

asbestos: rationale behind a proposed air quality standard

Leonard Bruckman and Robert A. Rubino 1975
Connecticut Department of Environmental Protection

The following discussion presents a proposed asbestos air quality standard and the rationale utilized in its formulation. The criterion of mesothelioma has been selected as the basis for developing the subject standard. A concentration mesothelioma incidence envelope has been constructed for the general population, based on mesothelioma incidence due to industrial exposure. An ambient air quality asbestos standard of 30 nanograms per cubic meter, based on a 30-day average sample is recommended. This standard is projected to result in 150 nationwide fatalities. Available ambient air asbestos data indicates that non-urban and remote non-urban areas have asbestos levels typically less than 1 nanogram per cubic meter while urban areas are usually below 30 nanograms per cubic meter.

Dispersion calculations are used to determine the maximum allowable asbestos mass emission standard for manufacturing sources (24 grams per day) and a maximum allowable average asbestos concentration (25 micrograms per cubic meter). A family of curves for various workdays depicting the average allowable asbestos concentration as a function of total asbestos contaminated exhaust gas flow rate, consistent with the 24 grams per day emission standard are present-

ed. In addition, a preliminary asbestos stack sampling train has been developed requiring electron microscopy as the analytical procedure to be used to determine the sample asbestos content.

The implementation of the proposed emission standard will lower the expected annual asbestos emissions from manufacturing operations in Connecticut to below one ton compared to the 10 tons emitted if the Federal EPA asbestos regulation is accepted. The proposed air quality standard will further reduce Connecticut asbestos-related fatalities to only two persons annually.

"Asbestos," the generic name given to a variety of fibrous, silicate naturally occurring minerals, is present as an invisible and anonymous ingredient in a myriad of industrial products, such as paint, floor tiles, gaskets, cement, shingles, pipes, brake linings, fireproofing and insulating materials, and asbestos fabrics. The principal variety of asbestos used in industry is chrysotile (greater than 95%) with amosite and crocidolite being of lesser significance. Consumption of asbestos in the U. S. has risen from approximately 40,000 tons in 1905 to nearly 800,000 tons in the early 1970's, about a 20-fold increase.^{1,2}

HCL 019309

been well known for nearly half a century that persons subjected to asbestos dust during work developed a severely crippling and sometimes fatal lung disease, called asbestosis. Asbestosis can be classified as an occupational disease which occurs after relatively heavy exposure to asbestos fiber dust. The use of antibiotics in the 1930's dramatically reduced the death rate from infectious pulmonary diseases that often had to be feared as a complication of asbestosis; consequently, asbestos workers instead of dying from asbestosis, began living long enough to develop asbestos-induced cancer of the lung, gastrointestinal tract, stomach, and especially the lining of the stomach and lung (i.e., mesothelioma).^{3,4} Asbestos has become one of the largest industrial causes of cancer in the world.⁵ All the commercially used forms of asbestos can produce asbestosis, as well as the types of cancer previously mentioned.

Mesothelioma was regarded by pathologists, until recently as an extremely rare disease and at times, a medical curiosity. In 1965 Dr. Selikoff and his associates conclusively proved a causal link between asbestos fiber exposure and mesothelioma.^{6,7} A significant and somewhat frightening feature of mesothelioma is the extended lag period between first exposure to asbestos fibers and the first symptoms of this form of cancer. It has been reported in the literature that this time period may be anywhere from 20 to 40 years.⁸⁻¹⁰ This long time interval emphasizes the latent insidious problem asbestos fiber exposure continually creates for individuals, both occupationally and non-occupationally exposed.

Health Effects—Non-Occupational

Evidence that people other than those working directly with asbestos were also being subjected to asbestos fibers was of several types. In a few geographic regions where asbestos was mined and milled and asbestos-containing products manufactured, pathologic alterations (i.e., pleural calcification) were discovered in populations with no history of occupational exposure.¹¹⁻¹⁵ Recent studies have indicated that urban dwellers harbor high quantities of asbestos fibers in their lungs.^{3,8,16-18}

The most serious implication of the hazards of non-occupational asbestos fiber exposure is the risk of contracting mesothelioma. Several studies have done much to verify the connection between asbestos fiber exposure and the incidence of mesothelioma among the general population.^{3,6,19,20} Current cases of mesothelioma must be associated with asbestos fiber inhalation that took place 20, 30, or more years ago, when world consumption of asbestos was only about 500,000 tons/year, and that neoplasms associ-

ated with today's annual production of over 4,000,000 tons will not be evident until the 1990s. The health hazard that asbestos fiber exposure poses to the general public is compounded by the synergistic effect of tobacco smoke, and possibly other air pollutants, such as benzo(a)pyrene.⁹

Relationship between Occupational Asbestos Exposure and Incidence of Mesothelioma

The Federal EPA has recognized the need to control the emission of asbestos fibers into the ambient air and has recently promulgated National Emission Standards for Hazardous Air Pollutants (Asbestos, Beryllium, and Mercury).²¹ Alternatives to the EPA's required control strategies are conceivable and the following discussion presents one such option, the formulation of an asbestos ambient air quality standard and a compatible mass emission standard for manufacturing sources of asbestos emissions.

The probability of a person in the general community contracting mesothelioma has been chosen as the basis for developing the asbestos air quality standard. Most of the information presently available on exposure to asbestos has been collected in industrial environments. Studies presently available on mesothelioma incidences due to industrial exposure do not report the fiber concentration to which affected workers were exposed; however, a study by the National Institute for Occupational Safety and Health (NIOSH) reports short term fiber concentrations for a number of industrial settings.²²⁻²⁶ While these short term fiber concentrations are of recent origin and as such are not directly related to the epidemiological studies of mesothelioma incidence, they do reflect the difference between well and poorly controlled processes; and thus can be used as an estimate of low or severe exposure.²³ Table I summarizes mesothelioma incidence data from several different studies along with high and low concentrations from the NIOSH document for shipyards, textile processes, construction operations, and insulation plants. These data were used to construct a concentration incidence envelope, depicted in Figure 1.

Relationship between Fibers Greater than Five Microns to Nanograms of Asbestos

Asbestos fibers monitored in industrial settings (greater than 5 μm in length, length to width ratio of greater than 3) using phase contrast (PC) light microscopy (430X magnification) account for approximately 2% of all asbestos fibers present (by number).²⁷ Hence, in the occupational environment every asbestos fiber greater than 5 μm in length corresponds to an actual fiber count of 50 (i.e., $1/0.02 = 50$).

Table I. Incidence of mesothelioma and asbestos concentrations in occupational environments.²³

Industry	Cohort ^a Number of individuals	Meso- thelioma incidence %	Reference	Highest ^b average concentration fiber/ cm ³	Lowest ^b average concentration fiber/ cm ³
Insulations plants	689	2.18	(23)	74.4	0.1
Shipyards	3000	0.73	(23), (26)	8.7	0.3
Construction	632	0.63	(23), (29)	7.1	0.9
Textile plants	716	1.50	(23), (25)	29.9	0.1
	1300	1.00	(23), (25), (30)	29.9	0.1
	~1300	1.20	(23), (24), (31)	29.9	0.1

^a Most of the individuals in these studies had been followed for 20 years or longer.

^b Concentrations from NIOSH document.²³

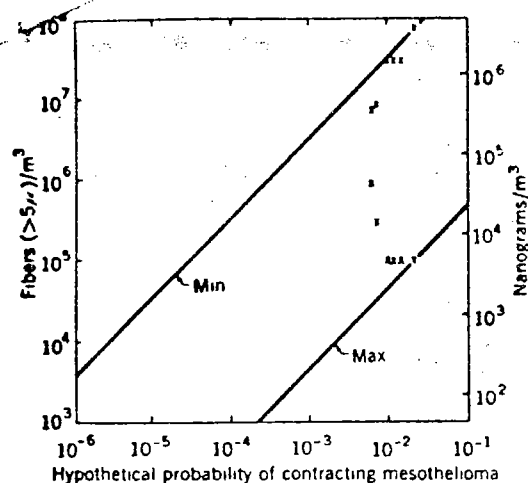


Figure 1. Expected incidence of contracting mesothelioma as a function of industrial air asbestos exposure (8-hr day, 5 day week).²³

Dr. Thompson (personal communication) reported that there are about 1000 total asbestos fibers per nanogram of asbestos.²⁸ Therefore, 20 asbestos fibers greater than 5 μ m long have been assumed equivalent to one nanogram of asbestos (i.e., $1000/50 = 20$). This appears to be a reasonable assumption since Lynch and his co-workers reported, based on magnesium determinations, that for certain manufacturing operations, one ng of asbestos was equivalent to 6.7 to 22.5 fibers greater than 5 μ m long as determined by PC.²⁷

Relationship between Non-Occupational Asbestos Exposure and Incidence of Mesothelioma

The mesothelioma incidence envelope depicted in Figure 1 appears as a function of both fibers greater than 5 μ m in length and ng of asbestos/ m^3 , respectively.

Occupational exposure concentrations, which are based on an 8 hr day, 5 day week must now be related to exposure levels for the general public (24 hr day, 7 day week) which contains many susceptible groups, such as the elderly and infants, who must non-voluntarily and continuously endure the ambient environment. This can be accomplished by dividing the occupational exposure levels by 4.2 (24 hr/8 hr $\times$ 7 days/5 days = 4.2).³² A concentration-mesothelioma incidence envelope for ambient air asbestos exposure for the general public has thus been constructed, as shown in Figure 2.

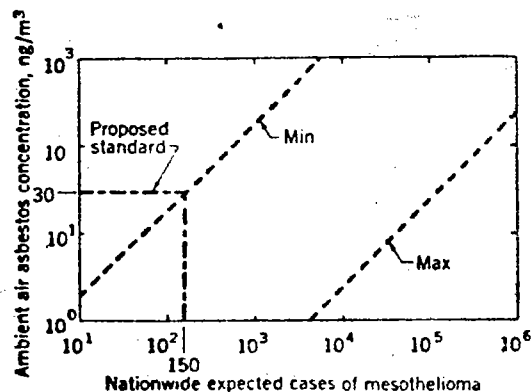


Figure 2. Nationwide expected cases of mesothelioma as a function of ambient air asbestos exposure (assumed population of United States was 230 million people).

Ambient Air Asbestos Standard

Now that a set of curves relating the nationwide expected cases of mesothelioma as a function of ambient air asbestos exposure has been constructed, a desired ambient air asbestos level can be decided on. This was accomplished by investigating the total number of nationwide fatalities attributed to automobile, aircraft, and railroad accidents for the year 1972. The *minimum* line (this reflects the smallest possible probability of contracting cancer) of the concentration-mesothelioma incidence curve was used. An ambient air asbestos standard of 30 ng/ m^3 was chosen. This level should result in about 1/10 the total nationwide number of fatalities (approximately 150) from airplane accidents and approximately the same number of deaths as from train mishaps; the maximum line, or greatest probability line, would have yielded over 100,000 expected fatalities. The use of the minimum line instead of an average line was chosen as a reasonable basis for setting the ambient air asbestos standard in order to account for some of the assumptions made in developing the concentration-incidence envelope.

Ambient Air Asbestos Levels

How reasonable is the desired ambient air standard of 30 ng of asbestos/ m^3 ? Nonurban and remote nonurban airborne asbestos concentrations are typically less than 1 ng/ m^3 .²⁸ Urban areas usually have asbestos concentrations below the desired 30 ng/ m^3 standard except in heavily industrialized areas (e.g., Manhattan, N.Y.C. and Philadelphia), around construction sites and toll booths.^{28,33,34} The desired standard appears to be reasonably set and attainable.

Comparison of OSHA and Ambient Air Asbestos Standards

The proposed American Conference of Government and Industrial Hygienists (ACGIH) Occupational Standard of 2 fibers greater than 5 μ m in length/ cm^3 (or $2/\text{cm}^3 \times 10^6 \text{ cm}^3/\text{m}^3 \times \text{ng}/20\text{f} = 10^5 \text{ ng}/\text{m}^3$) is designed specifically to protect the workman from contracting asbestosis, which results from heavy occupational exposure to asbestos. This standard does not take into account the effect of asbestos as a carcinogenic agent.^{8,22} The OSHA standard is equivalent to a general public exposure of 25,000 ng of asbestos/ m^3 (i.e., $10^5/4.2 = 25,000$). It is concluded that the proposed standard of 30 ng/ m^3 is not unreasonable even though it is approximately 1/1000 of the OSHA standard, since the latter standard is not nearly stringent enough to protect workmen from contracting cancer.

Asbestos Emission Standard

Figure 3 depicts the maximum allowable emission rate (consistent with the desired 30 ng/ m^3 ambient air standard based on both a 24 hr average and 30 day average sample) at corresponding distances from a source. These emission rate curves were constructed using dispersion estimates and local Connecticut meteorological assumptions.³⁴⁻³⁶ Thus, the maximum allowable emission rate, consistent with the aforementioned assumptions, would be either 20 or 24 g/day, respectively, at a distance of approximately either 300 or 350 ft from the source. A 24 hour sampling period is not really long enough to reflect properly the average ambient asbestos levels in any region and is very sensitive to short term asbestos emissions. An ambient air standard based on a 30 day average sampling period is more manageable from a monitoring standpoint and more descriptive of the average ambient air asbestos concentrations for a particular area. It is therefore concluded that the ambient air

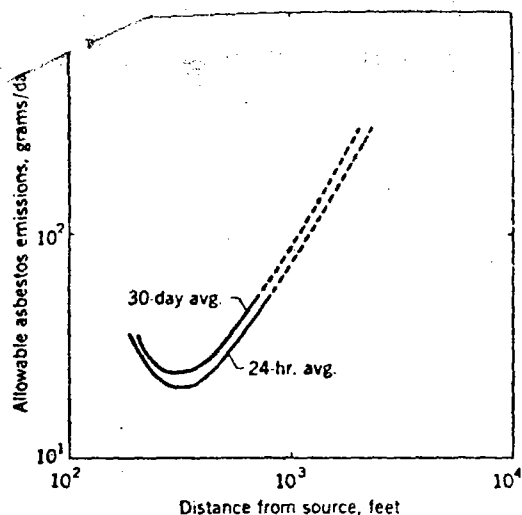


Figure 3. Calculated maximum allowable asbestos emission rate as a function of distance from source (assumed: 4 m/sec wind speed, 10m effective stack height, 40% frequency factor for wind direction, and C stability class).

asbestos standard should be based on a 30 day average sample and the corresponding maximum allowable asbestos emission rate is 24 g/day.

The aforementioned emission and ambient air quality standards result in a maximum daily inhaled asbestos dosage of 600 ng, ($20 \text{ m}^3/\text{day}$ (total average adult daily respiratory air intake) $\times 30 \text{ ng}/\text{m}^3 = 600 \text{ ng}/\text{day}$). However, under adverse local meteorological conditions a person walking near or close to an asbestos emission source might be subjected to the same asbestos concentration as that of the plume itself for a short period of time. In order to protect such an individual the maximum allowable average asbestos plume concentration could be limited to $25 \mu\text{g}/\text{m}^3$. A person walking "through the plume" will then experience an asbestos concentration for a 15 minute time period equivalent to 10 times his maximum allowable daily dosage.

Figure 4 depicts the average allowable asbestos concentration as a function of asbestos-contaminated exhaust gas flow rate for various periods of daily plant operation consistent with a maximum allowable emission rate of 24 g/day and the desired air quality standard. This Figure could be utilized as an emission standard of sorts; the larger the asbestos-contaminated gas flow rate the lower will be the allowable average asbestos plume concentration.

Manufacturing Sources of Asbestos Emissions

A survey of asbestos users in Connecticut, summarized in Table II, indicates that annual state uncontrolled asbestos emissions could amount to almost 200 tons. Comments solicited from manufacturers of particulate control equipment, in addition to other reports, indicate that there should be no problem in obtaining at least a 95% asbestos emission control efficiency using either a high energy scrubber or a fabric filter and in many instances control efficiencies as high as 99.9% are attainable. It should be understood that there is scant actual supportive test data available; however, the criterion used to support the quoted efficiencies (experience with similar fibrous material dusts) should be valid.^{37,38} Notwithstanding the employment of best available asbestos control technologies as required by the Federal EPA asbestos regulation almost 10 tons of asbestos might be emitted into the Connecticut atmosphere each year as a result of manufacturing operations. In order to evaluate properly the potential hazard of this airborne

asbestos it is important to understand that 1 g of asbestos could contain up to 1,000,000,000 asbestos fibers and one inhaled asbestos fiber could potentiate a cancer. The implementation of the proposed 24 g/day emission standard in Connecticut would lower the expected annual asbestos emissions from manufacturing operations to below 1 ton and, thereby, provide the impetus for the development of improved asbestos air pollution control equipment. In addition, greater than 95% of the sources should have no problem meeting the proposed emission standard employing state-of-the-art control technology.

Brake Lining Decomposition

Asbestos as a result of brake lining decomposition might contribute another 1.5 tons of airborne asbestos fibers into the Connecticut atmosphere each year.^{3,39-42} Several investigators have suggested that the asbestos emitted as a result of brake usage is not a significant source of atmospheric asbestos; however, in New York City air asbestos concentrations contiguous to a toll booth were 3 to 5 times background levels.^{43,44} Additional data are needed before the impact of brake lining decomposition on airborne asbestos levels can be ascertained.

Demolition

The demolition of asbestos sprayed highrise buildings could result in the emanation of a large amount of asbestos fiber dust unless adequate precautions are taken. If the asbestos emissions caused by demolition operations could be accurately accounted for the actual annual tonnage of asbestos emitted into the Connecticut atmosphere might be significantly increased.

Asbestos Stack Sampling Train

It has been suggested that an asbestos air quality regulation which employs an emission standard will suffer from the unavailability of an adequate stack sampling procedure; however, other investigators report that there should be no problems, either theoretical or physical in stack sampling for asbestos than there would be in sampling any complex source.⁴⁵ The stack sampling train depicted in Figure 5 (a modified version of the approved federal particulate train) might be used to measure asbestos stack emissions; however, this proposed train has never been field tested.

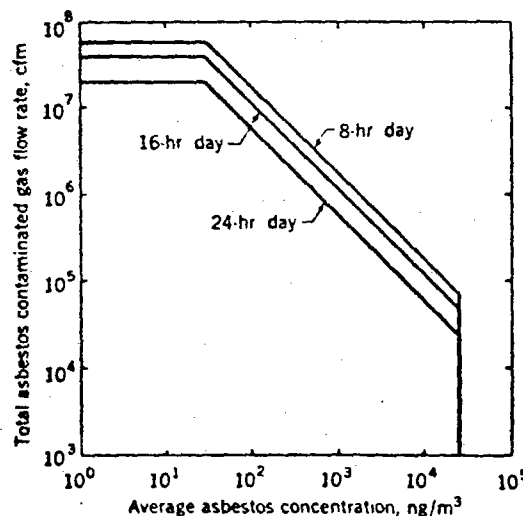


Figure 4. Total asbestos contaminated gas flow rate as a function of allowable asbestos air concentration.

Table II. A survey of asbestos users in Connecticut, 1972.

Type of source	Number of sources	Asbestos used, tons/year	Uncontrolled ^a emission factor, lb/ton	Potential uncontrolled emissions, tons/year
Asbestos cement, floor tile and paper mfg.	17	600	4	1.2
Friction product mfg.	4	3200	120	192
Textile mfg.	12	400	40	0.8
Miscellaneous mfg.	10	900	10	4.5
Vehicle brake lining & clutch facing erosion	1.56 x 10 ⁴	N/A	<30 µg/mile ^b	1.5
Demolition operations	~2500	Unknown	Unknown	Unknown

^a W. E. Davis & Assoc.³⁹

^b Jacko et al.⁴⁰

Analytical Asbestos Determination

In the last few years, techniques for determining chrysotile asbestos concentrations (in terms of ng/m³) in the ambient air, based on electron microscopy, have been developed and used with a reasonable degree of suc-

cess.^{28,33,38,41,44} Dr. Selikoff and his associates report that the unique morphology of chrysotile asbestos makes positive identification possible by transmission electron microscopy and the use of replicate analyses, repeated calibrations, and background measurements indicate that the accuracy of a given measurement is likely to be within 50% of the actual value and certainly within a factor of 2.^{16,33} This technique should be more than adequate in determining the asbestos content of stack samples.

Conclusion

In conclusion, it is believed that a reasonable approach aimed at controlling the dissemination of asbestos fibers into the ambient air has been presented. It is recommended that the rate of nationwide sampling of ambient air asbestos levels be stepped up and continued so as adequately to define the scope of the problem; studies around demolition sites and toll booths are particularly needed. In addition, epidemiological studies of persons who have died of mesothelioma would be useful since they would lead to a better definition of the maximum allowable daily asbestos intake.

Acknowledgments

Mr. Paul Norton, Air Pollution Engineer, Department of Environmental Protection, Engineering Section, Air Compliance Unit, Hartford, CT, Dr. Robert S. Pogrund, Environmental Health Resource Center, Illinois Institute for Environmental Quality, Chicago, IL, Dr. Richard J. Thompson, Chief, Analytical Chemistry Branch, U.S. Environmental Protection Agency gave valued assistance.

References

1. R. A. Clifton, personal communication, U. S. Bureau of Mines, February 1, 1973.
2. R. J. Sullivan and Y. C. Athanassidas, *Air Pollution Aspects of Asbestos*, U. S. Department of Commerce, National Technical Information Service, NTIS #PB 188 080, 1969.
3. *Asbestos: Need for and Feasibility of Air Pollution Controls*, Committee on Biologic Effects of Atmospheric Pollutants, Division of Medical Sciences, National Research Council, National Academy of Sciences, Washington, D. C., 1971.
4. I. J. Selikoff, C. Hammond, and J. Churg, "Carcinogenicity of amosite asbestos," *Arch. Environ. Health* 25: 183 (1972).
5. K. R. Boucot, W. Weiss, H. Seidman, W. K. Carnahan, and D. A. Cooper, "The Philadelphia Pulmonary Neoplasm Research Project: basic risk factors of lung cancer in older men," *Am. J. Epidemiol.* 95: 4 (1972)

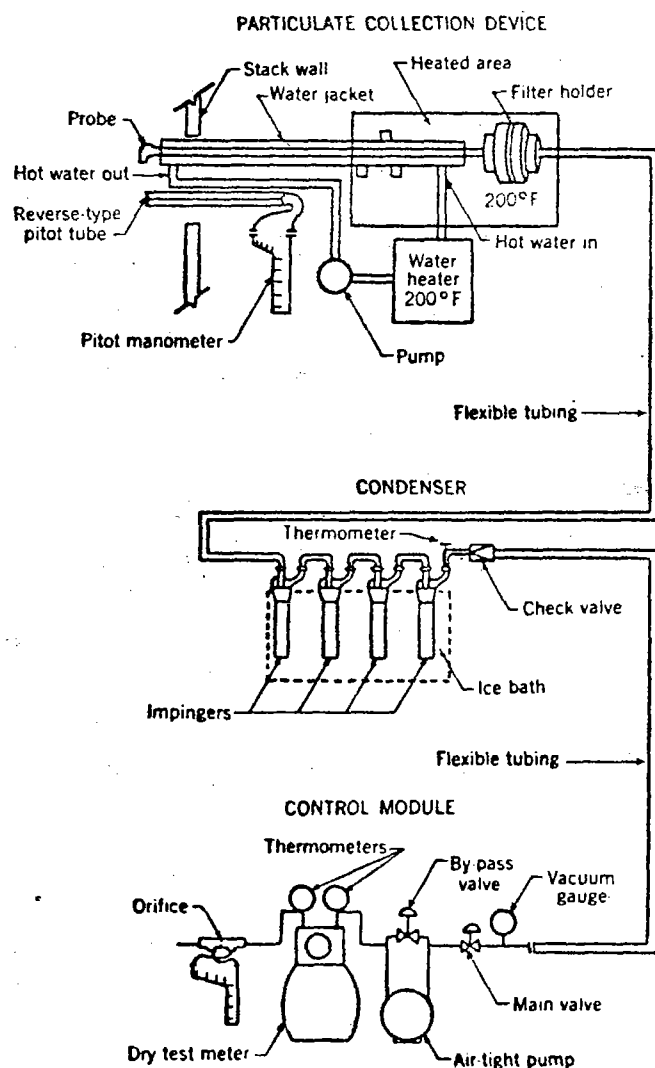


Figure 5. Proposed asbestos sampling train.

7. J. Churg and E. C. Hammond, "Relation between exposure of asbestos and mesothelioma," *New Eng. J. Med.* 272: 560 (1960).
8. R. Sherrill, "Asbestos, the savior of lives, has a deadly side," *N. Y. Times Magazine Section* pp. 12-13, 58-64 (January 21, 1973).
9. Hazards of Asbestos to Human Health, Minnesota Public Research Interest Group, Minneapolis, MN, 1972.
10. I. J. Selikoff and E. C. Hammond, "Environmental epidemiology, III. Community effects of non-occupational environmental asbestos exposure," *Amer. J. Publ. Health* 59: 1658 (1968).
11. J. C. Wagner, "Epidemiology of diffuse mesothelial tumors: evidence of an association from studies in South Africa and the United Kingdom," *Ann. N. Y. Acad. Sci.* 132: 575 (1965).
12. T. Ashcroft and A. G. Heppleston, "Mesothelioma and Asbestos on Tyneside: A Pathological Social Study," in *Pneumoconiosis, Proceedings of the International Conference, Johannesburg*, H. A. Shapiro (ed.), Oxford University Press, Cape Town, South Africa, 1970.
13. J. Hromek, "The mean incidence of characteristic pleural changes in citizens of the western part of former Jilalara Region," *Rozhl. Tuberk.* 22: 405 (1962).
14. R. Kiviluoto, "Pleural calcification as a roentgenologic sign of non-occupational Endemic Anthophyllite-Asbestosis," *Acta Radiol. Suppl.* 194: 1 (1960).
15. L. O. Meurman, "Asbestos bodies and pleural plaques in a Finnish series of autopsy cases," *Acta Path. Microbiol., Scan (Suppl.)* 181: 1 (1966).
16. V. Raunio, "Occurrence of unusual pleural calcification in Finland," *Ann Med. Inf. Fenn (Suppl.)* 49: 55: 1 (1966).
17. A. M. Langer and I. J. Selikoff, "Chrysotile Asbestos in Lungs of Residents of New York City," in *Proceedings of the Second International Clean Air Congress*, H. M. Englund and W. T. Beery (eds.), Academic Press, NY, 1971, pp. 161-165.
18. I. J. Selikoff, W. J. Nicholson, and A. M. Langer, "Asbestos air pollution," *Arch. Environ. Health* 25: 1 (1972).
19. M. D. Utidjian, P. Gross and R. T. P. deTreville, "Ferruginous bodies in human lungs: prevalence at random autopsies," *Arch. Environ. Health*, 17, 327 (1968).
20. J. Lieben and H. Pistawka, "Mesothelioma and asbestos exposure," *Arch. Environ. Health* 14: 559 (1967).
21. M. L. Newhouse and H. Thompson, "Mesothelioma of pleura and peritoneum following exposure to asbestos in the London area," *Brit. J. Ind. Med.* 22: 261 (1965).
22. "EPA sets final emission standards for asbestos, beryllium and mercury," *J. Air Poll. Control Assoc.* 23: 398 (1973), and *Federal Register* 38: (April 6, 1973).
23. *Criteria for a Recommended Standard for Occupational Exposure to Asbestos*, U. S. Department of Health, Education, and Welfare, Public Health Service, Health Services and Mental Health Administration, National Institute for Occupational Safety and Health, HSM # 72-10267, Washington, DC, 1973.
24. "Health Effects and Recommendations for Atmospheric Lead, Cadmium, Mercury, and Asbestos," Environmental Health Resources Center, State of Illinois Institute for Environmental Quality, Report #11 EQ-73-2, Chicago, IL, 1973.
25. H. C. Lewinsohn, "The medical surveillance of asbestos workers," *Roy. Soc. Health J.* 92: 69-77 (1972).
26. M. L. Newhouse, G. Berry, J. C. Wagner, and N. E. Turok, "A study of the mortality of female asbestos workers," *Brit. J. Ind. Med.* 29: 134 (1972).
27. J. Stumphuis, "Epidemiology of mesothelioma on Walcheren Island," *Brit. J. Ind. Med.* 28: 59 (1971).
28. J. R. Lynch, H. E. Ayer, and D. L. Johnson, "The interrelationships of selected asbestos exposure indices," *Amer. Ind. Hyg. J.* 31: 598 (1970).
29. R. J. Thompson, personal communication, preprint, R. J. Thompson and G. B. Morgan, "Determination of Asbestos in Ambient Air," May 2, 1973.
30. I. J. Selikoff, J. Churg, and E. C. Hammond, "Asbestos exposure and neoplasia," *J. Am. Med. Assoc.* 188: 22 (1964).
31. M. L. Newhouse, "A study of the mortality of workers in an asbestos factory," *Brit. J. Ind. Med.* 26: 294 (1969).
32. J. F. Knox, S. Holmes, R. Doll, and I. D. Hill, "Mortality from lung cancer and other causes among workers in an asbestos textile factory," *Brit. J. Ind. Med.* 25: 293 (1968).
33. W. Fulkerson and W. E. Goeller (eds.), "Cadmium: The Dissipated Element" Oak Ridge National Laboratory, Report #ORNL-NSF-EP-21, Oak Ridge, TE, 1973.
34. "Measurement of Asbestos in Ambient Air," Final Report, Contract #CPA-70-92, U. S. Environmental Protection Agency, Research Triangle Park, NC, 1970.
35. "Background Information on Development of National Emission Standards for Hazardous Air Pollutants: Asbestos, Beryllium, and Mercury," U. S. Environmental Protection Agency, Office of Air and Water Programs, Office of Air Quality Planning and Standards, APTD-1503, Research Triangle Park, NC, 1973.
36. D. B. Turner, *Workbook of Atmospheric Dispersion Estimates*, U. S. Environmental Protection Agency, Office of Air Programs, Research Triangle Park, NC, 1970.
37. G. Wight, personal communication, Connecticut meteorological data, Connecticut Department of Environmental Protection, 1973.
38. "Control Techniques for Asbestos Air Pollutants," U. S. Environmental Protection Agency, Office of Air and Water Programs, Office of Air Quality Planning and Standards, AP-117, Research Triangle Park, NC, 1973.
39. C. F. Harwood, "Asbestos Air Pollution Control," State of Illinois Institute for Environmental Quality, IIEQ Document #71-8, Chicago, IL, 1971.
40. "National Inventory of Sources of and Emissions: Asbestos: Section III," W. E. Davis and Associates, National Air Pollution Control Administration contract #22-69-131, PB 192-252, Leawood, KS, 1970.
41. M. G. Jacko, R. T. DuCharme, and J. H. Somers, "How much asbestos do vehicles emit?," *Auto Eng.* 81: 38 (1973).
42. M. G. Jacko and R. T. DuCharme, *Brake Emissions: Emission Measurements From Brake and Clutch Linings from Selected Mobile Sources*, Bendix Research Laboratories, U. S. Environmental Protection Agency report #68-04-0020, Southfield, MI, 1973.
43. J. R. Lynch, "Brake lining decomposition products," *J. Air Poll. Control Assoc.* 18: 824 (1968).
44. A. E. Anderson, R. L. Gealer, R. C. McCune, and J. W. Sprys, "Asbestos Emissions from Brake Dynamometer Tests," Paper 730549 presented at SAE Automobile Engineering Meeting, Detroit, MI, 1973.
45. W. J. Nicholson, A. N. Rohl, and E. F. Ferrand, "Asbestos Air Pollution in New York City," in *Proceedings of the Second International Clean Air Congress*, H. M. Englund and W. T. Beery, (eds.) Academic Press, New York, 1971, pp. 136-139.
46. W. S. Smith, personal communication, Entropy Environmentalists Inc., July 23, 1973.

Discussion

Lawrence A. Plumlee, M.D.

U. S. Environmental Protection Agency, Medical Science Adviser

It is always easy to find fault with another person's work, and it is difficult to come up with alternatives which cannot be criticized with just as much justification. Those of us associated with preparing the standards and regulations for environmental protection are often faced with the necessity of doing the best we can with a limited amount of information. This is particularly true when there is evidence that

damage to the public health will result if we fail to act. Nevertheless, it is my contention that Messrs. Bruckman and Rubino have made some maneuvers which render their asbestos standards unsupportable. One of these is their decision to base their standards on the risk of mesothelioma alone. They fail to take into account the possible environmental asbestos hazard of lung cancer. Among asbestos

Workers, various studies have all shown that the risk of dying of lung cancer is several times that of pleural and peritoneal mesothelioma put together. In one cohort Newhouse reported only 8 mesotheliomas as compared to a total of 36 cancers of the lung and pleura.¹ Selikoff reported 25 lung cancers in a cohort showing only 5 mesotheliomas² and in two additional cohorts, bronchiogenic carcinomas were almost 3 times as common as pleural and peritoneal mesothelioma.³ Admittedly, the high frequency of lung cancer in the general population makes it impossible to associate a given case with asbestos with the high degree of probability that exists for mesothelioma. Nevertheless, this is not adequate reason to disregard bronchiogenic carcinoma in my opinion.

There is another different reason for including lung cancer data on the dose-response curve: This is that the mesothelioma frequency which the authors report is not proportional to exposure. Indeed, the data they use show no change in the probability of contracting mesothelioma over an exposure range of three orders of magnitude. Thus, there seems to be no justification for drawing a mesothelioma incidence envelope which hypothetically assumes a decreasing risk of mesothelioma with decreasing dose. Admittedly this seems logical, but there are no quantitative data to support it. In fact, investigators at Mount Sinai have found that workers exposed to high levels of asbestos are less likely to develop mesothelioma than those exposed to lower levels. This is because those in the high exposure group died of asbestosis before mesothelioma, which has a long latent period, had time to develop. Thus, among 689 workers who were traced for 13 years, estimates of exposure varied directly with asbestosis, but inversely with mesothelioma.⁴ If, however, Bruckman and Rubino had lumped in the asbestos worker bronchiogenic carcinoma data with the mesothelioma data, both Enterline's^{5,6} and McDonald's⁷ data support the concept of increasing cancer accompanying increasing asbestos exposure. With the data presented here, it seems unwarranted to extrapolate a standard which is several orders of magnitude below the lowest levels at which effects were seen using a slope which does not even approximate the data points. Furthermore, the assumptions made in developing the concentration-incidence envelope are not elaborated, but do not appear to justify using the minimum expected risk of 150 mesotheliomas per year when the maximum number of mesothelioma deaths expected per year would be 100,000 fatalities. When this same method of constructing a concentration-incidence envelope was applied by scientists at the State of Illinois Institute for Environmental Quality,⁸ they assumed a risk of cancer midway between the minimum and maximum expected risks and based their recommended standard on this average expected risk, not on the minimum expected risk.

Indeed, it is likely that the incidence of mesothelioma is under reported in the studies used for drawing this curve, since most pathologists and physicians have never seen a mesothelioma. This should have led the authors to view the line predicting minimal probability of mesothelioma with suspicion, and to reject this line in favor of one predicting a higher probability of mesothelioma.

Furthermore, the minimum and maximum expected lines are drawn through the points representing the highest and lowest predicted exposure levels which resulted in mesothelioma. But there are only 12 points on the graph, and since these are highly variable, one would expect statistically that the addition of more points would lower the minimum and raise the maximum levels. Thus they do not really reflect the smallest and largest possible probabilities of contracting cancer at all, since the data presented strongly suggest that additional data would widen the envelope.

The authors state that occupational exposure concentrations must be modified to protect susceptible groups such as the elderly and infants, yet their extrapolation from occupational to environmental levels does not account for elderly and infants, but instead assumes that the risk of developing mesothelioma is the same for them as for workers except for the number of weekly hours of exposure. In actuality, the risk of developing cancer from asbestos is probably greater for infants if for no other reason than the fact that they may carry the inhaled irritant fibers in their bodies for a lifetime. The risk to the elderly is less than for workers, because the chance of their dying of other causes before the latent period for mesothelioma has passed is greatly increased.

I am glad the authors brought out clearly that the OSHA asbestos standard is not stringent enough to protect workers from contracting cancer. And I agree with their decision to assume that there is some risk of cancer at very low exposure levels, and to proceed to provide estimates of risk at these levels so that administrators, and the public as well, may choose a level of risk which seems necessary in the light of competing demands. For example, we may wish temporarily to suffer a slightly increased risk of cancer from a hypothetical persistent substance which we cannot get out of the food chain overnight, rather than confiscate vast amounts of food with consequent malnutrition and skyrocketing food prices. The authors determined an acceptable risk for asbestos apparently based on general public acceptance of a rate 10 times as high as annual deaths due to transportation accidents. I believe that most people consider transportation essential. I gave an example of when it might be essential to eat carcinogens in food. Asbestos may not be as essential as transportation. Some uses of it are not. If there are adequate substitutes for a carcinogen which can be used without intolerable economic penalty, then I believe that no risk from exposure to that carcinogen is acceptable. In other words, an administrator required to set a standard should be given several standards with the costs and benefits of each enumerated, and the political process will help him to make a wise decision. But to do such an analysis for asbestos will require a much more extensive data base than is presently available.

The authors state that EPA's standards would permit 10 tons of asbestos to be emitted yearly in Connecticut, but do not indicate the basis for deriving this estimate. This appears to assume that EPA standards will produce only 95% control, whereas large plants are expected to use baghouses which give 99% control or better. The authors do a good job of extrapolating from their ambient standard to an emission standard, except for one serious omission. They fail to consider the likelihood of reentrainment of fibers back into the air at a later time, or their ultimate fate, as for example, runoff into water and thus into the food chain. Nicholson has found that dust in attics of houses near a formerly active asbestos plant are still contaminated with asbestos 20 years later.⁴ Langer has preliminary data which indicate increasing asbestos in recent times in the Greenland ice cap.⁹ Thus the possibility of accumulation of asbestos in the environment, with the possibility of later reentrainment into the air, must be borne in mind.

A more serious problem is the authors' assumption that there are 1000 asbestos fibers per nanogram of asbestos. Dr. Richard Thompson, whose estimate they quote, has indicated that this estimate was an educated guess and now he believes that it is more likely that there are 100,000 or 1,000,000 electron microscopically visible asbestos fibers in a nanogram of asbestos.¹⁰ Nicholson has estimated that there is about 1 fiber (greater than 5µm in length) per nanogram of asbestos.⁴ But, equivalents which apply to the workplace may not apply to ambient air, because the larger fibers settle out of the air first, leaving a larger proportion

of small fibers. What this means is that even if everything else about the mesothelioma-asbestos exposure extrapolation which the authors used were reasonably correct—which it probably is not—there may still be an error of 10^3 or more due to the great inaccuracy which we incur when trying to convert fiber counts to nanograms. Furthermore, EPA has repeatedly experienced a much poorer degree of replicability than the factor of 2 which the authors believe can be attained by present analytic capability. This is particularly true when duplicate samples are sent to different experts. Testimony by Dr. Brown at the federal court in St. Louis, which is reviewing the Reserve Mining Case, indicated much greater inaccuracy of the analytic methods used.¹¹

The proposed asbestos sampling train is similar to that under test by EPA. The authors do not describe the kind of filter they would use, but membrane filters would not be suitable at very high gas temperatures, or would require cooling the stack gases first. It is not obvious nor stated in the text as to why a condenser is recommended in the authors' asbestos collection device.

In conclusion, I would like to emphasize that I respect the authors' intention to make the best use of available data in trying to derive solutions to the environmental asbestos problem. I realize that the authors must have been aware of some of the criticisms that I have made, but probably felt that the need for quantitative ambient air and emission standards warranted the proposal. However, my view is that the exposure level data from occupational studies, as well as present analytic methodology, are so unreliable as to make the results of this exercise meaningless. Until acceptable data are available, the most justifiable approach to protecting the public from asbestos health hazards is the control of the major sources into the environment to the maximum extent practicable.

Acknowledgment

Dr. Irving J. Selikoff and Mr. James U. Crowder, Jr. provided valuable assistance in the preparation of this critique.

Authors' Response

We are gratified to learn of the interest in our article. The points which Dr. Plumlee raise are certainly worthy of comment. Dr. Plumlee has correctly stated in the addendum to his review of the article that our objective was to derive a numerical asbestos standard which should not be exceeded at this time.

The criteria of mesothelioma was selected as the basis for developing the proposed ambient air quality asbestos standard of 30 ng/m^3 , based on a 30 day average sample,^{1,2} because:

1. As Dr. Plumlee stated, the high frequency of lung cancer in the general population does make it difficult to relate

Addendum

Following the writing of this review, it was pointed out to me that all of the assumptions made by Bruckman and Rubino are such as to make the standard they recommend less stringent. That is, their standard does not appear to be subject to the criticism that it may be too strict. If anything, it must be considered as too weak. A review of this paper confirms this comment. Bruckman and Rubino indicate that various sources exceed their standard. Their paper can be considered a reasonable basis for requiring asbestos emissions to be less than the standard which they derive. While their standard cannot be considered to be adequately protective, it does seem reasonable to believe that it should not be exceeded.

References

1. M. L. Newhouse, "Asbestos in the work place and the community," *Ann. Occup. Hyg.* 16: 97 (1973).
2. I. J. Selikoff, E. C. Hammond, and J. Churg, "Carcinogenicity of amosite asbestos," *Arch. Environ. Health* 25: 186 (1972).
3. I. J. Selikoff, E. C. Hammond, and H. Seidman, presented at the meeting of the Working Group to Assess Biological Effects of Asbestos, IARC, Lyon, France, October 4, 1972.
4. Private communication with Dr. William J. Nicholson.
5. P. E. Enterline, P. de Coufle, and V. Henderson, "Mortality in relation to occupational exposure in the asbestos industry," *J. Occup. Med.* 14: 897 (1972).
6. P. E. Enterline, P. de Coufle, and V. Henderson, "Respiratory cancer in relation to occupational exposures among retired asbestos workers," *Brit. J. Ind. Med.* 30: 162 (1973).
7. J. C. McDonald, "Cancer in Chrysotile Mines and Mills," paper 29 presented at Conference on Biological Effects of Asbestos, IARC, Lyon, France, October 2-5, 1972.
8. "Environmental Asbestos, Health Effects and Recommended Standards," Illinois Institute for Environmental Quality, December 1972 (available from Environmental Health Resource Center, P.O. Box 6998, Chicago, IL).
9. Private communication with Dr. Arthur M. Langer.
10. Private communication with Dr. Richard J. Thompson.
11. Reserve Mining Company, et al., v. United States of America, et al., (United States Court of Appeals, Eighth Circuit), June 4, 1974, p. 12.

a given case of bronchiogenic carcinoma to asbestos exposure with the high degree of probability that exists for mesothelioma, and

2. Some investigators maintain that the smaller asbestos fibers (those fibers less than 5 microns in length) most likely encountered in the ambient air (as Dr. Plumlee has stated, the larger asbestos fibers settle out of the air first, leaving a greater proportion of small fibers) are incapable of producing lung cancer; however, it has not been demonstrated that these short asbestos fibers are incapable of inducing mesothelioma.

Dr. Plumlee states that mesothelioma frequency is not proportional to exposure, however, investigations of the distribution of mesothelioma in occupationally exposed individuals indicate a definite correlation between exposure to asbestos fibers and mesothelioma incidence.^{3,9}

As was stated in the article, the boundaries of the asbestos concentration-mesothelioma incidence envelope were based on the extreme high and low data points. Because quantitative low level exposure information is not currently available, the Illinois Environmental Health Resource Center (EHRC) decided to extrapolate the available occupational data by using a linear dose-effect relationship.³ The authors agree with EHRC. Admittedly, more improved quantitative information concerning the relationship between low level ambient asbestos fiber exposure and the associated health effects, especially the risk of contracting mesothelioma, is needed as soon as possible to refine the asbestos exposure-mesothelioma incidence envelope.

The current EPA asbestos standard is essentially a no visible emission standard or the application of best available control technology. No visible emissions could easily be achieved by control equipment other than fabric filters which yield 95% control (e.g., wet cyclone). Plants which are currently meeting the no visible emissions standard without the use of fabric filters cannot be expected to install fabric filters. Furthermore, best available control technology can mean different things to different people. Who is to decide what is "best"? In our opinion, a numerical standard would be much easier to enforce, in addition to providing an incentive for manufacturers to develop more efficient control equipment, since some plants might not meet the proposed emission standard even with fabric filters.

The reentrainment of asbestos fibers into the ambient air at some later time, or their ultimate fate, was not considered when the emission standard was developed; however, the significance of this omission is unknown at this time.

Dr. Plumlee seems to have confused asbestos fibrils (i.e., ultimate asbestos fibers, 200 to 400 Å in diameter and often 2000 Å or smaller in length) with asbestos fibers, which in general can either be less than or greater than a micron long, with varying diameter sizes. Dr. Thompson estimated that there were approximately 100,000 to 1,000,000 asbestos fibrils (not fibers) in a nanogram of asbestos.¹⁰ Wesolowski reported that there were approximately 6000 to 280,000 (arithmetic mean of 110,000) asbestos fibrils per nanogram of asbestos.¹¹

Dr. Thompson also estimated that there were 100 asbestos fibrils per fiber visible under the electron microscope.¹⁰ Thus, assuming 100 fibrils per fiber and 100,000 to 1,000,000 fibrils per nanogram, there would be approximately 1000 to 10,000 asbestos fibers (not fibrils) per nanogram of asbestos. We used 1000 fibers per nanogram in the development of our conversion factor relating asbestos fibers greater than 5 microns in length to nanograms of asbestos. Dr. Nicholson's estimate of 1 fiber greater than 5 microns in length per nanogram of asbestos appears to be consistent with our assumption (i.e., 20 asbestos fibers greater than 5 microns in length per nanogram of asbestos). Furthermore, on the basis of assuming that most chrysotile asbestos fibers have a hollow cylindrical form with an average outer diameter 4 to 12 times the inner one, we calculate that there would be about 40 to 65 asbestos fibers 5 microns long, with a 3:1 length to width ratio, per nanogram of chrysotile asbestos.¹² The Battelle Columbus Laboratory reported that on the average (composite data from 88 am-

phibole analyses) there were 26 amphibole asbestos fibers greater than 5 microns in length per nanogram of amphibole asbestos.¹³ Thus, while the conversion used to relate asbestos fibers greater than 5 microns in length to nanograms of asbestos is not perfect, it seems to be reasonably accurate (20 vs. 26) and would certainly not introduce errors of 1000 or more into our asbestos exposure-mesothelioma incidence envelope as Dr. Plumlee claims.

The accuracy of the measurement technique used by Battelle to determine the chrysotile asbestos concentration of ambient air samples was reported to be $\pm 50\%$ based on the analyses of activated chrysotile sample.¹³ However, Dr. Plumlee's point concerning the problem of measuring the asbestos content of environmental samples is well taken. The accuracy of the techniques used to sample for asbestos and the analytical procedures used for the identification and counting of asbestos fibers should be improved and standardized as soon as possible.

In conclusion, we feel that our study provides a reasonable basis for requiring asbestos emissions to be less than our proposed standard, the implementation of which on a national basis would result in both significant reductions in asbestos emissions and projected asbestos related fatalities as compared to the current EPA no visible emissions standard.

References

1. Environmental Health Resource Center News, Illinois Institute for Environmental Quality, Chicago, IL, Issue # 12, 1974.
2. P. Gross, "Is short-fibered asbestos dust a biological hazard," *Arch. Environ. Health* 29, 115 (1974).
3. *Health Effects and Recommendations for Atmospheric Lead, Cadmium, Mercury and Asbestos*, Environmental Health Resources Center, State of Illinois Institute for Environmental Quality, Report #11 EQ-73-2, Chicago, IL, 1973.
4. J. F. Know, S. Holmes, R. Doll, and I. D. Hill, "Mortality from lung cancer and other causes among workers in an asbestos textile factory," *Brit. J. Ind. Med.* 25: 293 (1968).
5. H. C. Lewinsohn, "The medical surveillance of asbestos workers," *Roy. Soc. Health J.* 92: 69 (1972).
6. M. L. Newhouse, "A study of the mortality of workers in an asbestos factory," *Brit. J. Ind. Med.* 26: 294 (1969).
7. M. L. Newhouse, G. Berry, J. C. Wagner, and N. E. Turok, "A study of the mortality of female asbestos workers," *Brit. J. Ind. Med.* 29: 134 (1972).
8. I. J. Selikoff, J. Churg, and E. C. Hammond, "Asbestos exposure and neoplasia," *J. Am. Med. Assoc.* 188: 22 (1964).
9. J. Stumphuis, "Epidemiology of mesothelioma on Walcheren Island," *Brit. J. Ind. Med.* 28: 59 (1971).
10. R. J. Thompson, personal communication, May 7, 1975.
11. J. J. Wesolewski, "Asbestos in the California Environment," Air and Industrial Hygiene Laboratory Report, AIHL #164, California State Department of Health, Berkeley, California, May, 1974.
12. "Asbestos in the Great Lakes Basin with Emphasis on Lake Superior," A Report to the International Joint Commission from the Great Lakes Research Advisory Board, February, 1975.
13. C. W. Melton, personal communication, February 13, 1975.

Mr. Bruckman is principal air pollution control engineer and Mr. Rubino is assistant director of the Air Compliance Unit, Engineering Section, Connecticut Department of Environmental Protection, State Office Building, Hartford, CT 06115. This paper was presented as Paper No. 74-222 at the 67th Annual Meeting of APCA at Denver in June 1974. Dr. Plumlee, whose discussion of the paper is also presented here, is a medical science adviser with the U. S. Environmental Protection Agency.