Amianto Crisótilo

World Health Organization

Encontra-se disponível informação adicional da OMS sobre substâncias químicas que constituem um problema para a saúde pública, entre as quais, o amianto, no seguinte endereço electrónico:

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

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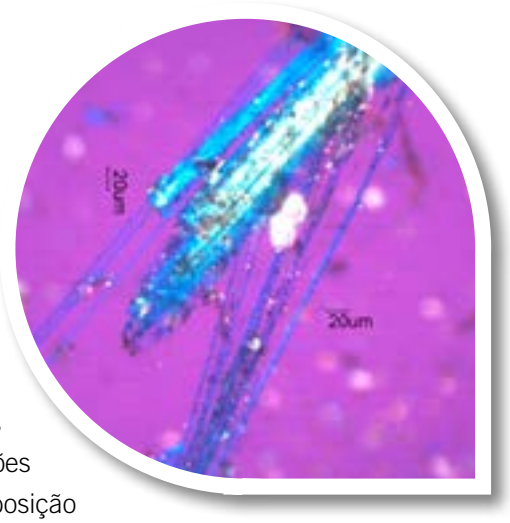
Prefácio



Muitos países já tomaram medidas a nível nacional no sentido da proibição do uso de todas as formas de amianto, de forma a limitar a exposição e assim controlar, prevenir e, em última análise, erradicar as doenças relacionadas com o amianto, as quais matam pelo menos 107 000 pessoas todos os anos, em todo o mundo. No entanto, outros existem que, por uma série de razões, terão de começar a actuar do mesmo modo. Nessa perspectiva, o primeiro propósito desta publicação é ajudar os Estados-Membros da Organização Mundial da Saúde (OMS) a tomar decisões informadas em matéria de gestão dos riscos para a saúde associados à exposição ao amianto crisótilo.

Este documento está dividido em três partes. A primeira parte reproduz um pequeno documento informativo da OMS, destinado a decisores, sobre a eliminação de doenças relacionadas com o amianto, actualizado em Março de 2014. A segunda parte aborda questões levantadas frequentemente quando se discutem políticas, exactamente para ajudar os decisores a adoptar uma posição. A terceira parte consiste numa síntese técnica dos efeitos do crisótilo sobre a saúde, a qual reúne e resume pela primeira vez as mais recentes avaliações oficiais da OMS levadas a cabo pelo Centro Internacional de Investigação do Cancro e pelo Programa Internacional de Segurança Química, ambos da OMS. A síntese técnica também analisa os resultados dos principais estudos publicados na sequência dessas avaliações e depois, de forma breve, as conclusões retiradas dos estudos de alternativas realizados pela OMS.

Recomendo esta publicação a ministros, funcionários governamentais e outros que desejem ou precisem de tomar decisões ou prestar aconselhamento em matéria de amianto, particularmente, as consequências para a saúde da exposição ao amianto crisótilo.

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Eliminação de Doenças Relacionadas com o Amianto

Actualizado em Março de 2014

O amianto é um dos mais relevantes agentes cancerígenos no local de trabalho, causando cerca de metade das mortes por cancro profissional *(1, 2)*. Em 2003, a Décima Terceira Sessão da Comissão Conjunta da Organização Internacional do Trabalho (OIT)/Organização Mundial da Saúde (OMS) sobre Saúde Ocupacional recomendou que fosse dedicada uma atenção especial à erradicação das doenças relacionadas com o amianto *(3)*. A resolução 58.22 de 2005 da Assembleia Mundial da Saúde (AMS) sobre a prevenção e controlo do cancro instava os Estados-Membros a observarem, sobretudo, os tipos de cancro para os quais a exposição evitável é um factor determinante, particularmente a exposição a agentes químicos no local de trabalho e no meio ambiente. Em 2007, a Resolução 60.26 da AMS apelava a campanhas a nível mundial para a eliminação das doenças relacionadas com o amianto e, em 2013, a Resolução 66.10 da AMS abordava a prevenção e controlo das doenças não transmissíveis, entre elas, o cancro.

O amianto é um dos mais relevantes agentes cancerígenos no local de trabalho

O termo "amianto" é a designação comercial do "asbesto", um grupo de minerais fibrosos, serpentínicos ou anfibólicos, de ocorrência natural, muito utilizado no passado e actualmente, devido à sua extraordinária resistência à ruptura, reduzida condução do calor e relativa resistência ao ataque químico. As principais variedades de amianto são o crisótilo, que é serpentínico, e a crocidolite, amosite, antofilite, tremolite e actinolite, que são anfibólicos *(4)*.

A exposição ao amianto, incluindo o crisótilo, causa cancro do pulmão, laringe e ovários, mesotelioma (cancro das membranas pleurais e peritoneais) e asbestose (fibrose pulmonar) *(5–7)*.

A exposição ao amianto e respectivo impacto na saúde pública são substanciais

A exposição ao amianto ocorre através da inalação de fibras, principalmente no ar contaminado do ambiente de trabalho, mas também do ar ambiente na vizinhança de fontes pontuais ou ar interior de habitações ou edifícios que contêm materiais com amianto friáveis. Os níveis mais elevados de exposição ocorrem durante a reembalagem de contentores de amianto, a mistura com outras matérias-primas e o corte a seco de produtos contendo amianto com ferramentas abrasivas. A exposição pode também ocorrer durante a instalação e utilização de produtos contendo amianto na manutenção de veículos. Os materiais que contêm crisótilo e/ou anfibólio friáveis estão ainda presentes em muitos edifícios e continuam a dar origem à exposição ao crisótilo e aos anfibólios, quando ocorre manutenção, reforma, eliminação ou demolição *(5)*. A exposição pode também dar-se quando os edifícios são danificados em resultado de catástrofes naturais.

Actualmente, cerca de 125 milhões de pessoas no mundo estão expostas ao amianto no local de trabalho *(1)*. De acordo com estimativas mundiais, pelo menos 107 000 pessoas morrem

todos os anos de cancro do pulmão, mesotelioma e asbestose resultantes da exposição ao amianto nas suas profissões *(1, 2, 8)*. Para além disso, quase 400 mortes foram atribuídas à exposição não profissional ao amianto. O número de doenças relacionadas com o amianto continua a subir, mesmo nos países que proibiram a sua utilização no início dos anos 90. Devido aos longos períodos de latência associados às doenças em questão, cessar a utilização do amianto agora só daqui a muitas décadas resultará num decréscimo do número de mortes por doenças com ele relacionadas.

Todos os tipos de amianto causam cancro no ser humano

O amianto (actinolite, amosite, antofilite, crisótilo, crocidolite e tremolite) foi classificado como agente cancerígeno para o ser humano pelo Centro Internacional de Investigação do Cancro *(7)*. A exposição ao crisótilo, amosite e antofilite e às misturas que contenham crocidolite resulta em risco acrescido de contrair cancro do pulmão *(7)*. Foram observados mesoteliomas após a exposição à crocidolite, amosite, tremolite e crisótilo no local de trabalho, bem como entre a população residente na proximidade de fábricas e minas de amianto e em pessoas que vivem com trabalhadores do amianto *(7)*.

A incidência das doenças relacionadas com o amianto está relacionada com o tipo, tamanho e dose de fibra e com a transformação industrial do amianto *(6)*. Não foi identificado qualquer limiar relativamente ao risco carcinogénico do amianto, incluindo o crisótilo *(5, 7)*. O consumo de cigarros aumenta o risco de contrair cancro do pulmão por exposição ao amianto *(5, 9)*.

Pelo menos, 107 000 pessoas morrem todos os anos de cancro do pulmão, mesotelioma e asbestose, resultantes da exposição ao amianto na actividade profissional

O crisótilo continua a ser muito utilizado

O amianto tem sido utilizado em milhares de produtos para um vasto número de aplicações, a saber, telhas, condutas de água, mantas ignífugas e materiais isolantes, bem como revestimentos, juntas e pastilhas de travão e embraiagens para automóveis. A utilização de amianto decresceu em muitos países, em resultado das crescentes preocupações com a saúde. O uso da crocidolite e produtos que contenham esta fibra e a pulverização de quaisquer formas de amianto são proibidos nos termos da Convenção da OIT sobre a segurança na utilização do amianto (nº 162) de 1986. Apesar disso, o crisótilo é ainda vastamente utilizado, aproximadamente 90% em materiais de construção em fibrocimento, sendo os países em desenvolvimento os seus maiores utilizadores. Acrescem ainda outras utilizações do crisótilo em materiais de fricção (7%), têxteis e outras aplicações *(10)*.

Até ao momento (final de 2013), mais de 50 países, incluindo todos os Estados-Membros da União Europeia, proibiram o uso de todas as formas de amianto, incluindo o crisótilo. Outros países estabeleceram restrições menos rigorosas. Apesar disso, alguns mantiveram ou inclusivamente aumentaram a sua produção e utilização do crisótilo nos últimos anos *(11)*. O acréscimo de utilização mais proeminente registou-se na região Ásia/Pacífico. A produção mundial de amianto no período de 2000–2012 manteve-se relativamente estável, em aproximadamente 2 milhões de toneladas por ano *(12, 13)*.

Recomendações da OMS sobre a prevenção das doenças relacionadas com o amianto

Tendo em conta que não há quaisquer provas para estabelecer um limiar relativo ao efeito carcinogénico do amianto, incluindo o crisótilo, e que foram observados riscos acrescidos de contrair cancro em populações expostas a níveis muito baixos *(5, 7)*, a via mais eficaz para a erradicação das doenças relacionadas com o amianto é deter a utilização de qualquer das suas formas. O uso continuado do fibrocimento na indústria da construção é particularmente preocupante, dado que envolve grande número de trabalhadores, a exposição é difícil de controlar e os materiais instalados são passíveis de se degradarem e constituírem um risco para quem proceda a reformas, manutenção ou demolição *(5)*. Nas suas diversas aplicações, o amianto pode ser substituído por determinados materiais em fibra *(14)* e por outros produtos com riscos menores ou nulos para a saúde.

Os materiais que contêm amianto devem ser encapsulados e, de uma forma geral, não é recomendável proceder a trabalhos que possam perturbar as suas fibras. Caso seja necessário, trabalhos tais como o encapsulamento, processos húmidos, ventilação por exaustão local com filtragem e limpeza regular devem ser efectuados apenas sob medidas de controlo rigorosas, a fim de evitar a exposição ao amianto. Tal requer ainda a utilização de equipamento de protecção pessoal – respiradores especiais, óculos de protecção, luvas e vestuário de protecção – e a disponibilização de instalações especiais para a sua descontaminação *(15)*.

A OMS está empenhada em trabalhar com os países para a erradicação das doenças relacionadas com o amianto cumprindo as orientações estratégicas que se seguem:

- reconhecendo que a forma mais eficaz de erradicar as doenças relacionadas com o amianto é acabar com a utilização de todos os tipos de amianto;
- fornecendo indicações sobre soluções de substituição do amianto por alternativas mais seguras e desenvolvendo mecanismos económicos e tecnológicos que estimulem a sua substituição;
- tomando medidas para evitar a exposição ao amianto instalado ou durante a sua remoção (redução);
- melhorando os serviços de diagnóstico precoce, tratamento e reabilitação das doenças relacionadas com o amianto e implementando registos das pessoas que a ele estão ou estiveram expostas.

A OMS recomenda veementemente o planeamento e implementação destas medidas, como parte de uma abordagem exaustiva para a erradicação das doenças relacionadas com o amianto. Tal abordagem deverá também incluir o desenvolvimento de perfis nacionais, acções de sensibilização, reforço das capacidades, um enquadramento institucional e um plano de acção nacional para a erradicação das doenças relacionadas com o amianto.

A OMS irá colaborar com a OIT na implementação da Resolução relativa ao amianto, adoptada pela Nonagésima Quinta Sessão da Organização Internacional do Trabalho *(16)*, e irá trabalhar com outras organizações intergovernamentais e a sociedade civil para a erradicação das doenças relacionadas com o amianto em todo o mundo.

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Crisótilo em estado bruto

Questões Frequentes e Respectivas Respostas

Esta secção aborda questões levantadas frequentemente por decisores políticos sobre a utilização do crisótilo.

É verdade que o crisótilo não é propriamente uma forma de amianto?

Não. O crisótilo é uma das seis formas de amianto, sendo as outras a crocidolite, amosite, tremolite, actinolite e antofilite.

Qual a política da OMS no que respeita ao amianto?

A política da OMS relativamente ao amianto é inequívoca. O amianto provoca cancro do pulmão, laringe e ovários, mesotelioma (cancro das membranas pleurais e peritoneais) e asbestose (fibrose pulmonar). As doenças relacionadas com o amianto podem e devem ser prevenidas e a forma mais eficaz de o fazer é banir a utilização de toda e qualquer forma de amianto a fim de evitar a exposição. As campanhas da OMS a nível mundial para erradicar as doenças relacionadas com o amianto visam apoiar os países na consecução desse objectivo.

Porque é que a OMS está tão preocupada com o amianto?

Existem provas científicas inequívocas de que o amianto provoca cancro e doenças respiratórias crónicas no ser humano. A OMS está a trabalhar para a redução do fardo mundial das doenças não transmissíveis, incluindo o cancro e as doenças respiratórias crónicas, reconhecendo que a prevenção primária reduz os custos dos serviços de cuidados de saúde e ajuda a assegurar a sustentabilidade das despesas da saúde. O cancro é a segunda maior causa de morte a nível mundial. Em 2008, registaram-se 7,6 milhões de mortes por cancro, a par de 12,7 milhões de novos casos. Estima-se que, aproximadamente, 19% de todos os cancros são imputáveis ao ambiente, nomeadamente, o do local de trabalho.

Presentemente, cerca de 125 milhões de pessoas no mundo encontram-se expostas ao amianto no local de trabalho. Segundo as estimativas da OMS, pelo menos 107 000 pessoas morrem todos os anos de cancro do pulmão, mesotelioma e asbestose relacionados com o amianto, resultantes de exposição profissional. Estima-se que aproximadamente metade das mortes por cancro profissional sejam causadas pelo amianto.

Qual é a autoridade que a OMS consulta em matéria de crisótilo e outras formas de amianto e respectiva gestão?

A OMS é a autoridade responsável pela direcção e coordenação da saúde do sistema das Nações Unidas. É sua responsabilidade assegurar a liderança em questões de saúde mundial, definir a agenda da investigação em matéria de saúde, estabelecer normas e padrões, articular opções políticas baseadas em evidências, fornecer apoio técnico aos países e monitorizar e avaliar as tendências da saúde.

A Assembleia Mundial da Saúde (AMS) é o órgão de decisão supremo da OMS; reúne anualmente e é constituído por delegações de 194 Estados-Membros. A principal função da AMS é definir a política da OMS.

A política da OMS em matéria de amianto provém de três resoluções da AMS: a WHA 58.22, de 2005, a WHA 60.26, de 2007 e a WHA 66.10, de 2013. A WHA 58.22 aborda os tipos de cancro para os quais a exposição evitável a agentes cancerígenos é determinante na sua causa; a WHA 60.26 apela para a realização de campanhas a nível mundial para a erradicação das doenças relacionadas com o amianto; e a WHA 66.10 aborda a prevenção e controlo das doenças não transmissíveis, incluindo o cancro.

De que forma estão as pessoas expostas ao amianto?

A exposição ao amianto ocorre por via da inalação e, em menor escala, da ingestão durante a extracção e trituração do amianto, bem como durante a produção e utilização de produtos que o contêm. Tal inclui a exposição que ocorre quando os materiais com amianto são cortados e instalados durante a construção, manutenção ou demolição de edifícios. O amianto é, geralmente, ou tem sido utilizado na forma de uma mistura fibrosa, misturada com outros materiais (por exemplo, cimento, plásticos e resinas) ou tecido numa matéria têxtil. O leque de aplicações em que o amianto tem sido utilizado é vasto e inclui coberturas de telhado, placas de cimento para pavimentos, condutas em cimento (por exemplo, para abastecimento de água), isolamento térmico e eléctrico, nomeadamente, mantas ignífugas e cortinas corta-fogo industriais, juntas e materiais de fricção (por exemplo, calços e pastilhas de travão e embraiagens para automóveis). Hoje em dia, a exposição às fibras de amianto ocorre sobretudo em circunstâncias em que os produtos de amianto se degradaram, tal como acontece no decurso da manutenção e demolição de edifícios e na eliminação dos resíduos de obras, bem como no contexto de catástrofes naturais.

Existem evidências científicas claras de que o amianto causa cancro e doenças respiratórias crónicas no ser humano

Por que é tão importante combater o amianto enquanto carcinogéneo quando tantos outros podem ser encontrados no meio ambiente?

Algumas formas de cancro imputáveis a factores ambientais crê-se terem múltiplos determinantes cancerígenos. Outras, no entanto, têm como causa um único agente cancerígeno identificável, caso do tabaco e do amianto, podendo a exposição ser evitada. (Nota: isto não se aplica a muitos dos outros agentes pertencentes ao Grupo 1, segundo a classificação do Centro Internacional de Investigação do Cancro [CIIC], que são cancerígenos para o ser humano, e muitos deles não têm o mesmo peso na doença[1]).

Uma das razões pelas quais é importante que os países tomem medidas sobre o amianto o mais rapidamente possível deve-se ao período excepcionalmente longo de latência entre a exposição e o desenvolvimento do mesotelioma, que atinge muitas vezes os 40 anos. Daí que o número das doenças relacionadas com o amianto vá continuar a aumentar, para já, mesmo nos países que proibiram a utilização do amianto há muitos anos.

Todas as formas de amianto causam cancro no ser humano (incluindo o crisótilo, a principal forma de amianto, que continua a ser produzido e utilizado), não tendo sido identificado qualquer limiar para os riscos cancerígenos. Esta é a conclusão da OMS e do CIIC numa série

[1] Para informação pormenorizada acerca dos carcinogéneos da categoria I do CIIC, consultar http://monographs.iarc.fr/ENG/Classification/ClassificationsGroupOrder.pdf.

de avaliações internacionais de especialistas realizadas durante um período de mais de 15 anos, tendo as mais recentes sido publicadas pelo CIIC em 2012. Estas conclusões reflectem o consenso científico internacional de peritos reunidos pela OMS para avaliar os efeitos do amianto sobre a saúde.

Além disso, ficou demonstrado que a co-exposição ao fumo do tabaco e às fibras do amianto aumenta substancialmente o risco de contrair cancro do pulmão, e o efeito é, no mínimo, cumulativo – isto é, quanto maior o consumo de tabaco, maior o risco.

Podemos ter a certeza de que as avaliações científicas do amianto realizadas pela OMS e pelo CIIC são totalmente isentas de influência externa?

Sim. Em qualquer caso, foram tomadas medidas para assegurar que potenciais conflitos de interesses foram identificados e resolvidos, que as avaliações foram extremamente rigorosas e independentes das posições dos governos, instituições nacionais e grupos de interesse especiais, e que levaram em linha de conta as opiniões oriundas de todas as regiões do mundo e foram sujeitas a uma rigorosa avaliação pelos pares a nível internacional.

Que acções têm sido desenvolvidas pelos países a nível nacional?

Muitos países já legislaram para proibir a utilização de amianto, o que foi feito, até à data (final de 2013), por mais de 50 Estados-Membros da OMS, com vista à protecção e promoção da saúde pública[2]. Habitualmente, a decisão era assumida após consulta intragovernamental, para levar em conta os interesses sectoriais, mas evitando que tivessem uma predominância excessiva na decisão final. Ao considerar tomar medidas legislativas contra a utilização de amianto, foi necessário ter em conta uma série de custos e benefícios, nomeadamente, os custos da prestação de cuidados de saúde e os associados com a perda de produtividade, por problemas de saúde crónicos da força de trabalho, para além de considerações de carácter económico e comercial convencionais.

Que acções têm sido desenvolvidas ou estão a ser propostas pelos países a nível internacional?

A Convenção de Basileia sobre o controlo de Movimentos Transfronteiriços de Resíduos Perigosos e sua Eliminação, que entrou em vigor em 1992 e da qual 181 países são Partes, destina-se a proteger a saúde humana e do ambiente contra os efeitos adversos dos resíduos perigosos. O amianto (pó e fibras) consta da lista de resíduos controlados, no âmbito da Convenção. As Partes da Convenção devem proibir ou não autorizar a exportação destes resíduos para as Partes que proibiram a sua importação ao abrigo da Convenção.

[2] Estão incluídos a Argélia, Argentina, Austrália, Barém, Brunei Darussalam, Chile, Egipto, os 28 Estados-Membros da União Europeia, Gabão, Honduras, Islândia, Israel, Japão, Jordânia, Kuwait, Moçambique, Noruega, Omã, Catar, República da Coreia, Arábia Saudita, Sérvia, Seicheles, África do Sul, Suíça, Turquia e Uruguai. O amianto está também interdito em dois estados do Brasil: Rio de Janeiro e Rio Grande do Sul.

Mais recentemente, uma maioria dos 154 países que são Partes da Convenção de Roterdão relativa ao Procedimento de Prévio Consentimento Informado para Determinados Produtos e Pesticidas Químicos Perigosos no Comércio Internacional (que entrou em vigor em 2004) declararam que o crisótilo deveria constar da lista do Anexo 3 da Convenção. Tal significaria que o crisótilo ficaria sujeito a um procedimento segundo o qual seria necessária uma decisão fundamentada por parte de um país antes de consentir, ou não, uma futura importação da substância. No entanto, até à data, a inclusão do crisótilo na referida lista tem sido suspensa por um pequeno número de países, predominantemente, mas não exclusivamente, por aqueles que continuam a ter interesse no comércio e utilização do crisótilo e produtos que o contêm.

É verdade que o crisótilo é menos nocivo do que outros tipos de amianto e, por isso, não deveria estar sujeito às mesmas medidas de controlo?

As evidências científicas são claras. A conclusão definitiva das avaliações da OMS e do CIIC é que o crisótilo provoca cancro do pulmão, laringe e ovários, mesotelioma e asbestose, seja, ou não, o seu efeito menos potente do que os tipos anfibólicos de amianto. As alegações acerca de propriedades físico-químicas divergentes, a questão de os estudos epidemiológicos feitos no passado se terem baseado, ou não, em crisótilo contaminado com tipos anfibólicos de amianto e o confinamento físico do crisótilo (no momento de fabrico) no actual cimento de alta densidade não alteram estas conclusões.

A conclusão inequívoca das avaliações da OMS e do CIIC é que o crisótilo causa cancro do pulmão, laringe e ovários, mesotelioma e asbestose

Uma grande preocupação é que, mesmo quando a sua utilização é devidamente regulamentada, os materiais de construção que contêm crisótilo (por exemplo, coberturas de telhado, condutas de água) se danificam, libertando fibras de amianto no ambiente, no decurso da manutenção ou demolição de edifícios, na remoção de resíduos de obras e como consequência de catástrofes naturais. Esta exposição pode ocorrer algum tempo após a instalação inicial (controlada). O risco pode ser inteiramente evitado, cessando a utilização destes produtos. As organizações nacionais, regionais e internacionais dispõem de informações acerca de materiais e produtos alternativos que podem ser utilizados com segurança.

A investigação da toxicidade do crisótilo, em curso ou a realizar, pode vir a mudar a actual posição da OMS e do CIIC relativamente à incidência de cancro?

De modo algum. A firme convicção da OMS e do CIIC, baseada em avaliações repetidas das evidências científicas, é que o crisótilo provoca cancro do pulmão, laringe e ovários, mesotelioma e asbestose, e que a forma mais eficaz de erradicar as doenças relacionadas com o amianto é parar com a utilização de todos os seus tipos, incluindo o crisótilo, para evitar a exposição. Embora o potencial cancerígeno do crisótilo tenha sido claramente identificado, poucos estudos incluíram mulheres. Existem ainda outros tipos de cancros que se suspeita estarem relacionados com o crisótilo, mas para os quais os estudos existentes são inadequados. Existe, portanto, uma necessidade permanente de novas pesquisas que investiguem os riscos da exposição ao crisótilo para outros tipos de cancro, particularmente os específicos da mulher.

Que informação está disponível sobre produtos alternativos, sobretudo materiais de construção, dado o argumento de que as actuais fibras substitutas do crisótilo são também elas tóxicas, ou de toxicidade por determinar?

Muitos governos nacionais, organismos regionais e organizações internacionais têm identificado alternativas e substitutos para as utilizações do amianto, assim como têm sido publicadas avaliações dos efeitos dos materiais de substituição sobre a saúde. Por exemplo, foi organizado um seminário pela OMS/CIIC em 2005 e tem havido publicações do governo do Reino Unido, da Comissão Europeia e do Escritório Regional da OMS para a Europa. As avaliações dos perigos para a saúde humana dos materiais substitutos do crisótilo concentraram-se em tipos alternativos de materiais fibrosos, devido aos potenciais riscos associados à inalação de fibras. Contudo, deve notar-se também que, para algumas das suas utilizações, o crisótilo pode ser substituído por materiais não fibrosos – por exemplo, policloreto de vinilo não plastificado (PVC-U) e chapa metálica.

A ausência de casos notificados de mesotelioma num dado país indica que não existe um fardo significativo da doença resultante do amianto e, logo, nenhuma razão para actuar, uma vez que o mesotelioma é um marcador tão específico da exposição ao amianto?

Não. A detecção de casos de mesotelioma e a medição rigorosa do seu número exigem sistemas de vigilância sistemática a nível nacional, mas eles são muitas vezes inexistentes. Há também que ter em mente que o período de latência entre a exposição ao amianto e o desenvolvimento do mesotelioma pode estender-se aos 40 anos ou mais e que, portanto, tais sistemas têm de ser duradouros.

É mais provável o amianto provocar cancro do pulmão do que mesotelioma (rácio de risco estimado 6:1) e a probabilidade é maior em indivíduos que fumam tabaco. O cancro do pulmão é muito mais comum do que o mesotelioma e de origem multifactorial. Pode acontecer que a história da exposição ao amianto (e isso pode incluir ambientes que não os do local de trabalho, ver abaixo) muitos anos antes seja facilmente negligenciada. A actual ausência de evidências a nível nacional não é prova de ausência, devendo ser tidos em conta os ensinamentos retirados das experiências de outros países em que ainda ocorrem grandes epidemias de mesotelioma, mesmo muitos anos depois de terem cessado as exposições generalizadas.

A exposição ao amianto é um problema apenas profissional, constituindo um risco nulo ou muito reduzido para a população em geral?

Não. Muitos casos de mesotelioma têm sido descritos em mulheres e filhos de trabalhadores do amianto, resultantes de exposição doméstica (pelo menos 376 casos), em trabalhadores não afectos à produção manual da indústria do amianto e em residentes nas imediações de minas de amianto, devido à poluição atmosférica; também tem sido notificada asbestose em mulheres e filhos de trabalhadores do amianto. Têm sido

descritos casos de mesotelioma em indivíduos expostos ao amianto ou minerais semelhantes de ocorrência natural no solo de regiões da Turquia, Grécia, Chipre, Córsega, Sicília, Nova Caledónia, província chinesa de Yunnan e Califórnia. Embora o grupo final não fosse protegido por medidas de controlo da produção e utilização do amianto, os outros grupos sê-lo-iam.

Ocorrem também outros tipos de exposição ambiental. Relatórios da Austrália e do Reino Unido identificaram elevadas concentrações de fibras de amianto no ar ambiente em cruzamentos de vias muito movimentadas, provocadas pelos produtos de atrito dos veículos. As exposições não ocupacionais decorrem de actividades de recuperação de edifícios e manutenção de veículos. Para além da exposição profissional dos trabalhadores da construção civil (uma vez que as medidas de controlo da exposição ao amianto são difíceis de implementar numa força de trabalho tão vasta e fragmentada que pode incluir muitos trabalhadores não declarados), por outro lado existe um potencial de exposição não profissional aos resíduos de construção que contêm amianto, se estes não forem devidamente armazenados e eliminados. Não é de descartar também a possibilidade de estes resíduos com amianto serem vasculhados e reaproveitados em construções informais.

A actual preocupação dos responsáveis políticos não é tanto em relação à exposição profissional nos sectores de extracção e fabrico dos produtos de amianto, mas sobretudo em relação à utilização de materiais que o contêm na indústria da construção. As preocupações estendem-se à exposição profissional durante as actividades de construção e à exposição involuntária da população em geral, através da degradação dos materiais de construção (por exemplo, telhas onduladas de amianto danificadas) e a eliminação inadequada dos resíduos de obras. É especialmente preocupante a utilização de materiais de construção que contêm amianto nas comunidades mais pobres, levando as famílias a uma grande proximidade com as fontes de exposição às fibras do crisótilo.

Existe risco de exposição não profissional ao amianto existente nos resíduos das construções

Informação Adicional

Outras publicações da OMS sobre o amianto

Título	Descrição	Website
Outline for the Development of National Programmes for Elimination of Asbestos-Related Diseases. International Labour Organization and World Health Organization; 2007	Este documento destina-se a facilitar aos países a elaboração dos respectivos programas nacionais para a erradicação das doenças relacionadas com o amianto. Aborda também as iniciativas dos países para a prevenção das doenças com ele relacionadas, resultantes da actual exposição às suas diversas formas, devido à sua utilização no passado. Disponível em inglês, francês, russo, espanhol, árabe e chinês.	http://www.who.int/occupational_health/publications/elimasbestos/en/, acesso em 11 de Março de 2014
Asbestos – hazards and safe practices for clean up after earthquake. World Health Organization; 2008	Esta nota técnica informativa fornece orientações em matéria de controlo dos riscos associados ao amianto, durante a limpeza e remoção de resíduos que o contenham de edifícios danificados ou destruídos após um terramoto ou outra catástrofe natural.	http://www.who.int/hac/crises/chn/asbestos/en/, acesso em 11 de Março de 2014

Avaliações de materiais alternativos publicadas

Título	Descrição	Website
Review of substitutes for asbestos construction products by a WHO temporary advisor. In: National Programmes for Elimination of Asbestos-related Diseases: Review and Assessment. WHO Regional Office for Europe; 2012: Annex 4	Relatório sobre a existência e segurança de materiais alternativos ao amianto, preparado por um consultor interino da OMS para servir de documento de referência numa reunião sobre o controlo do amianto na Região Europeia da OMS. Disponível em inglês e russo.	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, acesso em 11 de Março de 2014
Opinion on chrysotile asbestos and candidate substitutes. Scientific Committee on Toxicity, Ecotoxicity and the Environment (CSTEE), European Commission; 1998	Avaliação dos riscos para a saúde humana apresentados por três fibras alternativas – fibras de celulose, fibras de álcool polivinílico e fibras de para-aramida, realizada por uma comissão de peritos da Comissão Europeia.	http://ec.europa.eu/health/scientific_committees/environmental_risks/opinions/sctee/sct_out17_en.htm, acesso em 11 de Março de 2014
Harrison et al. Comparative Hazards of Chrysotile Asbestos and Its Substitutes: A European Perspective. Environ Health Perspect. 1999;107:607-611	Avaliação dos materiais alternativos ao amianto preparada para a Comissão de Saúde e Segurança do Reino Unido (Londres, Reino Unido) e posteriormente publicada na literatura científica.	http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1566482/, acesso em 11 de Março de 2014

Technical summary of WHO evaluations of chrysotile

Introduction

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

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

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

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

Chrysotile production, use and exposure

Production

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

Use

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

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

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

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

Non-occupational exposure

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

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

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)*.

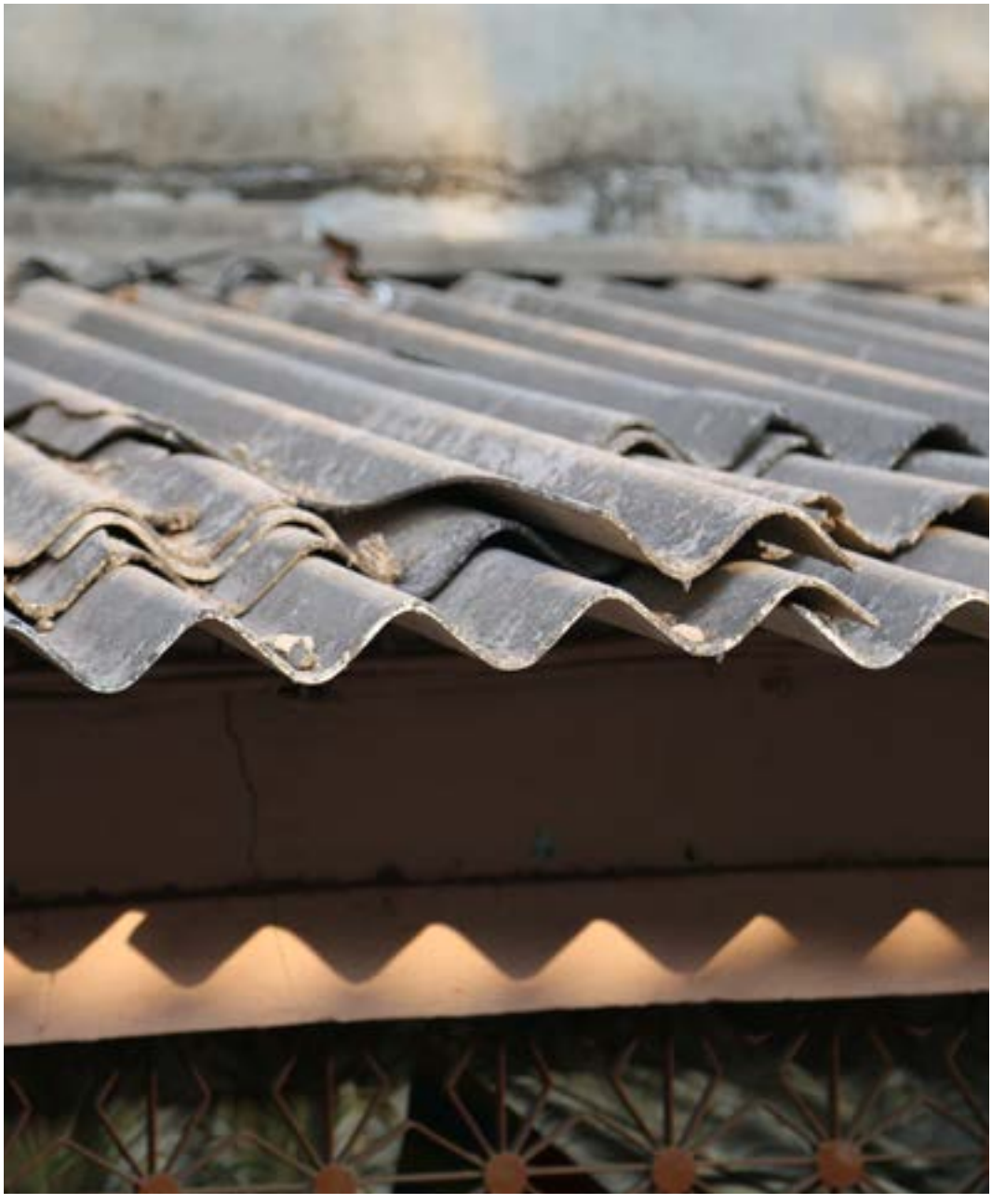
In 2006, the geometric mean asbestos fibre count in the air in the largest chrysotile mine in China was 29 fibres/mL, as estimated from gravimetric dust measurements. Available data indicated that up to 1995, dust concentrations had been 1.5–9 times higher *(11)*.

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

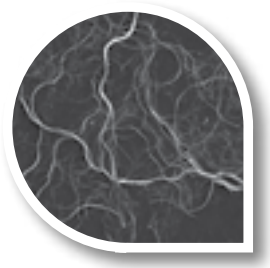
Mesothelioma

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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SAÚDE PÚBLICA E AMBIENTE

O amianto – grupo de minerais que inclui o crisótilo, a crocidolite, amosite, antofilite, tremolite e actinolite – é um dos agentes cancerígenos ocupacionais mais importantes. Pelo menos, 107 000 pessoas morrem todos os anos de doenças relacionadas com o amianto, nomeadamente, cancro do pulmão. Embora a utilização de amianto tenha decrescido em muitos países, o crisótilo continua a ser muito utilizado, particularmente em países em desenvolvimento.

Esta publicação sobre o amianto crisótilo está dividida em três partes. A primeira reproduz um pequeno documento informativo da OMS, destinado a decisores, sobre a erradicação de doenças relacionadas com o amianto. A segunda parte aborda questões levantadas frequentemente quando se discutem políticas, precisamente para auxiliar as instâncias de decisão. A terceira consiste numa síntese técnica dos efeitos do crisótilo sobre a saúde, a qual reúne e resume pela primeira vez as mais recentes avaliações oficiais da OMS levadas a cabo pelo Centro Internacional de Investigação do Cancro e pelo Programa Internacional de Segurança Química, ambos da OMS. A síntese técnica também analisa os resultados dos principais estudos publicados na sequência dessas avaliações e as conclusões retiradas dos estudos de alternativas realizados pela OMS.

Esta publicação será de interesse para todos os funcionários governamentais que precisem de tomar decisões informadas acerca da gestão dos riscos para a saúde associados à exposição ao amianto crisótilo.

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