AN ASSESSMENT OF THE RISK OF HEALTH EFFECTS RESULTING FROM THE USE OF MOBIL PRODUCTS BEARING THE NAME "DUM DUM"

WITH AN UPDATE OF SCIENTIFIC KNOWLEDGE TO 1999.

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PREFACE

In June 1992, I issued a report entitled "An assessment of the risk of health effects resulting from the use of Mobil Products bearing the name "Dum Dum". The report was prepared at the request of Mr Thomas Campion, Shanley & Fisher, Morristown, New Jersey. The task undertaken was to prepare a document which evaluated the risk of asbestos-related disease for persons using Dum Dum products.

This current report contains information updating that original report to take account of the scientific literature since the original report was prepared.

Ms Julia Kingsley Evans and/or other legal counsel acting for Mobil provided no specific direction concerning the scientific aspects of the report.

The Preface in the original report was as follows:

> This report was prepared at the request of Mr Thomas Campion, Shanley & Fisher, Morristown, New Jersey. The task undertaken was to prepare a document which evaluated the risk of asbestos-related disease for persons using Dum Dum products.
>
> Mr Campion provided no specific direction other than the report should advise " whether, on the basis of the available scientific evidence, the encapsulated asbestos in the products caused injury in the application or removal process."

REPORT ORGANIZATION

The report guides the reader through a logical sequence of steps, critically evaluating the factors which determine the occurrence of health effects in workers exposed to "asbestos" fibres. These factors are then examined in making an assessment of the risks for persons using "Dum Dum" products.

For convenience, technical terms and abbreviations are provided as footnotes on the pages where they occur. When more detail is required to support a particular evaluative step, this is provided in an appendix to the report.

The various sections of the report are numbered according to subject matter. The studies to which references are made in the report are listed in alphabetical and date order in the BIBLIOGRAPHY.

In order to facilitate reading this updated report, the original report layout and content have been retained. Information updating the report has been added at the end of each section and additional sections added when necessary. For convenience, references to the updated information have been incorporated into the original BIBLIOGRAPHY.

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1.0 OBJECTIVES

The objectives of the original June 1992 report were:

a. to describe a systematic approach which might be applied in evaluating whether the "asbestos" used in certain "Dum Dum" products has, or might reasonably be expected, to have caused adverse health effects in persons who applied or removed the products.

b. to apply this approach in critically evaluating the available scientific evidence concerning the health risks for workers applying or removing products bearing the name "Dum Dum".

The purpose of this updated report remains as originally stated. However, as this report incorporates information which has appeared in the scientific literature since the original report was prepared it also examines whether the scientific literature published between the early 1990s and 1999 has changed the conclusions recorded in the 1992 report. Those conclusions were:

- It is highly improbable that the levels of asbestos exposure by persons using Dum Dum products continuously over a 20 year period would be adequate to give rise to detectable "radiological changes" compatible with asbestosis.

- It is highly improbable that the levels of asbestos exposure by persons using Dum Dum products continuously over a 20 year period would be adequate to produce detectable increases in lung cancer or primary malignant mesothelioma death rates.

- The risks of mesothelioma, lung cancer and lung fibrosis associated with the use of the Dum Dum Products are so low that other sources of exposure and other

factors deserve a much higher priority for consideration in determining the etiology of cancers or radiological changes.

2.0 "DUM DUM" TERMINOLOGY

This report addresses "DUM DUM" products manufactured and marketed by Mobil. While preparing this report, it was found that a product known as "Red Dum Dum Boiler Putty" was currently being manufactured by "The Presco Co. Ltd", Mississauga, Ontario. In a telephone conversation, Mr R.C. Smith of that company informed me that this product has been manufactured by them since at least 1935 and probably earlier. The most recent Material Safety Data Sheet (MSDS) for the "Presco" product (APPENDIX A) does not report the presence of asbestos, although an earlier MSDS for a "Dum Dum Hi-Temp Boiler Putty" (date unknown - APPENDIX B) did note asbestos as a constituent. "Red Dum Dum Boiler Putty" product was not, as far as I could ascertain, ever produced by Mobil. The products discussed in this report will be restricted to those produced and/or marketed by Mobil.

UPDATE:

There has been no change in the nomenclature applied to these products.

3.0 BACKGROUND

3.1 SCOPE

This report was prepared in response to the question posed by Mr Thomas F. Campion, Shanley & Fisher, Morristown, New Jersey. Referring to Dum Dum products he asked ".... whether the encapsulated[1] asbestos in Mobil's products caused an injury to those who applied it or removed it."

The report specifically targets the risks for persons working with the manufactured products as marketed by Mobil and does not include an assessment of health risks for workers involved in manufacturing the products or handling raw materials used in their manufacture. "Working with" has been defined as applying, removing and disposing of the products.

UPDATE:

There is no change in the broad scope of the report.

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[1] Encapsulated denotes the enclosure of the asbestos by substances which cover or form a seal over the individual or bundles of fibres forming a mass in which the fibres are now an integral part of the whole and no longer free asbestos fibres.

3.2 PERIOD OVER WHICH PRODUCTS WERE SOLD

In 1963, Mobil acquired the paints and coatings business of the Martin-Marietta Corporation and began to manufacture and sell coatings products which contained small amounts of encapsulated asbestos. Several of these products were discontinued at various dates between 1963 and 1980, with the asbestos containing materials being totally discontinued by the end of 1980. This report, while drawing on information gathered outside this time-frame, addresses only products produced and sold in the period 1963-1980.

UPDATE:

There has been no information forthcoming since the preparation of the original report to indicate any modification to the time period 1963 - 1980.

4.0 THE ASBESTOS MINERALS

Asbestos is not a single substance. In later sections of this report, it will be seen that there are clear differences in the risk of asbestos-related diseases associated with exposure to the different fibrous minerals referred to as "asbestos". That they should pose different levels of risk should not be surprising as each of the "fibre types" has its own distinct chemical, mineralogical and physical characteristics.

Asbestos is a generic term applied to the naturally occurring hydrated fibrous silicates. There are six main varieties (FIGURE 1). Four of them, chrysotile, amosite, crocidolite and anthophyllite have been of commercial importance. Differences in the general appearance of these minerals under the electron microscope are shown in FIGURE 2.

UPDATE:

While there have been advances in understanding the importance of potential contaminants in commercially produced chrysotile asbestos, there has been no new information that would change the very general descriptions of the asbestos minerals given in sections 4.0 - 4.24. While the medical literature has given more attention to specific fibre types, the generic term "asbestos" is still widely used and in most instances the medical literature is still very imprecise in respect to mineral terminology.

4.1 CHRYSOTILE (WHITE ASBESTOS)

Chrysotile is an hydrated magnesium silicate and a member of the serpentine group of minerals. It usually occurs as white flexible fibres and is sometimes referred to as "white asbestos".

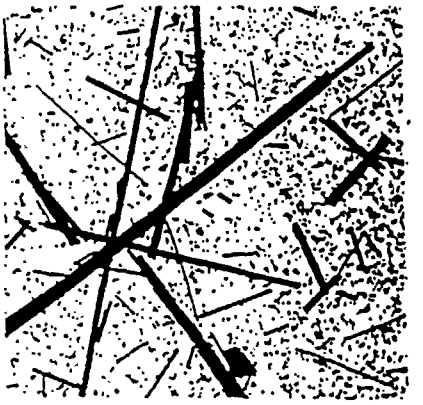
The main asbestos fibre type referred to in this report will be "CHRYSOTILE" as chrysotile was the

FIGURE 2

ELECTRON MICROGRAPHS OF CROCIDOLITE, AMOSITE, ANTHOPHYLLITE AND CHRYSOTILE AT THE SAME MAGNIFICATION (x 1700)[3,4]

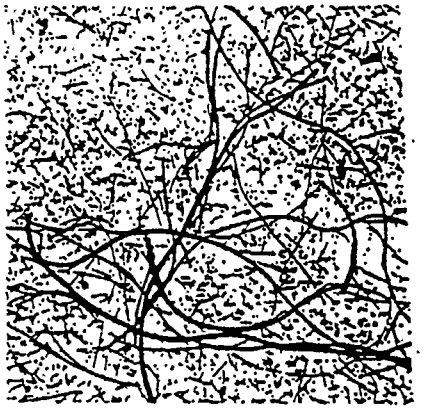
CROCIDOLITE

AMOSITE



ANTHOPHYLLITE

CHRYSOTILE



10 μm

[3] The sources of the fibres were: crocidolite: North-Western Cape Province, South Africa; amosite: Transvaal, South Africa; anthophyllite: Finland; chrysotile: Canada.

[4] Note the straight amphibole fibres (crocidolite, amosite and anthophyllite) in contrast to the curly chrysotile fibres; the relative order of magnitude of the diameters of the amphibole fibres..crocidolite<amosite<anthophyllite and the longitudinal fragmentation of the chrysotile fibres and the small diameter of the ultimate fibril (approx 0.03um) (Timbrell 1973). The dimensions of fibres are conventionally expressed in micrometres (μm). 1μm = 1 x 10^{-6}m.

In order to avoid confusion in the use of mineralogical terms, the term used throughout this text to identify crocidolite, the mineral mined in South Africa and Australia, will be "BLUE ASBESTOS"

UPDATE:

The production in South Africa has ceased.

4.2.2 Amosite (Brown Asbestos)

Amosite is a brown-grey or white fibre mined in South Africa.

UPDATE:

The production of amosite in South Africa has ceased.

4.2.3 Anthophyllite

Anthophyllite is a white fibre which was mined mainly in Finland and has had limited application commercially.

4.2.4 Tremolite and Actinolite

Tremolite and actinolite are two minerals in a series of minerals in which the iron/magnesium ratio determines the mineral name. Tremolite is not of direct commercial importance at this time However, tremolite has become quite important in understanding the health risks associated with chrysotile asbestos as tremolite would appear to occur in extremely small amounts in some chrysotile deposits.

When Quebec chrysotile miners and millers have inhaled mine and mill dusts, tremolite fibres appear to have been preferentially concentrated in their lungs. Some chrysotile miners in Quebec have been found to have as much tremolite as chrysotile in their lungs at autopsy (Rowlands et al 1982) Tremolite can be a contaminant of vermiculite and talc. Mesotheliomas have been reported in vermiculite miners (McDonald et al 1986a), and in talc miners (Vianna et al 1981). This has led to an hypothesis, increasingly supported by scientific research results, that the tremolite fibre contamination of chrysotile may explain the occasional occurrence of mesothelioma in workers in the Quebec chrysotile mines and mills.

Talc was used in some of the Dum Dum products. Some talcs have tremolite associated with them. It should be emphasized that not all talcs contain tremolite and not all tremolite is asbestiform. Whether or not talcs used by Mobil prior to the mid 1970's were contaminated by asbestiform tremolite is not known. However, after that time, Mobil stipulated that only tremolite-free talcs be purchased for use in the Dum Dum product line.

UPDATE:

In view of the importance of asbestiform tremolite, it should be noted that while fibrous tremolite is a potential contaminant in some chrysotile deposits, it is not a contaminant of all commercial chrysotile. The tremolite in the Quebec chrysotile mines is associated with mineralogical alterations in the rock usually close to igneous intrusive rocks (Williams-Jones 1999; Gibbs 1999). The fibrous tremolite was not in the ore body but in other rocks occurring in the mine. Tremolite fibres occur at very high concentrations in the lungs of some Quebec chrysotile miners [McDonald et al 1997).

5.0 THE DUM DUM PRODUCTS

UPDATE:

There have been no changes to the list of Dum Dum Products considered in this report or in the compositional data for these Dum Dum products.

5.1 GENERAL CHARACTERISTICS

A list of Dum Dum products is provided in Appendix C. For convenience, they have been classified according to their application, asbestos content and period over which they were manufactured.

The applications were.

* Filling nail holes
* General purpose caulking
* High temperature caulking
* Exterior masonry coatings
* Chimney coatings

The shared characteristics of "Dum Dum" products were that they were designed to.....

* harden only at the surface so that they could be painted.
* remain pliable underneath the surface during application and throughout the product lifetime to expand and contract with the building
* bridge cracks and flaws in the surfaces to which applied
* in most cases have a weatherproof exterior.
* reseal automatically if the surface were cracked because the exposed sub-surface material would reform a new coating.

The products were sold in a form ready for use and the components were thoroughly mixed in oils

so that the products were essentially of putty consistency making it unlikely that they would produce any dusts or respirable aerosols during use.

5.2 THE SPECIFIC PRODUCTS

Brief descriptions of the products taken from product literature, product labels and material safety data sheets provided by Mobil follow....

A. Dum Dum Nail Hole

A number of nail hole products were manufactured until 1973, the only differences between the various products were in the additives used to produce different colours (See APPENDIX C1). This material was of putty-like consistency, for knife application to fill nail holes in pre-finished wood.

B. Caulking Compounds

A number of products were manufactured and marketed as caulking materials (See APPENDIX C2).

The Mobil Chemical Product Data sheet for "DUM DUM Calking, 46-F-5" shows that this product was a mixture of pigment and asbestos fibre in a vegetable oil vehicle (Mobil 1976a). At 24°C its viscosity was described as putty and would build one inch (1") on the vertical without slumping. The label for this particular product was clearly marked "For industrial use only. Do not use around a household or dwelling" (Mobil 1976a; Mobil 1970a).

The product label indicated that "Dum Dum Calking" was designed for all conventional exterior caulking work, with flexibility to withstand building stresses, settling and shrinkage. It was described as an oil based caulking compound for glazing and filling operations by knife or gun for joints or cracks (Mobil 1976b). The product was clearly under oil as instructions to stir it, if oil had risen to the top, were provided. The surface was reported to "skin" in 18 hours and could be painted in 24 hours.

C. High temperature Caulking compounds

The high temperature caulking materials are described in APPENDIX C3. There appear to have been two such products.

"Hi-Heat Dum Dum, 46-F-7" was marketed from 1964 through October 1980. A Material Safety Data Sheet dated September 26 1980 states that this product was at that time "asbestos-free" (Mobil 1980).

Hi- Heat Dum Dum is described in the Mobil product literature as a heavy semi-plastic fibred coating which acts as a joint sealer and pliable gasket for boilers, furnaces and dry kilns. It retains its elasticity

at constant heat up to 175°C and at intermittent heat to 350°C (Mobil 19XXa)[5].

Its viscosity at 24°C was described as mastic (Mobil 1976c). The product after application could be painted over after 24 hours (Mobil 1970b). This product contained chrysotile asbestos and talc in a linseed oil blend (Chamberlain 1991).

"Dum Dum Armorcote, 46-J-9" was a specially compounded heavy bodied product containing asphalt, reinforcing pigment and high boiling point hydrocarbons. It was resistant to fumes, moisture and sustained temperatures to 300°F. It was used to protect and increase the efficiency of brick boiler settings, as a dry kiln liner and in similar refractory applications (Mobil 19XXb; Mobil 1973a). Its viscosity at 24°C was described as a putty (Mobil 1976d).

D. Masonry Coatings

The "95 series DUM DUM Masonoc" was marketed from 1964 to 1979 and contained not less than 10 and not more than 15% chrysotile asbestos. Its viscosity was mastic and while forming a "skin" in 1 hour the under surface remained pliable (Mobil 1976e). The product was described as a heavy bodied coating of mastic consistency designed for "hi build" application for filling and bridging cracks and crevices, with application by special brush or heavy duty mastic spray equipment (Mobil 19XXc). The product was noted to be used in applications for the waterproofing and restoration of exterior masonry and for the protection of steel surfaces and for industrial use only (Mobil 1971a; Mobil 1976e).

E. Chimney Coatings

The "97 Series Chimney Dum Dum" was marketed in the period 1964-1979. The product was a mixture containing chrysotile asbestos fibre (4-5%) and color fast pigments in a resinated vegetable oil (Mobil 1976f; Mobil 1970c). According to the product literature, "Chimney Dum Dum" provides "the same outstanding protective and waterproofing qualities as the 95 Series Masonoc except that it has been formulated for use on concrete chimneys where additional heat resistance up to 210°F may be required" (Mobil 1976g). The viscosity is described as mastic. Although the surface "skins" in 4-6 hours, the product was designed so that the under-surface remained pliable (Mobil 1976f).

F. Primers

Literature relating to the use of "Dum Dum Masonoc" recommended that the surface be prepared with primers. Examples of primers for use with the "Dum Dum Masonoc 95 series" were "Masonoc Primer, 47-W-21" and "Masonoc Primer,47-B-1". There were others which differed in colour but with essentially the same constituents except for the pigment. The 47-W-1 product was described

[5]The date 19XX has been used in references when the product literature exists but is undated.

as "a normal paint like material to be used beneath the Masonoc coating on all types of masonry surfaces to seal off and eliminate surface porosity" (Mobil 1976h).

A primer recommended for use with the Chimney Dum Dum was "Chimney Dum Dum Primer 38-V-5".

Commercial asbestos was not a component in the formulation of these primers (Chamberlain 1991). Hence, these primers will not be considered further.

6.0 OCCUPATIONALLY RELATED DISEASES

Occupational diseases result from exposures at work and are essentially of three types. The first is a disease which occurs very rarely in the general population. When a few cases occur in a working population in relation to a specific occupational exposure this is sufficient to raise a suspicion that the disease is occupational in origin. However, there are often factors other than occupation which are common to the cases and working population. Thus the observation of a clustering of cases of a rare disease should signal the need to seek confirmatory data. This usually involves an independent epidemiological study. An example is primary malignant mesothelioma.

The second type of occupational disease occurs quite commonly in the general population. In this situation it becomes necessary to determine whether the disease occurs more frequently in the workers exposed to a specific agent than in workers of the same age, sex, socio-economic status, etc. who are not so exposed. This is the situation in regard to linking health effects such as lung cancer to asbestos exposure. In such instances, several different agents may independently be associated with the disease. They may also interact to increase or decrease risks.

The third type of occupational disease is one where the disease has been established as occurring in occupationally exposed persons or is so defined. Such a disease is classical silicosis which occurs in persons exposed to high levels of respirable crystalline silica dust, often over relatively long periods of time. These are conditions which are typically encountered occupationally.

The key factors which determine whether or not a worker contracts or is identified with a dust related disease are as follows:

i. The characteristics and properties of the dust.

ii. The concentrations of the respirable dust to which the worker is exposed.

iii Duration of exposure to the airborne dust.

iv. Pattern of exposure.

v. Period since first exposure to time that the worker is examined for health effects.

vi. Personal habits of the worker which might directly or indirectly:

a. influence the risk of occurrence of a dust related disease(s) or health effect(s) or influence the course of the disease (eg: smoking).

b. produce a disease indistinguishable from that produced by the dust.

vii. Individual personal characteristics or pre-disposing conditions which might directly or indirectly influence the occurrence or course of a disease in that worker.

There are several essential considerations in determining the link between exposure and disease in both groups and in individuals.

First, the presence of the disease or health effect must be clearly defined and demonstrated. Second, it must make mechanistic sense for the disease to be associated with the agents or dusts to which the individual or group are exposed. Third, the nature and level of the dust exposure must be appropriate to explaining the nature of the disease or health effect and frequency with which it occurs. Fourth, other agents or factors known to be associated with, give rise to, or influence the occurrence or course of the disease must be evaluated. In the following sections, these are examined in relation to the diseases associated with the various asbestos minerals.

UPDATE:

The types of occupationally related diseases and the factors responsible for them remain as described above. In relation to asbestos, the same diseases have been emphasized (mesothelioma, lung cancer and asbestosis). However, over the past 10 years, there has been an increasing tendency to classify exposures as occupational, paraoccupational, domestic, building and environmental. This has been stimulated by increased concern about the risks for occupants and workers in buildings (Harvard 1988, HEI-AR 1991; Browne 1994); the finding that mesotheliomas were occurring more frequently in workers outside of the conventional production and manufacturing sectors (Peto et al 1999; Hodgson et al 1997; Hutchings et al 1996; Albin et al 1999; Peto et al 1995) and continued reports of spousal or familial mesotheliomas. As it has been claimed that an increasing number of persons working periodically with asbestos or in the vicinity of asbestos workers are at an increased risk of asbestos-related diseases, this new nomenclature has been introduced.

General definitions seem to be as follows:

- Occupational exposure refers to exposure which occurs while working directly with asbestos.

Para-occupational exposure refers to the exposure of workers through the work of others. It normally results from working in the vicinity of workers applying or removing insulation products.

Domestic exposure (and sometimes para-occupational exposure) has been used to refer to exposure in the home as a result of workers bringing work clothes home after working in asbestos producing, manufacturing, or using occupations. Such exposures can potentially be important for household members, especially spouses and children.

While measurements of exposure are few, the fact that domestic exposures can be relatively high has been confirmed by fibre lung burden measurements (Gibbs et al 1990) and by measurements made in the homes of workers (Nicholson 1980).

Neighbourhood exposure refers to exposures resulting from living in the vicinity of a factory or other source of airborne asbestos.

Building exposure refers to exposure resulting from living or working in a building containing asbestos. In a properly maintained building containing chrysotile asbestos the average building resident is not at any practically detectable increased risk of mesothelioma, lung cancer or asbestosis.

Environmental exposure refers to exposure resulting from the natural background of fibres in the environment.

Handyman exposure refers to the occasional exposure of maintenance personnel or handymen to asbestos by working directly with asbestos products.

While the above terms are widely used, such categorisation of exposures is not, in my view, appropriate as low exposures can occur in occupational settings and high exposures can occur under para-occupational circumstances.

There have been no major changes in the science underlying the importance of the factors (i - vii) noted in the original June 1992 report. However, the increasing emphasis on genetics in the mid-1990's is leading to an increased understanding of the genetic parameters affected by chemical exposures and of the genetic factors that might put persons at greater risk of certain diseases. The possibility that different genetic alterations occur in the same tumour as the result of different environmental insults has been suggested. Such factors may become important parameters to consider in the future, but are not yet at a point where they can be addressed from an epidemiological standpoint.

7.0 DISEASES ASSOCIATED WITH ASBESTOS EXPOSURE

The diseases or health effects which are frequently studied in relation to asbestos exposure are.....asbestosis[6], lung cancer and mesothelioma. Other diseases such as gastrointestinal cancer, cancer of the larynx, cancer of the ovary as well as cancers of other sites remain controversial.

UPDATE:

There has been no change in the main diseases (ie. lung cancer, mesothelioma and asbestosis) studied for their relationship to exposure to the various asbestos minerals. IPCS (1998) in addressing other diseases stated:

> "There has been considerable unresolved controversy regarding the possible carcinogenic effect of asbestos on the larynx, kidney and gastrointestinal tract. Moreover there is little evidence that permits an assessment of chrysotile in particular as a risk factor for these cancers. In four of the cohorts exposed almost exclusively to chrysotile, data were presented on SMRs for laryngeal cancer (Hughes et al 1987; Piolatto et al 1990; McDonald et al 1993, Dement et al 1994). Non-significant excesses were observed in some of the studies. It is not possible to draw conclusions about the association with laryngal cancer because confounding may play an important role in creating associations. Where examined, laryngeal cancer was strongly associated with cigarette smoking (McDonald et al 1993) and alcohol consumption (Piolatto et al 1990)."

IPCS (1998) also pointed out that because kidney cancer is rare, cohort studies have limited statistical power to detect even moderate excesses. Referring to McDonald et al (1993) they noted that there was no overall excess of kidney cancer in Quebec miners and millers although some increases occurred in subgroups stratified by mine and exposure but the number of cases "precludes meaningfully interpretation". In an asbestos cement production plant (chrysotile) Hughes et al (1987) reported an SMR of 225 for kidney cancer based on only 4 cases but the SMR from lung cancer was only 117.

IPCS (1998) also mentioned that there were no data on kidney cancer risks reported in other chrysotile worker chohorts.

In predominantly chrysotile exposed cohorts no consistent evidence of excess mortality from stomach or colorectal cancer and no systematic relation between gastric cancer and exposure in the Quebec chrysotile mining cohort (McDonald et al 1993) was noted. See also Liddell et al (1997).

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[6] Asbestosis is the scarring of the lung tissue resulting from exposure to normally high concentrations of asbestos minerals over many years.

7.1 MESOTHELIOMA

7.1.1 Occurrence

The tumour occurs on the pleura and peritoneum, membranes that surround the lung and line the thoracic and abdominal cavities. The pathological and clinical characteristics of mesotheliomas are now described in standard texts (Hunter 1987; Parkes 1975).

UPDATE:

Mesothelioma descriptions now appear in almost all standard medical texts (eg: Parkes 1994; Watson in Peckham et al 1995; Churg and Green 1998; Roggli et al (1994).

7.1.2 Diagnosis

Kannerstein et al (1979) drew attention to the difficulties of diagnosing this tumour and the value of "mesothelioma panels" to review cases. They noted that due to the "pathologic overlap with other neoplasms ... there is scarcely another variety of tumour which is so ill-defined and which admits of so much doubt as to its true nature".

Microscopically the tumour shows considerable variation and the appearance varies from tumour to tumour making accurate diagnosis difficult. Parkes (1975) notes that the diagnosis has either been by biopsy alone or at autopsy. He states that diagnosis made by biopsy alone is not reliable.

The difficulties noted above are exemplified in the study by McDonald et al (1973). Six pathologists reviewed 119 of 165 cases reported as primary malignant mesothelial tumours between 1960 and 1968 in a Canadian national survey. Based on histologic observations only, the panel was in favour of the diagnosis in 50% of cases, uncertain in 14% and against in 36%. This illustrates the tendency at that time to "over diagnose mesothelioma". In the United Kingdom, Greenberg & Davies (1974) reported that of 413 notifications of mesotheliomas: 76 were "definitely not" mesotheliomas when reviewed; 35 of these 76 had been identified as mesothelioma on the death certificate.

In a quite recent study of 78 autopsied cases in Canada, pathological review resulted in 33 (42%) being considered as definite mesotheliomas, 25 (32%) probable, 7 (9%) possible, 4 (5%) doubtful but 9 could not be classified because of inadequate tissue samples (McDonald et al 1989).

While it is likely that the tendency is towards being more rigorous in diagnosis, diagnosis is difficult and must be based on stringent standards of post mortem examination (Parkes 1975). Review by panels of pathologists is now the accepted method of obtaining agreement on such cases.

The early cases identified by Wagner et al (1960) were referred as having tuberculosis. More recently the possibility was raised that some of the women with "cancer of the ovary" who worked with blue

asbestos (crocidolite) while making military gas masks may have had mesotheliomas (Acheson et al 1982). The existence of benign mesothelioma must also be considered. For example, Daya & McCaughey (1990) reported 22 cases (18 women) of well differentiated papillary mesothelioma of the peritoneum (WDPMP) which they described, for practical purposes, as benign. The cause of these tumours is not known[7]. This further illustrates diagnostic complexity.

UPDATE:

The 1992 report drew attention to the considerable difficulties in diagnosis. This still appears to be a problem. As Wang (1996) stated: "After a century of debate and more than 3 decades of intense epidemiological, clinical and morphological studies, a specific and definitive diagnostic method of mesothelioma still eludes detection". Referring to McCaughey et al (1991) who reported on North American mesothelioma diagnosis, Wang notes: "Discrepancy in scoring among members is common, especially when only haematoxylin-eosin stained slides are available and the clinical history and gross findings inadequate. Similar observer variations appear to persist even when the results of electron (EM) microscopy and immuno-histochemistry are available for analysis". This is extremely important as diagnoses are sometimes made by pathologists who see very few cases, and diagnosis are sometimes based only on biopsies.

A major segment of the recent literature on mesothelioma is devoted to differentiating between lung cancer and mesothelioma and proposed methods of improving diagnosis (eg: Dejmek 1996; Nash et al 1999; Attanoos & Gibbs 1997; Churg & Green 1998; Colt 1997).

An example of a diagnostic/classification problem was described in Denmark where an unexpectedly large number of peritoneal mesotheliomas in women was reported to the Danish Cancer Registry during the year 1960 - 1985. A review found that only 37% were verified mesotheliomas and 33% possible mesotheliomas. Misclassification of other cancers was the reason for the peritoneal mesothelioma excess (Nielsen et al 1994).

Bias in reporting is also very important. For example a bias in reporting was identified in Sweden where asbestos exposure was found in 93% of cases reported to the Swedish Register of Reported Occupational Diseases while only 47% of the unreported cases had an asbestos exposure (Andersson & Toren 1995). While other registries report that mesothelioma reporting is good or complete, not all have undergone such a review.

As noted in the 1992 report, the presence of a disease or health effect must be clearly defined and demonstrated before any search for etiology is undertaken. The potential for a major problem with diagnosis exists when the fact or claim of asbestos exposure, regardless of the type or level of exposure is used as a criterion for diagnosis. Perhaps equally important is the fact that other potential

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[7] In their report they noted that although diffuse malignant mesothelioma is a highly malignant cancer, there are other mesothelial neoplasms that are less aggressive or are benign. They listed multicystic mesothelioma, adenomatoid tumour and "histogenetically contentious" localized fibrous mesothelioma as well as WDPMP.

etiologies are ignored. In Scandinavia, mesotheliomas have been reported in persons immigrating from the area of Turkey where mesothelioma rates are high (Ozesmi et al 1990).

7.1.3 Survival

In view of the high fatality rate with this tumour and short survival times, cases with very long survival times should be examined critically to ensure that the diagnosis is correct.

UPDATE:

In 1992, Ribak & Selikoff (1992) reported on the survival of 457 consecutive cases of pleural and peritoneal mesothelioma occurring among 17,800 asbestos insulation workers followed January 1, 1967 to January 1, 1987.

The mean survival time from initial presentation of pleural mesothelioma to death was 11.4 months compared to 7.4 months for those with peritoneal mesothelioma. This difference in survival was statistically significant. For pleural mesotheliomas only 1 case (0.7%) survived 36 months from initial symptoms to death. For peritoneal mesotheliomas only 1 case (0.4%) survived more than 36 months. Various treatment modalities were compared. They concluded that "no effective therapy is available yet."

While many new treatment regimes have been introduced and tested in the 1990's, this conclusion appears sadly to be as valid today as in 1992.

7.1.4 Etiological Factors

Etiological factors refer to those factors which are associated with a disease and which may explain its occurrence.

It is now well accepted that several factors are important in the occurrence of primary malignant mesothelioma. These fit well with the factors identified in Section 6.0 as essential in assessing the risk of occupationally related diseases:

* agent
* level of exposure
* duration of exposure
* period since first exposure to diagnosis
* other factors

UPDATE:

No changes.

7.1.4.1 Agent

Non-pulmonary pleural tumours have been reported in the literature for more than 100 years. However it would appear that the first formal link between primary malignant mesothelial tumours and an environmental factor was in 1959 when Dr. Christopher Wagner first diagnosed primary malignant mesotheliomas in persons living and/or working in the blue asbestos mines of South Africa (Wagner et al 1960). Since that time there has been considerable research carried out and some findings are now well established.....

* The majority of mesotheliomas in workers to date, have been shown to be associated with exposure to asbestos particularly to the amphibole fibres - blue asbestos, amosite and tremolite. This high proportion of mesotheliomas linked to asbestos exposure may, in part, be the result of biasses in reporting because major efforts have been directed at confirming asbestos associations, not at identifying other etiological factors. In spite of this...

    * In about 25-30% of mesothelioma cases, a clear history of asbestos exposure has not been established. While this may reflect differences in the methods used to establish exposure, those reporting this proportion appear to be reasonably confident that not all mesotheliomas are asbestos related.

    * Mesothelioma risks have been shown to be increased in persons exposed to a mineral other than asbestos, specifically a fibrous zeolite known as erionite (Baris et al 1987). Mesotheliomas have also been produced in experimental animals by fibres other than asbestos and zeolites.

* Fibre length, diameter, durability and biopersistence play important roles in a fibre's potential to induce mesothelioma, pulmonary fibrosis and pulmonary tumours in experimental systems.

* The tumour occurs in both sexes.

* The tumour risk appears to be unrelated to smoking

* Mesothelioma has a long "latency" with periods between first exposure and tumour detection of generally 20-40 years or more.

UPDATE:

In spite of another decade of research and an increased emphasis on tissue analyses to confirm exposure, there still remains a substantial proportion of mesothelioma cases in which asbestos exposure has not been established. A number of other causes for mesothelioma appear to be increasingly plausible and the possibility that other agents might play a role in asbestos related mesotheliomas is now receiving serious consideration.

Spirtas et al (1994) reported that the proportion of pleural mesotheliomas in men attributable to asbestos exposure was 88%. The attributable risk for peritoneal mesothelioma in men was 58%. For both sites combined the attributable risk in women was 23%.

Some caution is needed in interpreting the results of various studies as the findings will vary with the likelihood of exposure to asbestos by the study population.

Yates et al (1996) reported that among 272 mesotheliomas, 87% had documented asbestos exposure, while in the remainder, no asbestos exposure or asbestos bodies were found. Thus in South East England, most cases were asbestos related. There were no clinical differences between those in which asbestos exposure was established and those where it was not.

It now appears that between 10 and 25% of mesotheliomas occur where an asbestos etiology cannot be confirmed, although even in 1989, there were 33% of mesotheliomas at one urban centre where there was no historic evidence of asbestos exposure (Shepherd et al 1989). The occurrence of mesotheliomas in childhood and the existence of the stable background rate of mesothelioma in women in most countries suggest that non-asbestos etiologies exist. This is consistent with the multiple etiologies of cancer in general.

Clearly the key question of whether or not there are non-asbestos related mesotheliomas or mesotheliomas at low levels of exposure must relate to the agents, exposure and dose. A recent review by Hillerdal (1999) illustrates one opinion on this complex question of background rates and low level exposure. Another view is expressed by McDonald & McDonald (1996).

7.1.4.2 Asbestos Fibre Type

The differences in the risk of mesothelioma for workers exposed to different asbestos fibre types are quite marked. These risks will be described for the various fibre types and then compared.

a. Blue Asbestos

TABLE 1 shows the mesothelioma experience of persons who were occupationally exposed to blue asbestos. It is clear from these results that mesothelioma is strongly associated with working with blue asbestos and that the risks for workers in occupations where this material has been used can be very high.

b. Amosite

Amosite has been implicated in mesothelioma as an important cause of death in a US amosite factory (Seidman et al 1979) and in a British amosite factory (Acheson et al 1984). See TABLE 2.

c. Mixed fibre exposures

The pattern of mesothelioma mortality in workers exposed to mixtures of amphiboles and chrysotile are very similar to those among amphibole only exposed workers. See TABLE 3.

TABLE 1. MESOTHELIOMA IN VARIOUS COHORTS - AMPHIBOLE ONLY - BLUE ASBESTOS -

a. Gas mask filter manufacture - Canada
McDonald & McDonald (1978)

Total Cohort	Dead	Mesothelioma
199	56	9 (16.1%)[8]

b. Gas mask filter manufacture - UK
Acheson et al (1982)

Total Cohort	Dead	Mesothelioma
757	219	5 (2.3%)

c. Mining Blue Asbestos - Australia
Hobbs et al (1980)

Total Cohort	Dead	Mesothelioma
6200	526	17 (3.2%)

TABLE 2. MESOTHELIOMA IN VARIOUS COHORTS - AMPHIBOLE ONLY - AMOSITE ASBESTOS -

a. Amosite factory - US
Seidman et al (1979)

Total Cohort	Dead	Mesothelioma
820	528	14 (2.7%)

b. Amosite factory - UK
Acheson et al (1984)

Total Cohort	Dead	Mesothelioma
5969	422	5 (1.2%)

[8] As used in tables 1-5, parentheses represent percentage of deaths due to mesothelioma

TABLE 3. MESOTHELIOMA IN VARIOUS COHORTS - MIXED FIBRE - AMPHIBOLE & CHRYSOTILE -

a. Insulation workers - NY - New Jersey
Selikoff et al (1979a)

Total Cohort	Dead	Mesothelioma
632	478	38 (7.9%)

b. Insulation workers - US & Canada
Selikoff et al (1979a)

Total Cohort	Dead	Mesothelioma
17800	2271	175 (7.7%)

c. Dockyards - UK
Rossiter & Coles (1980)

Total Cohort	Dead	Mesothelioma
6292	1043	31 (3.0%)

d.Insulators in shipyards - USA
Selikoff et al (1979b)

Total Cohort	Dead	Mesothelioma
440	79	8 (10.1%)

e. Asbestos factory workers in London - UK
Newhouse & Berry (1979)

Total Cohort	Dead	Mesothelioma
M 4600	775	46(5.9%)
F 992	225	21(9.3%)

d. Tremolite

Fibrous tremolite has been implicated in the occurrence of mesothelioma in vermiculite miners where tremolite is a contaminant of the vermiculite (McDonald et al (1986a) and Amandus & Wheeler (1987). See TABLE 4. Mesotheliomas have also been reported in talc miners where the talc contained asbestiform tremolite.

TABLE 4. MESOTHELIOMA IN VARIOUS COHORTS - TREMOLITE ONLY -

a. Vermiculite mining - USA
McDonald et al (1986a)

Total Cohort	Dead	Mesothelioma
406.	165	4 (2.4%)[9]

b. Vermiculite mining - USA
Amandus & Wheeler (1987)

Total Cohort	Dead	Mesothelioma
575	161	2 (1.2%)[9]

e. Chrysotile

Mesothelioma has been relatively rarely described in pure chrysotile exposed workers. See TABLE 5. As noted earlier, the finding of tremolite fibres in high concentrations in the lungs of some Quebec chrysotile miners and millers (Rowlands et al 1982) has led to the hypothesis that the tremolite contamination of chrysotile may explain the increased mesothelioma risk in that mining and milling industry.

TABLE 5. MESOTHELIOMA IN VARIOUS COHORTS - CHRYSOTILE ONLY -

a. Chrysotile miners & millers
McDonald et al (1980)

Total Cohort	Dead	Mesothelioma
11379	4547	11 (0.24%)[10]

[9] These studies were conducted at the same mine.

[10] Some persons known to have worked with amphibole fibres.

TABLE 5 (CONT.) MESOTHELIOMA IN VARIOUS COHORTS - CHRYSOTILE ONLY -

b. Chrysotile miners & millers
Rubino et al (1979)

Total Cohort	Dead	Mesothelioma
900	332	1 (0.3%)[11]

c. Chrysotile miners & millers
Nicholson et al (1979)

Total Cohort[12]	Dead	Mesothelioma
544	178	1 (0.56%)

d. Chrysotile products factory
Weiss (1977)

Total Cohort	Dead	Mesothelioma
264	66	0 (-)

e. Asbestos cement plant
Thomas et al (1982)

Total Cohort	Dead	Mesothelioma
1970	351	0[13]

f. Chrysotile textile plant
McDonald et al (1983a)

Total Cohort	Dead	Mesothelioma
2543	863	1 (0.1%)[14]

[11] Not supported by histological examination.

[12] Cohort comprised men with at least 20 years seniority.

[13] There were 2 mesothelioma exposed to blue asbestos (pre 1936) but 0 in those exposed to chrysotile only.

[14] There was one peritoneal mesothelioma with no autopsy.

TABLE 5 (CONT.) MESOTHELIOMA IN VARIOUS COHORTS - CHRYSOTILE ONLY -

g. Friction materials manufacture - USA
McDonald et al (1984)

Total Cohort	Dead	Mesothelioma
3641	1267	0 (-)

h. Friction materials manufacture - UK
Newhouse & Sullivan (1989)

Total Cohort	Dead	Mesothelioma
13450	2577	2[15]

f. Comparisons of risks

The occurrence of 9 mesotheliomas among 56 deaths of crocidolite gas mask workers (16.1%) and 11 among 4,247 deaths of chrysotile miners (0.26%) illustrate clear differences in the mesothelioma risk posed by these two fibres (McDonald & McDonald 1978). See TABLE 6a. This difference and the strong association between blue asbestos and mesothelioma is paralleled by observations in several other studies.

TABLE 6a.

MESOTHELIOMA IN CHRYSOTILE MINERS & MILLERS AND BLUE ASBESTOS GAS MASK WORKERS
McDonald & McDonald (1978)

	Blue Asbestos Gas Mask Filter Workers	Chrysotile Miners & Millers
Total Cohort	199 (55% male)	11,379(96% male)
Dead	56	4,247(since 1935)
Mesothelioma	9 (16.1%)	11(0.26%)[16]

[15] There were 13 mesotheliomas at the plant: 11 had contact with blue asbestos; in the remainder, the diagnosis of 1 was uncertain and the work history of the other was not well established.

[16] Two cases were common to both series. Some chrysotile workers may have been exposed to other fibre types by work in a factory in one of the mining towns and/or in experimental/pilot plants.

In the study of workers manufacturing civilian gas masks using chrysotile asbestos there were no mesothelioma that could be linked to the chrysotile exposures. At the plant manufacturing military gas masks using blue asbestos 2.3% of the deaths were due to this cancer (Acheson et al 1982). See TABLE 6b.

TABLE 6b.

MESOTHELIOMA IN MILITARY (BLUE) & CIVILIAN (CHRYSOTILE) GAS MASK WORKERS
Acheson et al (1982)

	Blue Asbestos Gas Mask Filter Workers	Chrysotile Gas Mask Filter Workers
Total Cohort	757	570
Dead	219	177
Mesothelioma	5(2.3%)	1[17]

The mesothelioma experience of insulation workers[18] has been considerably worse than that of chrysotile miners and millers, chrysotile textile workers, friction manufacturing workers and other chrysotile-only exposed populations which makes it likely that their mesotheliomas are not linked to their chrysotile exposure.

Amosite has been implicated in mesothelioma as an important cause of death in an amosite factory in the US (Seidman et al 1979), with 14 of 528 amosite worker deaths (2.7%). It seems most probable that the mesothelioma experience of amosite and insulation workers is explained by their exposure to amphibole fibres. This association between amphibole fibre exposure and mesothelioma has been further supported by several case-control studies in which the lung fibre contents of mesothelioma cases and controls have been determined.

In a recent study in Canada, lung tissues from 78 mesothelioma cases and matched "referents"[19] were analyzed (McDonald et al 1989). The different fibre types were classified by length (greater than or equal to 8um and less than 8 um). More than one type of fibre and both long and short fibres were found in the lung tissue from some individuals. Despite the fact that concentrations of short fibres were highly correlated with the concentrations of long ones "analyses in which both short and long fibre concentrations were entered simultaneously into risk models showed likelihoods barely increased from those for long fibres alone indicating that short fibres were not significantly associated with excess risk, after accounting for the effect of long fibres".

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[17] This case was also considered to have been exposed to blue asbestos at another factory. There was also an excess number of persons with cancer of the ovary at the blue fibre plant. These were considered by the authors to possibly be additional mesotheliomas.

[18] Insulation workers, in general have usually worked with chrysotile and amosite and certainly in many countries with blue asbestos.

[19] Persons from the same autopsy registry who did not die of malignancy or respiratory disease but who were of the same age, sex, date of death, and for whom the same type of tissue (paraffin block, or wet) was available.

When the possible confounding effect of more than one fibre type in the lung was taken into account through the use of multivariate methods, risk increments were derived. See TABLE 7.

TABLE 7.

MESOTHELIOMA RISK INCREMENTS PER FIBRE PER MICROGRAM
(MCDONALD ET AL 1989)

Fibre	Risk Increment Risk(%)	(95% C.I.)[20]	Attributable[21]
Long Fibres (>8um)			
Amphiboles(all)	32.1	(8, 120)	68
Amosite	93.7	(20, 500)	28
Crocidolite	24.9	(1.4, 120)	10
Tremolite	69.7	(9,300)	30
Talc/Anthophyllite	1.8	(<0, 50)	2
Chrysotile	<0	(<0, 8)	--
Short Fibres (<8um)			
Amphiboles (all)	0.7	(0.1, 3)	55
Amosite	13.5	(0, 980)	20
Crocidolite	1.6	(0, 110)	13
Tremolite	0.6	(0, 3)	18
Talc/Anthophyllite	0.2	(0, 17)	6
Chrysotile	0.02	(<0, 0.2)	6

[20] 95% C.I. = Confidence Intervals. The lower limit is shown on the left and the upper limit on the right. This means that the true value of the risk increment lies within these confidence intervals unless a one in twenty mischance has occurred.

[21] Attributable risk is the proportion of cases occurring in the total population which can be explained by the risk factor.

The "increment in relative risk" is the slope of the line expressing the relationship between the relative risk of mesothelioma and the number of fibres per microgram of dry lung tissue. It should be noted that the confidence intervals for the 4 amphibole fibres are wide and differences between them were no more than could be accounted for by chance ($p > 0.10$). The much lower risk for chrysotile could not be accounted for by chance ($p < 0.001$). The attributable risk suggests that perhaps 30% of the mesotheliomas were attributable to tremolite exposure. All the mesothelioma from the Quebec mines and mills (9 cases) were among these.

A similar study was conducted in Australia, but controls were taken from only one hospital and not matched (Rogers et al 1991). The greatest risk of mesothelioma in Australia was associated with crocidolite fibres >10 um in length but that study claimed a significant contribution by short chrysotile fibres. Recognising that hospitals are often catchment areas for the industries in their district, the use of "control" tissues from one single centre in a national study may be a serious design flaw.

A second important factor is that the large numbers of short chrysotile fibres may reflect recent exposures to this fibre type. Chrysotile is also well recognised to be removed from the lung over time (Davis 1991) and this may have prompted Rogers et al to note the difficulty of assessing the risks of chrysotile alone due to the almost universal exposure to both chrysotile and amphiboles.

Although the levels of exposure of the various occupational groups described in this section have not been as well defined as one might like, the experience of workers using different fibre types have been so different that it is clearly inappropriate to extrapolate from the mesothelioma experience of persons working with one fibre type to another. Thus, in this report, only the experience of persons exposed to the asbestos fibre types likely to be found in Dum Dum products will be used in assessing the risk associated with these products.

UPDATE:

By 1992, differences in the risk of mesothelioma for workers exposed to different asbestos fibre types were quite marked and there have been no studies since to contradict this conclusion. Indeed, the difference between the mesothelioma producing potential of commercial chrysotile exposure and amphibole fibre exposure was clearly defined at a meeting of experts in 1994 (See Gibbs et al 1994). However, in the 1990s there were several reviews claiming that the risk of mesothelioma was the same for chrysotile and the amphiboles, that chrysotile under all circumstances caused mesothelioma or that the widespread use of chrysotile made it responsible for most of the mesotheliomas occuring in the world. In this section, this debate will be critiqued and new information concerning the mesothelioma risks associated with the various asbestos fibre types which has been published over the past decade will be summarized. In almost all instances these claims include mesotheliomas where exposure has been to amphibole as well as chrysotile.

The Amphibole Hypothesis.

Since 1990 there have been several articles claiming that chrysotile is responsible for mesothelioma occurrences. One such paper is that by Stayner et al (1996). Cullen (1996) also wrote on this subject. Both received critical comment from Wagner (1997) who first linked mesothelioma to

crocidolite asbestos exposure in 1959. He stated very clearly that; "The vast majority of mesotheliomas are associated with exposure to crocidolite asbestos..... No mesotheliomas have been shown to have occurred in chrysotile exposed workers unless the exposure has been intense and for more than 20 years. In addition there must be tremolite contamination of the chrysotile".

Langer and Nolan (1997), pointed out evidence which supported Wagner's contention. Mossman and Gee (1997) pointed out that both the articles by Stayner et al and Cullen did not include recent data and noticed errors in Stayner et al's claim that the carcinogenicity of crocidolite was based primarily on in-vitro studies. While Stayner et al (1996) accepted that there was a difference in the mesothelioma producing potential of amphibole and chrysotile fibres, their literature review on chrysotile was incomplete, selective, contained errors and failed to reflect the observations of the original researchers.

A second paper was published by Smith et al (1996). This article included a number of technically incorrect statements, errors and omissions. By only including studies in which high mesothelioma risks were reported, they were able to exclude from consideration those industries where risks were not reported because no cases had occurred, namely the chrysotile using industries. In fact their "evidence" was based on industries involving mixed exposures to amphibole and chrysotile. In making broad generalisations, they ignored the fact that not all chrysotile is contaminated with fibrous tremolite. They ignored the fact that exposure level is important. They also ignored the fact that the likelihood of downstream users being exposed to fibrous tremolite in chrysotile is extremely low if not nil. The inference was made that because most of the world's asbestos is chrysotile, most of the world's mesotheliomas are chrysotile related. This does not automatically equate. Most, if not all asbestos-related mesothelioma cases have had amphibole or mixed fibre exposures. Liddell (1997) commenting on the paper by Smith et al summed it up: "Therefore, these 16 studies which, by the definition of a low mesothelioma PMR included every one of those cohorts exposed to chrysotile alone, were ignored. Further comment is surely unnecessary- although a great deal more would be fully justified."

A review by McDonald and McDonald (1996) provides a rather different and more rational interpretation of the world's literature on mesothelioma.

Two other papers claiming that chrysotile is responsible for mesotheliomas were published by Nicholson and Landrigan (1994) and Nicholson and Raffn (1995). The articles depend on their assertion that amphibole exposures did not occur in the USA prior to about the mid 1930s. Evidence which I have gathered concerning the importation, mining and use of amphibole fibres in the USA prior to 1940 demonstrates that there was ample opportunity for workers to be exposed to amphiboles well prior to 1935. Those exposures included amosite, crocidolite and anthophyllite.

The fallacy in the argument used by Nicholson and colleagues is perpetuated by Smith et al (1996), Stayner et al (1996) and others later - ie: that a little amphibole usage is not important. The work of Koyhama and Suzuki (1991) which shows commercial amphibole fibres in the lungs of all the US insulation workers studied by them, raises serious doubts about any claims of non-amphibole related mesotheliomas in US insulation workers.

Amosite was also found in the lungs of all insulation workers in a study by Langer & Nolan (1998). Crocidolite was present in the lungs of 13% of insulation workers and 38.3% of shipyard workers. Crocidolite and amosite are not minerals commonly found in high concentration in the lungs of the general population.

Since the 1992 report, some studies of the mortality experience of asbestos exposed workers have been updated and new studies have been reported. The following section incorporates this new and updated information

a. Blue Asbestos

TABLE U1 shows the mesothelioma experience of persons who were occupationally exposed to blue asbestos. It is even clearer now, that mesothelioma is strongly associated with exposure to blue asbestos.

TABLE U1. MESOTHELIOMA IN VARIOUS COHORTS - AMPHIBOLE ONLY - BLUE ASBESTOS -

ua. Mining Blue Asbestos - Australia
Berry G (1991)

Total Cohort	Dead	Mesothelioma
6258	983	84 (8.5%)

ub. Mining Blue Asbestos - Australia
de Klerk et al (1993)

Total Cohort	Dead	Mesothelioma
1106	193	12 (6.2%)

uc. Mining Blue Asbestos - South Africa
Sluis-Cremer et al (1992)

Total Cohort	Dead	Mesothelioma
3430	423	20*(4.7%)

* Best evidence - 17 pleural

TABLE U1 (CONT). MESOTHELIOMA IN VARIOUS COHORTS - AMPHIBOLE ONLY - BLUE ASBESTOS -

ud. Blue Asbestos Cigarette filter Production
Talcott et al (1989)

Total Cohort	Dead	Mesothelioma
35	28	5*(17.8%)

* 1 pleural, 4 peritoneal,

Another Australian study was reported by Musk et al (1992) with 6505 men in the cohort and 32 mesotheliomas but the number of deaths was not clear. However, information on this Wittenoon cohort has been adequately described by the other Australian authors.

b. Amosite

Amosite has been directly implicated in mesothelioma as an important cause of mesothelioma in the US amosite factory reported by Seidman et al 1979, in a British amosite factory (Acheson et al 1984), and now by further studies in Texas (Levin et al 1998) and in South Africa (Sluis-Cremer et al 1992). See TABLE U2.

TABLE U2. MESOTHELIOMA IN VARIOUS COHORTS - AMPHIBOLE ONLY - AMOSITE ASBESTOS -

ua. Mining Amosite Asbestos - South Africa
Sluis-Cremer et al (1992)

Total Cohort	Dead	Mesothelioma
3212	648	4 (0.6%)

ub. Amosite factory - Tyler Texas - US
Levin et al (1998)

Total Cohort	Dead	Mesothelioma
1130	315	6** (1.9%)

** 4 pleural and 2 peritoneal. 2.7% of persons with more than 10 years from first exposure.

In an experiment with Baboons, 5 out of 12 developed malignant diffuse mesothelioma, three peritoneal, and two pleural after exposure to amosite at 1100-1200 f/cc for up to 898 days (Webster et al 1993).

c. Mixed asbestos fibre exposures

TABLE U3. MESOTHELIOMA IN VARIOUS COHORTS - MIXED FIBRE - AMPHIBOLE & CHRYSOTILE -

ua.Insulators in shipyards - Sweden
Jarvholm& Sanden (1998)

Total Cohort	Dead	Mesothelioma
248	86	7@ (8.1%)

@ All peritoneal

ub. Insulation workers - US & Canada
Selikoff & Seidman (1991)

Total Cohort	Dead	Mesothelioma
17800	4951	458 (9.3%)#

#Best estimate.
Pleural mesotheliomas 173.
Peritoneal msotheliomas 285.

d. Tremolite

While there have been many studies of fibre burden, there do not appear to have been any new occupational cohort studies.

e. Chrysotile

There have been claims of mesothelioma resulting from chrysotile only exposures in the former East Germany (Sturm et al 1994). I met with these researchers and discussed these cases. The cases were examined at one central laboratory but there were no tissue analyses for fibre type. The denominator for these cases is not well known. There was some blue asbestos used in some parts of Germany in insulation materials although most of their asbestos came from Russia. Without tissue analyses to verify the exposures of the "chrysotile only" exposed persons, this study cannot be interpreted. I was informed that because of the amalgamation of the two Germany's, no further work would be done on this question.

Only 1 probable mesothelioma was reported from Zimbabwe chrysotile mining operations as of 1991. It appears that there was no autopsy (Cullen & Baloyi 1991). Elmes (1994) reported that:"The chrysotile mines in the Transvaal, Swaziland and Zimbabwe appear to have exposed large numbers of workers to high levels of of pure chrysotile ... and yet there have been very few cases of lung cancer let alone mesotheliomas among the miners and mine mill workers (Webster, personal communication 1993; Baloyi, this workshop)."

TABLE U4. MESOTHELIOMA IN VARIOUS COHORTS - CHRYSOTILE ONLY -

ua. Chrysotile miners & millers
McDonald et al (1997)

Total Cohort	Dead	Mesothelioma
9780	8009	38 (0.47%)
T 5041	4125	25 (0.61%)
A 4031	3331	8 (0.2%)
F 708	553	5 (0.9%)

F is a factory where crocidolite & amosite were used. Out of about 11,000 men born 1891 - 1920 in the original cohort, 9780 survived into 1936.

ub. Chrysotile textile plant
Dement et al (1994)

Total Cohort	Dead	Mesothelioma
3022	1259	2 (0.16%)@

@ Sebastien et al (1989) and Case and Dufresne (1999) showed that some workers in this cohort had amphibole fibers in their lungs

uc. Mining chrysotile -Italy
Piolatto (1990)

Total Cohort	Dead	Mesothelioma
1058	427	2 (0.46%)@

@ At the Medichem Symposium in Vienna (1999) Dr Gruber,Switzerland reported that amphibole fibres had been milled at this mine.

f. Anthophyllite

TABLE U5. MESOTHELIOMA IN ANTHOPHYLLITE COHORTS - ANTHOPHYLLITE ONLY -

Anthophyllite miners -Finland
Karjalainen et al (1994)

Total Cohort	Dead	Mesothelioma
999	503	4* (0.8%)

* 3 pleural and 1 peritoneal

TABLE U5. (CONT.) MESOTHELIOMA IN ANTHOPHYLLITE COHORTS
- ANTHOPHYLLITE ONLY -

Anthophyllite miners -Finland
Meurman et al (1994)

Total Cohort	Dead	Mesothelioma
735	137	4* (2.9%)

* 3 pleural and 1 peritoneal.

f. Comparisons of risks

The evidence from all the new and updated studies, listed above, is consistent with the risk of mesothelioma being associated with amphibole exposures. When "chrysotile only" workers have been reported, they have been exposed to very high concentrations and tremolite has been present (eg: Quebec miners and millers). There have been cases of mesothelioma in which only chrysotile has been found in tissues, but usually these laboratories have not studied referent cases and issues concerning the representativity of the tissue analyzed and likelihood that the fibres reflect recent exposures have not been adequately addressed. There has continued to be a consistent association between amphibole asbestos exposure and mesothelioma based on tissue burden studies.

7.1.4.3 Other Fibres

a. Zeolites:

Evidence that fibres other than asbestos may be related to mesothelioma in humans is found in Turkey, where, in several small villages, the rates of mesothelioma are very high.

Research concentrated in this area has shown that the most likely etiological factor is exposure to the fibrous zeolite, erionite, which occurs in the rock of that area (Baris et al 1987). This zeolite was shown to produce high rates of mesotheliomas in experimental animals (Wagner et al 1985).

b. Man-Made Mineral Fibres:

Certain of these fibres have the characteristics, size and durability that are widely accepted as making them candidates for the production of mesothelioma and experiments in animals have been reviewed by Hesterberg 1991a. Indeed, it is possible to produce mesothelioma in experimental animals by inoculation with a wide variety of naturally occurring and man-made mineral fibres (Monchaux et al 1985; Stanton 1973; Stanton & Layard 1978; Pott et al 1980; Pott et al 1987). Recent animal experiments in which animals inhaled ceramic fibres by a nose only route, yielded a high percentage of mesotheliomas (Hesterberg 1991b). To date, no excess of mesothelioma has been reported in any occupational groups producing man-made mineral fibres.

c. Biogenic Fibres:

The possibility that organically produced fibres might be related to mesothelioma was raised by observations of mesothelioma in sugar cane workers in India (Das et al 1976) where after burning the cane, there remain long silica fibres (Newman 1983). McDonald and McDonald (1991) note that there is support for this observation with the observed higher than expected rates of mesothelioma in the sugar producing areas of Louisiana (Rothchild and Mulvey 1982) and in sugar refinery workers in Sweden, although there, asbestos exposure may also have been involved (Steinbeck et al 1983; Malker et al 1983).

UPDATE:

In spite of considerable research, it still appears that some 10-25% of mesotheliomas occur in which an asbestos exposure cannot be ascertained. As noted earlier, the reasons for these other mesotheliomas continues to be a subject of scientific debate. Evidence for spontaneous and non-asbestos etiologies include the experience with erionite in Turkey (see below), spontaneous mesothelioma occurrence in children the occurrence of malignant pleural mesotheliomas before the industrial exploitation of asbestos at the end of the last century. There has also been an increasing number of other possible etiological factors identified including biogenic silica fibres in sugar cane, ionising radiation, and pleural scars following empyema or therapeutic pneumothorax (Hubbard 1997).

a. Zeolites:

In the previous report it was stated that there was evidence that fibres other than asbestos were associated with the occurrence of mesothelioma in Turkey, where, in several small villages, the rates of mesothelioma were extremely high when the population was exposed to fibrous erionite. Since then, Baris et al (1996) have further reported on the experience in 3 villages in the Cappadocian region of Central Anatolia, Karain, Tuzkoy and Sarihidir. They found that between 1970 and 1994 there were 305 deaths in Karain, 177 (58%) were cancer related including 150 (49.2%) malignant pleural mesothelioma and seven (2.3%) peritoneal mesothelioma. Between 1980 and 1994, there were 519 deaths in the other two villages, with 257 cancer related with 120 malignant pleural mesotheliomas and 64 malignant peritoneal mesotheliomas.

These rates of mesothelioma exceed even those associated with crocidolite gas mask work. Survival in the Turkish cases has been 13.52 months for erionite-associated pleural mesothelioma compared to 21.6 months for asbestos associated mesothelioma (Selcuk et al 1992). However, Ribak and Selikoff (1992) reported shorter survival periods for asbestos related mesotheliomas (see 7.1.3).

Evidence that cases originating in Turkey can turn up in other countries may be very important. As noted elsewhere in this report, among 150 immigrants to Stockholm from this region of Turkey, there were, by 1985, 7 cases of malignant mesothelioma in still, very young people (Ozesmi et al 1990) .

As noted in the 1992 report, erionite has been shown to produce very high rates of mesothelioma in experimental animals (Wagner et al 1985). The erionite fibres used in those experiments were

from Rome Oregon. More recent data, while confirming the carcinogenic potential of erionite using fibres from Pine Valley, Nevada did not produce the same high rates of mesothelioma (Fraire et al 1997).

Caution is needed in interpreting these findings as the physico-chemical properties, in particular, differences in size distributions have been found to be critical in other circumstances where differences in fibre carcinogenicity have been identified.

b. Man-Made Mineral Fibres:

In 1992 it was reported that certain of the man-made or synthetic fibres have the characteristics, size and durability that are widely accepted as making them candidates for the production of mesothelioma.

The statement that no excess of mesothelioma has been reported in any occupational group producing synthetic mineral fibres appears to be still valid in 1999. However, it must be remembered that the cohorts have been production workers where exposures have been extremely low. Boffetta et al (1997) in a study of 22,002 production workers with 4521 deaths found 5 deaths from pleural mesothelioma. This is a PMR of 0.1%. They note that this may not represent an excess. Overall they did find a possibly increased lung cancer risk in rock and slag wool workers. There were 2 cases of mesothelioma with more than 12 years of employment in rock and slag wool production [PMR = 0.16] and 1 in glass wool production [PMR = 0.06%] with 25 years of employment.

The lung cancer risk for rock and slag wool workers increased with time since first exposure and with duration of exposure. They concluded: "These results are not sufficient to conclude that the increased lung cancer risk is the result of exposure to rock/slag wool; however, insofar as respirable fibres were an important component of the ambient pollution of the working environment, they may have contributed to the increased risk." A Case-control study is now underway to eliminate possible "confounders".

In a US Study (Marsh et al 1996), only 1 mesothelioma was reported in cohort of 443 workers from one factory with 237 deaths, but none among a larger cohort of 3035 workers with 781 deaths. Cohorts of refractory ceramic fibre workers are small, have had low exposure and it is probably premature to see mesotheliomas in these cohorts even if there were a risk. It should be noted that there are many different types of synthetic vitreous and ceramic fibre.

c. Biogenic Fibres:

The possibility that biogenically produced fibres might be related to mesothelioma is still under investigation.

Bhatt et al (1991) using fibres from the surfaces of the grains of P. canariensis failed to produce mesotheliomas experimentally. However, in rats injected intra-peritoneally with the carcinogen 15,16- dihydro-11-methylcyclopent(a)phenantren-17-one (11-methyl 17-ketone), silica fibres implanted on the pleura induced mesotheliomas indicating that they can act as promoters.

To date, epidemiological investigations have not produced any firm evidence of a relationship between biogenic fibre exposure and mesothelioma. In Hawaii, there was no statistically significant excess risk of mesothelioma in sugarcane workers (OR = 1.3; 95%CI = 0.4-3.8). Studies have not identified any sugarcane workers who developed mesothelioma and worked in jobs where high exposure levels to biogenic silica fibres had been measured (Sinks et al 1994). In Florida, Brooks et al (1992) found a slight but non significant increased risk of lung cancer and one mesothelioma case and no controls who worked in the sugarcane industry. Maltoni and colleagues (1995) reported 12 cases of mesothelioma in sugar refinery workers but concluded that they were exposed to asbestos.

7.1.4.4 Ionizing Radiation

The possibility that ionising radiation may be responsible for isolated cases of malignant mesothelioma has been noted in the literature (Stock et al 1979; Pelnar 1988).

UPDATE:

In the 1992 report, the possibility that ionizing radiation may be responsible for isolated cases of malignant mesothelioma was noted.

Since then, there have been several reports addressing the question of radiation effects. These are summarized below:

Therapeutic radiation

There have been several case reports and case series reported since 1990 which suggest a link between therapeutic radiation and both localized benign and malignant mesothelioma. See TABLE U6.

There have now been many pleural and peritoneal mesotheliomas reported after radiotherapy for Hodgkin's disease, testicular carcinoma, cervical cancer, Wilms tumour and breast carcinoma as well as after radiation treatment for nonmalignant disease. There have also been mesothelioma reported after Thorotrast exposure. The time interval between irradiation and mesothelioma has ranged from a few years to 35 years or more. Radiation has been shown to be carcinogenic to the pleura and peritoneum and animal studies suggest a possible interaction with asbestos in inducing mesothelioma.

All the human evidence is from case reports except for the study by Neugut et al (1997) which did not find a statistically significant relationship between radiation treatment and mesothelioma, although the relative risk was 1.56. Carvazza (1996) noted that post-irradiation malignant mesothelioma show an approximately equal male:female ratio and average age at diagnosis of 45 which contrasts with the male predominance and older mean age seen in asbestos-related mesotheliomas.

Thorotrast

The results of various thorotrast studies are summarized in the TABLE U7.

TABLE U6.

MESOTHELIOMA OCCURENCES IN PERSONS RECEIVING THERAPEUTIC RADIATION TREATMENT

REFERENCE	CASE DESCRIPTION	ASBESTOS EXPOSURE
Lerman et al 1991	Malignant pleural mesothelioma in a woman 20 years after radiotherapy for Hodgkin's disease.	Sought but none found.
Pappo et al (1997)	3 mesotheliomas after childhood treatment. 1 Hodgkin's disease. Interval between treatment and secondary cancer = 11 years.	Sought but none found.
Cavazza et al (1996)	8 cases with mesothelioma at sites of radiotherapy for a prior tumour. Mean age at diagnosis = 45 years (range 22-78 years). Average interval between radiotherapy and mesothelioma was 21 years (range 11-29). 6 cases were treated for Hodgkin's disease and 2 for breast cancer. They also reviewed 27 previously reported cases of post-irradiation malignant mesotheliomas of the pleura and peritoneum.	Sought - none found in 4, unknown in 4.
Hoffman et al (1994)	A mesothelioma occured after radiation therapy for Hodgkin's disease. Interval between treatment and mesothelioma was 9 years.	Sought -none found.
Weissman et al (1996)	4 cases following radiation treatment of Hodgkin's disease. Mean interval = 15 years.	Sought - none found. 250 ferruginous bodies found in tissue from one case.
Neugat et al (1997)	Retrospective cohort study of 251,750 women with breast cancer. The overall estimated relative risk of malignant pleural mesothelioma after treatment was 1.56 (95% CI 0.18-5.63). No cases found in Hodgkin's disease treated patients.	No specific information available.
Shannon et al (1995)	Retrospective random review of 1000 irradiated breast cancer cases- 3 cases of malignant pleural mesothelioma reported after thoracic irradiation.	Persons with history compatible with asbestos exposure excluded from review.

REFERENCE	CASE DESCRIPTION	ASBESTOS EXPOSURE
Falchero et al (1996)	Case of malignant pleural mesothelioma after mantle radiotherapy for Hodgkin's disease. Interval 17 years.	Sought - none found.
Hill et al (1997)	Case report of benign localized pleural mesothelioma in a woman following adjuvant radiation of breast and axillary region.	Not mentioned.

TABLE U7. THOROTRAST

Nationality	Thorotrast Exposed	Control
German (Ishikawa et al 1995)	9/2241 (5 pleural and 4 peritoneal).	0/1575
Danish (Andersson et al 1995)	The risk for malignant mesothelioma reached 2.5% based on 7 cases, with an actuarial risk of 7-8% for patients receiving 20 ml of Thorotrast. A possible gradient was found between Thorotrast and malignant mesothelioma.	
Japanese (Ishikawa et al 1995)	1/258 (peritoneal). Combined pleuro-peritoneal and retroperitoneal malignances were five fold more frequent (1.1%) than in the controls (0.2%).	0/ 1630
Stey et al (1995)	Case report - peritoneal mesothelioma in a 63 year old male.	

Andersson (1995) notes that for malignant mesothelioma, the risk has been demonstrated to be elevated in the Danish, Japanese, and the German Studies.

Plutonium

Sanders (1992) showed that pleural mesothelioma was infrequent following deposition of $^{239}PuO2$ in the lung or pleura of rats with 5 tumours in 2105 rats. In a previous study, 4 tumours in 527 rats were found. The risk of pleural mesothelioma was not significantly increased by intrapleural injection of 30kBq $^{239}Puo2$, but a much higher incidence was found following intraperitoneal injection of $^{239}PuO2$ considered to be due to aggregation of particles. It was argued that these findings fit with pleural particle clearance limiting the a-irradiation.

In a postmortem research study using the United States Transuranium and Uranium Registries, there

were 6 mesotheliomas in the first 260 deaths (in a biased self selected cohort). Five of the 6 cases were known to have had potential for asbestos exposure. One was a uranium miner with no asbestos exposure. There was no apparent relationship with internal or external dose and mesothelioma (Gold and Kathren 1998). The authors caution against drawing conclusions, recognising the fact that the original cohort was voluntary.

Atomic Bomb

A case of mesothelioma has been reported in a man who developed mesothelioma of the left hemithorax 50 years after the atomic bomb was dropped on Nagasaki in 1945 (Mizuki et al (1997). An asbestos exposure cannot be ruled out.

Overall Radiation

While the case reports and anecdotal evidence are strong, the link between radiation and mesothelioma cannot be accepted as proven on present evidence. On the other hand, the data are highly suggestive of an association for some types of radiation exposure so a link cannot be dismissed without additional carefully controlled studies which are negative.

7.1.4.5 Other Chemicals

While the evidence from experimental animals has shown that certain chemicals and drugs can induce mesothelioma (Kurakawa et al 1983; Okada et al 1989) there is, to date, no systematic evidence of such effects in humans. Beryllium has also been suggested (Oels et al 1971).

UPDATE:

A review of Potassium Bromate by Kurokawa et al (1990) showed statistically significant increases in peritoneal mesotheliomas in male F344 rats given 500 ppm of postassium bromate in drinking water for various periods. Donna et al (1991) showed that 2, 6-Dichloronbenzonitrile (Dichlobenil), a principle in a commercial herbicide, both by intraperitoneal and subcutanesous routes induced a significant increase in tumours including a small number of mesotheliomas.

7.1.4.6 Other Factors

a. Smoking:

Smoking does not appear to increase the risk of mesothelioma.

b. Diet:

A recent case-control study has suggested that a low vegetable diet might influence mesothelioma occurrence (Schiffman et al 1988).

c. Genetic Predisposition:

It has been suggested that certain familial cases in which siblings or parents and children in the same family get mesothelioma might be genetically linked (Risberg et al 1980; Martennsson et al 1984). The early childhood cases may fit this category. In practice, however, the close interaction of genetic and environmental factors makes it very difficult to separate them.

d. Viruses

In his review of non-asbestos mesothelioma, Peterson et al (1984) noted that an avian virus had been shown to induce mesotheliomas in chickens which were similar to those in mammals. To date, there is no comparable human evidence.

e. Non-asbestos exposed mesothelioma cases

There is a certain consistency in the epidemiological literature that clear evidence of asbestos exposure cannot be established for 25-30% of cases. Peterson et al (1984) have reviewed this problem and quote a range (0-87%) without documented asbestos exposure. In most countries where women have not been a major part of the workforce, the rates of mesothelioma in women have remained reasonably constant at the 1-2 per million population "background" rate. These might represent genetically controlled or background environmentally induced rates (Hirsch et al 1982; McDonald & McDonald 1986; Huncharek 1989; Peto et al 1981).

UPDATE:

a. Smoking:

There is still no evidence to suggest that smoking is important. The possibility that certain cigarettes with a crocidolite filter could have a role has neither been demonstrated nor ruled out.

b. Diet:

A Vitamin A based cancer prevention program (β-carotene or retinol) has been carried out on former crocidolite miners in Australia. Program participants had signifanctly lower mortality than nonparticipants, but the rates of the two groups converged with time (Musk et al 1998). The relative rate of mesothelioma in those on retinol compared to β-carotene was 0.24 (95% CI, 0.07 - 0.86), a significantly lower rate of mesothelioma (deKlerk et al 1998).

c. Genetic Predisposition:

While genetic predisposition continues to be suggested, the difficulties of separating environmental from genetic effects remain. However, this may be changed with the ongoing rapid developments in genetics.

d. Viruses

In the 1992 report the review of non-asbestos mesothelioma by Peterson et al (1984) was mentioned noting that an avian virus had been shown to induce mesotheliomas in chickens which were similar to those in mammals. More recently England et al (1991) inoculated chickens with a v-src-positive DNA fragments and found a peritoneal based tumour which was diffuse mesothelioma.

An important development since 1992 has been the finding of fragments of the DNA from SV40 in mesothelioma tissues. SV40 is a virus which contaminated some of the polio vaccines used in the late 1950's early 1960's.

The finding of SV40-like DNA sequences in human mesotheliomas (Carbone et al 1994) and the induction of mesotheliomas in hamsters by injection of wild type SV40 into the pleura or peritoneum (Cicala et al 1993) raised the possibility that the SV40 virus might act as an independent carcinogen or co-carcinogen with asbestos. When the SV40 was injected in the pleural space, pericardial and pleural tumours identified as mesotheliomas were observed in 100 percent of Syrian hamsters. To date, there is as yet no comparable evidence in humans.

Since the early 1990's, SV40 DNA sequences have been reported in a substantial portion of British mesotheliomas (Gibbs et al 1998; Pepper et al 1996; Stenton 1997), in 5 of 7 asbestos associated mesotheliomas in New Zealand (Mayall et al 1999) but in none of the non-asbestos associated mesotheliomas. Pepper et al (1996) suggested that vaccination with live SV40 through contaminated polio vaccines given between 1959 and 1961 might be a factor in the continuing rise in the incidence of malignant mesothelioma in some countries.

Hirvonen et al (1999) failed to find SV40 in Finnish mesothelioma specimens. This may be related to the fact that the Finnish poliovaccines were not contaminated with SV40 or other presently unknown factors (Carbone et al 1999).

In Belgium, the SV40 Ltag-like DNA sequences were found, but they did not detect nuclear expression of the viral oncoprotein which they concluded made a pathogenic role of SV40-tag in mesothelioma carcinogenesis questionable (Dhaene et al 1999). However, they intend to repeat their study.

In France, Galateau-Salle (1998) reported sequences related to SV40 Large T antigen (Tag) in 28.6% of bronchopulmonary carcinomas, 47.6% of mesotheliomas and 16% of cases with non-neoplastic and pulmonary disease. While the difference between pulmonary cancer and mesothelioma findings were not significant, the difference between mesotheliomas and non-malignant cases was statistically significant.

Testa et al (1998) reported on the results of a multi-institutional study which confirmed the presence and expression of Simian Virus 40 in human mesotheliomas with 10 of 12 (83 %) of human mesotheliomas tested revealing SV40 sequences and SV40 large T antigen expression. Electron

microscopy demonstrated variable amounts of asbestos fibres in 5 (71%) of 7 lung tissues available for analysis. Based on their work it has been suggested that the tumerogenic potential of SV40 Tag in some human mesotheliomas may arise from its ability to interact with and thereby inactivate several tumour and/or growth suppressive proteins in cooperation with asbestos fibres in inducing pleural mesotheliomas (Mutti et al 1998b, Matker et al 1998). Stenton (1997) concluded that there is circumstantial evidence which suggests that SV40 may act as a cofactor with asbestos in inducing human mesotheliomas.

The source of the SV40 human infections has not been completely identified even though the administration from 1957-1965 of SV40 contaminated coat or vaccine is highly suspected (Pass et al 1996; Mutti et al 1998a). Mutti et al (1998a) reported that horizontal infection by sexual transmission has also been hypothesized.

To date, only one epidemiological study has been undertaken (Strickler et al 1998). They carried out a retrospective cohort study using data from the Surveillance Epidemiology and End Results program (1973-1993) and the Connecticut Cancer registry (1950-1969) as well as national mortality statistics (1947-1973). They assumed that the cohorts who were likely to have received SV40 contaminated polio virus vaccine as infants were born in 1956 through 1962. Those who received it as children were born in 1947 through 1952. They assumed that persons born 1964 through 1969 would not have received the vaccine. After more than 30 years of follow-up Strickler et al concluded that exposure to SV40 contaminated polio virus vaccine was not associated with a significant increase in mesothelioma rates. This study did not take into account exposure to asbestos. The Relative risks for the cohort exposed as infants was 3.0 (95% CI - 0.67-13.11) and as children was 2.45 (95% CI - 0.50=12.03). However they recommended that as the exposed cohorts mature it is important to continue monitoring cancer risks, noting that the birth cohorts studied had not yet reached the age at which most mesotheliomas occur "resulting in few cases (71) and imprecise estimates of risk".

In a very recent paper, Carbone et al (1999) summarized the molecular and epidemiological issues. The current situation was sumarized as follows:

- SV40 was introduced into a significant portion of the human population between 1955 and 1963 through polio vaccines and adenovaccines contaminated with the virus.

- By 1961, 80-90% of all US children under the age of 20 had received at least one polio vaccination potentially contaminated with SV40. During the 9 year distribution, it is estimated that 98 million people, children and adults may have been injected with an SV contaminated vaccine in the US.

- In 1960, Sweet & Hillman demonstrated that SV40 contaminated both the Salk and Sabin poliovaccines which were prepared in the kidneys of rhesus monkeys.

- In 1962, Eddy et al showed that rhesus monkey cells when injected into hamsters caused sarcomas.

- By 1964, SV40 had been shown to be capable of infecting and transforming hamster, rodent and human cells and were capable of human growth when injected subcutaneously in terminally ill human volunteers.

- Short term studies and one long term epidemiological study of 1073 children (up to 19 years of follow-up) did not suggest that the virus was oncogenic in humans. (A 22 year follow-up study in Germany involving mainly oral polio vaccine was also negative).

- In the study by Strickler, direct exposure should not be considered as the only source of SV40 infection as it appears that the virus can infect and spread in human populations.

Clearly there is adequate information to question a role for SV40 in the occurrence of mesothelioma. However, it is premature to reach any conclusions as to whether SV40 has caused mesothelioma in asbestos exposed or non-asbestos exposed persons. However a role for the SV40 virus in mesothelioma causation in humans cannot, on present evidence, be ruled out.

e. Non asbestos exposed mesothelioma cases

The data remain consistent, that in most countries where women have not been a major part of the workforce, the rates of mesothelioma in women have remained reasonably constant at the 1-2 per million population "background" rate. This was discussed in Section 7.1.4.1.

7.2 MECHANISTIC CONSIDERATIONS

Experimental work in animals and other biological systems are consistent with fibre length and diameter being important in both the ability of fibres to produce experimental fibrosis and to produce mesothelial tumours when introduced directly to the pleural surface.

Experiments where "long" asbestos fibres were placed on the pleura, produced mesothelioma from all fibre types (Wagner & Berry 1973; Stanton 1973), but experience in humans has shown no clear high risk of mesothelioma for workers exposed to chrysotile asbestos fibres. While the reasons for such differences remain conjecture, a convincing argument is that in experimental animals, with a lifespan of approximately 2 years, the lung protection / removal mechanisms have less than 2 years over which to act. Thus if solubility of the fibre or removal mechanisms are important in reducing the number of fibres at a site, the time over which these mechanisms can act is truncated. Although the processes of tumour development occur within the lifetime of the animal, exposure levels in animals tend to be high and might further impede fibre removal mechanisms. Moreover, experiments where fibres are placed directly on the pleura exclude consideration of the mechanisms impeding

fibres from reaching the pleura under normal circumstances. In humans, time frames are measured in tens of years, which provides the fibres with the opportunity to be dissolved or removed from the lung.

In spite of the apparent difference between the human and animal experience, experimental data are never-the-less useful because they provide a possible mechanistic perspective on the importance of fibre shape and dimensions. Studies by Stanton & Wrench (1972); Stanton (1973), Stanton et al (1977) and Stanton & Layard (1978) showed that fibres greater than 8um in length and less than 0.25 um in diameter had a higher probability of producing tumours than did shorter and larger diameter fibres. They also demonstrated that reducing the length of fibres by pulverization decreases the carcinogenicity as far as mesothelial tumour production is concerned. They concluded that pulverized blue asbestos of length less than 1.25-3.75 um could be discounted in mesothelioma production.

Demonstrating that glass fibres and aluminium oxide fibres produced mesothelioma when in contact with the pleura suggested that mesothelial tumour carcinogenicity was linked primarily to length, diameter and, possibly, durability.

UPDATE:

Reaching the pleura:

The mechanism by which fibres reach the pleura (Nishimura & Broaddus 1998) or even whether they need to reach the pleura is still not known.

A possibly important finding was the identification of "black spots" on the parietal pleura which contained anthracotic pigment and high concentrations of amphibole fibres compared to normal pleura [Boutin et al 1996]. They suggested that this could explain why the parietal pleura is the target for plaques and mesothelioma

Fibre size:

The British Health & Safety Executive (Meldrum 1996) concluded that there is good evidence that longer fibres are more toxic than equal masses of shorter fibres of the same composition. They noted that results from intraperitoneal (IP) and inhalation studies in rats with long and short fibre amosite suggests that short fibres (<5um) pose little if any concern for disease development at any site.

Donaldson & Golyasnya (1995) showed that chromosomal abnormalities were caused by long fibres but not by short ones. They concluded that fibres with length of at least 10 to 15 micrometres are necessary to induce disease in the pulmonary parenchyma, but shorter fibres in the region of 8 to 10 micrometres can in their view cause mesothelioma.

It appears that short fibres of chrysotile have been reported on the pleura or in tumours on the pleura. This finding has little relevance, as it is not known whether the fibres arrived before or after tumour

growth was initiated and the evidence shows that short fibres do not induce mesotheliomas.

Biopersistence:

Fibre biopersistence is an important parameter in the evaluation of the fibrogenicity and carcinogenicity of fibres. It is now well established that the removal of fibres from the lung involves two phases. The first, is the rapid phase involving macrophage clearance of fibres from the lung. In this phase, fibre length seems to be the most important parameter. A second phase involves the removal of the longer fibres from the lung. In this phase, fibre length and fibre durability or dissolution are dominant.

It has been shown that when biopersistence is measured in rats following the inhalation of synthetic mineral fibres, clearance of the size range greater than 5μm in length (WHO fibres), fails to differentiate between the different fibre types. However, when the bioperistence of fibres longer than 20μm is examined, differences in the behaviour of the fibres become very apparent (Bernstein 1995,1996). In the rat, there was an important difference in the removal of long fibres >20μm compared to short ones (<20 μm). Measurement of the biopersistence of man-made fibres is easier than for the asbestos minerals, in particular chrysotile, as the latter breaks into fibrils increasing the fibre number concentration with time. An important factor in such experiments is the length distribution of the originally deposited fibres.

The half-times in days for crocidolite of various fibre length in rats (Bernstein et al 1996) has been reported in TABLE U8:

TABLE U8.

BIOPERSISTENCE OF CROCIDOLITE

Half-times for Crocidolite in days
Fibre Length

L<5μm			L5-20μm			L>20μm		
T½-1	T½-2	w-T½	T½-1	T½-2	w-T½	T½-1	T½-2	w-T½
2.7	430	172	1	442	262	1.9	806	536

-1 = half-time fast clearance phase; $T_{\frac{1}{2}}$-2 = half-time slow clearance phase; W-T½ = half-time combined weighted clearance.

As far as chrysotile is concerned, Coin et al (1992; 1994) reported that fibres longer than 16μm were cleared slowly, if at all. They estimated, a pulmonary retention half-time for fibres longer than 16μm of 114 days. They did not see transportation of chrysotile fibres from the central regions of the lung toward the sub-pleural regions. Caution is needed in interpreting these findings as the size of a rat macrophage is less than that in humans and a rat's life span is considerably less. Oberdorster (1994) noted that fibres up to 8μm in length were cleared very rapidly with pulmonary retention times

ranging 8-30 days. He concluded that dissolution is a very important clearance mechanism in the rapid disappearance of pulmonary chrysotile fibres. Assuming that all mammals behave similarly, Oberdorster (1996), ignoring sequestration of fibres, estimated that the overall retention half-time for chrysotile in a primate would be about 105 days.

Searl (1997) also found the rapid removal of short chrysotile fibres in rats but retention of long fibres. Unfortunately in none of these experiments were the fibres retained in the lung tissue analyzed to determine if they were tremolite not chrysotile fibres. However, Wagner (1974), when he looked, did find tremolite in the lungs of exposed animals.

Churg & Wright (1994) noted that very little information is available on actual fibre clearance rates from human lungs. They indicated that for amosite and crocidolite, the estimated clearance half-times are measured in years to decades whereas the data that exist suggest that the vast majority of chrysotile fibres are cleared within months although some fibres may be sequestered and cleared very slowly.

Short Coalinga amphibole free chrysotile fibres were found not to produce fibrosis or tumours and to have low biopersistence (Ilgren and Chatfield 1997, 1998a, 1998b). That study produced no mesotheliomas.

Recently, an experiment was conducted with "pure" chrysotile from Brazil. The same method as used in synthetic mineral fibre tests was used. The study found that fibres longer than 20µm chrysotile had a half-time of about 1 1/3 days (Bernstein 1999).

It is known that the solubility of fibres depends on whether they are inside or outside the cells as the ph differs. In vitro experiments of fibre durability have been undertaken to see if in-vitro fibre durability predicts biopersistence which it seemed to do for six synthetic mineral fibres and amosite fibres longer than 20µm (Searl et al 1999).

McDonald (1998) noted that a small number of well controlled studies using lung burden analyses have shown that amphibole fibres differ from chrysotile in as far as being more durable and having greater biopersistence in lung tissue.

Boffetta (1994) concluded that there are few data available concerning biopersistence in humans. He notes that studies analyzing asbestos lung burden in workers following cessation of exposure suggest a decreasing concentration over time that is independent of duration of exposure. However, the data in his paper suggests that this finding applies more closely to Canadian miners and Norwegian asbestos cement workers than to amosite workers.

A review of the situation for man-made fibres concluded: " Intraperitoneal injection studies showed clear dose-response relationships with many fibres and differences between their carcinogenic potency. These results support the concept that the durability of fibres in the body is an important factor in the production of fibre-related cancer" (Pott & Roller 1996).

In summary, the data are all consistent with long biopersistent fibres being considerably more important in disease causation than short or non-biopersistent fibres.

Tissue burden

The importance of tissue burden studies has been outlined by Becklake & Case (1994). McDonald (1998) concludes that research over the past forty years, demonstrates with increasing clarity that amphibole asbestos fibres-crocidolite, amosite and tremolite are more carcinogenic than chrysotile.

It is evident that without tissue analyses, potentially important exposures would go undetected. This is very important in the search for etiology. For example, a Jewelry worker with a mesothelioma was assumed to have been exposed to chrysotile only until it was found through tissue analysis that he was exposed to amosite (Kern et al 1992). Gibbs et al (1994) demonstrated the importance of tissue analyses for confirming past exposure.

There are numerous reports describing the tissue content of fibres in cases occupationally exposed to asbestos and in cases of asbestosis, lung cancer, and mesothelioma (Nolan et al 1994, Paoletti et al 1993; Langer & Nolan 1994; Murai & Kittagawa 1992; Tossavainen et al 1994; Sakai et al 1993; Dufresne et al 1995; Roggli 1995).

The importance of tissue analyses in establishing etiology depends on the use of appropriate controls or referents. For example workers are likely to have in their lungs, the fibres to which they are exposed, regardless of whether or not those fibres are related to health outcome. Inferences about association can only be made when the exposures of cases are compared to those of referents of similar age and circumstance who do not have mesothelioma or the other dieseases under investigation (McDonald 1994).

Many studies have reported ferruginous body concentrations in lung tissues. Criteria for evaluating tissue and ferruginous body burden study results have been reported (Helsinki Criteria Document 1997; Gibbs 1993).

UICC samples.

Frank (1995) claims that a study of the UICC chrysotile asbestos used in many animal experiments did not contain any tremolite. In fact tremolite was only found in the Quebec mines and mills by analysis of the fibres present in the lungs of miners. Few investigators have examined the fibre types in the lungs of the experimental animals after exposure.

7.3 LEVEL OF EXPOSURE

There is epidemiological evidence that the risk of mesothelioma is dose related (Newhouse et al 1985 and Newhouse & Berry 1979). See TABLE 8. In studies where exposure has not been quantitative, duration of employment has often been used as a surrogate. For example, in the British textile

industry there was no case of mesothelioma in men with less than 6 years of employment (Peto et al 1985) compared to 33 such cases where individuals were exposed for more than 6 years with 23 of the cases exposed for 20 years or more. The existence of exposure-response relationships has been strengthened by measurements of fibres in tissues where progressive increases in relative risk have been observed with increasing fibre content.

TABLE 8. MESOTHELIOMA -EVIDENCE OF EXPOSURE-RESPONSE.
Newhouse & Berry (1979)[22]

Exposure category	Number of cases	Rate / 100,000 s.yrs[23]
Low- Moderate[24]		
<2	4	33
>2	7	93
Severe[24]		
<2	16	104
>2	19	243

Rogers et al (1991) found relative risks to increase more rapidly with concentrations of fibre in lung tissue greater than 3×10^5 fibres /g by light microscopy and 10^6-$10^{6.5}$ fibres /g by transmission electron microscopy. Rogers et al (1991) used data from various studies, evaluated by Berry to illustrate how the risk of mesothelioma increased with the concentration of fibres in lung tissue. He did this by calculating odds ratios[25] which are shown in TABLE 9. These show that when the odds of mesothelioma occurrence is set at 1 for concentrations of less than 10^6 fibres/g of tissue, the odds of mesothelioma occurring for concentrations greater than 10^6 fibres/g of lung tissue are consistently greater than 1. This shows that the risk of mesothelioma increases with increasing exposure.

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[22] Data shown for men only.

[23] s.yrs = Subject or person years of exposure.

[24] The categories of exposure <2 and >2 represent less than or more than 2 years of exposure. The "low-moderate" exposure category was "probably 5-10 fibres/ml. The "severe" exposure category included concentrations which before 1945 averaged 20 fibres/ml or higher.

[25] An "odds ratio" is the ratio of the "odds" of disease occurrence in the exposed and non-exposed subgrt..ps.

TABLE 9. MESOTHELIOMA ODDS RATIOS IN RELATION TO LUNG TISSUE FIBRE CONTENTS RE-CALCULATED FROM VARIOUS STUDIES BY BERRY
From Rogers et al (1991)

		Concentration[26] $< 10^6$fibres/g	Concentration $>10^6$fibres/g
Berry et al 1989[27]	cr.a	1	3.8 (1.8- 8.0)
Berry et al 1989[28]	cr.a	1	7.4 (3.5-16.0)
Mowe et al 1985		1	8.5 (2.3-31.0)

In the Quebec chrysotile mines and mills the relative risk of mesothelioma increased with cumulative exposure. The case with the lowest cumulative exposure had 8.8 years of employment at a concentration of 6.7 fibres/ml (Liddell 1989). The mesotheliomas in the Quebec chrysotile mines and mills were also highly correlated with the concentrations of tremolite >8um long in their lungs as described earlier in this report (McDonald et al 1989).

UPDATE:

There has been no new evidence to suggest that mesothelioma risk is not exposure dependent.

Roggli (1995) found a significant correlation between duration of exposure and asbestos bodies as measured by light microscopy and between the duration of exposure and total uncoated fibres of 5 μm or longer as well as commercial amphiboles as determined by scanning electron microscopy.

In a US series of mesotheliomas, Dodson et al (1997) found that 46 of 55 individuals with a pathological diagnosis of mesothelioma had ferruginous bodies at concentrations of more than 1000 per gram dry lung tissue. The majority of the cores contained amosite. Roggli et al (1993) identified amosite in 81 percent of cases which accounted for 58 percent of all fibres of five micrometres or greater in length. Hughes & Weill (1994) pointed out, quite correctly, that by not including controls, the Roggli et al study cannot be used to deduce the relative importance or potencies of different fibre types.

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[26] < means less than or equal to stated value; > means more than stated value.

[27] recalculated data from McDonald et al 1982.

[28] recalculated data from Wagner et al 1982.

In a study of asbestos cement workers, exposed to chrysotile asbestos and small amounts of amphibole, the exposed workers with mesothelioma had a significantly higher total content of asbestos fibres in their lungs than those without mesothelioma who in turn had higher concentrations than controls. Chrysotile was the major type of fibre identified. However the difference between the various groups (exposed with mesotheliomas, exposed without mesotheliomas and controls) was most pronounced for the amphibole fibre (62, 4.7, 0.15 f/g) especially crocidolite (54, 1.8, <0.001 f/g) but were evident also for tremolite (2.9, <0.001, <0.001 f/g) and anthophyllite (1.7, <0.001, <0,001 f/g). For amosite, there was no statistically significant difference between the lung burdens from workers with and without mesothelioma. Fibrosis was correlated with tremolite but not with chrysotile in the lungs. (Albin et al 1990)

Churg et al (1993) determined the fibre content of the lungs of 94 long-term chrysotile miners & millers from the region of Thetford Mines. They found that mesothelioma, airway fibrosis, and asbestosis were strongly associated with a high tremolite fibre concentration, whereas pleural plaques and carcinoma of the lung showed no relationship to tremolite burden.

It is important to point out that in the Quebec chrysotile mining industry there was no case of mesothelioma in men employed for less than 2 years.

Studies of fibre in the tissues of the general population case control series have consistently shown higher amphibole concentrations in the lungs of mesothelioma cases compared to controls.

7.3.1 Neighbourhood Exposure

Another possible indirect measure of exposure is distance from a source. In this regard, the absence of high rates of mesothelioma in persons living near asbestos factories would be supportive of zero or very low risks at low levels of exposure, neighbourhood exposures being presumably less than those in the workplace. In fact, the only evidence of neighbourhood exposure increasing risk has been in cases where large quantities of blue asbestos was involved (Wagner et al 1960, Newhouse et al 1985). Other studies in Canada and the USA have not shown such an association (McDonald & McDonald 1980; Hammond et al 1979a). These latter studies were concerned with chrysotile and amosite respectively.

Quebec chrysotile mining and milling communities were included in the mesothelioma survey conducted by McDonald & McDonald (1980). There, even in the 1970's, airborne fibre concentrations[29] ranged 160-11000 ng/m^3 . These concentrations were orders of magnitude greater than the 0.1-100 ng/m^3,reported in other rural and urban areas (Gibbs et al 1980). While subject to many errors, the mean fibre concentrations at the "dustiest" site in these communities as measured by phase contrast microscopy was about 0.015 fibres/ml[30].

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[29] The concentrations were determined using transmission electron microscopy. Because fibres are disrupted in the preparation procedures, concentrations are expressed on a mass basis as ng/m^3. One ng = 1x10^{-9}g.

[30] This is the mean value for the results of 14 samples counted using two different phase contrast membrane filter mounting and counting methods (NIOSH and BOHS) and 4 counters. The main sources of error are particle overlap which would likely lead to underestimation of fibre concentration and lack of fibre differentiation which would overestimate the concentration.

UPDATE:

An understanding of the possible impact of neighbourhood exposures is not possible without an understanding of background rates of mesothelioma.

Background rates of mesothelioma

McDonald and McDonald (1993, 1996) have reviewed this question. They provided as evidence for the existence of background rates:

1. High rates of mesothelioma have occurred in certain villages in Turkey related to the natural occurrence of fibrous erionite.

2. Deposits of fibrous erionite are widespread on the earth and there is some suggestion of erionite related cases in proximity to deposits in the Rocky Mountains States of North America (Enterline and Henderson 1987).

3. The occurrence of mesothelioma in childhood unless the usual latency with asbestos related cases is shorter in childhood (Fraire et al 1988).

4. In tissue burden studies there have been a certain proportion of mesothelioma cases that cannot be attributed to asbestos exposure.

5. Backward extrapolation of trends to before the divergence in rates between men and women occurred indicates a mortality rates of about 1 to 2 per million population per annum in both sexes.

6. There are studies where there remains a low mesothelioma rate after exclusion of occupationally related cases.

7. There were mesotheliomas diagnosed prior to the commercial exploitation of asbestos.

Neighbourhood exposure

As reported before, the only evidence of neighbourhood exposures increasing mesothelioma risk has been where large quantities of blue asbestos was involved. Further evidence of risks for residents possibly confounded by domestic exposures, has been reported in the blue asbestos mining town of Wittenoom Australia (Hansen et al 1993, 1998).

The possibility of environmental exposure could not be excluded in five cases of mesothelioma, who did not have occupational exposure, four of whom lived within one half mile of a factory in Leeds using blue asbestos to manufacture asbestos mattresses for the insulation of steam boilers (Arblaster et al 1995). In another UK study in Calderdale, neighbourhood exposure cases again associated with

crocidolite were also identified (Edward et al 1996).

Mesotheliomas have been reported in persons possibly exposed to a natural background of fibres throughout childhood with tremolite fibres considered most likely to be responsible (Rey et al 1993). In Greece malignant mesotheliomas have occurred in persons using a material containing tremolite to white wash buildings. (Constantopoulos et al 1991; Sakellariou et al 1996). A study in Italy claims an excess of mesothelioma in persons living near a crocidolite using factory, but that study did not take adequate account of other possible occupational exposures (Magnani et al 1991; Browne 1991).

In a study of persons living in Manville, New Jersey where a major asbestos manufacturing facility which used chrysotile and amphibole asbestos was located, Berry (1997) suggests an increased rate of mesothelioma in residents due to past exposures to asbestos. However the possibility of other occupational exposures has not been adequately taken into account to conclude that the mesotheliomas are environmentally induced.

A study of persons living in the vicinity of chrysotile asbestos mines and mills in the province of Quebec Canada has been reported (Camus et al 1998). Seven pleural cancers were reported. It is not known whether these were mesotheliomas or if they were, whether they had occupational exposure. Churg (1998) observed high concentrations of amosite and crocidolite asbestos in the lungs from three women, from this region, who died of mesothelioma between 1983 and 1988. They had not worked in the mines and mills but had worked in an asbestos bag repair operation. This suggests that other exposures are important. Case (1998) noted that this mortality rate (seven cases) could be entirely explained by a few cases among women in the area who had occupational exposure to amphiboles in the manufacture of gas masks, the repair of burlap bags which contained imported fibre and possibly in one case, tremolite exposure brought home on a miner's clothes. He reported that seven such women received workers' compensation in Quebec during the period of the study by Camus et al. In view of the experience of miners and millers exposed in the community as well as being exposed to concentrations many orders of magnitude above that of the general population this seems to be the most logical explanation for these cases.

In summary there have been no studies indicating an increase in the risk of environmentally induced mesothelioma for persons living in the vicinity of chrysotile only using plants.

7.3.2 Household Exposure

There have been instances where it appears that household exposure from living with a person who is occupationally exposed to asbestos may have been involved in increases in mesothelioma risks, with cases in case-control studies reporting more domestic contact than controls. (McDonald & McDonald 1980; Vianna & Polan 1978; Lieben & Pistawa 1967).

The studies by McDonald & McDonald (1980) involved chrysotile (and tremolite contamination if present), other mine dusts and dusts from a factory where blue asbestos was also used. Fibre types

were not identified by Vianna & Polan (1978) but the occupations of household contacts included, brake-lining worker, pipe fitter, heat insulation worker, heat electric-wire worker, elevator insulation worker and electric wire insulation worker. Lieben & Pistawa (1967) noted that the asbestos fibre types to which mesothelioma cases may have been exposed by family contacts in the three cases were: chrysotile and amosite; an unidentified fibre used in an insulation plant; and, unidentified fibres used in insulation work in a shipyard.

The levels of fibre exposures associated with the household contacts were not described in any of the reports. However radiological changes similar to those found in asbestos workers have been described in persons living in the same household as amosite asbestos workers (Anderson et al 1979). Such radiological changes in workers have usually involved long term exposures at levels in excess of occupational exposure limits of 1-2 fibres per ml. Thus, it is likely that past household exposures, may have been quite high.

UPDATE:

Valic and Beritic-Stahujak (1993) drew attention to the fact that Nicholson et al (1980) had measured concentrations in the homes of miners and non-miners in a chrysotile mining community. On a mass basis they found that concentrations were much higher in the former, where 61% of samples were between 100 and 2,000 ng/m3 compared to the latter (32, 45 and 65 ng/m3).

While there continue to be cases of domestic exposure reported (eg: Schneider et al 1996), there are no findings which alter the view that household exposures were high.

The fact that mesothelioma incidence in females in North America and Western Europe has shown little or no increase during the last 20 to 30 years suggests that neither household or environmental exposures have increased mesothelioma risks significantly. IPCS (1998) reported "In several case-control studies reviewed therein "(Referring to an earlier IPCS Criteria Document 53)," there were more mesothelioma cases with household exposure than in controls, after exclusion of occupation. However for most of these investigations, it is not possible to distinguish the form of asbestos to which household contacts were exposed on the basis of the information included in the published reports". It is clear that this is very important as shown in Quebec.

7.4 LATENCY

There was no mesothelioma death in the Quebec chrysotile mines and mills below 20 years from first exposure (McDonald 1980). Peto (1980) found no mesotheliomas in the British textile industry under 20 years from first exposure. While the latter used predominantly chrysotile, some amphibole (blue asbestos) was used at the plant. Overall, the literature would appear to support the statement by Browne (1983) that in asbestos related cases the onset of the illness is usually 35-40 years after first exposure, rarely less than 20 years and perhaps never less than 15 years.

UPDATE:

The Helsinki criteria document (Consensus report 1997) states that: "a minimum of 10 years from first exposure is required to attribute the mesothelioma to asbestos exposure, though in most cases the latency is longer (e.g. on the order of 30 to 40 years).

In the Quebec chrysotile mining and milling industry there was only 1 case with a period from first exposure to death of less than 30 years and the latency period ranged up to 60 years (McDonald et al 1997).

In 1992, Lanphear & Buncher (1992) reviewed 21 articles that documented the latency periods for 1690 malignant mesothelioma. Of these 1105 (65%) fulfilled strict histologic and exposure criteria. Of these, 99% had a latency of more than 15 years, 96% had a latent period of at least 20 years. They calculated that the probability of a mesothelioma occurring within a decade of the first exposure was 0.0000, 10-14 years after first exposure was 0.0045, between 15 and 19 years was 0.0317 and after more than 20 years from first exposure was 0.9638. The estimated median latent period was at least 32 years after initial exposure. They did not analyze the results by fibre type or distinguish between pleural and peritoneal tumours.

As was pointed out by Peto et al (1982) and discussed later in this report, the major predictor of risk of mesothelioma appears to be period from first exposure. Mathematically, this can be expressed as $p = b.t^k$ where p = incidence in any one year, b is a constant, t is time since first exposure and k is an exponential between 3 and 4. Morgan (1991) argued that this could be used to work backwards from disease to probable cause. Based on this argument and assuming that exposure to asbestos is the cause of the mesothelioma, he adapted the model to

$$p_o + \sum_{i=1}^{n} p_i = 1$$

where p_o = background if it is assumed that the disease in this instance is caused by asbestos, and not background, the model becomes

$$\sum_{i=1}^{n} p_i = 1$$

where $p_i = t_i^k / \Sigma t^k$

As an example of an application, Morgan considered a former insulator with mesothelioma diagnosed in 1988, who was exposed to asbestos from 1942 through 1944 and again in 1950. The table provides the data that allows the conclusion that for this person there was an 84.4% likelihood that his 1942 through 1944 job caused the disease, and a 15.6% likelihood for the 1950 exposure.

Probability of Each Year as Cause

Exposure	Time (t)*	$t^{3.5}$	Casual Probability $t^{3.5}/\Sigma t^{3.5}$
1942	46	660,165	.303
1943	45	611,285	.281
1944	44	565,047	.260
1950	38	338,254	.156
Total		2,174,751	

* t = time in years since first exposure.

The causal probability = $t_i^{3.5} / \sum t_i^{3.5}$. Using this relationship, the causal likelihoods could be calculated. Peto showed that this equation (actually t^4) was approximately correct for exposure durations of up to 10 years. While the model is appealing it has several weaknesses. It assumes that asbestos exposure is the cause and does not take fibre type or exposure levels or industry sector into account.

A residence time model used by Hughes was described later in the 1992 report in which Incidence I at time t = KC $(t^{3.2} - (t-d)^{3.2})$ where t = time since first exposure and d = duration of exposure, C is the concentration and K is a constant depending on the process. Various models approximating a cubic model have been employed in various studies. It was noted by Hillerdahl (1999) that even in the most heavily exposed cohorts only a proportion of the cohort dies of mesothelioma so the formula is not applicable in all cases. Berry (1995) refined this model in which the increase in risk with time is attenuated by a factor representing a decline in risk corresponding to elimination of asbestos from the lungs. He further introduced a lag period to allow the tumour time to develop. This was shown to apply in experiments in which rats were inoculated with fibres. The new formula became:
$I = ce^{-z}(t-w)^k$ where I = incidence, c=cumulative exposure, z = elimination rate, t = time since first exposure, w=lag period and k is a constant.

Berry applied this formula to residents and visitors to the Wittenoom crocidolite mining area. With the elimination factor, incidence was relatively stable from 40-60 years after first exposure in contrast to the risks without fibre elimination. It assumes a linear non-threshold model. The lifetime risks of mesothelioma based on this model for residents estimated to be exposed continuously for 10 years to 0.001 f/ml from age 25 was 9/million. This was an estimate for crocidolite. The estimate for visitors exposed for 1 week at 0.01f/ml at age 20 was 0.27 - 1.6 per million. If there is a background of 2 per million per year, the lifetime risk is about 140 or 200 according to HEI-AR (1991).

If chrysotile itself can cause mesotheliomas in humans, its potency is at least 1/40th of that of crocidolite and may be zero. Extrapolating for Dum Dum, the lifetime risk for persons exposed continuously for 10 years to chrysotile at 0.001 f/cc from age 25 would be 9/40 per million = 0.225/million at most.

8.0 ASBESTOSIS

8.1 DEFINITION AND DIAGNOSIS

The definitions applied to asbestosis are many and highly variable although Harber & Smitherman (1991) found that over the last decade there was "stability of the criteria for asbestosis case definitions".

As damage to the tissues (fibrosis or scarring) can only strictly be observed at autopsy or biopsy, asbestosis should be a pathological diagnosis. In practice, the observation of signs and symptoms in life are used for "clinical diagnosis". Thus, "asbestosis" is usually a clinical term applied to fibrosis of the lungs which has been caused by asbestos. The diagnosis is a clinical opinion which takes into account a variety of observations such as changes on the chest radiograph, worker complaints of breathlessness, crepitations (noises in the lungs), certain lung function deficits and evidence of asbestos exposure. The simple knowledge that the person being examined has been exposed to asbestos is likely to introduce a bias into a clinical diagnosis.

There is probably little disagreement in the literature that high grades of certain radiological abnormalities reflecting scarring of the lung, combined with certain characteristic symptoms, pulmonary function deficits and clear evidence of long term exposure to high levels of asbestos might be called "asbestosis". However, minor radiological abnormalities or lung function changes may or may not be indicative of "asbestosis". The application of newer techniques in diagnosis, such as CAT and High Resolution CAT scans, positive gallium scans, exercise and other tests, has not improved the situation because the relationship between these techniques and the currently more accepted definitions of clinical asbestosis remains unclear.

It is for these reasons that the term "asbestosis" is rarely used in epidemiological studies. In most epidemiological studies the frequency of changes on a chest radiograph or frequency of symptoms etc., are treated independently and examined in relation to exposure. In some studies, combinations of changes to define asbestosis have been used but this definition has been for study purposes only.

UPDATE:

Browne (1994) reports that even in the 1990's, accurate statistics on the incidence and prevalence of asbestosis are bedeviled by problems of definition of both disease and exposure. Browne describes the parameters used to "diagnose" asbestosis for epidemiological purposes as radiographic change

(measured by ILO subcategories), pulmonary function deficits and presence of basal crackles. He points out that asbestosis possesses no pathognomonic, clinical, physiological or radiological features. Hence, given evidence of "occupational asbestos exposure" the diagnosis of asbestosis rests upon establishing clinical features and excluding diffuse interstitial pulmonary fibrosis due to other causes or diseases. Ferruginous bodies and/or radiological evidence of pleural plaques may support the likelihood of exposure but do not establish the existence of asbestosis.

The Helsinki document (Criteria document 1997) reports that the chest radiograph is still the basic tool for identifying asbestos related diseases such as asbestosis, pleural abnormalities, lung cancer and mesothelioma. They note that the limitations of the chest radiograph in the detection of asbestosis and asbestos associated pleural abnormalities are widely recognized and that computed tomography (CT) and high-resolution computed tomography (HRCT) can facilitate the detection of asbestosis and asbestos related pleural abnormalities but were not recommended as screening tools. They suggested that analysis of lung tissue for asbestos fibres and asbestos bodies could provide data to supplement the occupational history.

Some of the advantages and limitations of various methods of examining the pleura have been described by Muller (1993) and several others.

For clinical purposes the Helsinki Criteria document recommended that to identify persons with a high probability of past exposure to asbestos the results of tissue burden study results should be:

> "Over 0.1 million amphibole fibres (>5um in length per gram of dry lung tissue as measured by electron microscopy in a qualified laboratory OR
>
> Over 1000 asbestos bodies per gram of dry tissue (100 asbestos bodies per gram of wet lung tissue) OR
>
> Over 1 asbestos body per milliliter by bronchoalveolar lavage as measured by light microscopy in a qualified laboratory."

It was recommended that each laboratory establish its own reference values and that efforts be made to standardize the fibre burden analytical methods used by different laboratories.

The Helsinki document recommended that the term "pleural asbestosis" not be used and recognised that pleural plaques are often asymptomatic and without clinically important findings.

These conclusions are not unlike those of a workshop in Japan in 1991 (Gibbs et al 1993) where it was concluded that:

> "The finding of a lung parenchymal content of greater than 1000 ferruginous bodies/g dry lung is highly suggestive of occupational exposure to asbestos, as long as the value is obtained with standard methods in a laboratory with experience in the application of the technique."

The possibility that idiopathic pulmonary fibrosis cases might occur in asbestos-exposed workers has been reported (Gaensler et al 1991).

8.1.1 Radiological Changes

The chest radiograph may show changes. These include: lung markings which obscure the normal lung appearance; pleural thickening; pleural calcification; and possibly changes to the cardiac outline. These changes can be graded and an internationally agreed classification system exists for the reading of radiographs known as the ILO/UC Classification.

Interpreting these readings is not straightforward for the following reasons:

* There is almost always significant variation between even "expert" readers in terms of the existence or degree of radiological change. It is for this reason that scientific studies involve the review of films by multiple readers.

  An idea of the effect that this might have has been reported by Parker et al (1989). They reported that an initial reading of 566 radiographs found 30% of films to be classified as positive for pleural change. However, only 4% were considered positive by at least 2 out of 3 readers from a NIOSH panel reading radiographs under blind conditions[31].

* Readings by a single reader, especially when they have knowledge of exposure and other clinical information may be quite unreliable from the standpoint of obtaining objective information on which to establish etiological factors.

* Some radiological changes such as pleural calcification may be quite marked but have little or no importance in producing respiratory disability.

* As the radiological changes become less pronounced, there can be serious difficulties in the interpretation of results. Readings of the radiographs of persons with and without asbestos exposure may reveal the same types and degrees of abnormality. It is for this reason that, before reaching conclusions that there is a disease or indicator of a health effect in a group of workers, it is necessary to know that the frequency and degree of change is greater in those exposed than in a comparable group of non-exposed workers or that the degree of change and the incidence of such changes relate to exposure levels.

* Becklake (1991) notes that while the presence of small irregular opacities are usually considered important in the diagnosis of asbestosis (Canadian Thoracic Society 1979; American Thoracic Society 1986; Council report 1984) they are nonspecific for this condition

[31] "Blind" means without information on age, occupation, exposures, other clinical information etc

Fibrosis may be the result of exposure to other agents or to pathophysiological processes. Moreover, there is evidence that milder grades of radiological abnormality may be related to smoking or enhanced in asbestos exposed smokers (Hnizdo and Sluis-Cremer 1988; Rosenstock et al 1988; Lilis et al 1986; Weiss 1984; Blanc & Gamsu 1988; Blanc & Gamsu 1989; Weiss 1991).

UPDATE:

While there have been no major changes in the nature of radiological changes, the issue concerning the importance of pleural plaques remains. The relationship between pleural and parenchymal changes and lung cancer are discussed elsewhere. (See Section 9.1).

Pleural plaques

Sanden & Jarvholm (1991) did not find pleural plaques to be associated with mesothelioma in a prospective cohort study of 3893 insulation workers. Likewise in Canada definite pleural calcification was not associated with malignant mesotheliomas in the Quebec chrysotile miners (McDonald et al 1997). This absence of an association was originally noted by Gibbs (1972) and observed in Greece by Contantopoulos et al (1992). On the other hand, Hillerdal (1993, 1994) in a general population study in Sweden, reported that pleural plaques without asbestosis were associated with a slightly increased risk of lung cancer. However, in that study the fibre types and exposure levels were not known. The SMR for men with asbestosis was 2.3 and for men without asbestosis was 1.4.

A study of residents in Japan suggested that the prevalence of pleural plaques was associated with an increased concentration of anthophyllite fibres in the lung (Murai et al 1997). This is consistent with the findings many years ago of endemic pleural plaques associated with anthophylite exposure in Finland. Endemic pleural calcification has been reported too in Metsovo in North-west Greece, where persons white washed their homes with a material made from white rocks composed of tremolite fibres (Sakellariou et al 1996).

Areas were identified in other parts of Greece where similar materials containing tremolite were used as whitewash. Malignant pleural mesothelimoas have occured in some of these areas (Constantopoulos et al 1991).

8.1.2 Breathlessness

While breathlessness relates well to cumulative exposure in asbestos exposed workers, the symptom alone is non-specific.

UPDATE:

There have been no significant additions to the literature.

8.1.3 Lung Function Changes

While certain changes may correlate well with cumulative exposure to asbestos, not all tests are equally predictive. Some changes are completely non-specific and are influenced by other factors such as smoking, age, race etc.

UPDATE:

Symptoms and pulmonary function as measures of morbidity were reviewed by Becklake (1994).

8.1.4 Clinical observations

These are rarely used in epidemiological studies because of observer variation. However, certain well standardized observations may be useful (eg; presence of "rales" or "crepitations")[32].

In epidemiological studies the frequencies of certain measured outcomes in exposed workers are compared to those in non-exposed workers. The relationships between observations and levels of dust/fibre exposure are also examined to determine if the rate of adverse outcomes increases sensibly with exposure.

UPDATE:

There have been no significant additions to the literature.

8.1.5 Mechanistic considerations

In studies of fibre size in relation to fibrosis of the lung in experimental animals, Wright & Kuschner (1977) found that short fibres (glass and blue asbestos) less than 10 um in length did not produce lung fibrosis but that the long fibres did. The explanation for this would appear to be the removal of these shorter fibres from the lung. This and removal of chrysotile through solubility may also explain the observations by McDonald et al (1989) of different dose-response (radiographic outcome) curves for tremolite and chrysotile.

UPDATE:

The major new information available since 1990 relates to the importance of biopersistence which was discussed earlier.

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[32]Rales were proposed as one of the more sensitive indicators of "asbestosis" when the British Occupational standard for asbestos was published (BOHS 1968).

The fundamental finding is that the parameters which distinguish man-made fibres in terms of their carcinogenicity are fibre length, and fibre biopersistence. The dimension of fibres which distinguish the fibres on the basis of biopersistence are much longer than 5 μm (ie: > about 20 μm).

Churg et al (1990), based on tissue analyses of amosite exposed workers found that fibrosis grade was positively correlated with amosite concentration and negatively with mean fibre length parameters including fibre length, surface area and mass. When fibrosis grade vs. fibre burden was examined for amosite, chrysotile and tremolite, the relationships were statistically different. These findings indicate that the amosite concentrations like chrysotile and tremolite concentrations are closely and directly related to fibrosis at the local lung level. The concentration comparison data indicates that fibre for fibre amosite is more fibrogenic than chrysotile or tremolite and that the tremolite is more fibrogenic than chrysotile.

The apparent inconsistency in the results from these tissue analyses and the results from the animal studies which show that long fibres are more fibrogenic and carcinogenic than short fibres might be explained in several ways. Timbrell has suggested that fibrotic lungs retain more fibres than do non-fibrotic lungs. The bulk of airborne fibres are short fibres therefore the use of mean or median length may not accurately reflect the existence of potent very long fibres. In addition as long fibres break apart they are likely to increase the number of short fibres. Certainly in humans, short fibres are unlikely to be important generators of fibrosis because populations exposed to enormous numbers of short chrysotile fibres (eg: in mining and milling) do not show higher rates of asbestosis than those in industries where longer fibres are used (eg: in textiles).

Dufresne et al. (1996) found that lung cancers in workers with and without asbestosis from Asbestos Township had equal concentrations of retained fibres in the lungs but that those with lung cancer showed a tendency to a higher length to diameter ratio of amphiboles. They also had a 29 percent higher average cumulative smoking index.

They found that the geometric means (GM) concentrations were higher in cases than in the controls for chrysotile fibres 5 to 10 μm long in patients with asbestosis with or without lung cancer; for tremolite fibres 5 to 10 μm long in all patients; for crocidolite talc or anthophyllite fibres 5 to 10 μm long in patients with mesothelioma; for chrysotile and tremolite fibres greater or equal to 10 μm in length in patients with asbestosis; and crocidolite, talc or anthophyllite fibres longer than 10 μm in patients with mesothelioma.

Green et al (1997] studying textile workers reported a significant positive correlation between lifetime human exposure to asbestos and total lung burden of asbestos fibres. Fibrosis was correlated with both exposure to asbestos and concentration of asbestos fibres in the lung. The concentration of tremolite fibres in the lung provided a better estimate of lung fibrosis than did the concentration of chrysotile.

Mechanisms in the pathogenesis of asbestosis have been reviewed by Mossman and Churg (1998].

9.0 LUNG CANCER

The occurrence of lung cancer in certain age groups is not uncommon due to past smoking habits. In North America, almost one quarter of all cancer deaths are due to lung cancer.

The American Cancer Society Statistical report for 1978 (American Cancer Society 1978) reported the eventual risk of dying of lung cancer for white males 20 years of age in 1970 as 5.07%. Thus in a cohort of 100,000 men, approximately 5070 deaths from lung cancer would be predicted to occur if men were followed from age 20 to death.

Hughes (1989) noted a lifetime risk in 1982 of approximately 0.07 for a man dying of lung cancer. At this rate, approximately 7000 lung cancer deaths would be expected over the lifetime of a cohort of 100,000 men.

While the estimates of "background " may differ somewhat depending on the method of calculation and assumptions made, the numbers are quite large and it is against this background that the risks of lung cancer from asbestos exposure must be measured. The normal variation in background rates can make it very difficult to detect small increases in risk.

UPDATE:

Carcinoma of the lung associated with asbestos exposure has been reviewed in some detail by Churg (1993).

The mortality of the Quebec chrysotile miners has now been followed up to the point where more than 80% of the cohort has died. The findings from these studies are discussed in the following sections of this report.

An important new study is that of Camus et al (1998). They found no measurable excess risk of lung cancer mortality among women in two chrysotile asbestos-mining regions. These studies included estimates of exposure. The authors concluded that the EPA's model overestimated the risk of chrysotile asbestos-related lung cancer by a factor of at least 10.

9.1 LUNG CANCER RISK, TYPE OF ASBESTOS AND OCCUPATION

As noted in the discussion of mesothelioma, differentiating between asbestos fibre types is important because there is good evidence that different fibres pose different levels of health risk. These differences in risk not only exist between mesothelioma and fibre type, but also between lung cancer and fibre type. Although lung cancer differences are not as striking as for mesothelioma, they are worthy of consideration.

Hughes (1991) recently compiled the observed and expected numbers of deaths from lung cancer for various occupational groups, using only deaths 20 years after hire or when data were not available,

10 years after hire. The results of combining these, by fibre type and industry sectors in which more than one fibre type is represented, are shown in TABLE 10. In all industry sectors, the risks of lung cancer are lower for chrysotile only exposed persons than for those with mixed or amphibole only exposures. The lung cancer risk in the chrysotile textile industry is substantially greater than in the other chrysotile exposed occupations.

Since differences in the exposure levels for persons in the various studies reflected in table 10 were not taken into account, the validity of adding the studies together might be questioned. However, this problem is somewhat eliminated in FIGURE 3, where lung cancer risks in different industry sectors can be compared for the "same" levels of cumulative exposure[37]. These risks range from not detectable in the friction materials manufacturing industry, to risks in textile workers, which are approximately 50 times greater than those in Quebec chrysotile miners and millers (McDonald et al 1983a).

The reason for lung cancer risks being so much higher in textile plants is not yet known. It is hypothesized that it may be related to other materials used in the textile manufacturing process such as mineral oils which were sprayed on the fibres (Sebastien et al 1989). The possibility that the proportion of very long fibres in the airborne dust may be responsible still needs further investigation. This difference in risk of lung cancer is not explained on the basis of the tremolite content of the airborne dust (Sebastien et al 1989).

In the case of the asbestos textile industry where chrysotile has been the main fibre type used, lung cancer risks may be elevated but mesotheliomas are rare or absent. Thus, in all chrysotile industry sectors, mesothelioma occurs rarely or is absent.

In many studies, persons have been exposed to more than one fibre type. The health experience of these workers may not be directly relevant in considering the possible risk to workers using Dum Dum products where only chrysotile was used.

The clear difference in the lung cancer experience of workers in the chrysotile textile industry compared to that of workers in other chrysotile industries suggests that there is something special about that industry. Extrapolation of the high risks from that industry to the Dum Dum product industry is unlikely to be appropriate, except to err on the side of overestimating risks.

UPDATE:

Mining and Milling Industry Mortality

The updated experience of Quebec chrysotile miners and millers has been reported in a number of papers (McDonald et al 1993; McDonald et al 1997; Liddell et al 1997, 1998). Liddell et al (1997) reported the mortality for 9780 Quebec miners and millers followed from 1935 through 1992. The results (TABLE U9) showed no discernable association between chrysotile exposure and SMR below 300 mpcf.y (roughly 1000 f/cc-yrs). However, higher exposures led to excess mortality from lung

cancer. At higher levels of exposure, the risk increased with cumulative exposure. The effect of cigarette smoking was far more deleterious as the SMR for a 20+ cigarette per day smoker was 4.6 times higher than that of a non-smoker.

TABLE 10. THE OBSERVED & EXPECTED NUMBERS OF DEATHS FROM LUNG CANCER BY FIBRE TYPE AND INDUSTRY SECTOR[33]
Modified from Hughes (1991).

INDUSTRY	FIBRE TYPE: CHRYSOTILE[34]			MIXED[35]			AMPHIBOLES[36]		
	O	E	O/E	O	E	O/E	O	E	O/E
Mining & Milling	239	192.7	1.24				91	34.5	2.64
Gas Mask	6	4.8	1.25				33	15.8	2.09
Insulation				397	93.7	4.24	84	17.5	4.80
Misc. Manufacturing	4	4.3	0.93	77	72.4	2.71			
Asbestos Cement	58	56.3	1.03	351	225.1	1.56			
Textiles	94	55.6	1.69	342	188.9	1.81			
TOTAL	401	313.7	1.28	167	536.1	2.18	208	67.8	3.07

[33] This table has been reproduced with some modification from that presented by Hughes (1991). The friction products industry was eliminated from the original compilation because it had no "mixed" or "amphibole" equivalent for comparison. The totals reflect this change. The table has otherwise retained the classification used by Hughes although it is noted that Hughes included as "chrysotile textile", the plant studied by Robinson et al (1979). While this plant used mainly chrysotile, varying quantities of amosite and blue asbestos were also used.

[34] Based on the following publications...

McDonald et al (1980); Rubino et al (1979); Acheson et al (1982); Weiss (1977); Thomas et al (1982); Ohlson & Hogstedt (1985); Gardner et al (1986); McDonald et al (1983a); Robinson et al (1979).

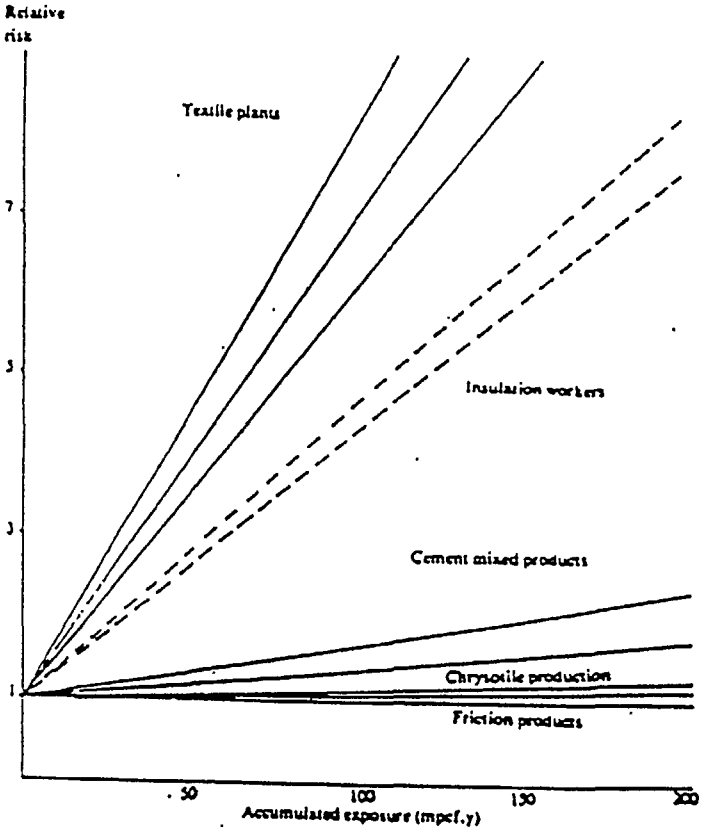
[35] Based on the following publications..

Selikoff et al (1979); McDonald et al (1983b); Enterline et al (1987); Peto et al (1985); Hughes et al (1987); Newhouse et al (1985); Albin et al (1984); Raffin et al (1989); Finkelstein (1984); Alies-Patien & Valleron (1985); Laquet et al (1980);

[36] Based on the following publications..

Armstrong et al(1988); Jones et al (1980); McDonald & McDonald (1981); Acheson et al (1984); Seidman et al (1986)

FIGURE 3 EXPOSURE - RESPONSE RELATIONSHIPS FOR VARIOUS OCCUPATIONAL GROUPS (AFTER MCDONALD 1984)



[37] Cumulative exposures are the sum of the products of exposure level and duration of exposure over a working lifetime and are expressed in concentration-years. The concentration unit, mpcf refers to millions of particles/cubic foot. Prior to the late 1960's, asbestos dust concentrations in North America were measured using a method known as the midget impinger. The method used today is the membrane filter phase contrast microscopic method in which concentrations are reported in fibres/cc. These curves (FIGURE 3) were constructed using the exposure measurements used in the original studies. If exposures were expressed in fibres/cc-years the axes would have changed but the overall pattern of differences in risks in the different industry sectors would remain.The units, fibres/ml (f/ml) and fibres/cc m(f/cc) are treated synonymously.

When the Quebec chrysotile miners' lung cancer and mesothelioma experience was examined in a case-referent study by the geographic area in which mines were located, the following was found:

- Miners in a Central area (A) had a statistically significant excess of lung cancer and of mesothelioma (ORs 1.98 and 2.55 respectively).

- The odds ratios for the peripheral area (B) were not significant (1.09 and 1.11 respectively).

- The Likelihood ratio comparing the two areas for lung cancer was 8.25 (or $p < 0.004$ for lung cancer).

- The rates of mesothelioma in the two areas were statistically significantly different ($p = 0.03$).

- The time weighted exposure of the referents in areas A and B for lung cancer were 15.1 and 20.7 mpcf respectively, and for mesothelioma were 17.4 and 16.3 mpcf respectively. Thus the effect was not due to differences in levels of exposure.

- The tremolite content of the lungs of workers employed in the central area were much higher than those in the periferal area. Thus, the results indicate that mesothelioma and lung cancer risks in chrysotile miners are related to the fibrous tremolite and not to the chrysotile exposure (McDonald & McDonald 1997).

Textile Lung Cancer Risks

Since 1990, attempts have been made to better understand the reasons for the high cancer risks in the textile industry. Several hypotheses have been proposed including chrysotile fibre length, amphibole fibre length, amphibole exposure and oils.

Gibbs (1997, 1999) raised the possibility that long amphibole fibres may be responsibile for the difference between the miners and textile industry lung cancer risks. He pointed out that based on the limited data from the study of fibre in tissues from miners and textile workers by Sebastien et al (1989) that the percentage of tremolite fibres longer than 20 um was 11 times greater in the lungs of textile workers than in the lungs of miners. The size distributions of the chrysotile fibres were only slightly different.

Recent analyses of lung tissue by Case and Dufresne (1999) confirm the earlier findings of Sebastien et al (1989) that the lungs of almost 40% of the textile workers' lungs examined contained crocidolite and amosite.

More importantly, these studies showed that the amphibole fibres in the lungs of textile workers were longer. Thus it seems quite plausible that the excess risk in this industry may be due to long amphibole fibres.

Dement (1991; 1998) examined the question of oil use as a factor in explaining the high lung cancer risk in textiles. He concluded that only a modest reduction (at most) in the slope occurred after controlling for mineral oil exposures. He considered the possible role of longer, thinner, more carcinogenic fibres in textile as a plausible explanation needing further investigation. Scientific opinions differ on whether oils are still to be considered a factor.

Lung cancer risk estimates

Based on his studies Dement estimated that the lung cancer risk increment per fibre/cc-year was 0.031 (Dement et al (1994). The slope reported by Lash et al (1997) was very similar (0.028) based on these same data.

The slope derived from the Quebec chrysotile mining data published by McDonald et al (1993) was 0.0002.

The lung cancer risk estimates reported by Lash et al (1997) who reviewed the various exposure-response studies for chrysotile are discussed in section 16.2. They re-calculated slopes for studies in which exposure response data existed. Differences between the risks in different industry sectors are clear. Lash claims that between sector differences are greater than the between fibre-type differences. However, it is most probable that there are major between fibre-type differences within individual Industrial sectors. An example of such a difference is the difference in the mesothelioma risks between chrysotile and blue asbestos in gas mask workers.

Thresholds of risk.

Over the past 10 years there has been growing acceptance of the possibility that a real or at least practical threshold exists. Meldrum (1996) in an HSE report stated: "However, the balance of toxicological evidence does not support the no-threshold model for asbestos-induced lung cancer."

Others have also examined this concept. While proving the negative is impossible, the evidence is certainly compatible with such a concept or at least non-linearity, with, for practical purposes undetectable risks at low exposures. Ilgren & Browne (1991) examined the evidence for a threshold in animals and humans and the question was examined specifically for chrysotile by Browne & Gibbs (1998).

Using Quebec chrysotile mining data, Vacek (1997) found a threshold effect using a linear relative risk model and found exposure intensity to be a better fit than cumulative exposure.

However, Stayner et al (1997) applying models to textile data did not find support for a threshold type of response. In view of two studies now which show that a substantial fraction of the textile workforce was exposed to amphibole fibres, this study should no longer be considered a pure chrysotile cohort.

Asbestosis and Lung Cancer

In the 1990's significant scientific discussion has centred on the relationship between asbestosis and lung cancer with views expressed in favour and against asbestosis, pathological or radiological being an essential pre-cursor (Churg 1993, Hughes & Weill 1991; Weill 1994; Jones et al 1996; Weiss 1999; Abraham JL 1994; Roggli et al 1994; Egilman and Reinert 1996; Wilkinson et al 1995; Banks et al 1999; and Case & Dufresne 1997).

While authors have invoked animal studies, case reports and anecdotes to support their contentions, the answer to the question must come from epidemiological studies. There are to date only three specifically designed to answer this question. The first showed that the lung cancer risk 20 years after hire was not increased in the absence of parenchymal changes of 1/0 or greater. The study involved 839 men examined in a cross-sectional morbidity study in 1969 (Hughes and Weill 1991; Weill 1994; and Jones et al 1996). This study was strong in the sense that the authors had quantitative exposure estimates for each individual based on measurements in the industry and the study was based only on workers who had radiological changes on entering the study. This study also found no relationship between pleural changes and lung cancer. A criticism leveled at the study was principally its statistical power.

Sluis-Cremer (1991) examined the relationship between asbestosis as diagnosed at autopsy and lung cancer. He was able to take into account age, smoking, residence time and cumulative exposure to amphibole asbestos in fibres/ml-years. The most significant predictor of lung cancer was the presence of asbestosis with an improvement in the likelihood chi-square of 30.1. The study results are shown in Table U11.

TABLE U11. RELATIONSHIP BETWEEN ASBESTOSIS AND LUNG CANCER (FROM SLUIS-CREMER 1991)

Asbestosis	No of persons	Expected	Cancer diagnosed at necropsy	SPMR	Cancer as on death certificate	SPMR
None	302	12.4	11	88.7	8	64.5
Slight	69	3.6	15	416.7	11	305.6
Moderate-pronounced	28	1.6	9	562.5	6	375.0

In the third study (Wilkinson et al 1995), occupational histories and smoking histories were obtained from 271 patients with a confirmed diagnosis of primary lung cancer and 678 referents. Workers were assigned to definite or probable exposure to asbestos more than 15 years before diagnosis. The extent of fibrosis was read by three readers. A previous reader blanked out changes such as tumour mass or lymphadenopathy using a silhouette over no more than two quadrants. These authors

concluded that their results did not suggest that radiological evidence of fibrosis was a prerequisite for asbestos-related lung cancer. The many weaknesses of this study were pointed out by Browne (1994); Weill et al (1995) and Weiss (1995). Jones et al (1996) reviewed the whole field and also identified weaknesses in the study by Wilkinson et al (1995). In contrast to the studies by Sluis-Cremer and Hughes and Weill in which well defined populations, personal quantitative exposures were available and biases largely avoided, the evidence from the Wilkinson study must be interpreted in terms of its weaknesses.

Jones et al presented their evidence for asbestosis being a pre-cursor of lung cancer as follows:

- In a series of post-mortem lung specimens from patients with asbestos and silicosis there were 13.2% and 1.3% of lung cancers respectively.
- In the study by Doll (1955), all lung cancer cases had asbestosis.
- Kipen et al (1987) reported that of 138 cases of lung cancer in insulation workers, all had histological evidence of lung fibrosis and 85% also had radiographic evidence of fibrosis.
- If all the lung cancer cases in insulation workers have asbestosis, then all the excess lung cancer must be in persons with asbestosis.
- Newhouse and Wagner reported moderate or severe histological asbestosis in necropsy specimens of confirmed lung cancer cases in ex-workers in an asbestos factory.
- Excess lung cancer deaths in populations exposed to asbestos generally start at about the same cumulative exposure levels as those at which asbestosis begins to appear.
- The Hughes & Weill study was prospective, had individual smoking and exposure information and radiologic status at outset of observation. The excess risk of lung cancer was restricted to workers with radiographic evidence of asbestosis, a finding consistent with the view that asbestos is a lung carcinogen because of its fibrogenicity.
- Sluis-Cremer & Bezeuidenhout found histological asbestos was significantly associated with the presence of bronchial cancer.
- Increased risks of lung cancer have been reported in studies in which fibrosis has resulted from factors other than asbestos (ie: idiopathic pulmonary fibrosis)

Based on their analysis they concluded that "While the issue of whether asbestos is a necessary precursor to asbestos-attributable lung cancer cannot at this time be considered settled, the weight of the available evidence strongly supports this proposition".

They also noted:

- Lung fibrosis of many causes - known and unknown - is associated with an increased risk of lung cancer.
- The much discussed synergism between asbestos exposure and smoking found in mortality studies of insulation workers turns out to be a synergism involving asbestosis not just asbestos exposure.
- The site of origin and cell type of a lung cancer are not regarded as reliable indicators of causation (or non-causation) by asbestos.
- In asbestos inhalation experiments, animals develop excess lung tumours only when lung fibrosis is produced.
- Pleural plaques have not proved to be a reliable marker for an increased risk of lung cancer.

The Helsinki report (Criteria document 1997) states that in their view "Heavy exposure in the absence of radiologically diagnosed asbestosis, is sufficient to increase the risk of lung cancer". They note that asbestosis is an "indicator of high exposure". Further they suggest that "Asbestosis may also contribute some additional risk of lung cancer beyond that conferred by asbestos exposure alone. Asbestosis diagnosed clinically, radiologically (including HRCT, or histologically can be used to attribute a substantial causal or contributory role to asbestos for an associated lung cancer". Pleural plaques alone were not considered indicative of adequate exposure to attribute lung cancer. In this case, a history of substantial asbestos exposure or high tissue burden of fibres should be required.

Unfortunately different studies still use different definitions of asbestosis.

The relevance of the debate concerning lung cancer and asbestosis to Dum Dum exposures is two fold:

1. If radiological changes are predictors of risk, the levels of exposure associated with Dum Dum products even with a lifetime of continuous use (say 20 years at 0.01 f/cc) is still more than two orders of magnitude below that at which one might begin to be able to discern any change radiologically. Therefore there can be no doubt that working with Dum Dum cannot increase lung cancer risk if radiologically defined asbestosis is a pre-cursor to lung cancer Asbestosis occurs as a result of long and high exposures. In fact, Dum Dum workers are never exposed to high fibre concentrations.
2. If pathological changes are predictors of risk, it is still highly improbable that at these levels of exposure even pathological changes compatible with asbestosis would occur.

As smoking and other exposures can give rise to parenchymal fibrosis, the level of chrysotile exposure associated with working with Dum Dum is such that it is highly improbable that any pathological change, even in a worker continuously with Dum Dum for 20 years is due to any Dum Dum related exposure.

It is clear that in the absence of any parenchymal radiological changes consistent with asbestosis, the likelihood that a lung cancer is asbestos related, if not nil, must be extremely small. Therefore at cumulative lifetime exposure levels such as those associated with the use of Dum Dum the risk of lung cancer may in fact be zero and for practical purposes is zero.

9.2 SMOKING

It is important to consider the magnitude of the risk of lung cancer associated with smoking. TABLE 11 modified from Hammond et al (1979), clearly indicates the importance of smoking. The asbestos workers were insulation workers and the "control" population was the 73763 men in the Cancer Society's prospective cancer prevention study. The risk of lung cancer for non-smoking asbestos insulation workers is 5 times that of non-smoking, non-asbestos exposed workers. The risk of lung cancer for asbestos exposed insulation workers who smoke is 53.24 which is more than 10 times that of non-smoking, asbestos exposed insulation workers. This effect of smoking is termed "multiplicative" because smoking multiplies the risk posed by the asbestos exposure.

McDonald (1984) showed that effects were more additive than multiplicative in chrysotile mine and mill workers as shown in TABLE 12. The risk of lung cancer for chrysotile miners and millers who are heavily exposed non-smokers is 6.9 times that of the non-smoking miner or miller with little exposure. The risk of lung cancer for the heavily exposed but moderate smoker is 12.8 times that of the "little" exposed non-smoker. Thus, moderate smoking almost doubles the risk so it is not possible to say whether the risk is simply the sum of the individual risks of heavy exposure (6.9) plus the risk of moderate smoking (6.3) or a risk of 6.9 multiplied by 2. These risks might be considered to be "additive" as opposed to multiplicative.

TABLE 11. EFFECTS OF SMOKING & ASBESTOS EXPOSURE ON LUNG CANCER RISKS

Asbestos insulation workers
Hammond et al 1979

Group	Exposure to Asbestos	History Cigarette Smoking?	Death Rate /100000	Mortality[28] Ratio
Control	No	No	11.3	1.00
Asbestos	Yes	No	58.4	5.17
Control	No	Yes	122.6	10.85
Asbestos	Yes	Yes	601.6	53.24

[28]Mortality ratio is the ratio of death rate relative to non exposed non-smoking population.

TABLE 12. EFFECTS OF SMOKING & ASBESTOS EXPOSURE ON LUNG CANCER RISKS

Quebec chrysotile miners & millers[39]
McDonald 1984

Asbestos Exposure

	Little	Moderate[40]	Heavy[41]
Non Smokers	1	2.0	6.9
Moderate Smokers	6.3	7.5	12.8
Heavy Smokers	11.8	13.3	25.0

[39]These are risks relative to the risk for non-smoking workers with "little" exposure.

[40]Moderate exposure is cumulative exposure of 30-300 mpcf-yrs accumulated to age 45.

[41]Heavy exposure is cumulative exposure greater than 300 mpcf-yrs accumulated to age 45.

Smoking habits must be taken into account in study design or in interpreting findings. It might be noted that for both chrysotile miners and millers and insulation workers the risk of lung cancer is increased more by smoking than by asbestos exposure. For example, the risk of lungcancer for moderate smokers with little asbestos exposure in the chrysotile mining & milling industry is more than 6 times that of non-smokers with the same exposure while moderate asbestos exposure increases the risk for a non-smoker twofold. In insulation workers, the lung cancer risk in non-smoking, asbestos exposed workers is 5 times that of non-smokers who are not asbestos exposed whereas the risk for workers who smoke but are not exposed to asbestos, is almost 11 times that of non-smokers who are not asbestos exposed. See TABLES 11 & 12.

UPDATE:

There have been several developments concerning smoking in recent years. The American Cancer Society follow-up study has shown that there was a major increase in the risk of lung cancer associated with cigarette smoking between the earlier study (CPS1) and the more recent study (CPS II).(See TABLE U12).

A study of smokers in Quebec Canada by Siemiatycki et al (1994) has shown that lung cancer risk increases considerably with cumulative exposure to cigarettes. The magnitude of this increase in risk is shown in TABLE U13.

Thus smoking is a very important parameter. This is especially important because studies of friction product manufacturing workers exposed to low levels of chrysotile (Berry & Newhouse 1983, Newhouse & Sullivan 1989, Berry 1994) did not show any increase in lung cancer risk. As those workers would have included smokers, there is clearly no evidence of synergistic effects at these levels of exposure (at least adequate for any risk to be detected).

McDonald et al (1993) reported on the mortality from 1976 to 1988 of the 1891-1920 birth cohort of Quebec chrysotile miners and millers and examined the relationship between exposure and cigarettes smoking. They showed that the asbestos effect is greater for the non-smoker than for the smokers or ex-smokers.

The authors examined the relationship between lung cancer and smoking which showed standardized mortality ratios for lung cancer of 0.47 for nonsmokers; 0.67 for ex-smokers, 1.09 for those smoking less than 20 cigarettes per day and 2.41 for those smoking 20 + cigarettes per day. This increase in lung cancer risk from nonsmokers to a 20 + cigarettes per day smoker is about five fold. The overall asbestos effect was only 1.13 being highest for the non-smoker 1.65 and lowest for the greater than 20 + cigarette per day smoker (0.90). They concluded that the interaction between cigarette smoking and asbestos was "less than multiplicative and far from simple".

TABLE U12. COMPARISON OF LUNG CANCER DEATH RATES BETWEEN CPS-1 AND CPS-11 FOR MALES OF ALL RACES WITH PREVALENT CANCERS INCLUDED

(Data from the Smoking and Tobacco Control Monograph, Appendix 4 provided by CDC)

Lung Cancer Death Rates for Males of All Races, 20 Cigarettes Per Day, Duration Fixed at Entry into the Study, 5+ Deaths in Cell

	CPS-1 Rates					CPS-11 Rates				
	Duration					Duration				
Age	30-34	35-39	40-44	45-49	Age	30-34	35-39	40-44	45-49
50-54	64.5	154.7			50-54	100.0	107.4		
55-59	122.8	127.4	149.9		55-59	101.3	225.9	289.5	
60-64		220.7	269.5	312.6	60-64		139.9	323.9	440.5
65-69			330.2	449.5	65-69		550.5	583.7	563.2
70-74				304.1	70-74			722.9	551.9
75-79					75-79			2,225.5	

Lung Cancer Death Rates for Males of All Races, 40 Cigarettes Per Day, Duration Fixed at Entry into the Study, 5+ Deaths in Cell

	CPS-1 Rates					CPS-11 Rates				
	Duration					Duration				
Age	30-34	35-39	40-44	45-49	Age	30-34	35-39	40-44	45-49
50-54	67.8				50-54	188	174.4		
55-59	136.1	189.8	327.1		55-59	161.5	235.2	354.8	
60-64		319.7	478.6	374.6	60-64		540.7	526.0	404.4
65-69			803.5	785.7	65-69		811.1	851.7	836.6
70-74					70-74			1,380.3	1,356.2

Note: Rates are presented for all cells with five or more lung cancer deaths. Person-years of observation and deaths accrue in the age group that an individual was in at the year of followup (age advance) but accrue to the duration category at the time of entry to the study (duration fixed).
Key: CPS=Cancer Prevention Study.

TABLE U13. ODDS RATIOS* BETWEEN CIGARETTE SMOKING AND LUNG CANER, FOR EVER-SMOKERS AND FOR SUBGROUPS DEFINED BY CUMULATIVE AMOUNT SMOKED, BY TWO CONTROL GROUPS (from Siemiatycki et al 1994).

*OR1: adjusted for age; OR2: also adjusted for socioeconomic status and ethnic group; OR3: also adjusted for a priori occupational confounders; OR4: also adjusted for data-based occupational confounders; 95% CI = 95% confidence interval for OR4.

Control Group and Smoking Status	Number of Cases	Number of Controls	Odds Ratios OR_1	OR_2	OR_3	OR_4	95% CI for OR_4
Cancer controls							
Nonsmoker	13	336	1.0	1.0	1.0	1.0	
Ever-smoker	844	1,371	14.7	13.1	14.1	15.8	8.7-29.1
Cigarette-years							
1-500	60	350	4.3	4.1	4.4	4.9	2.6-9.4
501-1,000	209	463	10/7	10.0	10.7	12.3	6.6-22.6
1,001-1,500	248	324	20.1	17.8	19.1	21.3	11.5-39.5
≥ 1,501	327	234	34.6	30.5	33.0	37/5	20.2-69.5
Population controls							
Nonsmoker	13	105	1.0.	1.0	1.0	1.0	
Ever-smoker	844	428	16.1	14.7	15.0	17.3	9.2-32.5
Cigarette-years							
1-500	60	101	4.8	4.6	4.7	5.4	2.7-10.8
501-1,000	209	143	11.6	10.8	11.3	12.8	6.6-24.7
1,001-1,500	248	111	20.4	19.0	19.6	23.0	11.8-45.0
≥ 1,501	327	73	37.2	34.7	35.6	42.2	21.4-83.2

9.3 OTHER FACTORS

It is well established that agents other than asbestos and cigarette smoking are associated with increased risks of lung cancer. Some of the chemicals listed as confirmed or suspected human carcinogens by the American Conference of Governmental Industrial Hygienists are shown in TABLE 13. Many of these have the lung as a possible site for the cancer. Workers may encounter one or more of these as part of their work, hobbies or even by virtue of where they live. Elsewhere in the literature, materials such as man made mineral fibres, silica, herbicides and cadmium among others are mentioned as possibly increasing the risk of lung cancer (Hunter 1987). The radioactive gas radon and radon daughters, which can be present in homes in some parts of the country, has been well established as increasing lung cancer risk in uranium and certain other underground mining industries.

These are just a few of the other factors which need consideration in evaluating lung cancer aetiology.

TABLE 13. CHEMICALS LISTED AS CONFIRMED (A1) OR SUSPECTED CARCINOGENS (A2) BY ACGIH

A1

4-AMINODIPHENYL
BIS-CHLOROMETHYL ETHER
HEXAVALENT CHROMIUM COMPOUNDS
BETA-NAPHTHYLAMINE
4-NITRODIPHENYL
ZINC CHROMATES
BENZIDINE
CHROMATE PROCESSING (CHROMATE)
COAL TAR PITCH VOLATILES
NICKEL SULPHIDE ROASTING
VINYL CHLORIDE

A2

ACRYLAMIDE
ACRYLONITRILE
ARSENIC TRIOXIDE PRODUCTION
BENZENE
BENZO(A)PYRENE
BERYLLIUM & COMPOUNDS
1,3 BUTADIENE
CHLOROMETHYL METHYL ETHER
CHRYSENE
3.3' -DICHLOROBENZIDINE
DIMETHYL CARBAMOYL CHLORIDE
1,1- DIMETHYL HYDRAZINE
DIMETHYL SULPHATE
ETHYLENE DIBROMIDE
ETHYLENE OXIDE
FORMALDEHYDE
HEXACHLOROBUTADIENE
HEXAMETHYL PHOSPHORAMIDE
LEAD CHROMATE
METHYLENE CHLORIDE
METHYLENE BIS (2-CHLOROANILINE)
4,4'METHYLENE DIANILINE
METHYL HYDRAZINE
METHYL IODIDE
NITROPROPANE
N-NITROSODIMETHYLAMINE
N-PHENYL-BETA-NAPTHYLAMINE
PHENYLHYDRAZINE
PROPANE SULTONE
BETA-PROPIOLACTONE
PROPYLENE IMINE
O-TOLIDINE
O-TOLUIDINE
P-TOLUIDINE
VINYL BROMIDE
VINYL CYCLOHEXANE DIOXIDE

UPDATE:

In the 1992 report it was noted that it was well established that agents other than asbestos and cigarette smoking are associated with increased risks of lung cancer.

Other chemical substances

Some of the chemicals listed as confirmed or suspected human carcinogens by the American Conference of Governmental Industrial Hygienists were shown in TABLE 13, many of which had the lung as a possible site for the cancer. This table has now been updated (TABLE U14). There are several new lists of substances classified as carcinogens which might be consulted to identify possible

carginogenic exposures. IARC, as of Nov 1998 listed 75 Group 1 chemicals, mixtures or exposure circumstances classified as carcinogenic to humans, 59 probably carcinogenic, and 227 possibly carcinogenic. The National Toxicology Program - Management status reports are produced from NTP Chemtrack system and identify carcinogenic substances.

There is still scientific debate concerning the carcinogenicity of some of the substances on these lists.

TABLE U14. CHEMICALS LISTED AS CONFIRMED (A1) OR SUSPECTED CARCINOGENS (A2) BY ACGIH

A1

4-AMINODIPHENYL
ASBESTOS
BENZIDINE
BIS-CHLOROMETHYL ETHER
CHROMIUM
HEXAVALENT CHROMIUM COMPOUNDS
NICKEL
NICKEL SULFIDE ROASTING
VINYL CHLORIDE
ZINC CHROMATES
ARSENIC
BENZENE
BERYLLIUM
CHROMATE PROCESSING (CHROMATE)
COAL TAR PITCH VOLATILES
β NAPHTHYLAMINE
NICKEL SUBSULPHIDE
URANIUM
WOOD DUST

A2

ACRYLONITRILE
BENZ(A)ANTHRACENE
BENZO(A)PYRENE
1,3 BUTADIENE
CALCIUM CHROMATE
CHLOROMETHYL METHYL ETHER
1,4 DICHLORO-2-BUTENE
ETHYLENE OXIDE
LEAD CHROMATE
4-NITRODIPHENYL
SULFURIC ACID
VINYL FLOURIDE
ANTIMONY TRIOXIDE
BENZO(B)FLUORANTHENE
BENZOTRICHLORIDE
CADMIUM
CARBON TETRACHLORIDE
DIAZOMETHANE
DIMETHYL CARBAMOYL CHLORIDE
FORMALDEHYDE
4,4'-METHYLENE BIS
STRONTIUM CHROMATE
VINYL BROMIDE

Radon

In the 1992 report, the radioactive gas radon and radon daughters were noted as substances which could be present in homes in some parts of the country. It was also noted that radon and radon daughters had been well established as increasing lung cancer risk in uranium and certain other underground mining industries. Since that time, there have been several studies of homes, some of which have claimed or reported an increased risk and others reporting that they did not find any

increased risk (Samet 1992; Chaffey & Bowie 1994; Auvinen et al 1996; Letourneau et al 1994; Pershagen et al 1994; Lubin & Steindorf 1995; Leenhouts 1999).

Environmental Tobacco Smoke (ETS)

In the 1990's, a debate centred around the risk of lung cancer for persons exposed to sidestream or environmental tobacco smoke (ETS). In a review, Woodward& McMichael (1991) concluded there was an increased risk. Fonthom et al (1994) in 5 metropolitan areas of the US found an increased risk for non-smoking women. A recent international multicenter study found "weak evidence" of a dose-response relationship between the risk of lung cancer and spousal and workplace ETS (Boffetta et al 1998). Other publications offer other opinions (Lee & Forey 1999; Denson 1999).

Lung Cancer Latency

The point at which a cancer process is initiated is not known and depends on the actual mechanism by which the cancer occurs.

If epidemiological studies have shown a link between exposure and the occurrence of lung cancer then some indication of the exposures which might have potential to contribute to risk can be inferred from the data. For example, Mustacchi (1996) argues that because insulation workers exposed less than 15 years before death did not show a statistically significant excess mortality from lung cancer, while all longer categories of latency did, the latency for lung cancer (at least in insulation workers) most probably exceeds 15 years. On this basis, any exposures occurring in the last 15 years before death are unlikely to have contributed to the occurrence of the cancer or death. He goes on to argue that exposures in the period prior to the last 15 years should be considered cumulative contributors to a decedent's fully materialized risk of 100% (ie: in the case of an individual who died with lung cancer). Based on the risks of dying from lung cancer in the various periods (TABLE U15) and cumulating all those risks he estimated the proportionate contributions to risk.

Unfortunately, a serious limitation of the insulation worker's study is that level of exposure or nature of exposure was not taken into account. There is ample evidence that these are important. The calculations cannot be applied to industrial sectors where there is no human evidence of an increased lung cancer risk.

While the application of this latency concept for risk apportionment is appealing it would only be a reasonable way of apportioning risk if the level and nature of exposure and other factors are taken into account and if there is epidemiological evidence that at these latencies there is a detectable increase in lung cancer risk in workers with that type and level of exposure. This is not the case with some chrysotile cohorts such as friction product manufacturing workers.

Mustacchi (1996) attempts to overcome the issue of exposure level using the experience of amosite factory workers where he argues that the latency shortens with increasing cumulative exposure and that at 50f-y/ml the risk falls below that suffered by the insulation workers. Therefore it would be

reasonable to consider as unlikely an asbestos etiology for lung cancer developing in less than 15-19 years in an individual with a cumulative exposure of 25-49 f-y/ml or occurring in less than 20-24 years in the presence of a cumulative exposure of 6-24 f-y/ml or in less than 25-29 years when the cumulative exposure is less than 6 f-yr/ml.

TABLE U15.

ESTIMATES OF CONTRIBUTIONS TO LUNG CANCER RISK BY LATENCY PERIOD BASED ON INSULATION WORKER DATA.

LATENCY	RISK/100000 PERSON/YEAR	PROPORTIONATE CONTRIBUTION %
<15		0
15-19	64.5	5.2
20-24	147.6	12.0
25-29	340.5	27.7
30-34	679.0	55.1
TOTAL	1230.6	

10.0 GASTROINTESTINAL CANCER

Selikoff et al (1964), in a study of insulation workers, reported that there was an excess of gastrointestinal cancer. As mentioned earlier these workers were most likely exposed to mixed chrysotile/amphibole fibres. Since that time there have been a number of cohorts studied where an excess of gastrointestinal cancer has been claimed and others where the results have been negative. The epidemiological studies have not shown consistent excesses of gastrointestinal cancers of specific sites. Thus, there is still controversy over whether gastrointestinal cancer risks are increased. Edelman(1988) reassessed the results of 32 published cohort studies and he concluded that "asbestos workers are not at an increased risk of gastrointestinal cancer".

As will be shown later in this report, the levels of exposure to fibres from "Dum Dum" are such that gastrointestinal cancer risks do not require further consideration.

UPDATE:

Liddell (1994) reporting on mortality in chrysotile miners concluded, "There is no evidence that the risk of stomach cancer is adversely affected by exposure to chrysotile; and there is no evidence of increased risk of other abdominal malignancies".

An excess of GI cancer has been reported in insulation workers who have been exposed to chrysotile and amphibole fibres (Selikoff & Seidman 1991).

11.0 OTHER CANCERS

The question of whether laryngeal cancers are associated with exposure to the various asbestos minerals is doubtful but still debated. Liddell (1991) in reviewing previous evidence opined that " the evidence on the link between exposure to asbestos and laryngeal cancer has failed to satisfy accepted criteria for causation". He noted that the Industrial Injury Advisory Council (UK) had recently recommended that, on the balance of the evidence, cancer of the larynx should not be added to the schedule of prescribed diseases in respect of occupations involving exposure to asbestos.

There have been studies of women working with blue asbestos where an excess of cancer of the ovary has been reported (Acheson et al 1982). Whether these were cancers of the ovary or mesothelioma remains unclear, as does the possibility of an association between asbestos and cancers of this site.

There have been claims in the literature that cancers of other sites, such as the kidney, might be associated with asbestos exposure, but the cases appear to be isolated. My review of the evidence concerning kidney cancer indicates that if there is an increased risk, it is not associated with chrysotile.

UPDATE:

There has been no information to change the situation as stated in 1992 and elsewhere in this report.

12.0 TALC

Although chrysotile was the only commercial asbestos used in the Dum Dum products, some contained talc. Talc, under some circumstances, has been shown to be contaminated with asbestiform tremolite, anthophyllite and chrysotile. Contaminants of the talc will be encapsulated in the Dum Dum products in the same way as the talc, chrysotile asbestos and other constituents. None-the-less, talc and the possibility of tremolite contaminants will be considered for completeness.

Talcs do not always contain tremolite, much less asbestiform tremolite or other asbestos fibres. After the mid-1970's Mobil purchased only tremolite free talc for use in its Dum Dum products. This does not mean that the talc before that time contained asbestiform tremolite but provides assurance that it did not, after that time. The presence of tremolite does not necessarily mean the presence of asbestiform tremolite.

There is considerable debate concerning the importance of cleavage fragments of tremolite. However as will be seen later in this report, the very low dust concentrations associated with applying or removing Dum Dum compounds do not make lung fibrosis from the asbestos minerals or tremolite contamination of talc a viable consideration.

UPDATE:

There has been no information to change the situation as stated in 1992.

13.0 ASSESSING HAZARD AND RISKS

In an earlier section it was noted that one of the key factors in the initiation of a disease process was "the characteristics and properties of the dust to which the worker is exposed."

In this section, characteristics other than fibre type will be considered.

The characteristics and properties of a dust which are important in determining its potential to produce a health effect include:

a. Particle size, shape and particle density (number of grams/cubic centimetre of the particle). These determine the aerodynamic behaviour and respirability of the dust, as well as whether and where the particle deposits in the respiratory system.

b. Particle size, particle shape and solubility. These determine whether and how long the deposited particle remains at the site of deposition or whether it is removed from the lung.

c. Chemistry and mineralogy. These determine the likely reactivity of the dust in the lung or system.

d. The toxicity or potential for the dust to produce adverse health effects.

e. Associated contaminants which might, in their own right, have the potential to cause health effects or influence the dust effects.

UPDATE:

No new information.

13.1 PARTICLE SIZE & SHAPE

The size, shape and density of particles are extremely important in determining whether they get into the lung, and if they do, how well lung defense mechanisms can remove them. The ability of fibres to experimentally induce disease is also size dependent.

UPDATE:

No new information.

13.1.1 Aerodynamic behaviour, respirability and deposition

Particles of significance from the standpoint of evaluating the health effects of asbestos, are those suspended in air (collectively known as aerosols) which may be inhaled.

Airborne particulates may range in size from molecular dimensions up to about 100 μm in diameter[42].

The rate at which spherical particles settle out in air is governed by Stokes' Law. This relates the terminal settling velocity of a particle (rate at which it falls in the air) to its density and radius as it falls in non-turbulent air. The aerodynamic diameter is the diameter of a sphere of unit density which has the same terminal settling velocity as the particle of interest.

As most dust clouds are made up of particles with a wide range of sizes and densities, it is convenient to consider particles equivalent if they exhibit the same settling rates which are defined by the aerodynamic diameter mentioned above. The physical dimensions of particles are important as they determine the aerodynamics of the particle, its ability to enter the respiratory tract and its behaviour in the respiratory system after deposition.

Particles are deposited in the lung by essentially four mechanisms, gravitational settlement, inertial impaction, Brownian movement[43] and interception. Particles which can penetrate deep into the alveolar region of the lung are defined as "respirable".

Timbrell (1965, 1970, 1973); Timbrell & Skidmore (1971) and Timbrell et al (1970) studied the aerodynamic behaviour of fibres and the relationships between fibre and non-fibrous particle aerodynamic behaviour by measuring particle and fibre deposition rates. They were able to show that length of the fibre was of little importance in determining fibre deposition rates but that, aerodynamically, fibre diameter was extremely important.

Based on these relationships and assuming that the upper limit of the diameter of a spherical particle of unit density reaching the lung alveolar region is 10μm, then the expected upper limit of the diameter of asbestos fibres penetrating to the alveolar region would be approximately 3.5μm. Assuming that they are straight, differences in the aerodynamic behaviour of fibres of different types are governed by their fibre densities and their diameter size distributions.

The sizes of compact particles found in the alveolar or gas exchange region of the lung are generally 10 μm or less in diameter. On the other hand, fibres of lengths exceeding 100 μm can be found deep in the lung at autopsy. This fits with the lesser importance of length in explaining aerodynamic behaviour.

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[42] μm = micrometre or one millionth of a metre and is the standard unit used in describing the sizes of small particles.

[43] Brownian movement means that particles are moved about in the lung air by bombardment by gas molecules. In this way they get close to surfaces and deposit. Fibre deposition in the lung is governed by essentially the same mechanisms as for spherical particle deposition.

Interception as a mechanism for fibre deposition in the respiratory airways is very important. Timbrell (1965) showed that there was a definite tendency for fibres longer than 5μm to align in laminar air flow. He also showed that asymmetry led to a lower rate of alignment which is important because it means that curly fibres or those with adhering particles have a lower chance of penetrating deep into the lung. Timbrell (Davies 1970) suggested that curliness was an important factor in explaining the apparent lower risks of health effects associated with chrysotile asbestos. Using UICC chrysotile fibre samples[44] and glass fibres of various shapes, he was able to show a significant reduction in the penetration into the alveolar regions of the lung of curly compared to straight fibres.

In a person who breathes through the nose, long fibres, curly fibres and fibres with adhering particles are likely to be trapped in the nose and to be removed by sneezing or by blowing the nose. If a person breathes through the mouth, the larger airborne fibres, normally trapped in the nose, are deposited by impaction in the upper part of the respiratory system. Whether breathed through the nose or mouth, fibres are rapidly transferred to the mouth, swallowed and excreted.

UPDATE:

There has been no new information to change the information provided. The importance of aerodynamics, size and shape was reviewed by Lippmann (1994).

13.1.2 Role of particle size in particle removal from the lung

The moment that either inert or toxic particles are deposited in the lung, mechanisms to remove them come into play. These can be divided into those that depend on ciliary action and those that act in the non-ciliated regions of the lung.

As in everyday life, particles and fibres which are deposited on the tracheobronchial part of the respiratory system are transported by mucociliary action upwards to be swallowed. This method of particle removal can be hindered by damage to the cilia, as through the effects of cigarette smoking, with clearance of particles severely impaired (Cohen et al 1979).

If particles are deposited more deeply in the lung, one of the most rapid removal mechanisms involves recruitable macrophages (phagocytic cells contained in the alveolar surface epithelium). A particle is engulfed by the macrophage or phagocyte which moves the particle towards the ciliary escalator for removal. Phagocytes may also serve to render the particles incapable of injuring or irritating the tissues.

Particle size and shape are important in clearance from the alveolar surface because shape and dimension may hinder phagocytosis. Allison (1973) examined the limit to the size of particles that

[44] UICC is the International Union Against Cancer and under their auspices special samples were prepared for use in animal experiments around the world. There were 2 chrysotile samples, one from "Rhodesia" and another from Canada. The latter was a mixture of fibres from various mills in Quebec. There were also samples of amosite and crocidolite from South Africa and of anthophyllite from Finland.

could be ingested by phagocytes, using fractions of chrysotile and blue asbestos containing high and low percentages of short and long fibres. Independently of fibre type, the short fibres (<5 μm) were rapidly and completely taken up by the phagocytes; fibres longer than 20 μm were never completely taken up; and fibres of length 5-20 μm were sometimes completely ingested. The observation that short (less than 5 μm) fibres are readily removed from the lung has been reported many times in the literature.

UPDATE:

Bioperistence is very important and has been discussed elsewhere in this report.

13.1.3 Particle solubility

Particle solubility is important in assessing health risks. In the case of chrysotile it has been well established that its outer magnesium layer makes it possible for body fluids to dissolve or leach out magnesium. The chrysotile structure is thus steadily dissolved and at a much greater rate than for the amphibole asbestos minerals (Morgan & Cralley 1973). Thus, increased solubility over the amphiboles may be another factor in the decreased retention of chrysotile in the lung.

UPDATE:

Particle durability is very important and has been discussed elsewhere in this report.

14.0 RISKS OF HEALTH EFFECTS FROM WORKING WITH DUM DUM

In section 6.0 it was noted that the risk of disease was related to the following....

i. The characteristics and properties of the dust to which the worker is exposed.

ii. The concentrations of the respirable fraction of the dust to which the worker is exposed.

iii. Duration of exposure to the airborne dust.

iv. Pattern of exposure.

v Period since first exposure to time that the worker is examined for health effects.

vi. Personal habits of the worker which might directly or indirectly:

a. influence the risk of occurrence of a dust related disease(s) or health effect(s) or influence the course of the disease.

b. produce a disease indistinguishable from that produced by the dust.

vii. Individual personal characteristics or pre-disposing conditions which might directly or indirectly influence the occurrence or course of a disease in that worker.

In the following sections the information available for Dum Dum products will be systematically applied in the assessment of the health risks for persons using Dum Dum products.

14.1 CHARACTERISTICS AND PROPERTIES OF AIRBORNE ASBESTOS DUSTS FROM DUM DUM

14.1.1 Asbestos fibre types

Earlier in this report it was noted that chrysotile was the only commercial asbestos fibre used in the Dum Dum products. It was also noted that talc was used in some of the products. In order to ensure that risks are not in any way underestimated, products will be considered to fit into 4 separate categories.

CATEGORY A: Products which contained neither commercial asbestos nor talc. Products in this category include - Chimney Dum Dum Primer

CATEGORY B: Products which contained only chrysotile but no talc.

Products in this category include

- Chimney Dum Dum
- Nail Hole Dum Dum
- Dum Dum Masonoc
- Dum Dum Armorcote

CATEGORY C: Products which contained no commercial asbestos as part of their formulations but contained talc. There is no information on whether tremolite was present in the talcs used. Two scenarios can be envisaged.

1. Products containing no asbestos - This is assured after the mid-1970's when the talc used was tremolite free. It may always have been tremolite free.

2. Products containing some tremolite by virtue of use of talc containing asbestiform tremolite. This is an assumption for which there is no direct evidence.

A product in this category is - Dum Dum Masonoc Primer CATEGORY D. Products which contained chrysotile asbestos as part of their formulations plus talc.

Two scenarios can be envisaged.

1. Product containing chrysotile only. This can be assured after the mid 1970's when the talc used was tremolite free. It may have always been tremolite free.

2. Product containing chrysotile plus tremolite by virtue of the use of talc containing asbestiform tremolite. This is an assumption for which there is no direct evidence.

Products in this category include - Hi -Heat Dum Dum

UPDATE:

There has been no new information since 1992 to change the products or their categorization.

14.1.2 Sizes of airborne fibres

The composition and size distribution of the fibres to which a worker might be exposed are rarely the same as those of the bulk material with which he or she may be working. This is because particles of different sizes may have different compositions and because size, shape and density determine whether particles, dispersed in air, remain airborne.

In the case of Dum Dum products, the commercial grade of chrysotile fibre used was short (grade 7) (Chamberlain 1991). The production process called for this fibre to undergo several stages of intimate mixing with various resins and oils as well as other inorganic materials and solvents. Thus the product was quite unlike the free asbestos used in its production.

Dum Dum products are of putty or mastic consistency. Scanning electron images[45] show how the surface of the Dum Dum product forms to encapsulate the fibre and this is evident in the removed product (PLATE 1). This binding of fibres to the matrix and to other fibres is seen in the broken cross-section of a piece of Chimney Dum Dum (PLATE 2) and in the surface clump (PLATE 3). The latter clump of fibres is about 20µm in diameter and non-respirable. For comparison, scanning electron images of free chrysotile at the same magnifications are shown in PLATES 4 - 6 The difference between the encaspulated and non-encapsulated fibre is quite evident.

The material after application dries with a hard surface. A sample of the Hi-Heat Dum Dum after removal, which was dark in colour, was provided to me by Dr Peters (1991). This sample when broken, was still moist and putty-like under the surface layer. There were no overtly visible fibres in the surface of the specimen. While much thinner, fragments of the "dried" Dum Dum Masonoc and Chimney Dum Dum were still very pliable. It is clear at both the macroscopic and electron microscopic level that the asbestos is encapsulated and not likely to give rise to high concentrations of fibres during use or removal of the product.

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[45] The micrographs were made available to me courtesy of Dr E.T. Peters of Arthur D. Little Inc.,Cambridge Massachusetts.

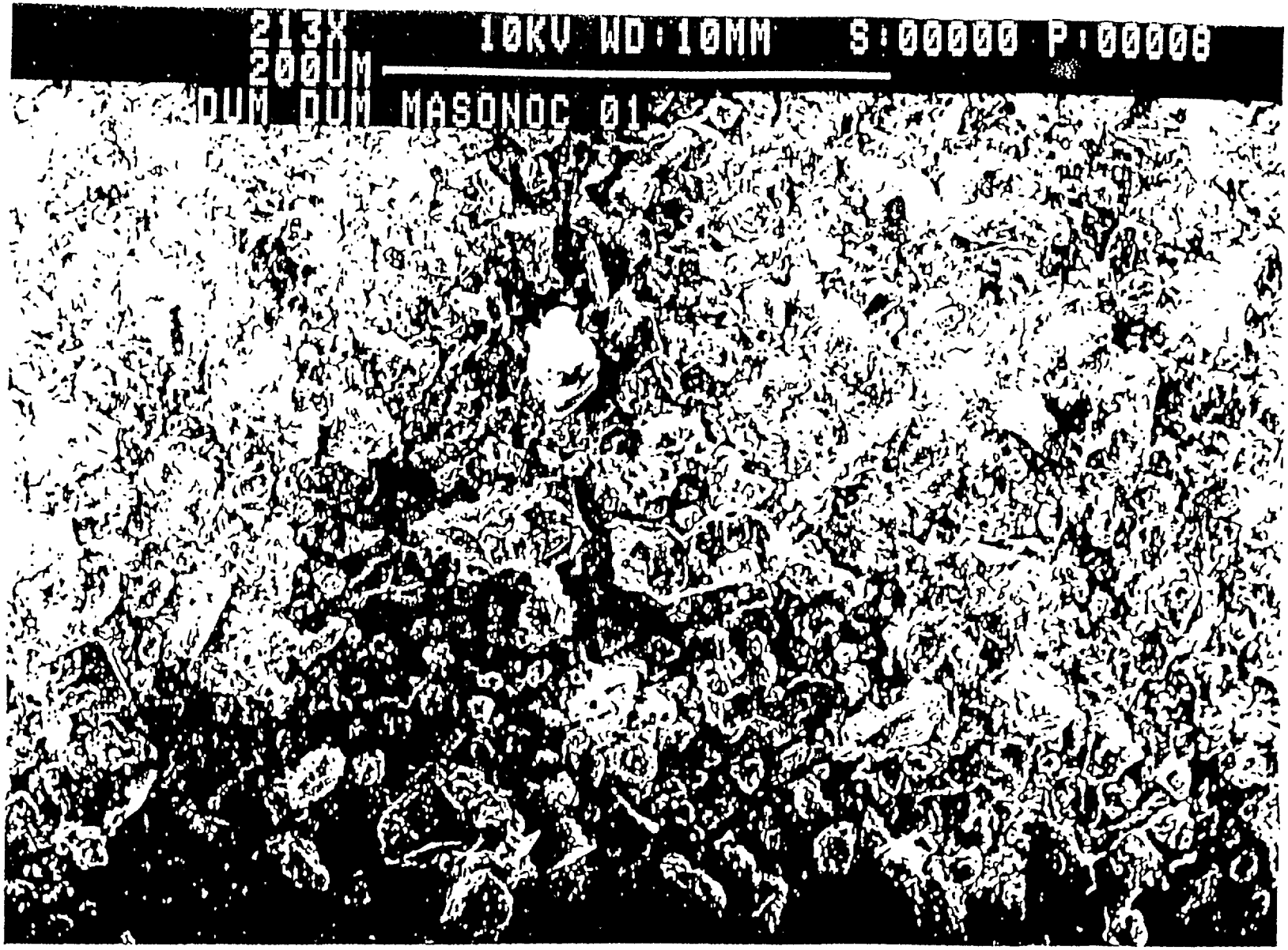

PLATE 1. SURFACE OF DUM DUM MASONOC TO SHOW ENCAPSULATION (SEM)

PLATE 2. BROKEN CROSS-SECTION OF CHIMNEY DUM DUM SHOWING HOW FIBRES ARE ENCAPSULATED. (SEM)

PLATE 4. FREE CHRYSOTILE FIBRES AT THE SAME MAGNIFICATION AS FIGURE 1. SAMPLE PREPARED IN SAME LABORATORY AS FIGURE 1.

PLATE 5. FREE CHRYSOTILE FIBRES AT THE SAME MAGNIFICATION AS FIGURE 2. SAMPLE PREPARED IN SAME LABORATORY AS FIGURE 2

PLATE 6. FREE CHRYSOTILE FIBRES AT THE SAME MAGNIFICATION AS FIGURE 3. SAMPLE PREPARED IN SAME LABORATORY AS FIGURE 3

PLATE 7. AIRBORNE ASBESTOS STRUCTURES - DUM DUM MASONOC REMOVAL EXPERIMENT.

PLATE 8. AIRBORNE ASBESTOS STRUCTURES - DUM DUM MASONOC APPLICATION EXPERIMENT.

PLATE 9. AIRBORNE ASBESTOS STRUCTURES - DUM DUM MASONOC APPLICATION EXPERIMENT.

PLATE 10. AIRBORNE ASBESTOS STRUCTURES - CHIMNEY DUM DUM APPLICATION EXPERIMENT.

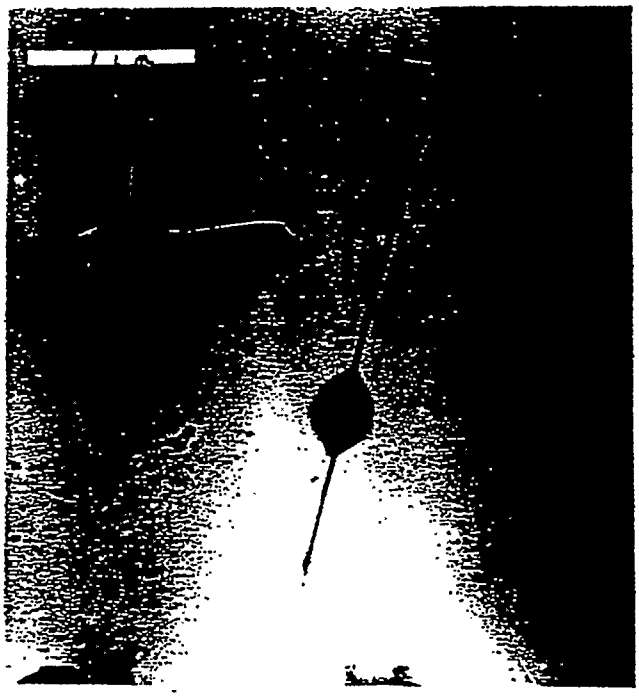

PLATE 11. AIRBORNE ASBESTOS STRUCTURES - CHIMNEY DUM DUM APPLICATION EXPERIMENT.

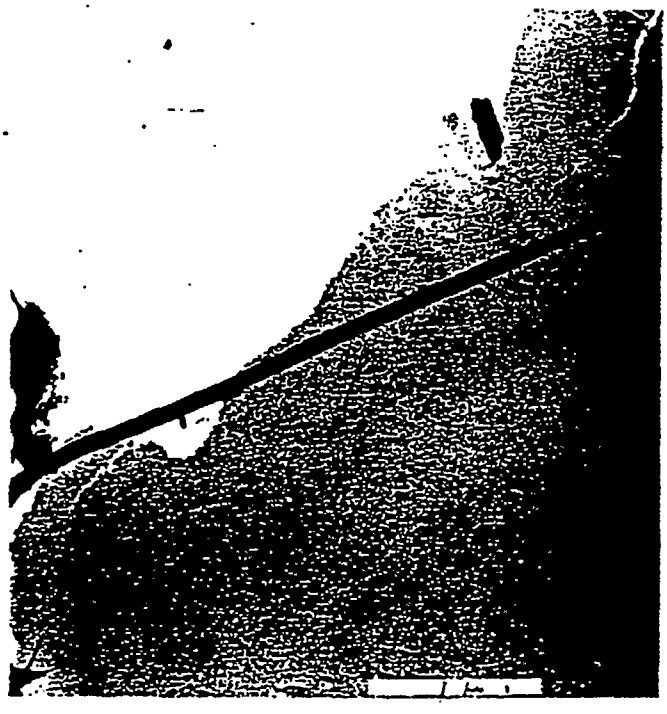

PLATE 12. AIRBORNE ASBESTOS STRUCTURES - CHIMNEY DUM DUM APPLICATION EXPERIMENT.

PLATE 13. AIRBORNE ASBESTOS STRUCTURES - HI-HEAT DUM DUM REMOVAL EXPERIMENT.

PLATE 14. AIRBORNE ASBESTOS STRUCTURES - HI-HEAT DUM DUM REMOVAL EXPERIMENT.

PLATE 15. AIRBORNE ASBESTOS STRUCTURES - HI-HEAT DUM DUM REMOVAL EXPERIMENT.

PLATE 16. AIRBORNE ASBESTOS STRUCTURES - HI-HEAT DUM DUM REMOVAL EXPERIMENT.

PLATE 17. AIRBORNE ASBESTOS STRUCTURES - HI-HEAT DUM DUM REMOVAL EXPERIMENT.

From a health effects standpoint, it is the airborne respirable fibres which are important.

Dr E.T. Peters (1990, 1991) performed a series of tests with three Dum Dum products, measuring the concentrations of asbestos structures (fibres, bundles, clusters and matrix)[46] associated with the trowelled on, spray application and removal of the products. Counts of airborne fibre were made using transmission electron microscopy.

Chimney Dum Dum was applied to bricks, Dum Dum Masonoc was applied to concrete and Hi Heat Dum Dum was applied to steel sheets; each such product was then removed.

As will be described later, the concentrations rarely exceeded the minimum detection limit for the experiments (0.01 asbestos structures/ml). Thus, there were few airborne asbestos structures to examine. Dr Peters provided me with the raw data concerning the dimensions of the structures in the airborne dust samples collected and analyzed by him (Peters 1991).

a. Dum Dum Masonoc

The dimensions of asbestos structures associated with ten application and removal experiments of this product are shown in APPENDIX D1. The concentrations measured in these experiments are summarized in TABLE 14. The total number of structures observed in all the Dum Dum Masonoc experiments was 20. Of the 20 observed structures, 3 would not have been visible by light optical microscopy (limit of visibility is approx 0.23 μm) and another had a diameter well exceeding 3 μm and would not be respirable. Thus 3 of these structures would not have been counted under the rules for counting fibres for compliance with the US OSHA standard[47]. These fibres have been removed and concentrations recalculated to reflect the concentrations that would have been reported under OSHA rules. These concentrations are identified as "PCM Equivalents". See TABLE 14

Based on transmission electron micrographs provided by Dr Peters (1991), the structures observed during the application of this product tended to be bundles of fibres with adhering particles rather than individual fibres (See PLATES 7-8). Similar results were seen during removal (See PLATE 9). Such adhering particles were, in some cases, quite large and could possibly have rendered these fibres non-respirable..

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[46] Dr Peters considered an asbestos structure to have a length to breadth ratio of 3:1 or greater and a length of 5μm or more.

[47] The OSHA regulations (Appendix A to 763.121-EPA/OSHA Reference Method-Mandatory - Federal Register 52 No 36-37 February 25 1987 page 5629) require that "fibres counts be made by positive phase contrast microscope" at "total magnification of approximately 400 X" and counting "only fibre equal to or longer than 5 micrometers" and counting " all particles as asbestos that have a length to width ratio (aspect ratio) of 3:1 or greater". The method also requires that the microscope used not be able to resolve the finest lines 6 & 7 on the HSE phase shift test slide. The width of the smallest ridge 7 is 0.25μm. The assumption of a limit of visibility of 0.23μm still allows for the inclusion of some fibres that would not be counted by the OSHA method.
This means that the concentrations that would have been associated with use of this product, had they been determined using phase contrast light optical microscopy, as required for measuring compliance with the OSHA permissible exposure limit, would have been much lower. These "OSHA" equivalent concentrations are recalculated and shown in TABLE 14.

TABLE 14. CONCENTRATIONS OF AIRBORNE STRUCTURES DURING THE APPLICATION AND REMOVAL OF VARIOUS Dum Dum PRODUCTS IN A SERIES OF EXPERIMENTS BY ARTHUR D LITTLE INC. (BASED ON DATA PROVIDED BY PETERS 1991)

PRODUCT	PROCESS	TEM[48] CONCN s/cc	PCM[49] EQUIV. f/cc
Hi- Heat Dum Dum	Application 1	<0.01	<0.01
	2	<0.01	<0.01
	Removal 1	<0.01	<0.01
	Replicate 1	<0.01	<0.01
	Removal 2	<0.01	<0.01
	Removal 3	0.01	0.01
Dum Dum Masonoc	Application 1P	0.01	<0.01
	A	<0.01	<0.01
	Application 2P	<0.01	<0.01
	A	<0.01	<0.01
	Removal 1P	<0.01	<0.01
	Replicate 1P	<0.01	<0.01
	Removal 1A	<0.01	<0.01
	Removal 2p	<0.01	<0.01
	Replicate 2p	<0.01	<0.01
	Removal 2A	<0.01	<0.01
Chimney Dum Dum	Application 1P	0.088	0.02
	Replicate 1P	0.024	<0.01
	Application A	0.024	<0.01
	Removal 1P	<0.01	<0.01
	Removal 1P	<0.01	<0.01
	Removal 1P	<0.01	<0.01

b. Chimney Dum Dum.

The dimensions of fibres in the samples collected during the application and removal of this product are shown in APPENDIX D-2. The concentrations measured in these experiments are shown in TABLE 14.

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[48] TEM = Transmission electron microscopy. Concentrations are reported as structures/cc(s/cc). s/cc = number of structures meeting the "asbestos structure" definition per 1 cc of air.

[49] PCM-EQUIV = Phase Contrast Microscopic (PCM) equivalent concentration. This concentration represents the concentration that would have been reported had the filter been counted using phase contrast microscopy and OSHA counting rules. The structures counted by transmission electron microcopy are considered to be asbestos fibres. These OSHA-equivalent fibres are considered to be those longer than 5μm in length with diameters greater than of 0.23μm. Concentrations are expressed in fibres/cc in line with the practice in the OSHA standard to express concentrations in fibres/cc (f/cc).

The vast majority of the structures observed in the airborne samples taken during tests of this product were short and thin. In fact, the majority of fibres observed had diameters less than would be visible by light optical microscopy (PLATES 10-12).

The relatively high proportion of fibres of 5-6μm length would suggest that fibres from this product would be dealt with relatively efficiently by macrophages as part of the lung protective mechanism if they were to enter and be deposited in the lung (Allison 1973).

Combining the size distributions of all the fibres recorded in the Chimney Dum Dum experiment, 37 of the 45 had diameters of 0.2 μm or less (78.7%) and would not be visible by light optical microscopy. This has an important effect on the concentration relative to the OSHA permissible exposure limit. See TABLE 14.

c. Hi-Heat Dum Dum

The dimensions of fibres in the samples collected during the application and removal of this product are shown in APPENDIX D-3. The concentrations measured in these experiments are shown in TABLE 14.

There were only 11 structures observed in the whole series of experiments with Hi- Heat Dum Dum, 1 during product application and 10 during the 3 removal experiments with replicate. While a few of the structures observed in samples during removal of the Hi-heat Dum Dum appeared as relatively straight "fibres" (PLATE 13-14), others were quite bent or had large adhering particles which would influence their respirability. PLATES 15-17. None of these fibres had diameters less than 0.23 μm, so all would be visible by phase contrast optical microscopy.

UPDATE:

There has been no new information since 1992 to change the information on the sizes of airborne fibres. The results as presented in the final reports by Arthur D Little Inc (1992) are shown in TABLE U16.

14.1.3 Asbestos exposure of workers applying and removing Dum Dum products

UPDATE:

The reports describing the production test procedures used by A.D. Little are given in Appendix UI.

TABLE U16. CONCENTRATIONS OF AIRBORNE STRUCTURES DURING THE APPLICATION AND REMOVAL OF VARIOUS DUM DUM PRODUCTS IN A SERIES OF EXPERIMENTS BY ARTHUR D LITTLE INC. (FROM REPORTS BY ARTHUR D. LITTLE INC (1992).

PRODUCT	PROCESS		TEM[48] CONCN s/cc	PCM[49] EQUIV. f/cc
Hi- Heat Dum Dum	Application	1	<0.01	<0.01
		2	<0.01	<0.01
	Removal	1	<0.01	<0.01
	Replicate	1	<0.01	<0.01
	Removal	2	<0.01	<0.01
	Removal	3	0.01	0.01
Dum Dum Masonoc	Application	1P	0.01	<0.01
		A	<0.01	<0.01
	Application	2P	<0.01	<0.01
		A	<0.01	<0.01
	Removal	1P	<0.01	<0.01
	Replicate	1P	<0.01	<0.01
	Removal	1A	<0.01	<0.01
	Removal	2p	<0.01	<0.01
	Replicate	2p	<0.01	<0.01
	Removal	2A	<0.01	<0.01
Chimney Dum Dum	Application	1P	0.088	0.02
	Replicate	1P	0.024	<0.01
	Application	A	0.024	<0.01
	Removal	1P	<0.01	<0.01
	Removal	1P	<0.01	<0.01
	Removal	1P	<0.01	<0.01

[48] TEM = Transmission electron microscopy. Concentrations are reported as structures/cc(s/cc). s/cc = number of structures meeting the "asbestos structure" definition per 1 cc of air.

[49] PCM-EQUIV = Phase Contrast Microscopic (PCM) equivalent concentration. This concentration represents the concentration that would have been reported had the filter been counted using phase contrast microscopy and OSHA counting rules. The structures counted by transmission electron microcopy are considered to be asbestos fibres. These OSHA-equivalent fibres are considered to be those longer than 5μm in length with diameters greater than of 0.23μm Concentrations are expressed in fibres/cc in line with the practice in the OSHA standard to express concentrations in fibres/cc (f/cc).

14.1.3.1 Levels of exposure

Hodgson (1985), reviewing alternatives to asbestos, noted "Mastics and sealants based on pitch and asbestos are used in coating applications where felts are inappropriate..... In these materials, asbestos imparts the property whereby the mixture remains in a consistent state. Mastics and sealants are marketed in sealed tins and the components must not settle or separate. If the mastic is to be applied to a vertical or sloping surface it must not run or sag, and again this is prevented by the presence of asbestos..... At present it would appear that asbestos based mastics and sealants have not been superseded by any wholly viable and workable alternative." Cogley et al (1982), in a report on asbestos in commercial and industrial use, described the composition of asbestos roofing products, coatings and paints and bituminous coatings as virtually all using grade 7 fibre. He noted " The asbestos fibres are thought to be completely bound by all of these additives in the final product". In describing the primary manufacture of sealants mixed with asphalt, tar and solvents or other additives, they noted " shortly after mixing, the asbestos fibre is bound by the asphalt and upon completion of mixing, the asbestos is considered completely bound in the asphalt with little chance of fibre dust exposure."

In discussing the release of fibres from wastes, weathering, corrosion, wear and scrap through demolition, they state

> "...In nearly all of these losses the asbestos fibres should be completely encased in the bituminous binder. Consequently, only small quantities of free fibre are likely to be released"

The Consumer Products Safety Commission under the product "Mastic" noted "Since asbestos as an additive reduces embrittlement, the danger of free fibre exposure appears limited" (CPSC 1978).

In the guidelines for assessment and abatement of asbestos containing materials in buildings, non-friable materials are defined as "matrix-bonded composite products" prepared by mixing fibres with various bonding agents such as starch, glue, plastics, cements and asphalt. The degree of bonding of asbestos fibres within the composite material or the fibre's immobilization covers a wide range. Caulking putties and mastics are described as non-friable (NBS 1983).

As the fibres in Dum Dum are coated with oleoresinous substances and by design will adhere to other particles, they will if rendered airborne, tend towards being far less respirable than free fibres encountered in the industries where health risks have been assessed.

Crucial to the potential of the product to pose a risk of health effects is the airborne respirable fibre concentration. In the following subsections, the potential for generating airborne asbestos fibre concentrations and the order of magnitude of the concentrations are discussed.

The various products under consideration are applied in a viscous "putty-like" state with the asbestos fibres and other components intimately mixed into a matrix.

Let us consider the various products in the categories described in section 14.1.1.

CATEGORY A. (Chimney Dum Dum Primer)

There will be no asbestos exposure as the product does not contain asbestos minerals.

CATEGORY B. (Nail Hole Dum Dum; Chimney Dum Dum, Dum Dum Masonoc, Dum Dum Armorcote)

i. Nail Hole Dum Dum.

As far as could be determined, measurements of exposure to chrysotile asbestos when using this product do not appear to have been described in the literature. However the following is known or can be deduced:

* The potential for a worker to be exposed to chrysotile asbestos fibre from this product is limited by the asbestos content of the product (only 2% chrysotile).

* The product is putty-like in consistency which will not readily give rise to aerosols of free fibre or any dust.

* The product is applied as a solid encapsulated material and physically pressed into a nail hole. The method to set nails in wood is well known and described in readily available manuals (Readers' Digest 1973). As shown in FIGURE 4, the nail is countersunk below the surface of the wood by placing a "nail set" of slightly smaller dimension than the nail head onto it. The nail is driven in such away as to sink the head below the surface of the wood. It is the small space above the nail head which would be filled with the "Dum Dum Nail Hole." In order to avoid damage to the wood surface, this would be pressed into the hole and the surface left flat or level with the wood surface. As the material is a putty-like consistency with the fibre mixed within the "putty", it would be virtually impossible for the applicator to be exposed to fibres during this work. The time taken to fill a hole might be measured in seconds.

* As the "Dum Dum Nail Hole" would be applied to make the surface of the filler level with the wood, sanding would not be necessary and the surface could be painted or varnished further sealing in any asbestos. The nail hole fillers were produced in colours to match the wood colour and texture of pre-finished woods so that there is only a very remote possibility that the surface containing this filler would ever be sanded.

* In the event that the surface of the wood was sanded, the surface area of the Dum Dum exposed would be extremely small and the depth of any sanding would be minimal, as the purpose of a nail hole filler is to just cover the head of nails. As the asbestos content of the filler was only 2%, it would be virtually impossible even with aggressive sanding for workers

to begin to approach a fraction of the asbestos exposure limits in effect in the USA even today.

* The quantity of "Nail Hole Dum Dum " used on a single board would be extremely small. As the product was recessed in the wood on which the product was used, it is difficult to identify even hypothetical situations under which it could give rise to any measurable exposures for persons disposing of the wood after its useful life.

* It is not known when this product was first manufactured, but it was not manufactured or marketed after 1973.

Overall, it is difficult to conceive of any situation under which "Dum Dum Nail Hole" might have been used, where the concentrations of respirable free asbestos fibres would be measurable above background levels by either conventional phase contrast microscopy or electron microscopy.

Support for a conclusion that chrysotile asbestos fibre exposure when using Dum Dum Nail Hole would be virtually zero is found in the very low levels of fibre exposure associated with the application of similarly encapsulated Dum Dum products described later in this section.

ii. Dum Dum Masonoc.

The 95 series Dum Dum Masonoc contained 12-15% asbestos and was a heavy bodied coating of mastic consistency designed for high build application for filling and bridging of cracks and crevices with application by special brush or heavy duty mastic spray equipment.

There are several reasons to conclude that during the normal application of this product the exposures would be extremely low if not zero:

* The product is of a putty consistency which will not make it easy to generate an airborne dust or aerosol.

* The products are typically applied in the open air as the masonry coatings are designed and recommended only for exterior use.

* It is recommended that if there is an enclosed area in which the product is used, an air supplied respirator be used. For example the label for "Dum Dum Masonoc Soft White 95-W-9" dated 4-73 states...

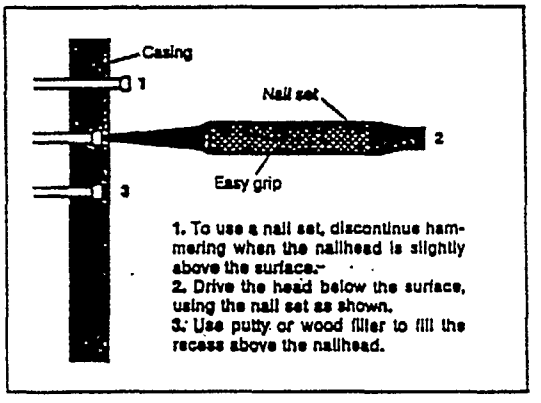
  ".....Use adequate ventilation. Wear an air supplied mask to avoid breathing concentrated fumes in enclosed areas. Keep container closed....."

  Air supplied respirators provide total protection from solvent and fibre exposure as the air is completely separate from that in the immediate work area.

FIGURE 4

- THE USE OF NAIL HOLE FILLER -

From Readers Digest (1973)



* Measurements of the concentrations of airborne asbestos structures during application and removal of Dum Dum Masonoc were all very low. (TABLE 14).

Using the highest concentrations measured, the concentrations were all still less than 0.01 asbestos structures/cc. At 0.01 structures /cc, this represents less than 1/20th of the present OSHA permissible exposure limit of 0.2 fibres/cc and 1/500th of the permissible exposure limit when the product was being widely used in commerce in the early 1970's. SEE APPENDIX E.

iii. Chimney Dum Dum

The 97 Series Chimney Dum Dum provides "the same outstanding protective and waterproofing qualities as the 95 Series Masonoc except that it has been formulated for use on concrete chimneys where additional heat resistance up to 210°F may be required".

The exposures associated with the application and removal of this product were likely to be low for the following reasons:

* As with the previous Dum Dum products, Chimney Dum Dum was of a putty consistency which would make it difficult to generate an airborne respirable dust or aerosol.

* The product was usually applied in the open air as the chimney coatings were designed and recommended only for exterior use.

* The same recommendation as appeared in chemical product data for the Dum Dum Masonoc 95 Series appeared in the literature concerning Chimney Dum Dum 97 Series in 1976 (Mobil 1976E, 1976F). This recommended that, if there was an enclosed area in which the product was used, an air supplied respirator be used. Air supplied respirators provide total protection as the air to be breathed comes from a completely separate supply of air from that in the immediate work area. I f the recommendation by Mobil was properly followed, this would make it impossible for the worker to be exposed to any asbestos.

* The results of the tests carried out by Arthur D Little Inc. (Peters 1990, 1991) showed that the concentrations associated with the application and removal of this product were low. TABLE 14. Indeed, after adjusting the concentrations for fibres that would not be visible by light microscopy, the concentrations were less than 0.01f/cc in all cases but one, which gave 0.02f/cc. A replicate of that measurement in a second test carried out while applying the product gave results of <0.01 f/cc and it is reasonable to conclude that the fibre concentration associated with the use of this product is generally less than 0.01f/cc. Even when the total transmission electron microscopic fibre counts were considered, the highest concentration recorded was still low (0.088f/cc), and well below the current OSHA standard based on phase contrast light optical microscopic fibre counts.

Under normal conditions of application and removal of this product, the concentrations of fibres to

which workers would have been exposed would have been less than one twentieth (1/20th) of the present OSHA permissible exposure limit and less than one five hundredth (1/500th) of the OSHA standard in effect or ACGIH recommended TLV in the early 1970's (5 fibres/cc).

iv Armorcote Dum Dum

This product is asphalt based. Petroleum based products which primarily included asphalt and tar based sealants were studied by Anderson et al (1982) in a report to EPA. They noted that the petroleum sealants contained 5-30% asbestos (primarily chrysotile) and 55-80% asphalt. They noted that other petroleum derivatives added to achieve proper consistency included naphtha, mineral spirits and lighter weight solvents. Other ingredients including rust proofing chemicals, pigments, heat reflecting powdered metals such as aluminium, emulsifiers, resins and clay fillers, were also present in those products.

Of special interest in the EPA study was that airborne fibre concentration measurements were collected for a variety of different materials and operations. The operations monitored varied from spraying cutback asphalt containing 5.8 - 7.7% asbestos on a roof surface to sand blasting a 2.1% asbestos content high performance exterior resin coating from a steel tank. The airborne fibre concentrations measured in the period 1974-1976 did not exceed 0.6f/ml for any of the asphalt tests. The results of those tests are shown in TABLE 15.

The duration of the activity or sampling times were recorded only in the cutback asphalt spraying and lasted approximately 6-7 hours. It is important to note that all these activities are carried on out of doors and not in confined spaces. The EPA report noted that there is no secondary processing necessary of the product and that the fibre releasability was classified as "low". The CPSC, while noting that the product is designed to form a watertight seal for the roofs of industrial plants, warehouses and other flat roofs, concluded under their scenario for fibre release.... "Roof coatings are formulated to remain somewhat flexible, therefore fibre release during abrasion or demolition is not likely". This conclusion is consistent with the conclusion reached in the EPA report in which fibre concentrations reported were very low.

Although only ranges were reported in the report by Anderson et al (1982), means and medians were probably of the same order of magnitude as determined for the Dum Dum materials tested by Peters (1990, 1991). A clear indication exists that these encapsulated products give rise to, at most, extremely low fibre exposure levels for workers. The findings are also consistent in that the fibre concentrations during removal are very low.

While measurements made during the application and removal of Armorcote Dum Dum products were not available, there is good support for concluding that exposure to asbestos during application and removal of the product would be very remote.

Reasons for such a conclusion might be sumarized as follows:

* The product was of a consistency that would make it difficult to generate respirable dusts or aerosols.

* The product was applied in the open air as it was designed and recommended only for exterior use.

* Measurements made in several US States have provided consistent evidence that the concentrations of airborne fibres recorded during the spraying of chrysotile-containing asphalt products and during the removal of such products are very low.

TABLE 15. FIBRE CONCENTRATIONS ASSOCIATED WITH THE SPRAY APPLICATION OF ASBESTOS CONTAINING PETROLEUM - BASED COATING PRODUCTS
(Summarized from Anderson et al 1982)

PRODUCT	ACTIVITY	CONCN *f/ cc*	TIME *mins*	DATE OF TEST
Cut back asphalt	Spraying	0.003- 0.15	342-432	1974
Asphalt-emulsion	Spraying	0.01 - 0.3	NR[50]	1974,76
Built-up roofing	Tear-off	0.1 - 0.4	NR	1974
	Tear-off & Replace	0.0 - 0.3	NR	1974,76
	Spraying	0.0 - 0.6	NR	1974,75,76

* There was a strong recommendation on the labels of containers of Dum Dum Armorcote Black 46-J-9 dated 4-73 stating..

> "..... Use adequate ventilation. Wear an air supplied mask to avoid breathing concentrated fumes in enclosed areas. Keep container closed....."

If followed, this recommendation would make it impossible for such workers to be exposed to any asbestos even if any respirable fibres were rendered airborne.

[50] NR = Not recorded. Phase contrast microscopy was assumed in the original report. Monitoring was done in several US States.

In summary, the levels of exposure for persons using or removing Armorcote Dum Dum products might reasonably be expected to range from zero (if proper procedures are followed) to average workplace concentrations, certainly below 0.1 fibre/cc and most probably below 0.01 fibres/cc.

CATEGORY C (Dum Dum Masonoc Primer)

After the mid-1970's, there would have been no asbestos exposure as the product does not contain commercial asbestos and any talc would then be tremolite fibre free. If any tremolite was present, prior to that time, it would be as a minor contaminant of the talc. As talc represents about 20 % of this product, a 1% tremolite content in the talc would represent only 0.2% of the product.

The product is applied as an ordinary paint and is unlikely to give rise to respirable fibre aerosols during application. After application it is covered by the Dum Dum Masonoc which has already been shown not to give rise to significant fibre exposures.

CATEGORY D

The asbestos contents of the Dum Dum caulking products were in the range 2-13% Product data (Mobil 1976) notes that the materials are present in vegetable oil and that at 24°C the caulking has a viscosity of "putty". It skinned over in 18 hours and could be painted with conventional paints in 24 hours. The caulking products were applied using a caulking gun, trowel or knife. It is evident that the consistency of this product and its application is such that exposure to fibres during application is virtually impossible.

The effectiveness of "encapsulation " in reducing asbestos fibre release was reported by Strassburg (1981) who found that when handling graphite, silicone or teflon-treated asbestos packs there was so little dust production that changeover from asbestos to substitute materials was not required.

The only situation where exposure to asbestos could occur, would be if the user attempted to thicken the caulking by mixing more asbestos into the caulking material. It is evident that under such circumstances the potential would exist for exposure to the free asbestos before mixing but the quantities of asbestos involved would be small, the time adding the fibre short, the occasion rare and lifetime exposures insignificant when compared to those of occupational groups where increased health risks have been demonstrated. As far as can be ascertained Mobil did not supply raw asbestos to add to their product.

As with regular putty, the Dum Dum caulking materials are oil based and designed so that the surface hardens but the underlying product remains in a putty state. In their review of asbestos use in consumer products for the US Consumer Product Safety Commission, Kearney Management Consultants (CPSC 1978) noted that caulking compounds contained 0.5-1% asbestos, somewhat less than in commercial products. The purpose of the asbestos appears to be to "lend viscosity and retains by absorption the oils which insure ductility, thus preventing cracking" . Their scenario for fibre release involved hardening of the compounds through weathering and loss of oils. They postulated

that cracking and embrittlement may result and fibres might then be released during removal of the brittle putty. Their opinions about the release of fibres were not supported by measurements and appear to be contradicted by the measurements made with Hi- Heat Dum Dum.

First & Love (1982) carried out measurements of airborne asbestos dust in a factory manufacturing a high asbestos content caulking compound (putty tape) over a 17 month period in 1979-81. The product was manufactured by kneading asbestos fibres into a mixture of mastic materials. Pertinent to the products under study in this report are the comments by the authors who when referring to the conclusion of the mixing period when the charge was dropped from an open hatch onto a pallet stated....

> "By this point in the process, all of the asbestos and talc has become thoroughly incorporated into a sticky non-dusting plastic mass and release of asbestos fibres is no longer possible"

Concentrations of fibres were determined using the methods recommended by NIOSH. The exposure of the shipping and receiving room worker as measured using a personal sampler was 0.007 f/cc, on the extruder 0.003 f/cc, on the batchmaker 0 - 0.047 f/cc and in all areas of the plant at least an order of magnitude below the then proposed standard of 0.5 f/cc. This would likely be similar to the materials sold under the name " Dum Dum" caulking and exposures associated with the use of the Dum Dum caulking materials might be closer to those of the extruder (0.003 fibres/cc) if measurable.

The information available on Hi-Heat Dum Dum from the experiment by Peters (1990) is more likely to be relevant to the low temperature materials and fit well with the very low concentrations measured during caulking manufacture. See TABLE 14.

i. Hi-Heat Dum Dum

High temperature resinous caulking materials of putty-like consistency are used in sealing joints and crevices on furnaces and boilers to prevent heat loss. One such product, Hi-Heat Dum Dum, is described as a mastic at 24°C. According to product literature, it was designed to remain pliable and withstand expansion and contraction associated with intermittent use. The product consisted of a linseed oil-base vehicle (resin) containing various solid fillers, including clay and chrysotile asbestos added for its resiliency and bridging properties.

The fibre concentrations associated with the application and removal of the material were determined in a special test carried out for Mobil by Arthur D Little (Peters 1990). In the experiment, steel panels were coated and heated to accelerate aging. Subsequently the product was removed from these panels by scraping in three separate 80 minute experiments. During application and removal the concentrations as determined by transmission electron microscopic counts of structures were 0.01 structures/cc or less, the limit of detection of the technique applied. The results are shown in TABLE 14. A reasonable conclusion based on the above results, is that there is insignificant occupational exposure to free asbestos fibres during the application and removal of Hi-Heat Dum Dum.

Based on the fact that this product was exposed to heat (225-250°F for 18 days), increasing its possibility of drying out, results might be expected to provide an upper estimate of the fibre release during removal. Oil based materials which are not so heated would be expected, because of the low vapour pressures of vegetable oils, to remain in a semi solid state throughout their lives.

It is clear that fibre exposures from the use or removal of this product are extremely low, with all "OSHA" fibre concentrations less than 0.01 fibres/cc which is 1/20th of the present OSHA permissible exposure level of 0.2 fibres/cc.

ii. Heating & Ventilating "Dum Dum" .

A product known as " Heating and Ventilating Dum Dum" (46-X-9) was produced in the period 1964-1969. This material was recommended specifically for sealing joints of air ducts in heating and air conditioning systems. The product would have been unlikely to be subjected to high temperatures and so would readily retain its semi-solid state. Experience with all the Dum Dum products tested suggest that they have the same basic characteristics of being associated with low or undetectable levels of fibre release. As it would be highly unlikely that such a material would have been applied, other than using a caulking gun or trowel, exposures associated with its use would be anticipated to be similar to other trowelled on products - very low. This product would be expected to be associated with low or undectectable fibre concentrations during removal also.

UPDATE:

Court decision concerning regulations governing roofing sealants and coatings.

In the original report, in 1992, some analogies were made between the Dum Dum products and asbestos fibres bound in asphalt. Since that time, the OSHA regulations governing asphalt roofing mastics were challenged and the rules overturned.

The Federal Reporter reports that on July 21 1997, the Asbestos Information Association of North America (AIA/NA) petitioned the Court for review of a final rule promulgated by the Occupational Safety & Health Administration (OSHA).

The facts relevant to AIA/NA's claim were as follows:

During the manufacturing process, solid asphalt is liquified by dissolving it in paint thinner, resulting in a thin black syrup. Enough chrysotile or powdered asbestos is mixed into the syrup to make a stiff paste that can be used, for example to seal the crack around a chimney. When the solvent evaporates, the asphalt becomes a tough leather-like film that shuts out water. Roof coatings are manufactured in the same way, except that they have a thinner consistency so that they can be applied with a brush.

The position taken by AIA/NA was that "the manufacturing process encapsulates the fibres in asphalt so that they cannot become airborne and workers cannot inhale or swallow the fibres inadvertently.

There is no evidence in the record that these products have ever been found to cause any worker exposure to asbestos. AIA/NA therefore objects to OSHA's regulations requiring warning labels on the products, notification to building owners when the products are installed and work practice requirements that increase the cost of removal".

The record review by the Court confirmed that: "there was no evidence in the record that asbestos fibres can ever escape from roofing sealants and become airborne; in fact the evidence in the record indicates that they cannot." Neither OSHA nor the AFL-CIO seriously disputed this assertion.

The argument was made by OSHA and the AFL-CIO that it is not possible to distinguish roofing coating from built-up roofing for purposes of asbestos regulations because "other" kinds of roofing materials break down due to weathering.

The decision of the Court was:

" We grant review and vacate the Agency's shipyard and construction standards in so far as they regulate asphalt roof coatings and sealants which contain asbestos."

"We hold that, because of the lack of substantial evidence in the record, the challenged regulations are invalid as to asbestos containing asphalt coatings and sealants."

This decision is fully supportive of arguments made in the original report concerning certain similarities between roofing sealants and Dum Dum products.

For example:

- The scientific evidence leaves little if any doubt that in the case of the Dum Dum products described in the report, the asbestos fibres are encapsulated. This is the situation with asphalt roof coatings and sealants.

- Fibres in Dum Dum are coated with oleoresinous substances which do not "dry out". This renders the chance of releasing free asbestos fibres as low if not lower than that of asphalt coatings and sealants.

- Measurements made during the spraying of asphalt asbestos coatings (Table 14 of original report) and application of Dum Dum products show the concentration of fibre structures to be, if anything lower for the Dum Dum (Table 16 of original report) than for the asphalt product.

- The 8 hour time weighted average elongated structure exposures of roofers during removal of asbestos roofing products ranged from not detectable ND to 0.036 f/cc (Emission Standards & Engineering Division 1990). The elongated structure concentrations during removal of Dum Dum were 0.01 f/cc or less.

The asbestos regulation was challenged by AIA/NA because asbestos roof sealants and coatings are still manufactured. Dum Dum products performed some of the same functions as the sealants (eg: cracks around chimneys) but are no longer sold.

Based on the available evidence, it is reasonable to conclude that if the asbestos containing Dum Dum products were still sold today (in 1999), they would also now be exempt from OSHA regulatory requirements. Clearly, like asphalt sealants, they would result in little or no exposure to free asbestos fibres in a form or at a concentration adequate to pose any practically measurable level of health risk for workers.

The conclusions regarding levels of exposure associated with the use of the various Dum Dum products are unchanged.

14.1.3.2 Duration of exposure

The total exposure of a worker over his/her lifetime is often expressed as a cumulative exposure. This is the sum of the products of concentration and duration of exposure over a working lifetime. In this equation some consideration must be given to the number of hours worked per day with the product. The nature of the products and the most probable users (construction workers) make it unlikely that workers would work continuously with the product day after day. A possible exception might be Dum Dum Nail Filler which may have been used in prefabricated house manufacture where a worker might work all day filling nail holes.

The period of exposure and hence maximum cumulative exposure possible for a worker from working with Mobil-supplied Dum Dum products would be limited by the period over which the products were on the market. Making the following assumptions.

* The worker was exposed for the total period over which Mobil sold the product.

* The worker worked 8 hours/day applying or removing Dum Dum products.

* The average exposure levels associated with the use/removal of the various products were less than or equal to 0.01 fibres /ml.

* No air supplied respirator was used.

The maximum durations of exposure and maximum possible cumulative exposures are shown in TABLE 16.

UPDATE:

There are no changes to the possible exposure periods.

15.0 THRESHOLD LIMIT VALUES AND LEGAL EXPOSURE LIMITS

The American Conference of Governmental Industrial Hygienists (ACGIH) is an organisation which has reviewed and published annually a list of Threshold Limit Values. These have been adopted as guidelines or even legal standards in many countries.
They refer to "airborne concentrations of substances and represent conditions under which it is believed that nearly all workers may be repeatedly exposed day after day without adverse effect."

The levels have changed with time and values for the asbestos minerals are shown in Appendix E. Legal standards for the workplace in the US have been set by the Occupational Safety and Health Administration. They have also changed permissible exposure limits over the years and values are shown in Appendix F.

It appears from the experimental test results that it would have been virtually impossible for workers using Dum Dum products to be exposed to concentrations which exceeded the standards in effect to protect health at the time that the material was used and would not, even during removal of the product, give rise to exposures that would exceed present day exposure limits (FIGURE 5).

UPDATE:

TLVs for the asbestos minerals updated to 1999 are shown in Appendix UE.

Legal standards for the workplace in the US have been set by the Occupational Safety and Health Administration. They have also changed permissible exposure limits. Values updated to 1999 are shown in Appendix UF.

Based on the test results it would have been impossible for workers using Dum Dum products to be exposed to concentrations which exceeded the standards in effect to protect health at the time that the material was used and would not, even during removal of the product, give rise to exposures that would exceed even the Threshold Limit Values or legal standard in existence in 1999 (FIGURE U1).

16.0 RISKS OF HEALTH EFFECTS FOR USERS OF DUM DUM PRODUCTS

There can be several ways of determining whether the risks faced by workers using Dum Dum products were unreasonable or unwarranted. The first makes direct comparisons of the levels of exposure of the workers with the occupational exposure standards[51] that have been established for the substance under consideration. This was done in section 15.0. See FIGURE 5

[51] Occupational exposure standards or limits are generally set by Governments to provide protection for workers within the confines of social acceptability at any point in time.

Another approach is to estimate the risks of disease for persons at their measured or assessed levels of exposure and to situate these risks in relation to other day-to-day risks or risks faced by non-exposed persons. In the following sections such risks are assessed.

TABLE 16. MAXIMUM POSSIBLE DURATIONS OF EXPOSURE AND MAXIMUM CUMULATIVE EXPOSURES OF WORKERS APPLYING DUM DUM PRODUCTS MARKETED BY MOBIL

PRODUCT	YEARS OF EXP.	YEARS No.	CONCN[52] f/cc	CUM.EXP[53] (fibres/cc-yrs)
Dum Dum Armorcote	64-79	15	0.01	0.15
Chimney Dum Dum	64-79	15	0.01	0.15
Dum Dum Masonoc	64-79	15	0.01	0.15
Heating & vent-ilating Dum Dum	64-69	6	0.01	0.06
Dum Dum Calk	64-69	5	0.01	0.05
Dum Dum Nail Hole	64-73	10	0.01	0.10
Hi Heat Dum Dum	64-80	16	0.01	0.16

Workers using Dum Dum products studied in this report have either not been exposed at all (if air supplied respirators were used) or were exposed to extremely low concentrations, orders of magnitude less than the prevailing standards. This would infer that they were protected at levels deemed safe at the time.

UPDATE:

There have been no developments that change this section, other than the lower Occupational Exposure Limit which is still not exceeded.

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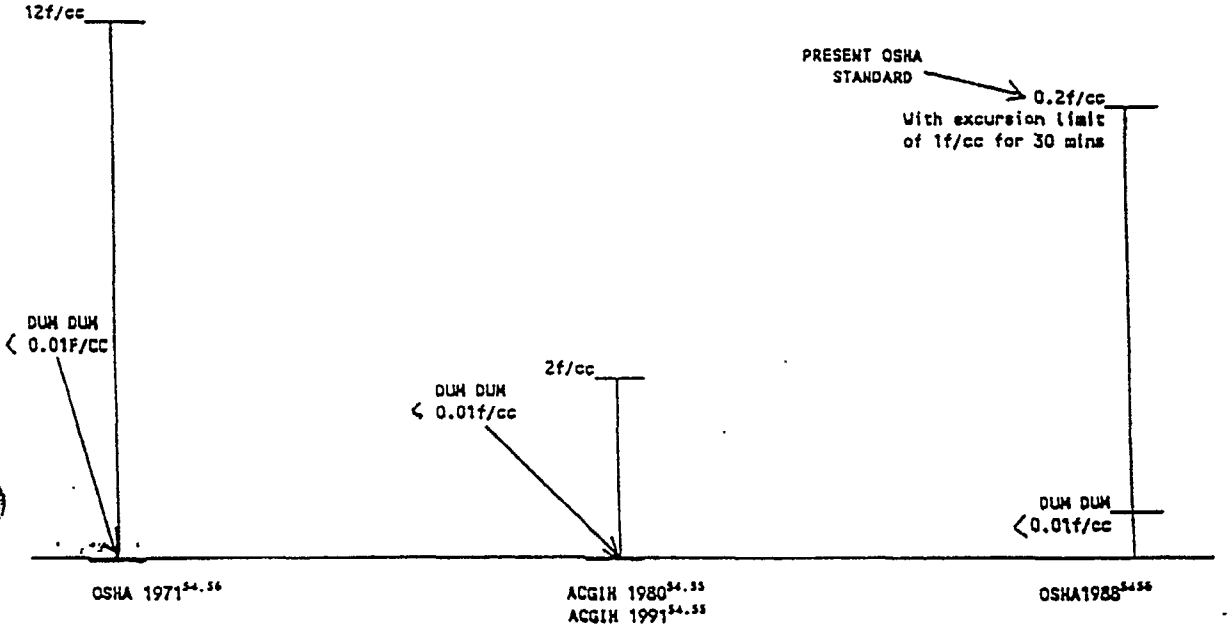
[52] It has been assumed that the maximum PCM equivalent concentrations in TABLE 14 (which were modified from the concentrations expressed in structures/cc measured by Peters (1990, 1991)) applied throughout the period of exposure.

[53] Cumulative exposure has been expressed in fibres/cc-years. Under OSHA counting rules all particles meeting the definition of a fibre are counted as asbestos fibres and concentration expressed as fibres/cc which is synonymous with fibres/ml.

FIGURE 5

OSHA PERMISSIBLE EXPOSURE LIMITS, ACGIH TLV'S FOR CHRYSOTILE ASBESTOS AND THE CONCENTRATION OF ASBESTOS STRUCTURES ASSOCIATED WITH THE APPLICATION AND REMOVAL OF *DUM DUM* PRODUCTS



AGENCY OR ORGANIZATION AND YEAR STANDARD WAS IN PLACE FOR CHRYSOTILE ASBESTOS

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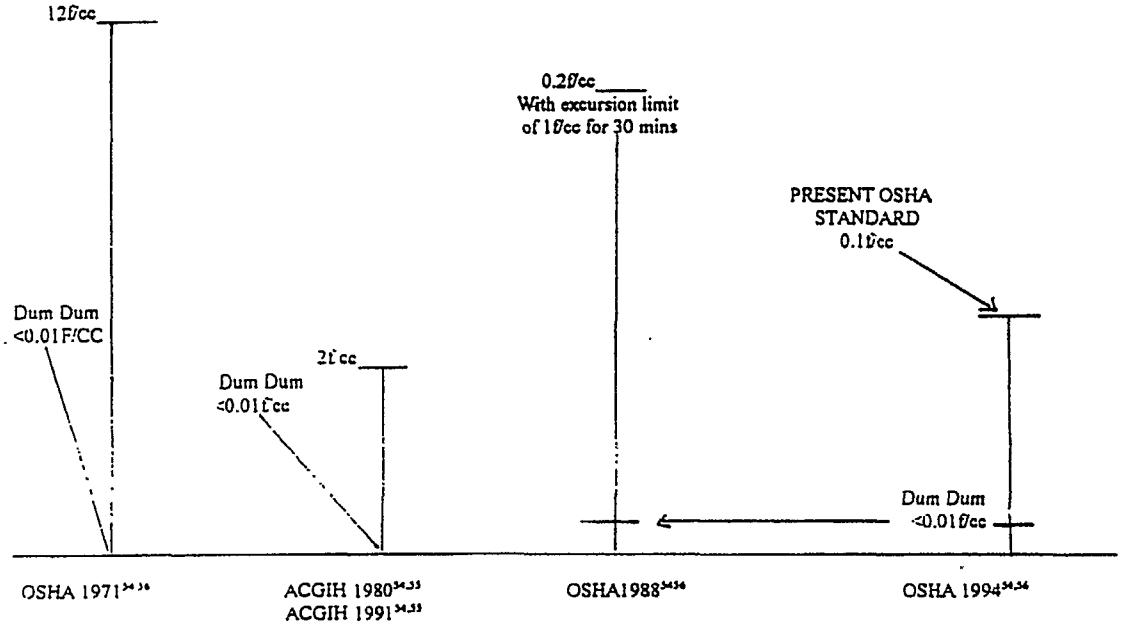
[54] TWA - Time weighted average exposure (8hrs).

[55] TLV - Threshold Limit Value

[56] Permissible exposure limit

FIGURE U1

OSHA PERMISSIBLE EXPOSURE LIMITS, ACGIH TLV'S FOR CHRYSOTILE ASBESTOS AND THE CONCENTRATION OF ASBESTOS STRUCTURES ASSOCIATED WITH THE APPLICATION AND REMOVAL OF DUM DUM PRODUCTS



AGENCY OR ORGANIZATION AND YEAR STANDARD WAS IN PLACE FOR CHRYSOTILE ASBESTOS

[4] TWA - Time weighted average exposure (8HR).

[5] TLV - Threshold Limit Value

[6] PEL - Permissible exposure limit

16.1 RADIOLOGICAL CHANGES

As mentioned earlier in this report, epidemiological studies are rarely based on clinical diagnoses so the true incidence of "asbestosis" is rarely known. More often radiological changes, pulmonary function or clinical signs are assessed and examined in relation to various indices of exposure.

There have been several such studies which, in the absence of studies of Dum Dum workers, might be useful in assessing risks. These include a study of workers exposed to tremolite, which permits an estimation to be made of the effect of exposure if the talc in certain of the Dum Dum products contained tremolite and studies of chrysotile asbestos miners and millers because they were exposed to chrysotile.

An idea of the order of magnitude of tremolite exposures associated with radiological changes was reported in a radiological survey of vermiculite miners (McDonald et al 1986b). This study included workers who had left the company as well as current employees, eliminating to a large extent a common criticism of cross-sectional studies where only healthy workers are examined. They showed that age, smoking and exposure levels contributed to the observed prevalence of radiological change. The relationship between cumulative exposure and the prevalence of pleural changes was statistically less strong than for small opacities.

Estimates, as is usual in such studies, are associated with uncertainties because population size, exposure estimates, reading errors, age, smoking and other environmental variables also contribute to radiological change. However, using dose-response relationships, they estimated that by retirement age, the increase in prevalence of small opacities greater than 1/0 using the ILO-UC classification lay between 5 and 10% for workers with 100 f/cc-years of exposure to tremolite.[57]

There have been at least 4 studies of Quebec chrysotile miners and millers which have provided factors for radiological prevalence. Increases in prevalence per 100 fibre/ml-years of cumulative exposure been identified as 0.2% (Liddell et al 1982); 0.6 (Rossiter et al 1972) and 4% (McDonald et al 1984). The higher value in the latter study probably relates to the inclusion of current and past employees in the survey.

McDonald et al (1986b) noted that no radiological change would be detectable at an average concentration of 0.1 tremolite fibres/cc for 40 years (cumulative exposure of 4 fibre/ml-years) because the variation and errors associated with radiology would be considerably greater than any expected level of risk.

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[57] A study at the same mine by other investigators gave somewhat lower estimates (Amandus et al 1987b). McDonald et al(1986b) concluded that Amandus et al (1987a,b) may have underestimated risks because the 188 men in their study were all current employees.

The upper limit of cumulative exposures associated with the application of Dum Dum products calculated above was 0.16 fibre/cc-yrs. If it was assumed that a worker worked 40 years applying and removing Dum Dum products full time, the maximum total cumulative exposure would be 40 X 0.01 f/cc which is 0.4 fibre/cc-years, which is one tenth of the cumulative exposure level at which McDonald et al (1986b) reported that no radiological change due to asbestos exposure would be detectable.

In fact, the probability of detecting changes in workers today as a result of work with Dum Dum would be even less for several reasons:

a. The exposure-response relationships used in the above calculations were based on the experience of tremolite exposed workers. Workers who used Dum Dum may not have been exposed to tremolite at all. The rates of radiological change resulting from chrysotile exposures are 1.25 to 2.5 times lower than those for tremolite.

b. By 1992, a worker starting to use Mobil marketed Dum Dum in 1964, if working full time on applying and removing it, could only have aquired 28 years of exposure. This would result in a cumulative exposure of 0.28 fibres/cc-years whereas 40 years was used in the calculation.

c. As regulations requiring respiratory protection for persons involved in removing asbestos, have been in place for several years, exposure during removal will have been less than 28 years.

d. It is highly improbable that a worker would work full time, 8 hours/day for 28 years applying or removing only Dum Dum products. Any lesser duration of exposure reduces the risk of radiological change further.

In conclusion, at levels of cumulative exposure associated with the use and removal of the Dum Dum products in this report, radiological changes would not be detectable even after 40 or more years of full time work with them.

UPDATE:

There have been no developments that change the conclusion of this section.

16.2 LUNG CANCER

Information on lung cancer risks in relation to levels of exposure are available for a number of cohorts. The risks vary quite considerably between different occupational groups as shown in FIG 3.

There have been no studies of persons who have spent their working lives with Dum Dum products; indeed, such groups are not likely to exist. On the other hand, construction workers and others who are likely to have used Dum Dum products are exposed to asbestos fibres from other sources. Thus,

data do not exist to directly derive risk estimates and it is necessary to extrapolate from the experience of other groups to the experience of workers using Dum Dum products. This is far from ideal as we need to choose the occupational group whose lung cancer experience will be used to estimate lung cancer risks for workers using Dum Dum.

In order to arrive at risk estimates it has been necessary to make some assumptions. Because the only commercial asbestos fibre type used in Dum Dum products was chrysotile and because it is possible that for some years some of the Dum Dum products may have contained asbestiform tremolite in talc, the lung cancer experience in three industrial situations will be used in assessing risks for the Dum Dum products.

a. the chrysotile textile industry which has been the chrysotile sector associated with the highest levels of lung cancer risk.

b. the tremolite exposure in vermiculite mining.

c. the chrysotile mining & milling industry which has been associated with a low risk/unit exposure.

The use of the first two studies will lead to overestimates of risk. Lung cancer risks in the chrysotile textile industry are considerably greater than in any other chrysotile industry, as are the risks from tremolite exposure; tremolite was only a minor constituent of the airborne dust and over a limited time period, if it was present at all.

Because the exposure-response relationships for lung cancer have been developed in industries where exposures have been orders of magnitude higher than those associated with working with Dum Dum products, it is necessary to extrapolate from these high doses to levels of exposure where the shape of the relationship is not known. For these estimates,it has been assumed that the exposure-response relationships are linear and pass through zero. This is a convenient model. However, it is unlikely that the line passes linearly through zero. Some suggest that a threshold exists below which no effect occurs. There is also a body of opinion that asbestos related lung cancers do not occur in the absence of "fibrosis" and, recently, evidence was presented that this may be the situation (Hughes & Weill 1991). As "fibrosis" may not occur in persons with very low exposures, the assumption of a threshold for lung cancer may be valid. It was shown earlier that the level of exposure anticipated from using Dum Dum products is a small fraction of those at which any radiological change could be detected, one of the markers used in the clinical diagnosis of asbestosis.

If we assume a working lifetime of continuous exposure to Dum Dum of 20 years (4 more years than the period over which Dum Dum products were marketed by Mobil) and a concentration of 0.01 fibres/cc associated with the use and removal of Dum Dum products, the total lifetime cumulative fibre exposure due to working with this product would be at most 20 x 0.01 = 0.20 fibre/ml-years.

This figure is likely to be a gross overestimate of cumulative exposure for several reasons:

* The PCM Equivalent concentrations in only 2 out of 22 tests reached or exceeded a concentration of 0.01 structure/ml. The values were 0.01 and 0.02 respectively and a replicate of the second measurement resulted in a concentration of less than 0.01.

* Workers would rarely be expected to work with a Dum Dum product continuously for 8 hours per day every day for 20 years.

* Some of the products were identified for exterior use only and/or others were recommended to be used with an air supplied respirator, thereby eliminating asbestos exposure.

The number of observed deaths (O) from lung cancer in a working population = the number expected (E) in the workforce without asbestos exposure + the additional number due to the asbestos exposure.

$$O = E + E(y) \times C$$

C = cumulative lifetime exposure and y is the increase in risk per unit fibre/cc-year of cumulative exposure.

As discussed above, the cumulative exposure for workers working 20 years at a concentration of 0.01f/cc = 0.20 f/cc-years.

If we assume that y = 0.01[58] the relationship becomes....

$$O = E + E\,(0.01) \times C$$

Let us further assume a cohort of 1000 men are involved in applying and removing Dum Dum products for a total of 20 years each and calculate the risks in a variety of ways.

a. The crude annual death rate from cancer of the lung, trachea and bronchus in the period 1985-87 in the USA was 75/100,000. Thus if the average life expectancy for working men in the 1000 man cohort were 74, the number of deaths expected over the 74 year period would be 55.50.

$$O = 55.50 + 55.50\,(0.01)x(0.20)$$
$$= 55.50 \div 0.11$$

There would be 0.11 lung cancers found in the cohort in addition to the number expected at US death rates. If we assume that exposure started at age 20 there would be 54 years of life expectancy left when the 1000 men started work with the Dum Dum. Hence the number of additional lung cancer deaths per annum due to exposure would be 0.11/54 = 0.002/annum.

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[58]This 0.01 is based on the estimated increase in risk with tremolite of 1% for each fibre/ml-year of exposure (McDonald et al 1986a).This is about the same as determined by Hughes et al (1987) for an asbestos cement plant and by McDonald et al (1983a), McDonald et al (1983b) and Peto et al (1985) for textile plants. The use of a 0.01 figure will represent an upper limit of risk, the risk based on other chrysotile dose-response curves being between perhaps 1/5th and 1/50th or more of this value.

b. If the above calculation is carried out using the eventual probability of developing lung cancer for a person of 20 years of age based on the cancer statistics for 1970 (American Cancer Society 1978), the expected number of deaths from lung cancer would be 1000 x 5.07 = 50.70

$$O = 50.70 + 50.70\ (0.01)x(0.20)$$
$$= 50.70 + 0.10$$

This would translate into 0.0019 additional lung cancer deaths /annum in the cohort.

c. A third method is to use the fractional increases in risk /unit exposure calculated by Nicholson (1986). He determined the fractional increases in lung cancer risks per fibre/ml-year of exposure (K_L), along with adjustments for possible biases, estimates of statistical variation and uncertainties associated with exposure estimates in various studies. He used a K_L of 0.01 in calculating lifetime risks for persons with 0.01 f/cc-yrs of exposure. He calculated, where smoking habits were not considered, that the lifetime risk for men with an age at first exposure of 20 and with 20 years of exposure was 59.5/100,000. As this reflects exposures for 24 hours/day it must be divided by 4.2 to bring the rate back to that for an 8 hr /day exposure which yields a risk of 14.2 /100,000. For a cohort of 1000 men this would be 0.14/1000. This would mean an additional 0.0026 lung cancer deaths per annum in the cohort for a life expectancy of 74.

d. Calculations by Doll and Peto (1985) quoted in the Nicholson (1986) report gave a lifetime risk of 25.2/100,000 for men exposed 35 years from age 20 at a concentration of 0.01f/ml. Assuming linearity, this would mean a risk of about 14.4/100,000 for men exposed 20 years from age 20 at 0.01 fibres/ml. This is 0.14/1000.

Thus, there is considerable consistency in the estimates of risk, all life-time risks being in the range 0.10-0.14/1000 or 0.0019 -0.0026 additional lung cancers due to exposure each year in a cohort of 1000 men. These figures are already small compared to other risks. See TABLE 17.

Exposure in the exposure-response curves on which risks for occupational groups are derived, are based on phase contrast microscopic (PCM) fibre counts. This is why the PCM-equivalent values have been used. However, even if we were to use the mean of the three highest concentrations recorded by transmission electron microscopy, in the experiments by Arthur D. Little Inc (0.045 fibres/cc), there would still be less than 1 death predicted as due to asbestos exposure in the cohort of 1000 men. There will be approximately 50 lung cancer deaths due to other factors.

In practice, these risks will be considerably lower because the values used to calculate risk were the same or similar to those for the textile industry which carried risks much greater than for any other chrysotile industry sectors. The slopes of the lines from various dose-response relationships for chrysotile can range as high as two order of magnitude. McDonald et al (1983a) has estimated that the risk in the chrysotile textile industry sector are 50 times those in chrysotile mining and milling. The ratio of the K_L values for predominantly chrysotile textiles and chrysotile mining and milling calculated by Nicholson(1986) was 20.

If the non-textile risks for chrysotile exposure were considered, as is probably more appropriate, the annual increase in the rate of lung cancer due to asbestos exposure from the product based on mining and milling experience would be at most an average of 0.00013 lung cancer deaths /annum in a cohort of 1000 men. This has assumed 20 years continuous exposure which is extremely unlikely, so actual risks are smaller still.

While calculations based on estimated increases in risk per unit exposure developed using different assumptions may provide slightly different risk estimates, the order of magnitude of the calculated risks at this concentration are in fact very similar, all are very low and in practice not detectable.

An increased risk of lung cancer was not detected at levels of exposure several fold greater than these, in the friction materials manufacturing industry with a workforce of 13,460 persons (Newhouse and Sullivan 1989).

UPDATE:

Lash et al (1997) carried out a meta-analysis of the relation between cumulative exposure to asbestos and relative risk of lung cancer. They assumed linear relationships and there were other assumptions made in order to carry out the meta-analysis. They reported that estimates of the study specific dose-response coefficient ($K_{1,i}$) ranged from zero to 42 x 10^{-3} ml/fibre-year.

Under the fixed effect model, a maximum likelihood estimate (MLE) of the summary measure of the coefficient (K_i) equal to 0.42 x 10^{-3} (95% confidence intervals (95% CI) = 0.221 to 0.69 x 10^{-3}) ml/fibre-years was obtained. Thus under a fixed effects model, the estimate of K_i by Lash et al was 24-fold lower than OSHA's (ie: the value reported by Nicholson of 10 x 10^{-3} noted in calculation c. above).

Under the random effects model implemented because there was substantial heterogeneity in the estimates of K and zero dose intercepts (A_i), (the model that they preferred), a MLE of K_i = 2.6 x 10^{-3} (95% CI 0.65 to 7.4 x 10^{-3}) was found. The potency K_i was fourfold lower than calculated by the US Occupational Safety and Health Administration (OSHA).

The reported values for the various "chrysotile only" cohorts summarized by Lash et al are listed below. In fact, all the textile cohorts had some amphibole fiber exposure, some substantial.

Chrysotile textiles:

Dement et al (1994)

K_i= 24 x 10^{-3} (95% CI = 11 - 48 x 10^{-3})
(A= 1.32)

Dement et al (1983)

$K_i = 28 \times 10^{-3}$ (95% CI = 8.4 - 90 x 10^{-3})
(A= 1.56)

McDonald et al (1983)

$K_i = 42 \times 10^{-3}$ (95% CI = 15 - 120 x 10^{-3})
(A= 1.09)

Peto et al (1985)

$K_i = 4.1 \times 10^{-3}$ (95% CI = 0.8 - 9.8 x 10^{-3})
(A= 1.09)

McDonald et al (1982)

$K_i = 36 \times 10^{-3}$ (95% CI = 13 - 110 x 10^{-3})
(A= 0.53)

Friction products

McDonald et al (1984)

$K_i = 0$ (95% CI = 0 - 3 x 10^{-3})
(A= 1.6)

It should be noted that the study by Berry & Newhouse which also did not have a slope different from zero (ie: $K_i = 0$) was not included in the study.

Mining & milling

Piolatto et al (1990)

$K_i = 0.2 \times 10^{-3}$ (95% CI = 0 - 3 x 10^{-3})
(A= 1.01)

Liddell et al (1977)

$K_i = 0.5 \times 10^{-3}$ (95% CI = 0.2 - 0.9 x 10^{-3})
(A= 0.76)

McDonald et al (1980)

$K_i = 0.5 \times 10^{-3}$ (95% CI = 0.2 - 0.9 x 10^{-3})
(A= 0.96)

McDonald et al (1993)

$K_i = 0.2 \times 10^{-3}$ (95% CI = 0.1 - 0.4 x 10^{-3})
(A=1.22)

The effect of applying either of the new estimates (ie: 0.0026 or 0.00042) instead of 0.01 would be to reduce the risks based on Nicholson (1986) in the 1992 report even further. When the mining and milling slope noted by Lash et al was applied using the mining risks of McDonald et al (1993), which probably represent the closest to pure commericial chrysotile exposure,

$$E_{i,j} = A_i (1 + K_{i,i} d_{i,j}) e_{i,j} \quad K_{i,i} \geq 0.$$

$e_{i,j}$ = population based expected number of deaths from lung cancer.
$d_{i,j}$ = cumulative exposure.
$E_{i,j}$ = number of deaths from lung cancer expected under the model for exposure $_i$ in study $_i$.
A_i = fitted interent corresponding to the relative risk of lung cancer among the cohort at zero exposure.
$K_{i,i}$ = a measure of potency and the coefficent relating cumulative exposure to asbestos to relative risk of lung cancer in study $_i$.

Assume that the crude lung cancer rate in the US is 75/100,000 and that there are 1000 men with a life expectancy of 74. The number of deaths over the 74 year period would be 55.5 (ie: 75/100,000 x 74) . If we assume that these men were continuously exposed at 0.01 f/cc for 8 hours per day for 20 years.

The expected number of deaths $E = 1.22 (1 + 0.2 \times 10\text{-}3 \times 0.01 \times 20) e_{i,j}$

$E = 1.22 (1 + 0.2 \times 10^{-3} \times 0.01 \times 20) 55.5$

The excess lung cancer deaths $= 1.22 (0.2 \times 10^{-3} \times 0.20) 55.5 = 2.7 \times 10^{-3}$

Thus 0.0027 men would die of lung cancer in a population of 1000 workers exposed to chrysotile from Dum Dum for 20 years at an assumed concentration of 0.01 fibre/ml. The fibre concentration is in fact less than this. This is a lifetime risk of 0.0000027 or 2.7/million.

In fact the risk will be even lower than this. In conclusion, the more recent data, show that as reported in the 1992 report, the lung cancer risks for workers working with Dum Dum are extremely low and possibly zero.

Based on their study of lung cancer in women residents living near chrysotile mines Camus et al (1998) reported that the EPA overestimated lung cancer risks associated with chrysotile 10 fold. They found an SMR of 0.99 (95% CI = 0.78 - 1.25) ie: no increased risk of lung cancer at cumulative exposure of less than 25 fibres-year/ml.

As noted in the 1992 report, the studies by Newhouse and Sullivan found no increased risk of lung cancer in friction product manufacturing workers and that cohort included workers with cumulative exposures as high as 356 f/cc-years. Also the K_i value for the other friction product plant was reported by Lash et al (1997) as zero.

16.3 MESOTHELIOMA RISKS

The factors which may be involved in mesothelioma causation were described in an earlier section. The characteristics of the Dum Dum products were also described. If we examine these in relation to the likely use of the Dum Dum products, the following would appear to be the situation.

* The asbestos used in Dum Dum is chrysotile. There is general agreement in the scientific literature that the risks of mesothelioma for workers exposed to chrysotile are considerably less than those for workers exposed to amphibole fibres. The mesothelioma risk may not be due to chrysotile but to amphibole contaminants associated with certain chrysotile fibre occurrences.

* The concentrations as measured using Hi-Heat Dum Dum were 0.01 asbestos structures/cc or less.

As in the case of lung cancer, it is necessary to extrapolate from risks at high levels of exposure in industries where the mesothelioma exposure data exist in order to quantify risks at low dose levels. Unlike the lung cancer situation, mesothelioma is a rare disease and reliable background rates to calculate the expected numbers of deaths from this cause are not available. The incidence of the disease is therefore calculated using a model based on observations in a number of studies. This model assumes that incidence rates increase as

a power of the time since exposure began, that incidence is proportional to concentration and includes duration of exposure based on multi-stage carcinogenicity.

The model used by Hughes (1989) can be expressed mathematically as...

Incidence of Mesothelioma at time t = $KC\ (t^{3.2}-(t-d)^{3.2}$ where t>d

C = concentration ; t = time since exposure began; d = duration of exposure

Hughes proposed that while there was considerable variability in the estimates of K from various studies "for mixed asbestos exposures, the median value of 0.2×10^{-8} provides a reasonable estimate" of K (Hughes & Weill 1986). This value would take account of any tremolite contamination in the talc and has been used here to provide an upper estimate of risk.

Hughes (1989) calculated, using lifetable methods, that for a cohort of 100,000 men exposed 20 years to 0.001f/ml starting at age 20 there would be 0.9 mesothelioma deaths by age 80. If these values

are expressed in terms of the number of deaths that would be seen in a cohort of 1000 workers exposed for 20 years at 0.01f/ml there would be about 0.09 deaths. This, over 60 years would be an average of 0.0015 mesothelioma deaths per annum in the 1000 man cohort.

Hughes used a factor of five to calculate the number of mesotheliomas that would be associated with chrysotile exposures[59]. Under this scenario there would be 0.018 deaths from mesothelioma if 1000 men were followed from age 20 until 80 years of age.

The levels of exposure of workers who worked with Dum Dum products are so low that one would not expect to see a single mesothelioma related to work with Dum Dum in a cohort of 50,000 workers working full time with the products for 20 years.

UPDATE:

It is now almost certain that workers in the Quebec chrysotile mining cohort did not get their mesotheliomas as a result of chrysotile exposure but through amphibole fibre exposures.

Based on the latest mining data there were at most 38 mesothelioma deaths (several of them not mining related) in about 10,000 workers and 8000 deaths followed from 1935-1993 (ie almost a lifetime for all cohort members). Some of these mesothelioma diagnoses are also questionable, so the number of mesotheliomas may be less.

No case of mesothelioma had less than 2 years of employment in the Quebec asbestos mines and mills. It is known that exposures were extremely high in the past (100s of fibres/cc). Based on the data from McDonald & McDonald (1997) the average exposure of mesothelioma referents was 17.4 mppcf in the central area of Thetford Mines where most of the risk was found. On this basis, if we assume 20 years of exposure and a particle to fibre conversion factor of 3:1, this would be about 1000 f/cc-years. Workers exposed to Dum Dum products for 20 years continuously would have had lifetime exposures of less than 0.2 fibre/cc-years.

If the risk is linearly related to cumulative exposure, this would mean that the proportional mortality from mesothelioma in workers using Dum Dum products would be 38/8000 x 0.2/1000 = 0.00000095 = 0.95/million deaths. This is a lifetime risk which is a tiny fraction of the lifetime risk due to the background risk of mesothelioma of about 2 per million population per annum or 140 per million.

As it appears that the mesotheliomas in the Quebec cohort are related to crocidolite (in the factory) and tremolite exposure (in the mine and mills) the risk would be lower still.

[59] This assumes that any mesotheliomas in chrysotile exposed populations are due to the chrysotile exposure which may not be the case.

16.4 COMPARISONS

In order to understand statements of risk, it is useful to place them in the perspective of risks faced on a daily basis. One way to do this is to express risks as an annual rate or lifetime risks/100,000 persons as has been done by Commins (1985). See TABLE 17.

In the previous section (16.3), it was found that, assuming a chrysotile only exposure, the number of deaths due to mesothelioma among 1000 Dum Dum workers exposed to 0.01f/ml for 20 years and followed from age 20 to age 80 was 0.018.

This assessed risk of death from mesothelioma could be expressed as a balance of lifetime risk of about 1.8/100,000 which is less than the lifetime risk of being struck by lightning.

It must be remembered that calculations of risk have assumed a conservative linear extrapolation through zero. It is probable that at very low concentrations, risks cease to be linear as clearance and other mechanisms cannot be overwhelmed and can act effectively to protect cells. A less conservative view which may be correct is that their exists a threshold of exposure below which no effects occur. In practice, this threshold exists because at such low risks, the risk becomes undetectable epidemiologically or experimentally.

The assessed risk of death from lung cancer was expressed as an increase in the annual average death rate from lung cancer which, for the same exposure scenario as for mesothelioma, resulted in an annual average death rate increase for men of 0.10 -0.26/100,000. This is equivalent to a balance of lifetime increase in risk of 5.4 to 14/100,000. This is a very low risk and much lower than the risk of cancer from x-rays or dying in a transcontinental airline crash.

In considering whether an individual who has worked with Dum Dum might have a cancer related to that exposure, it is necessary to recognise that the risk associated with the asbestos exposure due to that product is so low that almost all other identified non-asbestos and societal factors pose considerably greater risks of lung cancer and are the more likely etiological factors.

If we compare the exposure of someone using Dum Dum full time for 20 years, with the exposure of mesothelioma cases in the Quebec chrysotile mining and milling industry, we find: that the lowest lifetime cumulative exposure of a mesothelioma case in the Quebec chrysotile mining and milling industry was 59 fibre/ml years (6.7 fibres/ml for 8.8 years). A person exposed to Dum Dum at the concentrations determined during the use of the Hi-Heat product would need to work at least 5,900 years to achieve this exposure.

TABLE 17. LIFETIME RISK VALUES FOR SELECTED SITUATIONS
Commins (1985)

Selected risk situations, mainly U.S. data	Lifetime risk per 100,000
EXTRA HIGH RISK	
Smoking (all causes of death)	21,900
Smoking (cancer only)	8,800
HIGH RISK	
Motor Vehicle, U.S.A 1975 (deaths)	1,600
ELEVATED RISK	
Frequent airline passenger (deaths)	730
Cirrhosis of liver, moderate drinker	290
Motor Accidents, pedestrians U.S.A 1975 (deaths)	290
Skiing 40 hours per year (deaths)	220
MODERATE RISK	
Light drinker, one beer per day (cancer)	150
Drowning deaths,all recreational causes	140
Air Pollution U.S.A Benzo(a) pyrene cancer	110
Natural Background radiation, sea level (cancer)	110
Frequent airline passenger, cosmic rays cancer	110
LOW RISK	
Home accidents, U.S.A.,1975 (deaths)	88
Cycling (deaths)	75
Person sharing a room with a smoker (cancer)	75
Diagnostic x-rays U.S.A (cancer)	75
Risk level where few would commit own resources to reduce risk; Royal Society, London (1983)	**70**
VERY LOW RISK	
Person living in brick building, additional natural radiation (cancer)	35
Vaccination for small pox, per occasion (death)	22
One transcontinental air flight/year (death)	22
Saccharin, average USA consumption (cancer)	15
Risk level where few would consider action necessary, unless clear causal links with consumer products, Royal Society, London 1983	**7**
EXTREMELY LOW "RARE EVENT" RISK	
One transcontinental air flight per year, natural radiation (cancer)	4
Lightning	3
Hurricane deaths	3
Charcoal Broiled steak,one per week	3
Environmental asbestos risk,[60] 1985, (cancer)(around 1 per 100,000 or lower)	1
"Acceptable" risk: World health Organization for drinking water, 1984, (cancer)	**1**
(Further control not justified, Royal Society, London 1983	**0.7**

[60] Excludes possible effects of smoking.

UPDATE:

Cigarette smoking risks can put into perspective the order of magnitude of lung cancer risk from Dum Dum. Based on Siemiatycki et al (1994), a person smoking 20 cigarettes per day (ie: 1 pack) for 40 years would have a cumulative smoking history of 800 cigarette years (or 40 pack years). Their risk of dying with lung cancer would be 13.2 times that of a non-smoker.

The annual death rate of a non-smoker based on the British physicians study was 10/100,000 and for a person smoking 14-24 cigarettes per day was 127/100,000 (Doll and Peto 1976). This was based on rates derived from the 1950's and 1960's. The CPS II rate for a 50-60 year old man smoking 20 cigarettes/day for 40-44 years is 323.9/100,000. The CPS II rate for 55-59 year old male never smokers is 5.3/100,000.

The additional risk of dying from lung cancer for a worker, even if continuously exposed to chrysotile from Dum Dum for 20 years was calculated to be 2.7 per million as a lifetime risk which is on average 0.04 per million annual risk or a tiny fraction of the usual risk for a non-smoker dying of lung cancer.

Given the nature of the product, no worker would work with it continuously for 20 years, or even a small fraction of that, so risks would be even less.

Dr John Paling (1992) has introduced the "Paling Perspective scale" and has described risks on this scale. Some of the risks reported by Dr Paling are shown in TABLE U17.

It can be seen that the risks of cancer associated with working with Dum Dum are below those that EPA regulates and at a level that FDA considers too low to be of concern and well below that associated with eating a peanut butter sandwich each day for a year.

It should be noted that as the Quebec cohort has now been followed up for many more years, it is not known whether 59 fibres/cc-years is still the lowest exposure of a mesothelioma case in that industry as an updated exposure has not been published. However that comparison is not needed to conclude that the risks of working with Dum Dum are well below those where Governmental action is taken to reduce the risk. This is intrinsically recognised by the decision to eliminate regulatory controls on asphalt products which fall into a very similar category to the Dum Dum products.

TABLE U17

EXAMPLES OF RISKS REPORTED BY DR JOHN PALING.

RISKS/100,000	CATEGORY	RISKS WITH WHICH WE ARE AT HOME
0.1-100		US EPA regulates so that risks fall in or below this range.
0.1	Homebase	US Food & Drug Administration point below which any risk from a food additive is considered too small to be of concern.
0.18	Homebase	Risk of drowning in tub this year.
0.4	Homebase	Risk of resident being killed by crashing airplane.
1.0	Homebase	Extra risk of cancer from eating peanut butter every day for 1 year.
1.0	Homebase	Extra risk from living in Denver compared to New York for 1 year.
8	Homebase	Risk of mother dying in birth of single child.
10	Rapidly increasing risk	Risk of death from accidents at home in one year.
10		Risk of being murdered in the US.
1000	Rapidly increasing risk	Risk of highway death from 50 yrs of driving.
10,000	Risk massive	Risk of dying from cancer-smoking 1 pack of cigarettes per day for 30 years.

17.0 CONCLUSIONS

* It is highly improbable that the levels of asbestos exposure by persons using Dum Dum products continuously over a 20 year period would be adequate to give rise to detectable "radiological changes" compatible with asbestosis.

* It is highly improbable that the levels of asbestos exposure by persons using Dum Dum products continuously over a 20 year period would be adequate to produce detectable increases in lung cancer or primary malignant mesothelioma death rates.

* The risk of mesothelioma, lung cancer and lung fibrosis associated with the use of the Dum Dum Products are so low that other sources of exposure and other factors deserve a much higher priority for consideration in determining the etiology of cancers or radiological changes.

The basis for these conclusions is as follows:

* It is extremely improbable that workers could be exposed to significant levels of asbestos fibres during the normal application and removal of Nail Hole Dum Dum, Dum Dum Masonoc, Hi-Heat Dum Dum, Chimney Dum Dum, Dum Dum Calking or Dum Dum Armorcote and it is likely that the same applies to other similar Dum Dum products.

    - The products are putty like and generally applied outside.

    - Mobil product literature recommends that workers use air supplied respirators during application to protect themselves from solvent exposures in enclosed areas. This would totally eliminate even the potential for exposure during application of the products so labelled.

- Air measurements using personal and area samples made during the application and removal of Dum Dum Masonoc, Chimney Dum Dum and Hi-Heat Dum Dum have shown very low concentrations of "fibrous" structures (0.01 structures/cc or less).

    - These concentrations are almost all less than 0.01 asbestos structures/cc, low even when all structures visible by transmission electron microscope are considered.

- Measured concentrations are consistent with the literature concerning low concentrations associated with the production and use of similar products.

* Concentrations, when expressed in terms to assess their compliance with OSHA permissible exposure limits, show them to be approximately 1/10th - 1/20 th of the present standard of 0.2 fibres/cc and as much as 1/500th of the OHSA standard of 5 fibres/cc in effect in the early 1970's when the products were in use. Thus, workers have not been exposed at levels which would put them at a level of risk above that determined to be acceptable to the US Government.

* In the absence of studies of "Dum Dum" product applicators and removers, risks have been estimated using assumptions which would tend to overestimate risk. Assuming daily exposure for 8 hours for 40 years at the maximum measured airborne fibre concentrations associated with the use of Hi-Heat Dum Dum, Chimney Dum Dum and Dum Dum Masonoc (< 0.01 fibres/cc), even very minor radiological changes would not be detectable. Forty (40) years is more than twice the time that Mobil has marketed the product, and it will not be possible for a person to achieve this exposure before the year 2003.

* The risk of lung cancer at the levels of exposure associated with the application and removal of the Dum Dum products over 20 years may be 0 and at most classifiable as an extremely low or rare event. Even after 20 years of continuous exposure, at levels determined by laboratory tests of the product, the risk is considerably less than that calculated for sharing a room with a smoker.

* The risk of mesothelioma for workers spending 20 years applying and removing Dum Dum products tested by AD Little and Dum Dum products giving rise to the same levels of exposure is less than the lifetime risk of being struck by lightning.

UPDATE:

There has been no new information since 1990 to change these conclusions. Rather, the most recent data reinforces them.

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APPENDICES

A. MSDS FOR RED DUM DUM BOILER PUTTY

B. MSDS FOR DUM DUM HIGH TEMP BOILER PUTTY

C. DUM DUM PRODUCT LISTS

C1. DUM DUM NAIL HOLE PRODUCTS

C2. CAULKING PRODUCTS

C3. CAULKING PRODUCTS-HIGH TEMPERATURE CAULKINGS

C4. OTHER DUM DUM PRODUCTS

D. SIZE OF FIBRES OBSERVED IN AIRBORNE "DUST" SAMPLES

D1. SIZES OF "ASBESTOS STRUCTURES" IN AIRBORNE DUST SAMPLES COLLECTED DURING THE TESTING OF DUM DUM MASONOC

D2. SIZES OF "ASBESTOS STRUCTURES" IN AIRBORNE DUST SAMPLES COLLECTED DURING THE TESTING OF CHIMNEY DUM DUM

D3. SIZES OF "ASBESTOS STRUCTURES" IN AIRBORNE DUST SAMPLES COLLECTED DURING THE TESTING OF HI-HEAT DUM DUM

E. TLV'S 1946-1991

UE. THRESHOLD LIMIT VALUES (TLV'S) ESTABLISHED BY THE GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH)

F. FEDERAL REGULATORY PROGRAM - OSHA

UF. FEDERAL REGULATORY PROGRAM OSHA REVISED AND UPDATED TO INCLUDE 1971-1999

G. DUM DUM PRODUCT LABELS

H. DUM DUM PRODUCT DATA

UI. REPORTS FROM ARTHUR D. LITTLE INC.

APPENDIX A

MATERIAL HEALTH AND SAFETY DATA SHEET

Product: Red Dum Dum Boiler Putty Chemical Formula:

Other Names:

Manufacturer – Name: The Presco Co., Ltd.

Address: 6345 Netherhart Rd., Mississauga, Ont. L5T 1B8

Emergency Phone No.: (416) 670-8282 Name of Contact: R. C. Smith

II. COMPONENTS

Chemical Ingredients	TLV	%
Soybean Oil, Binding Oil, Petrofin 100, Petrofin 0, Red Oxide pigment, Crushed Limestone, Nytal 300, (talc Filler), Dicalite powdered filler, Hedmanite Hi-Temp earth filler, Varsol, Traces of Clearate, Fatty Acid Oil, Cobalt – trace, Exkin,- Trace.		

(Include any chemicals listed in the ACGIH TLV Booklet or Ontario Government Regulation)

III. HEALTH HAZARD INFORMATION

Route	Effects (Include both immediate and long-term effects)
INHALATION ☐	None Non Hazardous
EYE CONTACT ☐	Wash eyes with cold water. If inflamed, contact Doctor.
SKIN CONTACT ☐	None. Non-hazardous.
SKIN ABSORPTION ☐	Insignificant. Non-hazardous.
INGESTION ☐	A caulking type mastic. If ingested, see Doctor. Ingestion to be avoided.

IV. HANDLING PRECAUTIONS

None known.

(Include recommendations for personal protective equipment and controls)

INDUSTRIAL ACCIDENT PREVENTION ASSOCIATION
23rd FLOOR, 2 BLOOR ST. EAST, TORONTO, ONT. M4W 3C2

Print

FIRE AND EXPLOSION DATA

...ing ...int	Flashpoint	Flammable Limits	LEL / UEL
nil	nil	Dries out at 550°F	

REACTIVITY

STABLE [X]	UNSTABLE [ ]	HAZARDOUS DECOMPOSITION PRODUCTS	INCOMPATIBILITY
	(List conditions to avoid) None known	(List) None	(List materials to avoid) None known

EMERGENCY AND FIRE FIGHTING PROCEDURE

Non flammable

VI. SPILLAGE CLEAN-UP AND WASTE DISPOSAL PROCEDURE

Non spillable
No special waste disposal required.

...I. FIRST AID

INHALATION	Not required
EYES	Wash eyes with cold water..
SKIN	Not required
INGESTION	See Doctor

VIII. STORAGE INSTRUCTIONS

No special requirements

IX. ADDITIONAL INFORMATION

Date Aug 12/91 Signed

APPENDIX B

M S D S

Canadian Centre for Occupational Health and Safety

*** IDENTIFICATION ***

RECORD NUMBER : 47412
LANGUAGE : ENGLISH
PRODUCT NAME(S) : Dum Dum Hi Temp. Boiler Putty

*** MANUFACTURER INFORMATION ***

MANUFACTURER : The Presco Co Ltd
ADDRESS : 6345 Netherhart Road
Unit 7
Mississauga Ontario
Canada L5T 1B8
Telephone: 416-670-8282
EMERGENCY TELEPHONE NO.(S) : 416-670-8282
ESSENTIAL INFORMATION : For further information please contact R.C. Smith

*** MATERIAL SAFETY DATA ***

MATERIAL HEALTH AND SAFETY DATA SHEET

I.

Product Dum Dum Hi Temp. Boiler Putty Chemical Formula

Other Names

II. COMPONENTS

Chemical Ingredients	TLV	%
Soybean Oil, Binding Oil, Petrofin 100, Petrofin O, Red Oxide Pigment, Crushed Limestone, Nytal 300 Talc Filler, Dicalite Powdered Filler, Asbestos, Varsol. Traces of Clearate, Fatty Acid Oil, Cobalt, exkin.		

(Include any chemicals listed in the ACGIH TLV Booklet or Ontario Government Regulation)

III. HEALTH HAZARD INFORMATION

ROUTE		EFFECTS (INCLUDE BOTH IMMEDIATE AND LONG-TERM EFFECTS)
INHALATION	[ ]	None Non-hazardous.
EYE CONTACT	[ ]	Wash Eyes with cold water. If inflamed, contact Doctor.
SKIN CONTACT	[ ]	None Non-hazardous.
SKIN ABSORPTION	[ ]	Insignificant Non-hazardous
INGESTION	[ ]	This product is a caulking type mastic. If ingested, see Doctor. Ingestion to be avoided.

IV. HANDLING PRECAUTIONS

None known

(Include recommendations for personal protective equipment and controls)

V. FIRE AND EXPLOSION DATA

Boiling Point Nil
Flashpoint Nil
Flammable Limits Dries out at 550 deg F
LEL
UEL

REACTIVITY

STABLE [X]
UNSTABLE [ ] (List conditions to avoid) None known
HAZARDOUS DECOMPOSITION PRODUCTS (List) None
INCOMPATABILITY (List materials to avoid) None known

EMERGENCY AND FIRE FIGHTING PROCEDURE

Non flammable

VI. SPILLAGE CLEAN-UP AND WASTE DISPOSAL PROCEDURE

Non spillable

No special waste disposal required.

VII. FIRST AID

INHALATION	Not required
EYES	Wash eyes with water.
SKIN	Not required
INGESTION	See Doctor.

VIII. STORAGE INSTRUCTIONS

No special requirements

IX. ADDITIONAL INFORMATION

Date Signed

APPENDIX C

APPENDIX C1

DUM DUM NAIL HOLE PRODUCTS.

The date of initial production was unknown. The reported year of cessation of marketing of this product was 1973. Nail Hole Dum Dum products had various designations.

77-739 Dum Dum Nail Hole Dark Walnut
Asbestos 2%
Start -
Stop 1973
Trade Name Dum Dum Nail Hole Dark Walnut.
G-H Number 3720-599-LX32

77-171 Dum Dum Nail Hole Natural Walnut
Asbestos 2%
Start -
Stop 1973
Trade Name Dum Dum Nail Hole Natural Walnut
G-H Number 3721-199-LX115

77-539 Dum Dum Nail Hole Lt Luan
Asbestos 2%
Stop 1973
Trade Name Dum Dum Nail Hole Lt Luan
G-H Number 3720-499-LX203.

Dum Dum Nail Hole Walnut
Asbestos 2%
Start -
Stop 1973
Trade Name Dum Dum Nail Hole Walnut
G-H Number 3721-499-LX405.

171 Dum Dum Nail Hole White
Asbestos 2%
Start -
Stop 1973
Trade Name Dum Dum Nail Hole White
G-H Number 3720-110.

739 Dum Dum Nail Hole Dark Walnut
Asbestos 2%
Start -
Stop 1973
Trade Name Dum Dum Nail Hole Dark Walnut
G-H Number 3721-499-LX406

77-740 Dum Dum Nail Hole Allspice
Asbestos 2%
Start -
Stop 1973
Trade Name Dum Dum Nail Hole Allspice
G-H Number 3721-499-LX247.

742 Dum Dum Nail Hole Natural Walnut
Asbestos 2%
Start -
Stop 1973
Trade Name Dum Dum Nail Hole Natural Walnut
G-H Number 3721-499-LX315

569-W-1379 Dum Dum Nail Hole Bone White
Asbestos 2%
Start -
Stop 1973
Trade Name Dum Dum Nail Hole Bone White
G-H Number 3721-1

Dum Dum Travertine White[1]
Asbestos 2%
Start -
Stop 1973
Trade Name Dum Dum Travertine White
G-H-Number 3721-199-LX276

Mahagony Dum Dum[2]
Asbestos 2%
Start -
Stop 1973
Trade Name Mahogany Dum Dum
G-H Number 3721-399-LX313

Dum Dum Super White[3]
Asbestos 2%
Start -
Stop 1973
Trade Name Dum Dum Super White
G-H Number 3720-110.

[1,2,3] These Dum Dum products contained 2% asbestos and are probably Nail Hole products, but this is unconfirmed.

APPENDIX C2

CAULKING PRODUCTS

CODE	PRODUCT NAME	ASB %	YR START	YR DISC.	DESCRIPTION
46-F-3	Dum Dum Calk Gun-Grade Natural	10%	1964	Au1969	A Caulking of putty consistency for application by caulking gun
46-F-6	Dum Dum Calk	10%	1964	1969	Same as 46-F-3 but supplied in unspouted cartridges
46-W-3	Dum Dum Calk Non shrink Off White	U[4]	1964	Au1969	Similar to 46-F-3 but off white colour
46-W-4	Dum Dum non-Cartridge Calk Non shrink Off White.	U	1964	Ja1968	Same as 46-W-3 but supplied in unspouted cartridges
46-W-5	Dum Dum Cartridge Calk Non Shrink Off white	U	1964	1969	Same as 46-W-3 but supplied in nozzle type cartridges
46-F-5	Dum Dum Caulk Natural	2-3%	1964	Ja1979	A calking compound of putty consistency used for glazing and filling applications by putty knife and calking gun.
46-X-9	Heating and Ventilating Dum Dum	U	1964	Au1969	A caulking material for sealing joints on air ducts in heating and air conditioning systems. A heavy putty- type consistency, for knife or trowel application

[4] Asbestos content not known

APPENDIX C3

CAULKING PRODUCTS

HIGH TEMPERATURE CAULKINGS

CODE	PRODUCT NAME	ASB %	YR START	YR DISC.	DESCRIPTION
46-F-7	Hi-Heat Dum Dum	10-13	1964	Ja1980	A caulking of mastic consistency used for sealing joints and crevices on furnaces and boilers applicable only with caulking gun,trowel,special Dum Dum brush or heavy duty mastic spray equipment.
46-J-9	Armorcote Dum Dum	5-6	1964	Ja1979	A heavy bodied product for refractory application by trowel.

APPENDIX C4

OTHER DUM DUM PRODUCTS

CODE	PRODUCT NAME	ASB %	YR START	YR DISC.	DESCRIPTION
95	Series Dum Dum Masonoc	12-15	1964	1969	A heavy-bodied coating of mastic consistency designed for hi-build application and for bridging cracks and crevices with application by trowel, special brush or heavy-duty mastic spray equipment.
97	Series Chimney Dum Dum	4-5	1964	1979	A heavy-bodied coating of mastic consistency designed for hi-build application for filling and bridging cracks and and surface sealing of concrete chimneys with application by trowel, special brush or heavy-duty mastic spray equipment.

APPENDIX D

APPENDIX D1

SIZES OF "ASBESTOS STRUCTURES" IN AIRBORNE DUST SAMPLES COLLECTED DURING THE TESTING OF DUM DUM MASONOC BY ARTHUR D LITTLE (PETERS 1991).

PRODUCT APPLICATION

	DIAMETERS							
	0.1	0.15	0.2	0.25	0.3	0.4	0.5+	TOTAL
LENGTH 5.0-5.9					1		2	3
LENGTH 6.0-6.9							1	1
LENGTH 7.0-7.9	1						2	3
LENGTH 8.0-8.9							1	1
LENGTH 9.0-9.9								
LENGTH 10+	2				1		7	10
TOTAL	3				2		13	18

* Includes 1 fibre of diameter = 6um.

PRODUCT REMOVAL

	DIAMETERS							
	0.1	0.15	0.2	0.25	0.3	0.4	0.5+	TOTAL
LENGTH 5.0-5.9								
LENGTH 6.0-6.9								
LENGTH 7.0-7.9								
LENGTH 8.0-8.9								
LENGTH 9.0-9.9								
LENGTH 10+							2	2
TOTAL							2	2

APPENDIX D2

SIZES OF "ASBESTOS STRUCTURES" IN AIRBORNE DUST SAMPLES COLLECTED DURING THE TESTING OF CHIMNEY DUM DUM BY ARTHUR D LITTLE (PETERS 1991).

PRODUCT APPLICATION

	DIAMETERS							
	0.1	0.15	0.2	0.25	0.3	0.4	0.5+	TOTAL
LENGTH 5.0-5.9	9	1	5		1			16
LENGTH 6.0-6.9	2		4	2		1		9
LENGTH 7.0-7.9	1		1		1			3
LENGTH 8.0-8.9	2		4		1			7
LENGTH 9.0-9.9	2		1		1			4
LENGTH 10 +	1		3					4
TOTAL	17	1	18	2	4	1		43

PRODUCT REMOVAL

	DIAMETERS							
	0.1	0.15	0.2	0.25	0.3	0.4	0.5+	TOTAL
LENGTH 5.0-5.9								
LENGTH 6.0-6.9								
LENGTH 7.0-7.9								
LENGTH 8.0-8.9								
LENGTH 9.0-9.9								
LENGTH 10 +	1						1	2
TOTAL	1						1	2

APPENDIX D3

SIZES OF "ASBESTOS STRUCTURES" IN AIRBORNE DUST SAMPLES COLLECTED DURING THE TESTING OF HI-HEAT DUM DUM BY ARTHUR D LITTLE (PETERS 1991).

PRODUCT APPLICATION

	DIAMETERS							
	0.1	0.15	0.2	0.25	0.3	0.4	0.5+	TOTAL
LENGTH 5.0-5.9								
6.0-6.9								
7.0-7.9								
8.0-8.9								
9.0-9.9								
10 +							1	1
TOTAL							1	1

PRODUCT REMOVAL

	DIAMETERS							
	0.1	0.15	0.2	0.25	0.3	0.4	0.5+	TOTAL
LENGTH 5.0-5.9							2	2
6.0-6.9						1		1
7.0-7.9								
8.0-8.9								
9.0-9.9								
10 +						1	6	7
TOTAL						2	8	10

APPENDIX E

APPENDIX E

THRESHOLD LIMIT VALUES (TLV'S) ESTABLISHED BY THE AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH) 1946-1991

M.P.P.C.F.= Million Particles per Cubic Foot of air as measured using midget impinger sampler and standard light field count

Fibres/cc measured using phase contrast light optical microscopy.Fibres longer than 5um with aspect ratios greater than 3:1

YEAR	SUBSTANCE	CONCENTRATION TLV	
		M.P.P.C.F	Fibres/cc
1946	Asbestos	5	
1947			5
1948		*	
1949		*	
1950			*
1951			5
1952			5
1953			5
1956			5
1957			5
1958			5
1959			5
1960		5	
1961		-	5
1962			5
1963		5	
1964			5
1965		5	
1966			*
	Tremolite	5	
1967			5
1968			5
	Intended change.....	2	12 +

* Asbestos not listed

TABLE cont.

YEAR	SUBSTANCE	CONCENTRATION TLV M.P.P.C.F	Fibres/cc
1969			-?
1970	Asbestos,all types	5	
	Intended change.....		5 +$
	Talc (fibrous) USE ASBESTOS LIMIT		
1971	Asbestos,all types	-	
	Intended change		5 +#$
	Talc (fibrous) USE ASBESTOS LIMIT		
	Tremolite SEE TALC FIBROUS		
1972	Asbestos,all types	-	
	Intended change.....		5 +$# Ala
	Tremolite SEE TALC FIBROUS		
	Talc (fibrous) USE ASBESTOS LIMIT		
1973	Asbestos,all forms		5 +@# Ala
	Talc (fibrous) USE ASBESTOS LIMIT		
1974	Asbestos,all forms		5 +@# Ala
	Tremolite,SEE ASBESTOS		
	Talc (fibrous) USE ASBESTOS LIMIT		
1975	Asbestos,all forms		5 +@# Ala
	Tremolite,SEE ASBESTOS		
	Talc (fibrous) USE ASBESTOS LIMIT		
1976	Asbestos,all forms		5 +@# Ala
	Tremolite,SEE ASBESTOS		
	Talc (fibrous) USE ASBESTOS LIMIT		
1977	Asbestos,all forms		5 +@# Ala
	Tremolite,SEE ASBESTOS		
	Talc (fibrous) USE ASBESTOS LIMIT		

TABLE cont.

YEAR	SUBSTANCE	CONCENTRATION TLV	
		Fibres/cc	
1978	Asbestos,all forms	5	+@# Ala
	Tremolite,SEE ASBESTOS		
	Talc (fibrous) USE ASBESTOS LIMIT		
1979	Asbestos,all forms	5	+# Ala %
	Tremolite,SEE ASBESTOS		
	Talc (fibrous) USE ASBESTOS LIMIT		
1980	Asbestos		
	Amosite	0.5	Ala +#
	Chrysotile	2.0	Ala +#
	Crocidolite	0.2	Ala +#
	Other Forms	2.0	Ala +#
	Talc (fibrous) USE ASBESTOS LIMIT		
1981	Asbestos		
	Amosite	0.5	Ala +#
	Chrysotile	2.0	Ala +#
	Crocidolite	0.2	Ala +#
	Other Forms	2.0	Ala +#
	Talc (fibrous) USE ASBESTOS LIMIT		
1982	Asbestos		
	Amosite (12172-73-5)	0.5	Ala +#
	Chrysotile (12001-29-5)	2.0	Ala +#
	Crocidolite (12001-28-4)	0.2	Ala +#
	Other Forms	2.0	Ala +#
	Talc (fibrous) USE ASBESTOS LIMIT		
1983	Asbestos		
	Amosite (12172-73-5)	0.5	Ala +#
	Chrysotile (12001-29-5)	2.0	Ala +#
	Crocidolite (12001-28-4)	0.2	Ala +#
	Other Forms	2.0	Ala +#
	Talc (fibrous) USE ASBESTOS LIMIT		

TABLE cont.

YEAR	SUBSTANCE	CONCENTRATION TLV Fibres/cc	
1984	Asbestos		
	Amosite (12172-73-5)	0.5	Ala +#
	Chrysotile (12001-29-5)	2.0	Ala +#
	Crocidolite (12001-28-4)	0.2	Ala +#
	Other Forms	2.0	Ala +#
	Talc (fibrous) USE ASBESTOS LIMIT		
1985	Asbestos		
	Amosite (12172-73-5)	0.5	Ala x#
	Chrysotile (12001-29-5)	2.0	Ala x#
	Crocidolite (12001-28-4)	0.2	Ala x#
	Other Forms	2.0	Ala x#
	Talc (containing asbestos fibres)- USE ASBESTOS TLV. However,should not exceed 2mg/m3 respirable dust.		
1986	Asbestos		
	Amosite (12172-73-5)	0.5	Ala x#
	Chrysotile (12001-29-5)	2.0	Ala x#
	Crocidolite (12001-28-4)	0.2	Ala x#
	Other Forms	2.0	Ala x#
	Talc (containing asbestos fibres) USE ASBESTOS TLV-TWA.However,should not exceed 2mg/m^3 respirable dust.		
1987	Asbestos		
	Amosite (12172-73-5)	0.5	Ala x#
	Chrysotile (12001-29-5)	2.0	Ala x#
	Crocidolite (12001-28-4)	0.2	Ala x#
	Other Forms	2.0	Ala x#
	Talc (containing asbestos fibres) USE ASBESTOS TLV_TWA.However,should not exceed 2mg/m^3 respirable dust.		

TABLE cont.

YEAR	SUBSTANCE	CONCENTRATION TLV Fibres/cc	
1988	Asbestos		
	Amosite (12172-73-5)	0.5	Ala x#*k
	Chrysotile (12001-29-5)	2.0	Ala x#*k
	Crocidolite (12001-28-4)	0.2	Ala x#*k
	Other Forms	2.0	Ala x#*k
1989	Asbestos		
	Amosite (12172-73-5)	0.5	Ala x#*k
	Chrysotile (12001-29-5)	2.0	Ala x#*k
	Crocidolite (12001-28-4)	0.2	Ala x#*k
	Other Forms	2.0	Ala x#*k
1990	Asbestos		
	Amosite (12172-73-5)	0.5	Ala x#*k
	Chrysotile (12001-29-5)	2.0	Ala x#*k
	Crocidolite (12001-28-4)	0.2	Ala x#*k
	Other Forms	2.0	Ala x#*k
1991	Asbestos		
	Amosite (12172-73-5)	0.5	Ala x#*k
	Chrysotile (12001-29-5)	2.0	Ala x#*k
	Crocidolite (12001-28-4)	0.2	Ala x#*k
	Other Forms	2.0	Ala x#*k

In 1991 ACGIH gave "notice of intended change" with a proposed TLV =0.2 fibres/cc for all forms of asbestos

EXPLANATION OF NOTES

TLV Threshold Limit Value

TWA Time weighted average

÷ Fibres/cc >5um in length

x Fibres/cc longer than 5um and with an aspect ratio equal or greater than 3:1

@ A more stringent TLV for crocidolite may be required

As determined by the membrane filter method at 400-450 x magnification (4mm objective) phase contrast illumination.

$ Concentrations 5 fibres/ml but not to exceed 10,may be permitted for 15-minute periods each hour up to 5 times daily.

Ala Human Carcinogens.Substances or substances associated with industrial processes,recognised to have carcinogenic or cocarcinogenic potential with an assigned TLV

% Cigarette smoking can enhance the incidence of respiratory cancers from this or others of these substances or processes.

* Substance identified by other sources as a suspected or confirmed human carcinogen

k Substance for which OSHA and/or NIOSH has a permissible exposure limit (PEL) or a recommended exposure limit (REL) lower than the TLV.

APPENDIX UE

APPENDIX UE

THRESHOLD LIMIT VALUES (TLV'S) ESTABLISHED BY THE AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH)

REVISED AND UPDATED TO INCLUDE THE PERIOD 1946-1999

DEFINITIONS

M.P.P.C.F.= Million Particles per Cubic Foot of air as measured using impinger samplers and standard light field count.

Fibres/cc = Number of fibers per cubic centimeter of air as measured using phase contrast light optical microscopy and designated fiber definitions. Fibre counts were determined using the membrane filter method at 400-450X magnification [4-mm objective] phase contrast illumination.

Fibre = The definition of a fibre used in the TLV is a fibre longer than 5um with aspect ratio equal to or greater than 3:1.

THRESHOLD LIMIT VALUES (TLVS) BY YEAR

YEAR	SUBSTANCE	CONCENTRATION TLV	
		M.P.P.C.F	Fibres/cc
1946	Asbestos	5	
1947		5	
1948		*	
1949		*	
1950		*	
1951		5	
1952		5	
1953		5	
1956		5	
1957		5	
1958		5	
1959		5	
1960		5	

TABLE cont.

YEAR	SUBSTANCE	CONCENTRATION TLV M.P.P.C.F	Fibres/cc
1961		5	
1962		5	
1963		5	
1964		5	
1965		5	
1966		-	
	Tremolite	5	
1967		5	
	Tremolite	5	
1968		5	
	Tremolite	5	
	Intended change	2	12 +
1969		-?	
1970	Asbestos, all types	5	
	Intended change	5	÷$
	Talc (fibrous) USE ASBESTOS LIMIT		
	Tremolite SEE ASBESTOS		
1971	Asbestos, all types	-	
	Intended change	5	+#$
	Talc (fibrous) USE ASBESTOS LIMIT		
	Tremolite SEE TALC FIBROUS.		
1972	Asbestos, all types	-	
	Intended change	5	÷S# Ala
	Tremolite SEE TALC FIBROUS		
	Talc (fibrous) USE ASBESTOS LIMIT		
1973	Asbestos, all forms		
	Intended change	5	+@# Ala
	Talc (fibrous) USE ASBESTOS LIMIT		
	(A more stringent TLV for crocidolite may be required)		

1974 Asbestos, all forms	5	+@# Ala
Tremolite, SEE ASBESTOS		
Talc (fibrous) USE ASBESTOS LIMIT		
1975 Asbestos, all forms	5	+@# Ala
Tremolite, SEE ASBESTOS		
Talc (fibrous) USE ASBESTOS LIMITTABLE cont.		
1976 Asbestos, all forms	5	+@# Ala
Tremolite, SEE ASBESTOS		
Talc (fibrous) USE ASBESTOS LIMIT		
1977 Asbestos, all forms	5	+@# Ala
Tremolite, SEE ASBESTOS		
Talc (fibrous) USE ASBESTOS LIMIT		
1978 Asbestos, all forms	5	+@# Ala
Intended change		
Amosite	0.5	Ala +#
Chrysotile	2.0	Ala +#
Tremolite	0.5	Ala +#
Crocidolite	0.2	Ala +#
Other Forms	2.0	Ala +#
Tremolite, SEE ASBESTOS		
Talc (fibrous) USE ASBESTOS LIMIT		
1979 Asbestos, all forms	5	+# Ala %
Intended change		
Amosite	0.5	Ala +#
Chrysotile	2.0	Ala +#
Tremolite	0.5	Ala +#
Crocidolite	0.2	Ala +#
Other Forms	2.0	Ala +#
Talc {fibrous}	0.5	
Tremolite,SEE ASBESTOS		
Talc (fibrous) USE ASBESTOS LIMIT		
1980 Asbestos		
Amosite	0.5	Ala +#
Chrysotile	2.0	Ala +#
Crocidolite	0.2	Ala +#
Other Forms	2.0	Ala +#
Talc (fibrous) USE ASBESTOS LIMIT		

1974 Asbestos, all forms	5	+@# Ala
Tremolite, SEE ASBESTOS		
Talc (fibrous) USE ASBESTOS LIMIT		
1975 Asbestos, all forms	5	+@# Ala
Tremolite, SEE ASBESTOS		
Talc (fibrous) USE ASBESTOS LIMITTABLE cont.		
1976 Asbestos, all forms	5	+@# Ala
Tremolite, SEE ASBESTOS		
Talc (fibrous) USE ASBESTOS LIMIT		
1977 Asbestos, all forms	5	+@# Ala
Tremolite, SEE ASBESTOS		
Talc (fibrous) USE ASBESTOS LIMIT		
1978 Asbestos, all forms	5	+@# Ala
Intended change		
Amosite	0.5	Ala +#
Chrysotile	2.0	Ala +#
Tremolite	0.5	Ala +#
Crocidolite	0.2	Ala +#
Other Forms	2.0	Ala +#
Tremolite, SEE ASBESTOS		
Talc (fibrous) USE ASBESTOS LIMIT		
1979 Asbestos, all forms	5	+# Ala %
Intended change		
Amosite	0.5	Ala +#
Chrysotile	2.0	Ala +#
Tremolite	0.5	Ala +#
Crocidolite	0.2	Ala +#
Other Forms	2.0	Ala +#
Talc {fibrous}	0.5	
Tremolite,SEE ASBESTOS		
Talc (fibrous) USE ASBESTOS LIMIT		
1980 Asbestos		
Amosite	0.5	Ala +#
Chrysotile	2.0	Ala +#
Crocidolite	0.2	Ala +#
Other Forms	2.0	Ala +#
Talc (fibrous) USE ASBESTOS LIMIT		

YEAR	SUBSTANCE	CONCENTRATION TLV		
		M.P.P.C.F	Fibres/cc	Comments
1986	Asbestos			
	Amosite (12172-73-5)	0.5	Ala x#	
	Chrysotile (12001-29-5)	2.0	Ala x#	
	Crocidolite (12001-28-4)	0.2	Ala x#	
	Other Forms	2.0	Ala x#	
	Talc (containing asbestos fibres)			
	USE ASBESTOS TLV-TWA. However, should not exceed 2mg/m^3 respirable dust.			
1987	Asbestos			
	Amosite (12172-73-5)	0.5	Al x#	
	Chrysotile (12001-29-5)	2.0	Al x#	
	Crocidolite (12001-28-4)	0.2	Al x#	
	Other Forms	2.0	Al x#	
	Talc (containing asbestos fibres)			
	USE ASBESTOS TLV_TWA. However, should not exceed 2mg/m^3 respirable dust.			
1988	Asbestos			
	Amosite (12172-73-5)	0.5	Al x#*k	
	Chrysotile (12001-29-5)	2.0	Al x#*k	
	Crocidolite (12001-28-4)	0.2	Al x#*k	
	Other Forms	2.0	Al x#*k	
1989	Asbestos			
	Amosite (12172-73-5)	0.5	Al x#*k	
	Chrysotile (12001-29-5)	2.0	Al x#*k	
	Crocidolite (12001-28-4)	0.2	Al x#*k	
	Other Forms	2.0	Al x#*k	
1990	Asbestos			
	Amosite (12172-73-5)	0.5	Al x#*k	
	Chrysotile (12001-29-5)	2.0	Al x#*k	
	Crocidolite (12001-28-4)	0.2	Al x#*k	
	Other Forms	2.0	Al x#*k	

YEAR	SUBSTANCE	CONCENTRATION TLV		
		M.P.P.C.F	Fibres/cc	Comments
1986	Asbestos			
	Amosite (12172-73-5)	0.5	Ala x#	
	Chrysotile (12001-29-5)	2.0	Ala x#	
	Crocidolite (12001-28-4)	0.2	Ala x#	
	Other Forms	2.0	Ala x#	
	Talc (containing asbestos fibres)			
	USE ASBESTOS TLV-TWA. However, should not exceed 2mg/m^3 respirable dust.			
1987	Asbestos			
	Amosite (12172-73-5)	0.5	Al x#	
	Chrysotile (12001-29-5)	2.0	Al x#	
	Crocidolite (12001-28-4)	0.2	Al x#	
	Other Forms	2.0	Al x#	
	Talc (containing asbestos fibres)			
	USE ASBESTOS TLV_TWA. However, should not exceed 2mg/m^3 respirable dust.			
1988	Asbestos			
	Amosite (12172-73-5)	0.5	Al x#*k	
	Chrysotile (12001-29-5)	2.0	Al x#*k	
	Crocidolite (12001-28-4)	0.2	Al x#*k	
	Other Forms	2.0	Al x#*k	
1989	Asbestos			
	Amosite (12172-73-5)	0.5	Al x#*k	
	Chrysotile (12001-29-5)	2.0	Al x#*k	
	Crocidolite (12001-28-4)	0.2	Al x#*k	
	Other Forms	2.0	Al x#*k	
1990	Asbestos			
	Amosite (12172-73-5)	0.5	Al x#*k	
	Chrysotile (12001-29-5)	2.0	Al x#*k	
	Crocidolite (12001-28-4)	0.2	Al x#*k	
	Other Forms	2.0	Al x#*k	

TABLE cont.

YEAR	SUBSTANCE	CONCENTRATION TLV M.P.P.C.F	Fibres/cc	Comments
1995	Asbestos.			
	Amosite (12172-73-5)	0.5	Al x#*k	
	Chrysotile (12001-29-5)	2.0	Al x#*k	
	Crocidolite (12001-28-4)	0.2	Al x#*k	
	Other Forms	2.0	Al x#*k	
	Notice of intended change for "Asbestos, all forms [1332-21-4]" to 0.2f/cc for fibres longer than 5um and with an aspect ratio equal to or greater than 3:1 as determined by the membrane filter method at 400-450X magnification (4 mm objective) phase contrast illumination.			
1996	Asbestos			
	Amosite (12172-73-5)	0.5	Al x#*k	
	Chrysotile (12001-29-5)	2.0	Al x#*k	
	Crocidolite (12001-28-4)	0.2	Al x#*k	
	Other Forms	2.0	Al x#*k	
	Notice of intended change for "Asbestos, all forms [1332-21-4]" to 0.2f/cc for fibres longer than 5um and with an aspect ratio equal to or greater than 3:1 as determined by the membrane filter method at 400-450X magnification (4 mm objective) phase contrast illumination.			
1997	Asbestos			
	Amosite (12172-73-5)	0.5	Al x#*k	
	Chrysotile (12001-29-5)	2.0	Al x#*k	
	Crocidolite (12001-28-4)	0.2	Al x#*k	
	Other Forms	2.0	Al x#*k	
	Notice of intended change for "Asbestos, all forms [1332-21-4]" to 0.1f/cc for fibres longer than 5um and with an aspect ratio equal to or greater than 3:1 as determined by the membrane filter method at 400-450 x magnification (4 mm objective) phase contrast illumination.			
1998	Asbestos, all forms (1332-21-4)	0.1	Al x#	Critical effects. asbestosis; cancer.
1999	Asbestos, all forms (1332-21-4)	0.1	Al x#	Critical effects. asbestosis; cancer.

EXPLANATION OF NOTES

TLV Threshold Limit Value

TWA Time weighted average

\+ Fibres/ml >5um in length

x Fibres/cc longer than 5um and with an aspect ratio equal or greater than 3:1

@ A more stringent TLV for crocidolite may be required

As determined by the membrane filter method at 400-450 x magnification (4 mm objective) phase contrast illumination.

$ Concentrations 5 fibres/ml but not to exceed 10, may be permitted for 15-minute periods each hour up to 5 times daily.

% Cigarette smoking can enhance the incidence of respiratory cancers from this or others of these substances or processes.

* Substance identified by other sources as a suspected or confirmed human carcinogen

k Substance for which OSHA and/or NIOSH has a permissible exposure limit (PEL) or a recommended exposure limit (REL) lower than the TLV.

A1a Human Carcinogens. Substances or substances associated with industrial processes, recognised to have carcinogenic or cocarcinogenic potential with an assigned TLV

A1	1987, 1988	Confirmed Human carcinogen
	1989, 1990	Confirmed human carcinogens: Substances, or substances associated with industrial processes, recognized to have carcinogenic potential.
	1991	Confirmed human carcinogens: Substances, or substances associated with industrial processes, recognized to have carcinogenic potential Intended change to Confirmed human carcinogen: The agent is carcinogenic to humans based on the weight of evidence from epidemiologic studies of or convincing clinical evidence in exposed humans.

EXPLANATION OF NOTES cont.

A1	1992, 1993,1994, 1995, 1996, 1997,	Confirmed human carcinogen: The agent is carcinogenic to humans based on the weight of evidence from epidemiologic studies of or convincing clinical evidence in exposed humans.
	1998, 1999	Confirmed human carcinogen: The agent is carcinogenic to humans based on the weight of evidence from epidemiologic studies.

APPENDIX F

APPENDIX F

FEDERAL REGULATORY PROGRAM
OSHA

1971-1988

DATE	RULE
29.05.71	PEL of 12 f/cc over 8 hour time weighted average (TWA) Section 6(a) of the OSH Act codified at 29 U.S.C.S 655 (a) (36 FR 10466) (29 C.F.R.Part 1910)
12.07.71	PEL of 5 f/cc over 8 hour time weighted average (TWA) and peak exposure of 10 f/cc as Emergency Temporary Standard (36 FR 234207) (29 C.F.R.Part 1910.93a)
7.06.72	New Final rule-PEL of 5 f/cc over 8 hour time weighted average (TWA).To be lowered to 2f/cc in 1976 (37 FR 11318) (29 C.F.R.Part 1910)
09.10.75	Proposed Rule PEL of 0.5 f/cc over 8 hour time weighted average (TWA).Ceiling limit of 5f/cc for 15 minutes (1980)-withdrawn. (40 FR 47652)
07.76	PEL of 2f/cc as 8 hour time weighted average became effective as in 1972 final rule (29 CFR 1910.1001)
04.11.83	Emergency temporary Standard (ETS) of 0.5 f/cc as 8 hour time weighted average.
03.84	1983 ETS Overturned

TABLE cont.

DATE	RULE
10.04.84	Proposed rule - PEL to be lowered to 0.5 or 0.2 f/cc as time weighted average. (49 FR 14116)
12.07.85	EPA Proposed rule Immediately effective to extend OSHA provisions to state and local workers. (50 C.F.R.28530)
25.04.86	EPA Final Rule-extends OSHA protections to state & local workers. (51 FR 15722) (40 C.F.R.Part 763,Subpart G)
20.06.86	PEL lowered to 0.2f/cc as 8 hour time weighted average. (Final Rules 51FR 22612) (29 C.F.R.Part 1910, Subpart Z and 29 C.F.R.Part 1926,subpart D)
17.10.86	Partial Stay of 20.06.86 standards as they applied to non asbestiform tremolite.anthophyllite and actinolite pending further review and rule making. (51 FR 37002)
25.02.87	EPA Final Rule-Revised rule extended current OSHA requirements to state local workers involved in abatement and lifted repair exemption for projects 3 feet or less. (52 FR 5618) (40 C.F.R.Part 763,Subpart G)
14.09.88	Amendment - excursion limit of 1 f/cc over a 30 minute period added to cover employees involved in jobs with irregular peak whose total exposure may not reach 0.2 f/cc as time weighted average over an 8 hour exposure period. (53 FR 35610) (29 C.F.R.Part 1910,Subpart Z and 29 C.F.R.1926,Subpart D).

APPENDIX UF

APPENDIX UF

FEDERAL REGULATORY PROGRAM
OSHA

REVISED AND UPDATED TO INCLUDE THE PERIOD 1971-1999

DATE	RULE
05.29.71	PEL of 12 f/cc over 8 hour time weighted average (TWA) Section 6(a) of the OSH Act codified at 29 U.S.C.S 655 (a) (36 FR 10466) (29 C.F.R.Part 1910)
07.12.71	PEL of 5 f/cc over 8 hour time weighted average (TWA) and peak exposure of 10 f/cc as Emergency Temporary Standard (36 FR 234207) (29 C.F.R.Part 1910.93a)
06.07.72	New Final rule - PEL of 5 f/cc over 8 hour time weighted average (TWA). To be lowered to 2f/cc in 1976. Ceiling limit of 10f/cc. (37 FR 11318) (29 C.F.R.Part 1910)
10.09.75	Proposed Rule - PEL of 0.5 f/cc over 8 hour time weighted average (TWA).Ceiling limit of 5f/cc for 15 minutes.Applied to all industries except construction. (In 1980) - withdrawn. (40 FR 47652)
07.XX.76	PEL of 2f/cc as 8 hour time weighted average became effective as in 1972 final rule (29 CFR 1910.1001)
11.04.83	Emergency Temporary Standard (ETS) of 0.5 f/cc as 8 hour time weighted average. (48 FR 51096)

TABLE cont.

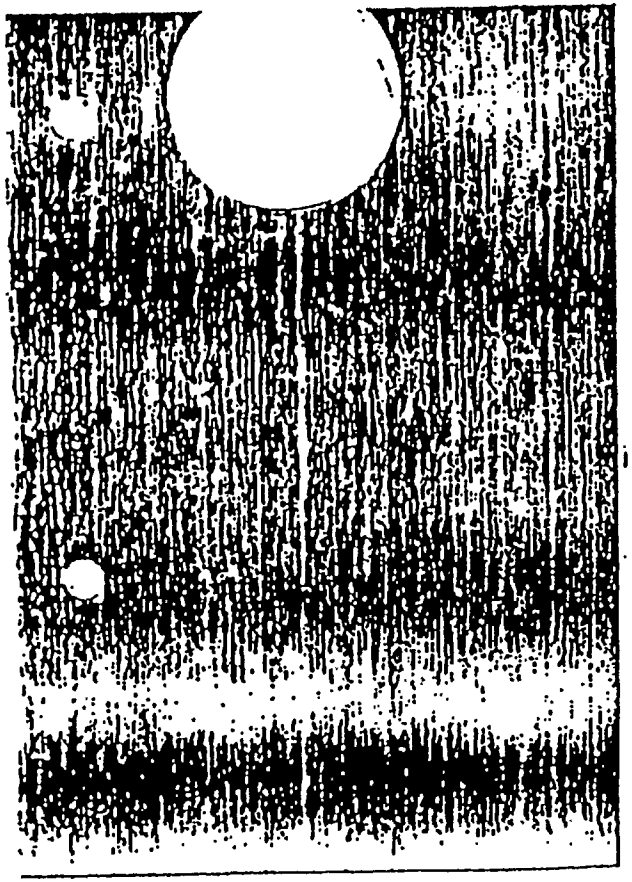
DATE	RULE
03.XX.84	1983 ETS - Overturned by US Circuit Court of Appeals for the Fifth Circuit.
04.10.84	Proposed rule - PEL to be lowered to 0.5 or 0.2 f/cc as time weighted average. (49 FR 14116)
07.12.85	EPA Proposed rule Immediately effective to extend OSHA provisions to State and local workers. (50 C.F.R.28530)
04.25.86	EPA Final Rule-extends OSHA protections to state & local workers. (51 FR 15722) (40 C.F.R.Part 763,Subpart G)
06.17.86	Two revised standards issued, one governing exposure to asbestos in general industry and the other applying to construction workplaces [06.20.86].
06.20.86	PEL lowered to 0.2f/cc as 8 hour time weighted average. (Final Rules 51FR 22612) (29 C.F.R.Part 1910,Subpart Z and 29 C.F.R.Part 1926,subpart D)
07.21.86	Revised standards amended OSHA's previous asbestos standard issued in 1972. The 1986 standard explicitly applied to non-asbestiform tremolite, actinolite and anthophyllite
10.17.86	Partial Stay of 06.20.86 standards as they applied to non asbestiform tremolite.anthophyllite and actinolite pending further review and rule making. (51 FR 37002)
02.25.87	EPA Final Rule-Revised rule extended current OSHA requirements to state local workers involved in abatement and lifted repair exemption for projects 3 feet or less. (52 FR 5618) (40 C.F.R.Part 763,Subpart G)

TABLE cont.

DATE	RULE
09.14.88	Amendment - excursion limit of 1 f/cc over a 30 minute period added to cover employees involved in jobs with irregular peak whose total exposure may not reach 0.2 f/cc as time weighted average over an 8 hour exposure period. (53 FR 35610) (29 C.F.R.Part 1910,Subpart Z and 29 C.F.R.1926,Subpart D).
10.11.94	Final Rule - The final standards amended the standards issued June 17 1986 (51 FR 22612, 29FR 1910.1001, June 20 1986) for occupationa l exposure in general industry and in the construction industry. They also include a separate standard covering asbestos exposure in the shipyard (29 CFR 1915.1001). PEL (TWA) = 0.1 f/cc for all asbesos work in all industries. (59 FR 40964).
07.01.98	Revisions - Safety & Health regulations for construction. PEL (TWA) = 0.1 f/cc as an 8 hour time weighted average. Excursion limit = 1 f/cc as averaged over 30 minutes. (29 CFR Ch XVII 7-1-98 Edition Part 1926).
07.01.98	Revisions - Occupational Safety & Health Standards. PEL (TWA) = 0.1 f/cc as an 8 hour time weighted average. Excursion limit = 1 f/cc as averaged over 30 minutes. (29 CFR Ch XVII 7-1-98 Edition Part 1910).

APPENDIX G

Mobil
Dum Dum Calking
Gray 46-F-5
FOR INDUSTRIAL USE ONLY. DO NOT USE IN OR AROUND A HOUSEHOLD OR DWELLING
CONFORMS TO HYDROCARBON EMISSION CONTROL REGULATIONS
KR
NET 1 GALLON



General: Dum Dum Caulking is designed for all conventional exterior caulking work, with flexibility to withstand building stresses, settling and shrinkage. It has outstanding adhesion to wood, masonry, glass, metal and ceramic tile.

Preparation: Surface must be clean, dry and free of dust. Loose paint, mortar or crumbling putty should be removed. All masonry cracks and mortar joints should be raked clean. Deep [illegible] openings should be packed with [illegible] of fine glass to about ⅛" from surface. Before caulking, prime bare wood surfaces with linseed oil or Exterior First Coater 17-W-4; Masonry surfaces with Masonoo Primer or 79 Series Exterior Latex Paint.

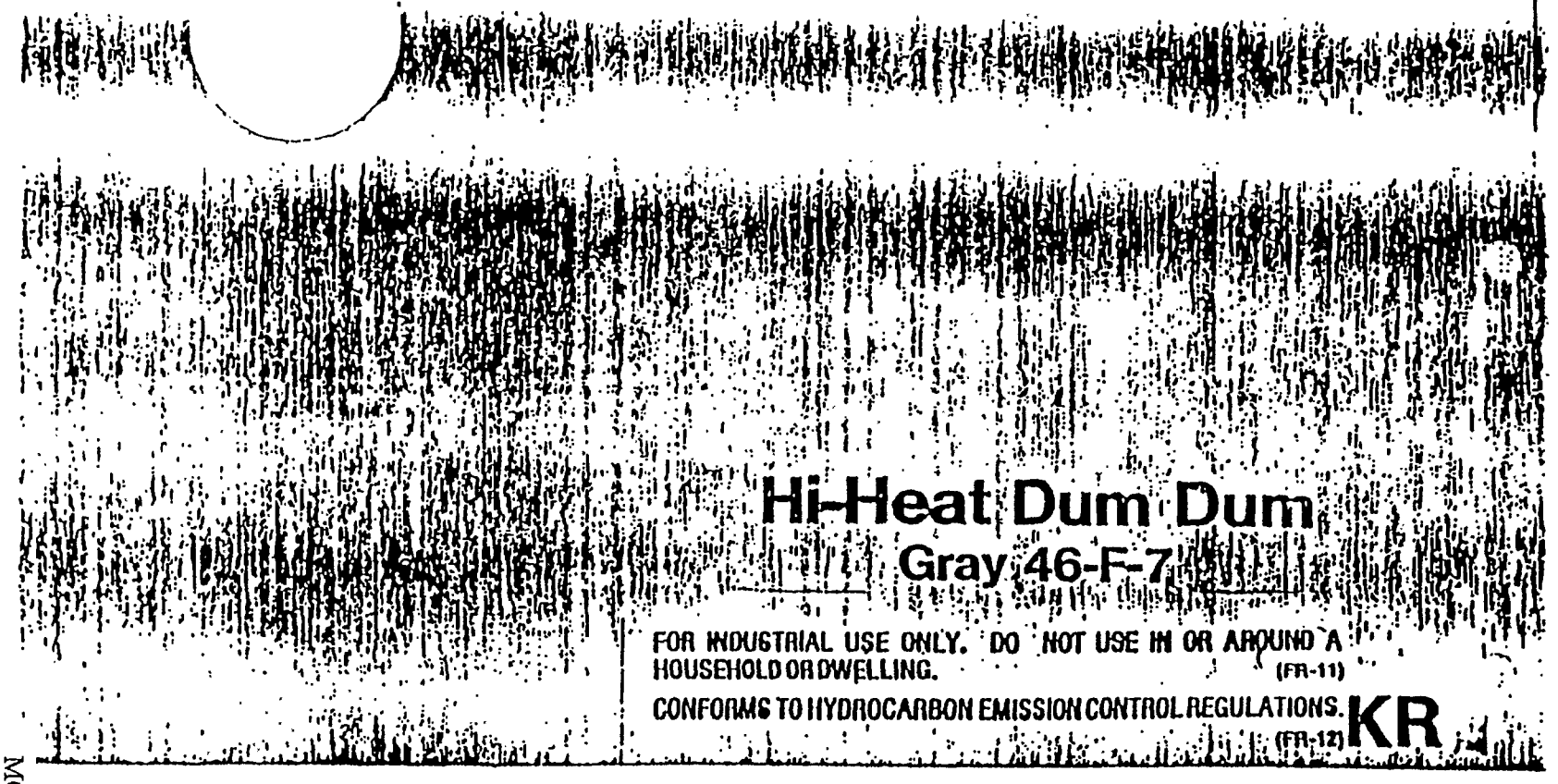
Application: If oil has risen to the top, stir thoroughly to a uniform mixture. Apply with a caulking gun or tool. Use as heavy a bead as possible assuring full contact on sides and bottom of bead. Leave a well rounded bead on the outside. When a smoothed bead is required for appearance, dip tool in Kerosene or water to eliminate sticking. To prevent skin forming in can when not in use, pour a liberal amount of water over the unused compound and keep container tightly sealed. Pour off water before re-using.

Note: Temperature influences the consistency of Dum Dum Calk. If too heavy, mix in thoroughly one cup of raw linseed oil to five gallons of Dum Dum Calk. If too thin, add dry, lump free, asbestos fibre as needed. ML-228 12-70

Mobil Chemical Company

[illegible]

Mobil®



Hi-Heat Dum Dum

Gray 46-F-7

FOR INDUSTRIAL USE ONLY. DO NOT USE IN OR AROUND A HOUSEHOLD OR DWELLING. (FR-11)

CONFORMS TO HYDROCARBON EMISSION CONTROL REGULATIONS. (FR-12) KR

NET 1 GALLON

MOB-HarrisMaster
00471

Mobil 1970b

General: Hi-Heat Dum Dum is recommended for use on furnaces, boilers, kilns and similar installations. May be used as a joint sealer and pliable gasket for boiler settings, as a caulking material for cracks and broken mortar joints, or for application overall on the unit. Use for continuous temperatures up to 175°F. and where short intermittent heat exposure does not exceed 650°F.

Preparation: Surface should be clean and dry. For masonry use, all cracks should be raked clean and any previously applied caulk or putty removed, then fill with Hi-Heat Dum Dum. On metal, best results will be had if metal is at normal temperature at time of application.

Application: Trowel, spray or brush where overall sealing work is being done. Bulk Caulking gun with leather cups, or trowel when filling cracks and joints.

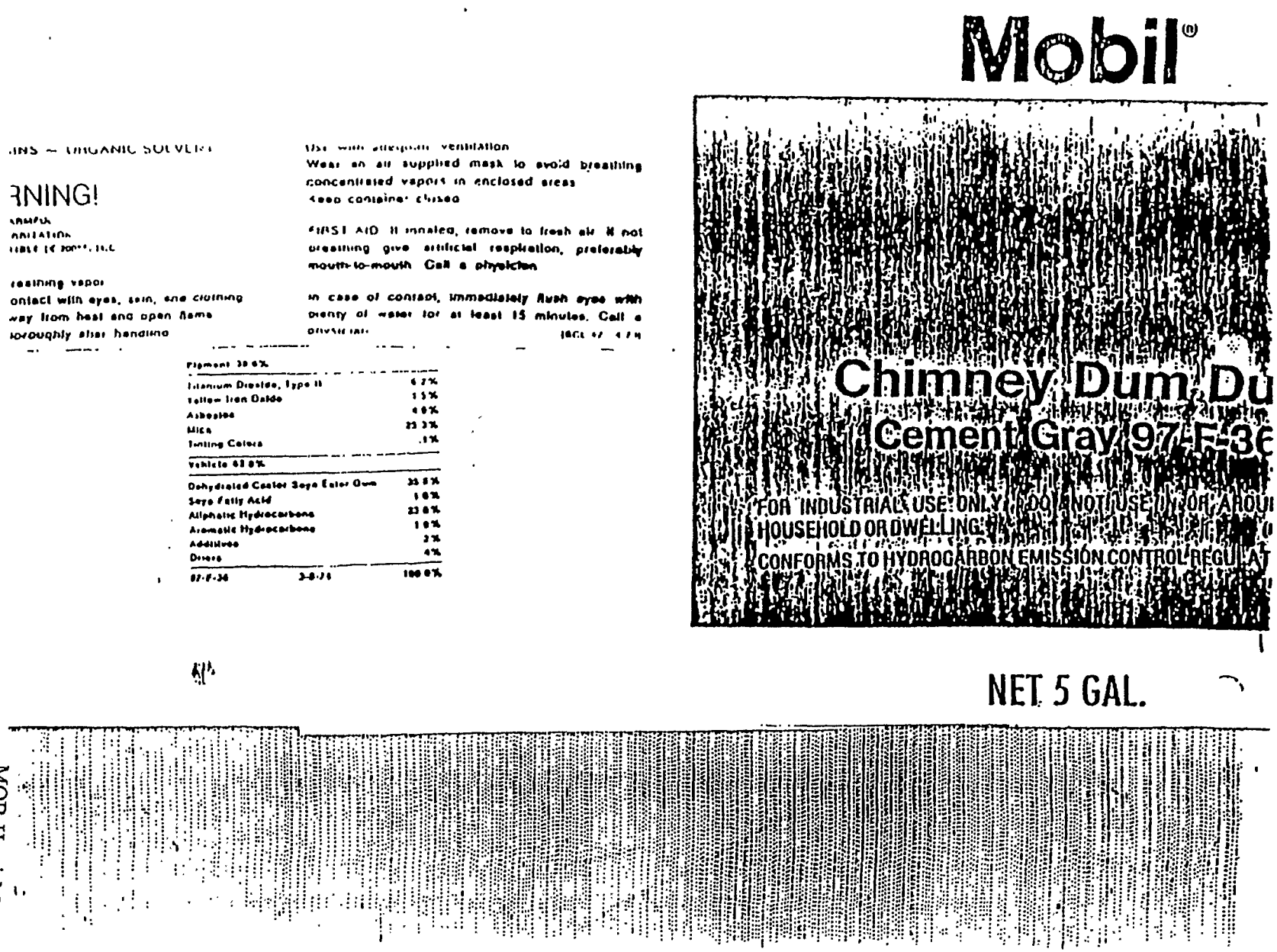
Coverage: Estimate 144 lineal feet per gallon at ¼ inch bead for filling and caulking and 15 to 20 square feet per gallon for entire surface work. Actual coverage will vary with application technique, job conditions and type of surface.

Dry Times: Surface skin forms in about 18 hours at 75°F. May be overcoated with most conventional paints after 24 hours. ML-234 12-70

Mobil Chemical Company

MAINTENANCE & MARINE COATINGS DEPARTMENT

EDISON, N.J. 08817/BEAUMONT, TEX. 77704/KANKAKEE, ILL. 60901/LOS ANGELES AREA/AZUSA, CALIF. 91702



General: Chimney Dum Dum is a heavy coating for the exterior of Masonry stacks. Fills and bridges hairline cracks. Provides flexible weatherproof protection under a tough outer skin.

Chimney linings must be inspected for integrity and freedom from air leaks.

Surface Preparation: Must be clean and dry. Surface should be thoroughly wire brushed to remove dirt, grease, soot and old coating. Cracks larger than hairline up to ⅛" wide should be raked, cleaned out and primed with 38-V-5 Chimney Dum Dum Primer and filled with Calk. Fill larger cracks with quick-setting cement. New cement patches should be treated to neutralize alkali cement. Prime entire surface with 38-V-5 Chimney Dum Dum Primer.

Application: Brush or spray—A special Dum Dum brush should be used for brush application. Spray equipment should be of the heavy mastic type. On porous surfaces apply at 30 or less square feet per gallon. On good, dense concrete apply at not more than 35 square feet per gallon. May be applied without completely cooling stack.

Thinner: Use material as packaged. If thickening occurs because of low temperatures, material should be placed in warm area. Never add thinner or other material as a reducer. Use 7-T-8 Thinner for clean-up only.

Service Temperature: 210° F. Maximum for continuous operation.

Caution: New concrete stacks must weather at least three months before coating. Any membrane curing agent must be completely weathered off or otherwise removed.

ML-223 12/78

Mobil Chemical Company

Mobil®

Dum Dum Mason[illegible]
Soft White 95-W-9

FOR INDUSTRIAL USE ONLY. DO NOT USE IN OR AROUND HOUSEHOLD OR DWELLING.

FIVE GALLONS

ITAINS — ORGANIC SOLVENT

ARNING!

HARMFUL.
S IRRITATION.
ATIBLE (< 300°F/1CC)

breathing vapor.
contact with eyes, skin, and clothing
away from heat and open flame.
thoroughly after handling

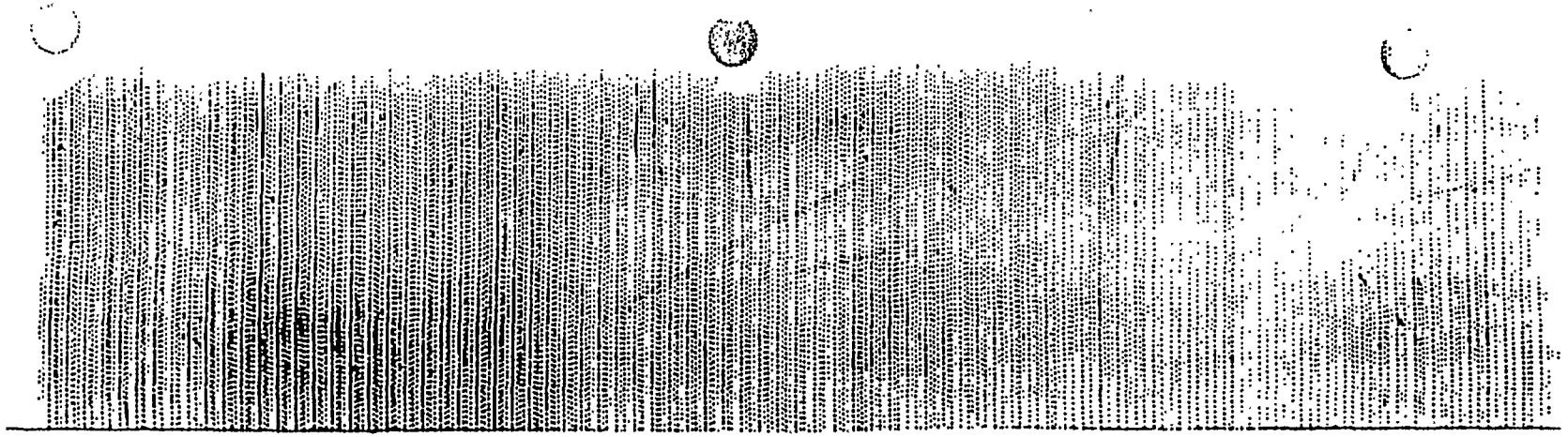
Use with adequate ventilation.
Wear an air supplied mask to avoid breathing concentrated vapors in enclosed areas.
Keep container closed.

FIRST AID: If inhaled, remove to fresh air. If not breathing give artificial respiration, preferably mouth-to-mouth. Call a physician.

In case of contact, immediately flush eyes with plenty of water for at least 15 minutes. Call a physician.

(MCL-42 4-73)

Pigment 21.8%		
Titanium Dioxide, Type I		10.2%
Asbestos		11.6%
Vehicle 78.2%		
Tung-Linseed-Soya Ester Gum		19.6%
Tung Rosin		13.3%
Soya Fatty Acid		1.8%
Aliphatic Hydrocarbons		41.3%
Aromatic Hydrocarbons		1.4%
Additives		.8%
95-W-9	12-12-72	100.0%



General: Dum Dum Masonoc is a heavy bodied, textured coating desired for use in hi-build applications for waterproofing and restoration of exterior masonry and for protection of steel surfaces.

Surface Preparation: Old masonry surfaces should be prepared by blast cleaning or other suitable methods to remove dust, dirt, surface contaminants, and loose particles. Any spalling concrete, loose mortar and unsound masonry must be removed and the surface repaired and brought to level as needed. Portland cement patches must be allowed to cure and then neutralized prior to overcoating. Surface must be completely dry before coating. Prime all bare masonry surface with a coat of Masonoc Primer, 47-U-1. Allow primer an overnight dry and then fill all cracks $\frac{1}{16}$ to $\frac{1}{4}$ inch with suitable caulking material prior to topcoating. New masonry surfaces must be allowed to cure for a sufficient length of time to reduce surface alkalinity. A minimum of 30 days is required with at least 6 months desired. Any membrane curing compounds must be completely weathered away or removed. Steel surfaces should be cleaned by blasting or wire brushing. Dum Dum Masonoc may be applied directly to bare steel or over a suitable Mobil steel primer.

Application: Use heavy duty spray equipment. Airless spray is recommended for best, uniform applications. Material may be applied by brush.

Thinner: Dum Dum Masonoc normally should be used as packaged. Slight thinning may be required in cold weather. Use Thinner 7-T-8 for clean-up and thinning.

Coverage: Apply at a uniform film thickness of 64 mils wet for a theoretical coverage of 25 sq. ft. per gallon. Actual coverage will be less depending upon application technique and type of surface.

This paint is resistant to the deteriorating action of molds (mildew) and to discoloration of the paint film by molds. No claim is made for preservative action to other than the paint film.

ML-218 11/71

Mobil Chemical Company

Mobil®

Dum Dum Armorcote

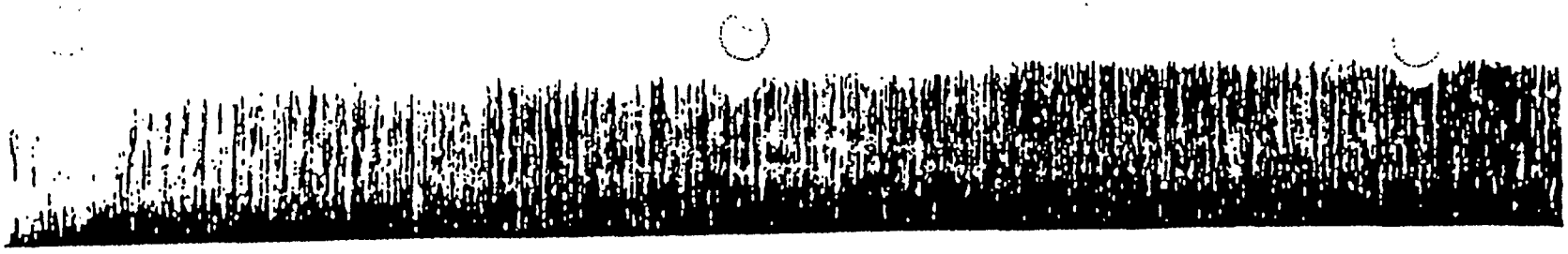
Black 46-J-9

MT7B053

FOR INDUSTRIAL USE ONLY. DO NOT USE IN OR AROUND A HOUSEHOLD OR DWELLING. (FR-11)

CONFORMS TO HYDROCARBON EMISSION CONTROL REGULATIONS. (FR-12) KR

NET 1 GALLON



General: Dum Dum Armorcote is a resilient material which provides protection and increased efficiency for brick boiler settings, as a dry kiln liner, and similar refractory applications. Used as a complete cover, Armorcote forms an air tight shield which offers substantial fuel savings by eliminating air leaks. Especially useful around and as a gasketing material for boiler settings. Also for use on the interior of dry kilns where cracks and pores allow heat loss and temperature fluctuations.

MAY BE APPLIED TO HOT SURFACES AND WILL RESIST SUSTAINED HEAT TO 300°F.

Preparation: Surface must be clean and dry. Remove all loose mortar or brick particles by wire brushing. Open mortar joints and cracks over ⅛" should be raked and filled with quick drying Portland Cement and allowed to set hard.

Application: Using a trowel, spread in uniform thickness. Boiler does not have to be shut down during application.

Coverage: (Theoretical) 75 sq. ft. per gallon for ⅛" thickness. Actual coverage may be less depending on type of surface and application technique.

Thinner: Use 7-T-12 for clean-up of equipment.

ML-235 12-78

CONTAINS — ORGANIC SOLVENT

WARNING!

FLAMMABLE [illegible]°F TCC
VAPOR HARMFUL
CAUSES IRRITATION

Keep away from heat, sparks, and open flame.
Avoid breathing vapor
Avoid contact with eyes, skin, and clothing
Wash thoroughly after handling

Use with adequate ventilation
Wear an air supplied mask to avoid breathing concentrated vapors in enclosed areas.
Keep container closed

FIRST AID If inhaled, remove to fresh air. If not breathing give artificial respiration, preferably mouth-to-mouth. Call a physician

In case of contact, immediately flush eyes with plenty of water for at least 15 minutes. Call a physician

IBCt-43 6-78

Mobil Chemical Company

APPENDIX H

Mobil Chemical Product Data

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Always consult your Mobil representative for latest information and recommendations for Mobil Products.

DUM DUM® CALKING

CONFORMS WITH AIR POLLUTION RULES AND REGULATIONS

46-F-5

COLOR	Natural
FINISH	Low Sheen
VEHICLE TYPE	Vegetable oil
PIGMENT TYPE	Inert pigment and Asbestos Fibre
SOLVENT TYPE	None
FLASH POINT, MINIMUM	Non-flammable
% SOLIDS BY VOLUME	100%
COVERAGE (THEORETICAL)	Size of joint 1/8" x 1/8" — 1230 Lineal Feet per gallon 1/4" x 1/4" — 300 Lineal Feet per gallon 3/8" x 3/8" — 135 Lineal Feet per gallon 1/2" x 1/2" — 77 Lineal Feet per gallon The actual coverage will be less, depending on application technique, job conditions and type of surface to be coated.
VISCOSITY AT 75°F. (24°C.)	Putty
AVERAGE DRY TIME AT 75°F. (24°C.)	Skins over in 18 hours — Paint over after 24 hours with conventional coatings
RECOMMENDED THINNER	If material stiffens in cold weather add up to 1 cup linseed oil per 5 gallons of calking. Use Thinner 7-T-38 for clean-up.
RESISTANCE TO	Fumes — Very Good Moisture — Excellent Expansion & Contraction — Very Good Cracking — Very Good Dry Heat — To 250°F. (121°C.)
RECOMMENDED PRIMER STEEL GALVANIZED WOOD MASONRY	 None None Exterior First Coater 17-W-4 or Exterior Primer White 17-W-5 Masonoc Primer 17-W-21 or Exterior Latex Paint 79-W-9
APPLICATION	Calking Gun Glazing Knife Will build 1" on vertical without slumping
CAUTION	Store in warm area in cold weather.

DUM DUM® CALKING 46-F-5

PREPARATION: Surface must be clean, dry and free of dust. Loose paint, mortar, or crumbling putty should be removed. All masonry cracks and mortar joints should be raked clean. Deep cracks and openings should be packed with oakum or fibre glass to about 3/8" of surface. Prime bare surfaces prior to application of Calk.

APPLICATION: If oil has risen to the top, stir thoroughly into the pigment matter. Apply with a calking gun or tool using as heavy a bead as possible. Apply in a manner assuring full contact on sides and bottom of bead. Leave a well rounded bead on the outside. When a smoothed bead is required for appearance, dip tool in water to eliminate sticking. To prevent skin forming when not in use, pour a liberal amount of water over the unused compound and keep container tightly sealed. Pour off water before re-using.

NOTICE: This product is for industrial use only and is not intended or suitable for use in or around a household or dwelling.

Mobil Chemical Company / MAINTENANCE & MARINE COATINGS DEPARTMENT

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Beaumont, Texas 77704
P.O. Box 3431 / Tel: (713) 835-5324

Kankakee, Illinois 60901

Los Angeles Area / Azusa, California 91702
1004 West Tenth Street / Tel: (213) 334-8251

Louisville, Kentucky 40210 (Railroad Coatings)
1630 West Hill Street / Tel: (502) 774-4411

These products also available from C

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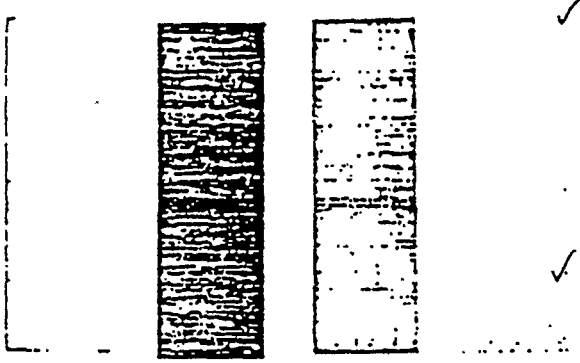

Also available: Chimney Dum Dum 37-Y-3 International Orange

DUM DUM MASONOC / 95 Series

Masonoc is a heavy bodied textured coating designed as a weather seal, waterproofing, and restoration coating for all types of masonry structures. Pliability and elasticity allow for building movement without cracking of the coating while a tough outer skin provides protection for normal usage. Its high build qualities permit hairline cracks to be bridged and sealed, and surface irregularities to be filled and uniformed.

Masonoc also provides fine protection for steel with its high film build and impermeability. Its excellent surface wetting properties recommend use of this material in areas that cannot be cleaned too well, where there are numerous angles and edges to protect, or when a single coat application is all that can be provided.

CHIMNEY DUM DUM PRIMER / 38-V-5

A clear 100 percent solid heat resistant penetrating primer designed for application to masonry surfaces beneath Chimney Dum Dum Coatings.

DUM DUM CAULK / 46-F-5

An oil based caulking compound recommended for glazing and filling operations by knife or gun for joints and cracks where movement is not a critical factor. Recommended specifically for use with 95 Series Masonoc. Excellent adhesion to previously primed surfaces and can be overcoated with conventional finishes after 24 hours.

CHIMNEY DUM DUM / 97 Series

Chimney Dum Dum provides the same outstanding protective and waterproofing qualities as the 95 Series Masonoc, except that it has been formulated for use on concrete chimneys where additional heat resistance up to 210°F. is required.

HI-HEAT DUM DUM / 46-F-7

A heavy semi-plastic fibred coating, which acts as a joint sealer and pliable gasket for boilers, furnaces and dry kilns. Improves boiler efficiency by preventing heat loss. Retains its elasticity at constant heat up to 175° and intermittent heat to 350°. May be used as a complete cover to eliminate excess fuel consumption and can be painted over in 24 hours.

DUM DUM ARMORCOTE / 46-J-9

A specially compounded, heavy bodied product containing asphalt, reinforcing pigment and high boiling hydrocarbons. Resistant to fumes, moisture, and sustained temperatures up to 300°F. Used to protect and increase the efficiency of brick boiler settings. May be applied to boilers even while they are in operation.

MASONOC PRIMER / 47-W-21

Masonoc primer is a normal paint like material which is to be used beneath the Masonoc coating on all types of masonry surfaces to seal off and eliminate surface porosity.

Brief product descriptions are given for the purpose of facilitating selection. Always refer to the individual Mobil Chemical Product Data Sheets for further specific product recommendations and use instructions.

Mobil Chemical Company / MAINTENANCE & MARINE COATINGS DEPARTMENT

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P.O. Box 250 / Tel: (201) 287-2626

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P.O. Box 3431 / Tel: (713) 835-5324

Los Angeles Area / Azusa, California 91702
1004 West Tenth Street / Tel: (213) 334-8251

Louisville, Kentucky 40210 (Railro
1630 West Hill Street / Tel: (502) 7

Mobil Chemical Product Data

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HI-HEAT DUM DUM®

CONFORMS WITH AIR POLLUTION RULES AND REGULATIONS

46-F-7

COLOR	Natural Gray
FINISH	Low
VEHICLE TYPE	Linseed Oil Blend
PIGMENT TYPE	Inerts and Asbestos Fibre
SOLVENT TYPE	Aliphatic hydrocarbons
FLASH POINT, MINIMUM	112°F (44°C.) PMC
% SOLIDS BY VOLUME	85%
RECOMMENDED DRY FILM (per coat)	1/16-3/32 inch (1.6-2.4 mm)
COVERAGE (THEORETICAL)	11 square feet per gallon (0.27 m^2/l) per 1/8" (3.2 mm) dry. The actual coverage will be less, depending on application technique, job conditions and type of surface to be coated.
VISCOSITY AT 75°F. (24°C.)	Mastic
AVERAGE DRY TIME AT 75°F. (24°C.)	Skins in 18 Hours. Paint over after 24 Hours with conventional coatings.
RECOMMENDED THINNER	None – Use at package consistency Clean-up – use Thinner 7-T-38
RESISTANCE TO	Fumes – Good Moisture – Very Good Dry Heat – to 350°F. (177°C.) Intermittent. To 175°F. (79°C.) Sustained
RECOMMENDED USE	Boilers, Furnaces, Kilns, etc., as joint seal, pliable gasket and for overall application during installation and repair.
APPLICATION	Caulking gun, trowel, brush or spray For spray – Heavy duty mastic equipment 6:1 pump with surge control binks 7E2 gun or DeVilbiss MBC 516 with 3/8" Nozzle and fluid tip. Brush – Special Dum Dum Brush.

HI-HEAT DUM DUM®46-F-7

Hi-Heat Dum Dum is recommended for use on furnaces, boilers, kilns and similar installations. It is specially blended to remain pliable and will withstand the expansion and contraction developed by intermittent use.

Hi-Heat Dum Dum can be used as a joint sealer and pliable gasket for boiler settings, as a caulking material for cracks and broken mortar joints, or for application overall on the unit.

Surface should be clean and dry. For masonry use, all cracks should be raked clean and any previously applied caulk or putty removed, then fill with Hi-Heat Dum Dum. On metal, best results will be had if metal is at normal temperature at time of application.

NOTICE: This product is for industrial use only and is not intended or suitable for use in or around a household or dwelling.

WARNING: VAPOR HARMFUL. CAUSES IRRITATION. COMBUSTIBLE. CONTAINS – ORGANIC SOLVENTS. Avoid breathing vapor. Avoid contact with eyes, skin, and clothing. Keep away from heat and open flame. Wash thoroughly after handling. Use with adequate ventilation. Wear an air supplied mask to avoid breathing concentrated vapors in enclosed areas. Keep container closed. FIRST AID: If inhaled, remove to fresh air. If not breathing give artificial respiration, preferably mouth-to-mouth. Call a physician. In case of contact, immediately flush eyes with plenty of water for at least 15 minutes. Call a physician.

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Beaumont, Texas 77704
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Kankakee, Illinois 60901
901 North Greenwood Avenue / Tel: (815) 933-556[illegible]

Los Angeles Area / Azusa, California 91702
[illegible]04 West Temple Street / Tel. (213) 334-8251

Louisville, Kentucky 40210 (Railroad Coatings)
[illegible]830 West Hill Street Tel (502) [illegible]

These products are also available from Mobil Chemical Toronto Canada
Essex Co Canada Sao Paulo Brazil Mexico City Mexico

Mobil Chemical Product Data

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This supersedes all previous publications.
Always consult your Mobil representative for latest information and recommendations for Mobil Products.

DUM DUM® ARMORCOTE

CONFORMS WITH AIR POLLUTION RULES AND REGULATIONS

46-J-9

COLOR	Black
FINISH	Low Sheen
VEHICLE TYPE	Modified asphalt
PIGMENT TYPE	Inert pigments and asbestos fibre
SOLVENT TYPE	Aliphatic hydrocarbons
FLASH POINT, MINIMUM	80°F *(27°C.)* PMC
% SOLIDS BY VOLUME	62%
RECOMMENDED DRY FILM (per coat)	1/16 to 1/8 inches *(1.6 to 3.2 mm)*
COVERAGE (THEORETICAL)	16 square feet per gallon *(0.4 m^2/l)* at 1/16 inch *(1.6 mm)* dry. The actual coverage will be less, depending on application technique, job conditions and type of surface to be coated.
VISCOSITY AT 75°F. *(24°C.)*	Putty
AVERAGE DRY TIME AT 75°F. *(24°C.)*	Surface skin in 1-2 hours Sets up in 24 hours
RECOMMENDED THINNER	Use Thinner 7-T-38 for clean-up only
RESISTANCE TO	Fumes – Very Good Moisture – Excellent Dry Heat – To 300°F. *(149°C.)*
APPLICATION	Trowel

DUM DUM® ARMORCOTE 46-J-9

Armorcote is a blend of specially selected bitumens to provide a low cost resilient material for refractory applications. Used as a complete cover, Armorcote forms an air tight shield which offers substantial fuel savings by eliminating air leaks. It is especially useful around and as a gasketing material for boiler settings.

Because Armorcote is moisture and fume resistant it lends itself to all types of environments including interior of dry kilns where cracks and pores allow heat loss and temperature fluctuations.

Armorcote may be applied up to 1/8 inch thick and will absorb expansion and contraction. It will prevent disintegration of plaster, mortar, concrete, brick, tile and cement block.

PREPARATION — Surface must be clean and dry. Remove all loose mortar or brick particles by wire brushing. Open mortar joints and cracks over 1/8 inch should be raked and filled with quick drying Portland Cement and allowed to set hard.

NOTICE: This product is for industrial use only and is not intended or suitable for use in or around a household or dwelling.

WARNING: FLAMMABLE. VAPOR HARMFUL. CAUSES IRRITATION. CONTAINS – ORGANIC SOLVENTS. Keep away from heat, sparks, and open flame. Avoid breathing vapor. Avoid contact with eyes, skin, and clothing. Wash thoroughly after handling. Use with adequate ventilation. Wear an air supplied mask to avoid breathing concentrated vapors in enclosed areas. Keep container closed. FIRST AID: If inhaled, remove to fresh air. If not breathing give artificial respiration, preferably mouth-to-mouth. Call a physician. In case of contact, immediately flush eyes with plenty of water for at least 15 minutes. Call a physician.

Mobil Chemical Company / MAINTENANCE & MARINE COATINGS DEPARTMENT

Edison, New Jersey 08817
P.O. Box 250 / Tel: (201) 287-2626

Beaumont, Texas 77704
P.O. Box 3431 / Tel: (713) 835-5324

Kankakee, Illinois 60901
901 North Greenwood Avenue / Tel: (815) 933-8561

Los Angeles Area / Azusa, California
[illegible] West Tenth Street / Tel: [illegible]

Louisville, Kentucky
[illegible] Westport Road / Tel: [illegible]

These products are also available from Mobil Chemical Company Distributors throughout the United States and Canada.

Mobil Chemical Product Data

NOVEMBER 1, 1976 / Printed in U.S.A.
This supersedes all previous publications.
Always consult your Mobil representative for latest information and recommendations for Mobil Products.

DUM DUM MASONOC®

CONFORMS WITH AIR POLLUTION RULES AND REGULATIONS

95 SERIES

FINISH	Low sheen
VEHICLE TYPE	Resinated Vegetable Oils
PIGMENT TYPE	Titanium Dioxide – Color pigments – Asbestos Fibre
SOLVENT TYPE	Aliphatic hydrocarbons
FLASH POINT, MINIMUM	105°F. *(41°C.)* PMC
% SOLIDS BY VOLUME	42% depending on color
RECOMMENDED DRY FILM (per coat)	25 mils *(625 µm)*
COVERAGE (THEORETICAL)	674 square feet per gallon *(16.5 m^2/ltr)* per dry mil *(25 µm)* average, depending on color. The actual coverage will be less, depending on application technique, job conditions and type of surface to be coated.
VISCOSITY AT 75°F. *(24°C.)*	Mastic
AVERAGE DRY TIME AT 75°F. *(24°C.)*	Skins over in 1 hour – Under surface remains pliable
RECOMMENDED THINNER	Do not thin for application – In cold weather store in warm areas before use. For clean-up use Thinner 7-T-38.
RESISTANCE TO	Dry Heat – To 150°F. *(66°C)* Salt Air – Excellent Weather extremes – Excellent Chemical Fumes – Excellent Moisture – Excellent
RECOMMENDED PRIMER STEEL GALVANIZED WOOD MASONRY	 None required None required Not recommended 47-W-21 Masonoc Primer
APPLICATION	Heavy duty spray – Trowel – Special brush. CONVENTIONAL SPRAY (Suggested Equipment) Heavy duty mastic equipment, 10:1 pump, 1" I.D. fluid hose. Binks 7E2 gun (45 x 3/8F nozzle) and DeVilbiss MBC 516 gun (MB-78 cap ¼") have given good results. Approximate settings – pump pressure 40 psi, atomizing pressure 70 psi.
NOTE	Cracks in excess of 1/32" *(0.8 mm)* should first be caulked with Dum Dum Caulking Compound.
CAUTION	Do not use on glazed brick without consulting Mobil representative.

DUM DUM MASONOC®95 SERIES

Dum Dum Masonoc is a heavily bodied, textured coating desired for use in hi-build applications for the waterproofing and restoration of exterior masonry and for the protection of steel surfaces.

PREPARATION: Old masonry surfaces should be prepared by blast cleaning or other suitable methods to remove dust, dirt, surface contaminants, and loose particles. Any spalling concrete, loose mortar, and unsound masonry must be removed and the surface repaired and brought to level as needed. Portland cement patches must be allowed to cure and then neutralized prior to overcoating.

Prime all bare masonry surface with a coat of Masonoc Primer 47-W-21. Do not apply more primer than is needed to seal the surface. Allow primer an overnight dry and then fill all cracks with suitable caulking material prior to top-

New masonry surfaces must be allowed to cure for a sufficient length of time to reduce alkalinity. A minimum of 30 days time is required with at least 6 months desired.

Steel surfaces should be cleaned by blasting or wire brushing. Dum Dum Masonoc may be applied directly to the bare steel if desired, or over a suitable Mobil steel primer.

NOTICE: This product is for industrial use only and is not intended or suitable for use in or around a household or dwelling.

WARNING: VAPOR HARMFUL. CAUSES IRRITATION. COMBUSTIBLE. CONTAINS – ORGANIC SOLVENTS. Avoid breathing vapor. Avoid contact with eyes, skin, and clothing. Keep away from heat and open flame. Wash thoroughly after handling. Use with adequate ventilation. Wear an air supplied mask to avoid breathing concentrated vapors in enclosed areas. Keep container closed. FIRST AID: If inhaled, remove to fresh air. If not breathing give artificial respiration, preferably mouth-to-mouth. Call a physician. In case of contact, immediately flush eyes with plenty of water for at least 15 minutes. Call a physician.

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Los Angeles Area / Azusa, California 91702
[illegible]24 West Tenth Street / Tel (213) 334-8251

Louisville, Kentucky 402[illegible] (Railroad Coatings)
[illegible]30 West Hill Street / Tel (502) 774-4411

[illegible] products also available from [illegible] Holland/Toronto, Canada
Bogota, Colombia Sao Paulo, Brazil Mexico City, Mexico

Mobil Chemical Product Data

NOVEMBER 1, 1976 / Printed in U.S.A.
This supersedes all previous publications.
Always consult your Mobil representative for latest information and recommendations for Mobil Products.

CHIMNEY DUM DUM®

CONFORMS WITH AIR POLLUTION RULES AND REGULATIONS

97 SERIES

FINISH	Medium gloss
VEHICLE TYPE	Resinated Vegetable Oils
PIGMENT TYPE	Titanium Dioxide, Asbestos Fibre, Mica plus color fast pigments.
SOLVENT TYPE	Aliphatic hydrocarbons
FLASH POINT, MINIMUM	101°F. *(38°C.)* PMC
% SOLIDS BY VOLUME	63% average, depending on color
RECOMMENDED DRY FILM (per coat)	25 mils *(625 µm)*
COVERAGE (THEORETICAL)	1010 square feet per gallon *(24.8 m^2/ltr)* per dry mil *(25 µm)* average, depending on color. The actual coverage will be less, depending on application technique, job conditions and type of surface to be coated.
VISCOSITY AT 75°F. *(24°C.)*	Mastic
AVERAGE DRY TIME AT 75°F. *(24°C.)*	Surface skin 4-6 hrs. Under surface remains pliable.
RECOMMENDED THINNER	Do not thin for application. Use 7-T-38 for clean-up.
RESISTANCE TO	Moisture — Excellent Chemical Fumes — Very Good Dry Heat — To 210°F. *(99°C.)* constant surface temperature.
RECOMMENDED PRIMER	Chimney Dum Dum Primer 38-V-5
APPLICATION	Brush — Spray CONVENTIONAL SPRAY (Suggested Equipment) Heavy duty mastic equipment, 10:1 pump, 1" I.D. fluid hose. Binks 7E2 gun (45 x 3/8F nozzle) and DeVilbiss MBC 516 gun (MB-78 cap ¼") have given good results. Approximate settings — pump pressure 40 psi, atomizing pressure 70 psi.
NOTE	On stacks that are in part-time use, apply thin brush coats of Chimney Dum Dum to approx. 10 mils *(250 µm)* wet total. Normal heavy coats could be damaged by rain collection on the interior of the stack.
CAUTION	NEW CONCRETE MUST WEATHER A MINIMUM OF 3 MONTHS BEFORE COATING! MEMBRANE CURING AGENTS MUST BE COMPLETELY WEATHERED OR OTHERWISE REMOVED BEFORE COATING.

CHIMNEY DUM DUM®97 SERIES

Chimney Dum Dum is a rugged heavy coating for the exterior of chimneys that provides flexible weatherproof protection. It fills and bridges hairline cracks with a flexible coating under a tough outer skin. Should the leathery skin become fractured, the soft under film will harden on contact with air. May be applied without completely cooling stack.

LININGS MUST BE INSPECTED FOR INTEGRITY AND FREEDOM FROM AIR LEAKS.

PREPARATION: Must be clean and dry. Surface should be thoroughly wire brushed to remove dirt, grease, soot and old coating. Cracks larger than hairline up to ½" wide should be raked, cleaned out and primed with 38-V-5 Chimney Dum Dum Primer and filled with Calk. Fill larger cracks with quick-setting cement. New cement patches should be treated to neutralize alkali content. Prime entire surface with 38-V-5 Chimney Dum Dum Primer.

NOTICE: This product is for industrial use only and is not intended or suitable for use in or around a household or dwelling.

WARNING: VAPOR HARMFUL. CAUSES IRRITATION. COMBUSTIBLE. CONTAINS – ORGANIC SOLVENTS. Avoid breathing vapor. Avoid contact with eyes, skin, and clothing. Keep away from heat and open flame. Wash thoroughly after handling. Use with adequate ventilation. Wear an air supplied mask to avoid breathing concentrated vapors in enclosed areas. Keep container closed. FIRST AID: If inhaled, remove to fresh air. If not breathing give artificial respiration, preferably mouth-to-mouth. Call a physician. In case of contact, immediately flush eyes with plenty of water for at least 15 minutes. Call a physician.

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901 North Greenwood Avenue / Tel: (815) 933-5561

Los Angeles Area / Azusa, California 91702
1004 West Tenth Street / Tel: (213) 334-2251

Louisville, Kentucky 40210 (Railroad Coatings)
1630 West Hill Street / Tel: (502) 774-4411

These products also available from Delft, Holland; Toronto, Canada; Bogota, Colombia; Sao Paulo, Brazil; Mexico City, Mexico

NOVEMBER 1975 / Printed in U.S.A.
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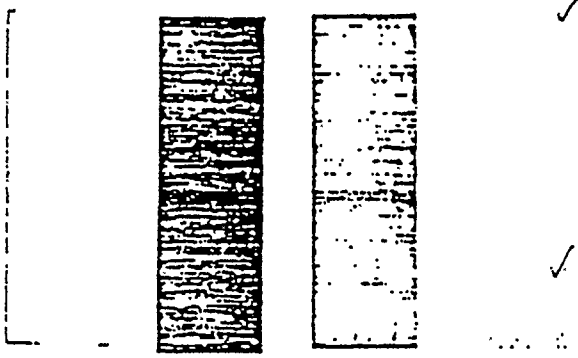

Also available: Chimney Dum Dum 97-Y-3 International Orange

DUM DUM MASONOC / 95 Series

Masonoc is a heavy bodied textured coating designed as a weather seal, waterproofing, and restoration coating for all types of masonry structures. Pliability and elasticity allow for building movement without cracking of the coating while a tough outer skin provides protection for normal usage. Its high build qualities permit hairline cracks to be bridged and sealed, and surface irregularities to be filled and uniformed.

Masonoc also provides fine protection for steel with its high film build and impermeability. Its excellent surface wetting properties recommend use of this material in areas that cannot be cleaned too well, where there are numerous angles and edges to protect, or when a single coat application is all that can be provided.

CHIMNEY DUM DUM PRIMER / 38-V-5

A clear 100 percent solid heat resistant penetrating primer designed for application to masonry surfaces beneath Chimney Dum Dum Coatings.

DUM DUM CAULK / 46-F-5

An oil based caulking compound recommended for glazing and filling operations by knife or gun for joints and cracks where movement is not a critical factor. Recommended specifically for use with 95 Series Masonoc. Excellent adhesion to previously primed surfaces and can be overcoated with conventional finishes after 24 hours.

CHIMNEY DUM DUM / 97 Series

Chimney Dum Dum provides the same outstanding protective and waterproofing qualities as the 95 Series Masonoc, except that it has been formulated for use on concrete chimneys where additional heat resistance up to 210°F. is required.

HI-HEAT DUM DUM / 46-F-7

A heavy semi-plastic fibred coating, which acts as a joint sealer and pliable gasket for boilers, furnaces and dry kilns. Improves boiler efficiency by preventing heat loss. Retains its elasticity at constant heat up to 175° and intermittent heat to 350°. May be used as a complete cover to eliminate excess fuel consumption and can be painted over in 24 hours.

DUM DUM ARMORCOTE / 46-J-9

A specially compounded, heavy bodied product containing asphalt, reinforcing pigment and high boiling hydrocarbons. Resistant to fumes, moisture, and sustained temperatures up to 300°F. Used to protect and increase the efficiency of brick boiler settings. May be applied to boilers even while they are in operation.

MASONOC PRIMER / 47-W-21

Masonoc primer is a normal paint like material which is to be used beneath the Masonoc coating on all types of masonry surfaces to seal off and eliminate surface porosity.

Brief product descriptions are given for the purpose of facilitating selection. Always refer to the individual Mobil Chemical Product Data Sheets for further specific product recommendations and use instructions.

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Louisville, Kentucky 40210 (Railroad Coatings)
1630 West Hill Street / Tel: (502) 774-4[illegible]

NOVEMBER, 1976 / Printed in U.S.A.
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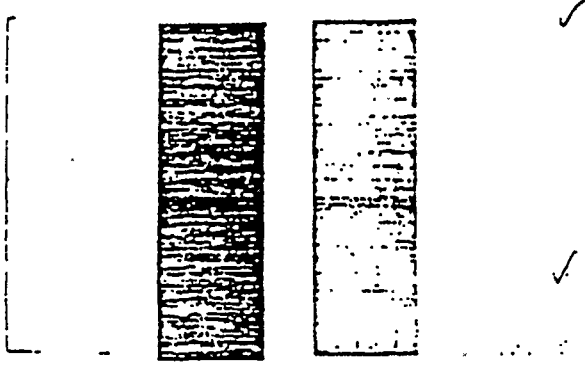

Also available: Chimney Dum Dum 97-Y-3 International Orange

DUM DUM MASONOC / 95 Series

Masonoc is a heavy bodied textured coating designed as a weather seal, waterproofing, and restoration coating for all types of masonry structures. Pliability and elasticity allow for building movement without cracking of the coating while a tough outer skin provides protection for normal usage. Its high build qualities permit hairline cracks to be bridged and sealed, and surface irregularities to be filled and uniformed.

Masonoc also provides fine protection for steel with its high film build and impermeability. Its excellent surface wetting properties recommend use of this material in areas that cannot be cleaned too well, where there are numerous angles and edges to protect, or when a single coat application is all that can be provided.

CHIMNEY DUM DUM PRIMER / 38-V-5

A clear 100 percent solid heat resistant penetrating primer designed for application to masonry surfaces beneath Chimney Dum Dum Coatings.

DUM DUM CAULK / 46-F-5

An oil based caulking compound recommended for glazing and filling operations by knife or gun for joints and cracks where movement is not a critical factor. Recommended specifically for use with 95 Series Masonoc. Excellent adhesion to previously primed surfaces and can be overcoated with conventional finishes after 24 hours.

CHIMNEY DUM DUM / 97 Series

Chimney Dum Dum provides the same outstanding protective and waterproofing qualities as the 95 Series Masonoc, except that it has been formulated for use on concrete chimneys where additional heat resistance up to 210°F. is required.

HI-HEAT DUM DUM / 46-F-7

A heavy semi-plastic fibred coating, which acts as a joint sealer and pliable gasket for boilers, furnaces and dry kilns. Improves boiler efficiency by preventing heat loss. Retains its elasticity at constant heat up to 175° and intermittent heat to 350°. May be used as a complete cover to eliminate excess fuel consumption and can be painted over in 24 hours.

DUM DUM ARMORCOTE / 46-J-9

A specially compounded, heavy bodied product containing asphalt, reinforcing pigment and high boiling hydrocarbons. Resistant to fumes, moisture, and sustained temperatures up to 300°F. Used to protect and increase the efficiency of brick boiler settings. May be applied to boilers even while they are in operation.

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1630 West Hill Street / Tel: (502) 7

Mobil 1980

MATERIAL SAFETY DATA SHEET

NPCA 1

FOR COATINGS, RESINS AND RELATED MATERIALS

(Approved by U S Department of Labor Essentially Similar to Form OSHA-20)

DATE OF PREP 9/26/80

Section I

MANUFACTURER'S NAME THE GIBSON-HOMANS COMPANY

STREET ADDRESS 1755 Enterprise Pkwy. CITY, STATE, AND ZIP CODE Twinsburg, OH 44087

EMERGENCY TELEPHONE NO. 216/425-3255

PRODUCT CLASS

MANUFACTURERS CODE IDENTIFICATION AX-2789-H

TRADE NAME ASBESTOS FIBRE-FREE MOBIL 46F7

Section II — HAZARDOUS INGREDIENTS

INGREDIENT	PERCENT	TLV PPM	TLV mg/M³	LEL	VAPOR PRESSURE
Mineral Spirits	3.8	500			2.0

Section III — PHYSICAL DATA

BOILING RANGE Mineral Spirits 310°F. VAPOR DENSITY [XX] HEAVIER [ ] LIGHTER THAN AIR

EVAPORATION RATE [ ] FASTER [XX] SLOWER THAN ETHER PERCENT VOLATILE BY VOLUME 4% WEIGHT PER GALLON 12.5#

Section IV — FIRE AND EXPLOSION HAZARD DATA

FLAMMABILITY CATEGORY Combustible FLASH POINT Closed Cup 105°F. minimum

EXTINGUISHING MEDIA

CO_2, Dry Chemical, Foam

UNUSUAL FIRE AND EXPLOSION HAZARDS

SPECIAL FIRE FIGHTING PROCEDURES

Contains solvent, water not effective.

MOB-HarrisMaster
00493

Section V – HEALTH HAZARD DATA

OLD LIMIT VALUE See Section II.

S OF OVEREXPOSURE

Inhalation - possible dizziness, light headedness.

MERGENCY AND FIRST AID PROCEDURES

See physician. Flush with water.

Section VI – REACTIVITY DATA

STABILITY ☐ UNSTABLE ☒ STABLE CONDITIONS TO AVOID Sources of ignition.

NCOMPATABILITY (Materials to avoid)

AZARDOUS DECOMPOSITION PRODUCTS

AZARDOUS POLYMERIZATION ☐ MAY OCCUR ☒ WILL NOT OCCUR

ONDITIONS TO AVOID

Section VII – SPILL OR LEAK PROCEDURES

TEPS TO BE TAKEN IN CASE MATERIAL IS RELEASED OR SPILLED

Contain spill. Remove all sources of ignition. Scoop up and return to container.

E DISPOSAL METHOD

Place in trash or use for landfill according to regulations.

Section VIII – SPECIAL PROTECTION INFORMATION

ESPIRATORY PROTECTION

ENTILATION Not normally required.

ROTECTIVE GLOVES Not normally; if needed, use rubber gloves.

YE PROTECTION Not normally.

THER PROTECTIVE EQUIPMENT

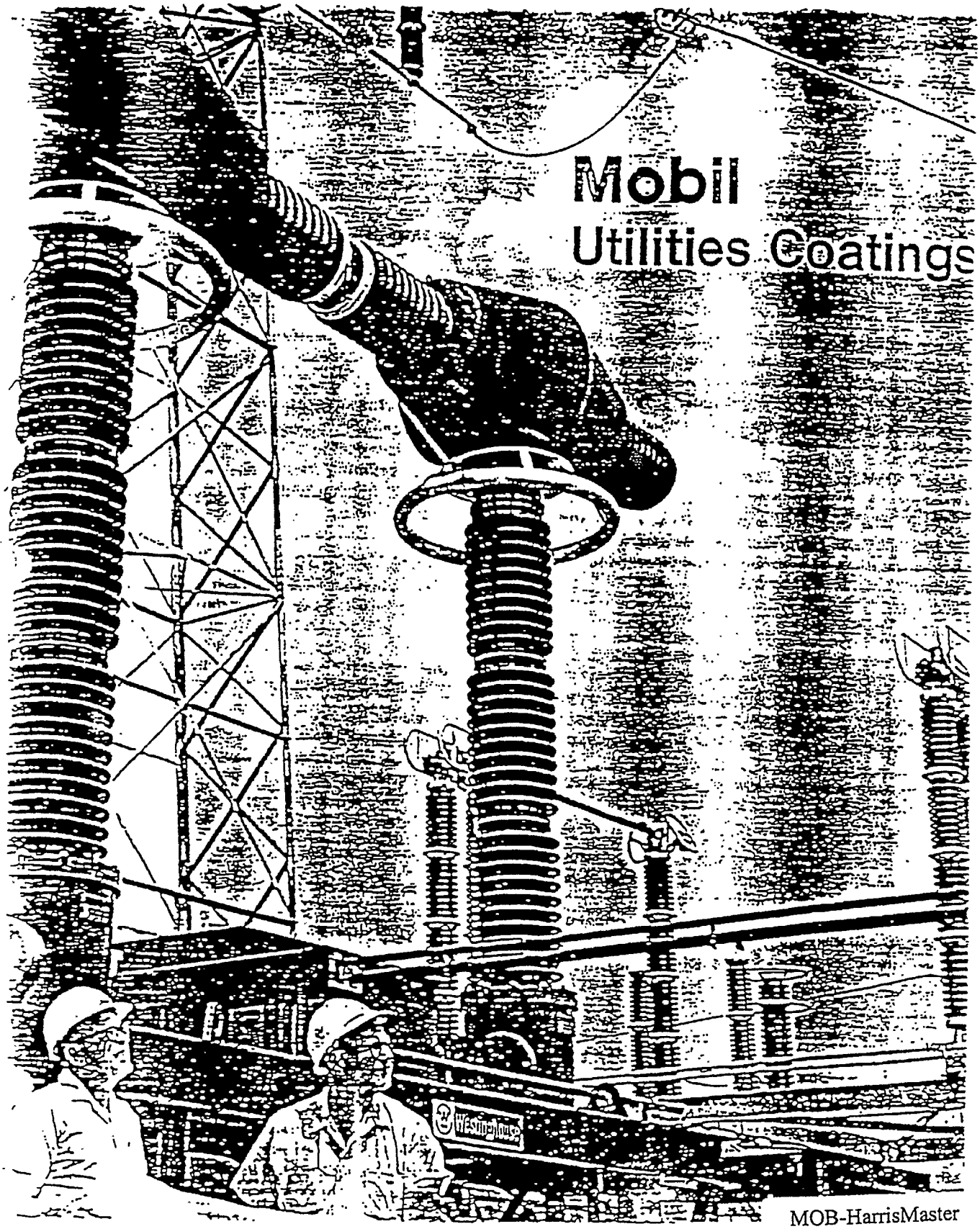
Section IX – SPECIAL PRECAUTIONS

RECAUTIONS TO BE TAKEN IN HANDLING AND STORING

Keep away from open flame.

THER PRECAUTIONS

Store in accordance to NFPA, State and local regulations.


Mobil
Utilities Coatings

PANORAMA COATINGS / 12 Series

A complete line of attractive colors, designed to provide the "modern look" for all types of industrial installations. These materials are formulated primarily for service in the industrial-environments to combine weather durability, resistance to mildly corrosive exposures, with good film build and ease of application with all generally used methods. They provide excellent color retention and appearance throughout a long service life.

STAINLESS STEEL PAINTS

Stainless steel pigmented coatings in both one coat high build quality and finish coat materials for previously primed surfaces. Provide excellent durability and steel protection.

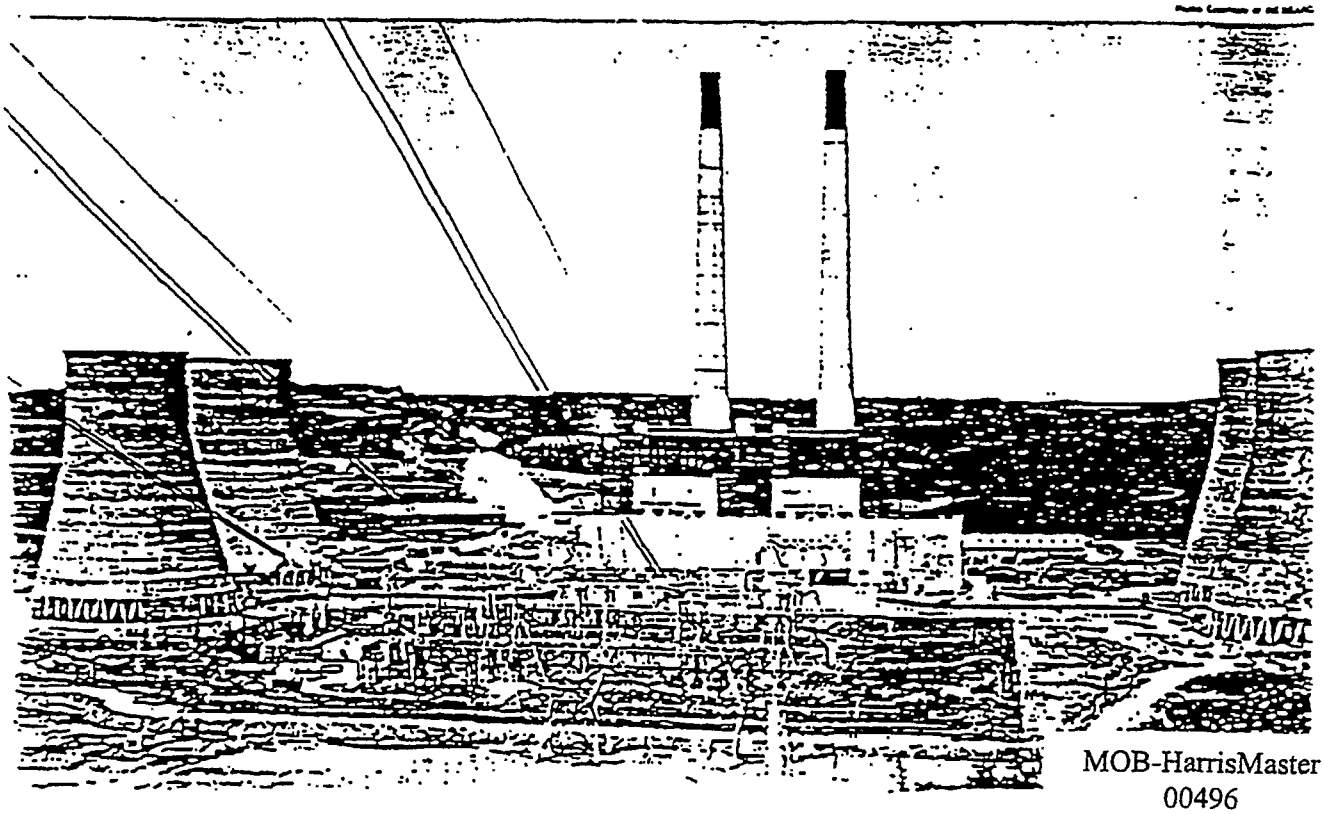
MOBILTONE LATEX FLAT WALL FINISH / 77 S

This Vinyl Latex is a flat, interior finish that pr uniformity of color and sheen . . . rapid drying . . . e application. Wide range of attractive colors ma ideal for use on walls, ceilings and woodwork.

HI-HEAT DUM DUM / 46-F-7

This material acts as a joint sealer and pliable g for boilers, furnaces and dry kilns. It improves efficiency by preventing heat loss. Retains its ela at constant heat up to 175° and intermittent heat tc

A heavy semi-plastic fibred coating, it can be us a complete cover to eliminate excess fuel consur and can be painted over in 24 hours.



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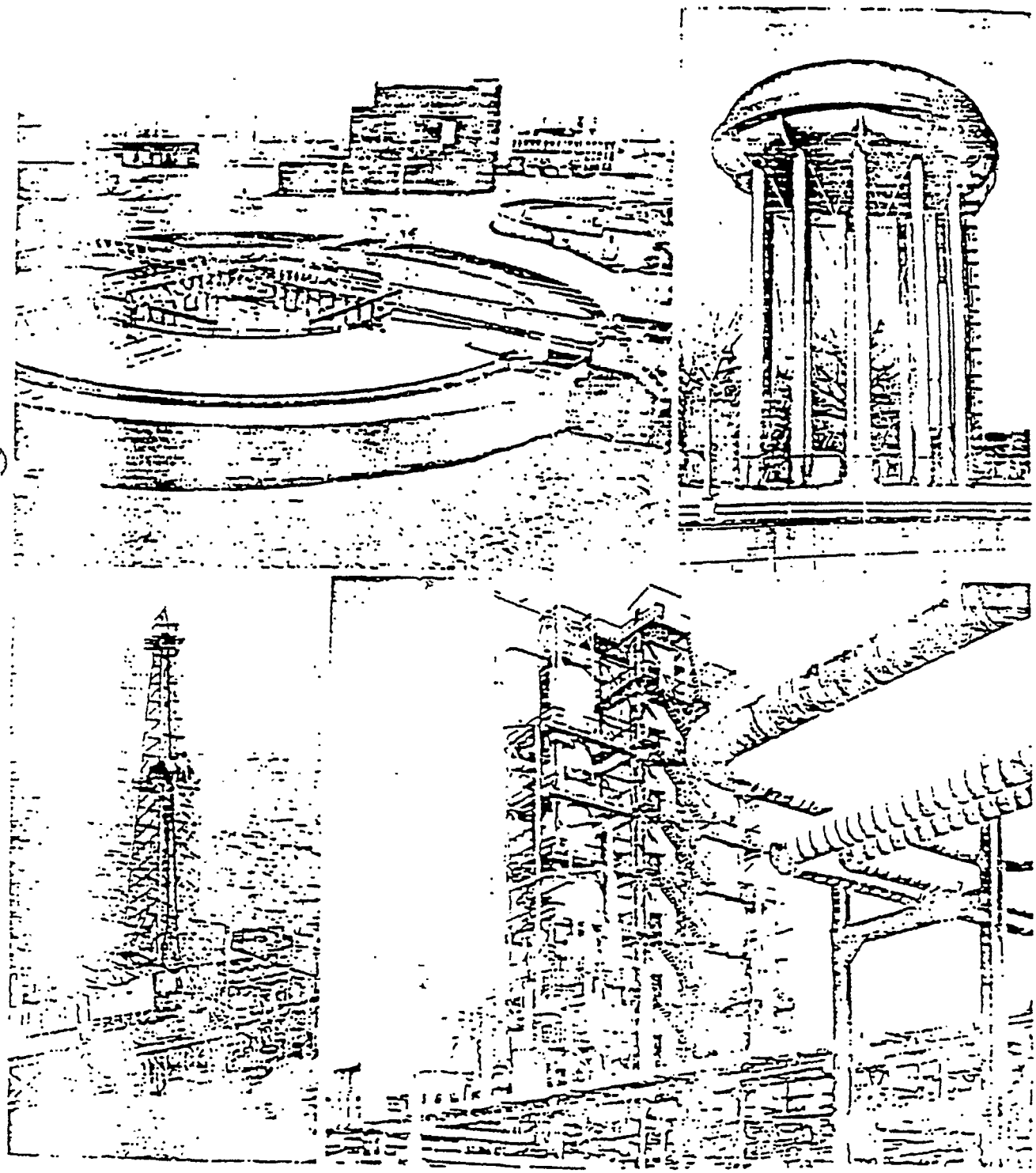
Mobil Chemical
Protective Coatings
Products Catalog



Dum Dum Products

DUM DUM MASONOC / 95 Series

Masonoc is a heavy bodied textured coating designed as a weather seal, waterproofing, and restoration coating for all types of masonry structures. Flexibility and elasticity allow for building movement without cracking of the coating while a tough outer film provides protection for normal usage. Its high thixotropic qualities permit hairline cracks to be bridged and sealed, and surface irregularities to be filled and uniformed.

Masonoc also provides fine protection for steel due to its high film build and impermeability. Its excellent surface wetting properties recommend use of this material in areas that cannot be cleaned too well or where there are numerous angles and edges to protect, or when a single coat application is all that can be provided.

CHIMNEY DUM DUM PRIMER / 38-V-5

A clear 100 percent solid heat resistant penetrating primer designed for application to masonry surfaces beneath Chimney Dum Dum Coatings.

DUM DUM CAULK / 46-F-5

Oil based caulking compound recommended for caulking and filling operations by knife or gun for joints and cracks where movement is not a critical factor. Recommended specifically for use with 95 Series Masonoc. Excellent adhesion to previously primed surfaces and can be overcoated with conventional finishes after 24 hours.

CHIMNEY DUM DUM / 97 Series

Chimney Dum Dum provides the same outstanding protective and waterproofing qualities as the 95 Series Masonoc, except that it has been formulated for use on concrete chimneys where additional heat resistance up to 210°F. is required.

HI-HEAT DUM DUM / 46-F-7

A heavy semi-plastic fibred coating, which acts as a joint sealer and pliable gasket for boilers, furnaces and dry kilns. Improves boiler efficiency by preventing heat loss. Retains its elasticity at constant heat up to 175° and intermittent heat to 350°. May be used as a complete cover to eliminate excess fuel consumption and can be painted over in 24 hours.

DUM DUM ARMORCOTE / 46-J-9

A specially compounded, heavy bodied product containing asphalt, reinforcing pigment and high boiling hydrocarbons. Resistant to fumes, moisture, and sustained temperatures up to 300°F. Used to protect and increase the efficiency of brick boiler settings. May be applied to boilers even while they are in operation.

MASONOC PRIMER / 47-W-21

Masonoc primer is a normal paint like material which is to be used beneath the Masonoc coating on all types of masonry surfaces to seal off and eliminate surface porosity.

ACRYLIC LATEX CALK / 46-W-6

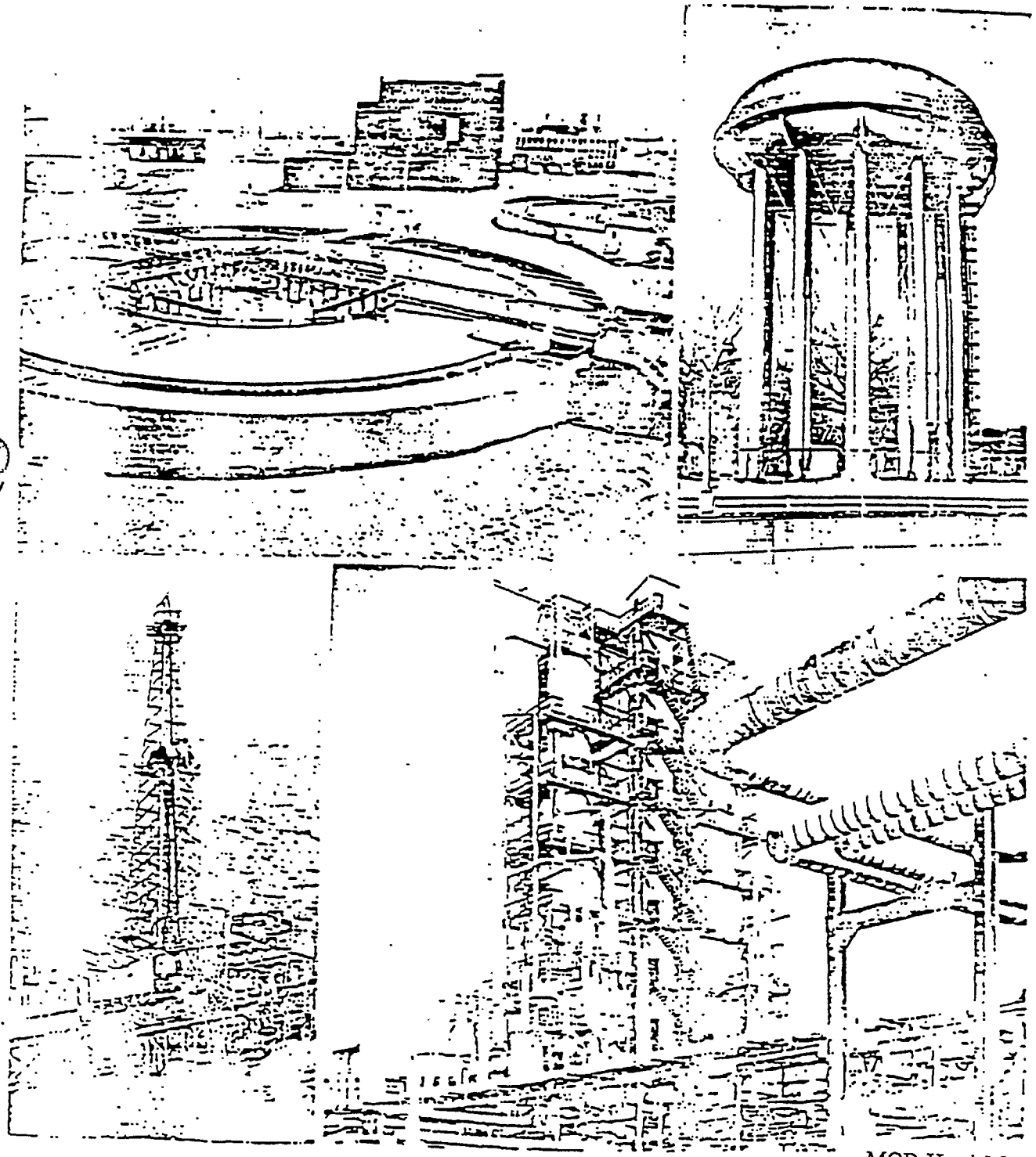
Acrylic Latex Calk 46-W-6 is a high quality caulking material designed for use around windows, doors, flashings, wall board joints or similar openings that require sealing. It is particularly effective for mortar joints and for filling masonry cracks. Acrylic Latex Calk exhibits excellent weatherability, flexibility and good adhesion to wood, masonry, glass and metal.

JOINTITE / 46-X-10

Jointite is a pipe thread compound for sealing joints of pipes, caps, etc., to prevent air and liquid leaks. It is made in a semi-paste consistency. Jointite will never harden and allows disjoining years later with ease. For use on joints in steam, gas, water, and air service. Can be used on potable water lines. Not recommended for use on gasoline or solvent lines.

Mobil 19xxc

Mobil Chemical
Protective Coatings
Products Catalog



Dum Dum Products

DUM DUM MASONOC / 95 Series

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Masonoc also provides fine protection for steel due to its high film build and impermeability. Its excellent surface wetting properties recommend use of this material in areas that cannot be cleaned too well, where there are numerous angles and edges to protect, or when a single coat application is all that can be provided.

CHIMNEY DUM DUM PRIMER / 38-V-5

A clear 100 percent solid heat resistant penetrating primer designed for application to masonry surfaces beneath Chimney Dum Dum Coatings.

DUM DUM CAULK / 46-F-5

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CHIMNEY DUM DUM / 97 Series

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HI-HEAT DUM DUM / 46-F-7

A heavy semi-plastic fibred coating, which acts as a joint sealer and pliable gasket for boilers, furnaces and dry kilns. Improves boiler efficiency by preventing heat loss. Retains its elasticity at constant heat up to 175° and intermittent heat to 350°. May be used as a complete cover to eliminate excess fuel consumption and can be painted over in 24 hours.

DUM DUM ARMORCOTE / 46-J-9

A specially compounded, heavy bodied product containing asphalt, reinforcing pigment and high boiling hydrocarbons. Resistant to fumes, moisture, and sustained temperatures up to 300°F. Used to protect and increase the efficiency of brick boiler settings. May be applied to boilers even while they are in operation.

MASONOC PRIMER / 47-W-21

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ACRYLIC LATEX CALK / 46-W-6

Acrylic Latex Calk 46-W-6 is a high quality caulking material designed for use around windows, doors, fittings, wall board joints or similar openings that require sealing. It is particularly effective for mortar joints and for filling masonry cracks. Acrylic Latex Calk exhibits excellent weatherability, flexibility and good adhesion to wood, masonry, glass and metal.

JOINTITE / 46-X-10

Jointite is a pipe thread compound for sealing joints, unions, caps, etc., to prevent air and liquid leaks. Sets up in a semi-paste consistency. Jointite will never harden and allows disjoining years later with ease. For use on joints in steam, gas, water, and air service. Can be used on potable water lines. Not recommended for use on gasoline or solvent lines.

APPENDIX UI

Evaluation of Asbestos Release from Exterior Masonry Weatherproofing Mastic During Application and Removal

By

Arthur D. Little, Inc.
Acorn Park
Cambridge, MA 02140-2390

1992

Reference No. 65191

Product Background

From the mid-1960's through the 1970's, Mobil Chemical Company manufactured and sold a line of mastic-type protective coatings under the name Dum Dum® Products. Dum Dum Masonoc/Series 95 was a restoration product for weatherproofing masonry structures. The Mobil Chemical Product Catalog states:

> "Dum Dum Masonoc/95 Series Masonoc is a heavy bodied textured coating designed as a weather seal, waterproofing, and restoration coating for all types of masonry structures. Pliability and elasticity allow for building movement without cracking of the coating while a tough outer skins provides protection for normal usage. Its high build qualities permit hairline cracks to be bridged and sealed, and surface irregularities to be filled and uniformed.
>
> Masonoc also provides fine protection for steel with its high film build and impermeability. Its excellent surface wetting properties recommend use of this material in areas that cannot be cleaned too well, where there are numerous angles and edges to protect, or when a single coat application is all that can be provided."

Masonoc consists of a resinated vegetable oil vehicle with about 22% solid fillers (by weight), including titanium dioxide, the chrysotile form of asbestos and various coloring pigments; the total solids content is 42 percent by volume. After application, Masonoc skins over in 1 hour, but the undersurface remains pliable to allow for expansion and contraction. The Mobil Chemical Product Data Sheet for Dum Dum Masonoc claims excellent resistance to dry heat (to 150°F), weather extremes, chemical fumes, moisture and salt air.

Product Testing During Application and Removal

Three 5 gallon pails of Dum Dum Masonoc (soft white 95-W-9) were obtained from an electric utility seeking information on product disposal. A sample was taken from one pail, about a third full, and ashed at 500°C. Microscopic analysis of this ash confirmed the presence of the chrysotile form of asbestos. The other two pails were intact with no visible signs of having been opened. After thorough mixing and being satisfied that the product viscosity was in the range of a mastic, these two pails of Masonoc were used for application and removal experiments. This work was conducted at our facilities in compliance with United States and Massachusetts rules and regulations applying to asbestos and in accordance with an approved Arthur D. Little, Inc. Health and Safety Plan.

A test plan was developed to simulate typical work practices for product application and removal[1]. In addition, a sampling and analysis protocol was developed to

[1]Although intended for exterior use, this product was tested in an interior (controlled) environment.

Table 1 Asbestos Exposures During Application and Removal of Dum Dum Masonoc®			
Sample No.	Description	Sample Type	Exposure (s/cc)[4]
072701	First Application	Personal	0.01
072702	First Application	Area	<0.01
072703	Second Application	Personal	<0.01
072704	Second Application	Area	<0.01
111406	First Removal	Personal	<0.01
111407	First Removal (replicate)	Personal	<0.01
111403	First Removal	Area	<0.01
050104	Second Removal	Personal	<0.01
050107	Second Removal (replicate)	Personal	<0.01
050105	Second Removal	Area	<0.01

Conclusions

The conclusions reached in this study are as follows:

- After thorough mixing, the obtained pails of Dum Dum Masonoc®, confirmed to contain chrysotile asbestos, were smooth and of mastic consistency.
- Application to concrete panels required heavy duty mastic spray apparatus.
- After aging at 150°F for 42 days and additional periods of 67 and 168 days at ambient conditions, the product was observed to be pliable and elastic when depressed with a tool.
- Results of worker exposure measurements during application and removal of Dum Dum Masonoc® in a controlled environment did not exceed the minimum detection limit of 0.01 structures/cc.
- Based upon the above results, occupational exposure to airborne asbestos fibers during the application or removal of Dum Dum Masonoc® is virtually non-existent.

[4]Asbestos structures per unit volume of air.

quantify the extent to which asbestos structures[2] may be released during the experimental application and removal activities. This protocol was designed so that all applicable NIOSH and EPA requirements for analysis by transmission electron microscopy were met or exceeded. The minimum detection limit for the analytical protocol was 0.01 structures/cc[3].

To evaluate the release of asbestos during product application and removal, concrete panels 4 feet by 2-1/2 feet by 1-1/2 inch were prepared in wooden frames and cured for 42 days. The panels were then each coated with Masonoc Primer 47-W-21, "a clear 100 percent solid heat resistant penetrating primer", dried overnight and then coated with Dum Dum Masonoc, using a Binks 7E2 spray gun and 2 gallon pressure pot, to a thickness of approximately 30-40 mils. Approximately 15 panels were coated during each of two 80 minute application experiments. Personal exposure and area air samples were collected during both experiments. The area sample was located about 10 feet from the worker (sprayer) at an elevation of 5 feet.

After product application, the coated panels were stacked (with spacers), allowed to dry for 24 hours and were then moved to an oven and exposed to flowing dry air at 150°F (the maximum recommended use temperature) for 42 days to accelerate the aging process followed by an additional 67 days at ambient conditions.

After aging, the product was found to be pliable and elastic when pressed with a screwdriver blade. A product removal experiment was performed using a putty knife and scraper. Eight panels were cleaned over an approximately 80 minute period during which personal samples were collected. After a further ambient aging period of 168 days (a total of 278 days after application), and again observing the product to be pliable and elastic, a second replicate removal experiment was performed on ten panels.

The collected filter samples from these experiments were prepared and analyzed by transmission electron microscopy. The results of these analyses are given in Table 1. These results may be overstated compared to actual exposures which would occur in an open air environment. As such, they are considered to represent a "worst case" condition.

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[2]Asbestos structures are defined herein as particles 5um or more in extent that satisfy the counting guidelines given in 40 CFR Part 763 Asbestos-Containing Materials in Schools, Appendix A, Figure 5 (Federal Register 52 (210) October 30, 1987, 41866-41867). Structures include fibers, bundles, clumps and matrix. OSHA regulates exposure to fibers, which are defined as "a particulate form of asbestos, tremolite, anthophyllite or actinolite, 5 micrometers or longer, with a length-to-diameter ratio of at least 3 to 1" (29 CFR 1910.1001)

[3]The minimum detection limit (MDL) for this experiment is the concentration where occupational exposure cannot be differentiated from general background.

Resiliency of Mobil Hi-Heat Dum Dum after High Temperature Exposure

By

Arthur D. Little, Inc.
Acorn Park
Cambridge, MA 02140-2390

1992

Reference No. 65191

Product Background

High temperature resinous materials of mastic (putty-like) consistency are used in sealing joints and crevices on furnaces and boilers to prevent heat loss. Mobil Chemical Company manufactured and sold such a caulking product from the mid-1960s through the 1970s called Hi-Heat Dum Dum. This product, No. 46-F-7, consisted of a linseed oil-base vehicle (resin) containing various solid fillers, including clay and the chrysotile variety of asbestos (added for its resiliency and bridging properties). The Mobil Chemical Products Catalog provides the following description:

> "Hi-Heat Dum Dum/46-F-7, a heavy semi-plastic fibred coating, which acts as a joint sealer and pliable gasket for boilers, furnaces and dry kilns. Improves boiler efficiency by preventing heat loss. Retains its elasticity at consistent heat up to 175° and intermittent heat to 350°. May be used as a complete cover to eliminate excess fuel consumption and can be painted over in 24 hours."

The purpose of a joint or crevice sealant is to bridge gaps between the furnace enclosure and attachments (doors, ports, flanges, etc.). This requires a product that is pliable and resilient (i.e., withstands shock without permanently deforming, tearing, rupturing or pulling away from the furnace surface), and does not degrade over time by becoming friable. The resinous formulation of Hi-Heat Dum Dum provides these properties and also effectively encapsulates the asbestos component, preventing its release.

Purpose of the Evaluation

The objective of the thermal exposure experiments was to determine the useful service life of Hi-Heat Dum Dum for temperatures within and above the recommended service temperature range described in the product literature.

Product Testing Experiments and Results

Approximately 30 gallons of 46-F-7 Hi-Heat Dum Dum was remanufactured, as observed by Arthur D. Little, Inc. staff who provided chain-of-custody transmittal to our facility. Approximately one pint was used for the tests described herein.

The aging experiments were conducted by filling steel fixtures with Dum Dum and placing the fixtures into ovens at different temperatures. The fixtures, consisting of a steel ring spotwelded to a piece of sheet steel, provided for a one inch diameter and three different thicknesses of the material - 0.062 inches, 0.125 inches and 0.187 inches. Fifteen fixtures of each thickness were prepared for a total of 45 fixtures, individually numbered by stamping.

Each fixture was tared on an analytical balance (scale) that measured the weight to 0.0001 grams. The fixtures were then filled with the Dum Dum product, reweighed to determine the

weight of the Dum Dum in the fixture and placed into ovens in triplicate at temperatures of: 175, 250, 350, 425 and 500 °F.

The three replicate samples at each thickness represented a total of nine samples at each temperature. At times of 22, 46, 70, 93.5, 133.5, 400.5 and 763 hours, the samples were removed from the ovens, allowed to cool to room temperature and again weighed. The percent change from initial weights are provided in Table 1.

All of the samples underwent a change in the original natural gray color after aging. The colors ranged from a darker gray (175°F) to chocolate brown (250°F) to dark brown (350°F) to black (425°F and 500°F). During the initial heating of the 500°F samples, smoke was emitted from the oven leading us to believe that the solvent within the sample ignited and burned. The surface of the 500°F samples became swollen and expanded out of the fixture in a spherical shape. This occurred to a lesser extent for the 425°F samples.

The samples also exhibited surface hardening. To determine any product brittleness or friability, we tested the flexibility of five samples aged for 763 hours by a bending experiment. For this purpose, a test sample was cut from the fixture such that there would be no interference from the fixture on the bend experiment. The tests were done using a three point bend fixture on an Instron 1332 mechanical testing machine with a 1000 pound load cell. A schematic representation of the bend condition is shown in Figure 1. Crosshead displacement control was used with a displacement rate of 0.1 inches/minute. The applied load and the crosshead displacement was continuously recorded. Test events such as the onset of surface cracking were noted. The experimental results of the bend tests are given in Table 2.

Discussion

Hi-Heat Dum Dum contains about 10 percent by weight mineral spirits, to provide a suitable viscosity for application, and a linseed oil blend as the vehicle which may have some volatiles; the solids content is stated as 85 percent by volume. Loss in weight of Hi-Heat Dum Dum exposed to elevated temperatures occurs by evaporation of the solvent and oxidation (ablation) of the resinous vehicle.

To estimate the useful life of the product, defined as retention of flexibility as demonstrated by a strain of 1 percent, the observed loss in initial weight as a result of furnace aging experiments is plotted in Figure 2, where the averages for three thicknesses tested are pooled. After 22 hours of high temperature exposure, much of the solvent has evaporated, more so as the exposure temperature increases. For longer exposures at elevated temperature, the 175, 250 and 350°F exposures demonstrate a linear change in weight loss when plotted against the logarithm of exposure time. The samples aged at 350°F for 763 hours still exhibit a 1 percent strain in bending (Table 2), having lost 12.5 percent of initial weight, representing essentially all of the non-solids content of Hi-Heat Dum Dum. From the data, product life at a sustained temperature of 250°F is conservatively estimated at more than 10 years.

Exposures of Hi-Heat Dum Dum at temperatures greater than 350°F results in rapid degradation due to both very rapid evaporation of the solvent and oxidation of the resin. Further, exposure of this product to sustained temperatures of 350°F could result in product life as short as one month. Accordingly, the use of this product under conditions where it is exposed to consistent heat of up to 175°F and intermittent heat of up to 350°F is considered prudent.

Conclusions

The conclusions reached in this study are as follows:

- Hi-Heat Dum Dum survives constant heating to 250°F and intermittent heating to 350°F and behaves as described in the product literature. Heating the Dum Dum above 350°F causes rapid loss in weight and probable failure of the product.
- After 763 hours of aging at 250 and 350°F, the product can be strained by one to one and a half percent, demonstrating that it has retained elasticity.
- Based upon extrapolation of the data, Hi-Heat Dum Dum is expected to retain its elasticity for more than 10 years when used in accordance with the manufacturers recommendations.

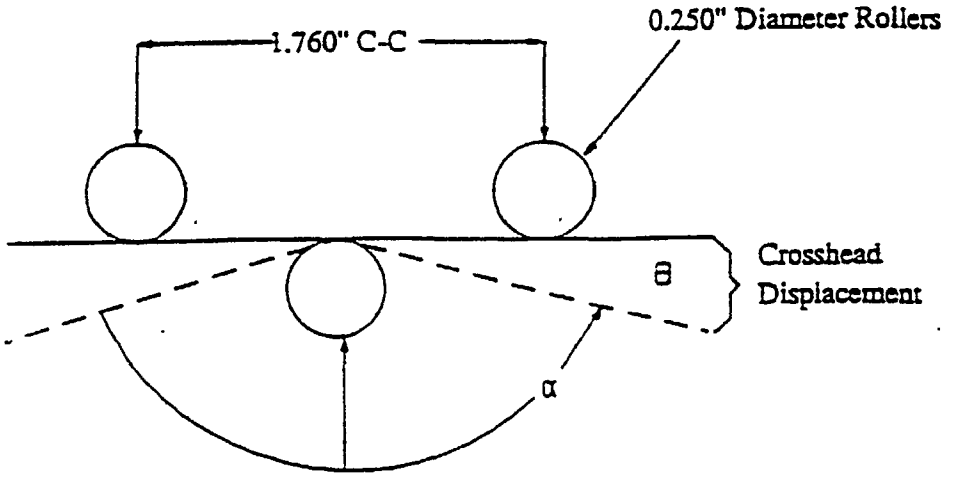
Table 1 Hi-Heat Dum Dum: Percent Weight Loss (Average of 3 Samples)							
Temp.	Time - Cumulative Hours at Temperature						
(°F)	22	46	70.5	93.5	133.5	400.5	763
Thickness = 0.063"							
175	8.90	8.95	8.87	8.87	8.94	9.52	8.99
250	9.47	9.50	9.55	9.57	9.65	9.93	9.75
350	9.91	10.67	10.91	11.00	11.39	12.10	12.18
425	10.96	11.73	12.07	12.34	12.74	14.65	16.11
500	14.60	18.39	21.36	23.98	28.47	40.41	45.53
Thickness = 0.125"							
175	7.86	8.92	9.27	9.43	8.94	9.52	9.79
250	8.88	9.51	9.71	9.73	9.65	9.93	9.95
350	10.24	10.86	11.02	11.13	11.39	12.10	12.10
425	11.04	11.55	12.17	12.80	12.74	14.65	18.98
500	15.08	17.35	22.28	24.98	28.47	40.41	45.24
Thickness = 0.183"							
175	6.25	7.79	8.52	8.90	9.31	10.13	10.00
250	7.79	9.21	9.71	9.82	10.05	10.27	10.27
350	9.68	10.50	10.75	10.90	11.15	11.90	13.31
425	11.21	11.70	12.25	12.68	13.94	16.42	18.28
500	14.15	16.87	19.41	21.75	24.22	37.88	44.30

Table 2
Hi-Heat Dum Dum: Flexibility after 763 Hours at Temperature

Sample	Thickness (inches)	Aging Temp. (°F)	Angle of Deflection		Percent Strain
			Crack Starts at One End	Fully Cracked	
37	0.125	250	10	16	1.7
27	0.187	250	4	11	1.2
33	0.062	350	4	10	1.1
8	0.125	350	5	10	1.1
28	0.187	350*	N/A	N/A	N/A

* Material separated from steel holder.

Figure 1
Schematic of Bend Test Conditions and Bend Angle Calculations



Crosshead Displacement

$\sin \theta = 0.5\ (1.760)$

$\alpha = 180° - 2\ \theta$

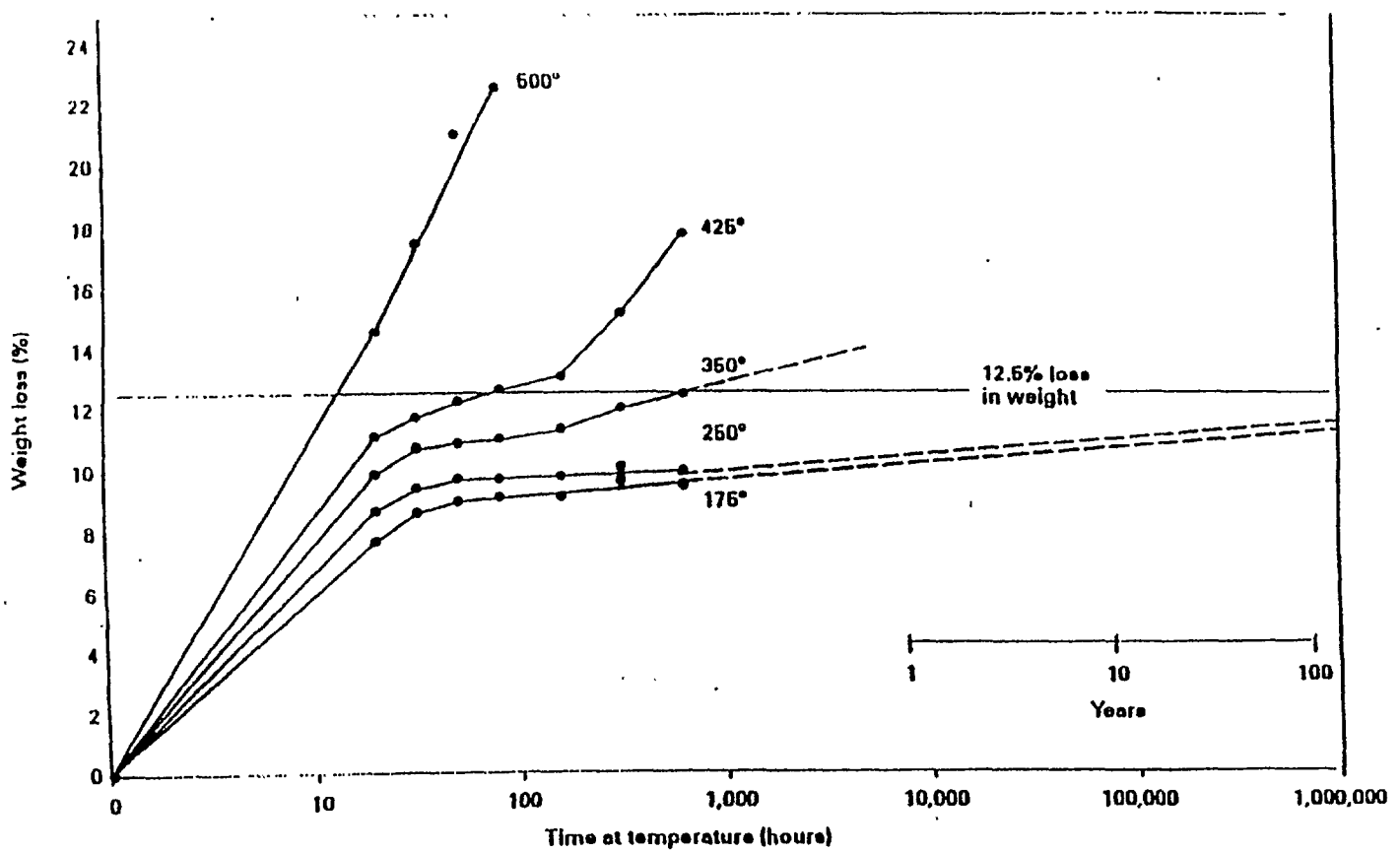


Figure 2. Ill-Heat Dum Dum Weight Loss with Time at Temperature

Evaluation of Asbestos Release from Hi-Heat Dum Dum Caulking During Application and Removal

By

Arthur D. Little, Inc.
Acorn Park
Cambridge, MA 02140-2390

1992

Reference No. 65191

Arthur D Little

Product Background

High temperature resinous caulking materials of mastic (putty-like) consistency are used in sealing joints and crevices on furnaces and boilers to prevent heat loss. Mobil Chemical Company manufactured and sold such a caulking product from the mid-1960s through the 1970s called Hi-Heat Dum Dum. This product, No. 46-F-7, consisted of a linseed oil-base vehicle (resin) containing various solid fillers, including clay and the chrysotile variety of asbestos (added for its resiliency and bridging properties). The Mobil Chemical Products Catalog provides the following description:

> "Hi-Heat Dum Dum/46-F-7, a heavy semi-plastic fibred coating, which acts as a joint sealer and pliable gasket for boilers, furnaces and dry kilns. Improves boiler efficiency by preventing heat loss. Retains its elasticity at consistent heat up to 175° and intermittent heat to 350°. May be used as a complete cover to eliminate excess fuel consumption and can be painted over in 24 hours."

The purpose of a joint or crevice sealant is to bridge gaps between the furnace enclosure and attachments (doors, ports, flanges, etc.). This requires a product that is pliable and resilient (i.e., withstands shock without permanently deforming, tearing, rupturing or pulling away from the furnace surface), and does not degrade over time by becoming friable[1]. The resinous formulation of Hi-Heat Dum Dum provides these properties and also effectively encapsulates the asbestos component, preventing its release.

Product Testing During Application and Removal

Hi-Heat Dum Dum was tested by Arthur D. Little, Inc. to determine if asbestos was released during application or removal. Approximately 30 gallons of 46-F-7 Hi-Heat Dum Dum was remanufactured, as observed by Arthur D. Little, Inc. staff who provided chain-of-custody transmittal to our facility. Experiments (described below) were conducted at our facilities in compliance with all current United States and Massachusetts rules and regulations relating to asbestos and in accordance with an approved Arthur D. Little, Inc. Health and Safety Plan.

An application and removal plan was developed to simulate typical work practices. In addition, a sampling and analysis protocol was developed to quantify the extent to which asbestos structures are released during the experimental application and

[1]The U.S. Environmental Protection Agency defines friable as "capable of being crumbled, pulverized, or reduced to powder by hand pressure".

removal activities[2]. This protocol was designed so that all applicable NIOSH and EPA requirements for analysis by transmission electron microscopy were met or exceeded. The minimum detection limit for the analytical protocol was 0.01 fibers/cc[3].

To evaluate the release of asbestos during product application and removal, steel panels (1/32 inch thick 1040 steel, cleaned and degreased, measuring about 2 1/2 x 3 1/2 feet) were coated by troweling at an estimated thickness of 1/8" (and feathered at the panel edges). Approximately 15 panels were coated during each of two 80-minute application experiments. Personal exposure samples were collected during both experiments.

After product application, the Hi-Heat Dum Dum was heated to accelerate the aging process[4]. The neutral gray of the as-applied coating changed in color to a chocolate brown (225-250°F for 20 days).

After aging, product removal experiments were conducted using a putty knife and scraper. Although the thicker, centrally coated area of each panel was very pliable and quite easily removed, the feathered edge regions were somewhat harder and required substantial mechanical effort to scrape down to the bare metal. Approximately 10 panels were cleaned in each of three, separate 80-minute experiments during which personal filter samples were collected.

The personal filter samples were prepared and analyzed by transmission electron microscopy. The results of these analyses are given in Table 1.

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[2]Asbestos structures are defined herein as particles 5μm or more in extent that satisfy the counting guidelines given in 40 CFR Part 763 Asbestos-Containing Materials in Schools, Appendix A, Figure 5 (Federal Register 52 (210) October 30, 1987, 41866-41867). Structures include fibers, bundles, clumps and matrix. OSHA regulates exposure to fibers, which are defined as "a particulate form of asbestos, tremolite, anthophyllite or actinolite, 5 micrometers or longer, with a length to diameter ratio of at least 3 to 1" (29 CFR 1910.1001).

[3]The minimum detection limit (MDL) for this experiment is the concentration where occupational exposure can not be differentiated from general background.

[4]During preliminary laboratory furnace experiments with coated steel coupons, we observed that the product discolored from a neutral grey to a dark chocolate brown after one day at 350°F and to a medium brown after four days at 265°F. Even after these exposures to temperatures which exceeded the recommended continuous use temperature, the product remained pliable and gummy when probed with a screwdriver. Therefore, although the product was designed for a continuous temperature of 175°F, a treatment of two weeks to dry, flowing air at 250°F was chosen to not only accelerate aging but also to account for intermittent temperatures greater than 175°F. The actual conditions of exposure were 225-250°F for 18 to 23 days.

Table 1

Asbestos Exposures During Application and Removal of Hi-Heat Dum Dum: Personal Samples Analyzed by Transmission Electron Microscopy

Sample No.	Description	Exposure (s/cc)[5]
042504	First Application	<0.01
042604	Second Application	<0.01
051703	First Removal	<0.01
051705	First Removal (replicate)	<0.01
052203	Second Removal	<0.01
052207	Third Removal	0.01

Conclusions

The conclusions reached in this study are as follows:

- The Hi-Heat Dum Dum that was tested was found to be non-friable and provided encapsulation of asbestos during application and removal experiments.
- Exposure of the product to 225-250°F for 18 to 23 days changes its color from a neutral gray to a chocolate brown, indicating modification of the resin. Even so, the product was elastic and pliable except at the feathered, thin edges where it was hardened but not friable.
- Results of worker exposure measurements during use of Hi-Heat Dum Dum did not exceed the minimum detection limit of 0.01 asbestos structures/cc in either product application or product removal experiments.
- Based upon the above results, occupational exposure to airborne asbestos fibers during the application or removal of Hi-Heat Dum Dum is virtually non-existent.

[5]Exposure represents asbestos structures per unit volume of air.

Evaluation of Asbestos Release from Chimney Weatherproofing Mastic During Application and Removal

By

Arthur D. Little, Inc.
Acorn Park
Cambridge, MA 02140-2390

1992

Reference No. 65191

Product Background

From the mid-1960's through the 1970's, Mobil Chemical Company manufactured and sold a line of mastic-type protective coatings under the name Dum Dum® Products. Chimney Dum Dum was a protective and waterproofing coating specially formulated for use on concrete and brick chimneys where heat resistance up to 210°F is required. The Mobil Chemical Product Catalog states:

> "Chimney Dum Dum/97 Series Chimney Dum Dum is a rugged heavy coating for the exterior of chimneys that provides flexible weatherproof protection. It fills and bridges hairline cracks with a flexible coating under a tough outer skin. Should the leathery skin become fractured, the soft under film will harden on contact with air. May be applied without completely cooling stack."

Chimney Dum Dum consists of a resinated vegetable oil vehicle, containing 41% pigments, including titanium dioxide, mica and the chrysotile form of asbestos; the total solids content is 63%. Application is by brush, or more typically, by spray. At 75°F, the surface skins over in 4-6 hours, and the under surface remains pliable. Resistance to chemical fumes and moisture is very good to excellent and the product is resistant to constant dry heat at 210°F.

Product Testing During Application and Removal

To determine if asbestos is released during the application or removal of Chimney Dum Dum, approximately 18 gallons of the product was prepared on the basis of the formula master and mixing instructions provided by Mobil. The oil base resin was no longer commercially available, and a special batch was prepared for us by McWhorter Corp. All other components were obtained commercially. The product was made with a Myers mixer in 6 gallon batches which were subsequently combined, blended and stored in 5 gallon pails. The prepared product was observed to be smooth and uniform in appearance and of mastic consistency. This work, as well as subsequent application and removal experiments, was conducted at our facilities in compliance with all current United States and Massachusetts rules and regulations relating to asbestos and in accordance with an approved Arthur D. Little, Inc. Health and Safety Plan.

A test plan was developed to simulate typical work practices for product application and removal[1]. In addition, a sampling and analysis protocol was developed to

[1]Although intended for exterior use, this product was tested in an interior (controlled) environment.

quantify the extent to which asbestos structures[2] may be released during the experimental application and removal activities. This protocol was designed so that all applicable NIOSH and EPA requirements for analysis by transmission electron microscopy were met or exceeded. The minimum detection limit for the analytical protocol was 0.01 structures/cc[3].

To evaluate the release of asbestos during product application and removal, ten concrete panels and twelve facing brick panels with concaved mortar joints, both sets four feet by two and one-half feet by one and one-half inches, prepared about one year earlier, were primed (Chimney Dum Dum Primer 38-V-5 equivalent), dried overnight and spray coated with Chimney Dum Dum at an estimated coating thickness of 30-40 mils using a Binks 2 gallon pressure pot with follower and a 7E2 spray gun. The coating operation required 55 minutes during which personal and area air samples were taken. The area sample was about ten feet from the spray site at an elevation of 5 feet.

After an ambient cure of 24 hours during which the coating skinned over, the panels were aged for 14 days at 200-210°F and eight days at ambient. The purpose of the furnace treatment was to accelerate the natural aging process. After the elapsed 23 days of aging, the product was hardened at the surface yet pliable and elastic when depressed with a screwdriver blade.

Product removal was carried out on eight brick panels and six concrete panels over 88 minutes during which personal and area filter samples were collected.

The collected filter samples from these experiments were prepared and analyzed by transmission electron microscopy. The results of these analyses are given in Table 1. These results may be overstated compared to actual exposures which would occur in an open air environment. As such, they are considered to represent a "worst case" condition.

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[2]Asbestos structures are defined herein as particles 5μm or more in extent that satisfy the counting guidelines given in 40 CFR Part 763 Asbestos-Containing Materials in Schools, Appendix A, Figure 5 (Federal Register 52 (210) October 30, 1987, 41866-41867). Structures include fibers, bundles, clumps and matrix. OSHA regulates exposure to fibers, which are defined as "a particulate form of asbestos, tremolite, anthophyllite or actinolite, 5 micrometers or longer, with a length-to-diameter ratio of at least 3 to 1" (29 CFR 1910.1001).

[3]The minimum detection limit (MDL) for this experiment is the concentration where occupational exposure cannot be differentiated from general background.

Table 1 Asbestos Exposures During Application and Removal of Chimney Dum Dum®			
Sample No.	Description	Sample Type	Exposure (s/cc)[4]
052802	Application	Personal	0.088
052803	Application	Personal (replicate)	0.024
052805	Application	Area	0.024
062001	Removal	Personal	<0.01
062002	Removal	Personal (replicate)	<0.01
062003	Removal	Area	<0.01

Conclusions

The conclusions reached in this study are as follows:

- Remanufactured Chimney Dum Dum was smooth and uniform with mastic-like consistency.
- Application to brick and concrete panels required heavy duty mastic spray apparatus.
- After 23 days of aging (14 days at 200-210°F), the product was skinned over but pliable and elastic when depressed with a tool.
- Results of worker exposure measurements during application of Chimney Dum Dum in a controlled environment were below the current OSHA action level[5].
- Results of worker exposure measurements during removal of Chimney Dum Dum in a controlled environment did not exceed the minimum detection limit of 0.01 structures/cc.
- Based upon the above results, there is insignificant occupational exposure to airborne fibers during the application of Chimney Dum Dum and virtually no exposure to airborne asbestos fibers during the removal of this product.

[4]Asbestos structures per unit volume of air. The structures observed were fibers (or fibers and bundles, corresponding to the EPA definition).

[5]OSHA defines action level as "an airborne concentration of asbestos, tremolite, anthophyllite, actinolite, or a combination of these minerals, of 0.1 fiber per cubic centimeter (f/cc) of air calculated as an eight (8) hour time weighted average" (29 CFR 1910.1001, promulgated June 20, 1986).

Memorandum from T. E. ALLEN, M. D.
NEW YORK, N. Y.

Date 13 August 1973

To

Dr. H.H. Golz
API

Dr. Allen has asked me to send the attached to you for your information.

Joan DiGangi
Dr. Allen's Secretary

Memo to File

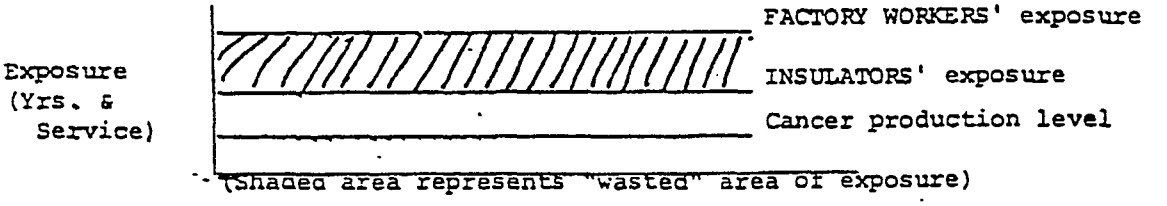
As noted in a memorandum dated 11 July 1973, a meeting was arranged, following an invitation from Dr. Irving Selikoff, to meet with him at the Mt. Sinai Medical Center. Attending the meeting were:

Dr. Harry Hermann - Associate of Dr. Selikoff - NY
Dr. W. Nicholson - Associate of Dr. Selikoff - NY
Dr. N. Roberts - Assoc. Medical Director, Exxon Corp.-NY
Dr. M. Goldman - Ass't. Medical Director, Exxon Corp.-NY
Dr. H.A. Sinclaire- Assoc. Medical Director, Mobil-NY
Dr. T.E. Allen - Medical Director, Mobil-NY

The purpose of the meeting was to present findings that Dr. Selikoff had made following examination of a group of represented Mobil employees (Paulsboro Refinery) on the request of the union at Paulsboro. In addition, Dr. Selikoff hoped that, on discussion of these findings, the group might be able to advise him whether such findings might be considered typical and usual of an average refinery population.

The meeting lasted approximately two and one half hours and consisted principally of a presentation of asbestosis as an occupational illness, with major points of same being illustrated by x-rays, statistics, and data gathered on the Mobil population examined. No list of examinees was given, and, at no time, was any data or x-ray identified by a specific name.

Initially, Dr. Selikoff gave a brief discussion of the disease, asbestosis. In this coverage, he covered the highlights of work being done here and in U.K. He mentioned historical work done in shipyards in USA and U.K. He made it clear that, during the discussions, he would be talking in terms of "insulators, brick workers, etc. in user companies," rather than in terms of "factory workers" (where asbestos products, piping, etc. are made such as Johns Manville, etc.). In spite of this, he pointed out that if exposure to asbestos fibers in insulators was enough to produce malignancy, the great exposure normally experienced in factory employees was "excess exposure" as far as cancer was concerned. Graphically shown, this would be as follows:



Dr. Selikoff pointed out that the typical x-ray findings in a case of asbestosis were:

1) General thickened pleura (the pleura is the thin, transparent covering of the lungs)
2) Pleural plaques (a concentration of thickening of the pleura in certain specific areas)
3) Reticular fibrosis (intensified marking throughout lung tissue, particularly in lower lung fields producing "ground glass" appearance.

4) A shaggy appearance to the heart outline.
5) Blunting or even complete obliteration of the area where the diaphragm touches the rib area.

It was emphasized that the above findings were typical of but not pathognomonic (specifically distinctive or diagnostic) of asbestosis. These findings, along with an extensive work or exposure history, can be considered diagnostic. One does not necessarily need to have positive pulmonary function tests to clinch the diagnosis. The development of asbestosis need not produce death in any person sufficiently exposed, but, there is developed in cases of asbestosis susceptibility to lung cancer, and even to mesothelioma, a specific type of malignancy.

In regard to Mobil's employee group at Paulsboro, Dr. Selikoff stated he was requested by the "union" there to examine a group of represented employees. This group of employees was presented by the union to Dr. Selikoff who did not have a voice in the choice and he had no previous knowledge of the examinees' medical, work or social history. He stated emphatically that, as a result, the study was a biased one, but he could not state whether it was biased in favor of a sick, average or well group. The only point on which there was certainty was that all examinees had had at least 20 years of work in the refinery. On several occasions, the size of the examined group was stated to be 137, this is not quite accurate because only 118 were examined. The remaining 19 were those employees who, at an earlier date, had been seen only for a chest x-ray and chest examination and, on whom, the union requested the company x-rays be forwarded to Dr. Selikoff at his Pate rson, N.J. office.

Dr. Selikoff handed out six sheets showing in graph form some of the findings resulting from his examination of the 118 employees. (It should be noted that the examinations were done by a team of 9 people from Mt. Sinai Medical Center who, with the aid of mobile x-ray unit, completed these examinations during one week-end.) After the charts were handed out, discussion was so continuous that it was not possible to examine these charts thoroughly. Constant references were made to the charts, however, during the discussion.

On the days following the conference, when the undersigned was able to thoroughly review these charts, it was evident that there were many errors in calculation, as well as some ambiguity in reporting the population studied and the results found in same. A call was made to Dr. Selikoff's office and some corrections were offered via the phone, and others were made by sending revised charts to replace those in question.

Attached are copies of the revised tables issued to us at the meeting. Table VI was not given at the meeting. It was included in a later mailing to the undersigned. General comments on the tables, which are self-explanatory, include the following.

It will be immediately obvious from Table I that the medical review was not restricted to "active" employees as was first thought. We are told by Dr. Selikoff's office that the breakdown of the 137 examinees is as

follows:

annuitants	13
"active" employees	105 (full medical review)
"active" employees	19 (partial medical review)

The 19 employees listed above were employees (no annuitants) who were seen at an earlier date by Dr. Selikoff and on whom only a chest examination and a chest x-ray were done. This was the group of employees on whom the union requested their company x-rays to be sent to Dr. Selikoff.

We are not aware in which group, i.e. service, the 13 annuitants fall. We can assume they may be in the 30-39 and 40-49 because it is in this group that ages in excess of normal retirement age are listed. It is possible that some might be in the first service category, 20-29 yrs., if they retired at an age younger than 65 years.

The remaining tables are self-explainatory but indicate, among other things, that

1) none of the cases in this series showing classic signs of asbestosis are "serious" cases. There is no doubt that, in x-ray films shown to us (some taken in the mobile unit - some previously in the group of 19) there are some x-ray signs indicative of asbestosis. Unfortunately, we were not given the names of the patients represented in the x-rays shown as samples. Dr. Selikoff feels, from his long experience, that x-rays alone (if not correlated with pulmonary function tests, etc.) tend to "unread" the time picture of asbestosis.

2) Clinical findings such as bronchitis and positive x-ray findings and pulmonary function tests are higher in "smokers" than "non'smokers" and higher in "ex smokers" than in the group which has no smoking history.

3) A high percentage of this group are "no hisotry of smoking" (28%) and "ex smokers" (39%) making a total group of "non smokers" - 67%.

4) The study is unique in one aspect. With the expected exposure of this group of employees to firebrick, there is no indication of silicosis. Firebrick is about 85% quartz in composition.

We are not prepared, at this time, to dispute or confirm Table VI. It is interesting to note that cancer of the lung in this group studied is recorded as 0.8%. The national average of cancer of the lung (males) is 3+%.

Probably the most revealing and appropriate chart is Table VII. This shows that of all the categories of workers represented in the study, that of the insulators, masons and bricklayers (13), 81% show positive signs of both pleural and parenchymal involvement of asbestos exposure. This is markedly

higher than the findings in all other occupations represented. Again, it was stated by Dr. Selikoff that although asbestosis is unquestionably established as a diagnosis, these do not represent presently "serious" cases.

Attached with the statistical tables is a copy of the recording form used in the analysis of x-rays of patients whose occupation might make them liable to pneumoconioses (chronic fibrous reaction in the lungs to the inhalation of any type of dust) such as asbestos, iron dust, cloth fibers, and silica. It should be noticed that this form and its legend is the official classification established by the I.L.O., and generally accepted as a classification by most pulmonary and occupational health specialists.

Dr. Selikoff feels that, in an ideal program of preventive health, workers with 20 years or more exposure in jobs involving insulating materials, fire-brick and masonary cement, chest x-rays should be done every three months. He also thinks many other preventive procedures to protect their respiratory systems should be instituted. Thus, he advocates the administration of flu vaccine each year for such a group. Above all, he urges all workers to stop smoking.

Dr. Selikoff was most professional in his conduct during this session. At no time did he mention the company's name and only, as identification of location of the worker studied, was the name Paulsboro used. Furthermore, he feels that the union is unaware of "99% of the great work done by the company medical department." "They only see and talk about the bad things when they happen." He cited a common union complaint that "our men naturally are sick breathing in that contaminated air at the refinery every working day." He quickly referred again to Table V showing that chronic bronchitis, if the union complaint had merit, would be appearing in all represented employees, when actually his statistics show it occurs primarily and predominately in the "smokers."

He urged consideration of a joint management - union study of the asbestos workers (those who have been exposed during their work life) in at least five refineries, among which should be included the "best" (as far as modern, but not recent construction) and the "worse." Thus assumes more than one company would be so involved. The whole research project should be jointly sponsored - developed and each side totally aware of the progress from planning through completion stages.

Such a study can be done by any one of many agencies. There is no doubt that it could be done by the federal government - through the agency of NIOSH. If done by them, he feels it would be a fair, well done and complete study. However, NIOSH could and would probably look at such a project only once and not on a continuing basis, and this type of study needs at least a 20-30 year follow-up since this can be the "incubation" period of asbestosis.

The study could be done completely by any outside agency, such as an academic group or a private research group. Needless to say, choice of the group would have to be mutually agreeable.

The ideal project, in Dr. Selikoff's opinion, is one planned with, guided and supervised by capable outside experts, but with all the examinations, x-rays, lab. work done by the company inplant medical department. He feels this is the only source of ongoing interested medical supervision available to the men. He does feel that, in Paulsboro's case, it may well take a gigantic effort to get the union and management to mutually trust each other in such an undertaking. He does not feel that it is impossible, and he feels every effort should be made to accomplish this goal. Again, he pointed out that, in the general male population of the USA, there is a 3-4% death rate due to lung cancer. Admittedly, there are multiple etiological factors in development of lung cancer. However, if and when lung cancer occurs in a company worker exposed to such materials as asbestos, the first and most obvious conclusion is that this is due to a carcinogenic material in the work environment. This is difficult to combat, and admittedly, can be unfair to managements. Research and careful clinical follow-up can establish data that could remove the haziness as to responsibility in all such cases.

In summation, Dr. Selikoff presented findings to the group in a most professional and non spectacular fashion. He admitted repeatedly the group studied is an ideal one in many respects. A more ideal study, jointly sponsored by management and union, should be undertaken. Mobil is not the only petroleum company with this problem (cases in Texaco were cited). Dr. Selikoff did not pretend to be an expert on refinery health since he has never had an opportunity to visit a refinery.

No commitment was made to him as to follow up to his presentation or suggestions.

After the close of the meeting, Dr. Selikoff was asked if he would explain his relationship to either OCAW or independent unions in the petroleum industry. He answered that he has no contract with OCAW or any union. OCAW and independent unions have and do request the Mt. Sinai Medical School - Department of Environmental Medicine to do projects for them as indicated in this study of some Paulsboro employees. He emphasized his commitment is not unilateral, and both the school and he, as an individual, are free at any time to consider research or consultant capacities with any agency, management, union or government.

Table I

Age distribution of 137 refinery workers, by years since first exposure

Years since first exposure	Length of exposure (years)	Number	Age range	Average age
20-29	20-29	65	41-65	50.0
30-39	30-39	55	48-70	53.4
	15	1	81	81
40-49	40-47	12	59-66	63.1
	36-39	4	64-70	68.2

Table II

Work Activities of 137 Production and Maintenance Employees

Type of Work	Active	Retirees
"Clean Work" (Docks, Case-Can, Auto Shop)	10	1
"Dusty" Production Work (Bead Plant, Filter Dept.)	5	
Fume Exposure (All other production units)	28	7
Mixed Fumes and Dust	6	
"Clean" Maintenance (Carpenters, Machinists, Electricians, etc.)	21	2
High Asbestos Exposure (Insulators, Masons, and Bricklayers)	13	3
Other Maintenance (Pipefitters, Welders, and Boilermakers)	41	
Totals	124	13

X-ray changes

Length of exposure (years)	Number of Persons	Parenchymal 1	Parenchymal 2	Pleural (only)	Total pleural changes	Total changes Parenchymal and/or pleural	No changes
20-29	65	18 28%	2 3%	10 15%	15 23%	30 46%	35 54%
30-39	55*	20 36%	2 4%	3 5%	3 5%	25 45%	31 56%
>40	16	9 57%	1 6%	0	3 19%	10 62%	6 38%
Total	136*	47 34%	5 4%	13 10%	21 15%	65 48%	72 52%

* A X-ray of one individual was unsatisfactory and could not be read

Table III

Pulmonary function abnormalities
in relation to X-ray changes
and smoking

	Restriction				Obstruction			
	Smokers	Ex-smokers	Non-smokers	Total	Smokers	Ex-smokers	Non-smokers	Total
Parenchymal and/or pleural changes Number of persons 65	7 11%	6 9%	3 5%	16 25%	11 17%	9 14%	0	20 31%
No changes Number of persons 71	2 3%	2 3%	2 3%	6 9%	8 11%	5 7%	3 4%	16 22%

Table IV

Smoking Habits

Length of exposure (years)	Number of Workers	Smokers	Ex-smokers	Non-smokers
20-29	65	22 34%	23 35%	20 31%
30-39	56	19 34%	21 37%	16 29%
40+	16	4 25%	10 52%	2 13%
Total	137	45 33%	54 39%	36 28%

Table V

Prevalence of Chronic Bronchitis

Length of exposure (years)	Number of Persons	Smokers	Ex-Smokers	Non-Smokers	Total
20-29	65	9	4	3	16 25%
30-39	56	11	3	1	15 27%
40+	16	1	1	0	2 12.5%
Total	137	21	8	4	33 24%

Table VI

History of previous cancer in 118 refinery workers

Cancer	Number of cases	
Skin	2	1.6%
Bladder	2	1.6%
Colon	1	0.8%
Abdominal	1	0.8%
Lung	1	0.8%
Total	7	6 %

T-a-VII

X-ray Results of Refinery Productio. and Maintenance Employees

	Negative		Positive						
Type of Work	0/0	0/1	1/0	1/1	1/2	2/1	2/2	Pleural	% Positive
"Clean Work" (Docks, Case-Can, Auto Shop	5	4	1	1					18
Dust (Dead Plant, Filter Dept.)	1	3						1	20
Fumes (All other production units)	9	9	8	5		1	1	1	47
Mixed Fumes and Dust	1	3				1		1	33
"Clean"maintenance (Carpenters, Machinist, Electricians, etc.)	6	5	3	7			1	1	52
Other maintenance (Pipefitters, Welders, and Boilermakers)	11	11	4	6	5			4	46
Insulators, Masons, and Bricklayers	2	1		8	2		1	2	81
								overall	48

JVIRONMENTAL SCIENCES LABORATORY

INT SINAI SCHOOL OF MEDICINE OF THE CITY UNIVERSITY OF NEW YORK

Number _____

Research Group _____

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ment: _____

U/C International Classification of Radiographs of Pneumoconioses BIT U/C Classification Internationale des Radiographies de moconios

Quality	ROUNDED SMALL OPACITIES Type	Profusion	Zones	IRREGULAR SMALL OPACITIES Type	Profusion	Zones	SMALL OPACITIES Combined Profusion	LARGE OPACITIES Type	Size	PLEURAL THICKENING Costo-phrenic angle	Site	Width	Extent	ILL DEFINED DIAPHRAGM	ILL DEFINED CARDIAC OUTLINE	PLEURAL CALCIFICATION Diaph	Wall	Other	Gen.	Symbols
+	p	0/- 0/0 0/1	s	0/- 0/0 0/1		0/- 0/0 0/1		0	0				0	0	0				0	ax bu ca co co
±	q	1/0 1/1 1/2	t	1/0 1/1 1/2		1/0 1/1 1/2	wd	A	R	R	a	I	R	I		R			I	ep ev di ef em
+	r	2/1 2/2 2/3	u	2/1 2/2 2/3		2/1 2/2 2/3	id	B	L	L	b	2	L	2		L			2	es hi ka k od

Number of zones involved

MOB-HarrisMaster
00536

Memorandum from T. E. ALLEN, M. D.
NEW YORK, N. Y.

Date 9 July 1973

To

Dr. H.H. Golz
Mr. J.M. McNerney

As promised during our telephone conversation.

T.E. Allen, M.D.

28 June 1973

Memo for File

This morning, Dr. Irving Selikoff of Mt. Sinai Medical Center, called me. For the record, the undersigned has never talked or met with Dr. Selikoff at any time previously and the telephone call was unsolicited.

Dr. Selikoff stated that, at an earlier date, at the union's request, he had examined approximately 18 employees from the Paulsboro Refinery. The employees for this examination had been selected by the union and general physical examinations with laboratory and x-ray procedures were carried out.

About six months ago, again at the union's request, Dr. Selikoff examined approximately 118 employees. These examinations were done during a week-end. Although it was not stated specifically, it was my understanding that the examinations were done by Dr. Selikoff and staff and not necessarily that each examination was done by Dr. Selikoff himself. Furthermore, Dr. Selikoff stated that the 118 employees were "volunteers" and thus represented "a potentially biased population." If he were doing a more scientific study, Dr. Selikoff would have picked his population of examinees at random. All individuals examined in the group of 118 were people with 20 or more years service with Mobil. The population generally represented those employees working with insulation, bricklayers, and general utility workers employed in the area where insulating materials might have been present but the population was not necessarily restricted to those who have direct and/or constant exposure to insulating materials. In short, the population represented not young people and not people with "very little" exposure to insulating materials.

The results of these examinations to date have shown that in slightly less than 50% of the total x-ray findings indicating a marked reticular fibrosis (a classic finding in asbestosis cases). No case was found to be of a "very severe type." Three were considered to be Grade II-2 and two cases were Grade II-1. These gradations are made in accordance with the ILO Classifications of pulmonary fibrosis as seen in cases of asbestosis. Dr. Selikoff's team also made other findings which have not yet been completely tallied. Such findings would have included notations of pulmonary function, finger-clubbing, cigarette smoking, etc., and their relationship to signs of asbestosis.

Dr. Selikoff readily admitted that on several occasions during the conversation, that his population was not a random sample, but more or less a selected sample. He is, however, concerned at the percentage of cases, i.e. five cases out of approximately 50.

At this point, he stated that with these findings in mind, he is wondering whether Paulsboro is unique with these findings because it is an "old" refinery and/or because it has an "old" population. If the answer to these questions is "no," are the findings here in Paulsboro typical of any refinery in the petroleum industry, particularly with any age group and service group similar to that of Paulsboro. Realizing that there is much to be duscussed on this general subject, he thought it best to call me and asked if I would meet with him for discussions on the subject. He was planning also to call Dr. Norbert Roberts, Associate

Medical Director, Exxon Corporation, since he knows Dr. Roberts and does not know any other petroleum company medical directors. He realizes that Dr. Roberts, having never worked in a refinery might not be intimately acquainted with these subjects. He thought if this is so that Dr. Roberts might refer him to a Medical Director of Exxon's domestic operation or to one of Exxon's refinery physicians experienced in this area. He felt that if such discussions were held all of us would tend to gain. He added if this information he has already gathered were given to the union without the benefit of discussion with petroleum medico representatives, the union might press the matter in a manner that might not be ideal for anybody.

Theodore E. Allen

Theodore E. Allen, M.D.

TEA/jd

Evaluation of Asbestos Release from Boiler Caulking During Application and Removal

Arthur D. Little, Inc.
Acorn Park
Cambridge, MA 02140

June 1990

C-65191

Product Background

High temperature resinous caulking materials of mastic (putty-like) consistency are used in sealing joints and crevices on furnaces and boilers to prevent heat loss. Mobil Chemical Company manufactured and sold such a caulking product from the mid-1960s through the 1970s called Hi-Heat Dum Dum. This product, No. 46-F-7, consisted of a linseed oil-base vehicle (resin) containing various solid fillers, including clay and the chrysotile variety of asbestos (added for its resiliency and bridging properties). The Mobil Chemical Products Catalog provides the following description:

> "Hi-Heat Dum Dum/46-F-7, a heavy, semi-plastic fibred coating, which acts as a joint sealer and pliable gasket for boilers, furnaces and dry kilns. Improves boiler efficiency by preventing heat loss. Retains its elasticity at consistent heat up to 175° and intermittent heat to 350°. May be used as a complete cover to eliminate excess fuel consumption and can be painted over in 24 hours."

The purpose of a joint or crevice sealant is to bridge gaps between the furnace enclosure and attachments (doors, ports, flanges, etc.). This requires a product that is pliable and resilient (i.e., withstands shock without permanently deforming, tearing, rupturing or pulling away from the furnace surface), and does not degrade over time by becoming friable[1]. The resinous formulation of Hi-Heat Dum Dum provides these properties and also effectively encapsulates the asbestos component, preventing its release.

Product Testing During Application and Removal

Hi-Heat Dum Dum was tested by Arthur D. Little, Inc. to determine if asbestos was released during application or removal. Approximately 30 gallons of 46-F-7 Hi-Heat Dum Dum was remanufactured, as observed by Arthur D. Little, Inc. staff who provided chain-of-custody transmittal to our facility. Experiments (described below) were conducted at our facilities in compliance with all current United States and Massachusetts rules and regulations relating to asbestos and in accordance with an approved Arthur D. Little, Inc. Health and Safety Plan.

A test protocol was developed to simulate typical work practices for product application and removal. Since these experiments involved an asbestos containing product, precautions were taken so that experimenters and the ambient environment were fully protected from any potential release of asbestos fibers. A sampling and analysis protocol was developed to quantify the extent to which asbestos fibers may

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[1]The U.S. Environmental Protection Agency defines friable as "capable of being crumbled, pulverized, or reduced to powder by hand pressure".

be released during the experimental application and removal activities[2]. This protocol was designed so that all applicable OSHA, NIOSH and EPA requirements were met or exceeded. The minimum detection limit for the analytical protocol was 0.01 fibers/cc[3].

To evaluate the release of asbestos during product application and removal, steel panels (1/32 inch thick 1040 steel, cleaned and degreased, measuring about 2 1/2 x 3 1/2 feet) were coated by troweling at an estimated thickness of 1/8" (and feathered at the panel edges). Approximately 15 panels were coated during each of two 80-minute application experiments. Personal exposure samples were collected during both experiments.

After product application, the Hi-Heat Dum Dum was heated to accelerate the natural aging process. During preliminary laboratory furnace experiments with coated steel coupons, we observed that the product discolored from a neutral grey to a dark chocolate brown after one day at 350°F and to a medium brown after four days at 265°F, indicating chemical modification of the resin by oxidation. Even after these exposures to temperatures which exceeded the recommended continuous use temperature, the product remained pliable and gummy when probed with a screwdriver. Although the product was designed for a continuous temperature of 175°F, a treatment of two weeks to dry, flowing air at 250°F was chosen to not only accelerate aging but also to account for interminent temperatures greater than 175°F.

After accelerated aging (actual conditions were 225-250°F for 18 to 23 days), product removal experiments were conducted using a putty knife and scraper. Although the thicker, centrally coated area of each panel was very pliable and quite easily removed, the feathered edge regions were somewhat harder and required substantial mechanical effort to scrape down to the bare metal. Approximately 10 panels were cleaned in each of three, separate 80-minute experiments during which personal filter samples were collected.

The personal filter samples were prepared and analyzed by transmission electron microscopy. The results of these analyses are given in Table 1.

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[2]Fiber is defined as a particle 5μm or more in length with substantially parallel sides and an aspect ratio (length to width) of three or more.

[3]The minimum detection limit (MDL) is the concentration where occupational exposure can not be differentiated from general background.

Table 1

Asbestos Exposures During Application and Removal of Hi-Heat Dum Dum: Personal Samples Analyzed by Transmission Electron Microscopy

Sample No.	Description	Exposure (s/cc)[4]
042504	First Application	<0.01
042604	Second Application	<0.01
051703	First Removal	<0.01
051705	First Removal (replicate)	<0.01
052203	Second Removal	<0.01
052207	Third Removal	0.01

Conclusions

The conclusions reached in this study are as follows:

- Hi-Heat Dum Dum is non-friable and provides encapsulation of asbestos during application and removal experiments.
- Exposure of the product to 225-250°F for 18 days changes its color from a natural gray to a chocolate brown, indicating oxidation of the resin (overheating). Even so, the product was elastic and pliable except at the feathered, thin edges where it was hardened but not friable.
- Results of worker exposure measurements during use of Hi-Heat Dum Dum did not exceed the minimum detection limit of 0.01 asbestos structures/cc in either product application or product removal experiments.
- Based upon the above results, there is no potential for occupational exposure to free asbestos fibers during the application, removal or other use of Hi-Heat Dum Dum.

[4]Exposure represents asbestos structures per unit volume of air. Structures include fibers, bundles (an arrangement of parallel fibers that touch), clusters (an arrangement of random fibers that touch) and matrix (a fiber or fibers embedded in a particulate).

Appendix A
Photographs

Exhibit No.	Description
1	Hi-Heat Dum Dum as Applied and Aged at Ambient Temperature for 20 Days.
2	Hi-Heat Dum Dum as Applied and Aged at 225-250°F for 20 Days.

Exhibit No. 1

MOB-HarrisMaster
00545

Exhibit No. 2

MOB-HarrisMaster
00546

STATE OF MASSACHUSETTS

COUNTY OF MIDDLESEX, SS.

AFFIDAVIT

I, Edward T. Peters, being duly sworn according to law, hereby depose and says as follows:

1. I am a senior staff scientist in the Materials and Applied Physics Unit of the Product Technology Section of Arthur D. Little, Incorporated, Cambridge, Massachusetts. I have been a staff scientist at Arthur D. Little, Inc. since 1969.

2. Arthur D. Little, Incorporated, is a research firm experienced in the physical, structural and chemical characterization of materials.

3. I received undergraduate degrees from DePauw and Purdue Universities, a Master of Science degree from the University of Wisconsin and the degree of Doctor of Science in Metallurgy from the Massachusetts Institute of Technology.

4. I served as a member of the faculty in the Department of Metallurgical Engineering at the University of Wisconsin from 1958 to 1959.

5. From 1963 until 1969 I was employed by ManLabs Incorporated, in Cambridge, Massachusetts. At ManLabs, I specialized in the structural and physical characterization of materials.

6. I am a Registered Professional Engineer in the State of Massachusetts, a member of the American Society for Metals and the American Industrial Hygiene Association.

7. While at Arthur D. Little, Inc., I have been responsible for several laboratory based programs designed to evaluate and characterize materials as well as corporate responsibility for technical studies relating to asbestos.

[redacted]

8. I have written several papers on materials characterization as well as lectured numerous times on the subject.

9. Arthur D. Little, Inc., was retained by Mobil Oil Corporation to test an asbestos-containing product known as Hi-Heat Dum Dum to evaluate any occupational exposure during product application or removal.

10. The attached report describes the results of a study carried out under my direction to evaluate personal exposure to asbestos in working with Hi-Heat Dum Dum. The report accurately sets forth the methodology of the study and the conclusions I reached as a result of the experiments performed.

Edward T. Peters
Edward T. Peters

Sworn and subscribed to, before me, this 19th day of June, 1990.

Elaine M. Kenney
Elaine M. Kenney
Notary Public

My Commission Expires: 7/9/93

Evaluation of Asbestos Release from Chimney Weatherproofing Mastic During Application and Removal

By

Arthur D. Little, Inc.
Acorn Park
Cambridge, MA 02140-2390

1992

Reference No. 65191

Product Background

From the mid-1960's through the 1970's, Mobil Chemical Company manufactured and sold a line of mastic-type protective coatings under the name Dum Dum® Products. Chimney Dum Dum was a protective and waterproofing coating specially formulated for use on concrete and brick chimneys where heat resistance up to 210°F is required. The Mobil Chemical Product Catalog states:

> "Chimney Dum Dum/97 Series Chimney Dum Dum is a rugged heavy coating for the exterior of chimneys that provides flexible weatherproof protection. It fills and bridges hairline cracks with a flexible coating under a tough outer skin. Should the leathery skin become fractured, the soft under film will harden on contact with air. May be applied without completely cooling stack."

Chimney Dum Dum consists of a resinated vegetable oil vehicle, containing 41% pigments, including titanium dioxide, mica and the chrysotile form of asbestos; the total solids content is 63%. Application is by brush, or more typically, by spray. At 75°F, the surface skins over in 4-6 hours, and the under surface remains pliable. Resistance to chemical fumes and moisture is very good to excellent and the product is resistant to constant dry heat at 210°F.

Product Testing During Application and Removal

To determine if asbestos is released during the application or removal of Chimney Dum Dum, approximately 18 gallons of the product was prepared on the basis of the formula master and mixing instructions provided by Mobil. The oil base resin was no longer commercially available, and a special batch was prepared for us by McWhorter Corp. All other components were obtained commercially. The product was made with a Myers mixer in 6 gallon batches which were subsequently combined, blended and stored in 5 gallon pails. The prepared product was observed to be smooth and uniform in appearance and of mastic consistency. This work, as well as subsequent application and removal experiments, was conducted at our facilities in compliance with all current United States and Massachusetts rules and regulations relating to asbestos and in accordance with an approved Arthur D. Little, Inc. Health and Safety Plan.

A test plan was developed to simulate typical work practices for product application and removal[1]. In addition, a sampling and analysis protocol was developed to

[1]Although intended for exterior use, this product was tested in an interior (controlled) environment.

quantify the extent to which asbestos structures[2] may be released during the experimental application and removal activities. This protocol was designed so that all applicable NIOSH and EPA requirements for analysis by transmission electron microscopy were met or exceeded. The minimum detection limit for the analytical protocol was 0.01 structures/cc[3].

To evaluate the release of asbestos during product application and removal, ten concrete panels and twelve facing brick panels with concaved mortar joints, both sets four feet by two and one-half feet by one and one-half inches, prepared about one year earlier, were primed (Chimney Dum Dum Primer 38-V-5 equivalent), dried overnight and spray coated with Chimney Dum Dum at an estimated coating thickness of 30-40 mils using a Binks 2 gallon pressure pot with follower and a 7E2 spray gun. The coating operation required 55 minutes during which personal and area air samples were taken. The area sample was about ten feet from the spray site at an elevation of 5 feet.

After an ambient cure of 24 hours during which the coating skinned over, the panels were aged for 14 days at 200-210°F and eight days at ambient. The purpose of the furnace treatment was to accelerate the natural aging process. After the elapsed 23 days of aging, the product was hardened at the surface yet pliable and elastic when depressed with a screwdriver blade.

Product removal was carried out on eight brick panels and six concrete panels over 88 minutes during which personal and area filter samples were collected.

The collected filter samples from these experiments were prepared and analyzed by transmission electron microscopy. The results of these analyses are given in Table 1. These results may be overstated compared to actual exposures which would occur in an open air environment. As such, they are considered to represent a "worst case" condition.

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[2]Asbestos structures are defined herein as particles 5μm or more in extent that satisfy the counting guidelines given in 40 CFR Part 763 Asbestos-Containing Materials in Schools, Appendix A, Figure 5 (Federal Register 52 (210) October 30, 1987, 41866-41867). Structures include fibers, bundles, clumps and matrix. OSHA regulates exposure to fibers, which are defined as "a particulate form of asbestos, tremolite, anthophyllite or actinolite, 5 micrometers or longer, with a length-to-diameter ratio of at least 3 to 1" (29 CFR 1910.1001).

[3]The minimum detection limit (MDL) for this experiment is the concentration where occupational exposure cannot be differentiated from general background.

Table 1 Asbestos Exposures During Application and Removal of Chimney Dum Dum®			
Sample No.	Description	Sample Type	Exposure (s/cc)[4]
052802	Application	Personal	0.088
052803	Application	Personal (replicate)	0.024
052805	Application	Area	0.024
062001	Removal	Personal	<0.01
062002	Removal	Personal (replicate)	<0.01
062003	Removal	Area	<0.01

Conclusions

The conclusions reached in this study are as follows:

- Remanufactured Chimney Dum Dum was smooth and uniform with mastic-like consistency.
- Application to brick and concrete panels required heavy duty mastic spray apparatus.
- After 23 days of aging (14 days at 200-210°F), the product was skinned over but pliable and elastic when depressed with a tool.
- Results of worker exposure measurements during application of Chimney Dum Dum in a controlled environment were below the current OSHA action level[5].
- Results of worker exposure measurements during removal of Chimney Dum Dum in a controlled environment did not exceed the minimum detection limit of 0.01 structures/cc.
- Based upon the above results, there is insignificant occupational exposure to airborne fibers during the application of Chimney Dum Dum and virtually no exposure to airborne asbestos fibers during the removal of this product.

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[4]Asbestos structures per unit volume of air. The structures observed were fibers (or fibers and bundles, corresponding to the EPA definition).

[5]OSHA defines action level as "an airborne concentration of asbestos, tremolite, anthophyllite, actinolite, or a combination of these minerals, of 0.1 fiber per cubic centimeter (f/cc) of air calculated as an eight (8) hour time weighted average" (29 CFR 1910.1001, promulgated June 20, 1986).

Evaluation of Asbestos Release from Hi-Heat Dum Dum Caulking During Application and Removal

By

Arthur D. Little, Inc.
Acorn Park
Cambridge, MA 02140-2390

1992

Reference No. 65191

Arthur D Little

Table 1

Asbestos Exposures During Application and Removal of Hi-Heat Dum Dum: Personal Samples Analyzed by Transmission Electron Microscopy

Sample No.	Description	Exposure (s/cc)[5]
042504	First Application	<0.01
042604	Second Application	<0.01
051703	First Removal	<0.01
051705	First Removal (replicate)	<0.01
052203	Second Removal	<0.01
052207	Third Removal	0.01

Conclusions

The conclusions reached in this study are as follows:

- The Hi-Heat Dum Dum that was tested was found to be non-friable and provided encapsulation of asbestos during application and removal experiments.
- Exposure of the product to 225-250°F for 18 to 23 days changes its color from a neutral gray to a chocolate brown, indicating modification of the resin. Even so, the product was elastic and pliable except at the feathered, thin edges where it was hardened but not friable.
- Results of worker exposure measurements during use of Hi-Heat Dum Dum did not exceed the minimum detection limit of 0.01 asbestos structures/cc in either product application or product removal experiments.
- Based upon the above results, occupational exposure to airborne asbestos fibers during the application or removal of Hi-Heat Dum Dum is virtually non-existent.

[5]Exposure represents asbestos structures per unit volume of air.

Product Background

High temperature resinous caulking materials of mastic (putty-like) consistency are used in sealing joints and crevices on furnaces and boilers to prevent heat loss. Mobil Chemical Company manufactured and sold such a caulking product from the mid-1960s through the 1970s called Hi-Heat Dum Dum. This product, No. 46-F-7, consisted of a linseed oil-base vehicle (resin) containing various solid fillers, including clay and the chrysotile variety of asbestos (added for its resiliency and bridging properties). The Mobil Chemical Products Catalog provides the following description:

> "Hi-Heat Dum Dum/46-F-7, a heavy semi-plastic fibred coating, which acts as a joint sealer and pliable gasket for boilers, furnaces and dry kilns. Improves boiler efficiency by preventing heat loss. Retains its elasticity at consistent heat up to 175° and interminent heat to 350°. May be used as a complete cover to eliminate excess fuel consumption and can be painted over in 24 hours."

The purpose of a joint or crevice sealant is to bridge gaps between the furnace enclosure and attachments (doors, ports, flanges, etc.). This requires a product that is pliable and resilient (i.e., withstands shock without permanently deforming, tearing, rupturing or pulling away from the furnace surface), and does not degrade over time by becoming friable[1]. The resinous formulation of Hi-Heat Dum Dum provides these properties and also effectively encapsulates the asbestos component, preventing its release.

Product Testing During Application and Removal

Hi-Heat Dum Dum was tested by Arthur D. Little, Inc. to determine if asbestos was released during application or removal. Approximately 30 gallons of 46-F-7 Hi-Heat Dum Dum was remanufactured, as observed by Arthur D. Little, Inc. staff who provided chain-of-custody transmittal to our facility. Experiments (described below) were conducted at our facilities in compliance with all current United States and Massachusetts rules and regulations relating to asbestos and in accordance with an approved Arthur D. Little, Inc. Health and Safety Plan.

An application and removal plan was developed to simulate typical work practices. In addition, a sampling and analysis protocol was developed to quantify the extent to which asbestos structures are released during the experimental application and

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[1]The U.S. Environmental Protection Agency defines friable as "capable of being crumbled, pulverized, or reduced to powder by hand pressure".

removal activities[2]. This protocol was designed so that all applicable NIOSH and EPA requirements for analysis by transmission electron microscopy were met or exceeded. The minimum detection limit for the analytical protocol was 0.01 fibers/cc[3].

To evaluate the release of asbestos during product application and removal, steel panels (1/32 inch thick 1040 steel, cleaned and degreased, measuring about 2 1/2 x 3 1/2 feet) were coated by troweling at an estimated thickness of 1/8" (and feathered at the panel edges). Approximately 15 panels were coated during each of two 80-minute application experiments. Personal exposure samples were collected during both experiments.

After product application, the Hi-Heat Dum Dum was heated to accelerate the aging process[4]. The neutral gray of the as-applied coating changed in color to a chocolate brown (225-250°F for 20 days).

After aging, product removal experiments were conducted using a putty knife and scraper. Although the thicker, centrally coated area of each panel was very pliable and quite easily removed, the feathered edge regions were somewhat harder and required substantial mechanical effort to scrape down to the bare metal. Approximately 10 panels were cleaned in each of three, separate 80-minute experiments during which personal filter samples were collected.

The personal filter samples were prepared and analyzed by transmission electron microscopy. The results of these analyses are given in Table 1.

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[2]Asbestos structures are defined herein as particles 5μm or more in extent that satisfy the counting guidelines given in 40 CFR Part 763 Asbestos-Containing Materials in Schools, Appendix A, Figure 5 (Federal Register 52 (210) October 30, 1987, 41866-41867). Structures include fibers, bundles, clumps and matrix. OSHA regulates exposure to fibers, which are defined as "a particulate form of asbestos, tremolite, anthophyllite or actinolite, 5 micrometers or longer, with a length to diameter ratio of at least 3 to 1" (29 CFR 1910.1001).

[3]The minimum detection limit (MDL) for this experiment is the concentration where occupational exposure can not be differentiated from general background.

[4]During preliminary laboratory furnace experiments with coated steel coupons, we observed that the product discolored from a neutral grey to a dark chocolate brown after one day at 350°F and to a medium brown after four days at 265°F. Even after these exposures to temperatures which exceeded the recommended continuous use temperature, the product remained pliable and gummy when probed with a screwdriver. Therefore, although the product was designed for a continuous temperature of 175°F, a treatment of two weeks to dry, flowing air at 250°F was chosen to not only accelerate aging but also to account for intermittent temperatures greater than 175°F. The actual conditions of exposure were 225-250°F for 18 to 23 days.

Evaluation of Asbestos Release from Exterior Masonry Weatherproofing Mastic During Application and Removal

By

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Acorn Park
Cambridge, MA 02140-2390

1992

Reference No. 65191

Arthur D Little

Product Background

From the mid-1960's through the 1970's, Mobil Chemical Company manufactured and sold a line of mastic-type protective coatings under the name Dum Dum® Products. Dum Dum Masonoc/Series 95 was a restoration product for weatherproofing masonry structures. The Mobil Chemical Product Catalog states:

> "Dum Dum Masonoc/95 Series Masonoc is a heavy bodied textured coating designed as a weather seal, waterproofing, and restoration coating for all types of masonry structures. Pliability and elasticity allow for building movement without cracking of the coating while a tough outer skins provides protection for normal usage. Its high build qualities permit hairline cracks to be bridged and sealed, and surface irregularities to be filled and uniformed.
>
> Masonoc also provides fine protection for steel with its high film build and impermeability. Its excellent surface wetting properties recommend use of this material in areas that cannot be cleaned too well, where there are numerous angles and edges to protect, or when a single coat application is all that can be provided."

Masonoc consists of a resinated vegetable oil vehicle with about 22% solid fillers (by weight), including titanium dioxide, the chrysotile form of asbestos and various coloring pigments; the total solids content is 42 percent by volume. After application, Masonoc skins over in 1 hour, but the undersurface remains pliable to allow for expansion and contraction. The Mobil Chemical Product Data Sheet for Dum Dum Masonoc claims excellent resistance to dry heat (to 150°F), weather extremes, chemical fumes, moisture and salt air.

Product Testing During Application and Removal

Three 5 gallon pails of Dum Dum Masonoc (soft white 95-W-9) were obtained from an electric utility seeking information on product disposal. A sample was taken from one pail, about a third full, and ashed at 500°C. Microscopic analysis of this ash confirmed the presence of the chrysotile form of asbestos. The other two pails were intact with no visible signs of having been opened. After thorough mixing and being satisfied that the product viscosity was in the range of a mastic, these two pails of Masonoc were used for application and removal experiments. This work was conducted at our facilities in compliance with United States and Massachusetts rules and regulations applying to asbestos and in accordance with an approved Arthur D. Little, Inc. Health and Safety Plan.

A test plan was developed to simulate typical work practices for product application and removal[1]. In addition, a sampling and analysis protocol was developed to

[1]Although intended for exterior use, this product was tested in an interior (controlled) environment.

quantify the extent to which asbestos structures[2] may be released during the experimental application and removal activities. This protocol was designed so that all applicable NIOSH and EPA requirements for analysis by transmission electron microscopy were met or exceeded. The minimum detection limit for the analytical protocol was 0.01 structures/cc[3].

To evaluate the release of asbestos during product application and removal, concrete panels 4 feet by 2-1/2 feet by 1-1/2 inch were prepared in wooden frames and cured for 42 days. The panels were then each coated with Masonoc Primer 47-W-21, "a clear 100 percent solid heat resistant penetrating primer", dried overnight and then coated with Dum Dum Masonoc, using a Binks 7E2 spray gun and 2 gallon pressure pot, to a thickness of approximately 30-40 mils. Approximately 15 panels were coated during each of two 80 minute application experiments. Personal exposure and area air samples were collected during both experiments. The area sample was located about 10 feet from the worker (sprayer) at an elevation of 5 feet.

After product application, the coated panels were stacked (with spacers), allowed to dry for 24 hours and were then moved to an oven and exposed to flowing dry air at 150°F (the maximum recommended use temperature) for 42 days to accelerate the aging process followed by an additional 67 days at ambient conditions.

After aging, the product was found to be pliable and elastic when pressed with a screwdriver blade. A product removal experiment was performed using a putty knife and scraper. Eight panels were cleaned over an approximately 80 minute period during which personal samples were collected. After a further ambient aging period of 168 days (a total of 278 days after application), and again observing the product to be pliable and elastic, a second replicate removal experiment was performed on ten panels.

The collected filter samples from these experiments were prepared and analyzed by transmission electron microscopy.. The results of these analyses are given in Table 1. These results may be overstated compared to actual exposures which would occur in an open air environment. As such, they are considered to represent a "worst case" condition.

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[2]Asbestos structures are defined herein as particles 5μm or more in extent that satisfy the counting guidelines given in 40 CFR Part 763 Asbestos-Containing Materials in Schools, Appendix A, Figure 5 (Federal Register 52 (210) October 30, 1987, 41866-41867). Structures include fibers, bundles, clumps and matrix. OSHA regulates exposure to fibers, which are defined as "a particulate form of asbestos, tremolite, anthophyllite or actinolite, 5 micrometers or longer, with a length-to-diameter ratio of at least 3 to 1" (29 CFR 1910.1001).

[3]The minimum detection limit (MDL) for this experiment is the concentration where occupational exposure cannot be differentiated from general background.

Table 1 Asbestos Exposures During Application and Removal of Dum Dum Masonoc®			
Sample No.	Description	Sample Type	Exposure (s/cc)[4]
072701	First Application	Personal	0.01
072702	First Application	Area	<0.01
072703	Second Application	Personal	<0.01
072704	Second Application	Area	<0.01
111406	First Removal	Personal	<0.01
111407	First Removal (replicate)	Personal	<0.01
111403	First Removal	Area	<0.01
050104	Second Removal	Personal	<0.01
050107	Second Removal (replicate)	Personal	<0.01
050105	Second Removal	Area	<0.01

Conclusions

The conclusions reached in this study are as follows:

- After thorough mixing, the obtained pails of Dum Dum Masonoc®, confirmed to contain chrysotile asbestos, were smooth and of mastic consistency.
- Application to concrete panels required heavy duty mastic spray apparams.
- After aging at 150°F for 42 days and additional periods of 67 and 168 days at ambient conditions, the product was observed to be pliable and elastic when depressed with a tool.
- Results of worker exposure measurements during application and removal of Dum Dum Masonoc® in a controlled environment did not exceed the minimum detection limit of 0.01 structures/cc.
- Based upon the above results, occupational exposure to airborne asbestos fibers during the application or removal of Dum Dum Masonoc® is virtually non-existent.

[4]Asbestos structures per unit volume of air.

Resiliency of Mobil Hi-Heat Dum Dum after High Temperature Exposure

By

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1992

Reference No. 65191

Arthur D Little

Product Background

High temperature resinous materials of mastic (putty-like) consistency are used in sealing joints and crevices on furnaces and boilers to prevent heat loss. Mobil Chemical Company manufactured and sold such a caulking product from the mid-1960s through the 1970s called Hi-Heat Dum Dum. This product, No. 46-F-7, consisted of a linseed oil-base vehicle (resin) containing various solid fillers, including clay and the chrysotile variety of asbestos (added for its resiliency and bridging properties). The Mobil Chemical Products Catalog provides the following description:

> "Hi-Heat Dum Dum/46-F-7, a heavy semi-plastic fibred coating, which acts as a joint sealer and pliable gasket for boilers, furnaces and dry kilns. Improves boiler efficiency by preventing heat loss. Retains its elasticity at consistent heat up to 175° and intermittent heat to 350°. May be used as a complete cover to eliminate excess fuel consumption and can be painted over in 24 hours."

The purpose of a joint or crevice sealant is to bridge gaps between the furnace enclosure and attachments (doors, ports, flanges, etc.). This requires a product that is pliable and resilient (i.e., withstands shock without permanently deforming, tearing, rupturing or pulling away from the furnace surface), and does not degrade over time by becoming friable. The resinous formulation of Hi-Heat Dum Dum provides these properties and also effectively encapsulates the asbestos component, preventing its release.

Purpose of the Evaluation

The objective of the thermal exposure experiments was to determine the useful service life of Hi-Heat Dum Dum for temperatures within and above the recommended service temperature range described in the product literature.

Product Testing Experiments and Results

Approximately 30 gallons of 46-F-7 Hi-Heat Dum Dum was remanufactured, as observed by Arthur D. Little, Inc. staff who provided chain-of-custody transmittal to our facility. Approximately one pint was used for the tests described herein.

The aging experiments were conducted by filling steel fixtures with Dum Dum and placing the fixtures into ovens at different temperatures. The fixtures, consisting of a steel ring sportwelded to a piece of sheet steel, provided for a one inch diameter and three different thicknesses of the material - 0.062 inches, 0.125 inches and 0.187 inches. Fifteen fixtures of each thickness were prepared for a total of 45 fixtures, individually numbered by stamping.

Each fixture was tared on an analytical balance (scale) that measured the weight to 0.0001 grams. The fixtures were then filled with the Dum Dum product, reweighed to determine the

weight of the Dum Dum in the fixture and placed into ovens in triplicate at temperatures of: 175, 250, 350, 425 and 500 °F.

The three replicate samples at each thickness represented a total of nine samples at each temperature. At times of 22, 46, 70, 93.5, 133.5, 400.5 and 763 hours, the samples were removed from the ovens, allowed to cool to room temperature and again weighed. The percent change from initial weights are provided in Table 1.

All of the samples underwent a change in the original natural gray color after aging. The colors ranged from a darker gray (175°F) to chocolate brown (250°F) to dark brown (350°F) to black (425°F and 500°F). During the initial heating of the 500°F samples, smoke was emitted from the oven leading us to believe that the solvent within the sample ignited and burned. The surface of the 500°F samples became swollen and expanded out of the fixture in a spherical shape. This occurred to a lesser extent for the 425°F samples.

The samples also exhibited surface hardening. To determine any product brittleness or friability, we tested the flexibility of five samples aged for 763 hours by a bending experiment. For this purpose, a test sample was cut from the fixture such that there would be no interference from the fixture on the bend experiment. The tests were done using a three point bend fixture on an Instron 1332 mechanical testing machine with a 1000 pound load cell. A schematic representation of the bend condition is shown in Figure 1. Crosshead displacement control was used with a displacement rate of 0.1 inches/minute. The applied load and the crosshead displacement was continuously recorded. Test events such as the onset of surface cracking were noted. The experimental results of the bend tests are given in Table 2.

Discussion

Hi-Heat Dum Dum contains about 10 percent by weight mineral spirits, to provide a suitable viscosity for application, and a linseed oil blend as the vehicle which may have some volatiles; the solids content is stated as 85 percent by volume. Loss in weight of Hi-Heat Dum Dum exposed to elevated temperatures occurs by evaporation of the solvent and oxidation (ablation) of the resinous vehicle.

To estimate the useful life of the product, defined as retention of flexibility as demonstrated by a strain of 1 percent, the observed loss in initial weight as a result of furnace aging experiments is plotted in Figure 2, where the averages for three thicknesses tested are pooled. After 22 hours of high temperature exposure, much of the solvent has evaporated, more so as the exposure temperature increases. For longer exposures at elevated temperature, the 175, 250 and 350°F exposures demonstrate a linear change in weight loss when plotted against the logarithm of exposure time. The samples aged at 350°F for 763 hours still exhibit a 1 percent strain in bending (Table 2), having lost 12.5 percent of initial weight, representing essentially all of the non-solids content of Hi-Heat Dum Dum. From the data, product life at a sustained temperature of 250°F is conservatively estimated at more than 10 years.

Exposures of Hi-Heat Dum Dum at temperatures greater than 350°F results in rapid degradation due to both very rapid evaporation of the solvent and oxidation of the resin. Further, exposure of this product to sustained temperatures of 350°F could result in product life as short as one month. Accordingly, the use of this product under conditions where it is exposed to consistent heat of up to 175°F and intermittent heat of up to 350°F is considered prudent.

Conclusions

The conclusions reached in this study are as follows:

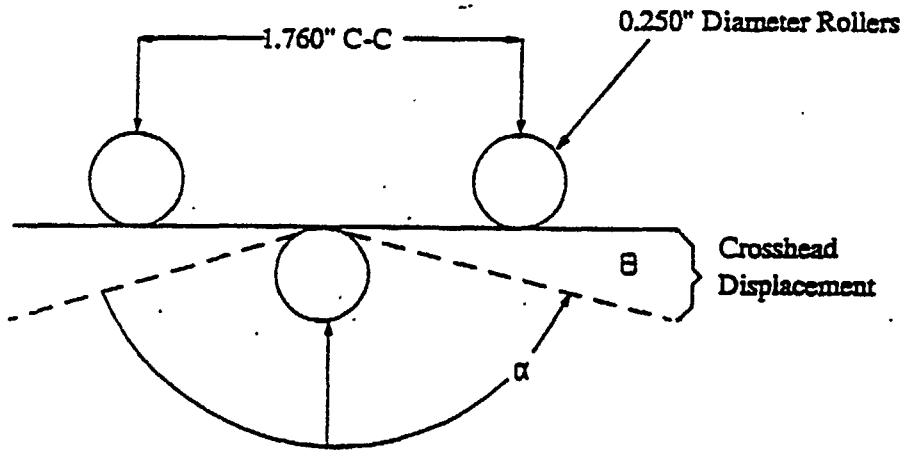
- Hi-Heat Dum Dum survives constant heating to 250°F and interminent heating to 350°F and behaves as described in the product literature. Heating the Dum Dum above 350°F causes rapid loss in weight and probable failure of the product.
- After 763 hours of aging at 250 and 350°F, the product can be strained by one to one and a half percent, demonstrating that it has retained elasticity.
- Based upon extrapolation of the data, Hi-Heat Dum Dum is expected to retain its elasticity for more than 10 years when used in accordance with the manufacturers recommendations.

Table 1
Hi-Heat Dum Dum: Percent Weight Loss
(Average of 3 Samples)

Temp.	Time - Cumulative Hours at Temperature						
(°F)	22	46	70.5	93.5	133.5	400.5	763
Thickness = 0.063"							
175	8.90	8.95	8.87	8.87	8.94	9.52	8.99
250	9.47	9.50	9.55	9.57	9.65	9.93	9.75
350	9.91	10.67	10.91	11.00	11.39	12.10	12.18
425	10.96	11.73	12.07	12.34	12.74	14.65	16.11
500	14.60	18.39	21.36	23.98	28.47	40.41	45.53
Thickness = 0.125"							
175	7.86	8.92	9.27	9.43	8.94	9.52	9.79
250	8.88	9.51	9.71	9.73	9.65	9.93	9.95
350	10.24	10.86	11.02	11.13	11.39	12.10	12.10
425	11.04	11.55	12.17	12.80	12.74	14.65	18.98
500	15.08	17.35	22.28	24.98	28.47	40.41	45.24
Thickness = 0.183"							
175	6.25	7.79	8.52	8.90	9.31	10.13	10.00
250	7.79	9.21	9.71	9.82	10.05	10.27	10.27
350	9.68	10.50	10.75	10.90	11.15	11.90	13.31
425	11.21	11.70	12.25	12.68	13.94	16.42	18.28
500	14.15	16.87	19.41	21.75	24.22	37.88	44.30

Table 2 Hi-Heat Dum Dum: Flexibility after 763 Hours at Temperature					
			Angle of Deflection		
Sample	Thickness (inches)	Aging Temp. (°F)	Crack Starts at One End	Fully Cracked	Percent Strain
37	0.125	250	10	16	1.7
27	0.187	250	4	11	1.2
33	0.062	350	4	10	1.1
8	0.125	350	5	10	1.1
28	0.187	350*	N/A	N/A	N/A
* Material separated from steel holder.					

Figure 1
Schematic of Bend Test Conditions and Bend Angle Calculations



Crosshead Displacement

$\sin\theta = 0.5\ (1.760)$

$\alpha = 180° - 2\ \theta$

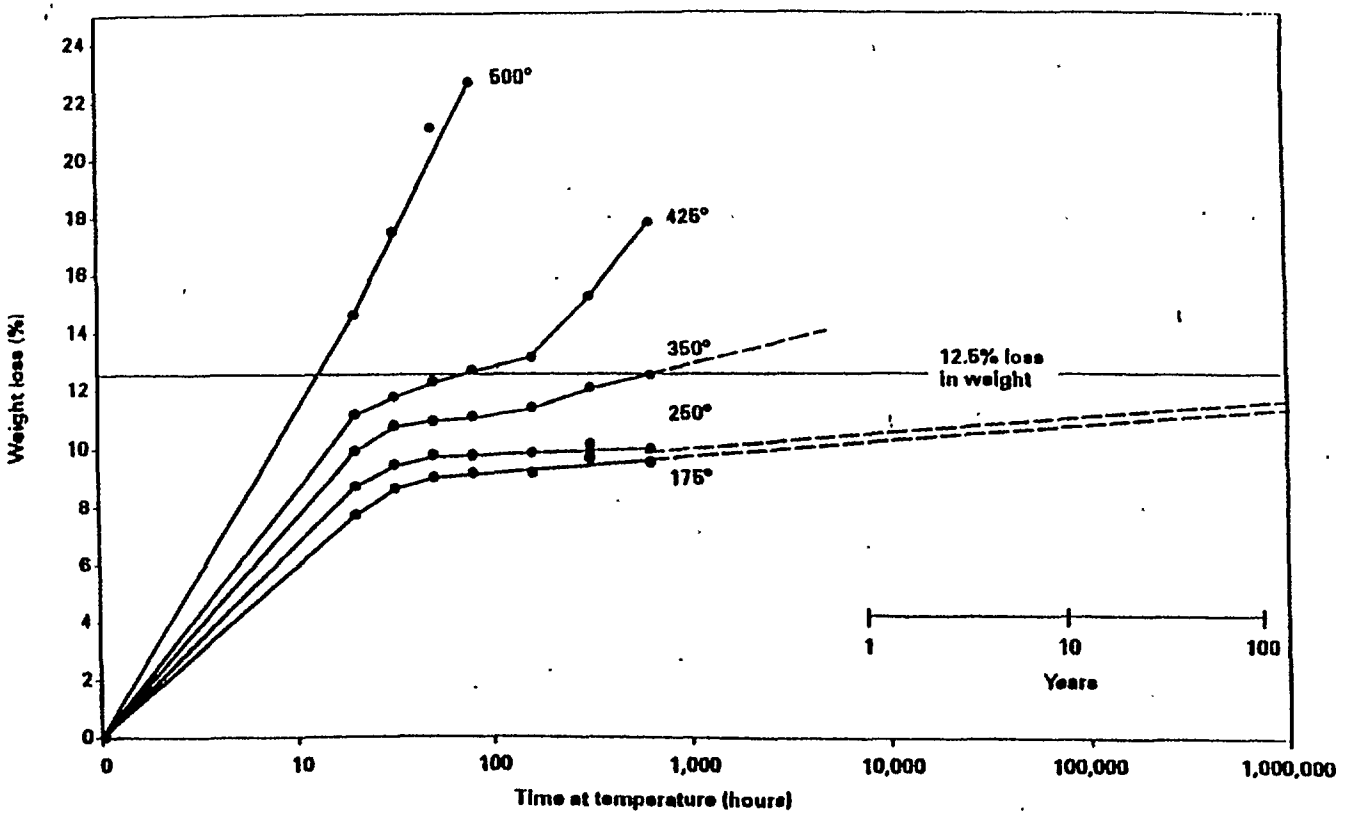


Figure 2. Hi-Heat Dum Dum Weight Loss with Time at Temperature