EPA/SAB/81/001

Review of "Technical Support Document for Regulatory Action Against Friable Asbestos-Containing Materials in School Buildings" (Draft dated September 1980)

A Report of the Toxic Substances Subcommittee

February 1981

Science Advisory Board
U.S. Environmental Protection Agency
Washington, D.C.

EPA NOTICE

This report has been written as part of the activities of the Science Advisory Board, a public advisory group providing extramural scientific information to the Administrator and other officials of the Environmental Protection Agency. The Board is structured to provide a balanced expert assessment of the scientific matters related to problems facing the Agency. This report has not been reviewed for approval by the Agency, hence its contents do not necessarily represent the views and policies of the Environmental Protection Agency, nor does mention of trade names or commercial products constitute endorsement or recommendation for use.

The Science Advisory Board's Toxic Substances Subcommittee met on December 2-3, 1980 to consider the "Technical Support Document for Regulatory Action Against Friable Asbestos-Containing Materials in School Buildings" (Draft dated September 1980). The Subcommittee reviewed and commented on the scientific merits of the document and responded to specific questions, relating to the document, posed by the Office of Toxic Substances. It should be noted here that the Subcommittee is a general scientific advisory committee, not a panel of experts on asbestos.

Copies of the Agenda, the Subcommittee roster, and a listing of SAB and EPA participants in the meeting are appended (Appendices A, B, and C). A rough transcript of the meeting was prepared and has been submitted to the Agency.

The meeting was open to the public. The Asbestos Information Association provided the Subcommittee with their extensive comments on the document, the same comments previously filed with EPA. Representatives of the Association made an oral presentation and submitted a brief written statement, a copy of which is appended (Appendix D).

Briefly, the Subcommittee found the document to be scientifically credible but in need of some revisions and generally endorsed it with the understanding that necessary revisions will be made.

Major points which the Subcommittee felt need to be addressed are as follows:

1. The problems associated with the extrapolation of available asbestos exposure data to long-term, low-level effects were noted. The Subcommittee recommends that these problems be discussed more fully in the document. Further, case studies on the major risk of mesothelioma following low level asbestos exposure should be emphasized.

2. The concept of using only a linear model to predict risk was questioned. The Subcommittee recommends that, in addition to the linear model, two other models be included: the one hit model and the Weibull model.

3. The role of smoking in connection with exposure to asbestos should be clarified. It should be clearly noted that, while smoking affects mortality rates in patients with asbestosis, it is not an important factor in increasing an individual's susceptibility to asbestosis, and all evidence indicates that smoking does not have an effect on the incidence of mesothelioma.

4. The differential effect of asbestos exposure on children, as distinct from adults, should be reevaluated. The greatest risk, in humans, to low-level environmental exposure is that of mesothelioma. The latency period for mesothelioma appears to be approximately the same whether an individual is six years old or thirty years old.

5. There are a number of areas in which definitions should be clarified, e.g., a discussion of concentrations and exactly what is meant by exposure and "lifetime risk." There are also a number of inferential statements which should be reviewed and revised.

6. The four external reviews from Life Systems, Inc. were reviewed by the Subcommittee and seen as objective and, overall, of very good quality. The Subcommittee feels that it would be of value to have these four reviews examined once more by the Agency.

7. There should be further detail on the measurements involved in asbestos studies.

> The measurement of similar samples of asbestos fiber in different laboratories or even in the same laboratory shows considerable variation. See, for example Tables C-1 and C-2 (pages C-9 and C-10) in "Asbestos: An Information Resource," DHEW Publication Number (NIH) 79-1681, May 1978. Variability of asbestos concentration as a result of measurement seriously flaws estimates of risk from concentration-time-risk curves. (See discussion on p.C-8, "Asbestos in Air," in above cited DHEW publication.)
>
> If EPA assumes that the nature of the asbestos fibers in schools is similar to that in an exposed working group, this should be clearly articulated. The conversion number (30), presumably derived from published data, needs to be justified.
>
> The use of concentration multiplied by durations (cumulative exposure) as a measure of exposure is of dubious merit. If possible, some data in which individuals are exposed for fixed times but at ranging concentrations should be included.
>
> Risk estimates may also be seriously affected by conversion of old exposure data to "new" asbestos concentrations with the use of fudge numbers. This should be avoided even if some of the data base is lost. It would be better to present two sets of untampered data to maintain impartiality.

8. Finally, there should be an executive summary, attached to the final document, which clearly reflects the content of the document.

In addition to these recommendations, the Subcommittee also responded to questions by the Office of Toxic Substances (OTS) sent to the Subcommittee prior to the meeting. The OTS questions, the Subcommittee responses, and detailed written comments on the Technical Support Document provided by Dr. Ruth Lilis are appended (Appendices E, F, and G).

U.S. ENVIRONMENTAL PROTECTION AGENCY
SCIENCE ADVISORY BOARD
TOXIC SUBSTANCES SUBCOMMITTEE
Conference Room 503A
Hubert H. Humphrey Building
Washington, D.C.

December 2-3, 1980

FINAL AGENDA

Tuesday, December 2, 1980

9:00 a.m.	Introductions and Opening Remarks	Dr. Vernberg
9:15 a.m.	Remarks by Representative of Office of Toxic Substances	Dr. Muir
9:45 a.m.	Discussion	Subcommittee
10:15 a.m.	Statement by Representatives of Office of Enforcement	Ms. Russell
10:30 a.m.	Statement by Representatives of Asbestos Information Association	Mr. Pigg Mr. Hardy
10:45 a.m.	Discussion	Subcommittee
11:15 a.m.	BREAK	
11:30 a.m.	OTS Questions on Asbestos-In-Schools Risk Assessment	Dr. Vernberg Subcommittee Dr. Teitelbaum OTS Staff
12:45 p.m.	LUNCH	
2:00 p.m.	OTS Questions on Asbestos-in-Schools Risk Assessment (Continued)	Dr. Vernberg Subcommittee Dr. Teitelbaum OTS Staff
4:30 p.m.	RECESS	

SCIENCE ADVISORY BOARD
TOXIC SUBSTANCES SUBCOMMITTEE

December 2-3, 1980

AGENDA
(Continued)

Wednesday, December 3, 1980

9:00 a.m.	Opening Remarks	Dr. Vernberg
9:15 a.m.	Chapter by Chapter Review of Technical Support Document for Regulatory Action Against Friable Asbestos-Containing Materials in School Buildings (Draft dated September 1980)	Dr. Vernberg Subcommittee Dr. Teitelbaum OTS Staff
10:30 a.m.	BREAK	
10:45 a.m.	Chapter by Chapter Review of Technical Support Document (Continued)	Dr. Vernberg Subcommittee Dr. Teitelbaum OTS Staff
12:45 p.m.	LUNCH	
1:30 p.m.	General Discussion and Plans for Development of Subcommittee Report	Dr. Vernberg Subcommittee
2:30 p.m.	Concluding Remarks	Dr. Vernberg

ADJOURNMENT

Agenda Notes

Dr. Winona Vernberg, Dean, School of Public Health, University of South Carolina
Dr. Warren R. Muir, Deputy Assistant Administrator for Toxic Substances, Office of Pesticides and Toxic Substances, EPA
Ms. Pamela Russell, Policy and Strategy Branch, Office of Enforcement, EPA
Mr. B.J. Pigg, Executive Director, Asbestos Information Association, Arlington, Virginia
Timothy S. Hardy, Esq., Kirkland and Ellis, Washington, D.C.
Dr. Harry Teitelbaum, Project Manager for Risk Assessment, Assessment Division, Office of Pesticides and Toxic Substances, EPA

U.S. ENVIRONMENTAL PROTECTION AGENCY
SCIENCE ADVISORY BOARD
TOXIC SUBSTANCES SUBCOMMITTEE

Chairperson

Dr. Winona Vernberg, Dean, School of Public Health, University of South Carolina, Columbia, South Carolina 29205

Members

Dr. Martin Alexander, Professor of Soil Microbiology, Department of Agronomy, Cornell University, Room 708, Bradfield Hall Ithaca, New York 14853

Dr. Joan M. Cranmer, Associate Professor, Department of Pharmacology and Interdisciplinary Toxicology, University of Arkansas Medical Center, Little Rock, Arkansas 72201

Dr. J. Clarence Davies, III, Executive Vice President, Conservation Foundation, 1717 Massachusetts Avenue, N.W., Suite 300 Washington, D.C. 20036

Dr. James Douglas, Associate Fellow, John B. Pierce Foundation, 290 Congress Avenue, New Haven, Connecticut 06519

Dr. Sheldon Murphy, Professor, Department of Toxicology, University of Texas Medical School at Houston, Houston, Texas 77025

Dr. Eva Pell, Professor, Department of Plant Pathology, Pennsylvania State University, 211 Buckhout Laboratory, University Park, Pennsylvania 16802

Consultants

Dr. Robert J. Kohut, The Boyce Thompson Institute at Cornell University Tower Road, Ithaca, New York 14853

Dr. Ruth Little, Associate Professor, Mount Sinai School of Medicine Department of Community Medicine, Fifth Avenue and 100th Street New York, New York 10029

Liaison Members

Dr. Margaret Mattson, Behavioral Medicine Branch, Division of Heart and Vascular Diseases, National Heart, Lung and Blood Institute Bethesda, Maryland 20014

Dr. Carol M. Schitter, Head, Gastrointestinal and Toxicology Work Group Laboratory of Organ Function and Toxicology, National Institute of Environmental Health Sciences, Research Triangle Park, North Carolina, 27709

Acting SAB Staff Officer

Mr. Ernst Linde, Scientist Administrator, Science Advisory Board A-101, U.S. Environmental Protection Agency, Washington, D.C. 20460 Telephone: (202) 472-9444

TOXIC SUBSTANCES SUBCOMMITTEE

December 2-3, 1980

Participants
(Actual)

Members, Liaison Members, Consultants

Dr. Winona Vernberg
Dr. Joan M. Cranmer*
Dr. J. Clarence Davies
Dr. James Douglas
Dr. Ruth Lilis
Dr. Margaret Mattson
Dr. Carol M. Schiller

Special Guest

Dr. Clifton Brooks **

SAB Staff

Mr. Ernst Linde
Ms. Bernie Davis
Mrs. Patti Howard

Office of Pesticides and Toxic Substances

Dr. Warren R. Muir, Deputy Assistant Administrator for Toxic Substances
Mr. Joseph J. Breen, Chemist, Exposures Evaluation Division
Ms. Frances Loebenstein, Executive Assistant to Associate Deputy Assistant Administrator for Toxic Substances
Mr. Larry C. Longanecker, Chemical Engineer, Chemical Controls Division
Mr. Charles Poole, Epidemiologist, Health and Environmental Review Divisio
Dr. James N. Rowe, Pharmacologist, Health and Environmental Review Divisio
Dr. Harry Teitelbaum, Project Manager for Risk Assessment, Assessment Division

Office of Enforcement

Ms. Pamela Russell, Policy and Strategy Branch

Office of General Counsel

Mr. Allan Carpien, Toxic Substances Division

* December 3 only
** Physician, Clinical Ecology Medical Group, Santa Ana, California; Member, Environmental Measurements Committee, Science Advisory Board

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

SUMMARY OF AIA/NA COMMENTS ON EPA'S TECHNICAL SUPPORT DOCUMENT RISK ASSESSMENT FOR ASBESTOS IN SCHOOLS

EPA's Technical Support Document issued in connection with its asbestos in school identification and notification proposal contains a risk assessment that has been found seriously flawed by all independent experts who have reviewed it. Those flaws, leading to an assessment that substantially over-estimates any risk school residents may face from presence in buildings with friable asbestos, have been emphasized in written critiques by four experts chosen by EPA to review the assessment early this summer:

Philip Cole of the University of Alabama

Kenneth Rothman of Harvard University

Julian Peto of Oxford University

Frank Carlborg, a consulting biostatistician

And by a member of the Department of Education Asbestos Task Force:

Ann Wylie of the University of Maryland.

First, the flaws in the risk assessment lead to a significant over-estimate of exposure through:

- An estimate of the number of schools with friable asbestos that is based on neither a representative, nor a random, sample of U.S. schools.

-- The failure to recognize that much friable asbestos in schools will be in areas of the schools where teachers and students are unlikely to be present for any appreciable portion of the school day.

-- A highly biased use of asbestos measurements collected by Sebastien in Paris. Although the median measurements found by Sebastien were well below 10 ng/m^2, EPA says the most likely prevalent asbestos level is 270 ng/m^2.

The net result of this biased and incomplete exposure assessment is to overestimate the likely exposure of American school residents by more than an order of magnitude.

Second, the flaws in the risk assessment overestimate the likely health risk for the assumed exposure through:

-- Reliance on but a single epidemiologic study for determining health effects even though numerous epidemiology studies have been conducted and the single study relied on by EPA is not among the most appropriate studies for risk assessment purposes, particularly because no direct exposure information (either on an individual-by-individual or even on an average basis) is available for this cohort.

-- The failure to take into account the extent to which smoking accounts for much of the disease in the epidemiology study used to predict risks and the great likelihood that smoking patterns among school residents will vary greatly from those of that cohort.

— The failure to predict risks on the basis of models other than a linear dose response model despite the absence of data indicating this to be the only appropriate model.

The result of these flaws in EPA's use of health evidence to predict risks for school residents is to over-estimate potential risk and indeed to obscure completely the likelihood that in fact such residents face no risk at all, or at most a de minimis and highly speculative risk.

Questions for the Science Advisory Board (SAB) by the Office of Toxic Substances (OTS) Relating to the "Technical Support Document for Regulatory Action Against Friable Asbestos-Containir Materials in School Buildings" (Draft dated September 1980).

1. When smoking-specific data become available from the underlying study of asbestos insulation workers, OTS plans to estimate lung cancer risks separately for school occupants who do and do not smoke. The interaction (i.e., greater than additive effects) between smoking and asbestos leads to greatly increased risks for exposed smokers. The percentage of students who smoke can be estimated from recent surveys of smoking habits in the U.S.

OTS tentatively plans to conduct two lung cancer analyses when the data arrive: one assuming the interaction takes place in asbestos-exposed students who smoke and one assuming no interaction. We would advise decision-makers that the prudent course would be to adopt the first assumption.

Can the SAB determine a more appropriate way to make use of the smoking-specific data for lung cancer when they become available?

2. The hazard assessment contends that the assumption should be re-examined that smokers exposed to asbestos are at no greater risk of pleural mesothelioma than similarly exposed nonsmokers. The primary source of data for questioning this assumption is the insulation workers study, though the evidence is not persuasive one way or the other at this time. Given this state of the evidence, what factors would the SAB recommend that OTS consider in deciding whether to calculate smoking-specific risk estimates for pleural mesothelioma when the data arrive?

3. The current assessment makes no attempt to estimate risks of morbidity or mortality from asbestosis, even for custodians. Can the SAB suggest a way to make such estimates, perhaps by use of the morbidity data of Berry et. al. for British asbestos-textile workers?

4. The current assessment makes no attempt to estimate risks of nonfatal (i.e., successfully treated) cancer for exposed school occupants. For some types of cancer (e.g., cancer of the larynx), sole dependence upon mortality data undoubtedly creates underestimates of overall risk. To date OTS has not

found case-fatality ratios (i.e., the proportion of cases that die from the cancers) that would easily enable such estimates to be made. The only relevant data that appear to be available are 3-year and 5-year survival rates from the Third National Cancer Survey. Can the SAB identify case-fatality ratios or develop a method to the use survival rates to estimate increased risks of cancer morbidity?

5. The preamble to the proposed rule shows that a custodian could easily double his or her "prevailing" asbestos exposure by sweeping for only 5 minutes a day. Other reasonable scenarios of this type are not presented, nor are risk estimates for such "typical individuals," yet these are the exposures expected to be affected most by the notification provisions of the rule. Can the SAB recommend methods for developing additional scenarios of peak exposures and estimating the resultant risks to be expected?

6. The greater remaining life expectancy of children exposed to asbestos places them at a greater risk than similarly exposed adults. Can the SAB direct us to studies dealing with the possible physiological reasons for variation in risk and/or latency by age?

7. The document uses the observed number of deaths among the insulation workers as recorded on the death certificate for all types of cancer other than pleural and peritoneal mesothelioma. For the mesotheliomas, the observed numbers based on all available evidence (e.g., autopsy reports, review of histopathologic material, etc.) are used, at the recommendation of the study's authors and one of the OTS extramural reviewers. The reason is that mesotheliomas are often misdiagnosed and, because the expectation of these rare cancers in the general population is virtually zero, comparison with expected deaths would not be adversely affected. OTS agrees that this is the appropriate course to take.

Nevertheless, it is true that individual deaths are "doubly counted" by this procedure, persons whose death certificate diagnoses of lung cancer were changed by the researchers to pleural mesothelioma in particular. OTS views this "double counting" as an apparent inconsistency and not a real one. We would appreciate the advice of the SAB on how to make this judgment clear to readers.

8. Of the plausible dose response curves that could not be dismissed by providing a poor fit to available dose-response data, linear regression "usually" leads to the highest predictions of increased risk. This was the rationale for using linear regression for the quantitative risk estimation. Scientists at the Consumer Product Safety Commission informally pointed out that when, as in the case of the insulation workers study, responses exceeds 10%, the one-hit model yields higher risk estimates than linear regression.

OTS has not yet applied the one-hit model in this assessment, with the understanding that risk estimates even higher than those we obtained using linear regression would not alter regulatory decision-making. Nevertheless, strict adherence to the decision criteria laid out in the document would require using the one-hit model. Would the SAB recommend applying the one-hit model to obtain the highest risk estimates that cannot be prudently ruled out?

9. At the suggestion of one of the extramural reviewers, OTS performed the attached calculations using "attributable risk" (the difference between observed and expected death rates from asbestos-related cancers) instead of the "lifetime risk" measure. As shown in the Attachment, this technique could not be applied completely because of a limitation in the readily available U.S. lifetable; nevertheless, it showed that the result would lie within 40% of the prediction obtained by the "lifetime risk" method. Does the SAB believe that the attached analysis would make the conclusions in the Technical Support Document clearer and more supportable?

Appendix to Question #9

Dr. Rothman's suggestion to use attributable risk, the difference between observed and expected mortality rates, is a good one. The observed mortality rate from asbestos-related types of cancer among the asbestos insulation workers (see Table 17 in the new draft) was 692 deaths/77,391 person-years = 8.94×10^{-3} deaths/person-year. (The number of person-years was obtained from the published report of this study.) The expected rate was 145.8/77,391 = 1.88×10^{-3} deaths/person-year. The attributable risk is the difference between the two rates: $(8.94 \times 10^{-3}) - (1.88 \times 10^{-3}) = 7.06 \times 10^{-3}$ deaths/person year. The most reasonable estimate of relevant cumulative exposure for these workers is 6.0×10^{4} ng-yr/m^3 (see p. 64 of the new draft).

If the number of exposed school children continues to average 3×10^{6} for the next 30 years (the expected remaining useful life of the buildings), and each student spends an average of 6 years attending a contaminated school, a total of 1.5×10^{7} children will become exposed. The most reasonable estimate of exposure in the schools is 270 ng/m^3 (pp.82-83, new draft). Because a school year is approximately one-half as long as a work year (p.84, new draft), the cumulative asbestos exposure for each of the children would be 270 ng/m^3 x 3 years = 810 ng-yr/m^3.

Assuming proportionality (the "linear nonthreshold" assumption), the attributable risk for the children would be 7.06×10^{-3} deaths/person-year x (810 ng-yr/m^3/6.0×10^{4} ng-yr/m^3) = 9.53×10^{-7} deaths/person-year. Assuming that the minimum induction period for the exposed school children is 20 years and that the attributable death rate will apply to the survivors in each subsequent year, the following calculations may be made. Using the 1976 U.S. Lifetable for all races and sexes, 97.8% of a group of people alive at age 12 would be expected to survive to age 32. Thus, $(1.5 \times 10^{7}) \times (.978) = 1.467 \times 10^{7}$ of the exposed school children are expected to survive long enough for the minimum induction period to transpire. The 1976 lifetable may then be used to construct the following table:

Age	No. still alive	Expected deaths from life table %	Expected deaths from life table No.	Expected deaths from asbestos Rate*	Expected deaths from asbestos No.
32	14,670,000	0.799	117,213	4.765×10^{-6}	70
37	14,552,717	1.173	170,703	4.765×10^{-6}	69
42	14,381,945	1.874	269,510	4.765×10^{-6}	68
47	14,112,359	2.932	413,774	4.765×10^{-6}	67
52	13,698,118	4.450	609,566	4.765×10^{-6}	65
57	13,088,487	6.661	898,881	4.765×10^{-6}	62
62	12,190,424	9.918	1,209,046	4.765×10^{-6}	58
67	10,981,320	14.045	1,542,326	4.765×10^{-6}	52
72	9,438,942	27.081	2,556,160	4.765×10^{-6}	45
77	6,882,737	30.869	2,126,009	4.765×10^{-6}	33
82	4,756,695			TOTAL	589

9.53×10^{-7} deaths/person-year $\times$ 5 years = 4.765×10^{-6} deaths/person during a 5-year period.

By the time the group of children reaches age 82, 589 of them would be estimated by this method to have died from cancers induced by asbestos exposure in schools. The published life table does not permit extending the calculation to another 5-year interval. Consequently, the subsequent risk among the 32% of the original group surviving to age 82 and beyond cannot be predicted. Nevertheless, the resulting estimate would differ by less than 40% from the prediction of 960 obtained by the "lifetime risk" method (see Table 21 in the new draft).

The assumption that the predicted attributable death rate would apply unchanged in each year following the passage of the minimum induction period cannot be judged more or less tenuous than the assumption that the "lifetime risk" measure is transferable across age strata. Nonetheless, as Dr. Rothman notes, the attributable death rate does enjoy more ready conceptualization and more frequent use in epidemiology. In any event, disagreement of <50% between the two approaches must be considered minor in light of all the other uncertainties in quantitative risk estimation--especially the choice of the dose-response function, which can result in predictions that differ by several orders of magnitude. Pages 84-88 of the new draft point out the need for prudence when faced with the considerable uncertainty of choosing among dose-response curves that have equal degrees of admittedly theoretical, plausibility.

Subcommittee Responses to Questions by the Office of Toxic Substances (OTS) Relating to the "Technical Support Document for Regulatory Action Against Friable Asbestos-Containing Materials in School Buildings" (Draft dated September 1980).

1. When smoking-specific data become available from the underlying study of asbestos insulation workers, OTS plans to estimate lung cancer risks separately for school occupants who do and do not smoke. The interaction (i.e., greater than additive effects) between smoking and asbestos leads to greatly increased risks for exposed smokers. The percentage of students who smoke can be estimated from recent surveys of smoking habits in the U.S.

   The Subcommittee feels that data are available to indicate smoking patterns among high school and elementary school students and agreed that there should be some evaluation.

2. The hazard assessment contends that the assumption that smokers exposed to asbestos are at no greater risk of pleural mesothelioma than similarly exposed nonsmokers should be reexamined. The primary source of data for questioning this assumption is the insulation workers study, though the evidence is not persuasive one way or the other at this time. Given this state of the evidence, what factors would the SAB recommend that OTS consider in deciding whether to calculate smoking-specific risk estimates for pleural mesothelioma when the data arrive?

   None

3. The current assessment makes no attempt to estimate risks of morbidity or mortality from asbestosis, even for custodians. Can the SAB suggest a way to make such estimates, perhaps by use of the morbidity data of Berry et al. for British asbestos-textile workers?

   Only morbidity data should be estimated for both custodians and teachers.

4. The current assessment makes no attempt to estimate risks of nonfatal (i.e., successfully treated) cancer for exposed school occupants. For some types of cancer (e.g., cancer of the larynx), sole dependence upon mortality data undoubtedly creates underestimates of overall risk. To date OTS has not found case-fatality ratios (i.e., the proportion of cases that die from the cancers) that would easily enable such estimates to be made. The only relevant data that appear to be available are 3-year and 5-year survival rates from the Third National Cancer Survey. Can the SAB identify case-fatality ratios or develop a method to use survival rates to estimate increased risks of cancer morbidity?

   The Subcommittee suggests that the Agency utilize data available from the National Cancer Institute. Cancers of the colon and rectum should be considered as well as cancer of the larynx.

5. The preamble to the proposed rule shows that a custodian could easily double his or her "prevailing" asbestos exposure by sweeping for only 5 minutes a day. Other reasonable scenarios of this type are not presented, nor are risk estimates for such "typical individuals," yet these are the exposures expected to be affected most by the notification provisions of the rule. Can the SAB recommend methods for developing additional scenarios of peak exposures and estimating the resultant risks to be expected?

> Peak exposures are of very great importance and must be taken into account. The Subcommittee suggests that the Agency inquire whether data from NIOSH are available and could be used to estimate risk of asbestosis, lung cancer, and mesothelioma.

6. The greater remaining life expectancy of children exposed to asbestos places them at a greater risk than similarly exposed adults. Can the SAB direct us to studies dealing with the possible physiological reasons for variation in risk and/or latency by age?

> The Subcommittee recommends that Dr. Peto's comments be considered as well as the Turkish studies on environmental exposure of children.

7. The document uses the observed number of deaths among the insulation workers as recorded on the death certificate for all types of cancer other than pleural and peritoneal mesothelioma. For the mesotheliomas, the observed numbers based on all available evidence (e.g., autopsy reports, review of histopathologic material, etc.) are used, at the recommendation of the study's authors and one of the OTS extramural reviewers. The reason is that mesotheliomas are often misdiagnosed and, because the expectation of these rare cancers in the general population is virtually zero, comparison with expected deaths would not be adversely affected. OTS agrees that this is the appropriate course to take.

Nevertheless, it is true that individual deaths are "doubly counted" by this procedure, persons whose death certificate diagnoses of lung cancer were changed by the researchers to pleural mesothelioma, in particular. OTS views this "double counting" as an apparent inconsistency and not a real one. We would appreciate the advice of the SAB on how to make this judgment clear to readers.

> The Subcommittee endorses the procedure that EPA currently follows.

8. Of the plausible dose response curves that could not be dismissed by providing a poor fit to available dose-response data, linear regression "usually" leads to the highest predictions of increased risk. This was the rationale for using linear regression for the quantitative risk estimation. Scientists at the Consumer Product Safety Commission informally pointed out that when, as in the case of the insulation workers study, responses exceed 10%, the one-hit model yields higher risk estimates than linear regression.

OTS has not yet applied the one-hit model in this assessment, with the understanding that risk estimates even higher than those we obtained using linear regression would not alter regulatory decisionmaking.

Nevertheless, strict adherence to the decision criteria laid out in the document would require using the one-hit model. Would the SAB recommend applying the one-hit model to obtain the highest risk estimates that cannot be prudently ruled out?

> The Subcommittee recommends use of three models, all of which will be based upon qualitative epidemiological data: the linear model, the one-hit model, and the Weibull model.

9. At the suggestion of one of the extramural reviewers, OTS performed calculations using "attributable risk" (the difference between observed and expected death rates from asbestos-related cancers) instead of the "lifetime risk" measure. This technique could not be applied completely because of a limitation in the readily available U.S. lifetable; nevertheless, it showed that the result would lie within 40% of the prediction obtained by the "lifetime risk" method. Does the SAB believe that the attached analysis would make the conclusions in the Technical Support Document clearer and more supportable?

> The Subcommittee believes that the attached analysis does make the conclusions in the technical support document clearer and more supportable.

COMMENTS by Dr. Ruth Lilis on
"Technical Support Document for Regulatory Action [TSCA: Section 6(a)] Against Friable Asbestos-Containing Materials in School Buildings"
(Draft dated September 1980)

1. USE AND PRESENCE OF FRIABLE ASBESTOS CONTAINING MATERIALS IN SCHOOLS

Page 7. It is not clear how the EPA estimated the number of public schools with friable asbestos-containing materials. It is indicated that out of the 768 school districts which responded to the survey (these represented 8% of the nation's total), there were 6422 (out of a total of 7378 schools) built or renovated after 1945. 5797 schools were inspected and 1916 (or 33% of these) were identified as having friable asbestos-containing materials.

Since 1916 schools were identified out of a sample representing 8% of the nation's schools, it is unclear how the final estimate of 8545 was reached. There must have been additional elements entering this projection, and this is an important issue which should be fully presented.

2. ASSESSMENT OF RISK FROM ASBESTOS IN SCHOOLS

a. Hazard Assessment

Page 18, last paragraph.

There is no convincing evidence indicating that smoking increases the risk of developing asbestosis.

b. Pleural and Peritoneal Mesothelioma

Page 22.

In estimating potential risk for asbestos-related disease in school children, teachers and other employees exposed in schools, the major emphasis should be on mesothelioma, since this adverse effect has clearly been associated with low levels of exposure, such as household exposure and neighborhood exposure. Another body of information could be used in evaluating the risk for mesothelioma. Several reports from Turkey have indicated the occurrence of numerous cases of mesothelioma with environmental exposure to asbestos, present in outcroppings of rocks in various areas of the country. The use of such materials for whitewash of dwellings or as construction material seems to be the major source of exposure to the general population. Attached are copies of relevant publications on endemic mesothelioma in Turkey.

c. Asbestosis

Page 32

The definition of asbestosis does not include the severity criterion. Asbestosis is the interstitial pulmonary fibrosis due to inhalation of asbestos fibers. The pathologic process can progress from early and slight changes, to more marked abnormalities and eventually to severe abnormalities. To restrict the definition of asbestosis to the "severest form" is erroneous.

Page 33, end of first paragraph, to be changed:

"..... individuals have to be examined for radiologic and clinical abnormalities." The radiologic method is the single most important one for the diagnosis of pneumoconioses in general, and for asbestosis in particular.

Page 34, second paragraph.

There is general agreement, at present, that asbestosis is not to be restricted for "advanced stages of the disease" or for "certified" asbestosis.

Page 39.

It is difficult to understand why the highly questionable definitions for "possible asbestosis" and "certified asbestosis" (McVittie, 1965) are used. The attempt to construct dose-response relationships for three different definitions of asbestosis is of little relevance.

Page 39, last paragraph.

With asbestos exposure in the lower range, such as in household exposure, the most prevalent abnormalities are pleural fibrosis and pleural calcifications. Such abnormalities occur often in the absence of definite parenchymal changes (interstitial fibrosis), and they can be quite extensive. With environmental asbestos exposure, such as that reported from Turkey, pleural abnormalities (pleural fibrosis, pleural calcifications and pleural effusions) are to be found with a higher prevalence than radiographically detectable interstitial pulmonary fibrosis.

Page 42, last paragraph.

The conclusion should indicate that, based on the available information on effects of non-occupational asbestos exposure, there is a high probability for pleural abnormalities to occur as a result of exposure in school buildings.

d. Factors that Modify the Risk of Asbestos-Induced Disease

Smoking

Page 49.

While there is no doubt that smoking affects mortality rates of patients with asbestosis, this does not warrant the statement "Smoking may also be an important factor in increasing an individual's susceptibility to asbestosis." There is no convincing evidence to prove this.

Page 49.

There is no effect of smoking on the incidence of mesothelioma. The data presented in Table 12 do not indicate this. Information of endemic mesothelioma from Turkey, where hundreds of cases have occurred, clearly indicate similar trends in females as in males; females are non-smokers in Turkey while most males are smokers.

Page 51.

The conclusion of IARC 1977 according to which mesothelioma "occur with equal frequency among smoking and non-smoking asbestos workers" is entirely valid. It does not seem appropriate for the document under review to suggest the need for a reevaluation of this conclusion.

e. Exposure Assessment

Page 57.

Peak concentrations are important in assessing risk. An effort to estimate how frequent the occurrence of peak exposures in the school environment is, and for how long the higher concentrations (above "prevalent concentration") persist should be made.

Asbestos Dispersion Mechanisms

Page 59.

The choice of the study by Sebastien et al. of several buildings in Paris as a data base for estimation of levels of exposure in U.S. schools could be subjected to criticism. It would be appropriate to use data on U.S. schools, either existing data (Sawyer, Nicholson) or, if these are not entirely satisfactory, other data which should be collected according to a protocol which would satisfy the criteria agreed upon by EPA. A more comprehensive discussion on the reasons for which U.S. data on asbestos in schools were not used would be appropriate.

Estimate of Prevalent Exposures

Page 65, first paragraph.

The criteria outlined as reasons for accepting data from Sebastien et al. are understood not to have been fulfilled by U.S. studies on schools. It would be appropriate to present more detailed comments on the reasons for which the U.S. studies did not fulfill these criteria.

f. Risk Assessment

Selection of the Underlying Study

Page 73.

Under the attributes that make the selected study (Selikoff et al., Hammond et al.) suitable to be taken as a basis for making quantitative estimates of risk the following is listed:
"Each of the diseases identified as hazards of asbestos exposure was investigated and was found to be in excess." This is, in itself, not necessarily an attribute making the study under consideration more appropriate as a basis for estimation of risk of exposure in schools.

Asbestos Exposure Among the Insulation Workers

Page 77.

The concept of "wasted" exposure is confusing and not at all helpful. I suggest the phrase "asbestos exposure not affecting outcome" be used.

Increased Risk Among the Asbestos Insulation Workers

Page 81.

The EPA definition of "lifetime risk" is unclear (as presented in the text, this is "excess risk," but not "lifetime risk").

Asbestos Exposure in Schools

Page 83.

The assumption is made that the average student is first exposed at age 12.

Page 84, first paragraph.

It is then estimated that for students the prevalent concentration has to be multiplied by "15 work years." It is rather difficult to understand how this 15 years figure was chosen.

The paragraph on page 84, providing background information on the reasoning followed, is from a biological point of view, highly questionable.

3. Final Comments

The extrapolation (or intrapolation) from a high exposure group, to assess risks of low level exposure, is fraught with inherent difficulties, especially when the extrapolation covers exposure levels approximately four orders of magnitude apart. The major risk, at lowest known asbestos exposure, is mesothelioma. The numerous cases of mesotheliomas in family members of asbestos workers, with neighborhood exposure and with environmental exposure, strongly indicate that a risk of developing mesothelioma as a result of exposure in schools is real.

I agree with most of the comments by Peto, and with his suggestions for the assessment of health hazards due to asbestos exposure in schools.