Chrysotile Asbestos

World Health Organization

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http://www.who.int/ipcs/assessment/public_health/chemicals_phc

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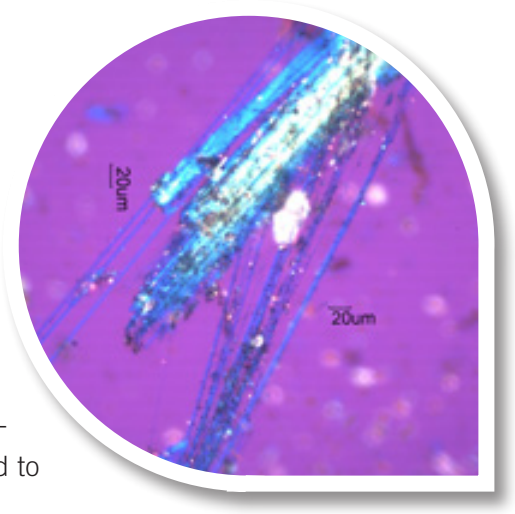
Foreword



Many countries have already taken action at a national level to prohibit the use of all forms of asbestos to limit exposure and so control, prevent and ultimately eliminate asbestos-related diseases, from which at least 107 000 people die each year globally. However, there are other countries that, for a range of reasons, have yet to act in the same manner. With that in mind, the prime intent of this publication is to assist Member States of the World Health Organization (WHO) in making informed decisions about management of the health risks attached to exposure to chrysotile asbestos.

The document is divided into three parts. The first part reproduces a WHO short information document for decision-makers on the elimination of asbestos-related diseases, updated in March 2014. The second part addresses questions commonly raised in policy discussions, specifically to assist decision-makers in coming to a view. The third part is a technical summary of the health effects of chrysotile, which brings together and summarizes for the first time the most recent authoritative WHO evaluations performed by its International Agency for Research on Cancer and its International Programme on Chemical Safety. The technical summary also reviews results from key studies published after those evaluations and then, briefly, the conclusions drawn from WHO assessments of alternatives.

I commend this publication to ministers, government officials and others who may wish or need to take decisions on, or provide advice related to, asbestos and in particular chrysotile asbestos and the health consequences of exposure.

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World Health Organization

Elimination of asbestos-related diseases

Updated March 2014

Asbestos is one of the most important occupational carcinogens, causing about half of the deaths from occupational cancer *(1, 2)*. In 2003, the Thirteenth Session of the Joint International Labour Organization (ILO)/World Health Organization (WHO) Committee on Occupational Health recommended that special attention should be paid to the elimination of asbestos-related diseases *(3)*. World Health Assembly (WHA) Resolution 58.22 from 2005 on cancer prevention and control urged Member States to pay special attention to cancers for which avoidable exposure is a factor, particularly exposure to chemicals at the workplace and in the environment. In 2007, WHA Resolution 60.26 called for global campaigns to eliminate asbestos-related diseases, and in 2013, WHA Resolution 66.10 addressed prevention and control of noncommunicable diseases, including cancer.

Asbestos is one of the most important occupational carcinogens

The term "asbestos" designates a group of naturally occurring fibrous serpentine or amphibole minerals with current or historical commercial usefulness due to their extraordinary tensile strength, poor heat conduction and relative resistance to chemical attack. The principal varieties of asbestos are chrysotile, a serpentine material, and crocidolite, amosite, anthophyllite, tremolite and actinolite, which are amphiboles *(4)*.

Exposure to asbestos, including chrysotile, causes cancer of the lung, larynx and ovary, mesothelioma (a cancer of the pleural and peritoneal linings) and asbestosis (fibrosis of the lungs) *(5–7)*.

Exposure to asbestos and its impact on public health are substantial

Exposure to asbestos occurs through inhalation of fibres primarily from contaminated air in the working environment, as well as from ambient air in the vicinity of point sources or indoor air in housing and buildings containing friable asbestos materials. The highest levels of exposure occur during repackaging of asbestos containers, mixing with other raw materials and dry cutting of asbestos-containing products with abrasive tools. Exposure can also occur during installation and use of asbestos-containing products and maintenance of vehicles. Friable chrysotile- and/or amphibole-containing materials are still in place in many buildings and continue to give rise to exposure to both chrysotile and the amphiboles during maintenance, alteration, removal and demolition *(5)*. Exposure can also occur as a consequence of natural disasters causing damage to buildings.

Currently, about 125 million people in the world are exposed to asbestos at the workplace *(1)*. According to global estimates, at least 107 000 people die each year from

asbestos-related lung cancer, mesothelioma and asbestosis resulting from occupational exposures *(1, 2, 8)*. In addition, nearly 400 deaths have been attributed to non-occupational exposure to asbestos. The burden of asbestos-related diseases is still rising, even in countries that banned the use of asbestos in the early 1990s. Because of the long latency periods attached to the diseases in question, stopping the use of asbestos now will result in a decrease in the number of asbestos-related deaths only after a number of decades.

All types of asbestos cause cancer in humans

Asbestos (actinolite, amosite, anthophyllite, chrysotile, crocidolite and tremolite) has been classified by the International Agency for Research on Cancer as being carcinogenic to humans *(7)*. Exposure to chrysotile, amosite and anthophyllite and to mixtures containing crocidolite results in an increased risk of lung cancer *(7)*. Mesotheliomas have been observed after occupational exposure to crocidolite, amosite, tremolite and chrysotile, as well as among the general population living in the neighbourhood of asbestos factories and mines and in people living with asbestos workers *(7)*.

The incidence of asbestos-related diseases is related to fibre type, size and dose and to industrial processing of the asbestos *(6)*. No threshold has been identified for the carcinogenic risk of asbestos, including chrysotile *(5, 7)*. Cigarette smoking increases the risk of lung cancer from asbestos exposure *(5, 9)*.

At least 107 000 people die each year from asbestos-related lung cancer, mesothelioma and asbestosis resulting from occupational exposures

Chrysotile is still widely used

Asbestos has been used in thousands of products for a vast number of applications, such as roofing shingles, water supply lines, fire blankets and insulation materials, as well as clutches and brake linings, gaskets and pads for automobiles. As a result of increasing health concerns, the use of asbestos has declined in many countries. The use of crocidolite and products containing this fibre and spraying of all forms of asbestos are prohibited under the ILO Convention concerning Safety in the Use of Asbestos (No. 162) from 1986. However, chrysotile is still widely used, with approximately 90% being employed in asbestos cement building materials, the largest users of which are developing countries. Other remaining uses of chrysotile are in friction materials (7%), textiles and other applications *(10)*.

To date (end of 2013), more than 50 countries, including all member states of the European Union, have banned the use of all forms of asbestos, including chrysotile. Other countries have introduced less stringent restrictions. However, some countries have maintained or even increased their production or use of chrysotile in recent years *(11)*. Increased usage has been most prominent in the Asia-Pacific region. World production of asbestos in the period 2000–2012 was relatively stable, at approximately 2 million tonnes per annum *(12, 13)*.

WHO recommendations on prevention of asbestos-related diseases

Bearing in mind that there is no evidence for a threshold for the carcinogenic effect of asbestos, including chrysotile, and that increased cancer risks have been observed in populations exposed to very low levels *(5, 7)*, the most efficient way to eliminate asbestos-related diseases is to stop using all types of asbestos. Continued use of asbestos cement in the construction industry is a particular concern, because the workforce is large, it is difficult to control exposure, and in-place materials have the potential to deteriorate and pose a risk to those carrying out alterations, maintenance and demolition *(5)*. In its various applications, asbestos can be replaced by some fibre materials *(14)* and by other products that pose less or no risk to health.

Materials containing asbestos should be encapsulated, and, in general, it is not recommended to carry out work that is likely to disturb asbestos fibres. If necessary, such work should be carried out only under strict control measures to avoid exposure to asbestos, such as encapsulation, wet processes, local exhaust ventilation with filtration, and regular cleaning. It also requires the use of personal protective equipment – special respirators, safety goggles, protective gloves and clothing – and the provision of special facilities for their decontamination *(15)*.

WHO is committed to working with countries towards the elimination of asbestos-related diseases in the following strategic directions:

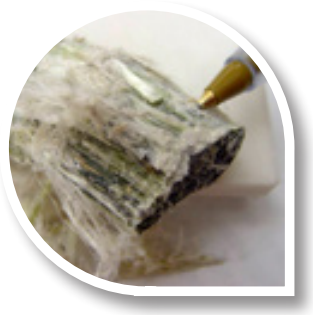
- by recognizing that the most efficient way to eliminate asbestos-related diseases is to stop the use of all types of asbestos;
- by providing information about solutions for replacing asbestos with safer substitutes and developing economic and technological mechanisms to stimulate its replacement;
- by taking measures to prevent exposure to asbestos in place and during asbestos removal (abatement);
- by improving early diagnosis, treatment and rehabilitation services for asbestos-related diseases and establishing registries of people with past and/or current exposure to asbestos.

WHO strongly recommends planning for and implementing these measures as part of a comprehensive national approach for the elimination of asbestos-related diseases. Such an approach should also include developing national profiles, awareness raising, capacity building, an institutional framework and a national plan of action for the elimination of asbestos-related diseases.

WHO will collaborate with ILO on implementation of the Resolution concerning asbestos, adopted by the Ninety-fifth Session of the International Labour Conference *(16)*, and will work with other intergovernmental organizations and civil society towards the elimination of asbestos-related diseases worldwide.

References

1. Concha-Barrientos M, Nelson D, Driscoll T, Steenland N, Punnett L, Fingerhut M et al. Chapter 21. Selected occupational risk factors. In: Ezzati M, Lopez A, Rodgers A, Murray C, editors. Comparative quantification of health risks: global and regional burden of disease attributable to selected major risk factors. Geneva: World Health Organization; 2004:1651–801 (http://www.who.int/healthinfo/global_burden_disease/cra/en/, accessed 11 March 2014).
2. Driscoll T, Nelson DI, Steenland K, Leigh J, Concha-Barrientos M, Fingerhut M et al. The global burden of disease due to occupational carcinogens. Am J Ind Med. 2005;48(6):419–31.
3. ILO, WHO. Summary report of the Thirteenth Session of the Joint ILO/WHO Committee on Occupational Health, 9–12 December 2003, Geneva. JCOH/2003/D.4. Geneva: International Labour Organization; 2003 (http://www.ilo.org/wcmsp5/groups/public/---ed_protect/---protrav/---safework/documents/publication/wcms_110478.pdf, accessed 13 March 2014).
4. 6.2 Asbestos. In: Air quality guidelines for Europe, second edition. WHO Regional Publications, European Series, No. 91. Copenhagen: World Health Organization Regional Office for Europe; 2000 (http://www.euro.who.int/__data/assets/pdf_file/0005/74732/E71922.pdf, accessed 11 March 2014).
5. Environmental Health Criteria 203: Chrysotile asbestos. Geneva: World Health Organization, International Programme on Chemical Safety; 1998 (http://www.inchem.org/documents/ehc/ehc/ehc203.htm, accessed 11 March 2014).
6. Environmental Health Criteria 53: Asbestos and other natural mineral fibres. Geneva: World Health Organization, International Programme on Chemical Safety; 1986 (http://www.inchem.org/documents/ehc/ehc/ehc53.htm, accessed 13 March 2014).
7. International Agency for Research on Cancer. Asbestos (chrysotile, amosite, crocidolite, tremolite, actinolite, and anthophyllite). IARC Monogr Eval Carcinog Risks Hum. 2012;100C:219–309 (http://monographs.iarc.fr/ENG/Monographs/vol100C/index.php, accessed 11 March 2014).
8. Driscoll T, Nelson DI, Steenland K, Leigh J, Concha-Barrientos M, Fingerhut M et al. The global burden of non-malignant respiratory disease due to occupational airborne exposures. Am J Ind Med. 2005;48(6):432–45.
9. International Agency for Research on Cancer. Tobacco smoke and involuntary smoking. IARC Monogr Eval Carcinog Risks Hum. 2006;83.
10. Perron L. Chrysotile. In: Canadian minerals yearbook, 2003. Ottawa: Natural Resources Canada; 2003:18.1–18.11.
11. Virta RL. Worldwide asbestos supply and consumption trends from 1900 through 2003. Circular 1298. Reston (VA): United States Department of the Interior, United States Geological Survey; 2006 (http://pubs.usgs.gov/circ/2006/1298/c1298.pdf, accessed 11 March 2014).
12. Virta RL. Asbestos [Advance release]. In: 2012 minerals yearbook. Reston (VA): United States Department of the Interior, United States Geological Survey; 2013:8.1–8.7 (http://minerals.usgs.gov/minerals/pubs/commodity/asbestos/myb1-2012-asbes.pdf, accessed 11 March 2014).
13. Virta RL. Asbestos statistics and information. In: Mineral commodity summaries 2013. Reston (VA): United States Department of the Interior, United States Geological Survey; 2013 (http://minerals.usgs.gov/minerals/pubs/commodity/asbestos/mcs-2013-asbes.pdf, accessed 11 March 2014).
14. Summary consensus report of WHO Workshop on Mechanisms of Fibre Carcinogenesis and Assessment of Chrysotile Asbestos Substitutes, 8–12 November 2005, Lyon. Geneva: World Health Organization; 2005 (http://www.who.int/ipcs/publications/new_issues/summary_report.pdf, accessed 11 March 2014).
15. International Chemical Safety Card 0014: Chrysotile. Geneva: World Health Organization, International Programme on Chemical Safety; 2010 (http://www.inchem.org/documents/icsc/icsc/eics0014.htm, accessed 13 March 2014).
16. Annex: Resolution concerning asbestos. In: Provisional Record 20 of the Ninety-fifth Session of the International Labour Conference, 31 May – 16 June 2006, Geneva: Report of the Committee on Safety and Health. Geneva: International Labour Organization; 2006:20/69 (http://www.ilo.org/public/english/standards/relm/ilc/ilc95/pdf/pr-20.pdf, accessed 13 March 2014).



Chrysotile in raw state

Commonly raised questions and answers

This section addresses questions commonly raised by policy-makers on the use of chrysotile.

Is it true that chrysotile is not really a form of asbestos?

No. Chrysotile is one of six forms of asbestos, the others being crocidolite, amosite, tremolite, actinolite and anthophyllite.

What is WHO's policy on asbestos?

WHO's policy on asbestos is unequivocal. Asbestos causes cancer of the lung, larynx and ovary, mesothelioma (a cancer of the pleural and peritoneal linings) and asbestosis (fibrosis of the lungs). Asbestos-related diseases can and should be prevented, and the most efficient way to prevent them is to stop the use of all forms of asbestos to prevent exposure. WHO's global campaigns to eliminate asbestos-related diseases aim to support countries in achieving that objective.

Why is WHO so concerned about asbestos?

There is clear scientific evidence that asbestos causes cancer and chronic respiratory diseases in humans. WHO is working to reduce the global burden of noncommunicable diseases, including cancer and chronic respiratory diseases, recognizing that primary prevention reduces health-care service costs and helps to ensure the sustainability of health expenditures. Worldwide, cancer is the second leading cause of death. In 2008, there were 7.6 million deaths from cancer, alongside 12.7 million new cases. Roughly 19% of all cancers are estimated to be attributable to the environment, including work settings.

Currently, about 125 million people in the world are exposed to asbestos at the workplace. According to WHO estimates, at least 107 000 people die each year from asbestos-related lung cancer, mesothelioma and asbestosis resulting from occupational exposures. Approximately half of all deaths from occupational cancer are estimated to be caused by asbestos.

With what authority does WHO speak on chrysotile and other forms of asbestos and their management?

WHO is the directing and coordinating authority for health within the United Nations system. It is responsible for providing leadership on global health matters, shaping the health research agenda, setting norms and standards, articulating evidence-based policy options, providing technical support to countries and monitoring and assessing health trends.

The World Health Assembly (WHA) is the supreme decision-making body for WHO; it meets annually and is composed of delegations from 194 Member States. The main function of WHA is to determine WHO policy.

WHO's policy on asbestos derives from three WHA resolutions: WHA 58.22 in 2005, WHA 60.26 in 2007 and WHA 66.10 in 2013. WHA 58.22 addresses cancers for which avoidable exposure to carcinogens is a factor in their causation, WHA 60.26 calls for global campaigns to eliminate asbestos-related diseases and WHA 66.10 deals with the prevention and control of noncommunicable diseases, including cancer.

How are people exposed to asbestos?

Exposure to asbestos occurs by inhalation and, to a lesser extent, ingestion during the mining and milling of asbestos and in the production and use of asbestos-containing products. This includes exposure from trimming and fitting of asbestos materials during building construction, maintenance and demolition. Asbestos is generally used or has been used as a fibrous mixture, bonded with other materials (e.g. cement, plastics and resins) or woven as a textile. The range of applications in which asbestos has been used is large and includes roofing, cement sheets for floors and walls, cement pipes (e.g. for supplying water), thermal and electrical insulation, including fire blankets and industrial fire curtains, gaskets and friction materials (e.g. vehicle brake shoes and brake pads and clutches). Today, exposure to asbestos fibres occurs particularly in circumstances where asbestos products have become degraded, such as during the course of building maintenance and demolition and the disposal of building waste, and also in the context of natural disasters.

There is clear scientific evidence that asbestos causes cancer and chronic respiratory diseases in humans

Why is it so important to tackle asbestos as a carcinogen when there are so many other carcinogens to be found in the environment?

Some cancers attributable to environmental factors are believed to have multiple carcinogenic determinants. Others, though, have as their causes single identifiable carcinogens, such as tobacco and asbestos, to which exposure is preventable. (Note: This is not the case for many of the other agents classified by the International Agency for Research on Cancer [IARC] as being in Group 1, carcinogenic to humans, and neither do many of them carry the same burden of disease.[1])

One of the reasons it is important that countries take action on asbestos as soon as possible is because of an unusually long latent period between exposure and the development of mesothelioma, often as long as 40 years. For this reason, the burden of asbestos-related diseases will continue to rise, for the moment, even in those countries that banned the use of asbestos many years ago.

All forms of asbestos cause cancer in humans (this includes chrysotile, the principal form of asbestos still in production and use), and no threshold has been identified for the carcinogenic risks. This is the conclusion of WHO and IARC in a series of authoritative international assessments conducted over a period of more than 15 years, the

[1] For details of IARC Group 1 carcinogens, see http://monographs.iarc.fr/ENG/Classification/ClassificationsGroupOrder.pdf.

most recent having been published by IARC in 2012. These conclusions reflect an international consensus of scientific experts convened by WHO to evaluate the health effects of asbestos.

In addition, it has been shown that co-exposure to tobacco smoke and asbestos fibres substantially increases the risk for lung cancer, and the effect is at least additive – that is, the heavier the smoking, the greater the risk.

Can we be certain that the scientific evaluations of asbestos by WHO and IARC are wholly independent of outside influence?

Yes. In every case, measures were taken to ensure that potential conflicts of interest were identified and addressed, that the assessments were extremely rigorous and independent of the views of governments, national institutions and special interest groups, and that they took account of opinions from all regions of the world and were subject to extensive international peer review.

What actions have been taken by countries at a national level?

Many countries have already legislated to prohibit the use of asbestos, with more than 50 WHO Member States now (end of 2013) having done so in order to protect and promote public health.[2] Typically, the decision was undertaken after cross-government consultation, to take account of sectoral interests but to avoid their over-predominance in the final decision. When considering taking legislative action against the use of asbestos, it has been necessary to take into account a range of costs and benefits, including the costs of providing health-care services and those associated with the loss of workforce productivity due to chronic ill-health, in addition to conventional economic and trade considerations.

What actions have been taken or are being proposed by countries at an international level?

The Basel Convention on the Control of Transboundary Movements of Hazardous Wastes and their Disposal, which entered into force in 1992 and to which 181 countries are Parties, aims to protect human health and the environment against the adverse effects of hazardous wastes. Asbestos (dust and fibres) is listed as a category of controlled waste under the Convention. Parties to the Convention are required to prohibit or not permit the export of such waste to Parties that have prohibited its importation under the Convention.

[2] These include Algeria, Argentina, Australia, Bahrain, Brunei Darussalam, Chile, Egypt, the 28 member states of the European Union, Gabon, Honduras, Iceland, Israel, Japan, Jordan, Kuwait, Mozambique, Norway, Oman, Qatar, Republic of Korea, Saudi Arabia, Serbia, Seychelles, South Africa, Switzerland, Turkey and Uruguay. Asbestos is also banned in two states of Brazil, Rio de Janeiro and Rio Grande do Sul.

More recently, a majority of the 154 countries that are Parties to the Rotterdam Convention on the Prior Informed Consent Procedure for Certain Hazardous Chemicals and Pesticides in International Trade (which entered into force in 2004) have indicated a wish to see chrysotile listed under Annex 3 of the Convention. This would mean that chrysotile would become subject to a procedure whereby an informed decision of a country would be needed before consenting or not to future importation of the substance. However, to date, listing of chrysotile has been blocked by a small number of countries, predominantly but not exclusively by those with a continued interest in the trade in, and use of, chrysotile and chrysotile-containing products.

Is it true that chrysotile is less harmful than other types of asbestos and should not, therefore, be subject to the same control measures?

The scientific evidence is clear. The firm conclusion of the WHO and IARC assessments is that chrysotile causes cancer of the lung, larynx and ovary, mesothelioma and asbestosis, whether or not it is less potent than amphibole types of asbestos in doing so. Assertions about differing physicochemical properties, the question of whether or not historical epidemiological studies may have been dealing with chrysotile contaminated with amphibole types of asbestos, and the physical containment of chrysotile in modern high-density cement (at the time of manufacture) do not alter this finding.

The firm conclusion of the WHO and IARC assessments is that chrysotile causes cancer of the lung, larynx and ovary, mesothelioma and asbestosis

A major concern is that even where use is appropriately regulated, chrysotile-containing building products (e.g. roof tiling, water pipes) become damaged and release asbestos fibres into the environment during the course of building maintenance, demolition and disposal of building waste, and as a consequence of natural disasters. Such exposure may occur some time later than the original (controlled) installation. This risk can be wholly averted by ceasing to use such products. Information on substitute materials and products that can be used safely is available from national, regional and international organizations.

Could ongoing or future research into the toxicity of chrysotile change the current view of WHO and IARC regarding the occurrence of cancer?

Absolutely not. The firm view of WHO and IARC, based on repeated assessments of the scientific evidence, is that chrysotile causes cancer of the lung, larynx and ovary, mesothelioma and asbestosis, and that stopping the use of all forms of asbestos, including chrysotile, to prevent exposure should be recognized as the most effective way to eliminate asbestos-related diseases. Although the carcinogenic potential of chrysotile has been clearly identified, few studies have included women. There are also additional cancers suspected to be related to chrysotile, but for which existing studies are inadequate. There is therefore an ongoing need for further research to investigate the risks of chrysotile exposure for additional types of cancer, in particular for female-specific cancers.

What information is available on alternative products, especially as building materials, given assertions that modern fibre substitutes for chrysotile are either themselves toxic or of undetermined toxicity?

Many national governments, regional bodies and international organizations have identified alternatives and substitutes for the uses of asbestos, and human health evaluations of substitute materials have also been published. For example, a WHO/IARC workshop was convened in 2005, and there have been publications from the government of the United Kingdom, the European Commission and the WHO Regional Office for Europe. Evaluations of the human health hazard of chrysotile substitute materials have concentrated on alternative types of fibrous materials due to the potential risks associated with inhalation of fibres. However, it should also be noted that for some of its uses, chrysotile may be replaced by non-fibrous material – for example, unplasticized polyvinyl chloride (uPVC) and sheet metal.

Does an absence of reported cases of mesothelioma in a country indicate that there is no significant burden of disease resulting from asbestos and therefore no reason for action, given that mesothelioma is such a specific marker of asbestos exposure?

No. Detection of cases of mesothelioma and accurate measurement of their number require systematic surveillance systems at the national level, and these are frequently absent. It should also be borne in mind that the latent period between exposure to asbestos and the development of mesothelioma can be as long as 40 years or more, and such systems therefore need to be of long standing.

Asbestos is more likely to cause cancer of the lung than mesothelioma (estimated risk ratio 6:1), and the likelihood is greater in individuals who smoke tobacco. Cancer of the lung is much more common than mesothelioma and is multifactorial in origin. A history of prior exposure to asbestos (and this can include non-working environments, see below) many years previously may easily be overlooked. Current absence of evidence at a national level is not evidence of absence, and lessons learnt by other countries where large epidemics of mesothelioma are still occurring, even many years after widespread exposures have stopped, should be taken into account.

Is asbestos exposure only an occupational issue, with no or little risk to the population at large?

No. Many cases of mesothelioma have been described in wives and children of asbestos workers, as a result of domestic exposure (at least 376

cases), in white collar workers within the asbestos industry, and in individuals living in the vicinity of asbestos mines, as a result of air pollution; asbestosis has also been reported in the wives and children of asbestos workers. Cases of mesothelioma have been described in individuals exposed to naturally occurring asbestos or asbestos-like minerals in soil in regions in Turkey, Greece, Cyprus, Corsica, Sicily, New Caledonia, Yunnan province in China and California. Although the final group would not be protected by control measures on the production and use of asbestos, the other groups would be protected.

Other types of environmental exposure also occur. Reports from Australia and the United Kingdom have identified elevated concentrations of asbestos fibres in ambient air at busy traffic intersections from friction products in vehicles. Non-occupational exposures arise from home renovation and car maintenance activities. In addition to the occupational exposures of construction workers (because measures to control asbestos exposure are difficult to put in place for a large, fragmented workforce that may include many informal workers), there is also potential for non-occupational exposure to asbestos-containing building waste if the waste is not stored and disposed of correctly. This includes the potential for asbestos-containing building waste to be scavenged and reused in informal settlements.

The concern for policy-makers today is less in relation to occupational exposure within the mining and manufacture of asbestos products sectors and more in relation to the use of asbestos-containing materials within the construction industry. Concerns extend to occupational exposure during construction activities and inadvertent exposure of the wider population from degradation of building materials (e.g. broken corrugated asbestos roof tiles) and inappropriate disposal of building waste. The use of asbestos-containing building materials in the poorest communities, bringing families into close proximity to sources of exposure to chrysotile fibres, is of particular concern.

There is potential for non-occupational exposure to asbestos-containing building waste

Additional Information

Other WHO publications on asbestos

Title	Description	Website
Outline for the development of national programmes for elimination of asbestos-related diseases. International Labour Organization and World Health Organization; 2007	This document is intended to facilitate countries in establishing their national programmes for elimination of asbestos-related diseases. It also addresses countries' efforts to prevent asbestos-related diseases arising from exposure to the various forms of asbestos already in place and as a result of their use in the past. Available in English, French, Russian, Spanish, Arabic and Chinese.	http://www.who.int/occupational_health/publications/asbestosdoc/en/, accessed 11 March 2014
Asbestos – hazards and safe practices for clean up after earthquake. World Health Organization; 2008	This technical information note provides guidance on how to control the risks associated with asbestos during the clean-up and disposal of asbestos-containing waste from damaged and destroyed buildings following an earthquake or other natural disaster.	http://www.who.int/hac/crises/chn/asbestos/en/, accessed 11 March 2014

Published evaluations of substitute materials

Title	Description	Website
Review of substitutes for asbestos construction products by a WHO temporary advisor. In: National programmes for elimination of asbestos-related diseases: review and assessment. WHO Regional Office for Europe; 2012: Annex 4	A review of the availability and safety of asbestos substitute materials, prepared as a background document for a meeting on asbestos control in the WHO European Region by a WHO temporary advisor. Available in English and Russian.	http://www.euro.who.int/en/health-topics/environment-and-health/occupational-health/publications/2012/national-programmes-for-elimination-of-asbestos-related-diseases-review-and-assessment, accessed 11 March 2014
Opinion on chrysotile asbestos and candidate substitutes. Scientific Committee on Toxicity, Ecotoxicity and the Environment (CSTEE), European Commission; 1998	Evaluation of the risks to human health posed by three substitute fibres – cellulose fibres, polyvinyl alcohol fibres and *p*-aramid fibres – by an expert committee of the European Commission.	http://ec.europa.eu/health/scientific_committees/environmental_risks/opinions/sctee/sct_out17_en.htm, accessed 11 March 2014
Harrison et al. Comparative hazards of chrysotile asbestos and its substitutes: a European perspective. Environ Health Perspect. 1999;107:607–11	An evaluation of asbestos substitute materials prepared for the United Kingdom Health and Safety Commission (London, United Kingdom) and subsequently published in the scientific literature.	http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1566482/, accessed 11 March 2014

Technical summary of WHO evaluations of chrysotile

Introduction

This technical summary on the health effects of chrysotile summarizes the most recent authoritative World Health Organization (WHO) evaluations performed by its International Agency for Research on Cancer (IARC) and its International Programme on Chemical Safety (IPCS). Key studies published after these evaluations are also briefly reviewed. The purpose of this technical summary is to assist policy-makers in assessing the importance of undertakings to prevent the adverse health effects – cancer and lung fibrosis – associated with exposure to chrysotile.

WHO has conducted a number of evaluations of the health effects associated with exposure to chrysotile over the past 20 years *(1, 2)*. These evaluations have concluded that all forms of asbestos, including chrysotile, are carcinogenic to humans, causing mesothelioma and cancer of the lung, larynx and ovary. Chrysotile also causes non-malignant lung diseases, which result in deterioration of lung function (asbestosis). Many scientific studies linking domestic and environmental exposure to asbestos with adverse health effects have also been identified, alongside the large number of studies in occupational settings.

Most informative in the evaluation of the effects of chrysotile exposure in humans *(1)* have been the studies performed in chrysotile mines in Quebec, Canada (most recent cohort update) *(3)*, a chrysotile mine in Balangero, Italy *(4, 5)*, cohorts of textile workers in South Carolina *(6)* and North Carolina, United States of America (USA) *(7)*, and two cohorts of asbestos factory workers in China *(8, 9)*. More recently, studies on chrysotile miners *(10–12)* and chrysotile textile workers in China *(13–17)* and two meta-analyses *(18, 19)* have further consolidated the database. All types of asbestos cause asbestosis, mesothelioma and cancer of the lung, larynx and ovary *(1, 2)*. This text concentrates on cancer of the lung, mesothelioma and asbestosis, as these have been the principal areas of research until relatively recently.

"There is sufficient evidence in humans for the carcinogenicity of all forms of asbestos (chrysotile, crocidolite, amosite, tremolite, actinolite and anthophyllite). Asbestos causes mesothelioma and cancer of the lung, larynx and ovary." *(1)*

Chrysotile production, use and exposure

Production

Chrysotile has always been the main asbestos species mined; in the peak year of production (1979), chrysotile comprised more than 90% of all asbestos mined *(20)*. With the exception of small amounts (approximately 0.2 Mt annually, in 2007–2011) of amphibole asbestos mined in India, chrysotile is at present the only asbestos species mined. World production in 2012 was estimated to be 2 Mt, the main producers being the Russian Federation (1 Mt), China (0.44 Mt), Brazil (0.31 Mt) and Kazakhstan (0.24 Mt); production has stopped in Canada, which until 2011 was one of the main producers. Although world production has decreased considerably from its peak of 5.3 Mt in 1979, it has remained stable during the 2000s (2–2.2 Mt) *(21–23)*.

Use

Asbestos is used as a loose fibrous mixture, bonded with other materials (e.g. Portland cement, plastics and resins) or woven as a textile. The range of applications in which asbestos has been used includes roofing, thermal and electrical insulation, cement pipe and sheets, flooring, gaskets, friction materials (e.g. brake pads and shoes), coating and caulking compounds, plastics, textiles, paper, mastics, thread, fibre jointing and millboard *(1)*.

Organizations that track the usage of chrysotile globally report that all asbestos (including chrysotile) use had been prohibited in 32 countries by 2007, rising to approximately 50 countries by 2014 *(24)*. The form of prohibition in countries can vary (e.g. exemptions for limited, highly specialized engineering uses can be permitted),

which complicates the process of determining the status of a country at any given time. However, countries that have prohibited all widespread and large-scale uses of all types of asbestos (including chrysotile) include Algeria, Argentina, Australia, Bahrain, Brunei Darussalam, Chile, Egypt, the 28 member states of the European Union, Gabon, Honduras, Iceland, Israel, Japan, Jordan, Kuwait, Mozambique, Norway, Oman, Qatar, Republic of Korea, Saudi Arabia, Serbia, Seychelles, South Africa, Switzerland, Turkey and Uruguay. Asbestos is also banned in two states of Brazil, Rio de Janeiro and Rio Grande do Sul *(25)*.

Although asbestos has not been banned in the USA, consumption decreased from 668 000 t in 1970 to 359 000 t in 1980, 32 t in 1990, 1.1 t in 2000 and 1.0 t in 2010 *(22, 23)*. Consumption of asbestos (mainly chrysotile) was 143 000 t in the United Kingdom in 1976, decreasing to 10 000 t in 1995; as the use of asbestos is banned in the European Union, it is expected to be zero at present. France imported approximately 176 000 t of asbestos in 1976; imports stopped by 1996, when France banned asbestos use. In Germany, the use of asbestos amounted to approximately 175 000 t annually from 1965 to 1975 and came to an end in 1993. In Japan, asbestos consumption was approximately 320 000 t in 1988 and decreased steadily over the years to less than 5000 in 2005; asbestos use was banned in 2012 *(26)*. In Singapore, imports of raw asbestos (chrysotile only) decreased from 243 t in 1997 to 0 t in 2001 *(27)*. In the Philippines, the importation of raw asbestos was approximately 570 t in 1996 and 450 t in 2000 *(28)*. However, in some countries, such as Belarus, Bolivia (Plurinational State of), China, Ghana, India, Indonesia, Pakistan, Philippines, Sri Lanka and Viet Nam, the use of chrysotile increased between 2000 and 2010. In India, use increased from 145 000 t in 2000 to 462 000 t in 2010 *(21, 23)*; in Indonesia, the increase was from 45 045 t in 2001 to 121 548 t in 2011 *(29)*.

Non-occupational exposure

Non-occupational exposure, also loosely called environmental exposure, to asbestos may be due to domestic exposure (e.g. living in the same household with someone exposed to asbestos at work), air pollution from asbestos-related industries or the use of asbestos-containing friction materials, or naturally occurring asbestos minerals.

In studies of asbestos concentrations in outdoor air, chrysotile is the predominant fibre detected. Low levels of asbestos have been measured in outdoor air in rural locations (typical concentration, 10 fibres/m^3).[3] Typical concentrations are about 10-fold higher in urban locations and about 1000 times higher in close proximity to industrial sources of exposure. Elevated levels of chrysotile fibres have also been detected at busy traffic intersections, presumably from braking vehicles *(30)*. In indoor air (e.g. in homes, schools and other buildings), measured concentrations of asbestos are in the range of 30–6000 fibres/m^3 *(1)*.

[3] 1 fibre/m^3 = 1 × 10^{-6} fibres/mL; 1 fibre/mL = 1 × 10^6 fibres/m^3.

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Occupational exposure

Exposure by inhalation and, to a lesser extent, ingestion occurs in the mining and milling of asbestos (or other minerals contaminated with asbestos), the manufacturing or use of products containing asbestos, and the construction, automotive and asbestos abatement industries (including the transport and disposal of asbestos-containing wastes) *(1)*. In estimates published in 1998, when most European Union countries had already banned the use of all asbestos, it was estimated that the proportion of the European Union workforce still exposed to asbestos (mainly chrysotile) in different economic subsectors (as defined by the United Nations) *(31)* was as follows: agriculture, 1.2%; mining, 10.2%; manufacturing, 0.59%; electrical, 1.7%; construction, 5.2%; trade, 0.3%; transport, 0.7%; finance, 0.016%; and services, 0.28% *(32, 33)*.

In 2004, it was estimated that 125 million people were exposed to asbestos (as stated above, mainly to chrysotile) at work *(34)*.

The National Institute for Occupational Safety and Health (NIOSH) in the USA estimated in 2002 that 44 000 miners and other mine workers may have been exposed to asbestos during the mining of asbestos and some mineral commodities in which asbestos may have been a potential contaminant. In 2008, the Occupational Safety and Health Administration (OSHA) in the USA estimated that 1.3 million employees in construction and general industry face significant asbestos exposure on the job *(1)*. In Europe, based on occupational exposure to known and suspected carcinogens collected during 1990–1993, the CAREX (CARcinogen EXposure) database estimates that a total of 1.2 million workers were exposed to asbestos in 41 industries in the (then 15) member states of the European Union. Over 96% of these workers were

employed in the following 15 industries: "construction", "personal and household services", "other mining", "agriculture", "wholesale and retail trade and restaurants and hotels", "food manufacturing", "land transport", "manufacture of industrial chemicals", "fishing", "electricity, gas and steam", "water transport", "manufacture of other chemical products", "manufacture of transport equipment", "sanitary and similar services" and "manufacture of machinery, except electrical" *(1)*. According to an unpublished report, in China, 120 000 workers of 31 asbestos mines come in direct contact with asbestos, and 1.2 million workers are involved in the production of chrysotile asbestos products *(35)*. Another unpublished report indicated that in 31 asbestos factories in China with 120 000 workers, all these workers could have come in contact with asbestos either directly or indirectly *(35)*. In India, approximately 100 000 workers in both organized and unorganized sectors were estimated to be exposed to asbestos directly, and 30 million construction workers were estimated to be subjected to asbestos dust on a daily basis *(36)*. The number of exposed workers in Brazil was estimated to be 300 000 *(25)*.

In Germany, there was a steady decline in asbestos exposure between 1950 and 1990; the 90th percentile of the fibre count was between 0.5 and 1 fibre/mL in textile, paper/seals, cement, brake pad and drilling/sawing activities in 1990 *(37)*.

In 2004, it was estimated that 125 million people were exposed to asbestos at work

In France, median asbestos concentrations were highest in the building (0.85 fibre/mL in 1986–1996 and 0.063 fibre/mL in 1997–2004), chemical industry (0.34 and 0.1 fibre/mL, respectively) and services (0.07 and 0.1 fibre/mL, respectively) sectors *(38)*.

In 1999, the median asbestos (almost exclusively chrysotile) fibre counts in the air, as measured by personal samplers, in a Chinese asbestos textile plant were 6.5, 12.6, 4.5, 2.8 and 0.1 fibre/mL in the raw material (opening), raw material (bagging), textile, rubber plate and asbestos cement sections of the plant; in 2002, the median asbestos fibre counts were 4.5, 8.6 and 1.5 fibres/mL in the raw material, textile and rubber plate parts of the plant *(15)*.

In 2006, the geometric mean asbestos fibre count in the air in the largest chrysotile mine in China was 29 fibres/mL, as estimated from gravimetric dust measurements. Available data indicated that up to 1995, dust concentrations had been 1.5–9 times higher *(11)*.

The geometric mean occupational exposures to asbestos fibres were 0.40, 1.70 and 6.70 fibres/mL in the construction, asbestos friction and asbestos textile industries in 1984 in the Republic of Korea; in 1996, the corresponding figures were 0.14, 0.55 and 1.87 fibres/mL *(39)*. Park and colleagues *(40)* analysed 2089 asbestos exposure data sets compiled from 1995 through 2006 from 84 occupational sites. Asbestos exposure levels decreased from 0.92 fibre/mL in 1996 to 0.06 fibre/mL in 1999, possibly in part because of enforcement of 1997 legislation banning the use of amosite and crocidolite. During the periods 2001–2003 and 2004–2006, mean asbestos exposure levels declined further to 0.05 and 0.03 fibre/mL, respectively. The mean concentration in the major primary asbestos production plants was 0.31 fibre/mL, and in the secondary asbestos industries (handlers and end uses of asbestos-containing

materials), 0.05 fibre/mL. In particular, a substantial reduction in asbestos exposure levels was evident among primary industries handling raw asbestos directly. In this industry, exposure dropped from 0.78 fibre/mL (period 1995–1997) to 0.02 fibre/mL (period 2003–2006).

In Thailand, breathing zone asbestos concentrations in 1987 in roof tile, cement pipe, vinyl floor tile, asphalt undercoat and acrylic paint plants and in brake and clutch shops were < 1.11, 0.12–2.13, < 0.18, < 0.06 and 0.01–58.46 fibres/mL, respectively. The brake and clutch shops were small-scale enterprises, in contrast to the others; they had high asbestos air concentrations also in 2000 (0.24–43.31 and 0.62–2.41 fibres/mL for the brake and clutch shops, respectively) *(41)*.

The occupational exposure limit for chrysotile has been lowered in the USA since the 1970s: from 12 fibres/mL in 1971 to 5 fibres/mL in 1972, 2 fibres/mL in 1976, 0.2 fibre/mL in 1986 and 0.1 fibre/mL in 1994 *(42)*. The occupational exposure limit for all asbestos species is also 0.1 fibre/mL in the Bolivarian Republic of Venezuela *(43)*, the European Union *(44)*, India *(36)*, Indonesia *(45)*, Malaysia *(46)*, Norway *(47)*, the Republic of Korea *(39)*, Singapore *(27)* and the provinces of Alberta and British Columbia in Canada *(48)*. Other occupational exposure limits for all asbestos fibres include 0.01 fibre/mL in the Netherlands *(49)*; 0.15 fibre/mL in Japan *(26)*; 0.2 fibre/mL in South Africa *(50)*; 0.8 fibre/mL in China *(11, 35)*; and 2 fibres/mL in Brazil *(48)* and the Philippines *(28)*. In Thailand, the labour law sets the limit for airborne asbestos at 5 fibres/mL *(41, 45)*. In Canada, the occupational exposure limit for chrysotile is 1 fibre/mL *(51)*.

Health effects

The key studies on the main health end-points associated with exposure to chrysotile have been summarized in Table 1 (see page 39).

Cancer of the lung

Studies in experimental animals

Bronchial carcinomas were observed in many experiments in rats after inhalation exposure to chrysotile fibres. There was no consistent increase in tumour incidence at other sites (except mesothelioma, see below) *(1)*.

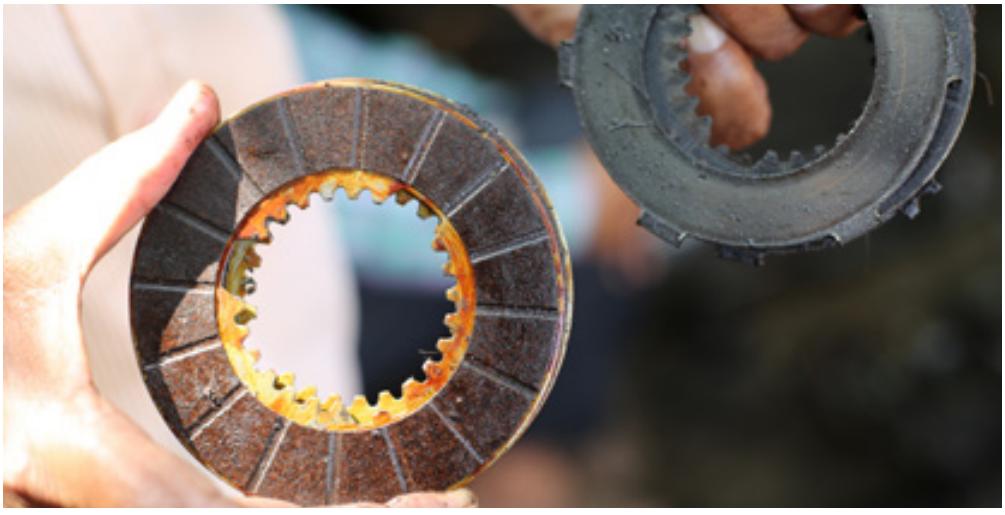
Studies in humans

Occupational exposure

In the final report on male workers in chrysotile mines in Quebec, Canada *(3)*, there was an exposure-related increase in mortality from lung cancer, reaching a standardized mortality ratio (SMR) of 2.97 (95% confidence interval [CI]: 2.18–3.95) in the most heavily exposed group. There was little difference between workers in the Asbestos and Thetford Mines areas of Quebec; in the latter area, the chrysotile was (to a small extent) contaminated with tremolite.

An elevated mortality from lung cancer (SMR: 1.49; 95% CI: 1.17–1.87) was observed in a cohort of chrysotile friction product plant workers in Connecticut, USA. Some anthophyllite was used in some product lines during the last 20 years of the follow-up *(52)*.

The risk of lung cancer was greatly increased among asbestos textile workers, mainly exposed to chrysotile, who received compensation for work-induced asbestosis in Italy (SMR: 6.82; 95% CI: 3.12–12.95). There was no quantitative estimation of what the exposure to "mainly chrysotile" represented *(53)*.



Among workers with at least 1 year's work experience between 1946 and 1987 in a chrysotile mine in Balangero, northern Italy, the lung cancer SMR was 1.27 (95% CI: 0.93–1.70) during the follow-up to 2003 *(5)*. No fibrous amphiboles were found, but 0.2–0.5% of a fibrous silicate, balangeroite, was identified in the chrysotile mined *(54)*.

Among workers of eight chrysotile asbestos factories in China with at least 15 years of work experience and followed from 1972 to 1986, the mortality from lung cancer was elevated (relative risk [RR]: 5.3; 95% CI: 2.5–7.1). The lung cancer risk was especially high among heavy smokers (chrysotile-exposed non-smokers: RR: 3.8 [95% CI: 2.1–6.3]; chrysotile-exposed light smokers: RR: 11.3 [95% CI: 4.3–30.2]; chrysotile-exposed medium smokers: RR: 13.7 [95% CI: 6.9–24.6]; chrysotile-exposed heavy smokers: RR: 17.8 [95% CI: 9.2–31.3]) *(8)*.

In a study in an asbestos textile plant in South Carolina, USA, the exposure was almost exclusively to chrysotile (part of the time, approximately 0.03% of the total amount of fibre used was crocidolite, which was never carded, spun or twisted and was woven wet). The lung cancer SMR was 1.95, with a 95% CI of 1.68–2.24. Exposure–response modelling for lung cancer, using a linear relative risk model, produced a slope coefficient of 0.0198 fibre-years/mL[4] (standard error 0.004 96) when cumulative exposure was lagged 10 years *(6)*.

Elevated mortality from lung cancer has been observed in chrysotile mine workers, chrysotile friction product plant workers and textile mill workers exposed to chrysotile

In a cohort study in four asbestos textile mills in North Carolina, USA, workers with at least 1 day's work between 1950 and 1973 were followed for mortality to 2003. In one of the plants, a small amount of amosite was used between 1963 and 1976, whereas the others used exclusively chrysotile *(7)*. In subsequent analysis of fibres from North Carolina and South Carolina by transmission electron microscopy, 0.04% of the fibres were identified as amphiboles *(55)*. Lung cancer mortality was elevated in an exposure-related fashion and reached an SMR of 2.50 (95% CI: 1.60–3.72) in the high-exposure category. The risk of lung cancer increased with cumulative fibre exposure (rate ratio: 1.102 per 100 fibre-years/mL, 95% CI: 1.044–1.164, for total career exposure) *(7)*.

Non-occupational exposure

There are few studies on lung cancer in people with non-occupational exposure to asbestos and even fewer in which chrysotile specifically has been investigated.

In a cohort of 1964 wives (not working in the asbestos mills) of asbestos cement workers in Casale Monferrato, Italy, the risk of dying from lung cancer was slightly elevated (SMR: 1.50; 95% CI: 0.55–3.26). The asbestos used was mainly chrysotile, but included approximately 10% crocidolite *(56)*. A slightly elevated lung cancer risk was observed among spouses of workers in an amosite factory in New Jersey, USA (SMR for male spouses of workers with more than 20 years of exposure, 1.97 [95% CI: 1.12–3.44], and for female spouses of workers with more than 20 years of exposure, 1.70 [95% CI: 0.73–3.36]) *(57)*.

[4] Cumulative exposure is expressed in units of (fibres/mL) × years. These units are given hereafter as fibre-years/mL.

Meta-analyses

In an informal meta-analysis of 13 studies with dose–response information available in 1986, WHO estimated the risk of lung cancer and mesothelioma in asbestos-exposed smokers and non-smokers *(58)*. Most of these studies have since been updated, new studies have become available and formal meta-analyses of studies on lung cancer among chrysotile-exposed workers have been performed, with the main aim to investigate the carcinogenic potency of chrysotile, especially in comparison with that of amphibole asbestos species. Another objective of the meta-analyses has been the elucidation of possible differences in the carcinogenic potency of fibres of different dimensions (i.e. length and thickness).

Lash et al. *(59)* conducted a meta-analysis based on the findings from 22 published studies on 15 asbestos-exposed cohorts with quantitative information on asbestos exposure and lung cancer mortality. Substantial heterogeneity was found in the slopes for lung cancer between these studies. The heterogeneity was largely explained by industry category (mining and milling, cement and cement products, or manufacturing and textile products), considered to reflect the stages of asbestos fibre refinement, dose measurements, tobacco habits and standardization procedures. There was no evidence that differences in fibre type (predominantly chrysotile, chrysotile mixed with other, or other) would explain the heterogeneity of the slope – in other words, there was no difference in the potency to cause lung cancer between the different fibre types.

Hodgson & Darnton *(60)* performed a meta-analysis based on 17 cohort studies with information on the level of asbestos exposure. Marked heterogeneity was observed in the potency slope derived from different chrysotile-exposed cohorts; the risk estimated from the South Carolina, USA, asbestos textile plants (approximately 6% per fibre-year/mL) was similar to the average in the amosite-exposed cohorts (5% per fibre-year/mL), whereas that from the Quebec, Canada, mine studies was only 0.06% per fibre-year/mL, and the studies in asbestos cement and friction product plants were intermediate in risk. Hodgson & Darnton *(60)* decided to exclude the South Carolina study from the calculation, mainly because the risk derived for the cohorts with mixed exposure (chrysotile + amphibole) was approximately 10% of that with pure amphibole exposures, and concluded that the potency of chrysotile to cause lung cancer was 2–10% of that of the amphiboles. Their "best estimate" for excess lung cancer from exposure to pure chrysotile was 0.1% per fibre-year/mL. However, the IARC Working Group *(1)* noted that there is no justification for exclusion of the South Carolina cohort, because it is one of the highest-quality studies in terms of the exposure information used in the study. An alternative explanation of the large difference in the risk estimates from the mining studies and the asbestos textile studies (also observed in the meta-analysis of Lash et al. *(59)*) could be the differences in fibre dimensions: a larger percentage of long fibres was found in samples from the South Carolina cohort *(61)* compared with what was previously reported in samples from the Quebec mines and mills *(62)*. A further possible cause of the difference is the difference in the quality of the exposure data *(18)*.

Berman & Crump *(63, 64)* published a meta-analysis that included data from 15 asbestos cohort studies. Lung cancer risk potency factors, based on a linear exposure–cancer risk relationship, were derived for fibre type (chrysotile versus amphiboles) and fibre size (length and width).

As with the previous analyses, substantial variation was found in these studies, with results for lung cancer varying by 2 orders of magnitude. The slope factor for chrysotile was 0.000 29 (fibre-year/mL)$^{-1}$ for Quebec mining and 0.018 (fibre-year/mL)$^{-1}$ for the South Carolina textile workers. That for tremolite (vermiculite mines and milling operations in Libby, Montana, USA) was 0.0026 (fibre-year/mL)$^{-1}$, with an upper uncertainty level of 0.03 (fibre-year/mL)$^{-1}$, and that for amosite insulation, 0.024 (fibre-year/mL)$^{-1}$ *(64)*.

In a further analysis of the fibre dimensions, the hypothesis that long chrysotile fibres are equipotent to long amphibole fibres was rejected for thin fibres (width < 0.2 μm), but not for fibres of all widths or for thick fibres (width > 0.2 μm). When the South Carolina cohort was dropped in a sensitivity analysis, the potency in the remaining studies in the meta-analysis was significantly greater for amphiboles than for chrysotile ($P = 0.005$). Dropping the Quebec cohort resulted in there being no evidence of a significant difference in potency between the fibre types ($P = 0.51$) *(63)*.

It is not possible to draw firm conclusions concerning the relative potency of chrysotile and amphibole asbestos fibres

The IARC Working Group *(1)* noted that both the Hodgson & Darnton *(60)* and Berman & Crump *(63, 64)* analyses reveal a large degree of heterogeneity in the study findings for lung cancer and that findings are highly sensitive to the inclusion or exclusion of the studies from South Carolina or Quebec. The reasons for the heterogeneity are unknown; until they are explained, it is not possible to draw firm conclusions concerning the relative potency of chrysotile and amphibole asbestos fibres.

IARC conclusions on cancer of the lung

In respect of cancer of the lung, IARC concluded that there is *sufficient evidence* of carcinogenicity in humans for all types of asbestos, including chrysotile. This is the strongest IARC category for describing the strength of evidence *(1)*.

Key new studies

Hodgson & Darnton *(65)* updated their meta-analysis of the lung cancer and mesothelioma risks from exposure to different asbestos species following the publication of data for the North Carolina, USA, chrysotile textile workers and noted that their original "best estimate", 0.1% per fibre-year/mL, was practically identical to the estimate from the North Carolina cohort (RR: 1.102 per 100 fibre-years/mL).

In a cohort study in the largest chrysotile mine in Quinghai, China, all male workers (n = 1539) employed at the beginning of 1981 were followed until the end of 2006. Mortality from different causes was compared with the national rates. Using a method with a sensitivity of 0.001%, no amphiboles were detected in the ore. The fibre exposure (estimated from gravimetric dust measurements in 2006) was 2.9–63.8 fibres/mL. The SMR for lung cancer was 4.71 (95% CI: 3.57–6.21). The SMR for the non-smoking chrysotile-exposed workers (miners and millers) was 1.79 (95% CI: 0.49–6.51), and that for the non-smoking referents (rear services and administration), 1.05 (95% CI: 0.19–5.96). For the smoking miners/millers, the SMR was 5.45 (95% CI: 4.11–7.22), and for the smoking referents, 1.66 (95% CI: 0.71–3.88) *(11)*. Lung cancer mortality increased with increasing estimated fibre exposure, and the SMR was 1.10 (95% CI: 0.47–2.28), 4.41 (95% CI: 2.52–7.71), 10.88 (95% CI: 6.70–17.68) and 18.69 (95% CI: 12.10–28.87) in the groups with estimated cumulative exposures of < 20, 20–100, > 100–450 and > 450 fibre-years/mL, respectively *(12)*. In an overlapping study of all 1932 workers employed for at least half a year between 1981 and 1988 and followed until 2010, the lung cancer SMR among the group considered directly exposed was 2.50 (95% CI: 1.85–3.24) *(10)*.

In the largest chrysotile factory in China, situated in Chongqing, in a follow-up of 584 male workers for 37 years, the SMR for lung cancer was 4.08 (95% CI: 3.12–5.33) *(14, 15)*. The risk increased with estimated exposure and was seen in both non-smokers and smokers. In females ($n = 277$), with a total employment time of only 19 years, a statistically non-significant excess of lung cancer was observed (SMR: 1.23; 95% CI: 0.34–4.50). The chrysotile used in the factory was from a single source in China, and the content of tremolite was less than 0.001% *(66)*. An RR of 1.23 (95% CI: 1.10–1.38) per 100 fibre-years/mL was estimated by fitting a log-linear model with a 10-year exposure lag *(67)*.

In 2011, Lenters and co-workers *(18)* analysed the association of the quality of exposure assessment with the estimated lung cancer potency of asbestos exposure in a meta-analysis of 18 industrial cohorts and 1 population-based case–referent study. Stratification by exposure assessment characteristics revealed that studies with well documented exposure assessment, larger contrast in exposure, greater coverage of the exposure history by exposure measurement data and more complete job histories had higher potency slope values than did studies without these characteristics. Differences in potency for chrysotile compared with amphibole asbestos were less evident when the meta-analysis was restricted to studies with higher-quality exposure data *(18)*.

In order to better evaluate the carcinogenic potency of asbestos fibres at low exposure levels, van der Bij and collaborators *(19)* applied, in addition to linear dose–exposure models, a spline function to the lung cancer and exposure data from the studies with no fewer than two risk estimates at different exposure levels. The spline function has the advantage that responses at high exposures do not excessively determine the dose–response relationships at low exposure levels. They found that in exposure to chrysotile alone, the relative lung cancer risks at lifetime exposures to 4 and 40 fibre-years/mL were 1.006 and 1.064, respectively (natural spline function with correction for intercept). After stratification by fibre type, a non-significant 3- to 4-fold difference in RRs between chrysotile and amphibole fibres was found for exposures below

40 fibre-years/mL. The difference in potency between chrysotile and amphiboles thus was considerably smaller than in the earlier analyses *(60, 63)*. As in the other meta-analyses, risk estimates for chrysotile were very different for the South Carolina, USA, and Quebec, Canada, studies.

Kumagai and coworkers *(68)* assessed the relationship between lung cancer mortality and asbestos exposure in the vicinity of an asbestos factory, based on meteorological modelling of the town of Hashima, Japan, where an amosite–chrysotile plant operated in 1943–1991. Excluding individuals with occupational exposure to asbestos or silica, lung cancer risk was elevated among those with highest estimated environmental asbestos exposure (SMR: 3.5; 95% CI: 1.52–5.47).

Malignant mesothelioma has been linked to occupational, domestic and environmental exposure to asbestos

The standardized incidence ratio (SIR) for lung cancer during a 10-year period in 15 villages in Turkey with environmental asbestos exposure was 1.82 (95% CI: 1.42–2.22) in men and 1.80 (95% CI: 1.43–2.00) in women, in comparison with 12 villages with no asbestos exposure. The estimated lifetime asbestos exposure range was 0.19–4.61 fibre-years/mL; the fibre type was either tremolite or a mixture of tremolite + actinolite + chrysotile or anthophyllite + chrysotile. Lung cancer risk was elevated in both non-smokers (SIR: 6.87; 95% CI: 3.58–13.20) and smokers (SIR: 12.50; 95% CI: 7.54–20.74) *(69)*.

Mesothelioma

Studies in experimental animals

After intrapleural or intraperitoneal injection of chrysotile, mesothelioma induction was consistently observed in rats, when samples contained a sufficient number of fibres with a fibre length of greater than 5 µm. In several studies in rats, mesotheliomas were also observed after inhalation exposure to chrysotile *(1)*.

Studies in humans

Occupational exposure

An excess of mesothelioma has been reported in cohort studies of chrysotile-exposed miners and millers (38 cases out of a total of 6161 deaths) in Quebec, Canada *(3)*, and of asbestos textile workers (3 cases out of 1961 deaths) in South Carolina, USA, who were predominantly exposed to chrysotile asbestos imported from Quebec *(6)*. However, the fact that chrysotile mined in Quebec is contaminated with a small percentage (< 1%) of amphibole asbestos (tremolite) complicates the interpretation of these findings. McDonald et al. *(70)* found that in the Quebec mining areas, the mortality from mesothelioma was 3 times higher among workers from mines in Thetford Mines, a region with higher concentrations of tremolite, than among those from mines in Asbestos, with lower concentrations of tremolite. However, Begin et al. *(71)* noted that although tremolite levels may be 7.5 times higher in Thetford Mines than in Asbestos, the rate of mesothelioma in the asbestos mine/mill workforce of these two towns was similar. This does not support the notion that the tremolite content of the ores is the determinant of mesothelioma risk in Quebec chrysotile workers.

No cases of mesothelioma among the total of 803 deaths were observed in the Connecticut, USA, friction material plant workers exposed to chrysotile *(52)*.

There were two cases of malignant pleural tumours among asbestos textile workers who received compensation for work-induced asbestosis in Italy; this represents a greatly increased risk (SMR: 22.86; 95% CI: 2.78–82.57). There was a more pronounced increase in the risk of peritoneal tumours. The exposure was described as "mainly chrysotile", but no quantitative data on the exposure were provided *(53)*.

Among 126 cases of mesothelioma identified in six referral hospitals in South Africa, 23 cases had mined Cape crocidolite; 3 had mined amosite; and 3, crocidolite plus amosite. None had purely chrysotile exposure *(72)*. It should be noted that chrysotile mining began later, and production levels were lower than in the crocidolite and amosite mines of South Africa.

Cases of mesothelioma have been reported among asbestos miners in Zimbabwe *(73)*. Chrysotile from Zimbabwe has been reported to contain 3 orders of magnitude less tremolite than that from Thetford Mines, Quebec *(74)*.

Asbestos textile workers in North Carolina, USA, were primarily exposed to chrysotile imported from Quebec, Canada. Large excesses of both mesothelioma (SMR: 10.92; 95% CI: 2.98–27.96) and pleural cancer (SMR: 12.43; 95% CI: 3.39–31.83) were observed *(7)*.

Two cases of mesothelioma were observed in the 1990 study in the Balangero, Italy, chrysotile mine *(54)*. However, in a follow-up until 2003, four pleural and one abdominal mesothelioma were identified, giving SMRs of 4.67 (95% CI: 1.27–11.96) for pleural mesothelioma and 3.16 (95% CI: 1.02–7.36) for all mesothelioma *(5)*.

Non-occupational exposure

Since the first large case-series published by Wagner and co-workers *(75)* linking malignant mesothelioma to occupational, domestic and environmental exposure to asbestos, at least 376 cases of mesothelioma for which domestic exposure to asbestos has been considered the causative agent have been published in some 60 scientific papers *(76)*.

Three cases of mesothelioma were identified in 1980–2006 from the mesothelioma registry in Piedmont, northern Italy, among white collar workers of the Balangero chrysotile mine, three among employees of a subcontractor working as lorry drivers in the mine, four among persons living in the vicinity of the mine, one the wife of a mine worker and five cases who had had contact with the main tailings *(4)*. No fibrous amphiboles were found, but 0.2–0.5% of a fibrous silicate, balangeroite, was identified in the chrysotile mined in Balangero *(54)*.

In a cohort of 1780 wives (not working in the asbestos mills) of asbestos cement workers in Casale Monferrato, Italy, the risk of dying from malignant pleural tumours was elevated in 1965–2003 (SMR: 18.00; 95% CI: 11.14–27.52). The asbestos used was mainly chrysotile, but included approximately 10% crocidolite *(56, 77)*. The incidence of histologically verified pleural mesothelioma in 1999–2001 was also elevated in a roughly latency- and exposure duration–dependent way, reaching an SIR of 50.59 (95% CI: 13.78–129.53) in the group with a latency of at least 40 years and duration of exposure of at least 20 years.

In a population-based case–referent study in a local health area of Casale Monferrato, Italy, the association between non-occupational asbestos exposure and malignant mesothelioma was examined for 116 cases of mesothelioma diagnosed in 1987–1993 and 330 referents. The odds ratio (OR) for the cases to be a spouse of an asbestos worker was 4.5 (95% CI: 1.8–11.1); the OR for the cases to be a child of an asbestos worker was 7.4 (95% CI: 1.9–28.1). The risk was inversely related to the distance between the residence and the asbestos factory, reaching an OR of 27.7 (95% CI: 3.1–247.7) for those ever living less than 500 m from the factory. In 1984, the average asbestos concentrations in the air were reported to be 0.011 fibre/mL close to the plant and 0.001 fibre/mL in the residential area. In different studies, the proportion of amphiboles varied between 3% and 50% of total asbestos fibres *(78)*.

Of the 162 female cases of fatal mesothelioma in Canada and the USA in 1966–1972, three occurred in wives of workers in Quebec chrysotile mines *(79)*. In a case–referent study among wives of workers in Quebec chrysotile mines, the risk of living with a mine worker for less than 40 years was associated with a mesothelioma risk of 3.9 (95% CI: 0.4–35); the risk of living with a mine worker for more than 40 years was associated with a risk of 7.5 (95% CI: 0.8–72). All cases had lived with a worker from the mine in Thetford Mines, where the chrysotile ore was contaminated with tremolite *(80)*.

In several countries or regions in different parts of the world – Turkey, Greece, Cyprus, Corsica, Sicily, New Caledonia, Yunnan province, China, and California, USA – there are areas with a high incidence of mesothelioma, apparently caused by asbestos or erionite in soil *(1, 81)*.

In a case–referent study of 1133 mesothelioma cases and 890 referents in California, the risk of mesothelioma was observed to be inversely related to the distance of the residence from naturally occurring asbestos ultramafic rocks, which contain serpentinic asbestos. The mesothelioma risk decreased with an SMR of 0.937 (95% CI:

0.895–0.982) per 10 km of distance, adjusted for age and probability of occupational asbestos exposure *(82)*.

In a case–referent study of 68 cases of mesothelioma in New Caledonia, the prevalence of mesothelioma in different parts of the island was related to the serpentinite content of the soil, not to mining activity or the use of the traditional lime, "pö", to cover houses *(83)*.

Meta-analyses

From a meta-analysis of cohort studies with quantitative information on exposure, Hodgson & Darnton *(60)* estimated that the excess mesothelioma risk was 0.1% per fibre-year/mL for cohorts exposed to chrysotile.

The meta-analysis conducted by Berman & Crump *(64)* was based on the analysis of the slopes that were estimated assuming that the mortality rate from mesothelioma increases after exposure ceases approximately as the square of time since first exposure (lagged 10 years). The slope factor, indicating potency, was estimated to be 0.15×10^{-8} per year^2 × fibres/mL for the South Carolina, USA, plants and 0.018×10^{-8} per year^2 × fibres/mL for the Quebec, Canada, mines, representing exposure to chrysotile, whereas the estimate for the Patterson, New Jersey, USA, factory where the asbestos species used was amosite was 3.9×10^{-8} per year^2 × fibres/mL. In a further analysis in which fibre size was considered, the hypothesis that chrysotile and amphibole forms of asbestos are equipotent was strongly rejected ($P \leq 0.001$), and the hypothesis that the potency of chrysotile asbestos was zero was not rejected ($P \geq 0.29$).

The IARC Working Group *(1)* noted that there is a high degree of uncertainty concerning the accuracy of the relative potency estimates derived from the Hodgson & Darnton *(60)* and Berman & Crump *(64)* analyses because of the severe potential for exposure misclassification in these studies.

The study of textile workers in North Carolina, USA *(7)*, was not included in the meta-analyses. Based on the approach used by Hodgson & Darnton *(60)*, the authors of the North Carolina study *(7)* estimated that the percentage of deaths was 0.0098% per

fibre-year/mL for workers followed for at least 20 years. This estimate is considerably higher than the original estimate developed by Hodgson & Darnton *(60)* of 0.001% per fibre-year/mL for cohorts exposed to chrysotile.

Bourdes and coworkers *(84)* performed a meta-analysis of available studies on household and neighbourhood exposure to asbestos and mesothelioma risk and came up with estimated summary RRs of 8.1 (95% CI: 5.3–12) for household exposure and 7.0 (95% CI: 4.7–11) for neighbourhood exposure.

IARC conclusions on mesothelioma

In respect of mesothelioma, IARC concluded that there is *sufficient evidence* of carcinogenicity in humans for all types of asbestos, including chrysotile. This is the strongest IARC category for describing the strength of evidence *(1)*.

Key new studies

Hodgson & Darnton *(65)* updated their meta-analysis of the potency of different asbestos fibres to cause mesothelioma following the publication of the North Carolina, USA, study *(7)* and revised their potency estimate upward to 0.007% per fibre-year/mL.

Of a total of 259 deaths in the Chinese asbestos factory workers *(16)*, 2 were from mesothelioma, whereas no mesotheliomas were reported among the 428 total deaths in the Chinese chrysotile miner cohort *(11)*. The tremolite content of the chrysotile studied in these studies was less than 0.001%. In a brief report, it was stated that the mesothelioma incidence in the asbestos (almost exclusively chrysotile) production areas in China was 85/1 000 000, whereas it was 1/1 000 000 in the general population *(35)*. It is not clear what proportion of the excess risk observed is due to environmental exposure and what proportion is due to occupational exposure.

Exposure to asbestos was studied among 229 malignant mesothelioma patients identified from the Australian Mesothelioma Registry and diagnosed between 2010 and 2012. For 70, no occupational exposure was discovered; these included 37 who had performed a major renovation of their housing with asbestos-containing materials, 35 who had lived in a house during a renovation with asbestos-containing materials, 19 who had lived in a house built of fibro (asbestos cement sheet), 19 who had lived with someone working in an asbestos-exposed job, 12 who had performed brake/clutch work (non-professionally), 10 who had visited Wittenoom (the western Australian city

with a crocidolite mine) and 8 who lived in the vicinity of an asbestos mine or asbestos products factory (total does not add to 70 because a number of participants were counted in more than one category) *(85)*.

In a case–referent study in the United Kingdom, exposure to asbestos was studied by detailed interview of 622 mesothelioma patients and 1420 population referents. The OR for living with an exposed worker before the age of 30 years was 2.0 (95% CI: 1.3–3.2). No information was available on the fibre type *(86)*.

The prevalence of malignant pleural mesothelioma was elevated in the vicinity of a chrysotile asbestos plant in north Cairo, Egypt. The increased prevalence was limited to the immediate vicinity of the factory and people estimated to have had a cumulative exposure of 20 fibre-years/mL *(87)*. (This study was not included in the meta-analysis of Goswami and co-workers *(88)* described below.)

In a cohort study of inhabitants of 15 villages in Turkey with environmental asbestos exposure and 12 villages with no such exposure, there were 14 deaths from mesothelioma in men out of a total of 79 cancer deaths; for women, the number of mesothelioma deaths was 17 out of a total of 40 cancer deaths. The estimated lifetime asbestos exposure range was 0.19–4.61 fibre-years/mL; the fibre type was either tremolite or a mixture of tremolite + actinolite + chrysotile or anthophyllite + chrysotile *(69)*. (This study was not included in the meta-analysis of Goswami and co-workers *(88)* described below.)

Occupational exposure to chrysotile also causes non-malignant lung diseases

In a meta-analysis of 12 cohort and case–referent studies on mesothelioma after domestic exposure to asbestos, Goswami and coworkers *(88)* estimated a summary RR of 5.02 (95% CI: 2.48–10.13). In six studies, the fibre type was not specified; in one, it was chrysotile; and in four, it was chrysotile with other fibres.

Asbestosis

Of 8009 deaths among Quebec, Canada, miners and millers in 1972–1992, 108 were caused by pneumoconiosis *(3)*. In the South Carolina, USA, cohort, the SMR for pneumoconiosis and other pulmonary diseases was 4.81 (95% CI: 3.84–5.94), and that for asbestosis, 232.5 (95% CI: 162.8–321.9); there were 36 deaths from asbestosis and 86 from pneumoconiosis out of a total of 1961 deaths *(6)*. In the North Carolina, USA, chrysotile textile worker cohort, the SMR for pneumoconiosis was 3.48 (95% CI: 2.73–4.38) *(7)*.

The SMR for asbestosis in the Chinese chrysotile textile cohort was 100 (95% CI: 72.55–137.83) *(14)*. In the Balangero, Italy, mine cohort, there were 21 cases of asbestosis out of a total of 590 deaths *(5)*.

One should note, however, that the pneumoconioses have never been reliably recorded as a cause of death on death certificates. Additionally, mortality studies are generally not sufficient to detect clinically significant morbidity. Equally, in studies of morbidity, the etiological or diagnostic specificity of the usual methods of assessment (i.e. chest radiography, physiological testing and symptom questionnaire) is limited. Many

studies show that exposure to chrysotile induces decrement in lung function, radiological changes consistent with pneumoconiosis and pleural changes *(2)*.

A dose-related reduction in vital capacity ($P = 0.023$) and expiratory volume ($P < 0.001$) was observed with increasing cumulative exposure (i.e. > 8 fibre-years/mL) to chrysotile asbestos in miners and millers in Zimbabwe who were exposed for more than 10 years *(89)*.

Chest X-ray changes among textile and friction product workers in China were reported by Huang *(90)*. A cohort of 824 workers employed for at least 3 years in a chrysotile products factory from the start-up of the factory in 1958 until 1980, with follow-up through to September 1982, was studied. Overall, 277 workers were diagnosed with asbestosis during the follow-up period, corresponding to a period prevalence of 31%. Exposure–response analysis, based on gravimetric data converted to fibre counts, predicted a 1% prevalence of Grade I asbestosis at a cumulative exposure of 22 fibre-years/mL.

Asbestosis was also detected in 11.3% of wives of asbestos-exposed shipyard workers with a 20-year work history and in 7.6% of their sons. The asbestos type was not specified *(91)*. One or more radiological signs of asbestosis were observed in 35% of the household contacts of amosite asbestos insulation workers *(92)*. The prevalence of pleural calcifications was increased 10.2-fold (95% CI: 2.8–26.3) among blood relatives of workers in chrysotile asbestos factories and 17.0-fold (95% CI: 7.7–32.2) among people living in the vicinity of a factory using Russian and Canadian chrysotile asbestos *(93)*.

IPCS conclusions

In addition to lung cancer and mesothelioma, occupational exposure to chrysotile also causes non-malignant lung diseases that result in deterioration in lung function, in particular a form of lung fibrosis described by the term asbestosis *(2)*.

Global burden of disease

No studies are available specifically on the global burden of disease caused by chrysotile. However, more than 90% of all asbestos used historically and practically all asbestos used today is chrysotile; thus, the estimates made of the populations exposed to asbestos are largely directly valid for chrysotile.

Cancer of the lung

Based on the methods of Driscoll et al. *(33)*, the burden of disease estimate for lung cancer was updated by Prüss-Üstün and collaborators *(94)*. Using the combined relative risk (SMR 2.0) of lung cancer in 20 cohort studies published by 1994 *(95)* and the estimated proportion of the population actually exposed to asbestos in the different WHO regions, Prüss-Üstün and collaborators *(94)* estimated that in the year 2004, asbestos caused 41 000 lung cancer deaths and 370 000 disability-adjusted life years (DALYs).

In an effort to estimate the global lung cancer burden from exposure to asbestos, McCormack and co-workers *(96)* studied the ratio of excess lung cancer deaths to excess mesothelioma deaths associated with exposure to different asbestos fibre types. This ratio was 6.1 (95% CI: 3.6–10.5) in the 16 available chrysotile-exposed cohorts. The authors were not able to derive an estimate for the total number of deaths or DALYs for asbestos-induced lung cancer. They concluded that in exposure to chrysotile, the observation of few mesothelioma deaths cannot be used to infer "no excess risk" of lung or other cancers.

In the year 2004, asbestos caused 41 000 lung cancer deaths

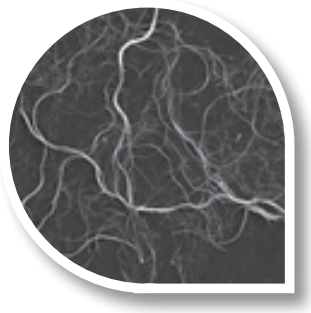
Mesothelioma

Driscoll and co-workers *(33)* estimated the global burden of mesothelioma deaths and DALYs based on the notion that mesothelioma is nearly always caused by exposure to asbestos, using the proportion of workers in different economic sectors (agriculture, mining, manufacturing, electrical, construction, trade, transport, finance and services) who are exposed to asbestos in Europe, the population numbers in these subsectors, as developed in the CAREX database by the Finnish Institute of Occupational Health, and an average mesothelioma risk for different asbestos species from the study of Hodgson & Darnton *(60)*. The global burden estimates, updated for the year 2004 worldwide, were 59 000 deaths and 773 000 DALYs from malignant mesothelioma *(33, 97)*.

Asbestosis

Driscoll and co-workers *(98)* estimated the global burden of asbestosis deaths and DALYs based on the notion that asbestos is

the only cause of asbestosis, using the proportion of workers in different economic sectors (agriculture, mining, manufacturing, electrical, construction, trade, transport, finance and services) who are exposed to asbestos in Europe, the population numbers in these subsectors, as developed in the CAREX database by the Finnish Institute of Occupational Health, and published risks of developing asbestosis at different levels of exposure to chrysotile *(99)*. The global burden estimates for the year 2000 worldwide were 7000 deaths and 380 000 DALYs from asbestosis.



Chrysotile substitute fibres[5]

A WHO Workshop on Mechanisms of Fibre Carcinogenesis and Assessment of Chrysotile Asbestos Substitutes *(100)* was convened at IARC in Lyon, France, in response to a request from the Intergovernmental Negotiating Committee for the Rotterdam Convention on the Prior Informed Consent Procedure for Certain Hazardous Chemicals and Pesticides in International Trade (INC). The substitutes considered by the WHO workshop included the 12 chrysotile substitutes identified by the INC for priority assessment by WHO, 2 substances from a second list provided by the INC to be assessed if resources allow and 1 further substance for which data were submitted in response to WHO's public "call for data" for the workshop.

Methodological aspects

The workshop established a framework for hazard assessment based on epidemiological data, in vivo experimental animal data on carcinogenicity and potential to cause lung fibrosis, and mechanistic information, genotoxicity data and biopersistence data as determinants of dose at the target site and possible indicators of carcinogenic potential. Noting that substitutes may be used in a variety of applications with different exposure potential, either alone or in combination with other substances, the

[5] This section is largely taken from reference *100*.

workshop did not embark on risk assessment, but rather limited its work to assessing the hazard.

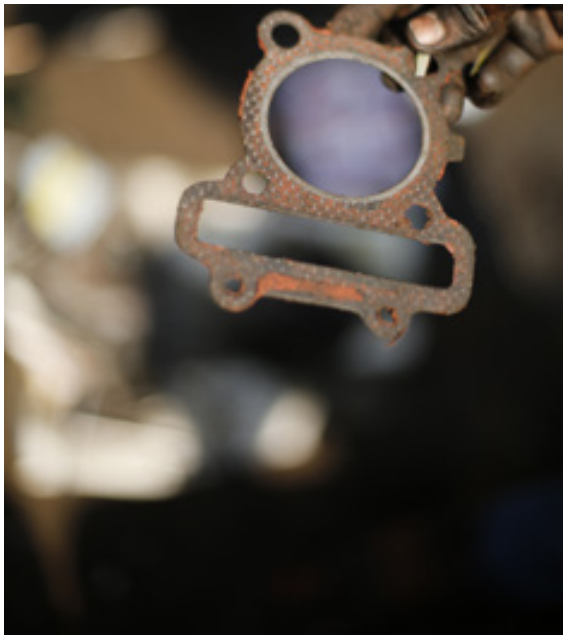
The workshop concluded that epidemiological studies on fibres have a clear advantage over toxicological studies, in that they involve studies of humans. They also have the advantage that they study the effects of exposure in the real world, where the effects of these exposures may be mitigated or enhanced by other factors. Despite these obvious advantages, the presence or absence of evidence of risk from epidemiological studies does not always override contrary findings from toxicological studies. The interpretation of either positive or non-positive epidemiological findings needs to be carefully considered in light of the strengths and weaknesses of the study design.

Carcinogenic response in experimental animals (lung cancer, mesothelioma) and fibrosis were considered to be the key effects; epithelial cell proliferation and inflammation were not regarded to be equally important indicators of human health hazard. From studies with asbestos, it is apparent that the sensitivity of the rat to fibre-induced lung tumours in inhalation studies is clearly lower than that of humans. This holds true when the effect is related to exposure concentrations and lung burdens. In comparison, testing of fibres by intraperitoneal injection represents a useful and sensitive assay, which also avoids the confounding effects of granular dusts.

The global burden estimates for the year 2000 worldwide were 7000 deaths and 380 000 DALYs from asbestosis

Fibres may act in principle on all steps in tumour development. However, of these interactions, the in vitro genotoxicity tests are mainly indicative of genotoxic effects involved in the first steps of tumour initiation. Effects related to biopersistence of fibres (e.g. continuous “frustrated phagocytosis”) and secondary genotoxicity arising from reactive oxygen and nitrogen species and mitogen release by macrophages and inflammatory cells are not detected in routinely used genotoxicity tests. Therefore, negative results indicate a lack of primary genotoxicity, but do not exclude effects on later steps of carcinogenesis.

The chemical composition of the substitutes is a key factor influencing their structure and physicochemical properties, such as surface area, surface reactivity and solubility. Attention should be paid not only to the chemical composition of the fibres, including their major and trace elements, but also to contaminants or accompanying elements, including their speciation. Fibre-derived free radical generation favours DNA damage and mutations. Surface properties are a determining factor in the inflammatory response. In relation to fibre dimension and deposition, one can assume that there exists a continuous variation in the carcinogenic potency of respirable fibres, which increases with



length. Biopersistence of a fibre increases tissue burden and therefore may increase any toxicity the fibre might possess. For synthetic vitreous fibres, there is evidence in experimental animals that the potential for carcinogenicity increases with biopersistence. This has not been demonstrated, however, for other fibres. For all fibres, the fibres must be respirable to pose an appreciable hazard.

Respirability is mainly determined by diameter and density; thus, with a given fibre diameter, a higher specific density is associated with lower respirability (note that the specific density of most organic fibres is lower than the specific density of inorganic fibres).

Hazard assessment

The workshop decided to group substitutes roughly into hazard groupings of high, medium and low. However, for some substitutes, there was insufficient information to draw any conclusion on hazard; in these cases, the workshop categorized the hazard as indeterminate (a category that is not comparable to the other groupings). The hazard groups high, medium and low should be considered in relation to each other and do not have reference to formal criteria or definitions, as such. It is important to note that for each substitute, the fibre dimensions of commercially available products may vary, and the workshop did not assess this variation. The substitutes are listed below in alphabetical order.

***para*-Aramid** releases respirable fibres with dimensions similar to those of known carcinogenic fibres. *p*-Aramid fibres have induced pulmonary effects in animal inhalation studies. Biopersistence was noted. The workshop considered the human health hazard to be **medium**.

Most natural deposits contain **attapulgite** fibres that are less than 5 µm in length; at workplaces, the mean fibre length was less than 0.4 µm. The hazard from exposure to respirable attapulgite is likely to be **high for long fibres** and **low for short fibres**. This assessment is mainly based on findings in long-term inhalation experiments in

animals, in which tumours were seen with long fibres; no tumours were seen in studies with short fibres.

The nominal diameter of **carbon fibres** ranges from 5 to 15 µm. Workplace exposure in production and processing is mostly to non-respirable fibres. The workshop considered the hazard from inhalation exposure to these fibres to be **low**.

Most **cellulose fibres** are not respirable; for these, the hazard is **low**. For respirable fibres, the available data do not allow the evaluation of the hazard; the hazard is thus **indeterminate**.

The dimensions of **graphite whiskers** indicate high respirability, and they have a long half-time in the lungs. However, in the absence of any further useful information, the hazard from inhalation exposure was considered to be **indeterminate**.

Magnesium sulfate whiskers did not induce tumours in limited inhalation and intratracheal administration studies, were negative in limited short-term tests and are very quickly eliminated from the lung. It was discussed whether the hazard grouping should be **low** or **indeterminate**. On the basis of the data available, in the time available, consensus was not reached.

The fibres must be respirable to pose an appreciable hazard

For respirable **polyethylene**, **polyvinyl chloride** and **polyvinyl alcohol fibres**, the data were insufficient for hazard classification, and the working group thus considered the hazard **indeterminate**.

In facilities producing **polypropylene fibres**, exposure to respirable fibres occurs. After intratracheal administration, respirable polypropylene fibres were highly biopersistent; however, no fibrosis was reported in a subchronic animal study. However, the data are sparse, and the human health hazard potential was considered to be **indeterminate**.

The workshop considered that respirable **potassium octatitanate fibres** are likely to pose a **high** hazard to humans after inhalation exposure. At workplaces, there is exposure to respirable fibres. There was a high and partly dose-dependent incidence of mesothelioma after intraperitoneal injection in two species (high incidence indicating high potency). There is evidence of genotoxicity. Biopersistence was noted.

Wool-like **synthetic vitreous fibres** (including glass wool/fibrous glass, mineral wool, special-purpose vitreous silicates and refractory ceramic fibre) contain respirable fibres. For these fibres, the major determinants of hazard are biopersistence, fibre dimensions and physicochemical properties. It was noted that the available epidemiological data are not informative, due to mixed (vitreous fibre) exposures or other design limitations. Based on inhalation exposure studies, intraperitoneal injection studies and biopersistence studies, it was concluded that the carcinogenic hazard could vary from high to low, with **high** for the biopersistent fibres and **low** for the non-biopersistent fibres.

Natural **wollastonite** contains respirable fibres. In occupational settings, exposure is mainly to short fibres. In chronic studies, wollastonite did not induce tumours after intraperitoneal injection in animals; however, samples of wollastonite were active in different studies for genotoxicity. After considering this apparent discrepancy, it was concluded that the hazard was likely to be **low.**

In a limited study with intraperitoneal implantation, **xonotlite** did not induce tumours. After intratracheal injection in a chronic study, no inflammatory or fibrotic reaction of the lung was observed. The chemical composition of xonotlite is similar to that of wollastonite, but it is more rapidly eliminated from the lung. The workshop considered the human health hazard to be **low**.

Table 1. Key findings of the cohort studies on the adverse health effects of chrysotile asbestos

Industry and location	Exposure to chrysotile	Exposure to other fibres	Deaths from all causes	Lung cancer deaths SMR (95% CI)	Mesothelioma deaths SMR (95% CI)	Pneumoconiosis/ asbestosis deaths	References
Chrysotile mining/milling in Quebec, Canada	Average 600 fibre-years/mL	< 1% tremolite	8 009	657 1.37 (1.27–1.48)	38	108/ND	*3, 60*
Friction products factory in Connecticut, USA	Average 46 fibre-years/mL	Some anthophyllite in use during the last 20 years of follow-up	803	73 1.49 (1.17–1.87)	0	12/0	*52, 60*
Asbestos textile mill in Italy, women with compensated asbestosis	ND	"Mainly chrysotile"[a]	123	9 6.82 (3.12–12.95)	ND	ND/21	*53*
Asbestos textile mills in South Carolina, USA	99% < 200 fibre-years/mL, average 26–28 fibre-years/mL	0.04% amphiboles	1 961	198 1.95 (1.68–2.24)	3	85/36	*6, 55*
Asbestos textile mills in North Carolina, USA	Average (range) 17.1 (< 0.1–2 943.4) fibre-years/mL	0.04% amphiboles	2 583	277 1.96 (1.73–2.20)	4[b]	73/36	*7, 55, 60*
Chrysotile mine in Balangero, Italy	< 100 – ≥ 400 fibre-years/mL	No amphiboles, 0.2–0.5% balangeroite	590	45 1.27 (0.93–1.70)	4 4.67 (1.27–11.96)	ND/21	*5*
Chrysotile mine in Quinghai, China	Average in 2006, 2.9–63.8 fibres/mL	≤ 0.001% amphiboles	428	56 4.71 (3.57–6.21)	0[c]	ND	*11*
Eight chrysotile textile factories in China	ND	ND[d]	496	65 5.3 (2.5–7.1)	2	ND/29[e]	*8*
Asbestos manufacturing factory in China	Median 1, 8 and 23 fibres/mL in different departments	≤ 0.001% amphiboles	259	53 4.08 (3.12–5.33)	2	ND/39	*15*

ND: no data

[a] No further data on other possible asbestos fibre types.

[b] Mesothelioma data available only for 1999–2003 of the total follow-up period of 1953–2003.

[c] The authors note that mesothelioma may be underreported.

[d] The published paper has no information on the asbestos species, but most likely it is the Chinese chrysotile with < 0.001% amphiboles.

[e] The text of the paper states that there were 148 cases of asbestosis, not 29 as in the tables.

References

1. International Agency for Research on Cancer. Asbestos (chrysotile, amosite, crocidolite, tremolite, actinolite, and anthophyllite). IARC Monogr Eval Carcinog Risks Hum. 2012;100C:219–309 (http://monographs.iarc.fr/ENG/Monographs/vol100C/index.php, accessed 11 March 2014).
2. Environmental Health Criteria 203: Chrysotile asbestos. Geneva: World Health Organization, International Programme on Chemical Safety; 1998 (http://www.inchem.org/documents/ehc/ehc/ehc203.htm, accessed 11 March 2014).
3. Liddell FD, McDonald AD, McDonald JC. The 1891–1920 birth cohort of Quebec chrysotile miners and millers: development from 1904 and mortality to 1992. Ann Occup Hyg. 1997;41(1):13–36.
4. Mirabelli D, Calisti R, Barone-Adesi F, Fornero E, Merletti F, Magnani C. Excess of mesotheliomas after exposure to chrysotile in Balangero, Italy. Occup Environ Med. 2008;65(12):815–9.
5. Pira E, Pelucchi C, Piolatto PG, Negri E, Bilei T, La Vecchia C. Mortality from cancer and other causes in the Balangero cohort of chrysotile asbestos miners. Occup Environ Med. 2009;66(12):805–9.
6. Hein MJ, Stayner LT, Lehman E, Dement JM. Follow-up study of chrysotile textile workers: cohort mortality and exposure–response. Occup Environ Med. 2007;64(9):616–25.
7. Loomis D, Dement JM, Wolf SH, Richardson DB. Lung cancer mortality and fibre exposures among North Carolina asbestos textile workers. Occup Environ Med. 2009;66(8):535–42.
8. Zhu H, Wang Z. Study of occupational lung cancer in asbestos factories in China. Br J Ind Med. 1993;50(11):1039–42.
9. Zhong F, Yano E, Wang ZM, Wang MZ, Lan YJ. Cancer mortality and asbestosis among workers in an asbestos plant in Chongqing, China. Biomed Environ Sci. 2008;21(3):205–11.
10. Du L, Wang X, Wang M, Lan Y. Analysis of mortality in chrysotile asbestos miners in China. J Huazhong Univ Sci Technolog Med Sci. 2012;32(1):135–40.
11. Wang X, Lin S, Yano E, Qiu H, Yu IT, Tse L et al. Mortality in a Chinese chrysotile miner cohort. Int Arch Occup Environ Health. 2012;85(4):405–12.
12. Wang X, Yano E, Lin S, Yu ITS, Lan Y, Tse LA et al. Cancer mortality in Chinese chrysotile asbestos miners: exposure–response relationships. PLoS One. 2013;8(8):e71899.
13. Wang X, Courtice MN, Lin S. Mortality in chrysotile asbestos workers in China. Curr Opin Pulm Med. 2013;19(2):169–73.
14. Wang X, Lin S, Yu I, Qiu H, Lan Y, Yano E. Cause-specific mortality in a Chinese chrysotile textile worker cohort. Cancer Sci. 2013;104(2):245–9.
15. Wang X, Yano E, Qiu H, Yu I, Courtice MN, Tse LA et al. A 37-year observation of mortality in Chinese chrysotile asbestos workers. Thorax. 2012;67(2):106–10.
16. Wang XR, Yu IT, Qiu H, Wang MZ, Lan YJ, Tse L et al. Cancer mortality among Chinese chrysotile asbestos textile workers. Lung Cancer. 2012;75(2):151–5.
17. Yano E, Wang X, Wang M, Qiu H, Wang Z. Lung cancer mortality from exposure to chrysotile asbestos and smoking: a case–control study within a cohort in China. Occup Environ Med. 2010;67(12):867–71.
18. Lenters V, Vermeulen R, Dogger S, Stayner L, Portengen L, Burdorf A et al. A meta-analysis of asbestos and lung cancer: is better quality exposure assessment associated with steeper slopes of the exposure–response relationships? Environ Health Perspect. 2011;119(11):1547–55.
19. van der Bij S, Koffijberg H, Lenters V, Portengen L, Moons KG, Heederik D et al. Lung cancer risk at low cumulative asbestos exposure: meta-regression of the exposure–response relationship. Cancer Causes Control 2013;24(1):1–12.
20. Black C, Lofty G, Sharp N, Hillier J, Singh D, Ubbi M et al. World mineral statistics 1975–1979. London: Institute of Geological Sciences; 1981 (http://www.bgs.ac.uk/mineralsuk/statistics/worldArchive.html, accessed 11 March 2014).
21. Virta RL. Asbestos [Advance release]. In: 2012 minerals yearbook. Reston (VA): United States Department of the Interior, United States Geological Survey; 2013:8.1–8.7 (http://minerals.usgs.gov/minerals/pubs/commodity/asbestos/myb1-2012-asbes.pdf, accessed 11 March 2014).
22. Virta RL. Asbestos statistics and information. In: Mineral commodity summaries 2013. Reston (VA): United States Department of the Interior, United States Geological Survey; 2013 (http://minerals.usgs.gov/minerals/pubs/commodity/asbestos/mcs-2013-asbes.pdf, accessed 11 March 2014).

23. Virta RL. Worldwide asbestos supply and consumption trends from 1900 through 2003. Circular 1298. Reston (VA): United States Department of the Interior, United States Geological Survey; 2006 (http://pubs.usgs.gov/circ/2006/1298/c1298.pdf, accessed 11 March 2014).

24. Kazan-Allen L. Current asbestos bans and restrictions. International Ban Asbestos Secretariat; 2014 (http://www.ibasecretariat.org/lka_alpha_asb_ban_280704.php, accessed 16 March 2014).

25. De Castro H. Aspectos Sobre la Producción del Amianto, Exposición y Vigilancia de los Trabajadores Expuestos al Amianto en Brasil. Cienc Trab. 2008;10(27):11–7.

26. Furuya S, Takahashi K, Movahed M, Jiang Y. National asbestos profile of Japan. Based on the national asbestos profile by the ILO and the WHO. Japan Occupational Safety and Health Resource Center and University of Occupational and Environmental Health, Japan; 2013 (http://envepi.med.uoeh-u.ac.jp/NAPJ.pdf, accessed 11 March 2014).

27. Lee H, Chia K. Asbestos in Singapore: country report. J UOEH. 2002;24(Suppl 2):36–41.

28. Villanueva M, Granadillos M, Cucuecco M, Estrella-Gust D. Asbestos in the Philippines: country report. J UOEH. 2002;24(Suppl 2):70–5.

29. Rahayu D, Wantoro B, Hadi S. 4. Indonesia. In: Kang D, Kim J-U, Kim K-S, Takahashi K, editors. Report on the status of asbestos in Asian countries November 2012. Pusan: World Health Organization; 2012:51–60.

30. Chrysotile asbestos: Priority Existing Chemical Report No. 9. Full public report. Canberra: National Industrial Chemicals Notification and Assessment Scheme; 1999 (http://www.nicnas.gov.au/__data/assets/pdf_file/0014/4370/PEC_9_Chrysotile-Asbestos_Full_Report_PDF.pdf, accessed 11 March 2014).

31. International Standard Industrial Classification of All Economic Activities, Revision 2. United Nations Statistics Division (http://unstats.un.org/unsd/cr/registry/regct.asp?Lg=1, accessed 25 March 2014).

32. Kauppinen T, Toikkanen J, Pedersen D, Young R, Kogevinas M, Ahrens W et al. Occupational exposure to carcinogens in the European Union in 1990–1993. CAREX International Information System on Occupational Exposure to Carcinogens. Helsinki: Finnish Institute of Occupational Health; 1998 (http://www.ttl.fi/en/chemical_safety/carex/Documents/1_description_and_summary_of_results.pdf, accessed 23 March 2014).

33. Driscoll T, Nelson DI, Steenland K, Leigh J, Concha-Barrientos M, Fingerhut M et al. The global burden of disease due to occupational carcinogens. Am J Ind Med. 2005;48(6):419–31.

34. Concha-Barrientos M, Nelson D, Driscoll T, Steenland N, Punnett L, Fingerhut M et al. Chapter 21. Selected occupational risk factors. In: Ezzati M, Lopez A, Rodgers A, Murray C, editors. Comparative quantification of health risks: global and regional burden of disease attributable to selected major risk factors. Geneva: World Health Organization; 2004:1651–801 (http://www.who.int/healthinfo/global_burden_disease/cra/en/, accessed 11 March 2014).

35. Wang X. 2. China. In: Kang D, Kim J-U, Kim K-S, Takahashi K, editors. Report on the status of asbestos in Asian countries November 2012. Pusan: World Health Organization; 2012:33–43.

36. Sane A. 3. India. In: Kang D, Kim J-U, Kim K-S, Takahashi K, editors. Report on the status of asbestos in Asian countries November 2012. Pusan: World Health Organization; 2012:44–50.

37. BK-Report 1/2007 Faserjahre. Sankt Augustin: Hauptverband der gewerblichen Berufsgenossenschaften (HVBG); 2007 (http://www.yumpu.com/de/document/view/5278685/bk-report-1-2007-faserjahre-deutsche-gesetzliche-, accessed 11 March 2014).

38. Kaufer E, Vincent R. Occupational exposure to mineral fibres: analysis of results stored on COLCHIC database. Ann Occup Hyg. 2007;51(2):131–42.

39. Paek D, Choi J. Asbestos in Korea: country report. J UOEH. 2002;24(Suppl 2):42–50.

40. Park D, Choi S, Ryu K, Park J, Paik N. Trends in occupational asbestos exposure and asbestos consumption over recent decades in Korea. Int J Occup Environ Health. 2008;14(1):18–24.

41. Taptagaporn S, Siriruttanapruk S. Asbestos in Thailand: country report. J UOEH. 2002;24(Suppl 2):81–5.

42. Martonik JF, Nash E, Grossman E. The history of OSHA's asbestos rulemakings and some distinctive approaches that they introduced for regulating occupational exposure to toxic substances. AIHAJ. 2001;62(2):208–17.

43. Mujica N, Arteta J. Asbesto en Venezuela. Cienc Trab. 2008;10(27):21–24.

44. European Commission. Directive 2009/148/EC of the European Parliament and of the Council of 30 November 2009 on the protection of workers from the risks related to exposure to asbestos at work. Off J Eur Union. 2009; L 330:28–36.

45. Kang D, Kim J-U, Kim K-S, Takahashi K. Report on the status of asbestos in Asian countries November 2012. Pusan: World Health Organization; 2012.

46. Rampal K, Chye G. Asbestos in Malaysia: country report. J UOEH. 2002;24(Suppl 2):76–80.

47. Forskrift om tiltaks- og grenseverdier. Trondheim: Direktoratet for arbeidstilsynet; 2014 (http://www.arbeidstilsynet.no/binfil/download2.php?tid=237714, accessed 24 March 2014).

48. Documentation of the TLVs® and BEIs® with other worldwide occupational exposure values [CD-ROM]. Cincinnati (OH): American Conference of Governmental Industrial Hygienists; 2007.

49. Asbestos. Risks of environmental and occupational exposure. The Hague: Gezondheidsraad (Health Council of the Netherlands); 2010 (http://www.gezondheidsraad.nl/sites/default/files/201010E.pdf, accessed 11 March 2014).

50. Asbestos Regulations, 2001. Department of Labour, Republic of South Africa; 2002 (http://www.labour.gov.za/DOL/legislation/regulations/occupational-health-and-safety/regulation-ohs-asbestos-regulations-2001/?searchterm=asbestos regulations, accessed 23 March 2014).

51. Canada Occupational Health and Safety Regulations. SOR/86–304. Ottawa: Minister of Justice; 2013 (http://laws-lois.justice.gc.ca/PDF/SOR-86-304.pdf, accessed 23 March 2014).

52. McDonald AD, Fry JS, Woolley AJ, McDonald JC. Dust exposure and mortality in an American chrysotile asbestos friction products plant. Br J Ind Med. 1984;41(2):151–7.

53. Germani D, Belli S, Bruno C, Grignoli M, Nesti M, Pirastu R et al. Cohort mortality study of women compensated for asbestosis in Italy. Am J Ind Med. 1999;36(1):129–34.

54. Piolatto G, Negri E, La Vecchia C, Pira E, Decarli A, Peto J. An update of cancer mortality among chrysotile asbestos miners in Balangero, northern Italy. Br J Ind Med. 1990;47(12):810–4.

55. Loomis D, Dement JM, Elliott L, Richardson D, Kuempel ED, Stayner L. Increased lung cancer mortality among chrysotile asbestos textile workers is more strongly associated with exposure to long thin fibres. Occup Environ Med. 2012;69(8):564–8.

56. Magnani C, Terracini B, Ivaldi C, Botta M, Budel P, Mancini A et al. A cohort study on mortality among wives of workers in the asbestos cement industry in Casale Monferrato, Italy. Br J Ind Med. 1993;50(9):779–84.

57. Anderson HA. Family contact exposure. In: Proceedings of the World Symposium on Asbestos. Montreal: Canadian Asbestos Information Centre; 1982:349–62.

58. 6.2 Asbestos. In: Air quality guidelines for Europe, second edition. WHO Regional Publications, European Series, No. 91. Copenhagen: World Health Organization Regional Office for Europe; 2000 (http://www.euro.who.int/__data/assets/pdf_file/0005/74732/E71922.pdf, accessed 11 March 2014).

59. Lash TL, Crouch EA, Green LC. A meta-analysis of the relation between cumulative exposure to asbestos and relative risk of lung cancer. Occup Environ Med. 1997;54(4):254–63.

60. Hodgson JT, Darnton A. The quantitative risks of mesothelioma and lung cancer in relation to asbestos exposure. Ann Occup Hyg. 2000;44(8):565–601.

61. Dement JM, Kuempel ED, Zumwalde RD, Smith RJ, Stayner LT, Loomis D. Development of a fibre size–specific job–exposure matrix for airborne asbestos fibres. Occup Environ Med. 2008;65(9):605–12.

62. Gibbs G, Hwang C. Dimensions of airborne asbestos fibres. IARC Sci Publ. 1980;30:69–78.

63. Berman DW, Crump KS. A meta-analysis of asbestos-related cancer risk that addresses fiber size and mineral type. Crit Rev Toxicol. 2008;38(Suppl 1):49–73.

64. Berman DW, Crump KS. Update of potency factors for asbestos-related lung cancer and mesothelioma. Crit Rev Toxicol. 2008;38(Suppl 1):1–47.

65. Hodgson JT, Darnton A. Mesothelioma risk from chrysotile. Comment on "Lung cancer mortality and fibre exposures among North Carolina asbestos textile workers" [Occup Environ Med. 2009]. Occup Environ Med. 2010;67(6):432.

66. Yano E, Wang ZM, Wang XR, Wang MZ, Lan YJ. Cancer mortality among workers exposed to amphibole-free chrysotile asbestos. Am J Epidemiol. 2001;154(6):538–43.

67. Deng Q, Wang X, Wang M, Lan Y. Exposure–response relationship between chrysotile exposure and mortality from lung cancer and asbestosis. Occup Environ Med. 2012;69(2):81–6.

68. Kumagai S, Kurumatani N, Tsuda T, Yorifuji T, Suzuki E. Increased risk of lung cancer mortality among residents near an asbestos product manufacturing plant. Int J Occup Environ Health. 2010;16(3):268–78.

69. Metintas S, Metintas M, Ak G, Kalyoncu C. Environmental asbestos exposure in rural Turkey and risk of lung cancer. Int J Environ Health Res. 2012;22(5):468–79.

70. McDonald AD, Case BW, Churg A, Dufresne A, Gibbs GW, Sebastien P et al. Mesothelioma in Quebec chrysotile miners and millers: epidemiology and aetiology. Ann Occup Hyg. 1997;41(6):707–19.

71. Begin R, Gauthier JJ, Desmeules M, Ostiguy G. Work-related mesothelioma in Quebec, 1967–1990. Am J Ind Med. 1992;22(4):531–42.

72. Rees D, Myers JE, Goodman K, Fourie E, Blignaut C, Chapman R et al. Case–control study of mesothelioma in South Africa. Am J Ind Med. 1999;35(3):213–22.

73. Cullen MR, Baloyi RS. Chrysotile asbestos and health in Zimbabwe: I. Analysis of miners and millers compensated for asbestos-related diseases since independence (1980). Am J Ind Med. 1991;19(2):161–9.

74. Lippmann M. Deposition and retention of inhaled fibres: effects on incidence of lung cancer and mesothelioma. Occup Environ Med. 1994;51:793–8.

75. Wagner JC, Sleggs CA, Marchand P. Diffuse pleural mesothelioma and asbestos exposure in the North Western Cape Province. Br J Ind Med. 1960;17:260–71.

76. Donovan EP, Donovan BL, McKinley MA, Cowan DM, Paustenbach DJ. Evaluation of take home (para-occupational) exposure to asbestos and disease: a review of the literature. Crit Rev Toxicol. 2012;42(9):703–31.

77. Ferrante D, Bertolotti M, Todesco A, Mirabelli D, Terracini B, Magnani C. Cancer mortality and incidence of mesothelioma in a cohort of wives of asbestos workers in Casale Monferrato, Italy. Environ Health Perspect. 2007;115(10):1401–5.

78. Magnani C, Dalmasso P, Biggeri A, Ivaldi C, Mirabelli D, Terracini B. Increased risk of malignant mesothelioma of the pleura after residential or domestic exposure to asbestos: a case–control study in Casale Monferrato, Italy. Environ Health Perspect. 2001;109(9):915–9.

79. McDonald AD, McDonald JC. Malignant mesothelioma in North America. Cancer. 1980;46(7):1650–6.

80. Case B, Camus M, Richardson L, Parent M, Desy M, Siemiatycki J. Preliminary findings for pleural mesothelioma among women in the Quebec chrysotile mining regions. Ann Occup Hyg. 2002;46(Suppl 1):128–31.

81. Baris YI, Grandjean P. Prospective study of mesothelioma mortality in Turkish villages with exposure to fibrous zeolite. J Natl Cancer Inst. 2006;98(6):414–7.

82. Pan XL, Day HW, Wang W, Beckett LA, Schenker MB. Residential proximity to naturally occurring asbestos and mesothelioma risk in California. Am J Respir Crit Care Med. 2005;172(8):1019–25.

83. Baumann F, Rougier Y, Ambroswi JP, Robineau BP. Pleural mesothelioma in New Caledonia: an acute environmental concern. Cancer Detect Prev. 2007;31(1):70–6.

84. Bourdes V, Boffetta P, Pisani P. Environmental exposure to asbestos and risk of pleural mesothelioma: review and meta-analysis. Eur J Epidemiol. 2000;16(5):411–7.

85. Mesothelioma in Australia 2012. Alexandria (NSW): Cancer Institute NSW, Australian Mesothelioma Registry, funded by Safe Work Australia and Comcare; 2012 (http://www.mesothelioma-australia.com/publications-and-data/publications, accessed 11 March 2014).

86. Rake C, Gilham C, Hatch J, Darnton A, Hodgson J, Peto J. Occupational, domestic and environmental mesothelioma risks in the British population: a case–control study. Br J Cancer. 2009;100(7):1175–83.

87. Madkour MT, El Bokhary MS, Awad Allah HI, Awad AA, Mahmoud HF. Environmental exposure to asbestos and the exposure–response relationship with mesothelioma. East Mediterr Health J. 2009;15(1):25–38.

88. Goswami E, Craven V, Dahlstrom DL, Alexander D, Mowat F. Domestic asbestos exposure: a review of epidemiologic and exposure data. Int J Environ Res Public Health. 2013;10(11):5629–70.

89. Cullen MR, Lopez-Carrillo L, Alli B, Pace PE, Shalat SL, Baloyi RS. Chrysotile asbestos and health in Zimbabwe: II. Health status survey of active miners and millers. Am J Ind Med. 1991;19(2):171–82.

90. Huang J. A study on the dose–response relationship between asbestos exposure level and asbestosis among workers in a Chinese chrysotile product factory. Biomed Environ Sci. 1990;3:90–8.

91. Kilburn KH, Lilis R, Anderson HA, Boylen CT, Einstein HE, Johnson SJ et al. Asbestos disease in family contacts of shipyard workers. Am J Public Health. 1985;75(6):615–7.

92. Anderson HA, Lilis R, Daum SM, Selikoff IJ. Asbestosis among household contacts of asbestos factory workers. Ann N Y Acad Sci. 1979;330:387–99.

93. Navratil M, Trippe F. Prevalence of pleural calcification in persons exposed to asbestos dust, and in the general population in the same district. Environ Res. 1972;5(2):210–6.

94. Prüss-Üstün A, Vickers C, Haefliger P, Bertollini R. Knowns and unknowns on burden of disease due to chemicals: a systematic review. Environ Health. 2011;10:9. doi: 10.1186/1476–069X-10–9.

95. Steenland K, Loomis D, Shy C, Simonsen N. Review of occupational lung carcinogens. Am J Ind Med. 1996;29(5):474–90.

96. McCormack V, Peto J, Byrnes G, Straif K, Boffetta P. Estimating the asbestos-related lung cancer burden from mesothelioma mortality. Br J Cancer 2012;106(3):575–84.

97. Global health risks: mortality and burden of disease attributable to selected major risks. Geneva: World Health Organization; 2009 (http://www.who.int/healthinfo/global_burden_disease/GlobalHealthRisks_report_full.pdf, accessed 11 March 2014).

98. Driscoll T, Nelson DI, Steenland K, Leigh J, Concha-Barrientos M, Fingerhut M et al. The global burden of non-malignant respiratory disease due to occupational airborne exposures. Am J Ind Med. 2005;48(6):432–45.

99. Stayner L, Smith R, Bailer J, Gilbert S, Steenland K, Dement J et al. Exposure–response analysis of risk of respiratory disease associated with occupational exposure to chrysotile asbestos. Occup Environ Med. 1997;54(9):646–52.

100. Summary consensus report of WHO Workshop on Mechanisms of Fibre Carcinogenesis and Assessment of Chrysotile Asbestos Substitutes, 8–12 November 2005, Lyon. Geneva: World Health Organization; 2005 (http://www.who.int/ipcs/publications/new_issues/summary_report.pdf, accessed 11 March 2014).

PUBLIC HEALTH AND ENVIRONMENT

Asbestos – a group of minerals that includes chrysotile, crocidolite, amosite, anthophyllite, tremolite and actinolite – is one of the most important occupational carcinogens. At least 107 000 people die each year from asbestos-related diseases, including lung cancer. Even though the use of asbestos has declined in many countries, chrysotile is still widely used, particularly in developing countries.

This publication on chrysotile asbestos is divided into three parts. The first part reproduces a WHO short information document for decision-makers on the elimination of asbestos-related diseases. The second part addresses questions commonly raised in policy discussions, specifically to assist decision-makers. The third part is a technical summary of the health effects of chrysotile, which brings together and summarizes for the first time the most recent authoritative WHO evaluations performed by its International Agency for Research on Cancer and its International Programme on Chemical Safety. The technical summary also reviews results from key studies published after those evaluations and the conclusions drawn from WHO assessments of alternatives.

The publication will be of interest to all government officials who need to make informed decisions about management of the health risks associated with exposure to chrysotile asbestos.

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