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European Parliament Pilot Project on Exposure to Indoor Air Chemicals and Possible Health Risks

Final Report

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The mission of the IHCP is to provide scientific support to the development and implementation of EU policies related to health and consumer protection.

The IHCP carries out research to improve the understanding of potential health risks posed by chemical, physical and biological agents from various sources to which consumers are exposed.

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Executive Summary

This report outlines the results of the 2-year pilot project on indoor air quality and potential health effects executed by the Joint Research Centre and funded by the European Parliament via the Directorate-General Health and Consumer Protection. It had four distinct objectives as follows:

1) to identify and quantify the main air pollutants present in public buildings, including indoor environments where children frequently stay, like schools and kindergartens,

2) to identify the main sources of these pollutants by applying source apportionment analyses,

3) to estimate people's exposure to these pollutants while working and/or living in these areas and combined with micro-environmental activity patterns during the day,

4) to evaluate possible health risks due to (chronic) exposure to air pollutants, in particular, for children.

In the frame of this work, measurement campaigns were conducted on chemical and biological pollution in the indoor air at selected public buildings, schools/kindergartens and personal dwellings in five European cities (capitals and medium-size cities alike) . These data were enhanced with results from the AIRMEX project previously obtained by the JRC. They include ca. 1000 measurements made in Nicosia (CY), Athens and Thessaloniki (EL), Catania and Milan (I), Dublin (IRE), Budapest (HUN), Brussels (B), Arnhem and Nijmegen (NL), Helsinki (FIN), and Leipzig (D). The chemical measurements focused on volatile organic compounds (VOCs), carbonyls and (in some cases) on fine and ultra-fine particulate matter in the indoor and ambient air. Biological measurements included both fungal contamination and estimation of the inflammatory potential of indoor air. In addition to the environmental measurements, personal exposure concentrations of VOCs and carbonyls were taken with the help of volunteers.

A literature review of potential health effects associated with the indoor air contaminants found in the aforementioned confined environments was undertaken and its results are reported here. In addition, a case study on health risks from indoor benzene exposure was completed using a biologically-based dose-response model to estimate the biologically effective dose of benzene and its metabolites in the presence of realistic and most common mixtures of VOCs. Whole DNA gene expression micro-array technology was used to investigate the definition of biomarkers of early biological events associated to chronic exposure to indoor and outdoor air mixtures and to their individual components, following a toxicogenomics approach.

The results indicate that indoor air pollution concentrations are consistently higher than the respective outdoor ones for the chemical families this study focused on. Differences attributable to variation in consumer behaviour, climate and type of building materials used, have been identified in the indoor/outdoor ratio of primary pollutants across Europe. These differences account for a small variance in the corresponding health risk to the local population across the EU. The project results will be presented on January 31, 2008 at a workshop to be held at the European Parliament.

It has to be noted that this was only a pilot project and its findings present limitations due to the restricted size of the samples taken and measured, which are by no means representative of the European Union as a whole. Markov chain Monte Carlo techniques have been used to

increase the statistical robustness of the health risk estimates presented herein. The latter were calculated based on stochastic simulations of risk for a population sample size of 20,000 for each European city participating in the study, simultaneously varying exposure levels and conditions, physiological and biochemical parameters that may modify actual health response at the individual level. Nevertheless, although the results are useful in highlighting factors that may influence the potential health risk from indoor air pollutants in different European professional and living settings, they cannot and should not be used to extract generalizable extrapolations on the actual health consequences of indoor air pollution in the European Union. Such conclusions would require the set-up of a much wider study designed specifically to identify the need for public health policy action at the Community level. This report outlines in its conclusions the main recommendations towards the strategic direction of such a large-scale follow-up project.

Scope

The objectives of the project are: 1) to identify and quantify the main air pollutants present in public buildings, including indoor environments which children frequent, like schools and kindergartens, 2) to identify the main sources of these pollutants applying source apportionment analyses, 3) to estimate people's exposure to these pollutants while working and/or remaining in these areas and combined with micro-environmental activity patterns during the day and 4) to evaluate possible health risks due to (chronic) exposure to air pollutants, in particular, for children.

Introduction

It is nowadays widely accepted that indoor air quality plays a significant role on total human exposure to air pollutants. The European Commission, through its Environment and Health Strategy (European Commission, 2003) and Action Plan (European Commission, 2004) and more recently through its Health and Consumer Protection Strategy (European Commission, 2005), promotes research on health impacts of environmental factors including consumer products and strongly supports efforts to identify the strength of indoor sources and reduce indoor emissions to improve human health and well-being.

Several EU-funded research programmes have been carried out, or are on-going, with the aim to individuate the main determinants of indoor air pollution, map them and explain their different role and distribution at geographical and national level. In the following paragraphs the projects which have given the most significant results are outlined in conjunction with their key findings.

EXPOLIS is a study on air pollution exposure distributions of adult urban populations in Europe. It started in 1996 as a project within the 4th Framework Programme for Research and Technology Development (RTD) (1994-98) and was completed in 1998 (http://www.ktl.fi/expolis). One of the most relevant results of this project is a database containing personal exposure data, home and work concentrations of main indoor pollutants (CO, $PM_{2.5}$, NO_2, about 30 VOCs), questionnaires. This represents a rich source of exposure-related information and a basis for further research (Jantunen et al, 1999).

The EXPOLIS-INDEX study, funded by CEFIC, investigated the factors which determine human exposures to air pollutants in indoor environments. This information is crucial for assessing the health risks related to indoor exposures and proposing mitigation strategies for harmful indoor contaminants. To achieve these goals, measurements of pollutant concentrations on person and air pollution levels in homes, at work and outdoors were combined with information on time spent in indoor and outdoor locations. The results concerning exposure to European indoor environments in Athens, Basel, Grenoble, Milan, Helsinki, Oxford and Prague (e.g. VOCs and PM2.5) were larger within city variation in exposure than on average between city variation (Künzli et al, 2004). The health consequence of these variations was deemed to be insufficiently understood.

The INDEX project (Critical Appraisal of the Setting and Implementation of Indoor Exposure Limits in the EU) started in 2002 and was concluded in 2005. The project was financially supported by DG SANCO and it was coordinated and carried out by the JRC in collaboration with leading European experts in the area of indoor air pollution. The scope of INDEX was to

identify priorities and to assess the needs for a Community strategy and action plan in the area of indoor air pollution (http://ec.europa.eu/health/ph_projects) (Kotzias et al, 2005) (http://www.jrc.ec.europa.eu/pce). Typical micro-environmental and personal exposure concentrations, as summarized from population studies reviewed in the project, are given in the table below. These data cover priority organic compounds such as aromatics, aldehydes and terpenes, as well as classical inorganic pollutants such as CO and NO_2. A comparative view of the summary results indicates that indoor concentrations of these compounds are significantly higher than outdoor values, while personal exposure concentrations are much higher than both.

Table 1: Typical European microenvironmental and exposure concentrations ($\mu g/m^3$, except CO, which is given in mg/m^3) summarized from population-based studies reviewed in the INDEX report.

	In[1]	W[1]	Out[1]	P[1]
Aromatics				
Benzene	2-13	4-14	1-21	3-23
Naphthalene	1-90	2-8	1-4	2-46
Styrene	1-6	3-7	1-2	1-5
Toluene	15-74	25-69	3-43	25-130
m&p-Xylenes	4-37	25-121	2-23	25-55
o-Xylene	2-12	7-29	1-8	8-15
Aldehydes				
Acetaldehyde	10-18	3	1-2	8
Formaldehyde	7-79	12	2-4	21-31
Terpenes				
a- Pinene	11-23	1-17	1-7	7-18
Limonene	6-83	11-23	5-9	19-56
Classical pollutants				
CO	0.5-1	1	2	0.8-1.7
NO_2	13-62	27-36	24-61	25-43

[1] In, W, Out, P = Indoor, workplace, outdoor and personal exposure concentrations

These data have been further enhanced via AIRMEX, a multi-annual programme of measurement campaigns across Europe executed by the European Commission's JRC with the help of local scientific partners in the hosting cities.

For reasons of comparison, table 2 reports on previous (particularly indoor) concentrations of typical components of indoor air given by the WHO in 1987.

Another European project designated 'Towards Healthy Air in Dwellings in Europe' (THADE) was designed to collect data about airborne pollutants in homes and to draw up recommendations that could serve as a basis for a European strategy to improve the quality of air in dwellings (http://www.efanet.org/activities/documents). This project focused on respiratory and allergic effects of the main indoor air pollutants (shown in table 1), the elaboration of guidelines and standards regarding indoor air quality in dwellings and recommendations for other European programmes on indoor air quality in dwellings (Franchi et al., 2006a-b).

Within the EP Pilot Project on Indoor air, the JRC-IHCP aims to systematically evaluate the relationship between indoor air pollution and human (chronic) exposure to pollutants. The experimental approach consists of field monitoring campaigns within several European cities in collaboration with local authorities and European institutions. These values are then used

in toxicogenomics experiments to identify biomarkers of early biological events linked to the onset or exacerbation of disease after long-term exposure to indoor air chemicals.

Table 2: 'Normal' residential indoor air concentrations of selected organic pollutants reviewed by a Working Group of WHO in 1987 (adapted from WHO 1989 and Maroni et al 1995).

	Concentration (µg/m^3)				
Pollutant	**10%**	**median**	**90%**	**AM**	**Sources**
Aromatics					
Benzene	2	10	20	10	fuel component, tobacco
Naphtalene		2	5		solvent moth balls
Styrene	< 1	1	5		fuel component
Toluene	30	65	150	80	fuel component, solvent
m,p-xylene	10	20	40	20	fuel component, solvent
o-xylene	3	5	10	10	fuel component, solvent
Aldehydes					
acetaldehyde		10	30		cigarette smoke
formaldehyde		25	60	40	chipboard, urea-formaldehyde insulation
Terpenes					
a-pinene	2	10	20	10	wax, wood product
limonene	2	15	70	30	odorant, detergent

10% and 90% = ith percentile, AM = arithmetic mean

Measurement of indoor, outdoor air pollution and personal exposure

To meet these objectives, a set of chemical compound classes (aromatics, carbonyls, terpenes) including priority indoor volatile organic compounds established by the INDEX project (Kotzias et al, 2005) have been selected for outdoor, indoor and personal monitoring in field measurement campaigns in various European cities (see table 3).

Table 3. Volatile organic compounds and carbonyl compounds investigated in this study

VOC	Carbonyls
Hexane	Formaldehyde
Benzene	Acetaldehyde
Toluene	Propanal
Ethylbenzene	Hexanal
m/p-Xylene	
o-Xylene	
1,2,4-Trimethylbenzene	
α-Pinene	
d-Limonene	

The coarse and fine fractions of particulate matter ($PM_{10-2.5}$, i.e. particles with aerodynamic diameter between 2.5 and 10 microns and $PM_{2.5}$, i.e. particles with aerodynamic diameter lower than 2.5 microns) that can induce inflammatory response are part of the parameters measured indoors. On-site analyses of relevant interior surfaces are also considered; the analysis of these data combined with those of the indoor/outdoor air environment are expected to allow us to identify the main sources of indoor air pollutants. A total of 991 samples were taken and analysed in the JRC analytical indoor air chemistry laboratories.

The measurements were conducted in public buildings, kindergartens and schools. Volunteers were identified among the employees and/or teachers working in the selected indoor environments for personal exposure monitoring. All measurements were carried out by means

of passive samplers (Radiello, charcoal type for VOCs and DNPH-covered for carbonyl compounds). The passive samplers were installed inside and outside the buildings and for personal monitoring. All the samplers used during the measuring campaigns were sent to the JRC from the local authorities and chemical analyses were performed. The use of passive samplers allows the determination of an average concentration for the time-length of the campaign (7 days). It is considered more suitable than active sampling (via pumps), which only provides information on the actual pollutant concentration (spot measurements over a very short period of time). Personal exposure monitoring is performed for VOCs and for carbonyl compounds.

The indoor/outdoor measuring campaigns always lasted one week, including weekends, for a total of 7 days. For personal exposure monitoring, volunteers received personal passive samplers to wear for two to three consecutive full days (the time spent at home, in the office, on transport and commuting was accounted for via dedicated questionnaires / time sheets, in order to allow us to investigate source-to-personal exposure scenarios during the 2nd phase of the study). Personal samplers are placed in the breathing area of each volunteer during the daytime and in the bedroom in the vicinity of the sleeping person during the night. Indoor (working place and home of each volunteer) and outdoor concentrations were also monitored. Volunteers also received a questionnaire for the registration of micro-environmental activity patterns. Private homes of the volunteers, upon their agreement, were monitored as well.

This final report builds on indoor, outdoor and personal data obtained by the JRC during measurement campaigns on public buildings, kindergartens and schools in various European cities in the frame of the project; these data are enriched with JRC measurements made in the context of the AIRMEX project (European Indoor Air Monitoring and Exposure Assessment Study). The data reported herein cover the period 2004-2007.

Current results indicate that personal exposure concentrations of the selected compounds were higher than indoor/outdoor concentrations, underlining the importance of individual behaviour and micro-environmental activity patterns on the determination of overall personal exposure. In most cases they were twice as high (or even higher) than indoor concentrations and significantly higher than outdoor concentrations. Generally, total VOC concentrations inside public buildings are higher and/or similar to the outdoor air concentrations. In some cities of Central Europe, VOC indoor concentrations are higher than outdoors (however the overall, indoor and outdoor concentrations are the lowest measured overall). In cities of Southern Europe, e.g. Athens, Catania, in buildings located in the city centre there is almost no difference between indoor and outdoor pollutant levels for the measured compounds. The lowest ratio between indoor and outdoor concentrations for benzene is 0.93 in Thesalloniki (GR) in May 2006, whilst the highest value (5.94) was observed in Leipzig in July 2006. This ratio is hardly ever lower than one, and can be as high as close to six. Similarly to benzene, the lowest value of the indoor:outdoor concentration ratio for toluene (0.74) was observed in Athens (GR) in December 2003; for xylene (0.83) in Catania (I) in May 2005; for ethylbenzene (0.86) in Catania (I) in October 2003. The highest value of the same ratio for toluene (5.25) was observed in Leipzig (D) in April 2005, while for xylene (2.33) and for ethylbenzene (3.11) in Leipzig (D) in July 2006. This trend may be attributed to different indoor ventilation regimes due to both consumer behaviour and climatic conditions and, partially, to different use of consumer goods in key sectors such as furniture or carpets. Other factors that may influence the indoor:outdoor ratio for these compounds are the ambient air temperature and humidity, the volume of the rooms, the age of the furniture and of the buildings.

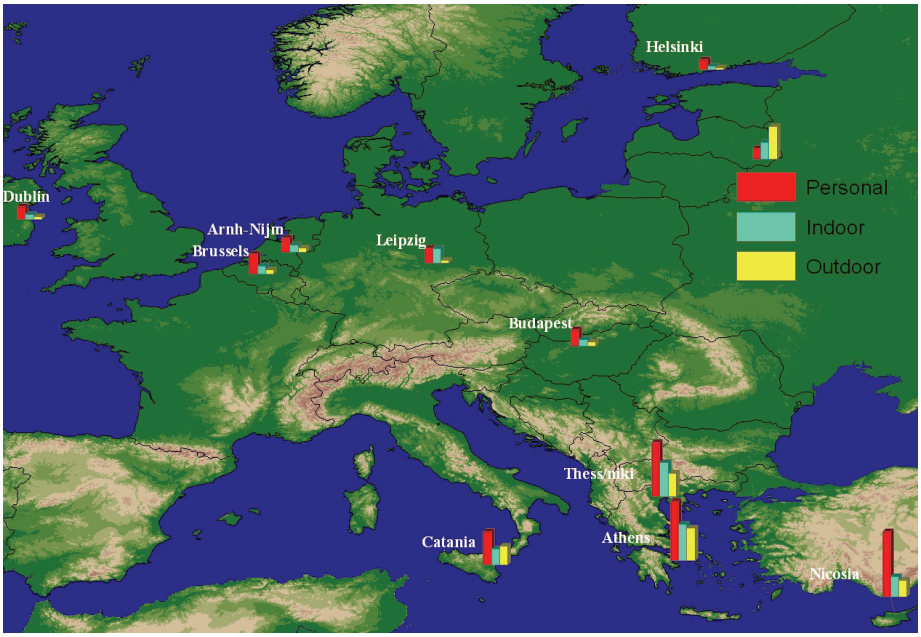

Figure 1: Geographical variation of total VOCs in public buildings across the EU

The sum of selected **VOCs measured** inside public buildings (see table 3) varies from a few micrograms (ca. 8 µg/m^3) to ca. 280 µg/m^3. **Outdoor** concentrations considering all sites vary from ca. 7 to ca. 150 µg/m^3. VOC concentrations in offices inside buildings can be very high depending on the smoking/non-smoking behaviour of the employees. Generally the aromatic compounds benzene, toluene, ethyl-benzene and the xylenes represent the major part of the measured VOCs.

Table 3: Total VOC and benzene concentrations (µg/m^3) measured in public buildings of ten European cities. Samples were simultaneously collected outside and inside buildings with personal samplers on volunteers working in such buildings. Minimum, maximum and mean values (in bracket) are shown.

City	VOC[t] Outdoor	VOC[t] Indoor	VOC[t] Personal	Benzene Outdoor	Benzene Indoor	Benzene Personal
Catania (October)	44.8 – 105.8 (67) n=3	39.6 – 157.1 (63.8) n=6	91 – 149 (112.5) n=6	5.5 – 8.0 (6.4) n=3	4.9 –17.1 (7.4) n=6	4.7 – 8.2 (6.2) n=6
Catania (May)	21.9 – 52.3 (43.6) n=3	20.9 – 40.4 (27.0) n=6	58.2 – 136.4 (79.1) n=6	2.8 – 4.7 (3.6) n=3	2.8 – 4.8 (3.9) n=6	3.1 – 7.0 (4.9) n=6
Athens (December)	49.4 – 125.2 (87.3) n=2	62.4 – 159.2 (112.4) n=4	174.4 – 312.6 (243.5) n=4	6.8 – 14.2 (10.5) n=2	7.3 – 13.3 (10.9) n=4	17 – 18.6 (17.8) n=4
Athens (October)	73.7 – 129.6 (101.6) n=2	60.3 – 136.9 (94.7) n=4	63.8 – 124.6 (98.3) n=4	6.7 – 12.4 (9.6) n=2	5.6 – 12.9 (8.8) n=4	5.0 – 9.9 (7.2) n=4
Nijmegen/Arnhem (August)	8.9 – 15.2 (12.3) n= 3	9.0 – 30.0 (18.4) n=6	11.6 – 72.9 (32.9) n=6	1.1 – 2.3 (1.7) n=3	1.0 – 2.5 (1.8) n=6	0.9 – 6.5 (2.9) n=6
Nijmegen/Arnhem (March)	7.7 – 15.0 (12.1) n=3	8.3 – 28.1 (22.5) n=6	28.0 – 74.8 (52.4) n=6	1.9 – 3.7 (3.0) n=3	1.8 – 6.2 (3.9) n=6	2.4 – 7.8 (4.6) n=6
Thessaloniki (November)	40.2 – 153.7 (80.7) n=3	58.5 – 281.8 (143.6) n=6	80.0 – 164.8 (131.7) n=6	4.4 – 15.2 (8.7) n=3	8.0 – 63.7 (33.0) n=6	8.8 – 14.2 (11.3) n=6
Thessaloniki (May)	22.4 – 88.7 (49.6) n=3	27.8 – 75.5 (49.2) n=6	64.5 – 478.7 (177.1) n=6	2.3 – 9.3 (4.8) n=3	3.1 – 7.9 (5.0) n=6	3.6 – 26.4 (11.2) n=6
Brussels (September)	10.5 – 17.3 (13.9) n=2	17.5 – 34.0 (22.7) n=8	37.4 – 101.5 (66.3) n=6	1.3 – 2.5 (1.9) n=2	1.9 – 3.9 (2.9) n=8	1.5 – 6.0 (3.4) n=6
Leipzig (April)	7.9 – 10.8 (9.7) n=4	20.1 – 81.9 (44.0) n=8	41.0 – 80.5 (59.3) n=4	1.5 – 1.8 (1.6) n=4	1.5 – 2.9 (2.0) n=8	1.9 – 5.6 (3.2) n=4
Leipzig (July)	4.6 – 7.2 (5.7) n=4	12.5 – 80.2 (38.9) n=8	10.6 – 71.4 (28.4) n=4	0.4 – 0.7 (0.6) n=4	0.7 – 3.8 (1.5) n=8	0.9 – 3.9 (1.9) n=4
Nicosia	35.6 – 71.8	32.6 – 85.3	117.2 – 279.2	4.6 – 7.8	3.7 – 9.6	4.4 – 23.3

(January)	(47.7) n=3	(57.9) n=9	(183.4) n=6	(5.7) n=3	(5.8) n=9	(10.9) n=6
Budapest (May 2007)	6.2 – 20.5 (13.0) n=4	14.3 – 23.0 (19.4) n=6	28.5 – 65.4 (47.5) n=4	0.9 – 2.1 (1.5) n=4	1.2 – 2.7 (1.9) n=6	1.2 – 2.8 (2.2) n=4
Dublin (May 2007)	6.1 – 11.9 (9.4) n=3	11.0 – 17.6 (14.2) n=6	9.2 – 67.8 (37.6) n=6	1.1 – 2.0 (1.6) n=3	1.8 – 2.9 (2.2) n=6	0.7 – 9.0 (3.6) n=6
Helsinki (August 2007)	4.6 – 11.5 (8.1) n=2	8.3 – 18.4 (11.7) n=6	13.3 – 69.6 (31.1) n=7	0.7 – 1.2 (1.0) n=2	0.7 – 1.3 (0.9) n=6	1.2 – 3.9 (1.8) n=7

Indoor/outdoor concentrations of VOCs in kindergartens and schools follow a similar trend as those found in public buildings, with higher concentrations in the indoor air than outdoors (fig. 2). **Mean VOC indoor levels (all sites) are in most cases double the corresponding outdoor values** ranging from ca. 15 µg/m^3 to 190 µg/m^3 (see table 4). Personal exposures vary, being strongly influenced by indoor and outdoor sources in association with micro-environmental activity patterns and personal behaviour.

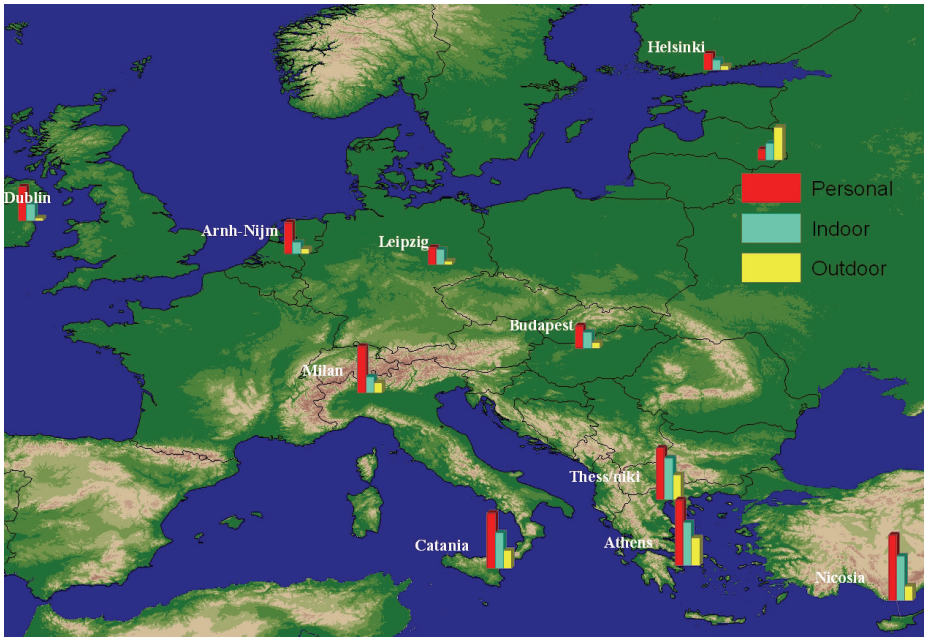

Figure 2: Total VOC levels in schools/kindergartens across Europe

In parallel to the measurements for volatile organic compounds, air samples were taken for the analysis of **carbonyl compounds**, in particular, formaldehyde and acetaldehyde, which are present both in- and outdoors. **In all sites air concentrations for aldehydes inside the buildings/kindergartens were up to 7-8 times higher than outside.** This is mostly valid for formaldehyde (HCHO), a widely used chemical, which appears to indicate that strong indoor sources exist, clearly determining indoor air concentrations. Values for HCHO in offices of public buildings vary from ca. 3 to 33 µg/m^3 (see figure 3), and in kindergartens from ca. 6 to 50 µg/m^3 (see figure 4 and table 4). The difference between the indoor and outdoor HCHO concentration varies between 3 and 14 µg/m^3. This denotes that indoor sources are dominant for this compound. Therefore there is room for reducing exposure to this compound if the main sources are identified and appropriate control measures are taken.

Table 4: Total VOC and benzene concentrations (μg/m^3) measured in schools and kindergartens in ten European towns. Samples were simultaneously collected outside and inside buildings with personal samplers on volunteers working in such buildings. Minimum, maximum and mean values (in bracket) are shown

City	$VOC_{[t]}$ Outdoor	$VOC_{[t]}$ Indoor	$VOC_{[t]}$ Personal	Benzene Outdoor	Benzene Indoor	Benzene Personal
Catania (October)	22.2 - 55.5 (36.1) *n=3*	(25.4 – 53.2) (36.6) *n=3*	68.1 – 100.8 (88) *n=3*	3.1 – 5.6 (4.2) *n=3*	3.1 – 4.4 (3.8) *n=3*	2.5 – 5.6 (3.8) *n=3*
Catania (May)	14.9 – 28.2 (22.0) *n=3*	17.9 – 192.7 (76.7) *n=3*	65.5 – 18.6 (85.7) *n=3*	2.0 – 2.9 (2.5) *n=3*	2.3 – 2.8 (2.6) *n=3*	4.0 – 6.1 (4.9) *n=3*
Athens (December)	31.7 – 39.5 (35.6) *n=3*	57.1 - 99.5 (78.3) *n=3*	104.2 – 130.7 (117.5) *n=3*	5.2 – 6.9 (5.9) *n=3*	4.9 – 10.7 (7.4) *n=3*	3.4 – 4.8 (4.2) *n=3*
Athens (October)	35.0 – 66.1 (50.5) *n=3*	35.3 – 78.2 (56.8) *n=3*	52.4 – 122.3 (87.4) *n=3*	3.6 – 6.2 (4.9) *n=3*	2.9 – 6.1 (5.0) *n=3*	4.0 – 4.6 (4.3) *n=3*
Nijmegen/Arnhem (March)	8.1 – 9.0 (8.6) *n=2*	19.6 – 36.1 (27.9) *n=2*	24.1 – 69.8 (47.0) *n=2*	2.3 – 2.5 (2.4) *n=2*	2.1 – 3.0 (2.6) *n=2*	2.3 – 7.7 (5.0) *n=2*
Nijmegen/Arnhem (August)	6.8 – 7.6 (7.2) *n=2*	8.6 – 10.9 (9.8) *n=2*	41.3 – 62.1 (51.7) *n=2*	0.9 – 1.0 (1.0) *n=2*	0.8 – 0.9 (1.0) *n=2*	2.6 – 4.3 (3.5) *n=2*
Thessaloniki (November)	13.3 – 74.9 (48.0) n=3	55.7 – 122.2 (88.0) n=3	78.5 – 150.6 (114.9) n=3	1.8 – 6.9 (4.6) n=3	2.6 – 7.5 (5.8) n=3	2.9 – 8.4 (6.1) n=3
Thessaloniki (May)	12.6 – 49.3 (29.2) *n=3*	21.1 – 63.4 (42.4) *n=3*	27.3 – 63.3 (48.0) *n=3*	1.2 – 4.7 (2.7) *n=3*	1.5 – 5.6 (3.1) *n=3*	2.3 – 4.7 (3.5) *n=3*
Leipzig (April)	7.5 – 8.6 (8.0) *n=3*	15.8 – 25.4 (20.3) *n=3*	29.1 – 53.7 (40.6) *n=3*	1.3 – 1.5 (1.4) *n=3*	1.0 – 1.8 (1.4) *n=3*	1.8 – 2.8 (2.3) *n=3*
Leipzig (July)	2.9 – 4.6 (4.0) *n=3*	18.0 – 46.7 (28.3) *n=3*	11.0 – 17.4 (14.2) *n=3*	0.4 – 0.6 (0.5) *n=3*	0.6 – 0.7 (0.7) *n=3*	0.7 – 1.2 (1.0) *n=3*
Nicosia (July)	9.1 – 14.9 (12.8) *n=3*	20.4 – 60.3 (40.5) *n=3*	46.8 – 59.5 (51.8) *n=3*	0.9 – 1.7 (1.4) *n=3*	1.1 – 2.7 (1.7) *n=3*	3.3 – 6.5 (4.9) *n=3*
Nicosia (January)	27.0 – 38.3 (33.7) *n=3*	79.7 – 118.8 (99.6) *n=3*	105.8 – 216.8 (154.6) *n=3*	3.5 – 4.9 (4.2) *n=3*	3.7 – 5.6 (4.4) *n=3*	4.4 – 6.6 (5.4) *n=3*
Budapest (May 2007)	8.8 – 9.3 (9.1) n=3	14.7 – 43.5 (24.9) n=6	26.1 – 50.9 (34.9) n=3	0.8 – 0.9 (0.8) n=3	0.9 – 5.1 (2.6) n=6	2.1 – 10.3 (4.8) n=3
Dublin (May 2007)	4.3 – 5.4 (4.8) n=3	9.0 – 50.9 (26.7) n=4	28.5 – 73.4 (53.5) n=3	0.8 – 0.9 (0.8) n=3	1.3 – 2.6 (1.9) n=4	1.3 – 12.3 (5.3) n=3
Helsinki (August 2007)	4.6 – 15.6 (8.4) n=3	16.4 – 20.1 (18.0) n=3	16.4 – 44.8 (26.6) n=6	0.8 – 1.5 (1.1) n=3	0.8 – 1.1 (0.9) n=3	1.2 – 3.5 (2.2) n=6

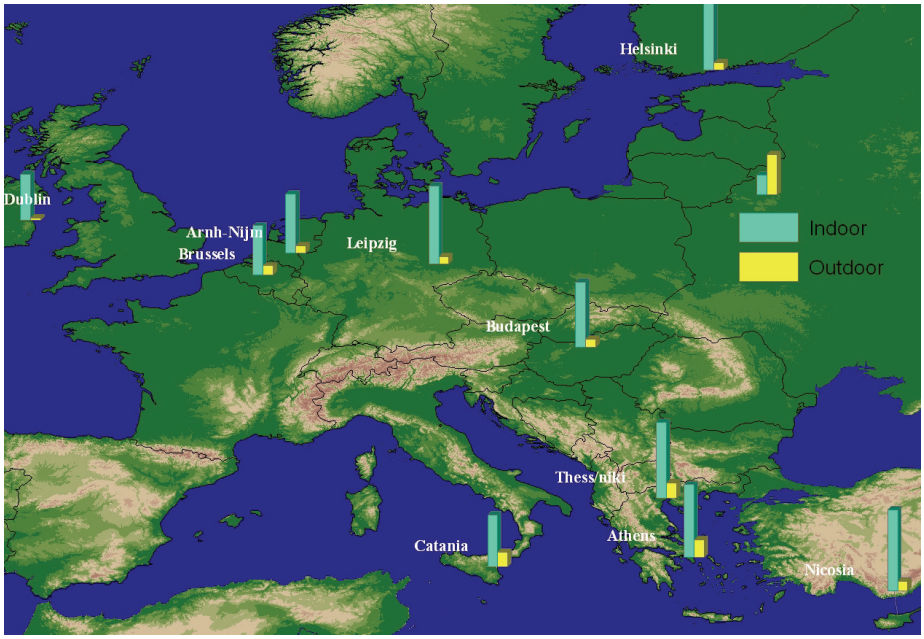

Figure 3: Formaldehyde concentrations measured in public buildings across the EU

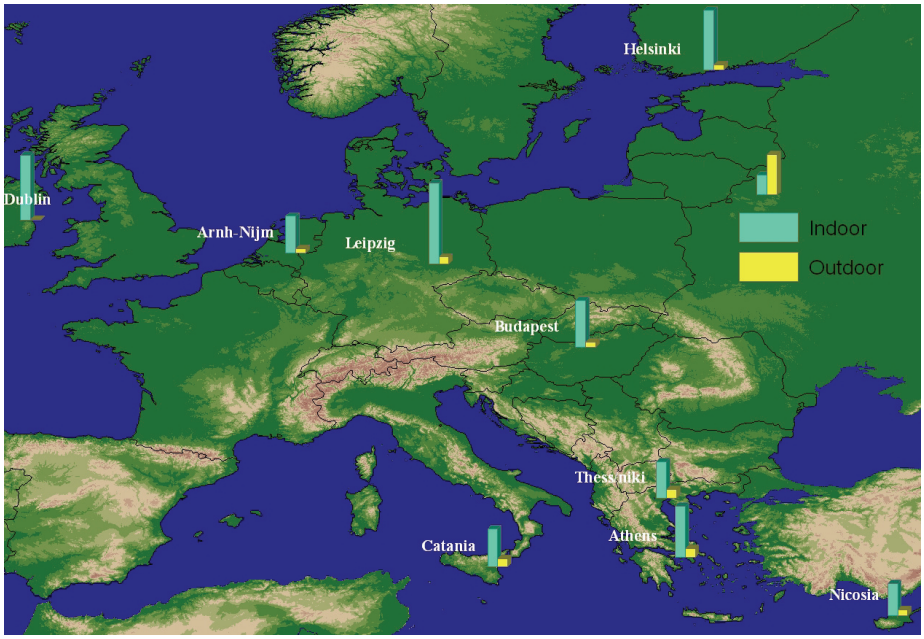

Figure 4: Levels of formaldehyde measured in schools/kindergartens in the EU

Table 5. Total carbonyl compounds and formaldehyde concentrations (μg/m^3) measured in public buildings of ten European towns. Samples were simultaneously collected outside and inside buildings. Minimum, maximum and mean values (in bracket) are shown

City	$Carb_{[t]}$ Outdoor	$Carb_{[t]}$ Indoor	$Carb_{[t]}$ Pers.	HCHO Outdoor	HCHO Indoor	HCHO Pers.
Catania (October)	7.7 – 9.2 (8.6) *n=3*	22.3 – 40.8 (29.5) *n=6*		4.3 – 4.9 (4.7) *n=3*	11.0 – 23.2 (16.1) *n=6*	
Catania (May)	6.5 – 10 (7.8) *n=3*	15.2 – 33.0 (21.3) *n=6*		3.1 – 4.4 (3.8) *n=3*	9.0 – 21.6 (13.2) *n=6*	
Athens (December)	7.4 – 11.6 (9.5) *n=2*	19.8 – 42.4 (32.8) *n=4*		3.7 – 5.6 (4.7) *n=2*	11.2 – 26.0 (20.0) *n=4*	
Athens (October)	9.8 – 13.7 (11.8) *n=2*	20.8 – 59.2 (40.1) *n=4*		4.1 – 5.8 (5.0) *n=2*	10.7 – 27.3 (20.6) *n=3*	
Nijmegen/Arnhem (March)	3.7 – 4.7 (4.3) *n=3*	8.4 – 44.3 (27.8) *n=6*		1.2 – 1.9 (1.7) *n=3*	3.1 – 12.5 (14.7) *n=6*	
Nijmegen/Arnhem (August)	2.9 -3.8 (3.6) *n=3*	13.9 – 75.7 (44.4) *n=6*		2.1 – 2.5 (2.4) *n=3*	7.6 – 33.6 (18.6) *n=6*	
Thessaloniki (November)	6.3 – 12.4 (9.2) *n=3*	34.6 – 65.5 (52.1) *n=6*		3.9 – 7.3 (5.4) *n=3*	14.1 – 29.9 (25.6) *n=6*	
Thessaloniki (May)	7.4 – 12.7 (9.7) *n=3*	36.3 – 56.8 (47.0) *n=6*		2.7 – 4.1 (3.3) *n=3*	13.9 – 21.7 (17.1) *n=6*	
Brussels (September)	3.9 – 4.5 (4.2) *n=2*	30.0 – 55.9 (33.5) *n=8*		2.7 – 3.2 (3.0) *n=2*	7.7 – 26.9 (16.6) *n=8*	
Leipzig (April)	4.9 – 6.1 (5.6) *n=4*	13.5 – 245.5 (92.4) *n=8*		2.1 – 2.4 (2.3) *n=4*	5.6 – 34.5 (21.1) *n=8*	
Leipzig (July)	3.6 – 5.7 (4.6) *n=4*	19.7 – 147.4 (81.1) *n=8*		1.1 – 2.2 (1.7) *n=4*	7.6 – 30.0 (22.6) *n=8*	
Nicosia (January)	8.3 – 12.6 (10.1) *n=3*	14.3 – 75.7 (58.2) *n=9*		2.0 – 3.7 (2.7) *n=3*	26.9 – 27.7 (27.3) *n=9*	
Budapest (May 2007)	6.2 – 7.7 (6.9) n=4	25.7 – 73.8 (51.6) n=6	57.9 – 99.2 (74.3) n= 3	1.9 – 2.6 (2.3) n=4	11.3 – 31.2 (18.5) n=6	13.8 – 22.6 (19.0) n=3
Dublin (May 2007)	3.7 – 3.9 (3.8) n=3	7.0 – 47.2 (28.3) n=6		2.0 (2.0) n=3	2.6 – 25.2 (12.9) n=6	
Helsinki (August 2007)	3.7 – 4.4 (4.1) n=2	7.4 – 125.8 (56.3) n=6	33.5 – 126.5 (71.9) n=7	1.8 – 2.4 (2.1) n=2	2.1 – 33.0 (18.9) n=6	8.3 – 29.9 (19.6) n=7

Table 6. Total carbonyl compounds and formaldehyde concentrations (μg/m^3) measured in shools and kindergartens of ten European towns. Samples were simultaneously collected outside and inside buildings. Minimum, maximum and mean values (in bracket) are shown

City	$Carb_{[t]}$ Outdoor	$Carb_{[t]}$ Indoor	$Carb_{[t]}$ Pers.	HCHO Outdoor	HCHO Indoor	HCHO Pers.
Catania (October)	4.5 – 7.2 (5.9) *n=3*	16.4 – 53.3 (31.7) *n=3*		2.9 – 4.0 (3.4) *n=3*	8.5 – 22.3 (15.7) *n=3*	
Catania (May)	6.5 – 7.1 (6.7) *n=3*	15 – 27.1 (21.2) *n=3*		2.4 – 3.2 (2.9) *n=3*	9.0 - 16.2 (13.0) *n=3*	
Athens (December)	4.2 – 7.1 (5.2) *n=3*	15.1 – 36.6 (24.5) *n=3*		2.3 – 4.0 (2.9) *n=3*	10.5 – 28.2 (18.3) *n=3*	
Athens (October)	7.3 – 9.3 (8.3) *n=3*	23.3 – 71.5 (47.4) *n=3*		3.3 – 4.1 (3.7) *n=3*	9.8 – 30.5 (20.2) *n=3*	
Nijmegen/Arnhem (March)	4.2 – 4.3 (4.3) *n=2*	19.2 – 33.2 (26.2) *n=2*		1.4 – 1.5 (1.5) *n=2*	6.1 – 11.8 (9.0) *n=2*	
Nijmegen/Arnhem (August)	2.4 – 4.2 (3.3) *n=2*	31.9 – 74.4 (53.2) *n=2*		1.5 – 2.3 (1.9) *n=2*	15.4 – 22.4 (18.9) *n=2*	
Thessaloniki (November)	4.4 – 8.7 (7.0) *n=3*	28.0 – 47.6 (35.4) *n=3*		2.7 – 4.6 (4.0) *n=3*	12.6 – 16.1 (13.9) *n=3*	
Thessaloniki (May)	7.4 – 12.0 (9.1) *n=3*	40.3 – 56.3 (48.1) *n=3*		2.1 – 3.8 (2.7) *n=3*	10.6 – 18.2 (13.8) *n=3*	
Leipzig (April)	4.7 – 4.8 (4.8) *n=3*	44.6 – 94.7 (62.7) *n=3*		2.5 – 2.6 (2.6) *n=3*	12.5 – 49.7 (29.1) *n=3*	
Leipzig (July)	5.8 – 6.7 (6.2) *n=3*	98.2 – 101.0 (99.2) *n=3*		2.5 – 2.7 (2.6) *n=3*	21.8 – 46.8 (31.9) *n=3*	
Nicosia (January)	7.0 – 9.9 (8.6) *n=3*	44.2 – 64.0 (51.7) *n=3*		1.7 – 2.6 (2.2) *n=3*	8.2 – 17.3 (12.0) *n=3*	
Budapest (May 2007)	5.3 – 5.8 (5.5) n=3	29.4 – 72.9 (54.1) n=6	45.3 – 52.8 (50.1) n=3	1.7 – 2.1 (1.9) n=3	10.5 – 27.1 (17.9) n=6	13.5 – 17.6 (15.6) n=3
Dublin (May 2007)	2.2 – 4.4 (2.9) n=3	16.7 – 144.0 (67.6) n=4		1.2 – 2.1 (1.6) n=3	5.6 – 48.8 (26.4) n=4	
Helsinki (August 2007)	3.3 – 5.1 (4.2) n=3	52.5 – 79.9 (64.3) n=3	40.6 – 76.6 (58.7) n=6	1.8 – 2.3 (2.0) n=3	18.6 – 29.4 (22.6) n=3	11.5 – 25.6 (17.5) n=6

Biological contamination and inflammatory potential of indoor air

Within the EP Pilot Project on Indoor Air, the effective exposure to allergens and microbiological pollutants of employees in public buildings was evaluated. As in the case of chemical indoor pollution, the experimental approach comprised field monitoring campaigns in several European cities in collaboration with local authorities and European institutions.

In each city, measurements were performed in one or two public buildings. In each building air was analyzed within three offices and at the entrance hall. Outdoor air samples were always taken as reference.

In order to assess the air quality, two methodological approaches were put in place:

1. the presence of pyrogenic components in the air was assessed by determining the total inflammatory capacity of air samples based on the *in vitro* pyrogen test (IPT);
2. microbiological contamination was determined by quantification of moulds and bacteria in the air.

The first method (IPT) provides general information on the presence of pyrogenic compounds. Indeed, it is sensitive to endotoxins, fungi spores, bacteria and their components such as lipopolysaccharide (LPS) and pyrogenic dust contaminants. All these components or organisms, which are commonly present in the air, can induce an inflammatory response in humans, through the increased production in human blood cells of the cytokine IL-1β, which is the endpoint of detection of this method.

The second method is based on classical microbiology and it provides data to calculate the following indices of contamination:

1. Global Index of Microbiological Contamination per cubic meter (GIMC/m^3). This is the sum of the number of bacteria and moulds detected per m^3 of air.
2. Index of Mesophilic Bacterial contamination (IMC). If IMC > 1 bacterial contamination is mainly due to human contamination.
3. Amplification Index (AI). This is the ratio between indoor and outdoor bacterial counts. If AI > 1 indoor air quality is worse than the outdoor due to a higher bacterial contamination.

Air samples were collected in two different ways during the monitoring campaigns. For the *in vitro* pyrogen test, air was filtered with personal air pumps (Zambelli, Italy) onto a membrane filter of monitoring cassettes to which they were connected. The pumps were placed on a table in the offices as this place is considered to be the most representative point for simulating the air inhaled by employees. At the entrance hall, pumps were placed on the reception desk. Air was collected in triplicate for 8 hours (a working day). Moreover, in one office per building, air was collected for 4, 8 and 24 hours in order to evaluate the biological pollution trend during the whole day.

For microbiological analysis, air was filtered using a SAS 100 microbial air sampler (International PBI) onto microbiological plates containing selective growth media for bacteria or for moulds. Each analysis was performed in duplicate. Afterwards plates were kept at 4°C to avoid microbial growth until they were processed.

Based on the results of the JRC measurements, no health hazards or risks were identified for all environments investigated. No specific source of biological contamination and no pathogens have been detected in the indoor environments analyzed.

However, the outdoor samples collected in Budapest show high levels of EEU per m^3 in the outdoor air (6.14 and 12.23 EEU/m^3). This trend was confirmed by microbiological analysis. Indeed the total microbial contamination, expressed by the GIMC value, is higher than 1000 CFU per m^3 of air, namely in the range of 2890-3575 CFU per m^3. A possible explanation of these results is the fact that the campaign took place in full spring blooming time and thus main contamination was due to pollens and plant microorganisms spread into the air at this time of year. This hypothesis is confirmed by the fact that the main fungi taxa present in the outdoor (88-92%) and indoor (56-89%) samples is *Cladosporium cladosporioides* and *Hyalodendron* sp., which are mainly associated to vegetation and their presence in indoor air may be considered as directly proportional to aerospore entry from the external environment. Based on taxonomical data, the main fungal source of contamination in these environments is outdoor pollution.

Data obtained from the measuring campaigns in all the other European cities are comparable. Data can be grouped by sampling place and the range of the results achieved is shown in Table 7.

Table 7: Summary of the biological pollution measurement campaigns results in 2007.

	CFU/m^3	CFU/m^3	CFU/m^3		EEU/m^3
place	**Meso. B.**	**Psychr. B.**	**Mycetes (moulds)**	**GIMC**	**I.C.**
Out	25-130	133-1010	265-555	451-1671	0.4.3.1
Entrance	115-660	245-375	35-686	468-1483	1.3-3.27
office	15-448	15-245	25-200	70-993	0.11-1.2
Archive	8-33	20-50	15-20	48-98	0.088-0.098

GIMC= global index of microbiological contamination = CFU/m^3 mesophilic bacteria + CFU/m^3 psychrophilic bacteria + CFU/m^3 mycetes

IMC= index of mesophilic bacterial contamination = (CFU/m^3 mesophilic bacteria) / (CFU/m^3 psychrophilic bacteria)

AI= amplification index = GIMC per m3 indoor air/ GIMC per m3 outdoor air

I.C= inflammatory capacity= human IL-1β (human pro-inflammatory molecule) release equivalent to that released in response to the reference LPS

Aerial microbial contamination is expressed in Colony Forming Unit per cubic meter of air (CFU/m^3). CFU is a measure of viable bacteria, based on the consideration that bacteria grow by binary division giving rise to a colony.

The "total inflammatory capacity" of the air samples is expressed in Endotoxin Equivalent Unit (EEU) per cubic meter of air. EEU indicates the IL-1β release equivalent to that released in response to the reference LPS (expressed in EU). The International Standard EU is defined as follow: 1 EU is defined as 100pg of the WHO reference LPS from *E. coli* O-113. In our experiments we used LPS from another *E. coli* strain and thus the EU is referred to 100pg of this type of LPS based on the *E. coli* LPS concentration-response curve.

Table 7 shows a different distribution of microorganisms in the different environments analyzed. Mesophilic bacteria, which are mainly of human source, are preponderant in indoor environments where the air is mainly affected by human activity. On the contrary, psychrophilic bacteria, which are of environmental source, as they have an optimum growing temperature at 25ºC, are mainly detected in outdoor samples. The concentration of moulds is higher outside than in indoor environments in this particular case.

In agreement with the data obtained during the measuring campaigns in other European cities too, the microbiological quality of the air at the entrance halls was worse compared to the air quality in the offices. This is connected with a high afflux of people during the day and therefore microbial anthropogenic contamination was preponderant in these environments (highest mesophilic bacteria counts and highest GIMC values).

In Dublin additional sampling was done in two book archives of a library. These archives are rooms with limited and restricted access and one of them has a special air cleansing system in order to preserve old books. In these two environments microbial contamination resulted very low with GIMC < 100.

The results on microbiological contamination are in accordance with results obtained by the *in vitro* pyrogen test (IPT), which provides information on the presence of pyrogenic components in the air and thus on the total inflammatory capacity (I.C.) of air samples. Indeed, indoor and outdoor samples are comparable in terms of I.C. (0.4-3.1 and 1.3-3.27 EEU/m^3 respectively) and in terms of microbial contamination (451-1671 CFU/m^3 and 468-1483 CFU/m^3 respectively). The I.C. values referred to offices are lower (I.C. < 1.2 EEU/m^3) compared to the entrance halls and outdoors, confirming low indoor contamination in the indoor environments analysed. Moreover, the I.C. values from air samples collected in the two archives are even lower than 0.1 EEU/m^3, in accordance with the microbiological results.

The results underline the good correlation of the two methodological approaches. Moreover, compared to conventional microbiological investigation which require at least one week to get the results, the *in vitro* pyrogen test (IPT) offers the advantage of providing data in two days.

Poor maintenance of the ventilation system could be a major potential source of indoor air biological contamination in indoor environments. Therefore, air quality at the outlets of the ventilation system has been investigated in the building where the heating/cooling system was on. On the basis of microbiological results, it can be concluded that the air at the outlets was, in general, clean and that the ventilation system in the buildings examined by the JRC was well maintained and did not affect the indoor air quality.

Source apportionment of the main indoor air pollutants

The causes of poor indoor air quality are many and diversified; they are of chemical, physical and biological nature. The main chemical sources are combustion products of solid fuels, such as CO, CO_2, SO_2, NO_2, particulate matter (PM_1, PM2.5, PM10), and volatile (benzene, toluene, carbonyls) or semi-volatile (phthalates, plasticizers) organic compounds and pesticides. Environmental tobacco smoke (ETS) represents another relevant source of indoor chemical pollutants. Three main domestic sources of indoor air pollution have been identified recenty: loaded particle filters, personal computers and other electronic equipment and building materials (Fanger, 2006). The former are very important because of their capacity to

store chemical and biological pollutants. In particular, volatile organic compounds (VOCs) are released from a variety of materials. Maroni et al. (1995) classified the sources of VOCs into major groups, mostly represented by consumer and commercial products; paints and associated supplies; pesticides; cosmetic care products; automotive products; furnishing and clothing, building materials, heating, ventilation and air-conditioning systems, personal sources, and outdoor sources.

In order to have a better understanding of sources contributing to the complexity of indoor air, the possibility and feasibility to carry out on-site emissions testing of relevant inner material surfaces by the use of emission cells was evaluated in some field measurement campaigns. Screening measurements of emissions from flooring surfaces (parquet, wall to wall carpets) and furniture (tables, chairs and sometimes cupboards) of different composition (see table 8) have been performed on site in 10 offices by using a glass cell positioned over the selected surface.

Table 8: Description of material surfaces for on-site analysis

Office	Floor	Table	Chair	Cupboard
1	Wall to wall carpet	Laminate	Textile/plastic	
2	Wall to wall carpet	Wood	Textile/plastic	
3	Wall to wall carpet	Wood	Textile/plastic	
4	Linoleum	Wood	Textile/plastic	Laminate
5	Linoleum	Laminate	Textile/Wood	
6	Linoleum	Laminate	Textile/plastic	
7	Parquet	Wood	Textile/Wood	
8	Wall to wall carpet	Laminate	Textile/plastic	
9	Wall to wall carpet	Laminate	Textile/plastic	
10	Wall to wall carpet	Laminate	Textile/plastic	

The cell resulted slightly attached to the surface when the air inside was sucked. Clean air was allowed to enter the cell and after an equilibration time (ca 30 min) without airflow passing through cell, air samples from the cell were taken. The air inside the cell, enriched in compounds emitted from the surface, was withdrawn into adsorbent cartridges for VOCs and aldehydes determination. Xylenes, toluene, ethylbenzene, formaldehyde, acetaldehyde, acetone, hexanal are frequently found among the compounds emitted from the surfaces analyzed.

In a first step, concentrations of chemical compounds coming from the specific surfaces (e.g. air inside the cell) were compared with the corresponding values measured in the indoor air of the room. An example of results thus obtained is shown in figure 5. Although an equal volume of air was sampled in all cases, the validity of direct comparison of values is limited and a direct comparison of the levels measured could only qualitatively and with limitations indicate surfaces with major contribution to chemical loading in the surrounding air.

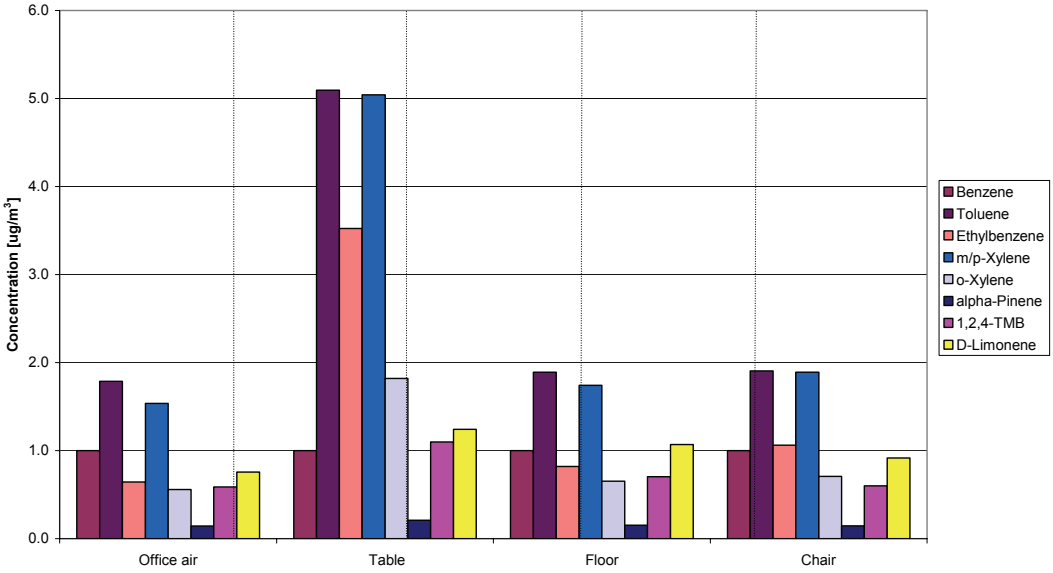

Figure 5: VOC concentrations measured in air and various surfaces inside an office

In a second step, profile diagrams were constructed to visually illustrate the chemical abundance in each category (office air, furniture, flooring material) of the monitored office.. A clear contribution of the surface emissions to the overall chemical loading of the indoor air has been observed in various environments. The qualitative and semi quantitative information obtained permitted, in some cases and for specific compounds, the identification of indoor surface sources that contributed to the loading of the indoor air of the room. In half of the offices studied, chairs were found to clearly contribute to the acetaldehyde loading in the air of the working environment. In figure 6 it can be observed that in office 5 the chair contributes more than the table and the linoleum floor to the overall acetaldehyde loading of the office air, whereas table and chair contribute equally to the hexanal loading. In other offices monitored, the flooring surface was the main source of hexanal.

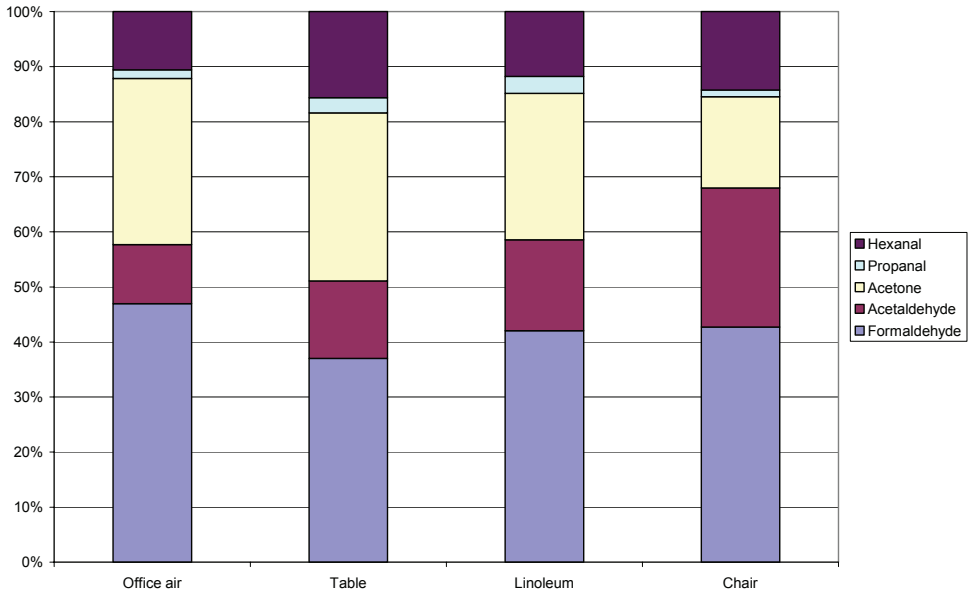

Figure 6: Profile diagrams of aldehydes measured in an office

Although the qualitative information obtained can be useful, as evidenced by the profile diagrams above, on site emission measurements have shown that the majority of the surfaces

analyzed emitted common compounds. Thus, attribution of a chemical marker to specific sources was rendered very difficult. Better understanding of indoor source apportionment would benefit from further experimental studies concerning the measurement of on-site specific emission rates and the comparison with laboratory chamber tests of the same inner surfaces or products.

Health effects of indoor air pollution

Indoor air quality (IAQ) has been a major concern for over three decades, when the expression "sick building syndrome" (SBS) was introduced to indicate a multiform set of medically unexplained illness symptoms (among others, headache, respiratory and ocular mucosa irritation, dizziness and nausea, fatigue, sensitivity to odours, irritable bowel syndrome), directly associated to household conditions and characteristics (Norbäck et al., 1991; US EPA, 1995; Wang et al., 2007). Apart from this "new" syndrome, other pathological conditions, such as allergies, asthma, skin and respiratory disorders and even cancer, have been reported as significantly increased and their increment attributed, at least in part, to poor indoor air quality. A recent review concluded that there is strong evidence for indoor air pollution as a cause of pneumonia and other acute lower respiratory infections (ALRI) among children under five years of age, and chronic obstructive pulmonary disease (COPD) and lung cancer (in relation to coal use) among adults. Immunological, neurobehavioral and toxic to reproduction effects have also been described. People do spend most part of their life in confined places: houses, offices, cars, and entertainment facilities. Some groups of people are more susceptible to developing these ill-health problems. These are generally children and the elderly, but also people affected by chronic and degenerative diseases, the carriers of particular polymorphisms, heavy smokers.

Poor indoor air quality has also been associated with low productivity and insufficient learning capacity, measured both qualitatively and semi-quantitatively (Allen et al., 2003; Fanger, 2006; Niemela et al., 2006) and in general with a low quality of life. Therefore, indoor air pollution currently represents a major health, social and economic issue. The WHO has recently published a comprehensive review of the national burden of disease attributed to indoor air pollution for non-industrialized countries (WHO, 2007). Indoor pollution in the non-industrialized world is mainly caused by use of solid fuel, including biomass and coal, for cooking and heating.

Chronic health effects

Asthma and allergic airway disease

The incidence of asthma and allergies has dramatically increased in the last 40 years. Asthma represents a disabling and often serious respiratory condition, mostly affecting children. It is a polygenic and multi-factorial disease, the molecular basis of which is currently under study by many scientific and clinical groups (Leikauf, 2002). A huge number of chemicals may trigger asthma. Firstly, volatile organic compounds (VOCs), a wide class of chemicals extensively used in industry and present as solvents, plasticizers, propellants in most household products, cosmetics, detergents, disinfectants. Furthermore, inorganic compounds and metals may play an important role. One of the earliest studies, carried out in the United States in the late '80s, reported an association between the concentrations of VOCs and chronic respiratory symptoms in children living in a large chemical manufacturing area (Ware et al., 1993). More recently, a case-control study carried out by Rumchev et al. (2007)

revealed that children of 6 months-3 years of age, exposed to total VOCs > 60 μg/m^3, were four times more likely to have asthma than those not exposed to such levels. Furthermore, children exposed to benzene > 20 μg /m^3 were eight times more likely to have asthma. Other VOCs that showed a significant independent effect on asthma were toluene, dichlorobenzene, and ethylbenzene (Rumchev et al., 2004**)**. A number of studies have reported a relation between formaldehyde concentration and respiratory symptoms and atopy (Maroni et al., 1995; Norback et al., 1995). A recent U.S. study compared the blood levels of 11 VOCs and pulmonary function measures in 953 adults (Elliott et al., 2006**)**. After adjusting for smoking, exposure to 1,2-dichlorobenzene was significantly associated with reduced pulmonary function in a study with asthmatic children. Exposure to VOCs was assessed by measuring the urinary concentration of hippuric acid, a biomarker of exposure to toluene, and muconic acid, a minor metabolite of benzene. The case group had significantly higher concentrations of muconic acid than did the control group, suggesting that VOCs have some role in asthma (Kim et al., 2005).

In occupational settings, polyisocyanates, especially toluene diisocyanate, have been historically associated with increased incidence of asthma (Bernstein et al., 1993). Current occupational standards assume that a threshold dose can be established at which no additional cases of asthma will develop. However, initiation of occupational asthma has been noted among workers wearing respiratory protective equipment and when exposures met existing TLVs. Besides, the existence of a threshold level for asthma development is still a matter of debate, and this has striking implications in the risk assessment procedures.

Irritant substances can also trigger asthma (possibly among susceptible individuals) by a non immuno-specific process. These disorders were termed “reactive airways dysfunction syndrome” (RADS) (Brooks et al., 1985). One attribute of RADS that differs from typical occupational asthma is the lack of a preceding latency period, because it is often initiated by a single exposure.

Microbial agents may play a role in the development of asthma and allergic airway diseases. Many fungi contain allergens, and sensitization is possible by indoor exposure to fungi due to dampness and mould growth. Additionally, virus infections may be transmitted by indoor air. In the first years of life, virus infections are common causes of wheezing. Some viruses are associated with an increase in asthma and allergy incidence (Schaub et al., 2006).

Cancer

The significant increase of cancer incidence in the second half of the 20th century is well known. This increase may be, at least in part, attributed to the huge amount of chemical products for professional and domestic use available on the market. During their life cycle, these products may release carcinogenic substances, which, taken up by inhalation or other route, can activate a series of biological responses culminating in one or more forms of cancer. In fact, cancer is the result of a multi-step process with diverse stages (initiation, promotion and progression), each of which may be inducted by different agents via independent events. Several forms of cancer are involved and associated with exposure to environmental pollutants: lung cancer to PAHs, asbestos, MMMF, radon; mesothelioma specifically to asbestos; leukaemia to benzene, electromagnetic fields; nasopharyngeal and sinonasal carcinoma to formaldehyde, dust. In a recent epidemiological investigation into the incidence of different types of cancer in Indiana (USA), Boeglin et al (2006) reported a strong correlation between the ambient emissions of VOCs and the incidence of cancers of the brain, nervous system, endocrine system and skin. Boffetta (2006), in a review of epidemiological studies about the association between environmental pollution and cancer,

has come to interesting conclusions. There is an increased risk of mesothelioma among individuals experiencing residential exposure to asbestos, while results for lung cancer are less consistent. Moreover, a causal association has been established between second-hand tobacco smoking and lung cancer, which may be responsible for 1.6% of lung cancers. Radon is another carcinogen present in indoor air, which may be responsible for 4.5% of lung cancers.

Assessing substances for carcinogenicity is an articulated process, based on evidence from essentially two sources: epidemiological studies and long-term animal tests. Other sources of information, such as structure-activity relations and short-term tests, can contribute to the overall evaluation. Therefore, a weight-of-evidence approach has been adopted at the international level (IARC, 1974-2007; US EPA, 2005). Generally, the categories of carcinogenicity individuate "proven, probably and suspect carcinogens" to humans. Many indoor and outdoor pollutants have been classified as proven or probable carcinogens. Among the most important carcinogens are asbestos, benzene, 1,3-butadiene, benzyl chloride, formaldehyde, vinyl chloride, trichloroethylene and perchloroethylene, many metal compounds and PAHs.

In addition, carcinogenic compounds can cause irritation and inflammation at sites of exposure and they are often antigenic (Ashby et al., 1993; Lee et al., 1993; Liekauf, 2002). Other compounds, directly reacting with proteins or DNA (genotoxic carcinogens), include polycyclic aromatics, aryl epoxides, bis-chloromethyl methyl ether, dimethyl carbonyl chloride, dimethyl sulfate, and propiolactone. Genotoxic chemicals are able to covalently link and cause damage to DNA; yet this does not inevitably lead to the creation of cancerous cells, because, as already said, cancer is a multi-stage process.

Inorganic and organic compounds that dominate the composition of fine particulate matter found indoors may cause genotoxic health effects, although the extent of genotoxicity is not well known (Krewski et al., 2007).

Respiratory and cardiovascular disease

Particles (PM10, PM2.5, fine particles, ultrafine particles) in ambient air have been associated with adverse health effects, including respiratory and cardiovascular effects (WHO, 2003; WHO, 2005). Particles from outdoor air may contribute to particle load in indoor air, but there are also indoor sources such as combustion (Lam et al., 2006), cooking, and particles may be formed by reactions between ozone and some VOCs (Wainman et al., 2000, Sarwar et al., 2004, Afsari et al., 2005). Man-made nanoparticles are increasingly used in consumer products but their impact as indoor air pollutants is not yet known and finally assessed. Studies in the USA show that PM2.5 is the most relevant metric for particulate matter. Long-term exposure to the coarse particle fraction, PM10-2.5, has not been clearly shown to be related to increased risks of mortality. In Europe, the principal available PM metric is black smoke. Relationships between adult mortality and long-term exposure to black smoke have also been found. There is also evidence linking long-term exposure to PM with prevalence and incidence of chronic bronchitis.

Acute health effects

Some indoor air substances have strongly acute health effects. Acute intoxication is generally under-evaluated, because it is not considered health-threatening, and it is underestimated, because victims are often not hospitalised (Mucci et al., 2006). However, such incidents are very frequent and cause of ailment and absenteeism. Carbon monoxide (CO), due to its

affinity for haemoglobin, represents, at high levels, one of the main causes of fatal poisoning (Lai et al., 2006, Watson et al., 2005). Furthermore, at lower concentrations, it may provoke headaches, dizziness, chest pains, nausea, confusion and disorientation. These symptoms of poisoning may be confused with the flu or food poisoning. However, foetuses, infants, elderly people, and people with anaemia or with a compromised cardio-respiratory system can be especially sensitive to CO exposures. Some indoor pesticides, such as carbamates and organophosphates, may affect the central nervous system, by reducing acetylcholinesterase activity (Lai et al., 2006, Watson et al., 2005). The US Six-Cities study has given evidence of relationships between particulate matter and acute episodes of bronchitis and other respiratory symptoms in children (Dockery et al, 1989; 1993). This is supported by evidence from other studies and age groups.

Benzene exposure as case study for human health effects

Benzene is a known carcinogenic compound for man principally inducing leukaemia and it is classified in Group 1 by the International Agency for Research on Cancer (IARC 1997). The World Health Organisation in its air quality guidelines stated that there is no absolute safety threshold for this compound and, based on a model of linear extrapolation derived from occupational exposure to benzene, they proposed a unit excess risk of $6x10^{-6}$. This means that an excess of six cases of leukaemia could be induced for a population of one million that has been exposed continuously, throughout its lifetime, to a benzene concentration of 1 $\mu g/m^3$. Benzene in the ambient air is regulated by Commission Directive 2000/69/EC that fixes a limit value of 5 $\mu g/m^3$ (annual mean) to be met by 2010. Due to the relevance of benzene for human health effects, detailed information on benzene concentrations (indoors in the working place, outdoors and personal) as measured in the field campaigns is presented (see figures 7-8 and tables 3-4).

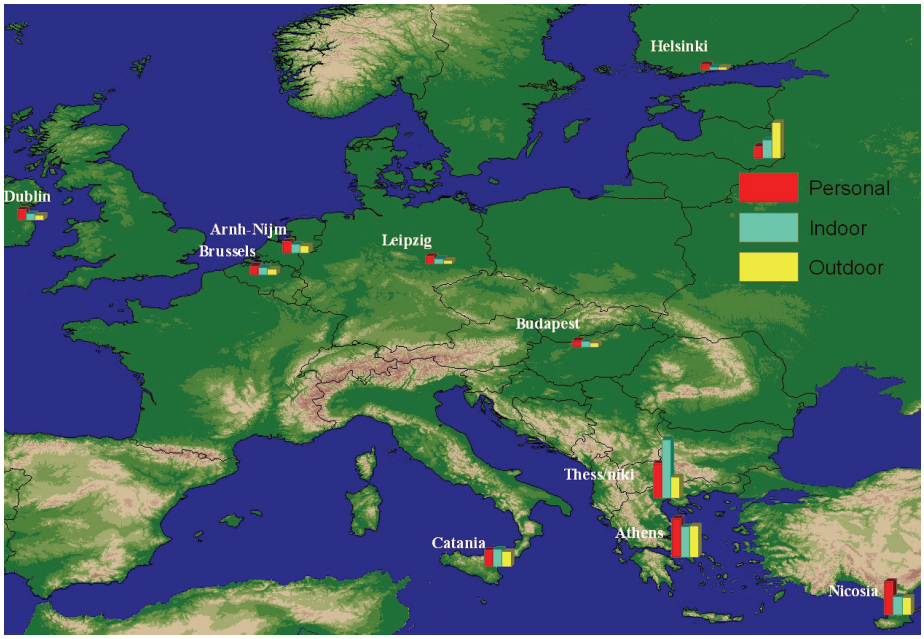

Figure 7: Benzene concentrations in public buildings across the EU

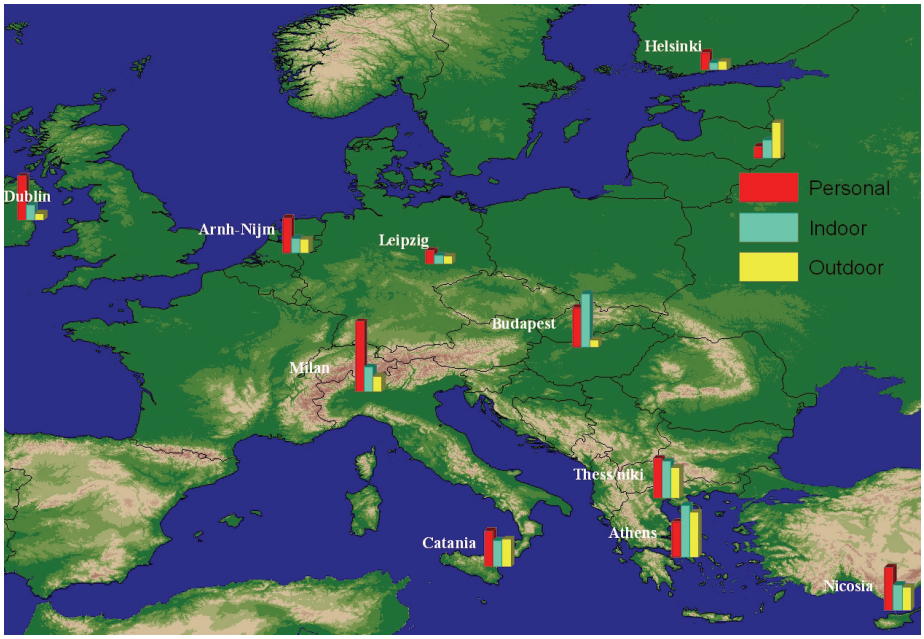

Figure 8: Benzene concentrations in schools/kindergartens across the EU

Benzene concentrations indoors in public buildings vary from 1 to 64 μg/m^3 with the highest level measured in Athens, Catania and Thessaloniki. Outdoor benzene concentrations ranged from 0.4 to 15 μg/m^3. In three cities located in Southern Europe (Athens, Catania, Thessaloniki) benzene (mean) concentrations indoors and outdoors exceed the limit value of 5 μg/m^3. Concentrations for benzene inside and outside schools and kindergartens are similar or slightly lower compared to the concentrations measured at public buildings. There are several explanations for this result. Most of the selected measurement sites are located outside heavy traffic zones. So outdoor air has relatively lower benzene levels. Strong benzene sources, e.g. smoking, do not generally affect indoor air quality in these areas. The results of the field campaigns indicate that about 25% of the outdoor concentrations, 30% of the indoor concentrations and 40.5% of the personal exposure concentrations exceeded the 5 μg/m^3 limit value (annual mean) established by the European legislation.

The distribution of personal inhalation exposure concentration of benzene based on the above data shows a median of 4.5 μg/m^3 (95th percentile: 14.4 μg/m^3). Following the linear (no effect threshold) exposure-response relationship for the unit risk of benzene-induced leukaemia given by the WHO[1], lifetime exposure to these benzene levels could induce a leukaemia risk of 27-86·10^{-6} (considering the median value or the 95th percentile value respectively) with an average at 33·10^{-6}. However, as exposure concentrations vary across Europe, climatic conditions and the different building materials and lifestyles at different locations across Europe are factors that might affect the levels of the indoor/outdoor pollutant concentrations and personal exposure levels. The reported data represent an indication of the actual situation regarding pollutant levels in the respective indoor and outdoor environments and on personal exposure of individuals selected randomly. Moreover, the results of gene expression experimentation are outlined in the section below, comprising an evaluation of the early biological response of human tissue to exposure to indoor and outdoor pollutants at the concentrations measured in the campaign sites.

[1] It should be noted that the WHO cancer risk estimate is estimated using a linear extrapolation to low chronic exposure levels from occupational exposure data measured in the so-called Goodyear Pliofilm cohort in the USA. Even though this estimate is validated at occupational exposure levels, it is not necessarily the most conservative cancer risk estimate for chronic exposure to low (environmental) levels of benzene.

Determination of early biological events from combined exposure to benzene and other VOCs

Environmental exposures occur to mixtures of chemicals rather than to individual agents, and such exposures occur in the context of numerous other potential risk-modifying physiological factors, such as genetic background, gender, stress, developmental period of exposure and diet. These co-occurring risk factors may enhance or mitigate the effects of chemical exposures, and do so in a dynamic fashion across the life span. As such, studies recognizing interactions and mixtures may have marked significance not only for evaluating the efficacy of risk assessment paradigms, but also for the determination of Community policies protecting public health. In this perspective, it is expected that the toxicogenomics approach would be the appropriate screening method for assessing biological effects of complex chemical mixtures, allowing us to review the whole spectrum of potential biological response rather than focusing on a pre-defined number of endpoints as in classical toxicological analysis.

Toxicogenomics consists of in vitro (on human cell lines) or in vivo (on laboratory animals) experiments whereby biological tissue is exposed to chemical toxicants and, via molecular biology techniques, the change in gene expression is measured with respect to a control sample of the same tissue. Whole genome toxicogenomics allows us to detect the cellular and biological processes modulated by exposure to the chemicals studied and, thus, infer potential health-related phenotypic responses and identify potential early biomarkers of health effects. The added value of the methodology rests on the possibility to explore all biological functions that can be modified by the toxicological insult. This capability expands the current capabilities of health risk assessment science, enlarging the scope of health effect research to endpoints beyond the ones addressed by conventional epidemiology and toxicology. It is also a valuable tool for enhancing our understanding of the biological mechanisms underlying pathologic responses to chemical exposure.

In reviewing the personal exposure measurements conducted across the European Union in the frame of this project, two main categories of indoor air mixtures of BTEX were identified, independent of the overall concentrations of VOCs:

(a) a mixture containing 20% benzene, 40% toluene, 10% ethylbenzene, 30% xylenes

(b) a mixture with 10% benzene, 60% toluene, 10% ethylbenzene, 20% xylenes

The chemical composition of all other indoor air mixtures measured at the European cities participating in the project fell within the range defined by mixtures (a) and (b) above. Therefore, these two mixtures were chosen for performing a comparative analysis of gene expression after in vitro exposure.

Cultures from a human lung cell line (A549) were exposed to different doses of the two BTEX mixtures for a period of 24 hours to derive a dose-response relationship with regard to gene expression modulation. Three doses (10 ng/l, 100 ng/l and 10 μg/l) were used for both mixtures (a) and (b). The results show that mixture (a) (the one richer with benzene) modulates more the overall genome than mixture (b) and that the number of genes the expression of which is modulated increases with rising exposure dose (see figure 9) even though the doses administered are below the no observed adverse effect level[2].

[2] The World Health Organisation has recently recommended a guideline for ambient toluene exposure of 260 $\mu g/m^3$ (68 ppb) as a weekly average concentration to protect against developmental neurotoxicity. The 8-hour time-weighted average permissible exposure limit of 100 ppm (375 $\mu g/m^3$ and 435 $\mu g/m^3$ for toluene and xylene isomers respectively) has been established in occupational settings.

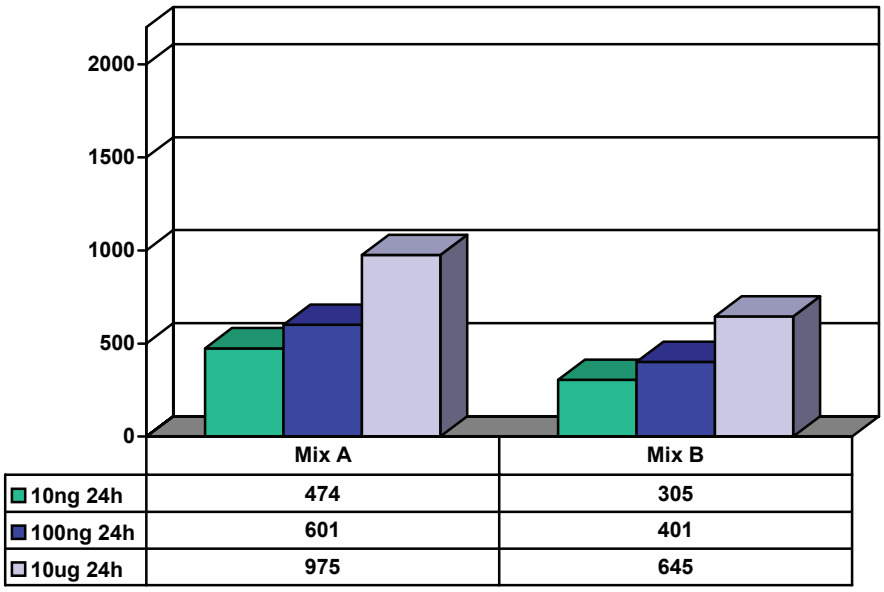

Figure 9: Dose-dependence of gene modulation given in terms of number of genes over- or under-expressed after 24 hours of exposure to the two BTEX mixtures considered in this study.

The presence of toluene in indoor air mixtures comprising other monoaromatic VOCs enhances non-carcinogenic responses like inflammation in *in vitro* experiments using human cell lines. Both time and dose of exposure influence the magnitude of the observed biological effect. **Early biological events** that may be precursors of later life pathologies **can be observed at doses lower than the currently observed no adverse effect level for non-cancer effects**. These results pave the way towards a better use of currently available human biomonitoring data. Specific biomarkers of, say, inflammation, or oxidative stress, can be readily identified and mechanistically linked to the onset or exacerbation of human pathologies associated to indoor air pollution (e.g. the link between inflammation and asthma exacerbation).

European Parliament workshop

In accordance to the technical annex of the project and its objectives, a workshop on indoor air in Europe presenting the results of the project and opening up a high-level discussion among international experts and stakeholders will be organised jointly by the JRC and DG SANCO under the auspices of the European Parliament at the Brussels seat of the Parliament on 31 January 2008.

Indoor air quality and health effects Commission Website

In addition, a dedicated website will be created by the JRC aimed at increasing public awareness of the health importance of air quality in indoor spaces. On the basis of the results of this project, the opinion of SCHER and contributions of the DG SANCO expert group on indoor air quality, this information hub could also give simple advice on how to improve indoor air quality at home with respect to different key pollutants. This might also become a tool for exchange of information on more technical issues between Member State authorities

and professionals, such as on best practices and on the dissemination of other important activities at the Community or national level.

Conclusions

Personal exposure concentrations to priority pollutants in this study were found in most cases to be higher than the respective indoor/outdoor concentrations. The concentrations of VOCs measured indoors, outdoors and on personal samplers depend on the relative contribution of indoor and outdoor source and time-activity patterns. Accurate exposure assessment that is critical to a credible and scientifically sound assessment of risk cannot be determined directly from measurements referring to fixed ambient background monitoring stations for these pollutants. Evaluation of possible long term health effects associated with the presence of pollutants indoors and outdoors should take into account personal exposure monitoring along with environmental activity patterns and personal behaviour. In reference to the ambient air limit value for benzene of 5 $\mu g/m^3$ (annual mean) established by the Commission to be met by 2010 (Directive 2000/69/EC), the measurements taken indicate that **about 25% of the outdoor concentrations, 30% of the indoor concentrations and 40 % of the personal exposure concentrations measured so far exceeded this limit value even though the average outdoor/indoor/personal exposure concentrations were below the limit**. On the contrary, biological contamination measurements identified no specific source of contamination or pathogens in any of the sites examined across the EU.

The potential adverse health effects currently associated with exposure to indoor air pollutants were reviewed and summarized in this report. Chronic health effects of indoor air pollution range from asthma and allergic airway disease to cancer, respiratory and cardiovascular disease. Acute health effects include insults to the central nervous system, dizziness, headache, chest pains, nausea, confusion and disorientation. Of particular interest is the potential interaction of different components of indoor air within the human body resulting in other additive effects. A toxicogenomics approach using whole-genome micro-arrays was used to identify early biological events leading to non-cancer health endpoints such as inflammation. Based on our results it may be assumed that **co-exposure to benzene and other VOCs found indoors such as toluene could modify the risk of leukemia** due to the metabolic inhibition of benzene to its toxic carcinogenic metabolites. It must be noted here that the results reported herein are only indicative and they are based on a restricted set of measurement data. They point out, however, the need for further and more comprehensive investigation of indoor air pollution and its potential health effects encompassing a statistically significant and representative set of measurements.

This study was a pilot project addressing human exposure to indoor air pollutants and their potential health effects. It provided information on levels of exposure and the associated health risk in cities across selected EU Member States. These findings highlight the need for further research to address the burden of indoor air pollution on public health in the European Union. The following are key research avenues that need to be explored in a Europe-wide programme:

- Develop a concept for a broader EU-wide monitoring study, focusing on free radicals and indoor air chemicals to identify chemical interactions resulting in the formation of toxic secondary products and on primary and secondary particles (coarse, fine and ultra-fine) in the indoor air.

- Make a systematic source apportionment of the pollutants in the indoor environment in quantitative terms. Identification of the main sources would help their mitigation. In this context, enhance our understanding of chemical emissions from consumer products and building materials.
- Study the mechanisms of chemical and biochemical interaction in the indoor air mixtures typically found in different geographical latitudes and develop the methodology to better inform the health risk assessment process as to the effect of such interactions on final health risk.
- Extend our knowledge on the adverse health effects of biological contaminants (microbes and bio-aerosols) present in indoor air, especially other than respiratory tract effects.

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Abstract

This report outlines the results of the 2-year pilot project on indoor air quality and potential health effects executed by the Joint Research Centre and funded by the European Parliament via the Directorate-General Health and Consumer Protection. It had four distinct objectives as follows:
1) to identify and quantify the main air pollutants present in public buildings, including indoor environments where children frequently stay, like schools and kindergartens,
2) to identify the main sources of these pollutants, applying source apportionment analyses,
3) to estimate people's exposure to these pollutants while working and/or living in these areas and combined with micro-environmental activity patterns during the day,
4) to evaluate possible health risks due to (chronic) exposure to air pollutants, in particular, for children.

The results indicate that indoor air pollution concentrations are consistently higher than the respective outdoor ones for the chemical families this study focused on. Differences attributable to variation in consumer behaviour, climate and type of building materials used, have been identified in the indoor:outdoor ratio of primary pollutants across Europe. These differences account for small variance in the corresponding health risk to the local population across the EU.

The mission of the JRC is to provide customer-driven scientific and technical support for the conception, development, implementation and monitoring of EU policies. As a service of the European Commission, the JRC functions as a reference centre of science and technology for the Union. Close to the policy-making process, it serves the common interest of the Member States, while being independent of special interests, whether private or national.

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