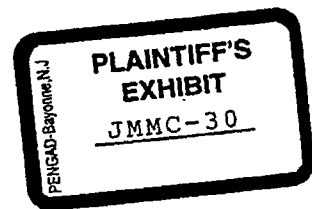


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State of California
AIR RESOURCES BOARD



Staff Report: Initial Statement of Reasons
for Proposed Rulemaking

Public Hearing to Consider the Adoption of a Regulatory
Amendment Identifying Asbestos as a
Toxic Air Contaminant

Agenda Item No.: 86-
Scheduled for Consideration: March 27, 1986
Release Date: February 10, 1986

(This report has been reviewed by the staffs of the California Air Resources Board and the California Department of Health Services and approved for publication. Approval does not signify that the contents necessarily reflect the views and policies of the Air Resources Board or the Department of Health Services, nor does mention of trade names or commercial products constitute endorsement or recommendation for use.)

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OVERVIEW AND RECOMMENDATION

I. INTRODUCTION

The Air Resources Board ("ARB" or "Board") identifies toxic air contaminants and develops regulations for the control of their emissions according to the requirements of state law. A toxic air contaminant (TAC) is an air pollutant that the Board or the Department of Food and Agriculture finds may cause or contribute to an increase in mortality or an increase in serious illness, or which may pose a present or potential hazard to human health. Health and Safety Code Section 39655 specifies that substances identified by the U.S. Environmental Protection Agency as hazardous air pollutants (Section 112 of the Clean Air Act) shall be identified as toxic air contaminants by the Air Resources Board. This report recommends that the Board find asbestos (in the following forms: chrysotile, actinolite, amosite, anthophyllite, crocidolite, and tremolite) to be a toxic air contaminant.

Section II of this Overview to the report presents the technical and toxicological information that supports the staff's recommendation. Section IIA is a summary of Part A, which presents data on the uses of asbestos, its emissions, and the concentrations of asbestos in the ambient air. Section IIB summarizes the Department of Health Services' (DHS) analysis in Part B of the health effects of asbestos. Section III presents the regulatory background and reviews the procedures by which the Board considers substances for the TAC designation. Section IV of this Overview discusses potential environmental effects of the recommended action, and Section V contains the staff's recommendation to the Board.

II. EVALUATION OF ASBESTOS

The ARB and the DHS prioritize candidate substances for evaluation and regulation as toxic air contaminants pursuant to Health and Safety Code (HSC)* Section 39660(f). That section states that the selection of a substance for consideration as a TAC is to be based on the risk to the public posed by the substance, the amount or potential amount of emissions from use of the substance, its manner of usage in California, its atmospheric persistence, and its concentration in the ambient air.

Under these guidelines, we selected asbestos for the Board's consideration as a TAC because it has been identified by the International Agency for Research on Cancer as a human carcinogen, it is emitted from many sources in the state, it is persistent in the atmosphere and in the environment at large, and its presence in the ambient air is documented. Asbestos has also been listed as a hazardous air pollutant by the U.S. Environmental Protection Agency and National Emission Standards have been promulgated. The standards are applicable to asbestos mills, roadways, manufacturing, demolition and renovation of buildings, spraying, fabricating, insulating materials, and disposal of asbestos containing waste.

A. EMISSIONS, PERSISTENCE AND AMBIENT CONCENTRATIONS OF ASBESTOS

The principal sources of asbestos emissions in California are a) mining and milling, b) manufacturing of asbestos products (both primary and secondary), c) automobile braking, and d) quarrying. Except for quarrying, these emission estimates include total asbestos fibers emitted into the atmosphere. For quarrying, the emission estimates are for asbestos fibers that have an effective aerodynamic diameter of 7 microns** or less. Other

* HSC; all statutory references are to the Health and Safety Code, except otherwise stated.

**A micron is 1×10^{-6} meters.

sources of emissions are demolition and renovation of buildings, roads surfaced with gravel containing asbestos, landfills, and natural weathering or human disturbance of serpentine (asbestos-containing mineral) deposits; however, emissions from these sources were not estimated due to lack of available information. Information on the emissions, persistence, and ambient concentrations of asbestos are summarized on Table I.

Asbestos supply and demand data for the United States from 1973 through 1984 shows a general decline in production and consumption. The demand for asbestos is expected to remain below the 1980 use through 1990.

Asbestos is exceptionally resistant to thermal degradation and chemical attack and fine fibers can remain airborne for long periods of time. Settled fibers are easily re-entrained back into the atmosphere. Atmospheric transport involves dispersion and reentrainment of asbestos fibers with deposition occurring due to wash-out and gravitational settling. The deposition and eventual burial of fibers in soils and sediments are the major processes by which asbestos fibers leave the atmosphere.

Asbestos has been documented in the air at several locations statewide by various studies. However, one study* was chosen for use in this report because the staff believes it provides the most suitable and most recent ambient data available for showing exposure to asbestos at several locations in California. The study found that in general, the highest concentrations (average 50 to 500 fibers/cubic meter)** of asbestos were measured at sampling

* Science Applications, Incorporated, 1983. Ambient Asbestos Concentrations in California - Volumes I and II

** Although measurement of ambient asbestos fibers is currently performed using transmission electron microscopy (TEM), cancer risk in this report is expressed in terms of phase contrast microscopy (PCM) fiber counts. Therefore, all concentrations in the Overview has been converted to PCM fibers per cubic meter to allow easier comparison of the health risk and exposure data. See Table III for sample calculations. Note: 100 to 1,000 TEM fibers/PCM fiber.

TABLE I
Summary of Data for Asbestos
Emissions

		Statewide		
<u>Source</u>	<u>Source Type</u>	<u>(Ton Per Year)</u>	<u>Inventory Year</u>	
<u>Mining</u>	Point	120	1984	
<u>Milling</u>	Point	340	1984	
<u>Manufacturing</u>				nd
Primary	Point	4	1982 & 1984	
Secondary	Point	2	1982 & 1984	
<u>Automobile Brakes</u>	Mobile	0.8	1984	
<u>Quarrying</u>	Point	0.5	1981	
		470		
Total*				

Atmospheric Fate

Asbestos can be readily subdivided into fibers of submicron diameter, which can remain airborne for long periods of time. In addition, asbestos is exceptionally resistant to thermal degradation and chemical attack, therefore settled fibers are persistent in the environment and subject to re-entrainment into the atmosphere.

Ambient Concentrations

Range of Total Asbestos
Fibers Measured at Ten Sampling
Locations (average of all
samples analyzed by transmission
electron microscopy and converted
to PCM fiber concentrations).

Averaged asbestos concentrations range from 8 to 80 PCM fibers per cubic meter at Sonora to 50 to 500 PCM fibers per cubic meter at South Gate. Generally, the highest concentrations were measured at sampling locations influenced by localized and industrial sources; the lowest concentrations were found at sites isolated from asbestos emission sources.

* The total emission estimate has been rounded off to one significant figure.

locations influenced by localized (where asbestos is processed, refined, or otherwise used) and industrial sources (areas with high density clusters of asbestos users). The lowest concentrations (average 8 to 80 fibers/cubic meter) were found at sites isolated from asbestos emission sources. In some of the locations with high concentrations, the population within ten kilometer grids is significant. The samples collected during the referenced study represent short averaging times thus, we are uncertain of how representative these concentrations are of annual averages.

In Part A, we also discuss an asbestos contamination problem in Alviso, California that DHS staff has been investigating. The contamination is thought to be caused by asbestos waste disposal and flooding in the area during the past 30 years. Ambient air monitoring studies by DHS have shown significant concentrations of asbestos fibers in the air throughout Alviso.

B. HEALTH EFFECTS AND RISK

In response to the ARB staff's request (Appendix A) and according to HSC Section 39660, the Department of Health Services (DHS) evaluated the health effects of asbestos and the risks from exposure to asbestos. To assist DHS, we provided DHS with a bibliography (Appendix B) of literature concerning the health effects of asbestos. The bibliography was obtained from the Toxline, Medline, and Biosis data bases available from NLM and Dialog information services. Also, we sent a letter (Appendix B) to all interested parties to request additional information. The information so obtained was forwarded to DHS.

In meeting the requirements in Section 39660 for DHS' evaluation, the DHS addresses these issues in Part B: 1) Is asbestos a carcinogen for animals and humans? 2) May health problems other than cancer occur from exposure to ambient concentrations? 3) Is there a "threshold" exposure level below which asbestos will not cause cancer? 4) What is the range of added risk of cancer during a lifetime of exposure to typical ambient concentrations of asbestos? 5) Should some particular fiber types (e.g., chrysotile, amosite) be considered more likely to cause cancer than others? 6) How does the relationship between fiber dimensions and carcinogenic potency affect the appropriateness of extrapolating health effects observed in occupational exposures to ambient air exposures? In response to these issues, the DHS makes the following conclusions and recommendation in Part B:

- 1) Asbestos is an undisputed human and animal carcinogen, and has been documented to cause cancer in humans in both occupational and nonoccupational settings.
- 2) Ambient asbestos levels in California are not expected to cause any acute health effects nor to result in asbestosis, a frequently disabling lung disease.
- 3) Although the mechanism of asbestos carcinogenicity is unknown, there is no compelling evidence that this process is characterized by a threshold.
- 4) The Department of Health Services has recommended the use of excess lung cancer lifetime risk values between 11 and 110 cases per million for each 100 PCM (phase contrast microscopy) fibers per cubic meter of asbestos exposure, and for mesothelioma a range of lifetime risk between 38 and 190 cases

for each 100 PCM fibers per cubic meter of asbestos exposure. Selection of these ranges was based on several health conservative assumptions. One effect of these assumptions is to produce estimated ranges of potential risk from exposure to ambient asbestos which, consistent with the health-conservative intent, include the highest, but not the lowest, reasonable projections. Estimated lifetime risks of lung cancer and mesothelioma by exposure group are shown in Table II. To calculate the risk estimates in Tables III and IV, DHS used average mean asbestos concentrations that have been measured in California. The concentrations used for exposure levels were 8 to 80 fibers/m³ for the lower range and 50 to 500 fibers/m³ for the upper range. Because of differences in counting methods, the average mean asbestos concentrations which were analyzed and counted by the transmission electron microscopy (TEM) method had to be converted to phase contrast microscopy (PCM) concentrations for DHS to do their risk analysis for those concentrations (see Table III). These values are consistent with the results of air sampling in a variety of locations in the United States (NRC, 1984).* As shown in these tables, risk estimates were also developed for a concentration of 2,000 fibers/m³, chosen by the NRC (1984) as representative of the 90th percentile of ambient asbestos concentrations, usually indicative of a local source of asbestos contamination.

* For information on the references cited in the Overview, see the Part B reference listing beginning on page R-1.

Table II

Estimated Lifetime Excess Risks of Lung Cancer and Mesothelioma Due to
Continuous Exposure to 100 Fiber/m³ of Asbestos
(Expressed as Cases per Million Population)^{1/}

<u>Exposure Group</u>	<u>Lung Cancer</u>	<u>Mesothelioma</u>
Male Smokers	11 (110)	24 (120)
Female Smokers	5 (50)	32 (160)
Male Nonsmokers	2 (15)	32 (160)
Female Nonsmokers	1 (6)	38 (190)

^{1/} Numbers in parentheses represent approximate upper confidence limits. The analysis corrected for competing causes of death using lifetables constructed from recent California vital statistics. Since risks for lung cancer and for other causes of death are dependent on smoking status, the lifetables were modified to account for age- and gender-specific smoking prevalence. Thus, risks are presented by gender and smoking status.

TABLE III

Estimated Lifetime Excess Lung Cancer Risk Due to
Continuous Exposure to Asbestos
(Expressed as Cases per Million Population)*

Exposure Group	Exposure Level (fibers/m ³)				
	8	50	80	500	2,000
Male Smokers	1(0-9)**	6(0-55)	9(0-88)	55(0-550)	221(0-2,210)
Female Smokers	1(0-5)	2(0-25)	5(0-41)	25(0-250)	101(0-1,010)
Male Nonsmokers	1(0-1)	1(0-8)	1(0-11)	8(0-75)	29(0-290)
Female Nonsmokers	1(0-1)	1(0-3)	1(0-5)	3(0-28)	11(0-110)

* Calculated with $C_1 = 0.01$. Ranges in parentheses were estimated with a lower limit of zero and an upper limit calculated with $C_1 = 0.1$. This upper bound is an approximate upper confidence limit.

** Sample Calculation For Excess Lung Cancer Risk - Male Smokers

Conversion from TEM = $\frac{(7,700 \text{ TEM fibers})}{\text{m}^3} \frac{(1 \text{ PCM fiber})}{(1000 \text{ TEM fibers})}$
to PCM concentrations

$$= 7.7 \text{ or } 8 \text{ fibers/m}^3$$

Estimated lifetime excess lung cancer risk-lower confidence limit = $\frac{(\text{PCM concentration})}{(\text{Estimated lifetime risk exposure concentration})} \times (\text{Lung cancer estimated lifetime risk})$

$$= \frac{(8 \text{ fiber/m}^3)}{(100 \text{ fibers/m}^3)} \times 11$$

$$= 0.88 \text{ or } 1$$

Estimated lifetime excess lung cancer risk - higher confidence limit = $\frac{(8 \text{ fibers/m}^3)}{(100 \text{ fibers/m}^3)} \times 110$

$$= 8.8 \text{ or } 9$$

TABLE IV

Estimated Lifetime Excess Mesothelioma Risk Due to
Continuous Exposure to Asbestos
(Expressed as Cases per Million Population)*

Exposure Group	Exposure Level (fibers/m ³)				
	8	50	80	500	2,000
Male Smokers	2(0-9)*	11(0-59)	19(0-95)	120(0-590)	470(0-2,400)
Female Smokers	2(0-12)	16(0-81)	26(0-120)	160(0-810)	640(0-3,300)
Male Nonsmokers	2(0-12)	16(0-79)	25(0-120)	160(0-790)	630(0-3,200)
Female Nonsmokers	3(0-16)	19(0-97)	31(0-160)	190(0-970)	780(0-3,800)

* Calculated with $C_m = 2.4 \times 10^{-8}$, $P = 3.0$, 20-year lag. Ranges in parentheses were estimated with a lower limit of zero and an upper limit calculated with $C_m = 1.2 \times 10^{-7}$, which is the highest estimated value for the proportionality constant C_m (from Finkelstein, 1983).

- 5) Risk assessment should not be segregated by fiber type.
- 6) DHS staff members believe that it is appropriate and reasonable to extrapolate from occupational exposure measurements to ambient air exposures.

The Department of Health Services' conclusions and recommendation were based upon the following:

- o Asbestos has been consistently demonstrated to be carcinogenic in animals and humans and is recognized as a human carcinogen by the International Agency for Research on Cancer.
- o In occupational cohort mortality studies, exposure to the three principal commercial forms of asbestos has been repeatedly linked with increased risks for lung cancer, mesothelioma and, to a lesser extent, other neoplasms.
- o Other than cancer, the most serious adverse health outcome ascribed to asbestos exposure is asbestosis, a consequence of extensive fibrosis of the lung due to the presence of asbestos fibers.
However, DHS staff members concur with the Consumer Products Safety Commission (CPSC) (1983) panel that knew of "no reports of disabling asbestosis occurring among persons whose maximum exposure to asbestos was of the order of 1 fiber/ml (by PCM) or less". The reserve capacity of the lung is sufficiently large that exposure to ambient asbestos levels does not yield asbestosis symptomatology.
- o Chronic exposure to asbestos can also result in pleural changes (thickening, plaque formation, and/or benign effusions), which are generally asymptomatic.
- o Lung cancer and mesothelioma are the outcomes of interest for the purpose of a quantitative risk assessment, since both are considered

to be nonthreshold processes posing potential population risks at ambient concentrations of asbestos. The risk assessment utilized the work done by the CPSC (1983), the National Academy of Sciences (NRC) (1984), Nicholson (1985), and the Ontario Royal Commission (1984).

- o DHS adapted models developed and/or used by earlier investigators to estimate risks of mesothelioma and lung cancer to the general population, and extrapolated risks observed in occupationally-exposed cohorts to lower levels of asbestos to which the general population may be exposed.
- o In estimating the maximum excess lifetime risks, DHS has used health conservative assumptions. Some of the more significant health conservative assumptions can be classified as relating to pathophysiology, to exposure or to the models used. These assumptions are listed below:

Pathophysiology. The carcinogenic effects of asbestos fibers are assumed to be exerted by short (<5 microns in length) fibers as well as long (≥ 5 microns) ones, at doses substantially lower than those in past occupational settings (see Sections 9.b and 9.d of Part B). Chrysotile is assumed to be as potent as the amphiboles in causing mesothelioma (see Section 9.f of Part B).

Exposure. Exposure to ambient levels of asbestos is assumed to occur 24 hours/day at ambient levels for a lifetime (see Section 9.f of Part B).

Models. The risk assessment models are nonthreshold and are linear with dose. The lung cancer proportionality constant

"C₁" used by DHS was derived omitting the results from the mining and milling epidemiologic studies (see Section 9.a.1.5 of Part B). The upper bound in DHS risk assessment was calculated with C₁ set at 0.1, which was the highest 95% upper confidence limit estimated for any of the epidemiologic studies under consideration (See Section 9.2 of Part B).

- o DHS' estimated lifetime risks of lung cancer and mesothelioma due to continuous exposure to 100 fibers/m³ of asbestos are compatible with the range of risks derived from the reports by NRC (1984), CPSC (1983) and Nicholson (1985). Mesothelioma risk estimates are also consistent with the recent incidence data obtained from the Bay Area Surveillance Epidemiology and End Results Program for 1973-1983.
- o Although some epidemiologic evidence suggests that the risk of mesothelioma from exposure to chrysotile may be lower than that from exposure to the amphiboles,* DHS concludes that there is no compelling reason to differentiate between fiber types in risk assessment. Mixed exposures, lack of good quantitative exposure data, and the physical effects of different industrial processes on asbestos fibers make the comparison of epidemiological studies problematic in this respect. Furthermore, in animal studies chrysotile has been shown to be at least as potent as the amphiboles in inducing mesothelioma.
- o The generally accepted hypothesis that longer and thinner fibers have greater carcinogenic potency than shorter and/or thicker fibers was based on Stanton's study in which animals exposed to large

* Amphibole asbestos is a general term for all varieties other than chrysotile.

numbers of long, thin fibers produced the highest incidence of mesotheliomas. However, DHS staff members have found limitations with the experimental basis for this hypothesis. These limitations are:

- 1) The experimental model can be directly applied only to mesothelioma;
- 2) The range of fiber dimensions used in the study is incomplete or uncertain due to the inherent problem of accurately measuring asbestos fibers and the difficulty of obtaining the exact distribution of fiber dimensions;
- 3) Further analysis of the experimental data indicates that carcinogenicity appears to be a continuous, increasing function of the aspect ratio of fiber dimensions;
- 4) Although the clearance of shorter fibers is more efficient such clearance is neither instantaneous nor complete;
- 5) Most asbestos fibers found at the pleura are short (< 5 microns), fine chrysotile, as opposed to the mixed fiber populations found in the lung parenchyma; and
- 6) The relationship of physical dimensions of fibers and their translocation and final deposition to target organs in humans has not been well characterized.

III. REGULATORY BACKGROUND AND PROCEDURES

Division 26, Chapter 3.5 of the HSC and Food and Agriculture Section 14021 et seq. set forth the procedure for identifying and controlling toxic air contaminants in California. (These provisions were enacted in September 1983 as Assembly Bill 1807, Stats. 1983, ch. 1047). The Department of Food and Agriculture is responsible for identifying and controlling TACs in their pesticidal uses. The ARB has authority over TACs in all their other uses.

HSC Section 39650 sets forth the Legislature's findings about substances which may be TACs. The Legislature has declared:

"That public health, safety, and welfare may be endangered by the emission into the ambient air of substances which are determined to be carcinogenic, teratogenic, mutagenic, or otherwise toxic or injurious to humans."

The findings also include directives on the consideration of scientific evidence and the basis for regulatory action. With respect to the control of TACs, the Legislature has declared:

"That it is the public policy of this state that emissions of toxic air contaminants should be controlled to levels which prevent harm to the public health."

The Legislature has further declared that, "while absolute and undisputed scientific evidence may not be available to determine the exact nature and extent of risk from toxic air contaminants, it is necessary to take action to protect public health."

In the evaluation of substances, the Legislature has declared that the best available scientific evidence, gathered from both public agencies and private sources, including industry, should be used. The Legislature has also determined that this information should be reviewed by a scientific review panel and by the public.

The Board's determination of whether or not a substance is a toxic air contaminant includes several steps specified by HSC. First, we request the DHS to evaluate the health effects of a substance (Section 39660). The evaluation includes a comprehensive review of all available scientific data. Upon receipt of a report on health effects from DHS and in consideration of

their recommendations, we prepare and submit a report to the Scientific Review Panel (SRP) for its review (Section 39661). The report consists of the DHS report (Part B), and material prepared by the ARB staff on the use, emissions and ambient concentrations of the substance (Part A). It serves as the basis for future regulatory action by the Board. The report is also made available to the public, who may submit comments on the report to the SRP.

After receiving the SRP's written findings on the report, the Board issues a public hearing notice and a proposed regulation which include the proposed determination that a substance is a toxic air contaminant. If, after a public hearing and other procedures to comply with Government Code Section 11340 et seq., the Board determines that a substance is a toxic air contaminant, its findings must be set forth in a regulation (Section 39662). The HSC also sets forth procedures for developing and adopting control measures for substances identified as TACs (Sections 39665-39667).

IV. ENVIRONMENTAL EFFECTS

The identification of asbestos as a TAC will not in itself have any environmental effects. If the Board lists asbestos as a TAC, the staffs of the ARB and the air pollution control districts will evaluate the need for, and appropriate degree of controls for emission sources. After this evaluation, the Board and the districts may adopt emission control measures which will result in the reduction of asbestos concentrations in the ambient air. Any environmental effects associated with control measures will be identified when such control measures are considered pursuant to HSC Sections 39665 and 39666.

V. RECOMMENDATION

Because asbestos is a known animal carcinogen and human carcinogen, has been listed by the U.S. Environmental Protection Agency as a hazardous air pollutant, and is known to be emitted in California, the ARB staff recommends its listing as a toxic air contaminant. In making this recommendation, we note that there is not sufficient available scientific evidence to support the identification of an exposure level below which carcinogenic effects would not occur.

State of California
AIR RESOURCES BOARD

TECHNICAL SUPPORT DOCUMENT

PUBLIC HEARING TO CONSIDER THE ADOPTION OF A REGULATORY
AMENDMENT IDENTIFYING ASBESTOS AS A
TOXIC AIR CONTAMINANT

Agenda Item No.: 86-
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Part C	Public Comments and Responses

PART A - A REVIEW OF ASBESTOS USES, EMISSIONS
AND PUBLIC EXPOSURE

Prepared by the Staff of the Air Resources Board

January 1986

(This report has been reviewed by the staffs of the California Air Resources Board and the California Department of Health Services and approved for publication. Approval does not signify that the contents necessarily reflect the views and policies of the Air Resources Board or the Department of Health Services, nor does mention of trade names or commercial products constitute endorsement or recommendation for use.)

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PART A - A REVIEW OF ASBESTOS USES, EMISSIONS
AND PUBLIC EXPOSURE

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I. NATURAL OCCURRENCE, PRODUCTION, USAGE, AND EMISSIONS

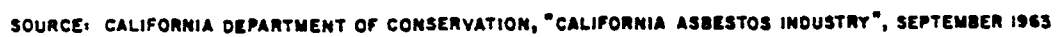
A. NATURAL OCCURRENCE

The word "asbestos" is not a mineral name, but a term applied to a group of naturally occurring mineral silicate fibers of the serpentine and amphibole series. Serpentine minerals are "layered silicates" and only one of these, chrysotile, is an asbestos mineral. The amphibole asbestiform minerals are "chain silicates" and the five varieties of commercial importance are commonly known as actinolite, amosite, anthophyllite, crocidolite and tremolite (Chronic Hazard Advisory Panel on Asbestos, 1983).

Chrysotile occurs only in serpentine, a fine-grained rock composed almost entirely of hydrous magnesium silicate minerals similar to chrysotile in chemical composition. Serpentine is a secondary rock, derived by alteration of several magnesium-rich types of igneous rocks, principally peridotite, and is abundant in the Coast Ranges, Sierra Nevada, and Klamath Mountains of California (California Department of Conservation, 1963).

Amphibole asbestos is a general term for all varieties other than chrysotile. Only a few of the amphibole minerals, primarily those that are aluminum poor, become sufficiently fibrous to be used as asbestos. Of these, tremolite and actinolite are the only ones that have been of significance in California. Amosite has not been found in California, and crocidolite and anthophyllite have not been found here in commercial quantities (California Department of Conservation, 1963). Figure I-1 shows the principal asbestos deposits in California and the location of the two operating mines.

I-2



B. PRODUCTION

Only three mines produce asbestos in the United States, two of these are in California: The Calaveras Asbestos Corporation which operates a mine in Calaveras County, and KCAC, Inc. which operates a mine in San Benito County. Both mines produce chrysotile asbestos by open pit mining (Burnett, 1982; U.S. Department of the Interior, 1976-1984; Versar Inc., 1984). Prior to 1980 as many as five asbestos mines were in operation in California (Roberts, 1980). In 1984, California mines and mills produced 57,308 tons of asbestos.

C. USAGE

Before asbestos from a mine can be used, it must be processed into fiber at a mill. Milling is a complex operation which separates the fiber from the mined rock by repetitively crushing the rock and then screening out the fiber. The fiber is then separated into various grades based on length. The processed asbestos fibers are then utilized by either primary or secondary manufacturers to produce various products for end users (U.S. Department of the Interior, 1980). The longest fibers are used for spinning and are woven into fabrics while the shorter fibers, nonspinning grades, are used in such products as cement or paper stock.

Primary manufacturers are companies that process raw asbestos fiber into either intermediate products (to be further processed or fabricated) or into finished products. Intermediate products from primary manufacturers include gasket paper, electrical insulating paper, textiles, packings and gaskets, and asbestos-cement sheets. Typical examples of finished products from primary manufacturers include coating mixtures, adhesives and sealants, floor covering, roofing felts, and asbestos-cement pipes. Some primary products

such as asbestos cement pipe, vinyl asbestos floor tile, and asbestos coatings and sealants undergo little or no secondary processing. Other products such as asbestos packings, gasketing materials, asbestos textiles, asbestos reinforced plastics, asbestos papers, and asbestos cement sheets must undergo further fabrication or modification before the product is finished (Versar Inc., 1984).

Secondary manufacturers are companies that further process the intermediate asbestos product to produce either another intermediate product or a finished product. Some examples of finished products from secondary manufacturers include tapes, spacers, washers and electrical component boards fabricated from electrical insulating paper; brake pads riveted or bonded to brake shoes; fume hood liners and laboratory table tops (Versar Inc., 1984).

Although processed asbestos fiber has more than 2000 uses, its major use has been in products connected with the construction industry (U.S. Department of the Interior, 1980). In 1984, the end use consumption of asbestos in the U.S. was as follows: friction products, 22 percent; flooring products, 21 percent; asbestos cement pipe, 12 percent; coatings, adhesives and sealant compounds, 11 percent; asbestos cement sheet, 8 percent; packings and gaskets, 6 percent; roofing products, 3 percent; paper and textiles, 1 percent; and other, 16 percent (U.S. Department of the Interior, 1985).

Of the total asbestos consumed in the United States in 1983, 97 percent was chrysotile and three percent was crocidolite. Small amounts of amosite were reported used (U.S. Department of the Interior Minerals Yearbook, 1984). A more recent breakdown of the types of asbestos used is not yet available.

Asbestos supply and demand data during the last 10 years shows a general decline in production and consumption. Figure I-2 depicts the general

decrease in production and consumption of asbestos from 1973 to 1984 (U.S. Department of the Interior, 1965-1974; U.S. Department of the Interior, 1976-1984). The demand for asbestos is expected to remain below the 1980 use through 1990 (U.S. Department of the Interior, 1985).

Consumption of asbestos could decrease at a greater rate if the U.S. Environmental Protection Agency (EPA) pursues a draft proposed asbestos ban under the Toxic Substances Control Act.

On June 15, 1984, EPA submitted to the Office of Management and Budget (OMB) a draft proposed rule that would have banned the use of asbestos in asbestos cement pipe, roofing tiles, floor tiles, and sealant compounds beginning in 1985. EPA submitted a second draft proposed rule on August 15, 1984 that would have banned the use of asbestos clothing immediately and would have banned all uses of asbestos by 1995. EPA's regulatory impact analysis of the proposed rules attributed 87 percent of the benefits to reduced exposure in the workplace. The other 13 percent were attributed to reduced ambient exposure.

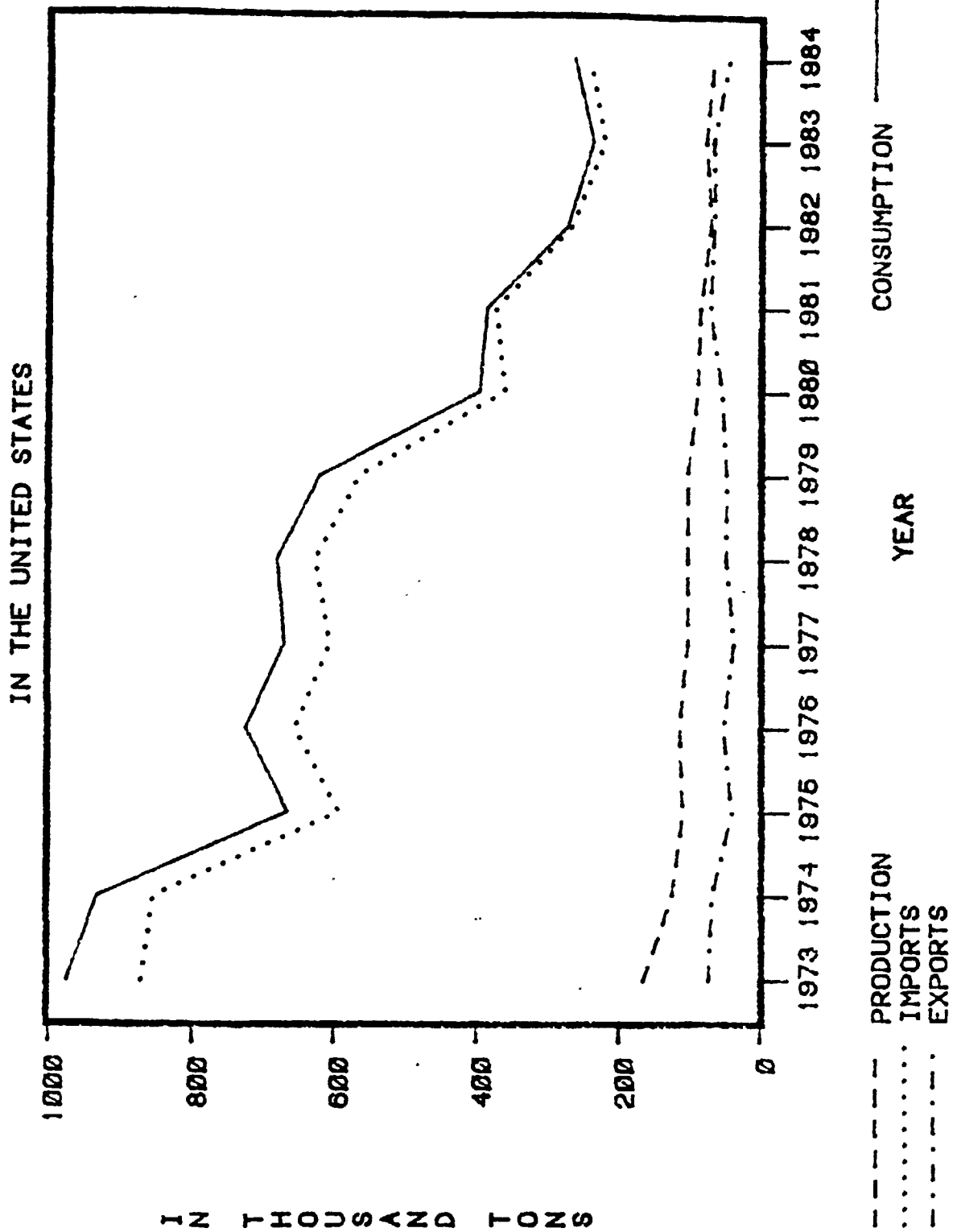
The EPA dropped its proposal to ban asbestos in February, 1985 based on OMB's legal analysis which concluded that it was appropriate for EPA to refer regulatory authority to another agency if that agency can reduce or prevent the risk associated with the substance. It is unclear at this time if EPA will pursue this again in the future.

D. ESTIMATED STATIONARY AND MOBILE SOURCE EMISSIONS

The principal sources of asbestos emissions in California are a) mining and milling, b) manufacturing of asbestos products (both primary and secondary), c) automobile brakes, and d) quarrying. Other sources of emissions are landfills, renovation and demolition of buildings, roads

Figure I-2

ASBESTOS PRODUCTION AND CONSUMPTION IN THE UNITED STATES



surfaced with gravel containing asbestos, and natural weathering or human disturbance of serpentine (asbestos containing mineral) deposits. A summary of estimated asbestos emissions is shown in Table I-1. Except for quarrying, the emission estimates include total asbestos fibers emitted into the atmosphere. For quarrying, the emission estimates are for asbestos fibers that have an effective aerodynamic diameter of 7 microns or less.

1. Mining and Milling

Mining and milling by the Calaveras Asbestos Corporation and KCAC, Inc. account for most of the estimated emissions of asbestos in the state (refer to Table I-1). These emissions arise from activities such as drilling, blasting, gathering and loading of ore, transportation of the ore, milling, and the removal of waste dust (Archer and Blackwood, 1979; Versar Inc., 1984). Figure I-3 shows a typical flow diagram of mining and milling operations.

Available asbestos emission factors for mining operations range from 5 to 8 pounds per ton of processed ore if emission control methods are used and from 9 to 10 pounds per ton if no control methods are used (Archer and Blackwood, 1979; Meylan, et al., 1978; Rajhans and Bragg, 1978; Versar Inc., 1984). Emission control methods for mines include spraying the ore and/or roadways to reduce dust, as well as fitting drilling equipment with cyclone dust collectors. Generally, it is difficult to completely control mining operations because many activities are carried out in the open. Since California mines are required to use control methods in order to comply with the National Emission Standard for Asbestos and to wet exposed ore, the lowest emission factor of 5 pounds per ton was used to estimate mining emissions.

Table I-1
Summary of Estimated Asbestos Emissions

<u>Source</u>	<u>Source Type</u>	<u>Emissions* (tons/year)</u>	<u>Inventory Year</u>	<u>Reference</u>
<u>Mining</u>	Point	120	1984	<u>1/</u>
<u>Milling</u>	Point	340	1984	<u>1/</u>
<u>Manufacturing</u>				
Primary	Point	4	1982 & 1984	<u>2/</u>
Secondary	Point	2	1982 & 1984	<u>2/</u>
<u>Automobile Brakes</u>	Mobile	0.8	1984	<u>3/</u>
<u>Quarrying</u>	Point	<u>0.5</u>	1981	<u>4/</u>
Total		470		

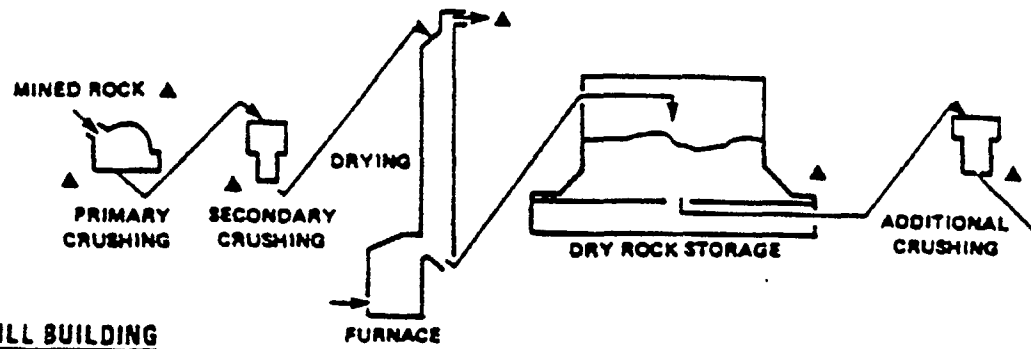
* The emission estimates may not total due to rounding.

- 1/ Archer and Blackwood, 1979; Roberts, 1980; U.S. Department of the Interior, 1976-1984; Versar Inc., 1984.
- 2/ California Air Resources Board, 1985; Versar Inc., 1984; Zwiacher et al., 1983.
- 3/ California Air Resources Board, 1984; Williams and Muhlbaier, 1982.
- 4/ Air and Industrial Hygiene Laboratory, 1981; Blackwood, Chalekode, and Wachter, 1978; U.S. Environmental Protection Agency, 1981.

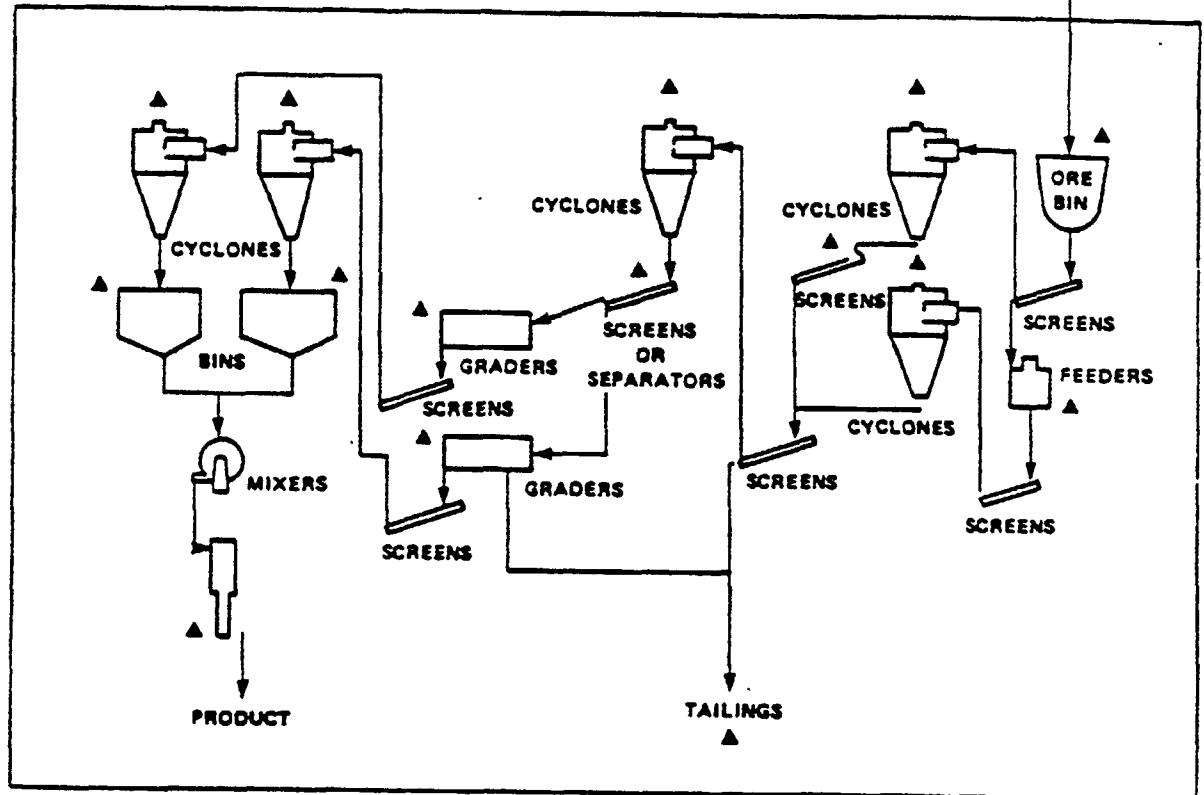
Figure I-3

FLOW DIAGRAM OF ASBESTOS MINING AND MILLING

OPEN AREA



MILL BUILDING



▲ Possible particulate emission source.

Source: Versar Inc., 1984.

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Asbestos emissions from mining operations are therefore estimated to be approximately 120 tons in 1984.

Reported milling-emission factors range from 12 to 80 pounds per ton if controlled and 100 pound per ton if uncontrolled (Archer and Blackwood, 1979; Meylan, et al., 1978; Rajhans and Bragg, 1978; Versar Inc., 1984). A number of control methods are used for milling operations. Cyclones are used first to concentrate and collect the fibers. Figure I-3 shows the typical locations where cyclones are used. Baghouses are then used to control the air emissions of asbestos from such processes as crushing, screening, fiberizing, product bagging, and conveying of waste rock material to the tailing pile (Harwood and Blaszkak, 1974; Meylan, et al., 1978; Versar Inc., 1984). Engineering estimates of collection efficiency for baghouses are about 99.99 percent for fibers greater than 1.5 um and about 98 percent for fibers less than 1.5 um in length (Harwood, C. F., et al., 1974). However, the overall reduction in asbestos emissions should be greater when cyclones and baghouses are used in series. Tailing emissions also arise from the milling operation. These are usually controlled by spraying with water and chemical stabilizers.

The Calaveras Asbestos Corporation uses baghouses or cyclones combined with baghouses to control emissions during ore preparation, drying, at the dryrock storage building, and on the milling facility. They also apply chemical stabilizers and water to control emissions from mill tailings . Assuming that both milling facilities in the state control asbestos emissions to the lowest reported level, an emission factor of 12 pounds per ton of processed asbestos was used to calculate emissions. The estimated emissions from California milling operations in 1984 is 340 tons.

2. Manufacturing Emissions

a. Primary

There are 42 identified primary manufacturers of asbestos products in the state. Of these, 33 are in the South Coast Air Basin. About three-fourths of the manufacturers produce coatings, adhesives and sealants. The rest are producers of asbestos cement pipe, asbestos reinforced plastic, floor tiles, paper, packings and gaskets or friction products. Reported emission factors for primary manufacturing of the various asbestos products range from 0.14 pound to 1.1 pounds asbestos per ton of asbestos used (Versar Inc., 1984). Applying these factors to the data derived from surveys of asbestos product manufacturers, asbestos emissions from primary manufacturing are estimated to be about 4 tons per year. Table I-2 presents estimated emissions by product category for the primary manufacturing facilities and Table I-3 shows where the facilities are located.

Although most of the manufacturers produce coatings, adhesives or sealants, over 70 percent of the estimated emissions come from the production of asbestos cement pipe. Emissions occur during the mixing of asbestos fiber with cement and silica, and during the receiving and storage of asbestos. Baghouses are typically used to control emissions where possible. The overall emission factor for the process was estimated to be 0.30 pound per ton (Meylan, et al., 1978; Versar Inc., 1984).

b. Secondary

There are 48 identified secondary manufacturers of asbestos products in California. About half are engaged in the fabrication of friction products, 27 percent in packings and gaskets and the rest in paper, coatings, adhesives and sealants, plastics, textiles and chlorine manufacture. The combined

Table I-2
Summary of Asbestos Emissions from
Primary Manufacturing^{1/}

	No. of Facilities	Total Tons Processed	Emission Factors (pound/ton)	Emissions* (ton/year)
Friction Products	2	350	0.60	0.1
Packings and Gaskets	2	3	0.60	< 0.1
Coatings, Adhesives and Sealants	32	4,080	0.20	0.4
Cement Pipe	2	16,700	0.30	3
Paper	1	30	1.10	< 0.1
Plastics	2	9,300	0.14	0.7
Floor Tile	<u>1</u>	<u>30</u>	<u>0.34</u>	<u>< 0.1</u>
Total	42	30,493		4

* The emission estimates may not total due to rounding.

^{1/} References used to develop this table were: Air Resources Board, 1985 and Zwiacher, 1983. Emission factors used were taken from Versar Inc., 1984.

Table I-3

Locations of Primary Manufacturing
Facilities in California

Product	Los Angeles County	Orange County	Other ^{1/} South Coast Counties	San Diego County	Contra Costa County	Alameda County	Other ^{2/} Bay Area Counties	San Joaquin County	Total
Friction Products	2								2
Packings and Gaskets	1	1							2
Coatings, Adhesives and Sealants	19	2	3	1	2	3	2		32
Cement Pipe			1					1	2
Paper					1				1
Plastics	1	1							2
Floor Tile	<u>1</u>	<u>—</u>	<u>—</u>	<u>—</u>	<u>—</u>	<u>—</u>	<u>—</u>	<u>—</u>	<u>1</u>
Total	24	4	4	1	3	3	2	1	42

^{1/} Riverside County and San Bernardino County
^{2/} San Francisco County and San Mateo County

Table I-4

Summary of Asbestos Emissions from
Secondary Manufacturing^{1/}

	No. of Facilities	Total Tons Processed	Emission Factors (pound/ton)	Emissions* (ton/year)
Friction Products	25	12,500	0.31	2
Packings and Gaskets	13	80	0.53	< 0.1
Coatings, Adhesives and Sealants	1	0.2	0.024	< 0.1
Paper	5	13	1.08	< 0.1
Plastics	1	0.1	0.14	< 0.1
Textiles	2	0.6	0.28	< 0.1
Chlorine Manufacturing	<u>1</u>	<u>30</u>	<u>0.14</u>	<u>< 0.1</u>
Total	48	12,624		2

* The emission estimates may not total due to rounding.

^{1/} References used to develop this table were: Air Resources Board, 1985 and Zwiacher, 1983. Emission factors used were taken from Versar Inc., 1984.

Table I-5

Locations of Secondary Manufacturing
Facilities in California

Product	Los Angeles County	Orange County	Other ^{1/} South Coast Counties	Contra Costa County	Alameda County	Total
Friction Products	20	2	1		2	25
Packings and Gaskets	6	3	1	2	1	13
Coatings, Adhesives and Sealants			1			1
Paper	3	2				5
Plastics		1				1
Textiles	2					2
Chlorine Manufacturing	—	—	—	<u>1</u>	—	<u>1</u>
Total	31	8	3	3	3	48

^{1/} Riverside County and San Bernardino County

asbestos emissions from these secondary facilities is estimated to be about 2 tons per year. Table I-4 presents a summary of asbestos emissions by product category from secondary manufacturing and Table I-5 shows where the facilities are located.

As in primary manufacturing, baghouses are the major emission control device used by secondary manufacturers.

3. Automobile Brake Emissions

The major constituent of automobile brake linings is chrysotile asbestos which makes up about 35-60 percent of the brake liner. The rest of the ingredients are binders which are phenolic type resins and modifiers such as carbon black, graphite, aluminum and silica (Meylan, et al., 1978).

Braking causes the brake lining to wear away and creates dust particles. These particles are first entrained around the brakes, but eventually settle onto the roadway or become airborne. Although the brake material contains about 50 percent asbestos, the emitted particles contain only about 0.029 percent asbestos. Apparently, most of the original asbestos fibers are broken down into nonfibrous materials (magnesium silicates) during the braking process (Lynch, 1968; Rowson, 1978; Williams and Muhlbaier, 1982).

On a statewide basis, emissions from automobile brakes are estimated to be a minor fraction (less than 1 ton/year) of the asbestos emissions. This emission estimate is based on an emission factor of 2.6 ug/km/vehicle (California Air Resources Board, 1984; Williams and Muhlbaier, 1982). However, in high braking areas such as toll booths, some researchers reported a noticeable increase in asbestos levels. Air concentrations of asbestos in an area adjacent to a toll booth has been reported to be 3-5 times higher than areas where little braking is expected to occur.

Other possible sources of asbestos emissions related to brake linings occur during the maintenance and repair of the liner. There are no available estimates for emissions from these activities. However, the concentration of asbestos may be significant due to the cloud of dust that is generated in the work area when loose dust is blown from the brake drums and back plates with compressed air (Meylan, et al., 1978).

4. Other Sources of Emissions

Other sources of asbestos emissions include 1) emissions related to rock quarrying operations in serpentine deposits and 2) natural weathering or other types of human disturbance of serpentine deposits (e.g., off-road vehicle use, road building). Eight rock quarries in the state are located in serpentine deposits and operate on a continuous basis. The chrysotile concentration in these quarries ranges from trace amounts to 20.0 percent by weight (Air and Industrial Hygiene Laboratory, 1981; U.S. Environmental Protection Agency, 1981). The number of quarries by county as identified by EPA are as follows: Santa Clara (2), Alameda (1), Marin (1), Tuolumne (3), and Shasta (1). Two of the quarries in Tuolumne County (Six Bits Quarry and Woods Creek Quarry) produce crushed gravel which is used for surfacing unpaved roads. Using the emission factor of 0.012 pound per ton of crushed stone produced (Blackwood, Chalekode and Wachter, 1978), the respirable asbestos emissions from the quarrying of serpentine was estimated to be 0.5 tons per year.

According to an EPA report, there are only 5 miles of county roads surfaced with serpentine material in the state. The ARB staff believes there may be a higher number of roads in California surfaced with serpentine material. Information from the Siskiyou County Air Pollution Control District indicated that a temporary rock quarry in Siskiyou County supplied serpentine

rock for use by the U. S. Forest Service in the building of roads in the Shasta-Trinity National Forest. Tests by the National Institute for Occupational Safety and Health (NIOSH) showed asbestos ranging from 1 to 30 percent by volume in the serpentine rock. The U.S. Forest Service hired Radian Corporation to obtain airborne samples of asbestos near the roadways. The study showed asbestos in the samples ranging from 0.01 to 0.27 fibers per cubic centimeter (see Part C). The ARB staff has not determined how the samples were analyzed and has requested a copy of the Radian study from the U.S. Forest Service. The Radian study points out the need to consider, in future inventories, the fugitive emissions from roads surfaced with serpentine rock. At the present time, emission factors are not available to estimate these emissions.

Some other potential sources of asbestos emissions for which emission factors have not been established are demolition and renovation of buildings, maintenance and installation of insulating materials, landfills and natural sources of asbestos. An EPA report and survey estimated that 95,556 public and private schools and 700,000 commercial, residential apartments, and federal buildings in the United States contain friable asbestos materials (Greenblatt, 1984; Zurer, 1985). If these buildings were demolished or renovated, the potential emissions to the atmosphere can be significant; however, there is no information available to allow one to estimate the emissions from the demolition and renovation of buildings. The ARB staff recognizes that emissions from this source and others for which emission factors have not been established can contribute to ambient levels of asbestos. As new information becomes available, these emissions should be added to the inventory.

It has been reported that about 90 percent of asbestos scrap (baghouse dust and product wastes) are eventually disposed of in landfills (Meylan, et al., 1978). The Department of Health Services considers waste materials containing asbestos as hazardous waste and has issued specific guidelines for asbestos handling and disposal. The Department's guidelines state that the asbestos waste should be wetted and sealed in non-returnable containers or in closed vehicles for handling and transport. For disposal, the guidelines specify that advance arrangements be made with the disposal site operator to assure that the asbestos containing waste is covered quickly with six inches of compacted soil. Landfill operators are also cautioned to take actions that ensure the compacting equipment does not become contaminated with asbestos dust.

Appendix C contains the calculations used for the emission estimates given in this chapter.

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REFERENCES FOR CHAPTER I

Air and Industrial Hygiene Laboratory Reports - Study Identification No. C81-014, 1981.

Archer, S. R. and T. R. Blackwood, 1979. Status Assessment of Toxic Chemicals: Asbestos. Final report prepared by Monsanto Research Corporation for the U.S. Environmental Protection Agency. PB 80-146327, EPA-600/2-79-2100.

Blackwood, T. R., P. K. Chalekode and R. A. Wachter, May 1978. Source Assessment: Crushed Stone. Final report prepared by Monsanto Research Corporation for the U.S. Environmental Protection Agency. EPA-600/2-78-004L.

Burnett, J. L., 1982. Mining Review - California Geology. California Department of Conservation.

California Air Resources Board, 1984. "BURD1984", Computer Printout Dated September 2, 1983, Emission Inventory Branch, Technical Support Division.

California Air Resources Board, 1985. Survey of Asbestos Manufacturers, Stationary Source Division.

California Department of Conservation, September 1963. "California Asbestos Industry." Volume 16, Number 9.

Chronic Hazard Advisory Panel on Asbestos, July 1983. Report to the U.S. Consumer Product Safety Commission.

Greenblatt, J., 1984. Evaluation of the Asbestos-In-Schools Identification and Notification Rule. Final report prepared by Westat, Inc. for the U.S. Environmental Protection Agency. EPA 560/5-84-005.

Harwood, C. F. and T. P. Blaszk, 1974. Characterization and Control of Asbestos, Emissions from Open Sources. Prepared by IIT Research Institute for the U.S. Environmental Protection Agency. EPA-650/2-74-090.

Harwood, C. F., P. Siebert, and T. P. Blaszk, 1974. Assessment of Particle Control Technology for Enclosed Asbestos Sources. Prepared by IIT Research Institute for the U.S. Environmental Protection Agency. Contract No. 68-02-1353, EPA-650/2-74-088. NTIS PB 239-926.

Lynch, J. R., 1968. "Brake Lining Decomposition Products." Journal of the Air Pollution Control Association. 18(12)524-526.

Meylan, W. M., et al., 1978. Chemical Market Input/Output Analysis of Selected Chemical Substances to Assess Sources of Environmental Contamination III - Asbestos. EPA 560/6-78-005, NTIS PB 285-531.

Rajhans, G. S., and G. M. Bragg, 1978. Engineering Aspects of Asbestos Dust Control.

Roberts, R. M., 1980. An Inventory of Carcinogenic Substances Released into the Ambient Air of California, Final Report Task II and IV, KVB, Inc. for Science Applications Inc.

Rowson, D. M., 1978. = "The Chrysotile Content of the Wear Debris of Brake Linings." Wear. 47:315-321.

U.S. Department of the Interior, 1980. Asbestos: A Chapter from Mineral Facts and Problems. 1980 Edition, Bureau of Mines.

U.S. Department of the Interior, 1985. Mineral Commodity Summaries, 1985. Bureau of Mines.

U.S. Department of the Interior, Minerals in the U.S. Economy: Ten-Year Supply-Demand Profiles for Mineral and Fuel Commodities, 1965-1974. Bureau of Mines.

U.S. Department of the Interior, Minerals Yearbooks for 1976, 1978, 1979, 1980, 1983, and 1984. Bureau of Mines.

U.S. Environmental Protection Agency, 1981. Assessment and Control of Chrysotile Asbestos Emissions from Unpaved Roads. EPA 450/3-81-006.

Versar Inc., 1984. Exposure Assessment for Asbestos. Draft final report prepared for U.S. Environmental Protection Agency. EPA contract no. 68-01-6271, Task No. 49.

Williams, R. L. and J. L. Muhlbaier, 1982. "Asbestos Brake Emissions." Environmental Research. 29:79-82.

Zwiacher, W. E., et al., 1983. Emissions of Potentially Toxic Hazardous Air Contaminants in the South Coast Air Basin. South Coast Air Quality Management District, Engineering Division.

Zurer, P.S., 1985, "Asbestos". Chemical and Engineering News. March 4, 1985, pages 28-41.

II. PERSISTENCE IN THE ATMOSPHERE

A. CHEMICAL AND PHYSICAL PROPERTIES

Unlike many substances that are discrete entities definable by a fixed chemical structure, asbestos fibers comprise a group of materials that are less easily defined. They have a broad range of chemical compositions, crystal structures, sizes, shapes, and properties. They have also been described with diverse terminology (Committee on Nonoccupational Health Risks of Asbestiform Fibers, 1984).

The term "asbestos" is not a mineral name, but a commercial-industrial term applied to a group of naturally occurring mineral silicate fibers of the serpentine and amphibole groups. Serpentine minerals are "layered silicates" and only one of these, chrysotile, is an asbestos mineral. The amphibole asbestiform minerals are "chain silicates" and the five varieties of commercial importance are known as actinolite, amosite, anthophyllite, crocidolite and tremolite (Chronic Hazard Advisory Panel on Asbestos, 1983). Table II-1 lists the names of the minerals included in the term asbestos.

The crystalline structure of chrysotile consists of a layer of magnesium oxide-hydroxide octahedra bonded to a layer of silicon dioxide tetrahedra in a somewhat mismatched fashion that produces a curvature in the sheet. The sheet, consequently, tends to roll itself into a hollow tube or possibly a tight spiral with the magnesium hydroxide on the outer surface. This hollow tube constitutes the basic fibril of chrysotile. Fibrils, bonded together, constitute fibers and the fibers in turn may be banded together to build up the macroscopic material. Theoretically, chrysotile can be successively split until the ultimate fibrils are reached, but the ease of fiberization varies with the particular ore (Selikoff and Lee, 1978).

Table II-1
Mineralogy of Commercial Asbestos^{1/}

Commercial Name	Mineral Name	Mineral Group
Chrysotile	Chrysotile	Serpentine
Crocidolite	Riebeckite	Amphibole
Anthophyllite	Anthophyllite	Amphibole
Amosite	Cummingtonite-grunerite ^{2/}	Amphibole
(No common commercial name; sometimes called amosite)	Actinolite-tremolite ^{3/}	Amphibole

^{1/} This table was taken from the report, Asbestiform Fibers - Nonoccupational Health Risks, by the Committee on Nonoccupational Health Risks of Asbestiform Fibers, 1984. Page 27.

^{2/} Hyphenated mineral names, such as cummingtonite-grunerite, represent mineral series. The minerals in the series are structurally identical but can contain variable proportions of two or more different cations in the same structural site. Thus, these mineral series may be regarded as solid solution series. The variable cations in the cummingtonite-grunerite series are magnesium and iron; most minerals in this series have both elements. The end members are identified by the unhyphenated names, e.g., cummingtonite, and grunerite. Sometimes the name of the end member includes the cation, e.g., ferroactinolite.

^{3/} Although asbestiform tremolite and actinolite occur in nature, large commercially mined deposits are rare. However, actinolite asbestos is found as a contaminant of amosite from South Africa, and tremolite asbestos is found as a contaminant of some talc and chrysotile deposits.

In contrast with the chrysotile crystal, amphiboles consist of double chains of linked silicon-oxygen tetrahedra lying parallel to the vertical crystallographic axis and bound laterally by metallic ions. There is no tendency for such layers to roll in tubes. The Si-O bonds along the chain are much stronger than the metallic ion bonds between chains, so that the amphiboles break lengthwise with ease, giving a fibrous appearance (Selikoff and Lee, 1978).

The best known chemical quality of all types of asbestos fibers is their heat resistance. All types of asbestos progressively break down to simpler structures through dehydroxylation or dehydrogenation when heated to temperatures between 400°C and 1000°C. It should be noted that asbestos fibers as such do not have melting points but the decomposition products formed on heating will themselves eventually melt.

The reactivity of asbestos towards acids and alkalines is fairly well known. Strong acids rapidly decompose chrysotile but amphibole fibers show various degrees of resistance against attack by strong acids. Strong alkalines have little influence on all asbestos fibers, in particular on chrysotile which makes the latter fiber a likely reinforcing agent in cement.

Since the biologically relevant physical properties of asbestos are described by the Department of Health Services staff in Part B, only the physical properties of asbestos that resulted in its widespread commercial use will be described here.

1. Tensile Strength

Tensile strength is the most important physical property of asbestos. Asbestos fibers with sufficiently small diameters have great strength and are used in many industrial-commercial products. Maximum tensile strength values

have been obtained for chrysotile and crocidolite of about $60,000 \text{ kg/cm}^2$, however, the tensile strength varies with length and diameter of fibers. These values are of the same order as those given for glass fiber and carbon steel and are somewhat greater than those for cotton and rock wool (Selikoff and Lee, 1978).

2. Surface Area

In industrial terminology, surface area means degree of openness or degree of fiberization. Each process involving asbestos fiber requires a degree of fiberization which, within limits, is critical for its purpose. For example, chrysotile fibers prepared for inclusion in moulded brake linings will have a different degree of fiberization from those prepared for the carding process which brushes, cleans and straightens fibers in the first stage of textile manufacture (Michaels and Chissick, 1979).

3. Thermal Insulative Value

The insulative value of asbestos has in the past been widely utilized. The asbestos fiber itself does not have low thermal conductivity, but when fibers are separated they trap air which has a very low conductivity and so provide insulation that, for heat flows in a constant direction, is comparable to that provided by similar materials made from other fibers. For heat loads that take place in alternate directions, such as on a roof exposed to the sun, the moderately high density of the material combines with the low thermal conductivity to give one of the lowest values for thermal diffusivity, the governing physical attribute under these conditions (Selikoff and Lee, 1978).

Table II-2 shows chemical and physical characteristics of the main types of asbestos. Note that the chemical composition of the amphiboles are

Table 11-2
CHARACTERISTICS OF THE MAIN TYPES OF ASBESTOS FIBERS^{1/}

Characteristic	Chrysotile	Crocidolite	Amosite	Anthophyllite	Tremolite	Actinolite
Theoretical formula	$Mg_3[Si_4O_{10}(OH)_2]$	$Na_2Fe_{11}Si_8Al_2[Si_{10}O_{22}(OH)_2]$	$(Fe,Mg)_7[Si_8O_{22}(OH)_2]$	$(Mg,Fe)_7[Si_{10}O_{22}(OH)_2]$	$Ca_2Mg_5[Si_8O_{22}(OH)_2]$	$Ca_2(Mg,Fe)_5[Si_{10}O_{22}(OH)_2]$
Chemical Analysis (range of major constituents - percent)						
SiO ₂	38-42	48-56	49-52	53-60	55-60	51-58
Al ₂ O ₃	(0-2) ^a	(0-1)	(0-1)	(0-3)	(0-3)	(0-3)
Fe ₂ O ₃	(0-5)	13-18	(0-5)	(0-5)	(0-5)	(0-5)
FeO	(0-3)	3-21	35-40	3-20	(0-5)	5-15
MgO	38-42	(0-13)	5-7	17-31	20-25	12-20
CaO	(0-2)	(0-2)	(0-2)	(0-3)	10-15	10-13
Mg ₂ O	(0-1)	4-8	(0-1)	(0-1)	(0-2)	(0-2)
H ₂ O ⁺	11.5-13	1.7-2.8	1.0-2.4	1.5-3.0	1.5-2.5	1.8-2.3

^a Bracketed figures denote substituents often present in asbestos.

Color	Usually white to pale green, yellow, pink	Blue	Light gray to pale brown	White to gray pale brown	White to gray	Pale to dark green
Decomposition temperature ^a (°C)	450-700	400-600	600-800	600-850	950-1040	620-960
Fusion temperature of residual material (°C)	1500	1200	1400	1450	1315	1400
Density g/cm ³	2.55	3.3-3.4	3.4-3.5	2.85-3.1	2.9-3.1	3.0-3.2
Resistance to acids	Undergoes fairly rapid attack	Good	Attacked slowly	Very good	Very good	Attacked slowly
Resistance to alkalis	Very good	Good	Good	Very good	Good	Good
Mechanical properties of fiber as taken from rock samples:						
Tensile strength 10 ³ kg/cm ²	31	35	17	(<7)	5	5
(Average)(10 ³ psi)	(440)	(495)	(250)	(<100)	(<70)	(<70)
Young's Modulus 10 ³ kg/cm ²	1,620	1,660	1,620	-	-	-
(Average)(10 ⁶ psi)	(23)	(27)	(23)	-	-	-
Texture	Usually flexible, silky and tough	Flexible to brittle and tough	Usually brittle	Usually brittle	Usually brittle	

^a Dehydroxylation or dehydrogenation accompanied by disruption of crystal lattice and major loss of strength.

^{1/} Table taken from Public Health Risks of Exposure to Asbestos, Commission of the European Communities, Directorate - General for Social Affairs, Health and Safety Directorate, 1977, page 23.

^{2/} From serpentinized dolomite deposits.

similar, therefore, fiber identification during analysis is difficult unless proper laboratory equipment and techniques are used. Some of these equipment and techniques are discussed in Section III-A.

4. Fiber Dimensions in Ambient Air and Occupational Settings

Available data on asbestos fiber dimensions in the ambient air indicate that ambient fibers tend to be smaller than fibers found in occupational environments. For example, the Committee on Nonoccupational Health Risk of Asbestiform Fibers estimated that for equal mass, there would be approximately 35 times more ambient air fibers than occupational air fibers. In the SAI report, the mean diameter and length for each sample analyzed by SAI is given. Table II-3 summarizes the SAI data in terms of average mean values for the samples taken at each location. There was only one location, South Gate, where the average mean length was greater than 5 microns.

Occupational asbestos samples are usually analyzed by phase contrast microscopy (PCM) and counted by a method prescribed by the U.S. Occupational Health and Safety Administration (OSHA). Only fibers greater than 5 microns and having an aspect ratio greater than 3:1 are counted. Therefore, small fibers are not counted. Because of this, it is not possible to characterize occupational samples in terms of mean diameter or length. The OSHA counting method has lead to problems when attempting to compare ambient air concentrations and occupational air concentrations. DHS included a detailed discussion in Appendix A of the Part B report on the ratio of fibers longer than 5 microns to total fibers counted in ambient air samples. DHS concluded that there are between 1 and 10 long fibers per 1,000 total fibers counted in ambient air samples.

Table II-3

Summary of Average Diameters and Lengths of Samples
Collected by SAI During 1981

<u>Sample Site</u>	Chrysotile		Amphiboles	
	<u>Average Mean Diameter (Microns)</u>	<u>Average Mean Length (Microns)</u>	<u>Average Mean Diameter (Microns)</u>	<u>Average Mean Length (Microns)</u>
King City	0.4	4.6	--	--
San Jose	0.14	1.3	0.14	2.0
Napa	0.12	1.1	0.18	1.1
Sonora	0.14	3.0	0.20	1.3
Century City	0.06	0.9	0.30	1.1*
San Fernando Valley	0.05	1.1	0.08	0.6
Bakersfield	0.08	0.7	0.17	2.1
South Gate	0.43	5.1	0.13	1.5
San Diego	0.12	1.2	0.18	1.0
Stockton	0.05	0.71*	0.15	1.4*

(1) For each sample taken by SAI, the mean diameter and length were reported.
These values are the averages of the mean dimension found at each location.

* Only one sample was reported.

B. FATE IN THE ATMOSPHERE

Transformation processes associated with the fate of asbestos in the environment are generally insignificant; the material is relatively inert. Degradation has been observed in the laboratory under extreme conditions of heat, mechanical stress and acidity. These conditions, however, are not normally observed or encountered in the ambient environment (Versar Inc., 1984).

Winds are capable of dispersing asbestos fibers from industrial sources and naturally occurring sources. Once in the atmosphere, the fibers are subject to removal by gravitational settling (dry deposition) and atmospheric washout by precipitation (Versar Inc., 1984) but are also easily reentrained from ground surfaces (U.S. Environmental Protection Agency, 1978). In a study conducted by Harwood and Ase (1977) in the vicinity of asbestos waste piles in Denison, Texas, nearly equal upwind and downwind concentrations of asbestos fibers were theorized to result from upwind resuspension of previously settled fibers.

The deposition and eventual burial of fibers in soils and sediments are the major natural processes by which asbestos leaves the atmosphere. Asbestos fibers could be reentrained if soils or sediments are disturbed by anthropogenic or natural activities.

REFERENCES FOR CHAPTER II

Commission of the European Communities, Directorate - General for Social Affairs, Health and Safety Directorate, 1977. Public Health Risks of Exposure to Asbestos.

Committee on Nonoccupational Health Risks of Asbestiform Fibers, 1984. Asbestiform Fibers - Nonoccupational Health Risks.

Harwood, C. and P. Ase, 1977. The Control of Fugitive Emissions from Asbestos Waste Piles. Draft final report prepared for the U.S. Environmental Protection Agency. EPA Contract No. CA-6-99-3380-A.

Michaels, L. and S. Chissick, Editors, 1979. Asbestos Volume 1 Properties, Applications and Hazards.

Selikoff, Irving J. and Douglas H. K. Lee, 1978. Asbestos and Disease.

Timbrell, V., 1965. The Inhalation of Fibrous Dust. Annals of the New York Academy of Science. 255-273.

U.S. Environmental Protection Agency, April 1978. Dispersion Model Analysis of the Air Quality Impact of Asbestos Emissions From Iron Ore Benefication Plants. EPA Contract No. 68-02-2507.

Versar Inc., 1984. Exposure Assessment for Asbestos. Draft final report prepared for the U.S. Environmental Protection Agency. EPA Contract No. 68-01-6271, Task No. 49.

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III. EXPOSURE TO ASBESTOS

A. Asbestos Measurement Techniques

Ambient asbestos air samples are usually analyzed by either optical (light) microscopy or electron microscopy methods. In some cases, the fibers must have an aspect ratio* of 3:1 or greater to be counted. Each microscopy method has its advantages and limitations and some of these are discussed in this section.

In optical microscopy, there are two methods used to analyze asbestos fibers: phase contrast microscopy (PCM) and polarized light microscopy (PLM). PCM enhances the visibility of fibers under low magnification powers of the phase contrast microscope, but cannot distinguish between asbestos and non-asbestos fibers. In addition, the poor resolution power of the microscope limits the detection of fibers to those larger than 0.3 microns. However, PCM is widely used for asbestos screening and counting since this is the prescribed method by U.S. Occupational Health and Safety Administration (OSHA) for determination of asbestos emissions in occupational settings. This method involves counting fibers with acceptable aspect ratios and greater than 5 microns in length; therefore, the smaller fibers are not counted.

There are two disadvantages with PCM. First, the count includes fibers within a specified size range, whether the fibers are asbestos or not. Second, due to the poor resolution power of the phase contrast microscope, fibers less than 0.3 microns will not be detected (Hayward 1985, Versar Inc., 1984).

*Aspect ratio means that the length of the fiber must be at least 3 times longer than the width of the fiber.

PLM can be used to identify and characterize asbestos and non-asbestos fibers based upon the crystal structure of the fibers. When used with dispersion staining, PLM may be able to distinguish different types of amphiboles within the sample. However, PLM has the same limitation as PCM which is poor resolution powers in detecting the smaller fibers.

To analyze and identify smaller asbestos fibers, electron microscopy (scanning electron microscopy (SEM) and transmission electron microscopy (TEM)) should be utilized. SEM has a resolution limitation in that it can only detect fibers larger than 0.1 microns. With respect to fiber identification, SEM equipped with an energy dispersive X-ray spectroscopic (EDXS) system can identify asbestos fibers by providing elemental analysis of the fibers, but it does not provide structural information to identify specific fiber types.

The preferred analytical method is TEM because of its ability to analyze and detect the smaller asbestos fibers that are found in the ambient air. A transmission electron microscope has the resolution capability of detecting fibers that are 0.2 nanometers in diameter. TEM can be used with selected area electron diffraction (SAED) which provides crystallographic (structural) information of the fibers being analyzed. TEM-SAED can identify chrysotile fibers, although X-ray diffraction analysis can be used for confirmation. To identify amphiboles, TEM-SAED must be supplemented by EDXS analysis in order to obtain elemental information.

Although TEM is the preferred analytical method, there is no standardized procedure for laboratories to use. Due to different instrument capabilities, operator proficiencies, and procedures, it is difficult to compare the results from various laboratories. In recognition of this problem, the EPA evaluated

the TEM/SAED method and procedures of different laboratories and developed a TEM/SAED provisional methodology manual to minimize the variability of results. The provisional methodology is found in Electron Microscope Measurement of Airborne Asbestos Concentrations, A provisional Methodology Manual by A. V. Samuda, Colin F. Harwood, and John D. Stockman which was published in June 1978. At the present time, EPA is revising the manual, but the changes have not been finalized.

B. ASBESTOS MEASUREMENTS

Several documents containing asbestos measurements taken in California were reviewed to show asbestos exposure to the general population (Appendix D). Only the report entitled Ambient Asbestos Concentrations in California: Volumes I and II, prepared by Science Applications, Incorporated (SAI) under contract to the Air Resources Board, contained asbestos measurements from several populated locations in the state. The samples were prepared and analyzed using EPA's provisional TEM methodology with only a minor modification which consisted of using a smaller pore size filter for better collection of smaller fibers.

With one exception, the remaining studies were older, had used different sample collection methods, sample preparation techniques and analytical techniques and were concerned with specific sources such as freeways, an asbestos mill, and off-road vehicles. Since different sample collection and analytical techniques were used for these studies, the results are not directly comparable. However, the measurements are useful for documenting that asbestos is present in the ambient air. Appendix E contains a table listing some of these measurements. In Section D of this chapter, a discussion of ambient asbestos monitoring data collected in Alviso, California

is presented. This area is contaminated with asbestos fibers as a result of disposal of asbestos waste in a nearby landfill.

We believe the asbestos data contained in the SAI report represent the most recent and suitable data that are available for evaluating ambient asbestos concentrations in California. Therefore, we are limiting our discussion to this study. (Science Applications, Inc., 1983). Samples were collected and analyzed by SAI at ten sites located throughout California. As with most asbestos data, samples represent very short averaging times and were collected over a very limited time period. Although ambient levels determined over the short-term are discussed, no method has been developed to extrapolate from short-term averages to long-term averages. Because of the nature of the measurements and the unknown variability of concentrations over time, annual average concentrations and associated population exposures cannot be determined for asbestos.

The samples were collected using a single-point filter sampler with a cyclone inlet providing a collection efficiency of 50 percent for 3.5 microns (μm) aerodynamic diameter particles. This particular sampler was chosen because of its ease of use in the field, ability to selectively sample particles in the respirable range, and a design which allows for uniform particle deposition on the filter. After the collection phase, samples were taken to the laboratory for analysis. All sample analyses were completed using a transmission electron microscope (TEM) with a resolution of six angstroms and equipped with selected-area electron diffraction (SAED) instrumentation. Chrysotile asbestos fibers can be identified using the SAED

technique, however, amphibole asbestos fibers cannot be differentiated. Appendix F presents more detailed sampling, sample preparation, analysis and quality control information.

In addition to measurement of asbestos concentrations, monitoring of meteorological conditions and of temporal variations in particulate matter concentrations were conducted at each site. The overall accuracy and precision of the data collected by SAI are not documented in the final report. The reason for this is that the actual number of samples collected and analyzed were too limited to statistically validate the precision and accuracy of the numbers reported for each site. However, it is now known that the high flow rate used for sampling in this study can result in significant fiber loss (20-50% of the total) probably as a result of electrostatic charging of the plastic filter holder (Hayward, June 1985).

Asbestos sampling sites were chosen based on emission and exposure potential. The project's goal was to measure airborne asbestos in areas known or suspected to have elevated levels as well as in areas isolated from asbestos sources. Six factors related to source type and potential population exposure guided site selection:

1. Localized Sources: Locations at or near which asbestos ore is processed, refined, or otherwise used, and at which asbestos can potentially be released into the air at elevated concentrations;
2. Natural Deposits: Areas where soil provides a source of asbestos fibers;
3. Non-Urban Locations: Rural areas with low population and which are located away from metropolitan asbestos emissions and natural deposits;

4. Urban Locations: Areas of potentially high population exposure to elevated particulate matter;
5. Industrial Sources: Metropolitan areas with high density clusters of asbestos users, as identified in the Environmental Protection Agency's register of sources regulated under the National Emission Standards for Hazardous Air Pollutants; and
6. Vehicular Braking: Areas where asbestos emissions from automobile brake and clutch friction materials are expected to be high.

Ten sites were selected for study. The site names, classification characteristics and population within a five kilometer and ten kilometer grid area of each are listed in Table III-1. The relative locations of the sites throughout the state are shown in Figure III-1. Two rural/suburban sources, a mill near King City and the asbestos cement pipe manufacturing plant in Stockton, were chosen to represent the localized source category. The mill is an asbestos processing plant and is the only asbestos source in this study located in a rural area; chrysotile asbestos ore is both refined and packaged at the King City mill. The asbestos pipe manufacturing plant is located at the border of the Stockton urban area; no other asbestos sources were identified in the vicinity of this plant.

Sonora in Tuolumne County was selected as a natural source site. Sonora is the only one of three serpentine-rich deposit areas within the state that does not have active mining nearby. Population surrounding the Sonora site is low, with a seasonal (summer and fall) influx of tourists.

Table III-1
Names, Classification Categories, and Population Near^{1/}
the Ten Sites Chosen for the SAI Asbestos Sampling Study

Site Name	Classification Category	Population at 5 Kilometers	Population at 10 Kilometers
King City	Localized Source Location	8,200	8,200
Stockton	Localized Source Location	15,000	34,000
Sonora	Natural Deposit Location	7,700	19,000
Napa	Non-Urban Background Location	48,000	66,000
Oildale	Non-Urban Background Location	4,300	23,000
San Diego	Urban Background Location	10,000	100,000
Sherman Oaks	Urban Background Location	6,400	120,000
San Jose	Industrial Source Location	180,000	620,000
South Gate	Industrial Source Location	150,000	680,000
Century City	Vehicular Braking Location	130,000	460,000

^{1/} Population estimates were made by summing the population of census tract centroids located within a 5 kilometer and 10 kilometer radius of each monitoring site.

Figure III-1

**California Map Showing Locations
of the
Ten Sampling Sites Used
in the
SAI Study**



SAI chose two non-urban background sites at Napa and Oildale (near Bakersfield) as being located in non-serpentine areas. These two sites represent differing climatological settings, especially with respect to dominant wind patterns. However, Napa County does have natural serpentine deposits (chrysotile) throughout the county as shown in Figure I-1. The Oildale site had the second highest concentration measured in the study. SAI was not able to explain this value. The ARB staff believes that it is important to note the presence of asbestos fibers in an area that was not suppose to have identifiable asbestos sources.

Sites in San Diego and Sherman Oaks (San Fernando Valley) were selected as urban background exposure sites. The sites were chosen to reflect dissimilar climatological conditions and are located away from both serpentine deposits and heavy automobile traffic. There were fewer than three sources using asbestos in each of the two communities.

San Jose and South Gate (Los Angeles County) were chosen to represent industrial source sites. Eighty-six asbestos users were identified in the San Jose area in 1981. Also in 1981, 147 users were identified in the Los Angeles area; the South Gate location represents a dense cluster of these asbestos users.

One of the major historical uses of asbestos has been as frictional material in vehicle braking and clutch facings. Therefore, Century City was chosen as a location representative of emissions from vehicular braking. In order to duplicate meteorological conditions at the Century City site and also quantify the affect of varying traffic volumes, samples were collected on an adjacent Sunday (low traffic volume) and Monday (high traffic volume).

Traffic counts between the two days were anticipated to vary by at least an order of magnitude.

C. AMBIENT ASBESTOS CONCENTRATIONS

All asbestos samples referenced in this discussion were collected during 1981. Samples were collected at each site during a single day except at Century City, where samples were collected on two consecutive days (Sunday and Monday) in an attempt to quantify the effect of traffic related sources on ambient asbestos concentrations. Two asbestos samplers were operated at each site location. Original sampling plans called for collection of at least four four-hour samples at each site between the approximate hours of eight o'clock in the morning and four o'clock in the afternoon. Because of ordinarily high particle concentrations, and therefore possibly high asbestos levels, samples at several locations were collected over shorter time intervals to preclude overloading the filters.

Short-term (single sample) results are summarized in Table III-2. Information given for each individual sample includes date of sample, sampling time, sampling duration, and concentrations of chrysotile and of amphibole asbestos(separately and combined) in fibers per cubic meter of air. Only valid sampling data are included in the summary. Concentrations listed include values below as well as concentrations at and above the analytical detection limit. The analytical detection limit is a function of the total area of collection filter scanned during analysis and also of the volume of air sampled. At the time of sample analysis, only those fibers with a length to diameter ratio of at least three to one were counted. When only one chrysotile or amphibole fiber was counted on the sample scanned, the concentration value based on that single fiber was used to define the

detection limit for the specific fiber type. Samples that were analyzed and not having any fibers counted during analysis were classified by SAI as being below the detection limit (DL). Nearly all fibers were less than five microns in length. Three samples contained asbestos fibers greater than five microns in length and only one sample had a mean fiber diameter of one and a half microns. The remaining samples had mean diameters less than one and a half microns.

Table III-2 suggests that asbestos sampling results from the ten sites are variable. In addition, individual samples collected at each of the sites are also highly variable. Overall, fiber-specific and combined concentrations range from levels below the analytical detection limit to a maximum concentration of 140,000 fibers per cubic meter (f/m^3). In addition, several samples collected at the same location had concentrations ranging from below the detection limit to quantifiable concentrations. SAI did not have an explanation for this, but they believe that meteorological conditions and humidity can influence the sampling results.

The highest asbestos concentration measured was near the mill at King City. The site represents a source-specific area in that it is located in a rural area with high levels of asbestos emitted from an isolated industrial source. All fibers sampled at the King City mill were chrysotile asbestos; this is not unexpected since the milling operation processes chrysotile ore. The maximum concentration, 140,000 f/m^3 , was measured in the early morning under reduced airflow conditions. This was intended to represent an upwind sample from a localized source. However, the data did not indicate this.

Although it is generally thought that wind conditions determine the amount of asbestos which can be picked up into the air, concentrations measured at this site indicate higher levels under reduced air flow conditions than under

Table III-2
Summary of Respirable Asbestos Samples Collected by SAI During 1981

Sample Site	Sampling Date	Sampling Midpoint	Sampling Time (min)	Chrysotile ^{1/} (f/m ³)	Amphibole ^{2/} (f/m ³)	Total Asbestos ^{3/}
King City (Local Source)	09/21/81	09:46	239	140,000	< DL	140,000
		11:07	240	9,400	< DL	9,400
		14:03	240	4,700	< DL	4,700
		15:45	239	2,400*	< DL	2,400
Stockton (Local Source)	07/23/84	10:25	227	< DL	2,500	2,500
		11:43	214	18,000	3,700*	21,700
Sonora (Natural Deposits)	09/24/81	10:05	240	12,000	2,400*	14,400
		10:40	247	6,900	2,300	9,200
		14:40	240	2,400*	2,400*	4,800
		14:39	239	< DL	< DL	< DL
Napa (Non-Urban)	09/23/81	09:48	240	4,700	< DL	4,700
		10:06	240	< DL	4,700	4,700
		14:01	240	9,400	< DL	9,400
		14:20	239	4,700	7,100	11,800
Oildale (Non-Urban)	10/21/81	10:10	177	< DL	< DL	< DL
		10:40	179	6,300	9,500	15,800
		13:30	182	3,100*	< DL	3,100
		14:35	169	52,000	52,000	104,000
San Diego (Urban)	12/17/81	09:40	195	< DL	2,900*	2,900
		09:38	190	3,000*	8,900	11,900
		13:05	210	2,700*	2,700*	5,400
		13:28	248	4,500	23,000	27,500
		15:30	69	< DL	16,000	16,000
Sherman Oaks (Urban)	10/20/81	10:05	241	< DL	< DL	< DL
		10:17	241	16,000	< DL	16,000
		13:37	119	14,000	4,800*	18,800
		14:30	180	28,000	< DL	28,000
San Jose (Industrial)	09/22/81	10:15	240	21,000	2,900	23,900
		10:31	241	14,000	9,400*	23,400
		14:30	240	9,400	2,400*	11,800
		14:43	240	14,000	7,100	21,100
South Gate (Industrial)	10/22/81	09:30	54	< DL	11,000*	11,000
		09:50	99	18,000	18,000	36,000
		12:04	59	9,600*	58,000	67,600
		14:25	59	56,000	< DL	56,000
Century City (Vehicle Braking) (Sunday)	10/18/81	09:10	158	< DL	< DL	< DL
		09:20	137	4,100*	< DL	4,100
		13:45	204	15,000	< DL	15,000
		14:05	86	46,000	20,000	66,000
Century City (Vehicle Braking) (Monday)	10/19/81	07:30	61	56,000	< DL	56,000
		09:10	69	< DL	< DL	< DL
		14:43	164	< DL	< DL	< DL
		15:30	60	28,000	9,400	37,400

* Indicates asbestos fiber concentration analyzed equals the detection limit for the sampling period.

1/ 11 out of 43 (26%) were below the detection limit.

2/ 19 out of 43 (44%) were below the detection limit.

3/ 6 out of 43 (14%) were below the detection limit.

higher wind conditions. Later in the afternoon, concentrations were much lower. Results of sampling at this site emphasize the importance of changing meteorological conditions and temporal variability on ambient asbestos concentrations.

Asbestos concentrations measured at the other two source-specific locations, Stockton (point source) and Sonora (natural source), are low in comparison to the King City site. The high concentration at King City is approximately six to ten times greater than the highest total asbestos level measured at the other two sites.

Concentrations measured at the Sonora site were all relatively low. During the day on which samples were collected, there was no measurable wind and visibility was clear. These types of meteorological conditions are atypical for the site, and measured asbestos concentrations were lower than expected. Consequently, it is difficult to draw any conclusions from the data and may be misleading to compare the Sonora results with those from other sampling locations. As was true of the King City location, samples collected at both the Stockton and Sonora sites indicate the importance of meteorological influences on resulting levels of airborne asbestos.

The remaining sites considered in the SAI study represent a variety of conditions statewide, designed to identify more normal ambient conditions than those influenced by specific sources. Included among the other sites were background sites which are located away from natural asbestos deposits, industrial sites located among a number of commercial asbestos users, and a site designed to identify the potential contribution of vehicle braking in an urban area.

Other sites chosen by SAI were located at Napa, Oildale, San Diego, and Sherman Oaks. A single observation of $104,000 \text{ f/m}^3$ was measured at Oildale in the afternoon. This is the second highest concentration measured at any of the ten sites. SAI was unable to explain this high value. Other concentrations measured at these locations range from levels below the detection limit to a concentration of $28,000 \text{ f/m}^3$. Eight of the seventeen observations are less than $10,000 \text{ f/m}^3$.

Total asbestos concentrations measured at the two industrial locations, San Jose and South Gate, are all above the analytical detection limit suggesting a more constant presence of airborne asbestos at these sites. Concentrations range from $11,000 \text{ f/m}^3$ to $67,000 \text{ f/m}^3$ with the majority of values above $20,000 \text{ f/m}^3$. Asbestos levels measured at the industrial sites are less variable than those observed at background and localized source sites.

Asbestos samples collected at the Century City location span a two day period. Samples collected on Sunday represent light traffic conditions while Monday samples represent heavy traffic conditions. Values for the two days range from below the detection limit on both days to maximums of $66,000 \text{ f/m}^3$ and $56,000 \text{ f/m}^3$ on Sunday and Monday, respectively. Comparison of the high value measured on each of the two days does not show any significant difference related to light traffic versus heavy traffic conditions. Of the total samples taken at Century City, only the two samples taken on Sunday and Monday, early in the morning, show a significant difference. The chrysotile asbestos level was higher by a factor of ten on Monday. SAI staff states in their report that conclusions from these data should be cautiously drawn because the sample with the higher level could be a result of a series of emergency brakings at some point during the sampling.

Averages of all concentrations measured at each of the sampling sites are summarized in Table III-3. In calculating these averages, concentrations reported as below the detection limit were assigned a value of one-half the detection limit as determined using Figure III-2.* For example, a sample collected over four hours and analyzed as below the detection limit was assigned a concentration value of $(0.5 \times 2,400 \text{ f/m}^3)$ or $1,200 \text{ f/m}^3$ for inclusion in the averaging calculation. The averaging process tends to smooth out the variability present among individual observations due to meteorological and source related parameters and gives a more representative indication of daily average concentrations and potential differences among the sites. As noted, individual concentration measurements include levels below the analytical detection limit as well as values at and above this limit. In the previous discussion, asbestos concentrations below the detection limit were so identified; in the following discussion of site averages, these values are treated differently.

All samples analyzed in the SAI study were collected at the same flow rate (15.5 liters of air per minute) and counted in an identical manner. Therefore, the analytical detection limit is a function only of sampling time. A graph of detection limit versus sampling time is shown in Figure III-2. Since sampling times in the SAI study ranged from one hour to four hours, net detection limits range from $9,100 \text{ f/m}^3$ to $2,400 \text{ f/m}^3$, respectively. Concentrations presented in the SAI study are reported as below

*This averaging method was used to strike a balance between other averaging methods that provide estimates lower than actual averages, if the non-detectable values are assumed equal to zero, or estimates higher than the actual average if the non-detectable values are assumed to equal the detection limit.

Table III-3

Summary of Averaged Respirable Asbestos Concentrations
 = Sampled by SAI During 1981

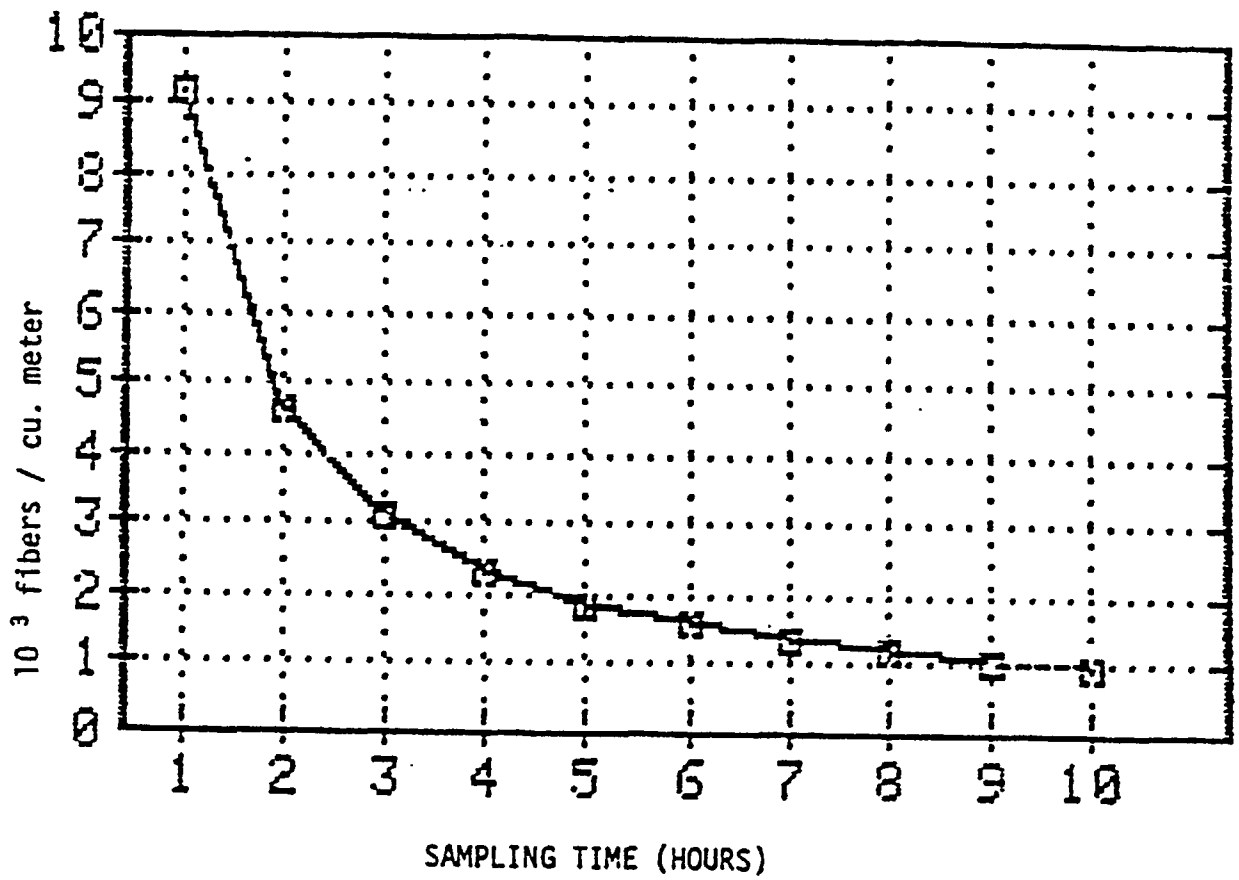
Site Name	Total # Samples	Chrysotile			Amphibole			Total*
		Samples DL	Peak (f/m ³)	Mean (f/m ³)	Samples DL	Peak (f/m ³)	Mean (f/m ³)	Mean (f/m ³)
King City	4	0	140,000	39,000	4	1,200	1,200**	40,000
Stockton	2	1	18,000	9,600	0	3,700	3,100	13,000
Sonora	4	1	12,000	5,600	1	2,400	2,000	7,600
Napa	4	1	9,400	5,000	2	7,100	3,500	8,500
Oildale	4	1	52,000	16,000	2	52,000	16,000	32,000
San Diego	5	2	4,500	3,200	0	23,000	11,000	14,000
Sherman Oaks	4	1	28,000	15,000	3	4,800	2,200	17,000
San Jose	4	0	21,000	15,000	0	9,400	5,500	21,000
South Gate	4	1	56,000	22,000	1	58,000	23,000	45,000
Century City								
Sunday	4	1	46,000	17,000	3	20,000	6,300	23,000
Monday	4	2	56,000	22,000	3	9,400	5,000	27,000

* The total mean concentrations may not total due to rounding.

**For consistency, the averaging method was used for all locations. We would not expect to find amphiboles at this location because the mill processes chrysotile ore.

Figure III-2

RELATIONSHIP BETWEEN DETECTION LIMIT AND SAMPLING TIME
USED IN SAI ASBESTOS STUDY



the detection limit if less than one fiber was counted on the portion of filter scanned. Simply because no fibers were counted, one cannot eliminate the sample from consideration or assume the asbestos concentration present in the sample is zero. Some asbestos could be present, but were not analyzed.

Average asbestos concentrations for the ten sites sampled range from 7,700 f/m³ at Sonora to 45,000 f/m³ at South Gate. Comparison of the average values in Table III-3 is similar to the comparison of individual values presented in Table III-2. South Gate shows the highest average concentration while King City had the second highest average concentration. Total asbestos concentrations tend to be higher at the industrial and localized sources than the other sampling sites.

In the discussion above, we have presented an analysis of the asbestos data by assigning a value of one-half the detection limit for each sample result that was reported below the detection limit. For comparative purposes, in Table III-4, we present mean asbestos concentrations for each sampling site that were calculated using three methods. The three methods are: 1) assuming non-detectable values are equal to zero, 2) assuming non-detectable values equal one-half the detection limit, and 3) assuming non-detectable values equal the detection limit.

Results of the SAI sampling study indicate that measurable airborne asbestos concentrations are present at a number of locations throughout the state. Quantifiable single sample concentrations vary by a factor of nearly sixty, ranging from 2,400 f/m³ to 140,000 f/m³ total asbestos. The variation of mean concentrations from site to site is nearly a factor of six (7,700 f/m³ to 45,000 f/m³ total asbestos). Generally, the highest concentrations were measured at sites influenced by localized and

Table III-4
Mean Respirable Asbestos Concentrations Calculated By
Three Averaging Methods
(Fibers/m)^a

Site Name	Chrysotile		Amphibole		Chrysotile & Amphibole		
	Assumed D.L. ^{1/}	Non-detectable Value	Assumed D.L.	Non-detectable Value	Assumed D.L.	Non-detectable Value	D.L.
	0	D.L.	0	D.L.	0	D.L.	D.L.
King City	39,000	39,000	1,200	0	40,000	39,000	42,000
Stockton	9,600	10,000	3,100	3,100	13,000	12,000	13,000
Sonora	5,600	5,900	2,100	1,800	7,700	7,100	8,300
Napa	5,000	5,300	3,600	3,000	8,600	7,700	9,500
Oildale	16,000	16,000	16,000	15,000	32,000	31,000	33,000
San Diego	3,200	4,300	11,000	11,000	14,000	13,000	15,000
Sherman Oaks	15,000	15,000	2,200	1,200	17,000	16,000	18,000
San Jose	15,000	15,000	5,500	5,500	20,000	20,000	20,000
South Gate	22,000	24,000	23,000	22,000	45,000	43,000	48,000
Century City							
Sunday	17,000	17,000	6,300	5,000	23,000	21,000	25,000
Monday	22,000	24,000	5,000	2,400	27,000	23,000	32,000

^a The respirable asbestos concentrations may not total due to rounding.

^{1/} D.L. means detection limit.

industrialized sources; the lowest concentrations were found at sites isolated from asbestos emission sources.

Although SAI's measured asbestos concentrations suggest these conclusions, individual measurements represent a very short averaging time, one hour to four hours. In addition, comparison of asbestos data with simultaneously collected meteorological data indicate ambient asbestos concentrations are greatly influenced by changes in parameters such as wind and humidity. The effect of changing meteorological conditions on resulting average asbestos concentrations over a longer timeframe cannot be extrapolated from the one day samples that are available.

In conjunction with asbestos sampling, SAI also monitored site-specific particle concentrations. The intent of these measurements was to determine whether asbestos and particle levels are related and consequently, if particle measurements could be used to predict ambient asbestos concentrations. SAI found no relationship between measured asbestos levels and concentrations of total suspended, inhalable, or fine particulate matter.

The ability to determine annual average concentrations is essential to the evaluation of population exposures and the associated risk of ambient levels of potentially toxic compounds. At the present time, however, no long-term asbestos sampling data are available and no method has been developed to extrapolate long-term average concentrations from limited short-term observations. Consequently, no estimate of annual average concentrations and associated population exposures can be made. However, in some sampling locations, a significant population resides in the area. In Table III-1, we presented the population within five and ten kilometer areas for each of the sampling locations.

D. SOURCE OF ASBESTOS CONTAMINATION

The DHS staff has been investigating an asbestos contamination problem in Alviso, California which is a small community located near San Jose (see Appendix G). The population in a 5 kilometer area surrounding central Alviso is approximately 31,000. Population located within a 10 kilometer radius of Alviso is approximately 270,000.

During the 1950's and 1960's, waste from an asbestos cement pipe manufacturing plant was disposed of in a landfill near Alviso. Subsequent flooding of the landfill at various times in the past 30 years have caused contamination of the soil in Alviso. Ambient air monitoring studies by DHS have indicated significant concentrations of asbestos fibers in the air throughout Alviso when compared to upwind concentrations (910,000 total fibers/m³ versus 12,000 total fibers/m³). This area has been designated as a federal and state superfund clean-up site.

E. EXPOSURE THROUGH OTHER MEDIA

Although the primary focus of this report is exposure to asbestos from the ambient air, asbestos can be taken into the body by either inhalation or ingestion of asbestos in other media. This section summarizes available information on exposure through other media. Table III-5 expresses asbestos exposure from various media in terms of the intake of fibers per year for an office person, house person, and child (see Appendix H for details). The relative amount of time a person spends in each exposure environment has a major impact on the levels of asbestos fibers taken into the body. For example, although there was no significant difference in the asbestos concentrations found in the home and the ambient air, the difference in yearly intake of asbestos fibers into the body from these two environments was significant. (see Table III-5)

Table III-5

Yearly Intake of Asbestos Fibers From Various Media For
An Office Worker, House Person, and Child

<u>Exposure Route</u>	<u>Child (10⁶ fibers per year)</u>	<u>House Person (10⁶ fibers per year)</u>	<u>Office Worker (10⁶ fibers per year)</u>
<u>Inhalation</u>			
Ambient Air	20-100	6-30	6-30
Indoor-Schools with asbestos	50-4,000	0	0
Indoor-Office buildings with asbestos	0	10-200*	60-1,500
Indoor-Homes with asbestos	1-200	1-300	1-200
Total**	70-4,300	20-500	70-1,700
<u>Ingestion</u>			
Treated Water System	400-90,000,000	500-140,000,000	500-140,000,000

* Accounts for time in stores, banks, and other similar types of buildings.

** Intake levels have been rounded off.

Note: The estimates presented in Table III-5 are based on limited data and should not be considered absolute. This data was tabulated to illustrate the combined effect of assumed exposure times and concentrations on the intake of asbestos fibers.

Indoor concentrations in office buildings and schools can exceed ambient air concentrations when asbestos has been used in buildings for specific purposes such as insulation and as a fire retardant on structural components. The asbestos intake levels in Table III-5 for exposures in buildings (home, office buildings, and schools) include estimates for buildings with asbestos present in the surrounding materials. The upper levels, therefore, may not be representative of the concentrations one would find in an "average" home, school, or office building but for the group of individuals affected, the intake levels can be significantly higher than those from ambient air.

Although intake of asbestos from drinking water can be thousands of times the intake from inhalation, DHS has concluded that experimental studies were not conclusive in determining the relationship of ingestion and various types of cancer. Therefore, the relative intakes of asbestos fibers for inhalation and ingestion should not be compared for risk assessment purposes.

1. Drinking Water Exposure

According to work done by Dr. Steven Hayward of the California Department of Health Services (Hayward, 1984), levels of asbestos in California drinking water can vary from one to 260,000 MFL (million fibers per liter) observed in a storage tank in the Klamath River Basin. In general, higher levels were found after storms occurred in areas with asbestos-containing serpentine rock formations. In the American River at Sacramento, from which drinking water is drawn, concentrations varied from 53 to 5600 MFL, depending upon season and recent storm history. In the California Aqueduct, levels of 300 MFL have been found at the head, compared to 15,000 MFL found just south of Coalinga. These

concentrations can be lowered through water treatment. However, a report done by EPA's Health Effects Research Laboratory in 1979, "Exposure to Asbestos from Drinking Water in the United States" (600/1-79-028), states that in the San Francisco Bay area, concentrations greater than 100 MFL have been found in treated water systems. In San Francisco's Crystal Springs Reservoir, a concentration 130 MFL has been found. These concentrations have been attributed to erosion of naturally occurring serpentine deposits. In contrast, Los Angeles water was found to contain less than 1 MFL. Another source of asbestos in drinking water is from asbestos cement pipe; however, there is insufficient information to estimate the amount of asbestos that is contributed by the asbestos cement pipe. Table III-6 summarizes the asbestos concentrations found in California water systems.

2. Indoor Exposure

Table III-7 shows a summary of indoor asbestos exposure samples collected in public and private schools and federal buildings. According to the report, "Indoor Pollutants", 1981, by the National Research Council's Committee on Indoor Pollutants, friable sprayed asbestos insulation on structural surfaces is the major source of airborne asbestos. Sprayed material has been applied for building insulation and fire proofing. The EPA has now banned spray-on application of asbestos-containing materials, except where the fibers are encapsulated with a binder during spraying and are not friable after drying.

Some estimates are that over half the buildings in the United States contain asbestos in a form that could give rise to indoor airborne exposures. In these buildings, the sprayed asbestos material is friable and susceptible to damage and disintegration by hand pressure. Most contamination from these sources is dependent on human activity, by contact disruption, and by re-entrainment of settled fibers. In addition, airborne exposure may result

Table III-6
Asbestos Concentrations Found In California Water

<u>Treated Water</u>	<u>Asbestos Concentrations</u> <u>(106 fibers/liter)</u>
Los Angeles	1
San Francisco	100-130
Klamath River	260,000
 <u>Untreated Water</u>	
American River	53-5,600
California Aqueduct	300-15,000

Table III-7
Summary of Indoor Asbestos Samples^{1/}

Sample Set	No. of Samples	Measured Concentration (ng/m ³)		Equivalent Concentration (fibers/m ³) ^{2/ 3/}	
		Median	90th Percentile	Median	90th Percentile
Air in U.S. school-rooms without asbestos	31	16.3	72.7	540	2,420
Air in U.S. buildings with cementitious asbestos	28	7.9	19.1	260	640
Air in U.S. buildings with friable asbestos	54	19.2	96.2	640	3,210
Air in U.S. school-rooms with asbestos surfaces	54	62.5	550	2,080	18,330
Air in U.S. schools with damaged asbestos surfacing materials	27	121.5	465	4,050	15,500

1/ Table adapted from Committee on Nonoccupational Health Risks of Asbestiform Fibers, 1984. Page 220.

2/ Based on a conversion factor of 30 ug/m³ = 1 fiber/cm³.

3/ Equivalent phase contrast microscopy measurement.

from breakdown of a hard asbestos surface such as vinyl-asbestos floor tiles (Sebastien, et al., 1982).

Dr. Steven Hayward also provided the information regarding indoor asbestos exposures at four locations in the San Francisco Bay area (Hayward, May 1985). Measurements were provided in fibers per cubic meter and in nanograms per cubic meter, as analyzed by transmission electron microscopy. Table III-8 shows the results of the indoor measurements taken at four locations.

3. Other Nonoccupational Exposure Routes

In 1977 the Consumer Product Safety Commission banned production of patching compounds and artificial fire logs that contained asbestos. In a series of regulations issued between 1972 and 1975, the Food and Drug Administration banned the interstate commerce of asbestos-containing garments and disallowed the use of asbestos in food, food additives, drugs, and drug components. Manufacturers have voluntarily curtailed the use of asbestos in other consumer products such as hair-dryers.

Nonoccupational exposures attributable to the use of manufactured asbestos products have often been assumed to be relatively low, because almost all these products contain asbestos in a binding matrix, such as cement, plastic, rubber or resin. However, exposures can occur if fibers are liberated from these matrices (Committee on Nonoccupational Health Risks of Asbestiform Fibers, 1984).

Exposures to asbestos may also result from the use of products made from asbestos contaminated substances. An example is talc, which is widely used as a pigment, extender, or processing aid in ceramic tile, paint, paper, and plastics. In smaller quantities talc is used as a component of cosmetic powders, foods, drugs, pesticides, and many other products (Committee on Nonoccupational Health Risks of Asbestiform Fibers, 1984).

Table III-8
Indoor Asbestos Measurements Taken in a
California Building and Three Homes

Location	Fibers/m ³	Nanograms/m ³	Description
DHS Laboratory ^{1/}	34,000	.06	Day one
Room 1	2,600	.003	Day two
DHS Laboratory ^{1/}	18,000	.02	Day one
Room 2	93,000	2.5	Day two (after maintenance activity above ceiling)
Menlo Park Home ^{2/} (asbestos ductwork)	<11,000	--	No fibers detected
Berkeley Home ^{3/} (front of heat register)	6,000 63	.14 1.1	Indoor-small fibers Indoor-large fibers
Corte Madera Home ^{4/} (before asbestos paper removal)	56,000	.3	All fiber sizes
(after asbestos paper removal)	32,000 4,000	2.1 29	Indoor-small fibers Indoor-large fibers

- ^{1/} Asbestos had been sprayed on girders above the ceiling.
^{2/} The ductwork was made of asbestos paper.
^{3/} The ventilation system ductwork was made of metal but was lined inside and out with asbestos paper.
^{4/} The ventilation system ductwork was made of metal but was lined on the outside with asbestos paper.

REFERENCES FOR CHAPTER III

Beaman, D. R. and D. M. File, 1976. "Quantitative Determination of Asbestos Fiber Concentrations." Anal. Chem. 48:101-110.

Committee on Nonoccupational Health Risks of Asbestiform Fibers, 1984. Asbestiform Fibers - Nonoccupational Health Risks.

Hayward, Dr. Steven B., March 1984. "Field Monitoring of Chrysotile Asbestos in California Waters." Journal of the American Water Works Association. 66-73.

Hayward, Dr. Steven B., May 31, 1985. Research Specialist, Air and Industrial Hygiene Laboratory, California Department of Health Services. Personal communication with Ralph Propper of the Air Resources Board.

Hayward, Dr. Steven B., June 18, 1985. Research Specialist, Air and Industrial Hygiene Laboratory, California Department of Health Services. Personal communication with Janette Munson of the Air Resources Board.

Hayward, Dr. Steven B., October 26, 1985. Research Specialist, Air and Industrial Hygiene Laboratory, California Department of Health Services. Personal communication with Todd Wong of the Air Resources Board.

Ontario Ministry of the Attorney General, 1984. Report of The Royal Commission on Matters of Health and Safety Arising from the Use of Asbestos in Ontario.

Science Applications, Incorporated, December 1983. Final Report Ambient Asbestos Concentrations In California: Volumes I and II.

Sebastien, P. J. Bignon and M. Martin, June 1982. "Indoor Airborne Asbestos Pollution From the Ceiling and the Floor." Science. 216:1410-1413.

U.S. Environmental Protection Agency, June 1978. Evaluating and Optimizing Electron Microscope Methods for Characterizing Airborne Asbestos. EPA-600/2-78-038.

Versar Inc., January 9, 1984. Draft Final Report - Exposure Assessment For Asbestos. Prepared for the U.S. Environmental Protection Agency, Contract No. 68-01-6211, Task 49.

Appendix A

**Department of Health Services
Health Effect Evaluation Request**

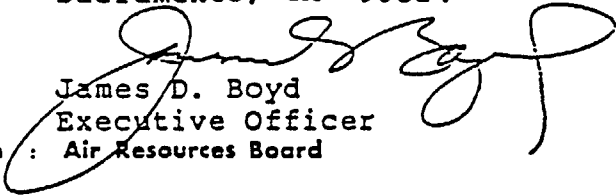
D 004276

Memorandum

To : Stanley Cubanski, Acting Director
Department of Health Services
714 P Street
Sacramento, CA 95814

Date : February 19, 1985

Subject: Evaluation of
Asbestos


James D. Boyd
Executive Officer
From : Air Resources Board

I am writing to formally request that the Department evaluate the health effects of asbestos (e.g., actinolite, amosite, anthophyllite, chrysotile, crocidolite and tremolite) as a candidate toxic air contaminant in accordance with Assembly Bill 1807 (Tanner). According to Health and Safety Code Sections 39660-62, your Department has ninety days to submit a written evaluation and recommendations on health effects of asbestos to the Air Resources Board. If required, your Department may request a thirty-day extension of the deadline.

Attached for your staff's consideration in evaluating asbestos are:

Attachment I - A list of references on asbestos health effects which were identified in an Air Resources Board letter of public inquiry and received in response to the inquiry letter;

Attachment II - Suggested issues which should be considered in preparing the health effects document on asbestos fibers;

Attachment III - Ambient asbestos concentration data which should be used to estimate the range of risk to California residents as required in Health and Safety Code Section 39660(c), sources of asbestos emissions and emission trends; and

Attachment IV - Descriptions of the sampling and analytical methods used to determine the ambient asbestos concentrations.

My staff is available for consultation in conducting this health effects evaluation. We look forward to continuing to work closely with you and your staff in carrying out this

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D 004277

legislative mandate. If you have any further questions regarding this matter, please contact me at 445-4383 or have your staff contact Peter D. Venturini, Chief of the Stationary Source Division, at 445-0650.

Attachments

cc: Gordon Duffy
Alex Kelter, DHS w/attachments
Raymond Neutra, DHS w/attachments
Emil Mrak, Chairman and Scientific
Review Panel Members w/attachments
Peter D. Venturini, ARB
John Holmes, ARB
Assemblywoman Sally Tanner
Senator Ralph Dills
Senator Art Torres
Claire Berryhill, DFA

Attachment I

ASBESTOS REFERENCES (11/9/84)

- 1 Acheson, E. D., Bennett, C., Gardner, M. J. and Winter, P. D. (1981) Mesothelioma in a factory using amosite and chrysotile asbestos. *Lancet* 2(8260-61):1403-1406
- 2 Acheson, E. D. and Gardner, M. J. (1980) Possible synergism between chrysotile and amphibole asbestos (Letter). *Lancet* 1(8170):706
- 3 Acheson, E. D. and Gardner, M. J. (1981) Dose-response relations from epidemiological studies. *Proc R Soc London, (Ser) A*. 376(1764):79-85
- 4 Acheson, E. D., Gardner, M. J., Pippard, E. C. and Grime, L. P. (1982) Mortality of two groups of women who manufactured gas masks from chrysotile and crocidolite asbestos: a 40-year follow-up. *Br J Ind Med* 39(4):344-348.
- 5 Acheson, E. D., Gardner, M. J., Winter, P. D. and Bennett, C. (1984) Cancer in a factory using amosite asbestos. *Int J Epidemiol*. 13(1):3-10.
- 6 Aisner, J. and Wiernik, P. H. (1981) Asbestos-related neoplasm. *Semin Oncol*. 8(3):241-242
- 7 Archer, V. E. (1979) Carcinogenicity of fibers and films: a theory. *Med Hypotheses*. 5(11):1257-1262
- 8 Archer, V. E. and Rom, W. N. (1983) Trends in mortality of diffuse malignant mesothelioma of pleura (Letter). *Lancet*. 2(8341):112-113.
- 9 Armstrong, B. K. et al (1984) Epidemiology of malignant mesothelioma in Western Australia. *Med J Aust* 141(2):86-88.
- 10 Artvinli, M. and Baris, Y. I. (1982) Environmental fiber-induced pleuro-pulmonary diseases in an Anatolian village: an epidemiologic study. *Arch Environ Health*. 37(3):177-181
- 11 Artvinli, M. and Baris, Y. I. (1982) Environmental fiber induced pleuro pulmonary diseases in an anatolian Turkey village-an epidemiologic study. *Arch Environ Health* 37(3):177-181
- 12 Arul, K. J. and Holt, P. F. (1980) Clearance of asbestos bodies from the lung: a personal view. *Br J Ind Med* 37(3):273-277
- 13 Barry, B. E. et al (1983) Reaction of rat lungs to inhaled chrysotile asbestos following acute and subchronic exposures. *Exp Lung Res* 5(1):1-22.
- 14 Beck, E., Konetzke, G. W. and Strum, W. (1982) Asbestos and mesothelioma in east Germany. *Arch Immunol Ther Exp* 30(3-4):229-234
- 15 Beck, E. G. (1980) Experimental pathology-in vitro studies-related to asbestos and other mineral fibers. *IARC Sci Publ* 1(30):385-400
- 16 Becklake, M. R. (1982) Exposure to asbestos and human disease (editorial). *N Engl J Med* 306(24):1480-1492
- 17 Becklake, M. R. (1982) Asbestos-related diseases of the lungs and pleura: current clinical issues. *Am Rev Respir Dis* 126(2):187-194

- 18 Begin, R et al (1981) Early lung events following low-dose asbestos exposure
Environ Res 26(2) 392-401
- 19 Berry, G and Newhouse, M L (1983) Mortality of workers manufacturing friction
material using asbestos Br J Ind Med 40(1) 1-7
- 20 Bignon, J et al (1979) Human and Experimental Data on Translocation of Asbestos
Fibers through the Respiratory System NY Academy of Science 745-50
- 21 Bignon, J and Jaurand, M C (1983) Biological in vitro and in vivo responses of
chrysotile versus amphiboles - Environ Health Perspect 51 72-80.
- 22 Blot, W J et al (1980/September 1980) Lung and Laryngeal Cancers in Relation to
Shipyard Employment in Coastal Virginia. JNCI. 65(3):571-5.
- 23 Bolton, R E et al (1982) Variations in the carcinogenicity of mineral fibers. Ann
Occup Hyg 26(1-4) 569-582
- 24 Bolton R E et al (1982) Variations in the carcinogenicity of mineral fibers. Ann
Occup Hyg 26 (1-4) 569-582
- 25 Boman, G et al (1982) Malignant mesothelioma in Turkish immigrant residing in
Sweden Scand J Work Environ Health 8(2):108-112
- 26 Briselli, M, Mark, E J. and Dickerson, G. R. (1981) Solitary fibrous tumors of the
pleura eight new cases and review of 360 cases in the literature. Cancer
47(11) 2678-2689.
- 27 Brody, A R. and Hill, L. H (1981) Deposition pattern and clearance pathways of
inhaled chrysotile asbestos Chest 80(1 Suppl):64-67.
- 28 Brown, R C, Poole, A and Fleming GTA (1983) The influence of asbestos dust on the
oncogenic transformation of C-3H-10T-1-2 cells Cancer Lett. 18(2):221-228.
- 29 Browne, K (1983) The epidemiology of mesothelioma. J Occup Med. 33(4):190-194.
- 30 Browne, K. (1983) Asbestos-related mesothelioma: epidemiological evidence for
asbestos as a promoter Arch Environ Health 38(5):261-266
- 31 Browne, K. and Coffe, T (1984) Mesothelioma due to domestic exposure to asbestos
(Letter). Br Med J (Clin Res) 289(6437):110-111.
- 32 Browne, K and Smither, W J (1983) Asbestos-related mesothelioma. factors
discriminating between pleural and peritoneal sites Br J Ind Med 40(2) 145-152
- 33 Casey, G (1983) Sister-chromatid exchange and cell kinetics in CHO-K1 cells, human
fibroblasts and lymphoblastoid cells exposed in vitro to asbestos and glass fiber
Mutat Res 116(3-4) 369-377
- 34 Chovil, A and Stewart, C (1979) Latency period for mesothelioma (Letter) Lancet
2(8147) 853
- 35 Churg, A M and Warnock, M. L (1981/March 1981) Asbestos and Other Ferruginous
Bodies, Their Formation and Clinical Significance. Am J Pathol. 102(3) 447-56
- 36 Churg, A and Golden, J (1982) Current problems in the pathology of asbestos-related
disease Pathol Annu 17(Pt 2) 33-66

- 37 Churg, A. and Wiggs, B. (1984) Fiber size and number in amphibole asbestos induced mesothelioma. *Am J Pathol* 115(3): 437-442.
- 38 Churg, A. and Wood, P. (1983) Observation on the distribution of asbestos fibers in human lungs. *Environ Res* 31(2): 347-380.
- 39 Cook, P. M., Palekar, L. D. and Coffin, D. L. (1982) Interpretation of the carcinogenicity of amosite asbestos and ferro actinolite on the basis of retained fiber dose and characteristics in vivo. *Toxicol Lett* 13(3-4): 151-158.
- 40 Craighead, J. E. and Mossman, B. T. (1982)) The Pathogenesis of Asbestos-Associated Diseases. *New England Journal of Medicine* 306(24): 1446-55.
- 41 Davis, J. M. (1979) The Use of Animal Models and Primary Prevention of Cancer. *NY Acad of Science* 330: 795-8.
- 42 DiPaolo, J. A., DeMarinis, A. J. and Doniger, J. (1983) Asbestos and Benzo(a)pyrene Synergism in the Transformation of Syrian Hamster Embryo Cells. *Pharmacology* 27(2): 65-73.
- 43 Dodson, R. F. et al (1984) Asbestos Content in Lungs of occupationally exposed individual. *JAMA* 252(1): 68-71.
- 44 Donham, K. J. et al (1980) The effects of long-term ingestion of asbestos on the colon of F344 rats. *Cancer* 45(5 Suppl): 1073-1084.
- 45 Englund, A. and Engholm, G. (1982) Asbestos related cancer in Swedish construction workers. *Arch Immunol Ther Exp* 30(3-4): 157-160.
- 46 Enterline, P. E. (1983) Risk assessment of asbestos carcinogenicity in nonoccupationally exposed populations. *VDI-Ber* 475: 297-303.
- 47 Enterline, P. E. (1983) Cancer produced by nonoccupational asbestos exposure in the United States. *J Air Pollut Control Assoc* 33(4): 318-322.
- 48 Enterline, P. E. (1981) Proportion of cancer due to exposure to asbestos. *Banbury Report* 9: 19-36.
- 49 Epler, G. R., McCloud, T. C. and Gaensler, E. A. (1982) Prevalence and incidence of benign asbestos pleural effusion in a working population. *JAMA* 247(5): 617-622.
- 50 Findley, M. E. et al (1983) An assessment of the Environmental Protection Agency's (USA) asbestos hazard evaluation algorithm. *Am J Public Health* 73(10): 1179-1181.
- 51 Finklestein, M. W. (1983) Mortality among long-term employees of an Ontario asbestos-cement factory. *Br J Ind Med* 40(2): 138-144.
- 52 Finklestein, M. W. (1984) Mortality among employees of an Ontario asbestos-cement factory. *Am Rev Respir Dis* 129(5): 754-761.
- 53 Fischbein, A. and Rohl, A. N. (1984) Pleural mesothelioma and neighborhood asbestos exposure. Findings from microchemical analysis of lung tissue. *JAMA* 252(1): 86-87.
- 54 Fleury, J. et al (1983) Interactions of asbestos fibers with hepatocytes in vitro, an ultrastructural study. *Toxicol Lett* 19(1-2): 15-22.
- 55 Frank, A. L. (1980) Clinical observations following asbestos exposure. *Environ Health Perspect* 34: 27-30.

- 56 Gardner, M. J., Acheson, E. D. and Winter, P. D. (1982) Mortality from mesothelioma of the pleural during 1968-78 in England and Wales. *Br J Cancer* 46(1) 81-88
- 57 Gladfelter, T. (1982) Malignant mesothelioma: an occupation disease (clinical conference). *J Fam Pract* 14(5) 827, 830-832
- 58 Glickman, L. T. et al. (1983) Mesothelioma in pet dogs associated with exposure of their owners to asbestos. *Environ Res.* 32(2) 305-313
- 59 Goldsmith, J. R. (1982) Asbestos as a systemic carcinogen: the evidence from eleven cohorts. *Am J Ind Med* 3(3) 341-348
- 60 Goschick, J. W. and Indulski, J. A. (1982) The Polish studies on occupational exposure to asbestos and its biological effects. *Arch Immunol Ther Exp* 30(3-4):169-176
- 61 Griffiths, L. C. et al. (1983) Deposition of crocidolite asbestos and glass microfibers inhaled by the Beagle dog. *Am Ind Hyg Assoc J* 44(3) 216-222
- 62 Hallenback, W. H. (1983) Asbestos PENETRATION OF THE GASTROINTESTINAL TRACT. *Environ Health Perspect* 53 153-154.
- 63 Hallenback, W. H., Markey, D. R. and Dolan, D. G. (1981) Analyses of tissue, blood, and urine samples from a baboon gavages with chrysotile and crocidolite asbestos. *Environ Res* 25(2) 349-360.
- 64 Hamilton, J. A. (1980) Macrophage stimulation and the inflammatory response to asbestos. *Environ Health Perspect.* 34 69-74
- 65 Harington, J. S. (1981) Fiber carcinogenesis: epidemiologic observations and the Stanton hypothesis. *JNCI* 67(5) 977-989
- 66 Harvey, G., Page, M. and Dumas, L. (1984) Binding of environmental carcinogens to asbestos and mineral fibers. *Br J Ind Med.* 41(3):396-400.
- 67 Haugen, A. and Harris, C. (1982) Asbestos carcinogenesis: asbestos interactions and epithelial lesion in cultured human tracheobronchial tissues and cells. *Recent Results Cancer Res (Germany, West)*. 82:32-42.
- 68 Henderson, D. W. (1982) Asbestos-related pleuropulmonary diseases: asbestosis, mesothelioma and lung cancer. *Pathology (Australia)*. 14(3):239-243.
- 69 Henderson, V. L. and Enterline, P. E. (1979) Asbestos exposure, factors associated with excess cancer and respiratory disease mortality. *Ann NY Acad Sci.* 330 117-126
- 70 Hesterberg, T. W. et al. (1982) Asbestos induces morphological transformation in Syrian hamster embryo cells in culture. *J Cell Biol.* 95(2 part 2) 449A
- 71 Hillerdal, G. (1983) Malignant mesothelioma 1982: review of 4170 published cases. *Br J Dis Chest* 77(4) 321-343
- 72 Holt, P. F. (1982) Translocation of asbestos dust through the bronchiolar wall. *Environ Res* 27(2) 255-260
- 73 Hopkins, J. (1984) Epidemiological studies on ingested asbestos. *Food Chem Toxicol* 22(2) 179-181.
- 74 Huang, S. L. (1979) Amosite, chrysotile and crocidolite asbestos are mutagenic in Chinese hamster lung cells. *Mutat Res* 68(3) 265-274

75. Humphrey, E. W. et al (1981) The production of malignant tumors of the lung and pleural in dogs from intratracheal asbestos instillation and cigarette smoking. *Cancer* 47(8) 1994-1999.
76. Huukonen, M. S. (1980) Asbestos and cancer in Finland. *J Toxicol Environ Health* 6(5-6) 1261-1265.
77. Huukonen, M. S. (1982) Asbestos and cancer. *Eur J Respir Dis* 123 145-152.
78. Jones, J. S. et al (1980) The pathology and mineral content of lungs in cases of mesothelioma in the United Kingdom in 1976. *IARC Sci Publ.* 1(30):187-199.
79. Joseph, L. B. et al (1983) Morphological transformation in vitro of normal human fibroblasts by chrysotile. *Environ Health Perspect* 51:17-22.
80. Kagan, E. (1981) The alveolar macrophage: immune derangement and asbestos-related malignancy. *Semin Oncol.* 8(3):258-267.
81. Kahn, E. I. et al (1980) Primary pericardial mesothelioma following exposure to asbestos. *Environ Res* 23(2):270-281.
82. Kannerstein, M. (1980) Recent advances and perspectives relevant to the pathology of asbestos-related diseases in man. *IARC Sci Publ.* 1(30):149-162.
83. Kannerstein, M. and Churg, J. (1980) Mesothelioma in man and experimental animals. *Environ Health Perspect.* 34:31-36.
84. Kaw, J. L., Tilkes, F. and Beck, E. G. (1982) Reaction of cells cultured in vitro to different asbestos dusts of equal surface area but different fiber length. *Br J Exp Pathol* 63(1):109-115.
85. Kolev, K. (1982) Experimentally induced mesothelioma in white rats in response to intra peritoneal administration of amorphous crocidolite asbestos preliminary report. *Environ Res.* 29(1) 123-133.
86. Kolev, K. (1982) Experimentally induced mesothelioma in white rats in response to intraperitoneal administration of amorphous crocidolite asbestos: preliminary report. *Environ Res* 29(1) 123-133.
87. Lafuma, J. et al (1980) Mesothelioma induced by intrapleural injection of different types of fibers in rats; synergistic effect of other carcinogens. *IARC Sci Publ.* 1(30):311-320.
88. Langer, A. M. et al (1980) Asbestos as a cofactor in carcinogenesis among nickel-processing workers. *Science* 209(4454) 420-422.
89. Lemen, R. A., Dement, J. M. and Wagoner, J. (1980) Epidemiology of asbestos-related diseases. *Environ Health Perspect.* 34 1-11.
90. Lewinsohn, H. C. et al (1980) The influence of occupational and environmental asbestos exposure on the incidence of malignant mesothelioma in Connecticut. *IARC Sci Publ.* 2(30):655-660.
91. Lillis, R. et al (1979) Asbestos disease in maintenance workers of the chemical industry. *Ann NY Acad Sci* 330 127-135.
92. Lipkin, L. E. (1980) Cellular effects of asbestos and other fibers: correlations with in vivo induction of pleural sarcoma. *Environ Health Perspect* 34:91-102.

- 93 Livingston, G. K., Rom, W. N. and Morris MV (1980) Asbestos-induced sister chromatid exchanges in cultured Chinese hamster ovarian fibroblast cells. *J Environ Pathol Toxicol* 4(2-3) 373-382
- 94 Lopez-Areal Del Amo L (1980) Diseases associated with asbestos in Spain. *IARC Sci Publ* 1(30) 201-206
- 95 Maltoni, C., Minardi, F. and Morisi, L. (1982) The relevance of the experimental approach in the assessment of the oncogenic risks from fibrous and nonfibrous particles. The ongoing project of the Bologna Institute of Oncology. *Med Lav* 73(4) 394-407.
- 96 Mancuso, T. F. (1983) Mesothelioma among machinists in railroad and other industries. *Am J Ind Med* 4(4) 501-513.
- 97 Masse, R. et al (1980) Experimental demonstration of the penetration of asbestos fibers into the gastrointestinal tract. *IARC Sci Publ* 1(30) 321-328.
- 98 McConell, E. E. et al (1983) Chronic effects of dietary exposure to amosite and chrysotile asbestos in Syrian golden hamsters. *Environ Health Perspect* 53:11-25.
- 99 McConell, E. E. et al (1983) Chronic effects of dietary exposure to amosite asbestos and tremolite in F344 rats. *Environ Health Perspect* 53:27-44
- 100 McCullagh, S. F. (1980) Amosite as a cause of lung cancer and mesothelioma in humans. *J Soc Occup Med* 30(4) 153-156
- 101 McDonald, A. D. et al (1984) Dust exposure and mortality in an American chrysotile asbestos friction products plant. *Br J Ind Med* 41(2):151-157
- 102 McDonald, A. D. et al (1983) Dust exposure and mortality in an American factory using chrysotile, amosite, and crocidolite in mainly textile manufacture. *Br J Ind Med* 40(4) 368-374
- 103 McDonald, A. D. et al (1983) Dust exposure and mortality in an American chrysotile textile plant. *Br J Ind Med* 40(4):361-367
- 104 McDonald, J. C. (1980) Asbestos-related disease: an epidemiological review. *IARC Sci Publ* 2(30) 587-601.
- 105 McDonald, J. C. et al (1980) Dust exposure and mortality in chrysotile mining, 1910-1975. *Br J Ind Med* 37(1):11-24
- 106 McDonald, J. C. and Liddell, F. D. (1979) Mortality in Canadian miners exposed to chrysotile. *Ann NY Acad Sci* 330:1-9
- 107 McDonald, J. C. and MacDonald, A. D. (1981) Mesothelioma as an index of asbestos impact. *Banbury Report* 9:73-86
- 108 Menchaux, G. et al (1981) mesothelioma in rats following inoculation with acid-leached chrysotile asbestos and other mineral fibers. *Carcinogenesis* 2(3) 229-236
- 109 Morgan, A. (1980) Effect of length on the clearance of fibers from the lung and on body formation. *IARC Sci Publ* 1(30) 329-335
- 110 Mossman, B., Light, W. and Wei, E. (1983) Asbestos: mechanisms of toxicity and carcinogenicity in the respiratory tract. *Annu Rev Pharmacol Toxicol* 23 595-615

- 111 Mossman, B. T. and Craighead, J. E. (1981) Mechanisms of asbestos carcinogenesis. *Environ Res* 25(2) 269-280.
- 112 National Research Council (1984) Non-occupational exposure to asbestiform fibers. NAS Report.
- 113 Newhouse, M. L. (1983) Asbestos-related diseases. *Practitioner* 227(1383) 1399-1411.
- 114 Newhouse, M. L. (1981) Epidemiology of asbestos-related tumors. *Semin Oncol* 8(3) 250-257.
- 115 Newhouse, M. L. and Berry, G. (1979) Patterns of mortality in asbestos factory workers in London. *Ann NY Acad Sci.* 330:53-60.
- 116 Newhouse, M. L., Berry, G. and Skidmore, J. W. (1982) A mortality study of workers manufacturing friction materials with chrysotile asbestos. *Ann Occup Hyg.* 26(1-4):899-909.
- 117 Nicholson, W. J. et al (1981) Cancer from occupational asbestos exposure projections. 1980-2000. Banbury Report 9:87-111.
- 118 Nicholson, W. J. (1984) Asbestos Health Assessment Update. EPA Report (Review draft) EPA-600/8-84-003A:131.
- 119 Nicholson, W. J. et al (1979) Long-term mortality experience of chrysotile miners and millers in Thetford Mines, Quebec. *Ann NY Acad Sci.* 330:11-21.
- 120 Nicholson, W. J., Perkel, G. and Selikoff, I. J. (1982) Occupational exposure to asbestos: population at risk and projected mortality--1980-2030. *Am J Ind Med.* 3(3):259-311.
- 121 Norseth, T. (1980) Asbestos and metals as carcinogens. *J Toxicol Environ Health.* 6(5-6):1021-1028.
- 122 Ordonez, N. G. and Smith, J. L. Jr (1983) Peritoneal malignant mesothelioma with multiple distant skin metastases. *Arch Dermatol.* 119(10):827-830.
- 123 Peto, J. (1980) The incidence of pleural mesothelioma in chrysotile asbestos textile workers. *IARC Sci Publ* 2(30):703-711.
- 124 Peto, J. (1979) Dose-response relationships for asbestos-related disease. implications for hygiene standards. Part II. Mortality. *Ann NY Acad Sci.* 330:195-203.
- 125 Peto, J. (1980) Lung cancer mortality in relation to measured dust levels in an asbestos textile factory. *IARC Sci Publ* 2(30):829-836.
- 126 Peto, J., Henderson, B. E. and Pike, M. C. (1981) Trends in mesothelioma incidence in the USA and the forecast epidemic due to asbestos exposure during World War II. Banbury Report 9:51-72.
- 127 Peto, J., Seidman, H. and Selikoff, I. J. (1982) Mesothelioma mortality in asbestos workers: implications for models of carcinogenesis and risk assessment. *Br J Cancer* 45(1) 124-135.
- 128 Pott, F. (1982) Animal experiments on biological effects of mineral fibers. *IARC Sci Publ* 1(30):261-272.
- 129 Price-Jones, M. J., Gubbings, G. and Chamberlain, M. (1980) The genetic effects of crocidolite asbestos: comparison of chromosome abnormalities and sister chromatid exchanges. *Mutat Res* 79(4) 331-336.

- 130 Pylev, L. N. et al (1982) Pleural mesothelioma in the Rhesus monkey *Macaca mulatta* induced by intra tracheal injections of chrysotile asbestos. *Eksp Onkol.* 4(6):36-38
- 131 Pylev, L. N. (1980) Pretumorous lesions and lung and pleural tumors induced by asbestos in rats, Syrian golden hamsters and *Macaca mulatta* (rhesus) monkeys. *IARC Sci Publ* 1(30) 343-355
- 132 Rahman, O., Das, B. and Viswanathan, P. N. (1983) Biochemical mechanisms in asbestos toxicology. *Environ Health Perspect* 51:299-303
- 133 Reiss, B. et al (1983) Absence of mutagenic activity of three forms of asbestos in liver epithelial cells. *Environ Res* 31(1):100-104.
- 134 Rocco, K. (1979) Based on available data, can we project an acceptable standard for industrial use of asbestos? Absolutely. *Ann NY Acad Sci.* 330:205-210
- 135 Rogol, P. R., Ryu, J. H. and Ginns, L. C. (1983) Reduced natural killer cell activity in asbestos workers. *Am Rev Respir Dis.* 127(4 part 2):73
- 136 Rom, W. N. et al (1983) Sister chromatid exchange frequency in asbestos workers. *J Natl Cancer Inst* 70(1) 45-48
- 137 Rubino, G. F. et al (1979) Mortality of chrysotile asbestos workers at the Balangero Mine, Northern Italy. *Br J Ind Med.* 36(3):187-194.
- 138 Scansetti, G. et al (1981) Pleural mesothelioma after a short interval from first exposure in the wine filter industry. *Am J Ind Med* 5(4) 335-339.
- 139 Schenker, M. B., Garshick, E. and Speizer, F. E. (1984) A case control study of incident mesothelioma deaths among railroad workers. *Am Rev Respir Dis.* 129(4 suppl) A146
- 140 Seidman, H., Selikoff, I. J. and Hammond, E. C. (1979) Short-term asbestos work exposure and long-term observation. *Ann NY Acad Sci.* 330 61-90
- 141 Seidman, H., Selikoff, I. J. and Hammond, E. C. (1982) Mortality of brain tumors among asbestos insulation workers in the United States and Canada. *Ann NY Acad Sci* 381 160-171
- 142 Selikoff, I. J., Churg, J. and Hammond, E. C. (1984) Classics in oncology: Asbestos exposure and neoplasia. *CA* 34(1):48-56
- 143 Selikoff, I. J., Hammond, E. C. and Seidman, H. (1980) Latency of asbestos disease among insulation workers in the United States and Canada. *Cancer* 46(12) 2736-2740
- 144 Selikoff, I. J., Hammond, E. C. and Seidman, H. (1979) Mortality experience of insulation workers in the United States and Canada, 1943-1976. *Ann NY Acad Sci* 330 91-116
- 145 Selikoff, I. J., Lillis, R. and Nicholson, W. J. (1979) Asbestos disease in United States shipyards. *Ann NY Acad Sci.* 330:295-311.
- 146 Selikoff, I. J., Seidman, H. and Hammond, E. C. (1980) Mortality effects of cigarette smoking among amosite asbestos factory workers. *JNCI.* 65(3):507-513.
- 147 Selikoff, I. J. (1981) Constraints in estimating occupational contributions to current cancer mortality in the USA. *Banbury Report* 9 3-18

- 148 Stanton M. F. et al (1981) Relation of particle dimension to carcinogenicity in amphibole asbestoses and other fibrous minerals JNCI 47(5):965-975
- 149 Stevens, J. B. et al (1984) Asbestos metabolism in-vivo. Am Rev Respir Dis. 129(4 suppl) A168
- 150 Stumphius, J. (1979) Mesothelioma incidence in a Dutch shipyard. Ann NY Acad Sci 330 317-322
- 151 Stryba, K. and Lange, A. (1982) Effects of asbestos on benzo(a)pyrene mutagenicity. Mutat Res 97(3) 227-228
- 152 Taylor, R. A. and Johnson, L. P. (1981) Mesothelioma: current perspectives. West J Med 134(5) 379-383.
- 153 Teta, M. J. et al (1982) Occupational asbestos exposure and mesothelioma in Connecticut USA. "Occupational lung disease" edited by Gee, J.B. et al. Raven Press, New York:249pp
- 154 Teta, M. J. et al (1983) Mesothelioma in Connecticut, 1955-1977. Occupational and geographic associations. J Occup Med. 25(10) 749-756.
- 155 Thomas, H. F. et al (1982) Further follow-up study of workers from an asbestos cement factory. Br J Ind Med 39(3):273-236.
- 156 Topping, D. C. and Nettesheim, P. (1980) Two-stage carcinogenesis studies with asbestos in Fischer 344 rats. JNCI. 65(3):627-630.
- 157 Topping, D. C., Nettesheim, P. and Martin, D. H. (1980) Toxic and tumorigenic effects of asbestos on tracheal mucosa. J Environ Pathol Toxicol. 3(5-6):261-275
- 158 Valerio, F. et al (1983) Chromosomal aberrations induced by chrysotile and crocidolite in human lymphocytes in vitro. Mutat Res. 122(3-4):397-402
- 159 Vicent, J. H. et al (1981) Static electrification of airborne asbestos: a study of its causes, assessment and effects on deposition in the lungs of rats. Am Ind Hyg Assoc J 42(10):711-721
- 160 Wagner, J. C. (1979) Diseases associated with exposure to asbestos dusts. Practitioner 223(1333):28-33
- 161 Wagner, J. C. (1982) Experimental studies and the pathology of asbestos induced lung diseases. Arch Immunol Ther Exp. 30(3-4) 221-228
- 162 Wagner, J. C., Berry, G. and Pooley, F. D. (1982) Mesotheliomas and asbestos type in asbestos textile workers: a study of lung contents. Br Med J 285(6342) 603-606.
- 163 Wagner, J. C., Berry, G. and Pooley, F. D. (1980) Carcinogenesis and mineral fibers. Br Med Bull 36(1):53-56
- 164 Wagner, J. C., Berry, G., Skidmore, J. W. and Pooley, F. D. (1980) The comparative effects of three chrysotiles by injection and inhalation in rats. IARC Sci Publ 1(30) 363-372
- 165 Wagner, J. C., Griffiths, D. M. and Hill, R. J. (1984) The effect of fibre size on the in vivo activity of UICC crocidolite. Br J Cancer 49(4):453-458
- 166 Wagner, M. M. (1980) Immunology and asbestos. IARC Sci Publ 1(30) 247-251

- 167 Wagner, M. M. and (1983) HLA-A and B antigen frequencies and mesothelioma in relation to asbestos exposure. *Br J Cancer*. 48(5).727-730
- 168 Walker, A. M. et al (1983) Projections of asbestos related disease 1980-2009. *J Occup Med* 25(5) 409-425
- 169 Varnock, M. L., Kuwahara, T. J. and Wolery, G. (1983) The relation of asbestos burden to asbestosis and lung cancer. *Pathol Annu.* 18(pt 2) 109-145
- 170 Whitaker, D., Shilkin, K. B. and Walters MNI (1984) Cytologic and tissue culture characteristics of asbestos induced mesothelioma in rats. *Acta Cytol.* 28(2) 185-189
- 171 Winkler, G. C. and Ruettnier, J. R. (1982) Penetration of asbestos fibers in the visceral peritoneum of mice a scanning electron microscopic study. *Exp Cell Biol* 50(4) 187-194.
- 172 Woitowitz, H. J. et al (1982) Asbestos related diseases in West Germany. *Arch Immunol Ther Exp* 30(3-4).143-144
- 173 Woitowitz, H. J. et al (1981) Asbestos-related diseases in the Federal Republic of Germany. *Am J Industr Med* 2(1).71-78.



ASBESTOS INFORMATION ASSOCIATION

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January 17, 1985

William V. Loscutt, Chief
Toxic Pollutants Branch
Re: Asbestos
California Air Resources Board
P.O. Box 2815
Sacramento, CA 95812

Dear Mr. Loscutt:

I am writing in response to the Air Resources Board's request for information regarding asbestos. I have had the opportunity to review the ARB's bibliography and to discuss the program with Mr. John Batchelder. The greatest omission in the ARB's bibliography appears to be in not listing a number of large, overview studies of asbestos which are valuable for their scope and for the many individual references assembled in them. I would, therefore, recommend that the ARB collect and consider the following studies:

Acheson, E.D., Gardner, M.J. (1983) Asbestos. The Control Limit for Asbestos. Her Majesty's Stationery Office, London.

British Advisory Committee. (1979) Asbestos. (The Simpson Report). Her Majesty's Stationery Office, London.-

CPSC. (1983) Chronic Hazard Advisory Panel on Asbestos. U.S. Consumer Product Safety Commission, Directorate for Health Sciences.

Report of the Royal Commission on Matters of Health and Safety Arising from the Use of Asbestos in Ontario. (1984).

On the issue of ingestion of asbestos, there is a recent animal bioassay from the National Toxicology Program:

NTP. (1983) Lifetime carcinogenesis studies of amosite asbestos in Syrian golden hamsters. NIH publication number 84-2505.

I am also enclosing a copy of the EPA's response from the December 19, 1984 Federal Register regarding the use of asbestos in brakes. Mr. Batchelder noted that this is one area which may be a concern of the ARB.

Finally, I am enclosing a copy of a paper by Wagner and Elmes entitled "The Mineral Fibre Problem." As the use of asbestos declines, the use of other fibrous materials increases. The Wagner and Elmes paper cautions, on the basis of animal and human evidence, against the indiscriminant and uncontrolled use of these fibrous materials. The purpose of sending this paper is to make the point to the ARB that these fibrous materials constitute an area of investigation which has received scant attention, as opposed to asbestos, which has been very thoroughly studied and regulated. Yet another survey study of asbestos, as is presently contemplated by the ARB, has a certain aura of "reinventing the wheel" about it, whereas, with the exception of the Danish government, there has not been a regulatory body which has addressed the issue of fibers generally.

I would appreciate being kept informed of the progress of this project of the ARB, and if there is a mailing list established, please include my name on it. If I may be of any assistance, please feel free to contact me.

Very truly yours,



Nicholas J. Muchyi, Esq.
Government Affairs Council

40 CFR Part 763

(OPTS-211015, TSM-FRL 2724-4)

Asbestos; Response to Citizens' Petition.

AGENCY: Environmental Protection Agency (EPA).

ACTION: Response to Citizens' Petition.

SUMMARY: The Environmental Protection Agency (EPA) is granting a petition filed under section 21 of the Toxic Substances Control Act (TSCA) by the Natural Resources Defense Council, Inc., to prohibit the use of asbestos in automobile and truck brakes. EPA has commenced an appropriate proceeding to address the risks which may be posed by this use of asbestos.

DATE: Submit written comments on or before March 16, 1985.

ADDRESS: Submit written comments in triplicate identified by the document control number (OPTS-211015) to: TSCA Public Information Office (TS-793), Office of Toxic Substances, Environmental Protection Agency, Rm. E-107, 401 M St. SW., Washington, D.C. 20460.

A copy of the petition and related information (with any confidential business information deleted) is located in Room E-107, Environmental Protection Agency, 401 M St. SW., Washington, DC 20460.

This material is available for viewing and copying from 8 a.m. to 4 p.m. Monday through Friday, excluding legal holidays.

FOR FURTHER INFORMATION CONTACT: Edward A. Klein, Director, TSCA Assistance Office (TS-799), Office of Toxic Substances, Environmental Protection Agency, Rm. E-343, 401 M St. SW., Washington, D.C. 20460. Toll-free: (800-424-6065). In Washington, D.C.: (554-1404). Outside the USA: (Operator-202-354-1404).

SUPPLEMENTARY INFORMATION**I. Introduction**

Section 21 of the Toxic Substances Control Act (TSCA), 15 U.S.C. 2620, provides that any person may petition the Administrator of EPA to initiate a proceeding for the issuance, amendment, or repeal of a rule under various sections of the Act. EPA may hold a public hearing or may conduct an appropriate investigation to determine whether the petition should be granted. EPA must either grant or deny the petition within 90 days. If EPA grants the petition, EPA shall promptly commence an appropriate proceeding. If EPA denies the petition, the reasons for denial must be published in the Federal Register, and the petitioner may

commence a civil action in a district court of the United States to compel EPA to initiate a rulemaking proceeding as requested in the petition. Any such civil action must be filed within 60 days after EPA's denial of the petition or, if EPA fails to grant or deny the petition within 90 days after the petition is filed, within 60 days following expiration of the 90-day response period.

II. Evaluation of the Petition**A. Introduction**

On September 12, 1984, EPA received a petition from the Natural Resources Defense Council, Inc. (NRDC), requesting that EPA prohibit the further use of asbestos in automobile and truck brakes under section 6 of TSCA. The petition requested a prohibition of asbestos in both brakes for new cars and trucks and in replacement brakes for existing vehicles. The petition argued that the risks posed by asbestos in brakes are unreasonable and that economically and technically feasible substitutes are available.

In order to promulgate any rules under TSCA section 6, the Agency must consider a number of factors, including, among other things, the effects of a chemical substance on human health and the magnitude of exposure; the benefits of utilizing the substance; and the availability of substitutes for the use or uses of the substance being assessed. 15 U.S.C. 2605(c)(1). The Agency has conducted a review of the available information pertaining to the use of asbestos in brakes, including the information in NRDC's petition. A summary of that review, including an evaluation of the risks posed by this use and the availability of substitutes, is presented below.

B. Risk Presented by Asbestos

Asbestos is a demonstrated human carcinogen that causes lung cancer and mesothelioma (a cancer of the chest and abdominal linings), as well as other lung disorders. People are exposed to asbestos throughout the life cycle of the substance—when asbestos is mined, milled, processed, fabricated into industrial and consumer products, and when those products are used, repaired, and disposed of.

With regard to the use of asbestos in brakes, it has been estimated that about 2,750 people are potentially exposed during primary manufacturing of brake friction materials, and that about 350,000 people are potentially exposed to asbestos during servicing and repair of vehicle brakes (Ref. 6). For example, persons in brake service and repair shops typically are exposed to asbestos

when dust is blown out of brake drums being replaced, when brake flanges are roughened to increase friction properties, and when brake shoes are relined.

Use of asbestos in vehicle brakes may also result in increased asbestos fiber concentrations in the ambient air. For example, EPA has evidence that motor vehicle braking most likely contributes between 0.23 to 1.3 percent of the concentration of asbestos in the ambient air (Ref. 7). Both general population and workplace exposures to asbestos fibers from its use in brakes may result in an increased number of asbestos-related illnesses, including cancer.

C. Availability of Substitutes

The petitioner asserts that economically and technically feasible substitutes, most prominently semimetallic friction materials and aramid fibers, are available to replace asbestos in brakes. EPA has analyzed the availability of substitutes for many asbestos products, including brakes, and that analysis is summarized in Appendix A of the "Regulatory Impact Analysis of Controls on Asbestos Products" (Ref. 2), which is included in the public record established for NRDC's section 21 petition. EPA acknowledges that new substitutes for asbestos use in brakes are being developed and that EPA's analysis (summarized below) may not include recent developments.

1. Heavy vehicle brake blocks. Brake blocks are components of brakes that are riveted or bolted to the insides of brake shoes to provide protection against the heat and wear caused by braking. Heavy vehicle brake blocks are used on heavy duty trucks, buses, and other heavy duty vehicles. About 34 percent of all asbestos used in brakes is in this category of use. Asbestos heavy vehicle brake blocks account for about 99 percent of the market for heavy vehicle brake blocks.

Until recently, the only commercially available substitute for asbestos heavy vehicle brake blocks was a semimetallic brake block using brass and zinc chips in an organic binder. It is not considered as good as asbestos because it performs erratically at different temperatures. It is also considered inferior to the asbestos brake block in resisting wear and minimizing brake fade. Recently, aramid fiber products, such as Kevlar, have been introduced, but aramid fiber products are now more expensive than asbestos products, and there is not sufficient evidence to determine whether aramid fiber products will be as effective as asbestos in this application. Information suggests that such products may last longer than asbestos-based

products, but verification of this information as well as information on price and efficacy of the products are needed before EPA can determine whether continued use of asbestos in brake blocks presents an unreasonable risk.

2. *Light and medium vehicle drum brake linings.* Drum brake linings are made of friction materials which cover curved metal shoes in a drum brake. About 40 percent of all asbestos used in brakes is in this category of use. Light vehicle asbestos drum brake linings may be used in both the front and rear brakes of light and medium vehicles, primarily on the front wheels. However, most passenger vehicles still use drum brake linings on the rear wheels. At least 90 percent of drum brake linings are still asbestos.

Until recently the only substitutes for asbestos brake linings which appeared to have any potential were semimetallic brake linings. However, these products tend to perform erratically at different temperatures, fade, and produce more noise than asbestos-based linings. More recently, brake linings made with aramid fiber have been developed. However, these are more expensive than the asbestos product and there is not currently enough information available to judge the performance of aramid fiber brake linings. Furthermore, EPA's evidence indicates that large volume production of aramid fiber brakes may require substantial retooling by brake manufacturers. Therefore, aramid fiber brakes may not be available in substantial quantities for several years.

3. *Heavy vehicle disc brake pads.* Disc brake pads are steel plates lined with friction materials which rub against a rotor. Heavy vehicles rarely use disc brakes. About 0.1 percent of all asbestos used in brakes is in this category of use.

Semimetallic disc brake pads are about 20 percent more expensive than the asbestos disc brake pads but they last about 30 percent longer. The semimetallic disc brake pads are not considered to be good general replacements for asbestos disc brake pads for heavy vehicles because semimetallics perform better than asbestos only in hostile, high-friction, high-heat environments.

4. *Light and medium vehicle disc brake pads.* These are the same as disc brake pads for heavy vehicles, except they are smaller. About 20 percent of all asbestos used in brakes is in this category of use. In the disc brake market, semimetallic brakes have already made larger inroads, and as in the drum brake linings market aramid-based brakes are just beginning to be introduced. Approximately 50 percent of

disc brakes in the original equipment market are asbestos and 50 percent are semimetallic. In the replacement brakes after-market approximately 70 percent of disc brakes are asbestos and 30 percent are semimetallic.

Semimetallic disc brakes cost approximately 2 times as much as the asbestos disc brake pads, but they last about 40 percent longer than the asbestos pads. Semimetallic disc brake pads cannot substitute for asbestos disc brake pads in every application because of inferior performance characteristics. EPA has information indicating that semimetallic disc brakes should be used only in cars with power brake systems because otherwise the semimetallic brakes may not provide enough stopping power. EPA also has information that the semimetallic brakes are slightly inferior because the user has to push harder on them before they are warmed up to get the same performance as with asbestos brakes.

EPA is aware that a good deal of product research is being done to develop effective substitutes for asbestos in brakes. EPA is seeking additional information on substitutes for asbestos in brakes.

III. Conclusion

EPA has decided to grant NRDC's petition because the Agency believes that the use of asbestos in brakes does present risks to human health. EPA is initiating a proceeding to gather information on the desirability and feasibility of reducing risks associated with the use of asbestos in brakes. EPA will analyze the exposure from uses of asbestos, the risk presented by those uses, and the substitutes for such uses. EPA will also gather information of the price, efficacy, and availability of substitutes for asbestos in brakes. After analysis of this information, EPA will determine what further action is appropriate to address the risks which may be posed by this use of asbestos.

Based on information reviewed to date, EPA is not prepared to make an unreasonable risk finding for an immediate ban of all uses of asbestos in brakes at this time. Effective substitutes may not be available for certain applications of asbestos in brakes and substitutes for other applications are not available in sufficient quantity because of limited production capacity. Retooling may be required to make substitutes available in large volumes.

The Agency invites the public to submit comments on issues relating to the petition. EPA is particularly interested in receiving information on both the current availability of substitutes for asbestos in brakes and

on new substitute products that are currently being developed. The Agency has an ongoing workgroup, which will review these comments, will continue to investigate the availability of effective asbestos-free substitutes for use in vehicle brakes, and consider appropriate options to address the risk presented by asbestos in brakes.

IV. Record

EPA has established a record for information relating to the NRDC petition. The record includes information considered by EPA in developing this response and consists of the following categories of information:

- (1) The NRDC petition.
- (2) Appendix A of the "Regulatory Impact Analysis of Controls on Asbestos Products."
- (3) Other information on substitutes for asbestos in brakes.
- (4) Information received from the public concerning the petition.
- (5) Memoranda summarizing meetings and telephone conversations with the public concerning the petition.
- (6) Appendix J of the "Regulatory Impact Analysis of Controls on Asbestos Products."
- (7) Appendix N of the "Regulatory Impact Analysis of Controls on Asbestos Products."

A public version of the record, without any confidential business information, is available to the public in the Office of Toxic Substances Public Information Office, from 8 a.m. to 4 p.m. Monday through Friday, except legal holidays. The Public Information Office is located in Rm. E-107, 401 M St., SW., Washington, D.C.

Dated: December 14, 1984.

William D. Ruckelshaus,

Administrator.

[FR Doc. 84-33120 Filed 12-17-84; 1:14 pm]

GILLIAM CODE 1440-60-2

1. The mineral fibre problem

J. C. Wagner P. C. Elmes

For many years interest in the biological effect of mineral fibres was mainly confined to the commercial types of asbestos. Recently many other fibrous minerals have been recognised as potentially dangerous pollutants of the environment. The majority of these materials are naturally occurring, others are synthetic. The natural fibres are either specifically exploited for commercial purposes or else occur as atmospheric contaminants which are released during mining or tunnelling operations. Industry has been developing other mineral fibres as a substitute for asbestos to meet an increasing need for cheap and reliable materials for reinforcement, friction products and insulation. The latter demand has been emphasised by the present fossil fuel crisis. Minerals being exploited for a variety of purposes other than insulation and reinforcement are known to consist of fibres or elongated crystals, for example, some clays and some zeolites. Thus, these minerals can be considered under the following groups:

1. Asbestos minerals
 - a. Of commercial value
 - b. As potential environmental contaminants
2. Synthetic mineral fibres
3. Other naturally occurring fibrous minerals

ASBESTOS

Asbestos of commercial value

Practically all the knowledge that is available about hazards associated with the inhalation of fibrous mineral dusts has been obtained in studies of asbestos. Asbestos consists of six naturally occurring minerals: chrysotile, crocidolite, amosite, anthophyllite, tremolite and actinolite. Chrysotile is a member of a group of minerals referred to as the serpentines and is composed almost exclusively of magnesium in combination with silica. It has a sheet structure which curls to produce hollow tube-like fibres. The other five are members of one mineralogical group referred to as the amphiboles. They are very similar in crystal structure, being chain silicates, but they vary in chemical composition. Crocidolite and amosite are iron-rich varieties, anthophyllite is a magnesium rich mineral, while tremolite and actinolite contain a large amount of calcium together with magnesium.

The annual world production of asbestos in 1976 was 5×10^9 kg, of which 97 per cent was chrysotile and the remainder crocidolite and amosite. The commercial production of the other three amphiboles has been on a small scale in the past, but they are important as contaminants of other minerals and agricultural soil.

Chrysotile is widely distributed, with the largest production from the Ural

With the development of more sophisticated techniques it is now obvious that a correct estimation of the number of fibres in tissue or environmental samples can only be obtained by examination under a transmission electron microscope, otherwise the large number of fibres of less than $0.5 \mu\text{m}$ in diameter will not be observed. The crucial question of the amount, size and type of fibre found in tissue which can be related to the diseases which will be described later, cannot be stated with confidence at this stage. In macerated specimens of dried lung 10^6 fibres per gramme can be found without evidence of disease; in cases of asbestosis the count is usually over 10^4 . With a light microscope seldom less than 250 000 fibres per gramme lung tissue are found in cases of asbestosis.

2. PLEURAL PLAQUES AND DIFFUSE PLEURAL FIBROSIS

The presence of circumscribed areas of fibrous thickening below the mesothelium on the lower portion of the chest wall, over the diaphragm or on the pericardium are characteristic of exposure to fibrous mineral dusts. These plaques may be extensive, are leaf-shaped, often bilateral and have an irregular embossed surface. They consist of woven collagen fibres and as they mature become acellular and avascular. This avascularity leads to necrosis and sometimes to the gradual deposition of calcium in the lesions (Meurman, 1966). It can take 20 years or more for sufficient calcium to be deposited for the plaques to become radio-opaque and visible on chest radiography. Therefore, the plaques are seen much more frequently by the pathologists at necropsy than by the radiologist. In some cases there is generalised pleural fibrosis, leaving the lungs *en cuirasse* completely sheathed in a thick layer of fibrous tissue. Unlike plaques, generalised pleural thickening can restrict the expansion of the lungs and cause breathlessness.

3. ASBESTOSIS

Asbestosis is a slowly progressive and persistent interstitial fibrosis of the lung associated with the inhalation of asbestos dust and characterised by asbestos bodies and fibres in large numbers in the tissue. If sufficient dust has been retained, the individual lesions in the alveoli join up until the individual acini become linked in a fibrous mesh, the process starting at the base of the lung and gradually spreading upwards. This process is fairly well established before there is recognition on radiological or by physiological examination, the latter often being obscured by the effects of cigarette smoking. If exposure has been sufficient the disease will progress after the worker has left the industry (Becklake et al, 1979).

4. CANCER OF THE LUNG

Carcinoma of the bronchus is a frequent cause of death among workers with radiological evidence of asbestosis (Liddell and McDonald, 1980). The risk of a cigarette smoking worker heavily exposed to asbestos developing lung cancer is 25 to 50 times greater than an age matched non-smoker who has not worked with asbestos (see J. C. McDonald, 1980). Initially the carcinomas reported in the asbestos workers were peripherally situated, with adenocarcinomas being the most common (Buchanan, 1965). With the increase in cigarette smoking, all types of endobronchial tumours are being seen, but the number of adenocarcinomas is still more frequent than in non-exposed cigarette smokers (Kannerstein and Churg, 1972).

of fibre; apart from mining areas, pure exposure is rare. South African experience with crocidolite has been repeated on a smaller scale at Wittenoom in Western Australia, where mesotheliomas have occurred, both in those employed in the mines and in the environmentally exposed population (Hobbs et al, 1980). Nothing comparable has been reported for chrysotile, amosite or anthophyllite mining. The gas mask workers investigated by Jones et al (1976) and some of those by McDonald and McDonald (1978) appeared to have had a pure exposure. Pooley's analysis of the lungs of the Nottingham cases also showed significant amounts of chrysotile, but not more than is found in autopsy material generally. The technique developed by Pooley (1975) for the identification of asbestos and other mineral fibres in lung tissue is the most useful method available for identifying individual exposures and the complexity of the situation gives emphasis to the need for the parallel examination of appropriate controls. The comparisons made between the fibres in the lungs of the mesothelioma cases and controls in Britain by Jones et al (1980) when compared with those obtained by A. D. McDonald (1980) in the United States and Canada has shown that chrysotile fibres are found in considerable quantity equally in cases and controls. In Britain, crocidolite and to a lesser extent amosite were associated with mesothelioma, whereas in the USA it was predominantly amosite and less often crocidolite. Selikoff et al (1972) found a considerable excess of mesotheliomas in factory workers exposed to amosite but has not reported on the fibre content of their lungs. The Advisory Committee to the Secretary of State for Employment (Health and Safety Commission, 1979) concluded that in the causation of mesotheliomas, crocidolite was more dangerous than chrysotile but that amosite might be intermediate between the two.

Experimental evidence has complicated the situation by showing that many types of mineral fibre can cause mesothelioma. This evidence has come in the main from intrapleural implantation studies which were initiated by Wagner (1962) and continued in collaboration with Stanton and Wrench (1972); Pott et al (1972) undertook similar intraperitoneal investigations. This work has indicated that the size of the fibres was more important than their nature.

IMPORTANCE OF FIBRE SIZE

The significance of the physical characteristics of fibres in explaining the biological effects of asbestos was first emphasised by Timbrell (1965). He demonstrated that diameter was the most important factor in determining whether a fibre would be inhaled. The finer the fibre the more easily would it reach the lung parenchyma. Later, Timbrell et al (1970) showed that this could be applied to the amphiboles and that the ultimate diameter of crocidolite was less than that of amosite. Although the individual fibres of chrysotile have a diameter less than that of crocidolite, they occur in a woven coil formation, the total diameter of which affects its aerodynamic behaviour. Thus chrysotile behaves as a coarse fibre and finds difficulty in reaching the pleural surfaces through the peripheral airways. However, chrysotile fibres in aqueous solution can divide longitudinally into fibrils which under some circumstances are straight and have similar properties to very fine amphiboles. The typical electron microscopic appearance of asbestos fibres is shown in Figure 1.1.

Calculations and experiments with casts of the lower respiratory tract showed that fibres up to $3.0\text{ }\mu\text{m}$ in diameter would reach the respiratory bronchioles. The length of fibres most likely to cause fibrosis would appear to be greater than $10\text{ }\mu\text{m}$ (Timbrell

At this stage we must assume that all mineral fibres of similar size range are potentially hazardous to man, whether of asbestos or other type.

Asbestos minerals as potential environmental contaminants

Under this heading the following situations will be considered:

1. asbestiform minerals contaminating banded ironstone
2. tremolite as a contaminant of other mineral deposits
3. possible contamination of agricultural soil.

Asbestiform minerals contaminating banded ironstone

Although it has been known for many years that banded ironstone deposits frequently contain small seams of fibrous silicates, occasionally the fibrous deposits are large, and may then be the source of amphibole asbestos as exploited in South Africa and Australia. Other deposits occur which are of no commercial value, for example, there are the taconite fibres in the Mesabi Range on the shores of Lake Superior. Although it has been shown that iron ore mining in this region is causing both contamination of the atmosphere and the water of the lake, no evidence of a hazard to man has been established. All the fibres are below $5.0\ \mu\text{m}$ in length. In a large South Dakota gold mine the ore-bearing rock was cummingtonite — grunerite (a close relation of amosite) and some fibres were found in the dust. It was suggested that these fibres were associated with an increased incidence of carcinoma of the lung, but these findings were not confirmed by the investigations of McDonald et al (1978). Nevertheless, the possibility that hazardous fibres may sometimes be released from iron deposits in the vicinity of amphibole mines remains.

Tremolite as a contaminant of other mineral deposits

The fibre dimensions of tremolite vary even more between deposits than the other amphiboles. A coarse flake-like tremolite occurs as a contaminant of talc in California; this material does not cause tumours when implanted intrapleurally in rats, and there is no published evidence of disease among the miners. A coarse fibrous tremolite is found as a contaminant of the chrysotile deposits in Quebec Province in Canada. This fibre has been found in the lungs of miners and millers from these mines (Pooley, 1976); whether it plays any part in the etiology of pleural plaques and pulmonary fibrosis is still uncertain. In the talc mines in the northern part of New York State there is contamination by a finer fibrous tremolite, and a few mesotheliomas and carcinomas of the lung have been reported among the miners (Kleinfeld et al, 1967). Practically pure tremolite of a coarser type has been used in Eastern Turkey for stuccoing houses. Yazicioglu (1976) found a significant incidence of pleural plaques among the inhabitants. Tremolite with very fine long fibres has been mined in South Korea (see Fig. 1.1); this fibre has been shown to produce a high incidence of mesotheliomas in experimental animals. We have been informed that the mining operations have now been suspended because of suspected cancer among the workers.

Possible contamination of agricultural soil

A further source of fibrous mineral exposure only recently appreciated, may prove of consequence. Evidence is still fragmentary, and the only confirmed situation is in Bulgaria, where Burilkov and Michailova (1970; 1972) found pleural plaques in



GLASS WOOL



ROCK WOOL

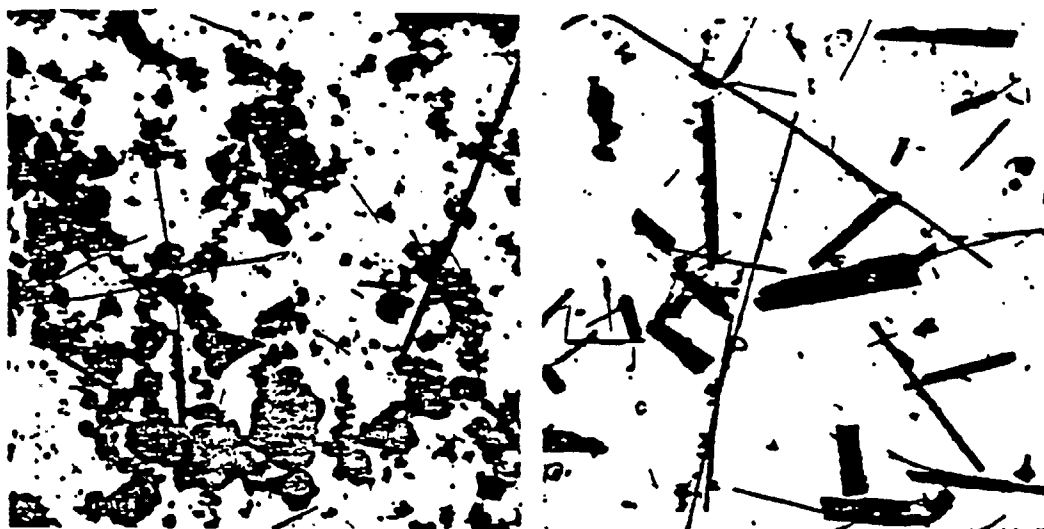


GLASS FIBRE (CODE 100)

—
10 μm

Fig. 1.2 Electron micrographs of synthetic vitreous fibres

particularly on factory floors, and occasionally for agricultural and pharmaceutical purposes. Information is scanty concerning the size and shape which particles of these fibrous minerals assume under a range of potentially relevant natural and artificial conditions; nor is much yet known about their biological activity in various *in vitro* tests. Animal experiments have been recently initiated in which sepiolite and attapulgite fibres are inhaled or implanted intrapleurally; the results will not be



TURKISH FIBRE

ERIONITE FROM USA

Fig. 1.3 Electron micrographs of dispersed samples of synthetic and naturally occurring fibrous erionite

To date, synthetic substitutes for asbestos (the man-made vitreous and ceramic fibres) do not appear to have caused lung fibrosis, lung cancer or mesothelioma in man. However, animal experiments indicate that it would be unwise to create materials which include fibres smaller than $0.5 \mu\text{m}$ in diameter if the risk of mesothelioma is to be avoided.

Experimental work suggests that both synthetic and natural non-asbestos mineral fibres of less than $0.5 \mu\text{m}$ in diameter and greater than $8 \mu\text{m}$ in length may be hazardous. Some fibrous clays have already been shown to cause mesothelioma experimentally while epidemiological evidence suggests that fibrous erionite may well have been responsible for a very high incidence of mesotheliomas in man.

Changes in industry and commerce are leading to human exposure from a variety of natural and synthetic mineral fibres. Further research is urgently needed to avoid replacing the hazard of asbestos with others as serious.

REFERENCES

- Artvinli M, Baris Y I 1979 Malignant mesotheliomas in a small village in the Anatolian region of Turkey: an epidemiological study. *Journal of the National Cancer Institute* 63: 17-22
- Baris Y I et al 1978 An outbreak of pleural mesothelioma and chronic fibrosing pleurisy in the village of Karain/Urgup in Anatolia. *Thorax* 33: 181-192
- Becklake M R, Liddell F D K, Manfreda J, McDonald J C 1979 Radiological changes after withdrawal from asbestos exposure. *British Journal of Industrial Medicine* 36: 23-28
- Buchanan W D 1965 Asbestosis and primary thoracic neoplasms. *Annals of the New York Academy of Sciences* 132: 507-518
- Burilkov T, Michailova L 1970 Asbestos content of the soil and endemic pleural asbestosis. *Environmental Research* 3: 443-451
- Burilkov T, Michailova L 1972 Über den Sepiolitgehalt des Bodens in Gebieten mit Endemischem Pleuraverkalkungen. *Internationales Archiv für Arbeitsmedizin* 29: 95-101

- faserförmiger Staube und ihre Deutung im Hinblick auf die Tumorentstehung beim Menschen. Zentralblatt für Bakteriologie, Parasitenkunde, Infektionskrankheiten und Hygiene: erste Abteilung: Originale, Reihe B: Hygiene, Präventive Medizin 162: 467-505
- Pott F, Huth F, Friedrichs K H 1972 Tumoren der Ratte nach i.p. Injektion von gemahlenem Chrysotil und Benzo (a) pyren. Zentralblatt für Bakteriologie, Parasitenkunde, Infektionskrankheiten und Hygiene: erste Abteilung: Originale, Reihe B: Hygiene, Präventive Medizin 155: 463-469
- Selikoff I J, Hammond E C, Chung J 1972 Carcinogenicity of amosite asbestos. Archives of Environmental Health 25: 183-186
- Stanton M F, Layard M, Tegeris A, Miller E, May M, Kent E 1977 Carcinogenicity of fibrous glass: pleural response in the rat in relation to fiber dimension. Journal of the National Cancer Institute 58: 587-603
- Stanton M F, Wrench C 1972 Mechanisms of mesothelioma induction with asbestos and fibrous glass. Journal of the National Cancer Institute 48: 797-821
- Stell P M, McGill T 1973 Asbestos and laryngeal carcinoma. Lancet 2: 416-417
- Thomson J G, Kaschula R O C, MacDonald R R 1963 Asbestos as a modern urban hazard. South African Medical Journal 37: 77-81
- Timbrell V 1965 The inhalation of fibrous dusts. Annals of the New York Academy of Sciences 132: 255-273
- Timbrell V, Pooley F D, Wagner J C 1970 Characteristics of respirable asbestos fibres. In: Shapiro H A (ed) Pneumoconiosis: Proceedings of the International Conference Johannesburg 1969. Cape Town Oxford University Press, London, pp 120-125
- Timbrell V, Rendall R E G 1971/2 Preparation of the UICC standard reference samples of asbestos. Powder Technology 5: 279-287
- Timbrell V, Skidmore J W 1968 Significance of fiber length in experimental asbestosis. In: Holstein E (ed) Internationale Konferenz der biologische Wirkungen des Asbestos, Dresden. Dtsch. Zentralinst, Arbeitsmed, Berlin, DDR pp 52-56
- Wagner J C 1962 Experimental production of mesothelial tumours of the pleura by implantation of dusts in laboratory animals. Nature 196: 180-181
- Wagner J C 1979 Diseases associated with exposure to asbestos dusts. The Practitioner 223: 28-33
- Wagner J C, Berry G, Pooley F D 1980b Carcinogenesis and mineral fibres. British Medical Bulletin 36: 53-56
- Wagner J C, Berry G, Skidmore J W 1976 Studies of the carcinogenic effects of fiber glass of different diameters following intrapleural inoculation in experimental animals. In: Occupational exposure to fibrous glass — proceedings of a symposium. (DHEW Publication no (NIOSH) 76-151.) U.S. Department of Health, Education and Welfare, Washington DC, pp 193-197
- Wagner J C, Sleggs C A, Marchand P 1960 Diffuse pleural mesothelioma and asbestos exposure in the North Western Cape Province. British Journal of Industrial Medicine 17: 260-271
- Wagner J C, Berry G, Skidmore J W, Timbrell V 1974 The effects of the inhalation of asbestos in rats. British Journal of Cancer 29: 252-269
- Wagner J C, Berry G, Hill R J, Munday D E, Skidmore J W 1980a Animal experiments with man-made mineral fibres. In: Wagner J C (ed) Biological effects of mineral fibres. WHO/IARC, Lyon, pp 361-362
- Webster I 1973 Asbestos and malignancy. South African Medical Journal 47: 165-171
- Yazicioglu S 1976 Pleural calcification associated with exposure to chrysotile asbestos in south east Turkey. Chest 70: 43-47
- Yazicioglu S, Ilcayto, R, Balci K, Sayli B S, Yorulmaz B 1980 Pleural calcification, pleural mesotheliomas and bronchial cancers caused by tremolite dust. Thorax 35: 564-569



DEPARTMENT OF THE ARMY
U. S. ARMY ENVIRONMENTAL HYGIENE AGENCY
ABERDEEN PROVING GROUND, MARYLAND 21010-6422

JAN 30 1985

REPLY TO
ATTENTION OF

Occupational and Environmental
Medicine Division

William V. Loscutoff
Chief, Toxic Pollutants Branch
California Air Resources Board
P.O. Box 2815
Sacramento, California 95812

Dear Mr. Loscutoff:

This Agency does not have data on asbestos per your request. However, we are aware of a document on the health effects of asbestos which you may want to review. The title is: Asbestos - An Update of Epidemiology and Pathology since 1976. This document was published by the USAF Occupational and Environmental Health Laboratory, Aerospace Medical Division, Brooks Air Force Base, Texas in February 1984. A copy can be obtained by requesting report number 84-125C011BOB from either the National Technical Information Service or the Defense Technical Information Center.

Any questions concerning this reply should be directed to Major Robert W. Petzold, M.D. at telephone number 301-671-2464.

Sincerely,

for Robert W. Petzold
Noel C. Gaydos
Colonel, Medical Corps
Director, Occupational and
Environmental Health

Attachment II

SUGGESTED ISSUES WHICH SHOULD BE CONSIDERED IN PREPARING THE HEALTH EFFECT DOCUMENT ON ASBESTOS FIBERS

Three publications may be useful for providing the background information on the health effects of asbestos (IARC, 1977; EPA review draft, 1984; and NRC, 1984; see attached reference list for full references). The IARC publication (1977) provides an adequate summary of asbestos health effects through 1976. The other two publications provide summary reviews on more recent health information and quantitative risk estimates of lung cancer and mesothelioma. Based on its evaluation of the health effects of asbestos, IARC has found "sufficient evidence" for carcinogenicity to humans and animals. Evidence for activity in short-term tests has been found to be "inadequate" (IARC, 1982).

The following is an outline of issues in the format desired by the SRP:

I. Asbestos As a Toxic Air Contaminant

- A. Asbestos is a generic name for a category of fibrous minerals that are used commercially. In EPA's National Emission Standard for Asbestos, the minerals defined as asbestos are actinolite, amosite, anthophyllite, chrysotile, crocidolite and tremolite. Because of the provisions of Article 39655 of the Health and Safety Code, it will be important to include at least those minerals in the California definition of toxic air contaminants.
- B. The physical properties of fibers such as respirability, size and aspect ratio, durability, flexibility and tensile strength, surface area and surface charge have been considered in relationship to observed health effects of asbestos minerals. A discussion of these relationships of fiber characteristics to health effects of asbestos will be very useful in eventually establishing control approaches.
- C. Evidence of carcinogenicity of several asbestiform fibers has been demonstrated in occupational studies, in studies of other human populations and in animal studies. Because the evidence of carcinogenicity is so strong in humans and animals it is unlikely

that other toxic effects need to be considered in order to list asbestos as a toxic air contaminant.

II. Threshold Determination of Asbestos

- A. There is no strong positive experimental or epidemiological evidence that asbestos has a carcinogenic threshold. Therefore, it should be treated as a substance without a threshold.
- B. Because of the evidence that asbestos is not directly genotoxic, it might be argued that asbestos is likely to have a threshold. Therefore, the possibility that a single fiber of asbestos is capable of producing cancer would appear to need discussion.

III. Dose-Response Estimates of the Carcinogenicity of Asbestos

Choices and related considerations are as follows:

- A. The choice of using data from human epidemiologic studies or animal studies or both.
- B. The choice of using lung cancer or mesothelioma or both as the biological endpoints for the carcinogenic effects of asbestos.
- C. The choice of using one or more of the different types of asbestos as the causative agents.
- D. The choice of using one or more of the indices for quantifying the dose. For example: mass or number of fibers or fibers at specific size distributions or total surface areas.
- E. The establishment of a method or factor to relate the fiber counts and size distribution obtained from optical and electron microscopy measurements.
- F. The establishment of a method or factor to relate fiber counts to mass measurements.

- G. The synergistic effect of asbestos fibers and cigarette smoke or benzo(a)pyrene may be considered as an additional risk.
- H. An estimation of the potential risk related to indoor air exposure to asbestos.
- I. An estimation of the potential risk associated with asbestos exposure via drinking water.

References:

EPA Review Draft (1984) Asbestos Health Assessment Update. U.S.E.P.A. report (EPA-600/8-84-003A), 133pp.

IARC (1982) IARC Monographs on The Evaluation of The Carcinogenic Risk of Chemicals to Humans. IARC Monography Supplement 4, P.52-53. World Health Organization.

IARC (1977) IARC Monographs on The Evaluation of Carcinogenic Risk of Chemicals to Man--Asbestos. Vol. 14, 106pp. World Health Organization.

NRC (1984) Asbestiform Fibers--Nonoccupational Health Risks. Committee on Nonoccupational Health Risks of Asbestiform Fibers, National Research Council, National Academy Press, 334pp.

Attachment III

Ambient Asbestos Concentrations, Asbestos Sources and Emission Trends

Data from a study done under contract to the Air Resources Board, entitled Final Report - Ambient Asbestos Concentrations in California, Volume 1, (December 1983), show mean concentrations of total asbestos fibers in the range of 9,500-66,000 fibers/cubic meter. The study also shows mean concentrations of chrysotile fibers in the range of 4,500-51,000 fibers/cubic meter, and mean concentrations of amphibole fibers in the range of below the detection limit to 31,000 fibers/cubic meter. As further discussed in Attachment IV, the sampler used has a collection efficiency of 50 percent for 3.5 um aerodynamic diameter particles.

Of the reports containing measured ambient asbestos concentrations in California, we used the results from the above referenced study because it was the only one containing measurements from several populated locations in the State and because the samples were analyzed by transmission electron microscopy. Although we believe this data represents the best available, it is not possible to estimate an annual average concentration from these measurements with an acceptable degree of accuracy.

Sources that lead to community exposure to asbestos are:

- o asbestos mining and milling operations;
- o floor= tile production;
- o gaskets and packing production;
- o manufacturing of friction products;
- o paints, coatings and sealants production;
- o asbestos reinforced plastics production;
- o asbestos cement pipe production;
- o asbestos textiles production;
- o asbestos paper production; and
- o asbestos cement sheet production.

We are currently investigating the asbestos mining/milling and manufacturing/fabricating source categories in California in order to estimate asbestos releases to the atmosphere. Other potential sources contributing to community exposure to asbestos are:

- o Natural weathering or human disturbance of mineral deposits (off-road vehicles, road-building);
- o Reentrainment of asbestos fibers that have settled out by dry deposition;
- o Transportation (consumption of brake and clutch linings);

- o Demolition operations; and
- o Fiber loss from unpaved roads surfaced with crushed serpentinite (contains chrysotile) as a result of vehicular traffic.

Asbestos fiber production at the mines and mills in California decreased from 1979 to 1983.^{1/} National consumption of asbestos decreased from 1977 to 1982.^{2,3/} We are determining whether this trend has occurred in manufacturing/fabricating source categories in California. At the present time, we are unable to assess the emission trends of the potential sources.

^{1/} Telephone conversation with Fred Carrillo, U.S. Bureau of Mines, January 1985.

^{2/} Draft Final Report-Exposure Assessment For Asbestos prepared by Versar Inc. for the U.S. Environmental Protection Agency, January 9, 1984, pg. 24.

^{3/} Asbestiform Fibers-Nonoccupational Health Risks by the Committee on Nonoccupational Health Risks of Asbestiform Fibers, National Research Council, National Academy Press, 1984, pg. 56.

Attachment IV

= Sampling and Analytical Methods

The goal of Science Applications, Inc. (SAI) project was to establish "worst case" respirable asbestos concentrations in a representative cross-section of California locations. Given the project objective, sampling and analysis methods focused on applying state-of-the-art sampling and analysis techniques to a cross-section of geographical sites based on emission/exposure potential. To examine maximum asbestos level conditions, sampling was done during the dry period of the year. Sampling methods adopted for the study followed protocols established by Dr. Walter John of the Air and Industrial Hygiene Laboratory in 1976, 1978, and 1980. The samples were analyzed by transmission electron microscopy according to the measurement and verification procedures specified by the U.S. Environmental Protection Agency (EPA) in their provisional method (1978).

Since previous SAI surveys using electron microscopy showed that 90 to 95 percent of asbestos fibers in the ambient environment are shorter than 5.0 μm , a single-point cyclone sampler with a collection efficiency of 50 percent for 3.5 μm aerodynamic diameter particles at a flow rate of 15.5 l/minute was chosen for all filter sampling. An 8.0 μm pore size 47 mm diameter Millipore backing filter was used in conjunction with a Nuclepore 0.2 μm pore size collection filter. The backing filter

was used to ensure even distribution of particles across the filter face.

=

The project was intended to measure airborne asbestos concentrations in areas that are known or are suspected to have elevated levels and in those that are isolated from asbestos sources. An additional site criterion was to co-locate, where possible, field measurements with existing particulate matter monitoring stations. Ten sampling sites were chosen: Napa, San Jose, Stockton, Sonora, King City, Bakersfield, San Fernando Valley, San Diego, South Gate and Century City.

Original sampling plans called for collection of at least four four-hour samples at each site. Paired replicate samples were taken morning and afternoon at King City, San Jose, Napa, San Fernando Valley, and Sonora. Century City, Bakersfield, South Gate and San Diego normally have high particulate concentration levels, so samples were collected at these locations over shorter time intervals to preclude overloading the filters. At these sites, SAI took five to nine samples per day. Sampling time was based on prior calibrations of filter loading, as a function of suspended particle count levels, by scanning electron microscopy.

A JEOL 6C transmission electron microscope (TEM), with a resolution of six angstroms and equipped with selected area

electron diffraction was used for all sample analyses. Calibration of instrument magnification was performed using a 21,400 lines/inch carbon replicate standard against a scale etched on the fluorescent screen of the TEM.

In reporting the analytical results, values shown as zero are below the detection limit. Detection limit is defined as a function of the total area of the filter scanned and the volume of air that is sampled. When only one fiber was found on the sample scanned, the value obtained was used to derive the detection limit. Samples having less than one fiber found during analysis were defined as being below the detection limit. All the samples analyzed were counted in an identical manner and had been collected at the same flow rate, therefore, the detection limit was a function only of the duration of sampling time. Since sampling times ranged from one to four hours depending on mass concentration, net detection limits ranged from 9100 to 2400 fibers/m³ of air, respectively.

To fulfill quality assurance requirements, the University of Washington Transmission Electron Microscopy Center was provided with replicate filter samples for an interlaboratory comparison. The samples were analyzed according to the EPA provisional method and the data returned to SAI for computer reduction. In addition to a background control (Napa site),

sites selected for inter-comparison analyses were King City, Sonora, Century City, and San Diego. The Napa sample was done in duplicate by both laboratories to provide or measure reproducibility. The detection limit for TEM asbestos analysis in this study was approximately 2400 fibers/m³, the result of counting only one fiber in the filter area analyzed. The greatest difference between comparative samples was for the duplicated background control samples done by the University of Washington. Although values for these two replicates are a factor of 11 apart, the difference represents the counting of four fibers vs. one fiber per total counting area, respectively. Differences between the two laboratory's analytical results for the other replicate samples are in all cases less than the variation in the single duplicate analysis.

Appendix B
Information Request
and
Public Responses

D 004311

AIR RESOURCES BOARD

1102 Q STREET
P.O. BOX 2815
SACRAMENTO, CA 95812



December 7, 1984

Dear Sir or Madam:

Subject: Request for Information Regarding Asbestos

I am writing to request information on the health effects of asbestos (e.g., chrysotile, crocidolite, actinolite-tremolite, amosite, anthophyllite, etc.) as part of our toxic air contaminant program. This program is based on Health and Safety Code Sections 39650, et seq. which require the ARB to identify compounds as toxic air contaminants and once identified to develop and adopt control measures for such compounds. After consultation with the staff of the Department of Health Services (DHS), we have selected asbestos as a candidate toxic air contaminant to be evaluated in accordance with the provisions of Health and Safety Code Sections 39650, et seq.

Before the ARB can formally identify a compound as a toxic air contaminant, several steps must be taken. First, the ARB must request the Department of Health Services to evaluate the health effects of candidate compounds. Second, the ARB staff must prepare a report which includes the health effects evaluation and then submit the report to a Scientific Review Panel for its review. The report submitted to the Panel will be made available to the public. Information submitted in response to this request will be considered in the ARB report to the Panel. Although any person may also submit information directly to the Panel for its consideration, I urge you to submit all information at this time for our consideration in the development of the report for the Panel. The Panel reviews the sufficiency of the information, methods, and data used by the DHS in its evaluation. Lastly, after review by the Scientific Review Panel, the report with the written findings of the Panel will be considered by the Air Resources Board and will be the basis for any regulatory action by the Board to officially identify a compound as a toxic air contaminant.

Prior to formally requesting the DHS to prepare a health effects evaluation of asbestos, we are providing, pursuant to the provisions of Section 39660(e) of the Health and Safety Code, an opportunity to interested parties to submit information on the health effects of asbestos which they believe would be

December 7, 1984

important in DHS's evaluation of asbestos as a candidate toxic air contaminant.

In late October 1984, ARB staff received a reference search on health effects of asbestos using the Toxline, Medline, and Biosis databases available from NLM and Dialog Information Services. The search was limited to material available to the public between January, 1980 and July, 1984. The attached bibliography lists the references from this information search. We are requesting pertinent information on the health effects of asbestos, including any material that may not be available to the public, that is not included in the attached bibliography.

Pursuant to the provisions of the Public Records Act (Government Code Sections 6280 et seq.), the information you provide will be a public record and subject to public disclosure, except for trade secrets which are not emission data or other information which is exempt from disclosure or the disclosure of which is prohibited by law. The information may also be released to the Environmental Protection Agency, which protects trade secrets and confidential information in accordance with federal law, and to other public agencies, which are also required to protect such information.

To expedite the review process, we ask that any information which you believe should be regarded as "trade secret" be clearly marked and separated from other information. You may identify portions of the information you submit as "trade secret" in accordance with Health and Safety Code Section 39660(e). The claim of trade secrecy must be supported upon the request of the Air Resources Board. Other information claimed to be trade secret and information otherwise claimed to be exempt from disclosure may be identified as confidential in accordance with Section 91011, Title 17, California Administrative Code. Section 91011 requires that the claim of confidentiality be accompanied by specified supporting information.

I would appreciate receiving any relevant information you wish to submit by January 21, 1985. Your help in expediting our review will be greatly appreciated. Please send the information to the attention of:

William V. Loscutt, Chief
Toxic Pollutants Branch
Re: Asbestos
California Air Resources Board
P. O. Box 2815
Sacramento, CA 95812

If you have any further questions regarding health effects information, please contact Mr. John Batchelder at (916) 323-1505. For any other questions, please contact Mr. Robert Barham at (916) 322-7072.

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If you are not the person to whom this request should be addressed, please forward it to the appropriate person in your organization. Also, please let us know whether you would like to continue to receive information inquiries for other candidate compounds, and if not, if there is anyone in your organization to whom such requests should be sent.

Sincerely,



Peter D. Venturini, Chief
Stationary Source Division

cc: Alex Kelter, DHS
Lori Johnston, DFA
Wayne Morgan, President, CAPCOA
Jan Bush, Executive Secretary, CAPCOA
David Howekamp, EPA Region IX
Assemblywoman Sally Tanner
Senator Ralph Dills
APCOs
Emil Mrak, Chairman, and
Scientific Review Panel Members

Attachment

ASBESTOS REFERENCES (11/9/84)

1. Acheson, E. D., Bennett, C., Gardner, M. J. and Winter, P. D. (1981) Mesothelioma in a factory using amosite and chrysotile asbestos. *Lancet*. 2(8260-61):1403-1406.
2. Acheson, E. D. and Gardner, M. J. (1980) Possible synergism between chrysotile and amphibole asbestos (Letter). *Lancet*. 1(8170):706.
3. Acheson, E. D. and Gardner, M. J. (1981) Dose-response relations from epidemiological studies. *Proc R Soc London, (Ser.) A*. 376(1764):79-85.
4. Acheson, E. D., Gardner, M. J., Pippard, E. C. and Grime, L. P. (1982) Mortality of two groups of women who manufactured gas masks from chrysotile and crocidolite asbestos: a 40-year follow-up. *Br J Ind Med*. 39(4):344-348.
5. Acheson, E. D., Gardner, M. J., Winter, P. D. and Bennett, C. (1984) Cancer in a factory using amosite asbestos. *Int J Epidemiol*. 13(1):3-10.
6. Aisner, J. and Wiernik, P. H. (1981) Asbestos-related neoplasm. *Semin Oncol*. 8(3):241-242.
7. Archer, V. E. (1979) Carcinogenicity of fibers and films: a theory. *Med Hypotheses*. 5(11):1257-1262.
8. Archer, V. E. and Rom, W. N. (1983) Trends in mortality of diffuse malignant mesothelioma of pleura (Letter). *Lancet*. 2(8341):112-113.
9. Armstrong, E. K. et al (1984) Epidemiology of malignant mesothelioma in Western Australia. *Med J Aust*. 141(2):86-88.
10. Artvinli, M. and Baris, Y. I. (1982) Environmental fiber-induced pleuro-pulmonary diseases in an Anatolian village: an epidemiologic study. *Arch Environ Health*. 37(3):177-181.
11. Artvinli, M. and Baris, Y. I. (1982) Environmental fiber induced pleuro pulmonary diseases in an anatolian Turkey village-an epidemiologic study. *Arch Environ Health*. 37(3):177-181.
12. Arul, K. J. and Holt, P. F. (1980) Clearance of asbestos bodies from the lung: a personal view. *Br J Ind Med*. 37(3):273-277.
13. Barry, B. E. et al (1983) Reaction of rat lungs to inhaled chrysotile asbestos following acute and subchronic exposures. *Exp Lung Res*. 5(1):1-22.
14. Beck, E., Konetske, G. W. and Strum, W. (1982) Asbestos and mesothelioma in east Germany. *Arch Immunol Ther Exp*. 30(3-4):229-234.
15. Beck, E. G. (1980) Experimental pathology-in vitro studies-related to asbestos and other mineral fibers. *IARC Sci Publ*. 1(30):385-400.
16. Becklake, M. R. (1982) Exposure to asbestos and human disease (editorial). *N Engl J Med*. 306(24):1486-1487.
17. Becklake, M. R. (1982) Asbestos-related diseases of the lungs and pleura: current clinical issues. *Am Rev Respir Dis*. 126(2):157-164.

18. Begin, R. et al (1981) Early lung events following low-dose asbestos exposure. *Environ Res.* 26(2):392-401.
19. Berry, G. and Newhouse, M. L. (1983) Mortality of workers manufacturing friction material using asbestos. *Br J Ind Med.* 40(1):1-7.
20. Bignon, J. et al. (1979) Human and Experimental Data on Translocation of Asbestos Fibers through the Respiratory System. *NY Academy of Science.* 745-50.
21. Bignon, J. and Jaurand, M. C. (1983) Biological in vitro and in vivo responses of chrysotile versus amphiboles. *Environ Health Perspect.* 51:72-80.
22. Blot, W. J. et al. (1980/September 1980) Lung and Laryngeal Cancers in Relation to Shipyard Employment in Coastal Virginia. *JNCI.* 65(3):571-5.
23. Bolton, R. E. et al (1982) Variations in the carcinogenicity of mineral fibers. *Ann Occup Hyg.* 26(1-4):569-582.
24. Bolton, R. E. et al (1982) Variations in the carcinogenicity of mineral fibers. *Ann Occup Hyg.* 26 (1-4):569-582.
25. Boman, G. et al (1982) Malignant mesothelioma in Turkish immigrant residing in Sweden. *Scand J Work Environ Health.* 8(2):108-112.
26. Briselli, M., Mark, E. J. and Dickerson, G. R. (1981) Solitary fibrous tumors of the pleura: eight new cases and review of 360 cases in the literature. *Cancer.* 47(11):2678-2689.
27. Brody, A. R. and Hill, L. H. (1981) Deposition pattern and clearance pathways of inhaled chrysotile asbestos. *Chest.* 80(1 Suppl):64-67.
28. Brown, R. C., Poole, A. and Fleming GTA (1983) The influence of asbestos dust on the oncogenic transformation of C-3H-10T-1-2 cells. *Cancer Lett.* 18(2):221-228.
29. Browne, K. (1983) The epidemiology of mesothelioma. *J Occup Med.* 33(4):190-194.
30. Browne, K. (1983) Asbestos-related mesothelioma: epidemiological evidence for asbestos as a promoter. *Arch Environ Health.* 38(5):261-266.
31. Browne, K. and Goffe, T. (1984) Mesothelioma due to domestic exposure to asbestos (Letter). *Br Med J (Clin Res).* 289(6437):110-111.
32. Browne, K. and Smither, W. J. (1983) Asbestos-related mesothelioma: factors discriminating between pleural and peritoneal sites. *Br J Ind Med.* 40(2):145-152.
33. Casey, G. (1983) Sister-chromatid exchange and cell kinetics in CHO-K1 cells, human fibroblasts and lymphoblastoid cells exposed in vitro to asbestos and glass fiber. *Mutat Res.* 116(3-4):369-377.
34. Chovil, A. and Stewart, C. (1979) Latency period for mesothelioma (Letter). *Lancet.* 2(8147):853.
35. Churg, A. M. and Warnock, M. L. (1981/March 1981) Asbestos and Other Ferruginous Bodies, Their Formation and Clinical Significance. *Am J Pathol.* 102(3):447-56.
36. Churg, A. and Golden, J. (1982) Current problems in the pathology of asbestos-related disease. *Pathol Annu.* 17(Pt 2): 35-66.

37. Churg, A. and Wiggs, B. (1984) Fiber size and number in amphibole asbestos induced mesothelioma. *Am J Pathol.* 115(3):437-442.
38. Churg, A. and Wood, P. (1983) Observation on the distribution of asbestos fibers in human lungs. *Environ Res.* 31(2):347-380.
39. Cook, P. M., Palekar, L. D. and Coffin, D. L. (1982) Interpretation of the carcinogenicity of amosite asbestos and ferro actinolite on the basis of retained fiber dose and characteristics in vivo. *Toxicol Lett.* 13(3-4):151-158.
40. Craighead, J. E. and Mossman, B. T. (1982) The Pathogenesis of Asbestos-Associated Diseases. *New England Journal of Medicine.* 306(24):1446-55.
41. Davis, J. M. (1979) The Use of Animal Models and Primary Prevention of Cancer. *NY Acad of Science.* 330:795-8.
42. DiPaolo, J. A., DeMarinis, A. J. and Doniger, J. (1983) Asbestos and Benzo(a)pyrene Synergism in the Transformation of Syrian Hamster Embryo Cells. *Pharmacology.* 27(2):65-73.
43. Dodson, R. F. et al (1984) Asbestos Content in Lungs of occupationally exposed individual. *JAMA.* 252(1):68-71.
44. Donham, K. J. et al (1980) The effects of long-term ingestion of asbestos on the colon of F344 rats. *Cancer.* 45(5 Suppl):1073-1084.
45. Englund, A. and Engholm, G. (1982) Asbestos related cancer in Swedish construction workers. *Arch Immunol Ther Exp.* 30(3-4):157-160.
46. Enterline, P. E. (1983) Risk assessment of asbestos carcinogenicity in nonoccupationally exposed populations. *VDI-Ber.* 475:297-303.
47. Enterline, P. E. (1983) Cancer produced by nonoccupational asbestos exposure in the United States. *J Air Pollut Control Assoc.* 33(4):318-322.
48. Enterline, P. E. (1981) Proportion of cancer due to exposure to asbestos. *Banbury Report.* 9:19-36.
49. Epler, G. R., McCloud, T. C. and Gaensler, E. A. (1982) Prevalence and incidence of benign asbestos pleural effusion in a working population. *JAMA.* 247(5):617-622.
50. Findley, M. E. et al (1983) An assessment of the Environmental Protection Agency's (USA) asbestos hazard evaluation algorithm. *Am J Public Health.* 73(10):1179-1181.
51. Finklestein, M. W. (1983) Mortality among long-term employees of an Ontario asbestos-cement factory. *Br J Ind Med.* 40(2):136-144.
52. Finklestein, M. W. (1984) Mortality among employees of an Ontario asbestos-cement factory. *Am Rev Respir Dis.* 129(5):754-761.
53. Fischbein, A. and Rohi, A. N. (1984) Pleural mesothelioma and neighborhood asbestos exposure. Findings from microchemical analysis of lung tissue. *JAMA.* 252(1):86-87.
54. Fleury, J. et al (1983) Interactions of asbestos fibers with hepatocytes in vitro: an ultrastructural study. *Toxicol Lett.* 19(1-2):15-22.
55. Frank, A. L. (1981) Clinical observations following asbestos exposure. *Environ Health Perspect.* 34:27-31.

54. Gardner, M. J., Acheson, E. D. and Winter, P. D. (1982) Mortality from mesothelioma of the pleural during 1968-78 in England and Wales. *Br J Cancer*. 46(1):81-88.
57. Gladfelter, T. (1982) Malignant mesothelioma: an occupation disease (clinical conference). *J Fam Pract*. 14(5):827,830-832.
58. Glickman, L. T. et al (1983) Mesothelioma in pet dogs associated with exposure of their owners to asbestos. *Environ Res*. 32(2):305-313.
59. Goldsmith, J. R. (1982) Asbestos as a systemic carcinogen: the evidence from eleven cohorts. *Am J Ind Med*. 3(3):341-348.
60. Goschicki, J. W. and Indulski, J. A. (1982) The polish studies on occupational exposure to asbestos and its biological effects. *Arch Immunol Ther Exp*. 30(3-4):169-176.
61. Griffia, L. C. et al (1983) Deposition of crocidolite asbestos and glass microfibers inhaled by the Beagle dog. *Am Ind Hyg Assoc J*. 44(3):216-222.
62. Hallenback, W. H. (1983) Asbestos PENETRATION OF THE GASTROINTESTINAL TRACT. *Environ Health Perspect*. 53:153-154.
63. Hallenback, W. H., Markey, D. R. and Dolan, D. G. (1981) Analyses of tissue, blood, and urine samples from a baboon gavages with chrysotile and crocidolite asbestos. *Environ Res*. 25(2):349-360.
64. Hamilton, J. A. (1980) Macrophage stimulation and the inflammatory response to asbestos. *Environ Health Perspect*. 34:69-74.
65. Harington, J. S. (1981) Fiber carcinogenesis: epidemiologic observations and the Stanton hypothesis. *JNCI*. 67(5):977-989.
66. Harvey, G., Page, M. and Dumas, L. (1984) Binding of environmental carcinogens to asbestos and mineral fibers. *Br J Ind Med*. 41(3):396-400.
67. Haugen, A. and Harris, C. (1982) Asbestos carcinogenesis: asbestos interactions and epithelial lesion in cultured human tracheobronchial tissues and cells. *Recent Results Cancer Res (Germany, West)*. 82:32-42.
68. Henderson, D. W. (1982) Asbestos-related pleuropulmonary diseases: asbestosis mesothelioma and lung cancer. *Pathology (Australia)*. 14(3):239-243.
69. Henderson, V. L. and Enterline, P. E. (1979) Asbestos exposure: factors associated with excess cancer and respiratory disease mortality. *Ann NY Acad Sci*. 330:117-126.
70. Hesterberg, T. W. et al (1982) Asbestos induces morphological transformation in Syrian hamster embryo cells in culture. *J Cell Biol*. 95(2 part 2):449A.
71. Hillerdal, G. (1983) Malignant mesothelioma 1982: review of 4170 published cases. *Br J Dis Chest*. 77(4):321-343.
72. Holt, F. F. (1982) Translocation of asbestos dust through the bronchiolar wall. *Environ Res*. 27(2):255-260.
73. Hopkins, J. (1984) Epidemiological studies on ingested asbestos. *Food Chem Toxicol*. 22(1):179-181.
74. Huang, E. L. (1979) Amosite, chrysotile and crocidolite asbestos are mutagenic in Chinese hamster lung cells. *Mutat Res*. 68(3):263-274.

75. Humphrey, E. W. et al (1981) The production of malignant tumors of the lung and pleural in dogs from intratracheal asbestos instillation and cigarette smoking. *Cancer*. 47(8):1974-1979.
76. Huukonen, M. S. (1980) Asbestos and cancer in Finland. *J Toxicol Environ Health*. 6(5-6):1241-1245.
77. Huukonen, M. S. (1982) Asbestos and cancer. *Eur J Respir Dis*. 123:145-152.
78. Jones, J. S. et al (1980) The pathology and mineral content of lungs in cases of mesothelioma in the United Kingdom in 1974. *IARC Sci Publ*. 1(30):187-199.
79. Joseph, L. B. et al (1983) Morphological transformation in vitro of normal human fibroblasts by chrysotile. *Environ Health Perspect*. 51:17-22.
80. Kagan, E. (1981) The alveolar macrophage: immune derangement and asbestos-related malignancy. *Semin Oncol*. 8(3):258-267.
81. Kahn, E. I. et al (1980) Primary pericardial mesothelioma following exposure to asbestos. *Environ Res*. 23(2):270-281.
82. Kannerstein, M. (1980) Recent advances and perspectives relevant to the pathology of asbestos-related diseases in man. *IARC Sci Publ*. 1(30):149-162.
83. Kannerstein, M. and Churg, J. (1980) Mesothelioma in man and experimental animals. *Environ Health Perspect*. 34:31-36.
84. Kaw, J. L., Tilkes, F. and Beck, E. G. (1982) Reaction of cells cultured in vitro to different asbestos dusts of equal surface area but different fiber length. *Br J Exp Pathol*. 63(1):109-115.
85. Kolev, K. (1982) Experimentally induced mesothelioma in white rats in response to intra peritoneal administration of amorphous crocidolite asbestos preliminary report. *Environ Res*. 29(1):123-133.
86. Kolev, K. (1982) Experimentally induced mesothelioma in white rats in response to intraperitoneal administration of amorphous crocidolite asbestos: preliminary report. *Environ Res*. 29(1):123-133.
87. Lafuma, J. et al (1980) Mesothelioma induced by intrapleural injection of different types of fibers in rats; synergistic effect of other carcinogens. *IARC Sci Publ*. 1(30):311-320.
88. Langer, A. M. et al (1980) Asbestos as a cofactor in carcinogenesis among nickel-processing workers. *Science*. 209(4454):420-422.
89. Lemen, R. A., Dement, J. M. and Wagener, J. (1980) Epidemiology of asbestos-related diseases. *Environ Health Perspect*. 34:1-11.
90. Lewinsohn, H. C. et al (1980) The influence of occupational and environmental asbestos exposure on the incidence of malignant mesothelioma in Connecticut. *IARC Sci Publ*. 1(30):655-660.
91. Lillis, R. et al (1979) Asbestos disease in maintenance workers of the chemical industry. *Ann NY Acad Sci*. 330:127-128.
92. Lippman, L. E. (1980) Cellular effects of asbestos and other fibers: correlations with in vivo induction of pleural sarcoma. *Environ Health Perspect*. 34:91-100.

93. Livingston, G. K., Rom, W. N. and Morris MV (1980) Asbestos-induced sister chromatid exchanges in cultured Chinese hamster ovarian fibroblast cells. *J Environ Pathol Toxicol.* 4(2-3):373-382.
94. Lopez-Areal Del Amo L (1980) Diseases associated with asbestos in Spain. *IARC Sci Publ.* 1(30):201-206.
95. Maltoni, C., Minardi, F. and Morisi, L. (1982) The relevance of the experimental approach in the assessment of the oncogenic risks from fibrous and nonfibrous particles. The ongoing project of the Bologna Institute of Oncology. *Med Lav.* 73(4):394-407.
96. Mancuso, T. F. (1983) Mesothelioma among machinists in railroad and other industries. *Am J Ind Med.* 4(4):501-513.
97. Masse, R. et al (1980) Experimental demonstration of the penetration of asbestos fibers into the gastrointestinal tract. *IARC Sci Publ.* 1(30):321-328.
98. McConnell, E. E. et al (1983) Chronic effects of dietary exposure to amosite and chrysotile asbestos in Syrian golden hamsters. *Environ Health Perspect.* 53:11-25.
99. McConnell, E. E. et al (1983) Chronic effects of dietary exposure to amosite asbestos and tremolite in F344 rats. *Environ Health Perspect.* 53:27-44.
100. McCullagh, S. F. (1980) Amosite as a cause of lung cancer and mesothelioma in humans. *J Soc Occup Med.* 30(4):153-156.
101. McDonald, A. D. et al (1984) Dust exposure and mortality in an American chrysotile asbestos friction products plant. *Br J Ind Med.* 41(2):151-157.
102. McDonald, A. D. et al (1983) Dust exposure and mortality in an American factory using chrysotile, amosite, and crocidolite in mainly textile manufacture. *Br J Ind Med.* 40(4):368-374.
103. McDonald, A. D. et al (1983) Dust exposure and mortality in an American chrysotile textile plant. *Br J Ind Med.* 40(4):361-367.
104. McDonald, J. C. (1980) Asbestos-related disease: an epidemiological review. *IARC Sci Publ.* 2(30):587-601.
105. McDonald, J. C. et al (1980) Dust exposure and mortality in chrysotile mining, 1910-1975. *Br J Ind Med.* 37(1):11-24.
106. McDonald, J. C. and Liddell, F. D. (1979) Mortality in Canadian miners exposed to chrysotile. *Ann NY Acad Sci.* 330:1-9.
107. McDonald, J. C. and MacDonald, A. D. (1981) Mesothelioma as an index of asbestos impact. *Banbury Report.* 9:73-86.
108. Monchaux, G. et al (1981) mesothelioma in rats following inoculation with acid-leached chrysotile asbestos and other mineral fibers. *Carcinogenesis.* 2(3):229-236
109. Morgan, A. (1980) Effect of length on the clearance of fibers from the lung and on body formation. *IARC Sci Publ.* 1(30):329-335.
110. Mossman, B., Light, W. and Wei, E. (1983) Asbestos' mechanisms of toxicity and carcinogenicity in the respiratory tract. *Annu Rev Pharmacol Toxicol.* 23:595-615

111. Mossman, B. T. and Craighead, J. E. (1981) Mechanisms of asbestos carcinogenesis. *Environ Res.* 25(2):269-280.
112. National Research Council (1984) Non-occupational exposure to asbestiform fibers. NAS Report.
113. Newhouse, M. L. (1983) Asbestos-related diseases. *Practitioner.* 227(1383):1399-1411.
114. Newhouse, M. L. (1981) Epidemiology of asbestos-related tumors. *Semin Oncol.* 8(3):250-257.
115. Newhouse, M. L. and Berry, G. (1979) Patterns of mortality in asbestos factory workers in London. *Ann NY Acad Sci.* 330:53-60.
116. Newhouse, M. L., Berry, G. and Skidmore, J. W. (1982) A mortality study of workers manufacturing friction materials with chrysotile asbestos. *Ann Occup Hyg.* 26(1-4):899-909.
117. Nicholson, W. J. et al (1981) Cancer from occupational asbestos exposure projections 1980-2000. Banbury Report. 9:87-111.
118. Nicholson, W. J. (1984) Asbestos Health Assessment Update. EPA Report (Review draft). EPA-600/8-84-003A:131.
119. Nicholson, W. J. et al (1979) Long-term mortality experience of chrysotile miners and millers in Thetford Mines, Quebec. *Ann NY Acad Sci.* 330:11-21.
120. Nicholson, W. J., Perkel, G. and Selioff, I. J. (1982) Occupational exposure to asbestos: population at risk and projected mortality--1980-2030. *Am J Ind Med.* 3(3):259-311.
121. Norseth, T. (1980) Asbestos and metals as carcinogens. *J Toxicol Environ Health.* 6(5-6):1021-1026.
122. Ordonez, N. G. and Smith, J. L. Jr (1983) Peritoneal malignant mesothelioma with multiple distant skin metastases. *Arch Dermatol.* 119(10):827-830.
123. Peto, J. (1980) The incidence of pleural mesothelioma in chrysotile asbestos textile workers. *IARC Sci Publ.* 2(30):703-711.
124. Peto, J. (1979) Dose-response relationships for asbestos-related disease: implications for hygiene standards. Part II. Mortality. *Ann NY Acad Sci.* 330:195-203.
125. Peto, J. (1980) Lung cancer mortality in relation to measured dust levels in an asbestos textile factory. *IARC Sci Publ.* 2(30):829-836.
126. Peto, J., Henderson, B. E. and Pike, M. C. (1981) Trends in mesothelioma incidence in the USA and the forecast epidemic due to asbestos exposure during World War II. Banbury Report. 9:51-72.
127. Peto, J., Seidman, H. and Selikoff, I. J. (1982) Mesothelioma mortality in asbestos workers: implications for models of carcinogenesis and risk assessment. *Br J Cancer.* 45(1):124-135.
128. Pott, F. (1982) Animal experiments on biological effects of mineral fibers. *IARC Sci Publ.* 1(30):261-272.
129. Price-Jones, M. J., Gubbings, G. and Chamberlain, M. (1980) The genetic effects of crocidolite asbestos: comparison of chromosome abnormalities and sister chromatid exchanges. *Mutat Res.* 79:4 331-336.

130. Pylev, L. N. et al (1982) Pleural mesothelioma in the Rhesus monkey *Macaca-mulatta* induced by intra tracheal injections of chrysotile asbestos. *Eksp Onkol.* 4(6):36-38.
131. Pylev, L. N. (1980) Pretumorous lesions and lung and pleural tumors induced by asbestos in rats, Syrian golden hamsters and *Macaca mulatta* (rhesus) monkeys. *IARC Sci Publ.* 1(30):343-355.
132. Rahman, G., Das, B. and Viswanathan, P. N. (1983) Biochemical mechanisms in asbestos toxicology. *Environ Health Perspect.* 51:299-303.
133. Reiss, B. et al (1983) Absence of mutagenic activity of three forms of asbestos in liver epithelial cells. *Environ Res.* 31(1):100-104.
134. Robock, K. (1979) Based on available data, can we project an acceptable standard for industrial use of asbestos? Absolutely. *Ann NY Acad Sci.* 330:205-210.
135. Rogol, P. R., Ryu, J. H. and Ginns, L. C. (1983) Reduced natural killer cell activity in asbestos workers. *Am Rev Respir Dis.* 127(4 part 2):73.
136. Rom, W. N. et al (1983) Sister chromatid exchange frequency in asbestos workers. *J Natl Cancer Inst.* 70(1):45-48.
137. Rubino, G. F. et al (1979) Mortality of chrysotile asbestos workers at the Balangero Mine, Northern Italy. *Br J Ind Med.* 36(3):187-194.
138. Scansetti, G. et al (1981) Pleural mesothelioma after a short interval from first exposure in the wine filter industry. *Am J Ind Med.* 5(4):335-339.
139. Schenker, M. B., Garshick, E. and Speizer, F. E. (1984) A case control study of incident mesothelioma deaths among railroad workers. *Am Rev Respir Dis.* 129(4 suppl):A146.
140. Seidman, H., Selikoff, I. J. and Hammond, E. C. (1979) Short-term asbestos work exposure and long-term observation. *Ann NY Acad Sci.* 330:61-90.
141. Seidman, H., Selikoff, I. J. and Hammond, E. C. (1982) Mortality of brain tumors among asbestos insulation workers in the United States and Canada. *Ann NY Acad Sci.* 381:160-171.
142. Selikoff, I. J., Churg, J. and Hammond, E. C. (1984) Classics in oncology: Asbestos exposure and neoplasia. *CA.* 34(1):46-56.
143. Selikoff, I. J., Hammond, E. C. and Seidman, H. (1980) Latency of asbestos disease among insulation workers in the United States and Canada. *Cancer.* 46(12):2736-2740.
144. Selikoff, I. J., Hammond, E. C. and Seidman, H. (1979) Mortality experience of insulation workers in the United States and Canada, 1943-1976. *Ann NY Acad Sci.* 330:91-116.
145. Selikoff, I. J., Lillis, R. and Nicholson, W. J. (1979) Asbestos disease in United States shipyards. *Ann NY Acad Sci.* 330:295-311.
146. Selikoff, I. J., Seidman, H. and Hammond, E. C. (1980) Mortality effects of cigarette smoking among amosite asbestos factory workers. *JNCI.* 63(3):507-513.
147. Selikoff, I. J. (1981) Constraints in estimating occupational contributions to current cancer mortality in the USA. *Banbury Report.* 9:3-16.

148. Stanton, M. F. et al (1981) Relation of particle dimension to carcinogenicity in amphibole asbestoses and other fibrous minerals. *JNCI*. 47(5):945-975.
149. Stevens, J. B. et al (1984) Asbestos metabolism in-vivo. *Am Rev Respir Dis*. 129(4 suppl):A168.
150. Stumphius, J. (1979) Mesothelioma incidence in a Dutch shipyard. *Ann NY Acad Sci*. 330:317-322.
151. Szybka, K. and Lange, A. (1982) Effects of asbestos on benzo(a)pyrene mutagenicity. *Mutat Res*. 97(3):227-228.
152. Taylor, R. A. and Johnson, L. P. (1981) Mesothelioma: current perspectives. *West J Med*. 134(5):379-383.
153. Teta, M. J. et al (1982) Occupational asbestos exposure and mesothelioma in Connecticut USA. "Occupational lung disease" edited by Gee, JB et.al.. Raven Press, New York:249pp.
154. Teta, M. J. et al (1983) Mesothelioma in Connecticut, 1955-1977. Occupational and geographic associations. *J Occup Med*. 25(10):749-756.
155. Thomas, H. F. et al (1982) Further follow-up study of workers from an asbestos cement factory. *Br J Ind Med*. 39(3):273-236.
156. Topping, D. C. and Nettesheim, P. (1980) Two-stage carcinogenesis studies with asbestos in Fischer 344 rats. *JNCI*. 65(3):627-630.
157. Topping, D. C., Nettesheim, P. and Martin, D. H. (1980) Toxic and tumorigenic effects of asbestos on tracheal mucosa. *J Environ Pathol Toxicol*. 3(5-6):261-275.
158. Valerio, F. et al (1983) Chromosomal aberrations induced by chrysotile and crocidolite in human lymphocytes in vitro. *Mutat Res*. 122(3-4):397-402.
159. Vicent, J. H. et al (1981) Static electrification of airborne asbestos: a study of its causes, assessment and effects on deposition in the lungs of rats. *Am Ind Hyg Assoc J*. 42(10):711-721.
160. Wagner, J. C. (1979) Diseases associated with exposure to asbestos dusts. *Practitioner*. 223(1233):28-33.
161. Wagner, J. C. (1982) Experimental studies and the pathology of asbestos induced lung diseases. *Arch Immunol Ther Exp*. 30(3-4):221-228.
162. Wagner, J. C., Berry, G. and Pooley, F. D. (1982) Mesotheliomas and asbestos type in asbestos textile workers: a study of lung contents. *Br Med J*. 285(6342):603-606.
163. Wagner, J. C., Berry, G. and Pooley, F. D. (1980) Carcinogenesis and mineral fibers. *Br Med Bull*. 36(1):55-56.
164. Wagner, J. C., Berry, G., Skidmore, J. W. and Pooley, F. D. (1980) The comparative effects of three chrysotiles by injection and inhalation in rats. *IARC Sci Publ*. 143C: 363-372.
165. Wagner, J. C., Griffiths, D. M. and Hill, E. J. (1984) The effect of fibre size on the in vivo activity of UICC crocidolite. *Br J Cancer*. 49(4):453-458.
166. Wagner, J. C. (1985) Immunology and asbestos. *IARC Sci Publ*. 143C:247-251.

167. Wagner, M. M. and (1983) HLA-A and B antigen frequencies and mesothelioma in relation to asbestos exposure. Br J Cancer. 48(5):727-730.
168. Walker, A. M. et al (1983) Projections of asbestos related disease 1980-2009. J Occup Med. 25(5):409-425. =
169. Warnock, M. L., Kuwahara, T. J. and Wolery, G. (1983) The relation of asbestos burden to asbestosis and lung cancer. Pathol Annu. 18(pt 2):109-145.
170. Whitaker, D., Shilkin, K. B. and Walters MNI (1984) Cytologic and tissue culture characteristics of asbestos induced mesothelioma in rats. Acta Cytol. 28(2):185-189.
171. Winkler, G. C. and Ruettnner, J. R. (1982) Penetration of asbestos fibers in the visceral peritoneum of mice a scanning electron microscopic study. Exp Cell Biol. 50(4):187-194.
172. Voitowitz, H. J. et al (1982) Asbestos related diseases in West Germany. Arch Immunol Ther Exp. 30(3-4):143-144.
173. Voitowitz, H. J. et al (1981) Asbestos-related diseases in the Federal Republic of Germany. Am J Industr Med. 2(1):71-78.



UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

MAR 4 1985

OFFICE OF
RESEARCH AND DEVELOPMENT

Mr. William V. Loscutoff, Chief
Toxic Pollutants Branch
Re: Asbestos
California Air Resources Board
P. O. Box 2815
Sacramento, CA 95812

Dear Mr. Loscutoff:

A copy of your request for information regarding asbestos, dated December 7, 1984, has been forwarded to my office. The Office of Health and Environmental Assessment has published a document entitled, Asbestos Health Assessment Update. The document is a First External Review Draft, dated February 1984, and we plan to revise it later this year. Unfortunately, we have run out of copies, but the document is available from:

National Technical Information Service
5285 Port Royal Road
Springfield, VA 22161

The order number is PB-84-186832.

I hope the information will be useful to you. If you have questions, please feel free to call me at 202/382-7345.

Sincerely yours,

Marie C. Pfaff
Technical Information Staff
Office of Health and Environmental
Assessment

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Ford Motor Company

The American Road
Dearborn, Michigan 48121

February 18, 1985 .

Mr. William V. Loscutoff, Chief
Toxic Pollutants Branch
Re: Cadmium and Asbestos
California Air Resources Board
P.O. Box 2815
Sacramento, CA 95812

Subject: Response to Mr. P. D. Venturini's Requests for Information
Regarding Cadmium and Asbestos

Dear Mr. Loscutoff:

The Ford Motor Company has not undertaken independent scientific studies to evaluate the health effects relating to either cadmium or asbestos. Rather, the Company quantitatively measures the ambient concentrations within the plant environments of regulated and suspected toxic air contaminants. These concentrations are evaluated with respect to the current Occupational Safety and Health Administration permissible exposure limits, National Institute of Occupational Safety and Health recommended standards, and American Conference of Governmental Industrial Hygienists Threshold Limit Values.

We regret that we are unable to submit information pursuant to your inquiries concerning health effects but we would like to continue to receive information on your progress in regulating toxic air contaminants.

Yours truly,

A handwritten signature in cursive script, appearing to read "Frank P. Partee".

F. P. Partee
Principal Staff Engineer
Air/Noise Compliance
Stationary Source Environmental
Control Office

10:DKJ7/L



DEPARTMENT OF THE ARMY
U. S. ARMY ENVIRONMENTAL HYGIENE AGENCY
ABERDEEN PROVING GROUND, MARYLAND 21010-5422

JAN 30 1985

REPLY TO
ATTENTION OF

Occupational and Environmental
Medicine Division

William V. Loscutoff
Chief, Toxic Pollutants Branch
California Air Resources Board
P.O. Box 2815
Sacramento, California 95812

Dear Mr. Loscutoff:

This Agency does not have data on asbestos per your request. However, we are aware of a document on the health effects of asbestos which you may want to review. The title is: Asbestos - An Update of Epidemiology and Pathology since 1976. This document was published by the USAF Occupational and Environmental Health Laboratory, Aerospace Medical Division, Brooks Air Force Base, Texas in February 1984. A copy can be obtained by requesting report number 84-125C01180B from either the National Technical Information Service or the Defense Technical Information Center.

Any questions concerning this reply should be directed to Major Robert W. Petzold, M.D. at telephone number 301-671-2464.

Sincerely,

for Robert W. Petzold
Joel C. Gados
Colonel, Medical Corps
Director, Occupational and
Environmental Health



ASBESTOS INFORMATION ASSOCIATION

1745 Jefferson Davis Highway, Crystal Square 4, Suite 509
Arlington Virginia 22202 • (703) 979-1150

Sub

January 17, 1985

William V. Loscutoff, Chief
Toxic Pollutants Branch
Re: Asbestos
California Air Resources Board
P.O. Box 2815
Sacramento, CA 95812

Dear Mr. Loscutoff:

I am writing in response to the Air Resources Board's request for information regarding asbestos. I have had the opportunity to review the ARB's bibliography and to discuss the program with Mr. John Batchelder. The greatest omission in the ARB's bibliography appears to be in not listing a number of large, overview studies of asbestos which are valuable for their scope and for the many individual references assembled in them. I would, therefore, recommend that the ARB collect and consider the following studies:

Acheson, E.D., Gardner, M.J. (1983) Asbestos. The Control Limit for Asbestos. Her Majesty's Stationery Office, London.

British Advisory Committee. (1979) Asbestos. (The Simpson Report). Her Majesty's Stationery Office, London.

CPSC. (1983) Chronic Hazard Advisory Panel on Asbestos. U.S. Consumer Product Safety Commission, Directorate for Health Sciences.

Report of the Royal Commission on Matters of Health and Safety Arising from the Use of Asbestos in Ontario. (1984).

On the issue of ingestion of asbestos, there is a recent animal bioassay from the National Toxicology Program:

NTP. (1983) Lifetime carcinogenesis studies of amosite asbestos in Syrian golden hamsters. NIH publication number 84-2505.

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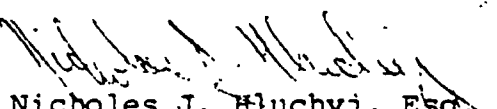
JAN 24 '85

I am also enclosing a copy of the EPA's response from the December 19, 1984 Federal Register regarding the use of asbestos in brakes. Mr. Batchelder noted that this is one area which may be a concern of the ARB.

Finally, I am enclosing a copy of a paper by Wagner and Elmes entitled "The Mineral Fibre Problem." As the use of asbestos declines, the use of other fibrous materials increases. The Wagner and Elmes paper cautions, on the basis of animal and human evidence, against the indiscriminant and uncontrolled use of these fibrous materials. The purpose of sending this paper is to make the point to the ARB that these fibrous materials constitute an area of investigation which has received scant attention, as opposed to asbestos, which has been very thoroughly studied and regulated. Yet another survey study of asbestos, as is presently contemplated by the ARB, has a certain aura of "reinventing the wheel" about it, whereas, with the exception of the Danish government, there has not been a regulatory body which has addressed the issue of fibers generally.

I would appreciate being kept informed of the progress of this project of the ARB, and if there is a mailing list established, please include my name on it. If I may be of any assistance, please feel free to contact me.

Very truly yours,


Nicholes J. Hluchyj, Esq.
Government Affairs Council

40 CFR Part 783

(OPTS-211016, TSM-JRL 2726-4)

Asbestos; Response to Citizens' Petition

AGENCY: Environmental Protection Agency (EPA).

ACTION: Response to Citizens' Petition.

SUMMARY: The Environmental Protection Agency (EPA) is granting a petition filed under section 21 of the Toxic Substances Control Act (TSCA) by the Natural Resources Defense Council, Inc. to prohibit the use of asbestos in automobile and truck brakes. EPA has commenced an appropriate proceeding to address the risks which may be posed by this use of asbestos.

DATE: Submit written comments on or before March 18, 1985.

ADDRESS: Submit written comments in triplicate identified by the document control number (OPTS-211015) to: TSCA Public Information Office (TS-793), Office of Toxic Substances, Environmental Protection Agency, Rm. E-107, 401 M. St. SW., Washington, D.C. 20460.

A copy of the petition and related information (with any confidential business information deleted) is located in: Room E-107, Environmental Protection Agency, 401 M. St. SW., Washington, DC 20460.

This material is available for viewing and copying from 8 a.m. to 4 p.m. Monday through Friday, excluding legal holidays.

FOR FURTHER INFORMATION CONTACT: Edward A. Klein, Director, TSCA Assistance Office (TS-799), Office of Toxic Substances, Environmental Protection Agency, Rm. E-343, 401 M. St. SW., Washington, D.C. 20460. Toll-free: (800-424-9063). In Washington, D.C.: (554-1404). Outside the USA: (Operator-202-354-1404).

SUPPLEMENTARY INFORMATION:
I. Introduction

Section 21 of the Toxic Substances Control Act (TSCA), 15 U.S.C. 2620, provides that any person may petition the Administrator of EPA to initiate a proceeding for the issuance, amendment, or repeal of a rule under various sections of the Act. EPA may hold a public hearing or may conduct an appropriate investigation to determine whether the petition should be granted. EPA must either grant or deny the petition within 90 days. If EPA grants the petition, EPA shall promptly commence an appropriate proceeding. If EPA denies the petition, the reasons for denial must be published in the Federal Register, and the petitioner may

commence a civil action in a district court of the United States to compel EPA to initiate a rulemaking proceeding as requested in the petition. Any such civil action must be filed within 60 days after EPA's denial of the petition or, if EPA fails to grant or deny the petition within 90 days after the petition is filed, within 60 days following expiration of the 90-day response period.

II. Evaluation of the Petition

A. Introduction

On September 12, 1984, EPA received a petition from the Natural Resources Defense Council, Inc. (NRDC), requesting that EPA prohibit the further use of asbestos in automobile and truck brakes under section 8 of TSCA. The petition requested a prohibition of asbestos in both brakes for new cars and trucks and in replacement brakes for existing vehicles. The petition argued that the risks posed by asbestos in brakes are unreasonable and that economically and technically feasible substitutes are available.

In order to promulgate any rules under TSCA section 8, the Agency must consider a number of factors, including, among other things, the effects of a chemical substance on human health and the magnitude of exposure; the benefits of utilizing the substance; and the availability of substitutes for the use or uses of the substance being assessed. 15 U.S.C. 2605(c)(1). The Agency has conducted a review of the available information pertaining to the use of asbestos in brakes, including the information in NRDC's petition. A summary of that review, including an evaluation of the risks posed by this use and the availability of substitutes, is presented below.

B. Risk Presented by Asbestos

Asbestos is a demonstrated human carcinogen that causes lung cancer and mesothelioma (a cancer of the chest and abdominal linings), as well as other lung disorders. People are exposed to asbestos throughout the life cycle of the substance—when asbestos is mined, milled, processed, fabricated into industrial and consumer products, and when those products are used, repaired, and disposed of.

With regard to the use of asbestos in brakes, it has been estimated that about 2,750 people are potentially exposed during primary manufacturing of brake friction materials, and that about 350,000 people are potentially exposed to asbestos during servicing and repair of vehicle brakes (Ref. 6). For example, persons in brake service and repair shops typically are exposed to asbestos

when dust is blown out of brake drums being replaced, when brake linings are roughened to increase friction properties, and when brake shoes are relined.

Use of asbestos in vehicle brakes may also result in increased asbestos fiber concentrations in the ambient air. For example, EPA has evidence that motor vehicle braking most likely contributes between 0.23 to 1.3 percent of the concentration of asbestos in the ambient air (Ref. 7). Both general population and workplace exposures to asbestos fibers from its use in brakes may result in an increased number of asbestos-related illnesses, including cancer.

C. Availability of Substitutes

The petitioner asserts that economically and technically feasible substitutes, most prominently semimetallic friction materials and aramid fibers, are available to replace asbestos in brakes. EPA has analyzed the availability of substitutes for many asbestos products, including brakes, and that analysis is summarized in Appendix A of the "Regulatory Impact Analysis of Controls on Asbestos Products" (Ref. 2), which is included in the public record established for NRDC's section 21 petition. EPA acknowledges that new substitutes for asbestos use in brakes are being developed and that EPA's analysis (summarized below) may not include recent developments.

1. **Heavy vehicle brake blocks.** Brake blocks are components of brakes that are riveted or bolted to the insides of brake shoes to provide protection against the heat and wear caused by braking. Heavy vehicle brake blocks are used on heavy duty trucks, buses, and other heavy duty vehicles. About 34 percent of all asbestos used in brakes is in this category of use. Asbestos heavy vehicle brake blocks account for about 99 percent of the market for heavy vehicle brake blocks. Until recently, the only commercially available substitute for asbestos heavy vehicle brake blocks was a semimetallic brake block using brass and zinc chips in an organic binder. It is not considered as good as asbestos because it performs erratically at different temperatures. It is also considered inferior to the asbestos brake block in resisting wear and minimizing brake fade. Recently, aramid fiber products, such as Kevlar, have been introduced, but aramid fiber products are now more expensive than asbestos products, and there is not sufficient evidence to determine whether aramid fiber products will be as effective as asbestos in this application. Information suggests that such products may last longer than asbestos-based

products, but verification of this information as well as information on price and efficacy of the products are needed before EPA can determine whether continued use of asbestos in brake blocks presents an unreasonable risk.

2. Light and medium vehicle drum brake linings. Drum brake linings are made of friction materials which cover curved metal shoes in a drum brake. About 46 percent of all asbestos used in brakes is in this category of use. Light vehicle asbestos drum brake linings may be used in both the front and rear brakes of light and medium vehicles, primarily on the front wheels. However, most passenger vehicles still use drum brake linings on the rear wheels. At least 99 percent of drum brake linings are still asbestos.

Until recently the only substitutes for asbestos brake linings which appeared to have any potential were semimetallic brake linings. However, these products tend to perform erratically at different temperatures, fade, and produce more noise than asbestos-based linings. More recently, brake linings made with aramid fiber have been developed. However, these are more expensive than the asbestos product and there is not currently enough information available to judge the performance of aramid fiber brake linings. Furthermore, EPA's evidence indicates that large volume production of aramid fiber brakes may require substantial retooling by brake manufacturers. Therefore, aramid fiber brakes may not be available in substantial quantities for several years.

3. Heavy vehicle disc brake pads. Disc brake pads are steel plates lined with friction materials which rub against a rotor. Heavy vehicles rarely use disc brakes. About 0.1 percent of all asbestos used in brakes is in this category of use.

Semimetallic disc brake pads are about 20 percent more expensive than the asbestos disc brake pads but they last about 30 percent longer. The semimetallic disc brake pads are not considered to be good general replacements for asbestos disc brake pads for heavy vehicles because semimetallics perform better than asbestos only in hostile, high-friction, high-heat environments.

4. Light and medium vehicle disc brake pads. These are the same as disc brake pads for heavy vehicles, except they are smaller. About 20 percent of all asbestos used in brakes is in this category of use. In the disc brake market, semimetallic brakes have already made larger inroads, and as in the drum brake linings market, aramid-based brakes are just beginning to be introduced. Approximately 50 percent of

disc brakes in the original equipment market are asbestos and 50 percent are semimetallic. In the replacement brakes after market approximately 70 percent of disc brakes are asbestos and 30 percent are semimetallic.

Semimetallic disc brake pads cost approximately 3 times as much as the asbestos disc brake pads, but they last about 40 percent longer than the asbestos pads. Semimetallic disc brake pads cannot substitute for asbestos disc brake pads in every application because of inferior performance characteristics. EPA has information indicating that semimetallic disc brakes should be used only in cars with power brake systems because otherwise the semimetallic brakes may not provide enough stopping power. EPA also has information that the semimetallic brakes are slightly inferior because the user has to push harder on them before they are warmed up to get the same performance as with asbestos brakes.

EPA is aware that a good deal of product research is being done to develop effective substitutes for asbestos in brakes. EPA is seeking additional information on substitutes for asbestos in brakes.

III. Conclusion

EPA has decided to grant NRDC's petition because the Agency believes that the use of asbestos in brakes does present risks to human health. EPA is initiating a proceeding to gather information on the desirability and feasibility of reducing risks associated with the use of asbestos in brakes. EPA will analyze the exposure from uses of asbestos, the risk presented by those uses, and the substitutes for such uses. EPA will also gather information of the price, efficacy, and availability of substitutes for asbestos in brakes. After analysis of this information, EPA will determine what further action is appropriate to address the risks which may be posed by this use of asbestos.

Based on information reviewed to date, EPA is not prepared to make an unreasonable risk finding for an immediate ban of all uses of asbestos in brakes at this time. Effective substitutes may not be available for certain applications of asbestos in brakes and substitutes for other applications are not available in sufficient quantity because of limited production capacity. Retooling may be required to make substitutes available in large volumes.

The Agency invites the public to submit comments on issues relating to the petition. EPA is particularly interested in receiving information on both the current availability of substitutes for asbestos in brakes and

on new substitute products that are currently being developed. The Agency has an ongoing workgroup, which will review these comments, will continue to investigate the availability of effective asbestos-free substitutes for use in vehicle brakes, and consider appropriate options to address the risk presented by asbestos in brakes.

IV. Record

EPA has established a record for information relating to the NRDC petition. The record includes information considered by EPA in developing this response and consists of the following categories of information:

- (1) The NRDC petition.
- (2) Appendix A of the "Regulatory Impact Analysis of Controls on Asbestos Products."
- (3) Other information on substitutes for asbestos in brakes.
- (4) Information received from the public concerning the petition.
- (5) Memoranda summarizing meetings and telephone conversations with the public concerning the petition.
- (6) Appendix J of the "Regulatory Impact Analysis of Controls on Asbestos Products."
- (7) Appendix N of the "Regulatory Impact Analysis of Controls on Asbestos Products."

A public version of the record, without any confidential business information, is available to the public in the Office of Toxic Substances Public Information Office, from 8 a.m. to 4 p.m. Monday through Friday, except legal holidays. The Public Information Office is located in Rm. E-107, 401 M St. SW., Washington, D.C.

Dated: December 14, 1984.

William D. Ruckelshaus,
Administrator.

[FR Doc. 84-33120 Filed 12-17-84; 1:14 pm]

GILLING CODE 1688-00-01

1. The mineral fibre problem

J. C. Wagner P. C. Elmes

For many years interest in the biological effect of mineral fibres was mainly confined to the commercial types of asbestos. Recently many other fibrous minerals have been recognised as potentially dangerous pollutants of the environment. The majority of these materials are naturally occurring, others are synthetic. The natural fibres are either specifically exploited for commercial purposes or else occur as atmospheric contaminants which are released during mining or tunnelling operations. Industry has been developing other mineral fibres as a substitute for asbestos to meet an increasing need for cheap and reliable materials for reinforcement, friction products and insulation. The latter demand has been emphasised by the present fossil fuel crisis. Minerals being exploited for a variety of purposes other than insulation and reinforcement are known to consist of fibres or elongated crystals, for example, some clays and some zeolites. Thus, these minerals can be considered under the following groups:

1. Asbestos minerals
 - a. Of commercial value
 - b. As potential environmental contaminants
2. Synthetic mineral fibres
3. Other naturally occurring fibrous minerals

ASBESTOS

Asbestos of commercial value

Practically all the knowledge that is available about hazards associated with the inhalation of fibrous mineral dusts has been obtained in studies of asbestos. Asbestos consists of six naturally occurring minerals: chrysotile, crocidolite, amosite, anthophyllite, tremolite and actinolite. Chrysotile is a member of a group of minerals referred to as the serpentines and is composed almost exclusively of magnesium in combination with silica. It has a sheet structure which curls to produce hollow tube-like fibres. The other five are members of one mineralogical group referred to as the amphiboles. They are very similar in crystal structure, being chain silicates, but they vary in chemical composition. Crocidolite and amosite are iron-rich varieties, anthophyllite is a magnesium rich mineral, while tremolite and actinolite contain a large amount of calcium together with magnesium.

The annual world production of asbestos in 1976 was 5×10^9 kg, of which 97 per cent was chrysotile and the remainder crocidolite and amosite. The commercial production of the other three amphiboles has been on a small scale in the past, but they are important as contaminants of other minerals and agricultural soil.

Chrysotile is widely distributed, with the largest production from the Ural

With the development of more sophisticated techniques it is now obvious that a correct estimation of the number of fibres in tissue or environmental samples can only be obtained by examination under a transmission electron microscope, otherwise the large number of fibres of less than $0.5 \mu\text{m}$ in diameter will not be observed. The crucial question of the amount, size and type of fibre found in tissue which can be related to the diseases which will be described later, cannot be stated with confidence at this stage. In macerated specimens of dried lung 10^6 fibres per gramme can be found without evidence of disease; in cases of asbestosis the count is usually over 10^4 . With a light microscope seldom less than 250 000 fibres per gramme lung tissue are found in cases of asbestosis.

2. PLEURAL PLAQUES AND DIFFUSE PLEURAL FIBROSIS

The presence of circumscribed areas of fibrous thickening below the mesothelium on the lower portion of the chest wall, over the diaphragm or on the pericardium are characteristic of exposure to fibrous mineral dusts. These plaques may be extensive, are leaf-shaped, often bilateral and have an irregular embossed surface. They consist of woven collagen fibres and as they mature become acellular and avascular. This avascularity leads to necrosis and sometimes to the gradual deposition of calcium in the lesions (Meurman, 1966). It can take 20 years or more for sufficient calcium to be deposited for the plaques to become radio-opaque and visible on chest radiography. Therefore, the plaques are seen much more frequently by the pathologists at necropsy than by the radiologist. In some cases there is generalised pleural fibrosis, leaving the lungs *en cuirasse* completely sheathed in a thick layer of fibrous tissue. Unlike plaques, generalised pleural thickening can restrict the expansion of the lungs and cause breathlessness.

3. ASBESTOSIS

Asbestosis is a slowly progressive and persistent interstitial fibrosis of the lung associated with the inhalation of asbestos dust and characterised by asbestos bodies and fibres in large numbers in the tissue. If sufficient dust has been retained, the individual lesions in the alveoli join up until the individual acini become linked in a fibrous mesh, the process starting at the base of the lung and gradually spreading upwards. This process is fairly well established before there is recognition on radiological or by physiological examination, the latter often being obscured by the effects of cigarette smoking. If exposure has been sufficient the disease will progress after the worker has left the industry (Becklake et al, 1979).

4. CANCER OF THE LUNG

Carcinoma of the bronchus is a frequent cause of death among workers with radiological evidence of asbestosis (Liddell and McDonald, 1980). The risk of a cigarette smoking worker heavily exposed to asbestos developing lung cancer is 25 to 50 times greater than an age matched non-smoker who has not worked with asbestos (see J. C. McDonald, 1980). Initially the carcinomas reported in the asbestos workers were peripherally situated, with adenocarcinomas being the most common (Buchanan, 1965). With the increase in cigarette smoking, all types of endobronchial tumours are being seen, but the number of adenocarcinomas is still more frequent than in non-exposed cigarette smokers (Kannerstein and Churg, 1972).

of fibre; apart from mining areas, pure exposure is rare. South African experience with crocidolite has been repeated on a smaller scale at Wittenoom in Western Australia, where mesotheliomas have occurred, both in those employed in the mines and in the environmentally exposed population (Hobbs et al, 1980). Nothing comparable has been reported for chrysotile, amosite or anthophyllite mining. The gas mask workers investigated by Jones et al (1976) and some of those by McDonald and McDonald (1978) appeared to have had a pure exposure. Pooley's analysis of the lungs of the Nottingham cases also showed significant amounts of chrysotile, but not more than is found in autopsy material generally. The technique developed by Pooley (1975) for the identification of asbestos and other mineral fibres in lung tissue is the most useful method available for identifying individual exposures and the complexity of the situation gives emphasis to the need for the parallel examination of appropriate controls. The comparisons made between the fibres in the lungs of the mesothelioma cases and controls in Britain by Jones et al (1980) when compared with those obtained by A. D. McDonald (1980) in the United States and Canada has shown that chrysotile fibres are found in considerable quantity equally in cases and controls. In Britain, crocidolite and to a lesser extent amosite were associated with mesothelioma, whereas in the USA it was predominantly amosite and less often crocidolite. Selikoff et al (1972) found a considerable excess of mesotheliomas in factory workers exposed to amosite but has not reported on the fibre content of their lungs. The Advisory Committee to the Secretary of State for Employment (Health and Safety Commission, 1979) concluded that in the causation of mesotheliomas, crocidolite was more dangerous than chrysotile but that amosite might be intermediate between the two.

Experimental evidence has complicated the situation by showing that many types of mineral fibre can cause mesothelioma. This evidence has come in the main from intrapleural implantation studies which were initiated by Wagner (1962) and continued in collaboration with Stanton and Wrench (1972); Pott et al (1972) undertook similar intraperitoneal investigations. This work has indicated that the size of the fibres was more important than their nature.

IMPORTANCE OF FIBRE SIZE

The significance of the physical characteristics of fibres in explaining the biological effects of asbestos was first emphasised by Timbrell (1965). He demonstrated that diameter was the most important factor in determining whether a fibre would be inhaled. The finer the fibre the more easily would it reach the lung parenchyma. Later, Timbrell et al (1970) showed that this could be applied to the amphiboles and that the ultimate diameter of crocidolite was less than that of amosite. Although the individual fibres of chrysotile have a diameter less than that of crocidolite, they occur in a woven coil formation, the total diameter of which affects its aerodynamic behaviour. Thus chrysotile behaves as a coarse fibre and finds difficulty in reaching the pleural surfaces through the peripheral airways. However, chrysotile fibres in aqueous solution can divide longitudinally into fibrils which under some circumstances are straight and have similar properties to very fine amphiboles. The typical electron microscopic appearance of asbestos fibres is shown in Figure 1.1.

Calculations and experiments with casts of the lower respiratory tract showed that fibres up to $3.0\text{ }\mu\text{m}$ in diameter would reach the respiratory bronchioles. The length of fibres most likely to cause fibrosis would appear to be greater than $10\text{ }\mu\text{m}$ (Timbrell

At this stage we must assume that all mineral fibres of similar size range are potentially hazardous to man, whether of asbestos or other type.

Asbestos minerals as potential environmental contaminants

Under this heading the following situations will be considered:

1. asbestiform minerals contaminating banded ironstone
2. tremolite as a contaminant of other mineral deposits
3. possible contamination of agricultural soil.

Asbestiform minerals contaminating banded ironstone

Although it has been known for many years that banded ironstone deposits frequently contain small seams of fibrous silicates, occasionally the fibrous deposits are large, and may then be the source of amphibole asbestos as exploited in South Africa and Australia. Other deposits occur which are of no commercial value, for example, there are the taconite fibres in the Mesabi Range on the shores of Lake Superior. Although it has been shown that iron ore mining in this region is causing both contamination of the atmosphere and the water of the lake, no evidence of a hazard to man has been established. All the fibres are below $5.0\ \mu\text{m}$ in length. In a large South Dakota gold mine the ore-bearing rock was cummingtonite — grunerite (a close relation of amosite) and some fibres were found in the dust. It was suggested that these fibres were associated with an increased incidence of carcinoma of the lung, but these findings were not confirmed by the investigations of McDonald et al (1978). Nevertheless, the possibility that hazardous fibres may sometimes be released from iron deposits in the vicinity of amphibole mines remains.

Tremolite as a contaminant of other mineral deposits

The fibre dimensions of tremolite vary even more between deposits than the other amphiboles. A coarse flake-like tremolite occurs as a contaminant of talc in California; this material does not cause tumours when implanted intrapleurally in rats, and there is no published evidence of disease among the miners. A coarse fibrous tremolite is found as a contaminant of the chrysotile deposits in Quebec Province in Canada. This fibre has been found in the lungs of miners and millers from these mines (Pooley, 1976); whether it plays any part in the etiology of pleural plaques and pulmonary fibrosis is still uncertain. In the talc mines in the northern part of New York State there is contamination by a finer fibrous tremolite, and a few mesotheliomas and carcinomas of the lung have been reported among the miners (Kleinfeld et al, 1967). Practically pure tremolite of a coarser type has been used in Eastern Turkey for stuccoing houses. Yazicioglu (1976) found a significant incidence of pleural plaques among the inhabitants. Tremolite with very fine long fibres has been mined in South Korea (see Fig. 1.1); this fibre has been shown to produce a high incidence of mesotheliomas in experimental animals. We have been informed that the mining operations have now been suspended because of suspected cancer among the workers.

Possible contamination of agricultural soil

A further source of fibrous mineral exposure only recently appreciated, may prove of consequence. Evidence is still fragmentary, and the only confirmed situation is in Bulgaria, where Burilkov and Michailova (1970; 1972) found pleural plaques in



GLASS WOOL



ROCK WOOL

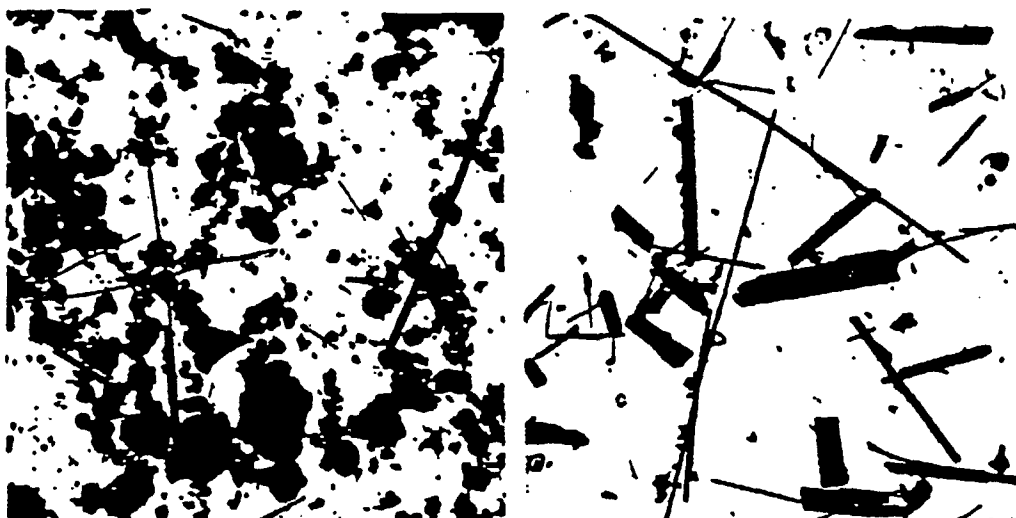


GLASS FIBRE (CODE 100)

10 μ m

Fig. 1.2 Electron micrographs of synthetic vitreous fibres

particularly on factory floors, and occasionally for agricultural and pharmaceutical purposes. Information is scanty concerning the size and shape which particles of these fibrous minerals assume under a range of potentially relevant natural and artificial conditions; nor is much yet known about their biological activity in various *in vitro* tests. Animal experiments have been recently initiated in which sepiolite and attapulgite fibres are inhaled or implanted intrapleurally; the results will not be



TURKISH FIBRE

ERIONITE FROM USA

Fig. 1.3 Electron micrographs of dispersed samples of synthetic and naturally occurring fibrous erionite

To date, synthetic substitutes for asbestos (the man-made vitreous and ceramic fibres) do not appear to have caused lung fibrosis, lung cancer or mesothelioma in man. However, animal experiments indicate that it would be unwise to create materials which include fibres smaller than $0.5\ \mu\text{m}$ in diameter if the risk of mesothelioma is to be avoided.

Experimental work suggests that both synthetic and natural non-asbestos mineral fibres of less than $0.5\ \mu\text{m}$ in diameter and greater than $8\ \mu\text{m}$ in length may be hazardous. Some fibrous clays have already been shown to cause mesothelioma experimentally while epidemiological evidence suggests that fibrous erionite may well have been responsible for a very high incidence of mesotheliomas in man.

Changes in industry and commerce are leading to human exposure from a variety of natural and synthetic mineral fibres. Further research is urgently needed to avoid replacing the hazard of asbestos with others as serious.

REFERENCES

- Arvinli M, Baris Y I 1979 Malignant mesotheliomas in a small village in the Anatolian region of Turkey: an epidemiological study. *Journal of the National Cancer Institute* 63: 17-22
- Baris Y I et al 1978 An outbreak of pleural mesothelioma and chronic fibrosing pleurisy in the village of Karain/Ürgüp in Anatolia. *Thorax* 33: 181-192
- Becklake M R, Liddell F D K, Manfreda J, McDonald J C 1979 Radiological changes after withdrawal from asbestos exposure. *British Journal of Industrial Medicine* 36: 23-28
- Buchanan W D 1965 Asbestosis and primary thoracic neoplasms. *Annals of the New York Academy of Sciences* 132: 507-518
- Burilkov T, Michailova L 1970 Asbestos content of the soil and endemic pleural asbestosis. *Environmental Research* 3: 443-451
- Burilkov T, Michailova L 1972 Über den Sepiolitgehalt des Bodens in Gebieten mit Endemischem Pleuraverkalkungen. *Internationales Archiv für Arbeitsmedizin* 29: 95-101

- faserformiger Staube und ihre Deutung im Hinblick auf die Tumorentstehung beim Menschen. Zentralblatt für Bakteriologie, Parasitenkunde, Infektionskrankheiten und Hygiene: erste Abteilung: Originale, Reihe B: Hygiene, Präventive Medizin 162: 467-505
- Pott F, Huth F, Friedrichs K H 1972 Tumoren der Ratte nach i.p. Injektion von gemahlenem Chrysotil und Benzo (a) pyren. Zentralblatt für Bakteriologie, Parasitenkunde, Infektionskrankheiten und Hygiene: erste Abteilung: Originale, Reihe B: Hygiene, Präventive Medizin 155: 463-469
- Selikoff I J, Hammond E C, Churg J 1972 Carcinogenicity of amosite asbestos. Archives of Environmental Health 25: 183-186
- Stanton M F, Layard M, Tegeris A, Miller E, May M, Kent E 1977 Carcinogenicity of fibrous glass: pleural response in the rat in relation to fiber dimension. Journal of the National Cancer Institute 58: 587-603
- Stanton M F, Wrench C 1972 Mechanisms of mesothelioma induction with asbestos and fibrous glass. Journal of the National Cancer Institute 48: 797-821
- Stell P M, McGill T 1973 Asbestos and laryngeal carcinoma. Lancet 2: 416-417
- Thomson J G, Kaschula R O C, MacDonald R R 1963 Asbestos as a modern urban hazard. South African Medical Journal 37: 77-81
- Timbrell V 1965 The inhalation of fibrous dusts. Annals of the New York Academy of Sciences 132: 255-273
- Timbrell V, Pooley F D, Wagner J C 1970 Characteristics of respirable asbestos fibres. In: Shapiro H A (ed) Pneumoconiosis: Proceedings of the International Conference Johannesburg 1969. Cape Town Oxford University Press, London, pp 120-125
- Timbrell V, Rendall R E G 1971/2 Preparation of the UICC standard reference samples of asbestos. Powder Technology 5: 279-287
- Timbrell V, Skidmore J W 1968 Significance of fiber length in experimental asbestosis. In: Holstein E (ed) Internationale Konferenz der biologische Wirkungen des Asbestos, Dresden. Dtsch. Zentralinst, Arbeitsmed, Berlin, DDR pp 52-56
- Wagner J C 1962 Experimental production of mesothelial tumours of the pleura by implantation of dusts in laboratory animals. Nature 196: 180-181
- Wagner J C 1979 Diseases associated with exposure to asbestos dusts. The Practitioner 223: 28-33
- Wagner J C, Berry G, Pooley F D 1980b Carcinogenesis and mineral fibres. British Medical Bulletin 36: 53-56
- Wagner J C, Berry G, Skidmore J W 1976 Studies of the carcinogenic effects of fiber glass of different diameters following intrapleural inoculation in experimental animals. In: Occupational exposure to fibrous glass — proceedings of a symposium. (DHEW Publication no (NIOSH) 76-151.) U.S. Department of Health, Education and Welfare, Washington DC, pp 193-197
- Wagner J C, Sleggs C A, Marchand P 1960 Diffuse pleural mesothelioma and asbestos exposure in the North Western Cape Province. British Journal of Industrial Medicine 17: 260-271
- Wagner J C, Berry G, Skidmore J W, Timbrell V 1974 The effects of the inhalation of asbestos in rats. British Journal of Cancer 29: 252-269
- Wagner J C, Berry G, Hill R J, Munday D E, Skidmore J W 1980a Animal experiments with man-made mineral fibres. In: Wagner J C (ed) Biological effects of mineral fibres. WHO/IARC, Lyon, pp 361-362
- Webster I 1973 Asbestos and malignancy. South African Medical Journal 47: 165-171
- Yazicioglu S 1976 Pleural calcification associated with exposure to chrysotile asbestos in south east Turkey. Chest 70: 43-47
- Yazicioglu S, Ilcayto, R, Balci K, Sayli B S, Yorulmaz B 1980 Pleural calcification, pleural mesotheliomas and bronchial cancers caused by tremolite dust. Thorax 35: 564-569



Oil, Chemical & Atomic Workers
International Union, AFL-CIO



Joseph Misbrenner, President
Michael Ricigliano, Secretary Treasurer
L. Calvin Moore, Vice President
Robert E. Wages, Vice President
International Offices
255 Union Blvd., Lakewood, CO 80228
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Mail P O Box 2812, Denver, CO 80201

December 17, 1984

Peter D. Venturini, Chief
Stationary Source Division
State of California
Air Resources Board
P. O. Box 2815
Sacramento, CA 95812

Dear Mr. Venturini:

Reference is made to your recent request for information regarding Asbestos.

Please be advised that we have no additional information or materials that you do not already have. Thank you for your inquiry.

Yours truly,

Dan C. Edwards, Director
Health and Safety Department

DCE:pl

cc: R. Wages, Vice President
J. Foley, Dir Dist. # 1

RECEIVED
DEC 21 1984
State of California
Air Resources Board

B-28

END

D 004339

DEPARTMENT OF HEALTH AND HUMAN SERVICES
DIVISION OF HEALTH SERVICES
COUNTY OF MARIN

HALL OF JUSTICE
CIVIC CENTER
SAN RAFAEL, CALIFORNIA
PHONE 499-6879

B-29 12/21
Mr. Scott
response
due 1/7

December 13, 1984

RECEIVED

12/24/84

DEC 20 . . .

Peter D. Venturini, Chief
Stationary Source Division
Air Resources Board
1102 Q Street
Sacramento, CA 95812

Stationary Source
Division
Air Resources Board

Re: Your letter 12/7/84 requesting information regarding asbestos

Dear Mr. Venturini:

This is not a response to your request, but is a heartfelt plea for clarity and accuracy. In accord with the dictum, "Do no harm," bureaucratic agencies should strive always not to pollute the English language.

Asbestos cannot be a "toxic air contaminant," not even if Ronald Reagan and the Congress say it is. Asbestos is not a toxin. It is not even a poison.

Here is Dorland's Medical Dictionary definition:

TOXIN -- A poison; frequently used to refer specifically to a protein or conjugated protein substance produced by higher plants, certain animals, and pathogenic bacteria that is highly toxic for other living organisms. Such substances are differentiated from the simple chemical poisons and the vegetable alkaloids by their high molecular weight and antigenicity.

Now that we have moved into the era of industrial toxins, we are merely adding chemists to the list (plants, animals, bacteria) of things that produce toxins. We should not be changing the meaning of the root word.

Asbestos is virtually inert in all the forms you mentioned. It cannot be a toxin. It is not even a "simple chemical poison."

Look at the confusion and tremendous cost engendered by bureaucratic and media repetition of the phrase "deadly toxic PCBs." PCBs are not deadly -- they were handled quite casually by thousands of workers for many years, with no reported deaths, just chloracne. Neither are PCBs toxic, being relatively inert, like asbestos. There is absolutely no reason for any person to react with fear to the presence of small quantities of PCBs in urban soil.

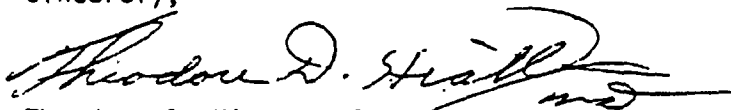
All that I say here is not meant in any way to lessen the legitimate concerns this society must have for keeping harmful, potentially carcinogenic (or mutagenic, or teratogenic) substances out of the air we breathe (like microscopic asbestos fibers) and out of the food we eat (like bioconcentrated PCBs), and out of the water pipes.

My plea is only for accuracy and preservation of the language upon which accuracy depends.

With accurate thinking it becomes obvious that a PCB-filled transformer atop a pole is no more dangerous than a room filled with polyvinyl furniture where people smoke cigarettes. Ignition of either one will seriously endanger firefighters or anyone else who might breathe the smoke. And the same is true of encapsulated asbestos. Yet this society spends billions of dollars cleaning up certain PCB - contaminated soils that don't need to be "cleaned up," and removing asbestos materials that don't yet need to be removed. The removal itself generally causes more danger to workers, the public, and the biosphere than would exist if the material were left alone.

It seems to me that both public and private funds should be spent more wisely in working toward a total prohibition of continued corporate contamination of the earth, air and water that make up our common heritage.

Sincerely,



Theodore D. Hiatt, M.D.
Marin County Health Officer

TDH:jm

CC: William V. Loscutoff, Chief
Toxic Pollutants Branch
California Air Resources Board

Assemblywoman Sally Tanner

Senator Ralph Dills

Alex Kelter
Dept. of Health Services

David Howekamp
EPA Region IX

P.S. DHEW Publication Number (NIH) 78-1681 May 1978 "Asbestos: An Information Resource" has a bibliography even more extensive than the one supplied by you. There are few, if any, hazardous substances more extensively documented.

AIR RESOURCES BOARD

1102 Q STREET

P.O. BOX 2815

SACRAMENTO, CA 95812



January 7, 1985

Theodore D. Hiatt, M.D.
Marin County Health Officer
Department of Health and Human Services
Hall of Justice - Civic Center
San Rafael, CA 94903

Dear Dr. Hiatt:

Thank you for your letter of December 13, 1984, responding to our request for information regarding asbestos. With respect to the question of whether or not asbestos can be a toxic air contaminant, we are constrained to follow the language of California Health and Safety Code Section 39655, which includes the following:

"...toxic air contaminant means an air pollutant which may cause or contribute to an increase in mortality or an increase in serious illness, or which may pose a present or potential hazard to human health." Substances which have been identified as hazardous air pollutants pursuant to Section 7412 of Title 42 of the United States Code shall be identified by the state Air Resources Board as toxic air contaminants (asbestos has been so identified pursuant to the cited U.S. Code). In addition, the intent of the legislation establishing our toxic air contaminant program was to protect the public from "...the emission into the ambient air of substances which are determined to be carcinogenic, teratogenic, mutagenic, or otherwise toxic or injurious to humans."

Although the definition for a toxic air contaminant may be inconsistent with the definition of toxin found in a medical dictionary, I do not believe that the intent, as stated in the statute, is in disagreement with your concerns. The intent of the law is to protect public health.

Please be assured that I share your concern with proper usage of the English language in government work. Thank you again for your letter. If you have

January 7, 1985

any further questions concerning our toxic air contaminant program, please feel free to contact me at (916) 445-0650 or William Loscutoff, Chief of the Toxic Pollutants Branch, at (916) 322-6023.

Sincerely,



Peter D. Venturini, Chief
Stationary Source Division

cc: W. Loscutoff
Assemblywoman Sally Tanner
Senator Ralph Dills
Alex Kelter, DHS
David Howekamp, EPA

Appendix C
Emissions Calculations*

* Sample calculations in this Appendix contains more significant figures than were used in the Part A of the report. All differences between estimates here and in Part A are due to rounding.

D 004344

Asbestos Emission Calculations for Mining and Milling, 1984

1. Mining

Process Rate

In 1984, 57,308 tons of asbestos were produced; 36,000 tons by Calaveras Asbestos and 21,308 tons by KCAC Corp. (formerly Union Carbide Corp.)

Mining emissions from Calaveras Asbestos:

Process Rate: 36,000 tons asbestos produced

Emission factor: 5 lb/ton at 50% control

	<u>Emission Factor at 50% Control</u>
Drilling & Blasting	2
Loading _____	1
Hauling _____	1
Unloading _____	<u>1</u>
Total	5 lb/ton

36,000 tons (5 lb/ton)
Emission = ----- = 90 tons
 2,000 lb/ton

Mining Emissions from KCAC Corp.

Process Rate: 21,308 tons asbestos produced

**Emission factor: 3 lb/ton at 50% control from the following
activities only: loading, hauling and unloading**

For 1984, KCAC obtained its ores from existing stockpiles. Mining is done every 2 or 3 years by stripping material from the asbestos deposit with push scrapers; no drilling or blasting is required.

$$\begin{array}{rcl} & 21,308 \text{ tons (3 lb/ton)} & \\ \text{Emissions} = & \frac{\text{-----}}{2,000 \text{ lb/ton}} & = 32 \text{ tons} \end{array}$$

$$\text{Total Mining Emissions} = 90 \text{ tons} + 32 \text{ tons} = 122 \text{ tons}$$

2. Milling

Process Rate

Asbestos produced in 1984 = 57,308 tons

Emission Factor*: 12 lb/ton

$$\begin{array}{rcl} & 57,308 \text{ tons (12 lb/ton)} & \\ \text{Emission} = & \frac{\text{-----}}{2,000 \text{ lb/ton}} & = 344 \text{ tons} \end{array}$$

*emission factor assumes best controls on dry process, KCAC uses a wet process for which no emission factors are available

3. Manufacturing

a. Primary

Process Rate

Data on process rate (amount of asbestos used) was obtained from: 1) 1983 South Coast Air Quality Management District's Toxics Survey and 2) 1985 asbestos survey conducted by the Stationary Source Division, Air Resources Board (ARB) (see Table C-1). ARB surveyed the manufacturing facilities identified by the U.S. Environmental Protection Agency (EPA) that were not included in the 1983 South Coast survey. Overall response to both surveys was about 70 percent. To calculate the total emissions from all manufacturing facilities surveyed, it was assumed that those that responded are representative of the whole population.

Emission Factor

The emission factors for primary manufacturing of the various asbestos product categories range from .14 lb/ton of asbestos used to 1.1 lb/ton of asbestos used (see Table I-2). The emission factors were cited from an EPA report (Versar, 1984).

Emission = Process rate x emission factor

Sample calculation:

50 tons asbestos used x .2 lb/ton = .005 ton/year
in manufacturing sealants -----

2000 lbs/ton

Table C-1
ASBESTOS EMISSIONS FROM THE MANUFACTURING OF ASBESTOS PRODUCTS

Product Type	Average Process Rate (ton)	Range of Process Rate (ton)	Total Primary Emission* (ton)	Average Process Rate (ton)	Range of Process Rate (ton)	Total Secondary Emission* (ton)
1. Friction	175	150-200	0.1	500	0.1-1775	2
2. Packings & Gaskets	-	1.67	< 0.1	6.15	0.025-40	< 0.1
3. Coatings, Adhesives & Sealants	127.50	0.025-1202	0.4	-	0.18	< 0.1
4. Cement Pipe	8350	7500-9212	3	0	0	0
5. Paper	-	30.3	< 0.1	2.60	0.25-5.00	< 0.1
6. Plastics	4650	0.548-18,283	0.7	-	0.13	< 0.1
7. Textiles	0	0	0	-	0.32	< 0.1
8. Floor Tile	-	29	< 0.1	0	0	0
9. Miscellaneous (Chlorine)	0	0	0	-	30	< 0.1
Total			4			2

* Emission estimates may not total due to rounding.

b. Secondary

Process Rate

Data on process rates were obtained from the 1983 South Coast Toxic Survey and the 1985 asbestos survey by ARB.

Emission Factor

The emission factors for secondary manufacturing are estimated from the emission factors used for primary manufacturing. The equation used for calculations is as follows:

$$\text{Emissions} = \text{Emfac} \times \text{process rate}$$

Where:

$$\text{Emfac} = (\text{EF}) \times \text{W}$$

EF = asbestos emission factor for primary manufacturing

W = asbestos content of the intermediate product used
in producing another product

4. Automobile Brakes

Process Rate

Total vehicle miles traveled in 1984 =
 $456,857 \times 10^3$ miles per day

Emission Factor

2.6 ug/km (Williams and Muhlbaier, 1982)

Emission

$$\begin{aligned} & (2.6 \times 10^{-6} \text{g/km})(456,857 \times 10^3 \text{ miles/day})(365 \text{ days/year}) \\ & \text{-----} = \\ & (454 \text{ g/lb})(.62 \text{ mile/km})(2000 \text{ lbs/ton}) \\ & = .77 \text{ ton/year} \end{aligned}$$

5. Quarrying

Table C-2 contains estimated asbestos emissions from quarries.

Asbestos Emissions = Amount of respirable particulate x % chrysotile asbestos content by weight.

Sample calculation for George Reed, Inc.:

Process Rate = 274,000 tons of crushed stone produced

Emission factor =

0.012 lbs respirable particulate per ton of crushed stone

0.20 lb of chrysotile asbestos per lb of respirable particulate

Emission:

$$274,000 \text{ tons} \times (0.012 \text{ lb/ton})(0.20) = 658 \text{ lbs asbestos}$$

Reference for emission factor for respirable particulate: Blackwood, T.R. and P.K. Chalekode, 1978. Source Assessment: Crushed Stone. Prepared by Monsanto Research Corporation for the U.S. Environmental Protection Agency. EPA-600/2-78-004L.

Table C-2

Asbestos Emissions from
Quarrying, 1984

	<u>Quarry</u>	<u>Production of Crushed Stone/Year</u>	<u>Respirable Particulate (lb)</u>	<u>Chrysotile Asbestos Content(%)</u>	<u>Asbestos Emissions (lb)</u>
1.	George Reed Inc, Tuolumne	274,000	3,288	20	658 1) reference
2.	Woods Creek Rock, Tuolumne	231,894	2,783	1	28 2)
3.	Raisch Products Santa Clara	435,000	5,220	1.6	84 1)
4.	Ghilotti Bros. Maria	156,000	1,872	1.4	26 2)
5.	Dumbarton Quarry Assoc., Alameda	1,000,000	12,000	0.5	60 1)
6.	Hillsdale Rock Santa Clara	250,000	3,000	2.7	<u>81</u> 1)
TOTAL					937 lbs or 0.468 tons

References for 1984 production of crushed stone:

- 1) ELLEN LINDER, BAAQMD for quarries located in the Bay Area.
- 2) JERRY BENICASA, Tuolumne County for quarries located in Tuolumne.

D 004351

REFERENCES FOR APPENDIX C

1. U.S. Environmental Protection Agency, 1981. Assessment and Control of Chrysotile Asbestos Emissions from Unpaved Roads. EPA 450/3-81-006.
2. Versar Inc., 1984. Exposure Assessment for Asbestos. Draft final report prepared for U.S. Environmental Protection Agency. EPA contract No. 68-01-6271, Task No. 49.
3. Williams, R. L. and J. L. Muhlbaier, 1982. "Asbestos Brake Emissions," Environmental Research. 29:79-82.

Appendix D

**Documents Containing Asbestos Measurements
Taken in California**

D 004353

An Inventory of Carcinogenic Substances Released into the Ambient Air of California. Final Report Task II and IV. March 1980. KVB, Inc.

Inventory of Carcinogenic Substances Released into the Ambient Air of California: Phase II. November 1982. Science Applications, Inc.

Ambient Asbestos Concentrations in California, Volume 1. Final Report. December 1983. Science Applications, Inc.

Ambient Asbestos Concentrations in California - Field Number and Mass Concentration Summaries, Volume 2. December 1983. Science Applications, Inc.

An Inventory of Carcinogenic Substances Released into the Ambient Air of California: Volume 1. Final Report - Screening and Identification of Carcinogens of Greatest Concern. February 19, 1979. Science Applications, Inc.

"Chrysotile Asbestos in a California Recreational Area." Cooper, W.C., J. Murchio, W. Pendorf, and H.R. Wenk. Science 206:685-688, November 9, 1979.

Assessment and Control of Chrysotile Asbestos Emissions from Unpaved Roads. May 1981. U.S. Environmental Protection Agency.

Experimental Determination of the Number and Size of Asbestos Fibers in the Ambient Air. January 1976. Air and Industrial Hygiene Laboratory, Laboratory Services Branch, Department of Health Services. AIHL/SP-1.

Asbestos in the California Environment. June 1975. Air and Industrial Hygiene Laboratory, Laboratory Services Branch, Department of Health Services.

Asbestos Fibers in Ambient Air of California. March 1973. School of Public Health, University of California, Berkeley.

Report of Seasonal Variations in Asbestos Aerosols In The Clear Creek Recreational Area (1979). University of California, Berkeley.

"Summary of Airborne Asbestos Data - Alviso, California and Control Sites". April 15, 1985. Memorandum from Dr. Steven Hayward of the Air and Industrial Hygiene Laboratory to Ralph Propper of the Air Resources Board.

Appendix E

Ambient Asbestos Measurements
In California

D 004355

Ambient Asbestos Measurements in California

<u>Location in California</u>	<u>Number of Fibers/m³</u>	<u>References</u>
King City, downwind of a milling plant	6,000 to 1,600,000	John et al., 1976
King City, upwind of a milling plant	200 to 11,000	John et al., 1976
San Jose	0 to 3,500	Murchio et al., 1973
Berkeley	0 to 4,000	Murchio et al., 1973
Los Angeles (Downtown)	0 to 5,700	Murchio et al., 1973
Emeryville, near asbestos manufacturer	238,000	Murchio et al., 1973
White Mountain (desert)	20 to 100	Murchio et al., 1973
Santa Monica Freeway, up- wind (at 4th)	700*	Murchio et al., 1973
Santa Monica Freeway, down- wind (at 4th)	700*	Murchio et al., 1973
Harbor Freeway, upwind	1,100*	Murchio et al., 1973
Harbor Freeway, downwind (at 146th)	1,600*	Murchio et al., 1973
San Diego Freeway, up- wind (at National)	200*	Murchio et al., 1973
San Diego Freeway, down- wind (at National)	800*	Murchio et al., 1973
San Diego Freeway, up- wind (at 122nd)	900*	Murchio et al., 1973
San Diego Freeway, down- wind (at 122nd)	500*	Murchio et al., 1973
Los Angeles Freeways, upwind (four sites)	800**	Murchio et al., 1973
Los Angeles Freeways, downwind (four sites)	900**	Murchio et al., 1973
San Lucas	1,000,000	Wesolowski, 1975
Berkeley	.700,000	Wesolowski, 1975

*Mean values

** Mean value of 60 samples

Source: Science Applications, Inc., February 19, 1979. An Inventory of
Carcinogenic Substances Released Into the Ambient Air of California.
Volume I - Final Report Screening and Identification of Carcinogens of
Greatest Concern. Page 35.

Appendix F

SAI Sampling, Sample Preparation, Analysis and Quality Control Techniques

D 004357

SAI Sampling, Sample Preparation, Analysis
and Quality Control Techniques
for the 1983 Asbestos Study

I. Introduction

Given the project objective of providing an inventory of respirable airborne asbestos in California, the technical approach focused on applying state-of-the art sampling and analysis techniques to a cross-section of geographical sites based on emission/exposure potential. Sampling method adopted for this study followed protocols established by John et al. (1976, 1978, 1980). No exceptions were taken to the measurement and verification procedures specified by the U.S. Environmental Protection Agency (U.S. Environmental Protection Agency, 1978). Discussions with California Air Industrial Hygiene Laboratory (AIHL), the U.S. Environmental Protection Agency and the National Bureau of Standards (NBS) staff resulted in a number of minor changes and improvements.

To accomplish the program objective for quantifying asbestos fibers in the respirable range (less than 3.5-um aerodynamic diameter), a single-point filter sampler with a cyclone inlet providing a collection efficiency of 50% for 3.5-um aerodynamic diameter particles at a flow rate of 15.5 liters per minute was used for all filter sampling. An 8.0-um pore size backing filter was used in conjunction with a 0.2-um pore size collection filter. Although original protocols specified the use of a 0.4-um pore size collection filter, the 0.2-um pore size filter was chosen for better collection efficiency of smaller fibers. No other significant changes were incorporated.

The cyclone sampler shown in Figure F-1 was developed for ambient air size-selective monitoring. This sampler will collect particles as small as

2.5-um (21.7 liters per minute flow); Figure F-2 (from John et al., 1978) indicates the particle deposition efficiency, relative to 50% cutoff, of the cyclone design shown in Figure F-1. Design criteria for the sampler include:

- o A vertical cone with cap at bottom and outlet on top. This configuration minimizes loading and re-entrainment.
- o The after filters are 47 mm diameter which will allow the use of membrane filters, if chemical analysis is desired.
- o The cyclone is intended for 24 hour sampling at average flows of 15 to 20 liters per minute.
- o Both liquid and solid particles can be sampled with this device.

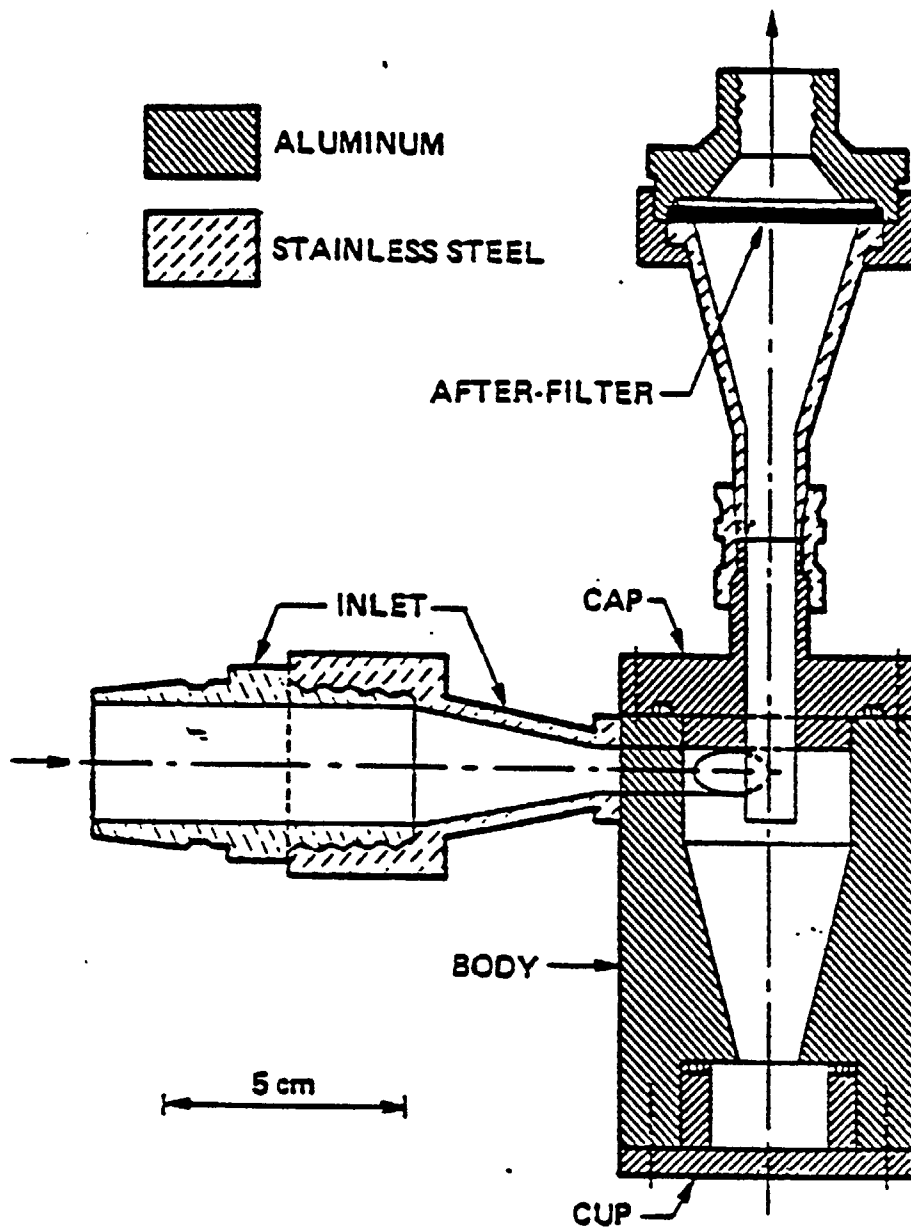
The cyclone was selected for use over other samplers for this study because of its ease of use in the field, its ability to selectively sample because of its ease of use in the field, its ability to selectively sample small particles and the design which allows for uniform particle deposit on the filter.

II. Sampling Procedures

Preparation and Handling of Filters

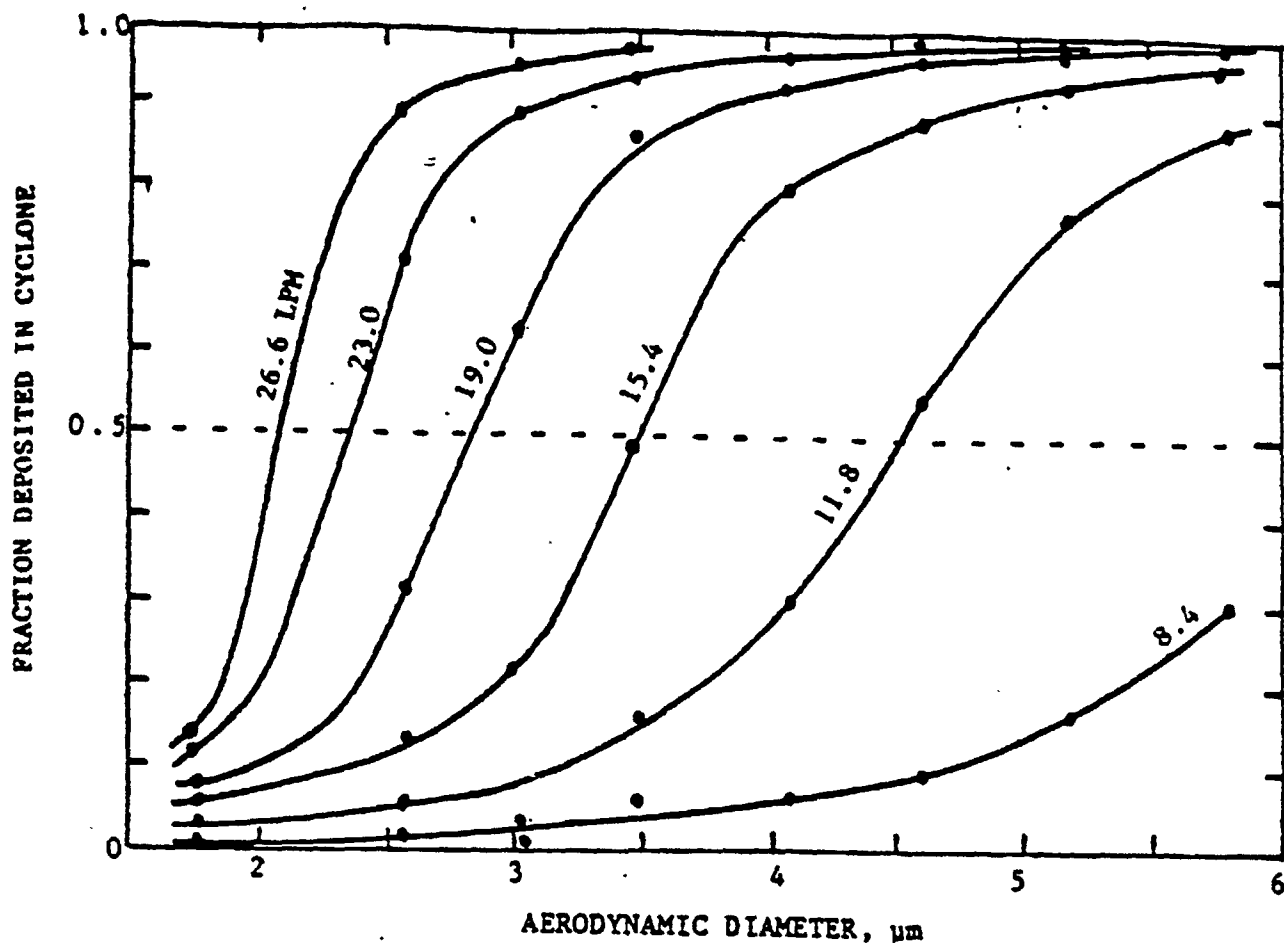
To reduce contamination from handling in the field, individual polyethylene cassettes were constructed to hold the backing and collection filters for each sample. These cassettes consisted of two rings that

Figure F-1



ASSEMBLY DRAWING OF THE CYCLONE SAMPLER DEVELOPED FOR AMBIENT AIR MONITORING (John and Reischl, 1980).

Figure F-2



Fraction of Methylene Blue Particles Deposited in the Cyclone as a Function of the Aerodynamic Particle Diameter. The curves are labeled with the flow rate (from John et al., 1978).

press-fit together to provide a rigid support for the filters. Before loading the filters, the cassettes were pre-washed in an ultrasonic bath of 0.1-um filtered, de-ionized water and allowed to dry in a "particle-free" clean room (class 100 certified). The filters were loaded into the cassettes inside the laminar flow bench of the clean room, using vacuum tweezers. Before sampling, an 8.0-um pore size, 47 mm diameter Millipore backing filter was placed under the Nuclepore 0.2-um pore size collection filter to insure even distribution of particles across the filter face.

After being loaded, each cassette was placed in a separate, clear plastic box (50mm x 80mm), which had been treated with Zerostat anti-static charge reducer. A secondary box specially constructed to reduce contamination and filter tipping was used to transport the filter boxes. Vibration during transportation was minimized by placing this secondary box containing a plastic bag full of water and styrofoam pellets.

Filter cassette loading was done on a damp-wiped table in the mobile laboratory; the entire cyclone was disassembled and rinsed with Freon 113. Cassettes and cyclone components were handled with polyethylene gloves, reducing possible contamination time to that required to remove the sample from its box and transfer it to the head of the cyclone (approximately ten seconds).

Air Sample Collection Procedures

Original sampling plans called for at least four four-hour samples at each site. Paired replicate samples were taken morning and afternoon at King City, San Jose, Napa, San Fernando Valley and Sonora. Century City, Bakersfield, South Gate and San Diego ordinarily have high particle

concentration levels, so samples were collected at these locations over shorter time intervals to preclude overloading the filters. Sampling time was based on prior calibrations of filter loading as a function of suspended particle count level, by scanning electron microscopy.

Sample filtering was carefully done, as described earlier in this section, with plastic gloves worn to transfer filter cassettes to the pre-cleaned cyclone assembly. The sampling pumps were operated at 15.5 liters per minute under a vacuum of six to seven inches of mercury. The pump and filter train were pre-tested to assure that there were no leaks in the six to seven-inch (mercury) vacuum range.

III. Analytical Techniques

Sample Preparation Procedures

Upon return to the laboratory, the plastic filter cassettes were wiped clean with a damp, lint-free cloth and placed in clean room facilities. Everything relating to filter handling was cleaned with 0.2-um pore size filtered water and blown dry with Freon dusting spray. Direct handling of the filter cassettes was done with plastic gloves under a class 100 (fewer than 100 particles per cubic foot of air) laminar air flow. Particulate matter levels under the laminar air flow were maintained at less than 5 particles per cubic foot in the 0.3-um diameter range; particulate matter in the clean room was kept at less than 10 particles per cubic foot in the 0.7 um size range. Mass levels measured by the RAM counter in the center of the room were 1 ug/m^3 .

Sectioning of Filters

Sectioning of Nuclepore filters with the least disturbance to the membrane surface was a critical step in sample preparation. The Nuclepore

filter with 8.0-um pore size Millipore backing filter was removed from the cassette and transferred to a 2 inch x 2 inch plexiglass plate. A new razor blade (cleaned with dusting spray) was slowly pressed on the filter pad using a downward vertical motion to cut the filter in half. The Nuclepore filter section from each half filter was carefully slid off the Millipore backing filter onto a clean piece of plexiglass. The filter was then tacked in place and a series of polyvinylchloride (PVC) cement beads were placed around the edges of each filter section. The tacked filters were allowed to dry under plastic petri dish covers. The plexiglass square containing the tacked filter section was placed in the bottom of a petri dish with a minimum clearance of 1.5 cm and fastened with double stick tape. One section was saved as an archive and for sub-sectioning for replicate analysis by the University of Washington. The other section was reserved for carbon coating as required by the EPA method.

Carbon Coating Procedures

Carbon coating was done with a Denton rotating stage carbon evaporator. The filters were carbon coated at 1×10^{-5} torr in short bursts with 30 to 40 nm of carbon. To minimize fiber dislodgement on the sampler filters, evacuation of the carbon coating chamber was performed slowly over a period of five minutes.

Modified Jaffe-Wick Grid Preparation

The filters were extracted using a modified Jaffe-Wick technique outlined in the provisional U.S. EPA methodology (EPA, 1978). All petri dishes and utensils were precleaned and spray-dusted. The foam supports and filter paper sections were ultrasonically cleaned with three washes of analytical grade acetone. Although the provisional method calls for the use of screens to

support Formvar sample grids, Whatman 42 filters papers were used because they produce more uniform grids. Another difference in the methodology was the use of 300-mesh instead of 200-mesh grids (300-mesh grids provide more support for the Formvar grid layering, as well as more accurate tracking of particle location within each grid hole by electron microscopy).

A clean section of 0.25 inch x 2 inch x 2 inch polyurethane foam with a 2 inch x 2 inch filter paper on top was placed in a 6 cm diameter petri dish. Approximately three to six 300-mesh sample grids were placed on top of the filter paper, Formvar side up. Three square sections (4 mm x 4 mm) of the sample filter were excised with a clean razor blade and placed against the Formvar side of the grid with the particle side facing against the grid. Each of the grids was then saturated with 5 to 8 ul of analytical grade chloroform by filling the petri dishes to a level that just covered the filter paper support, using a long-needle, 50 ml syringe. Each dish was transferred to a glass dessicator with a reservoir of chloroform below the support plate. The chloroform level in the petri dish was carefully raised to the top of the filter paper, the petri dish covered, the dessicator lid replaced and the filters allowed to dissolve for 24 hours. An outside light source just above the dessicator kept it at a temperature of about 26.7 C. After 24 hours, the chloroform from each dish was removed with a syringe and the grids were allowed to dry before being transferred to a grid storage box.

Sample Analysis Procedures

A JEOL 6C transmission electron microscope (TEM), with a resolution of six angstroms and equipped with selected area electron diffraction (SAED), was used for all sample analyses. Calibration of instrument magnification was

performed using a 21,400 lines/in carbon replicate standard against a scale etched on the fluorescent screen of the TEM.

The scale etched on the screen contained divisions of 1 cm, with a mm-division scale near the center. Both scales were set so that magnifications of 10,000X and 20,000X were accurate within 5%. All diameter measurements were done under the 10X viewing microscope on the TEM. At a magnification of 10,000X, 1 mm on the screen is equal to 0.1 μ m; at 20,000X, 1 mm is equal to 0.05 μ m. The counting magnification ranged from 15,000X to 20,000X.

Each lot of grids was calibrated in two ways. First optical calibration was performed on a Leitz Dialux 20 light microscope using a standard with divisions of 0.01 mm. Second, the area of the grid holes was checked by scanning electron microscopy (SEM: International Scientific Model III-A). Calibration of scanning electron microscope magnification was performed using latex sphere standards of 2.02 μ m diameters, respectively. Optical microscopy was not selected for analysis of the samples, since the optical technique is not able to measure fibers less than 5 μ m in length.

Quantitation Techniques

The count data from each sample were analyzed by a modified version of the computer program outlined in the U.S. EPA provisional methodology (EPA, 1978). The reported data included fiber concentrations (by fiber type: chrysotile, amphibole, indeterminate, non-asbestos) as fibers/ m^3 of air. Mass concentrations of asbestos (pg/m^3 air) were calculated, although these data are subject to error accumulated from squaring particle radii in the computation*. In addition to the standard EPA reporting format, an additional

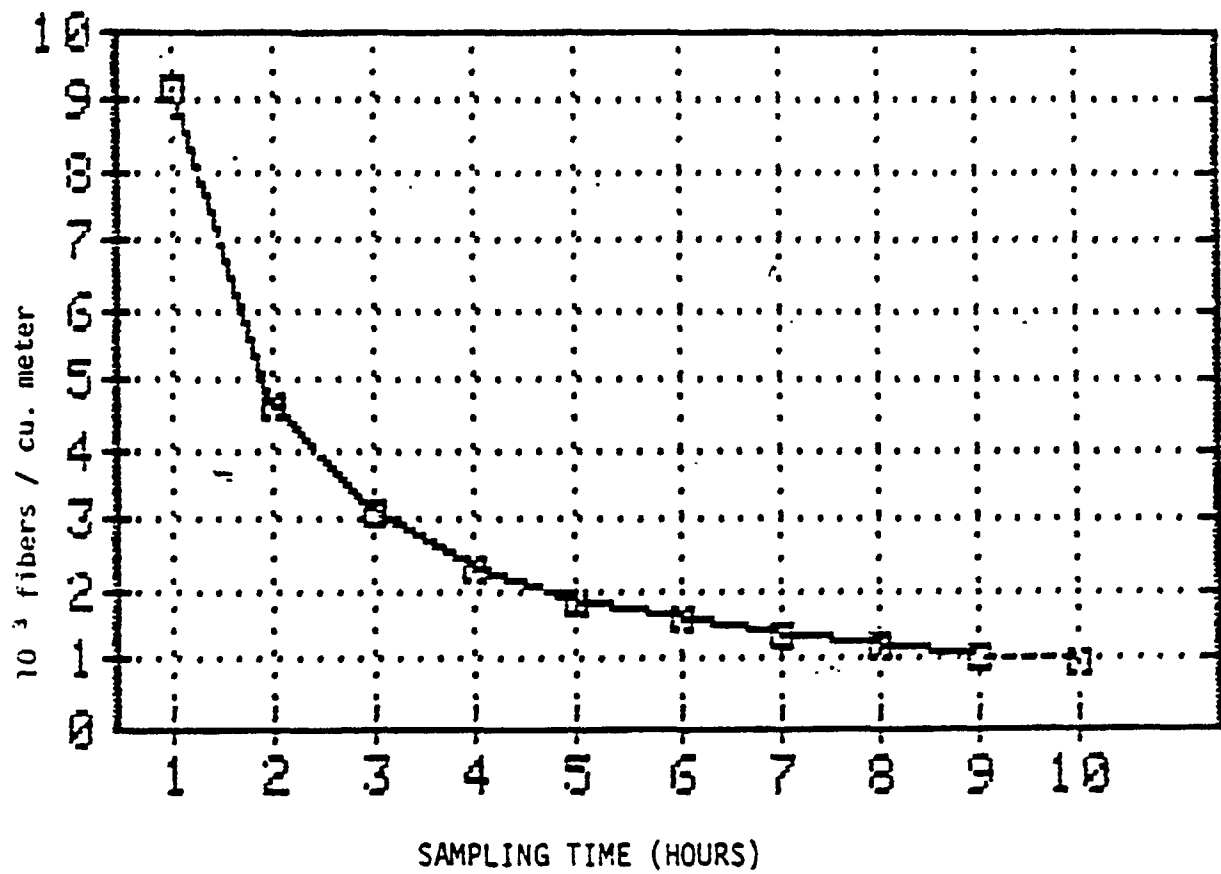
category to distinguish chrysotile fibers in the occupational exposure size range (length 5.0 μm) has been provided.

In reporting data, values shown as zero are below the detection limit (DL: a function of the total area of the filter scanned and the volume of air that is sampled). When only one fiber was found on the sample scanned, the value obtained was used to derive the detection limit. Samples having less than one fiber found during analysis were defined as being "below the detection limit" (DL). All the samples analyzed were counted in an identical manner and had been collected at the same flow rate (15.5 liters per minute), therefore, the detection limit was a function only of the duration of sampling time. A graph of detection limit vs sampling time is presented in Figure F-3. Since sampling times ranged from one to four hours, net detection limits ranged from 9,100 to 2,400 fibers/ m^3 of air, respectively.

IV. Quality Assurance

To fulfill the project's quality assurance requirements, the University of Washington Transmission Electron Microscopy Center was provided with replicate filter samples from different locations as part of an interlaboratory comparison. To insure proper handling in transit, the samples were carbon coated at SAI's laboratory and hand-carried to Seattle. The samples were analyzed according to the EPA provisional method and the data returned to SAI for computer reduction. In addition to a background control (the Napa site), sites selected for inter-comparison analyses (Table F-I) were King City, Sonora, Century City, and San Diego. The Napa sample was done in duplicate by both laboratories to provide a measure of reproducibility.

Figure F-3



Relationship Between Minimum Asbestos Detection Limit vs. Sampling Time

The detection limit for TEM asbestos analysis in this study was approximately 2,400 fibers/m³, the result of counting only one fiber in the filter area analyzed. The greatest difference between comparative samples was for the duplicated background control samples (Napa) done by the University of Washington. Although values for these two replicates are a factor of 11 apart (41,000 vs 3,600 fibers/m³), the difference represents the counting of four fibers vs one fiber per total counting area, respectively. Differences between the two laboratories' analytical results for the other replicate samples are in all cases less than the variation in the single duplicate analysis.

Table F-1

Interlaboratory Comparison of Asbestos Measurement by
Transmission Electron Microscopy

Sample	SAI TEC Laboratory		University of Washington	
	Chrysotile (fibers/m ³)	Amphibole (fibers/m ³)	Chrysotile (fibers/m ³)	Amphibole (fibers/m ³)
A-14 King City	9.4×10^3	< DL	1.5×10^4	3.6×10^3
A-24 Napa	4.7×10^3	7.1×10^3	4.1×10^4	7.2×10^3
	2.4×10^3	4.8×10^3	3.6×10^3	< DL
A-27 Sonora	2.4×10^3	2.4×10^3	2.1×10^4	1.2×10^4
A-100 Century City	1.5×10^4	< DL	1.7×10^4	2.1×10^3
A-202 San Diego	3.0×10^3	8.9×10^3	2.5×10^4	4.5×10^3

REFERENCES FOR APPENDIX F

1. John, W., A. Berner, G. Smith and J.J. Wesolowski, 1976. Experimental Determination of the Number and Size of Asbestos Fibers in Ambient Air. California Air Industrial Hygiene Laboratory Report, AIHL/ SP-1.
2. John, W., G. Reischl, and J. Wesolowski, 1978. Size-Selective Monitoring Techniques for particulate Matter in California Air. California Air and Industrial Hygiene Laboratory Report. AIHL/SP-12.
3. John, W. and G. Reischl, 1980. "A Cyclone for Size-Selective Sampling of Ambient Air." JAPCA. 3:872-876.
4. U.S. Environmental Protection Agency, 1978. Electron Microscope Measurement of Airborne Asbestos Concentrations, A Provisional Methodology Manual. U.S. Environmental Protection Agency Report, EPA-600/2-77-178, Revised June 1978.

Appendix G
Memorandum Regarding Asbestos
Contamination in Alviso, California

Sample Calculations of the Amount of Asbestos Fibers
Ingested or Inhaled into the Body

I. INHALATION OF ASBESTOS

A. AMBIENT AIR EXPOSURES

Assumptions of exposure for office worker

1. Mean asbestos concentrations range from 7,700 to 45,000 fibers/_m³ as measured by TEM (see Table III-3).
2. Breathing rate of air is 0.83m³/hr.
3. Time outside (10%) for 365 days/yr.

Sample calculation

$$\begin{aligned}\text{Yearly intake of fibers} &= \frac{7,700 \text{ fibers}}{\text{m}^3} \times \frac{0.83\text{m}^3}{\text{hr.}} \times \\ &\quad \frac{24 \text{ hr}}{\text{day}} \times 0.10 \times \frac{365 \text{ days}}{\text{year}} \\ &= 5.6 \times 10^6 \\ &\quad \text{or} \\ &= 6 \times 10^6 \text{ yearly intake of} \\ &\quad \text{fibers (estimates are rounded} \\ &\quad \text{off)}\end{aligned}$$

B. INDOOR AIR EXPOSURE - SCHOOL

Assumptions of exposure for school children

1. Breathing rate of air is 0.75m³/hr.
2. Time in school (15%) - normalize to 365 days/yr.
3. Concentrations are in PCM units. Convert to TEM units by using the conversion factor of 100 to 1000 TEM fibers/PCM fiber.
4. The median asbestos concentrations in schools range from 540 PCM fibers/m³ to 4,050 PCM fibers/m³ (see Table III-7). The low value for the estimated yearly fiber intake uses the 540 PCM concentration and the 100 TEM fibers per PCM fiber conversion factor, while the upper value uses the 4,050 PCM concentration and the 1000 TEM fibers per PCM fiber conversion factor.

Sample calculation

$$\begin{aligned}\text{Yearly intake of fibers} &= \frac{540 \text{ PCM fibers}}{\text{m}^3} \times \frac{0.75 \text{ m}^3}{\text{hr.}} \times \\ &\quad \frac{100 \text{ TEM fibers}}{\text{PCM fiber}} \times \frac{24 \text{ hr}}{\text{day}} \times 0.15 \times \frac{365 \text{ days}}{\text{year}} \\ &= 53.2 \times 10^6 \\ &\quad \text{or} \\ &= 50 \times 10^6 \text{ yearly intake of} \\ &\quad \text{fibers (estimates are rounded} \\ &\quad \text{off)}\end{aligned}$$

C. INDOOR AIR EXPOSURE - OFFICE BUILDING

Assumptions of exposure for office worker

1. Breathing rate of air is $0.83 \text{ m}^3/\text{hr.}$
2. Time in office building (33%) - normalize to 365 days/yr.
3. Concentrations are in PCM units. Convert to TEM units by using the conversion factor of 100 to 1000 TEM fibers/PCM fiber.
4. The median asbestos concentrations in office buildings range from 260 PCM fibers/ m^3 to 640 PCM fibers/ m^3 (see Table III-7). The low value for the estimated yearly fiber intake uses the 260 PCM concentration and the 100 TEM fibers per PCM fiber conversion factor, while the upper value uses the 640 PCM concentration and the 1000 TEM fibers per PCM fiber conversion factor.

Sample calculation

$$\begin{aligned}\text{Yearly intake of fibers} &= \frac{260 \text{ PCM fibers}}{\text{m}^3} \times \frac{100 \text{ TEM fibers}}{\text{PCM fiber}} \times \\ &\quad \frac{0.83 \text{ m}^3}{\text{hr}} \times \frac{24 \text{ hr}}{\text{day}} \times 0.33 \times \frac{365 \text{ days}}{\text{year}} \\ &= 62 \times 10^6 \\ &\quad \text{or} \\ &= 60 \times 10^6 \text{ yearly intake of} \\ &\quad \text{fibers (estimates are rounded} \\ &\quad \text{off)}\end{aligned}$$

D. INDOOR AIR EXPOSURE - HOMES

Assumptions of exposure for houseperson

1. Breathing rate of air is $0.83\text{m}^3/\text{hr}$.
2. Time in office building (85%) - for 365 days/yr.
3. Concentrations are in TEN units and range from 63 fibers/ m^3 to 56,000 fibers/ m^3 (see Table III-8).

Sample calculation

$$\begin{aligned}\text{Yearly intake of fibers} &= \frac{56,000 \text{ fibers}}{\text{m}^3} \times \frac{0.83\text{m}^3}{\text{hr}} \times \frac{24 \text{ hr}}{\text{day}} \times 0.85 \times \frac{365 \text{ days}}{\text{year}} \\ &= 347.3 \times 10^6 \\ &\text{or} \\ &= 350 \times 10^6 \text{ yearly intake of} \\ &\quad \text{fibers (estimates are rounded} \\ &\quad \text{off)}\end{aligned}$$

II. INGESTION OF ASBESTOS

A. TREATED WATER

Assumptions of exposure from drinking of water for children

1. Consumption rate is 1 liter/day (children) for 365 days/yr.
2. Asbestos concentrations in water range from 1×10^6 fibers/liter to $260,000 \times 10^6$ fibers/liter (see Table III-6).

Sample Calculation

$$\begin{aligned}\text{Yearly intake of fibers} &= \frac{100 \times 10^6 \text{ fibers}}{\text{liter}} \times \frac{1 \text{ liter}}{\text{day}} \times \frac{365 \text{ days}}{\text{year}} \\ &= 36,500 \times 10^6 \text{ or } 3.7 \times 10^{10} \\ &\quad \text{yearly intake of fibers.}\end{aligned}$$

III. DISCUSSION OF ESTIMATE

- A. Only exposures by the same route, either inhalation or ingestion, should be compared. It is not possible to compare health risk exposures of ingestion or inhalation because the health risk associated with ingestion have not been conclusively determined in animal and epidemiological studies (see DHS Part B report). Also, the fraction of asbestos fibers that is retained by the body has not been determined for the different exposure routes (Committee on Nonoccupational Health Risks of Asbestiform Fibers, 1984).
- B. Exposure estimates were calculated for three different lifestyles: office worker, houseperson, and school children. The amount of time that an individual is exposed to and at any given asbestos concentrations may vary considerably from the values that were assumed; however, because of the uncertainty in the measurement methods and concentrations, the range of exposures given should cover most individuals. Table H-1 lists the amount of time the office worker, houseperson, and child was expected to be in each environmental setting. The breathing rate was assumed to be $0.75 \text{ m}^3/\text{hour}$ for a child and $0.83 \text{ m}^3/\text{hr}$ for an adult. For drinking water, the consumption rate was assumed to be 1.0 liter/day for a child and 1.5 liter/day for an adult (Committee on Nonoccupational Health Risk on Asbestiform Fibers, 1984).

Table H-1

Annual Percentage of Time in Various Environments

	Office Worker (%)	Houseperson (%)	Child (%)
Ambient Air	10	10	35
Indoor-Home	57	85	50
Indoor-Office Building	33	5*	0
Indoor-Schools	<u>0</u>	<u>0</u>	<u>15</u>
	100%	100%	100%

* Accounts for time shopping and inside of other office complexes.