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.)
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 lord regulation as toxic air contaminants pursuant to Health and Safety Code (HSC)* section 39660(f). That
section states that the selection of a substance far 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 $1 \times 10^{-6}$ 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


** Although measurement of ambient asbestos fibers is currently performed using transmission electron microscopy (TEM), cancer risk in this report is expressed in teems 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

<table>
<thead>
<tr>
<th>Source</th>
<th>Source Type</th>
<th>Statewide (Ton Per Year)</th>
<th>Inventory Year</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mining</td>
<td>Point</td>
<td>120</td>
<td>1984</td>
</tr>
<tr>
<td>Milling</td>
<td>Point</td>
<td>340</td>
<td>1984</td>
</tr>
<tr>
<td>Manufacturing</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Primary</td>
<td>Point</td>
<td>4</td>
<td>1982 &amp; 1984</td>
</tr>
<tr>
<td>Secondary</td>
<td>Point</td>
<td>2</td>
<td>1982 &amp; 1984</td>
</tr>
<tr>
<td>Automobile Brakes</td>
<td>Mobile</td>
<td>0.8</td>
<td>1984</td>
</tr>
<tr>
<td>Quarrying</td>
<td>Point</td>
<td>0.5</td>
<td>1981</td>
</tr>
<tr>
<td><strong>Total</strong>*</td>
<td></td>
<td><strong>470</strong></td>
<td></td>
</tr>
</tbody>
</table>

#### 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
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.

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* 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/m$^3$ for the lower range and 50 to 500 fibers/m$^3$ 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/m$^3$, 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")

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

Numbers in parentheses represent approximate upper confidence limits. The analysis corrected for competing causes of death using life tables constructed from recent California vital statistics. Since risks for lung cancer and for other causes of death are dependent on smoking status, the life tables 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 Level (fibers/m$^3$)

<table>
<thead>
<tr>
<th>Exposure Group</th>
<th>8</th>
<th>50</th>
<th>80</th>
<th>500</th>
<th>2000</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male Smokers</td>
<td>1(0-9)**</td>
<td>6(0-55)</td>
<td>9(0-88)</td>
<td>55(0-550)</td>
<td>221(0-2,210)</td>
</tr>
<tr>
<td>Female Smokers</td>
<td>1(0-5)</td>
<td>2(0-25)</td>
<td>5(0-41)</td>
<td>25(0-250)</td>
<td>101(0-1,010)</td>
</tr>
<tr>
<td>Male Nonsmokers</td>
<td>1(0-1)</td>
<td>1(0-8)</td>
<td>1(0-11)</td>
<td>8(0-75)</td>
<td>29(0-290)</td>
</tr>
<tr>
<td>Female Nonsmokers</td>
<td>1(0-1)</td>
<td>1(0-3)</td>
<td>1(0-5)</td>
<td>3(0-28)</td>
<td>11(0-110)</td>
</tr>
</tbody>
</table>

* Calculated with C1 = 0.01. Ranges in parentheses were estimated with a lower limit of zero and an upper limit calculated with C1 = 0.1. This upper bound is an approximate upper confidence limit.

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

Conversion from TEM = (7,700 TEM fibers) (1 PCM fiber)

m$^3$ (1000 TEM fibers)

= 7.7 or 8 fibers/m$^3$

Estimated lifetime excess lung cancer risk-lower confidence limit = (PCM concentration) x (Lung cancer estimated lifetime risk)

(Estimated lifetime risk exposure concentration)

= (8 fibers/m$^3$) x 11

(100 fibers/m$^3$)

= 0.88 or 1

Estimated lifetime excess lung cancer risk - higher confidence limit = (8 fibers/m$^3$) x 110

(100 fibers/m$^3$)

= 8.8 or 9
## TABLE IV

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

**Exposure Level (fibers/m³)**

<table>
<thead>
<tr>
<th>Exposure Group</th>
<th>8</th>
<th>50</th>
<th>80</th>
<th>500</th>
<th>2000</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male Smokers</td>
<td>2(0-9)*</td>
<td>11(0-59)</td>
<td>19(0-95)</td>
<td>120(0-590)</td>
<td>470(0-2,400)</td>
</tr>
<tr>
<td>Female Smokers</td>
<td>2(0-12)</td>
<td>16(0-81)</td>
<td>26(0-120)</td>
<td>160(0-810)</td>
<td>640(0-3,300)</td>
</tr>
<tr>
<td>Male Nonsmokers</td>
<td>2(0-12)</td>
<td>16(0-79)</td>
<td>25(0-120)</td>
<td>160(0-790)</td>
<td>630(0-3,200)</td>
</tr>
<tr>
<td>Female Nonsmokers</td>
<td>3(0-16)</td>
<td>19(0-97)</td>
<td>31(0-160)</td>
<td>190(0-970)</td>
<td>780(0-3,800)</td>
</tr>
</tbody>
</table>

* Calculated with Cm = 2.4 x 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 Cm = 1.2 x 10⁻⁷, which is the highest estimated value for the proportionality constant Cm (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:

- 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.

- 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.

- 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.

- Chronic exposure to asbestos can also result in pleural changes (thickening, plaque formation, and/or benign effusions), which are generally asymptomatic.

- 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.

- 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.

- 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 "$C_1$" used by DHS was derived omitting the results from the mining milling epidemiologic studies (see Section 9.a.i.5 of Part B). The upper bound in DHS risk assessment was
calculated with $C_1$ 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).

- DHS' estimated lifetime risks of lung cancer and mesothelioma due to continuous exposure to 100 fibers/m$^3$ 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.

- 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.

- 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 numbers of long, thin fibers produced the

* Amphibole asbestos is a general term for all varieties other than chrysotile.
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 parenchyme; 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,
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 considerate or 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, it 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.