15

Air Pollution with Asbestos in Several Cities in Romania

Anca Maria Moldoveanu
University of Medicine and Pharmacy" Carol Davila" Bucharest
Romania

1. Introduction

The general population is exposed to low levels of asbestos primarily by inhalation. Small quantities of asbestos fibers are ubiquitous in air. They may arise from natural sources (e.g., the effect of weathering on asbestos containing minerals), from windblown soil from hazardous waste sites where asbestos is not properly stored, and from deterioration of automobile clutches and brakes or breakdown of asbestos-containing (mainly chrysotile) materials, such as insulation.
Because of these properties, asbestos fibers have been used in a wide range of manufactured goods. In 1989, EPA identified the following asbestos product categories. Many of these materials may still be in use (EPA, 2010).

asbestos-cement corrugated sheet	asbestos-cement flat sheet	asbestos-cement pipe	asbestos-cement shingle
roof coatings	flooring felt	pipeline wrap	roofing felt
asbestos clothing	non-roof coatings	vinyl/asbestos floor tile	automatic transmission components
clutch facings	disc brake pads	drum brake linings	brake blocks
commercial and industrial asbestos friction products	sheet and beater-add gaskets (except specialty industrial)	commercial, corrugated and specialty paper	millboard
rollboard			

Table 1. Representatives asbestos product categories

The exposure of the general population to asbestos has been found to be very low. The concentrations of asbestos fibers in outdoor air are highly variable, ranging from below 0.1 ng/m^3 (equivalent to 3x10^{-6} f/mL measured by phase contrast microscopy [PCM]) in rural areas to over 100 ng/m^3 (3x10^{-3} PCM f/mL) near specific industrial sources such as asbestos mines. Typical concentrations are 1x10^5 PCM (phase contrast microscopy) f/mL in rural areas and up to an order of magnitude higher in urban areas. In the vicinity of an asbestos mine or factory, levels may reach 0.01 f/mL or higher. The concentration of fibers in indoor

air is also highly variable, depending on the amount and condition of asbestos-containing materials in the building 1 to 200 ng/m^3 (3x10^{-5} to 6x10^{-3} PCM f/mL), (Nicholson 1987). For a human exposed for a lifetime (70 years), this range of exposures corresponds to cumulative doses of approximately 0.002–0.4 PCM f-yr/mL (PCM fiber-years/mL).

2. Units of airborne asbestos levels

Numerous measurements have been performed to determine the concentration of asbestos fibers in environmental media, primarily air. The results were reported in a variety of units, including ng/m^3 (measured by midget impinger counting analysis), TEM f/mL (fibers measured by transmission electron microscopy), and PCM f/mL (fibers measured by phase contrast microscopy). The most accurate and sensitive method for measuring asbestos fiber content in air is electron microscopy, and preferably transmission electron microscopy (TEM) must be used. Phase contrast microscopy cannot distinguish between asbestos and non-asbestos fibers or between different types of asbestos. However, in certain occupational settings where the predominant fiber is asbestos, PCM should give an adequate measure of asbestos concentration. In non-occupational environments where a large fraction of the fibers are not asbestos (e.g., wool, cotton, glass), PCM may greatly overestimate the asbestos levels in air.

Regulations regarding asbestos determine what fibers are counted in the analysis. Established methods define fiber material having a length >5 μm and a length to diameter ratio of >3:1. In the same air sample, the fibers counted by TEM can be 50–70 times higher than those counted by PCM. This relates to the fact that PCM cannot detect fibers less than about 0.20–0.30 μm in diameter while TEM is capable of detecting fibers with diameters as small as 0.01 μm. Therefore, PCM may miss thin fibers as well as include nonasbestos fibrous material. The conversion factors between fibers counted by PCM and those counted by TEM are highly variable. In 1984, the NRC (1984) recommended that a conversion be used to measure asbestos fibers. It was suggested that crude approximations could be achieved by assuming that 1 PCM f/mL is equal to 60 TEM f/mL. Both 1 PCM f/mL and 60 TEM f/mL are approximately equal to a mass concentration of 30 μg/m^3. Since the health effects data regarding inhalation exposure to asbestos are usually expressed in terms of PCM f/mL(fibre/mL), ambient air data reported in units of ng/m^3 or TEM f/mL (fibre/mL) are converted to units of PCM f/mL (fibre/mL) using the factors suggested by NRC (1984).

For a good understanding of the measurement units used it can be stipulated that 10 fibers are typically present in a cubic meter (fibers/m^3) of outdoor air in rural areas. (A cubic meter is about the amount of air that you breathe in 1 hour). Health professionals often report the number of fibers in a milliliter (mL) (equivalent to a cubic centimeter [cm^3]) of air rather than in a cubic meter of air. Since there are one million cm^3 (or one million mL) in a cubic meter, there typically would be 0.00001 fibers/mL of asbestos in air in rural areas. Typical levels found in cities are about 10-fold higher.

Close to an asbestos mine or factory, levels may reach 10,000 fibers/m^3 (0.01 fibers/mL) or higher. Levels could also be above average near a building that contains asbestos products and that is being torn down or renovated or near a waste site where asbestos is not properly covered up or stored to protect it from wind erosion.

In indoor air, the concentration of asbestos depends on whether asbestos was used for insulation, ceiling or floor tiles, or other purposes, and whether these asbestos-containing materials are in good condition or are deteriorated and easily crumbled. Concentrations

measured in homes, schools, and other buildings that contain asbestos range from about 30 to 6,000 fibers/m^3 (0.00003–0.006 fibers/mL).

3. Asbestos biomarkers

Biomarkers are broadly defined as indicators signaling events in biologic systems or samples. They have been classified as markers of exposure, markers of effect, and markers of susceptibility (NAS/NRC 1989).

Principal biomarkers of exposure to asbestos fibers include the detection and counting of fibers or asbestos bodies in bronchoalveolar lavage fluid samples (De Vuyst et al. 1982, 1988, 1997; Dumortier et al. 1990, 1998; Roggli et al. 1994a; Sebastien et al. 1988a; Teschler et al. 1994; 1996, Tuomi et al. 1991b),sputum samples (McDonald et al. 1988, 1992; Sebastien et al. 1988b), or in autopsied or surgically resected lung tissue samples (Case 1994; Churg 1982; Churg and Warnock 1981; Churg and Wright,1994; Churg et al. 1993; de Klerk et al. 1996; Dodson et al.,1999; Dufresne et al. 1995, 1996a, 1996b; Sebastien et al. 1989). Asbestos bodies are collections of fibers (usually of length >8 μm) with a protein -iron coating (also known as ferruginous bodies) that, when observed in lung tissue sections in conjunction with fibrosis, have been proposed to be used in the diagnosis of asbestosis (Churg 1989; Craighead et al.,1982). Whereas light microscopy can be used to detect and count asbestos bodies, most uncoated fibers in tissue or fluid samples are too small to be visible (Dodson et al. 1999). Transmission or scanning electron microscopy is used to detect and count uncoated asbestos fibers in lung tissue or fluid samples, and electron diffraction or energy-dispersive x-ray analysis is used to determine asbestos type (e.g. chrysotile, anthophyllite, tremolite) (NIOSH 1994b). These biomarkers provide indicators of retained internal dose, the cumulative net result of deposition and clearance of inhaled asbestos fibers.

Counting asbestos bodies in human tissue, BAL fluid and sputum is easy and reproducible. However, not all fiber types and lengths form asbestos bodies. Therefore, the technique of counting ferruginous bodies in tissue sections is not sensitive, especially for short fibers and chrysotile. The sensitivity increases when a digested sample is evaluated by light microscopy for the presence of ferruginous bodies combined with the evaluation of the digested material for core identification of ferruginous bodies as well as characterization of the uncoated asbestos burden.

Advantages to counting asbestos bodies in human tissue, BAL (bronchoalveolar lavage) fluid, or sputum: Asbestos bodies are easy to identify with light microscopy. The results are easy to reproduce. Asbestos bodies correlate well with the concentration of amphibole fibers, which are the fibers that correlate best with mesothelioma. Counting asbestos bodies in BAL fluid could be used as a screening technique to indicate that exposure to amphibole fibers has occurred.

Disadvantages to counting asbestos bodies in human tissue, BAL fluid, or sputum: Chrysotile does not readily form asbestos bodies. The presence of ferruginous bodies in any sample only represents a population of the longer fibers in that sample. These numbers tell us nothing about the actual numbers of uncoated fibers of any length or type. Counting asbestos bodies may underestimate exposures to fibers that are greater than 5 microns in length and less than 20 microns in length.

Other Considerations: The study should combine analyses of asbestos bodies by light microscopy and fibers by electron microscopy. Electron microscopy can also be used to find

smaller ferruginous bodies and define the presence and types of uncoated asbestos fibers in a sample. The presence of asbestos bodies is a marker of exposure, not of disease.
Pleural plaques and pleural calcification are markers of exposure and may develop 10 to 20 years after initial exposure. Plaques are opaque patches visible on chest x-rays that consist of dense strands of connective tissue surrounded by cells. All commercial types of asbestos induce plaques. Plaques can occur even when fibrosis is absent and do not seem to reflect the severity of pulmonary disease.

4. Asbestos health effects

Asbestos exposure can cause a number of disabling and fatal diseases. The principal rout of exposure is by inhalation through the nose and mouth. Asbestos, traditionally valued for its indestructibility, is especially resistant to the internal defenses of the human body. Once lodged inside the lungs, most fibers will not break up or dissolve, and they cannot be neutralized or removed.
Asbestosis is a disease which is characterized by pulmonary fibrosis, a progressive scarring of the lungs caused by the accumulation of asbestos fibers. Asbestosis is associated exclusively with chronic, occupational exposure. The buildup of scar tissue interferes with oxygen uptake through the lungs and can lead to respiratory and heart failure. Often, asbestosis is a progressive disease, even in the absence of continued exposure.
Asbestos is known to be a human carcinogen. The EPA has determined that asbestos is a human carcinogen (Group A). In addition, the International Agency for Research on Cancer (IARC) has determined that asbestos is carcinogenic to humans (Group 1). These conclusions are based primarily on the evidence that asbestos causes lung cancer and mesothelioma. The World Health Organization Task Group on Environmental Health Criteria for Chrysotile Asbestos (WHO, 1998) concluded that "asbestotic changes are common following prolonged exposures of 5 to 20 f/mL(fibre/mL,)"[these correspond to cumulative exposures of 50–200 f-yr/mL (PCM fiber-years/mL) for a 10-year exposure] and that "the risk at lower exposure levels is not known."
Mesothelioma, a malignant nodular type cancer of the membranes which line the lung cavity, is another disease related to asbestos exposure. Malignant mesotheliomas of these membranes (the pleura and the peritoneum) are extremely rare in persons with no history of asbestos exposure, but may account for 10% to 18% of excess deaths in workers exposed to asbestos. Generally, a latency period of at least 25 to 30 years is required in order to observe mesotheliomas, and some victims have had a latency period of forty years since their initial exposure to asbestos. This form of cancer is incurable and is usually fatal within a year after diagnosis. Mesothelioma has been associated with short term, incidental exposure.
Some health studies have observed increases in esophageal, stomach, colo-rectal, kidney, and possibly ovarian cancers as well as cancers in the nose and throat from exposure to asbestos. While the magnitude of increased cancer risk for these sites is not as great as for lung cancer and mesothelioma, the increased risk may be of considerable importance because of the high background rates of some of these tumors in the general population.
There are many factors which complicate studies of non-occupational exposure, including a lack of data on incidental exposures which may occur, lack of data on non-occupational levels (f/cc) of exposure, and the lack of a control group (zero, or at least known "near zero" exposure). Confounding variables such as migration into and from communities and multiple exposures to other toxic chemicals and carcinogens consistently frustrate attempts

to generalize about the risk of low level exposure. At low levels of exposure, for example, asbestos may serve only as a "cancer promoter", acting as a co-factor along with other substances and carcinogens to elevate the risk of developing cancer above normal. Because asbestos fibers do accumulate in the lungs, and because the risk of developing disease does increase as the ***cumulative*** dose increases, exposure to asbestos should be controlled or eliminated whenever possible. Even a relatively minor source of airborne asbestos fibers should be abated, avoided, or minimized in order to maintain the cumulative dose at a minimum.

5. Purpose of the present study

The aim of the study was to assess the presence of asbestos fibers in the air of several towns in Romania. The main source is traffic and only a single town has traffic and an industrial source. The second purpose of the study was to investigate the presence of asbestos bodies in the lung tissue as biomarkers of the environmental exposure to asbestos through air.

6. Material and methods

The study was carried out in 9 towns in Romania including the capital city, Bucharest. The presence of asbestos bodies in the lung tissue of deceased people was investigated in Bucharest. The study was performed in first year of the new millennium.

6.1 Assessment of air pollution with asbestos

For measuring the levels of air contamination with asbestos fibers, the method used was phase contrast microscopy. Phase contrast microscopy (PCM) accurately assesses fiber exposure levels for fibers >5 μm in length and >0.25 μm in diameter. PCM cannot differentiate between asbestos and nonasbestos fibers. The standard method for the determination of airborne asbestos particles in the workplace is by phase contrast microscopy (NIOSH Method 7400). In the study, air samples were taken with a pump with a flow rate of 2 l/min for 2 hours. Asbestos was collected on a 25 mm cellulose ester filter. The filter was treated to make it transparent and then it was analyzed by microscopy at 400–450x magnification, with phase contrast illumination. A fiber is defined as any particle with a length>5 μm and a length-to-diameter ratio >3:1. The PCM method is relatively fast and inexpensive but it does not distinguish between asbestos and nonasbestos fibers and, it cannot detect fibers thinner than 0.25 μm. Consequently, this method is most useful for the analysis of samples that are composed mainly of asbestos, but only where a significant fraction of the fibers are large enough to be counted. If samples are grossly contaminated by nonasbestiform fibers, then transmission electronmicroscopy (NIOSH Method 7402) should be used for positive identification. Due to some technical problems, we used in the study only the phase contrast microscopy (PCM).Concentrations are reported as fibers/cm^3.
The levels of air pollution with asbestos were measured twice, in the spring and in the fall.

6.2 Assessment of the presence of asbestos bodies in the lung tissue

For assessing the presence of biomarkers of exposure in human body, samples of lung tissues were taken to routine autopsies in a pathology service in one of the important hospitals in Bucharest. The subjects included in the group of investigation were people with different causes of death. All subjects did not have occupational exposure during the life. The group of subjects was made of 25 bodies, 11 female and 14 male. The average value of age was 63.9

years of age. The youngest subject was 47 years old and the oldest 82. The occupational history of the subjects showed that none of them were exposed during their active life to asbestos.
The causes of death were: myocardial infarction, hypertension, arrhythmia, rupture of heart with haemopericardium, heart failure, COPD, chronic bronchitis, acute pulmonary edema, pulmonary thromboembolism, upper gastrointestinal bleeding, gastric ulcer, intestinal infarction, liver cirrhosis, intestinal obstruction.
5 of 25 subjects had died from cancer, 3 cases of pulmonary metastases from breast cancer, 1 case of intestinal metastases of rectal cancer and 1 case of liver cancer.
The identification of the presence of asbestos bodies in the lung tissue was made using light microscopy.

7. Results

7.1 The levels of asbestos in air in the investigated cities

In Bucharest the level of asbestos in air was measured in 21 points.

Measuring point nr.	Summer (fiber/cm^3)	Autumn (fiber/cm^3)
1	0.0418	0.0498
2	0.0393	0.0398
3	0.0411	0.0378
4	0.0489	0.0500
5	0.0350	0.0367
6	0.0182	0.0237
7	0.0169	0.0168
8	0.0229	0.0126
9	0.0149	0.0177
10	0.0132	0.0202
11	0.0145	0.0158
12	0.0139	0.0325
13	0.0181	0.0104
14	0.0181	0.0088
15	0.0146	0.0108
16	0.0200	0.0220
17	0.0126	-
18	0.0305	0.0280
19	0.0390	0.0430
20	0.0250	0.0260
21	0.0280	0.0240

Table 2. Levels of asbestos in air in Bucharest

Levels of asbestos in air	Summer (fiber/cm^3)	Autumn (fiber/cm^3)
Average value	0.0288	0.0250

Table 3. Levels of asbestos in air in Bucharest - Average values

The levels of asbestos in air of Bucharest had a variation between 0.0126 fiber/cm^3 and 0.0489 fiber/cm^3 with an average value equal to 0.0288 fiber/cm^3 in the summer and a variation between 0.0088 fiber/cm^3 and 0.0500 fiber/cm^3 with an average value equal to 0.0250 fiber/cm^3 in the autumn. The main source in Bucharest is the traffic and demolition of old buildings containing asbestos in their structure.
In Râmnicu Sărat city the level of asbestos in air was measured in 7 points.

Measuring point nr.	Summer (fiber/cm^3)	Autumn (fiber/cm^3)
1	0.0201	0.0271
2	0.0102	0.0358
3	0.0152	-
4	0.0130	-
5	0.0127	0.0340
6	0.0080	0.0143
7	-	0.0239

Table 4. Levels of asbestos in air in Râmnicu Sărat city

Levels of asbestos in air	Summer (fiber/cm^3)	Autumn (fiber/cm^3)
Average value	0.0132	0.02702

Table 5. Levels of asbestos in air in Râmnicu Sărat – average values

The levels of asbestos in air of Râmnicu Sărat had a variation between 0. 0080 fiber/cm^3 and 0.0201 fiber/cm^3 with an average value equal to 0.0132 fiber/cm^3 in the summer and a variation between 0.0143 fiber/cm^3 and 0.0358 fiber/cm^3 with an average value equal to 0.02702 fiber/cm^3 in the autumn. The source in this relatively little town is traffic but the main source is an industrial source.
In the summer, the minimum value was measured in the city of Târgu Jiu, the highest value was measured in the city of Ploieşti followed by the city of Bucharest and the highest average value was measured in Bucharest.
In two towns (Brăila and Alexandria) the minimum level was equal to 0. The maximum value was measured in Bucharest and the highest average value was measured in Râmnicu Sărat during the autumn.
In all 9 investigated towns the source of asbestos is traffic. Only the city of Râmnicu Sărat has an industrial source.
Traffic has the greatest contribution to air pollution with asbestos in Bucharest, town with a heavy traffic.

Name of the city	Minimum Value (fiber/cm^3)	Maximum value (fiber/cm^3)	Average value (fiber/cm^3)
București	0.0126	0.0489	0.0288
Constanța	0.0132	0.0249	0.0164
Rm. Sărat	0.0080	0.0201	0.0132
Ploiești	0.0050	0.0720	0.0060
Brăila	0.0060	0.0130	0.0097
Alexandria	0.0050	0.0080	0.0063
Craiova	0.0028	0.0120	0.0048
Tulcea	0.0028	0.0053	0.0037
Tg.Jiu	0.0010	0.0020	0.0140

Table 6. Levels of asbestos in air in 9 cities in Romania in summer

Name of the city	Minimum Value (fiber/cm^3)	Maximum value (fiber/cm^3)	Average value (fiber/cm^3)
București	0.0088	0.0500	0.0250
Constanța	0.0092	0.0398	0.0159
Rm. Sărat	0.0143	0.0130	0.0270
Ploiești	0.0090	0.0130	0.0110
Brăila	0.0000	0.0180	0.0077
Alexandria	0.0000	0.0070	0.0035
Craiova	0.0090	0.0014	0.0011
Tulcea	0.0037	0.0061	0.0021
Tg.Jiu	0.0010	0.0020	0.0104

Table 7. Levels of asbestos in air in 9 cities in Romania in autumn

An interesting approach will be, if the air pollution level with asbestos will be assess today especially in Bucharest taking into account the fact that the traffic is more intense in

comparison with other towns and to the previous period. Also, some technical modification to the disc brake pads and other brake components or clutch plates of cars were made especially to the new cars, trying to reduce the levels of air contamination with asbestos fibers.
The asbestos bodies were present in 4 of 25 investigated bodies.

Name	**Age**	**Sex**	**Occupation**	**Clinical diagnosis**	**Pathology diagnosis**	**Cause of death**	**Presence of asbestos bodies in the lung tissues**
D.A.	59	female	pensioner	lung cancer, breast cancer, respiratory failure	breast cancer, lung cancer – metastases, acute myocardial infarction	metastases, acute myocardial infarction	asbestos bodies present
M.A.	50	male	pensioner	COPD, renal failure	COPD, pulmonary thrombo-embolism cor pulmonale, pulmonary edema, upper gastrointestinal bleeding	-	asbestos bodies present
A.I.	59	female	pensioner	hart failure, liver cirrhosis	pulmonary edema, liver cirrhosis	intestinal obstruction	asbestos bodies present
J.E.	58	male	pensioner (former miner)	liver cancer, paralysis of the left side of the body	COPD cor pulmonale, silicosis, respiratory failure	respiratory failure	asbestos bodies present

Table 8. Presence of asbestos bodies in lung tissue in the investigated bodies in Bucharest

7.2 The presence of asbestos bodies in the lung tissue of the investigated bodies

Assessing the proportion of the presence of asbestos bodies in the subjects' bodies, the result showed that in 16% of investigated subjects the exposure markers were presents due to the environmental exposure.

8. Conclusions

Even if the method used overestimates the level of air pollution with asbestos fibers by including in the same category other types of fibers, the biological marker present in the lung tissue showed the risk of exposure to asbestos and risk to induce a specific morbidity due to the environmental exposure. The presence of asbestos bodies in the lung tissue of persons without occupational exposure shows that human health can be negatively affected by environmental exposure to asbestos and the efforts to reduce and even eliminate this pollutant from the environment may be beneficial for human health.

9. References

ATSDR (Agency for Toxic Substances and Disease Registry (2001),*Toxicological Profile for Asbestos.* Update. Department of Health and Human Services, Public Health Service, Atlanta, GA: U.S.,

ATSDR *Report on the Expert Panel on Biomarkers of Asbestos Exposure and Disease* , May 9-10, (2006), Atlanta, Georgia,

Case BW. (1994), Biological indicators of chrysotile exposure, *Ann Occup Hyg* 38:503-518,

Craighead JE, Abraham JL, Churg A, et al. (1982) The pathology of asbestos-associated diseases of the lungs and pleural cavities: Diagnostic criteria and proposed grading schema. *Arch Pathol Lab Med*106:544-597,

Craighead JE, Mossman BT. (1982) The pathogenesis of asbestos-associated diseases. *N Engl J Med* 306:1446-1455.

Churg AM, Warnock ML. (1981). Asbestos and other ferruginous bodies: Their formation and clinical significance. *Am J Pathol* 102:447-456.

Churg A. (1982) Fiber counting and analysis in the diagnosis of asbestos-related disease. *Hum Pathol* 13(4):381-392.

Churg A. (1989). The diagnosis of asbestosis. *Hum Pathol* 20(2):97-99.

Churg A, Wright JL, Vedal S. (1993) Fiber burden and patterns of asbestos-related disease in chrysotile miners and millers. *Am Rev Respir Dis* 148:25-31.

Churg A, Wright JL. (1994) Persistence of natural mineral fibers in human lungs: An overview.*Environ Health Perspect* 102(Suppl. 5):229-233.

de Klerk NH, Musk AW, Williams V, et al. (1996). Comparison of measures of exposure to asbestos in former crocidolite workers from Wittenoon Gorge, W. Australia. *Am J Ind Med* 30:579-587.

De Vuyst P, Dumortier P, Gevenois PA. (1997). Analysis of asbestos bodies in BAL from subjects with particular exposure. *Am J Ind Med* 31:699-704.

De Vuyst P, Dumortier P, Moulin E, et al. (1988) Asbestos bodies in bronchoalveolar lavage reflect lung asbestos body concentration. *Eur Resp J* 1:362-367.

De Vuyst P, Jedwab J, Dumortier P, et al. (1982) Asbestos bodies in bronchoalveolar lavage. *Am Rev Resp Dis* 126:972-976.

Dodson RF, Williams MG, Huang J, et al. (1999). Tissue burden of asbestos in nonoccupationally exposed individuals from east Texas. *Am J Ind Med* 35:281-286.

Dufresne A, Harrigan M, Masse S, et al. (1995). Fibers in lung tissues of mesothelioma cases among miners and millers of the township of Asbestos, Quebec. *Am J Ind Med* 27:581-592.

Dufresne A, Begin R, Churg A, et al. (1996a), Mineral fiber content of lungs in patients with mesothelioma seeking compensation in Quebec. *Am J Respir Crit Care Med* 153:711-718.

Dufresne A, Begin R, Masse S, et al. (1996b). Retention of asbestos fibres in lungs of workers with asbestosis, asbestosis and lung cancer, and mesothelioma in Asbestos township. *Occup Environ Med* 53:801-807.

Dumotier P, De Vuyst P, Strauss P, et al. (1990). Asbestos bodies in bronchoalveolar lavage fluids of brake lining and asbestos cement workers. *Br J Ind Med* 47:91-98.

Dumortier P, Cöplü L, de Maertelaer V, et al. (1998) Assessment of environmental asbestos exposure in Turkey by bronchoalveolar lavage. *Am J Respir Crit Care Med* 158:1815-1824.

McDonald JC, McDonald AD, Sebastien P, et al. (1988). Health of vermiculite miners exposed to trace amounts of fibrous tremolite. *Br J Ind Med* 45:63-634.

McDonald JC, Sebastien P, Case B.(1992). Ferruginous body counts in sputum as an index of past exposure to mineral fibers. *Ann Occup Hyg* 36(3):271-82.

NAS/NRC (1989) Biologic markers in reproductive toxicology. National Academy of Sciences/National Research Council. Washington, DC: National Academy Press, 15-35.

Nicholson WJ.(1987), Airborne levels of mineral fibres in the non-occupational environment. New York, NY: City University of New York, Mount Sinai School of Medicine, Division of Environmental and Occupational Medicine.

NIOSH. (1989a) Fibers - method 7400. In: *NIOSH manual of analytical methods. 3rd ed. Supplement.* Cincinnati, OH: National Institute for Occupational Safety and Health, 7400-1 - 7400-13.

NIOSH. (1989b) Asbestos fibers - method 7402. In: *NIOSH manual of analytical methods. 3rd ed. Supplement.* Cincinnati, OH: National Institute for Occupational Safety and Health, 7402-1 - 7402-8.

NIOSH. (1994b), Asbestos by TEM. In: *Manual of analytical methods, 4th ed*. Cincinnati, OH: U.S.Department of Health and Human Services, National Institute for Occupational Safety and Health.

NRC. 1984. National Research Council. Asbestiform fibers: nonoccupational health risks. Washington, DC: National Academy Press.

Roggli VL, Coin PG, MacIntyre NR, et al. (1994a) Asbestos content of bronchoalveolar lavage fluid: A comparison of light and scanning electron microscopic analysis. *Acta Cytol* 38:502-510,

Sebastien P, Armstrong B, Monchaux G, et al. (1988b). Asbestos bodies in bronchoalveolar lavage fluid and in lung parenchyma. *Am Rev Respir Dis* 137:75-78.

Sebastien P, McDonald JC, McDonald AD, et al, (1989) Respiratory cancer in chrysotile textile and mining industries: Exposure inferences from lung analysis. *Br J Ind Med* 46:180-187.

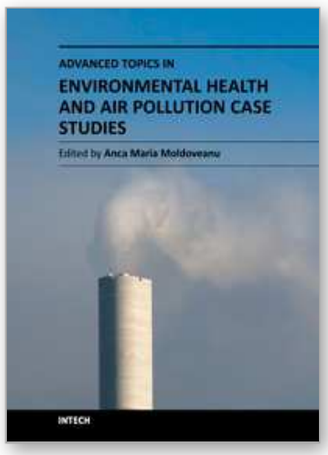
Teschler H, Friedrichs KH, Hoheisel GB, et al. (1994) Asbestos fibers in bronchoalveolar lavage and lung tissue of former asbestos workers. *Am J Resp Crit Care Med* 149:641-645.

Teschler H, Thompson AB, Dollenkamp R, et al. (1996). Relevance of asbestos bodies in sputum. *EurResp J* 9:680-686.

Tuomi T, Oksa P, Anttila S, et al. (1991b). Fibres and asbestos bodies in bronchoalveolar lavage fluids of asbestos sprayers. *Br J Ind Med* 49:480-485.

WHO (1998) Chrysotile asbestos*: Environmental Health Criteria*. Geneva: Switzerland: World HealthOrganization.

www.epa.gov/*asbestos*/ Environmental Protection Agency, June 2010,



Advanced Topics in Environmental Health and Air Pollution Case Studies

Edited by Prof. Anca Moldoveanu

ISBN 978-953-307-525-9
Hard cover, 470 pages
Publisher InTech
Published online 29, August, 2011
Published in print edition August, 2011

The book describes the effects of air pollutants, from the indoor and outdoor spaces, on the human physiology. Air pollutants can influence inflammation biomarkers, can influence the pathogenesis of chronic cough, can influence reactive oxygen species (ROS) and can induce autonomic nervous system interactions that modulate cardiac oxidative stress and cardiac electrophysiological changes, can participate in the onset and exacerbation of upper respiratory and cardio-vascular diseases, can lead to the exacerbation of asthma and allergic diseases. The book also presents how the urban environment can influence and modify the impact of various pollutants on human health.

INTECH
open science | open minds

InTech Europe
University Campus STeP Ri
Slavka Krautzeka 83/A
51000 Rijeka, Croatia
Phone: +385 (51) 770 447
Fax: +385 (51) 686 166
www.intechopen.com

InTech China
Unit 405, Office Block, Hotel Equatorial Shanghai
No.65, Yan An Road (West), Shanghai, 200040, China
中国上海市延安西路65号上海国际贵都大饭店办公楼405单元
Phone: +86-21-62489820
Fax: +86-21-62489821

IntechOpen