

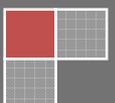
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# **Integrated Exposure for Risk Assessment in Indoor Environments (INTERA)**

## **A review of existing indoor air pollutant exposure data and models**

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## EXECUTIVE SUMMARY

The Integrated Exposure for Risk Assessment in Indoor Environments (INTERA) project was commissioned by CEFIC under the Long Range Research Initiative to develop novel methods for the integration of exposure and risk information relating to household air pollutants within EU home settings. This report represents a distillation of Work Package 1 (WP1) and presents information on a review of the scientific literature of inhalation, dermal and ingestion exposures to indoor pollutants in domestic environments within the past 15 years. The review additionally identifies existing and developing indoor pollutant modelling methods across all exposure routes.

Using a systematic approach and the online database Ovid Medline, a total of 57 scientific publications relevant to exposures generated by the use of household consumer products have been identified and reviewed. Additional material relevant to mould, biological material and fine particulate matter have also been generated although not reviewed for the purposes of the INTERA project.

A total of 29 indoor pollutant modelling methods are presented. Several of these are still under development. These models range from broad risk characterisation models through to exposure modelling systems for specific tasks.

The output of this review and a series of project meetings/expert webinars has been the establishment of an exposure determinant matrix for the main indoor pollutant chemical groups that will be considered by future work packages in the INTERA project. These groups are: radon, carbon monoxide, carbon dioxide, nitric oxides, polycyclic aromatic hydrocarbons

(PAHs), aldehydes, polybrominated diphenyl ethers (PBDEs), nicotine and volatile organic compounds (VOCs). The exposure determinant matrix and information on measurements and modelling methods will be utilised in the further development of a knowledge management system for the full chain approach that INTERA will take to characterize exposure and risk in indoor environments.

# 1. INTRODUCTION

## Project rationale

Exposure to indoor air pollutants is one of the primary environmental health stressors, especially given that people in developed economies tend to spend approximately 80-90% of their time within enclosed living spaces (Klepeis et al, 2001). Concentrations of indoor air pollutants are determined by the ability of outdoor source pollutants to infiltrate indoors and emissions from indoor sources such as building materials, furniture, burning of solid fuels, smoking (environmental tobacco smoke), electric appliances, use of cleaning products and other household chemicals, and indoor climatic variables. The variability of species and the levels of contamination within and between different indoor microenvironments can be very wide and this poses real problems for modeling and exposure estimation. Personal exposure levels are likely to vary even more widely given differences between and within individuals in behaviour and activities.

Moreover, three key parameters significantly increase the levels of uncertainty and variability for the characterization of exposure and risk in an indoor microenvironment:

- Possible interactions among the several contaminants, including indoor reactive chemistry before exposure, or after inhalation (toxicodynamics).
- Toxicity pathways that may be activated due to the cumulative exposure-mixture effect of contaminants that, when assessed individually, are within the proposed safety limits.
- The susceptibility of different populations to several contaminants. For example, exposure levels met in everyday settings may pose some risk for specific groups (e.g. young children or asthmatics), especially when considering the above two assumptions.

The characterization of an indoor microenvironment solely by monitoring levels of single contaminants and linking them to epidemiological data is an inadequate approach if protection of both individuals and the wider community population is to be achieved. In order to better understand, quantify and characterize an indoor environment, a full chain mechanistic approach is needed, dealing with several sub-tasks. These include:

- The relations among the sources of contamination (outdoor and indoor), and the levels of indoor contamination.
- After identifying the sources and the emissions strength, air pollution modeling is needed, including consideration of reactive chemistry of indoor contaminants in order to calculate the spatial and temporal pattern of the indoor concentrations.
- The actual exposure to indoor contamination. Time activity patterns need to be defined in order to link them properly to the spatial and temporal course of indoor contamination. Details of how time-activity patterns and behaviours differ between age groups, family structures and geographical location are vital to understand the variability in personal exposures. Data on product use is also essential. Moreover, the procedure needs to be conducted for the majority of the known contaminants in order to assess the cumulative exposure.
- Internal dose modelling is an essential element for describing these kinds of dynamic procedures. Physiology Based Toxicokinetic (PBTK) modelling is necessary to dynamically describe the fate of the contaminant (absorption, distribution, metabolism, excretion) in the human body. Moreover, possible interactions among the several contaminants (mixture effect) can be implemented, accounting, as much as possible, for accumulation based on the levels and patterns of exposure (single and repeated events, continuous).

- Health outcome assessments should be rather based on biology than statistics when adequate background knowledge exists. Biology Based Dose Response (BBDR) ensures the proper exploitation of the internal dose obtained by the PBTK. The effect of internal dose temporal variation (including “peaks”) is translated into meaningful effects.

The above bullet points represent a ‘full chain approach’ which the INTERA project will aim to implement within a dynamic simulation environment.

The main objective of INTERA is to define optimal methodologies for predicting indoor exposure to chemical and non-chemical contaminants and their inter-relationships. The project design includes the following elements:

- The characterisation and justification of a framework capable of being applied to indoor exposure data/information and covering parameters relevant to their wider interpretation.
- The development/incorporation of appropriate databases of quality assured source data.
- The development/incorporation of suitable models and statistical methodologies for the characterization and treatment of such data.
- The application of suitable models and/or statistical methods that serve to either fill gaps or offer refined exposure assessment where uncertainties are considered unacceptable.
- The ability to display exposure predictions in a number of formats in order that they can be better applied within the context of both research and policy development.

To achieve these outputs the INTERA project team has defined the following sub-objectives:

- 1) Determine the main parameters influencing exposure
- 2) Review and collate existing indoor exposure data, including the most prominent indoor exposure studies in Europe.

- 3) Collate all the above data and organise them into a comprehensive database/knowledge management system.
- 4) Develop full chain models using exposure reconstruction algorithms to fill data gaps and support refined exposure assessment.
- 5) Display exposure predictions at different spatial and temporal scales.
- 6) Implement the integrated approach in 3 case studies.
- 7) Disseminate research findings

This report is the output of sub-objectives 1 and 2 above and is the culmination of Work Package 1 of the INTERA project.

## 2. AIMS

Work Package 1 (WP1) of the INTERA project had the following primary aims:

- To identify the main studies describing inhalation, dermal and ingestion exposure to, or determinants of, indoor domestic pollutants published in the past 15 years;
- To identify ongoing studies and indoor exposure models for domestic environments;
- To identify and review existing and developing indoor pollutant modeling approaches;
- To develop a summary matrix of the exposure determinants for various indoor air pollutants used in existing models and previous studies.

## 3. METHODS

### 3.1 Literature review search strategy: inhalation exposure

A search of Ovid Medline was performed, limited to studies undertaken since 1995, with full text published in English, and involving human beings, which yielded the following number of scientific papers:

- Indoor air pollution with:
  - Homes (680 hits)
  - Domestic (239 hits)
  - Determinants (87 hits)

The remit of this review was to examine studies that had been carried out in countries that were members of the European Union (EU) (see Appendix). Studies that did not collect primary data in at least one home or domestic indoor environment were also excluded. Search results were cross-checked to eliminate duplicates. Following this process a total of 195 publications were exported into an online bibliographic database (RefWorks, Proquest Inc) for further scrutiny.

All 195 publication titles were examined, and at this stage a further 65 were discarded as not relevant to the study themes. The remaining 130 publications were separated into three categories: 46 in 'chemical'; 12 in 'particulates'; and 72 in 'biological'. Discussions at the INTERA project meetings in February, May and September 2010, together with input across the 3 webinars held in June-July 2010, finalized that the review would concentrate on publications identified as investigating concentrations of consumer product chemicals within domestic

homes. As a result the 84 identified publications classified as examining either biological or solely particulate exposure data were not included in the literature review.

Electronic copies of all 46 remaining papers or abstracts were sourced and the reference lists of these were hand checked for additional relevant publications. This revealed a further 20 possibly relevant papers for scrutiny. On further examination of the 46 papers examining chemicals, three were moved to the 'particulates' category, three did not collect indoor air measurements, and one had only the abstract published in the English language. The 20 additionally identified publications were examined and 5 were included in the chemical category. This gave a total of 44 papers to be reviewed. As an additional cross check, a search of the Ref Works database was performed using each pollutant as a search term and results compared with the 44 papers identified at the initial search. Two duplicate papers were discovered at this point due to different abbreviations being used and these were therefore removed from the database, leaving 193 papers. Figure 1 on page 14 presents the results of this search, which led to the inclusion of a further 5 papers into the review, giving a final total of 49 reviewed papers.

Several of these 49 papers examined more than one indoor air pollutant/chemical.

### 3.2 Literature review search strategy: ingestion/dermal exposure

A similar search strategy was employed to review the literature on ingestion and dermal exposure within domestic environments. In addition to a literature search of Ovid Medline, the PubMed database was also examined using the following search terms:

	(Medline)	(Pub Med)
• Indoor Air Pollution with:		
• Dermal exposure	(7 hits)	(52 hits)
• Ingestion	(69 hits)	(98 hits)

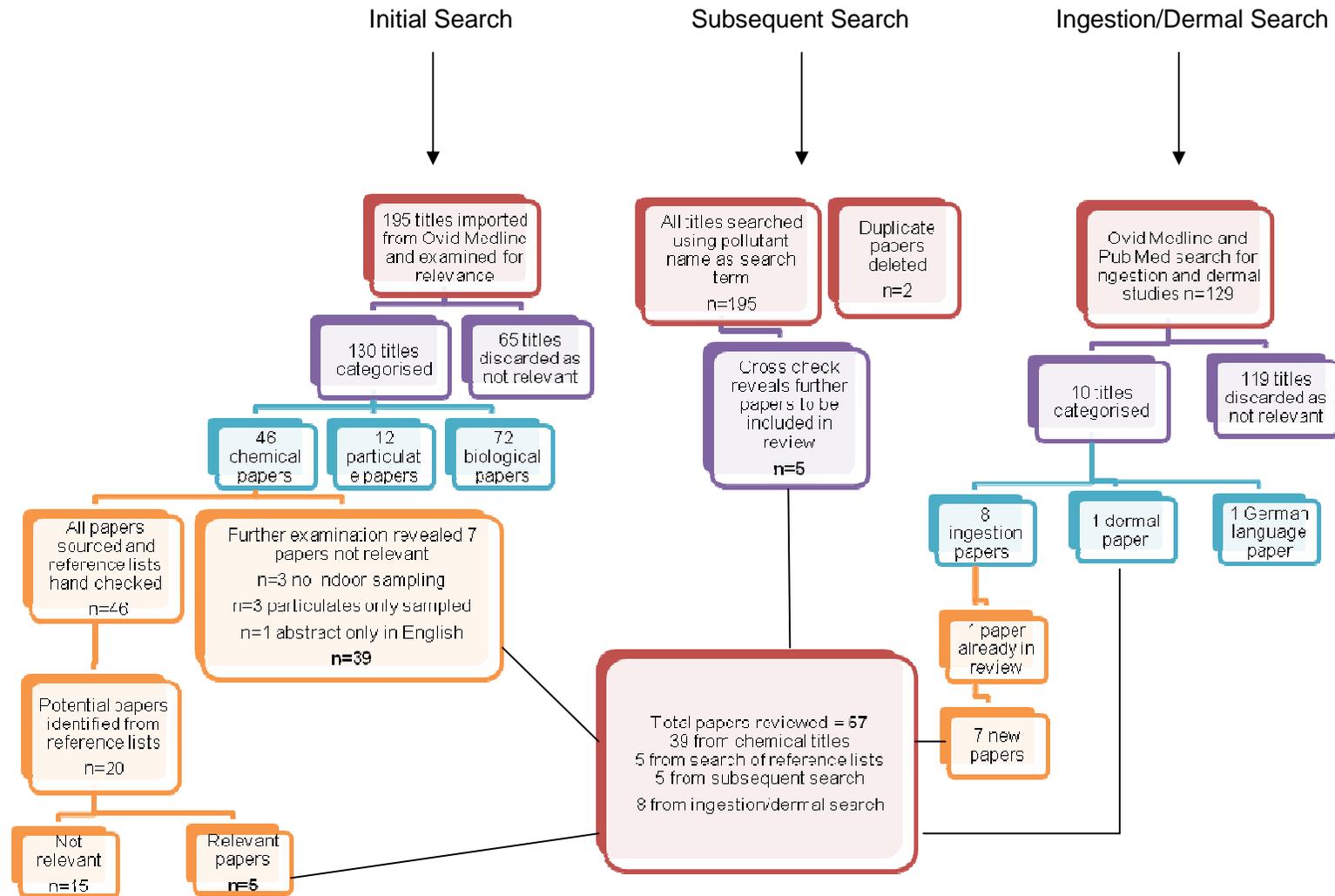
Other combinations of search terms used were as follows:

• Dermal exposure + domestic	(7 hits)	(26 hits)
• Dermal exposure + homes	(5 hits)	(7 hits)
• Dermal exposure + determinants	(33 hits)	(57 hits)
• IAP + dermal exposure + homes	(8 hits)	(8 hits)
• IAP + dermal exposure + domestic	(0 hits)	(2 hits)
• IAP + dermal exposure + determinants	(0 hits)	(2 hits)
• IAP + ingestion + domestic	(5 hits)	(7 hits)
• IAP + ingestion + homes	(16 hits)	(23 hits)
• IAP + ingestion + determinants	(1 hit)	(1 hit)

All titles from the Medline search were initially imported into the Ref Works database and alphabetized to enable the removal of duplicates. The results of the Pub Med searches were not

easily imported into the database and therefore these titles were imported into Excel where they were alphabetized and hand checked against the Medline results. After excluding all titles prior to 1995, 129 papers remained on the database for further scrutiny. All abstracts were checked to determine whether the work was carried out within European Union countries and involved humans. Studies not conducted in the EU were excluded, together with studies that looked only at occupational exposures. This left 10 full articles to be sourced for possible inclusion in the review. One of these papers was found to be published in German and therefore not included in this review, while one was already included under the inhalation category and was re-examined to extract information on ingestion exposure. The remaining 8 papers have been summarised in the results section of this report.

**Figure 1: Flow chart of search strategies used**



### **3.3 Review of current studies and existing models of indoor pollutant levels**

The search strategy for this component of the work package involved examining the grey literature and accessing literature and project reports available from academic institutions, funding bodies and other organizations with a research or consumer interest in indoor air quality (IAQ). From a list of IAQ models identified by the INTERA project team, an internet search was initially conducted using Google and Google Scholar search engines and then each specific source was checked for additional links, employing a 'snowball' technique. In addition to the collective experience and knowledge of the INTERA team, the main 'snowball' starting points were the US EPA IAQ webpage (<http://www.epa.gov/iaq>), the book "Exposure Analysis" (edited by Ott, Steinemann and Wallace, 2006, page 459), reports from the HEIMSTA project, (Health and Environment Integrated Methodology and Toolbox for Scenario Assessment) available from <http://www.heimtsa.eu/> and specific papers where indoor models have been used such as EXPOLIS from the project website at <http://www.ktl.fi/expolis>. In addition, 2 review studies, namely 1) a review performed in the framework of the EnVie study (de Oliveira Fernandez et al., 2009), and 2) a comprehensive overview of IAQ modeling techniques in the report of Milner et al. (2005), were also used as data sources for IAQ models discussed in this chapter.

The Results section of this report (Section 4) provides an overview of each study/model, together with links to web pages with embedded publications and final reports where available. In a first section, IAQ modeling techniques are discussed. In the second part, studies related to risk assessment of IAQ or databases used to populate IAQ modeling techniques (e.g. emission database) are discussed. The latter two types of studies do not fall strictly under the definition of 'IAQ models'; however, inclusion of these types of studies is justified because the output of IAQ modeling is to be used in risk assessment, and so the usefulness of IAQ modeling is put into perspective.

## 4. RESULTS

### 4.1 Indoor air pollutant levels measured in homes

#### Radon

Radon is a colourless, odorless noble gas, a decay product of uranium that occurs naturally in all rocks and soils in many areas of the world. Radon concentrations in outdoor air are generally low, but in some geological conditions radon can build up in confined areas such as basements of houses. It is estimated that annual mortality from exposure to radon in buildings accounts for 9% of lung cancer deaths and around 2% of all cancer deaths in the UK (Darby 2005). The UK Health Protection Agency (HPA) reports that the average radon concentrations in most UK homes is around 20 becquerels per cubic metre (Bq/m<sup>3</sup>) (HPA, 2011), and the National Radiological Protection Board (NRPB) advise that homes above an Action Level of 200 Bq/m<sup>3</sup> should take steps to reduce air concentrations (NRPB, 1990).

The UK Childhood Cancer Study (UK Childhood Cancer Study, 2002), a case-control study of 2226 cases and 3773 controls found no evidence of an association between higher radon concentrations and risk of any childhood cancers. This study collected measurements in homes using passive radon detectors. The overall mean radon concentration found in this study was 24 Bq/m<sup>3</sup>, with averages of 16.1 Bq/m<sup>3</sup> and 27.2 Bq/m<sup>3</sup> in most and least deprived homes respectively, showing a positive association between radon concentration and affluence.

Similar levels of indoor radon were found in a case-control study by Kaletsch carried out in Lower Saxony, Germany (Kaletsch et al. 1999). The overall median level of radon was 27 Bq/m<sup>3</sup> across homes of 209 controls and 164 cases of cancer including leukaemias and central nervous system (CNS) tumours. A study based in the Isle of Man found indoor radon exposure

at 48 Bq/m<sup>3</sup> to be around twice that of the UK average, but there was no statistical difference between the NRPB and Isle of Man seasonal levels (Grainger et al. 2000).

In the radon affected area of Northampton, UK, Briggs obtained data on radon concentrations in a representative home over an 18-day period (Briggs et al. 2003) and found an average hourly ground floor radon concentration of 467 Bq/m<sup>3</sup>. After applying time-activity modeling techniques, average hourly exposures ranged from around 250-340 Bq/m<sup>3</sup>. Another study in Northamptonshire, UK looked at radon levels measured before and after remediation measures were taken in a single home (Denman et al. 2008). Hourly measurements were taken on two floors over a 5 week period in this home situated in a designated radon affected area where previously measured levels had been >200 Bq/m<sup>3</sup>. After remediation, levels in this typical house fell to 25% of those measured previously, with diurnal variability basically eliminated, and personal exposure found to be directly proportional to time spent in the house, regardless of time of day.

Table 1 below provides a summary of the radon concentrations found in the papers reviewed in this report.

**Table 1: Summary of radon papers**

Country	Year pub	Domestic Environment	Averaging time	No samples/ persons	Concentration (Bq/m <sup>3</sup> ) <sup>†</sup>				Reference
					Mean	Median	Min	Max	
Germany	1999	Children <15 years Cases leukaemia	12 months	82	26.4	22			Kaletsch 1999
		Children <15 years Cases tumour		82	33.1	23.5			
		Children <15 years Controls		209	28.5	21			
UK: Isle of Man	2000	Living room and bedroom	3 months	285	48	26	4	518	Grainger 2000
UK	2002	Children <15 years*	6 months 6 months	2226 cases 3773 controls	21.1 25.5				UK CCS, 2002**
UK	2003	Kitchen (home with cellar)	24 hours	1	467				Briggs 2003
UK	2008	Upstairs before remediation	24 hours	1	299.06				Denman 2008
		Downstairs before remediation	24 hours	1	370.14				
		Upstairs after remediation	24 hours	1	99				
		Downstairs after remediation	24 hours	1	65.71				

<sup>†</sup>becquerels per cubic metre; \*diagnosed with malignant disease; \*\*United Kingdom Childhood Cancer Study  
Unless otherwise stated, mean values given are arithmetic means; empty cells indicate this information not available

## **Carbon monoxide**

Carbon monoxide (CO) is a colourless, odourless poisonous gas which is produced when carbon-based fuels such as gas, oil, wood and coal fail to burn properly, producing excess CO. Inhaling this toxic gas reduces the blood's ability to carry oxygen, thus depriving the brain and other organs of oxygen and can lead to nausea, unconsciousness and death.

Henderson and colleagues took continuous measurements of CO in 44 non-smoking homes in order to determine whether the World Health Organisation's (WHO) limits of  $7\mu\text{g}/\text{m}^3$  over 24 hours (WHO, 2010) are adhered to in domestic environments (Henderson et al., 2006). They found the homes monitored did not exceed 1ppm over the monitoring period while in an additional 20 homes with a resident smoker, values ranged from 0.1-21ppm.

As part of a study to validate a model designed to measure the Chance of Accumulation of Combustion Products (CACP), real-time CO measurements were made in 74 homes in the Netherlands over a one week period (Willers et al. 2006). This model included information on gas cooking, effectiveness of extractor fans, kitchen volume, air supply/ventilation in the kitchen and the presence of other sources of combustion. Mean levels of CO were mostly below the limit of detection but in homes with gas cookers or a high CACP, significantly higher concentrations were found (0.50ppm versus 0.03ppm), although the results indicate there was little incomplete gas combustion in these study homes.

Matthews et al were interested in the effect different types of residential heating would have on indoor CO levels (Matthews et al. 2010). They measured CO over one week during winter and found concentrations were below 1ppm, making it difficult to reach conclusions over different heating types based on CO exposure alone.

Table 2 below provides a summary of the CO concentrations found in the papers reviewed in this report.

**Table 2: Summary of Carbon monoxide papers**

Country	Year pub	Domestic Environment	Averaging time	No samples/ persons	Concentration (ppm) <sup>†</sup>				Reference
					Mean	Median	Min	Max	
The Netherlands	2005	Gas kitchens	7 days	50	0.50				Willers 2006 (PIAMA study)
		Electric kitchens		19	0.03				
		All kitchens		69	0.37				
UK	2006	Non-smoking	7 days	44	<1			Henderson 2006*	
		Smoking	2 days	20	0.1-21				
UK	2010	Winter	7 days		<1			Matthews 2010*	

<sup>†</sup>parts per million; \*from abstract only

Unless otherwise stated, mean values given are arithmetic means; empty cells indicate this information not available

## **Carbon dioxide**

Carbon dioxide (CO<sub>2</sub>) is expired by humans as part of the metabolic process and is also one of the primary products of combustion. The National Institute for Occupational Safety and Health (NIOSH) suggests indoor concentrations of more than 1000ppm are indicative of inadequate ventilation.

To further test the CACP model described in relation to CO, Willers measured combustion-generated CO<sub>2</sub> in 69 homes in the Netherlands, using a CO<sub>2</sub> probe with a measuring range of 0-10,000ppm (Willers et al. 2006). Overall mean levels of 659ppm were found and although there was no measured difference between homes with gas or electric cookers, during actual cooking there was a significant difference found for gas vs electric cooking appliances ( $t=2.32$ ,  $p=0.02$ ), indicating that concentrations were higher in gas homes.

Researchers from Sweden were interested in the effect of indoor CO<sub>2</sub> on asthma symptoms (Norback et al. 1995). Subjects were a sub-sample of a European Commission multi-centre study who had reported at least one respiratory symptom in the last 12 months, matched to randomly selected non-symptomatic individuals. Air monitoring of 88 homes was performed in both the living room and bedroom of the participants, with CO<sub>2</sub> measured using a direct reading infrared spectrometer. Symptoms were assessed using a modified but validated respiratory health survey: the International Union against Tuberculosis and Lung Disease (IUATLD) questionnaire (Burney et al. 1989). Average concentrations of CO<sub>2</sub> was significantly higher in homes of subjects who reported nocturnal chest tightness (1020ppm vs 850ppm), with the recommended level of 1000ppm being exceeded in 26% of homes measured. After adjusting for factors such as age, sex, smoking status, presence of wall to wall carpets and house dust mites, the odds ratio for an increase in CO<sub>2</sub> by 1000ppm was 20.0 (95% CI 2.7-146) for nocturnal breathlessness.

Table 3 below provides a summary of the CO<sub>2</sub> concentrations found in the papers reviewed in this report.

**Table 3: Summary of carbon dioxide papers**

Country	Year pub	Domestic Environment	Averaging time	No samples/ persons	Concentration (ppm) <sup>†</sup>				Reference
					Mean	Median	Min	Max	
Sweden	1995	Nocturnal shortness of breath	30 mins	26	1020				Norback 1995
		No nocturnal shortness of breath		62	850				
The Netherlands	2005	Gas kitchens	7 days	50	654.16				Willers 2006 (PIAMA study)
		Electric kitchens		19	671.47				
		All kitchens		69	658.93				

<sup>†</sup>parts per million

Unless otherwise stated, mean values given are arithmetic means; empty cells indicate this information not available.

### **Nitrogen dioxide / Nitrous oxide / Nitrous acid**

Nitrogen dioxide (NO<sub>2</sub>) is an irritant gas which at high concentrations can cause inflammation of the airways and lower resistance to respiratory infections, although there is conflicting evidence of its effect on the adult respiratory system. Indoor levels of NO<sub>2</sub> generally reflect the use of combustion appliances such as gas fires and cookers.

There is agreement among researchers that indoor concentrations of NO<sub>2</sub> are influenced both by outdoor levels and by the presence of gas appliances in the home (Mosqueron et al , 2002, Valero et al. 2009). Garcia-Algar however found that cigarette smoking, season of measurement, and the energy source were the main determinants of indoor concentrations (Garcia Algar et al. 2004). NO<sub>2</sub> was measured in living rooms in homes in Ashford, Barcelona and Menorca over 7-15 days with median levels found of 5.79ppb, 23.87ppb and 6.06ppb respectively. All three centres showed increased levels when gas was used either for heating or cooking. After adjusting for potential predictors, 52% of the variability in indoor NO<sub>2</sub> concentration could be explained by these significantly related variables.

Willers conducted passive measurements of NO<sub>2</sub> in kitchens of 55 homes in the Netherlands over a one week sampling period (Willers et al. 2006). Values reported for 48 hour average NO<sub>2</sub> concentrations were 27µg/m<sup>3</sup> for gas cooking homes and 22µg/m<sup>3</sup> for homes that used electricity. The authors found these concentrations to be lower than previous studies in the Netherlands.

Valero found that personal exposure of pregnant women to NO<sub>2</sub> was influenced both by outdoor and indoor levels in an area of Spain (Valero et al. 2009). Using Radiello samplers, 48 hour concentrations were measured in the homes of 108 pregnant women in two areas of Spain. Median levels of 36µg/m<sup>3</sup> and 32µg/m<sup>3</sup> were found in Valencia and Sabadell respectively. In a

multiple regression model, 32% of indoor NO<sub>2</sub> variability was explained by outdoor concentrations and the presence of gas appliances. Hagenbjork-Gustafsson studied 46 homes without gas appliances in Sweden and found higher concentrations in less urbanized control homes than those in a nearby urban area (Hagenbjork-Gustafsson et al. 1996). Twenty-four hour mean concentrations were 12µg/m<sup>3</sup> and 6µg/m<sup>3</sup> for urban and control areas. The researchers concluded that when no gas appliances are present, indoor concentrations reflect outdoor levels.

Sakai found a geometric mean concentration of 6.7µg/m<sup>3</sup> in 27 homes in Uppsala, Sweden, when measured over 24 hours (Sakai et al. 2004). The outdoor concentration was the same, indicating there were no important indoor sources impacting on NO<sub>2</sub> levels. This finding was consistent with the reported absence of ETS and lack of gas cooking among these homes.

One of the largest databases of NO<sub>2</sub> measurements was developed and populated within the framework of the Swiss Study on Air Pollution and Lung Diseases in Adults (SAPALDIA) (Monn et al. 1998). Bedrooms in a sub sample of 500 homes were measured over 1 week, as well as personal and outdoor measurements, to evaluate the relationship between personal levels with indoor and outdoor concentrations. Overall average indoor levels were 21µg/m<sup>3</sup>. Levels were significantly higher in homes with gas cookers (5-8µg/m<sup>3</sup>). Overall personal levels found were 27µg/m<sup>3</sup> and outdoor 31µg/m<sup>3</sup>.

Residential exposures of adults taking part in the EXPOLIS study were measured over 48 hours in homes in Switzerland, Finland and the Czech Republic (Kousa et al. 2001). Average levels were 36µg/m<sup>3</sup>, 27µg/m<sup>3</sup> and 30µg/m<sup>3</sup> in Basle, Helsinki and Prague respectively. These results are slightly higher than those measured in the SAPALDIA study mentioned above.

The Indoor Factors and Genetics in Asthma (INGA) study performed repeated indoor NO<sub>2</sub> measurements 6-13 months apart in 631 German homes (Topp et al. 2004). Passive samplers were placed in living rooms and bedrooms for a one week period on each occasion. Living room measurements were 14.9µg/m<sup>3</sup> on visit one and 15.2µg/m<sup>3</sup> on the second visit. Within-home correlations, after adjusting for region and season, ranged between 0.24-0.55, and they concluded that a single measurement was not sufficient to take account of variability over time in a moderate climate like Germany. Also in Germany, indoor concentrations of NO<sub>2</sub> were measured in living rooms and bedrooms in 204 homes in Erfurt and 201 homes in Hamburg (Cyrys et al. 2000). The authors found living room concentrations to be 15µg/m<sup>3</sup> and 17µg/m<sup>3</sup> in each of the two cities and in bedrooms levels were 15µg/m<sup>3</sup> and 18µg/m<sup>3</sup> respectively. When a linear regression analysis was applied, including variables such as place of residence, season, location of home to busy roads and outdoor levels, the major indoor sources in living room concentrations remained gas cooking and the presence of smokers.

In Denmark, researchers looked at the impact of outdoor temperature on indoor NO<sub>2</sub> levels and found significantly higher values during the cold season (Sorensen et al. 2005). Median NO<sub>2</sub> concentrations found in Copenhagen homes were 8.9µg/m<sup>3</sup> below 8°C and 6.6µg/m<sup>3</sup> when the temperature was above this level.

Other studies have looked at the effects of NO<sub>2</sub> on respiratory health (Jarvis et al. 2005, Osman et al. 2007, Venn et al. 2003). Osman et al measured NO<sub>2</sub> in the homes of 148 patients with Chronic Obstructive Pulmonary Disease (COPD) and found concentrations over one week to be 7.8ppb in the living room and 7.1ppb in the bedroom (Osman et al. 2007). They did not find an association with indoor NO<sub>2</sub> and health status in this patient group. Similarly, Venn found no effect of NO<sub>2</sub> on wheezing illness in children when measured objectively in the homes of cases and controls in Nottingham, UK (Venn et al. 2003). Jarvis measured indoor NO<sub>2</sub> and nitrous acid

(HONO) in over 200 hundred homes as part of the EC Respiratory Health Survey (Jarvis et al. 2005). While no significant association of indoor NO<sub>2</sub> with symptoms or lung function was found, for HONO an increase of 1ppb was associated with a decrease in forced expiratory volume in one second (FEV<sub>1</sub>) and a decrease in percentage of FEV<sub>1</sub>/FVC (forced vital capacity) after adjusting for confounders.

Looking at personal exposure in Lille, France Piechocki-Minguy used passive samplers to elicit exposure data in 46 volunteers over four micro-environments: home; other indoor places; transport; and outdoors (Piechocki-Minguy et al. 2006). Mean NO<sub>2</sub> concentrations for home environment differed between summer and winter and between weekdays and weekends, with subjects reporting spending between 62-88% of their time at home dependent on day of the week. Although the highest levels found were in the transport and outdoor micro-environment, the main contributor to total NO<sub>2</sub> personal exposure was found to be indoor pollution due to the high percentage of time spent indoors in this population.

Table 4 below provides a summary of the NO<sub>2</sub> concentrations found in the papers reviewed in this report.

**Table 4: Summary of nitrogen dioxide papers**

Country	Year pub	Domestic Environment	Averaging time	No samples/ persons	Concentration ( $\mu\text{g}/\text{m}^3$ ) <sup>†</sup>				Reference
					Mean	Median	Min	Max	
Sweden	1996	Urban bedrooms Less urban bedrooms	24 hours	23 20	12 8		6 6	25 26	Hagenbjork-Gustafsson 1996
Switzerland	1998	Bedrooms Gas cooking Geneva Gas cooking Basle Gas cooking Lugano	7 days	>500	21 40 29 36				Monn 1998 (SAPALDIA)
Germany	2000	Living rooms Erfurt Bedrooms Erfurt Living rooms Hamburg Bedrooms Hamburg All homes winter All homes summer	7 days	204 204 201 201 199 205	15 15 17 18		14.6 17.5		Cyrus 2000
Switzerland Finland Czech Republic	2001	Homes	48 hours	50 175 33	36 27 30				Kousa 2001
France	2002	Living rooms	48 hours	62	35.1	32.5	14	855	Mosqueron 2002
UK	2003	Bedroom; child wheeze Bedroom; child no wheeze	4 weeks	193 223			0 0	>58 >58	Venn 2003*
Germany	2004	Visit 1 living room Visit 2 living room Visit 1 bedroom Visit 2 bedroom	1 week	631 631 631 631		14.9 15.2 13.8 14.5		60.9 83.8 52.1 76.2	Topp 2004 (INGA)
UK & Spain	2004	Living room Ashford UK  Living room Menorca Living room Barcelona	7-15 days	642  456 340		5.53  5.79 22.82	0.27  0.20 1.19		Garcia-Algar 2004

**Table 4 (contd): Summary of nitrogen dioxide**

Country	Year pub	Domestic Environment	Averaging time	No samples/ persons	Concentration ( $\mu\text{g}/\text{m}^3$ ) <sup>†</sup>				Reference
					Mean	Median	Min	Max	
Sweden	2004	Bedrooms	24 hours	27	6.7				Sakai 2004
UK	2005	Kitchen	14 days	255		24.55	5.4	113.7	Jarvis 2005
Denmark	2005	Bedroom; outdoor temp <8°C	48 hours	38		8.9			Sorensen 2005
		Bedroom: outdoor temp >8°C		35		6.6			
The Netherlands	2006	Gas kitchens	7 days	49	26.59				Willers 2006** (PIAMA study)
		Electric kitchens		19	22.41				
		All kitchens		68	25.35				
France	2006	Winter working day	24 hours	13	22	19	11	38	Piechocki-Minguy 2006
		Winter weekend		13	27	20	10	60	
		Summer working day		31	14	11	5	24	
		Summer weekend		31	15	13	5	44	
UK	2007	Bedrooms COPD patients	7 days	148	6.8				Osman 2007
		Living rooms COPD patients			7.5				
Spain	2009	Valencia	48 hours	50		36			Valero 2009
		Sabadell		58		32			

<sup>†</sup>Micrograms per cubic metre; \*reported as no of cases/controls exposed to scale of levels of NO<sub>2</sub>; \*\*geometric mean. Unless otherwise stated, mean values given are arithmetic means; empty cells indicate this information not available.

## Aldehydes

Aldehydes are organic carbonyl compounds found in both indoor and outdoor environments. The main indoor sources that may impact on human health are acetaldehyde and formaldehyde, commonly found in consumer products for cleaning and personal hygiene, building materials, furniture made with pressed wood products, tobacco smoke and combustion sources. Domestic exposure on a large scale has not been fully explored but formaldehyde is known to be an irritant to eyes and airways.

Several studies have measured aldehydes in domestic homes, the most commonly measured being formaldehyde, acetaldehyde, pentanal and hexanal (Dassonville et al. 2009, Clarisse et al. 2003, Marchand et al. 2008). Using Radiello passive samplers, Dassonville measured five aldehydes on several occasions over the course of one year in the bedrooms of 196 Parisian infants randomly selected from a birth cohort study (Dassonville et al. 2009). The geometric mean concentrations for the four main aldehydes were  $19.4\mu\text{g}/\text{m}^3$  for formaldehyde,  $8.9\mu\text{g}/\text{m}^3$  for acetaldehyde,  $3.7\mu\text{g}/\text{m}^3$  for pentanal and  $25.3\mu\text{g}/\text{m}^3$  for hexanal. The main sources were building materials including new coverings, smoking and the use of air fresheners. They also found seasonal variability with higher levels of formaldehyde observed in warmer months. Another study measured aldehydes in Paris apartments where refurbishment had taken place within the previous three years (Clarisse et al. 2003). Measurements were carried out simultaneously in the living room, kitchen and bedroom over a 72 hour period. Concentrations in bedrooms were slightly higher than those found by Dassonville but their findings concur with regard to the main sources, ie floor/wall covering age, smoking and ambient parameters such as temperature and humidity. Clarisse found no difference between the concentrations in the different rooms measured and therefore concluded that one sample only was needed to measure indoor exposure.

Formaldehyde was measured in the homes of a sub-sample of a large-scale population study carried out in Germany in 1991-1992 (Seifert et al. 2000). Mean indoor concentrations over a 48-hour period were reported as being  $>70\mu\text{g}/\text{m}^3$ , although half the samples were above this and 14% exceeded the German indoor guideline for formaldehyde of 0.1ppm. Marchand et al found mean concentrations of  $32.2\mu\text{g}/\text{m}^3$ ,  $14.3\mu\text{g}/\text{m}^3$  and  $8.6\mu\text{g}/\text{m}^3$  for formaldehyde, acetaldehyde and hexanol respectively in 162 homes in Strasbourg which they regarded as similar to other studies in the United States and Australia (Marchand et al. 2008). In living rooms the age of ceiling covering was found to be the main explanatory variable, while in bedrooms it was the age of the furniture. Around 7% of the homes in this study had formaldehyde concentrations  $>50\mu\text{g}/\text{m}^3$ .

Similarly in Bari, South Italy, Lovreglio conducted passive monitoring of formaldehyde and acetaldehyde in 59 homes over a 24 hour period, reporting concentrations of  $16\mu\text{g}/\text{m}^3$  and  $10.7\mu\text{g}/\text{m}^3$  respectively (Lovreglio et al. 2009). This compared to outdoor concentrations of  $4.4\mu\text{g}/\text{m}^3$  and  $3.4\mu\text{g}/\text{m}^3$  for formaldehyde and acetaldehyde respectively. Levels were higher in the presence of new or newly restored furniture ( $p=0.03$ ) and while levels were significantly higher in winter months, they found no association with irritant or allergic complaints, concluding that such low concentrations could be considered not to pose a risk to human health. Venn, however, found that among cases of wheezing illness in children, formaldehyde was associated with more frequent nocturnal symptoms (OR: 1.45, 95% CI 1.06-1.98) (Venn et al. 2003).

Studies of domestic formaldehyde concentrations in Sweden found levels to be within the Swedish guidelines of  $12\text{-}60\mu\text{g}/\text{m}^3$  (Norback et al. 1995, Gustafson et al. 2005, Gustafson et al. 2007). One study by Gustafson measured bedroom concentrations in two campaigns: 24 hour concentrations were  $23\mu\text{g}/\text{m}^3$  while 6-day sampling returned a mean of  $29\mu\text{g}/\text{m}^3$  which was slightly higher than personal exposure levels recorded in 24 and 40 subjects over the two

sampling campaigns (Gustafson et al. 2005). Another study by the same researchers looked at concentrations of formaldehyde and acetaldehyde over winter in 14 wood-burning and 10 control homes in a small Swedish town (Gustafson et al. 2007). They found no significant difference between wood burners and control homes for either aldehyde.

Sakai reported a geometric mean concentration for formaldehyde of  $8.3\mu\text{g}/\text{m}^3$  measured over 24 hours in 27 Swedish bedrooms in Uppsala which was significantly higher than the outdoor concentration ( $p<0.01$ ) (Sakai et al. 2004). This study compared homes in Sweden to homes in Japan.

Norback only found one home that exceeded the earlier Swedish limit of  $100\mu\text{g}/\text{m}^3$  in his sample of 88 random subjects in Uppsala (Norback et al. 1995). Comparing subjects with and without asthma symptoms, Norback observed an adjusted odds ratio of 12.5 (95% CI: 2.9-77.9) for nocturnal breathlessness in relation to indoor concentrations of formaldehyde.

Table 5 below provides a summary of the aldehyde concentrations found in the papers reviewed in this report.

**Table 5: Summary of aldehyde papers**

Country	Year pub	Domestic Environment	Averaging time	No samples/ persons	Concentration ( $\mu\text{g}/\text{m}^3$ ) <sup>†</sup>				Reference
					Mean	Median	Min	Max	
Sweden	1995	Bedroom formaldehyde with nocturnal sob**	2 hours	26	29		<5	110	Norback 1995
		Bedroom formaldehyde no nocturnal sob**		62	17		<5	60	
Germany	2000	Homes: formaldehyde	48 hours	508	<70*				Seifert 2000
UK	2003	Bedroom: child wheeze	3 days	193			0	>32	Venn 2003 <sup>‡</sup>
		Bedroom: child no wheeze		223			0	>32	
France	2003	Kitchen formaldehyde	72 hours	61	21.7				Clarisse 2003*
		Kitchen acetaldehyde			10.1				
		Kitchen pentanal			5.7				
		Kitchen hexanal			20.5				
		Living room formaldehyde			24.3				
		Living room acetaldehyde			10				
		Living room pentanal			6				
		Living room hexanal			23.8				
		Bedroom formaldehyde			24.5				
		Bedroom acetaldehyde			10.2				
Bedroom pentanal	6.4								
Bedroom hexanal	25.5								
Sweden	2004	Bedroom	24 hours	27	8.3*				Sakai 2004
Sweden	2005	Bedroom formaldehyde campaign A	24 hours	24	26	23	9.9	58	Gustafson 2005
		Bedroom formaldehyde campaign B	6 days	40	35	29	8.6	120	

**Table 5 (contd): Summary of aldehyde papers**

Country	Year pub	Domestic Environment	Averaging time	No samples/ persons	Concentration ( $\mu\text{g}/\text{m}^3$ ) <sup>†</sup>				Reference
					Mean	Median	Min	Max	
Sweden	2007	Wood burning homes acetaldehyde	24 hours	14	17	13			Gustafson 2007
		Non wood burning homes acetaldehyde		10	12	11			
France	2008	Living room formaldehyde	30-95 mins	143	30.4	26.7	6	93	Marchand 2008
		Living room acetaldehyde		122	13.9	11.5	0	66	
		Living room hexanal		122	8.5	6.4	0	45	
		Bedroom formaldehyde		143	33.5	30.9	6	87	
		Bedroom acetaldehyde		122	14.6	11.8	0	59	
		Bedroom hexanal		122	9.2	6.3	0	67	
France	2009	Bedroom formaldehyde <sup>‡</sup>	7 days	187	19.4	19			Dassonville 2009 <sup>*</sup>
		Bedroom acetaldehyde		187	8.9	8.8			
		Bedroom pentanal		187	3.7	3.7			
		Bedroom hexanal		187	25.3	23.8			
Italy	2009	All homes formaldehyde	24 hours	59	16	14.2	5.4	42.4	Lovreglio 2009
		All homes acetaldehyde			10.7	8.4	0.3	38.8	

<sup>†</sup>Micrograms per cubic metre; <sup>\*</sup>geometric mean; <sup>\*\*</sup>shortness of breath; <sup>‡</sup>results for 1<sup>st</sup> of 4 home visits; <sup>\*</sup>reported as no of cases/controls exposed to total aldehydes  
 Unless otherwise stated, mean values given are arithmetic means; empty cells indicate this information not available.

## **Polycyclic aromatic hydrocarbons (PAHs)**

PAHs arise from incomplete burning of carbon-containing materials such as oil, wood, coal or tobacco. They are also found at low concentrations in consumer products that contain coal tars. Human exposure occurs when they breathe smoke, vehicle or industrial emissions, and PAHs can also be ingested via contaminated water or seafood. Several PAHs have been classified as probable human carcinogens (IARC, 2010).

Fromme et al measured PAHs in street side living rooms in 123 typical Berlin apartments over winter and summer (Fromme et al. 2004a). Participants were both smokers and non-smokers. Median levels of Benzo(a)pyrene (BaP) were  $0.65\text{ng}/\text{m}^3$  in winter and  $0.27\text{ng}/\text{m}^3$  in summer in smoking homes while for non-smoking homes the corresponding concentrations measured were  $0.25\text{ng}/\text{m}^3$  and  $0.09\text{ng}/\text{m}^3$ . The authors concluded that 75% of the variance was caused by BaP concentrations in air outside apartment windows. A significant correlation was found between indoor PAH concentrations and outdoor concentrations in the apartments of non-smokers which could be attributable mainly to traffic emissions.

Gustafson studied the impact of domestic wood burning on indoor levels of 27 PAHs in 23 homes over winter in a small town in Sweden (Gustafson, Ostman & Sallsten 2008). Thirteen of these homes had wood burning appliances and no smoking was permitted during the sampling in living rooms. They found that over 24 hours, levels of PAHs measured were 3-5 times higher in the wood burning homes. Median concentrations of BaP<sub>eq</sub> were  $0.88\text{ng}/\text{m}^3$  for wood,  $0.23\text{ng}/\text{m}^3$  for control homes and  $0.74\text{ng}/\text{m}^3$  for outdoors. Phenanthrene was the most abundant PAH found across all homes. The total PAH cancer potency (sum of BaP equivalents) was almost four times higher in the wood burning homes compared with the control homes, while the indoor BaP level of  $0.52\text{ng}/\text{m}^3$  was five times higher than the Swedish guideline of  $0.1\text{ng}/\text{m}^3$ .

Table 6 below provides a summary of the papers on domestic PAH concentrations reviewed in this report.

**Table 6: Summary of papers PAH papers reviewed**

Country	Year pub	Domestic Environment	Averaging time	No samples/ persons	Concentration (ng/m <sup>3</sup> ) <sup>†</sup>				Reference
					Mean	Median	Min	Max	
Germany	2004	Smokers winter	7-8 hours	57		0.65*			Fromme 2004
		Non smokers winter			0.25*				
		Smokers summer		58		0.27*			
		Non smokers summer				0.09*			
Sweden	2008	Wood burning homes	24 hours	14		0.52*		Gustafson 2008	
		Non wood burning homes		10		0.12*			

<sup>†</sup>nanograms per cubic metre; \*median indoor level of Benzo(a)pyrene (BaP);  
 Unless otherwise stated, mean values given are arithmetic means; empty cells indicate this information not available.

**Polybrominated diphenyl ethers (PBDEs) / Polychlorinated biphenyls (PCBs) / Tetrabromobisphenol-A (TBBP-A) / Organophosphates**

Polybrominated diphenyl ethers or PBDEs are common flame retardants used to reduce the risk of fire in products such as plastics for television cabinets, furniture foams and fabrics. They are structurally similar to PCBs. The flame retardant properties of these chemicals result in a slow ignition and rate of fire growth, thus allowing increased time to escape in the event of fire. However, there is growing evidence that PBDEs accumulate in living organisms and traces have been found in human breast milk, fish, aquatic birds although the mechanisms of transfer remain unknown (<http://www.epa.gov/opptintr/pbde/>).

In 2008, Mandalakis used passive samplers to conduct a study in Greece measuring airborne levels of PBDEs (19 congeners) in a number of microenvironments including 5 homes (Mandalakis et al 2008). The mean concentration found in the homes measured was 8pg/m<sup>3</sup>, compared with outdoor concentrations of 18pg/m<sup>3</sup>. They concluded that inhalation intake represented a small proportion (~1%) of overall daily exposure. Their results yielded lower average concentrations of PBDEs than other studies of indoor environments (Harrad et al 2006). Harrad found PBDE concentrations were higher in offices than in homes and they estimated exposure via inhalation and dust ingestion to be 9% and 37% in adults and 2% and 69% in toddlers (Harrad et al 2006). With regard to PCBs, they conclude that inhalation contributes between 4.2 and 63% of overall UK exposure for adults. These results were comparable with data from a study conducted in Germany (Fromme et al. 2009). Fromme found the mean concentration of PBDE congeners in indoor air in 34 residences to be 38.4pg/m<sup>3</sup> (sum of BDEs 47, 99, 100, 153, 154) and 73.1pg/m<sup>3</sup> (when BDE 183 was added).

Two widely used brominated flame retardants: tetrabromobisphenol A (TBBP-A) and hexabromocyclododecanes (HBCDs) were measured in indoor air (n=33) and indoor dust

(n=45) homes in Birmingham, UK, together with concentrations in outdoor air (Abdallah et al., 2008). The indoor air samples yielded concentrations of 180pg/m<sup>3</sup> for HBCD and 15pg/m<sup>3</sup> for TBBP-A using passive samplers. No significant difference was found in air or dust for either compound from homes and offices. Using typical activity patterns between different environments, they estimate that dust ingestion constitutes 34% TBBP-A and 24% HBCDs for overall adult exposure and 90% TBBP-A and 63% HBCDs for toddlers.

Organophosphorus compounds (OPs) are ubiquitous in indoor environments. They are used in flame retardants, and plasticizers utilized in materials such as PVC, polyester and polystyrene; and also used in products such as paint and varnish, upholstered furniture, floor coverings and polish. Duplicate air samples were collected by solid-phase extraction columns in 17 buildings in Sweden, representing occupational, public and domestic environments (Marklund et al., 2005). Exposure by inhalation of air was found to be highest for the chlorinated OPs: tris(2-chloroethyl) phosphate (TCEP), and tris(chloropropyl) phosphate (TCPP). On the whole domestic levels were 3-4 times lower than public buildings although this was based on only two homes measured. In the 17 environments sampled, exposure of adults and children to total OPs was estimated at 5.8µg kg<sup>-1</sup> and 57µg kg<sup>-1</sup> respectively, based on estimated indoor air inhaled and dust ingested.

See table 7 for a summary of these papers.

**Table 7: Summary of papers measuring concentrations of PBDEs**

Country	Year pub	Domestic Environment	Averaging time	No samples/ persons	Concentration (pg/m <sup>3</sup> ) <sup>†</sup>				Reference
					Mean	Median	Min	Max	
Sweden	2005	Living room ΣOPs <sup>**</sup> Bedroom ΣOPs <sup>**</sup>	10 hours	1	230,000 160,000				Marklund 2005
UK	2006	Living room & bedroom Σ-BDE <sup>††</sup> Living room & bedroom Σ-PCB <sup>‡</sup>	28 days	31	52 2823	24 1802	4 487	245 9764	Harrad 2006
Greece	2007	Total PBDEs	7-10 weeks	5	9	11	3	15	Mandalakis 2007
UK	2008	Living rooms: Σ-HBCD* Living rooms: TBBP-A <sup>**</sup>	30 days	33 5	250 16	180 15	67 9	1300 22	Abdallah 2008
Germany	2009	Living room : ΣPBDEs 47, 99, 100, 153, 154 Living room: ΣPBDEs 47, 99, 100, 153, 154 and 183	24 hours	34	38.4 73.1				Fromme 2009

<sup>†</sup>picograms per cubic metre; \*total hexabromocyclododecanes; \*\*tetrabromobisphenol A; <sup>††</sup>total brominated diphenyl ethers; <sup>‡</sup>total polychlorinated biphenyls; <sup>\*\*</sup>total organophosphates. Unless otherwise stated, mean values given are arithmetic means; empty cells indicate this information not available.

## **Nicotine**

Nicotine is one of several thousand chemicals present in tobacco smoke and is known to act as a stimulant. Measurement of nicotine is often used as a marker for the general hazard of Environmental Tobacco Smoke (ETS).

Air nicotine measurements using passive monitors over 2 weeks in living rooms were conducted in the homes of >300 children in each of three European countries: Germany, the Netherlands and Sweden (Gehring et al. 2006). Measured concentrations were compared with questionnaire reported smoking and Gehring found a dose response relationship in all three countries. The range of air nicotine concentrations were from  $<0.08\mu\text{g}/\text{m}^3$  (LOD) to  $14.3\mu\text{g}/\text{m}^3$  in Germany,  $10.2\mu\text{g}/\text{m}^3$  in the Netherlands and  $3.2\mu\text{g}/\text{m}^3$  in Sweden. Specificity and negative predictive values were excellent in all countries while sensitivity and positive predictive values were only moderate to good indicating some misclassification. Phillips also found reported exposure to ETS to be supported by measured indoor nicotine levels in around 80% of subjects in a study of 190 non-smokers in Sweden during 1994 (Phillips et al. 1996). The median exposures to nicotine of occupants living in smoking homes was  $1.1\mu\text{g}/\text{m}^3$  while this was  $0.05\mu\text{g}/\text{m}^3$  in non-smoking homes.

Brunekreef conducted a similar study in the homes of smokers and non-smokers as part of the Prevention and Incidence of Asthma and Mite Allergy (PIAMA) birth cohort study (Brunekreef et al. 2000). He found that the smoking data reported in the questionnaire showed an excellent level of agreement with the measured air nicotine concentrations and concluded that exposure classification based on questionnaire data was likely to be valid way of assessing domestic exposure.

Others have not found this level of agreement between measured levels and self-reported smoking. Looking at the effects of smoke-free legislation on domestic smoke levels in Northern Ireland, van Tongeren et al (2008) found that self-reported domestic secondhand smoke (SHS) exposure was reduced following the ban while measured airborne nicotine levels were generally higher. This increase remained after adjusting for the effects of smoking practices.

Table 8 summarises the nicotine papers.

**Table 8: Summary of nicotine papers**

Country	Year pub	Domestic Environment	Averaging time	No samples/ persons	Concentration ( $\mu\text{g}/\text{m}^3$ ) <sup>†</sup>				Reference
					Mean	Median	Min	Max	
Sweden	1996	Smoking homes Non-smoking homes	24 hours	190		1.1 0.5			Phillips 1996
**The Netherlands	2000	Non smoking homes  ††Smoking homes >15/day	14 days	43  6	<LOD <sup>‡</sup>  5.95		<LOD <sup>‡</sup>  1.34	0.2  15.4 7	Brunekreef 2000
*Germany *The Netherlands *Sweden*	2006	Living rooms Living rooms	14 days	347 335 354				14.3 10.2 3.2	Gehring 2006

<sup>†</sup>Micrograms per cubic metre; <sup>\*</sup> Part of AIRALLERG study which was a nested case-control study within 4 ongoing birth cohort studies; <sup>\*\*</sup>GINA and LISA in Germany, PIAMI in the Netherlands, and BAMSE in Sweden; <sup>‡</sup>LOD=0.05 $\mu\text{g}/\text{m}^3$ ; <sup>††</sup>data available for 4 other smoking levels in homes. Unless otherwise stated, mean values given are arithmetic means; empty cells indicate this information not available.

### **Volatile organic compounds (1,3-butadiene, aromatic amines, benzene, toluene, xylene)**

Volatile organic compounds (VOCs) include a variety of chemicals, some of which may have both short- and long-term health effects. They are emitted from a variety of products such as paint and paint strippers, building materials, furnishings, and equipment such as copiers and printers. This can result in much higher indoor than outdoor concentrations.

Aromatic amines are by-products of chemical manufacturing and contaminants of dyes, rubber and textiles. ETS also contains aromatic amines. Palmiotto studied the concentrations of ten aromatic amines in indoor and outdoor air in Italy (Palmiotto et al. 2001). They found the levels of one aromatic amine, aniline, to be extremely variable so this was not included in the comparisons between sites. In non-smoking homes Palmiotto found concentrations were between 5-11ng/m<sup>3</sup> but in the presence of smokers the levels increased to between 15-33ng/m<sup>3</sup>, based on 9 homes. Low levels are considered to be <20ng/m<sup>3</sup>.

A pilot for the German INGA study described earlier investigated the VOCs benzene, toluene, ethyl benzene and xylene (BTEX) in 20 homes (Schneider et al. 1999). Passive samplers were set up at three heights in living rooms (0.7, 1.2, and 2 metres, and at 2 metres in bedrooms and kitchens over 7 days. They found no significant difference between median values of weekly average air concentrations of BTEX in the different height measurements. Indoor levels were significantly higher than outdoor except for benzene, and with the exception of toluene, there was no difference between old and new buildings. Also within the INGA study, Topp measured BTEX in the living rooms and bedrooms of adults and children on two occasions in two regions of Germany over 1 week (Topp et al. 2004). Correlations between values for the two visits for all BTEX except toluene were ~1, indicating that there was no relationship between the measurements. In a within-home correlation, the correlation for toluene was 0.27 for living room concentrations, which rose to 0.39 when adjusted for region and season.

In 2006, Alexopoulos attempted to evaluate the exposure of Athens citizens to toluene and xylene using passive air samplers in 50 homes (Alexopoulos et al., 2006). Non-smoking volunteers were from a variety of occupational groups to encompass different degrees of outdoor and indoor exposure, eg postmen and traffic policeman as opposed to teachers. They took measurements 6 times throughout a one year period and found that factors influencing indoor concentrations were the type of floor covering, type of heating and recent painting but their results did not reach statistical significance. Annual average levels found were  $36\mu\text{g}/\text{m}^3$  for xylene and  $39\mu\text{g}/\text{m}^3$  for toluene. They concluded that groups likely to be exposed to higher concentrations are those whose occupations involve more time outdoors.

Also in Athens, Chatzis measured average benzene levels over six 5-day sampling campaigns over a one year period (Chatzis et al., 2005). They found proximity to busy roads a factor influencing indoor levels, and in the 50 non-smoking homes they measured, found an annual mean of  $10.2\mu\text{g}/\text{m}^3$  which is much lower than reported by Alexopoulos. The outdoor level over the same period was  $21.5\mu\text{g}/\text{m}^3$  which Chatzis states exceeds the limit of  $5\mu\text{g}/\text{m}^3$  proposed by EU guidelines. They suggest that climatic factors influence the type of wall and floor coverings used and exposures in Greece may differ greatly from other northern European towns. These different exposure patterns suggests that fixed point monitoring is not sufficient to estimate population exposure overall. Benzene was measured on five consecutive days in the homes of 21 non-smoking parents with children aged 2-3 years in Rouen, France (Kouniali et al. 2003). This study assessed personal exposure of children to benzene by computing time weighted averages of bedroom, day care centre and outside exposures, together with simultaneous collection of urine samples. The average concentration in children's bedrooms was  $10.9\mu\text{g}/\text{m}^3$ , with detectable levels of muconic acid and hydroquinone found in the urine samples.

Gustafson's study of homes in Sweden where wood is burned for fuel found increased levels of benzene in wood burning homes when compared to control homes ( $3\mu\text{g}/\text{m}^3$  versus  $1.5\mu\text{g}/\text{m}^3$ ) (Gustafson et al. 2007). These levels were considerably lower than those found by both Chatzis and Alexopoulos, although they were based on only 14 and 10 homes respectively (Alexopoulos, Chatzis & Linos 2006, Chatzis, Alexopoulos & Linos 2005). Wood burning homes also had higher median levels of 1,3-butadiene at  $0.23\mu\text{g}/\text{m}^3$  and  $0.11\mu\text{g}/\text{m}^3$ . In a linear regression model, type of appliance, number of hours burning and number of times wood was added were significant factors for indoor levels of 1,3-butadiene. Also in Sweden, Emenius measured total VOCs in 56 single storey homes and found that homes with mechanical ventilation had significantly lower concentrations compared with naturally ventilated homes ( $149\mu\text{g}/\text{m}^3$  versus  $373\mu\text{g}/\text{m}^3$ ,  $p < 0.0001$ ) (Emenius et al. 1998).

Fifteen VOCs were measured in a number of varied microenvironments in 12 homes (6 smoking, 6 non-smoking) by Kim (Kim, Harrad & Harrison 2001). Daytime variations in concentrations were represented by three measurement periods over 24 hours between November 1999 and February 2000 in Birmingham, UK. They found higher mean concentrations indoors than outdoors for all VOCs measured but no correlation was found between simultaneous measurements. In smoking homes, significantly higher concentrations of 1,3-butadiene were detected.

Some authors have looked at the effect of indoor levels of VOCs on prevalence of asthma and to determine risk and severity of wheezing illness in children. In Sweden, Norback measured VOCs in living rooms and bedrooms of 88 subjects (Norback et al. 1995). Homes in the study had no gas cooking or heating so effects were not confounded by emissions of both VOCs and  $\text{NO}_2$ . They found significant relations between measured indoor concentrations of various VOCs and symptoms related to asthma. On the contrary, Venn found no effect for total or

individual VOCs on wheezing illness in children (Venn et al. 2003). BTEX were measured in the bedrooms of cases and controls among young primary school children over a 4 week period.

The largest contribution of VOCs to personal exposure was found in homes in Leipzig (Gokhale, Kohajda & Schlink 2008). Concentrations of between 35-80 $\mu\text{g}/\text{m}^3$  were found in domestic air samples analysed for 25 organic compounds over 1 week using passive samplers. When a genetic algorithm (GA) model was applied to conduct source apportionment they found this gave comparable results to that of chemical mass balance.

Table 9 provides a summary of the papers that reported VOC exposures.

**Table 9: Summary of volatile organic compound papers**

Country	Year pub	Domestic Environment	Averaging time	No samples/ persons	Concentration ( $\mu\text{g}/\text{m}^3$ ) <sup>†</sup>				Reference
					Mean	Median	Min	Max	
Sweden	1995	Living room total VOCs: nocturnal SOB <sup>±±</sup>	2 hours	26	780		70	8350	Norback 1995
		Living room total VOCs: no nocturnal SOB <sup>±±</sup>		62	300		70	1670	
		Bedroom total VOCs: nocturnal SOB <sup>±±</sup>		26	790		90	9380	
		Bedroom total VOCs: no nocturnal SOB <sup>±±</sup>		62	310		70	2920	
Sweden	1998	Single storey homes: mechanical ventilation	14 days	22	149			Emenius 1998	
		Single storey homes: natural ventilation		29	373				
Germany	1999	Living room: 1.2m <sup>**</sup> : benzene	7 days	20	3.86	3.49	1.3	12.6	Schneider 1999
		Living room: 1.2m <sup>**</sup> toluene			50.13	52.51	18.8	96.6	
UK	2001	Smoking homes: benzene	8 hrs/day for 3 mths	6 homes (32 samples)	16.3	11.4	4.2	63.7	Kim 2001
		Smoking homes: toluene			29.2	28.4	9.6	78.2	
		Non-smoking homes: benzene			11.5	6.6	3.4	51.7	
Italy	2001	Smokers' homes	8 hours	4			15	Palmiotto 2001	
		Non smokers' homes		5			5		11
France	2003	Children's bedrooms: benzene	5 days	20		10.9		Kouniali 2003	
		Parent's bedrooms: benzene				9.1			
UK	2003	Bedroom: child wheeze	4 weeks	189			0	Venn 2003	
		Bedroom: child no wheeze		220			0		>506 <sup>±</sup>

**Table 9 (contd): Summary of volatile organic compound papers**

Country	Year	Year pub	Domestic Environment	Averaging time	No samples/ persons	Concentration ( $\mu\text{g}/\text{m}^3$ ) <sup>†</sup>				Reference
						Mean	Median	Min	Max	
Germany	1995-1998	2004	Living room benzene: Visit 1	7 days	631		2		91.7	Topp 2004
			Living room benzene: Visit 2				2.5		32.8	
			Living room toluene: Visit 1				35.7		918.5	
			Living room toluene: Visit 2				32.3		1325.9	
			Nursery benzene: Visit 1	7 days	631		1.7		114.0	
			Nursery benzene: Visit 2				2.1		40.7	
			Nursery toluene: Visit 1				21		678.4	
			Nursery toluene: Visit 2				20.3		1195.6	
Greece	1997-1998	2005	Homes: benzene	108 hrs x 6 campaigns	50	10.2		6.03	13.35	Chatzis 2005
Greece	1997-1998	2006	Homes: toluene Homes: xylene	7 days x 6 campaigns	50	49.2 43.7	38.9 36.2	19.4* 19.5*	82.5* 72.98*	Alexopoulos 2006
Sweden	2003	2007	Wood burning homes: benzene	24 hours	14	3.9	2.6			Gustafson 2007
			Non-wood burning homes: benzene		10	2	1.4			
			Wood burning homes: benzene	7 days	14	5.7	3			
			Non-wood burning homes: benzene		10	2.5	1.5			
			Wood burning homes: 1,3-butadiene	24 hours	14	0.38	0.2			
			Non- wood burning homes: 1,3-butadiene		10	0.11	0.1			
			Wood burning homes: 1,3-butadiene	7 days	14	0.31	0.23			
			Non-wood burning homes: 1,3-butadiene		10	0.11	0.11			
Germany	2005	2008	Homes; 25 VOCs	7 days	7			35	80	Gokhale 2008

<sup>†</sup>micrograms per cubic metre; \*10<sup>th</sup>-90<sup>th</sup> percentiles; \*\*measurements taken at other heights; <sup>‡</sup>cases/controls exposed to levels of total VOCs between 0->506  $\mu\text{g}/\text{m}^3$ ; <sup>‡‡</sup>shortness of breath. Unless otherwise stated, mean values given are arithmetic means; empty cells indicate this information not available.

## 4.2 Studies of ingestion and dermal exposure

### Ingestion

Eight out of the nine papers found in the literature search for papers relating to dermal and ingestion exposure to indoor pollutants in the home concentrated on measurement of exposure to chemicals via the ingestion route. Four of these papers investigated polybrominated diphenyl ethers and a further four looked at arsenic, perfluoroalkyl compounds (PFCs), the phenolic organic contaminants bisphenol-A, triclosan and tetrabromobisphenol-A, and phthalates.

In 2004, Fromme examined household dust in 30 Berlin apartments for the presence of phthalates, which are a compound of polyvinyl chloride (PVC) and commonly used as emulsifiers for cosmetics, glues, paints and dyes (Fromme et al. 2004b). In this study the main phthalate found in house dust was diethylhexyl, and the estimated intake via ingestion of household dust was 0.11 µg/kg body weight per day in adults, and 5.97 µg/kg body weight per day in children. The authors conclude that although the main route of ingestion of phthalates is through foodstuffs, non-dietary ingestion may be important for specific sub-groups of the population.

Dust ingestion comprises an important route of exposure to HBCDs and TBBP-A according to Abdallah and colleagues, representing 24% (of HBCD) and 34% (of TBBP-A) of overall exposure of adults and 63% and 90% of toddler's overall exposure respectively (Abdallah Mohamed et al. 2008). In the absence of comprehensive time-activity data for the UK population, the authors based their exposure estimates on typical US population activity patterns, thus for adults 63.8% of their time is spent at home while for toddlers this is typically 86.1% (Klepeis et al 2001). In common with other studies of ingestion exposure, Abdallah used average and high dust ingestion rates of 20 and 50mg/day for adults and 50 and 200mg/day for

toddlers as reported by Jones-Otazo et al (2005). Based on 45 homes measured in 2008, the median exposure of adults to total HBCDs was found to be 32.5 ng day<sup>-1</sup> with a corresponding estimate of median exposure of 86.9 ng day<sup>-1</sup> in children aged 6-24 months.

Abdallah went on to report personal exposure of 21 UK adults to HBCDs and their degradation products via ingestion of indoor dust in three microenvironments: home, office and car (Abdallah, Harrad 2009). Figures for dust ingestion of HBCDs within the home environment were not given separately in this paper but the authors conclude that house dust contributed the most to personal exposure to all compounds studied due to the proportion of time spent in the home rather than to significantly higher concentrations (personal exposure range 4.5 – 1851 ng total HBCD per day).

In 2008, Harrad looked at the brominated flame retardants known as PCBs in 28 homes in the UK (Harrad et al. 2009). They found concentrations of brominated diphenyl ethers (BDEs) in UK dust samples to be significantly higher than those in Canadian homes. Looking at  $\Sigma$ tri-hexa-BDEs day<sup>-1</sup>, the median exposure (using the mean ingestion rate for adults of 20 mg/day and 50 mg/day for toddlers), was 1.2 ng day<sup>-1</sup> and 2.9 ng day<sup>-1</sup> in adults and children respectively. For a mean ingestion rate, the intake of a specific BDE (BDE 209) was estimated at 56 ng/day for adults and 140 for toddlers. Despite the existence of restrictions on the manufacture and use of PCBs, Harrad and colleagues report that PCBs still persist in domestic homes resulting in appreciable exposure of adults and particularly young children. In a study of 20 homes in Birmingham, UK, they estimated exposure via dust ingestion and found the median exposure in an adult to be 0.95 ng  $\Sigma$ PCB d<sup>-1</sup> (average 2.3) while for toddlers aged 6-24 months the median exposure was 2.4 ng  $\Sigma$ PCB d<sup>-1</sup> (average 5.6) (Harrad et al. 2008).

Geens et al assessed human exposure to bisphenol-A, triclosan and tetrabromobisphenol-A (BPA, TCS and TBBPA) via dust ingestion in 18 homes in Flanders, Belgium in 2008 (Geens et al. 2009). These contaminants have a number of applications in domestic homes for example food containers, plastic bottles and small kitchen appliances such as mixers and microwaves. Jones-Otazo intake rates were used and activity patterns as proposed by Klepeis (Jones-Otazo et al. 2005, Klepeis et al. 2001). For average dust intake rates, Geens calculated a median daily intake ( $\text{ng d}^{-1}$ ) to BPA, TCS and TBBPA of 2.9, 4 and 0.2 in adults and 97, 35 and 34 in toddlers respectively. They too conclude that food intake is the most important route with dust contributing <5% according to median values.

Perfluoroalkyl compounds (PFCs) are chemicals used in many consumer products with perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA) being the most commonly studied. Björklund studied 10 houses and 38 apartments in Stockholm and found the concentrations of both PFOS and PFOA to be higher in apartments than in houses studied (Bjorklund, Thuresson & De Wit 2009). They estimated the mean dust ingestion of PFOS for a toddler living in an apartment was 10 ng/day but for those living in a house this fell to 5 ng/day. Estimated dust exposures were compared with data from Canada and Spain and although the authors conclude that diet is the most important exposure route, dust ingestion is still of interest particularly with regard to the exposure of toddlers.

Concentrations of arsenic were derived from household dust and garden soil both in an examining site and in a control location in southwest England (Rieuwerts, Searle & Buck 2006). Using the Contaminated Land Exposure Assessment (CLEA) model, estimates of arsenic intake for children from 0-6 years were derived. Compared to the UK Soil Guidance Value (SGV) index dose of  $0.03 \mu\text{g}$ , estimates for housedusts in this young population group deemed most at risk was up to  $3.53 \mu\text{g kg}^{-1} \text{bw day}^{-1}$  (DEFRA and EA (Department for Environment, Food and Rural

Affairs and the Environment Agency) 2002a). The authors express concern about the potential implications for the health of children in this area.

### **Dermal exposure**

One paper was found that looked at dermal exposure to captan which is a fungicide used by fruit growers in seasonal spraying (de Cock et al. 1998). Although basically an occupational exposure, this paper was included because it measured indoor exposure in the fruit growers' homes and dermal exposure of the fruit growers' families during harvesting season and two days after spraying. Captan was detected in all 10 homes studied with an average of 7.6  $\mu\text{g}/\text{m}^2$  (range 0.91-14.9). The average exposure of hands from rinsing was 0.27  $\text{mg}/\text{m}^2/\text{hour}$  although this appears to have been carried out in only 4 subjects.

### **4.3 Results of review of existing IAQ models and indoor air emission, exposure and risk assessment studies**

#### **4.3.1 Review of existing IAQ models**

##### **Introduction**

Two major types of computer simulation techniques for studying airflow and contaminant transport in buildings exist: 1) mass-balance models, and 2) computational fluid dynamic (CFD) techniques. Each approach has strengths and limitations for studying different building ventilation and IAQ problems.

CFD models take a microscopic view of IAQ by examining the detailed flow fields and pollutant concentration distributions within a room or rooms. CFD models require a high computational demand. Since it is outwith the scope of the INTERA project to assess variations in concentrations within one room, the CFD models will not be included in the overview of IAQ modeling tools.

Mass balance modeling takes a macroscopic view of IAQ by evaluating average pollutant concentrations in the different zones of a building as contaminants are transported through the building and its HVAC system (heating, ventilation and air conditioning system). Mass balance models constitute the majority of the currently available models, and can be split into 3 types: regression models; micro-environmental models; and multizone models.

## Mass balance models: principles and types

Mass-balance models are used to simulate average indoor air pollutant concentrations as a function of outdoor concentrations, building and pollutant characteristics and indoor sources. These models consider transport of air pollutants between outdoors and indoors, as well as between indoor compartments in the buildings, and are widely used due to the simplicity of the mathematics involved. The strength of these models is the simulation of air pollutant concentrations that may be well compared with results from experimental measurements. Mass-balance models are based on the following concept: the mass of pollutant in the room during a short time interval  $dt$ , over which the parameters may be assumed to be constant, increases by an amount  $dm$ , as a result of the penetration of outdoor air and indoor sources:

$$dm = \text{inward mass flow} + \text{internal production} - \text{outward mass flow}$$

Converting the pollutant mass to the concentration within the compartment,  $C$ , at steady state this is usually written as an differential equation, such as:

$$\frac{dC}{dt} = S - L \times C = 0$$

where  $dC/dt$  is the rate of change of the indoor concentration,  $S$  is the sum of all sources and  $L$  is the sum of all sinks. The above equation may be analytically written including further parameters such as a loss rate (adsorption and deposition), a decay rate (for radioactive contaminants), a filtration factor and a mixing factor. The 3 types of mass balance models will be discussed in the below section, together with some examples for each.

### *Statistical regression models*

Regression models are mass-balance models in steady-state form, and among the 3 types, they are the simplest models available to predict indoor air pollutant concentrations. These entirely empirical techniques are quick and easy to apply since they do not require complicated input parameters. Several forms of such statistical regression models exist, and in general require input parameters such as penetration factors, outdoor concentration, room/building volume, indoor surface area, air exchange rate, deposition/adsorption rate. An example of such a model is the one described by Kulkarni and Patil (2002):

$$C_{in} \approx k C_{out} + \frac{S}{Q}$$

Where  $C_{in}$  and  $C_{out}$  are the time-integrated indoor and outdoor concentrations respectively,  $k$  the penetration factor,  $Q$  the airflow rate and  $S$  the average emission rate (parameters  $K$ ,  $Q$  and  $S$  are characteristics of the particular building). Some examples of statistical regression models developed for particulate matter are described on p 20 of the report of Milner et al. (2005).

Such statistical models are often incorporated as indoor air module of integrated exposure assessment software packages. An example is the ConsExpo 4.0 software package (RIVM, 2005), dealing with exposure from consumer products, under others via indoor air exposure. In ConsExpo, two models, one for exposure to vapors (e.g. from painted walls) and one for exposure to sprays are incorporated. The two models both use the parameters air exchange rate and room volume as input parameter; the emission model is a bit more flexible than in the above described statistical model, and can, in addition to the classical constant rate model, also account for emissions via instantaneous release or evaporation mode. Like other statistical models, the indoor air module in ConsExpo does not provide the ability to take into account

airflows to adjacent rooms, and, hence differentiation of concentration among various rooms within a building is not possible.

### *Micro-environmental models*

These models are based on the concept of the micro-environment (ME). This is defined as a generic space with a homogeneous pollutant concentration in which people spend time (Duan, 1982). This has been the key concept in modeling of personal exposure and, as a result, micro-environmental models are often used together with exposure models.

A micro-environment may be a whole building, a section of a building (i.e. multiple rooms) or individual rooms. According to this definition, the air in a micro-environment is assumed to be instantaneously well-mixed (i.e. the pollutant concentration is the same everywhere). This assumption is only valid if the mixing in the indoor air occurs at a much greater rate than other factors affecting indoor air concentrations, such as air exchange and deposition (Kulmala et al, 1999). Micro-environmental models are used to predict average concentrations in one or two indoor compartments based on a simple parameterization of the flow rates between them and between the compartments and the outdoor air.

A typical example of micro-environmental models is the INTAIR model (Dimitroulopoulou et al., 2001a). INTAIR is a deterministic compartment model that allows indoor concentrations to be simulated into two separate micro-environments using the following mass-balance equation:

$$\frac{dC_i}{dt} = -v_d (A_i/V_i) C_i + \lambda_r f C_o - \lambda_r C_i + \lambda_z (C_j - C_i) + Q_i/V_i$$

where  $C_i$  is the indoor concentration,  $C_o$  is the outdoor concentration,  $v_d$  is the deposition velocity,  $A$  is the surface area,  $V$  is the indoor volume,  $\lambda_r$  is the indoor-outdoor air exchange rate,

$\lambda_i$  is the air exchange rate between the indoor micro-environments,  $f$  is a building filtration factor,  $Q$  is the indoor emission rate and the subscripts  $i$  and  $j$  refer to the two individual micro-environments.

INTAIR is linked to an exposure model (EXPAIR) and provides estimates of the contribution of indoor and outdoor sources on total exposure. In Dimitroulopoulou *et al.* (2001a), the INTAIR model was parameterized to predict NO<sub>2</sub> concentrations in five different indoor micro-environments (residential and non residential). In another study, Dimitroulopoulou *et al.* (2001b) parameterized the INTAIR model to estimate PM10 concentrations and to assess the contribution of smoking for typical homes in the UK.

The updated version of the deterministic INTAIR model is the INDAIR model (Dimitroulopoulou *et al.*, 2006). In its probabilistic form, INDAIR simulates analytically (based on pollutant and building characteristics), the frequency distributions of indoor air pollutant concentrations in the three major rooms of the home (kitchen, lounge and bedroom); concentrations in four other non-residential micro-environments were simulated using I/O ratios. The model parameters and I/O ratios were defined in the INDAIR model as probability density functions to provide frequency distributions of air pollutant concentrations in different micro-environments. Model parameterization was based on data (although limited) for the UK and the results were found to be consistent with measured micro-environmental concentrations in the UK. The model has also been linked with a time-activity based exposure model, EXPAIR. This version of INDAIR/EXPAIR modeling framework was designed to simulate indoor concentrations and personal exposures at specific locations, using an assumed and fixed activity pattern, in terms of smoking and cooking and was unable to accommodate time varying levels in outdoor concentrations caused by changes in traffic and meteorological conditions.

### *Multizone indoor air quality models*

Whereas statistical regression models and micro-environmental models assume the air within a building to be instantaneously well-mixed, the real airflow pattern within a building may be extremely complex and not adequately represented by a model with only a single compartment (or few compartments). Often some parts of a building, particularly basements or rooms with closed doors, exchange air only very slowly with other parts of the building and, thus, the actual mixing is far from instantaneous as assumed by statistical and micro-environmental models. Therefore, real buildings are often more suitably represented as a large number of connected well-mixed chambers. This is the approach used in multizone modelling techniques, which are especially used to simulate ventilation rates (Milner et al., 2005).

Multizone IAQ models require the user to identify and describe all the zones (rooms) of interest and the links (e.g. flow paths) between those zones (and with the outside air). They generally take into account mechanical ventilation, tightness of buildings, terrain, shielding and climate conditions. The outputs of these models include air flow rates across the envelopes, between the rooms and through the mechanical ventilation system. The network of links is described by a series of flow equations which are solved simultaneously to provide air flow rates between rooms. Assuming that air flow patterns are unaffected by any contaminant present, a mass balance calculation in each zone at each time step can be included in a multizone model to predict the variation of concentrations with time (de Oliveira Fernandes et al., 2009). Multizone models use average or representative values for the parameters describing the conditions in a single zone (pressure, temperature, etc.).

Currently, there are no probabilistic multizone mass-balance models. The model user must decide whether a micro-environmental or multizone model is appropriate depending on the

available information, such as the building geometry and whether air exchange rates are known in advance (Milner et al., 2005).

Although the equations that form the basis of both micro-environmental and multizone models are generally similar, there are significant differences between the two model types. First, in micro-environmental models, air exchange rates are specified in advance as model parameters, whereas multizone models allow the air exchange rates between zones to be predicted by the model. Additionally, there are differences in the uses of the models. Micro-environmental models are used more frequently for analysis of air quality, while multizone techniques are used predominantly to model building ventilation. However, some multizone models have been coupled with contaminant transport models (also based on mass-balance approaches) to predict air pollutant distributions within buildings using the modeled ventilation rates (Milner et al., 2005).

Examples of multizone models which are in widespread use are CONTAM and RISK (EPA).

A general description of these models, and common input and output parameters and formats, and (dis)advantages of multizone models are briefly described here below:

### **CONTAM**

CONTAM is a multizone indoor air quality and ventilation analysis program developed by National Institute of Standards and Technology" (NIST) (Walton and Dols, 2003). CONTAM allows the user to determine the following indoor air quality parameters:

- *airflows and pressures* – infiltration, exfiltration, and room-to-room airflows and pressure differences in building systems driven by mechanical means, wind

pressures acting on the exterior of the building, and buoyancy effects induced by temperature differences between the building and the outside;

- *contaminant concentrations* – the dispersal of airborne contaminants transported by these airflows and transformed by a variety of processes including chemical and radio-chemical transformation, adsorption and desorption to building materials, filtration, and deposition to building surfaces; and/or
- *personal exposure* – the prediction of exposure of building occupants to airborne contaminants for eventual risk assessment.

CONTAM can be useful in a variety of applications. Its ability to calculate building airflows and relative pressures between zones of the building is useful for assessing the adequacy of ventilation rates in a building, for determining the variation in ventilation rates over time, for determining the distribution of ventilation air within a building, and for estimating the impact of envelope air-tightening efforts on infiltration rates. The program has also been used extensively for the design and analysis of smoke management systems. The prediction of contaminant concentrations can be used to determine the indoor air quality performance of buildings before they are constructed and occupied, to investigate the impacts of various design decisions related to ventilation system design and building material selection, to evaluate indoor air quality control technologies, and to assess the indoor air quality performance of existing buildings. Predicted contaminant concentrations can also be used to estimate personal exposure based on occupancy patterns.

## **RISK (EPA)**

The Indoor Environment Management Branch of the US- EPA has developed an IAQ model for analyzing the impact of sources, sinks, ventilation, and air cleaners on indoor air quality.

The model uses data on source emissions, room-to-room air flows, air exchange with the outdoors, and indoor sinks to predict concentration/time profiles for all rooms. The concentration/time profiles are then combined with individual activity patterns to estimate exposure. The model allows analysis of the effects of air cleaners located in either/or both the central air circulating system or individual rooms on IAQ and exposure. The model allows simulation of a wide range of sources including long-term steady-state sources, on/off sources, and decaying sources. Several sources are allowed in each room. The model allows the analysis of the effects of sinks and sink reemissions on IAQ.

#### **4.3.2 Available software packages for indoor environment modelling**

Numerous models used for modelling IAQ or indoor pollutant exposure exist. In the previous section, some examples illustrating the 3 types of mass balance models were given (INDAIR, CONTAM, RISK). In this section, a broader list of available IAQ or indoor pollutant models is described.

Some of the models described below go beyond the strict domain of IAQ and are more related to indoor environment exposure, or where indoor air is a minor aspect in the modelling of the overall human exposure model (called hereafter as 'integrated human exposure assessment model'). In the majority of these integrated human exposure assessment models, IAQ is based on a simple statistical regression or single box model; although this is in many cases not explicitly mentioned in the guidance documents. Nevertheless, we decided not to discard these models from the list because the scope of INTERA is also broader than 'indoor air' extending to exposure to pollutants by the dermal and ingestion routes.

The models are listed in alphabetical order. Short summary details, primarily extracted from project and other relevant websites, are provided for each of the identified IAQ studies and

models. Where possible, current URL links to the project reports or relevant summaries are provided.

## **APEX (TRIM)**

*Type: micro-environmental IAQ model*

See also: [http://www.epa.gov/ttn/fera/human\\_apex.htm](http://www.epa.gov/ttn/fera/human_apex.htm) (1999-2003)

The Air Pollutants Exposure model (APEX) is part of EPA's overall Total Risk Integrated methodology (TRIM) model framework (EPA, 1999), in particular the inhalation exposure component (TRIM.ExpInhalation). TRIM is the third in a series of IAQ models developed by the Indoor Environment Management Branch of US EPA's National Risk Management Research Laboratory. It is a time series modeling system with multimedia capabilities for assessing human health and ecological risks from hazardous and criteria air pollutants.

APEX estimates human exposure to criteria and toxic air pollutants at the local, urban, and consolidated metropolitan area level using a stochastic, "microenvironmental" approach. That is, the model randomly selects data on a sample of hypothetical individuals in an actual population database and simulates each individual's movements through time and space (e.g., at home, in vehicles) to estimate their exposure to and, optionally, dose of the subject pollutant. APEX can assume people live and work in the same general area (i.e., that the ambient air quality is the same at home and at work) or optionally can model commuting and thus exposure at the work location for individuals who work.

Compared to conducting a field study that would involve identifying, interviewing, and monitoring specific individuals in a study area, APEX provides a vastly less expensive, more timely, and more flexible approach. Compared to other air exposure models, APEX provides a good

balance in terms of precision and resource expenditure between the more narrowly focused site-specific model and the broadly applicable national screening-level models. The model also allows different air quality data, exposure scenarios, and other inputs and thus is very useful for decision making.

## **CALENDEX**

*Type: integrated human exposure assessment model*

See also: [http://cfpub.epa.gov/crem/knowledge\\_base/crem\\_report.cfm?deid=76394](http://cfpub.epa.gov/crem/knowledge_base/crem_report.cfm?deid=76394)  
[http://www.exponent.com/calendex\\_software/](http://www.exponent.com/calendex_software/) (2000)

Calendex™ is an advanced exposure and risk analysis tool for estimating exposure of the U.S. population and more than 30 of its subgroups, to the following substances: pesticides in food and the residential environment; food, air, and water contaminants; chemical ingredients in formulated products. It uses an approach known as the calendar model to estimate exposures on each calendar day for the population of concern.

Calendex™ permits the estimation of exposure to single or multiple compounds for a wide variety of time periods (daily/acute, short-term, intermediate-term, and chronic (up to one year) time periods).

**CARES** (Cumulative and Aggregate Risk Evaluation System):

*Type: integrated human exposure assessment model*

See also: [http://cfpub.epa.gov/crem/knowledge\\_base/crem\\_report.cfm?deid=213910&view=pdf](http://cfpub.epa.gov/crem/knowledge_base/crem_report.cfm?deid=213910&view=pdf)  
(2002)

CARES (Cumulative and Aggregate Risk Evaluation System) is an object-oriented, windows-based PC program. CARES is comprised of modules (objects) that can be selected, linked and

executed using a programming canvas provided by an interface program called Notitia. The Notitia interface allows the user to choose from a menu of exposure sources (dietary, water, and residential) and exposure durations. The user inputs toxicity and chemical specific exposure data and conducts either an aggregated or cumulative risk evaluation for populations of interest. CARES has report writing and data querying subroutines that allows the user to view the results of the risk assessment, determine source contribution and print summary reports. CARES stores the detailed determinants and assumptions of each risk assessment so that the analysis can be reproduced.

**CASE** (Cumulative and Aggregate Simulation of Exposure)

*Type: integrated human exposure assessment model*

CASE, a physical-stochastic model, uses a micro-activity approach to estimate the exposure of children to multiple chemicals through various exposure routes. CASE has been developed by adding new algorithm modules to the existing Dermal Exposure Reduction Model (DERM) which was developed earlier at Stanford University for calculation of ingestion exposure, inhalation exposure and intake dose for multiple pesticides. It uses combinations of equations to describe physical exposure and environmental concentrations such as contaminants in indoor air, dust and surfaces, and exposure factors such as transfer efficiency, soil-skin adherence and contact area. The model makes the assumption that contaminants can be transferred instantly and each transfer event is considered to be an independent one.

**CEPST™** (Chemical Exposure Priority Setting Tool):

*Type: integrated human exposure assessment model*

See also: <http://www.thelifelinegroup.org/CEPTS/index.htm> (2006)

CEPST™ has evolved from the Complex Exposure Tool© (ComET©) that The LifeLine Group™ developed for Health Canada. CEPST™ currently exists as a proof of concept modeling construct. It demonstrates that for large groups of chemicals with little data and possibly multiple exposure scenarios, users can set priorities for chemicals relative to their exposure potential. The LifeLine Group has developed an approach to do this relative to human health risks. CEPST considers multiple routes of exposure, multiple subpopulations and different possible durations of exposure. Users can change the assumptions in the model and see the impacts. The full IT implementation of CEPST™ into a software tool is currently under further design and development.

#### **ConsExpo 4.1**

*Type: integrated human exposure assessment model, with a statistical regression model included as IAQ module in ConsExpo*

See also: <http://www.rivm.nl/en/healthanddisease/productsafety/ConsExpo.jsp> (2006)

ConsExpo is a computer program with mathematical models to assist in exposure assessments for compounds in consumer products. The program was initially developed in the framework of the RIVM project 'Risk assessment for consumers' to improve risk assessment for consumer products, on request of the Dutch Food and Food Consumer Product Safety Inspectorate (VWA). An exposure assessment in the current version of ConsExpo is a step-by-step process, starting with providing basic information on the product, compound and exposed person. The program is based on relatively simple exposure and uptake models. The starting point for these models is the route of exposure, i.e. the inhalatory, dermal or oral route.

In January 2010 a new version of the ConsExpo software, version 5 was released. This version facilitates the exposure assessment for different populations, to multiple products, in

various exposure scenarios, combining all routes and pathways of exposure into a total, aggregate exposure estimate for a population.

## **CONTAM**

*Type: multizone mass balance model (See page 61-2).*

See also: <http://www.bfrl.nist.gov/IAQanalysis/CONTAM/overview/1.htm>

## **DEEM™/DEPM:** (Dietary Exposure Evaluation Model/Dietary Exposure Potential Model)

*Type: integrated human exposure assessment model (with focus on dietary exposure)*

See also: <http://www.epa.gov/microbes/depm.htm> (2003)

The DEEM™ dietary exposure assessment modules can be used to estimate the intake of toxicants, nutrients, pesticides, food additives, and natural constituents - in short for any chemical component of food or water. These substances can include inorganic and organic chemicals as well as microorganisms or toxins produced by microorganisms. They can be naturally or synthetically created.

The goal of dietary exposure assessments is to characterize the exposure of the population of concern and to identify the variability of that exposure. Typically, the primary objectives are to estimate the level of ingestion of the substance and to identify the sources of both variability and uncertainty in the estimate. In addition, the exposure assessment can also be useful in aggregate exposure assessments to identify the potential importance of diet relative to other pathways of exposure and to indicate where consumption of a particular food commodity or other unique characteristic (i.e., age, regional and ethnic preferences), would indicate the potential for unique exposure patterns.

**E-FAST** (Exposure and Fate Assessment Screening Tool) V2.0:

*Type: integrated human exposure assessment model*

See also: <http://www.epa.gov/opptintr/exposure/pubs/efast.htm> (2007)

The Exposure and Fate Assessment Screening Tool, Version 2.0, also known as E-FAST V2.0, is a screening-level computer tool that allows users to estimate chemical concentrations in water to which aquatic life may be exposed, as well as generate human inhalation, drinking water ingestion, and fish ingestion exposures resulting from chemical releases to air, water, and land. E-FAST V2.0 is appropriate for use as a screening tool to assess potential exposures from chemical discharges to air (stack or fugitive releases), surface water, or land. E-FAST V2.0 can also be used to estimate potential inhalation and dermal exposures to consumer products, such as hard surface cleaners, soaps, air fresheners, paints, gasoline, and motor oil. The exposed populations assessed by the model are either some segment of the general population or consumers.

**EUSES** (European Union System for the Evaluation of Substances):

*Type: integrated human exposure assessment model; does not contain an indoor specific module*

See also: [http://ecb.jrc.ec.europa.eu/documents/Existing-Chemicals/EUSES/EUSES\\_2.1/EUSES\\_2.1\\_documentation/EUSES\\_2.1\\_User\\_Manual.pdf](http://ecb.jrc.ec.europa.eu/documents/Existing-Chemicals/EUSES/EUSES_2.1/EUSES_2.1_documentation/EUSES_2.1_User_Manual.pdf) (2008)

EUSES is a decision-support instrument, which enables the user to carry out rapid and efficient assessments of the general risks posed by substances to man and the environment.

EUSES is intended mainly for initial and refined risk assessments rather than comprehensive assessments. The system is fully described in the extensive EUSES documentation and is based on the EU Technical Guidance Documents (TGD) on Risk Assessment for New Notified

Substances, Existing Substances and Biocides. The new EUSES 2.1 version (2008) is an update of EUSES 2.0, containing all Emission Scenario Documents for biocides. The development of EUSES 2.1 was commissioned by the European Commission to the National Institute of Public Health and the Environment (RIVM) of The Netherlands. The work was supervised by an EU working group comprised of representatives of the JRC-European Chemicals Bureau, EU Member States and the European chemical industry.

**IAQX** (Simulation Tool Kit for Indoor Air Quality and Inhalation Exposure):

*Type: multizone mass balance model*

<http://www.epa.gov/nrmrl/appcd/mmd/iaq.html> (1991)

IAQX is a Microsoft Windows-based IAQ simulation software package that complements and supplements existing IAQ simulation programs (such as RISK) and is designed mainly for advanced users. In addition to performing conventional IAQ simulations, which compute the time/concentration profile and inhalation exposure, IAQX can estimate the adequate ventilation rate when certain air quality criteria are provided by the user, a unique feature useful for product stewardship and risk management.

**I-BEAM** (Indoor Air Quality Building Education and Assessment Model):

*Type: qualitative, educational IAQ model*

See also: <http://www.epa.gov/iaq/largebldgs/i-beam/index.html> (2008)

The Indoor Air Quality Building Education and Assessment Model (I-BEAM), released in 2002, is a guidance tool designed for use by building professionals and others interested in IAQ in commercial buildings. I-BEAM updates and expands EPA's Building Air Quality guidance (the Building Air Quality is still available via the web at [www.epa.gov/iaq/largebldgs/](http://www.epa.gov/iaq/largebldgs/)) and was

designed to be a comprehensive state-of-the-art guidance for managing IAQ in commercial buildings. I-BEAM contains text, animation/visual, and interactive/calculation components that can be used to perform a number of diverse tasks: conducting an indoor air quality (IAQ) building audit; diagnosing and resolving IAQ related health problems; establishing an IAQ management and maintenance program to reduce IAQ risks; planning IAQ compatible energy projects; protecting occupants from exposures to construction/renovation contaminants; and calculating the cost, revenue, and productivity impacts of planned IAQ activities. However, I-BEAM provides rather qualitative and not quantitative outputs, and provides a 'general contaminant' output and not pollutant-specific outputs.

**INDAIR-2: Indoor Air (2005)**

See above (micro-environmental models, page 59)

Micro-environmental model allowing calculation of outdoor concentrations, indoor sources and building characteristics.

**MCCEM v1.2 (Multi-Chamber Concentration and Exposure Model):**

*Type: multizone mass balance model*

See also: <http://www.epa.gov/oppt/exposure/pubs/mccem.htm> (2001)

MCCEM uses a mass balance approach to estimate average and peak indoor air concentrations of chemicals released from products or materials in houses, apartments, townhouses, or other residences. The data libraries contained in MCCEM are limited to residential settings. However, the model can be used to assess other indoor environments (e.g. schools, offices) if the user can supply the necessary inputs. Also estimates inhalation exposures to these chemicals, calculated as single day doses, chronic average daily doses, or lifetime average daily doses. The software package maintains a library of US residences,

containing data on zone or area volumes, interzonal air flows, and whole-house exchange rates, and allows the user to tailor his analysis to a particular location, and to model air concentrations in as many as four zones for a given residence. Estimates exposure for periods ranging from 1 hour to 1 year, and develops seasonal or annual exposure profiles using a long-term model. In addition, the model offers several different options for dealing with 'sinks' like carpeting and wallboard.

**MIAQ (Multi-Chamber Indoor Air Quality Model):** (2001)

*Type: multizone mass balance model*

See also: <http://www.exposurescience.org/MIAQ>

Research software for predicting the time-evolution of gaseous and particulate air pollutants in an arbitrary number of connected zones. MIAQ accounts for emissions, ventilation, deposition, coagulation, and chemical reactivity. The program is written in Fortran-77 and Compiles with GNU gfortran on a wide variety of platforms.

**PARAMS 1.0**

*Type: multizone mass balance model*

See also: <http://www.epa.gov/nrmrl/appcd/mmd/iaq.html>

This Microsoft Windows-based computer program implements 30 methods for estimating the parameters in indoor emissions source models, which are an essential component of indoor air quality (IAQ) and exposure models. PARAMS is an extension to the EPA-RISK model (see above).

**PROMISE** (Probabilistic Methodology for Improving Solvent Exposure Assessments)

See also: <http://www.sielkenassociates.com/DesktopDefault.aspx?tabid=1309>

PROMISE© is an ongoing modeling project (Version 7, released June, 2001) by Silken Inc for the Solvents Council of the American Chemistry Council. The software is designed to evaluate exposures and doses from single and multiple uses of products that contain volatile solvents (adhesives, nail polish, paints, floor cleaners, etc.), from activities surrounding the use of large volumes of solvents (open drums or open tanks), or from spills. The model can be used to investigate products used in the workplace and the home.

PROMISE© can calculate multi-route exposures from dermal, inhalation (indoor or outdoors), and/or ingestion routes of solvent exposure. The distribution of the dose can be evaluated and compared for various exposure pathways, alternative exposure and uptake models, and will allow a tiered analysis of exposure. PROMISE© is written FORTRAN and is designed to run on PCs in the Windows® environment (Windows95®, Windows98®, or WindowsNT®). The embedded document below is an evaluation of some current models including PROMISE©.

**SHEDS** (Stochastic Human Exposure and Dose Simulation):

See also: [http://www.epa.gov/heasd/products/sheds\\_multimedia/sheds\\_mm.html](http://www.epa.gov/heasd/products/sheds_multimedia/sheds_mm.html)

SHEDS-Multimedia version 3 is a probabilistic aggregate residential exposure model. Other SHEDS models, with similar approaches but addressing different chemical classes and exposure scenarios, have been developed by EPA/ORD's exposure modeling research program to address exposures to particulate matter (SHEDS-PM), air toxics (SHEDS-ATOX), and wood (SHEDS-Wood) This modeling tool can help predict ranges of exposure in a population, enhance dose model estimates, identify critical pathways and factors, quantify uncertainties,

and compare model predictions against real-world data. It has a number of unique features that advance the science of human exposure assessment, and has been peer reviewed and applied for research and regulatory support. SHEDS can link with measurements, source models, and PBPK models to quantify and reduce uncertainty in risk assessments.

Residential-SHEDS is a physically based probabilistic model which simulates dermal and non-dietary ingestion exposure of individuals to pesticides from residue on various surfaces in the home and from turf in the residential yard. It generates daily exposure and dose time profiles using mechanistic equations. Exposure is modeled as a series of transfers, using a micro-activity approach.

**SWIMODEL** (Swimmer Exposure Assessment Model): (2003)

See also: <http://www.epa.gov/oppad001/swimodel.htm>

The Swimmer Exposure Assessment Model was developed by EPA as a screening tool to conduct exposure assessments of pesticides found in indoor swimming pools and spas (these include the 200+ pesticides registered for swimming pool uses, as well as those which exist in water and run-off water). The SWIMODEL uses well-accepted screening exposure assessment equations to calculate the total worst-case exposure for swimmers expressed as a mass-based intake value (mg/event). The assessor has the option of using the default values available within the model or entering other available values. The model focuses on potential chemical intakes only and does not take into account metabolism or excretion of the chemical of concern.

**RISK (EPA)**

*Type: multizone mass balance model*

See above page 63.

**WPEM (Wall Paint Exposure Model):**

See also: <http://www.epa.gov/opptintr/exposure/pubs/wpem.htm>

WPEM estimates the potential exposure of consumers and workers to the chemicals emitted from wall paint which is applied using a roller or a brush. WPEM is a software product that uses mathematical models developed from small chamber data to estimate the emissions of chemicals from oil-based (alkyd) and latex wall paint. This is then combined with detailed use, workload and occupancy data (e.g., amount of time spent in the painted room, etc.) to estimate exposure.

Appendix 8.2 provides a summary table of the models discussed above.

### 4.3.3 Review of ongoing or recently completed indoor air emission, exposure and risk assessment studies

#### **AIRMEX**

See also: <http://web.jrc.ec.europa.eu/eis-chemrisks/toolbox.cfm> (2006)

AIRMEX (the 'European Indoor Air Monitoring and Exposure Assessment Project') investigated indoor air quality and personal exposure concentration by measuring 19 volatile organic compounds in indoor air, such as carbonyls, terpenoids and aromatics, in public buildings and kindergartens in a number of cities across Europe. AIRMEX is funded under the EU's Joint Research Centre (JRC).

The researchers performed analyses in Athens and Thessaloniki (Greece), Leipzig (Germany), Catania and Milan (Italy), Brussels (Belgium), Nicosia (Cyprus) and Arnhem and Nijmegen (the Netherlands). The full project also included the cities of Dublin (Ireland), Helsinki (Finland) and Budapest (Hungary). To facilitate pan-European inter-city relationships, the data were incorporated into the EIS-ChemRisks Toolbox AIRMEX database.

Links: [http://ihcp.jrc.ec.europa.eu/our\\_databases/airmex](http://ihcp.jrc.ec.europa.eu/our_databases/airmex)  
[http://ec.europa.eu/research/headlines/news/article\\_10\\_02\\_26\\_en.html](http://ec.europa.eu/research/headlines/news/article_10_02_26_en.html)

#### **BUMA (Prioritisation of Building Materials as indoor pollution sources)**

See also: <http://www.mech.uowm.gr/bumaproject/> (2003-2008)

The BUMA project produced a database of current quantified emitted compounds by construction products and other building materials including particleboards, wood products, paints, adhesives, vinyl tiles and coving etc. It aimed to assess human exposure to air hazards emitted by such building materials that are commonly used in Europe in order to improve

understanding of the sources of hazardous VOCs and other compounds that exist in the indoor environment. An indoor exposure modeling system with guidelines is linked to the database in order to estimate exposure.

**EDETOX** (Evaluations and Predictions of Dermal Absorption of Toxic Chemicals):

See also: <http://research.ncl.ac.uk/edetox/theedetoxdatabase.html> (2005)

This was a three year study funded by the EU Framework V research programme to generate data to improve knowledge of the dermal absorption process for a number of environmental and occupational contaminants and to provide relevant quantitative data which can be used directly in the risk assessment for dermal exposure, including information that will allow regulatory authorities to progress towards assignment of quantitative skin notations for potentially hazardous chemicals.

**EnVIE:** <http://www.envie-iaq.eu/> (2007)

The aim of the EnVIE project was to increase the understanding of Europe-wide public health impacts of IAQ by identifying the most widespread and significant indoor causes for these health impacts and evaluating the existing and optional building and housing related policies for controlling them. EnVIE identified literature on IAQ research projects as well as the EU, WHO, and expert groups that have existed during the past 20 years.

**EXPOLIS:** <http://www.ktl.fi/expolis/> (1996)

The EXPOLIS study represents the first venture into measurement of the exposure of populations in six European cities to key air pollutants. Exposure to fine particulates (PM<sub>2.5</sub>), carbon monoxide (CO), volatile organic compounds (VOCs) and nitrogen dioxide (NO<sub>2</sub>) were studied in working age populations in large European cities including Helsinki and Athens.

**HEIMTSA** (Health and Environment Integrated Methodology and Toolbox for Scenario Assessment):

See also: <http://www.heimtsa.eu/Home/tabid/152/language/en-GB/Default.aspx>

Publications link: <http://www.heimtsa.eu/Results/Publications/tabid/3427/Default.aspx>

This study developed methodology for health impact and cost benefit analysis, so that overall environment and health impacts caused by releases of substances into the environment from all relevant human activities could be evaluated at the European level, as reliably as practicable given current knowledge. A special WP of HEIMTSA was dedicated to case studies; among them the “Indoor air” case study dealt with contaminants of high interest in the EU, namely radon, naphthalene, ETS and formaldehyde. The methodology used a chain starting from emissions, through to indoor concentrations, human exposure, internal dose, health effects and finally monetary valuation for all EU countries. Alternatively, exposure proxies like statistics regarding living with a smoker, or indoor PM<sub>2.5</sub> concentration as proxy to ETS exposure were tested and applied in conjunction with either toxicologically or epidemiologically-derived exposure-response functions.

**INDEX:** (Critical Appraisal of the Setting and Implementation of Indoor Exposure Limits in the EU) (2002)

Links: [http://ec.europa.eu/health/ph\\_projects/2002/pollution/fp\\_pollution\\_2002\\_exs\\_02.pdf](http://ec.europa.eu/health/ph_projects/2002/pollution/fp_pollution_2002_exs_02.pdf)  
<http://jrc.ec.europa.eu/>  
Final report:  
[http://ec.europa.eu/health/ph\\_projects/2002/pollution/fp\\_pollution\\_2002\\_frep\\_02.pdf](http://ec.europa.eu/health/ph_projects/2002/pollution/fp_pollution_2002_frep_02.pdf)

To assess the health risks of indoor air pollutants at prevailing concentration levels in Europe, the Joint Research Centre (JRC) of the European Commission carried out a project called "Critical Appraisal of the Setting and Implementation of Indoor Exposure Limits in the EU" (INDEX). The aims of the project were: (1) to assess health risks of indoor-originated chemical pollutants that might be regulated in the EU and (2) to provide suggestions and recommendations on potential exposure limits or other risk management measures. The results of the INDEX project should contribute to the development of an EU strategy for the management of IAQ. The highest priority was given in this study to: formaldehyde, nitrogen dioxide, carbon monoxide, benzene and naphthalene. Exposure limits, recommendations and management options were also given to minimize the health risks for these compounds.

**THADE:** Towards Healthy Air in Dwellings in Europe

See also: <http://www.efanet.org/activities/documents/THADEHandout.pdf> (2002)

Following in the wake of the very successful 'Indoor Air Pollution in Schools' (2000) study, in 2002 the European Federation of Allergy and Airways Diseases Patients Associations (EFA) was awarded a grant by the European Commission (DG SANCO) for a project entitled 'Towards Healthy Air in Dwellings in Europe – THADE'.

The aim was to compile an overview of evidence-based data about exposure to indoor air pollution and its health effects, particularly as regards allergies, asthma and other respiratory

diseases such as COPD; review cost-effective measures and technology to improve indoor air quality; review legislation and guidelines on indoor air pollution; and recommend an integrated strategy that defines appropriate indoor air quality policies for implementation in Europe.

## 5. DETERMINANTS OF EXPOSURE: CONCENTRATION MATRIX

In light of the information from the literature review of recent studies on indoor pollutants and discussions within the INTERA project team, extending through to the 3 project webinars held during summer 2010, the following determinants of exposure matrix has been generated for the airborne concentrations (inhalation route). Ingestion and dermal exposure will be important for materials such as PBDEs, Nicotine and to a lesser extent VOCs and PAHs but much of that will be driven by behavioural/activity related parameters.

Chemical / Determinants	Outdoor Levels	Ventilation	ETS	Buildings	Household products	Presence of gas appliances	Furnishings	Animals, plants & dampness
Radon		++		++				
Carbon monoxide	+	++	+	+		+++		
Carbon dioxide		++	++			++		+
Nitrogen dioxide	+	++	+			+++		
Aldehydes	+	++	+	+++		+	+++	+
PAHs		++	++	+++				
PBDEs		++		+++	+		+++	
Nicotine		+	+++					
VOCs	++	++	++	+	++		++	

Influence of outdoor levels includes seasonal variation, urban pollution, and proximity to main roads or gasoline stations.

Building parameters such as age, whether the home is a house or a flat, on which level the measurements were taken, such as ground level versus first level, and construction materials used have an impact of the concentrations of certain chemicals. Building characteristics include features such as double glazing and central heating, type of flooring - presence of wall to wall carpets as opposed to smooth floors, recency of ceiling covering and painting or decorating.

Household products include cleaning agents and detergents, air fresheners, do-it-yourself (DIY) products such as solvents, paint remover and latex paints.

Other factors which should be taken into account are time activity patterns: how much time is spent indoors at home differs among individuals; smoking behavior and presence of household pets.

+ Symbols indicate a semi-quantitative assessment of contribution as follows: +some influence; ++considerable determinant of exposure; +++the primary concentration determinant for that pollutant.

## 6. CONCLUSIONS

This work package has reviewed the recent literature where measurement of inhalation, dermal and ingestion exposure to indoor pollutants has been performed in domestic or home-life settings within the EU. A comprehensive database of scientific literature has been established with a total of 57 papers satisfying the review inclusion criteria. Summary data is provided here with full copies of these scientific papers being compiled within an online resource and made available to members of the INTERA study team for assimilation in to WP2 and other elements of the full chain modeling process. The review process has also identified a total of 29 relevant model systems that deal in some way with indoor air exposure characterization. Some of these are broad in scope focusing primarily on risk while others are much narrower examining single stage processes of any given individual's exposure profile. The report provides current web-based links to all of these models and where possible details of final reports and any peer-reviewed publications arising from these models.

In tandem with this ongoing review the INTERA project team and invited experts from across the EU have engaged in a series of meetings and webinars throughout 2010. The result has been the generation of an exposure determinant matrix for the consumer chemicals that the INTERA project has decided to concentrate on. The matrix and other material presented in this report will be used by the second work package of INTERA to collate the available information on indoor contaminant exposures in to a comprehensive knowledge management system.

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## **8. APPENDICES**

### **8.1 MEMBER STATES OF THE EUROPEAN UNION (As at February 2011)**

Austria  
Belgium  
Bulgaria  
Cyprus  
Czech Republic  
Denmark  
Estonia  
Finland  
France  
Germany  
Greece  
Hungary  
Ireland  
Italy  
Latvia  
Lithuania  
Luxembourg  
Malta  
Netherlands  
Poland  
Portugal  
Romania  
Slovakia  
Slovenia  
Spain  
Sweden  
United Kingdom

From website:

[http://europa.eu/abc/european\\_countries/eu\\_members/index\\_en.htm](http://europa.eu/abc/european_countries/eu_members/index_en.htm)

## 8.2 SUMMARY OF MODELS

Model Acronym	Title	Type of model	Exposure route	Website
APEX (TRIM)	Air Pollutants Exposure Model (Total Risk Integrated Methodology)	Micro-environmental IAQ	Inhalation	<a href="http://www.epa.gov/ttn/fera/human_apex.html">http://www.epa.gov/ttn/fera/human_apex.html</a>
CALENDEX™	CALENDEX™	Integrated human exposure assessment	Inhalation, dermal, ingestion	<a href="http://cfpub.epa.gov/crem/knowledge_base/crem_report.cfm?deid=76394">http://cfpub.epa.gov/crem/knowledge_base/crem_report.cfm?deid=76394</a> <a href="http://www.exponent.com/calendex_software/">http://www.exponent.com/calendex_software/</a>
CARES	Cumulative and Aggregate Risk Evaluation System	Integrated human exposure assessment	Dermal, ingestion	<a href="http://cfpub.epa.gov/crem/knowledge_base/crem_report.cfm?deid=213910&amp;view=pdf">http://cfpub.epa.gov/crem/knowledge_base/crem_report.cfm?deid=213910&amp;view=pdf</a>
CASE	Cumulative and Aggregate Simulation of Exposure	Integrated human exposure assessment	Inhalation, dermal, ingestion	Canales RA and Leckie JO., Application of a stochastic model to estimate children's short term residential exposure to lead, <i>Stoch Environ Res Risk Assess</i> (2007), 21: 737-745.
CEPST	Chemical exposure priority setting tool	Integrated human exposure assessment	Inhalation, dermal, ingestion	<a href="http://www.thelifelinegroup.org/CEPTS/index.htm">http://www.thelifelinegroup.org/CEPTS/index.htm</a>

## SUMMARY OF MODELS (CONTINUED)

Model Acronym	Title	Type of model	Exposure route	Website
CONTAM	Multizone Airflow and Contaminant Transport Analysis Software	Multizone mass balance	Inhalation	<a href="http://www.bfrl.nist.gov/IAQanalysis/CONTAM/overview/1.htm">http://www.bfrl.nist.gov/IAQanalysis/CONTAM/overview/1.htm</a>
ConsExpo 4.1	Consumer Exposure	Integrated human exposure assessment	Inhalation, dermal, ingestion	<a href="http://www.rivm.nl/en/healthanddisease/productsafety/ConsExpo.jsp">http://www.rivm.nl/en/healthanddisease/productsafety/ConsExpo.jsp</a>
DEEM/DEPM	Dietary exposure evaluation model/dietary exposure potential model	Integrated human exposure assessment	Ingestion	<a href="http://www.epa.gov/microbes/depm.htm">http://www.epa.gov/microbes/depm.htm</a>
E-FAST	Exposure and fate assessment screening tool	Integrated human exposure assessment	Inhalation, dermal, ingestion	<a href="http://www.epa.gov/opptintr/exposure/pubs/efast.htm">http://www.epa.gov/opptintr/exposure/pubs/efast.htm</a>
EUSES	European Union system for the evaluation of substances	Integrated human exposure assessment	Inhalation, dermal, ingestion	<a href="http://ecb.jrc.ec.europa.eu/documents/Existing-Chemicals/EUSES/EUSES_2.1/EUSES_2.1_documentation/EUSES_2.1_User_Manual.pdf">http://ecb.jrc.ec.europa.eu/documents/Existing-Chemicals/EUSES/EUSES_2.1/EUSES_2.1_documentation/EUSES_2.1_User_Manual.pdf</a>

**SUMMARY OF MODELS (CONTINUED)**

<b>Model Acronym</b>	<b>Title</b>	<b>Type of model</b>	<b>Exposure route</b>	<b>Website</b>
IAQX	Simulation tool kit for IAQ and inhalation exposure	Multizone mass balance	Inhalation	<a href="http://www.epa.gov/nrmrl/appcd/mmd/iaq.html">http://www.epa.gov/nrmrl/appcd/mmd/iaq.html</a>
I-BEAM	Indoor air quality building education and assessment model	Qualitative, educational IAQ model	Inhalation	<a href="http://www.epa.gov/iaq/largebdgs/i-beam/index.html">http://www.epa.gov/iaq/largebdgs/i-beam/index.html</a>
INDAIR-2	Indoor air	Micro-environmental mass balance	Inhalation	Dimitroulopoulou C., Ashmore M.R., Hill M.T.R., Bryne M.A and Kinnersley R. 2006, "INDAIR: A probabilistic model of indoor air pollution in the UK." <i>Atmospheric Environment</i> , vol. 40, no. 33, pp. 6362-6379.
MCCEM v1.2	Multi-chamber concentration and exposure model	Multizone mass balance	Inhalation	<a href="http://www.epa.gov/oppt/exposure/pubs/mccem.htm">http://www.epa.gov/oppt/exposure/pubs/mccem.htm</a>
MIAQ	Multi chamber indoor air quality model	Multizone mass balance	Inhalation	<a href="http://www.exposurescience.org/MIAQ">http://www.exposurescience.org/MIAQ</a>

**SUMMARY OF MODELS (CONTINUED)**

<b>Model Acronym</b>	<b>Title</b>	<b>Type of model</b>	<b>Exposure route</b>	<b>Website</b>
PARAMS 1.0		Multizone mass balance	Inhalation	<a href="http://www.epa.gov/nrmrl/appcd/mmd/iaq.html">http://www.epa.gov/nrmrl/appcd/mmd/iaq.html</a>
PROMISE	PRObabilistic Methodology for Improving Solvent Exposure assessments		Inhalation, dermal, ingestion	<a href="http://www.sielkenassociates.com/DesktopDefault.aspx?tabid=1309">http://www.sielkenassociates.com/DesktopDefault.aspx?tabid=1309</a>
RISK		Multizone mass balance model	Inhalation	<a href="http://www.epa.gov/nrmrl/appcd/mmd/iaq.html">http://www.epa.gov/nrmrl/appcd/mmd/iaq.html</a>
SHEDS	Stochastic human exposure and dose simulation		Inhalation, dermal, ingestion	<a href="http://www.epa.gov/heasd/products/sheds_multimedia/sheds_mm.html">http://www.epa.gov/heasd/products/sheds_multimedia/sheds_mm.html</a>
SWIMODEL	Swimmer exposure assessment model		Ingestion	<a href="http://www.epa.gov/oppad001/swimodel.htm">http://www.epa.gov/oppad001/swimodel.htm</a>
WPEM	Wall paint exposure model		Inhalation	<a href="http://www.epa.gov/opptintr/exposure/pubs/wpem.htm">http://www.epa.gov/opptintr/exposure/pubs/wpem.htm</a>