



Asbestos and Toxicological Concerns

Gregory A. Cade* and Hilda Oltean

University of Alabama at Birmingham, USA

Abstract

Environmental toxicology is a relatively young field of science concerned with the study of environmental pollutants in air, dust, sediment, soil, and water in the environment and their effects.

Since the 1970s, scientists concerned with toxins in the environment focused their research on the impacts of various chemical agents on ecosystems and health hazards associated with certain chemicals including asbestos. Asbestos, the foremost among toxic fugitive dust, has the ability to resist heat, fire, and electricity. Asbestos is a group of naturally occurring fibrous silicate materials with known toxicity resulting from exposure.

Although the precise mechanisms by which asbestos fibers cause toxic injury have not yet been fully determined, it is well-documented that fibers that persist within the lung or the mesothelium are capable of producing fibrogenic and tumorigenic effects in these tissues.

Keywords: Toxicology; Mesothelium; Asbestos; Actinolite; Amosite

Introduction

The causal association between asbestos exposure and malignant and nonmalignant illnesses has been established by various epidemiologic studies. The major routes of entry for asbestos into the body are inhalation and ingestion. Exposure via the skin is quite minimal. When inhaled, the asbestos fibers can make their way down to the smallest airway level into the lungs.

As a result of the body's attempts and mechanisms to remove the fibers from the lungs, these can be transported from the lung parenchyma out to the pleura and the lining of the lung. Thus, inhalation of the asbestos fibers is also associated with pleural lung diseases other than mesothelioma, including benign asbestos effusion, benign pleural plaques, and visceral pleural fibrosis.

Mechanisms of Action

Asbestos-related cancers became better recognized towards the end of the 20th century. The International Agency for Research on Cancer has classified five types of asbestos fibers—actinolite, amosite, anthophyllite, crocidolite, and tremolite as human carcinogens [1].

Asbestos fibers may easily be released into the air by the disturbance of asbestos-containing material, demolition work, repair or remodeling, maintenance of asbestos products and breakdown of natural deposits. They are particularly resistant

to chemical and thermal degradations and will remain virtually unchanged over long periods.

Fiber parameters including differences in chemical compositions and specifically fiber dimension are important parameters in determining the toxicological and pathological consequences of asbestos exposure. According to the World Health Organizations (WHO), current regulations focalize as per following fiber definition:

WHO fibers = any particle that has Fiber length $>5\mu\text{m}$ and Fiber width $<3\mu\text{m}$, Aspect ratio $>3:1$

In order to assess the toxic potential of asbestos fibers, recent studies showed that several air samples collected in buildings with asbestos-containing material contained short asbestos fibers (SAF) in a concentration of ≥ 10 fibers L^{-1} . In the literature, SAF is less pathogenic than long asbestos fibers (LAF). The presence of SAF in air samples appears as an indicator of the degradation of asbestos-containing material [2]. OSHA Standards indicates that 0.1 fibers per cubic centimeter (f/cc) is 'safe' for asbestos workers. The National Institute for Occupational Health and Safety (NIOSH) states that all levels of asbestos exposure have demonstrated asbestos-related disease. OSHA admits in its Asbestos Final Rule, that the 0.1 f/cc level leaves a remaining significant risk.

Mutagenesis plays an important role in the development of asbestos-induced neoplasms but not in benign conditions [3]. However, this does not rule out the role of factors such as inflammation, cellular toxicity, and oxidative stress as these mechanisms of action are involved in the induction of non-neoplastic diseases as well. Substantial evidence suggests the role of oxidative stress in the etiology of pulmonary fibrosis [4] and mesothelioma [5]. The DNA damage caused by asbestos fibers was thought to be a result of mechanical interference to chromosome segregation caused by these fibers during the process of mitosis [6]. However, the current belief is that DNA damage is a result of the oxidation that occurs after sustained inflammation and subsequent reactive oxygen species (ROS) production. A combination of genetic and environmental

Submitted: 19 April 2019 | **Accepted:** 24 May 2019 | **Published:** 28 May 2019

***Corresponding author:** Gregory A. Cade, University of Alabama at Birmingham, Alabama, USA, Tel: 760-696-7959; Email: Hilda@elglaw.com

Copyright: © 2019 Cade GA and Oltean H. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Citation: Cade GA, Oltean H (2019) Asbestos and Toxicological Concerns. Ann Environ Sci Ecol 3: 4.



factors has been implicated in the development of malignant mesothelioma. The Simian virus 40 T antigens has been found in most mesotheliomas and thus believed to be involved in the etiology of the disease [7]. The inhaled asbestos fibers move downwards into the lungs and eventually reach the pleural surface, where macrophages interact and engulf the asbestos fibers. The phagocytosis of elongated fibers of asbestos is thought to lead to ROS production and cytokine release, which contributes to DNA damage and subsequent transformation of mesothelial cells [8].

Occupational Toxicology

Although asbestos has been totally banned in over 60 countries, some nations still allow its use for certain products if certain specific regulations are adhered to. Workers and the general public can still be exposed during incorrectly performed removal procedures or in old buildings with degraded asbestos-containing materials. As a result of its desirable properties and low cost, asbestos has been widely used in a very large number of applications and commercial products.

Asbestos is a fibrous silicate mineral extracted from below Earth's surface using industrial mining techniques. Once mined from the ground, it gets refined and developed into industrial materials. The major producers of asbestos include China, Brazil, Kazakhstan, and Russia. The Clean Air Act of 1970, U.S. federal law classified asbestos as a hazardous air pollutant and gave the Environmental Protection Agency authority to set regulations on the use, management and removal of asbestos. About 1.3 million workers in the United States are still exposed to asbestos at their workplaces, and therefore, asbestos continues to be a health hazard.

Discussion

Epidemiologic studies of asbestos-exposed workers indicate that the deposition of inhaled asbestos fibers mainly occurs in lung tissue leading to the development of pulmonary disease including asbestosis, lung cancer, mesothelioma of the pleura or peritoneum. The signs and symptoms of malignant mesothelioma have been reported with a latency period very rare of less than 15 years and a median latency of 30 to 49 years [9].

Most of the meta-analyses and reviews published in the past four decades failed to clarify the relationship between asbestos exposure and the risk of gastrointestinal cancers involving the esophagus, stomach, intestines, and colorectal region [5-10]. Recent evidence indicates that asbestos exposure may have increased the risk of cancer in the gastrointestinal tract in some groups of asbestos workers [10]. Furthermore, certain studies did establish a positive link between asbestos exposure and gastrointestinal cancer [11-18]. Asbestos exposure related stomach cancer has been well documented [19-23], and also there has been evidence of an association between asbestos exposure and colon and esophageal cancer. In a 2013 study, an increased risk of gastrointestinal cancer was detected among chrysotile miners in China [25]. A total of 285 cases of colorectal cancer were found to be linked to asbestos exposure thus confirming the well-established relationship between asbestos exposure

and peritoneal cancers [24]. In addition, studies have suggested that asbestos fibers that have been inhaled can get translocated from the lungs to other organs including brain, liver, and kidney; however, fibers have been demonstrated only in the pleura and peritoneum [25,26]. It can be concluded that asbestos-related stomach cancer that develops after occupational exposure has been best validated to date [27-30].

Asbestos fibers can migrate downward through the diaphragm into the abdominal cavity. If ingested, asbestos fibers can locate in different parts of the gastrointestinal tract, in colonic tissue specifically. These fibers make their way into the kidneys causing scarring and therefore cancer. A strong correlation between occupational asbestos exposure and kidney cancer emerged when asbestos fibers were found in the kidney tumor of a 76-year-old man who used to cut asbestos cement panels at his workplace [31]. Additionally, he had also developed malignant peritoneal mesothelioma. The cancer had not metastasized to his kidneys, meaning that the two cancers were unrelated in this respect, but both were caused by asbestos exposure. Notably, recent studies have also linked asbestos exposure to cancer of the larynx [32] and ovaries [33-38].

Several factors determine a diagnosis of an asbestos-related disease, including the patient's possible exposure history, symptoms and test results. Getting a proper diagnosis from a specialist preferably specialized in asbestos-related diseases holds the potential to assure a new lease on life.

Concluding Remarks

At any life cycle stage, organisms can be exposed to various kinds of toxicants. Humans, throughout their development, have been exposed to various toxic environmental agents through air, soil, water and food. Asbestos-related diseases are prime examples of environmentally-related illnesses and significant occupational and public health concern. In the U.S. asbestos kills between 12,000 - 15,000 people per year. An estimated 107,000 people worldwide die each year from occupational asbestos-related lung cancer, asbestosis, and malignant mesothelioma [39-40].

Environmental contamination with asbestos was recognized as a serious issue; therefore, health risk warnings and recommendations concerning the need for control are required. Currently, exposure to chrysotile type of asbestos either in pure form or contaminated with tremolite is predominant. Extending the life of a patient with asbestos-related illness mainly depends on correct and early diagnosis. Anyone with a history of asbestos exposure should take special note of their symptoms and immediately seek medical attention.

References

1. IARC Metals, arsenic, dust and fibers. IARC Monogr Eval Carcinog Risks Hum. 2012; 100: 11-465.
2. Guillaume Boulanger, Pascal Andujar, Jean-Claude Pairon, Marie-Annick Billon-Galland, Chantal Dion, et al. Quantification of short and long asbestos fibers to assess asbestos exposure: a review of fiber size toxicity. Environ Health. 2014; 13-59.

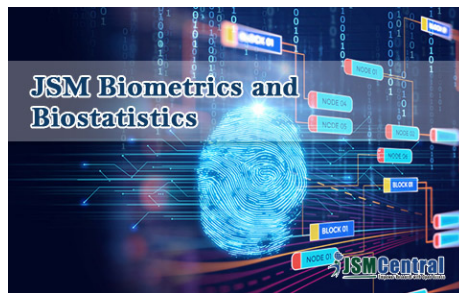
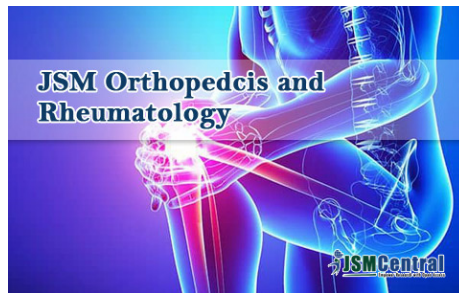


3. Huang SXL, Jaurand MC, Kamp DW, Whysner J, Hei TK. Role of mutagenicity in mineral fiber-induced carcinogenicity and other diseases. *J Toxicol Environ Health B Crit Rev.* 2011; 14: 179-245.
4. Cheres P, Morales-Nebreda L, Kim SJ, Yeldandi A, Williams DB, Cheng Y, et al. Asbestos-induced pulmonary fibrosis is augmented in 8-oxoguanine DNA glycosylase knockout mice. *Am J Respir Cell Mol Biol.* 2015; 52: 25-36.
5. Sezgi C, Taylan M, Sen HS, Evliyaoğlu O, Kaya H, Abakay O, et al. Oxidative status and acute phase reactants in patients with environmental asbestos exposure and mesothelioma. *Scientific World Journal.* 2014.
6. Lamote K, Nackaerts K, van Meerbeeck JP. Strengths, weaknesses, and opportunities of diagnostic breathomics in pleural mesothelioma-a hypothesis. *Cancer Epidemiol. Biomarkers Prev.* 2014; 23: 898-908.
7. Procopio A, Marinacci R, Marinetti MR, Strizzi L, Paludi D, Iezzi T, et al. SV40 expression in human neoplastic and non-neoplastic tissues: perspectives on diagnosis, prognosis and therapy of human malignant mesothelioma. *Dev Biol Stand.* 1998; 94: 361-367.
8. Ramos-Nino ME, Testa JR, Altomare DA, Pass HI, Carbone M, Bocchetta M, et al. Cellular and molecular parameters of mesothelioma. *J Cell Biochem.* 2006; 98: 723-734.
9. S van der Bij, H. Koffijberg, J.A. Burgers, P. Baas, van de Vijver MJ, de Mol BA, et al. Prognosis and prognostic factors of patients with mesothelioma: a population-based study. *Br J Cancer.* 2012; 107: 161-164.
10. Seok Jo Kim, David Williams, Paul Cheres, and Savid W Kamp. Asbestos-induced gastrointestinal cancer: an update. *J Gastrointest Dig Syst.* 2013; 3: 135.
11. Miller AB. Asbestos fibre dust and gastro-intestinal malignancies. Review of literature with regard to a cause/effect relationship. *J Chronic Dis.* 1978; 31: 23-33.
12. Morgan RW, Foliart DE, Wong O. Asbestos and gastrointestinal cancer. A review of the literature. *West J Med.* 1985; 143: 60-65.
13. Neugut AI, Wylie P. Occupational cancers of the gastrointestinal tract. I. Colon, stomach, and esophagus. *Occup Med.* 1987; 2: 109-135.
14. Weiss W. The lack of causality between asbestos and colorectal cancer. *J Occup Environ Med.* 1995; 37: 1364-1373.
15. Weiss W. Asbestos and colorectal cancer. *Gastroenterology.* 1990; 99: 876-884.
16. Homa DM, Garabrant DH, Gillespie BW. A meta-analysis of colorectal cancer and asbestos exposure. *Am J Epidemiol.* 1994; 139: 1210-1222.
17. Fang R, Le N, Band P. Identification of occupational cancer risks in British Columbia, Canada: a population-based case-control study of 1,155 cases of colon cancer. *Int J Environ Res Public Health.* 2011; 8: 3821-3843.
18. Aliyu OA, Cullen MR, Barnett MJ, Balmes JR, Cartmel B, Redlich CA, et al. Evidence for excess colorectal cancer incidence among asbestos-exposed men in the Beta-Carotene and Retinol Efficacy Trial. *Am J Epidemiol.* 2005; 162: 868-878.
19. Bunderson-Schelvan M, Pfau JC, Crouch R, Holian A. Nonpulmonary outcomes of asbestos exposure. *J Toxicol Environ Health B Crit Rev.* 2011; 14: 122-152.
20. Kjaerheim K, Ulvestad B, Martinsen JI, Andersen A. Cancer of the gastrointestinal tract and exposure to asbestos in drinking water among lighthouse keepers (Norway) *Cancer Causes Control.* 2005; 16: 593-598.
21. Andersen A, Glatte E, Johansen BV. Incidence of cancer among lighthouse keepers exposed to asbestos in drinking water. *Am J Epidemiol.* 1993; 138: 682-687.
22. Kanarek MS, Conforti PM, Jackson LA, Cooper RC, Murchio JC. Asbestos in drinking water and cancer incidence in the San Francisco Bay area. *Am J Epidemiol.* 1980; 112: 54-72.
23. Polissar L, Severson RK, Boatman ES. Cancer risk from asbestos in drinking water: summary of a case-control study in western Washington. *Environ Health Perspect.* 1983; 53: 57-60.
24. Clin B, Morlais F, Launoy G, Guizard AV, Dubois B, Bouvier V, et al. Cancer incidence within a cohort occupationally exposed to asbestos: a study of dose-response relationships. *Occup Environ Med.* 2011; 68: 832-836.
25. Miserocchi G, Sancini G, Mantegazza F, Chiappino G. Translocation pathways for inhaled asbestos fibers. *Environ Health.* 2008; 7: 4.
26. Suzuki Y, Kohyama N. Translocation of inhaled asbestos fibers from the lung to other tissues. *Am J Ind Med.* 1991; 19: 701-704.
27. Ramazzini C. Asbestos is still with us: repeat call for a universal ban. *Arch Environ Occup Health.* 2010; 65: 121-126.
28. Sun TD, Chen JE, Zhang XJ, Li XY. Cohort studies on cancer mortality of digestive system among workers exposed to asbestos: a meta-analysis. *Zhonghua Lao Dong Wei Sheng Zhi Ye Bing ZaZhi.* 2008; 26: 605-608.
29. Raffn E, Lynge E, Juel K, Korsgaard B. Incidence of cancer and mortality among employees in the asbestos cement industry in Denmark. *Br J Ind Med.* 1989; 46: 90-96.
30. Szeszenia-Dabrowska N, Wilczynska U, Szymczak W, Laskowicz K. Environmental exposure to asbestos in asbestos cement workers: a case of additional exposure from indiscriminate use of industrial wastes. *Int J Occup Med Environ Health.* 1998; 11: 171-177.
31. Candura SM, Boeri R, Teragni C, Chen Y, Scafa F. Renal cell carcinoma and malignant peritoneal mesothelioma after occupational asbestos exposure: case report. *Med Lav.* 2016; 107: 172-177.
32. Straif K, Benbrahim-Tallaa L, Baan R, Grosse Y, Secretan B, El Ghissassi F, et al. WHO International Agency for Research on Cancer Monograph Working Group. A review of human carcinogens-part C: metals, arsenic, dusts, and fibres. *Lancet Oncol.* 2009; 10: 453-455.
33. M. Constanza Camargo, Leslie T. Stayner, Kurt Straif, Margarita Reina, Umaima Al-Alem, Paul A. Demers, et al. *Environ Health Perspect.* 2011; 119: 1211-1217.
34. Seidman H, Selikoff J, Gelb SK. Mortality experience of amosite asbestos factory workers: dose-response relationships 5 to 40 years after onset of short-term work exposure. *Am J Ind Med.* 1986; 10: 479-514.
35. Hilt B, Langård S, Andersen A, Rosenberg J. Asbestos exposure, smoking habits, and cancer incidence among production and maintenance workers in an electrochemical plant. *Am J Ind Med.* 1985; 8: 565-577.
36. Chow WH, McLaughlin JK, Malker HS, Weiner JA, Ericsson JL, Stone BJ, et al. Occupation and stomach cancer in a cohort of Swedish men. *Am J Ind Med.* 1994; 26: 511-520.
37. Gerhardsson de Verdier M, Plato N, Steineck G, Peters JM. Occupational exposures and cancer of the colon and rectum. *Am J Ind Med.* 1992; 22: 291-303.
38. Garabrant DH, Peters RK, Homa DM. Asbestos and colon cancer: lack of association in a large case-control study. *Am J Epidemiol.* 1992; 135: 843-853.



39. Wang X, Courtice MN, Lin S. Mortality in chrysotile asbestos workers in China. *Curr Opin Pulm Med.* 2013; 19: 169-173.
40. Clin B, Morlais F, Launoy G, Guizard AV, Dubois B, Bouvier V, et al.

Cancer incidence within a cohort occupationally exposed to asbestos: a study of dose-response relationships. *Occup Environ Med.* 2011; 68: 832-836.



Our motto is to advance scientific excellence by promoting open access. We are committed in the widest possible dissemination of research and uplift future innovation



[Submit Manuscript](#)