford, RL, Garland, JL, Lipson, DA, Mills, AL, Stetzenbach, LD, (Eds.). Manual of Environmental Microbiology: ASM Press; 2007. p. 989-1004.

U.S. Department of Health and Human Services. (2006). The health consequences of involuntary exposure to tobacco smoke: a report of the Surgeon General. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, Coordinating Center for Health Promotion, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health.

US EPA (2011a). US Environmental Protection Agency air quality guidelines. Available at http://www.airnow.gov/index.cfm?action=aqibasics.aqi [Accessed 22nd July 2011].

US Environmental Protection Agency (2011b). The Benefits and Costs of the Clean Air Act from 1990 to 2020. Available at http://www.epa.gov/oar/sect812/feb11/fullreport.pdf

Watkiss P, Holland M, Hurley F, Hunt A, Pye S (2008). Assessing the Costs and Benefits of the European Air Pollution Policy (CAFE): Results and Lessons from Experience. In: J Le Roux, T Sherpa and E Williams (eds.). Economic Appraisal of Environmental Regulation. Stirling: Scottish Environmental Protection Agency.

World Health Organisation (WHO) (2010a) Global Status Report on non communicable diseases. WHO Press, World Health Organization, 20 Avenue Appia, 1211 Geneva 27, Switzerland.

World Health Organisation (2006). Health risks of particulate matter from long-range transboundary air pollution. WHO Regional Office for Europe, Copenhagen, Denmark. Available at http://www.euro.who.int/_data/assets/pdf_file/0006/78657/E88189.pdf

WHO Regional Office for Europe, ed. (2007). Health relevance of particulate matter from various sources: Report on a WHO Workshop. Bonn, Germany 26-27 March 2007. Copenhagen: World Health Organization.

World Health Organisation (1999). Environmental Health Criteria 213: Carbon Monoxide (second edition). Available at http://www.who.int/ipcs/publications/ehc/ehc_213/en/index.html [Accessed 22nd July 2011].

World Health Organisation (web site). Indoor air pollution, research and evaluation (http://www.who.int/indoorair/research/en/index.html [Accessed July 2011].

World Health Organisation- air quality guidelines. Global update 2005. http://www.who.int/phe/health_topics/outdoorair_aqg/en/ [Accessed on 22nd June 2011].

World Health Organisation. WHO guidelines for indoor air quality. (2010b). Selected pollutants. ISBN 978 92 890 0213 4. WHO, Copenhagen, Denmark.

Acronyms and Notations

µg/m³ Micrograms per meter cubed
ALRI Acute lower respiratory infections

CAFE Clean Air for Europe
CHD Coronary heart disease
CI Confidence Interval
CO Carbon monoxide
CO₂ Carbon dioxide

COMEAP Committee on the Medical Effects of Air Pollutants

COPD Chronic obstructive pulmonary disease
CRF Concentration-Response Functions
EHIA Environmental health impact assessment

E-R Exposure-response

EPA Environmental Protection Agency
ERFs Exposure-response functions
ETS Environmental tobacco smoke
EU/m³ Endotoxin unit per meter cubed

ExternE Externalities of Energy
GBD Global burden of disease

GM Geometric mean

Health and Environment Integrated Methodology and Toolbox for Scenario

HEIMTSA Assessment

HIA Health Impact Assessment

IAP Indoor Air Pollution

IAPAH Indoor Air Pollution and Health

IAQ Indoor air quality

IOM Institute of Occupational Medicine

LAL Limulus Amebocyte Lysate

INTARESE Integrated Assessment of Health Risks of Environmental Stressors in Europe

LPG Liquefied petroleum gas
LRIs Lower respiratory illnesses

n Number

NO₂ Nitrogen dioxide OR Odds Ratio

PAHs Polycyclic aromatic hydrocarbons

PM Particulate Matter

PM₁₀ Particulate matter smaller than 10 microns PM_{2.5} Particulate matter smaller than 2.5 microns

ppb Parts per billion ppm Parts per million RR Relative risk

SCHER Scientific Committee on Health and Environmental Risks

SCOTH Scientific Committee on Tobacco and Health

SFU Solid fuel use

SHCS Scottish House Condition Survey SIDS Sudden Infant Death Syndrome

TFC Total fuel consumption
WHO World Health Organisation

An Ghníomhaireacht um Chaomhnú Comhshaoil

Is í an Gníomhaireacht um Chaomhnú Comhshaoil (EPA) comhlachta reachtúil a chosnaíonn an comhshaol do mhuintir na tíre go léir. Rialaímid agus déanaimid maoirsiú ar ghníomhaíochtaí a d'fhéadfadh truailliú a chruthú murach sin. Cinntímid go bhfuil eolas cruinn ann ar threochtaí comhshaoil ionas go nglactar aon chéim is gá. Is iad na príomhnithe a bhfuilimid gníomhach leo ná comhshaol na hÉireann a chosaint agus cinntiú go bhfuil forbairt inbhuanaithe.

Is comhlacht poiblí neamhspleách í an Ghníomhaireacht um Chaomhnú Comhshaoil (EPA) a bunaíodh i mí Iúil 1993 faoin Acht fán nGníomhaireacht um Chaomhnú Comhshaoil 1992. Ó thaobh an Rialtais, is í an Roinn Comhshaoil, Pobal agus Rialtais Áitiúil.

ÁR bhfrfagrachtaí

CEADÚNÚ

Bíonn ceadúnais á n-eisiúint againn i gcomhair na nithe seo a leanas chun a chinntiú nach mbíonn astuithe uathu ag cur sláinte an phobail ná an comhshaol i mbaol:

- áiseanna dramhaíola (m.sh., líonadh talún, loisceoirí, stáisiúin aistrithe dramhaíola);
- gníomhaíochtaí tionsclaíocha ar scála mór (m.sh., déantúsaíocht cógaisíochta, déantúsaíocht stroighne, stáisiúin chumhachta);
- diantalmhaíocht:
- úsáid faoi shrian agus scaoileadh smachtaithe Orgánach Géinathraithe (GMO);
- mór-áiseanna stórais peitreail;
- scardadh dramhuisce.

FEIDHMIÚ COMHSHAOIL NÁISIÚNTA

- Stiúradh os cionn 2,000 iniúchadh agus cigireacht de áiseanna a fuair ceadúnas ón nGníomhaireacht gach bliain.
- Maoirsiú freagrachtaí cosanta comhshaoil údarás áitiúla thar sé earnáil - aer, fuaim, dramhaíl, dramhuisce agus caighdeán uisce.
- Obair le húdaráis áitiúla agus leis na Gardaí chun stop a chur le gníomhaíocht mhídhleathach dramhaíola trí comhordú a dhéanamh ar líonra forfheidhmithe náisiúnta, díriú isteach ar chiontóirí, stiúradh fiosrúcháin agus maoirsiú leigheas na bhfadhbanna.
- An dlí a chur orthu siúd a bhriseann dlí comhshaoil agus a dhéanann dochar don chomhshaol mar thoradh ar a ngníomhaíochtaí.

MONATÓIREACHT, ANAILÍS AGUS TUAIRISCIÚ AR AN GCOMHSHAOL

- Monatóireacht ar chaighdeán aeir agus caighdeáin aibhneacha, locha, uiscí taoide agus uiscí talaimh; leibhéil agus sruth aibhneacha a thomhas.
- Tuairisciú neamhspleách chun cabhrú le rialtais náisiúnta agus áitiúla cinntí a dhéanamh.

RIALÚ ASTUITHE GÁIS CEAPTHA TEASA NA HÉIREANN

- Cainníochtú astuithe gáis ceaptha teasa na hÉireann i gcomhthéacs ár dtiomantas Kyoto.
- Cur i bhfeidhm na Treorach um Thrádáil Astuithe, a bhfuil baint aige le hos cionn 100 cuideachta atá ina mór-ghineadóirí dé-ocsaíd charbóin in Éirinn.

TAIGHDE AGUS FORBAIRT COMHSHAOIL

 Taighde ar shaincheisteanna comhshaoil a chomhordú (cosúil le caighdéan aeir agus uisce, athrú aeráide, bithéagsúlacht, teicneolaíochtaí comhshaoil).

MEASÚNÚ STRAITÉISEACH COMHSHAOIL

■ Ag déanamh measúnú ar thionchar phleananna agus chláracha ar chomhshaol na hÉireann (cosúil le pleananna bainistíochta dramhaíola agus forbartha).

PLEANÁIL, OIDEACHAS AGUS TREOIR CHOMHSHAOIL

- Treoir a thabhairt don phobal agus do thionscal ar cheisteanna comhshaoil éagsúla (m.sh., iarratais ar cheadúnais, seachaint dramhaíola agus rialacháin chomhshaoil).
- Eolas níos fearr ar an gcomhshaol a scaipeadh (trí cláracha teilifíse comhshaoil agus pacáistí acmhainne do bhunscoileanna agus do mheánscoileanna).

BAINISTÍOCHT DRAMHAÍOLA FHORGHNÍOMHACH

- Cur chun cinn seachaint agus laghdú dramhaíola trí chomhordú An Chláir Náisiúnta um Chosc Dramhaíola, lena n-áirítear cur i bhfeidhm na dTionscnamh Freagrachta Táirgeoirí.
- Cur i bhfeidhm Rialachán ar nós na treoracha maidir le Trealamh Leictreach agus Leictreonach Caite agus le Srianadh Substaintí Guaiseacha agus substaintí a dhéanann ídiú ar an gcrios ózóin.
- Plean Náisiúnta Bainistíochta um Dramhaíl Ghuaiseach a fhorbairt chun dramhaíl ghuaiseach a sheachaint agus a bhainistiú.

STRUCHTÚR NA GNÍOMHAIREACHTA

Bunaíodh an Ghníomhaireacht i 1993 chun comhshaol na hÉireann a chosaint. Tá an eagraíocht á bhainistiú ag Bord lánaimseartha, ar a bhfuil Príomhstiúrthóir agus ceithre Stiúrthóir.

Tá obair na Gníomhaireachta ar siúl trí ceithre Oifig:

- An Oifig Aeráide, Ceadúnaithe agus Úsáide Acmhainní
- An Oifig um Fhorfheidhmiúchán Comhshaoil
- An Oifig um Measúnacht Comhshaoil
- An Oifig Cumarsáide agus Seirbhísí Corparáide

Tá Coiste Comhairleach ag an nGníomhaireacht le cabhrú léi. Tá dáréag ball air agus tagann siad le chéile cúpla uair in aghaidh na bliana le plé a dhéanamh ar cheisteanna ar ábhar imní iad agus le comhairle a thabhairt don Bhord.



Science, Technology, Research and Innovation for the Environment (STRIVE) 2007-2013

The Science, Technology, Research and Innovation for the Environment (STRIVE) programme covers the period 2007 to 2013.

The programme comprises three key measures: Sustainable Development, Cleaner Production and Environmental Technologies, and A Healthy Environment; together with two supporting measures: EPA Environmental Research Centre (ERC) and Capacity & Capability Building. The seven principal thematic areas for the programme are Climate Change; Waste, Resource Management and Chemicals; Water Quality and the Aquatic Environment; Air Quality, Atmospheric Deposition and Noise; Impacts on Biodiversity; Soils and Land-use; and Socio-economic Considerations. In addition, other emerging issues will be addressed as the need arises.

The funding for the programme (approximately €100 million) comes from the Environmental Research Sub-Programme of the National Development Plan (NDP), the Inter-Departmental Committee for the Strategy for Science, Technology and Innovation (IDC-SSTI); and EPA core funding and co-funding by economic sectors.

The EPA has a statutory role to co-ordinate environmental research in Ireland and is organising and administering the STRIVE programme on behalf of the Department of the Environment, Heritage and Local Government.



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