Asbesto crisotilo

Organización Mundial de la Salud

En la siguiente dirección web se encuentra más información de la OMS sobre sustancias químicas de especial importancia para la salud pública, incluido el asbesto:

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

Asbesto crisotilo

Organización Mundial de la Salud

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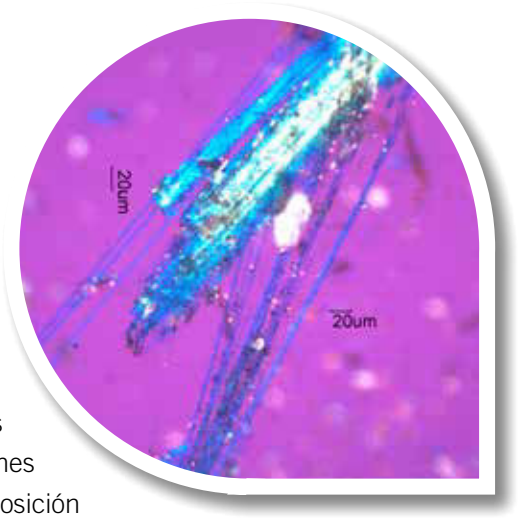

Muchos países han adoptado medidas a nivel nacional para prohibir el uso de todas las formas de asbesto a fin de limitar la exposición al mismo, y de ese modo, controlar, prevenir y –a la larga– eliminar las *enfermedades relacionadas con el asbesto*, que causan la muerte de al menos 107.000 personas cada año en todo el mundo. Sin embargo hay otros países que por diversas razones aún no lo han hecho. Teniendo eso en cuenta, el principal objetivo de esta publicación es ayudar a los Estados Miembros de la Organización Mundial de la Salud (OMS) a tomar decisiones informadas sobre la gestión de los riesgos para la salud asociados a la exposición al asbesto crisotilo.

El documento consta de tres partes. En la primera se reproduce un breve documento informativo de la OMS dirigido a las instancias decisorias acerca de la eliminación de las enfermedades relacionadas con el asbesto, el cual fue actualizado en marzo de 2014. La segunda parte aborda unas preguntas que con frecuencia se plantean en debates de política, con el objetivo específico de ayudar a los tomadores de decisiones a formarse una opinión. La tercera parte es un resumen técnico sobre los efectos del crisotilo en la salud, en el que se recogen y resumen por vez primera las evaluaciones oficiales más recientes de la OMS llevadas a cabo por su Centro Internacional de Investigaciones sobre el Cáncer y su Programa Internacional de Seguridad de las Sustancias Químicas. En este resumen técnico se analizan además los resultados de algunos estudios clave publicados después de dichas evaluaciones, y luego, se mencionan sucintamente las conclusiones de las evaluaciones realizadas por la OMS sobre productos sustitutos del asbesto.

Recomiendo esta publicación a ministros, funcionarios y demás personas que deseen o necesiten tomar decisiones, u ofrecer consejería a cerca del asbesto, y en particular del asbesto crisotilo y las consecuencias en la salud por exposición al mismo.

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Organización Mundial de la Salud

Eliminación de las enfermedades relacionadas con el asbesto

Actualizado en marzo de 2014

El asbesto es uno de los carcinógenos ocupacionales más importantes, que causa cerca de la mitad de las muertes por cáncer ocupacional (*1, 2*). En la 13ª reunión del Comité Conjunto de Salud Ocupacional OIT/OMS celebrada en 2003, se recomendó que se prestara especial atención a la eliminación de las *enfermedades relacionadas con el asbesto* (*3*). La Asamblea Mundial de la Salud, en su resolución WHA58.22 de 2005 sobre prevención y control del cáncer, instó a los Estados Miembros a que otorgaran especial atención a los cánceres relacionados con exposiciones evitables, en particular la exposición a sustancias químicas presentes en el lugar de trabajo y en el ambiente. En 2007, en la resolución WHA60.26 se exhortó a llevar a cabo campañas mundiales para eliminar las *enfermedades relacionadas con el asbesto*, y en 2013 la resolución WHA66.10 abordó la prevención y el control de las enfermedades no transmisibles, incluyendo el cáncer.

El asbesto es uno de los carcinógenos ocupacionales más importantes

El término «asbesto» designa un grupo de minerales naturales fibrosos que se presentan en forma de serpentinas o de anfíboles, que han tenido o siguen teniendo utilidad comercial debido a su extraordinaria resistencia a la tracción, su baja conductividad térmica y su relativa resistencia al ataque químico. Hay dos variedades principales de asbesto, a saber: las serpentinas, que corresponde al crisotilo o asbesto blanco; y los anfíboles, que incluyen la crocidolita, la amosita, la antofilita, la tremolita y la actinolita. (*4*).

La exposición al asbesto, incluido el crisotilo, causa cáncer de pulmón, laringe y ovario, mesotelioma (cáncer de pleura o peritoneo) y asbestosis (fibrosis pulmonar) (*5-7*).

La exposición al asbesto y su impacto en la salud pública son un problema relevante

La exposición al asbesto se produce por inhalación de las fibras, principalmente aquellas presentes en el aire contaminado del ambiente laboral; y también, en el aire próximo a puntos emisores o del interior de viviendas y locales construidos con materiales friables que contienen asbesto. Los mayores niveles de exposición se producen durante el re-empaque de contenedores de asbesto, durante la mezcla con otras materias primas, y al cortar productos que contienen asbesto en seco con herramientas abrasivas. También puede haber exposición durante la instalación y uso de productos que contienen asbesto y durante trabajos de mantenimiento de vehículos. Muchos edificios viejos todavía albergan materiales fabricados con crisotilo y/o anfíboles friables en su estructura, por lo cual siguen siendo una fuente de exposición a estas fibras en el curso de su mantenimiento, modificación, eliminación y demolición (*5*). La exposición puede deberse también a los daños sufridos por edificios como consecuencia de desastres naturales.

Actualmente hay cerca de 125 millones de personas expuestas al asbesto en su lugar de trabajo en el mundo (*1*). Se estima que a nivel global cada año mueren como mínimo 107.000

personas por cáncer de pulmón, mesotelioma y asbestosis debidos a la exposición ocupacional al asbesto (*1, 2, 8*). Además, cerca de 400 defunciones se han atribuido a exposiciones no ocupacionales al mismo. La carga de las enfermedades relacionadas con el asbesto sigue aumentando, incluso en países que prohibieron su utilización a principios de los años noventa. Debido al largo periodo de latencia de estas enfermedades, aunque se suprimiera su utilización de inmediato, el número de muertes que provoca solo comenzaría a disminuir después de varios decenios.

Todas las variedades de asbesto provocan cáncer en el hombre

El Centro Internacional de Investigaciones sobre el Cáncer ha clasificado el asbesto (actinolita, amosita, antofilita, crisotilo, crocidolita y tremolita) en la categoría de sustancias carcinógenas para el ser humano (*7*). La exposición al crisotilo, la amosita y la antofilita, así como a mezclas con crocidolita, aumenta el riesgo de desarrollar cáncer de pulmón (*7*). Se han detectado casos de mesotelioma tras la exposición laboral a la crocidolita, la amosita, la tremolita y el crisotilo, así como en poblaciones residentes en las cercanías de fábricas y minas de asbesto, y en personas que conviven con trabajadores que manipulan estos minerales (*7*).

La incidencia de enfermedades relacionadas con el asbesto depende del tipo, tamaño y cantidad (dosis) de fibras inhaladas, así como el proceso industrial de esas fibras (*6*). No se ha establecido un umbral de riesgo carcinogénico para el asbesto, incluido el crisotilo (*5, 7*). El tabaquismo aumenta el riesgo de cáncer de pulmón debido a la exposición al asbesto (*5, 9*).

Al menos 107.000 personas mueren cada año por causa de cáncer de pulmón, mesotelioma y asbestosis debidos a la exposición ocupacional al asbesto

El crisotilo se sigue usando ampliamente

El asbesto se ha utilizado para fabricar miles de productos destinados a aplicaciones muy diversas, tales como tejas para techos, tuberías para acueductos, cobijas incombustibles y material de aislamiento, así como componentes de embragues, frenos, juntas de culata y fieltros para automóviles. El uso del asbesto ha disminuido en muchos países debido al aumento de los problemas de salud que ocasiona. El uso de la crocidolita y los productos que contienen esta fibra, así como la pulverización de cualquier variedad de asbesto fue prohibido desde 1986, en virtud del Convenio n.º 162 de la OIT que trata *sobre utilización del asbesto en condiciones de seguridad.* Sin embargo el uso del crisotilo aún es muy amplio; aproximadamente el 90% de esta fibra se usa para fabricar materiales de fibrocemento para la construcción, cuyos principales consumidores son los países en desarrollo. El crisotilo también se utiliza en materiales de fricción (7%), textiles y otras aplicaciones (*10*).

Hasta la fecha (final de 2013), más de 50 países, incluida la totalidad de los Estados miembros de la Unión Europea, han prohibido el uso de todas las variedades de asbesto, incluyendo al crisotilo. Otros países han impuesto restricciones menos estrictas. Sin embargo, en los últimos años algunos países han mantenido o incluso aumentado la fabricación o el uso de crisotilo (*11*). Este incremento se observa sobre todo en la región de Asia-Pacífico. Entre 2000 y 2012 la producción mundial de asbesto se mantuvo relativamente estable, en aproximadamente 2 millones de toneladas por año (*12, 13*).

Recomendaciones de la OMS respecto a la prevención de las enfermedades relacionadas con el asbesto

Teniendo presente que se carece de evidencia para establecer el umbral del efecto carcinogénico del asbesto, incluido el crisotilo, y de que se ha observado un mayor riesgo de cáncer en poblaciones expuestas a niveles muy bajos (*5, 7*), la opción más eficiente para eliminar las enfermedades relacionadas con el asbesto es detener el uso de todas las variedades de asbesto. La persistencia del uso de materiales de asbesto-cemento en la construcción es motivo de especial preocupación debido a que la fuerza de trabajo es bastante numerosa, a la dificultad para controlar la exposición y al hecho de que los materiales utilizados pueden deteriorarse y poner en riesgo a los trabajadores que hacen restauración, trabajos de mantenimiento y demoliciones (*5*). En sus diversas aplicaciones, el asbesto puede ser reemplazado por algunos materiales fibrosos (*14*) y otros productos que tienen menor o ningún riesgo para la salud.

Los materiales que contienen asbesto deben aislarse, y como regla general, no se aconseja realizar trabajos que puedan alterar el estado de las fibras. Si es inevitable, este tipo de trabajo solo debe realizarse bajo estrictas medidas de control de la exposición, tales como el aislamiento, el procesamiento por vía húmeda, sistemas de ventilación local exhaustiva con filtros y limpieza sistemática. También es necesario utilizar equipo de protección personal –respiradores especiales, gafas de seguridad, así como ropa y guantes protectores– y prever instalaciones especiales para disponer de ellos y descontaminarlos (*15*).

La OMS está comprometida a trabajar con los países para eliminar las enfermedades relacionadas con el asbesto mediante las siguientes orientaciones estratégicas:

- reconocer que la opción más eficiente para eliminar las enfermedades relacionadas con el asbesto es detener el uso de todos los tipos de asbesto;
- suministrar información sobre las soluciones para reemplazar el asbesto con sustitutos más seguros y desarrollar mecanismos económicos y tecnológicos que fomenten la sustitución;
- tomar medidas para prevenir la exposición al asbesto instalado y durante los procesos de remoción del mismos;
- mejorar el diagnóstico precoz y el tratamiento de las enfermedades relacionadas con el asbesto y de los servicios de rehabilitación correspondientes, y establecer registros de las personas que estén o hayan estado expuestas a esas fibras minerales.

La OMS recomienda firmemente la planeación y aplicación de estas medidas en el marco de un plan nacional integral para la eliminación de las enfermedades relacionadas con el asbesto. Este enfoque deberá incluir también el establecimiento de perfiles nacionales, campañas de sensibilización, creación de capacidades, un marco institucional y un plan de acción nacional para la eliminación de las enfermedades relacionadas con el asbesto.

La OMS colaborará con la OIT para la aplicación de la Resolución relativa al asbesto aprobada en la nonagésima quinta reunión de la Conferencia Internacional del Trabajo (*16*); y también con otras organizaciones intergubernamentales y la sociedad civil hacia la eliminación de las enfermedades relacionadas con el asbesto en todo el mundo.

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El crisotilo en su estado natural

Preguntas y respuestas frecuentes

Esta sección se aborda algunas preguntas que se plantean a menudo los formuladores de políticas acerca del uso del crisotilo.

¿Es cierto que el crisotilo no es realmente una forma de asbesto?

No. El crisotilo es una de las seis formas de asbesto, siendo las otras la crocidolita, la amosita, la tremolita, la actinolita y la antofilita.

¿Cuál es la política de la OMS sobre el asbesto?

La política de la OMS sobre el asbesto es inequívoca. El asbesto causa cáncer de pulmón, laringe y ovario, mesotelioma (cáncer de pleura o peritoneo) y asbestosis (fibrosis pulmonar). Las enfermedades relacionadas con el asbesto pueden y deben prevenirse, y la opción más eficiente para lograrlo es dejar de usar cualquier forma de asbesto para prevenir la exposición. Las campañas mundiales emprendidas por la OMS para eliminar las enfermedades relacionadas con el asbesto tienen la finalidad de ayudar a los países a lograr dicho objetivo.

¿Por qué la OMS se preocupa tanto por el asbesto?

Hay clara evidencia científica de que el asbesto produce cáncer y enfermedades respiratorias crónicas en el ser humano. La OMS está trabajando para reducir la carga mundial de enfermedades no transmisibles, incluyendo el cáncer y las enfermedades respiratorias crónicas, reconociendo que la prevención primaria reduce el costo de la prestación de servicios de salud y contribuye a asegurar la sostenibilidad de costos sanitarios. El cáncer es la segunda causa de defunción a nivel mundial. En 2008 se registraron 7,6 millones de muertes por cáncer, y 12,7 millones de casos nuevos. Se estima que aproximadamente un 19% de todos los cánceres se pueden atribuir al ambiente, incluidos los entornos de trabajo.

En el mundo hay unos 125 millones de personas expuestas al asbesto en el lugar de trabajo. Según estimaciones de la OMS, cada año mueren como mínimo 107.000 personas por cáncer de pulmón, mesotelioma o asbestosis debidos a la exposición al asbesto en los lugares de trabajo. Se calcula que aproximadamente la mitad de todas las defunciones por cáncer ocupacional se deben al asbesto.

¿La OMS tiene competencia para hablar del crisotilo y otras formas del asbesto y su manejo?

La OMS es la autoridad directiva y coordinadora en el tema de salud dentro del Sistema de las Naciones Unidas. Es la entidad responsable de liderar los asuntos mundiales sobre la salud, configurar la agenda de investigación en salud, establecer normas y estándares, articular opciones políticas basadas en la evidencia, prestar asistencia técnica a los países y monitorear, vigilar y evaluar las tendencias mundiales de la salud.

La Asamblea Mundial de la Salud es el órgano decisorio supremo de la OMS; se reúne todos los años y asisten a ella las delegaciones de 194 Estados Miembro. Su principal función consiste en determinar las políticas de la Organización.

La política sobre el asbesto de la OMS se deriva de tres resoluciones de la Asamblea: la WHA58.22 de 2005, que trata el tema de los cánceres relacionados con exposiciones evitables a cancerígenos; la WHA60.26 de 2007, que exhorta a emprender campañas mundiales para eliminar las enfermedades relacionadas con el asbesto; y la WHA66.10 de 2013, que trata de la prevención y el control de las enfermedades no transmisibles, incluido el cáncer.

¿Cómo se expone la gente al asbesto?

La exposición al asbesto ocurre por inhalación, y en menor medida por ingestión, durante la extracción y la trituración del asbesto, y en la producción y uso de productos que lo contienen. Ello incluye la exposición en trabajos de construcción durante el corte y colocación de materiales con asbesto, el mantenimiento y la demolición de edificios. El asbesto generalmente se utiliza o se ha utilizado como una mezcla fibrosa, aglomerado con otros materiales (por ejemplo cemento, plásticos y resinas) o tejido como un textil. El asbesto se ha usado en una amplia gama de aplicaciones, tales como techos, placas de cemento para suelos y paredes, tuberías de cemento (por ejemplo para suministro de agua), sistemas de aislamiento térmico y eléctrico, incluidas cobijas y cortinas ignífugas industriales, juntas de culata y piezas de fricción (como pastillas o zapatas para frenos y embragues de vehículos). Hoy día la exposición a fibras de asbesto ocurre en circunstancias particulares en las que se ha producido un deterioro de los productos que lo contienen, como ocurre en el curso del mantenimiento y demolición de edificios y la disposición de residuos de construcción, así como en el contexto de desastres naturales.

Existen claras evidencias de que el asbestos causa cáncer y enfermedades respiratorias crónicas en los seres humanos

¿Por qué es tan importante ocuparse del asbesto como cancerígeno cuando hay tantos otros cancerígenos en el ambiente?

Se considera que algunos cánceres atribuibles a factores ambientales están determinados por múltiples cancerígenos. Otros, sin embargo, tienen como causa un carcinógeno único e identificable, como el tabaco o el asbesto, cuya exposición es prevenible. (Nota: no ocurre así con muchos de las otras sustancias clasificadas por el Centro Internacional de Investigaciones sobre el Cáncer [CIIC] en el Grupo 1, esto es, como cancerígenos para el ser humano, y la carga de morbilidad asociada a muchos de ellos tampoco es similar.[1])

Una de las razones por las cuales es importante que los países tomen medidas contra el asbesto lo antes posible es el periodo de latencia excepcionalmente largo entre la exposición y el desarrollo de mesotelioma, que con frecuencia es hasta de 40 años. Por esta razón, la carga de enfermedades relacionadas con el asbesto seguirá aumentando, incluso en los países que lo prohibieron hace muchos años.

Todas las formas de asbesto causan cáncer en el hombre (incluyendo al crisotilo, que es la principal variedad de asbesto que aún se produce y utiliza), y no se ha identificado un umbral de seguridad para su riesgo carcinogénico. Esta es la conclusión de la OMS y el CIIC tras una serie de evaluaciones internacionales acreditadas llevadas a cabo a lo largo de más de

[1] Para más información sobre los carcinógenos de la categoría 1 del CIIC, véase http://monographs.iarc.fr/ENG/Classification/ClassificationsGroupOrder.pdf.

15 años, siendo la más reciente publicada por el CIIC en 2012. Esas conclusiones reflejan el consenso internacional de los científicos expertos convocados por la OMS para evaluar los efectos del asbesto en la salud.

Además, se ha demostrado que la exposición coexistente al humo de tabaco y a las fibras de asbesto aumenta sustancialmente el riesgo de cáncer pulmonar, y ese efecto es cuando menos aditivo; es decir, cuanto más se fuma, mayor es el riesgo.

¿Podemos estar seguros de que las evaluaciones científicas del asbesto realizadas por la OMS y el CIIC son absolutamente independientes de cualquier influencia externa?

Sí. En todos los casos se adoptaron medidas para identificar y solucionar los posibles conflictos de interés, así como para garantizar que las evaluaciones fueran muy rigurosas e independientes de los puntos de vista de los gobiernos, las instituciones nacionales y de grupos de interés especiales; que se tuvieran en cuenta las opiniones de todas las regiones del mundo, y que fueran objeto de una exhaustiva revisión internacional inter pares.

¿Qué medidas han adoptado los países a nivel nacional?

Muchos países ya han promulgado leyes para prohibir el uso del asbesto. A día de hoy (final de 2013) más de 50 Estados Miembros de la OMS han actuado así para proteger y promover la salud pública.[2] Dicha decisión fue tomada tras una consulta interministerial, a fin de tener en cuenta los intereses sectoriales pero al mismo tiempo evitando que tuvieran demasiado peso en la decisión final. En el estudio de las posibles medidas legislativas contra la utilización del asbesto ha sido necesario tener en cuenta una serie de costos y beneficios, incluyendo los costos de la prestación de servicios de salud y aquellos relacionados con la pérdida de la productividad de la fuerza de trabajo por causa de problemas de salud crónicos, además de las consideraciones económicas y comerciales habituales.

¿Qué medidas han tomado o están proponiendo los países a nivel internacional?

El Convenio de Basilea sobre el Control de los Movimientos Transfronterizos de los Desechos Peligrosos y su Eliminación que entró en vigor en 1992 y del que son parte 181 países, tiene por objeto proteger la salud humana y el ambiente de los efectos adversos de los desechos peligrosos. El asbesto (polvo y fibra) figura como una categoría de desecho controlado en el marco del Convenio. Las Partes del Convenio deben prohibir o no permitir la exportación de ese tipo de desechos a las Partes que hayan prohibido su importación bajo el amparo de dicho tratado.

[2] Se trata de los siguientes países: Arabia Saudita, Argelia, Argentina, Australia, Bahrein, Brunei Darussalam, Chile, Egipto, Gabón, Honduras, Islandia, Israel, Japón, Jordania, Kuwait, Mozambique, Noruega, Omán, Qatar, República de Corea, Serbia, Seychelles, Sudáfrica, Suiza, Turquía, los 28 Estados miembros de la Unión Europea y Uruguay. El amianto está prohibido también en dos Estados del Brasil, Río de Janeiro y Rio Grande do Sul.

Un poco más reciente, una mayoría de 154 países que son Partes en el Convenio de Rotterdam sobre el procedimiento de Consentimiento Informado Previo aplicable a ciertos plaguicidas y productos químicos peligrosos que son objeto de comercio internacional (que entró en vigor en 2004), indicaron que desean que el crisotilo se incluya en el anexo 3 del Convenio. Ello significaría que el asbesto estaría sometido a un procedimiento por el cual se requeriría una decisión informada de un país antes de aceptar o no su importación en el futuro. Sin embargo, dicha inclusión en la lista ha sido bloqueada hasta ahora por un pequeño número de países; en forma predominante pero no exclusiva por los países que siguen teniendo intereses en el comercio y uso del crisotilo, y de los productos que lo contienen.

¿Es cierto que el crisotilo es menos nocivo que otros tipos de asbesto y, por consiguiente, no debería estar sometido a las mismas medidas de control?

Las evaluaciones hechas por la OMS y el CIIC concluyen firmemente que el crisotilo causa cáncer de pulmón, laringe y ovario, mesotelioma y asbestosis

La evidencia científica es clara. La firme conclusión de las evaluaciones de la OMS y el CIIC es que el crisotilo causa cáncer de pulmón, laringe y ovario, mesotelioma y asbestosis, bien si es o no menos potente que los anfíboles para hacerlo. Las afirmaciones acerca de las diferentes propiedades físico-químicas, la posibilidad de que estudios epidemiológicos históricos hayan sido realizados con formas de crisotilo contaminadas con variedades anfíboles, y la contención física del crisotilo en el cemento moderno de alta densidad (en el momento de la fabricación), son aspectos que no invalidan esa conclusión.

Una mayor preocupación surge cuando el uso del asbesto está adecuadamente regulado y los productos que contienen crisotilo (por ejemplo tejas para techos o tuberías de agua) sufren daños y liberan fibras de asbesto en el ambiente durante los trabajos de mantenimiento de edificios, en los procesos de demolición y de eliminación de los residuos de construcción, y como consecuencia de desastres naturales. Estas exposiciones pueden producirse algún tiempo después de la instalación original (controlada). Este riesgo puede evitarse totalmente si se deja de utilizar ese tipo de productos. La información sobre los materiales y sustitutos que se pueden usar con seguridad, se encuentra disponible en diversas organizaciones nacionales, regionales e internacionales.

¿Podrían las investigaciones actuales o futuras sobre la toxicidad del crisotilo llevar a la OMS y el CIIC a modificar la opinión que hoy tienen sobre el riesgo de cáncer?

De ninguna manera. La firme opinión de la OMS y el CIIC, basada en reiteradas evaluaciones de las pruebas científicas, es que el crisotilo causa cáncer de pulmón, laringe y ovario, mesotelioma y asbestosis; y que se debe reconocer que la suspensión del uso de todas las formas de asbesto, incluido el crisotilo, es la alternativa más eficaz para evitar la exposición y eliminar las enfermedades relacionadas con el asbesto. Ahora bien, aunque el potencial carcinógeno del crisotilo es incuestionable, son escasos los estudios que han incluido mujeres. Se sospecha que también hay otros tipos de cáncer que están relacionados con el crisotilo, para los cuales los estudios existentes son insuficientes. Así, pues, persiste la necesidad de llevar a cabo nuevos estudios para investigar los riesgos de la exposición al crisotilo en otros tipos de cáncer, en particular para aquellos específicos de la mujer.

¿De qué información disponemos sobre productos sustitutos, especialmente para materiales de construcción, teniendo presentes las afirmaciones que dicen que los productos sustitutos modernos del crisotilo también son tóxicos o tienen una toxicidad no determinada?

Muchos gobiernos nacionales, órganos regionales y organizaciones internacionales han identificado alternativas y productos sustitutos para prescindir del asbesto, y además se han publicado evaluaciones sobre los efectos de los materiales de sustitución en la salud humana. Por ejemplo, en 2005 se celebró un taller OMS/CIIC, y existen publicaciones al respecto del Gobierno del Reino Unido, la Comisión Europea y la Oficina Regional de la OMS para Europa. Los estudios sobre los peligros que entrañan los materiales de sustitución del crisotilo para la salud humana se han centrado en otros tipos de material fibroso, debido a los riesgos potenciales asociados a la inhalación de fibras. Sin embargo, también debe tenerse en cuenta que, en algunos de sus usos, el crisotilo puede ser reemplazado por material no fibroso, como por ejemplo cloruro de polivinilo no plastificado (uPVC) y chapas metálicas.

¿Dado que el mesotelioma es un marcador muy específico de la exposición al asbesto, la inexistencia de casos notificados de mesotelioma en un país indica que no hay una carga de enfermedad significativa por asbesto, y que por consiguiente no hay razón para adoptar medidas?

No. Para detectar los casos de mesotelioma y medir con precisión su número se requieren sistemas de vigilancia sistemática a nivel nacional, y con frecuencia tales sistemas no existen. También hay que tener presente que el periodo de latencia entre la exposición al asbesto y la aparición del mesotelioma puede ser hasta de 40 años o más, y por tanto esos sistemas deben funcionar durante mucho tiempo.

El asbesto tiende a causar más casos de cáncer de pulmón que de mesotelioma (se estima una razón de riesgo de 6:1), y esa probabilidad es aún mayor en los fumadores. El cáncer de pulmón es mucho más frecuente que el mesotelioma y tiene un origen multifactorial. Es fácil que una historia de exposición previa al asbesto (quizá en entornos no laborales, véase más abajo) pase desapercibida durante muchos años. La inexistencia de pruebas a nivel nacional no prueba la inexistencia de casos. Convendría tener en cuenta las enseñanzas de otros países en los que siguen apareciendo grandes epidemias de mesotelioma aún muchos años después de detener las exposiciones elevadas.

¿Es la exposición al asbesto un problema únicamente ocupacional, que plantea un riesgo mínimo o nulo para la población general?

No. Muchos de los casos de mesotelioma se han descrito en mujeres e hijos de trabajadores del asbesto, como consecuencia de exposición doméstica (al menos 376 casos), en oficinistas de la industria del asbesto, y en personas que viven en la vecindad de las minas de asbesto, como resultado de la contaminación del aire. También se han notificado casos de asbestosis en mujeres e hijos de trabajadores del asbesto. Se han descrito casos de mesotelioma en personas expuestas a asbesto de origen natural o a minerales parecidos al asbesto presentes en el suelo en regiones de Turquía, Grecia, Chipre, Córcega, Sicilia, Nueva Caledonia, la provincia china de Yunnan y California. Aunque este último grupo no se vería protegido por las medidas de control de la producción y el uso de asbesto, los otros grupos sí se beneficiarían.

La exposición ambiental puede tener también otros orígenes. Datos obtenidos en Australia y el Reino Unido han revelado concentraciones elevadas de fibras de asbesto en el aire ambiente en cruces de carreteras muy transitadas, provocadas por los materiales de fricción de los vehículos. En las actividades de reparación de viviendas y mantenimiento de automóviles se producen exposiciones no laborales al asbesto. Los trabajadores de la construcción tienen un riesgo de exposición no ocupacional adicional a la exposición ocupacional, debido a los residuos de construcción que contienen asbesto que no se almacenan y eliminan correctamente (las medidas de control de la exposición al asbesto son difíciles de aplicar para una mano de obra tan extensa y fragmentada, en la que pueden abundar trabajadores informales). Algunos de esos residuos son recuperados por recicladores y reutilizados en asentamientos informales.

Hoy la preocupación para los actuales formuladores de políticas es menor para la exposición ocupacional en los sectores de la minería y la fabricación de productos de asbesto, y es mayor para el uso de materiales de asbesto en la industria de la construcción. La preocupación se extiende a la exposición ocupacional durante las actividades de construcción y la exposición inadvertida de la población general por el deterioro de materiales de construcción (por ejemplo la ruptura de tejas onduladas de asbesto) y de la eliminación inadecuada de los escombros de construcción. También causa especial inquietud el uso de materiales de construcción con asbesto en las comunidades más pobres, que acercan a las familias a las fuentes de exposición a fibras de crisotilo.

Existe un riesgo de exposición no-ocupacional en residuos de construcción que contienen asbesto

Información adicional

Otras publicaciones de la OMS sobre el asbesto

Título	Descripción	Sitio web
Esquema para la elaboración de programas nacionales de eliminación de las enfermedades relacionadas con el asbesto. Organización Internacional del Trabajo y Organización Mundial de la Salud; 2007	El objetivo de este documento es ayudar a los países a establecer sus programas nacionales para la eliminación de las enfermedades relacionadas con el asbesto. También atiende los esfuerzos de los países para prevenir este tipo de enfermedades, debidas a la exposición a diversas formas de asbesto ya instalado o a su utilización en el pasado. Disponible en árabe, chino, español, francés, inglés y ruso.	http://whqlibdoc.who.int/hq/2007/WHO_SDE_PHE_07.02_spa.pdf?ua=1, consultado el 11 de marzo de 2014
Asbesto– peligros y prácticas seguras de limpieza depués de terremotos. Organización Mundial de la Salud; 2008	Esta nota técnica informativa brinda indicaciones sobre cómo controlar los riesgos asociados al asbesto durante la limpieza y eliminación de los escombros que lo contengan en edificios dañados y destruidos después de un terremoto u otro desastre natural.	http://www.who.int/hac/crises/chn/asbestos/en/, consultado el 11 de marzo de 2014

Evaluaciones publicadas sobre materiales sustitutivos

Título	Descripción	Sitio web
Análisis de sustitutos para los productos de construcción de asbesto por un consultor temporal de la OMS. En: Programas Nacionales para la eliminación de Enfermedades Relacionadas con el Asbesto: Análisis y evaluación. Oficina Regional de OMS para Europa; 2012: Anexo 4	Es un análisis sobre la disponibilidad y seguridad de los materiales sustitutos del asbesto, preparado un asesor temporal de la OMS como documento informativo de base para una reunión sobre el control del asbesto en la Región de Europa de la OMS. Disponible en inglés y ruso.	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, consultado el 11 de marzo de 2014
Opiniones sobre el asbesto Crisotilo y sus candidatos sustitutos. Comité científico sobre Toxicidad, Eco toxicidad y el Ambiente (CSTEE), Comisión Europea; 1998	Evaluación de los riesgos para la salud humana de tres fibras sustitutas: fibras de celulosa, fibras de PVA y fibras de P-aramida, a cargo de un comité de expertos de la Comisión Europea.	http://ec.europa.eu/health/scientific_committees/environmental_risks/opinions/sctee/sct_out17_en.htm, consultado el 11 de marzo de 2014
Harrison *et al.* Peligros comparativos del asbesto Crisotilo y sus substitutos: un perspectiva Europea. Environmental Health Perspectives, 1999;107:607-611	Evaluación de materiales de sustitución del asbesto preparada para la Comisión de Salud y Seguridad del Reino Unido (Londres, Reino Unido) y editada posteriormente como publicación científica.	http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1566482/, consultado el 11 de marzo de 2014

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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

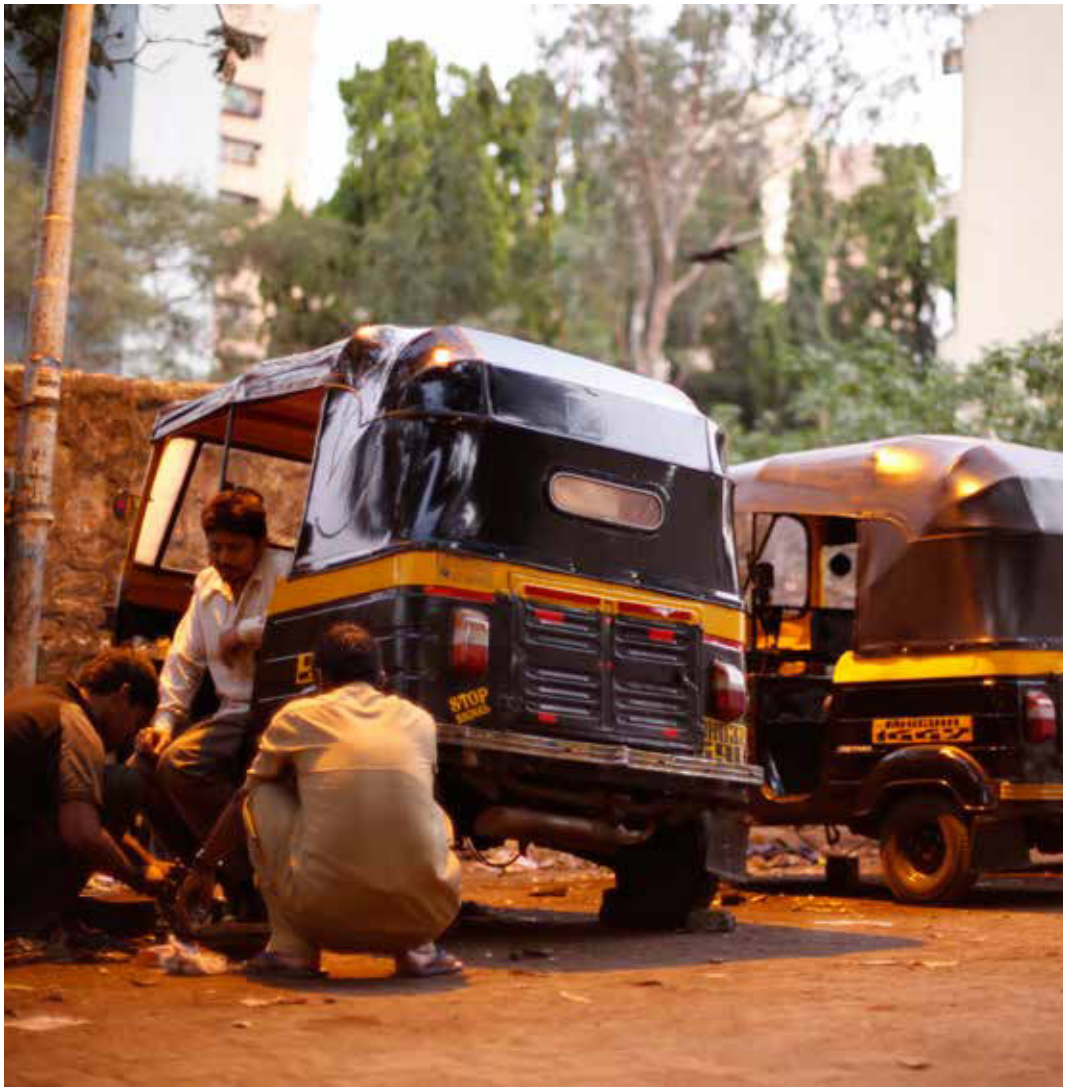
Non-occupational exposure, also loosely called environmental exposure, to asbestos may be due to domestic exposure (e.g. living in the same household with someone exposed to asbestos at work), air pollution from asbestos-related industries or the use of asbestos-containing friction materials, or naturally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

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

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



Elevated levels of chrysotile fibres have been detected at busy traffic intersections, presumably from braking vehicles

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

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)*.

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

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

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 $(\text{fibre-year/mL})^{-1}$ for Quebec mining and 0.018 $(\text{fibre-year/mL})^{-1}$ for the South Carolina textile workers. That for tremolite (vermiculite mines and milling operations in Libby, Montana, USA) was 0.0026 $(\text{fibre-year/mL})^{-1}$, with an upper uncertainty level of 0.03 $(\text{fibre-year/mL})^{-1}$, and that for amosite insulation, 0.024 $(\text{fibre-year/mL})^{-1}$ *(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)*.

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)*.

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

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).

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)*.

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

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 coworkers *(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.)

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.

Occupational exposure to chrysotile also causes non-malignant lung diseases

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.

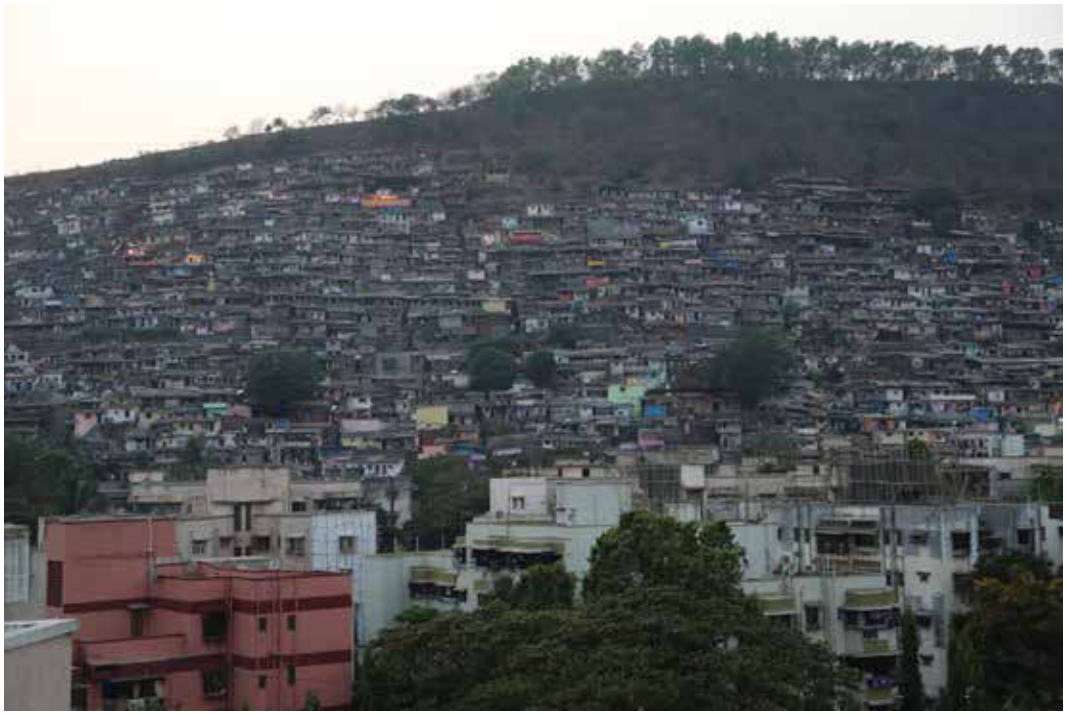
In the year 2004, asbestos caused 41 000 lung cancer deaths

Mesothelioma

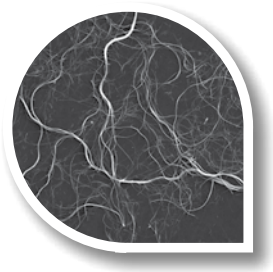
Driscoll and co-workers *(33)* estimated the global burden of mesothelioma deaths and DALYs based on the notion that mesothelioma is nearly always caused by exposure to asbestos, using the proportion of workers in different economic sectors (agriculture, mining, manufacturing, electrical, construction, 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 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

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

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.

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

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**.

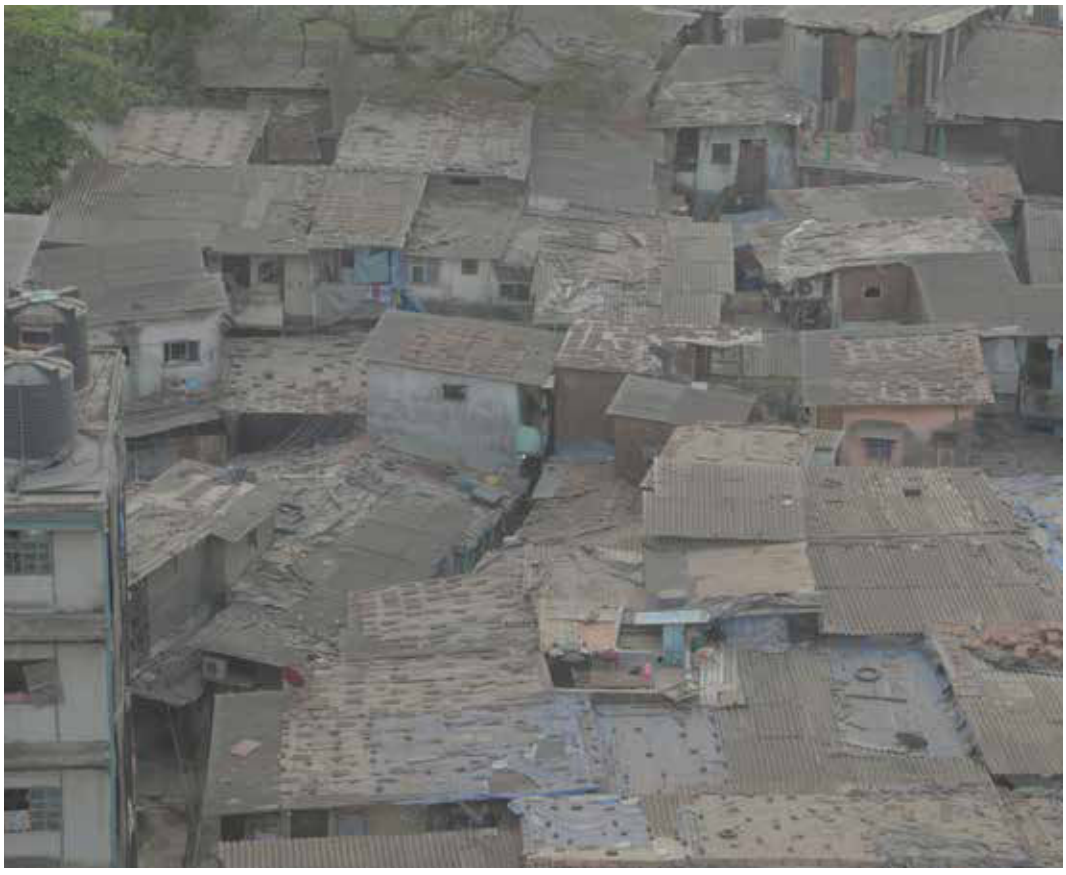
Magnesium sulfate whiskers did not induce tumours in limited inhalation and intratracheal administration studies, were negative in limited short-term tests and are very quickly eliminated from the lung. It was discussed whether the hazard grouping should be **low** or **indeterminate**. On the basis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

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 fibres must be respirable to pose an appreciable hazard

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 physico-chemical 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.

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SALUD PÚBLICA Y MEDIO AMBIENTE

El asbesto – un grupo de minerales conformado por el crisotilo, la crocidolita, la amosita, la antofilita, la tremolita y la actinolita – es uno de los carcinógenos ocupacionales más importantes. Cada año mueren al menos 107.000 personas por enfermedades relacionadas con el asbesto, incluyendo el cáncer de pulmón. Aunque el uso del asbesto ha disminuido en muchos países, el crisotilo se sigue utilizando ampliamente, sobre todo en los países en desarrollo.

Esta publicación sobre el crisotilo se divide en tres partes. La primera parte reproduce un documento informativo corto de la OMS dirigido a los tomadores de decisiones, que trata de la eliminación de las enfermedades relacionadas con el asbesto. La segunda parte aborda una serie de preguntas que frecuentemente se plantean en debates políticos, con el fin específico de prestar asistencia a los tomadores de decisiones. La tercera parte es un resumen técnico de los efectos del crisotilo en la salud, en el cual se recogen y resumen por primera vez las más recientes evaluaciones oficiales de la OMS, realizadas por su Centro Internacional de Investigaciones sobre el Cáncer y su Programa Internacional de Seguridad de las Sustancias Químicas. El resumen técnico también examina los resultados de los principales estudios publicados después de las susodichas evaluaciones, y las conclusiones derivadas de la evaluación de sustitutos del asbesto hecha por la OMS.

La presente publicación será de interés para todos los funcionarios públicos que necesiten tomar decisiones informadas sobre el manejo de los riesgos a la salud asociados con la exposición al crisotilo.

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