# ENVIS NEWSLETTER

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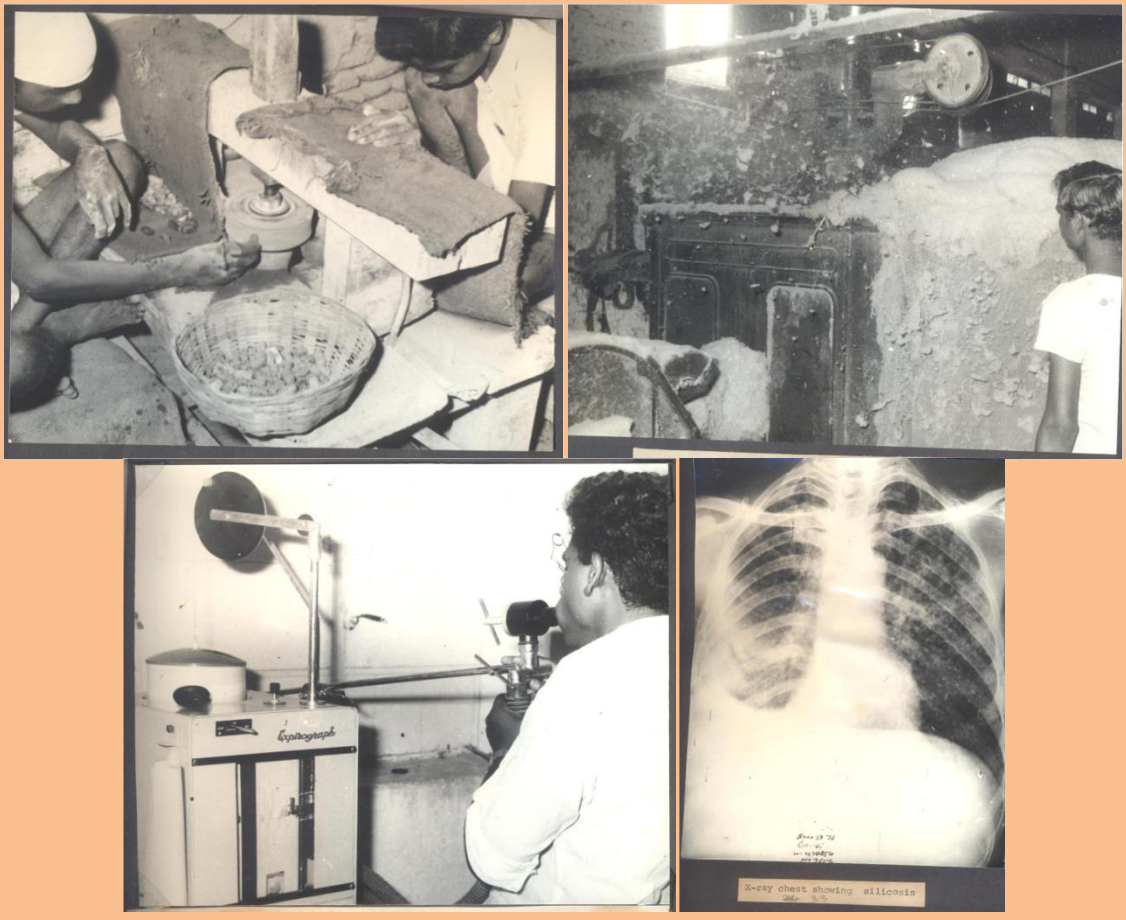
# *Special Issue on Occupational Lung Disease*




## CSIR- Indian Institute of Toxicology Research
## Lucknow, India

## IN THIS ISSUE

### ODDS & ENDS



**Editorial**

Various name for Occupational Lung Disease are Occupational Asthma or Silicosis or Farmer's Lung or Occupational Lung Cancer or Asbestosis This is a disease contracted as a result of an exposure of a person to work place risk factors. In this case there is a causal relationship between the disease and the exposure of the worker to certain hazardous agents at the workplace. Relationship is normally established on the basis of clinical and pathological data, occupational history and job analysis, the disease is usually caused by repeated and long-term exposure to particular type of toxic substances. Here it is important to mention that Article 21 of the Indian Constitution guarantees the protection of life and personal liberty of a person, there are various Supreme Court judgments, that upheld the right to employees' health under this "right to life". It is a fact that occupational lung diseases are preventable. It may be possible to slow down the progression of symptoms or lower your risk of developing complications. As per the ILO estimate 2.34 million people die each year from work-related accidents and diseases. Of these, the vast majority -an estimated 2.02 million- die from a wide range of work-related diseases. Of the estimated 6,300 work-related deaths that occur every day, 5,500 are caused by various types of work- related diseases. The ILO also estimates that 160 million cases of non-fatal work-related diseases occur annually. Technological, social and organizational changes in the environment of workplace brought about by vast globalization have also been accompanied by various emerging risks and challenges. Some traditional risks have declined due to improved safety measures, better regulations and advanced technologies, but till than these diseases continue to take an heavy toll on workers' health. Yet, globally, more than half of all countries still do not collect adequate statistics for occupational diseases. Available data concern mainly injuries and fatalities. In Indian system occupational health is not governed under primary healthcare, but it is under the purview of Labour Ministry, though it should have been governed by the Ministry of Health. For fund allocation occupational health has to compete with the budget of health as well as curative health. Now with this scenario, there is an urgent need to understand properly the risk factors for present occupational hazards. It is high time that India should formulate occupational health safety (OHS) legislation with proper and adequate enforcement machinery and also establish centres of excellence in the field of occupational medicine.

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Lucknow India

**Pneumoconioses.**

Occupational lung diseases are caused or made worse by exposure to harmful substances in the work-place. "Pneumoconiosis" is the term used for the diseases associated with inhalation of mineral dusts. While many of these broad-spectrum substances may be encountered in the general environment, many occur in the work-place for greater amounts as a result of industrial processes; therefore, a range of lung reactions may occur as a result of work-place exposure. Physicians in metropolitan cities are likely to encounter pneumoconiosis for two reasons: (i) patients coming to seek medical help from geographic areas where pneumoconiosis is common, and (ii) pneumoconiosis caused by unregulated small-scale industries that are housed in poorly ventilated sheds within the city. A sound knowledge about the various pneumoconioses and a high index of suspicion are necessary in order to make a diagnosis. Identifying the disease is important not only for treatment of the individual case but also to recognise and prevent similar disease in co-workers.

[Indian J Chest Dis Allied Sci. 2013 Jan-Mar; 55(1):25-34.]

**Giant cell interstitial pneumonia: an unusual finding in a case of preoperative death.**

Giant cell interstitial pneumonia (GIP) is an exceedingly rare, debatable, perplexing, occupational lung disease, which most commonly affects individuals exposed to hard metal dust. Authors report a case of GIP in a 60-year-old man, scheduled for coronary artery bypass graft surgery and died during induction of general anesthesia despite all efforts to resuscitate him. Patient's relatives lodged complaint with the police alleging the negligence by the attending physicians. Despite inaccessible data pertaining to the occupation, clinical history, and radiographic findings, the diagnosis was GIP due to the presence of intra-alveolar, bizarre, "cannibalistic" multinucleated giant cells-the histologic sine qua non of GIP. To the best of knowledge, this is the first case report of GIP in the world literature that was diagnosed on histopathologic examination of lung tissue obtained at medicolegal autopsy.

[Am J Forensic Med Pathol. 2013 Jun; 34(2):110-4.]

**Adverse respiratory effects associated with cadmium exposure in small-scale jewellery workshops in India**.

Cadmium (Cd) is an important metal with both common occupational and environmental sources of exposure. Although it is likely to cause adverse respiratory effects, relevant human data are relatively sparse. A cross-sectional study of 133 workers in jewellery workshops using Cd under poor hygienic conditions and 54 referent jewellery sales staffs was performed. Authors assessed symptoms, performed spirometry, measured urinary Cd levels in all study subjects and quantified airborne total oxidant contents for 35 job areas in which the studied workforce was employed. Author tested the association of symptoms with exposure relative to the unexposed referents using logistic regression analysis, and tested the association between urinary Cd levels and lung function using multiple regression analysis, adjusting for demographics, smoking and area-level airborne oxidants. Exposed workers had 10 times higher urinary Cd values than referents (geometric mean 5.8 vs 0.41 μg/dl; $p<0.01$). Of the exposed subjects, 75% reported respiratory tract symptoms compared with 33% of the referents (OR=3.1, 95% CI 1.4

to 7.3). Forced vital capacity (FVC) and forced expiratory volume in 1 s (FEV1) were also lower among the exposed workers than the referents (>600 ml decrement for each, $p<0.001$). For every 1 µg increase in urinary Cd there was a 34 ml decrement in FVC and a 39 ml decrement in FEV1 ($p<0.01$), taking into account other covariates including workplace airborne oxidant concentrations. This cohort of heavily exposed jewellery workers experienced frequent respiratory symptoms and manifested a marked deficit in lung function, demonstrating a strong response to Cd exposure.
[Thorax. 2013 Jun;68(6):565-70.]

**Silicosis in India: past and present.**

This particular review focuses on the burden of the problem of silicosis and its clinical manifestations reported from India. In recent estimates from India, there are over 3 million workers exposed to silica dust, whilst 8.5 million more work in construction and building activities, similarly exposed to quartz. Several recent reports on lung function assessment show both restrictive and obstructive patterns. Tuberculosis is a common complication reported in Indian studies. Occasionally, silico-mycosis, lung cancer and connective tissue disorders in association with silicosis are also reported. The National Human Rights Commission (NHRC) in response to the direction from the Supreme Court of India has made several recommendations on preventive, remedial and rehabilitative measures. The NHRC has been asked to work with various stakeholders such as individual organizations, state and central governments and other agencies to implement the measures. Silicosis is a common occupational disorder seen all over India, particularly in the Central and Western States. It is an important cause of respiratory morbidity. The problem has been highlighted on the national level as a major human-rights concern in India.
[Curr Opin Pulm Med. 2013 Mar;19(2):163-8.]

**Occupational safety and health in India: now and the future.**

India, a growing economy and world's largest democracy, has population exceeding 1.2 billion. Out of this huge number, 63.6% form working age group. More than 90% work in the informal economy, mainly agriculture and services. Less than 10% work in the organized sector; mainly industry, mining and some services. New service industries like Information Technology (IT), Business Process Outsourcing (BPO) are increasing rapidly; so is the proportion of females in the workforce. The occupational safety and health (OSH) scenario in India is complex. Unprecedented growth and progress go hand in hand with challenges such as huge workforce in unorganized sector, availability of cheap labour, meagre public spending on health, inadequate implementation of existing legislation, lack of reliable OSH data, shortage of OSH professionals, multiplicity of statutory controls, apathy of stakeholders and infrastructure problems. The national policy on OSH at workplace, adopted by the government in 2009, is yet to be implemented. Some of the major occupational risks are accidents, pneumoconiosis, musculoskeletal injuries, chronic obstructive lung diseases; pesticide poisoning and noise induced hearing loss. The three most important OSH needs are: 1. legislation to extend OSH coverage to all sectors of working life including the unorganized sector; 2. spreading the awareness about OSH among stakeholders; 3. development of OSH infrastructure and OSH professionals. Other issues include integration of

occupational health with primary health care.
[Ind Health. 2012;50(3):167-71.]

**Combined pulmonary fibrosis and emphysema in a welder.**
Combined pulmonary fibrosis and emphysema (CPFE) syndrome is an uncommon entity characterised by emphysema of the upper lobes and diffuse fibrosis of the lower lobes and carries a bad prognosis with the onset of pulmonary hypertension. Lung involvement due to exposures suffered by welders is generally considered benign though, rarely, a diffuse interstitial fibrotic disease has been reported. CPFE syndrome has however never been reported in welders. A 65-year-old man, welder by occupation and an ex-smoker, presented with progressive exertional dyspnoea associated with dry cough noticed for the last four months. On examination, there was mild tachypnea, clubbing and bilateral basal velcro crepitations on chest auscultation. Lung function test revealed mild mixed ventilatory impairment with severe diffusion defect. HRCT chest showed bilateral upper lobe emphysema and diffuse interstitial fibrosis in the lower lobes. Transbronchial lung biopsy revealed interstitial fibrosis, chronic inflammation and iron deposits. A diagnosis of combined pulmonary fibrosis with emphysema (CPFE) with interstitial pulmonary siderofibrosis (IPS) was established. A review of literature did not show any other report of a similar nature.
[Monaldi Arch Chest Dis. 2012 Mar; 77(1):26-8.]

**Evaluation of cytotoxic, genotoxic and inflammatory responses of micro- and nano-particles of granite on human lung fibroblast cell IMR-90.**
Occupational exposure of granite workers is well known to cause lung impairment and silicosis. Toxicological profiles of different size particles of granite dust, however, are not yet understood. Present evaluation of micro- and nano-particles of granite dust as on human lung fibroblast cells IMR-90, revealed that their toxic effects were dose-dependent, and nanoparticles in general were more toxic. In this study authors first demonstrated that nanoparticles caused oxidative stress, inflammatory response and genotoxicity, as seen by nearly 2 fold induction of ROS and LPO, mRNA levels of TNF-α and IL-1β, and induction in micronuclei formation. All these were significantly higher when compared with the effect of micro particles. Thus, the study suggests that separate health safety standards would be required for granite particles of different sizes.
[Toxicol Lett. 2012 Feb 5; 208(3):300-7.]

**A review on the occupational health and social security of unorganized workers in the construction industry.**
Construction is one of the important industries employing a large number of people on its workforce. A wide range of activities are involved in it. Due to the advent of industrialization and recent developments, this industry is taking a pivotal role for construction of buildings, roads, bridges, and so forth. The workers engaged in this industry are victims of different occupational disorders and psychosocial stresses. In India, they belong to the organized and unorganized sectors. However, data in respect to occupational health and psychosocial stress are scanty in author's country. It is true that a sizable number of the workforce is from the unorganized sectors - the working hours are more than the stipulated hours of work - the work place is not proper - the working conditions are non-congenial in most of the cases and involve risk factors. Their wages are also not adequate, making it difficult for them to run their families. The hazards

include handling of different materials required for construction, and exposure to harsh environmental conditions like sun, rain, and so on. On account of this, in adverse conditions, it results in accidents and adverse health conditions cause psychosocial strain and the like. They are victims of headache, backache, joint pains, skin diseases, lung disorders like silicosis, other muscular skeletal disorders, and so on. The repetitive nature of the work causes boredom and the disproportionate earning compared to the requirements puts them under psychological stress and strain and other abnormal behavioural disorders. The Government of India has realized the importance of this industry and has promulgated an Act in 1996. The state government are being asked to adhere to this, although only a few states have partially enforced it. In this article, attempts have been made to review some of the important available articles for giving a broad idea of the problem and for furtherance of research in this field.
[Indian J Occup Environ Med. 2011 Jan; 15(1):18-24.]

**ARDS following inhalation of hydrochloric acid.**

The clinical spectrum of Inhalation injury can range from mild cough to a devastating ARDS. Authors herewith present a patient who is a goldsmith by occupation and his work consists of dissolving gold in Hydrochloric acid. He had accidentally inhaled fumes of Hydrochloric acid and presented with cough and breathlessness, later on required mechanical ventilation for ARDS and improved. This highlights the importance of not to neglect mild symptoms like cough and dyspnea in such a scenario which may have some hidden catastrophe.
[J Assoc Physicians India. 2011 Feb; 59:115-7.]

**Nanotoxicity of dolomite mineral of commercial importance in India.**

The risk of occupational exposure to dolomite, an important mineral exists both in organized as well as unorganized sectors. Toxicological profiles of bulk dolomite are meagrely known in general and its nanotoxicity in particular. Effects of micro- and nano particles on cell viability, LDH leakage and markers of oxidative stress were observed. The study indicated that cytotoxicity of dolomite nanoparticles is significantly higher than the microparticles. The study thus suggests for the prescription of exposure limit for nanodolomite in the best interest of health of workers at risk of exposure under mining, milling and industrial environment.
[J Biomed Nanotechnol. 2011 Feb; 7(1):114-5.]

**Quantitative assessment of elemental carbon in the lungs of never smokers, cigarette smokers, and coal miners.**

Inhalation exposure to particulates such as cigarette smoke and coal dust is known to contribute to the development of chronic lung disease. The purpose of this study was to estimate the amount of elemental carbon (EC) deposits from autopsied lung samples from cigarette smokers, miners, and control subjects and explore the relationship between EC level, exposure history, and the extent of chronic lung disease. The samples comprised three subgroups representing never smokers (8), chronic cigarette smokers (26), and coal miners (6). Following the dissolution of lung tissue, the extracted EC residue was quantified using a thermal-optical transmission (TOT) carbon analyzer. Mean EC levels in the lungs of the control group were 56.68 ± 24.86 (SD) μg/g dry lung weight. Respective mean EC values in lung samples from the smokers and coal miners were 449.56 ± 320.3 μg/g and 6678.2 ± 6162 μg/g. These values were significantly higher than those obtained from the never-smoker

group. EC levels in the lung and pack-years of cigarette smoking correlated significantly, as did EC levels and the severity of small airway disease. This study provides one of the first quantitative assessments of EC in human lungs from populations at high relative risk for the development of chronic lung disease.
[J Toxicol Environ Health A. 2011; 74(11):706-15.]

**Bronchial anthracofibrosis: an emerging pulmonary disease due to biomass fuel exposure.**
The objective of this study was to document current knowledge of bronchial anthracofibrosis (BAF), an emerging pulmonary disease recognised just over a decade ago; to highlight the demographic profile, and clinical, radiological and bronchoscopic features peculiar to BAF; and to discuss the postulated causes and clinical conditions associated with BAF, emphasising the need to characterise and recognise it as a distinct clinical disorder. An extensive search of the literature was performed in Medline/PubMed and other databases with key terms 'anthracosis', 'biomass fuels', 'bronchial anthracofibrosis' and 'pulmonary tuberculosis'. The bibliographies of papers identified were searched for further relevant articles. A total of 17 studies and six case series/reports describing 1320 patients with bronchoscopically confirmed BAF were documented. BAF was predominantly observed in elderly housewives in rural areas with prolonged exposure to biomass fuel, and was associated with respiratory diseases such as tuberculosis (TB), chronic obstructive pulmonary disease, pneumonia and malignancy. Exposure to biomass fuel smoke emerged as the main causative factor, but the possibility of an occupational lung disorder was also raised. Characteristic clinical, thorax computed tomography and bronchoscopic features of BAF were identified and its differentiation from endobronchial TB and bronchogenic carcinoma was described. As a pulmonary disease, BAF is yet to be highlighted in both developing and industrialised countries. BAF is currently diagnosed only on bronchoscopy, whereas a suitable non-invasive diagnostic modality would enable rapid diagnosis and increased recognition. Approaches for patients with BAF need to be developed and the serious hazards of biomass fuel use should be emphasised.
[Int J Tuberc Lung Dis. 2011 May; 15(5):602-12.]

**The agate industry and silicosis in Khambhat, India.**
Agate stones have been shaped and polished into beads and other decorative items for thousands of years in Khambhat, India. Agate is a silicate quartz that produces a fine dust when shaped and polished. The people who shape and polish the stones in workshops in their homes are being sickened with silicosis, as are their families and neighbours. These home-based workshops are unregulated and the workers and their families have no access to occupational health services or workers' compensation when they become ill. Occupational health activists have tried to find an effective strategy to confront these working conditions and protect the health and livelihood of the agate workers. They have had limited success, and huge challenges remain.
[New Solut. 2011; 21(1):117-39.]

## DID YOU KNOW

One of the oldest known occupational diseases, silicosis is caused by silica dust inhalation. The full name of this disease is 45 letters long (the longest word in the English language) - **pneumonoultramicroscopicsilicovolcanokoniosis**. Silica is abundant in the earth's crust, many types of occupations pose threat for inhalation of silica dust. Although silicosis is fatal and has no cure, it can be prevented if the inhalation of silica dust is minimized through preventive measures. Silica dust measuring two to five micron size, after inhalation, travels up to the alveoli of the lungs. larger size dust particles are filtered through the nose or thrown out by cilia in the windpipe. Silica dust is highly toxic and it is difficult to monitor its presence as it has no smell and also does not offers any warning to the worker.

Asbestosis (caused by prolong exposure to asbestos) is serious, long-term breathing disorder that permanently scars the lungs and makes it dificult to breathe. Reason for asbestosis is breathing in minute fibres of asbestos, a mineral used for insulation, vinyl floor tiles, cement, brake linings and many other products. Asbestosis is a kind of pneumoconiosis ("pneumoconiosis" or "pneumoconioses" is the general term for diseases caused by breathing in mineral dust). It is also called pulmonary fibrosis.

How does asbestos damage the lungs? When a a person works on asbestos by cutting, grinding or any other manner, minute asbestos fibres fly and accumulate the air and they stay there for a long time. People who breathe in these tiny asbestos fibres, can get stuck these deep in their lungs. In the lungs these fibres damage the alveoli and air. In normal course alveoli inflate and deflate like a balloon, by inhaling oxygen and exhaling of waste gas (carbon dioxide). Healthy alveoli are nice and stretchy. Asbestos causes damage and scarring and thus alveoli become stiff. When alveoli are stiff, lungs find it hard to take in oxygen, and the muscles of chest have to work hard to force the air in and out of the lungs. Here it is important to mention that family members of people who work closely with a large amount of asbestos may also be at risk, because the worker may bring home asbestos fibres on his clothes.

Jobs that put people at risk of asbestosis:

- asbestos and talc miners
- shipyard workers
- construction and demolition workers
- power plant workers
- auto brake mechanics
- workers who make asbestos-containing products such as firebricks, fire-retardant paint and asbestos cement
- firefighters and other emergency rescue workers
- sailors, navy servicemen and women who work and sleep on ships insulated with asbestos
- boilermakers
- steamfitters
- plumbers
- welders
- janitors

## CURRENT CONCERN

As per an ILO estimate 2.34 million people die each year from work-related accidents and diseases. Of these, the vast majority -an estimated 2.02 million- die from a wide range of work-related diseases. Of the estimated 6,300 work-related deaths that occur every day, 5,500 are caused by various types of workrelated diseases. The ILO also estimates that 160 million cases of non-fatal work-related diseases occur annually Occupational diseases cause huge suffering and loss in the world of work. Yet, occupational or work-related diseases remain largely invisible in comparison to industrial accidents, even though they kill six times as many people each year. Furthermore, the nature of occupational diseases is altering rapidly: technological and social changes, along with global economic conditions, are aggravating existing health hazards and creating new ones. Well-known occupational diseases, such as pneumoconioses, remain widespread, while relatively new occupational diseases, such as mental and musculoskeletal disorders (MSDs), are on the rise. While much progress has been made in addressing the challenges of occupational diseases, there is an urgent need to strengthen the capacity for their prevention in national OSH systems. It is the need of time that there should be a collaborative effort of government and employer and worker as well as workers' organizations, to fight against this epidemic which is hidden. It will have to appear prominently in global as wll as national agendas for safety and health of working population. Diagnosis, treatment and prevention of diseases of occupational origin requires specific knowledge and experience that are not available adequately in most of the developing nations, this is the major constrain in data collection and occupational health surveillance. Moreover, in some countries, like India, responsibility for health and safety at work is divided between labour and health ministries and this dual responsibility leads to difficulty in data collection.

## REGULATORY TRENDS

Working conditions and the nature of employment tend to have major repercussions on the health of a worker. The concept of 'Occupational health' has evolved from work-related ailments. Occupational health broadly means any injury, impairment or disease affecting a worker or employee during his course of employment. It not only deals with work-related disorders but also encompasses all factors that affect community health within it. The inadequate surveillance of employees is the most important reason for increased prevalence of work related and other non-communicable life style diseases at work place. Safety and health occupy a significant place in India's Constitution, which prohibits employment of children under the age of 14 in factories, mines and hazardous occupations. This policy aims to protect the health and strength of all workers by discouraging employment in occupations unsuitable to the worker's age and strength. It is the policy of the State to make provisions to secure just and humane conditions at work.

(http://www.articlesbase.com/diseases-and-conditions-articles/importance-of-occupational-health-in-india-5600954.html)

The Constitution provides a broad framework under which policies and programmes for occupational health and safety can be established. Legislation on occupational health and safety has existed in India for over 50 years. The principal health and safety laws are based on the British Factories Act. The Factories Act, 1948 has been amended from time to time, especially after the Bhopal gas disaster, which could have been prevented. The amendment demanded a shift away from dealing with disaster (or disease) to prevention of its occurrence. The Factories (Amendment) Act came into force on December 1, 1987. A special chapter on occupational health and safety to safeguard workers employed in hazardous industries was added. In this chapter, pre-employment and periodic medical examinations and monitoring of the work environment are mandatory for industries defined as hazardous under the Act. A maximum permissible limit has been laid down for a number of chemicals.
**(**http://infochangeindia.org/agenda/occupational-safety-and-health/status-of-occupational-safety-and-health-in-india.html**)**

The Act is implemented by state factory inspectorates, supported by industrial hygiene laboratories. There are similar provisions under the Mines Act. The Factories Act is applicable only to factories that employ 10 or more workers; it covers only a small proportion of workers.

Regulations dealing with Occupational health and safety
Factories Act, 1948, amended in 1954, 1970, 1976, 1987
Mines Act, 1952
Dock Workers (Safety, Health and Welfare) Act, 1986
Plantation Labour Act, 1951
Explosives Act, 1884
Petroleum Act, 1934
Insecticide Act, 1968
Indian Boilers Act, 1923
Indian Electricity Act, 1910
Dangerous Machines (Regulations) Act, 1983
Indian Atomic Energy Act, 1962
Radiological Protection Rules, 1971
Manufacture, Storage and Import of Hazardous Chemicals Rules, 1989

**THE FIRST SCHEDULE TO THE FACTORIES ACT, 1948**

LIST OF INDUSTRIES INVOLVING HAZARDOUS PROCESSES

1. Ferrous Metallurgical Industries. Integrated Iron and Steel. Ferro-alloys. Special Steels.
2. Non-ferrous Metallurgical Industries. Primary Metallurgical Industries, namely zinc, lead, copper, manganese and aluminium.
3. Foundries (ferrous and non-ferrous). Castings and forgings including cleaning or smoothening/roughening by sand and shot blasting.
4. Coal (including coke) Industries. Coal, Lignite, Coke, etc. Fuel Gases (including Coal Gas, Producer Gas, Water Gas).
5. Power Generating Industries.
6. Pulp and paper (including paper products) Industries.
7. Fertiliser Industries.Nitrogenous.Phosphatic.Mixed.
8. Cement Industries. Portland Cement (including slag cement, puzzolona cement and their products).
9. Petroleum Industries.Oil Refining.Lubricating Oils and Greases.
10. Petro-chemical Industries.
11. Drugs and Pharmaceutical Industries.Narcotics, Drugs and Pharmaceuticals.
12. Fermentation Industries (Distilleries and Breweries).

13. Rubber (Synthetic) Industries.
14. Paints and Pigment Industries.
15. Leather Tanning Industries.
16. Electro-plating Industries.
17. Chemical Industries.
18. Insecticides, Fungicides, Herbicides and other Pesticides Industries.
19. Synthetic Resin and Plastics.
20. Man-made Fibre (cellulosic and non-cellulosic) industry.
21. Manufacture and repair of electrical accumulators.
22. Glass and Ceramics.
23. Grinding or glazing of metals.
24. Manufacture, handling and processing of asbestos and its products.
25. Extraction of oils and fats from vegetable and animal sources.
26. Manufacture, handling and use of benzene and substances containing benzene.
27. Manufacturing processes and operations involving carbon disulphide.
28. Dyes and dyestuff including their intermediates.
29. Highly flammable liquids and gases.

## ON THE LIGHTER SIDE

While sitting in lab the other day, a discussion broke out about weekend plans. Naturally, these plans consisted mainly of drinking. Or, as one person referred to it, "killing brain cells". Someone observed that, even though alcohol supposedly kills brain cells, they seem to do better in classes during which they regularly take in modest amounts of alcohol than in classes where they don't. This turned out to be a fairly common phenomenon.

Since this was during a cell bio class, and we'd been studying biochemical pathways all day, authorsstarted theorizing about different mechanisms of how, exactly, this works. Authorscame up with two good ones: the "natural selection" mechanism, and the "exfoliation" mechanism.

The natural selection theory states that drinking: alcohol kills off the weak, old, and slow brain cells, leaving only the more fit and effective ones.

The exfoliation theory holds that alcohol removes the old, crusty, dead layer of brain cells, exposing young fresh ones which are much faster. Sort of like peeling an onion.

Either hypothesis fits the data, but in order to determine which is the true explanation, more "field study" will have to be done... such is the price of science.

## FORTHCOMING CONFERENCES

**Oncology Imaging for Drug Development,** 3/12/2014 to 3/13/2014, United Kingdom**,**

SMi's 10th annual Oncology Imaging for Drug Development conference will provide attendees with a comprehensive insight into the industry and its future. Authors will be considering the current landscape of personalised imaging within the field of oncology, as research suggests healthcare that is inherently more ‘personal’ is the way forward. In addition, this year’s event will explore current and future legislation and its impact on imaging. The use of imaging in clinical trials will also be examined and discussed together with the use of novel biomarkers. To view the full

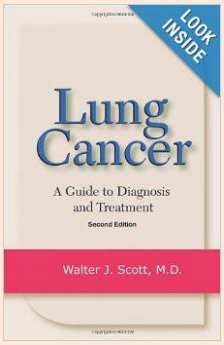
speaker line-up and conference programme visit the website.

http://www.cancer-imaging.com

**2014 Toxicology and Risk Assessment Conference (TRAC),** 4/7/2014 to 4/10/2014, USA,

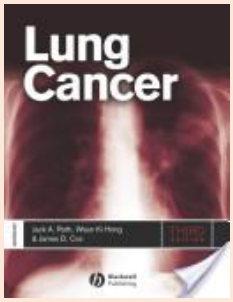
The 2014 Toxicology and Risk Assessment Conference (TRAC) will be a four-day meeting focusing on topics in risk assessment principles and practice. The conference provides attendees with an overview of current research, methodologic, and practice issues that are the focus of risk assessment efforts in various Federal agencies, academic institutions, industry, and other organizations. The theme of TRAC 2014 is "Toxicology and Risk Assessment Guidance: From Principles to Practice in the Age of Omics, Osomes, and New Opportunities." This theme will examine the 'omics fields, such as toxicogenomics, proteomics, and metabolomics, components of the concept of the exposome, and other emerging technologies, for current state-of-the-science opportunities for setting guidance and exposure limits.
http://www.cdc.gov/niosh/conferences/TRAC/

## BOOK STOP



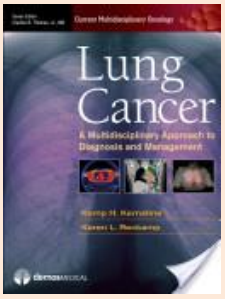

**Lung Cancer: A Guide to Diagnosis and Treatment, Editors Walter J. Scott , Addicus Books; Second Edition, Second edition, ISBN-10: 1886039097, ISBN-13: 978-1886039094, 2012**

What is my prognosis? What are my treatment options? Which therapies would be the most effective for my stage of lung cancer? These and other frequently asked questions are addressed in this crucial reference designed to help patients educate themselves and obtain the best possible treatments. The completely revised second edition has been updated to include a discussion of the movement towards customized chemotherapy; treatment options for early-stage lung cancer including minimally invasive surgery; and the most promising treatments, among them multimodality therapy—a combination of surgery, chemotherapy, and radiation. Dr. Scott also surveys tests for early detection of lung cancer, talks about the importance of cancer staging, examines alternative treatments, and offers advice on coping with emotions such as "smoker's guilt."




**Lung Cancer, Editors: Jack A. Roth, James D. Cox, Waun Ki Hong, John Wiley & Sons, 2011, ISBN 1444358669, 9781444358667, 2011.**

Lung cancer is a major cause of cancer-related deaths in men and women. However, since the first edition of *Lung Cancer* was published 14 years ago, rapid progress in the biology, prevention, diagnosis, and treatment of the disease has been made.




**Lung Cancer: A Multidisciplinary Approach to Diagnosis and Management, Volume 2 of Current Multidisciplinary Oncology, Springer Demos Medic Series. Editors: Kemp H. Kernstine, Karen L. Reckamp, Demos Medical Publishing, ISBN: 1936287064, 9781936287062, 2010**

Over the course of the last decade, the treatment of lung cancer has evolved quite rapidly. New scientific and clinical advances have modified the standard of care and led to improved patient outcomes. At the same time, the treatment of lung cancer has become increasingly complex, requiring the comprehensive review and assessment of multiple issues, genetics, radiology, surgery, reconstruction, chemotherapy, and more. As a result the harmony and open communication between these specialties facilitated by a multidisciplinary team approach are crucial in providing the best care to patients and ensuring successful treatment. *Lung Cancer: A Multidisciplinary Approach to Diagnosis and Management*, written by a multidisciplinary team of authors representing a range of disciplines, is a valuable resource for physicians, fellows, nurses, physician assistants, physical therapists, and all health care providers involved in the treatment of lung cancer. *Lung Cancer: A Multidisciplinary Approach to Diagnosis and Management* summarizes the state-of-the-art issues related to the treatment of lung cancer and describes an approach for optimal multidisciplinary care for individuals who have been diagnosed with lung cancer or who are at higher risk to develop lung cancer.

**About the Series:**
*The Current Multidisciplinary Oncology* series edited by Charles R. Thomas consolidates and integrates the varied aspects of multidisciplinary care for major topics in oncology, including breast, lung, prostrate, head and neck and more. The volumes in the *Current Multidisciplinary Oncology* series will represent all related topic areas, including oncology, radiation oncology, pain, pathology, imaging, psychological support and the

primary disease. In addition, each volume includes a chapter focusing on special populations and the disease's impact / difference on them, and discussion of future directions and quality of life issues. In addition each volume has a chapter written by a private practice oncologist.

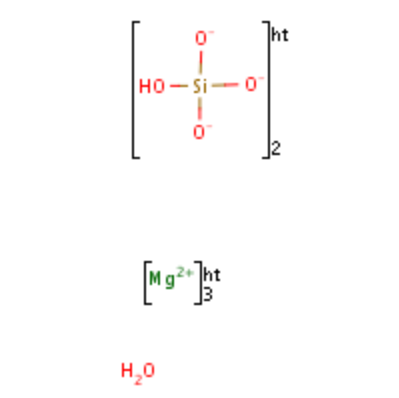
## MINI PROFILE OF CHRYSOTILE

**SYNONYMS**: Chrysotile sbestos-; avibest-c-; calidria-rg-100-; calidria-rg-144-; calidria-rg-600-; cassiar-ak-; chrysotile-; chrysotile- (mg3(oh)4(sio5)); hooker-no-1-chrysotile-asbestos-; plastibest-20-; rg-600-; serpentine-; serpentine-chrysotile-; sylode

**CAS RN:** 12001-29-5

**MOLECULAR FORMULA:** $Mg_3$-$Si_2$-$O_5$-$(OH)_4$

**MOLECULAR STRUCTURE:**




**MOLECULAR WEIGHT:** 277

**PROPERTIES:** Appearance: white or gray fibrous solid; Color: white or gray; pH: Not applicable; Odor: None; Vapor Pressure: Not applicable; Vapor Density (Air=1): Not available; Boiling Point/Range: Not available; Melting Point/Range: 800-850 °C; Specific Gravity: 2.2 to 2.6 g/cc; Solubility in water: None

**USES:** In cement products, floor tile, paper products, paint and caulking, textiles, plastics.
In different types of packings, woven brake linings to clutch facings, and electric insulation.

**TOXICITY DATA:**
NIOSH considers asbestos to be a potential occupational carcinogen.
OSHA: The employer shall ensure that no employee is exposed to an airborne concentration of asbestos in excess of 0.1 fiber/cu cm of air as an 8-hr TWA
TLV: +8 hr Time Weighted Avg (TWA): 0.1 fibers/cc. Respirable fibers: length greater than 5 m; aspect ratio greater than or equal to 3:1, as determined by the membrane filter method at 400-450X magnification (4-mm objective), using phase-contrast illumination. /Asbestos, all forms/
ipl-rat TDLo: 150 mg/kg
ipr-rat LDLo: 300 mg/kg
ihl-hmn TCLo: 2.8 fb/cc/5Y
ipr-mus TDLo: 100 mg/kg
itr-rat TDLo: 10 mg/kg

**PERSONAL PROTECTION**:.
Eye/Face Protection: Approved chemical safety glasses with side shield; Protective gloves: Rubber gloves; Protective clothing: Wear protective clothing to prevent skin contact. Do NOT take working clothes home; Respiratory Protection: Wear NIOSH approved respirator; Other: Wash prior to eating, drinking, or smoking. Avoid ingestion or breathing of dust.

**STORAGE:** Store in well-sealed container in cool, dry area.

| Route | Symptoms | First Aid | Target Organ |
|---|---|---|---|
| Inhalation/ Ingestion | There are not acute signs or symptoms associated with asbestos. Diseases associated with over exposure are chronic, generally taking from 10 to 40 years to become apparent | Ingestion: Get medical aid immediately Inhalation: Move the exposed person to fresh air at once. Support breathing. If symptoms persist contact physician. | Gastro Intestinal Respiratory Tract |
| Contact Eye/Skin | There are not acute signs or symptoms associated with asbestos. Diseases associated with over exposure are chronic, generally taking from 10 to 40 years to become apparent | Eyes: In case of contact, immediately flush eyes with copious amounts of flowing water for at least 15 minutes, retracting eye lids often. Get medical attention immediately. Contact lenses should not be worn when working with this product.<br><br>Skin: Wash skin thoroughly with mild soap and water. Flush with copious amounts of water for 15 minutes. | Eyes, Skin, |