INTERNATIONAL ENVIRONMENTAL RESEARCH FOUNDATION

Synopsis:
Health Effect of Chrysotile Containing Friction Products

By
Robert P Nolan, PhD
International Environmental Research Foundation
www.ierfinc.org
New York, NY

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Introduction

Although many countries have discontinued the use of friction products fabricated with chrysotile, these products remain in the global market place and are widely used. Questions have been raised if airborne asbestos released from these products during manufacture and/or use could be associated with an increased risk of developing any of the asbestos-related diseases.

A comprehensive review of this subject was published by Paustenbach *et al.*, 2004 which concluded that workers manufacturing friction products have a chrysotile exposure "perhaps 10 to 50 times greater than those of brake mechanics, but the risk of asbestosis, mesothelioma and lung cancer, if any, was not apparent, except in those who had some degree of exposure to amphibole asbestos during their careers".

The goal of this synopsis is to report any significant updates in the medical and scientific literature on the health effects of friction products and the use the exposure data reported by Paustenbach *et al.*, 2004 and others to estimate the expected magnitude of the increased risk of lung cancer and mesothelioma associated with the use of chrysotile containing friction products.

Three Principal Asbestos-Related Diseases: Historical Development

Our understanding the human health effects associated with exposure to airborne asbestos has developed slowly. Using this knowledge to implement public policies to effectively control these effects has met with limited success. The major policy challenges are that asbestos is often viewed as a single substance (although it is a group of six commercial minerals – five amphibole asbestos minerals - actinolite asbestos, anthophyllite asbestos, amosite, crocidolite, tremolite asbestos and one serpentine mineral - chrysotile) and exposure by inhalation to the different fiber-types is associated with a range of increased risks of asbestos-related diseases (Hodgson & Darnton 2000).

Observable asbestos-related health effects generally occur in historical occupational cohorts with poorly defined, but high cumulative exposures. Cumulative exposures are the concentration of airborne fiber (fibers/milliliter) multiplied by the duration of the exposure usually reported as f/mL x years.

Historically, workers exposed to high concentrations of airborne asbestos in mining, manufacturing and use of asbestos insulation materials developed asbestos-related diseases. The health effects literature regarding the commercial amphibole minerals – amosite, crocidolite - and chrysotile are the most highly developed and the main source of our knowledge of asbestos-related diseases and this synopsis will focus primarily on these three minerals.

The major producer of amosite was the Republic of South Africa (RSA), where production ended in 1992. RSA was also the major producer of crocidolite with some production at the Wittenoom Mine in Australia, production in these two countries ended in 1997 and 1966 respectively. Finland, the major producer of anthophyllite asbestos, ended production in 1977. Actinolite asbestos and tremolite asbestos were never commercially mined to any extent but these minerals have been found in other ore deposits and increased the risk of asbestos-related diseases. The only asbestos fiber-type that remains in commerce today is chrysotile.

An understanding of the risks associated with occupational exposure to asbestos developed slowly and the initial focus was on the generic asbestos and the development of asbestosis, asbestos-related lung cancer and mesothelioma.

The first case of asbestos-related disease entered the literature in 1906, was an unusual fibrosis of the lung that occurred in workers in the Barking factory in London (for a review see Murray, 1990). There were

about 10 fibrosis cases most of whom died around age 30. The workers also had tuberculosis, so the fibrosis could not be unambiguously related to their dust exposures. Asbestos exposed workers continued to develop fibrosis and in 1924 Cooke was the first to call the disease asbestosis (Cooke 1927). The following year the first case would be reported "…of pulmonary fibrosis, the result of the inhalation of asbestos dust" (Seiler, 1928). This disease would be called asbestosis.

In the United States, lung cancer associated with asbestos exposure was first reported in an asbestosis case (Lynch and Smith, 1935) and additional case reports of lung cancer and asbestosis would continue to appear for decades, mainly from the United Kingdom, Canada, Germany and the United States. In these early publications the fiber-type was not reported nor was the intensity or duration of the exposure. The focus of the early case reports was the presence of fibrosis and lung cancer. These case reports involved either a single case or a small number of cases.

Prior to World War II lung cancer was a relatively rare disease. In 1922 there were 612 lung cancer cases in Great Britain and by 1947 the number had increase to 9,287 (Doll and Hill 1950). The number of lung cancer cases in the United States and the United Kingdom increased markedly from after World War I until the late 1940s (Wynder & Graham 1950, Doll & Hill 1950).

Many investigators suspected cigarette smoking was an important etiological factor in the increase but strong evidence supporting this claim was lacking. Others thought air pollution from automobiles and paved roads were an important factor.

Wynder and Graham (1950) found bronchogenic cancer markedly higher in US male smokers than non-smokers. Among the males only 2% of the non-smokers, or minimal smokers (defined as less than 1 cigarette a day) developed lung cancer, while 51.2% of those who smoked 20 cigarettes per day over the same time period developed lung cancer. If you did not smoke you would rarely develop lung cancer.

Doll & Graham (1950) studied lung cancer among male and female - smokers and non-smokers. Of the 649 males with lung carcinoma only 2 cases occurred among the non-smokers. So 647 of the lung cancer occurred among the male smokers. Among the females in the study 31.7% of the lung carcinomas occurred among the non-smoking females. Smoking was much less common among the females compared to the males, so the proportion of non-smoking females with lung cancer was proportionally higher.

In the United Kingdom, Doll (1955) reported on a cohort where 113 male deaths had occurred. Each had at least 20 years of occupational exposure to chrysotile asbestos although the asbestos-type was not reported. Eleven lung cancers occurred in this cohort, where less than 1 case of lung cancer would have been expected, based on the rate of this disease among males in the general population. All of the lung cancer cases showed histological evidence of asbestosis. This report provided strong evidence that individuals with asbestosis were at an increased risk of developing lung cancer. No mention is made about smoking in general or the smoking habits of the 113 workers (Doll 1959).

Mesothelioma is a cancer of the pleura, pericardium, and peritoneal membranes, which surround the lung, heart, and abdominal cavities, respectively. The pleural mesothelial cells occur almost as a monolayer surrounding the outside of the lung and the inside of the chest wall. Mesothelioma occurs predominantly in those exposed to the amphibole asbestos dusts – amosite, crocidolite, tremolite asbestos (Wagner *et al*., 1960, Wagner 1991, Table 1). Mesothelioma of the pleura and peritoneum was reported in each of the 10 cohorts of workers exposed in mining and manufacturing of amphibole asbestos (crocidolite, amosite & tremolite) accounting for 5.3% of all deaths (Table 1).

Mesothelioma mortality among chrysotile mining and manufacturing workers is markedly lower than in the amphibole asbestos exposed cohorts with an inconsistent pattern, where no increase in observer in some of the chrysotile exposed to cohort (Table 1, Figure 1).

Of the 36 mesothelioma cases reported in the six chrysotile cohorts, 33 occurred in the chrysotile miners and millers after cumulative exposures of 600 fiber per milliliter x years (Table 1). All of the chrysotile-related mesothelioma cases occurred in the pleura while the amphibole asbestos cohorts always have peritoneal mesothelioma case.

Expectations of Asbestos-Related Diseases from Friction Products

The largest cohort study of friction product workers was done at the Ferodo factory in UK. The cohort included both males and females, the mortality of the males only will be summarized here. There were 8,404 males in the cohort with 2,055 deaths. There was no excess mortality from any cause in the cohort and no deaths from asbestosis (Newhouse & Sullivan 1989). There were 241 lung cancer where 242.5 were expected so the Standard Mortality Ratio was 0.99 (Hodgson & Darnton 2000). There were 13 pleural mesothelioma cases in total with 11 occurring in males. Newhouse & Sullivan (1989) reported, "…eleven were of subjects who had known contact with crocidolite asbestos, in one instance the diagnosis was uncertain and in the other the occupational history of the subject is not well established". The authors went on to conclude, "…under good environmental conditions chrysotile asbestos containing products can be manufactured with no detectable mortality".

The cumulative asbestos exposure in the Ferodo study was 35 f/mL x years is considerably higher than what would occur in modern chrysotile mining or manufacturing operation where airborne concentration of chrysotile are less than 0.0058f/mL, where a 40-year career would correspond to a cumulative exposure of 0.23f/mL x years (Nolan et al., 2015). More than 150-fold lower than the cumulative exposure reported in the Ferodo Study.

A PubMed search of the literature identified one new publication by Babieri *et al*., 2020 not included in the Paustenbach *et al*., 2004. The new paper reported no mesothelioma cases in the Italian friction product workers studied and the airborne fiber concentrations in 1982 were about 0.3f/mL while all the 1992 measurements were less than 0.1f/mL. Babieri *et al*., 2020 supports the data and conclusions of Paustenbach *et al*., 2004.

At the lower cumulative exposures associated with brake repair work asbestos-related disease is not expected to occur.

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Figure 1. The % of mesothelioma deaths is plotted against the cumulative asbestos exposure by asbestos-type.

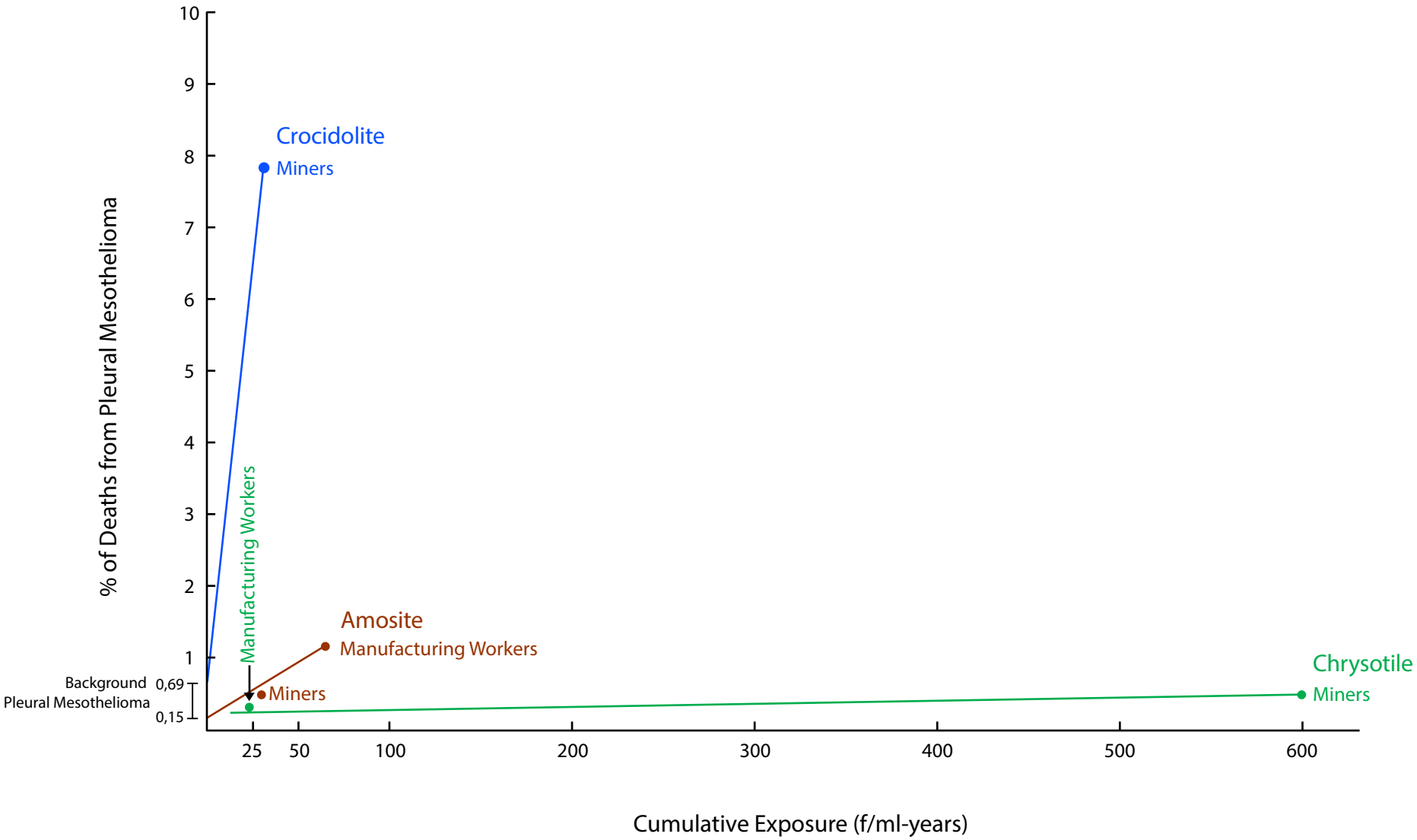

Table 1. Summary of the **cohort epidemiology studies by asbestos fiber-type.**			
Asbestos Fiber-Type	**Cohort Name**	**Total № of Mesothelioma Death (%)**	**Cumulative f/mL x Years§**
Crocidolite	***5 Occupational Cohorts***	***270/3,275(8.3%)***	***53***
Amosite	***4 Occupational Cohorts***	***32/1,943 (1.6%)***	**Unknown**
Tremolite‡	***1 Occupational Cohorts§***	***15/767(2.0%)***	**Unknown**
Chrysotile	***Miners & Millers***		
	Canadian Mines	33/7,456 (0.44%)	600
	Russian Mines	No Cases Reported	
	South African Mines	No Cases Reported	
	Manufacturers		
Chrysotile	**Charleston, South Carolina** *(Males only)*	3/1,186 (0.25%) §	28
Chrysotile	**New Orleans, LA**	0/259 (0%)	22
Chrysotile	**Connecticut**	0/557 (0%)	46
Chrysotile	***All Males Manufacturers***	***3/2,002 (0.15%)***	**32**
Chrysotile	***All Males Mining***	***33/7,456 (0.44%)***	**600**
Chrysotile	***TOTAL all studies***	***36/10,540 (0.34%)***	***170***
Crocidolite Amosite Tremolite	***TOTAL all studies***	***317/5,985(5.30%)***	***53***
‡Calcic amphiboles with increased sodium and potassium see Sullivan 2007.			
§ Hein et al., 2007 the other data are from Hodgson and Darnton, 2000.			