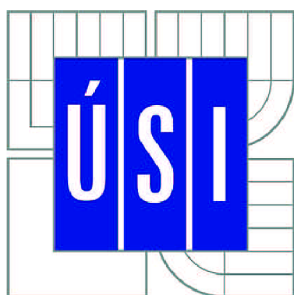# VYSOKÉ UČENÍ TECHNICKÉ V BRNĚ

BRNO UNIVERSITY OF TECHNOLOGY

ÚSTAV SOUDNÍHO INŽENÝRSTVÍ

INSTITUTE OF FORENSIC ENGINEERING

THE CONCENTRATION OF PARTICULATE MATTER IN THE AIR OF SELECTED AREAS OF THE CITY OF BRNO AND ITS HEALTH RISKS

KONCENTRACE PEVNÝCH ČÁSTIC V OVZDUŠÍ VYBRANÝCH LOKALIT MĚSTA BRNA A JEJICH ZDRAVOTNÍ RIZIKA

DIPLOMOVÁ PRÁCE
MASTER'S THESIS

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Ředitel ústavu Vám v souladu se zákonem č.111/1998 o vysokých školách a se Studijním a zkušebním řádem VUT v Brně určuje následující téma diplomové práce:

Koncentrace pevných částic v ovzduší vybraných lokalit města Brna a jejich zdravotní rizika

v anglickém jazyce:

The concentration of particulate matter in the air of selected areas of the city of Brno and its health risks

Stručná charakteristika problematiky úkolu:

Analýza dostupných literárních údájů o možném vlivu pevných částic na zdraví člověka. Analýza chemického složení pevných částic v ovzduší. Srovnání dosažených výsledků analýz s platnou legislativou. Vyhodnocení možných zdravotních rizik.

Cíle diplomové práce:

1. Analýza dostupných literárních údájů o možném vlivu pevných částic na zdraví člověka.
2. Analýza chemického složení pevných částic v ovzduší.
3. Srovnání dosažených výsledků analýz s platnou legislativou.
4. Vyhodnocení možných zdravotních rizik.

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Vedoucí diplomové práce: doc. Ing. Vladimír Adamec, CSc.

Termín odevzdání diplomové práce je stanoven časovým plánem akademického roku 2013/2014.

V Brně, dne 18.10.2013

L.S.

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    doc. Ing. Aleš Vémola, Ph.D.
    Ředitel vysokoškolského ústavu

Abstract

This thesis deals with air pollution caused by particulate matter (PM) in the city of Brno areas. The theoretical part focuses on potential sources of PM, its chemical composition, size classification and other physic-chemical properties. The next section is devoted to the health risks of PM. In this context, the work aims at the anatomy of respiratory system and the effects of PM on it. Health effects of dust pollution including the potential respiratory and cardiovascular disorders are also discussed. In experimental part, a collection of dust was carried out. The dust collection was carried out in four selected areas. Concentrations of airborne particles PM_{10} and $PM_{2.5}$ were determined, subsequently. Concentration of heavy metals and polycyclic aromatic hydrocarbons bound to dust particles was determined. Finally, the comparison between the regulatory standards and results of analysis was done as well as the health risk assessment.

Abstrakt

Tato práce se zabývá znečištěním ovzduší pevnými prachovými částicemi v lokalitách města Brna. V teoretické části je pojednáno o možných zdrojích prachových částic, o chemickém složení a velikostní klasifikaci polétavých částic a dalších fyzikálně-chemických charakteristikách. Další část je věnována zdravotním rizikům prašnosti. V této souvislosti práce pojednává o anatomii dýchacího systému a účincích prachu na něj. Jsou taktéž rozebrány zdravotní efekty prachového znečištění, včetně uvedení možných nemocí respiračního a kardiovaskulárního systému, jejichž může být prach příčinou. V praktické části byl proveden sběr prachu. Byly vybrány celkem čtyři lokality, kde byly prováděny odběry vzorků. Následně byly stanoveny koncentrace polétavých částic PM_{10} a $PM_{2.5}$. Dále byly u vybraných lokalit stanoveny koncentrace těžkých kovů a polyaromatických uhlovodíků vázaných na prachových částicích. V závěru bylo provedeno srovnání s legislativou a stanovena zdravotní rizika.

Declaration

I declare that I have worked out the diploma thesis by myself and that all the quotations from the used literary sources are accurate and complete.

Brno ………………..

…………………………………………

Student's signature

CONTENT

1   INTRODUCTION

Dust is the most widespread pollutant which human is being exposed on daily routine. The magnitude of harmful effects of dust to human is very broad. Origin, chemical properties, particle size distribution, the concentration in the atmosphere and many other factors has big influence on the evaluation of harmful effects. That is why the large legislation is on this field, including the regulations for working environment, outdoor environment as well as indoor residential areas (Hollerová, 2007). The study of dust has expanded in recent decades and there are many reviews on the importance of dust in the framework of the Earth ecosystem from a variety of disciplinary perspectives.

Dust particles can be of various origins. It is not only dust which we are being exposed in our dwellings but such particulate matter like that from streets (Joshi et al., 2009), vicinity of roads (Du et al., 2013; Liu et al., 2014; Duong and Lee, 2011; Valloto et al. 2014), iron and steel-making plants (Zhan and Guo, 2013; Hleis et al., 2013), item wind-blown dust plumes derived from dessicated soils of arid lands (Ernst, 2012; Goudie, 2014). Next peril, mainly in the case of working environment in specialized industrial fields, comes out of high concentration of dust particles in air which is associated with possibility of dust explosion. That is why many publications have been devoted to this problem (e.g. Klippel et al., 2013; Broumand and Bidabadi, 2013; Abuswer et al., 2013; Joseph, 2007; Snoeys, 2012; Amyotte et al., 2012 etc.).

Air pollution is a long-term problem in the Czech Republic and is the cause of many deaths and illnesses such as respiratory, cancer and heart diseases. The largest sources of air contamination include thermal power plants and industry, automobile traffic, local heating or waste combustion. In the 70s and 80s of the 20th century the air pollution in some industrial areas was one of the worst in Europe. After 1989, a number of steps to reduce the air pollution were introduced, especially in the energy and other industries. Therefore the reduction in air pollution happened in the case of series of substances (SO_2, NO_x, dust particles). After 2000, there was a reversal of the trend and concentrations of many pollutants rose again. Nowadays, the main problem of the air quality in the CR is, as in the case of the other countries of the European Union, suspended particles and ozone, in particular. Most of the population is exposed to these pollutants that exceed the regulatory limits. In areas with intense vehicular traffic and industry is main problem also benzo(a)pyrene pollution. The limits for NO_2 are exceeded in areas with heavy traffic. Pollution by dust particles is the very

next problem in the CR. Dust particles poses a significant risk to human health and come mainly from combustion processes in power engineering, home heating and transport. Transport causes not only direct exhalation but also whirling of dust particles (Macoun, 2009).

Epidemiological studies have shown a positive correlation between exposure to ambient particulate matter and the development and exacerbation of adverse respiratory and cardiovascular outcome (Breznan et al., 2013). Main problem remains not only in case of working environment and indoor areas but mainly in outdoor spaces as well. In fact, the atmosphere near roads as well as in urban areas may be considered to be most risky in terms of human health because over half of the global populations live in urbanized areas (Zhonggen et al., 2013).

The city of Brno is an area with deteriorated air quality as defined by the Ministry of Environment of the CR on a yearly basis. These are areas where the ambient air quality limit is exceeded in one or several pollutants. So the main effort of this work is to evaluate the data from dust collection and analysis, and appraise the concentration of particulate matter, polyaromatic hydrocarbons and other pollutants and assess their risks in terms of human health.

2   DEFINITION OF THE PROBLEM SITUATION

A problem situation is such a nonstandard state of an entity, which requires, due to objective or subjective reasons, a solution with defined aim. The solution process is not usual, so the solver has to use informational, evaluating, creative and decision-making operations and search for methods of the solution (Janíček, 2007; Janíček et al., 2013).

From the above definition point of view, the nonstandard state, in the context of this work, is increased concentration of particulate matter (PM) in the air (or generally in an environment). That means there are such amount and such kind of airborne particles, in certain locality, which can threaten the human health. This is the nonstandard situation which has to be solved due to the intersection of objective and subjective reasons. The objective reason is mainly a general endeavor to keep the population healthy without any serious health disorder. The next objective reasons are, in particular, the population mortality and the standard of living. A general effort of an individual (subject) to avoid a health disorder may be considered to be the subjective reason.

In this case the entity of our interest is a human. The nonstandard state of the entity may thus be defined as an adverse influencing of the entity from surroundings because there is increased concentration of PM, which activates and influences the entity. The necessity to solve this problem is the consequence of the objective reasons, primarily. That is the effort to reduce the amount of PM in the air and thereby preserve the health of population. It is beyond the terms of this work to solve this problem situation completely due to its wide extend, so only the analysis of current state will be carried out. This analysis may be served as a basis for implementation of appropriate steps to reduce and moderate the consequence of the above mentioned.

3   DEFINITION OF THE PROBLEM

A problem is the substantial part, of the problem situation that requires a solution, which is defined by a subject (Janíček 2007; Janíček et al., 2013).

The main problem of this work is to determine the concentration of PM in the air, what kind of dust it is and how dangerous it may be. It is important to determine size of airborne particles and chemical composition. Based on these informations it is possible to determine what particular diseases these airborne particles may cause, so the increased morbidity and mortality of population may be discovered and predicted.

4 ANALYSIS OF THE CURRENT STATE

4.1 AIRBOIRNE PARTICULATE MATTER

PM is an air pollutant consisting of a mixture of particles, that can be solid, liquid or both, are suspended in the air and represent a complex mixture of organic and inorganic substances, with particles varying in size, composition and origin (Mazzarella et al., 2007). Aerodynamic diameter of these particles mostly does not exceed 100 μm (Mateus et al., 2013). These particles are considered to be carriers of a wide range of inorganic and organic components. PM mass and composition is also highly variable in spatial-temporal terms and is strongly influenced by climatic and meteorological conditions. It can be emitted from both natural and man-made sources. Typically, PM is defined according to size or the diameter of the particles which make up a particular fraction, as this is what determines how long they will reside in the air, how far they may be transported and, in terms of health, how they will be deposited in the respiratory system. Air quality policy and emissions regulations are typically based on the mass of size fraction PM_{10} and/or $PM_{2.5}$. For these fractions are the most likely to impact human health as they are small enough to be inhaled and respired. In addition to size, PM mass and number concentrations, volatility, morphology and chemical composition (e.g. organic, metal, salt content) are critical factors to be considered in the assessment of risk. The evidence regarding the relationship between airborne PM mass exposures (i.e. $PM_{2.5}$) and patterns of cardiopulmonary morbidity and mortality is quiet solid. It is not clear, however, what chemical constituent may be particularly responsible for the observed effects (Zereiny and Wiseman, 2010).

Large amounts of dust particles are emitted also from arid soils of the Earth (500 – 1500 Mt/year), mostly located in the so called "dust belts", which extend throughout North Africa, Middle East, Central and South Asia to China. These emissions exert a considerable influence on the climate, biogeochemistry and air quality of our planet. Because of its ability to scatter and absorb radiation, dust interacts directly with incoming solar radiation. Poor urban air quality due to the arrival of dust from distant desert frequently occurs in Southern Europe, United States and Australia etc. (Rodríguez et al, 2012; Rashki et al, 2012). During the dust events an increase in mortality and in respiratory and cardiovascular diseases has been observed (Goudie, 2014).

4.1.1 Terminology

Particle aerodynamic diameter – the diameter of a sphere of density 1 $g \cdot cm^{-3}$ with the same terminal velocity due to gravitational force in calm air, as the particle, under the prevailing conditions of temperature, pressure and relative humidity;

Inhalable fraction – the mass fraction of total airborne particles which is inhaled through the nose and mouth;

Extrathoracic fraction – the mass fraction of inhaled particles failing to penetrate beyond the larynx;

Thoracic fraction – the mass fraction of inhaled particles penetrating beyond the larynx;

Tracheobronchial fraction – the mass fraction of inhaled particles penetrating beyond the larynx, but failing to penetrate to the unciliated airways;

Respirable fraction – the mass fraction of inhaled particles penetrating to the unciliated airways (BS EN 481, ČSN ISO 7708).

Aerosol – airborne particles and the gas (and vapour) mixture in which they are suspended; **Airborne dust** – finely divided matter, in solid form, dispersed in air;

Airborne particles – fine matter, in solid or liquid form, dispersed in air (ČSN EN 1540).

4.1.2 Size classification

According to United States Environmental Protection Agency (US EPA, 2013) the size of particles is directly linked to their potential for causing health problems. US EPA is concerned about particles that are 10 μm in diameter or smaller because those are the particles that generally pass through the throat and nose and enter the lungs. Once inhaled, these particles can affect the heart and lungs and cause serious health effects. The main size classification comes originally out of US EPA:

➢ Inhalable coarse particles (PM_{10}), such as those found near roadways and dusty industries, are large than 2.5 μm and smaller than 10 μm in diameter.

➢ Fine particles ($PM_{2.5}$), such as those found in smoke and haze, are 2.5 μm in diameter and smaller. These particles can be directly emitted from sources such as forest fires, or they can form when gases emitted from power plants, industries and automobiles react in the air.

However, there are also studies which are concerned with particulate matter of submicron sizes i.e. $PM_{1.0}$. Such studies were done e.g. in Italy (Vecchi et al., 2004), Germany (Wiseman and Zereini, 2014), China (Yu et al., 2012) etc. And the other works which deal even with particles smaller than 0.1 μm i.e. $PM_{0.1}$. Such works were carried out e.g. in Taiwan (Chen et al, 2010), Finland (Pakkanen et al. 2001), California (Ham and Kleeman, 2011) etc. Airborne particle size is one determinant of where particles may deposit in the respiratory tract upon inhalation, and is therefore a possible determinant of the health effect caused (Elihn et al., 2011).

Particle size is a main determinant of where in the respiratory tract the particle will come to rest when inhaled. Because of their small size, particles on the order of ~ 10 μm or less (PM_{10}) can penetrate the deepest part of the lungs such as the bronchioles or alveoli. Large particles are generally filtered in the nose and throat via cilia and mucus, but particles smaller than about 10 μm, can settle in the bronchi and lungs and cause health problems (Goudie, 2014).

4.1.3 Sources of dust

In general, dust can be derived from a number of sources ranging from natural geogenic, to biogenic and anthropogenic sources (US EPA, 2008), including forest fires, dust storms, traffic and industry, and is found both outdoors and indoors. In terms of latter, PM may be generated within the built environment or may be transported from outside via various mechanisms (Zereiny and Wiseman, 2010). Fugitive dust is an important contributor to atmospheric particulate matter. It was one kind of source type including soil dust, paved and unpaved road dust, construction dust and re-suspended dust deposited on building roofs or windowsills etc. In urban area road dust, soil dust and roof-deposited dust served as both sinks and sources for heavy metals which emitted from simplex sources types, such as vehicle wear (tires, body, brake lining etc.), industrial activities, road paint degradation, domestic heating, construction and demolition activities, etc. and then deposited on various receiving surfaces (road, topsoil, buildings and plants, etc.) (Kong et al., 2011).

The airborne particles can be divided according to source into two main categories – primary and secondary (US EPA, 2008).

Primary particles are released directly from their source, primarily by combustion, into the atmosphere. The main sources of primary PM are road transport, stationary combustion (mainly domestic coal burning) and industrial processes. The land and sea are also major

sources of primary particles, through soils carried by the wind and the generation of marine aerosol particles by bursting of air bubbles in breaking waves.
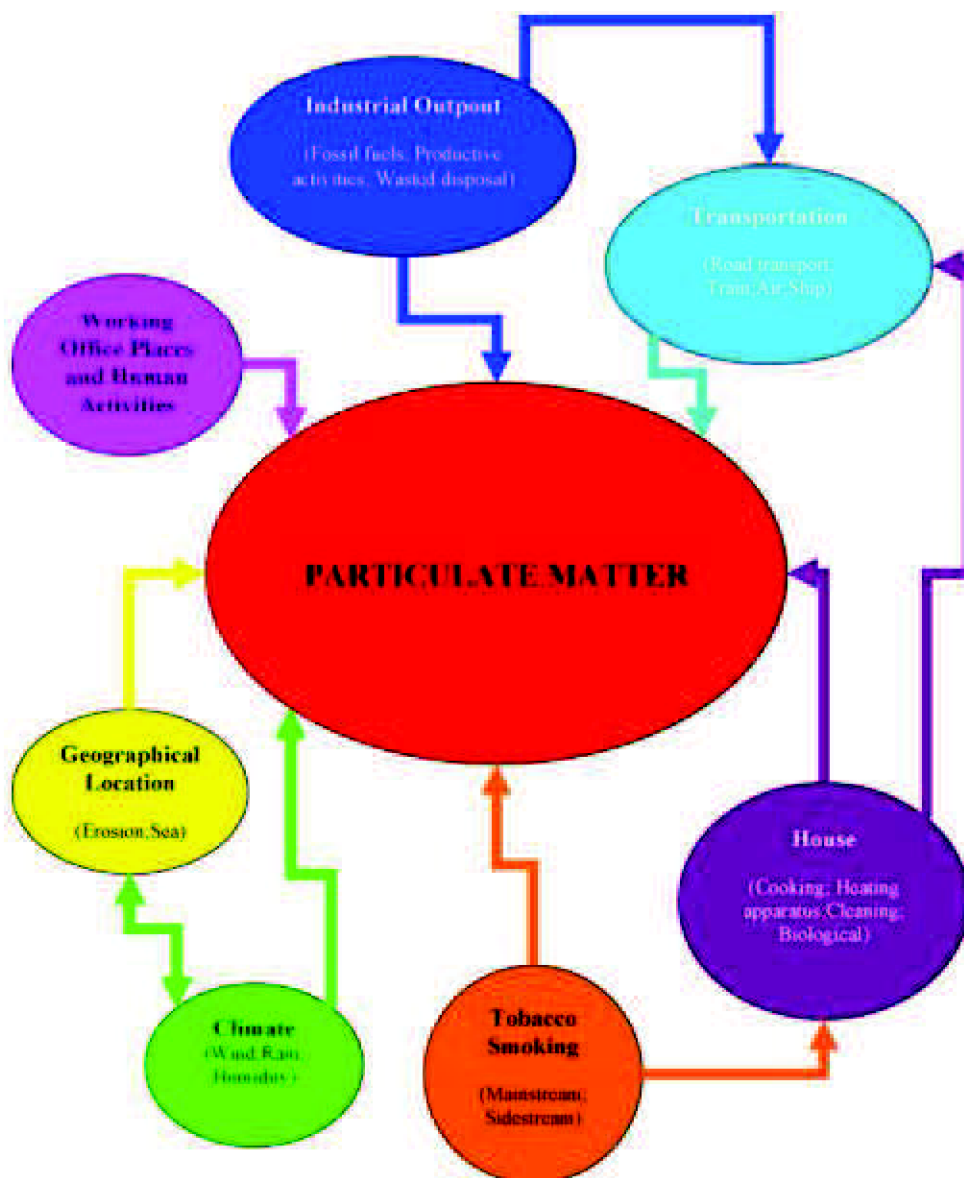
Fig. 4.1: Possible sources of PM (Polichetti et al., 2009)

Secondary particles are subsequently formed within the atmosphere as a result of chemical reactions, producing substances of low volatility, which consequently condense into solid or liquid phase, thereby becoming PM. Examples include sulphates and nitrates formed from the oxidation of sulphur dioxide (primarily from power generation and industrial combustion processes) and nitrogen dioxide (primarily derived from road transport and power generation) in the atmosphere to acids, which are then neutralised by atmospheric ammonia derived mainly from agricultural sources. Carbonaceous particulates also contain a secondary fraction formed from the oxidation of volatile organic compounds. Compared to primary

particles, the chemical processes involved in the formation of secondary ones are relatively slow and their persistence in the atmosphere is prolonged (Kelly and Fussel, 2012).
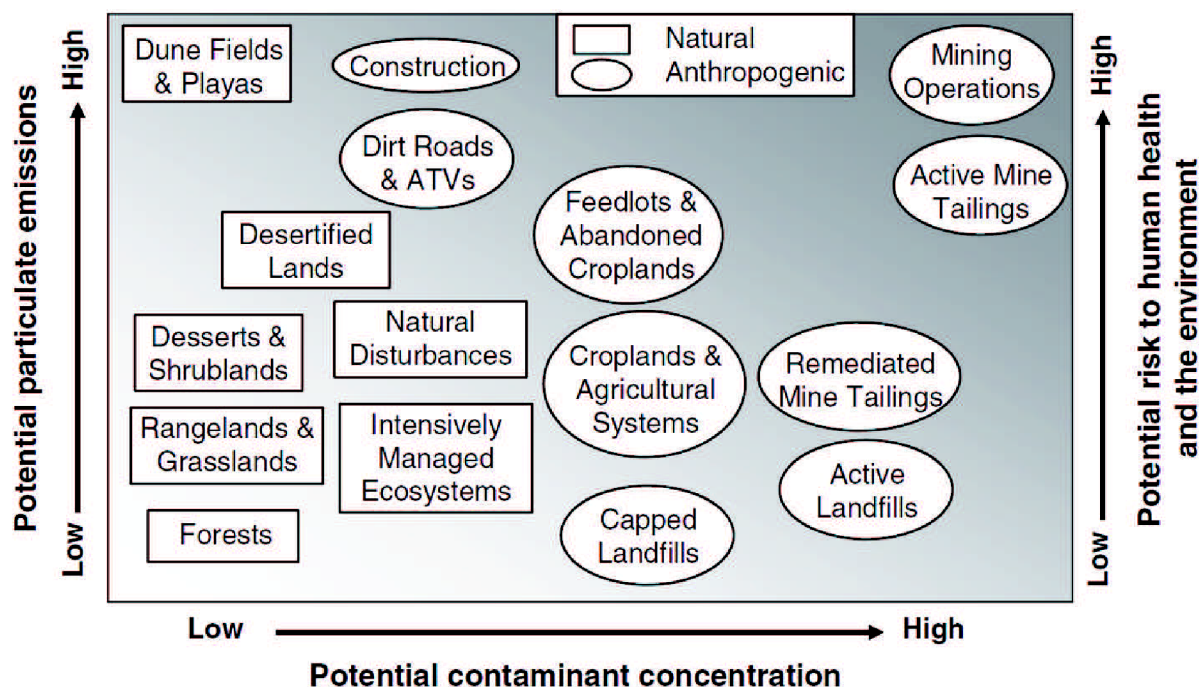
Fig. 4.2: Natural and anthropogenic sources of dust associated with relative amounts of emissions, contaminant concentration, and risk to human health and the environment (Csavina et al., 2012)

4.1.4   Chemical composition

The major components of PM are sulphates, nitrates, ammonia, sodium chloride, black carbon, mineral dust and water. It consists of a complex mixture of solid and liquid particles of organic and inorganic substances suspended in the air (WHO, 2014). The particles are carriers of other dangerous substances such as heavy metals and various organic compounds (Kopanakis et al., 2012; Saeedi et al, 2012; Klees, et al., 2013). The chemical composition, which varies depending on the source, is a major factor that contributes to the adverse health effects of PM. Several *in vitro* and *in vivo* studies reported the effects of metals, in particular transition metals, on PM induced inflammatory response and cytotoxic activities (Sun et al., 2012).

Metals, metalloids and metalic ions

Metallic cations play an important role in many biological and environmental systems. Some of them are essential for many organisms, on the other hand, many of them are toxic. Concentration of metals plays significant role as well as the ionic form of a given metal on

which bioavailability and physiological and toxicological aspects depend. For example chromium is a typical metallic element whose two ionic forms (trivalent Cr^{3+} and hexavalent Cr^{6+}) have various effects. Trivalent form of chromium is an essential part of food which is involved in digestion of saccharides, lipids and proteins and has no negative influence. Hexavalent form of chromium behaves in different way. It can cause nose irritation, sneezing, bleeding from the nose, ulcers or lung cancer (Vojtěšek et al, 2009).

Potential risks and the adverse impact of metallic pollutants in the air have been evaluated in many different respects (e.g. Zhang et al., 2014; Duong and Lee, 2011; Saffari et al., 2013; Kong et al., 2011 etc.). Potential harmful metals are important contaminants in street dust in urban areas. Their species and solubility affect significantly their mobility, bioavailability, and toxicity. Precipitation can also deposit potential harmful metals and discharge toxic components of street dust into street run-off, further polluting water bodies and food chains. High levels of potential harmful metals have adverse effects on the health of individuals. In urban regions, children might suffer more severe hazard than adults due to ingestion since children are typically hand-to-mouth active, and have much higher absorption rate of potential harmful metals from their digestion system and higher hemoglobin sensitivity to potential harmful metals (Zhang et al., 2014).

Heavy metals in street dust may originate from anthropogenic sources such as petroleum, diesel and coal combustion, as well as industrial activities and natural geochemical processes such as weathering. Heavy metals are not biodegradable and can remain in soil and dust over long periods of time. Human exposure to heavy metals in the urban environment likely occurs through food, drinks and water. Skin contact and hand-mouth contamination could be due to direct exposure to metal-contaminated dust, in particular unintentional uptake by children in playgrounds and city streets. Metal pollutants such as Cd, Cr, Ni and Pb have cumulative effects, causing growth retardation in children, kidney disease, cancer and many other adverse health effects (Saeedi et al., 2012; Li et al., 2013). Inhalation of arsenic oxides has been reported to be associated with a very large spectrum of common cancer type, including cancers of the lung, kidney, or liver.

The mechanisms of the action of metals and metalloids are not clear yet. They could act as cocarcinogens by activating pro-carcinogens in the liver. They could also act by replacing natural enzyme-associated metals and thereby inactivate their activity. Some metals and metalloids may also be mutagenic through other mechanisms, e.g. by interacting with DNA (Mena et al, 2009).

Polycyclic aromatic hydrocarbons

Polycyclic aromatic hydrocarbons (PAHs) are a group of organic compounds with multiple fused rings. Sixteen of them are classified as priority pollutants by the US EPA and they are extensively studied by environmental scientist in various matrices such as soil, dust, natural waters and sediments because of their mutagenic and carcinogenic properties (Lorenzi et al., 2011).
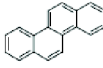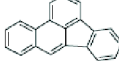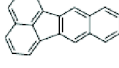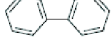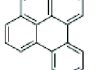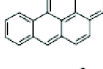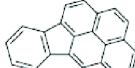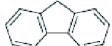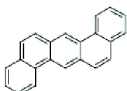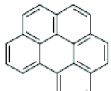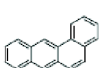
PAHs are an unavoidable byproduct of any kind of combustion, in particular incomplete combustion processes. Therefore, these substances are ubiquitous in the polluted atmospheric environment in the $ng \cdot m^{-3}$ concentration range. The gaseous state is predominant for the lighter molecular weight PAHs, while the substances with more than four rings are preferentially associated with the aerosol particles. The significance of PAH abundance is caused by the health hazard they pose upon inhalation. Some PAHs show mutagenic activity. Among atmospheric trace chemical substances, PAHs are considered to pose the highest human health risk. PAHs have been added to the list of persistent organic compounds to be regulated under the POPs protocol to the Convention on Long-range Transboundary Air Pollution (Zereiny and Wiseman, 2010).

PAHs are mainly released into the urban environment as a result of fossil fuels combustion, motor vehicle emissions, wood and waste burning, painting, asphalt pavement operations and solvent application in small industries and workshops. PAHs with large number of rings cause adverse health effects such as genetic mutations and cancer. Benzo(a)pyrene, one of the most hazardous PAHs, is categorized as a probable human carcinogenic substance (Saeedi et al, 2012). PAHs can generate reactive oxygen species, which induce lipid peroxidation and DNA damage (Koiko, et al, 2014).

PAHs can be classified in term of their source as either pyrogenic or petrogenic. The former is characterized as being mainly derived vehicle exhaust and combustion of fossil fuel, whereas petrogenic sources are usually derived from petroleum products and crude oil. In terms of PAH distribution pyrogenic sources are identified as those containing higher molecular weight PAHs i.e. those with 4–6 ring structures whereas petrogenic PAHs are identified as those containing lower molecular weight PAHs i.e. those with 3–4 ring structure. Vehicle exhausts have been reported to be a major source of pyrogenic PAHs e.g. benzo(a)anthracene, benzo(b)fluoranthene, benzo(a)pyrene etc. PAH distribution profile in urban road from both

industrial and non-industrial localities shows a predominance of pyrogenic over petrogenic PAHs (Lorenzi et al., 2011).

Table 4.1: Physico-chemical properties: saturation vapor pressure (p_{sat}), Henry's law constant (K^H), octanol-water partitioning coefficient (K_{ow}), and degradability in air as the reaction rate coefficients of the hydroxyl radical with the gaseous ($k_{g(OH)}$) and particulate phase molecules ($k_{p(OH)}$), of parent PAHs at T = 298 K (Zereiny and Wiseman, 2010)[1]

	p_{sat} (Pa)	K^H (M atm^{-1})	log K_{ow}	k_g OH (10^{-12} cm^3 molec^{-1})	k_p OH $^{(a)}$ (10^{-12} cm^3 molec^{-1} s^{-1})		p_{sat} (Pa)	K^H (M atm^{-1})	log K_{ow}	k_g OH (10^{-12} cm^3 molec^{-1})	k_p OH $^{(a)}$ (10^{-12} cm^3 molec^{-1} s^{-1})
Naphthalene (NAP)	1.0×10^1	2.3	3.37	22 (22)	5.0	Chrysene (CHR)	8.4×10^{-5}	191	5.91	(50)	5.0
Acenaphthylene (ACY)	8.9×10^{-1}	8.8	4.07	110 (75)		Benzo(b)fluoranthene (BBF9	6.7×10^{-5}	1522	6.12	(18)	
Acenaphthene (ACE)	2.9×10^{-1}	5.4	3.92	100 (67)		Benzo(k)fluoranthene (BKF)	1.3×10^{-8}	1712	6.84	(54)	3.5
Fluorene (FLN)	8.0×10^{-2}	10.4	4.18	16 (9)	3.1	Benzo(e)pyrene (BEP)	7.4×10^{-7}	3333	6.44	> 4.3 (50)	4.7
Phenanthrene (PHE)	1.5×10^{-2}	23.6	4.60	13 (13)		Benzo(a)pyrene (BAP)	7.3×10^{-7}	2188	6.50	3.5 (50)	4.1
Anthracene (ANT)	8.0×10^{-4}	17.7	4.50	17 (40)	4.4	Indeno(1,2,3-c,d)pyrene (IPY)	1.3×10^{-8}	2874	6.58	(64)	
Fluorene (FLN)	8.0×10^{-2}	10.4	4.18	16 (9)	3.1						
Phenanthrene (PHE)	1.5×10^{-2}	23.6	4.60	13 (13)		Dibenz(a,h)anthracene (DBA)	1.3×10^{-8}	7092	6.50	(50)	
Anthracene (ANT)	8.0×10^{-4}	17.7	4.50	17 (40)	4.4						
Fluoranthene (FLT)	1.2×10^{-3}	113	5.22	50 (29)		Benzo(g,h,i)perylene (BPE)	1.3×10^{-8}	3021	6.63	(87)	5.9
Pyrene (PYR)	6.0×10^{-4}	84	5.18	(50)	3.1	Benzo(a)anthracene (BAA)	2.8×10^{-5}	83	5.61		5.6

4.2   DUST AND HUMAN HEALTH RISKS

In recent decades, numerous adverse health effects have been associated with what is broadly identified as air pollution (Choi, et al, 2013; Orona et al., 2014). These effects include exacerbation of existing cardiovascular and respiratory illnesses, an increased risk of stroke

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[1] Estimated values in lack of experimental data are given in brackets;

^{a} On graphite particles

and death (Morman and Plumlee, 2013). An estimated 1.3 million deaths attributed to urban outdoor air pollution annually according to the World Health Organization (WHO). PM according to WHO affects more people than any other pollutant (WHO, 2014).

As this thesis deals with the health risks of airborne particles, it is generally known that the part of human organism which is exposed to these particles at most is the respiratory system. That is the reason why the next chapter is devoted to this field. To assess health risks which dust particles may cause it is necessary to have the basic idea about the anatomy of the respiratory system. It is also important to know how the air is in breath as it travels on its route and thus to be able to describe how the inhaled dust particles travels and have influence on the individual parts of this system.

4.2.1 Anatomy and physiology of human respiratory system

The organs of the respiratory system can be divided into two groups, or tracts. Those in the upper respiratory tract include the nose, nasal cavity, sinuses, and pharynx. Those in the lower respiratory tract include the larynx, trachea, bronchial tree, and lungs. The air enters the nasal cavity through the nose. From there it goes to the pharynx, to the larynx, to the trachea, to the bronchi (where it enters the lungs), to the bronchial tree, and finally to the tiny air sacs called alveoli. At the alveoli the second part of respiration – the exchange of gases – takes place.

The nose is covered with skin and is supported internally by muscle, bone and cartilage. Its two nostrils (external nares) provide openings through which air can enter and leave the nasal cavity. Many internal hairs guard these openings, preventing entry of large particles carried in the air.

The nasal cavity, a hollow space behind the nose, is divided medially into right and left portions by the nasal septum. This cavity is separated from the cranial cavity by the cribriform plate of the ethmoid bone and from the oral cavity by the hard palate. Nasal conchae (turbinate bones) curl out from the lateral walls of the nasal cavity on each side, dividing the cavity into passageways called the superior, middle, and inferior meatuses. They also support the mucous membrane that lines the nasal cavity and help increase its surface area. The upper posterior portion of the nasal cavity, below the cribriform plate, is slitlike, and its lining contains the olfactory receptors that provide the sense of smell. The remainder of the cavity conducts air to and from nasopharynx.
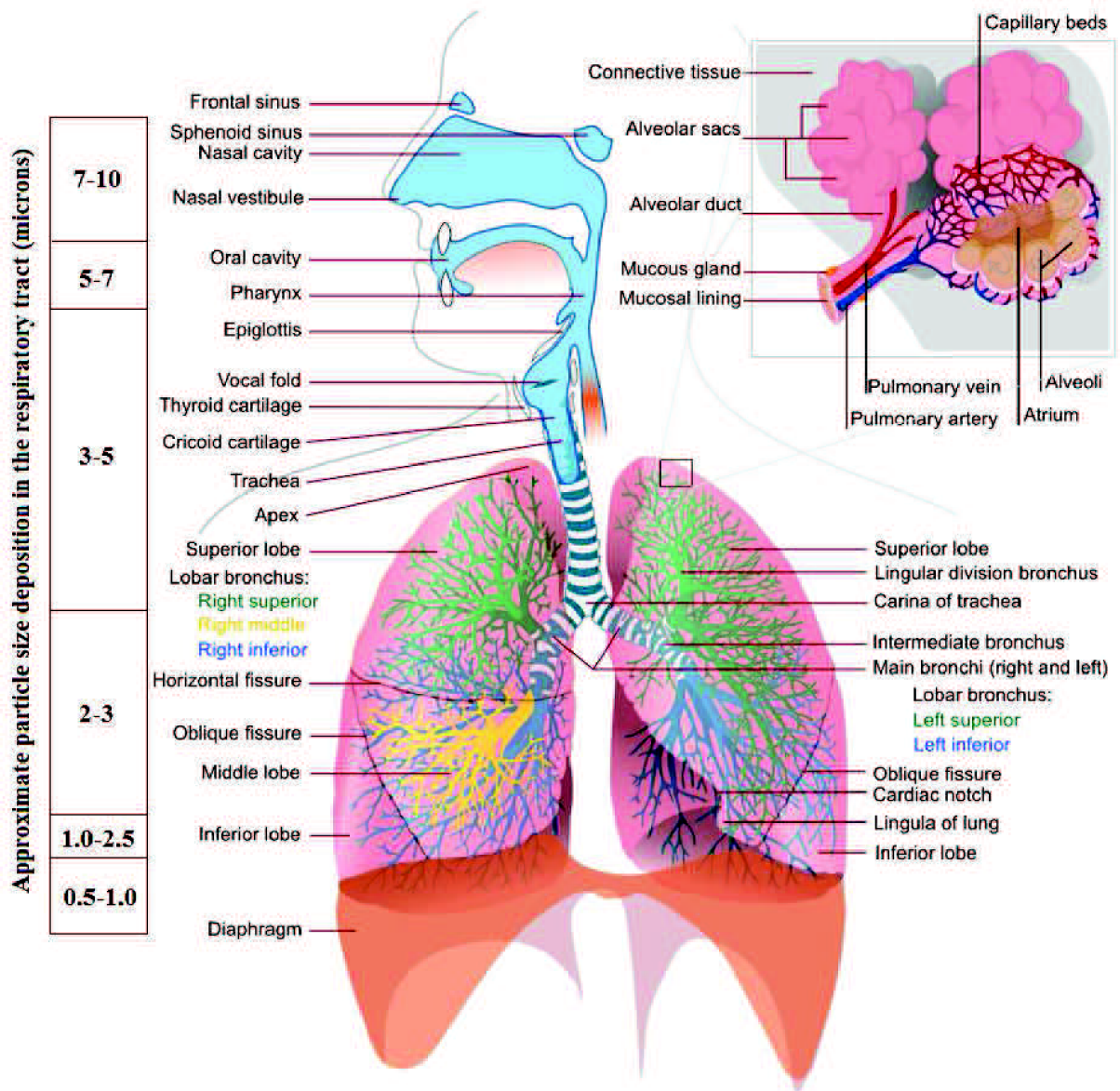
Fig. 4.3: Scheme of the human respiratory system with marked approximate particle size deposition that may occur in each section

The mucous membrane lining the nasal cavity contains pseudostratified ciliated epithelium that is rich in mucous-secreting goblet cells. It also includes an extensive network of blood vessels and normally appears pinkish. As air passes over the membrane, heat radiates from the blood and warms the air, adjusting its temperature to that of the body. In addition, evaporation of water from the mucous lining moistens the air. The sticky mucus the mucous membrane secretes entraps dust and other small particles entering with the air. As the cilia of the epithelial cells move, a thin layer of mucus and any entrapped particles are pushed toward the pharynx. When the mucus reaches the pharynx, it is swallowed. In the stomach, gastric juice destroys microorganisms in the mucus, including pathogens. Thus, the filtering from the

mucous membrane prevents particles from reaching the lower air passages as well as prevents respiratory infections.
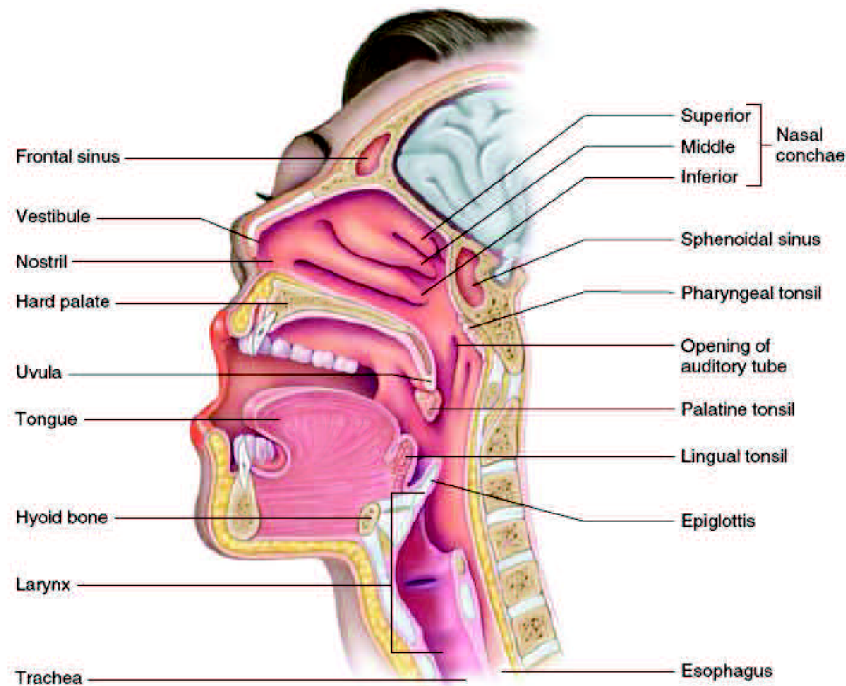
Fig. 4.4: Sagittal section showing internal anatomy (nasal septum has been removed)

Sinuses

The sinuses are air-filled spaces in the maxilliary, frontal, ethmoid and sphenoid bones of the skull, these spaces open into the nasal cavity and are lined with mucous membranes that are continuous with the lining of the nasal cavity. Consequently, mucus secretions drain from the sinuses into the nasal cavity. Membranes that are inflamed and swollen because of nasal infections or allergic reactions (sinusitis) may block this drainage, increasing pressure within a sinus and causing headache. The sinuses reduce the weight of the skull. They also serve as resonant chambers that affect the quality of the voice.

Pharynx

The pharynx (throat) is located posterior to the oral cavity and between the nasal cavity and the larynx. It is a passageway for food moving from the oral cavity to the esophagus and for air passing between the nasal cavity and the larynx. It also aids in producing the sounds of speech. The subdivisions of pharynx are the nasopharynx, oropharynx, and laryngopharynx.

The nasopharynx is located superior to the soft palate. It communicates with the nasal cavity and provides a passageway for air during breathing. The auditory tubes, which connect the pharynx with the middle ears, open through the walls of the nasopharynx. The oropharynx is posterior to the mouth. It opens posterior to the soft palate into the nasopharynx and projects downward to the upper border of the epiglottis. This portion is a passageway for food moving downward from the mouth and for air moving to and from the nasal cavity. The laryngopharynx is located just inferior to the oropharynx. It extends from the upper border of the epiglottis downward to the lower border of the cricoid cartilage of the larynx and is a passageway to the esophagus.
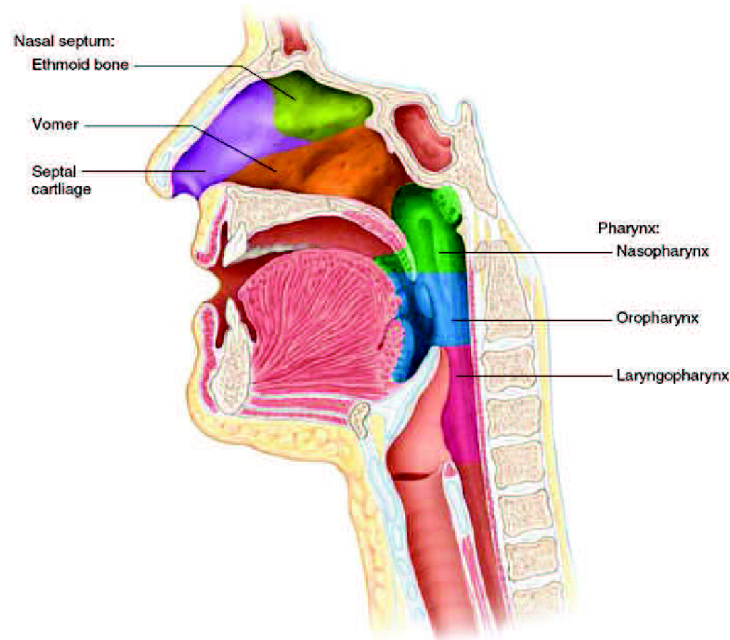
Fig. 4.5: Nasal septum and regions of the pharynx

Larynx

The larynx is an enlargement in the airway superior to the trachea and inferior to the pharynx. It is a passageway for air moving in and out of the trachea and prevents foreign object from entering the trachea. The larynx also houses the vocal cords.

The larynx is composed of a framework of muscles and cartilages bound by elastic tissue. The largest of the cartilages are the thyroid, cricoid, and epiglottic cartilages. These structures are single. The other laryngeal cartilages – the arytenoid, corniculate, and cuneiform cartilages – are paired.

Trachea

The trachea (windpipe) is a flexible cylindrical tube about 2.5 cm in diameter and 12.5 cm in length. It extends downward anterior to the esophagus and into the thoracic cavity, where it splits into right and left bronchi.

The inner wall of the trachea is lined with ciliated mucous membrane that contains many goblet cells. This membrane continues to filter the incoming air and to move entrapped particles upward into the pharynx where the mucus can be swallowed.
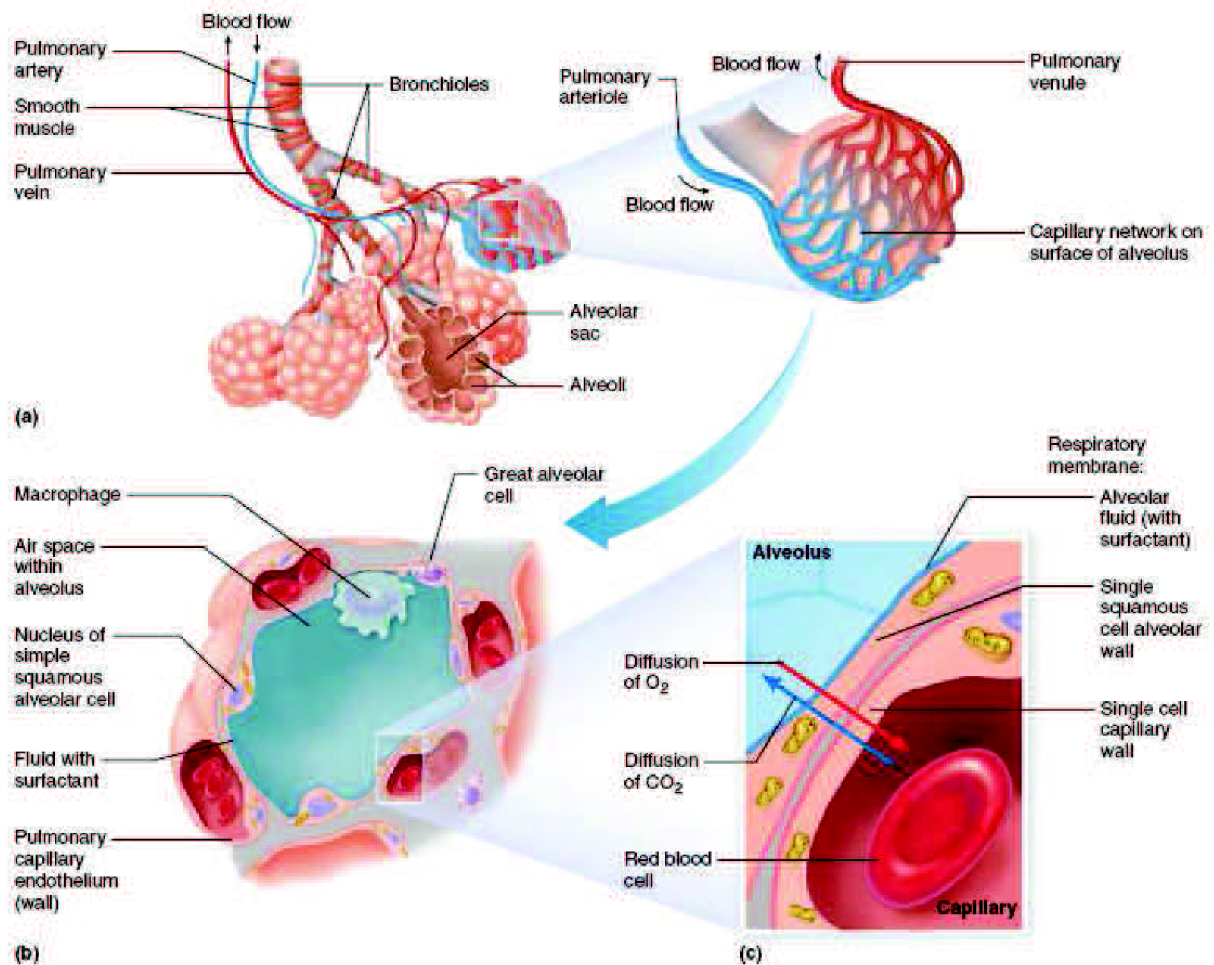
Fig. 4.6: Bronchiole, alveoli, and the respiratory membrane: (a) cluster of alveoli at the end of a bronchiole and the network of capillaries covering them, (b) cells of the alveoli, (c) respiratory membrane

Bronchial tree

The bronchial tree consists of branched airways leading from the trachea to the microscopic air sacs in the lungs. Its branches begin with the right and left primary bronchi, which arise from the trachea at the level of the fifth thoracic vertebrae. The openings of the

primary bronchi are separated by a ridge of cartilage called the carina, each bronchus, accompanied by large blood vessels, enters its respective lung.

Lungs

The lungs are soft, spongy, cone-shaped organs located in the thoracic cavity. The right and left lung are separated medially by the heart and the mediastinum, and they are enclosed by the diaphragm and the thoracic cage. Each lung occupies most of the thoracic space on its side and is suspended in the cavity by a bronchus and some large blood vessels. These tubular structures enter the lung on its medial surface through a region called the hilum. The right lung is larger than the left lung, and it is divided by fissures into three parts, called the superior, middle, and inferior lobes. The left lung is similarly divided and consists of two parts, a superior and an inferior lobe (Shier et al., 2007).

4.2.2 Respiratory exposure pathway

Particles are transported with inhaled air through the nose, an effective filter for larger particles (generally stated as > 10 µm), or the mouth, which is not. Particles may then be exhaled or deposited by coming in contact with wet airspace surfaces. Mouth breathing may occur during exercise or heavy labour allowing larger particles to enter the respiratory system.
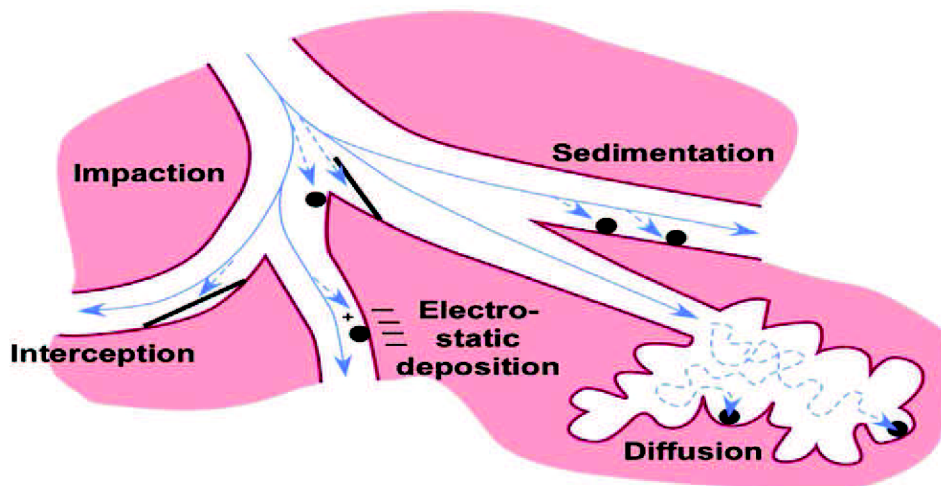
Fig. 4.7: Schematic picture of processes by which particles may be deposited in the lungs (Morman and Plumlee, 2013)

Particle deposition occurs by three mechanisms, impaction, sedimentation and Brownian diffusion. Impaction occurs when particles fail to follow airflow streamlines and contact airway walls. Impaction plays a greater role in the nasopharyngeal and upper portions of the tracheobronchial tree as airflow velocities are higher in these regions. For particles

greater than 0.5 μm in diameter, gravitational sedimentation is important and refers to the distance a particle may settle within a given time. Gravitational settling occurs predominately in the mid-size and smaller bronchioles and the alveolar region. For a particle that is less than 0.5 μm, the likelihood of it contacting the airway walls is governed by diffusional transport where collisions between gas molecules and particles cause small displacement of the particle (Morman and Plumlee, 2013).

4.2.3 Airborne particulate matter and human health disorders

Epidemiological studies have shown a positive correlation between exposure to ambient PM and the development and exacerbation of adverse respiratory and cardiovascular outcome (Sun et al., 2012; Rissler et al. 2012; Orona et al., 2014). However, increased levels of air PM have been associated also with other distant systems i.e. cerebrovascular (Leiva G et al., 2013; Martinelli et al., 2013) and nervous (Orona et al., 2014). A specific consequence of exposure to high levels particulate air pollution is increased susceptibility to infections often leading to the hospitalization of affected individuals (Breznan et al, 2013). Airborne PM contribution to health problems in different population groups, and their effect on morbidity and mortality are not well understood. The damage performed by PM_{10} and $PM_{2.5}$ to human health is manifested as mortality due to cardiac and respiratory causes, a decrease in lung capacity in children and asthmatic adults and an increase in chronic obstructive pulmonary diseases. The negative health effects caused by particle concentrations in the air depend on the pollutant (i.e. physical-chemical composition) and its concentration (the level and time of exposure) (Leiva G et al., 2013).

4.2.4 Respiratory disorders

The pathogenic effect of dust inhalation on respiratory system tissues can be attributed to the direct physical action of dust particles on the epithelium of the human airways and may be exacerbated by the toxic effects of both trace elements (including arsenic, etc.) and of biologically active compounds (bacteria, fungi, pollen, and viruses). It is likely that the populations most susceptible to suffering from the short-term effects[2] of suspended particles

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[2] The effects on health of acute exposure to air pollution, with emphasis on mortality and hospitalization, and include mostly time series analyses over a few days of exposure. Conversely, long-term effects are those which include cohort survival analyses over years of exposure, evaluate the impact of chronic exposure to air pollutants on the risk of a disease (Franchini and Mannucci, 2012).

are: (1) The elderly, due to their lower immunological capacity and the deterioration in their general health due to the ageing process, (2) subjects affected by chronic pulmonary disorders, and (3) the very young, whose lungs and airways have not fully developed yet (Goudie, 2014).

The respiratory disorders fall into three categories – infections, chronic obstructive pulmonary diseases (COPDs) and lung cancer. The first one includes cold, influenza, tuberculosis, pertussis (whooping cough) and pneumonia. The second one, COPDs, include chronic bronchitis, emphysema and asthma – the following text is devoted to these ones. For completeness, the third one includes squamous cell carcinoma, adenocarcinoma and oat cell carcinoma (Shier et al., 2007; Yang and Omaye, 2009). COPDs are the non-specific terminology commonly used to describe the spectrum of various diseases causing limitation of respiratory airflow (Yang and Omaye, 2009), i.e. asthma, chronic bronchitis, and emphysema, as mentioned above. COPD is a slowly progressive disease due to an exacerbated inflammatory process in lungs triggered by exposure to noxious particles or gases (Fortunato et al., 2014). These are described in detailed manner in subsequent text.

Allergies and asthma

An allergic reaction occurs when the human immune system reacts to a foreign substance from the outside world. The most common foreign substance may include molds, dust mites, animal dander, chemicals, foods, insect stings, etc. The immune system responds to these foreign substances that can act as potentially dangerous invaders by sending out antibodies (e.g. immunoglobulin E – IgE) to attack them. These foreign substances are referred to as allergens and individuals will experience the symptoms of an allergic reaction which includes sneezing, runny nose, sore eyes, itchy throat, eczema, and certain irritating skin conditions (Kim et al., 2013).

Scientists have characterized asthma as a special type of inflammation of the airways leading to the contraction of airway muscles, mucus production, and swelling in the airways. As the airways become overly responsive to environmental changes, the result is wheezing and coughing (Kim et al., 2013; Shier et al., 2007). Inflammation of the lining of the airways is a major factor in asthma. Inflammation is produced by the human immune system. The immune system's job is to defend our body against impurities with foreign and harmful bacteria, viruses, dust, chemical, etc. The immune system in people with asthma however overreacts by releasing many different kinds of cells and other chemicals to the airways.

These cells cause the following changes in the airways: (1) swell or inflame the inner linings of the airways with less room in the airways to transmit the air, (2) tighten the muscles surrounding the airways which narrows the airways even more, and (3) produce thick mucus from the mucus glands in the airways, which further blocks the airways (Kim et al., 2013). A person with asthma usually finds it harder to force air out of the lungs than to bring it in. This is because inspiration utilizes powerful breathing muscles, and, as they contract, the lungs expand, opening the air passages. Expiration, on the other hand, is a passive process due to elastic recoil of stretched tissues. Expiration also compresses the tissue and constricts the bronchioles, further impairing air movement through the narrowed air passages (Shier et al., 2007). Asthma and allergy have strong hereditary component and are influenced by air pollutants. It is important to note that asthma exacerbation is linked to air pollution, even at levels below regulatory standards (Yang and Omaye, 2009).

Chronic bronchitis

This disorder often results from long-term irritation of the epithelium of the bronchial tree. With the subsequent inflammation, cilia are lost and mucus is overproduced. Without the cilia escalator, mucus and debris accumulate, leading to further chronic inflammation and infections. The long-term effect is a decrease in the diameter of the bronchioles, which reduces ventilation of the alveoli. Chronic bronchitis often leads to emphysema (Shier et al., 2007).

Emphysema

Emphysema is a progressive degenerative disease that destroys many alveolar walls. As a result, clusters of small air sacs merge into larger chambers, which decrease the total surface area of the alveolar walls. At the same time, the alveolar walls lose their elasticity, and the capillary networks associated with the alveoli diminish. Because of the loss of tissue elasticity, a person with emphysema finds it increasingly difficult to force air out of the lungs. Abnormal muscular efforts are required to compensate for the lack of elastic recoil that normally contributes to expiration (Shier et al., 2007; Redlarsky and Jaworski, 2013).

4.2.5 Cardiovascular disorders

There is a link between the particulate air pollution and the deaths caused due to cardiovascular diseases such as myocardial infarction and arrhythmia. However any definite pathway that can explain the link between the two has not yet been determined. There are two

possibilities that link air pollution to heart diseases. These involve the classical pathway that explains the indirect effects mediated through pulmonary oxidative stress and inflammatory responses and the other is a direct pathway which explains the actions of pollutants on the cardiovascular system, blood and lung receptors.

The indirect effects contributing to classical pathway are less acute and occur after several hours or days of infection. Pulmonary oxidative stress/inflammation caused due to inhaled pollutants may be responsible for this, further leading to release of pro-thrombotic and inflammatory cytokines[3] into the circulation. Oxidative stress is a result of exposure to ultrafine carbon particles, diesel exhaust particles, ambient PM and cigarette smoke.
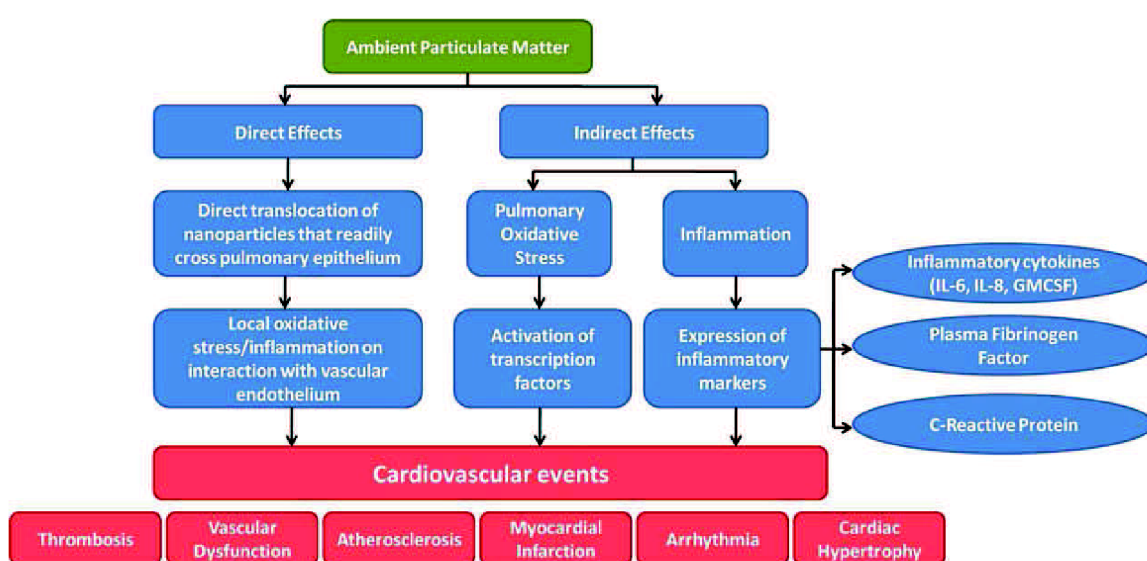
Fig. 4.8: Mechanisms by which PM leads to cardiovascular diseases

These lead to an increase in the reactive oxygen species in the heart and lung as a result of free radicals present within the PM. The size, surface area and transition metal content have been found to affect the degree of oxidative stress and the release of cytokines. Inflammation plays a substantial role in atherogenic[4] progression, alterations in endothelial[5] function and

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[3] Cytokine is a small protein released by cells that has a specific effect on the interactions between cells, on communications between cells or on the behaviour of cells (MedicineNet.com, 2012).

[4] Initiating, increasing, or accelerating atherogenesis – the process of forming atheromas, plaques in the inner lining of arteries (MedicineNet.com, 2012).

[5] Endothelium – a layer of flat cells lining the closed internal spaces of the body such as the inside of blood vessels and lymphatic vessels and the heart (MedicineNet.com, 2013).

potentially mediates acute plaque rupture. It has been found that concentrated PM produce an inflammatory response on exposure to human lung. Fine particles may lead to inflammation and oxidative stress by penetrating the alveolar epithelium.

Direct pathway involves direct translocations of inhaled fine particles or insoluble nanoparticles into the circulatory system thus affecting the cardiovascular system. Gases, ultrafine particles and nanoparticles present in the PM can readily cross the pulmonary epithelium or the lung-blood barrier owing to their particle size, charge, chemical composition and propensity to form aggregates. A possibility of particle translocation, either as naked particle or after ingestion by macrophages[6], in humans cannot be ruled out completely. Once such nanoparticles are in circulation, they lead to further deleterious effects such as local oxidative stress and inflammation on interacting with vascular endothelium. Also, they may lead to instability of atherosclerotic plaques resulting in thrombosis or even initiate cardiac arrhythmias. Other rapid responses such as myocardial infarctions may also succeed the direct effects of nanoparticle pollutants on cardiovascular system. A few major disorders are explained further.

Thrombosis

Injuries to the blood vessels activate the body's hemostatic mechanism to repair the damage and avoid loss of blood. This results in accumulation of platelets and fibrin hence forming a blood clot or thrombus. Such a thrombus formation inside the blood vessels (thrombosis) obstructs the blood flow in vessels further leading to other cardiovascular events such as ischemia. *In vitro* and *in vivo* studies show that PM can induce pro-thrombotic effects. A systemic increase in thrombotic tendency, secondary to the induction of inflammatory mediators produced in the lungs and released in the circulation or to the translocation of particles of smaller diameter from lungs into the circulation has been frequently proposed to account for the cardiac and cerebrovascular effects of particulate air pollution. It has been found that long-term exposure to particulate air pollution is associated with altered coagulation function and deep vein thrombosis risk. In addition to altering the properties of endothelial cells and platelets, particulate nanoparticles could themselves result in thrombus formation. These findings suggest that air pollution can result in increasing the risk of thrombosis, further promoting ischemic events.

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[6] Macrophage is a type of white blood cell that ingests foreign material (MedicineNet.com, 2012).

Atherosclerosis

It is a process in which deposits of fatty substances, cholesterol, cellular waste products, calcium and other substances build up in the inner lining of an artery.

Myocardial infarction

Blockage of coronary arteries due to the deposition of atherosclerotic plaque on arterial walls leads to ischemia or restricted blood supply to the heart muscles. This can cause damage or even myocyte[7] death, which may promote myocardial infarction. Air pollution has been found to trigger myocardial infarction. As myocardial infarction is linked to atherosclerosis and thrombosis, the potential mechanisms that link air pollution to myocardial infarction are similar (Shrey et al., 2011).

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[7] muscle cell

5 EXPERIMENTAL PART

5.1 SAMPLING LOCATIONS

Sampling was performed in four localities with various degree of automobile traffic. The content of selected organic and inorganic pollutants was studied as well as the concentration of PM in the air. Two localities signed as background (residential area) and two localities with high traffic load were explored. However, not all measured values were available from all location. For available data, see table 5.1 (measuring programs).
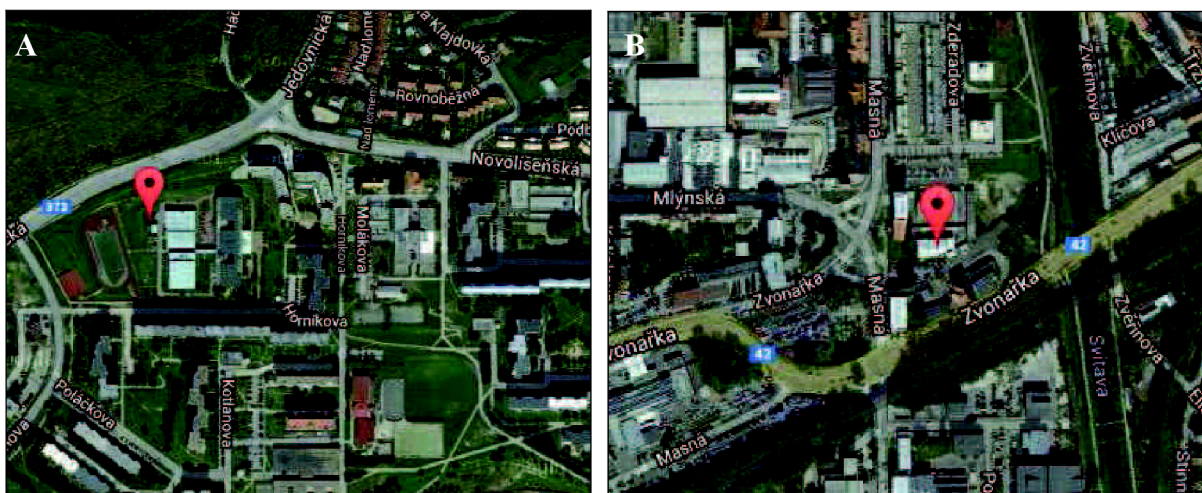
Fig. 5.1: Locations: Brno, Líšeň (A); Brno, Masná (B)

Fig. 5.2: Locations: Brno, Úvoz (A); Brno, Zvonařka (B)

The localities Masná and Líšeň are background types with lower automobile traffic load. Conversely, Úvoz is very traffic-loaded area in city centre. The position of the measuring station is near the crossroad of Údolní and Úvoz streets about 4 m from road. Similarly, Zvonařka is also highly loaded by automobile traffic. 43 thousand cars a day (10 %

haulage) passes through this street, the speed of traffic flow is about 40 km/h, the distance of measuring station from roadway and crossroad is 10 m and 50 m, respectively.

Table 5.1: Basic features of sampling locations

Location	*Líšeň*	*Masná*	*Úvoz*	*Zvonařka*
Station type	background	background	traffic	traffic
Zone type	urban	urban	urban	urban
Zone feature	residential	residential, trade	residential	trade
Latitude	49° 12′ 47.574″ (N)	49° 11′ 19.999″ (N)	49° 11′ 53.123″ (N)	49° 11′ 9.177″ (N)
Longitude	16° 40′ 40.647″ (E)	16° 37′ 36.997″ (E)	16° 35′ 37.115″ (E)	16° 36′ 49.179″ (E)
Altitude	340 m	214 m	235 m	200 m
Measuring programs (interval[8])	PM_{10} (1d/2d), $PM_{2.5}$ (1d), PAHs (1d/3d), heavy metals (1d/2d)	PM_{10} (1d), PAHs (1d/6d)	PM_{10} (1d)	PM_{10} (1h), $PM_{2.5}$ (1h)

5.2   SAMPLING AND ANALYTICAL METHODS

5.2.1   Automatic and gravimetric sampling of PM

In the case of automatic sampling, aerosol particles are caught on a filtration belt, which is made of glass fibres, using vacuum. The filtration belt is automatically unreeling between beta-emitter and Geiger-Müller counter. The difference between radiation before and after the aerosol particles are caught represents amount of dust aerosol particles on the filter. The aerosol particles are drawn in using vacuum pump with sampling head connected to top part of the analyser with 1 $m^3 \cdot h^{-1}$ flow rate. In the case of gravimetric method, the Sequential Particulate Sampler FH 95 SEQ was used for the manual mass concentration determination of suspended particulate in the ambient air. The sampled particulate is determined by balancing

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[8] d – day, h – hour

the filter before and after sampling. The sampler has a pressure and temperature compensated probe to allow a precise measuring of the air flow rate at ambient and standard conditions. The airflow rate ($1 \text{ m}^3 \cdot \text{h}^{-1}$) is controlled by an rpm carbon vane pump at a stability of about 1 %. Atmospheric concentration of particulates ($\mu g \cdot m^{-3}$) is ratio of the weight of particulates and volume of air which passed through the filter.
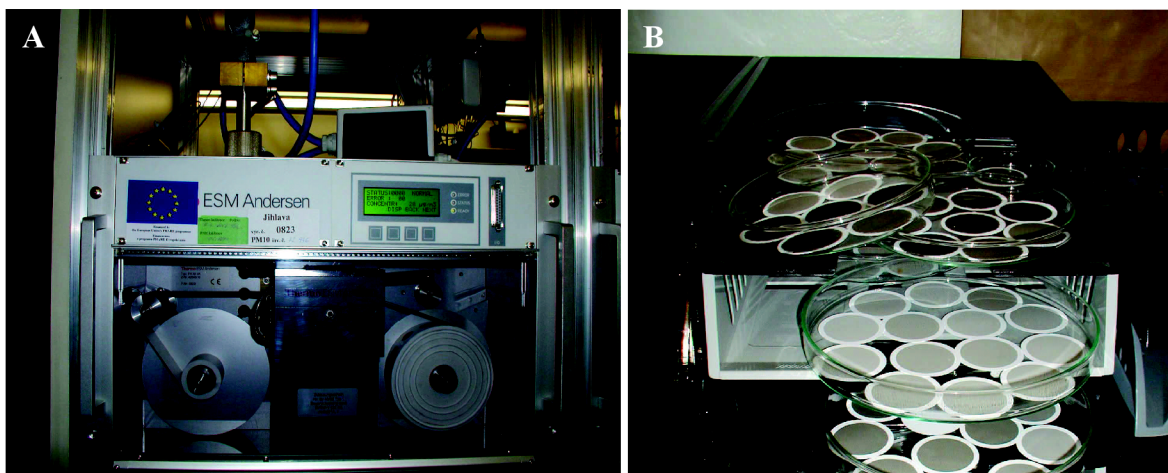
Fig. 5.3: Thermo ESM Andersen, FH 62 I-R automatic sampler (A), and stabilization of exposed filters for gravimetric method (B)

5.2.2 Determination of heavy metals

Heavy metals were determined in PM collected in Líšeň. Mn, Ni, Cu, As, Cd, Pb, V, Cr, Fe, Co, Zn, Se were determined using an inductively coupled plasma mass spectrometry (ICP-MS). A sample of aerosol particles on the filter is mineralized in microwave equipment using a solution of nitric acid and hydrogen peroxide. After the sample is transformed into the solution form, concentration of the elements can be determined.

Sample solution is drawn by a peristaltic pump into a nebulizer to form an aerosol. The aerosol is drifted by the current of argon into the inductively coupled plasma where it is vaporized and the elements are ionized. The flow of ions comes through an interface consisting of two cones into the mass spectrometer. Focused ion beam enters the quadrupole where the ions are separated according to ratio of mass and electric charge. The ions fall down on a detector to form a signal whose intensity is directly proportional to concentration of the element in the sample.

5.2.3 Determination of PAHs

PAHs were determined in samples from the background type localities (Líšeň, Masná, see table 5.2). The concentration of PAHs in Masná and Líšeň was determined using gas chromatography with mass spectrometer as detector and high performance liquid chromatography, respectively after previous extraction of sample and his purifying and concentrating using liquid chromatography column.

Table 5.2: PAHs determined in the background localities

Masná (2006 – 09/2013)	Líšeň (2009 – 06/2013)
indeno(1,2,3,c,d)pyrene (I123cdP)	indeno(1,2,3,c,d)pyrene
antracene (A)	benzo(b)fluoranthene
benzo(a)anthracene (BaA)	benzo(k)fluoranthene
benzo(a)pyrene (BaP)	benzo(a)pyrene
benzo(b)fluoranthene (BbF)	benzo(g,h,i)perylene
benzo(g,h,i)perylene (BghiPRL)	dibenzo(a,h)antracene
benzo(k)fluoranthene (BkF)	-
dibenzo(a,h)antracene (DBahA)	-
fenanthrene (Fen)	-
fluoranthene (Flu)	-
chrysene (Chry)	-

5.3 STATISTICAL ANALYSIS

Data that are available for this thesis was provided by Czech Hydrometeorological Institute. That includes data from collection and analysis of PM. The time series are expressed by month intervals. The correlation analysis was carried out which tells us if there is a dependence between single quantities. The most familiar measure of dependence between two quantities is the Pearson correlation coefficient. It is obtained by dividing the covariance of two variables by the product of their standard deviations. The Pearson correlation is 1 in the case of a perfect direct (increasing) linear relationship (correlation), −1 in the case of a perfect inverse (decreasing) linear relationship (anticorrelation) and some value between −1 and 1 in all other cases, indicating the degree of linear dependence between the variables. As it approaches to zero there is less of a relationship (closer to uncorrelated). The closer the coefficient is to either −1 or 1, the stronger the correlation between the variables. If the

variables are independent, Pearson correlation coefficient equals to zero, but the converse is not true because the correlation coefficient detects only linear dependencies between two variables (Meloun and Militký, 2004). Correlation coefficients were determined using MS Excel.

5.4   HEALTH RISK ASSESSMENT

Health effects of pollutants on living organism can be generally divided into short-term (acute) and long-term (chronic). Duration of exposure to the pollutant in the case of acute risk is assumed to be for one hour, in some cases it can be longer (several hours to one day). Chronic risk is assessed from long (up to several years) exposure using the average ambient concentration of the pollutant.

5.4.1   Substances with non-carcinogenic (treshold) effect

The risk assessment for these substances is based on the idea that their effect can be found on the target organism from a certain threshold concentration. So it is necessary to find the upper limit of the level of exposure that will be tolerated by the organism i.e. to quantify the relation dose-response.

Inhalation toxic risk (chronic or acute) can be calculated as the ratio of actually measured concentration of the pollutant C_{inh} and inhalation reference concentration RfC_i (amount of pollutant per unit volume of the air, which can be considered to be safe, in the case of selected type of exposure i.e. acute or chronic). It is expressed as a dimensionless coefficient of toxic hazard HQ.

$$HQ_i = \frac{C_{inh}}{RfC_i} \tag{1}$$

For the risk assessment of all major toxic substances monitored in the environment is applied additivity principle. That means that the total value of HQ, which is known as a toxic hazard index HI, is the sum of all partial HQ_i.

$$HI = \sum_{i=1}^{n} HQ_i \tag{2}$$

The result is a dimensionless coefficient having a value smaller or greater than one. The toxic hazards are considered to be small if the value of HI is significantly smaller than one. If the

HI approaches to one, it is still acceptable, but if the value exceeds one, the health risk increases depending on *HI*.

5.4.2   Substances with carcinogenic (non-treshold) effect

Risk assessment of carcinogenesis is difficult and complicated task of determining risks. In case of carcinogenic substances we take into account situations that the harmful effects may occur in any doses different from zero due to the fact that this process is very slow and time of exposure is counted in decades. The existence of threshold concentrations is not assumed and the risk assessment is based on linear relationship between dose and effect (with increasing dose the probability of occurrence of an undesirable effect increases). Results calculated in this way indicate the maximum risk i.e. the risk assuming lifetime exposure to the substance.

The result of the procedure for determining exposure at risk assessment is estimation of the absorbed dose. The procedure for calculating the daily intake of the substance is based on relation (3). Using this relation the average daily dose of a pollutant, that is the human body able to take in one day, can be calculated.

$$LADD_i = \frac{CA \times IR \times ET \times EF \times ED}{BW \times AT} \tag{3}$$

where $LADD_i$ is lifetime average daily intake of the pollutant by inhalation ($mg \cdot kg^{-1} \cdot day^{-1}$), CA is concentration of the pollutant in the air ($mg \cdot m^{-3}$), IR is a volume of air inhaled per day ($m^3 \cdot h^{-1}$), ET exposure time ($h \cdot day^{-1}$), EF exposure frequency ($day \cdot year^{-1}$), ED duration of exposure (year), BW body weight of the individual (kg), AT average exposure time (day).

For lifetime exposure, which is especially important for determination of exposure to carcinogenic contaminants, it is necessary to calculate the lifetime exposure of the individual (*LADD*). To take into account lifelong non-threshold carcinogenic effect of a pollutant, the average exposure time is calculated as $AT = 365 \cdot ED$ which equals to 25550 days. Duration of exposure in this relationship equals to 70 years. The calculated *LADD* value expresses the level of average lifetime exposure which is further used to quantify risk – estimating the probability of malignancy caused by the pollutant, i.e. *ILCR* (individual lifetime cancer risk, see equation 4) (Adamec, 2007; Pokorný, 2009).

5.4.3 Exposure scenario for carcinogenic risk assessment

Lifetime exposure scenario to outdoor air in the place of living of the population is shown in table 5.3. It should be taken into account that this scenario is based on statistical data of the average American population whose habits may slightly vary from the Czech population. Total inhalation rate is based on the assumption of distribution of inhalation rates depending on outdoor activity. In the case of average population it is 15 % of rest, 65 % of light load and 20 % medium load. The overall inhalation rate of child population is attributed to 5 % of rest, 80 % light load and 15 % of medium load. It is calculated for 350 days of stay in the place of exposure and a change of the environment is assumed for 15 days a year (holiday). It is necessary to emphasize that *LADD* is calculated for outdoor activities only so the zero exposition is assumed in the indoor environment. The health risk is, for simplicity, assessed for ideal average population without distinguishing the exposure data for male and female (Adamec, 2007).

Table 5.3: Basic exposure scenario of a standard person and a child under age of 14 years

Basic exposure data	standard person	child 0 – 14 years
Age (years)	70	14
Weight (kg)	70	32
Duration of exposure (years)	30	14
Frequency (days·year^{-1})	350	350
Exposure time (h·day^{-1})	3	3
Inhalation rate (m^3·hod^{-1})	0.83	3.2

5.4.4 Quantification of carcinogenic risk

An estimation of lifelong effect of the pollutant is used for health risk assessment of exposure to carcinogenic substances. In the case of short-term exposure, the recalculation on the total estimated lifetime exposure of the individual must be done. This recalculation is made using *LADD*. Lifetime exposure risk of the individual (*ILCR* – individual lifetime cancer risk) is then calculated according to following relation:

$$ILCR = 1 - e^{(-ICPF \cdot LADD)} \tag{4}$$

where ICPF is inhalation cancer potency factor (table 5.4). The risk calculated using this relation is considered to be theoretical increase of probability of cancer occurrence above the general population average.

Table 5.4: Cancer potency factors for selected substances (Budroe et al., 2011)

Noxious substance	BaP	As	Cd	Ni	Pb
ICPF [mg·kg^{-1}·day^{-1}]	3.9	12	15	0.91	0.042

In the risk estimate model (equation 4), an upper boundary limit of the risk cancer potency (*IPCF*) is used so the calculated *ILCR* value is the upper limit of the estimate. The real risk is likely lower. An *ILCR* value equaled to $1 \cdot 10^{-6}$ is considered to be acceptable level of carcinogenic risk. This value may be interpreted as the probability of malignancy due to harmful effects of a substance, which is over normal occurrence in the case of one man in a million, in an area with over than 100 exposed people.

5.4.5 Conception of the PAHs carcinogenic potential

For the evaluation of carcinogenic effect of PAHs, it is necessary to take into account various carcinogenic potential of single polyaromatic compounds in the mixture. Therefore the total concentration of PAHs is converted to concentration expressed as $\sum TEQ_{BaP}$ where individual polyaromatic compounds are transformed using toxic equivalency factors (TEF). The TEF value is proportional to multiple of carcinogenic potential due to BaP and thus obtained values are summed. Thus the mixture of PAHs is converted to hypothetical concentration of BaP with its carcinogenic potential. For the values of toxic factors TEF, see table 5.6 (Jung et al., 2010).

Table 5.6: TEF values for individual polyaromatic compounds

PAH	I123cdP	A	BaA	BaP	BbF	BghiPRL	BkF	DBahA	Fen	Flu	Chry
TEF	0.1	0.01	0.1	1	0.1	0.01	0.1	5	0.001	0.001	0.01

5.4.6 Substances with threshold and non-threshold effects

Substances, which have defined *RfC* values, exhibit systemic toxic effects. That means they can affect some of the target organs of human body. Substances, which have defined reference values ICPF, exhibit non-threshold effects. Pollutants such as arsenic, cadmium and other exhibit both of these types of effects to the human organism. Table 5.7 shows reference concentration (called also reference exposure level *REL*) of metals whose health risks are

dealt with in this work. The values of *RfC* depends on the source, some values may slightly vary (e.g. in US EPA and CAL EPA databases).

Table 5.7: Reference concentration of selected toxic metals (OEHHA, 2011)

Noxious substance	As	Cd	Ni	Pb
RfC [ng·m^{-3}]	30	20	50	500

5.4.7 Health risk quantification of exposure to PM$_x$

Long-term elevated concentrations of suspended particles contribute to occurrence of various symptoms of respiratory deterioration, increased morbidity and mortality. Mortality is often used to illustrate negative impacts of suspended particles.

For the risk assessment of long term exposure to PM, the conclusions of American Cancer Society, which are also recommended by WHO, were used. The authors concluded that the increase in average annual concentration of PM$_{2.5}$ by 10 µg·m^{-3} leads to 6% increase in total mortality of the exposed population. This relationship is modified for PM$_{10}$ where the increase in annual concentration by 10 µg·m^{-3} leads to 3% increase in total mortality of exposed population. The base concentration for PM$_{10}$ is 20 µg·m^{-3} (recommended by WHO), at which mortality should not increase with 95% probability (WHO, 2006). The maximal increase in mortality can be calculated according to following relation:

$$I_{M\%} = \frac{I_{MB\%}(C_{MAX} - C_{BASE})}{C_{INCR}} \qquad (5)$$

where $I_{M\%}$ is total maximal increase in mortality of exposed population, C_{MAX} is maximal annual concentration of PM$_x$, C_{BASE} is base concentration (it equals to 20 µg·m^{-3} and 10 µg·m^{-3} for PM$_{10}$ and PM$_{2.5}$, respectively), $I_{MB\%}$ (it equals to 3 % and 6 % for PM$_{10}$ and PM$_{2.5}$, respectively) is the increase in total mortality if the C_{BASE} is increased by C_{INCR} (10 µg·m^{-3}).

5.4.8 Legislation

Upper and lower bounds for assessing the level of pollution for the protection of ecosystems and vegetation for ground-level ozone, expressed as an exposure index AOT40, SO$_2$ and NO$_x$ are determined by Clean Air Act No. 201/2012 Coll. And Decree No 330/2012 Coll. about how to assess and evaluate the level of contamination, the extent of informing the

public about the level of pollution in smog situations. Table 5.8 shows upper limits of selected pollutants. In the case PM_{10}, it is distinguished between annual average and 24-hours average. The upper limit of 24-hours average equals to 50 $\mu g \cdot m^{-3}$ and it can be exceeded no more than 35 times a year.

Table 5.8: Air pollution limits - annual averages (Air Act No. 201/2012 Coll.)

Substance	PM_{10} (annual)	PM_{10} (24-h)	$PM_{2.5}$	BaP	As	Cd	Ni	Pb
Limit [$\mu g \cdot m^{-3}$]	40	50 / 35×/year	25	0.001	0.006	0.005	0.020	0.500

6 RESULTS AND DISCUSSION

6.1 PM CONCENTRATION

The charts of the development of PM concentrations are based on month average values. Figure 6.1A shows development of PM_{10} concentrations in study areas during the years 2009-2013. The highest concentration of PM_{10} is observable in Zvonařka locality and Úvoz which is reasonable due to the high traffic load. All curves have similar shape and are characteristic by downward trend. The highest values are observable at the beginning (2009) and as time goes by, the concentration of PM_{10} decreases (e.g. from about 70 $\mu g \cdot m^{-3}$ in 2009 to about 40 $\mu g \cdot m^{-3}$ in Zvonařka in 2013).
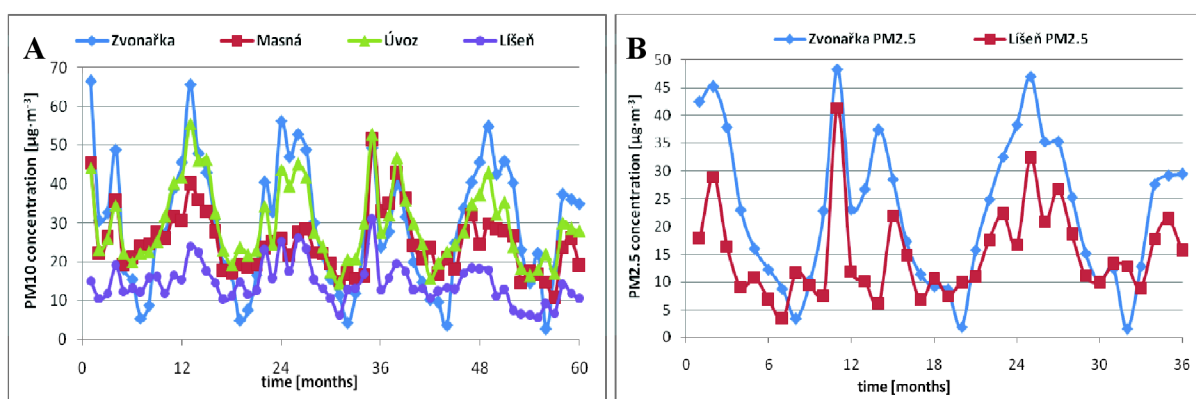
Fig. 6.1: Concentration of PM_{10} in years 2009–2013 (A) and $PM_{2.5}$ in years 2011–2013 (B): comparison of traffic and residential areas

At figure 6.1B is shown comparison of $PM_{2.5}$ between traffic (Zvonařka) and background (Líšeň) area. The difference between the concentrations (lower amount of PM in background area) is observable again. Compared to the amount of PM_{10}, the concentration of $PM_{2.5}$ is almost at the same level or slightly lower (figure 6.2) in the case of traffic area. In the case of background locality, the concentration of PM_{10} and $PM_{2.5}$ is alternating or $PM_{2.5}$ is slightly elevated.
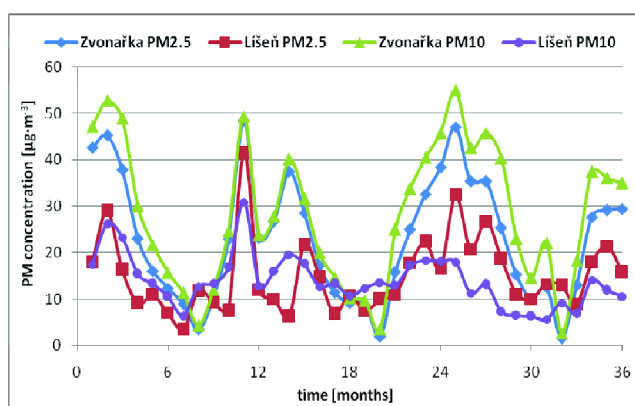
Fig. 6.2: Comparison of PM_{10} and $PM_{2.5}$ in traffic and background area 2011–2013

For all localities is typical increased concentration of PM in summer period compared to those in winter period. That is mainly caused by higher amount of combustion wastes which are released due to local heating which is in winter at high level. Moreover, it can be assumed that there is lower level of vehicular traffic in summer, due to holiday.

Table 6.1: Concentration of PM$_{10}$ – annual average / maximum / number of exceeded daily concentrations [µg·m^{-3}]

	Líšeň	**Masná**	**Úvoz**	**Zvonařka**
2008	-	33.50 / 147.3 / 39	44.00 / 174 / 106	34.62 / 146 / 73
2009	24.05 / 68 / 7	33.51 / 96 / 45	30.18 / 86 / 35	34.97 / 152.8 / 68
2010	27.14 / 97 / 13	31.39 / 78 / 26	34.46 / 149 / 59	35.68 / 170.9 / 75
2011	27.31 / 101 / 23	29.49 / 148 / 24	30.74 / 110 / 45	31.22 / 126.6 / 59
2012	24.16 / 84 / 15	33.35 / 103 / 30	30.31 / 111 / 33	28.72 / 120.1 / 44
2013	22.42 / 107 / 10	27.19 / 56 / 5	27.16 / 123 / 19	35.57 / 138.9 / 69

Table 6.1 shows annual averages of concentrations of PM$_{10}$, the highest value in the year and how many times the PM$_{10}$ concentration exceeded the daily upper limit which equals to 50 µg·m^{-3} 35 times a year (see table 5.8). The annual averages are not exceeded in most cases (except Úvoz in 2008), however, the number of days which is exceeded daily concentration is vast and in some cases the number of days is exceeded even more than two times. The highest daily values reach more three times of the limit. In the cases of background localities, the limits were exceeded in two cases, only.

Table 6.2: Concentration of PM$_{2.5}$ – annual average/maximum [µg·m^{-3}]

	2008	**2009**	**2010**	**2011**	**2012**	**2013**
Líšeň	-	-	-	19.63/127	17.64/90	18.63/100
Zvonařka	26.80/109.2	28.54/137.3	30.45/161.8	26.86/115.3	24.01/117.7	26.90/124.5

Table 6.2 shows the same values as table 6.1 except of the number of exceedances as these are required by Clean Air Act No. 201/2012 Coll. only in the case of PM$_{10}$. The upper limit for PM$_{2.5}$ amounts to 25 µg·m^{-3}. This value was exceeded in Zvonařka, only, but not in too significant manner.

6.2 PAHS CONCENTRATION

Concentration of PAHs was determined in Masná and Líšeň background localities. The series are not the same in length because of the data was not available. Figure 6A shows concentration of sum of eleven PAHs as BaP. Figure 6B compares PAHs situation between Masná and Líšeň as sum of six PAHs (see table 5.2). It can be seen that in Masná is more than 10 times higher PAHs concentration. Masná location is situated near highly loaded road and probably that is why the high concentrations of PAHs are here compared to Líšeň.
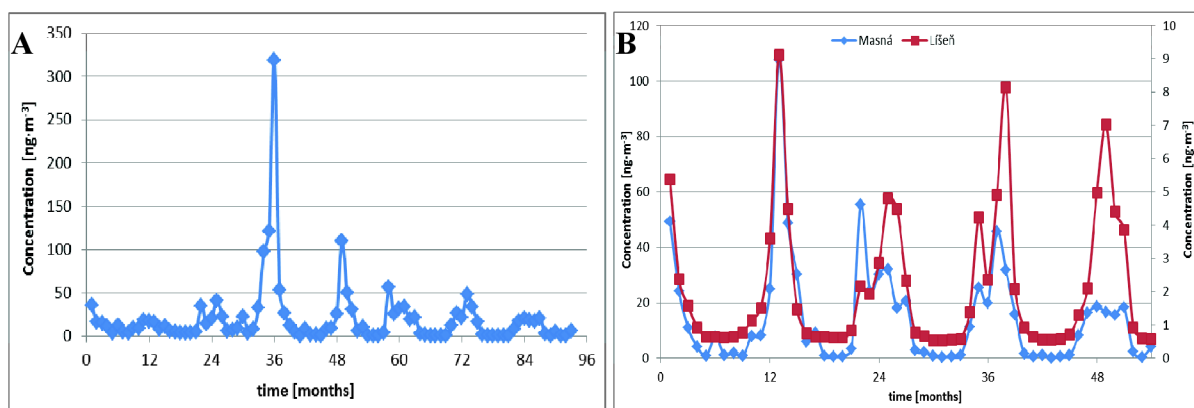
Fig. 6.3: Sum of 11 PAHs as BaP - Masná 2006–09/2013 (A) and sum of 6 PAHs for comparison of Masná and Líšeň 2009–06/2013 (B)

From figure 6.3 is also obvious concentration increase during winter. Conversely, the lowest concentrations are observed during summer. It may be caused by lower level of automobile traffic in Brno during holiday, but mainly due to lowest or no local heating in summer period.

Table 6.3 shows concentration of BaP, its annual average values and the highest values. The upper limit for BaP annual average concentration amounts to $1 \ ng \cdot m^{-3}$ so it is obvious that these are exceeded in Masná locality. The highest value exceeds the upper limit even more than 27 times (Masná, 2012).

Table 6.3: Concentration of BaP – annual average / maximum [ng·m^{-3}]

	2009	2010	2011	2012
Líšeň	0.53 / 2.8	0.80 / 6.0	0.73 / 5.6	0.97 / 7.7
Masná	1.21 / 9.7	1.24 / 6.6	1.10 / 5.7	1.22 / 27.7

6.3   HEAVY METALS CONCENTRATION

Concentration of heavy metals was determined in PM from Líšeň. Figure 6.4 shows concentration of selected metals. Nickel, arsenic, cadmium and lead were selected because of their impact on human health.
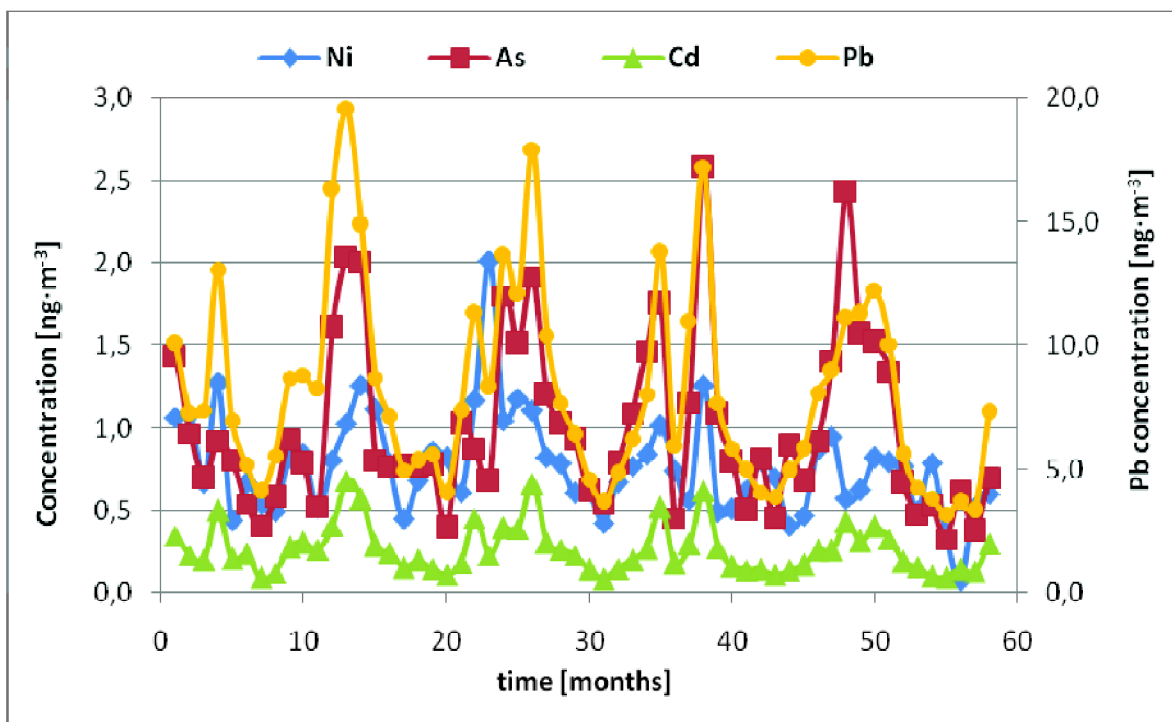
Fig. 6.4: Concentration of selected heavy metals during 2009–2013

Table 6.4 displays annual mean concentrations of selected heavy metals. Compared to legislation limits (table 5.8), we can see the resulting values of metals average concentration are not exceeding. In some cases, the daily values are exceeded but the legislation does not refer to daily concentrations but only to annual and these are not exceeded. Nevertheless, the health risks of these substances were assessed as these are non-threshold and they may cause harmful effects even at very low concentrations.

Table 6.4: Concentration of toxic metals – annual average / maximum [ng·m^{-3}]

	2009	**2010**	**2011**	**2012**
As	0.82 / 5.6	1.06 / 5.6	1.12 / 6.5	1.14 / 8.6
Cd	0.26 / 2.0	0.31 / 2.2	0.29 / 2.9	0.25 / 2.4
Ni	0.75 / 10.5	0.99 / 29.5	0.81 / 3.1	0.68 / 2.7
Pb	8.27 / 7.7	9.30 / 68.7	8.61 / 47.2	7.78 / 51.4

6.4 CORRELATION ANALYSIS

The correlation analysis was carried out only for Líšeň because there are available complex data i.e. values of PM_{10}, $PM_{2.5}$, PAHs and metals concentration and therefore it is possible to evaluate the correlation coefficient between these quantities. The concentration of metals is a sum of monthly average of Mn, Ni, Cu, As, Cd, Pb, V, Cr, Fe, Co, Zn and Se.

Figure 6.5 shows comparison of all quantities together. A similar course of the curves can be seen, so the quantities are in correlation between each other, which following figures prove. Figure 6.6 shows a correlation between content of PAHs and PM. A positive correlation between these quantities can be seen. That means if the PM concentration increases, the concentration of PAHs increases, too.
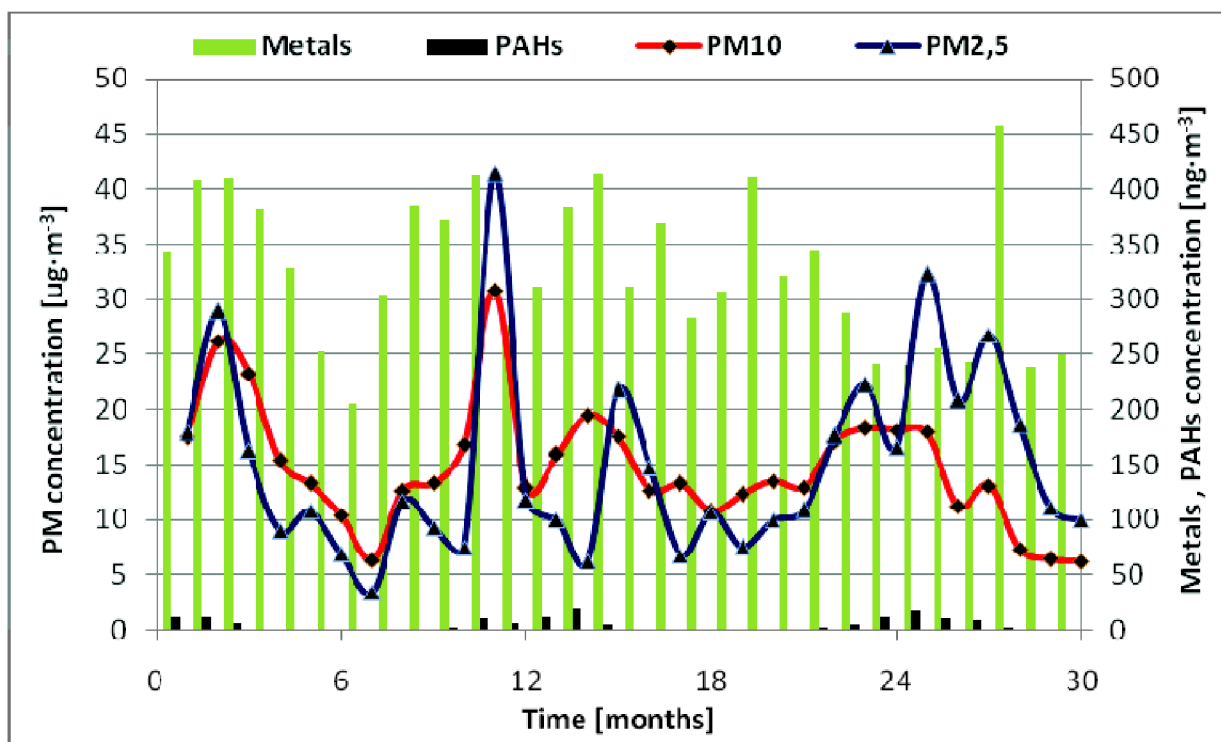
Fig. 6.5: Comparison of measured values in Líšeň area: 2011–6/2013

The same may be said for the dependences of metals on PM (figure 6.7). Lower correlation can be seen in the case of metal-$PM_{2.5}$ dependence which has smaller slope of the regression line and hence lower value of correlation coefficient which equals to 0.143 (see table 10). Figure 6.8 shows positive and correlation between PM_{10} and $PM_{2.5}$. This proves value of correlation coefficient which is highest of all (0.628). On the contrary, the scatter plot which represents dependence of PAHs on metals shows zero correlation. The value of

correlation coefficient equals to 0.003 and the regression line is of constant course. So it may be said that the amount of PAHs does not depend on concentration of metals.
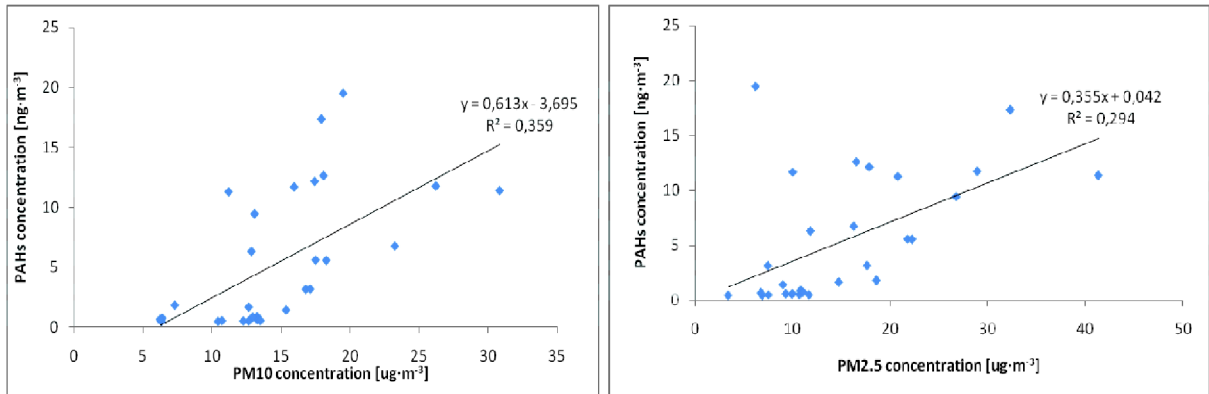
Fig. 6.6: Dependence of PAHs concentration on PM concentrations
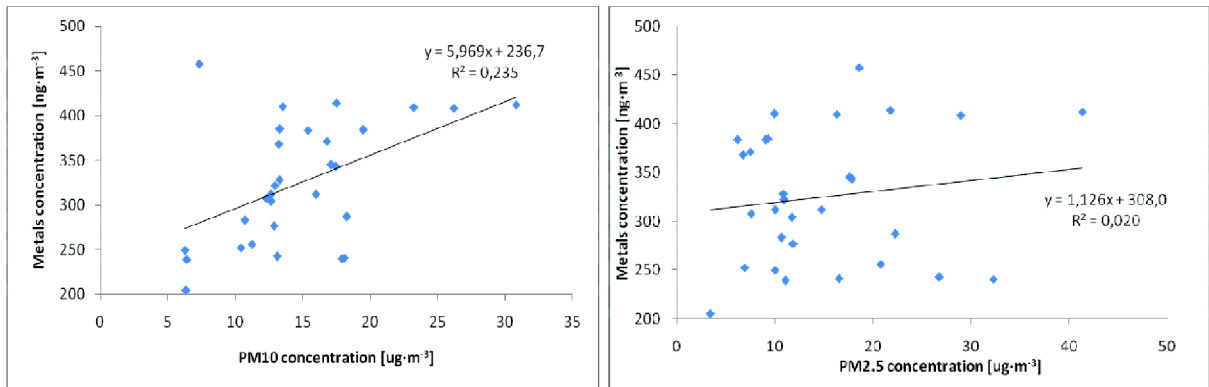
Fig. 6.7: Dependence of heavy metals concentration on PM concentrations
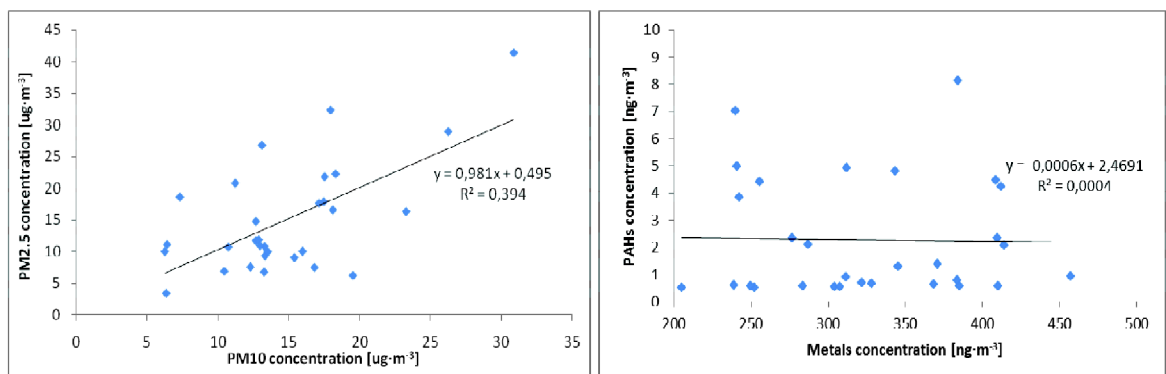
Fig. 6.8: Dependence of concentration of PM$_{2.5}$ on PM$_{10}$ concentration and PAHs concentration on heavy metals concentration

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Table 6.5: Correlation matrix

	PAHs	PM$_{10}$	PM$_{2.5}$	heavy metals
PAHs	1	0.600	0.543	0.003
PM$_{10}$	0.600	1	0.628	0.485
PM$_{2.5}$	0.543	0.628	1	0.143
heavy metals	0.003	0.485	0.143	1

6.5 RESULTS OF HEALTH RISK ANALYSIS

6.5.1 Estimate of health risks of systemic pollutants

Average annual concentrations of pollutants were used for the calculation of hazard quotient. The direct inhalation of the pollutant from the air was assumed. The data from long-term collection of dust were available and hence the concentrations of toxic metals which were analyzed in fraction of airborne dust. Therefore the health risks of their chronic systemic effect were assessed.

Table 6.6: Evaluation of chronic health risks due to exposure to toxic metals in Líšeň, 2012

Pollutant	C [ng·m^{-3}][9]	RfC [ng·m^{-3}]	HQ [-]	Legislation limit 201/2012 Coll. [ng·m^{-3}]
As	1.14	30	0.038	6
Cd	0.25	20	0.012	5
Ni	0.68	50	0.014	20
Pb	7.79	500	0.016	500

Table 6.6 shows evaluation of chronic health risk caused by toxic metals. It is shown in the case of year 2012. For the other values of hazard quotient, see figure 6.9. Table 6.7 contains calculated values of hazard index for selected organs of human organism (2012). The maximum value of hazard index does not exceed 0.054. As mentioned in chapter 5.4.1, the toxic hazards are considered to be small if the value of *HI* is significantly smaller than one. If the *HI* approaches to one, it is still acceptable, but if the value exceeds one, the health risk increases depending on *HI*. The value 0.054 may be considered as very small because the health risk increases if it exceeds one. So in this case the health risk is negligible and that is

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[9] Anual average concentration

why the calculated values are not displayed for the other years because those values are also very small even negligible which can be estimated from low values of *HQ*.
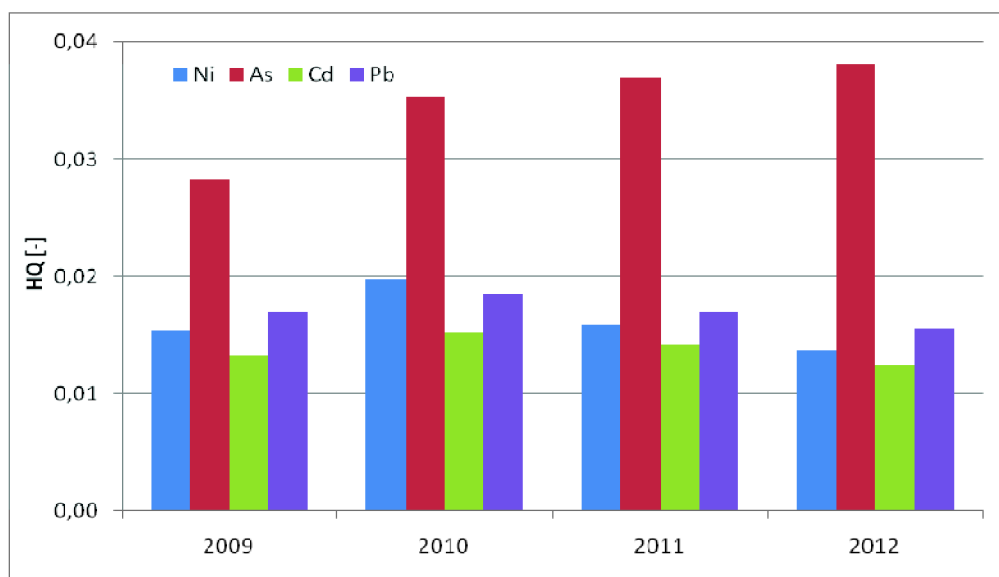
Fig. 6.9: Values of hazard quotient for single elements in Líšeň, 2009–2012

Chronic inhalation risk due to exposure to toxic metals in PM is negligible for all diagnosis and thus acceptable.

Table 6.7: Chronic hazard index (Líšeň, 2012)

Pollutant	Liver, kidney	Cardiovascular system	Reproductive system	CNS system	Hematologic effects	Respiration system
As	-	0.038	0.038	0.038	-	-
Cd	0.012	-	0.012	-	-	0.012
Ni	-	-	-	-	0.014	0.014
Pb	0.016	-	-	0.016	0.016	-
HI	0.028	0.038	0.050	0.054	0.030	0.026

6.5.2 Evaluation of risks of carcinogenic pollutants

As mentioned in chapter 5.4.6, some toxic elements possess both the systemic effects and also carcinogenic potential. The estimate of health risk was carried out using the exposure scenario displayed in table 5.3 which considers three hours long exposure to the outdoor air. The individual risk, with probability of malignant disease occurrence due to a harmful substance, equal to $1 \cdot 10^{-6}$ (i.e. one case per million) is considered to be acceptable. Under this

assumption, the carcinogenic risk of adults, from exposure to PAHs and toxic metals in Líšeň, may be evaluated to be acceptable (see figure 6.10A and 6.11A).
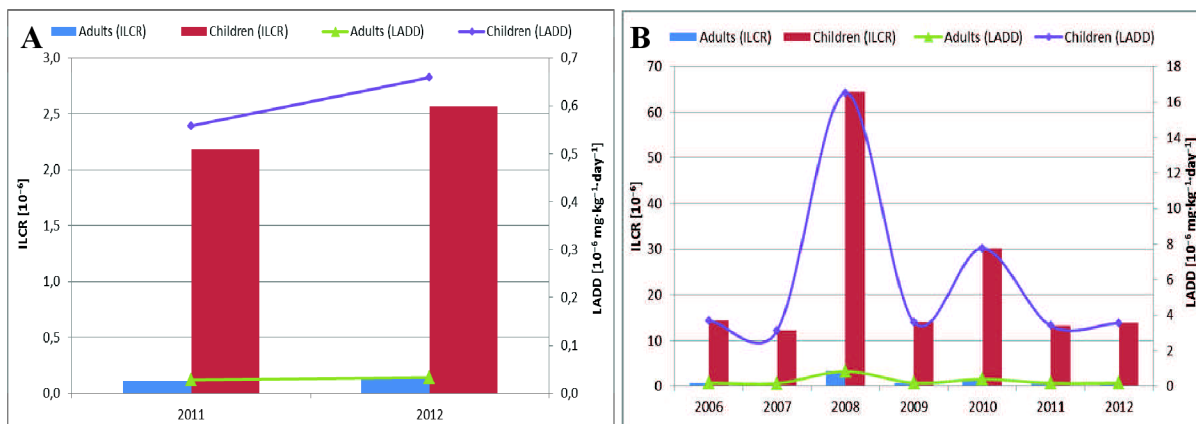
Fig. 6.10: Health risks of PAHs: adult and child population in Líšeň (A) and Masná (B)

Figure 6.10 shows values of carcinogenic risks of PAHs. In the Líšeň locality (figure 6.10A) the cancer risk of children is in the limit of acceptability. ILCR values for Masná (figure 6.10B) are also in the limit of acceptability but now for adults. The very high values refer to children population in Masná, which exceeded the limit more 60 times in 2008. The values are getting lower towards present but they are elevated more than 10 times compared to the limit of $1 \cdot 10^{-6}$. As the individual cancer risk for children is exceeded in significant manner, it is appropriate to talk about the increased carcinogenic risk from exposure to PAHs which are found in airborne PM. That should be a reason for implementation of appropriate steps towards reduction.

A similar situation can be seen in Líšeň. Individual risks for children from exposure to toxic metals are also elevated, especially in the case of arsenic, cadmium is in the limit of acceptability in some cases it is already behind it.
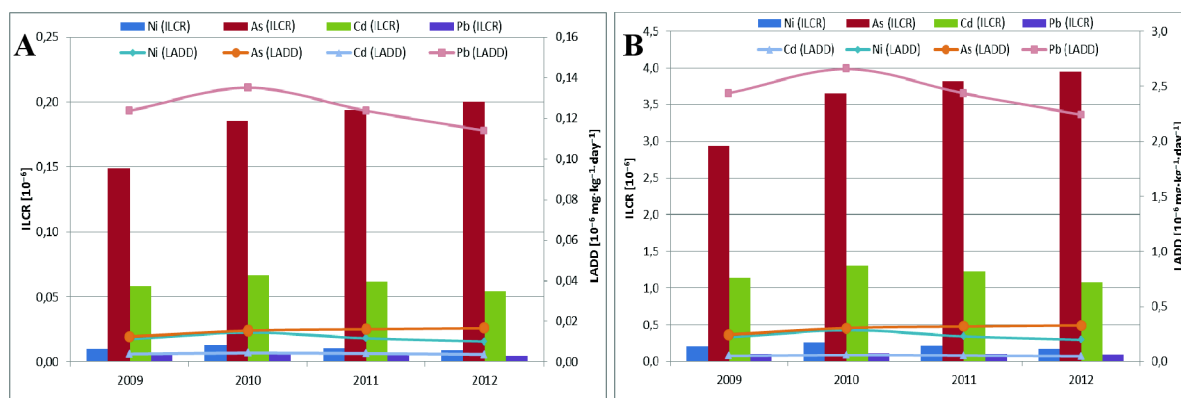
Fig. 6.11: Health risks of toxic metals in Líšeň: for adults (A) and children (B)

6.5.3 Estimate of health risks of PM_x

In this section are estimated health risks in the form of percentage of mortality increase using relation 5. Table 6.8 displays PM_{10} concentrations together with estimates of mortality increase. Lower percentage of mortality estimate is in the case of Líšeň which is not influenced by heavy traffic as the other ones. However, the maximum concentration reached 107 $\mu g \cdot m^{-3}$ in 2013 which is an increase compared to previous years. Calculated to increase in mortality, long-term exposure to this concentration may cause increase in mortality by 26.1 %.

Higher values are in the case of the other location. Masná is rather regarded as background locality but the values of maximum concentration are comparable with those of the localities with high traffic load. The annual mean concentrations are in range of 30 $\mu g \cdot m^{-3}$ but the number of days which it exceeded the limit of 50 $\mu g \cdot m^{-3}$ is higher than the legislation allows (see table 6.1). The worst situation is in streets Úvoz (which is usually to be said that it is the most polluted street in Europe) and Zvonařka. Maximal annual concentrations in these areas are long-term exceeded, which should be taken into account and find out some precautions a steps to reduction.

Table 6.8: Mortality increase due to suspended PM_{10}: maximum annual concentration [$\mu g \cdot m^{-3}$]/increase in mortality [%]

	Líšeň	Masná	Úvoz	Zvonařka
2008	-	147.3 / 38.2	174 / 46.2	146 / 37.8
2009	68 / 14.4	96 / 22.8	86 / 19.8	152.8 / 39.8
2010	97 / 23.1	78 / 17.4	149 / 38.7	170.9 / 45.3
2011	101 / 24.3	148 / 38.4	110 / 27.0	126.6 / 32.0
2012	84 / 19.2	103 / 24.9	111 / 27.3	120.1 / 30.0
2013	107 / 26.1	56 / 10.8	123 / 30.9	138.9 / 35.7

Table 6.9 shows the same as the table 6.8 but now for $PM_{2.5}$. The maximum annual concentrations exceed 100 $\mu g \cdot m^{-3}$ also in Líšeň. As this case counts to $PM_{2.5}$, the airborne particles smaller than 2.5 μm in diameter, the higher values of mortality increase can be seen compared to those of PM_{10}. It is reasonable because smaller particles can penetrate more deeply into the respiratory tract and cause more adverse, long-term health effects.

The worst state was in 2010, in Zvonařka. Maximum value exceed 160 $\mu g \cdot m^{-3}$. Converting this value to potential increase in mortality, it equals to 91.1 %. The concentration decrease in last three years but still it is range of 120 $\mu g \cdot m^{-3}$, which corresponds to increase in mortality in range of 65 %. These values of estimated mortality increase are calculated for maximum value of concentration which was observed in a given year. So to consider this true, the concentrations must be in a higher range for a longer time because this model assumes long-term exposure. Based on this we can consider these results to be overestimated. But these results represent the most conservative approach when it is supposed that the worst conditions may occur.

Table 6.9: Mortality increase due to suspended PM$_{2.5}$: maximum annual concentration [$\mu g \cdot m^{-3}$]/increase in mortality [%]

	2008	2009	2010	2011	2012	2013
Líšeň	-	-	-	127/ 70.2	90/ 48.0	100/ 54.0
Zvonařka	109.2/ 59.5	137.3/ 76.4	161.8/ 91.1	115.3/ 63.2	117.7/ 64.6	124.5/ 68.7

7   CONCLUSION

This work dealt with the air pollution by dust particles, their collection, chemical analysis and health risks assessment. Four localities were analyzed from the air pollution point of view. Based on the results presented above, it may be said that there is a large necessity to introduce steps to reduce the pollution. There are many programs for reduction of air pollution. However, it is very difficult to implement them because nowadays the development of industry and related transportation is at high level. So it is necessary to extend next reduction of air pollution and thus to reduce health and environmental risks. There is a need to focus on fine dust particles ($PM_{2.5}$) which have a significant impact on human health. The air pollution in Brno city is at very high level. The concentrations of particulate matter exceed all permitted limits, the number of exceedances of PM_{10} is almost three times higher in some cases. Concentration of PAHs and toxic metals are within the legislation limits. Nevertheless, the very next analyses have shown that the concentrations of these substances may have adverse health effects as concerned of carcinogenic risks, especially in the case of children population. So it is not only the problem of PM alone. The very next problems are the substances bounded to PM which can cause next exacerbation and deterioration of health. So in this case, there is increased carcinogenic risk for children population caused by polycyclic aromatic hydrocarbons which should be a basis for implementing appropriate steps to reduction of these compounds. It is very difficult almost impossible to prevent these health risks and air pollution. Vehicular traffic is on daily routine as well as the heating in power plant or local heating (especially in winter), the reconstructions of building structures are also a problem which causes increase in level of particle pollution.

The main aims of this work were to do an overview literature search about particulate matter and its health risk, evaluate results of chemical analysis and compare the results with legislation limits. Finally, the health risk assessment was carried out and summarized. It is not in aims of this work to come up with new regulation steps for pollution or to recommend some. Nonetheless, this work may serve as a basis for implementing next steps to reduction of the air pollution in areas of the city of Brno.

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9   LIST OF ABBREVIATIONS AND SYMBOLS

A	anthracene	
AT	average exposure time	day
BaA	benzo(a)anthracene	
BaP	benzo(a)pyrene	
BbF	benzo(b)fluoranthene	
BghiPRL	benzo(g,h,i)perylene	
BkF	benzo(k)fluoranthene	
BW	body weight	kg
CA	concentration of a pollutant	$mg \cdot m^{-3}$
C_{inh}	inhaled concentration	weight per m^3
COPD	chronic obstructive pulmonary disease	
DBahA	dibenzo(a,h)antracene	
ED	exposure duration	year
EF	exposure frequency	$day \cdot year^{-1}$
ET	exposure time	$h \cdot day^{-1}$
Fen	fenanthrene	
Flu	fluoranthene	
HI	hazard index	[-]
HQ	hazard quotient	[-]
Chry	chrysene	
I123cdP	indeno(1,2,3,c,d)pyrene	
ICPF	individual cancer potency factor	[-]
ILCR	individual lifetime cancer risk	[-]
IR	inhalation rate	$m^3 \cdot h^{-1}$
LADD	lifetime average daily dose	$mg \cdot kg^{-1} \cdot day^{-1}$

PAHs	polycyclic aromatic hydrocarbons	
PM	particulate matter	
REL	reference exposure level	
RfC_i	reference exposure concentration	weight per m^3
TEF	toxic equivalency factor	
TEQ_{BaP}	benzo(a)pyrene toxic equivalent	
USEPA	United States Environmental Protection Agency	
WHO	World Health Organization	