

HEALTH IMPACT ASSESSMENT OF AIR POLLUTION

ENHIS-1 PROJECT: WP5 HEALTH IMPACT ASSESSMENT

LOCAL CITY REPORT

Toulouse

Summary of main findings for Toulouse

In 2001 the PM_{10} annual mean (SD) was 22 (10) (25 (12) $\mu\text{g}/\text{m}^3$ when a correction is applied in order to compensate losses of volatile compounds due to the TEOM measurement method), above the 1999/30/EC Directive limit value for 2010 (20 $\mu\text{g}/\text{m}^3$), and below that established for 2005 (40 $\mu\text{g}/\text{m}^3$). For the summer period of the same year, the mean (SD), P5 (5th percentile) and P95 of the maximum daily 8-hour moving average concentration of ozone (O_3) were 91 (23), 63 and 132 $\mu\text{g}/\text{m}^3$.

Regarding children, infant mortality in Europe is quite low and consequently, the expected attributable number of deaths related to air pollution is also very low. All other things being equal, the reduction of the annual average levels of PM_{10} to 20 $\mu\text{g}/\text{m}^3$ would prevent a very low but significant number of total postneonatal deaths (0.2 death). Reducing PM_{10} daily mean values to 20 $\mu\text{g}/\text{m}^3$ would prevent 5 hospital respiratory admissions of children under 15 years old.

As far as short-term effects of O_3 in summer are concerned, all other things being equal, each reduction by 10 $\mu\text{g}/\text{m}^3$ of the daily maximum 8-hour moving average concentrations would delay 7 deaths per year in the general population in the study area, 3 from cardiovascular diseases, and 2 from respiratory causes. In terms of hospital admissions, this would represent 1 respiratory admission in the adult population and 5 in the population over 64 years.

Summary of HIA of outdoor air pollution in Toulouse in ENHIS-1

Health outcome	Population	Pollutant	Period	Mean type	RR (for 10 µg.m ³ increase)	References	Number of attributable cases by scenario ¹		
Mortality							Ozone: Reduction by 10 µg.m ³	PM10: Reduction by 5 µg/m ³	
Total mortality excluding external causes (ICD9 < 800 - ICD10 A00-R99)	All ages	O ₃ 8h max	Summer ²	Daily	1.0031 (1.0017-1.0052)	Gryparis et al 2004	6.64		
Cardiovascular mortality (ICD9 390-459 - ICD10 I00-J99)					1.0046 (1.0022-0.0073)		3.24		
Respiratory mortality (ICD9 460-519 - ICD10 J00-J99)					1.0113 (1.0074-1.0151)		1.89		
Total postneonatal mortality	1 month-1 year	Corrected PM ₁₀ ³	Year	Annual	1.048 (1.022-1.075)	Lacasaña et al 2005		0.21	
Postneonatal respiratory mortality (ICD9 460-519 - ICD10 J00-J99)					1.216 (1.102-1.342)			0.09	
Postneonatal Sudden Infant Death Syndrom Mortality (ICD9 798.0 - ICD10 R95)					1.12 (1.07-1.17)	Woodruff 1997		0.11	
Morbidity									
Emergency room visits for asthma (ICD-9 codes 493, ICD-10 codes J45, J46)	< 18 years	O ₃ 1h max	Year	Daily	1.0115 (1.0067-1.0163)	CARB 2004	not available		
Cough	< 18 years	Measured PM ₁₀			1.0407 (1.0202-1.0511)	Ward and Ayres 2004			not available
Lower respiratory symptoms LRS	< 18 years	Measured PM ₁₀			1.0407 (1.0202 -1.617)	Ward and Ayres 2004			not available
Hospital respiratory admissions (ICD9 460-519 - ICD10 J00-J99)	< 15 years	Measured PM ₁₀			1.010 (0.998-1.021)	Anderson et al 2004			
Hospital respiratory admissions (ICD9 460-519 - ICD10 J00-J99)	15 - 64 years	O ₃ 8h max	Summer	1.001 (0.991-1.012)	1.34				
Hospital respiratory admissions (ICD9 460-519 - ICD10 J00-J99)	> 64 years			1.005 (0.998-1.012)	5.03				

¹ For ozone: absolute reduction by 10 µg/m³. For PM₁₀: absolute reduction by 5 µg/m³.

² Definition of summer period : 01 April – 30 September

³ PM₁₀ reference papers for HIA on postneonatal mortality use gravimetric methods to measure PM₁₀. If the local air quality network uses automatic methods (TEOM or other) a correction factor is required to compensate for loss of volatile compounds: if available, a local correction factor recommended by the air quality network or, by default, the European factor 1.3.

Introduction *(please describe the following issues)*

- Since the Apehis 3 study, the study area has been reevaluated and includes now the city of Toulouse and 50 surrounding municipalities. The population living in this area in 1999 is estimated to 670,713 inhabitants. The area is characterized by a low population density compared to other urban centres, and by heavy traffic between the suburbs and the town centre.
- In the study area, as at the national level, sulfur dioxide air pollution has been decreasing for several years now. A decrease has also been observed for carbon monoxide and at a lower level for benzene. These decreases are mainly attributable to technical improvement of vehicles. Particular air pollution levels keep steady since 1999 (beginning of measurements). Nitrogen dioxide air pollution levels keep steady too for about 5 years. For ozone levels, no marked trends were observed, ozone levels being very dependent on summer climatic conditions.
- In Toulouse, as in other French urban areas of more than 250 000 inhabitants, the main political framework for public air quality management is the plan for air protection (PPA) which is in progress.
- In this study area, the life expectancy is higher than the French one. The main cause of deaths in the general population is represented by cardiovascular diseases. The second one is cancers. Both death rates are lower in the study area than the national French one. Regarding infant mortality, the main causes of death are conditions arising during the perinatal period and congenital anomalies.
- Air pollution Health impact assessments (HIA) have been previously carried out in the study area especially during the phases 2 and 3 of Apehis. The most recent analysis (Apehis 3) estimated that reduction of the long-term PM_{2.5} pollution to the levels of $15 \mu\text{g}/\text{m}^3$ would reduce mortality in Toulouse by 60 deaths in one year, which would save 28 years of expected life for starting year of simulation. If the daily means of PM₁₀ would be kept under $20 \mu\text{g}/\text{m}^3$, 15 deaths and 39 hospital respiratory admissions could have been avoided in the year 2000.
- This report presents the results obtained for the Toulouse study area. After a brief description of a air pollution sources, exposure and health data, the results of the HIAs conducted on postneonatal mortality, cardiovascular and respiratory mortality, and respiratory hospital admissions in relation with ozone (short-term) and PM₁₀ (short- and long-term) are presented.
- This work has been carried out within the framework of work package WP5 on health impact assessment of ENHIS-1 project (www.enhis.net).

Sources of air pollution

Regarding sulfur dioxide, about 43% of the emissions are represented by tertiary and residential sectors, 38% by industry/ garbage incineration, and 19% by traffic. Conversely, traffic is the main source of PM₁₀ emissions and represent about 70% of nitrogen oxides emissions. In the study area, the levels of nitrogen oxides measured near road traffic areas are higher than the national air quality guideline ($40 \mu\text{g}/\text{m}^3$).

Ozone is a secondary air pollutant. Its formation is the result of complex processes. It is hence not possible to attribute ozone to sources of air pollution.

Exposure data

- Data concerning air pollution were obtained from Oramip, the local air pollution monitoring network. Within the study area in 2001, 2 background monitoring stations (TEOM) measured the levels of PM₁₀, and 4 background monitoring stations measured the levels of ozone. One of these stations, measuring the levels of PM₁₀ and O₃, was destroyed on 21 September 2001 during the AZF factory explosion.
- For HIA purpose for chronic exposure, ENHIS recommended to correct TEOM PM₁₀ in order to compensate losses of volatile compounds, because the corresponding RRs were obtained using gravimetric PM₁₀ as a measure of exposure. In Toulouse, as part of the French national pilot program for PM surveillance, specific polynomial regression has been used for each city PM₁₀ correction. The coefficients of these regressions were derived from parallel PM₁₀ measurements within each city¹.
- How indicators have been calculated:
 - PM₁₀: daily exposure indicator has been calculated as the arithmetic mean of the daily concentrations of the stations.
 - Ozone: The daily maximum 1-hour concentration and the daily maximum 8-hour moving averages of each day have been calculated for the summer period (1st April to 30th September).
- AP data description: The annual mean level (SD) of TEOM PM₁₀ in Toulouse was 22 (10) µg/m³, and P5 and P95 of the daily mean values were, respectively, 11 µg/m³ and 36 µg/m³. The annual mean level (SD) of corrected PM₁₀ in Toulouse was 25 (12) µg/m³, and P5 and P95 of the daily mean values were, respectively, 11 µg/m³ and 41 µg/m³. The mean (SD), P5 and P95 of the daily maximum 8-hour moving average concentrations of O₃ were, respectively, 91 (23), 63 and 132 µg/m³, and those of the daily maximum 1-hour concentrations (entire year) 79 (31), 33 and 139 µg/m³ (Table 1 and figures 1-3)
- Both TEOM and corrected PM₁₀ levels were lower than the limit value for 2005 (40µg/m³). However, both TEOM and corrected PM₁₀ levels were slightly higher than the limit value for 2010 (20µg/m³). Concerning ozone, the daily maximum 8-hour moving average has been higher than 120µg/m³ during 27 days, whereas the target value for 2010 is 120 µg/m³ not to be exceeded on more than 25 days per calendar year averaged over three years.
- Daily ozone levels (both 1-hour and 8-hour maximum) show a large variability. There are a few days during the summer when ozone levels are very high. Among these days, one corresponds to situation where the information threshold (180 µg/m³ for 1-hour average) was overshoot. Daily corrected PM₁₀ levels show a smaller variability. During more than 74% of the days, the daily mean value is between 10 and 30 µg/m³. During slightly less than 38% of the days, the daily mean value is lower than 20µg/m³.

¹ Jean-Luc HOUDRET, François MATHE. Programme pilote national de surveillance des particules PM₁₀ et PM_{2.5}. Ecole des mines de Douai, Département Chimie et environnement, Etude n°10. 2003

Table 1. Descriptive statistics for ozone and PM₁₀ levels in Toulouse area, 2001

	O3 8h - summer	O3 1h max - year	Corrected PM10 - year
Number	183	365	364
Minimum	36	12	5
Percentile 5	63	33	11
Percentile 25	74	58	17
Median	86	77	23
Percentile 75	104	96	29
Percentile 95	132	139	41
Percentile 98	143	153	62
Maximum	165	194	111
Daily mean	91	79	25
standard error	23	31	12
% missing values	0,00%	0,00%	0,27%

Fig 1. Distribution of O3 8h max in Toulouse area - summer 2001

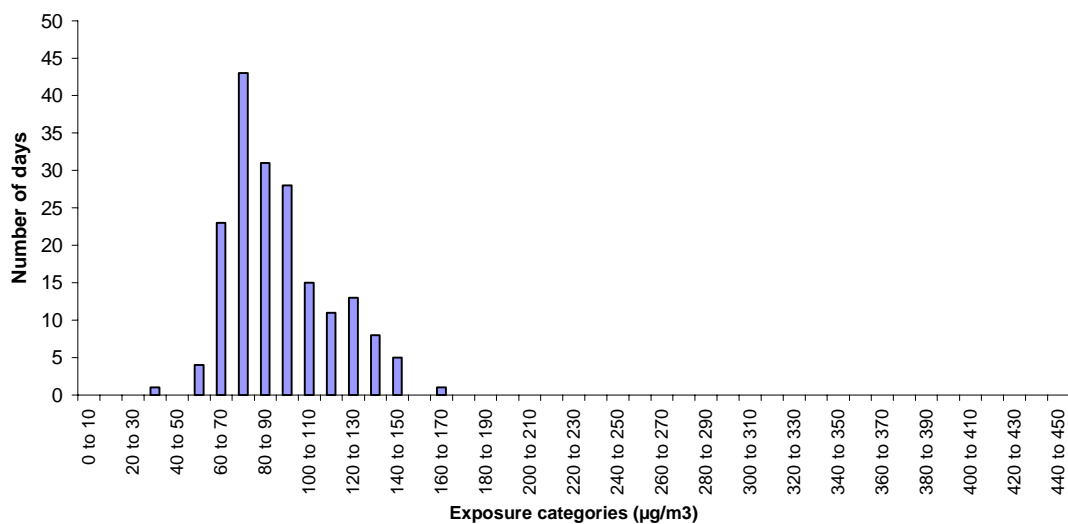


Fig 2. Distribution of O3 1h max in Toulouse area - year 2001

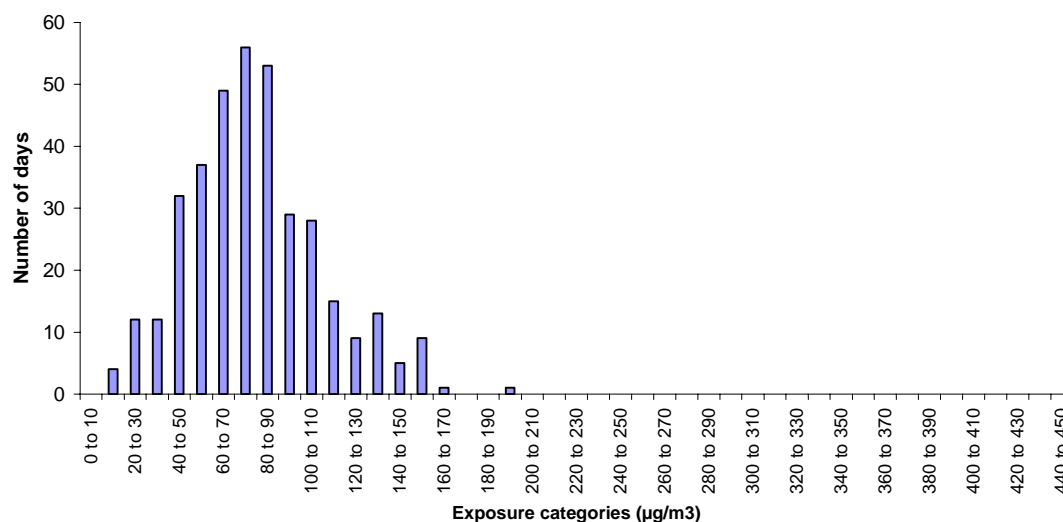
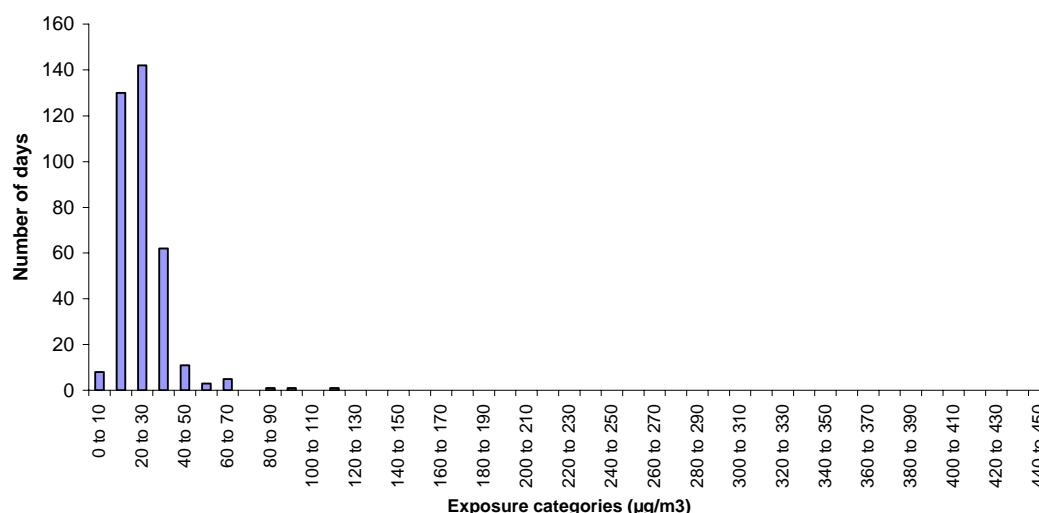


Fig 3. Distribution of corrected PM 10 in Toulouse area - year 2001



Health data

- Mortality data were obtained from the Institut National de la Santé et de la Recherche Médicale (CepiDC). The CepiDC is a register. There are no missing data, and a quality control program is applied. Death causes for year 2001 were coded according to ICD-10. Most of the coding (about 80%) was automated.
- Hospital admissions data concerned public and private hospitals and were extracted from the Information Systems Medicalisation Program (PMSI) by the French Institute of Public Health (InVS). These data are total hospital admissions data, and hence contain both emergency and scheduled hospital admissions. Hospital admissions causes for year 2001 were coded according to ICD-10.
- Data concerning specifically emergency hospital admissions, emergency room visit for asthma, cough or lower respiratory syndromes were not available for Toulouse study area, and hence no HIA was conducted for these indicators.

The total number of postneonatal deaths in 2001 was 9 (annual rate 123.1 per 100,000) , among which 1 was due to respiratory causes and 2 to sudden infant death syndrome.

The number of deaths in the general population (excluding external causes) was 4,291 (annual rate 639.77 per 100,000), among which 1386 (annual rate 206.65 per 100,000) were due to cardiovascular causes, and 237 (annual rate 35.36 per 100,000) were due to respiratory causes.

The annual rate of respiratory admissions was high particularly in elderly people : 2,277.71 per 100,000 (2,088 hospital admissions during 2001). It was lower for children under 15 : 939.93 per 100,000 (1,003 hospital admissions during 2001); then, for people age between 15 and 64 : 569.51 per 100,000 (2,690 hospital admissions during 2001).

Table 2. Descriptive statistics for health outcomes in Toulouse area, 2001

Health outcome	ICD9	ICD10	Annual deaths	Annual rate (per 100 000)	Daily mean (SD)	Daily rate (per 100 000)	Annual incidence rate (per 100 000)
POSTNEONATAL MORTALITY							
Total			9	123.10			
Respiratory ICD9 460-519 ICD10 J00-J99	460-519	J00-J99	1	13.59			
Sudden infant death syndrome ICD9 798.0 – ICD10 R95	798.0	R95	2	27.17			
GENERAL POPULATION MORTALITY							
Total mortality all causes ICD9 <800 ICD10 A00-R99	<800	A00-R99			12 (3.6)	1.75	
Cardiovascular mortality ICD9 390-459 ICD10 I00-I99	390-459	I00-I99			4 (2.0)	0.57	
Respiratory mortality ICD9 460-519 ICD10 J00-J99	460-519	J00-J99			1 (0.8)	0.10	
MORBIDITY							
Cough					not available		
Lower respiratory symptoms LRS					not available		
Emergency room visits for asthma - Age < 18 years ICD9 493, ICD10 J45 J46	493	J45-J46			not available	not available	
Hospital respiratory admissions - Age < 15 years ICD9 460-519 ICD10 J00-J99	460-519	J00-J99					939.93
Hospital respiratory admissions - Age 15 -64 years	460-519	J00-J99					569.51
Hospital respiratory admissions - Age > 64 years	460-519	J00-J99					2277.76

Health Impact Assessment

Methodology

Health impact of air pollution (AP) has been calculated as the annual number of health events attributable to AP in the target population. A causal relationship between AP and the effects is assumed, and therefore HIA can only be performed for those outcomes with sufficient evidence of causality. Once the effects with sufficient evidence of causal relationship with AP have been determined, the next step is to find the best exposure-response functions (ERFs) for each of the

selected outcomes. Table 3 shows the result of a systematic review on these issues carried out by the Bilbao Apehis team² for WP5 of ENHIS-1. This table summarizes the health outcomes and ERFs deemed suitable for HIA according to the criteria established by WP5 with the advice of the air pollution experts of WP5³.

Table 3. Health outcomes and Exposure-response functions (ERFs) selected for health impact assessment

	OUTCOME	POLLUTANT	ERFs	ORIGINAL SOURCE
CHILDREN - PARTICLES				
	Total postneonatal mortality (1 month-1 year)	PM ₁₀ Annual Mean	RR=1.048 (1.022-1.075) ↑10µg/m ³	Lacasaña et al 2005
	Postneonatal respiratory mortality ICD9 460-519 ICD10 J00-J99	PM ₁₀ Annual Mean	RR=1.216 (1.102-1.342) ↑10µg/m ³	Lacasaña et al 2005
	Postneonatal Sudden Infant Death Syndrome (SIDS) mortality (normal birth weight ≥2500g) ICD9 798.0 –ICD10 R95	PM ₁₀ Annual Mean	Adjusted Odds Ratio AOR=1.12 (1.07-1.17) ↑10µg/m ³	Woodruff et al. 1997
	Cough	PM ₁₀ Daily Mean	OR=1.041 (1.020-1.062) ↑10µg/m ³	Ward & Ayres 2004
	Lower respiratory symptoms LRS	PM ₁₀ Daily Mean	OR=1.041 (1.020-1.051) ↑10µg/m ³	Ward & Ayres 2004
CHILDREN – OZONE				
	Emergency room visits for asthma <18 Y ICD9 493, ICD10 J45 J46	Ozone Maximum 1 h	RR=1.0116 (1.0067-1.0165) ↑10µg/m ³	CARB 2004
ADULTS/GENERAL POPULATION				
	Total mortality all causes ICD9 <800 ICD10 A00-R99	Ozone Maximum 8 h Summer	RR= 1.0031 (1.0017-1.0052) ↑10µg/m ³	Gryparis et al 2004 (APHEA 2)
	Respiratory mortality ICD9 460-519 ICD10 J00-J99	Ozone Maximum 8 h Summer	RR= 1.0113 (1.0074-1.0151) ↑10µg/m ³	Gryparis et al 2004 (APHEA 2)
	Cardiovascular mortality ICD9 390-459 ICD10 I00-I99	Ozone Maximum 8 h Summer	RR= 1.0046 (1.0022-1.0073) ↑10µg/m ³	Gryparis et al 2004 (APHEA 2)

To be coherent with mortality findings, it was decided, with the experts' advice, to include RRs of hospital admissions in the health impact assessment calculations, even if they were not statistically significant. More concretely, it was decided that if there was not any new RR published by the time of making the calculations, the RRs for respiratory hospital admissions from Anderson's meta-analysis could be used, although they were not statistically significant (see Table 2). The rationale for that is that if there is sufficient evidence to accept a causal relationship between air pollution and respiratory mortality -both in children-PM and adults-O₃- we should easily accept that there will also be an impact on hospital admissions.

² Cambra K, Alonso E, Cirarda FB, Martínez-Rueda T. Bilbao APHEIS group. Selection of outcomes and exposure response functions for health impact assessment of particles and ozone. Review of the evidence. ENHIS project. WORK PACKAGE 5. Bilbao, February 2005. Http:

³ Ferran Ballester: Valencian School of Health Studies, Valencia, Spain; Sylvie Cassadou: National Institute of Public Health Surveillance, InVS, Toulouse, France; Fintan Hurley: Institute of Occupational Medicine, Edinburgh, Scotland, UK; Nino Künzli: University of Southern California, Division of Occupational and Environmental Health, Los Angeles, CA, USA; Odile Meckel: Institute of Public Health NRW (LOEGD), Bielfeld, Germany; Hans-Guido Mücke: WHO Collaborating Center (Air)-Federal Environmental Agency, Berlin, Germany; Nikolaos Stilianakis: Institute for Environment and Sustainability, European Commission – JRC, Ispra, Italy.

Table 4. Complementary Exposure-response functions (ERFs) for health impact assesment on respiratory hospital admissions for children (particles) and adults (ozone)

	OUTCOME	POLLUTANT	RR	SOURCE
CHILDREN - PARTICLES				
	Respiratory hospital admissions 0-14 Y ICD9 460-519 ICD10 J00-J99	PM ₁₀ Daily Mean	RR= 1.010 (0.998-1.021) ↑ 10µg/m ³	Anderson 2004
ADULTS/GENERAL POPULATION				
	Hospital respiratory admissions 15-64 Y ICD9 460-519 ICD10 J00-J99	Ozone Maximum 8 h	RR=1.001 (0.991-1.012) ↑ 10µg/m ³	Anderson et al 2004
	Hospital respiratory admissions >64 Y ICD9 460-519 ICD10 J00-J99	Ozone Maximum 8 h	RR=1.005 (0.998-1.012) ↑ 10µg/m ³	Anderson et al 2004

Finally, HIA needs defining the evaluation scenarios, i.e. the hypothetical scenario with which we want to compare the current air pollution situation. We calculate the impact on health of the (current) air pollution levels in the city that are above the pollution level of the evaluation scenario. In other words, the attributable number of health events (deaths, hospital admissions...) calculated for each scenario represents the number of events that would be prevented if, all other things being equal, air pollution levels were reduced to the evaluation scenario level. These evaluation scenarios are based on the objectives and limits established in 1999/30/CE, and 2002/3/CE Directives.

HIA scenarios

1 - HIA scenarios for PM₁₀

1.1.- Scenarios for HIA on **short-term** effects of PM₁₀ and **cough, lower respiratory symptoms** in people under 18 year (<18), and **hospital respiratory admissions** in people under 15 year (< 15)

1.1.1 Reduction of PM₁₀ levels to a 24-hour value of **50 µg/m³** in all days exceeding this value (Limit of 1999/30/CE Directive)

1.1.2. Reduction of PM₁₀ levels to a 24-hour value of **20 µg/m³** in all days exceeding this value

1.1.3 Reduction **by 5 µg/m³** of all the 24-hour values

1.2.- Scenarios for HIA on **long-term** effects of PM₁₀ and **postneonatal mortality** (total, respiratory and sudden infant death syndrome-SIDS)

1.2.1 Reduction of the annual mean value of PM₁₀ to a level of **40 µg/m³** (Limit of 1999/30/CE Directive for 2005)

1.2.2 Reduction of the annual mean value of PM₁₀ to a level of **20 µg/m³** (Limit of 1999/30/CE Directive for 2010)

1.2.3 Reduction **by 5 µg/m³** of the annual mean value of PM₁₀

2.- HIA scenarios on short-term effects of Ozone

1.2.1 Daily maximum 1-hour concentration and **emergency room visits for asthma** in people under 18 year (< 18)

1.2.1.1 Reduction of O₃ daily maximum 1-hour concentrations to a level of **180 µg/m³** in all days exceeding this value (Information threshold of 2002/3/CE Directive)

1.2.1.2 Reduction **by 10 µg/m³** of the daily maximum 1-hour concentrations

1.2.2 Daily maximum 8-hour moving average concentration and **mortality** in general population

1.2.2.1 Reduction of O₃ daily maximum 8-hour moving average concentrations to **120 µg/m³** in all days exceeding this value (Limit for health protection of 2002/3/CE Directive)

1.2.2.2 Reduction **by 10 µg/m³** in the daily maximum 8-hour moving average concentrations.

Findings

For long-term HIA, corrected PM10 levels were used as a measure of exposure. The following results were found:

- The annual number of postneonatal deaths attributable to PM₁₀ levels higher than 20 µg/m³ was 0.19 (95%CI: 0.09 – 0.30), which is equivalent to an annual rate of 2.58 deaths per 100 000 (95%CI: 1.22 – 4.08).
- The annual number of postneonatal respiratory deaths attributable to PM₁₀ levels higher than 20 µg/m³ was 0.09 (95%CI: 0.04 – 0.13), which is equivalent to an annual rate of 1.22 deaths per 100,000 (95%CI: 0.54 – 1.77).
- The annual number of postneonatal SIDS deaths attributable to PM₁₀ levels higher than 20 µg/m³ was 0.10 (95%CI: 0.06 – 0.14), which is equivalent to an annual rate of 1.36 deaths per 100,000 (95%CI: 0.82 – 1.90).

Short-term HIA of PM10 on hospital respiratory admissions were calculated using TEOM PM10, as the corresponding RRs were obtained using TEOM measured values as an assessment of PM10 exposure. The annual number of hospital admissions for respiratory causes of children aged less than 15 attributable to PM₁₀ levels higher than 20 µg/m³ was 4.51 (95%CI: -0.90-9.52), which is equivalent to an annual rate of 4.23 deaths per 100,000 (95%CI: -0.84-8.92).

Table 5. Potential benefits of reducing PM₁₀ levels. Absolute numbers and rates (per 100 000 children) (95% confidence limits) attributable to the health effects of PM₁₀.

	PM10 reduction	Number of attributable cases per year	Annual rates (per 100.000)
POSTNEONATAL MORTALITY	Corrected* annual mean levels		
Total	by 5 µg/m ³	0.21 (0.10 - 0.32)	2.85 (1.36 - 4.35)
	to 20 µg/m ³	0.19 (0.09 - 0.30)	2.58 (1.22 - 4.08)
	to 40 µg/m ³	NA	NA
Respiratory	by 5 µg/m ³	0.09 (0.05 - 0.14)	1.22 (0.68 - 1.90)
	to 20 µg/m ³	0.09 (0.04 - 0.13)	1.22 (0.54 - 1.77)
	to 40 µg/m ³	NA	NA
SIDS	by 5 µg/m ³	0.11 (0.06 - 0.15)	1.49 (0.82 - 2.04)
	to 20 µg/m ³	0.10 (0.06 - 0.14)	1.36 (0.82 - 1.90)
	to 40 µg/m ³	NA	NA

	PM10 reduction	Number of attributable cases per year	Annual rates (per 100.000)
MORBIDITY	Measured daily levels		
Cough <18 y	by 5 µg/m ³ to 20 µg/m ³ to 50 µg/m ³	Not available Not available Not available	
LRS <18 y	by 5 µg/m ³ to 20 µg/m ³ to 50 µg/m ³	Not available Not available Not available	
Hospital respiratory admissions <15 y	by 5 µg/m ³ to 20 µg/m ³ to 50 µg/m ³	4.91 (-0.98 - 10.28) 4.51 (-0.90 - 9.52) 0.37 (-0.07 - 0.79)	4.60 (-0.92 - 9.63) 4.23 (-0.84 - 8.92) 0.35 (-0.07 - 0.74)

NA: Not applicable if air pollution levels are lower than the scenario level

* PM₁₀ reference papers for HIA on postneonatal mortality use gravimetric methods to measure PM₁₀. In France, the automatic methods (TEOM) was used, and a correction factor is required to compensate for loss of volatile compounds: a local polynomial correction factor elaborated by the Ecole des Mines de Douai was used for each French city, see p. 5

Regarding short-term effects of O₃, each reduction by 10 µg/m³ of daily maximum 8-hour moving average concentrations would delay 6.64 (95%CI: 3.64 – 11.14) deaths per year in the study area, 3.24 (95%CI: 1.55 – 5.15) from cardiovascular diseases, and 1.89 (95%CI: 1.24 – 2.52) from respiratory causes.

Table 6. Potential benefits of reducing ozone daily levels. Absolute numbers and rates (per 100 000 inhabitants) (95% confidence limits) attributable to the health effects of ozone.

	OZONE reduction	Number of attributable cases per year	Annual rates (per 100.000)
MORTALITY	Daily 8-h max		
Total	by 10 µg/m ³ to 120 µg/m ³	6.64 (3.64 - 11.14) 1.20 (0.66 - 2.01)	0.99 (0.54 - 1.66) 0.18 (0.10 - 0.30)
Cardiovascular	by 10 µg/m ³ to 120 µg/m ³	3.24 (1.55 - 5.15) 0.60 (0.28 - 0.95)	0.48 (0.23 - 0.77) 0.09 (0.04 - 0.14)
Respiratory	by 10 µg/m ³ to 120 µg/m ³	1.89 (1.24 - 2.52) 0.37 (0.24 - 0.50)	0.28 (0.18 - 0.38) 0.06 (0.04 - 0.07)
MORBIDITY	Daily 1-h max		
Emergency room visits for asthma <18y	by 10 µg/m ³ to 180 µg/m ³		Not available Not available
	Daily 8-h max		
Hospital respiratory admissions 15-64 y	by 10 µg/m ³ to 120 µg/m ³	1.34 (-12.04 - 16.05) 0.24 (-2.11 - 2.84)	0.28 (-2.55 - 3.40) 0.05 (-0.45 - 0.6)
Hospital respiratory admissions > 64 y	by 10 µg/m ³ to 120 µg/m ³	5.03 (-2.01 - 12.06) 0.93 (-0.37 - 2.23)	5.49 (-2.19 - 13.16) 1.01 (-0.40 - 2.43)

NA: Not applicable if air pollution levels are lower than the scenario level

Discussion

Mortality data are highly reliable, and hence do not represent a major source of uncertainty for the results of the present HIAs. On the other hand, hospital admission data present a major source of uncertainty because they include both emergency hospital admissions and scheduled hospital admissions, that are certainly not temporally linked with the levels of air pollution. Hence, the numbers of attributable hospital admissions are certainly over-estimated.

In Toulouse study area, ozone levels in 2001 are slightly higher than the target value for 2010 ($120 \mu\text{g}/\text{m}^3$ not to be exceeded on more than 25 days per calendar year averaged over three years). The compliance with long term objectives for ozone (maximum daily 8-h mean within a calendar year lower than $120 \mu\text{g}/\text{m}^3$) would induce a slight but significant benefit in term of deaths (1.20 for total mortality). But above all, reduction of daily 8-h max levels of ozone by $10 \mu\text{g}/\text{m}^3$ would induce larger health benefits in terms of mortality (respectively 6.64, 3.24, 1.89 for total, cardiovascular and respiratory mortality).

PM10 levels are compliant with 2005 limit values ($40 \mu\text{g}/\text{m}^3$). Hence, there are no attributable cases for the scenario corresponding to a reduction of the annual mean to $40 \mu\text{g}/\text{m}^3$. For a reduction of the annual mean by $5 \mu\text{g}/\text{m}^3$ or a reduction of the annual mean to $20 \mu\text{g}/\text{m}^3$, the number of attributable cases in the study area is very slight, because of the low size of this age group, but it is significant.

The numbers of attributable cases may seem small, especially when compared with the number of deaths attributable to other risk factors, such as tobacco smoking. However, air pollution exposure concerns everyone, whereas exposure to other risk factors may be more easy to control at the individual level.

Conclusion

The results from the present HIAs may help promoting measures aiming at reducing air pollutant emissions, especially traffic linked emissions, as health benefits are a powerful way of motivating changes in individuals comportment.

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