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## Indoor Air Pollutants: Relevant Aspects and Health Impacts

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### 1. Introduction

During the last three decades, many efforts have been made to protect populations from harmful exposure to outdoor pollutants. Networks of air monitoring stations have been located in strategic places and these provide information on the outdoor pollutant concentrations to which populations are exposed. However, people spend about 80-90% of their time in various indoor ambiances (i.e. homes, offices, restaurants, etc.) and the quality of indoor air, is an important factor influencing human health. Indoor air quality is characterized by multiple determinants, such as physical parameters, chemical emissions and biological contaminations. It is a common belief that while indoors, one is safe from harmful pollutants. However, the scientific evidence has shown that indoor air at homes can be more seriously polluted than outdoor air of the largest and most industrialized cities (WHO, 2006; Franklin, 2007). Furthermore, people who constantly stay indoors, thus being chronically exposed to indoor pollution, are often the most susceptible individuals (infants, children and seniors). To understand the relationship between indoor air quality and health, it is important to further study the indoor pollutants that have the most significant effects on human health.

Tobacco smoke is one of the most significant indoor sources of air pollution. There is no doubt that tobacco smoke causes various adverse health effects; voluminous literature has been dedicated to this topic. The adverse effects of smoking have been widely recognized for several decades, but only recently public concerns have focused on indirect exposures to tobacco smoke.

From the chemical point of view, tobacco smoke is a very complex mixture of gaseous phase and particles of different sizes. To this date more than 5000 different chemicals have been identified in tobacco smoke (Perfetti & Rodgman, 2008, 2011). Many of these, namely N-nitrosamines, polycyclic aromatic hydrocarbons, and heavy metals are known or suspected carcinogens.

Considering the importance of this topic, this chapter is dedicated to indoor air pollution and its health impacts. A brief historical perspective of the problem is presented. A general

discussion on the aspects of indoor quality is given, covering indoor pollutants, their main sources, and impacts on human health. The chapter then focuses on tobacco smoke with particular emphasis on indoor particles.

## 2. Historical overview of indoor air pollution

Indoor air pollution has a much longer history than usually thought. Archaeological evidences suggest that indoor pollution was widely experienced in the distant past. Mummified lung tissues, preserved by tanning, freezing or desiccation, proved to be most useful to provide information on prehistoric exposure. The samples of these tissues were re-hydrated, allowing subsequent microscopic examination to identify solid materials deposited in the lungs (Brimblecombe, 1999). Various materials (mineral and wind-blown dust) that cause pneumoconiosis and silicosis were identified in samples of lung tissues throughout many different epochs and geographical locations (i.e. from a mummy of ancient Egypt, from Peruvian miner of sixteenth century, from East Anglian flint-knappers) (Brimblecombe, 1999); the most frequent occurrence was found for anthracotic particles being a result of lifelong exposure to smoke indoors. The smoke certainly was among the first major sources of indoor pollution. The soot found on the ceilings of prehistoric caves provides further evidence of indoor pollution associated with open fires as first human habitations were poorly ventilated (Spengler & Sexton, 1983). Perhaps even then, people were able to intuitively recognize negative impacts of smoke, as in the Romano - British Period, cooking was done outdoors or away from living areas (Brimblecombe, 1999). Nevertheless, during the Dark Age primitive huts still did not have chimneys. The smoke from the central hearth simply rose and then slowly escaped through holes in the roof; such conditions led to high levels of indoor pollution. The use of chimneys in the early modern period therefore represented a particularly relevant technological change. By the Elizabethan time chimneys became far more common and effective (Burr, 1997), and thus widely used. It is important to point out that these transformations were accomplished with some skepticism and prejudice; indoor smoke was considered important in hardening the timbers of the house and warding off the diseases among its habitants (Brimblecombe, 1999). Perhaps it was the reason why stoves, another step of technological development, never really took hold in England although some attempts were made; stoves grew especially popular on the continent significantly reducing exposure to indoor smoke.

Indirectly the historical evolution of indoor pollution was also related with outdoor pollution. For example, in 1952, a major air pollution disaster in London resulted in passage of the Clean Air Act (in 1956). Consequently, changes were made to the means of heating homes. Fireplaces, typically placed in each room and the use of soft coal, were successfully replaced by central or electrical heating systems (Boubel et al., 1994).

Although interest in indoor air quality has been increasing since the beginning of the twentieth century, prior to 1970s the scientific interest in these problems was rather low. However, the last two decades represented a positive change and recently public attention has focused on the risks associated with poor indoor air quality. Furthermore, exposure levels to various harmful indoor air pollutants have been increasingly considered for the protection of human health.

3. Indoor pollution

It is a common belief that when indoors one is safe from harmful pollutants. The general perception is that levels of pollution inside buildings are lower than outside as the walls protect us from external impacts. However, confined indoor spaces may cause the concentration of pollutants to rise to unacceptable levels. Furthermore, there are many

Pollutant		Emission source
Inorganic chemical substances	Carbon dioxide	Combustion activity, metabolic activity
	Carbon monoxide	Fuel burning, tobacco smoke, stoves, gas heaters, motor vehicles in garages
	Nitrogen dioxide	Outdoor air, fuel burning, motor vehicles in garages
	Sulfur dioxide	Outdoor air, fuel combustion
	Ozone	Photochemical reaction
	Radon	Soil and bedrock under houses, building materials, ground water
Organic chemical substances	Formaldehyde	Insulation, furnishings
	Polycyclic aromatic hydrocarbons	Tobacco smoke, fuel combustions
	Polychlorinated biphenyls	Heat transfer fluids used in lamp ballasts and TV capacitors, stabilizers used in PVC wire insulation materials, additives in sealants, adhesives, paints, and floor finishes
	Volatile organic compounds	Household products (paints, aerosol sprays, cleaning supplies), building materials and furnishings, office equipments (i.e. copiers and printers)
Biological pollutants	Allergens	Domestic animals, insects, house dusts
	Fungi	Internal surfaces, soils, plants, food
	Microorganisms	Occupants – people, animals, plants, air heating, ventilation, air-conditioning systems
	Pollens	Outdoor air, indoor vegetation
Other	Asbestos	Fire retardant material, insulation
	Particles	Tobacco smoke, combustions, resuspension
	Pesticides	Commercial and residential application of insecticides and herbicides

Table 1. Indoor air pollutants and their emissions sources, adapted from Jones, 2002; Król et al., 2011

additional sources of indoor pollution. They include combustion processes for house heating, lighting and cooking, emissions from the buildings materials, decorations, and activities of the indoor occupants (Jones, 2002). Indoor pollution also originates from biological sources, such as domestic animals, plants and insects. Due to a variety of these sources, the extents of indoor pollution differ significantly among places and over times. A substantial part of indoor pollution may result from outdoors. In some cases outdoor sources, such as vehicular traffic or industrial emissions can be major contributors to indoor pollution. This is especially important for homes situated in industrial areas or in urban areas in close proximity to roads. A study performed in the Netherlands showed increased mortality rates due to the exposure of particles found indoors from vehicular emissions; a relative risk of 1.95 was estimated for people living within 50 m of a major road (or 100 m from a highway) (Hoek et al., 2002). Several other authors provided evidence of human indoor exposure to traffic pollutants in relation to distance from major roads or to traffic density (Heinrich et al., 2005; Janssen et al., 2003; Martuzevicius et al., 2008; Meija et al., 2011). Thus, the actual extent of indoor pollution results from both outdoor and indoor sources, but also of other parameters such as building architecture, furniture position, etc. Undoubtedly, the “individuality” of each indoor environment implies further research difficulties for complete understanding of indoor pollution.

Initially indoor pollutants that received the greatest attention were pollutants that penetrated from outdoors, namely sulfur dioxide, nitrogen oxides, ozone, and particles. However, the pollutants present indoors include not only these gases and particles, but a whole range of other pollutants that only increase to significant levels in enclosed indoor environments. Thus, attention was subsequently focused on pollutants that were of particular concern indoors, i.e. formaldehyde, radon, asbestos, tobacco smoke, and volatile organic compounds (Weschler et al., 2009). Later on, pesticides and other organic compounds found indoors gained scientific attention (Weschler et al., 2009). Table 1 presents the most known indoor pollutants and their respective sources (Jones, 2002; Król et al., 2011).

Indoor air pollution has been associated with a wide range of health outcomes as most of the air pollutants directly affect the respiratory and cardiovascular systems. Table 2 gives a general overview of the mechanisms and respective health outcomes of the main pollutants from indoor combustion (Bernstein et al., 2008; Goyal & Khare, 2010).

In general there are two types of health effects arising from indoor air pollutants: short-term (acute) effects and long-term (chronic) effects. Short-term health effects, such as irritation of eyes, nose, throat, and skin, headache, dizziness, and fatigue appear after a single exposure or repeated exposures. If identified they are treatable, however, most of these effects are similar to those associated with the common cold or other viral diseases, so it is often difficult to determine if the symptoms result from exposure to indoor air pollution or another cause. Long-term health effects occur only after long or repeated periods of exposure to pollutants. For example, short-term effects of smoke inhalation from indoor fuel combustion include acute respiratory irritation and inflammation, and acute respiratory infection (Goyal & Khare, 2010). Long-term effects of smoke inhalation are chronic obstructive pulmonary disease (COPD), chronic bronchitis, adverse reproductive outcomes and pregnancy-related problems, such as stillbirths and low birth weight, and lung cancer (Goyal & Khare, 2010; Okona-Mensah & Fayokun, 2011).

Pollutant	Mechanism	Potential health effects
Carbon monoxide	Binding with hemoglobin to produce carboxyhemoglobin, which reduces oxygen delivery to key organs and developing fetus	Low birth weight Increased perinatal death
Nitrogen dioxide	Acute exposure increases bronchial reactivity Long-term exposure increases susceptibility to bacterial and viral lung infections	Wheezing, exacerbation of asthma Respiratory infection, Reduced lung function in children
Sulfur dioxide	Acute exposure increases bronchial reactivity Long term: difficult to dissociate from effects of particulates	Wheezing, exacerbation of asthma Exacerbation of COPD, cardiovascular disease
Polycyclic aromatic hydrocarbons	Carcinogenic	Lung cancer Cancer of mouth, nasopharynx, and larynx
Particles	Acute bronchial irritaion, inflammation, increased reactivity Reduced mucociliary clearance Reduced macrophage response and reduced immunity	Wheezing, exacerbation of asthma Respiratory infection Chronic bronchitis and COPD Excess mortality, including from cardiovascular disease

Table 2. Examples of health risks associated with exposures to pollutants from indoor combustion

4. Selected indoor pollutants

There have been major changes in products and building materials used indoors over the last five decades. Accompanied also by modifications in building operations these changes have led to different emission profiles for indoor pollutants. For example restrictions on use of some building materials have led to a reduction of asbestos indoors. On the other hand indoor environments are less ventilated than they were decades ago, which leads to increased levels of pollutants from biological sources, i.e. mold, allergens, and fungi (Weschler et al., 2009). Furthermore, in some parts of the world air-conditioned buildings are frequently built. All of these changes have altered the type and concentrations of chemicals that occupants are exposed to in indoor environments (Weschler et al., 2009). The personal habits of building occupants have also changed. Since tobacco smoke represents a serious risk to human health and is a major source of indoor particles, the following section focuses on this pollutant. A brief overview of other health-relevant indoor pollutants, common in all regions of the world, is then given.

## 4.1 Tobacco smoke

### 4.1.1 Tobacco smoke throughout history

A history of tobacco dates back to 5000–3000 BC when the agricultural product began to be cultivated in South America (Gately, 2001). By the arrival of Christopher Columbus in 1492 the use of tobacco reached every corner of American continent including islands such as Cuba. Tobacco was used in various ways and for various purposes. It was sniffed, chewed, eaten, drunk, smeared over bodies, used in eye drops and enemas (Gately, 2001); it was blown into the warriors faces during battle, and offered to gods. Perhaps the major use of tobacco was in medicine. Mild analgesic and antiseptic properties made tobacco useful to heal minor illness such as toothache when its leaves would be packed around the tooth (Gately, 2001). It was also believed to be a remedy for snake bites when the juice of tobacco leaves was applied directly to wound. Later, the consumption of tobacco smoke evolved into burning the plant substance either by accident or with intent of exploring other means of consumption. Tobacco is a powerful insecticide and blowing its smoke over seed corn or fruits trees was an effective way of controlling pests in some civilizations (Gately, 2001). The practice of tobacco combustion also worked its way into shamanistic rituals (Wilbert, 1987); many ancient civilizations, such as Indians and Chinese, burnt incense as a part of their religious rituals. Smoking of tobacco thus probably had its origins in these incense-burning ceremonies of shamans (Robicsek, 1979). The act of smoking was not merely a method for tobacco consumption but an integral part of the rituals. Nevertheless, later it was adopted for pleasure and as a social tool.

Tobacco was introduced to Europe in 1493 when Christopher Columbus returned from his discovery voyage to America (Thielen et al., 2008). Previously Europe did not have any precedent for tobacco smoking. In fact Europeans did even lacked the vocabulary to describe the act of smoking (Gately, 2001). The English language term “smoking” was created only in the late 18<sup>th</sup> century; before then the practice was called “drinking smoke” (Lloyd & Mitchinson, 2008). Although, Europeans reacted with horror and consternation when the returning sailors smoked, the habit spread; nowadays, it is estimated that there are  $1.1 \times 10^9$  smokers around the world (IARC, 2004). The medical properties of tobacco raised European’s curiosity. Originally, tobacco was planted in palace gardens where it was studied by royal physicians. The initial association of tobacco with royal society enhanced its reputation and helped its spread around the world. In Spain tobacco was populated via the Roma Catholic clergy, who developed a fondness to snuff (Gately, 2001). Frenchman Jean Nicot introduced snuff to France in 1560. Nicot (from whose name the word nicotine is derived) received tobacco cuttings from famous Portuguese botanist Damião de Goes during his official stay at Portuguese court; he later planted those in the gardens of the French embassy (Taylor, 2008). Afterwards, the use of tobacco spread to England. The first report of a smoking Englishman is of a sailor in Bristol in 1556, seen “emitting smoke from his nostrils” (Lloyd & Mitchinson, 2008). Around 1600 tobacco was introduced in what today is modern-day Gambia and Senegal. By the 1650s the Portuguese brought the commodity and the plant to southern Africa, establishing the popularity of tobacco throughout all of Africa (Gately, 2001). Tobacco, both product and plant, followed the most important trade routes to major ports and markets, and then on into the hinterlands. By the mid-17<sup>th</sup> century every major civilization had been introduced to tobacco smoking. In many cases tobacco smoking had assimilated into the native culture, despite the attempts to eliminate the practice with harsh penalties or fines (Gately, 2001). During the American Civil War in 1860s the primary labor force of tobacco production

shifted from slavery to share cropping (Burns, 2006). In 1881, the industrialization of tobacco production with the cigarette followed.

#### 4.1.2 Tobacco combustion and composition

The term “tobacco smoking” is the process where tobacco is burned and the vapors either tasted or inhaled. When smoking, air is drawn into a cigarette/cigar during each puff, and combustion takes place. This process forms tobacco mainstream smoke. Emissions produced in such manner are inhaled by smoker; burn temperature is up to 1000 °C (Colls, 1997). Although health-harmful pollutants such as N-nitrosamines are formed, they influence only the smoker.

Between the puffs, the cigarette smoulders, and from its lit end it forms sidestream tobacco smoke. The combustion temperature is lower (approximately around 400 °C; Colls, 1997), but it can lead to the formation of more toxic compounds than those found in mainstream smoke. Furthermore, before further dilution by air turbulences, the concentrations of toxicants in the sidestream smoke are extremely high. The mixture of smokes present in a room is then called “environmental tobacco smoke” (also known as second-handed tobacco smoke). Environmental tobacco smoke consists of mainstream smoke exhaled into environment and sidestream smoke, after dilution and aging (Thielen et al., 2008). The aging can last for minutes or hours, but during that time the composition of all pollutants, including particulates changes.

When tobacco combustion takes place, a large number of gaseous and particulate pollutants are produced; many of those being known or suspected carcinogens and more than 100 are considered chemical poisons (Colls, 1997). Various authors reported that over 4000 different chemical components were found in tobacco smoke. However, in their last update Perfetti and Rodgman (2011) reported 5685 components in tobacco smoke that accounted for more than 99% of the mass of whole smoke. Certainly, other tobacco smoke components are present in the mixture but the total mass of these remaining components is obviously quite small (Perfetti & Rodgman, 2011). Some of the tobacco smoke components, such as carbon monoxide, carbon dioxide, nitrogen, nitric oxide, formaldehyde, and benzene are present in gaseous phase, whereas others (e.g. phenols, cresols, and hydrogen cyanide, and light molecular weight polycyclic aromatic hydrocarbons) are portioned between both vapor and particulate phases (Castro et al., 2011; Rodgman & Perfetti, 2008). Carcinogenic metals, such as arsenic and cadmium, chromium, nickel, and lead (IARC, 2011) are mainly found in the particles (Slezakova et al., 2009a; Wu et al., 1997), as well as tobacco-specific nitrosamines (Thielen et al., 2008) and higher weight polycyclic aromatic hydrocarbons (Castro et al., 2011; Rodgman & Perfetti, 2006; Slezakova et al., 2009b). At present, as many as 570 PAHs have been identified in tobacco smoke (Perfetti & Rodgman, 2011), several of them being classified by the International Agency for Research on Cancer (IARC) as carcinogens (Rodgman & Perfetti, 2006, 2008). As most of these particulate-bound compounds have known adverse impacts on human health, the respective exposures represent a serious risk to human health (IARC, 2010; WHO, 2006).

#### 4.1.3 Health effects

Although originally little was known about the harmful health effects of tobacco smoke, by the twentieth century these had been widely recognized. Since the 1950s the health effects of

tobacco smoke have been intensively studied. Voluminous literature and public media have linked active tobacco smoking to lung and heart diseases and to cancers of various organ systems (Pasupathi et al., 2009; Shah & Cole, 2010). Smoking harms nearly every organ of the human body, but the full extent of the damage is still unknown. Even today, over 50 years after the first links between smoking and lung cancer were established, more diseases are being found to be caused by smoking. However, it is known that about half of all continuing regular smokers are killed by their smoking (EC, 2004); those smokers that die in middle age, lose approximately 22 years of life, with a larger proportion of that shortened life span being spent in ill health (EC, 2004). Over 3 million people are killed every year because they smoke, dying mostly due to lung cancer and aortic aneurysm, out of these over 650 000 are European citizens (EC, 2004).

Tobacco smoke affects not only people who smoke but also those who are somehow exposed to it. Undoubtedly, the exposure of smokers is much higher compared to those of nonsmokers. The exposure of non-voluntary smokers cannot be underestimated as it was found that higher levels of cancer-causing substances occurred in sidestream smoke than in mainstream smoke (Wu et al., 1997). Thus, the exposure to environmental tobacco smoke, also called passive smoking, has also become an important health issue and it has been established beyond any doubt that passive smoking poses hazards to human health. Among the most significant concerns associated with exposure to environmental tobacco smoke are potential respiratory and other effects associated with chronic exposures. Passive smoking increases the risk and frequency of respiratory symptoms (wheeze, cough, breathlessness and phlegm) and asthma (Horak et al., 2007; Kabir et al., 2009; Trude & Skorge, 2007), being a proven cause of respiratory diseases of the lower airways (croup, bronchitis, bronchiolitis, pneumonia) in childhood and during adulthood (Skorge et al., 2005); extended exposure to environmental tobacco smoke also induce various heart disease (Wu, 1997) as well as lung cancer for non-smokers (EC, 2004; Okona-Mensah & Fayokun, 2011). Thus in 1993, the US Environmental Protection Agency (USEPA) classified passive smoking as a “Class A” human carcinogen (USEPA, 1993); accordingly, IARC declared that exposure to environmental tobacco smoke is carcinogenic to humans (IARC, 2004). The concentrations of particles in environmental tobacco smoke can reach up to  $1000 \mu\text{g m}^{-3}$  compared to  $1\text{--}5 \mu\text{g m}^{-3}$  in clean ambient air or  $100 \mu\text{g m}^{-3}$  in polluted ambient air; the highest concentrations can be found in poorly ventilated and crowded pubs, clubs and coffee bars (Colls, 1997; Lee et al., 2010; Slezakova et al., 2009a). Many countries have implemented certain measures of interventions such as smoking bans or restriction in workplaces or public places in order to protect public health. However, these legislative interventions cannot apply for homes or other private indoor environments where exposure to environmental tobacco smoke has remained an important health issue. In Western countries, with an adult smoking prevalence of 30-50%, it is estimated that over 50% of homes are occupied by at least one smoker (WHO, 2000a), resulting in a high prevalence of exposure to environmental tobacco smoke. Young children, in particular, who spend most of their time at home, are at increased risks for even greater exposures to tobacco smoke if their mothers smoke; it was estimated that exposure to second-hand smoke in homes increases the risk of developing asthma by 40-200% (Bernstein et al., 2008).

The indirect exposure to tobacco smoke can also occur as a consequence of third-hand smoke (Winickoff et al., 2009). Third-hand smoke refers to the residue that is left behind on furniture, walls, and carpeting after a cigarette has been extinguished. This term first

appeared in print in 2006 but it became more widely known when used in 2009 by Jonathan Winickoff (Burton, 2011). Third-hand smoke may lead to contact of harmful compounds (Rehan et al., 2011) and has several exposure routes. It can remain on surfaces as a potential source of dermal exposure or be ingested by food that has been exposed to tobacco smoke (Matt et al., 2004). It can be re-emitted as source of inhalation exposure as dust can carry third-hand smoke to the lungs (Singer et al., 2002). Furthermore, after time the smoke residue can become airborne again. Petrick et al. (2011) has recently shown that residues of nicotine can interact with indoor air pollutants (i.e. ozone) resulting in the formation of secondary organic aerosols and gas and condensed phase products. The cumulative exposures to these airborne species may be greater for an infant than an adult when both breathing rate and body weight are considered (Winickoff et al., 2009). It is however rather difficult to quantify third-hand smoke contamination, because it depends largely on the respective spaces, in small confined places like a car the deposition might be really significant (Fortmann et al., 2010); personal exposures may continue to occur on the order of hours to days (Petrick et al., 2011). Although the health implications of third-hand smoke are currently unknown, children are especially susceptible to this exposure because they breathe near, crawl and play on, touch, and mouth contaminated surfaces; at up to 0.25 g per day, the dust ingestion rate in infants is more than twice that of adults (Winickoff et al., 2009). Emphasizing that third-hand smoke harms the health of children thus may be an important element in encouraging home smoking bans.

## 4.2 Particles

“Indoor particles”, “airborne particulates”, or “aerosols”, these are some of the terms once used when dealing with particles. The term “particulate matter” (with abbreviation PM) is often used as a synonym for particles in pollutants science. Particulate matter, as defined by World Health Organization (WHO) is a mixture of solid or solid/liquid particles suspended in air (WHO, 2000b). These particles vary in size, shape, origin, and chemical composition. It is usual to classify the particles by their aerodynamic characteristics. Typically these are summarized by aerodynamic diameter, i.e. a diameter of a spherical particle with a density of  $1 \text{ g cm}^{-3}$  that has the same inertial properties and settling velocity as the particle in question (Wilson et al., 2002).

When breathing, particles deposit in the human respiratory system thus causing various health effects. The deposition of particles within the human respiratory system is influenced by several parameters such as particles properties (size, density and shape, chemical composition), morphology of the respiratory tract, and breathing pattern. Among these parameters, size of the particles is especially important. During breathing, particles with an aerodynamic diameter of  $10 \mu\text{m}$  or less are naturally inhaled by humans; larger particles are less likely to enter the human respiratory tract. After inhalation larger particles are deposit in the nose and mouth. Particles with  $6\text{--}7 \mu\text{m}$  or less pass into the lower parts of respiratory tract where they deposit in the smaller conducting air ways and gas exchange regions of lungs. According to the entrances into the various compartments of the respiratory system, particles can be classified as (Wilson, 1998): inhalable, thoracic, and respirable. Inhalable particles refer to those that enter the respiratory system during breathing, including the head airways. Thoracic particles are those that enter the lower respiratory tract including the trachea, bronchi and the gas exchanges regions of lungs. Finally, respirable particles are those that are capable of reaching the alveolar regions of lungs. It is necessary to point out

that there is a great discrepancy in the literature on the use of the terms “inhalable”, “thoracic” and “respirable”. Thoracic particles are often used as equivalent for PM<sub>10</sub>, respirable particles are frequently used as synonym for PM<sub>2.5</sub> (Brunekreef & Holgate, 2002). It is important to differentiate between these terms as they are not completely identical. The term PM<sub>x</sub> originated from sampler cutpoint classification and refers to the collection of particles below or within a specified aerodynamic size range, usually defined by the upper 50% cutpoint size. Thus PM<sub>10</sub> stands for particulate matter with a 50% cutpoint at 10 μm of aerodynamic diameter, PM<sub>2.5</sub> are particles with a 50% cutpoint at 2.5 μm of aerodynamic diameter. However, respirable particle have 50% upper cutpoint at 4.0 μm whereas, as mentioned before it is 2.5 μm for PM<sub>2.5</sub> (Wilson, 1998). As for the PM<sub>10</sub> and thoracic particles, they both have 50% cutpoint at 10 μm, but thoracic particles have less precise size cut (Dockery et al., 1998).

The terms “fine” and “coarse” particles are also used in indoor pollution science. These terms originated from modal classification (Whitby, 1978) being based on the size distributions and formation mechanisms (Fig. 1). The coarse mode particles are with aerodynamic diameter greater than the minimum in the particle mass distribution, which generally occurs between 1–3 μm (Wilson et al., 2002). Particles of this mode are

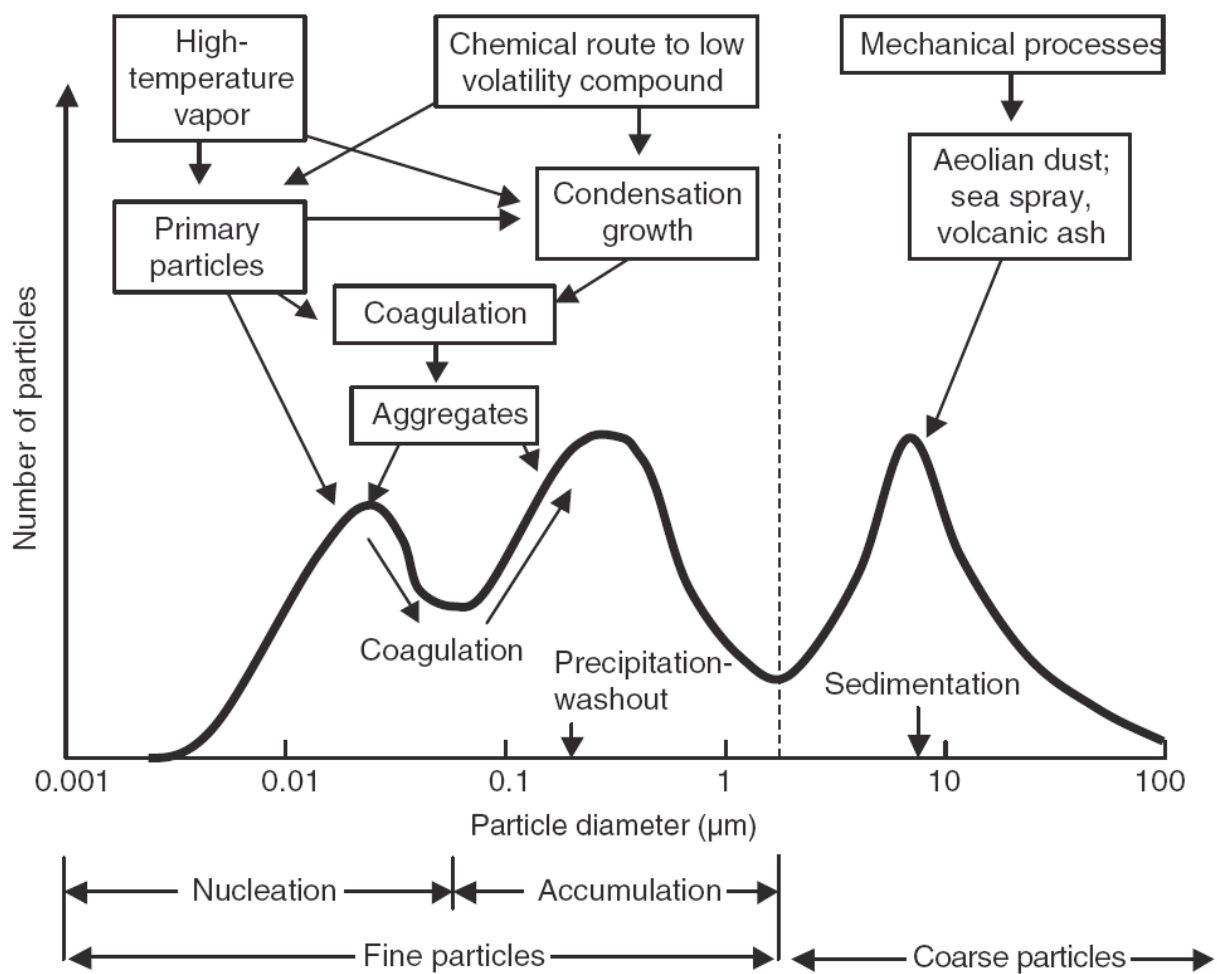


Fig. 1. Prototypical size distribution of particles, their sources and pathways of formations; dashed line corresponds approximately to 2.5 μm of diameter (Wilson et al., 2002)

mechanically produced by the break-up of larger solid particles. Pollen grains and mould spores as well as particles from plant fibers and leaves belong to this mode. The fine mode particles have an aerodynamic diameter mostly smaller than the minimum in the particle mass distribution. Based on the formation mechanisms of the particles, they are further subdivided into (WHO, 2000b): nuclei and accumulation mode particles. Nuclei (also called “ultrafine particles”) particles typically have an aerodynamic diameter smaller than 0.1  $\mu\text{m}$  (Oberdörster et al., 1995). These particles are directly emitted from combustion sources or formed by nucleation (i.e. condensation of low-vapor-pressure substances formed by high temperature vaporization or by chemical reactions in the atmosphere to form nuclei). Accumulation mode (Wilson et al., 2002) particles have an aerodynamic diameter between 0.1–1.0  $\mu\text{m}$ . These particles are formed from nuclei-mode ones that grow by coagulation (i.e. the combination of two or more particles to form a larger particle) or by condensation (i.e. condensation of gas or vapor molecules on the surface of existing particles). Coagulation is most efficient for large numbers of particles, and condensation is most efficient for large surface areas. Over the years the terms “fine” and “coarse”, as applied originally to the particle sizes, have lost their precise meaning. In many given articles the definition border is fixed by convention at 2.5  $\mu\text{m}$  of aerodynamic diameter due to the measurement facilities.

#### 4.2.1 Indoor sources

As it can be seen from Table 1 tobacco smoke and indoor combustion activities (i.e. wood and fossil fuel burning) are the main indoor sources of particles. Indoor particles are also produced by various activities, such as cleaning (vacuuming, dusting, and sweeping), cooking (broiling, baking, frying, toasting, barbecuing), by human movements, and from animals and plants (Abt et al., 2000a). In addition to these sources, emissions originated from outdoors are especially important for particulates (Castro et al., 2010; Chen & Zhao, 2010; Massey et al., 2009). Outdoor emissions are relevant particularly for fine particles as the contribution of outdoor particles indoors generally increases with decreasing particle sizes (Geller et al., 2002; Slezakova et al., 2011); it was found that outdoor 2-10  $\mu\text{m}$  particles accounted for 10% to 40% of indoor emissions, whereas it was between 35% to 92% for particles smaller than 2  $\mu\text{m}$  (Abt et al., 2000b; Slezakova et al., 2011).

The chemical composition of particulate matter is strongly related to its origin and sources. For example particles from combustions consist mainly of carbon and unburned or partially burned organic compounds. Except the carbonaceous materials, particles generally contain inorganic water insoluble material (i.e. various minerals) and inorganic water soluble material (i.e. sulfates, nitrates, chlorides). Although in much lower abundances, hazardous particulate components such as polycyclic aromatic hydrocarbons and heavy metals are relevant. Adsorbed onto the surface of the particles, they can contribute to adverse health effects of particulate matter. PAHs and heavy metals are predominantly found in fine particles (Castro et al., 2010, 2011; Slezakova et al., 2009a, 2009b, 2011); the large surface area of smaller particles allows them to carry greater amounts of these toxic compounds that are consequently deposit in the lower respiratory tract, thereby having a greater effect on the adverse health outcomes.

#### 4.2.2 Health effects

The extensive epidemiological research of the last two decades has provided much evidence on the exposure to particulate air pollution and adverse health effects. However, the health

impacts associated with these exposures have been much more extensively studied for outdoor particles than for indoor ones. Hence the majority of the knowledge arrives from the studies of outdoor air, whereas the number of epidemiological studies related to indoor environments is rather limited. There is still a lack of knowledge concerning health effects of different outdoor particle fractions, namely in relation to different size fractions of  $PM_{2.5}$ . Even more, this lack exists for indoor particles. Furthermore, the adverse effects of indoor particulate matter depend on deposition of particles in the respiratory tract, which is directly related to particle size and chemical composition. However, indoor particulates may differ substantially in composition from outdoor particulates hence their significance and contribution to the adverse health effects needs to be fully explored.

Particulate indoor air pollution has been linked to both acute and chronic health effects (Mitchell et al., 2006), including asthma, cardiac diseases, as well as impaired lung function and other conditions (Allen et al., 2008; Abbey, 1998; Pope et al., 1991; Viegi et al., 2004). Specifically fine particles have been shown to decrease forced expiratory volume in 1 second ( $FEV_1$ ) in asthmatic schoolchildren (Delfino et al., 2004). Furthermore, Delfino et al. (2008) found that  $FEV_1$  decrements were significantly associated with personal exposures to  $PM_{2.5}$ , but not ambient  $PM_{2.5}$  levels. These results emphasize the importance of indoor exposures. The personal exposures to indoor particles are often much higher than ambient air concentrations (Brown et al., 2008). Franck et al. (2011) investigated associations between indoor particle concentrations and the risks for respiratory diseases in young children; exposures to high indoor particle concentrations were associated with increased risks for the development of obstructive bronchitis, especially for particles smaller than 1  $\mu m$ . Apparently more detailed indoor measurements are necessary in order to fully understand health effects of indoor particle exposure.

#### 4.3 Carbon monoxide

Carbon monoxide is a toxic tasteless, odorless, and colorless gas that is produced by incomplete combustion of fuels such as wood, petrol, coal, natural gas and kerosene. Indoors, carbon monoxide is produced by these combustion sources (cooking and heating) and is also introduced through the infiltration from outdoor air into the indoor environment. Exposure to high levels of carbon monoxide might be lethal. Carbon monoxide is absorbed through the lungs and diffused across the alveolar capillary membrane. Once absorbed it passes across red blood cell membranes, and enters the red blood cell stroma where it binds to hemoglobin forming carboxyhemoglobin (COHb); the affinity of carbon monoxide to hemoglobin is about 200 times higher than that of oxygen (Tiwary and Colls, 2009). Such binding reduces the capacity of blood to carry oxygen and interferes with oxygen release at the tissues; the resulting impaired delivery of oxygen can interfere with cellular respiration and cause tissue hypoxia. Health effects of carbon monoxide are generally considered in relation to carboxyhemoglobin levels in blood. Except the increased daily mortality rate, health effects of carbon monoxide include early onset of cardiovascular disease, behavioral impairment, decreased exercise performance of young healthy men, reduced birth weight and sudden infant death syndrome (Bernstein et al., 2008). The severity of poisoning is dependent on concentration, length of exposure, and the general underlying health status of the exposed individual. Acute effects of carbon monoxide poisoning are particularly severe on the organs that require a high supply of oxygen, namely the brain and heart; the latter being well documented (Jones, 2002). Chronic

exposure to carbon monoxides causes symptoms that are easily misdiagnosed, such as headache, fatigue, dizziness and nausea (Jones, 2002).

#### 4.4 Nitrogen dioxide

Nitrogen dioxide is a reddish brown gas with a characteristic pungent odor. It is a key precursor of a range of secondary pollutants whose effects on human health are well-documented (WHO, 2006). The most important indoor sources include tobacco smoke and gas-, wood-, oil-, kerosene- and coal-burning appliances such as stoves, ovens, space and water heaters and fireplaces. Outdoor nitrogen dioxide from natural and anthropogenic sources also influences indoor levels. Inhalation is the major route of exposure to nitrogen dioxide. The link between exposure to nitrogen dioxide and adverse respiratory effects in susceptible populations has been explored extensively, but results are inconclusive (Franklin, 2007; WHO, 2006). There is recent evidence suggesting that children with atopy or asthma, infants who are at risk of developing asthma, and female adults are more sensitive to the respiratory effects of nitrogen dioxide exposure (Berstein et al., 2008). An increase in indoor nitrogen dioxide of  $28 \mu\text{g m}^{-3}$  was associated with a 20% increased risk of lower respiratory illness in children (WHO, 2010). Indoor exposure to nitrogen dioxide may also enhance asthmatic reactions to inhaled allergens (Berstein et al., 2008).

#### 4.5 Radon

Radon is a colorless and odorless radioactive gas that arises from the decay of radium-226. It is classified by the IARC as a human carcinogen (IARC, 2011). The main source of indoor radon is through the decay of radium in the soil subjacent to a house. Due to current construction methods, radon concentrations often become enhanced indoors. Radon itself is inert and causes little damage. However, it undergoes further radioactive decay which produces short-lived radioisotopes; the most stable of the isotopes is radon-222 which is universally referred as “radon” or “radon gas”. Some of isotopes are electrically charged and can be inhaled, both directly or bound to particles. Once inhaled, they deposit in lungs causing severe health effects (WHO, 2010). There is direct evidence from residential epidemiological studies that radon causes lung cancer, even at concentrations typically found in indoor air (Al-Zoughool & Krewski, 2009). Some studies suggested also an association with other cancers, in particular leukaemia and cancers of the extra-thoracic airways (WHO, 2010).

#### 4.6 Sulfur dioxide

Sulfur dioxide is nonflammable, nonexplosive, colorless gas with strong pungent smell. Its concentrations indoors are typically lower than those from outdoors. Inhalation is the major route of exposure to sulfur dioxide. Some studies have demonstrated that sulfur dioxide can cause bronchoconstriction (airway narrowing) in both healthy and asthmatic adults with clinical symptoms of shortness of breath, wheezing (Berstein et al., 2008), and impaired lung function (Jones, 2002). Such responses also occur at low levels of sulfur dioxide during moderate exercise, asthmatic children and adults are at higher risk. Data available from recent epidemiological study do not show any evidence of excess general and cause-specific mortalities (respiratory, cardiovascular) associated with exposures to sulfur dioxide in urban areas (Brunekreef et al., 2009).

#### 4.7 Formaldehyde

Formaldehyde is to the general public the most known volatile organic compound that has been associated with indoor air pollution. Indoor sources include combustion processes such as smoking, heating, cooking, or candle or incense burning. Major sources in non-smoking environments are building materials and consumer products that emit formaldehyde. This applies typically to new materials and products but in conditions with high relative humidity and high indoor temperatures it can last several months (WHO, 2010). Sources of formaldehyde indoors include furniture and wooden products containing formaldehyde-based resins, insulating materials, textiles, products such as paints, wallpapers, glues, adhesives, varnishes and lacquers, household cleaning, and electronic equipment. Formaldehyde is classified as a human carcinogen (IARC, 2011) and high occupational exposures are considered as a risk for nasopharyngeal cancer (Franklin, 2007). At lower concentrations formaldehyde has mostly been associated with irritation of the eyes, nose and upper airways (Berstein et al., 2008). Several epidemiological studies reported associations between exposure to formaldehyde in homes and schools with asthma, asthma severity, allergy, and airway inflammation in children (Franklin, 2007).

#### 4.8 Polycyclic aromatic hydrocarbons

Polycyclic aromatic hydrocarbons (PAHs) are a group of organic compounds with two or more aromatic rings. They occur in indoor air as complex mixtures, and their composition may vary from place to place. PAHs are known for their cytotoxic and mutagenic properties; although a number of PAHs are non-mutagenic, their metabolites or derivatives may be potential mutagens. PAHs also represent the largest known group of carcinogens. While some compounds are probable/possible carcinogens others are known human carcinogens (IARC, 2010). PAHs that are potent carcinogens are typically attached to particles. The primary exposure to carcinogenic PAHs found in air occurs via inhalation of these particles. Chronic exposure to PAHs may affect the pulmonary, gastrointestinal, renal and dermatological systems (Król et al., 2011). Certain PAHs can also affect the haematopoietic and immune systems, and can have teratogenic and neurological effects (Król et al., 2011). Health evaluation data suggest that lung cancer is the most serious health risk from exposure to PAHs in indoor air (WHO, 2010).

#### 4.9 Volatile organic compounds

Volatile organic compounds (VOCs) are organic chemicals that easily vaporize at room temperature. Over 300 individual compounds have been measured in indoor environments, some of them, such as benzene, dichloromethane, and tetrachloromethane are known carcinogens (IARC, 2011). Individual compounds have been associated with a variety of health effects including irritation, neurologic and respiratory symptoms (Bernstein et al., 2008). Associations between these measures of exposure and poor respiratory health have been observed in infants, preschool and school-aged children, but the findings were not completely consistent (Franklin, 2007).

#### 4.10 Pollutants from biological sources

As various biological materials (i.e. mold, yeasts, wood-rotting fungi, bacteria, and viruses) have been found indoors, the health impacts of inhaled biological pollutants should not be

underestimated (Dales et al., 2008). Biological agents can cause diseases through atopic mechanisms, infection processes, and direct toxicity. Numerous studies conducted worldwide have reported an association between indoor pollutants from biological sources and adverse acute and chronic health effects (Bernstein et al., 2008), including rhinitis and other upper respiratory symptoms, asthma, humidifier fever, extrinsic allergic alveolitis, atopic dermatitis, high blood pressure in adults, lung and immune system adverse effects (Bernstein et al., 2008; Srikanth et al., 2008).

## 5. Guidelines

The fulfillment of indoor climate requirements are stated as objectives of the Directive 2002/91/EC on Energy Performance of Buildings, however no specifications on how to achieve this are provided (Jantunen et al., 2011). Portugal is the only Member State that included an Indoor Air Quality assessment in the procedure of assessing energy performance according to the Directive (Jantunen et al., 2011). In 2008 the EU commission revised the directive. The new Directive 2010/31/EC still includes the requirement for a good indoor climate, but still does not specify any actions to guarantee this goal; the directive emphasizes energy efficiency, but does not require any information on indoor air climate. However, European Union set recommendation guidelines for levels of indoor exposure to radon (Commission Recommendation 90/143/Euratom), expressed as effective dose equivalent. For existing buildings the effective dose equivalent is 20 mSv per annum (for practical purposes, may be taken as equal to an annual average concentration of radon gas of 400 Bq/m<sup>3</sup>). For future constructions the dose is 10 mSv per annum, i.e. equivalent to an annual average concentration of 200 Bq/m<sup>3</sup>.

Despite the efforts of information campaigns on health effects of indoor pollution and on the maintenance of a healthy indoor environment, the public is still more aware of the adverse impacts of outdoor pollution rather than indoor pollution. Improving indoor air quality is a specific action of the European Union (CEC, 2004), with two key elements: addressing environmental tobacco smoke, and developing networks and guidelines on other factors affecting indoor air quality by using research and exchange of best practice. Concurrently, the World Health Organization continues with its efforts to define conditions for healthy air. In order to encourage the relevant policy developments for ensuring healthy indoor air, guidelines for a range of chemical substances most commonly polluting indoor air have been recently set (WHO, 2010). If sensibly applied as part of policy development, indoor exposure to air hazardous pollutants should decline thus leading to a reduction in adverse effects on health.

## 6. Conclusion

During last few decades indoor air quality has finally received much deserved scientific attention. Although considerable progress has been made in our knowledge about indoor pollution and its sources, the problem is still not completely understood. The unique individuality of each indoor environment (i.e. construction, inhabitants habits, etc.) implies further research difficulties and indoor ambiances also differ between regions as well as between continents. Despite that, the problems of poor indoor air that modern societies face are similar for many countries around the world. It is certainly necessary to continue with

research efforts. In particular associations between indoor air quality and health impacts need to be better addressed as so far much of the knowledge is derived from outdoor exposure studies.

Much effort has been invested in the reduction of outdoor pollution. However, reducing ambient pollution does not necessarily result in a proportionate decrease in indoor air pollution - a situation which has important implications for interventions. Therefore, regulatory aspects of indoor air quality also need to be considered. As the links between indoor air quality and health effects are becoming better understood, various attempts have been made to address this issue. For example, the USA and many European countries have successfully implemented interventions to protect public health from some hazardous pollutants, such as tobacco smoke. Asian or South American countries still have a high prevalence of second-hand tobacco smoke in public places, hence the need for future smoke-free regulation in those countries. As for the indoor particles there is still lack of scientific evidence which prevents the establishment of comprehensive indoor limit values and guidelines for health protection.

To ensure the protection of public health, it is necessary to combine all our available resources including scientific knowledge and regulatory power, thus providing the healthiest indoor environments possible for this and future generations.

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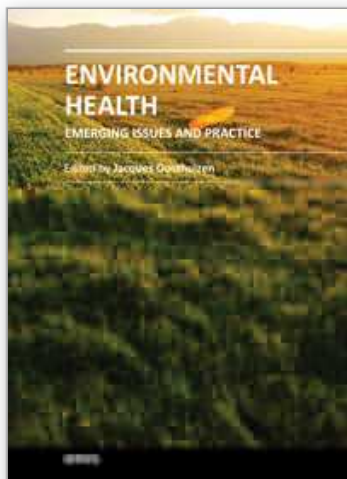
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Environmental health practitioners worldwide are frequently presented with issues that require further investigating and acting upon so that exposed populations can be protected from ill-health consequences. These environmental factors can be broadly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

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