PB85-221448

Review of the Scientific Basis for EPA's (Environmental Protection Agency's) School Asbestos Hazard Program, with Recommendations to State Health Officials

(U.S.) National Inst. for Occupational Safety and Health, Cincinnati, OH

Oct 84

U.S. Department of Commerce
National Technical Information Service
NTIS

PB85-221448

50272-101

REPORT DOCUMENTATION PAGE	1. REPORT NO.	2.	3. Recipient's Accession No.

4. Title and Subtitle	5. Report Date
A Review Of The Scientific Basis For EPA's School Asbestos HazardProposal With Recommendations To State Health Officials	84/10/00
	6.
7. Author(s) Anonymous	8. Performing Organization Rept. No.
9. Performing Organization Name and Address NIOSH, U.S. Department of Health and Human Services, Cincinnati, Ohio	10. Project/Task/Work Unit No.
	11. Contract(C) or Grant(G) No. (C) (G)
12. Sponsoring Organization Name and Address SAME AS BOX 9	13. Type of Report & Period Covered
	14.

15. Supplementary Notes

16. Abstract (Limit: 200 words)

The basis for the school asbestos (1332214) hazard program of the Environmental Protection Agency (EPA) is reviewed. Risk of disease following asbestos exposure is discussed For industrial exposures, lung cancer risks cluster between 1 and 10 percent per fiber year per milliliter For mesothelioma, estimates range from 0.01 to 0.06 percent. For nonoccupational exposure, lung cancer risks range from 2 to 40 per million exposed persons Mesothelioma risks range from 2 to 100 per million The indirect quantitative risk assessment of EPA for asbestos associated cancers due to exposures at schools in early life is discussed As of May, 1982, approximately 8,600 schools contain friable asbestos and approximately 2 to 6 million students and 100,000 to 300,000 teachers, administrators, and other staff are potentially exposed to airborne asbestos in these schools. EPA estimates that over the next 30 years, approximately 1,000 premature deaths will result from current and future exposures to asbestos released from friable building materials Attention to environmental sources of asbestos by EPA have focused on potential risks for children because they are more active than adults, breathe at higher rates, and breathe more often by mouth

17. Document Analysis a. Descriptors

b. Identifiers/Open-Ended Terms

NIOSH-Publication, NIOSH-Author, Respirable-dust, Airborne-fibers. Industrial-dusts, Risk-analysis, Environmental-exposure, Quantitative-analysis, Health-protection, Health-standards, Protective-measures

c. COSATI Field/Group

18. Availability Statement	19. Security Class (This Report)	21. No. of Pages 22
	20. Security Class (This Page)	22. Price

(See ANSI-Z39.18) See Instructions on Reverse OPTIONAL FORM 272 (4-77) (Formerly NTIS-35) Department of Commerce

ii

00145154

A REVIEW OF THE SCIENTIFIC BASIS FOR EPA'S SCHOOL ASBESTOS HAZARD PROGRAM, WITH RECOMMENDATIONS TO STATE HEALTH OFFICIALS
CENTERS FOR DISEASE CONTROL
October 1984

INTRODUCTION

In view of the carcinogenic potential of inhaled asbestos fibers, public health officials should identify the presence (or confirm the absence) and evaluate the potential hazards of environmental exposures to asbestos released from controllable bulk sources in consumer products. Evaluations of school buildings have revealed the widespread presence of potentially hazardous asbestos-containing materials, with some surfaces found to be heavily damaged or deteriorated.

The Environmental Protection Agency (EPA) has placed the legal burden of administering the mandatory school asbestos hazard program on local and State educational agencies; however, the lay public members of these agencies may lack sufficient guidance as to 1) the training, technical consultation, and standardized methods necessary to conduct valid and reliable environmental sampling and analysis of bulk asbestos, 2) the limitations (sensitivity, specificity, limits of detection, and quantification) of available bulk- and air-sampling methods, 3) the quantitative risk assessment of the airborne hazard potential of any bulk asbestos identified by the sampling and analytical program, 4) what to tell the nonoccupationally exposed groups (students, parents, and community members) about their level of risk for asbestos-associated diseases, 5) what to tell the occupationally exposed groups (the administrative, teaching, custodial, and maintenance staffs) about their risks, especially if the implementation of control measures requires contact with hazardous bulk asbestos, 6) how to decide whether to implement a control program, and 7) how to choose between alternative control measures.

Reliable and precise, standardized methods of sampling and analyzing bulk asbestos should precede the application of equally valid, standardized evaluation criteria in the process of recognizing, evaluating (predicting), and controlling environmental hazards caused by airborne asbestos. Quantification of airborne asbestos fiber concentrations by air sampling is not an appropriate first approach because 1) it requires a relatively high level of expertise and expense, especially in view of the large number of buildings involved, and 2) it indicates only current airborne fiber concentrations, and thus the risks for transient and peak exposures due to episodic releases of fibers from bulk material are not reflected.

A consistent national approach is essential if the desired public health benefits of this program are to be realized and the impact of the program is to be evaluated. The following review should be helpful to State health officials who may be called upon to assist in designing, implementing, interpreting, and evaluating nonindustrial asbestos hazard programs.

Enclosed for your information is EPA's document titled "Guidance for Controlling Friable Asbestos-Containing Materials in Buildings."

Under the authority of the Toxic Substances Control Act (1976), EPA promulgated a mandatory program (40 CFR, Part 763) requiring all local public school boards to assess the potential for hazardous inhalation exposures to asbestos in primary and secondary schools by June 28, 1983 (1). From March 1979-May 1982, the school asbestos program was voluntary, and EPA, in collaboration with the U.S. Department of Health and Human Services (nee U.S. Department of Health, Education, and Welfare), the Occupational Safety and Health Administration, the Consumer Product Safety Commission, and independent consultants, prepared and distributed several guidance documents (2-9). In 1980, EPA proposed the use of an Asbestos Exposure Assessment Algorithm, based on the presence and description of eight factors for nonoccupational indoor environments (10). In practice, each factor was to be rated and given a numerical score; the sum of the scores then would provide a numerical index that could be compared with a given corrective-action scale. In October 1982, the EPA Region VII Asbestos Coordinator published an inspection manual for use with the EPA algorithm (11). However, results with the algorithm have varied greatly among both trained and untrained observers, and experts' scores have shown poor comparability (12). To provide a less ambiguous basis for decision making, EPA recently published a new guidance document that prescribes a modified method for selecting a course of action based on the use of "yes" and "no" responses rather than on the rating and scoring of each factor (13).

This review is to assist public health officials in providing up-to-date advice and consultation to educational agency officials, often the lay public, who have the legal responsibility to implement, interpret, and act upon these asbestos hazard evaluations. This will update the Centers for Disease Control's (CDC's) public health recommendations regarding asbestos hazards in buildings, dated May 9, 1977 (14).

BACKGROUND

Exposures to asbestos vary in nature, frequency, and duration, and they decrease in approximately the following order of intensity: direct occupational exposure (e.g., mining, milling, fabricating, or using asbestos-containing materials); indirect occupational exposure (e.g., that of an electrician working near an asbestos insulation worker); family contact exposure ("take-home" from the workplace); and general environmental exposures (e.g., from communitywide contamination near waste disposal sites, from industrial point-source emissions and motor vehicle brake linings, and from consumer products and damaged or deteriorated building materials made or contaminated with asbestos) (15-28).

Risk of Disease After Industrial Exposures - Reliable population-based studies on the increased risk of asbestos-associated diseases (pulmonary fibrosis, pleural thickening and asbestosis, lung cancer, and pleural or peritoneal mesothelioma) have been reported for certain groups with nontrivial, well-documented occupational exposures (29-34). The risk for both types of asbestos-associated malignancies, lung cancer and pleural or peritoneal mesothelioma, varies in a fashion consistent with a linear (nonthreshold) dose-response relationship (29-34). However, we do not completely understand the pathogenic mechanisms of mineral fiber-induced carcinogenesis, the interactive effects of other risk factors, and the dose-response relationships at extremely low levels of frequent or transient exposures (35-39).

For lung cancer, excess risks per unit of exposure vary widely, but estimates cluster between 1% and 10% for increased cancer risk per fiber-year/ml (30).* In addition, the risk for lung cancer multiplies for cigarette smokers occupationally exposed to asbestos at either high or low levels (16,30,34). Age-standardized lung cancer death rates (deaths per 100,000 person-years) among a large cohort of insulators ranged from 11.3 for unexposed nonsmokers to 58.4 for exposed nonsmokers and from 122.6 for unexposed smokers to 601.6 for exposed smokers (16). Since most lung cancers in both exposed smokers and nonsmokers occur after age 60, the risk caused by asbestos exposure before age 50 (whether transient or continuous) is virtually independent of age at first exposure and is simply proportional to the cumulative dose (34).

For mesothelioma, most estimates range from 0.01% to 0.06% (cumulative risk after 35 years' latency) per fiber-year/ml; however, the risks may be five or more times higher than this when exposures begin early in life (30-34). Cigarette smoking does not appear to increase the risk for mesothelioma in exposed individuals (34).

Risk of Disease After Nonindustrial Exposures - Environmental contamination with natural and synthetic mineral fibers is now so common (40,41) that virtually all urban dwellers have some of these fibers in their lungs, especially if they have had occupational or avocational exposures to mineral-fiber dusts (42,43). Radiologically detectable plaques, or pleural thickening and/or pulmonary fibrosis, have been associated with nonoccupational (household contact) exposures (16). Although such roentgenographic abnormalities can give evidence of asbestos exposure, they are not diagnostic unless alternative traumatic, infectious, medical, surgical, and environmental etiologies are ruled out (44). Asbestosis, a potentially disabling, nonmalignant, fibrotic lung disease, is highly

* In measurements of low-level environmental asbestos contamination, the total mass concentration of asbestos fibers per cubic meter of air (ng/m^3) is estimated by electron microscopic (EM) techniques for counting and sizing fibers (34,40). However, the most extensive and reliable exposure data available for quantitative risk assessment are from studies of occupationally exposed groups, measured by phase contrast microscopic (PCM) and polarizing light microscopic (PLM) techniques and expressed in fiber concentration (f/m^3) for fibers detectable by light microscopic methods (i.e., only those fibers longer than 5 μm). Partly because of differences in the specificity and sensitivity of these methods for identifying and quantifying asbestos fibers, the conversion factor relating mass concentration to fiber concentration ranges from 5,000 to 150,000 ng/m^3 per 1,000,000 f/m^3, with a geometric mean of about 30,000 ng/m^3 per 1,000,000 f/m^3 (i.e., about 30 f/ng) and a geometric standard deviation of about 4,000 ng/m^3 per 1,000,000 f/m^3 (about 250 f/ng). The geometric mean of the range of conversion factors should be used for environmental risk assessment, with the low mass concentrations extrapolated from fiber count (34) and with the large magnitude of variability noted in this extrapolation (30,40). In this report, we will use the geometric mean conversion factor of 30 fibers (longer than 5 μm) per nanogram (30 f/ng) of asbestos, keeping in mind that the uncertainty about this conversion factor is considerable (34,41).

dose-dependent and clearly associated with industrial exposure (15,35-39); there is no convincing evidence that disabling asbestosis is caused by nonoccupational exposures to asbestos (34).

No reliable, population-based data are available on which to base a direct quantitative assessment of the risk of asbestos-associated cancer due to take-home or other nonindustrial exposures to asbestos (29-34). However, numerous quantitative risk assessments have been based on indirect methods, explicit but different assumptions, and various sources of data on environmental exposure concentrations (30,31,34). For individuals with nonoccupational exposures to asbestos, Schneiderman et al. estimated an excess lung cancer risk of 3-30 per million exposed persons (30), and Enterline estimated the excess risk to be 2-40 per million (31). For individuals with nonoccupational exposures to asbestos, the estimated excess mesothelioma risks were 4-24 per million (30) and 100 per million (31). A comprehensive review of the risk assessments for exposures to asbestos and asbestiform fibers is available in a report of the National Academy of Sciences (45).

Nelson et al. have provided the most recent and authoritative estimated risks of death from lung cancer (Table 1) and mesothelioma (Table 2) according to age at onset of nonoccupational exposure to asbestos, duration of such exposure, sex, and smoking status (34).

A person's age at first exposure to asbestos is an important determinant of risk of mesothelioma (34). For both pleural and peritoneal mesothelioma, incidence appears to rise as a function of the third or fourth power of time since first exposure. This rise occurs irrespective of cigarette smoking; however, the magnitude of the risk is related to both the concentration and the duration of exposure. When exposure begins before age 20, the risk of mesothelioma may be similar to that of lung cancer in smokers and may be greater than that of lung cancer in nonsmokers, perhaps because of differences in the pathogenic roles of asbestos in the multistage processes that produce these different cancers (34).

Although we cannot prove that there is a linear, nonthreshold dose-response relationship after nonindustrial exposures, it is thought that such a relationship does exist, that exposure to respirable-size asbestos fibers poses a carcinogenic risk for humans, that exposure beginning early in life increases the risk for mesothelioma, and that no safe level of exposure to a carcinogenic agent has been demonstrated; therefore, sources of asbestos that are likely to result in hazardous exposures should be identified and controlled (29-34).

Table 1*

Lung Cancer: Estimated Risks of Death Per 100,000 Person-Years
Due to Continuous Nonoccupational Asbestos Exposure,
by Age at Onset of Exposure, Duration of Exposure, and Smoking Status

Age at Onset of Exposure (in Years)	Years of Continuous Nonoccupational Exposure (10,000 f/m^3)			
	1	5	10	20
Male nonsmokers				
<1	0.1 - 0.8	0.5 - 4.6	0.9 - 8.8	1.8 - 17.6
10	0.1 - 0.8	0.5 - 4.6	0.9 - 8.8	1.8 - 17.6
20	0.1 - 0.8	0.5 - 4.6	0.9 - 8.8	1.8 - 17.6
30	0.1 - 0.9	0.5 - 4.6	0.9 - 8.8	1.8 - 17.2
50	0.1 - 0.8	0.4 - 3.8	0.7 - 6.7	1.2 - 11.8
Male smokers				
<1	0.8 - 8.4	4.2 - 41.6	8.4 - 83.6	16.7 - 166.7
10	0.8 - 8.4	4.2 - 42.0	8.4 - 84.0	16.8 - 167.6
20	0.8 - 8.4	4.2 - 42.4	8.4 - 84.4	16.7 - 166.7
30	0.8 - 8.4	4.2 - 42.4	8.4 - 84.0	15.8 - 158.3
50	0.7 - 7.1	3.2 - 32.3	5.7 - 56.7	8.1 - 80.6

* This table was adapted from the Final Report of the Chronic Hazard Advisory Panel on Asbestos to the Consumer Product Safety Commission (34). Calculations were based on U.S. mortality rates for 1977, adjusted to account for secular changes in the risk of lung cancer in male smokers compared with male nonsmokers (34,46,47). Patterns for female smokers and nonsmokers are similar to those given for males (34). From the authors' linear, nonthreshold dose-response model, the risks for lung cancer can be extrapolated from alternative assumptions of age at onset of exposure, duration of continuous exposure, smoking status, and level of exposure (34).

Table 2*

Mesothelioma: Estimated Risks of Death Per 100,000 Person-Years
Due to Continuous Nonoccupational Asbestos Exposure,
by Age at Onset of Exposure, Duration of Exposure, and Smoking Status

Age at Onset of Exposure (in Years)	Years of Continuous Nonoccupational Exposure (10,000 f/m^3)			
	1	5	10	20
Male nonsmokers				
<1	3.7 - 37.4	17.1 - 170.9	30.7 - 307.0	49.4 - 493.5
10	2.4 - 23.5	10.6 - 105.8	18.8 - 187.7	29.2 - 291.9
20	1.3 - 13.4	6.1 - 61.3	10.5 - 105.4	15.7 - 157.1
30	0.7 - 7.1	3.2 - 31.5	5.3 - 52.5	7.4 - 73.9
50	0.1 - 1.3	0.5 - 4.6	0.7 - 6.7	0.8 - 8.0
Male smokers				
<1	3.2 - 31.9	14.5 - 144.9	25.7 - 256.6	41.2 - 412.4
10	2.0 - 19.7	8.8 - 88.2	15.5 - 154.6	23.4 - 233.5
20	1.1 - 10.9	4.9 - 49.1	8.4 - 84.0	12.3 - 123.5
30	0.5 - 5.9	2.4 - 24.3	4.0 - 40.3	5.5 - 5[illegible].4
50	0.1 - 0.8	0.3 - 3.4	0.5 - 4.6	0.5 - 5.5

* This table was adapted from the Final Report of the Chronic Hazard Advisory Panel on Asbestos to the Consumer Product Safety Commission (34). Calculations were based on U.S. mortality rates for 1977 (34). Patterns for female smokers and nonsmokers are similar to those given for males (34). The risks for mesothelioma can be extrapolated from alternative assumptions of age at onset of exposure, duration of continuous exposure, smoking status, and level of exposure (34).

EPA'S INDIRECT QUANTITATIVE RISK ASSESSMENT FOR ASBESTOS-ASSOCIATED CANCERS DUE TO EXPOSURES AT SCHOOL IN EARLY LIFE

Because the onset of asbestos-associated cancers generally follows initial exposures only after long latency periods of 20 to 30 years or more, the early recognition, evaluation, and control of potentially hazardous exposures to asbestos are essential. This is especially true for environments in which infants, children, and young adults may be exposed to airborne asbestos fibers from a wide variety of consumer products. Such environments may include homes, day-care facilities, and schools where asbestos-containing construction and insulation materials (especially sprayed-on materials [3] and possibly floor tiles [48]) may be deteriorated, friable (easily crumbled), or otherwise likely to result in fallout (e.g., from frequent mechanical disruption). Asbestos was used extensively in school and other construction from 1946 to 1978 (4-7).

Between 1969 and 1970, concentrations of asbestos in ambient (outdoor) air were measured in 48 cities in the U.S.A. Asbestos was detectable in the air of virtually every metropolitan area; however, ambient levels never exceeded 100 ng/m^3 (about 3,000 f/m^3--see footnote on page 3 concerning the use of a conversion factor of about 30 f/ng), except near sources of asbestos emissions (e.g., within 0.5 miles of an ongoing asbestos spray fireproofing operation where levels as high as 500 ng/m^3—about 15,000 f/m^3—were measured) (41). In the homes of chrysotile asbestos mine and mill workers, five (38%) of thirteen 4- to 8-hour daytime air samples contained between 200 and 5,000 ng/m^3 (about 6,000 to 150,000 f/m^3), whereas airborne asbestos concentrations in the homes of nonminers in the same town were routinely less than 100 ng/m^3 (about 3,000 f/m^3) (41). In 10 public schools, evaluated because of visibly damaged areas of sprayed-on chrysotile asbestos, the airborne concentrations in 4- to 8-hour daytime indoor samples ranged from 9 to 1,950 ng/m^3 (270 f/m^3 to 60,000 f/m^3), with an average of about 220 ng/m^3 (6,600 f/m^3), whereas outdoor samples at three of these schools averaged 14 ng/m^3 (420 f/m^3) (41). A more representative, random survey of 25 schools with asbestos surfacing materials gave similar results, even though these schools were not selected because of the presence or absence of damaged materials. In that survey, average levels of about 240 ng/m^3 (7,200 f/m^3) were found in rooms with asbestos surfaces, 54 ng/m^3 (1,600 f/m^3) in rooms that were in the same buildings but that did not have asbestos surfaces, and 8 ng/m^3 (240 f/m^3) in samples of air outside these buildings.

On the basis of a survey of the nation's schools, EPA estimated that as of May 1982 about 8,600 schools contained friable asbestos (1). Although recognizing various limitations to the validity of these data, Nicholson has estimated that about 2 to 6 million students and 100,000 to 300,000 teachers, administrators, and other staff, including approximately 23,000 janitorial and maintenance workers, are potentially exposed to airborne asbestos in these schools (8,41).

Environmental asbestos exposure may increase the risk for preventable premature mortality due to lung cancer (beyond the proportion that could be attributable to other nonoccupational exposures such as cigarette smoke and ionizing radiation) and mesothelioma (30,31,34,45). In the absence of population-based data for nonindustrially exposed groups, EPA and others have provided indirect estimates of the general population risks by means of quantitative risk assessment methods (8,30,31,34,41,45).

Using a number of controversial but explicit assumptions, EPA has estimated that over the next 30 years about 1,000 premature deaths (minimal and maximal estimates = 100 and 7,000) will result from current and future exposures to asbestos released from friable building materials. Although an estimated excess lifetime risk of 1,000 premature deaths per 90,000,000 person-years (30 years of exposure for an average population size of 3 million people) may not seem to represent an unusually large individual risk ratio, EPA regards this as an important national public health problem, especially since 90% of these deaths would be expected to result from children's exposures that could have been prevented (8,41).*

The assumptions used in the EPA risk assessment included the following: reference exposure and epidemiologic data from mortality studies of asbestos-exposed insulation workers; estimates of the prevalent levels of airborne asbestos exposures in schools containing friable asbestos from data on buildings surveyed in European and American cities; the extent of contamination and size of the populations at risk, from the above-mentioned survey of U.S. schools in which asbestos was considered a potential hazard only if it was friable; no change in smoking habits (assumed to be the same as those of the reference population of insulation workers) over the next 30 years; an extrapolation of four orders of magnitude, from the exposure levels experienced by the insulation workers, with no consideration given to the influence that children's longer life expectancy would have on the risk for mesothelioma; and no peak exposures over the estimated mean levels (8). An additional assumption was that the cumulative exposures for 3.27 million current school occupants (about 90% students) were calculated as if they were a cohort that would be exposed for 1,000 hours per year (students) or 2,000

* The rule on asbestos hazards in schools was partly justified by EPA because of the need to control "peak" exposures (1). In buildings containing friable asbestos materials, peak exposures of up to 500,000 ng/m^3 (15,000,000 f/m^3) have been documented and may be common during simple maintenance or cleaning operations or after vandalism and other damage (1,8,41). The average adult male inhales about 9.6 m^3 of air per 8 hours of light physical activity, and the average 10-year-old child inhales about 6.24 m^3 in the same period of light physical activity. During periods of rest or maximal exercise, the volume of inspired air may be about one-third or five times the given values, respectively (49). A "school year" of exposure is about 1,000 hours (6 hours per day for 5 days per week and 33 weeks per year), whereas a "work year" of exposure is about 2,000 hours (8 hours per day for 5 days per week and 50 weeks per year). With these factors in mind, it is important to note that a peak childhood exposure to 500,000 ng/m^3 (15,000,000 f/m^3) for 1 hour results in inhalation of the same number of fibers as exposure to 500 ng/m^3 (15,000 f/m^3) over a full school year. Since adult school workers inhale about 50% more air at similar levels of activity and are exposed to the school environment for about twice as many hours per calendar year as students, they would inhale the same number of fibers at about one-third the peak or annual exposure levels given in the example for children.

hours per year (staff) over the 30-year period that the school buildings are expected to remain in service (8). Although EPA recognized that over this time the current students and staff would be replaced by others, the agency was able to simplify its risk estimates by assuming that the size of the exposed population would remain at about 3.27 million, by using cumulative exposures, and by assuming a linear nonthreshold dose-response relationship (8,30,34,45).

More cautious assumptions (e.g., the occurrence of peak exposures and the use of a time-dependent dose-response model), which reflect the greater magnitude of mesothelioma risk for exposed children, would considerably increase the above risk estimates.

THE RATIONALE BEHIND EPA'S SCHOOL ASBESTOS HAZARD PROGRAM

EPA's attention to controllable environmental sources of asbestos exposures has been focused on the relatively greater potential risks for children than for adults partly because children are more active, they breathe at higher rates and more often by mouth, they spend more time close to the floor where sedimented dust accumulates, and they have an anticipated longer remaining life span during which the chronic effects of asbestos exposure may be manifested (2-8).

The EPA policy assumes: 1) that valid and reliable methods of inspection, sample identification and collection, and analysis will be used by adequately trained individuals to detect the presence of bulk asbestos in school environments, 2) that evaluation criteria based on such data will permit a quantitative estimate of the hazard potential for deterioration, disturbance, fallout, and resuspension of airborne respirable-size fibers, 3) that such criteria may be used for selecting the most appropriate control strategy among several alternatives that vary in effectiveness and technical and economical feasibility, 4) that implementation of such control measures will significantly reduce the overall lung burden from environmental exposures to asbestos fibers in school populations, and 5) that such a reduction in lung burden will significantly reduce the risk for delayed onset of asbestos-associated cancer in these populations. However, regardless of the logic behind the program, EPA has proposed no means for evaluating the effectiveness of its implementation, and preliminary evidence indicates that, in practice, program operations will vary markedly (12,50). We know of only two States (South Carolina and Arizona) in which the State health department has prescribed and administered the training, certification, and methods to be used in each school asbestos hazard evaluation (51,52).

ENVIRONMENTS AFFECTED BY EPA'S RULE

The mandatory EPA rule calls for an asbestos hazard evaluation in all nonprofit public schools. More details on the legal definitions of "nonprofit," "public," and "schools" may be obtained from the rule itself (1). The EPA rule does not mandate an evaluation of asbestos hazards in other indoor or outdoor environments.

THE MANDATED PROCESS OF INSPECTION, IDENTIFICATION, AND NOTIFICATION

The steps involved in complying with the EPA rule consist of three phases: inspection, identification, and notification (13). The implementation of control measures is not mandated. Certainly, ethical and legal issues may arise when potentially hazardous asbestos-containing materials are found in the school environment (1,16,21,33).

In the following outline of the required process, some suggestions are included that, although not required under the letter of the rule, appear to be appropriate:

1. Inspect the entire school building for deteriorated, water-damaged, or friable material that may contain asbestos and be subject to fallout or mechanical disruption (4,5,11,13).

2. If such material is found (e.g., on floor or ceiling tiles, in pipe lagging, in sprayed materials, or on jackets of boilers or furnaces), take systematically selected random bulk samples by removing all layers of three or more representative portions of the material with a suitable sampling device (e.g., a scalpel or trephine) and putting them into a clean collecting device (e.g., a 35-mm film canister). Use appropriate respiratory protection and work practices when obtaining the samples to minimize potential personal and environmental exposures to asbestos fibers.*

3. Carefully label each container to show the sampling site, and submit the samples to a competent laboratory to determine if they contain asbestos. Specify the preferred analytical method (polarizing light microscopy with dispersion staining or electron microscopy), and require that the laboratory report its findings with quality control data on the sensitivity, specificity, limits of detection, possible interferences, and confidence limits of quantitation for the method as used in that laboratory.

4. Evaluate the potential for human exposure if the presence of asbestos is confirmed, using a standardized set of evaluation criteria that include the condition and type of product, the likelihood of water damage, the accessibility and amount of exposed surface area, air movement in the vicinity, human activity in the vicinity, friability, the number and age of occupants, the average duration of occupancy,

---

* To provide advice on sampling and analyses, including a list of laboratories that are competent in the polarized light microscopic method of analysis, EPA maintains a toll-free telephone number: 1-800-334-8571. EPA has advised that in the process of obtaining samples of random or suspect building materials, respiratory protection is unnecessary, although exposures of up to 100,000 f/m^3 may occur during sample collection (1). However, we believe that the use of personal respiratory protection and precautions against releasing fibers to the environment during sampling (such as enclosing and wetting the surface area to be sampled) would be prudent.

the frequency and methods of cleaning the exposed surface, and the percentage of various types of asbestos in the material, by weight (13).

5. Post warnings as prescribed by EPA, and notify potentially exposed teachers, custodians, other staff, and parent-teacher associations of the findings and any recommended control measures.

IDENTIFICATION AND EVALUATION OF ASBESTOS HAZARDS - SOME CAVEATS

In Industrial Occupational Settings Where Asbestos Is Known To Be Present - In the mining, milling, formulation, or application of a product that is known or suspected to contain asbestos, the hazard's recognition, evaluation, and control depends on the sampling and analysis of airborne respirable-size asbestos fibers. Fibers less than 3.5 µm in diameter are considered respirable (15).

The Occupational Safety and Health Administration (OSHA) standard for occupational exposure to airborne asbestos is based on the concentration of fibers that are longer than 5 micrometers (µm) and are thus resolvable by a 400-500 X magnification phase-contrast microscope (PCM) (53). Since 1976, the OSHA standard has limited a worker's 8-hour time-weighted average (TWA) exposure to 2,000,000 fibers (longer than 5 µm) per m^3 (f/m^3). In December 1976, NIOSH recommended to OSHA that this standard be lowered to 100,000 f/m^3 (8-hour TWA) (15). In November 1980, the 100,000-f/m^3 limit was selected by NIOSH again on the basis of the best available data concerning health risks and the validity and reliability of available methods for sampling and analyzing airborne asbestos fibers (19). Because of the well-documented human carcinogenicity of asbestos and the apparent lack of any threshold (no-effect level) in its carcinogenic effects, NIOSH's ultimate goal in recommending occupational exposure limits has been to eliminate asbestos exposure. Although the 100,000-f/m^3 limit was considered not feasible, partly because of the limitations imposed by currently accepted methods of sampling and analysis, NIOSH's recommendation was intended to 1) protect against the noncarcinogenic effects of asbestos, 2) materially reduce the risk of asbestos-induced cancer, and 3) be measurable by techniques that are valid, reproducible, and widely available to industry and to official agencies (19).

In November 1983, OSHA issued an emergency temporary standard that would have lowered the worker's 8-hour TWA exposure to 500,000 f/m^3. This emergency standard was suspended by judicial order (November 23, 1983), and the limit of 2,000,000 f/m^3 is the current OSHA standard for occupational exposure to airborne asbestos. In June 1984, NIOSH reiterated its recommendation for an occupational exposure limit of 100,000 f/m^3, noting recent improvements in the sensitivity and reliability of available methods for sampling and analyzing airborne fibers (54,55).

Some researchers believe that asbestos fibers less than 5 µm long may be carcinogenic and that they should be included in the airborne fiber count; however, only supplemental use of the more expensive and sophisticated analytical EM methods would permit detection of such short fibers. Other investigators believe that the main hazard is from asbestos fibers longer than 10 to 15 µm (those that cannot be fully ingested by single cells in the lung).

Still other investigators have hypothesized that any durable mineral fiber 0.25 µm or less in diameter and longer than 8 µm may be capable of inducing or promoting carcinogenesis if inhaled (56-58). The portion of very short or very long fibers in the total weight of fibers collected by air sampling varies greatly (15,30,34).

Clearly, the sensitivity of the method for identifying airborne asbestos depends on whether PCM or EM methods are used and on whether the distribution of fibers by size and the absolute fiber count are determined.

Of more fundamental concern is the fact that the PCM analytical method used under the OSHA standard for airborne asbestos is not specific for asbestos fibers. This may affect both the sensitivity and the specificity of the method. Under OSHA's standard, fibers are identified only by the requirements that the observed particulate must have a length-to-diameter (aspect) ratio of 3:1 or greater and be detectable by PCM methods. The physical, chemical, and mineralogical nature of the material need not be determined. Thus, glass fibers or other refractile fibrous minerals may be counted (false positives), and asbestos fibers or fibrils too small to be detected by light microscopic methods may not be noted (false negatives) (19).

In Nonindustrial Settings (Such as Schools) - There are no uniformly accepted, standardized evaluation criteria (at least seven algorithms have been used) for predicting the aerosolization potential of respirable fibers from asbestos-containing bulk material (12,13). State health departments have only limited economic, human, and technical resources available for evaluating the hazards of nonoccupational exposures to asbestos and other indoor air pollutants (59). In practice, some or all of the factors (listed in item 4 under the previous section) are scored for each sample analyzed; the total scores for each sample are then compared with predetermined criteria so that the relative hazard potential of each sampling site can be rated (10,12). The latest EPA guidance document (13) suggests the use of "yes" and "no" responses rather than a scoring system, thus reducing ambiguity; however, that method has not been independently evaluated (12).

Considerable controversy surrounds the adequacy of the PCM light microscopic method's sensitivity and specificity for identifying asbestos fibers in air samples; however, in bulk samples the use of a light microscopic method may be sufficiently sensitive (the size of fibers is not likely to limit detection) and specific if polarizing light microscopy (PLM) or PLM in conjunction with dispersion staining is used in a laboratory with good quality control (15).*

---

* Advice on the results of quality control tests by various laboratories may be obtained from EPA by telephone (1-800-334-8571). The cost of analyses by light microscopic methods varies from about $25 to $45 or more per sample. The cost of analyses by EM methods varies from about $100 to several hundred dollars per sample.

When air samples are collected (e.g., during routine periodic monitoring of an environment containing potentially hazardous bulk asbestos materials or after an asbestos abatement or removal program), the "action level" should conform with a policy of lowest feasible level.*

Use of the revised NIOSH PCM air sampling method, including modified rules for counting only fibers with aspect ratios of 5:1 or more in a 1,000-liter sample of air, will permit detection and quantitation of about 10,000 f/m^3 if a coefficient of variation of about 25% is considered acceptable for risk-management decisions (54,55,60). This variability is reasonable, since the conversion factor (30 f/ng) used to convert mass concentrations to fiber concentrations in environmental risk assessments has such a large uncertainty factor (250 f/ng). An "action level" of 10,000 f/m^3 may be useful as a guideline for monitoring a building with potentially hazardous asbestos surfaces as part of a comprehensive asbestos program or during abatement work, maintenance, etc. It is not a recommended "occupancy" or "safe" level.

Studies of occupational groups have shown no clear evidence that comparable exposures to different asbestos fiber types or formulations result in different levels of risk for asbestos-associated cancers (34). Only analytical EM and PLM methods can distinguish the specific mineralogical types of asbestos (15). When the revised NIOSH exposure monitoring method is applied to environmental settings, about 5% of the air samples below 10,000 f/m^3 and all of the samples that contain more than 10,000 f/m^3 should be further analyzed by EM or PLM methods for specifically determining the identity of fibers detected by the PCM method (54,55,60).

Investigators at NIOSH have developed a screening test for asbestos, the K^2 method (a colorimetric test interpreted visually by the investigator), which may be used in the field. It is extremely sensitive (61); however, recent experience indicates that false-negative results can occur with materials containing more than 1% asbestos (62). Since the specificity of a screening test is of considerable importance in determining the predictive value of a positive test, it is important to note that false-positive results are common with the K^2 asbestos screening test. Thus, positive samples must be confirmed by analytical EM or PLM methods (62). In a stratified random sample of Colorado schools, in which the method of dispersion staining with PLM was used for confirming positive K^2 tests, the specificity of the K^2 test was only about 21% (28). Under these circumstances, the predictive value of a positive K^2 test was only about 56% (28,62). The K^2 test probably should not be recommended for use as a screening test (62).

An algorithm developed for risk assessment of asbestos in the Colorado schools identified 31 of 41 randomly selected schools that had asbestos material in

* The concept of an environmental "action level" is not the same as that of a permissible exposure limit that is precisely monitored for compliance with regulatory standards. As used here, it is consistent with CDC's policy of recommending that asbestos exposures be reduced to the lowest feasible level; it is readily measured by using the revised NIOSH PCM method (54); and it should be helpful to authorities who must make risk-management decisions when the general public is potentially exposed to a well-documented human carcinogen.

one or more locations, and most of these had high exposure potentials, on the basis of relative scores for six evaluation criteria: condition or degree of deterioration of the material, its accessibility, air movement, human activity, friability, and percentage of asbestos. For each sample site, each criterion was ranked 1 to 3, except the percentage of asbestos, which was ranked 1 to 4. A score of 8 or less was considered a negligible hazard, and 9 or more indicated that the site required corrective action (28,62).

On the basis of these studies, 63%-89% of the public schools in Colorado were estimated to pose a potentially serious asbestos hazard to staff, children, and community groups who use these schools (28). This is about two to three times the national average estimated from EPA's survey (8); however, this average may reflect differences in the sampling and analytical methods and evaluation criteria of the "Colorado algorithm" rather than in the actual prevalence of hazardous asbestos problems at schools in Colorado (12).

NOTIFICATION: THE LEGAL PROCESS AND AN OPPORTUNITY FOR EDUCATION AND RISK REDUCTION

The principal legal requirement appears to be that potentially exposed occupational and nonoccupational school occupants should be notified that friable asbestos has been identified in their school. There is no mandate to provide the occupants with a quantitative estimate of their risk for asbestos-associated diseases. Such an estimate would be very difficult to make, since valid and reliable data on levels of exposure based on air-sampling are difficult to obtain in these settings.

The only other legal requirement appears to be that school employees should be notified of OSHA requirements (i.e., for training, supervision, protective equipment, monitoring, and medical surveillance) if the asbestos is removed or if their tasks result in more intense occupational exposures.

From a public health perspective, potential exposures to low levels of asbestos in nonindustrial settings may be less important than exposures to cigarette smoke (in relation to one's ultimate risk for premature morbidity and mortality). Therefore, when a potential asbestos exposure hazard is identified, the notification to the school should be accompanied by information on the numerous benefits of not smoking, including the reduced synergistic risk for lung cancer due to historical or future exposures to asbestos. It should be made clear, however, that no such benefit has been demonstrated for reducing the risk of pleural and peritoneal mesothelioma and that exposure to asbestos may carry a risk of lung cancer even for nonsmokers.

CONTROL MEASURES - HEALTH AND ECONOMIC IMPACT OF THE EPA RULE

The rule does not mandate that corrective or control measures be taken if a potentially hazardous exposure to asbestos is identified; however, EPA's regional offices can provide technical information and perhaps assistance regarding control measures. Advice and technical assistance to workers and their supervisors called upon to implement control measures may also be obtained from NIOSH or OSHA regional offices. This advice may include engineering controls, exhaust ventilation, work practices, personal protective equipment such as adequate respiratory protection, and medical examinations.

In selecting the most appropriate control measures, school officials should consider the following factors:

1. The location and amount of the asbestos-containing material(s).
2. The condition and function of the material(s).
3. The likelihood of present or future fallout or disruption of the material(s).
4. The economic cost, technical feasibility, and potential for hazardous occupational versus nonoccupational exposures in the course of various control measures.

The alternative control methods are as follows (13):

1. Encapsulation (with an effective sealant) reduces the likelihood that fibers will be released into the building environment as long as the sealant remains intact. If this method is used, a comprehensive asbestos hazard program should be instituted on the basis of current OSHA regulations and NIOSH recommendations. Such a program should include the designation of one competent administrator who would be responsible for organizing and conducting routine periodic inspections and environmental monitoring (using the lowest feasible action level, e.g., 10,000 f/m^3); education and training of potentially exposed individuals; respirator selection, maintenance, and use; and recordkeeping.
2. Enclosure (with a barrier such as a suspended or false ceiling) reduces the likelihood that incidental contact with the asbestos-containing material will occur as long as the barrier remains intact and entry into the enclosed space is not required. If this method is used, a comprehensive asbestos hazard program, as described above, would be advisable.
3. Administrative management may effectively minimize the problem if no action is required immediately and if potential sources are inspected periodically. If this method is used, a comprehensive asbestos hazard program, as described above, would be advisable.
4. Removal eliminates the source of the contamination. However, control by removal may cause considerable exposure risk for workers and for future occupants unless disrupted material is removed properly and completely, appropriate work practices are used, and respiratory protection is provided.

Under the EPA rule, it is not necessary to follow up the positive identification of a potentially hazardous exposure to bulk asbestos with a demonstration of airborne respirable asbestos fibers in the affected environment(s). In fact, a comprehensive evaluation (sampling and analysis) of airborne asbestos concentrations--even in a relatively circumscribed environment--is very costly, and highly sophisticated human and technical resources are required to obtain valid results. Furthermore, in a given sampling only the current airborne fiber concentration is measured, and thus the risks for transient and peak exposures due to episodic releases of fibers from bulk material are not reflected.

Since the financial and human costs of any control measure may be high, the avoidance of a false-positive identification of an asbestos hazard is an important consideration in implementing a program to comply with the EPA rule.

In August 1984, the Asbestos School Hazard Abatement Act of 1984 established an EPA program to provide financial assistance to local and State educational agencies that have identified sources of asbestos that are potentially hazardous to the health of schoolchildren and employees. This fall, EPA will send an informational package to the office of each State Governor concerning plans for implementing this Act. Application forms will be sent directly to the local educational agencies to be completed. These applications will be processed by the State, and EPA will assign priorities on the basis of the nature of the asbestos hazard and the financial need of the affected school. EPA's review and evaluation will determine who receives financial assistance. Since funding is limited, EPA strongly encourages local educational agencies and State governmental officials to begin abatement efforts and not delay or revise plans in anticipation of federal assistance. For further information on this Act, contact your EPA Regional Asbestos Coordinator as provided in this advisory.

RECOMMENDATIONS

The primary prevention of hazardous exposures to toxic agents is one of the goals that the Surgeon General identified in his 1980 report titled "Promoting Health/Preventing Disease: Objectives for the Nation." The early identification, evaluation, and control of occupational and nonoccupational exposures to previously unrecognized asbestos is consistent with these goals and should provide important public health benefits for the nation.

State health departments may be called upon to assist local and State educational agencies in implementing EPA's efforts to meet these goals. In addition, States may wish to identify and control other potentially hazardous asbestos exposures in environments and consumer products not covered under the EPA school asbestos hazard program. We hope that the preceding information and the following suggestions will be helpful in designing and conducting such efforts.

1. Standardized reliable and valid methods of asbestos hazard evaluation are necessary, especially if there is to be periodic reevaluation of asbestos hazards and an overall assessment of the effectiveness of the EPA rule (34,45,50,59).

2. Risk-management decisions regarding the implementation of alternative control measures for identified nonindustrial asbestos hazards should be based on an environmental carcinogen policy of control at the lowest feasible level (12,13).

3. EPA has announced that it will reevaluate the current regulation "Asbestos; Friable Asbestos-Containing Materials in Schools; Identification and Notification" (1). We recommend support of this reevaluation and any potential efforts on the part of the EPA to develop uniform methods for surveillance of school asbestos hazards and to develop uniform criteria for conducting remedial activities.

4. In selected settings where the potential for study exists, the effectiveness of alternative control measures should be evaluated in relation to the level of hazard determined by a set of standardized evaluation criteria. Effectiveness may be defined either in terms of assessing airborne asbestos concentrations in selected buildings, following cohorts of building occupants with appropriate medical-epidemiological surveillance, or in some other way, such as determining the lung burden (biological monitoring of exposure) of asbestos in randomly selected pets or children who die of unrelated causes (63).

5. Population-based epidemiologic data should be obtained on the magnitude and extent of nonindustrial inhalation exposures to asbestos and on the distribution and occurrence of asbestos-associated diseases among nonindustrially exposed groups. State health departments may be able to assist in developing such data by coordinating the results of mandated school asbestos hazard evaluations in their respective States, especially if standardized methods are used.

   The risk-assessment analysis noted earlier in this document suggests that of the various incidences of asbestos-related diseases, mesothelioma incidence is the most likely to be affected by school asbestos exposures. The surge in occupational exposure during and after World War II would also be expected to have a marked impact on mesothelioma incidence. A nationwide reporting system and surveillance for mesothelioma would be valuable for estimating the validity of the asbestos risk-assessment predictions made to date. It would also enable many specific studies to be done on the relationship between specific asbestos exposure situations and mesothelioma incidence. CDC and State health departments, therefore, should consider establishing a national reporting system for mesothelioma to reflect trends in incidence and to serve as a basis for epidemiologic studies.

6. Additional laboratory and epidemiological studies should be conducted to define safe exposure limits for fibrous and platy minerals used in consumer products and building materials (e.g., vermiculite, talc, perlite, wolastonite, or glass or rock wool), since the long-term health consequences of low-level exposures to these minerals are not well understood (45,56-58).

FURTHER ASSISTANCE

In Occupational Settings - Technical assistance in recognizing, evaluating, and controlling asbestos hazards may be obtained from CDC/NIOSH regional offices or from the Division of Surveillance, Hazard Evaluations, and Field Studies, Cincinnati, Ohio 45226.

In Nonoccupational Settings - State and local health departments may obtain technical assistance concerning health risks in private homes and multiple family dwellings from the Center for Environmental Health (CEH), CDC. CEH may provide consultation regarding the health effects of asbestos but has limited resources for field assistance in the recognition, evaluation, or control of indoor, nonoccupational exposures to asbestos. Assistance may also be obtained from the Consumer Product Safety Commission's regional offices or from those of the EPA. The EPA Regional Asbestos Coordinators' addresses are listed in Appendix B of the enclosed document, "Guidance for Controlling Friable Asbestos-Containing Materials in Buildings."

REFERENCES

1. Environmental Protection Agency. Asbestos; friable asbestos-containing materials in schools; identification and notification (final rule). Federal Register 1982;47:23360-89.

2. Nicholson WJ, Rohl AM, Weisman I. Asbestos contamination of the air in public buildings. (NTIS #PB-250-930.) Springfield, VA: Environmental Protection Agency, 1975 Oct.

3. Nicholson WJ. Control of sprayed asbestos surfaces in school buildings: a feasibility study. New York, NY: Environmental Sciences Laboratory, Mount Sinai School of Medicine, 1978 Jun.

4. Environmental Protection Agency. Asbestos-containing materials in school buildings: a guidance document, Part 1. (EPA/OTS C00090.) Washington, DC: Environmental Protection Agency, 1979 Mar.

5. Environmental Protection Agency. Asbestos-containing materials in school buildings: a guidance document, Part 2. (EPA/OTS C00090.) Washington, DC: Environmental Protection Agency, 1979 Mar.

6. Environmental Protection Agency. Friable asbestos-containing materials in schools; proposed identification and notification (notice of proposed rulemaking). Federal Register 1980;45:61966-97.

7. Environmental Protection Agency. Measurement of asbestos air pollution inside buildings sprayed with asbestos. (EPA/OPTS 560/13-80-026.) Washington, DC: Environmental Protection Agency, 1980 Aug.

8. Environmental Protection Agency. Support document for proposed rule on friable asbestos-containing materials in school buildings. (EPA/OPTS 560/12-80-003.) Washington, DC: Environmental Protection Agency, 1980 Oct.

9. Lentzen DE, Brantly EP, Gold KW, Myers LE. Interim method for the determination of asbestos in bulk insulation samples. (EPA/OPTS 600/4-82-020.) Washington, DC: Environmental Protection Agency, 1982.

10. U.S. Department of Health, Education, and Welfare. Asbestos detection and control: local educational agencies; asbestos detection and state plan: state educational agencies. Federal Register 1980;45:61950-64.

11. Brandner W. Asbestos exposure assessment in buildings: inspection manual. Kansas City, MO: Environmental Protection Agency, Region VII, 1982 Oct.

12. Findley ME, Rose VE, Cutter GR, Windsor RA. An assessment of the Environmental Protection Agency's asbestos hazard evaluation algorithm. Am J Public Health 1983;73:1179-81.

13. Environmental Protection Agency. Guidance for controlling friable asbestos-containing materials in buildings. (EPA/OPTS 560/5-83-002.) Washington, DC: Environmental Protection Agency, 1983.

14. Center for Disease Control. Public health recommendations regarding asbestos-spray building materials. Atlanta: Center for Disease Control, 1977 May.

15. Finklea JF. Revised recommended asbestos standard. (NIOSH Publication #77-169.) Cincinnati, OH: National Institute for Occupational Safety and Health, 1976.

16. Selikoff IJ, Hammond EC, eds. Health hazards of asbestos exposure. Ann NY Acad Sci 1979;330:1-811.

17. Lemen R, Dement JM, eds. Dusts and disease. Park Forest South, IL: Pathotox Publishers, Inc, 1979.

18. Centers for Disease Control, National Institute for Occupational Safety and Health. Occupational exposure to talc containing asbestos: morbidity, mortality, and environmental studies of miners and millers. (NIOSH Publication #80-115.) Cincinnati, OH: National Institute for Occupational Safety and Health, 1980.

19. Millar JD. Workplace exposure to asbestos: review and recommendations. (NIOSH Publication #81-103.) Cincinnati, OH: National Institute for Occupational Safety and Health, 1980.

20. Selikoff IJ, ed. Scientific basis for evaluation of asbestos-associated disease: course outline. New York, NY: Environmental Sciences Laboratory, Mount Sinai School of Medicine, 1982 Dec.

21. Selikoff IJ. Disability compensation for asbestos-associated disease in the United States. New York, NY: Environmental Sciences Laboratory, Mount Sinai School of Medicine, 1982.

22. Dement JM, Harris RL, Symons MJ, Shy CM. Exposures and mortality among chrysotile asbestos workers. Parts I (Exposure estimates) and II (Mortality). Am J Ind Med 1983;4:399-433.

23. Nicholson WJ, Selikoff IJ, Seidman H, Hammond EC. Mortality experience of asbestos factory workers: effect of differing intensities of asbestos exposure. Environ Res (in press).

24. Ing R, Falk H, Lemen RA, et al. Community asbestos exposure in Globe, Arizona. J Pediatr 1981;99:409-11.

25. Nicholson WJ. Control of sprayed asbestos surfaces in school buildings. (NIEHS Publication #1-7-2113.) Washington, DC: U.S. Government Printing Office, 1976.

26. Faich G. Asbestos hazard evaluation in Rhode Island schools. Am J Public Health 1980;70:162-4.

27. Novick LF, Rice C, Freedman MA, Jillson D. Asbestos in Vermont schools: findings of a statewide on-site investigation. Am J Public Health 1981; 71:744-6.

28. Baldwin CA, Beaulieu HJ, Buchan RM, Johnson HH. Asbestos in Colorado schools. Public Health Rep 1982;97:325-31.

29. International Agency for Research on Cancer (IARC). IARC monographs on the evaluation of carcinogenic risk of chemicals to man: asbestos. (IARC Monographs, vol 14.) Lyon, France: World Health Organization, 1977.

30. Schneiderman MS, Nisbet ICT, Brett SM. Assessment of risks posed by exposure to low levels of asbestos in the general environment. bga-Berichte 1981;4:1-28.

31. Enterline PE. Cancer produced by nonoccupational asbestos exposure in the United States. J Air Pollut Control Assoc 1983;33:318-22.

32. Peto J, Seidman H, Selikoff IJ. Mesothelioma mortality in asbestos workers: implications for models of carcinogenesis and risk assessment. Br J Cancer 1982;45:124-35.

33. Walker AM, Loughlin JE, Friedlander ER, Rothman KJ, Dreyer NA. Projections of asbestos-related disease 1980-2009. J Occup Med 1983;25:409-25.

34. Nelson N, Nicholson WJ, Hughes JM, et al. Final report of the Chronic Hazard Advisory Panel (CHAP) on Asbestos. Washington, DC: Consumer Product Safety Commission, 1983 Jul:1-148.

35. Craighead JE, Mossman BT. The pathogenesis of asbestos-associated diseases. N Engl J Med 1982;306:1446-55.

36. Lemen RA, Dement JM, Wagoner JK. Epidemiology of asbestos-related diseases. Environ Health Perspect 1980;34:1-11.

37. McDonald AD, McDonald JC. Malignant mesothelioma in North America. Cancer 1980;46:1650-6.

38. Craighead JE, Abraham JL, Churg A, et al. Asbestos-associated diseases. Arch Pathol Lab Med 1982;106:543-97.

39. Becklake MR. Asbestos-related diseases of the lungs and pleura. Am Rev Respir Dis 1982;126:187-94.

40. Sebastien P, Billon A, Dufour G, et al. Levels of asbestos air pollution in some environmental situations. Ann NY Acad Sci 1979;330:401-15.

41. Nicholson WJ. Environmental Protection Agency Update on Asbestos Health Effects. New York, NY: Environmental Sciences Laboratory, Mount Sinai School of Medicine, 1983.

42. Churg A, Warnock ML. Analysis of the cores of asbestos bodies from members of the general population: patients with probable low-degree exposure to asbestos. Am Rev Respir Dis 1979;120:781-6.

43. Churg A. Nonasbestos pulmonary mineral fibers in the general population. Environ Res 1983;31:189-200.

44. Albolda SM, Epstein DM, Gefter WB, Miller WT. Pleural thickening: its significance and relationship to asbestos dust exposure. Am Rev Respir Dis 1982;126:621-24.

45. Breslow L, Bates R, Bendixen H, et al. Nonoccupational health risks of asbestiform fibers. Washington, DC: National Academy Press, 1984.

46. Garfinkel L. Time trends in lung cancer mortality among nonsmokers and a note on passive smoking. JNCI 1981;66:1061-6.

47. Hammond EC. Smoking in relation to death rates of one million men and women. In: Epidemiological study of cancer and other diseases. NCI Monographs 1966;19:127-204.

48. Sebastien P, Bignon J, Martin M. Indoor airborne asbestos pollution: from the ceiling and the floor. Science 1982;216:1410-1413.

49. International Commission on Radiological Protection (ICRP). Report of the Task Group on Reference Man. New York, NY: Pergamon Press, 1975.

50. General Accounting Office (GAO). Asbestos in schools: a dilemma. Washington, DC: GAO Community and Economic Development Division, 1982.

51. Jackson RS. Procedures for asbestos detection in South Carolina. Columbia, SC: South Carolina Department of Health, 1982.

52. Charelle ML. Arizona's school asbestos program. J Environ Health 1982;45:135-37.

53. US Department of Labor. Asbestos standard. 29 CFR 1910.1001, 1976:510-4.

54. Carter JW, Taylor DG, Baron PA. Fibers (Method #7400). In: Eller PM, ed. NIOSH manual of analytical methods, 3rd ed, vol 1. Cincinnati: National Institute for Occupational Safety and Health, 1984.

55. Taylor DG, Baron PA, Shulman SA, Carter JW: Identification and counting of asbestos fibers. Am Ind Hyg Assoc J 1984;45:84-8.

56. Stanton MF, Layard M, Tegeris A, Miller E, May M, Morgan E, Smith A. Relation of particle dimension to carcinogenicity in amphibole asbestos and other fibrous minerals. JNCI 1981;67:965-75.

57. Harington JS. Fiber carcinogenesis: epidemiologic observations and the Stanton hypothesis. JNCI 1981;67:977-88.

58. Kilburn KH. Flame-alternated fiberglass: another asbestos? Am J Ind Med 1982;3:121-5.

59. Bernstein RS, Falk H, Turner DR, Melius JM. Nonoccupational exposures to indoor air pollutants: a survey of state programs and practices. Am J Public Health 1984;74:1020-3.

60. Leidel NA, Bayer SG, Zumwalde RD, Busch KA. USPHS/NIOSH membrane filter method for evaluating airborne asbestos fibers. Cincinnati: National Institute for Occupational Safety and Health (NIOSH) Pub. No. 79-127, 1979.

61. Kim WS, Carter JW, Kupel RE. Quick screening test for asbestos. Am Ind Hyg Assoc J 1981;42:198-201.

62. Baldwin CA, Beaulieu HJ, Buchan RM. Application of the K^2 asbestos screening test in Colorado schools. Am Ind Hyg Assoc J 1982;43:602-4.

63. Glickman LT, Domanski LM, Maguire TG, Dubielzig RR, Churg A. Mesothelioma in pet dogs associated with exposure of their owners to asbestos. Environ Res 1983;32:305-13.

B35/12