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Traffic-Related Air Pollution: Legislation *Versus* Health and Environmental Effects

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1. Introduction

Ambient air quality is a very topical issue as it has an important influence on human health. Exposure to atmospheric pollutants may result in various adverse health effects. The impacts of air pollution are not confined only to human health but also to the environment as a whole. In that regard, vehicular traffic emissions are especially important, because its volume is increasing every year. Consequently pollutants, such as nitrogen oxides (NOx), carbon monoxide (CO), particulate matter (PM), and polycyclic aromatic hydrocarbons (PAHs) are emitted into the atmosphere causing a significant decline of air quality across Europe, which results in hundreds of thousands of premature deaths every year. In order to improve the situation, the European Union has been defining legislation on ambient air quality with limits of the respective pollutants and aiming to increase the levels of public health protection. Despite reductions in emissions, concentrations of these pollutants remain high — often above existing targets — exposing populations to levels that reduce life expectancy, cause premature death and widespread aggravation to health.

In this chapter, various aspects of air pollution are discussed with specific emphasis on vehicular road traffic. An overview of the current legislation related to air quality is given. The work then focuses on the health impacts of important traffic related pollutants, with particular focus on polycyclic aromatic hydrocarbons (PAHs). The general description of PAHs is presented with further discussion on their health and environmental impacts.

2. Air pollution

Air quality is a very topical issue at the moment. Ultimately we are all surrounded by air both indoors and outdoors and we need air to live; the daily human requirement for air is around 15 kg. Generally air is freely available and we have come to regard access to air of acceptable quality as a fundamental human right.

Primary air pollutants are emitted directly into the atmosphere, whilst secondary pollutants are formed in it. Primary air pollutants include nitrogen oxides, sulfur dioxide, volatile

organic compounds, and particles that are released into the atmosphere from road transport emissions, stationary combustion sources, and from natural emissions. Secondary air pollutants are formed from chemical reactions of primary pollutants in the atmosphere and include ground level ozone and secondary particulates. The total emissions of the main air pollutants in 27 Member States of the European Union are presented in Table 1 (EEA, 2010).

Pollutant	Units	1990	2008	Change (%) 1990-2008
Nitrogen oxides (NOx)	Gg	17 152	10 397	– 39
Carbon monoxide (CO)	Gg	64 526	27 228	– 58
Sulphur oxides (SOx)	Gg	26 208	5 867	– 78
Particles ($PM_{2.5}$)	Gg	1 612[a]	1 403	– 13
Particles (PM_{10})	Gg	2 299[a]	2 126	– 8
Mon-methane volatile organic compounds (NMVOC)	Gg	16 807	8 296	– 51
Total polycyclic aromatic hydrocarbons (PAHs)	Mg	3 416	1 359	– 60
Amonium ($NH_{3)}$)	Gg	4 997	3 799	– 24
Lead (Pb)	Mg	22 398	2 293	– 90
Cadmium (Cd)	Mg	281	118	– 58
[a]data of 2000, once information for previous years is not available				

Table 1. Emissions of the main air pollutants during 1990-2008 in 27 Member States of European Union

Table 1 clearly demonstrates that among the main air pollutants, the largest reductions across 27 Member States of the European Union have been achieved for lead and SOx, which have decreased since 1990 by 90% and 78%, respectively. The implementation of regulations setting limits of lead and sulfur dioxide levels in urban areas has contributed to these significant reductions. Other parallel political actions devoted to the control of urban atmospheric emissions include a ban of the use of lead additives in gasoline (Directive 98/70/EC), sulfur abatement technologies in industrial facilities (EEA, 2011a) and the introduction of fuels with reduced levels of sulfur (Directive 98/70/EC; EN 590/2004). These initiatives have all contributed to a sharp decrease of the emitted amounts of sulfur dioxide. Emissions of other key air pollutants also decreased since 1990. It is noteworthy that these significant reductions include emissions of the three air pollutants primarily responsible for the formation of harmful ground-level ozone in the atmosphere, namely carbon monoxide (58% reduction), non-methane volatile organic compounds (51% reduction) and nitrogen oxides (39% reduction). The concentrations of particulate matter have not shown significant improvement since 1997. Emission trends compiled for the period 2000–2008 indicate that PM_{10} emissions decreased by 8%, while $PM_{2.5}$ was reduced by 13%. Fine particulate matter is now generally recognized as one the main threats to human health from air pollution, with transport being a significant source (EEA, 2011b). Data available on cadmium reveal that since 1990 significant emission reductions have occurred for this toxic heavy metal (around 60%). These reductions were due to improved abatement technologies for combustion facilities and in the metal refining and smelting

industries (EEA, 2009). However, despite the emissions reductions, concentrations of many of these pollutants remain high, often above existing standards (EEA, 2005).

Sources of air pollutants may be classified as stationary (fossil fuel power plants, petrochemical plants, petroleum refineries, food processing plants, other large and small industries, and home heating) or mobile (automobiles, industrial vehicles, trains, all types of vessels, and airplanes) (Godish, 2004). Among these, emissions from vehicle road transport are especially important as they are a significant source of pollution within urban areas throughout the world. Some authors (Fischer et al., 2000; Martuzevicius et al., 2008) reported twofold differences in the concentrations of several traffic-related primary pollutants (black carbon, fine particulate matter, benzo[a]pyrene, and benzene) in locations with high and low traffic activity.

In Europe emissions of some road transport–related pollutants, such as nitrogen oxides or non-methane volatile organic compounds have decreased since 1990 (EEA, 2010), mainly due to the introduction of new technologies (i.e. three way catalytic converters on passenger cars) and stricter regulation of emissions from heavy duty vehicles (Regulation 595/2009). Despite these decreases the Member States of the European Union still have difficulty complying with the legislative limits of traffic related pollutants (EEA, 2008), mainly due to the fact that the demand for road transport has been growing much faster than anticipated. Transport volumes are growing about 1.9% annually for passenger and 2.7% for freight transport (EEA, 2011b). Road transport remains the most important source of the ozone precursors, namely of nitrogen oxides and carbon monoxide in Europe, in 2008 contributing 40% and 34% of total European emissions, respectively (Table 2; EEA, 2008, 2010). Whereas passenger cars and heavy duty vehicles contribute the majority of road transport nitrogen oxide emissions, for carbon monoxide passenger cars alone contribute around 4/5 of the emissions from the road transport sector. Road transport is also a significant source of non-methane volatile organic compounds (Table 2) and of $PM_{2.5}$ and PM_{10} emissions (EEA, 2008, 2010).

Increases in urbanization and motor vehicle use have raised questions about the health effects of exposure to traffic pollutants. Kunzli et al. (2000) estimated that in European countries France, Switzerland and Austria, with a total population of 74 million inhabitants, 3% of total mortality per year (i.e. 20 000 deaths) are due to traffic emissions alone; hypothetically total omission of traffic emissions would lead to prolonged life expectancy of 0.35 years. Other studies indicate that living near roads with heavy traffic may considerably increase the risks of adverse health effects (Beelen et al., 2009; Heinrich et al., 2005; Janssen et al., 2001). Some of those studies also provided evidence of effects related to the distance from major roads and traffic density (Hoek et al., 2002). Recently, Brunekreef et al. (2009) reported results from a very comprehensive European cohort study on the effects of long-term exposure to traffic pollutants and cause-specific mortalities. Specifically, the authors observed effects of particulate matter ($PM_{2.5}$), nitrogen oxides and sulfur dioxides with relative risks estimated for concentration change of 10 $\mu g/m^3$ of $PM_{2.5}$, 30 $\mu g/m^3$ of NO_2, and 20 $\mu g/m^3$ of SO_2. The largest risk estimates were found for respiratory mortalities for which the relative risks were 1.37 (95 CI, 1.00–1.87) for $PM_{2.5,}$ 1.07 (0.75–1.52) for $NO_{2,}$ and 0.88 (0.64–1.22) for SO_2. For cardiovascular deaths the authors reported relative risks of 1.07 (95 CI, 0.94–1.21), 1.04 (0.90–1.21), and 0.94 (0.82–1.06) for $PM_{2.5}$, NO_2, and SO_2, respectively.

As it can be seen there was no association between SO_2 concentrations and mortalities as there was no traffic contribution to this pollutant. In view of this and other research studies the European Union recognizes road transport as significant pollution source and considers the reduction of its emissions fundamental in order to protect public health.

	Contribution of various sources (%)					
Pollutant	Passenger cars	Heavy duty vehicles	Light duty vehicles	Other road transport emissions	Total road transport	Other sources
Nitrogen oxides (NOx)	19	18	3		40	60
Carbon monoxide (CO)	28	2	2	2	34	66
Sulfur oxides (SOx)	-	-	-	-	0	100
Particles (PM_{10})	4	3	2	5	14	86
Particles ($PM_{2.5}$)	5	4	3	3	15	85
Mon-methane volatile organic compounds (NMVOC)	11	2	1		14	86
Total polycyclic aromatic hydrocarbons (PAHs)	8	1			9	91
Amonium (NH_3)	2	-	-	-	2	98
Lead (Pb)	4			4	4	96
Cadmium (Cd)	1	1			2	98

Table 2. Source contribution of air pollutants in 2008 in 27 Member States of European Union (EEA, 2008, 2010)

3. European standards for ambient air

Humans can be adversely affected by exposure to hazardous air pollutants in ambient air. Since the early 1970s, the European Union has made efforts to improve air quality by controlling emissions of harmful substances into the atmosphere, improving fuel quality, and by integrating environmental protection requirements into the transport and energy sectors. Thirty years of environment policy has led to a comprehensive system of environmental controls. In order to protect public health, the European Union has established and implemented a large number of health-based standards of pollutants in ambient air. Current European standards of pollutants in ambient air are summarized in Table 3.

Pollutant	Averaging period of time	Limit/Target value	Number of exceedances	Date of enforcement
Carbon monoxide	Maximum daily 8 hour mean	10 mg/m^3	n/a	Limit value entered into force 1.1.2005
Nitrogen dioxide	1 hour	200 µg/m^3	18	Limit value entered into force 1.1.2010
	1 year	40 µg/m^3	n/a	Limit value entered into force 1.1.2010
*Ozone	Maximum daily 8 hour mean	120 µg/m^3	25 days averaged over 3 years	Target value entered into force 1.1.2010
Sulfur dioxide	1 hour	350 µg/m^3	24	Limit value entered into force 1.1.2005
	24 hours	125 µg/m^3	3	Limit value entered into force 1.1.2005
Particles PM_{10}	24 hours	50 µg/m^3	35	Limit value entered into force 1.1.2005
	1 year	40 µg/m^3	n/a	Limit value entered into force 1.1.2005
Particles $PM_{2.5}$	1 year	25 µg/m^3	n/a	Target value entered into force 1.1.2010. Limit value enters into force 1.1.2015
Benzene	1 year	5 µg/m^3	n/a	Limit value entered into force 1.1.2010
*Polycyclic aromatic hydrocarbons	1 year	1 ng/m^3	n/a	Target value entered into force 31.12.2012
*Arsenic	1 year	6 ng/m^3	n/a	Target value enters into force 31.12.2012
*Cadmium	1 year	5 ng/m^3	n/a	Target value enters into force 31.12.2012
*Nickel	1 year	20 ng/m^3	n/a	Target value enters into force 31.12.2012
Lead	1 year	0.5 µg/m^3	n/a	Limit value entered into force 1.1.2005 (or in 1.1.2010 in the immediate vicinity of the specific industrial sources situated on sites contaminated by decades of industrial activities; and a 1.0 µg/m^3 limit value applied from 1.1.2005 to 31.12.2009)

n/a not available; *Target value

Table 3. European air quality standards (Directive 2008/50/EC; Directive 2004/107/EC)

With exception to so-called "fourth daughter directives" (Directive 2004/107/EC) most of the existing European legislation on ambient air (Directive 96/62/EC, daughter Directives 1999/30/EC, 2000/69/EC, 2002/3/EC, and Council Decision 97/101/EC) have been merged into a single directive in 2008 when a new Directive 2008/50/EC on ambient air quality entered into force.

As can be seen the standards apply over different periods of time, as it was estimated that health impacts associated with these pollutants occur over different exposure times. In terms of suspended particles, the directive 2008/50/EC represents a significant step forward as for the first time air quality objectives for $PM_{2.5}$ (i.e. fine particles) were set. Except for the annual $PM_{2.5}$ limit value (Table 3), the directive also introduced additional parameters that target the exposure of the population to fine particles. These parameters are exposure concentration obligations and national exposure reduction targets (Table 4). Both parameters are based on the average exposure indicator (AEI), which represents a 3-year running annual mean of $PM_{2.5}$ concentration averaged over the selected monitoring stations in agglomerations and larger urban areas, set in urban background locations to best assess the $PM_{2.5}$ exposure of the general population (Directive 2008/50/EC).

Parameter	Averaging period of time	Value	Number of exceedances	Date of enforcement
$PM_{2.5}$ Exposure concentration obligation	Based on 3 year average	20 μg/m^3 (AEI)	n/a	Legally binding in 2015 (years 2013, 2014, 2015)
$PM_{2.5}$ Exposure reduction target	Based on 3 year average	Percentage reduction + all measures to reach 18 μg/m^3 (AEI)	n/a	Reduction to be attained where possible in 2020, determined on the basis of the value of exposure indicator in 2010

Table 4. European $PM_{2.5}$ exposure parameters (Directive 2008/50/EC)

To meet the $PM_{2.5}$ exposure concentration obligation, AEI in 2015, should be less than 20 μg/m^3. The national exposure reduction target stipulates that between 2010 and 2020 Member States should reduce their $PM_{2.5}$ concentrations by certain percentages (0, 10, 15, or 20%), depending on the level of their AEI in 2010 (Directive 2008/50/EC). If AEI in 2010 is assessed to be over 22 μg/m^3, all appropriate measures need to be taken to achieve 18 μg/m^3 by 2020. The reduction is not necessary in cases where AEI in 2010 was equal to, or below 8.5 μg/m^3. There is no explanation given for this value, nevertheless, some authors observed (Brunekreef & Maynard, 2008) that in studies that evaluated relationships between $PM_{2.5}$ exposure and respective health responses, concentrations of 8.5 μg/m^3 represented $PM_{2.5}$ levels associated with lower risks (Laden al., 2000; Pope et al., 2002). The national exposure reduction target is provisionary. Depending on the outcome of the 2013 review it should be replaced by legally binding national exposure reduction obligations.

Other significant changes of the Directive 2008/50/EC include the possibility for Member States to discount natural sources of pollution when assessing compliance against limit

values. The Member states can also apply for possible time extensions of three years (for PM_{10}) or up to five years (for NO_2, benzene) for complying with the set limit values, based on conditions and the assessment by the European Commission.

Even though the regulatory efforts of the last decade, the levels of some health hazardous pollutants in ambient air, namely particulate matter and ozone have not shown any significant improvements despite the decrease of their respective emissions (Table 1). A number of countries are also likely to miss one or more legally binding 2010 emission ceilings. As many European citizens still live in cities where air quality limits set for the protection of human health are exceeded, the need to reduce exposure to air pollution remains an important issue.

4. Health effects of main traffic pollutants

4.1 Particulate matter

Particulate air pollution was one of the first types of pollution that demonstrated evidence of health effects even at low ambient levels. Thus there is a wealth of consistent evidence of particulate matter related health effects that include morbidity and mortality outcomes, both general and cause-specific. The evidence from numerous epidemiological studies on long-term responses indicated that an increase of 10 $\mu g/m^3$ in daily PM_{10} average concentration is associated with approximate risks of 1.013 and 1.009 for respiratory and cardiovascular deaths (WHO, 2006). Also increased hospitalizations and related health care visits are significant for various respiratory diseases and, to a lesser extent, for cardiovascular disease (Medina-Ramon et al., 2006; Vigotti et al., 2010). Increased symptom prevalence includes lower respiratory system symptoms, asthma, and cough (WHO, 2006). Although the mechanisms of underling respiratory morbidity and mortality due to PM exposure are not clear, it is thought that the fine particles (i.e. $PM_{2.5}$) are of greatest concern to health. Due to their smaller sizes these fine particles are breathed into the deepest parts of lungs. Thus the scientific attention has been focused on these fine particles. Studies on long-term exposure to $PM_{2.5}$, showed an association with different cardiac and pulmonary health effects. Recent studies have also reported very high associations between the atmospheric concentrations of $PM_{2.5}$ and daily mortality rates. Total mortality appears to increase approximately 2 to 4% for every 5 $\mu g/m^3$ increase in $PM_{2.5}$, associated in a higher extent with cardiopulmonary system (WHO, 2006). Furthermore, epidemiological studies have reported that there was a clear association between episodes of $PM_{2.5}$ and increases in respiratory disease (bronchitis), impaired lung function, coughing, infections of the lower respiratory tract, and respiratory symptoms in asthmatics (WHO, 2006).

4.2 Polycyclic aromatic hydrocarbons

PAHs represent a class of organic compounds with two or more fused aromatic rings. They originate from a wide variety of natural and anthropogenic sources. The largest releases of PAHs are due to the incomplete combustion of organic matter, such as coal, oil and gas (Shibamoto, 1998) during the course of industrial processes and other human activities. Forest fires, which may or may not be the consequence of human activity, are also a significant and usually unpredictable source of PAHs. In urban atmospheres, PAHs are mainly of anthropogenic origin; road vehicle traffic is one of the most important

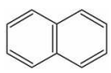
anthropogenic emission sources, in urban areas contributing by as much as 74% of PAH emissions (Omar et al., 2002). Polycyclic aromatic hydrocarbons are also emitted from a variety of stationary sources, burning of domestic fuels are a significant source of PAHs (WHO, 1998).

In general, PAHs are ubiquitous compounds with low solubility in water, high melting and boiling points, and low vapor pressures. The physical-chemical properties of PAHs are greatly influenced by their molecular structure, i.e. by number of rings and molecular weight. While the physical-chemical properties of PAHs vary considerably, the semi-volatile properties of some PAHs make them highly mobile throughout the environment, with deposition and re-volatilization processes distributing them between air, soil and water; some PAHs are subject to long-range transport through the atmosphere making them a transboundary environmental problem. PAHs, whether dissolved in water or present in the air, can undergo photodecomposition in the presence of the ultra violet light from solar radiation (Park et al., 2002).

PAHs exist as many different isomers. Out of the currently identified compounds, the United States Environmental Protection Agency (US EPA) has recommended sixteen PAHs as "priority pollutants", due to their potential carcinogenic and mutagenic properties. Table 5 summarizes the physical-chemical properties of the priority PAHs (WHO, 1998); as demonstrated in Fig. 1 all compounds are parental PAHs, i.e. aromatic rings without any alkyl substitution.

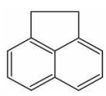
Compound	Molecular weight	Melting point	Boiling point	Vapor pressure at 25 °C	Solubility in water at 25 °C
	g/mol	(°C)	(°C)	(Pa)	(µg/L)
Naphthalene	128.17	81	218	10.4	3.17×10^{4}
Acenaphthylene	152.19	92-93	265	8.9×10^{-1}	3.93×10^{3}
Acenaphthene	154.21	95	279	2.9×10^{-1}	3.4×10^{3}
Fluorene	166.22	115-116	295	8.0×10^{-2}	1.98×10^{3}
Anthracene	178.23	216	342	8.0×10^{-4}	73
Phenanthrene	178.23	100	340	1.6×10^{-2}	1.29×10^{3}
Fluoranthene	202.25	109	375	1.2×10^{-3}	260
Pyrene	202.25	150	393	6.0×10^{-4}	135
Benz[a]anthracene	228.29	161	400	2.8×10^{-5}	14
Chrysene	228.29	254	448	8.4×10^{-5}	2.0
Benzo[b]fluoranthene	252.31	167	357	----------	1.2
Benzo[k]fluoranthene	252.31	216	480	1.3×10^{-7}	0.76
Benzo[a]pyrene	252.31	178	496	7.3 × 10-7	3.8
Dibenz[a,h]anthracene	278.35	267	524	1.3×10^{-8} (20 °C)	0.5 (27 °C)
Indeno[1,2,3-cd]pyrene	276.33	164	536	1.3×10^{-8} (20 °C)	62
Benzo[ghi]perylene	276.33	278	545	1.4×10^{-8}	0.26

Table 5. Physical-chemical properties of the priority PAHs (WHO, 1998)

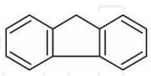


Naphthalene

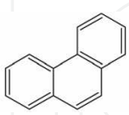
Acenaphthylene



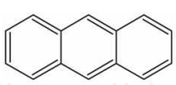
Acenaphthene



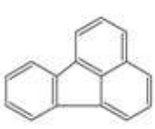
Fluorene



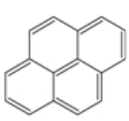
Phenanthrene



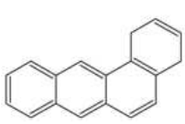
Anthracene



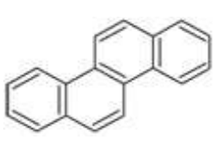
Fluoranthene



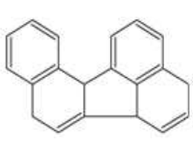
Pyrene



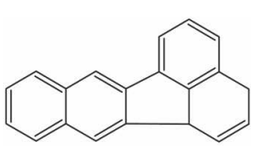
Benz[a]anthracene



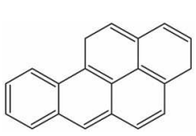
Chrysene



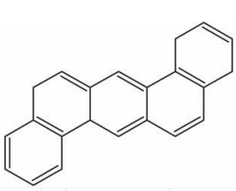
Benzo[b]fluoranthene



Benzo[k]fluoranthene

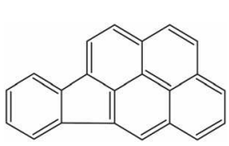


Benzo[a]pyrene



Dibenz[a,h]anthracene

Benzo[ghi]perylene



Indeno[1,2,3-cd]pyrene

Fig. 1. Molecular structures of 16 PAHs listed as priority pollutants by U.S. Environmental Protection Agency (Shibamoto, 1998)

In the ambient air PAHs are present both in the vapor phase as well as bound to particles. PAHs with low molecular weight are usually found more in the vapor phase, but the majority of compounds with four or more rings are mainly particulate–bound (Slezakova et al., 2011; Srogi, 2007). The series of related studies performed in Oporto, Portugal (Castro et al., 2009; Slezakova et al., 2010, 2011) showed that in urban environments with major influences of vehicular traffic emissions, on average 5 to 8% of total particulate PAH content was associated with bigger particles (i.e. $PM_{2.5-10}$) whereas 92–95% of total PAH content was present in $PM_{2.5}$; in a remote site 95 % of PAHs were $PM_{2.5}$–bound (Slezakova et al., 2010). When PAHs are adsorbed onto a particle, its size is then the key parameter influencing transport of the compounds within the atmosphere. Larger particles are removed from the air by gravitational settling or impaction, but generally PAHs are not adsorbed onto these large particles. The residence time of a particle smaller than 1 μm is between 4 and 40 days, and from 0.4 till 4 days for a particle with an aerodynamic diameter of 1−10 μm (Smith, 1984). Without wet deposition the residence time of a particle can be longer, consequently PAHs adsorbed on a particle surface can travel long distances before deposition from the atmosphere (Kiss et al., 1996). Hence distribution, residence time, transport, and wet and dry deposition of PAHs in the atmosphere are mainly influenced by the nature of particulate matter. However, the persistence of PAHs in the atmosphere also depends on atmospheric conditions, such as solar radiation intensity, temperature, relative humidity; precipitation is considered to be the dominant sink for atmospheric PAHs. Temperature is probably the most important physical parameter that influences distribution of PAHs between particulate and gaseous phases. Temperature increase promotes vaporization of PAHs and gaseous PAHs are more likely to be subjected to transformation and reduction by photochemical degradation (Fang et al., 2006; Tsapakis & Stephanou, 2005). PAH decay under low outdoor humidity conditions was slower than at high humidity (Kamens et al., 1988; Tsapakis & Stephanou, 2007). In the presence of sunlight PAHs can undergo a photo-oxidation reaction that is recognized as one of the important removal process of PAHs from the atmosphere (Fang et al., 2006). Finally, the levels of other pollutants also influence the transformation of PAHs in the atmosphere and the reaction of ozone and PAHs is considered as a degradation process of these compounds, reducing their atmospheric concentrations (Park et al., 2002; Tham et al., 2008).

PAHs are typically found in a mixture of many compounds. In studies that estimate human cancer risk from exposure to complex mixtures of PAHs, benzo[a]pyrene has been commonly used as a substitute for other compounds, due to its strong carcinogenicity. However, some authors have questioned the appropriateness of this approach (Pufulete et al., 2004). The concerns are related to the variability of the compositions of different PAH mixtures. For example it was observed that benzo[a]pyrene represented less than 3% of the total PAH content in emissions originated from combustion sources (Castro et al., 2011). In various mixtures, low-potency PAHs, such as phenanthrene may occur in high concentrations (Slezakova et al., 2011) or, as recently discovered, some PAH compounds could be present in minor amounts, nevertheless possessing higher carcinogenic potency (such as dibenzo[a,l]pyrene with potency two orders of magnitude higher than benzo[a]pyrene; Castro et al., 2010, 2011; Slezakova et al., 2009). The concept of Toxicity Equivalency Factor (TEF) estimates the human cancer risk from exposure to complex PAHs using TEF for each compound in a PAH mixture, thus allowing for the aggregation of all concentrations, weighted for their carcinogenetic potency relative to that of benzo[a]pyrene.

However, the studies evaluating toxic effects of PAH mixtures do not recognize interactions between the individual PAH compounds in mixtures, which could lead to significant increases of health risks. At this moment complete understanding of these interactions is not possible as the current knowledge is still limited, thus these problems are yet to be solved by the scientific community.

4.2.1 Health impacts

Individual PAHs are extremely hazardous to human health. Many of them are cytotoxic and mutagenic (WHO, 1998) and they constitute the largest group of known carcinogens. The carcinogenic potency of individual PAHs is widely varying. Out of sixteen PAHs recommend by US EPA as priority pollutants benzo[a]pyrene has been classified by the International Agency for Research on Cancer (IARC) as an known carcinogen to humans (Group 1; IARC, 2010), whereas other PAHs have been considered as probable (Group 2A) and possible (Group 2B) human carcinogens (IARC, 2002, 2010). Table 6 shows the carcinogenicity of 16 US EPA PAHs and dibenzo[a,l]pyrene using different classification systems of IARC, US EPA and the TEF concept.

Because of their hazardous properties there has been widespread interest in analyzing and evaluating human exposure to PAHs in ambient air. Nevertheless, for obvious reasons, there are no studies in which the humans were deliberately exposed to PAHs. The information on the effects of inhaled PAHs comes only from epidemiological biomarker studies of humans exposed to PAHs in work places or in urban environments. The first PAH studies were conducted in the 1990s in the heavily polluted northern region of the Czech Republic with high ambient concentrations of benzo[a]pyrene up to tens of ng/m^3 (Binkova et al., 1996; Dejmek et al., 2000). A significant correlation between individual exposures to carcinogenic PAHs and DNA adducts was found, this effect being significant especially for non-smokers (Binkova et al., 1995). Since then, other studies were performed in less polluted areas (concentrations of benzo[a]pyrene lower then 5 ng/m^3) around the world (Jung et al., 2010; Liao et al., 2011; Novotna et al., 2007; Palli et al., 2008). Although the results of all these studies were not completely consistent, they indicated that exposure to levels of PAHs present in urban air, even at relatively low concentrations, resulted in high levels of health risks.

The health concerns of PAHs have been traditionally focused on their potential carcinogenicity in humans, which seems to be beyond dispute. PAHs are genotoxic compounds and their carcinogenicity is probably mediated by their ability to damage the DNA (Irigaray & Belpomme, 2010; Novotna et al., 2007; Palli et al., 2008). Even exposure to low doses of PAHs might be associated with various cancers, indicating that there is no safe threshold. However, regarding the PAH carcinogenicity due to exposure to polluted air, it is important to point out that there is no epidemiological evidence showing that at levels present in urban air PAHs cause cancer. Until now the only evidence of PAH carcinogenicity in humans exists for long-term exposure (of many years) to polluted air of work places with high concentrations of PAHs, which exceed those in ambient air by orders of magnitude (Bostrom et al., 2002; Peluso et al., 2001; Srogi, 2007). Due to the lack of useful, good-quality data, the quantitative cancer risk estimates of PAHs as air pollutants are very uncertain, because they are based on extrapolation from substantially higher occupational concentrations, which makes it difficult to draw conclusions (Bostrom et al., 2002).

Compound	Classification			
	IARC[a]	US EPA[b]	TEF[c]	Unit risk[d] $(\mu g/m^3)^{-1}$
Naphthalene	2B	C	0.001	-
Acenaphthylene	not available	D	0.001	-
Acenaphthene	3	not available	0.001	-
Fluorene	3	D	0.001	-
Phenanthrene	3	D	0.001	-
Anthracene	3	D	0.01	-
Fluoranthene	3	D	0.001	2.8×10^{-4}
Pyrene	3	D	0.001	-
Chrysene	2B	B_2	0.1	8.7×10^{-4}
Benz[a]anthracene	2A	B_2	0.1	4.0×10^{-3}
Benzo[b]fluoranthene	2B	B_2	0.1	1.0×10^{-2}
Benzo[k]fluoranthene	2B	B_2	0.1	2.8×10^{-3}
Benzo[a]pyrene	1	B_2	1	8.7×10^{-2}
Dibenz[a,h]anthracene	2A	B_2	5	1.8×10^{-1}
Benzo[g,h,i]perylene	3	D	0.01	-
Indeno[1,2,3-c,d]pyrene	2B	B_2	0.1	1.1×10^{-2}
Dibenzo[a,l]pyrene	2A	not available	100[e]	8.7×10^{-0}

[a](IARC, 2002, 2010): Group 1 - carcinogenic to humans; Group 2A - probably carcinogenic to humans; Group 2B - possible carcinogenic to humans; Group 3 - unclassifiable as to carcinogenetic in humans; Group 4 - probably not carcinogenic to humans

[b](USEPA, 1986, 2005): Group A - human carcinogens; Group B - probable human carcinogens (B1: based on limited evidence of carcinogenicity in humans and sufficient evidence of carcinogenicity in animals; B2: based on sufficient evidence of carcinogenicity in animals);Group C - possible human carcinogens; Group D - not classifiable as to human carcinogenicity; Group E - evidence of non-carcinogenicity for humans

[c]Toxicity Equivalency Factor (TEF): estimation based on the relative potency to benzo(a)pyrene (Nisbet & LaGoy, 1992)

[d]Unit risk (WHO, 1998).

[e](Pufulete et al., 2004; Okona-Mensah et al., 2005)

Table 6. Classification of selected PAHs

Furthermore humans are never exposed only to a single PAH compound in ambient air, and the coexistence of PAHs in various mixtures implies further difficulties. To fully understand the carcinogenesis of PAHs and their role as air pollutants, these issues need to be correctly addressed by further research.

4.2.2 Environmental impacts

From a global point of view, the largest emissions of PAHs are found in the atmosphere. However, apart from release into air, PAHs can be also transferred directly to water, soil and sediments. Marine pollution by crude oil causes appreciable quantities of PAHs (Grueiro-Noche et al., 2010; Martins et al., 2011). Improper waste disposal and biomass burning have also caused serious PAH pollution of land in some localities (Chrysikou et al., 2008; Chung et al., 2007; Liu et al., 2010).

The most common effects of organisms that occur with long-term exposure to PAHs are bioaccumulation, behavioral alternation in some species, reduction in growth, reduced reproduction and deformities, and increased mortalities (Khanal, 2003). Tumor development has also been reported in fish exposed to benzo[a]pyrene (WHO, 1998) as well as various acute effects (Tintos et al., 2008; Viera et al., 2008). Some PAHs (naphthalene, phenanthrene, and fluoranthene) are acutely toxic to aquatic organisms and the toxicity is affected by metabolism and photosynthesis (Khanal, 2003); in the presence of ultraviolet light the toxicity of PAHs gets more intense (Arsften et al., 1996; WHO, 1998).

The effects of air pollution are not confined only to human health or environment but also to buildings and historical monuments. These impacts can have permanent consequences that might lead to potential losses of these, principally irreplaceable historical structures forever. One of the most important building deterioration phenomena is the deposition of pollutants on surfaces (Marioni et al., 2003) which consequently affects façades of buildings and monuments (Gaviño et al., 2004). Eventually the particles, together with dry-deposited gases such as SO_2 result in the formation of hard, grey - black crusts, in which airborne organic pollutants, such as PAHs and a wide range of particulate matter (including dust, pollen, and spores) are entrapped (Fig. 2). The pressure from the crystal growth breaks off small areas of stone thus exposing a more vulnerable surface. Recent studies showed that deposition of these pollutants is important also from a health hazard perspective. Historical monuments and buildings in urban areas can act as passive repositories for air pollutants present in the surrounding atmosphere and may lead to higher human exposures thus representing additional risks for human health (Slezakova et al., 2011).

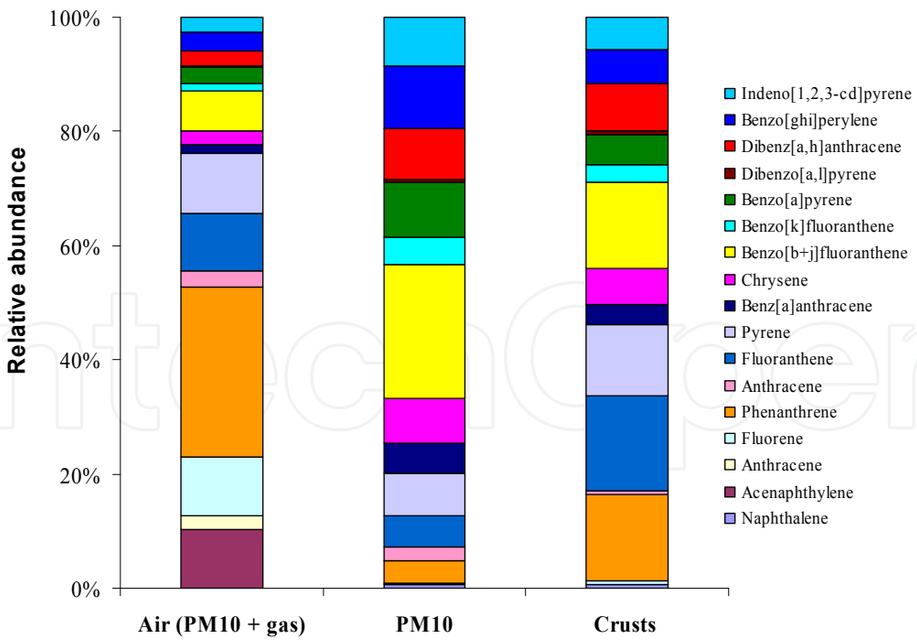


Fig. 2. Selected PAHs in Oporto, Portugal. The graph demonstrates the abundance of PAHs during the winter of 2008 in air (i.e. sum of both gas and particulate phase), in PM_{10}, and in black crusts of a selected historical monuments situated in an urban site. The similarity of the contribution profiles between PAHs in black crusts and in PM_{10} is obvious.

4.3 Nitrogen dioxide

Nitrogen monoxide is almost instantaneously oxidized to nitrogen dioxide. Of the two gases, nitrogen dioxide is much more toxic to humans. Nitrogen dioxide is less soluble than sulfur dioxide, so that a much higher proportion penetrates into the deep lung. Approximately 70–90% of nitrogen dioxide inhaled can be absorbed from the respiratory tract of humans (Tiwary & Colls, 2009). In human studies, nitrogen dioxide has been associated with adverse health effects even at low ambient concentrations. Exposure to high nitrogen dioxide levels from occupational exposure may have adverse effects such as pulmonary edema (Godish, 2004; WHO, 2006). Asthmatics appear to be the most reactive group upon exposure to nitrogen dioxide, although controlled studies on the effects of short-term exposure on the symptoms and severity of asthma have not led to clear findings (WHO, 2006). Short-term exposure studies have shown that asthma sufferers may experience enhanced sensitivity after exposure to nitrogen dioxide, and that those with normal respiratory function may experience increased airway resistance (WHO, 2006).

4.4 Ozone

Ozone is a colorless gas. As mentioned previously it differs from the other pollutants because of its secondary origin (i.e. formed in the atmosphere rather than being emitted). It is formed through a series of complex reactions in the atmosphere involving solar radiation and anthropogenic pollutants, such as non-methane volatile organic compounds and carbon monoxide, in the presence of nitrogen dioxide (Alvim-Ferraz et al., 2006). The concentration of ozone in the atmosphere depends on several factors: sunshine intensity, atmospheric convection, the height of the thermal inversion layer, and concentrations of nitrogen oxides and other precursors (WHO, 2006). As a consequence of anthropogenic activities, ground-level ozone represents a major concern because of its concentration increase. In densely populated areas of Europe and USA the levels of ozone can reach up to 200 $\mu g/m^3$ (1-hour mean; WHO, 2006). Much higher levels of ozone (up to 400 $\mu g/m^3$ during several days) can be observed in developing countries, where the combination of mega-cities with significant emissions of ozone precursors and a climate that favors photochemical reactions of ozone formation (WHO, 2006) is found. Exposure to ozone is almost exclusively by inhalation and has been associated with both acute and chronic effects. Short-term exposure to high ozone concentrations includes effects on the pulmonary and cardiovascular systems with evidence of both morbidity and mortality (WHO, 2006). Long-term exposure to relatively low levels is also of concern; it can lead to the development of atherosclerosis and asthma, reduction in lung function, and life expectancy (Sousa et al., 2009; WHO, 2006).

4.5 Carbon monoxide

Exposure to carbon monoxide may be lethal; however poisoning is typically caused in confined spaces (indoors, cars) by exposures to carbon monoxide at levels considerably higher than those existent in ambient air. Generally, the mechanism of carbon monoxide toxicity is tissue hypoxia. Carbon monoxide combines with blood haemoglobin about 200 times more readily than oxygen (Colls, 2003). The resulting carboxyhaemoglobin molecules can no longer transport oxygen from the lungs around the body, and hence the oxygen supply to the brain and other organs is reduced. The reaction is reversible, and exposure to clean air removes most of the gas from the body with a half-life of 3–4 h. Effects are

particularly severe at tissues where partial pressures of oxygen are already low. The quantity of carboxyhaemoglobin formed depends on a variety of factors, such as, the concentration of carbon monoxide in the air, duration of exposure, temperature, health status, and the activity of the individual and metabolism of the individual exposed (Tiwary & Colls, 2009). At lower concentrations carbon monoxide may cause headache, fatigue, nausea, and, in some cases, vomiting.

In general there has been little research into the potential health effects of exposure to ambient concentrations of carbon monoxide (Godish, 2004). Few authors reported associations between exposure to carbon monoxides and health outcomes (mortality rates, cardiovascular disease, hospital admissions, prenatal development; Maynard & Waller, 1999) that should not be underestimated. Further research, including time-series studies, is needed.

4.6 Metals

Metals gained the attention of the scientific community because they are an important class of human carcinogen. Five transition metals—arsenic, cadmium, chromium VI, beryllium, and nickel—are accepted as human carcinogens in one form or another or in particular routes of exposure (IARC, 2011). Exposures to small doses of these metals (Goyer et al., 2004) can result in diffuse or spotted hyper-pigmentation of the skin, and if continued for years can produce benign skin lesions (hyperkeratosis) and cancer of the skin. Chronic exposure to low doses of cadmium (through cigarette smoking) can cause kidney tubular dysfunction and osteoporosis in susceptible populations. Lung cancer also occurs with chronic inhalation exposure of arsenic and cadmium (Goyer et al., 2004). Almost all metals that occur in the atmosphere are associated with particles. Breathing heavy metal particles can have serious health effects. Virtually all aspects of human immune system function are compromised by the inhalation of heavy metal particulates. Nevertheless, the epidemiological evidence for health effects associated with inhalation exposure to particulate metals is far from comprehensive. In most environments particulate-bound metals exist in low concentrations and it has not been fully established if those quantities are sufficient/significant to cause the adverse health effects (Dominici et al., 2007). However, some studies showed lung injuries and inflammation associated with exposure to metal particles (Hirshon et al., 2008; Prieditis & Adamson, 2002).

5. Conclusion

Road transport presents one of the paradoxes of modern society. While the volume of road transport has been continually growing in European countries, the amounts of road emissions of air pollutants continue to decline in member States of the European Union. These decreases are due to technical developments as well as the implementation of various regulations. However, despite the significant reductions of road transport exhaust emissions across Europe, there have not been proportional improvements in concentrations of the respective pollutants in ambient air.

Emissions from road transport are the primary source of health hazardous pollutants, such as nitrogen oxides and carbon monoxide, and a significant source for fine particulate pollution. Exposures to these emissions are typically non voluntary and represent serious

risks to human heath. In order to protect public health it is necessary to reduce the levels of these exposures and to do so adequately a deeper understanding of health effects is needed. Characterizing the magnitude of those exposures and quantifying the average exposure burden imposed by living near traffic are among the problems that need to be addressed.

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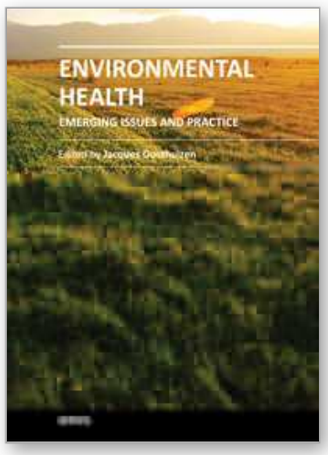

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Environmental health practitioners worldwide are frequently presented with issues that require further investigating and acting upon so that exposed populations can be protected from ill-health consequences. These environmental factors can be broadly classified according to their relation to air, water or food contamination. However, there are also work-related, occupational health exposures that need to be considered as a subset of this dynamic academic field. This book presents a review of the current practice and emerging research in the three broadly defined domains, but also provides reference for new emerging technologies, health effects associated with particular exposures and environmental justice issues. The contributing authors themselves display a range of backgrounds and they present a developing as well as a developed world perspective. This book will assist environmental health professionals to develop best practice protocols for monitoring a range of environmental exposure scenarios.

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