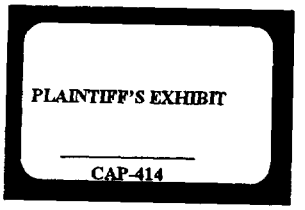

COMMENTS OF THE

ASSOCIATION OF ASBESTOS/CEMENT PIPE PRODUCERS

ON

EPA'S ADVANCE NOTICE OF PROPOSED RULEMAKING

ON

NATIONAL REVISED PRIMARY DRINKING WATER REGULATIONS

(40 C.F.R. Part 141; WH-FRL 2418-1)

John F. Welch
Association of Asbestos/
Cement Pipe Producers
1600 Wilson Boulevard
Suite 1008
Arlington, VA 22209
(703) 841-1556

Timothy S. Hardy
Kirkland & Ellis
1776 K Street, N W.
Washington, D.C. 20006
(202) 857-5042

Special Counsel to the
Association of Asbestos/
Cement Pipe Producers

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COMMENTS OF THE ASSOCIATION OF ASBESTOS/ CEMENT PIPE PRODUCERS ON EPA'S ADVANCE NOTICE OF PROPOSED RULEMAKING ON NATIONAL REVISED PRIMARY DRINKING WATER REGULATIONS

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* Given the great likelihood that EPA already has in its files almost all of the sources relied upon in these comments, we are not submitting copies of these authorities with our comments; should, however, the Agency wish copies of any of the referenced works, AACPP would be glad to supply them.

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COMMENTS OF THE ASSOCIATION OF ASBESTOS/ CEMENT PIPE PRODUCERS ON EPA'S ADVANCE NOTICE OF PROPOSED RULEMAKING ON NATIONAL REVISED PRIMARY DRINKING WATER REGULATIONS

INTRODUCTION

Consistent with our long history of providing information to and cooperating with the Environmental Protection Agency to understand the asbestos in drinking water issue, the Association of Asbestos/Cement Pipe Producers (AACPP) welcomes this opportunity to comment on EPA's Advance Notice of Proposed Rulemaking (ANPRM) on National Revised Primary Drinking Water Regulations, 48 Fed. Reg. 45502 (Oct. 5, 1983). AACPP is a trade association representing all domestic companies that produce and market asbestos-cement (A/C) pipe, as well as twenty-five companies manufacturing or marketing A/C pipe throughout the world. AACPP has supported EPA's intensive scientific activities over the past ten years to develop a data base from which reasonable conclusions can be drawn on the wisdom of promulgating a national primary drinking water standard for asbestos. Given the results of this research, EPA now has in-hand more than sufficient data to conclude that no such regulation is warranted under the Safe Drinking Water Act (SDWA), 42 U.S.C. §§ 300f et seq.

I. EPA HAS EXTENSIVELY STUDIED THE EFFECTS OF ASBESTOS IN DRINKING WATER OVER THE PAST TEN YEARS AND HAS CONFIRMED THE ABSENCE OF ANY EVIDENCE OF ADVERSE HEALTH EFFECTS.

Due to the known serious adverse effects of inhaled asbestos, substantial scientific inquiry has been directed toward determining whether ingested asbestos, and especially asbestos ingested from drinking water, poses any human risk. Numerous independent scientific groups throughout the world concluded in the mid-1970's that there was no cause for public health concern. These groups also recognized the need for additional scientific research to confirm their admittedly tentative conclusions.

Since the mid-1970's, that research has been conducted, much of it sponsored by EPA. As a result, the earlier tentative conclusions have been confirmed. The following history of the past ten years thus provides the essential starting point for responding to EPA's ANPR and demonstrates why no national primary drinking water standard is warranted.

A. Numerous Reports in the Mid-1970's Uniformly Found No Reason for Public Health Concern About Asbestos in Drinking Water, But Called for Additional Research.

Perhaps the first comprehensive review of the scientific evidence on asbestos in water was sponsored by the American Water Works Association Research Foundation. In 1974, at the urging of and supported by AACPP, AWWA convened

a panel of distinguished experts to "analyze[], to the extent possible, all the pertinent literature on the biological effects of asbestos."[1/] The panel concluded, "the slight or perhaps no risk posed by low level occupational exposure makes it even less likely that the exposure from ingestion of public water would pose a gastro-intestinal cancer hazard,"[2/] and noted further:[3/]

> Calculations comparing the probable ingestion exposure in occupational groups to that likely to occur as a result of ingestion of potable water from asbestos-cement pipe systems suggests that the probability of risk to health from the use of such systems is small -- approaching zero.

Three years later the Safe Drinking Water Committee of the National Academy of Sciences reached essentially the same conclusion: existing studies "do not suggest an immediate hazard to public health" from "asbestos orally ingested

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1/ "A Study of the Problem of Asbestos in Water," J. AWWA 1 (Part 2, Sept. 1974). The Panel included Dr. Marvin Kuschner, then Dean of the School of Medicine at SUNY (Stony Brook); Roger Lee and Gordon G. Robeck of the EPA Water Supply Division; John Rossum of the California Water Service Company; Dr. Marvin A. Schneiderman, then with the National Cancer Institute; Dr. E. Windle Taylor of the London, England, Metropolitan Water Board; and George W. Wright, M.D. Id.

2/ Id. at 5.

3/ Id.

through drinking water."[4/] An expert panel of Europeans agreed,[5/] as did the British Advisory Committee on Asbestos.[6/]

Many of the expert reviewing bodies recognized, however, that the data from which they had drawn reassuring conclusions were limited. They thus called for additional animal ingestion studies, epidemiology studies of populations who had been exposed to asbestos in drinking water, better data on asbestos levels in drinking supplies, studies of the ability of asbestos to migrate through the gastrointestinal wall, and development of improved monitoring methods.[7/]

4/ NAS Safe Drinking Water Committee, Drinking Water and Health 191 (1977), issued in response to SDWA § 1412(e), 42 U.S.C. § 300g-1(e).

5/ Zielhuis, R.L., Rapporteur, Comm. of the European Communities, Public Health Risks of Exposure to Asbestos 13 (1977) ("At this moment, there is no evidence that there exists any increased health risk from asbestos fibres in drinking water . . .").

6/ United Kingdom Health and Safety Executive, Asbestos: The Final Report of the Advisory Committee, Vol. I at 38, 60 (1979) ("studies to investigate the carcinogenicity of ingested asbestos have generally yielded negative results and [ ] there is no clear epidemiological evidence of increased incidence of carcinoma in the gastro-intestinal tract attributable to asbestos in non-occupationally exposed populations").

As the Chariman of the Advisory Committee's medical panel, Dr. Donald Acheson, has testified, "the Committee as a whole tended to discount risk relating to drinking water." Ontario Royal Commission on Asbestos, Tr. Vol. XIX, at 95 (July 1981).

7/ See, e.g., NAS, supra note 4, at 191; AWWA, supra note 1, at 22.

B. Since the Mid-1970's, EPA Has Conducted Extensive Research to Determine If There Are Any Adverse Effects from Asbestos in Drinking Water.

Given the expert panel recommendations, EPA embarked on a comprehensive research program. As the Agency reported to Congress in June 1975, even the few preliminary studies then initiated, relating primarily to Duluth water supplies, were expected to "contribute significantly to our understanding" of asbestos and health.8/

EPA's program has been outlined by the Agency's Industrial Environmental Research Laboratory.9/ This 1981 survey of on-going and completed research noted work at EPA's Cincinnati Municipal Environmental Research Laboratory on removal of asbestos from water, analysis of drinking water samples, erosion of asbestos from A/C pipe, and water treatment techniques; work at the Cincinnati Health Effects Research Laboratory on cell culture and *in vitro* asbestos assays, asbestos fiber fate in baboons, overall assessment of U.S. drinking water exposures, and epidemiology studies in the Puget Sound region, Duluth, Minnesota, five California counties, and Connecticut; work at the Athens, Georgia,

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8/ EPA Office of Toxic Substances, "Preliminary Assessment of Suspected Carcinogens in Drinking Water: Interim Report to Congress," 21 (June 1975).

9/ EPA Industrial Environmental Research Laboratory, "Research and Development: Asbestos/Asbestiform Research in EPA ORD," EPA-600/7-81-032 (March 1981).

Environmental Research Laboratory on monitoring methodology; and major state-of-the-art rat and hamster ingestion bioassays at the National Institute of Environmental Health Sciences in Research Triangle Park, North Carolina. EPA and NIEHS have invested well over $30 million over the past ten years to study asbestos in water.

As noted in the EPA research review, and as presented at EPA's Summary Workshop on Ingested Asbestos last fall,[10/] these many research projects have almost all been completed. EPA's major commitment to respond to the mid-1970's recommendations for further research has been fulfilled. Indeed, as Dr. Joseph A. Cotruvo, Director of Criteria and Standards in EPA's Office of Drinking Water, has noted, "[v]ery few other issues have had the volume and intensity of study and resultant information."[11/]

As detailed in Parts III and IV of these comments, the results of this research -- in particular the state-of-the-art bioassays and new epidemiology studies that have all been negative and the extensive monitoring that has confirmed the very low concentrations of asbestos in U.S. drinking

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10/ Environmental Protection Agency, Transcript of "Proceedings of the U.S. EPA Summary Workshop on Ingested Asbestos" (Briedenbach Research Center, Cincinnati, Ohio, Oct. 13-14, 1982) [hereafter cited as EPA Workshop Tr.], which has been peer and policy reviewed and is to be published in Environmental Health Perspectives (Nov. 1983).

11/ Cotruvo, J.A., "Asbestos in Drinking Water -- A Status Report," EPA Workshop Tr., supra note 10, at 607.

water supplies -- should lay to rest any lingering tentativeness in conclusions as to the absence of health effects due to ingested asbestos. It is certainly the case, as Dr. Cotruvo stated in summing up the 1982 Workshop that, "[w]hile sufficient data on which to base a decision may not have been available in the past, it appears that we are now at a point where there is adequate information along with the appropriate mechanism for reaching a decision in a reasonable period of time."[12/]

C. Consistent with Decisions of the World Health Organization and Other Nations, EPA Should Not Promulgate a Recommended Maximum Contaminant Level for Asbestos.

Through the ANPR process, it is now an appropriate time for EPA to close the book on ingested asbestos and to assure the public that it need not fear any adverse health effects from asbestos in U.S. drinking water. EPA should join the World Health Organization and other nations,[13/] the American

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12/ Id. at 612.

13/ Although proposing guideline limits for numerous other substances, the World Health Organization, "Guidelines for Drinking Water Quality" (1982), recently announced that no guidelines would be set for asbestos.

AACPP is aware of no nation that has established regulatory controls on asbestos concentrations in drinking water. The absence of such regulations is fully explained by the conclusions reached by health experts in numerous nations that no risk exists from existing asbestos concentrations:

(Footnote 13 continued on next page.)

Water Works Association,[14] and numerous states[15] in determining that no regulations need be established for monitoring or control of asbestos in drinking water.

(Footnote 13 continued from previous page.)

A recent United Kingdom Parliamentary Report concluded that asbestos in water poses "no risk to health." Cited in Commins, B.T., "Asbestos Fibres in Drinking Water," 7 (May 1983).

An official report of the German Health Agency concluded similarly. Aurand, K., and W.S. Kierski, "Assessment of Potential Health Hazards from Asbestos Fibres in Drinking Water and Food," 4 Bundesgesundheitsamt, Berichte (1981). ("All calculations [ ] so far appear to suggest even under the most unfavourable assumptions that the intake from asbestos fibres with food, drinking water or other beverages would present a very low risk, the existence of which appears doubtful . . .")

A recent South African review of the ingestion evidence, including the data presented at EPA's fall 1982 Workshop, see note 10 supra, reached the same conclusion. Editorial, "Asbestos in Drinking Water," 63 S. African Med. J. 217 (1983):

> No evidence whatsoever exists that ingested asbestos fibres in the concentrations possible in ordinary environments constitute a health hazard: fears relating to potable water in contact with asbestos under realistic conditions may now be abandoned.

14/ Although still subject to final approval by the AWWA Board of Directors, the position statement adopted after several years of deliberation by the AWWA Ad Hoc Committee on Asbestos in Drinking Water states: "Unless a definite causal relationship between asbestos in drinking water and adverse health effects is demonstrated, standards and regulations for asbestos in drinking water should not be promulgated." AWWA Mainstream 7 (Nov. 1983).

15/ See, e.g., letters to Johns-Manville Sales Corp. from state health and environmental officials of Kansas, Nebraska, Missouri, Wyoming, Utah, South Dakota, and Nevada (1979 and

(Footnote 15 continued on next page.)

II. THE STATUTORY STANDARDS OF THE SAFE DRINKING WATER ACT PRECLUDE EPA FROM REQUIRING BURDENSOME, COSTLY MONITORING OR TREATMENT OF ASBESTOS FIBERS IN WATER.

In enacting the Safe Drinking Water Act (SDWA) in 1974, Congress was concerned that a multitude of chemicals in the nation's drinking water could be causing adverse human health effects. It thus directed EPA, with the assistance of the National Academy of Sciences (NAS), to study all such chemicals. If the Agency found adverse effects were likely to occur, it was to set monitoring and/or treatment requirements through issuance of national primary drinking water standards. Given this stringent scheme of regulation, Congress recognized that resources should be employed to control those chemicals most likely to be causing adverse health effects and most amenable to control. Given the totally speculative, at best, health effects of asbestos in drinking water and the technological and economic infeasibility of monitoring and treatment, asbestos is not a candidate for a national primary drinking water standard.

(Footnote 15 continued from previous page.)

1980), and letter to State Senator Buchanan from the Virginia State Health Commissioner (Sept. 19, 1979) (all letters on file at AACPP). See also the comments of a California State Department of Public Health officer that there was "no discernible health hazard" from asbestos in the state's drinking water. Los Angeles Times, Oct. 11, 1983, at Part II, p. 5.

A. Congress Recognized the Multitude of Potential Chemicals for Which National Primary Drinking Water Regulations Should Be Considered and Expected EPA to Focus Attention on Those Chemicals Most Likely to Cause Adverse Effects.

Congress was aware in 1974 of the vast number of chemicals that enter the nation's drinking water supply. The House Report on the SDWA noted that "12,000 chemicals are now being used commercially" and "500 new chemical compounds are added each year."16/ Because the Committee recognized that "[i]t is, of course, impossible for EPA to regulate each of these contaminants," it directed the Agency to establish primary drinking water regulations for "groups" of contaminants "which may be harmful to health."17/ As stressed

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16/ "Safe Drinking Water Act," H. Rep. 93-1185 to Accompany H.R. 13002, 93d Cong., 2d Sess. (July 10, 1974), reprinted in "A Legislative History of the Safe Drinking Water Act," Comm. Print, Senate Committee on Environment and Public Works, 97th Cong., 2d Sess. 542 (1982) [hereinafter SDWA Leg. Hist.].

17/ Id. One of the "groups" of "contaminants" for which regulations were anticipated was asbestos. Id. This expectation, however, preceded not only the mid-1970's reports generally finding no reason for public health concern about asbestos in water, but also the last decade of research that has confirmed the absence of any scientific basis for concern.

In addition, review of the SDWA legislative history reveals that most of the Congressional asbestos concern was generated by the U.S. Court of Appeals for the Eighth Circuit's reversal of a District Court order to remedy taconite tailing discharges into Lake Superior. The concern was focused on an apparent belief that the Court of Appeals presumed no adverse health effects existed in the absence of

(Footnote 17 continued on next page.)

by Congressman Paul Rogers, chairman of the subcommittee from which the SDWA emerged, Congress was concerned about chemicals that "may pose a threat to the public health."[18/]

The need to be selective in determining what chemicals "may" cause "adverse effects" was reemphasized by the NAS in its first 1977 report responsive to the Congressional call for review of possible contaminants. Although it believed thresholds could not be established for long term effects of toxic agents, NAS set as a guiding principle the concept that compounds should be assessed "in terms of human risk, rather than as 'safe' or 'unsafe.'"[19/]

(Footnote 17 continued from previous page.)

definitive data and a feeling the SDWA should reverse that presumption. See, e.g., comments of Cong. Steelman, SDWA Leg. Hist., supra note 16, at 658; comments of Cong. Ruppe, id., at 690-91. To the extent the Act reversed that presumption, the comprehensive scientific data accumulated in the last decade has demonstrated that regardless of who bears the presumption, no adverse effects can be found to exist from asbestos in drinking water.

18/ SDWA Leg. Hist., supra note 16, at 652 (emphasis added).

19/ See 48 Fed. Reg. at 45508. EPA has extended this principle in seeking to respond to Congressional suggestions that RMCL's for carcinogens should be zero. SDWA Leg. Hist., supra note 16, at 543, 552. Although not relevant to assessment of ingested asbestos -- given the overwhelming data indicating it is not carcinogenic in man or animal -- the wisdom of this suggestion has been questioned by EPA in consideration of regulations for allegedly carcinogenic volatile synthetic organic chemicals, 47 Fed. Reg. 9350, 9357 (March 4, 1982), as indeed it should, given the total impracticability of regulating chemicals at very, very low trace levels when even conservative risk assessments predict human risks at such levels in the 10^{-5} and lower range.

The SDWA requires that once a recommended maximum contaminant level (RMCL) is set, either a legally enforceable maximum contaminant level (MCL) or a required treatment technique regulation be promulgated.[20/] Accordingly, were EPA to issue RMCL's on the basis of the vaguest of speculation about adverse health effects, even when such speculation was refuted by the available scientific data, the consequence would be innumerable regulations that would severely impede the ability of the nation's water suppliers to allocate their resources to meet the most pressing health needs.[21/] Congress clearly directed no such result.[22/] When, as with asbestos, no evidence suggests the likelihood of any adverse effects from drinking water concentrations, issuance of an RMCL is unwarranted under the SDWA.

20/ SDWA § 1412(b)(2), 42 U.S.C. § 300g-1(b)(2).

21/ Faced with a statute, the Occupational Safety and Health Act, that contained similar presumptions in favor of safety and health, the Supreme Court recognized that regulation in the absence of findings of "significant" health effects is unwarranted. Industrial Union Department, AFL-CIO v. American Petroleum Institute, 448 U.S. 607, 642 (1980).

As Justice Powell's concurrence noted, given the "[t]housands of toxic substances," any OSHA regulation that was not based on "comparative benefits" could result "in an effort to reduce a single risk ..., even though other significant risks remain unregulated." "I would not attribute such an irrational intention to Congress," the Justice concluded. Id. at 670 & n.7.

22/ In addition, in dealing with asbestos, for which the predominant source in drinking water is natural erosion, Congress created a special variance provision in SDWA § 1415, 42 U.S.C. § 300g-4, for jurisdictions that cannot meet national primary standards because of "characteristics of the raw water sources" from which drinking water is supplied.

B. The Need for RMCL's Is Particularly Weak When the Chemical in Question Is Found Only at Parts-per-Trillion Levels and Is Predominantly Due to Natural Raw Water Sources, and When Drinking Water Represents Only a Small Fraction of Human Exposure.

The absence of any need for an asbestos national primary drinking water standard is particularly emphasized by the very low levels of asbestos in the nation's drinking water supply. Even when attention has been focused (in admittedly non-representative sampling programs) on those drinking water supplies most likely to contain high levels of asbestos, only 20% of the nation's water supplies have been found to have concentrations above 1 million fibers/liter.[23/] A smaller, but more representative, sample found asbestos concentrations to be considerably lower, with 61% of the U.S. population's water supplies having concentrations below detectable levels, another 27% below 1 million fibers/liter, and only 7% and 5% between 1 and 10 and above 10 million fibers/liter, respectively. Indeed, as noted by EPA's expert on asbestos monitoring, Dr. James R. Millette, "[c]onsideration of the population served by the U.S. water supplies . . . suggests that the percentage of U.S. population receiving water with fiber concentrations exceeding

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23/ Millette, J. R., Clark, P. J., and Pansing, M. F., "Exposure to Asbestos from Drinking Water in the United States," Environmental Health Effects Research Report, Office of Research and Development, EPA-600/1-79-028 (1979); Millette, J. R., et al., "Concentration and Size of Asbestos in Water Supplies" 34 Environ. Health Perspect. 13 (1980).

10 MFL [million fibers/liter] is . . . somewhat less than 5% . . . ."[24/]

Moreover, although some water-borne asbestos derives from mining runoff, asbestos product manufacture discharges, or A/C pipe degradation, most areas with high fiber concentrations represent the results of natural erosion of fibers from asbestos outcroppings. As a result of their natural source, the great majority of asbestos fibers in water are shorter than 5 microns,[25/] a significant factor in reducing the likelihood of cancer causation, as discussed in Part IV of these comments.

The seemingly high concentrations represented by 1 million fibers/liter, moreover, are in fact not at all substantial when put in terms more typically used for other chemicals. That is, 1 million fibers/liter is roughly equivalent to 1 part-per-trillion on a weight basis, the basis used for measuring most other chemicals in water.[26/]

24/ Millette, J.R., et al., "Asbestos in Water Supplies of the United States," EPA Workshop Tr., supra note 10, at 127, 134, 135, 140-41.

25/ Id. at 135.

26/ Lawrence, J., et al., "Removal of Asbestos Fibres from Potable Water by Coagulation and Filtration," 9 Water Research 397 (April 1975) ("a concentration of $1x10^6$ fibres/liter corresponds to a mass concentration of only on the order of 1 ng/liter [1 part-per-trillion]").

Although such calculations cannot be made precisely, given varying sizes and weights of asbestos fibers, the average fiber weighs between 10^{-12} and 10^{-15} grams. See

(Footnote 26 continued on next page.)

These very low levels of asbestos in drinking water loom even smaller when considered in the context of other possible human sources of asbestos ingestion. A preliminary EPA assessment has concluded that gastrointestinal exposures to asbestos are much more likely from dietary sources (including mayonnaise, ketchup, meats, and various beverages) and ambient air, than from drinking water. The first two sources were estimated to contribute 1,000 to 10,000 times more fiber exposure to the gut than even drinking water concentrations of 1 to 10 million fibers/liter.27/ The contribution of drinking water exposures thus pale in significance.

In sum, prevailing U.S. asbestos drinking water concentrations are at the part-per-trillion level due primarily to natural erosion and are likely to represent only a very small fraction of total ingested asbestos exposures. Even if one were to have evidence of likely adverse effects from asbestos ingestion, little justification would exist for

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(Footnote 26 continued from previous page.)

Commins, supra note 13, at 61-62. Therefore, 10^6 fibers/liter is equivalent to 10^{-6} to 10^{-9} grams/10^3 grams (the weight of a liter of water), or 1 part-per billion to 1 part-per-trillion. The latter figure is more likely as most asbestos fibers found in water are short (less than 5 microns), and thus lighter. Millette, supra note 24, at 135.

27/ Rowe, J. N., "The Relative Source Contributions of Diet and Air to Ingested Asbestos Exposures," EPA Workshop Tr., supra note 10, at 356, 360, 367.

establishing a national primary drinking water standard with its concomitant monitoring and/or treatment requirements. Given, as discussed next, the overwhelming body of scientific data finding no ingested asbestos adverse health effects, it is clear no such standard is warranted under the SDWA.

III. NONE OF THE EVIDENCE ON INGESTED ASBESTOS INDICATES ANY "ADVERSE EFFECTS ON THE HEALTH OF PERSONS."

The starting point for assessing whether a national asbestos drinking water standard is called for by the SDWA is the wealth of scientific data exploring whether asbestos ingestion causes adverse health effects. Although much of the data was preliminary in the mid-1970's, the extensive additional research through animal bioassays, epidemiology studies of populations exposed to relatively high levels of asbestos in water, and further research on migration of fibers through the gastrointestinal tract, all confirm the earlier conclusions that ingested asbestos poses no health risk to humans at levels found in U.S. drinking water supplies.

A. The Recent State-of-the-Art Animal Studies Find No Adverse Effects in Asbestos Ingestion.

Completion of state-of-the-art rat and hamster lifetime bioassays within the past four years, and the absence of any negative health effects in each study, have led scientific

reviewers uniformly to conclude that there is no meaningful evidence of any asbestos ingestion risk to animals. The following review of the significant animal bioassays demonstrates the definitiveness with which such conclusions can be reached.

As part of the comprehensive Government program to study asbestos ingestion, a subcommittee of the Department of Health, Education and Welfare's Committee to Coordinate Toxicology and Related Programs prepared and widely distributed a draft research protocol to select the appropriate animal species and dose levels to maximize the potential for detecting any possible carcinogenic effects of asbestos ingestion. It then held a public meeting to obtain further comment.

Based on this comprehensive planning, the Government initiated lifetime, including in utero exposure, chronic bioassays of various forms of asbestos in male and female rats and hamsters as part of the National Toxicology Program (NTP). The bioassays also included co-administration of known intestinal carcinogens to determine if asbestos had cancer promoting properties. The asbestos used in these NTP bioassays was "the same asbestos one would find in the environment," and indeed, "[i]f anything," would have been expected to be more carcinogenic, as "there was a higher percentage of longer fibers in the feeding studies' materials, than would be found in any drinking water samples."28/

28/ Comments of Drs. McConnell and Millette of NIEHS and EPA, respectively, EPA Workshop Tr., supra note 10, at 117.

In the first of the NTP bioassays, NIEHS administered 1% amosite asbestos in pelletized diets to 252 male and 254 female Golden Syrian hamsters for their lifetime, as well as to the dams from which these animals were derived. No adverse effects were found in body weight gain or survival. NIEHS concluded:[29/]

> Neither of the amosite groups [male or female] showed an increased rate of neoplasia in any organ or tissue compared to the control groups. Under the conditions of this bioassay, the ingestion of amosite was not carcinogenic . . .

Similar negative results were reported from NIEHS's lifetime bioassay of chrysotile asbestos in Syrian Golden hamsters.[30/] Both short range and intermediate range chrysotile fibers were fed to groups of 125 to 254 hamsters and their mothers at 1% dosages in pelletized diets. Again, there were no adverse effects on body weight gain or survival, and NIEHS concluded that both lengths of chrysotile "were not carcinogenic when ingested by male and female Syrian golden hamsters."[31/]

29/ McConnell, E.E., et al., "Chronic Effects of Dietary Exposure to Amosite and Chrysotile Asbestos in Syrian Golden Hamsters," EPA Workshop Tr., supra note 10, at 20.

30/ National Toxicology Program, "Carcinogenesis Bioassay of Chrysotile Asbestos in Syrian Golden Hamsters," NTP Draft Report (June 23, 1981); McConnell, supra note 29, at 41.

31/ Id. at 41. NIEHS also concluded no significance should be attributed to the increased adrenal cortical adenomas in the male and female intermediate range chrysotile groups as

(Footnote 31 continued on next page.)

Asbestos administered to hamsters in drinking water by Smith, et al., has also been found not to be carcinogenic. Amosite and taconite tailings were administered at doses of 0.5, 5 and 50 mg/liter to groups of 60 hamsters.[32/] No malignant tumors were found in the highest dose amosite group, and 3, 1, 1 and 1, tumors were found, respectively, in the middle and low amosite groups, low dose taconite group, and controls. Dr. Smith, who has conducted a number of studies of asbestos in rodents, concluded the study "does not indicate that ingestion of . . . UICC amosite caused an excess of tumors, [but] the findings do show that hamsters of the strain used are capable of developing a variety of tumors, and were therefore suitable for testing for carcinogenicity . . .."[33/]

Several recent large bioassays, including additional bioassays conducted as part of the National Toxicology

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those tumors' incidence was not significantly different from the temporal controls. Id. at 37.

In this bioassay, hamsters were also administered both chrysotile and 1, 2-dimethylhydrazine dihydrochloride (DMH, a known animal intestinal carcinogen) to test asbestos cancer promoting properties. No conclusions could be drawn as none of the groups had any increased intestinal neoplasia. Id. at 39.

32/ Smith, W.E., et al., "Health of Experimental Animals Drinking Water With and Without Amosite Asbestos and Other Mineral Particles," 3 J. Environ. Pathol. & Toxicol. 277, 282 (1980).

33/ Id. at 296.

Program, have also found no evidence of carcinogenicity when asbestos was ingested by rats.

In the NTP rat bioassay, blocky (non-fibrous) tremolite and amosite alone and in combination with the known gastrointestinal carcinogen 1,2-dimethylhydrazine dihydrochloride (DMH) were administered to mothers and groups of 100 to 250 Fischer 344 rats at 1% dosages in pelletized diets. One group received chrysotile asbestos by gavage during lactation. No adverse effects on survival were found for any of the asbestos groups, and no toxic or neoplastic effects were found in any of the amosite groups in the target gastrointestinal organs. Further, no enhanced or protective effect was found from amosite in the rats also administered DMH. Thus, as chief NIEHS investigator McConnell reported:[34/]

> [A]mosite asbestos did not adversely affect the gastrointestinal tract [in terms of either neoplastic or non-neoplastic lesions] of either male or female F344 rats.

The NIEHS results with 1% crocidolite pelletized diets in Fischer 344 rats were similar. Groups of 118 male and female rats were fed control diets, and 250 male and female

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34/ McConnell, E.E., et al., "Chronic Effects of Dietary Exposure to Amosite and Tremolite in Fischer 344 Rats," EPA Workshop Tr., supra note 10, at 60, 63-64, 78-84, 85-89. In addition, no toxicity or increase in neoplasia was found in the tremolite exposed groups, and the finding of increased C-cell carcinomas in the amosite groups was concluded by the authors not to be biologically significant.

rats were on the treated diet through their lifetimes, as were their dams prior to and following birth. Food consumption and survival were comparable in all groups, and no overt toxicity was observed in the crocidolite-fed animals. The only increased incidence of tumors in treated groups were thyroid C-cell tumors, and because concurrent control incidences were low relative to control rates in other recent NIEHS rat studies, the authors concluded these increases were not "biologically significant." NIEHS thus concluded that "[u]nder the conditions of this study, crocidolite asbestos . . .did not cause a carcinogenic response."[35/]

Groups of 240 rats were fed 10% chrysotile asbestos or cellulose fiber in the diet for 32 months by Donham, et al. No carcinogenic effects of asbestos, even with these very high doses, were found. Four, two and three colon cancers were found in the chrysotile, cellulose, and control groups, respectively (with a control group half the size of the treated groups).[36/]

35/ National Toxicology Program, "Carcinogenesis Bioassay of Crocidolite Asbestos in Fischer 344/N Rats (Feed Study)," NTP Draft Technical Report, 1 (1983).

36/ Donham, K.J., et al., "The Effects of Long-Term Ingestion of Asbestos on the Colon of F344 Rats," 45 Cancer Supp. 1073 (March 1980).

Although the authors suggested that the overall results constituted "suggestive evidence" of colon carcinogenesis, id. at 1080, other reviewers of their data have labeled the results "equivocal," particularly in light of the absence of any statistical significance in the tumor comparison.

(Footnote 36 continued on next page.)

No tumors were found by Ward, et al., in groups of 21 rats administered either amosite or chrysotile intragastrically for 34 weeks. Azoxymethane (a known intestinal animal carcinogen) and amosite or chrysotile administration produced fewer tumors than azoxymethane administration alone. In a second experiment, conducted for the rats' lifetime, no significant differences were found between amosite-and-azoxymethane and azoxymethane-only groups (15 small intestine and 29 colon tumors in the asbestos group and 12 small intestine and 27 colon tumors in the amosite-plus group).37/

(Footnote 36 continued from previous page.)

McConnell, EPA Workshop Tr., supra notes 28, 34, at 38, 90. Commins, supra note 13 at 45, concludes "[t]he study must be regarded as negative in terms of any carcinogenic properties," and notes further that the one mesothelioma in the chrysotile group "could have been caused by intracheal introduction of asbestos into the lungs rather than via the gastro-intestinal tract."

37/ Ward, J.M., et al., "Ingested Asbestos and Intestinal Carcinogenesis in F344 Rats," 3 J. Environ. Pathol. & Toxicol. 301 (1980).

Although Ward, et al., attributed significance to the 16 colon tumors in the 49 rats administered saline plus amosite, reviewers of their data disagree. McConnell of NIEHS, for example, EPA Workshop Tr., supra notes 28, 34, at 39, 92, finds these data should "be viewed with some suspicion," as there were no concurrent controls and because it appears these chrysotile rats were "inadvertently exposed to AOM" [azoxymethane, the known potent animal carcinogen with which other groups were being treated at the same time.] EPA's Dr. Lyman Condie agreed that "[o]ne has reason to doubt the authors' conclusions." Condie, L. W., "Review of Published Studies of Orally Administered Asbestos," EPA Workshop Tr., supra note 10, at 9.

No gastrointestinal cancers were detected by Hilding, et al., in any group (of 20 to 30) rats administered water containing 1, 100, 5,000, and 100,000 million fibers/liter, or 20 or 300 mg/day, chrysotile/amosite and amosite, respectively. Between 1 and 7 malignant tumors of other organs were found in various groups, but no significance was attributed to these scattered tumors, especially since the highest tumor finding was in the lower of the two amosite dosage groups, and the lowest tumor finding in the highest amosite dosage group.38/

Finally, no excess gastrointestinal cancers were found by Bolton, et al., in groups of 22 to 24 rats fed 250 mg/week amosite, crocidolite or chrysotile mixed with margarine. Nor were any gastrointestinal mucousal abnormalities or other adverse effects found in the treated groups, and scanning electron micrscopy did not detect any gastrointestinal penetration.39/

These recent state-of-the-art chronic animal bioassays demonstrate the absence of any carcinogenic effect from asbestos ingestion. They confirm the numerous previous,

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38/ Hilding, A.C., et al., "Biological Effects of Ingested Amosite Asbestos, Taconite Tailings, Diatomaceous Earth and Lake Superior Water in Rats," 36 Arch. Environ. Health 298 (1981).

39/ Bolton, R.E., et al., "The Pathological Effects of Prolonged Asbestos Ingestion in Rats," 29 Environ. Res. 134 (1982).

smaller animal studies[40] and show the absence of any significance in the few purportedly positive studies.[41]

This large animal data base demonstrates that ingested asbestos is not an animal carcinogen. As one recent reviewer concluded:[42]

> The overall evaluation of all these ingestion studies (involving practically 10,000 animals in all), suggests that the evidence for ingested asbestos being carcinogenic to animals is completely negative. This conclusion is reinforced

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40/ Bonser, G.M. and Clayson, D.B., "Feeding of Blue Asbestos to Rats," in 45th Annual Report of the British Empire Cancer Campaign For Research, 242 (1967) (crocidolite fed in diet at 0.15%); Gross, P., et al., "Ingested Mineral Fibers," 29 Arch. Environ. Health 341 (1974) (rat administration of 5% chrysotile and 5 or 1 mg/week crocidolite in the diet); Cunningham, H.M., et al., "Chronic Effects of Ingested Asbestos in Rats," 6 Arch. Environ. Contam. Toxicol. 507 (1977) (chrysotile administered as 1% of the diet); Wagner, J.C., et al., "Animal Experiments with Talc" in Inhaled Particles IV, W.H. Walton, ed., 647 (1977) (100 mg/day chrysotile fed to rats); Webster, I., "The Ingestion of Asbestos Fibers," 9 Environ. Health Perspect. 199 (1974) (baboons administered asbestos for up to 5 years); and Smith, W.E., "Asbestos, Talc and Nitrites in Relation to Gastric Cancer," 34 Am. Ind. Hyg. Ass'n. J. 227 (1973) (lifetime administration of 1% chrysotile or amosite diets to hamsters).

41/ See, e.g., Gibel, W., et al., "Tierexperimentelle Undersuchungen uber eine Kanserogene Wirkung von Asbesfiltermaterial nach oraler Aufnahme" (Experimental Study of Carcinogenic Activity of Asbestos-Filter Material Following Ingestion) Arch. 46 Geschwulstforsch Heft 437 (1976).

42/ Commins, supra note 13, at 47 (emphasis added).

As Dr. Commins further notes, at 6-7, a 1% chrysotile diet (as was administered or exceeded in many of the animal studies) would equate to human drinking water exposures one billion times more contaminated than even unusually highly contaminated sources with more than 1 million fibers/liter.

> by the fact that numerous studies were carried out (at least a dozen) and massive doses were administered in some experiments.

In the words of Dr. Lyman Condie of EPA's Health Effects Research Laboratory:[43/]

> The bulk of the experimental evidence indicates that the long-term, high-level ingestion exposure to various types of asbestos fibers failed to produce any definite, reproducible, organ-specific carcinogenic effect.

And of Dr. Cotruvo in summarizing the 1982 EPA Workshop:[44/]

> The results of animal feeding studies as presented, including the National Toxicology Program study, indicate essentially that no toxicity was demonstrated in whole animal lifetime exposures.

This animal evidence is particularly relevant to human health effects given the finding in numerous other animal studies that asbestos inhalation effects in humans also occur in animals -- indicating the appropriateness of animal studies to determining potential asbestos human effects. Both lung cancers and mesotheliomas have been produced in rats (including rats of the same strain as those in the NIEHS feeding studies) and hamster inhalation studies,[45/] thus

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43/ Condie, supra note 37, at 9-10 (emphasis added).

44/ Cotruvo, supra note 11, at 609 (emphasis added).

45/ McConnell comments, EPA Workshop Tr., supra note 10, at 115, 125; Condie comments, id. at 111. Notably, however, increases in gastrointestinal cancer were not found in these inhalation studies. Wagner, J.C., et al., "The Effects of the Inhalation of Asbestos in Rats," 29 Br. J. Cancer 252 (1974); Davis, J. M. G., et al., "Mass and Number of Fibres in the Pathogenesis of Asbestos-Related Lung Disease in Rats," 37 Br. J. Cancer 673 (1978).

leading Dr. Condie to "expect to be able to produce a neoplastic response within the lifetime of conventional laboratory animals with massive doses of ingested asbestos such as those employed in some of the studies."[46/] This expectation was unfulfilled.

In sum, all recent bioassays and reviews thereof have agreed that the animal ingestion studies are negative and provide no evidence for concern about potential human health effects of asbestos in water.[47/]

46/ Condie, supra note 37, at 10; see also McConnell comment, EPA Workshop Tr., supra note 10, at 115; and Commins, supra note 13, at 43 (the animal inhalation results "strongly support[] the thesis that animal experimentation with asbestos can be a good model for human exposure"). Dr. David Coffin, Senior Science Advisor of EPA's RTP Health Effects Research Laboratory, agrees: "I think that, by analogy, these rats probably live long enough to develop a tumor of such organs as the kidney, colon, etc., as might be expected from the GI tract port of entry." EPA Workshop Tr., supra note 10, at 122.

47/ See also, National Academy of Sciences, Safe Drinking Water Committee, Drinking Water and Health, Vol. IV, "Asbestos," 223, 248 (1983); Meyer, E., "Investigations About the Presence of Asbestos Fibres in Drinking Water in the Federal Republic of Germany and Evaluation of the Results in Respect of Potential Health Hazards," 2 G.W.F.-Wasser/Ab Wasser 85-96 (1982); Aurand, supra note 13, at 2.1 ("[a]nimal experiments about asbestos ingestion have not in any way shown carcinogenic effects"); "Chronic Hazard Advisory Panel on Asbestos: Report to the U.S. Consumer Product Safety Commission," II-51 (July 1983) (terming the animal results "inconsistent," but noting that "recent tests conducted as part of the National Toxicology Program have given consistently negative results"); Toft., P., and M.E. Meek (of the Department of National Health and Welfare, Canada), "Asbestos in Drinking Water -- A Canadian View," EPA Workshop Tr., supra note 10, at 591, 599-600 ("there is no conclusive evidence that ingested asbestos is carcinogenic or cocarcinogenic in animal species").

B. The Many Epidemiology Studies of Asbestos Ingestion Also Typically Find No Adverse Effects.

Paralleling the negative animal ingestion studies are numerous epidemiology studies of populations exposed to much higher than typical concentrations of asbestos in drinking water that have found no evidence of increased cancer risk. Only one of the many studies has been interpreted to suggest any cancer risk, and that study is characterized by its authors as of limited significance for drawing conclusions about human ingestion risks. To be sure, each of the epidemiology studies is of limited sensitivity to detect small increases in risk; but the absence of evidence of increased risk in numerous studies provides more than ample data upon which EPA can conclude that the epidemiology data do not indicate any adverse health effects are to be anticipated from asbestos drinking water ingestion.

Asbestos ingestion concerns first focused in the Duluth, Minnesota area in the early 1970's. The earliest epidemiology studies, since updated, thus sought to determine whether increased cancer risks could be found among the Duluth population, which had been consuming Lake Superior water with concentrations of 1 to 65 million fibers/liter since the late 1950's. The studies of Duluth residents,[48/] employing various

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48/ Mason, T.J., et al., "Asbestos-Like Fibers in Duluth Water Supply: Relation to Cancer Mortality," 228(8) JAMA 1019

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methods of collecting and analyzing the data, reached consistent conclusions of absence of health effects. The 1974 Mason study noted that asbestos ingestion would be expected to show a greater excess of cancer in the esophagus and stomach than in the rectum, but "this did not occur;"[49/] the 1976 Levy study comparing Duluth and St. Paul residents found "no consistent pattern of statistically significant rate differences for any GI cancer;"[50/] and a 1981 Sigurdson update concluded:[51/]

> [T]here are currently no observed etiologic or causal associations between exposures to amphibole fibers in the Duluth drinking water supply and the development of cancer.

Successive studies were also conducted of residents of Connecticut comparing cancer rates in areas that had and had not extensively used A/C pipe.[52/] EPA participated in both

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(1974); Levy, B. S., et al., "Investigating Possible Effects of Asbestos in City Water Surveillance of Gastrointestinal Cancer Incidence in Duluth, Minnesota," 31 Am. J. Epidem. 362 (1976); Sigurdson, E.E., "Interim Report of Cancer Incidence in Duluth During 1969-1976 as an Investigation of Health Effects of Amphibole Fibers in the Municipal Water Supply," Minnesota Department of Health (1981); and Sigurdson, E.E., "Observations of Cancer Incidence Surveillance in Duluth, Minnesota, "EPA Workshop Tr., supra note 10, at 184.

49/ Mason, supra note 48, at 1020.

50/ Levy, supra note 48, at 366.

51/ Sigurdson 1981, supra note 48, at 11.

52/ Harrington, J.M., et al., "An Investigation of the Use of Asbestos Cement Pipe for Public Water Supply and the

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studies. The 1978 Harrington study "detected no changes in incidence rates or patterns in Connecticut for cancers of the stomach, colon, or rectum over the period 1935-1973, that could be construed as related to the introduction around 1950 of A/C pipes . . ."[53/] The 1980 Meigs study, using more sensitive and refined techniques, found the same absence of any pattern of increased cancer in towns using A/C pipes (with asbestos concentrations up to 700,000 fibers/liter) and concluded that "the lack of coherent evidence for cancer risks from use of A/C pipe is reassuring" and demonstrates the absence of any reason to change "current water distribution policies."[54/]

Successive studies of residents of areas with high natural asbestos concentrations in the Province of Quebec have similarly found no increased cancer risks. Scientists from the Canadian Health and Welfare Department analyzed cancer mortality in populations exposed to concentrations as high as 1.3 billion fibers/liter. The 1977 Wigle study of

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Incidence of Gastrointestinal Cancer in Connecticut, 1935-1973," 107(2) Am. J. Epidem. 96 (1978); Meigs, J. W., et al., "Asbestos Cement Pipe and Cancer in Connecticut 1955-1974," 42 J. Environ. Health 187 (1980); and Meigs, J.W., "An Assessment of Studies on Cancer Risks from Asbestos in Connecticut Drinking Water," EPA Workshop Tr., supra note 10, at 334.

53/ Harrington, supra note 52, at 101.

54/ Meigs 1980, supra note 52, at 191.

22 Quebec municipalities (grouped as known high, possible high, and probable low exposures) "did not reveal excess cancer mortality that could be related to the presence of asbestos fibers in drinking water supplies."[55/] The 1980 Toft and Wigle study again found, this time for two groups of localities with high (greater than 100 million fibers/liter) and low (less than 5 million fibers/liter) asbestos concentrations in their water supply "no consistent increase of mortality rates for any cancer."[56/]

The same absence of any indication of increased cancer risks from natural asbestos ingestion with drinking water concentrations of 37 to 556 million fibers/liter has been found in studies of residents of the Puget Sound, Washington, area. The first, an ecologic study employing techniques similar to those previously used in Duluth, Connecticut and Quebec, once again found "little evidence that asbestos in community water supplies has altered the risk of any cancer."[57/]

55/ Wigle, D.T., et al., "Cancer Mortality in Relation to Asbestos in Municipal Water Supplies," 32 Arch. Environ. Health 185, 189 (1977).

56/ Toft, P., and D.T. Wigle, et al., "Asbestos and Drinking Water in Canada," 18 The Science of the Total Environment 77 (1981).

In both Canadian studies, some excess cancer risks (lung and stomach) were found among males, but the authors attributed these results to asbestos inhalation exposures that would have been experienced by the many asbestos miners and millers living in these localities.

57/ Polissar, L., et al., "Cancer Incidence in Relation to Asbestos in Drinking Water in the Puget Sound Region," 116(2) Am. J. Epidem. 314 (1982).

The second Puget Sound study employed more sensitive case-control techniques comparing exposures of 382 cases with various cancers to 462 controls chosen from the general population. The authors found "no convincing evidence for increased cancer risk from imbibed asbestos. Confidence intervals for relative risks from almost all sites included unity." The only significantly elevated risks were for male stomach and pharyngenal cancers and these "sex-inconsistent results" were attributed by the authors to "other factors" than asbestos.[58/] Although noting the limited sensitivity of the study (an 80% probability of detecting at the 5% significance level relative risks less than two), the authors also noted the strengths of the study including collection of individualized exposure data, the high asbestos levels for 60 years in the community studied, and the high participation rates of the selected cases and controls.[59/]

EPA has itself also conducted an epidemiologic study of residents of Escambia County Florida where A/C pipe has been used for 30 or more years and asbestos concentration levels up to 32.7 million fibers/liter have been recorded.[60/]

58/ Polissar, L., et al., "Cancer Risk from Asbestos in Drinking Water: Summary of a Case-Control Study in Western Washington," EPA Workshop Tr., supra note 10, at 170, 173.

59/ Id. at 180.

60/ Millette, J.R., et al., "An Epidemiologic Study of the Use of Asbestos-Cement Pipe for the Distribution of Drinking Water in Escambia County, Florida," EPA Workshop Tr., supra note 10, at 278.

Based on comparisons between census tracts that had and had not used A/C pipe for 25 years, the Agency concluded, "results of the analyses in this study do not show any statistical association between the deaths due to cancer types and the use of A/C pipe."

The only epidemiology study ever even to suggest ingestion risks was that conducted in the San Francisco Bay area, where asbestos concentrations from natural sources of up to 36 million fibers/liter have been found.[61/] An indirect ecological study, like most of the other asbestos ingestion epidemiology studies, the Kanarek study found some statistical correlations between esophageal, stomach and pancreatic cancers and residence in census tracts with high asbestos fiber drinking water counts. EPA and the study's authors have, however, repeatedly emphasized that this ecologic study "can only specify associations, and cannot pinpoint

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61/ Kanarek, M.S., et al., "Asbestos in Drinking Water and Cancer Incidence in the San Francisco Bay Area," 112 Am. J. Epidem. 54 (1980); Conforti, P. M., et al.; "Asbestos in Drinking Water and Cancer in the San Francisco Bay Area: 1969-1974 Incidence," 34 J. Chronic Dis. 211 (1981); Conforti, P. M., "Effect of Population Density on the Results of the Study of Water Supplies in Five California Counties," EPA Workshop Tr., supra note 10, at 205; Tarter, M.E., et al., "A Graphical Analysis of the Interrelationships Between Waterborne Asbestos, Digestive System Cancer, and Population Density," EPA Workshop Tr., supra note 10, at 236; and Kanarek, M.S., "Commentary: The San Francisco Bay Epidemiology Studies on Asbestos in Drinking Water and Cancer Incidence: Relationship to Studies in Other Locations and Pointers for Further Research," EPA Workshop Tr., supra note 10, at 329.

definite causation,"[62/] "do[es] not prove that there is a link between Bay Area water and cancer,"[63/] is based on "underlying assumptions of the method [that] make definitive conclusions untenable," and presents "findings [that] in no way lend themselves to the interpretation regarding the possible regulation of asbestos in drinking water."[64/]

62/ Kanarek, M.S., "Asbestos in Drinking Water and Cancer Incidence," at 155, Doctoral Dissertation, Univ. of California (Berkeley) (1978).

As Dr. Abraham Ibrahim, Chairman of the Epidemiology Department at the University of North Carolina, has noted in his review of various types of epidemiology studies: "[E]cologic stud[ies] must be interpreted with great caution and used only to generate hypotheses since the exposure information is so far removed from its potential effect." Direct Testimony before EPA in Suspension Hearings on 2,4,5-T, Docket Nos. 415, et al., at 10 (Nov. 4, 1980).

63/ Environmental Protection Agency, "EPA Position on Study Entitled 'Asbestos in Drinking Water and Cancer Incidence'" (1978).

64/ Conforti, EPA Workshop Tr., supra note 61, at 219, 220.

Reviewers of the Kanarek study had questioned whether the failure to control for population density was responsible for the various increased cancer rates. The Conforti and Tartar papers presented at the Workshop, supra note 61, sought to answer these criticisms. As noted by Dr. Robert C. Cooper, "Commentary: Comments on the California Studies," EPA Workshop Tr., supra note 10, at 340, 343, although the authors felt these new analyses had answered the population density question, "in the process of this [ ] analysis, it was seen that the data from San Francisco City and County per se has a major effect upon the correlation that is not related to population density . . . We are now attempting to describe this phenomenon in more detail and to determine if it is independent of asbestos dose." Dr. Meigs, the author of one of the Connecticut studies, also noted "the [] [new] suggested possible nonasbestos-related explanations for the original findings" of the Kanarek study. Meigs (1982), supra note 52, at 338.

(Footnote 64 continued on next page.)

In sum, epidemiologic studies in five different areas with relatively high asbestos concentrations in water supplies for decades do not provide any significant evidence of a cancer risk.[65/] Some commentators have been unwilling to conclude definitively that ingested asbestos is not a human carcinogen on the basis of these studies because of the possibility that none of the studies was sensitive enough to detect slight increased cancer risks.[66/] However, the

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Although attention has been focused on the possible confounding effect of population density, as Dr. Kenny S. Crump, "Review of a Study of Asbestos in Drinking Water and Cancer in the San Francisco Bay Area" (1982), has noted, the study was also unable to control a large number of other potential confounding factors, any of which could account for the associations found: "smoking, eating, and other personal habits; occupational exposures to carcinogens; and exposures to other environmental pollutants, such as organic contaminants in drinking water" (at 9-13, 24). As Dr. Crump further noted, the "associations found by Kanarek et al. were small by traditional epidemiologic standards and therefore particularly sensitive to confounding effects" (at 8). In addition, Dr. Crump has identified a number of unexplained internal inconsistencies in the results presented by Kanarek and Conforti (at 13-18).

65/ In addition, an ecologic survey comparing cancer incidence in U.S. counties with and without known natural deposits of asbestos also found no difference in cancer incidence between the two groups of counties. Fears, T. R., "Cancer Mortality and Asbestos Deposits," 104 Am. J. Epidem. 523 (1976).

66/ See, e.g., L. Polissar comment, EPA Workshop Tr., supra note 10, at 378 ("even if there are risks, they are probably relatively small"); and Marsh, G.M., "A Critical Review of

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studies do clearly demonstrate that no significant risk exists from prevailing asbestos levels in the U.S. drinking water supply. The studies further demonstrate -- especially when combined with the animal evidence where massive doses were ingested and sensitivity was great[67/] -- that it would

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Epidemiologic Studies Related to Ingested Asbestos," EPA Workshop Tr., supra note 10, at 144 (Despite an evident bias -- note his concluding comment that case-control studies "are now needed to firmly establish risk levels to ingested asbestos" (at 160) -- Marsh concludes, (at 147, emphasis added) merely that, despite an absence of any consistent pattern of tumors in male and females in the 13 studies reviewed, some of the scattered positive associations "may have a biological basis related to ingested asbestos").

Some reviewers, however, have reached definitive conclusions. The Committee on Occupational Medical Practice of the Journal of Occupational Medicine recently wrote:

> Epidemiological studies conducted in Minnesota and Canada have permitted the conclusion that no cancer risk is associated with asbestos fibers in water. . . . [O]ur Committee believes that at this time there is little evidence to support a health risk from asbestos fibers in drinking water.

"Occupational Medical Forum," J. W. Mitchell, ed., 25 J. Occup. Med. 361 (1983).

67/ The importance of interpreting the epidemiology studies in context with the animal ingestion evidence was stressed by Dr. Crump, supra note 64, at 19-20. He calculates that if one took the highest relative risk found in the Kanarek study (1.4 for stomach cancers) and projected a conservative linear dose-response relationship based on the highest fiber concentration found in the San Francisco Bay area (36 million fibers/liter), one would predict that the relative risk ratio in the NTP hamster study should have been 32 million. Thus, as Dr. Crump concludes:

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be unreasonable to conclude that ingested asbestos is likely to cause adverse human health effects.68/

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> [I]f the risk ratios observed by Kanarek et al. are truly due to waterborne asbestos fibers, and the hamster susceptibilities are at all comparable to human susceptibilities, all of the hamsters should have died many times over from cancer. Since they did not, unless hamsters are a totally inadequate model for humans, it is highly unlikely that the low levels of asbestos fibers in SF-O SMSA water caused the increased cancer incidences observed by Kanarek et al.

Linda S. Erdreich of EPA's Environmental Criteria and Assessment Office has made the same point. "Comparing Epidemiologic Studies of Ingested Asbestos for Use in Risk Assessment," EPA Workshop Tr., supra note 10, at 306. Noting first that previous attempts, such as the EPA Ambient Water Quality Criterion for Asbestos, quantified asbestos ingestion risks from inhalation studies, Erdreich asserts that "data on the health effects of human exposure via ingestion are necessary to verify this route extrapolation" (at 310, emphasis added). Erdreich then reviews the epidemiology studies and finds they "suggest that the risk is not greater than estimated from the inhalation studies and may be less" (at 320, emphasis added). She finds that more sensitive epidemiologic studies would be needed to quantify how much less is the ingestion risk, but concludes: "If data from animal studies clearly did not support the association between cancer and ingested asbestos [as the many reviewers at the Workshop concluded they did not], the need for human studies might be circumvented." Id.

68/ Dr. Cotruvo of the Office of Drinking Water, EPA Workshop Tr., supra note 11, at 610, has reached the same conclusion:

> It can be said, then, that the epidemiologic evidence of risk from ingestion of water containing asbestos fiber is not convincing, and that, in view of the

(Footnote 68 continued on next page.)

C. Findings of Gastrointestinal Cancer in Some Cohort Studies of Heavily Inhalation-Exposed Asbestos Workers Do Not Suggest Adverse Effects Should Be Expected from Ingested Asbestos.

The search for additional data on ingested asbestos risks was initiated following increased gastrointestinal risk findings in some cohorts of heavily inhalation-exposed asbestos workers. As was recognized then, when recommendations for ingestion research were made, and as most commentators recognize now, the inhalation risks can serve as no more than suggestive evidence of a need for more research. Inhalation findings did not then, and do not now, provide any scientific basis for predicting adverse effects from asbestos ingestion. Nonetheless, some have suggested

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> lack of confirmation by animal studies, the existence of a risk has not been satisfactorily demonstrated.

As has Dr. Commins, supra note 13, at 59 (emphasis added):

> There appears to be no firm evidence for adverse health effects in human populations, drinking even high levels of asbestos in drinking water over a long time scale. The separate findings from each of these [animal bioassay, gut penetration, and epidemiology] studies taken separately would suggest that there is no significant evidence for any health effects from ingesting asbestos. However the combination of all three study approaches indicates that there appears to be no adverse effects in the normal population who drink water containing asbestos.

drinking water standards might be set based on the cohort inhalation data. Given these suggestions, it is useful to review their tenuousness.

1. Conflicting evidence and divergent views in the scientific community exist as to whether heavy exposures to inhaled asbestos cause gastrointestinal cancer.

The foundation for suggestions that drinking water standards might be set based on cohort inhalation studies is itself open to considerable scientific dispute. Although increased gastrointestinal cancers have been found in some heavily exposed worker cohorts, such findings are by no means consistent. A substantial body of scientific opinion holds that an association between inhaled asbestos and gastrointestinal cancers has not been demonstrated.

The relationship of gastrointestinal cancers and inhaled asbestos was most recently and comprehensively assessed by the Advisory Panel on Asbestos to the Consumer Product Safety Commission. The Panel explicitly noted that it could not reach agreement on whether a gastrointestinal cancer/asbestos inhalation association existed.[69/]

69/ CPSC Panel, supra note 47, at I-2. The expert panel, chaired by Norton Nelson of New York University Medical Center, also included William J. Nicholson of Mount Sinai School of Medicine, Janet M. Hughes of Tulane University Medical Center, Nancy K. Kim of the New York State Department of Health, Julian Peto of Oxford University, Marvin A. Schneiderman of Clement Associates (who also served on the 1974 AWWA panel, supra note 1), and Carl M. Shy of the University of North Carolina.

Reviewing numerous worker cohort studies, in each of which very substantial asbestos exposures occured under conditions prevailing decades ago, the CPSC panel found statistically significant increases for esophagus, stomach, colon and rectum cancers in only 8 of the 28 studies (with 2 of the increases in the 8 studies sensitive enough to detect 50% increases in relative risk and 6 of the increases in the other 20 studies).[70/] The Panel noted that regional and ethnic differences (such as the high proportion of persons of Slavic origin among the New York and New Jersey insulators in some of the cohorts) could account for the increased risks in some of the studies.[71/] Further, the Panel emphasized that a dose-response relationship -- one of the essential elements for reaching determinations of causation -- [72/] had not been demonstrated in any of the studies.[73/] Thus, the Panel's report concluded: "For these reasons, some members of the Panel believe the causal relationship of gastro-intestinal cancer with asbestos exposure has yet to be resolved."[74/]

70/ Id. at II-60 to II-61.

71/ Id. at II-62.

72/ Hill, A.B., Principles of Medical Statistics 309-23 (9th Ed. 1971).

73/ CPSC Panel, supra note 47, at II-64.

74/ Id. The absence of sufficient evidence to demonstrate a gastrointestinal cancer/asbestos inhalation association

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2. Even if inhaled asbestos is assumed to cause gastrointestinal cancer, the cohort studies do not demonstrate that ingested asbestos is the cause of such disease.

Those who would project ingested asbestos risks from the inhalation studies further assume that the risks in such studies were due to asbestos fibers that were ingested upon elimination from the lungs.[75/] Such an assumption is contrary to what evidence exists as to the nature of any conceivable gastrointestinal cancer risk to heavily inhalation-exposed asbestos workers. As EPA's Dr. Lyman Condie has noted, the overwhelming negative findings in the animal ingestion studies "cast some doubt on the hypothesis that peritoneal mesotheliomas and gastrointestinal cancers result from the ingestion of asbestos fibers cleared from the lungs following inhalation exposure."[76/]

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has also been noted by Craighead, J.E., and B.T. Mossman, in their comprehensive review, "The Pathogenesis of Asbestos-Associated Diseases," 306 New Eng. J. Med. 1446 (1982); in an Editorial, "Asbestos and Health," The Lancet (Oct. 27, 1979); and by the International Labor Organization in its just adopted "Code of Practice on Safe Use of Asbestos," Part A-6.3 (Nov. 16, 1983) ("Other types of cancer (e.g. of the gastrointestinal tract) have been attributed to [airborne] asbestos exposure though the evidence at present is inconclusive.")

75/ See NAS 1983, supra note 47, at 233-34.

76/ Condie, supra note 37, at 11.

As noted previously, one of the areas where a need for further research was identified in the mid-1970's was the issue of whether ingested asbestos fibers penetrate the gastrointestinal mucousa, have some opportunity to reside in tissue, and thus may pose any cancer risk to the gut. Considerable additional research completed within the past decade was summarized at the EPA Workshop by EPA's Dr. Philip M. Cook. Dr. Cook first noted that "movement of a large number of fibers [through the mucousa] is [likely] a necessary precursor for carcinogenesis following ingestion of asbestos."77/ Paying especially close attention to experimental and analytic limitations of each of the studies, Dr. Cook then noted that it would be impossible to prove no penetration occurred. He, nonetheless, concluded that the many studies "indicate the involvement of a very small fraction of ingested fibers in penetration and consequently low probability for significant tissue accumulations and increased risk of cancer."78/

Two other preliminary studies presented at the EPA Workshop provide additional data indicating the unlikelihood

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77/ Cook, P.M., "Review of Published Studies on Gut Penetration by Ingested Asbestos Fibers," EPA Workshop Tr., supra note 10, at 406, 410 (emphasis added).

78/ Id. at 423. Dr. Commins, supra note 13, at 6, 48, finds from review of the gut penetration studies that:

> If in fact fibres penetrate the gut wall, the event would seem to be very rare, perhaps only one fibre in every 10,000 or even perhaps one in 100,000.

that migration of ingested asbestos fibers would lead to gastrointestinal cancer. Boatman, et al., from the University of Washington, under an EPA grant, reported finding no differences between urine asbestos concentrations in persons with asbestos levels in their drinking water 100 times less than concentrations of Everett area residents whose tapwater had concentrations of about 200 million fibers/liter.[79/] M.E. Meek reported on studies by the Canadian Health and Welfare deprtment to determine whether migration would occur even in ulcerated intestinal tissues.[80/] No intracellular fibers were observed, leading Meek to conclude, "the gut wall of rats may present an effective barrier to the penetration of asbestos even under conditions of loss of the epithelium."[81/]

These recent conclusions that very few, if any, asbestos fibers entering the gastrointestinal tract are likely to penetrate the gastrointestinal tissue, along with the negative findings in the animal studies, thus cast serious doubt on the assumption that it is inhaled and subsequently ingested

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79/ Boatman, E.S., et al., "The Use of Quantitative Analysis of Urine to Assess Exposure to Asbestos Fibers in Drinking Water in the Puget Sound Region," EPA Workshop Tr., supra note 10, 430, 433.

80/ Meek, M.E., "Transmigration of Ingested Asbestos," EPA Workshop Tr., supra note 10, at 486; see also Meek, M. E., "An Investigation of the Penetration of Ingested Asbestos into the Normal and Abnormal Intestinal Mucosa of the Rat," 21(2) Fd. Chem. Toxicol. 193 (1983).

81/ Meek, M. E., "Transmigration of Ingested Asbestos," at 495.

asbestos that would account for any gastrointestinal cancer in the worker cohorts. More plausible is the theory that any gastrointestinal cancer increase may be due to the inhaled fibers themselves. As Dr. J.R. Goldsmith demonstrated in a recent article reviewing the cohort inhalation studies, "increases in gastrointestinal cancer are not distinguishable from increases of cancer at all nonpulmonary sites." Thus, concludes Goldstein, "it is plausible to designate asbestos as a systemic carcinogen, and to reject the inference that orally ingested asbestos is specifically linked to the observed increase in gastrointestinal cancer rates reported."82/

An additional factor that casts severe doubt on the assumption that the inhalation cohort studies can be employed to predict ingestion risks is the significant divergence between the fiber sizes in the inhalation environment and those found in drinking water. As noted previously, most water-borne fibers are less than 5 microns in length. Given the extensive animal evidence indicating that it is the

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82/ Goldsmith, J.R., "Asbestos as a Systematic Carcinogen: The Evidence from Eleven Cohorts," 3 Am. J. Industr. Med. 341, 342 (1982).

See also, Commins, supra note 13, at 41, and scientific studies cited therein ("[i]f gastro-intestinal cancer does result from industrial exposure to asbestos, it would be more likely to arise as a result of systemic translocation of certain fibres originally deposited and absorbed in the depths of the lung"); and Lee, K.P., et al., "Pulmonary Response and Transmigration of Inorganic Fibers by Inhalation Exposure," 102 Am. J. Pathol. 314 (1981).

longer fibers (especially those longer than 8 microns) that are most likely to be carcinogenic, the absence of a human health risk from ingestion is even more likely.[83/]

In sum, the inconsistent results in the cohort inhalation studies, as well as the absence of evidence of any significant pentration of gastrointestinal tissue by ingested fibers or of carcinogenicity in the animal ingestion studies, cast extreme doubt on the likelihood that the human inhalation studies have any plausible connection to prediction of adverse effects from drinking water asbestos ingestion.

D. The Recent National Academy of Sciences Attempt to Quantify Risks of Ingested Asbestos Based on the Most Tenuous of Assumptions Does Not Warrant Serious EPA Consideration.

Despite the implausibility of attempting to quantify asbestos ingestion risks on the basis of the cohort inhalation studies,[84/] this is the course followed by NAS in its

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83/ See, e.g., Stanton, M.F., et al., "Carcinogenicity of Fibrous Glass: Pleural Response in the Rat in Relation to Fiber Dimension," 58 J. NCI 587 (1977); Stanton, M.F. and C. Wrench, "Mechanisms of Mesothelioma Induction with Asbestos and Fibrous Glass," 48 J. NCI 797 (1972); Davis, J.M.G., et al., "Mass and Number of Fibres in the Pathogenesis of Asbestos-Related Lung Disease in Rats," 37 Br. J. Cancer 673 (1978); Kenny S. Crump Testimony, Ontario Royal Commission on Asbestos, Tr. Vol. XXVI at 60-66 (Aug. 1981).

84/ The previous quantitative risk assessment of ingested asbestos based on cohort inhaltion results -- EPA's Ambient Water Quality Criterion for Asbestos, 44 Fed. Reg. 56628 (Oct. 1, 1979), 45 Fed. Reg. 79318 (Nov. 28, 1980) -- was deemed "utterly useless" by Dr. Ervin Bellack, Senior Chemist in the Health Effects Branch of the EPA Office of Drinking Water. Memorandum to Joseph Cotruvo (June 6, 1979).

(Footnote 84 continued on next page.)

1983 report on drinking water contaminants. This NAS document does not, however, even purport to be a reasoned review of the asbestos ingestion evidence. More appropriately, it should be characterized as an attempt to predict risk numbers on the basis of evidence that does not suggest any risk. Nowhere does the NAS report even discuss whether the occupational inhalation risks can reasonably be said to demonstrate any ingestion risk; instead, it calculates numbers without ever considering whether the numbers deserve to be manipulated.

Adoption of the NAS recommended risk assessment would require acceptance of each of the following unwarranted assumptions:85/

(Footnote 84 continued from previous page.)

AACPP previously commented on the failure of this criteria document to reflect accurately the latest scientific knowledge. See Comments of the Asbestos Information Association/North America and Association of Asbestos/Cement Pipe Producers Association, submitted to EPA on February 15, 1980 in response to Water Quality Criteria, 44 Fed. Reg. 56628 (Oct. 1, 1979).

85/ Beyond the fundamental flaws in the NAS risk assessment discussed in the text, any attempt to rely on the NAS work would require scrutiny of numerous other assumptions in the report, most of them undocumented, including the assumptions that:

a. The ingested asbestos risk is multiplicative of, rather than additive to, underlying cancer risks from other exposures;

b. The dose-response relationship between ingested asbestos and cancer is linear;

(Footnote 85 continued on next page.)

a. The overwhelming negative animal ingestion evidence should be completely ignored.

b. Because the overwhelming negative epidemiology evidence includes no single very sensitive study, it too should be ignored.

c. All worker inhalation studies that did not find any increase in GI cancer should be ignored.

d. Only those worker inhalation studies with the highest relative risks should be employed to assess risks.

e. Risks should be assessed on the basis of studies (in four out of five cases) that collected no exposure data.

f. The absence of any dose/response information on asbestos and gastrointestinal cancers should be ignored.

EPA's own epidemiologists and experts on asbestos in water have emphasized that such manipulation of the cohort inhalation studies to determine ingestion risks is "open to question" and that use of such data to test the sensitivity of various negative epidemiology studies "should in no way imply its acceptance as a standard-setting value."[86/] Given the numerous unjustified and tenuous assumptions upon which the NAS document is based, these cautionary notes should be heeded. EPA should place no reliance on the 1983 NAS report in its SDWA proceedings.

(Footnote 85 continued from previous page.)

c. Thirty percent of all fibers inhaled in the cohort studies were subsequently swallowed; and

d. One fiber measured by light microscopy is equivalent to 50 fibers measured by transmission electron microscopy.

86/ Millette (1982), supra note 10, at 293.

IV. BOTH MONITORING AND TREATMENT OF ASBESTOS IN DRINKING WATER WOULD BE UNDULY COSTLY, TIME CONSUMING, AND DIFFICULT TO ACCOMPLISH.

Beyond the evidence confirming the absence of adverse health effects from asbestos in drinking water, consideration of whether an MCL or treatment technique is warranted under the SDWA entails an understanding of the technical and economic infeasibility of measuring and removing asbestos in drinking water. Prerequisite to proposing a maximum contaminant level (MCL) for asbestos in drinking water, EPA must determine "... it is economically and technologically feasible to ascertain the level of such contaminant in water in public water systems." The Act also contemplates treatment technique requirements where there is an inability to specify an MCL, with such requirements leading "to a reduction in the level of such contaminant sufficient to satisfy the requirements of section [1412]."[86/]

As a "primary drinking water regulation," an MCL or treatment technique must meet standards of technical feasibility. Thus, any such regulation must contain:[87/]

> criteria and procedures to assure a supply of drinking water which dependably complies with such maximum contaminant levels; including quality control and

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86/ SDWA § 1401(1)(C)(i), 42 U.S.C. § 300f(1)(C)(i).

87/ SDWA § 1401(1)(D), 42 U.S.C. § 300f(1)(D) (emphasis added).

> testing procedures to insure compliance with such levels and to insure proper operation and maintenance of the system... .

For national revised primary drinking water regulations, standards of technical and economic feasibility are clearly defined:[88/]

> ... the term "feasible" means feasible with the use of the best technology, treatment techniques, and other means, which the Administrator finds are generally available (taking cost into consideration).

The limited availability and high costs of analytical methods for measuring asbestos in drinking water raise serious questions whether such techniques are "economically and technically feasible." A single measurement technology is available -- transmission electron microscopy (TEM). TEM is costly ($150,000 to $300,000 per instrument or $300 to $600 per analysis) and time-consuming (approximately six to eight hours per sample). More significantly, the precision and accuracy of TEM measurement are quite limited. Collectively, these high costs and technological limitations do not allow the level of asbestos in drinking water to be ascertained dependably, even with considerable expense. Treatment techniques for minimizing asbestos levels in drinking water are also expensive to design, construct, operate and maintain. Even if an RMCL were warranted, no

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88/ SDWA § 1412(b)(3), 42 U.S.C. § 300g-1(b)(3) (emphasis added).

conceivable MCL nor required treatment technique for asbestos in drinking water is likely to meet the statute's standards of technical and economic feasibility.

When monitoring and treatment costs are balanced against the evidence demonstrating no adverse health effects from the ingestion of asbestos, there is no reasonable basis for EPA to set a national primary drinking water standard for asbestos.

A. Measurement of Asbestos Concentrations in Water Is Extremely Expensive and of Dubious Accuracy at Parts-per-Trillion Levels.

As previously discussed,[89/] asbestos is found in many U.S. water supplies, primarily due to natural sources, i.e., geological formations through or over which water sources pass. But, "the majority of U.S. water consumers are not exposed to concentrations of asbestos fibers above one million fibers per liter," and "[t]he majority of persons receiving water from asbestos-cement pipe distribution systems are not exposed to significant numbers of fibers from the pipe."[90/]

As the vast majority of water supplies have asbestos concentrations below 1 million fibers/liter, their weight

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89/ See pp. 13-14, supra.

90/ Millette 1979, supra note 23, at 2.

basis concentrations are below one part-per-trillion.[91] The lowest MCL's for any inorganic and organic chemicals currently regulated by national interim primary drinking water regulations, 42 C.F.R. §§ 141.11(b) and 141.12(a), are 0.002 milligrams per liter (mercury), and 0.0002 milligrams per liter (Endrin), respectively, or 2,000 and 200 parts-per-trillion -- two and three orders of magnitude greater than even rarely occurring asbestos levels of 1 million fibers/liter. Were an MCL to be considered by EPA for asbestos, therefore, the Agency would necessarily be establishing regulations at contamination levels far below those set previously for any other substance.

These low levels of asbestos in drinking water, coupled with the extremely small (micrometer to sub-micrometer) size of the fibers themselves, contributes to the difficulty of detection and measurement. These difficulties are summarized concisely in an EPA-sponsored study:[92]

> Primary reasons for the extreme difficulty in determining asbestos fiber concentrations in water include (1) asbestos fiber concentration in potable water is generally very low, (2) chemical analytical methods are not applicable because elements present are common to all rock-forming minerals, (3) asbestos fibers cannot be concentrated or separated

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91/ See p. 14 supra.

92/ DeBerry, D. W., et al., "Final Report - Corrosion in Potable Water Systems," 5-12, EPA Contract No. 68-01-5834 (February 1982).

> from other inorganic solids present in the water, and (4) fiber sizes are often below the resolution of the optical microscope.

As described next, each of these difficulties contributes to the infeasibility and unavailability of a methodology for monitoring asbestos in the nation's water supplies.

1. Sample collection and preparation processes require numerous samples and are subject to considerable error.

Extraordinary care must be taken during the collection and preparation of water samples for asbestos analysis. This process is so exacting and sensitive that one experienced scientist has likened it to a clinician obtaining "a sample of body fluid or tissue for laboratory analysis":93/

> In practice, it is to be appreciated that the sequence of manipulative operations required to accomplish this are not without errors.

The extremely small size and ubiquitous nature of asbestos makes sample collection preparation highly sensitive to contamination and interference from other substances. Special measures must be taken to prevent contamination through proper sample containment and preservation techniques:94/

93/ Boatman, E. S., "Analyzing Asbestos Fibers in Water by Means of Transmission Electron Microscopy," J. AWWA 533 (October 1982).

94/ Id.

> The difficulty associated with asbestos fiber analysis is that indigenous asbestos contaminants, unlike microbiological laboratory contaminants, cannot be eliminated by using appropriate germicides or ultraviolet radiation but remain potentially ever present.
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> Ideally, the sample preparation room should be similar to a surgical suite. The room should contain a laminar flow, bio-hazard cabinet; be ventilated by filtered, positive-pressure air; and be reserved for asbestos analysis only. Air samples from the work areas should be taken periodically to monitor room air contaminants, particularly those of asbestos.

Nearly all asbestos drinking water samples are taken on a "grab" basis, typically involving collection of a single water sample or a sample pair, i.e., a single sample of water collected before treatment and another sample after treatment, or comparable samples collected before and after passage through A/C pipe. Although grab sampling may be an acceptable practice for other substances, its limitations for asbestos in drinking water have been emphasized:95/

> In order to more fully characterize the asbestos content of these water systems, it may be necessary to sample a small portion of a very large flow of water. This would minimize the problems of utilizing grab samples and reduce the

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95/ Tarter, M. E., and C. J. Leong, "Asbestos Sampling Plan for the San Francisco Bay Area, California," 58, Environmental Protection Agency Contract, Order No. C3253 NAET (July 1980).

> number of water samples to be analyzed. This would also take into account the intermittent and non-uniform occurrence of asbestos in water from various sources.

In spite of the desirability of "large flow" sampling, such a technique does not exist.

The absence of a method for "large flow" sampling requires that numerous samples be taken to produce valid monitoring data. The EPA Interim Method for Determining Asbestos in Water (EPA Interim Method) explicitly emphasizes this need, particularly in sampling open water supplies:96/

> If a representative sample of a water supply is required, a carefully designated set of samples should be taken representing the vertical as well as the horizontal distribution and these samples should be composited for analysis.

When asbestos levels in the water distribution system are being characterized, the need for numerous samples also is of vital importance. As EPA researchers have observed: "Collecting a single sample for an asbestos fiber count is often insufficient to judge the actual behavior of A-C pipe in a given situation."97/

To bypass the need for numerous samples, inexperienced investigators sometimes collect a single water sample before

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96/ Anderson, C. H., and J. M. Long, "Interim Method for Determining Asbestos in Water," 3, EPA-600/4-80-005 (January 1980).

97/ Buelow, R. W., et al., "The Behavior of Asbestos-Cement Pipe Under Various Water Quality Conditions," J. AWWA 102 (February 1980).

passage through the distribution system, and then take multiple samples after passage. This technique cannot, however, produce meaningful data because it does not result in independent "before/after" pairs. The "before" samples are artificial because they are based on a single sampling. The samples are not independent of each other and, therefore, cannot be used to ascertain asbestos levels in the distribution system. Only "matched" sample pairs can be expected to provide reliable analyses of the distribution system. Accurate characterization of asbestos levels in the water supply or distribution system results only when repeated samples are taken. This information is necessary to determine variability of fiber counts over time and to establish limits on the uncertainty associated with the number of counts for a given sample.

In addition to the numerous errors that may be introduced during sample collection, preparing samples for TEM analysis offers yet more "[s]ources of error . . . [that] may include settling of particulates in the water with time, clumping of the fibers during filtration, a nonuniform deposition of particulates on the membrane surface, loss of fibers during carbon coating or dissolution of the filter, and masking of fibers by other organic or inorganic particulates."98/

98/ Boatman, supra note 93, at 534.

2. Available analytic methods for determining asbestos levels in drinking water are limited.

After nearly a decade of research, there is only one method generally accepted as appropriate for detecting and quantifying asbestos in drinking water: transmission electron microscopy or TEM. Only TEM has the capability of magnifying the extremely small asbestos fibers and determining morphology, crystal structure and elemental analysis.

The availability of sophisticated TEM equipment is limited. Of the 250 to 300 instruments in the U.S., most are used in university metallurgical and health sciences laboratories or in private research facilities. Of these, it is estimated that only 20 analytical TEM instruments suitable for measuring asbestos in drinking water are in operation. Only two state health departments (Connecticut and California) have TEM capabilities for measuring asbestos in drinking water.

Properly trained and experienced operators are even more limited. There may be no more than a dozen microscopists in the U.S. truly skilled in dependably analyzing asbestos levels in drinking water.[99/] This situation has prompted one microscopist experienced in asbestos analysis to observe that "only a few analytical laboratories can or will undertake this type

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99/ Stewart, I. (Walter C. McCrone & Associates), Anderson, C.H. (EPA), private communications (December 1983).

of investigation."[100/] Moreover, neither EPA, nor the National Bureau of Standards, nor any other standards organization, has promulgated criteria or procedures for certifying TEM laboratories and operators.

The outlook is not optimistic for alternative analytic methods that might alleviate these equipment and trained manpower limitations. None of the EPA research and development involving two-phase liquid separation[101/] or multiple-detector light scattering methods[102/] has resulted in acceptable alternative analytical techniques. Moreover, the feasibility of surrogate methods, such as using turbidity as a reliable indicator for asbestos levels in finished drinking water, has not been demonstrated consistently.[103/] Even if analytical and detection technology progresses, it is likely that more sophisticated techniques will only exacerbate current equipment and operator limitations:[104/]

100/ Boatman, supra note 93, at 533.

101/ Melton, C. W., et al., "Development of a Rapid Analytical Method for Determining Asbestos in Water," EPA-600/4-78-066 (December 1978).

102/ Diehl, S. R., et al., "Optical Detection of Fiber Particles in Drinking Water," EPA-600/2-79-127 (August 1979).

103/ McGuire, M. J., et al., "Optimizing Large-Scale Water Treatment Plants for Asbestos-Fiber Removal," 364 J. AWWA (July 1983).

104/ Boatman, supra note 93, at 536.

> [S]uch a setup [asbestos fiber "tagging" for automatic pattern recognition and computer analysis] would probably mean that even fewer testing laboratories than are now operating would be able to afford the additional equipment.

Significant questions thus exist about the availability of equipment and trained personnel to monitor asbestos in the nation's drinking water.

3. The only available analytic monitoring method is not sufficiently precise nor accurate to ensure dependable compliance with an MCL.

Although the general approach to asbestos analytical methodology has improved over the past ten years, precision and accuracy of the EPA Interim Method remains limited and not well-documented. The analytic procedures in the EPA Interim Method are numerous and complicated. Each can introduce substantial errors into the analysis. Detection limits vary depending on extraneous particulate matter in the water sample, as well as the contamination level in the laboratory. By their own admission, the authors of the EPA Interim Method caution that it "is not intended to furnish a complete characterization of all the [asbestos] fibers in water."105/ EPA's Ambient Water Quality Criteria for Asbestos reaffirms this observation, noting that "[t]he analytical techniques for the measurement of asbestos minerals in . . .

105/ Anderson and Long, supra note 96, at 1.

water samples collected in . . . general environmental circumstances are time-consuming, and the results are often highly variable."106/

Nowhere is the EPA Interim Method burdened heavier than in terms of precision and accuracy.107/ The Method acknowledges that it is not even possible to quantify errors involved in counting:108/

> As no standard reference materials are available, only approximate estimates of the accuracy of the procedure can be made. At 1 MFL, it is estimated that the results should be within a factor of 10 of the actual asbestos fiber content.

It is noteworthy that EPA's own assessment of the accuracy of the method is only for fiber concentrations of 1 million fibers/liter -- levels of asbestos not found in 90-plus percent of U.S. drinking water. The accuracy of the EPA Interim Method has thus not been documented at asbestos levels most likely to occur in the vast majority of drinking waters in the United States.

106/ Environmental Protection Agency, Criteria and Standards Division, Office of Water Planning and Standards, "Asbestos-Ambient Water Quality Criteria" (1979) at C-1.

107/ The terms "precision" and "accuracy" describe statistical characteristics of a measurement process. The American Society of Testing and Materials (ASTM) defines these terms as follows: "Precision of a measurement process refers to the degree of mutual agreement between individual measurements from the process, while accuracy refers to the degree of agreement of such measurements with an accepted reference level . . .." ASTM Standard Recommended Practice for Use of the Terms Precision and Accuracy as Applied to Measurements of a Property of a Material, E-177, at 195 (1982).

108/ Anderson and Long, supra note 96, at 27 (emphasis added).

Few studies have attempted to document the precision and accuracy of the EPA Interim Method. The intra-laboratory precision of the method is dependent on the number of fibers counted. Under optimal conditions, i.e., maximum volume of water filtered, fiber loading of at least 3.5 fibers per grid square, and 100 fibers counted, precision (expressed as relative standard deviation) of 10% may be attained. This level of precision is rarely achieved in actual practice where suspended solids in drinking water limit the volume that can be filtered and the fibers are not uniformly distributed or are present in low concentrations. Then, the precision diminishes as the EPA Interim Method notes:[109/]

> The relative standard deviation of analyses of the same water sample in the same laboratory will increase as a result of sample preparation errors and a relative standard deviation of about ± 25 to 35% will occur. As the number of fibers counted decreases, the precision will also decrease approximately proportional to $\sqrt{N}$ where N is the number of fibers counted.

The limited data available indicate that intra-laboratory precision, based on a study involving a different analyst for each of three water samples, ranged from 24% to 37%.[110/] The inter-laboratory precision of the Method is substantially less. A study involving the analysis of filters prepared from

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109/ Anderson and Long, supra note 96, at 27.

110/ Id. at 28.

nine water samples by 51 laboratories showed that precision ranged from 35% to 66% or more than a factor of two.[111] Since this study side-stepped the difficult sample preparation process, it is reasonable to conclude that the results of an ab initio study would have shown even greater imprecision. Another comparison of inter-laboratory analyses between EPA and an independent laboratory showed that 33% of the total samples were "somewhat divergent." The differences in asbestos content of the "divergent" samples ranged from 91 to 161 million fibers/liter. The results of so-called non-divergent samples were no closer than 300,000 fibers/liter and as far apart as 37 million fibers/liter.[112]

In sum, a reasonable expectation from the EPA Interim Method is a level of accuracy within a factor of 10 of the actual asbestos fiber content and precision of about 50%. An analytic method with such inherent variability does not fulfill the statutory requirement of "quality control and testing procedures to insure compliance" with an MCL or treatment requirement.

4. The only available analytical monitoring method is unduly costly and time-consuming.

There are substantial costs in equipping a laboratory to analyze for asbestos in drinking water. A basic TEM

111/ Id.

112/ Boatman, supra note 93, at 536.

costs approximately $150,000. An analytical TEM equipped with selected area electron diffraction and energy dispersive spectroscopy capabilities to determine conclusively that observed fibers are asbestos costs from $235,000 to $295,000. As previously noted,[113] a clean room facility for sample preparation is necessary. Ancillary equipment including a vacuum evaporator, low temperature plasma asher, data processor, Jaffe-wick washer, and filtering apparatus, also must be purchased. Thus, a conservative estimate for constructing and equipping a laboratory for the analysis of asbestos in drinking water would be $250,000. In addition, salary and overhead for a qualified microscopist, a TEM maintenance contract and consumable items such as reagents, sample containment vessels, membrane filters, glassware, power, etc., would add another $50,000 of annual recurring expenses.

Assuming that 20% of the 60,000 community water supply systems to which a regulation might apply have asbestos in their water supplies,[114] the total cost of putting into place asbestos analytical capability would be $3 billion plus $600 million in yearly operation and maintenance costs.

113/ See p. 51, supra; Boatman, supra note 93 at 533.

114/ For purposes of this estimate, it is assumed that the 163,000 non-community water systems cannot make a capital investment of $250,000 for asbestos analytical capability.

Such costs do not meet reasonable standards of economic feasibility.

The alternative to purchasing analytical capabilities -- use of commercial laboratories -- also would be very costly. Typical costs for determining the chrysotile asbestos content of drinking water range from $300 to $480 per sample. Amphibole asbestos analysis and determination generally is $50 to $75 more per sample.[115/] Even this seemingly practical monitoring alternative has substantial economic impacts, as Gary Logsdon, Research Sanitary Engineer at EPA's Municipal Environmental Research Laboratory, has noted:[116/]

> The annual cost of submitting only two samples per week to a laboratory for analysis would be in the range of $30,000 to $40,000. Only the largest utilities can absorb such a high analytical cost.

Using Logsdon's average estimate ($35,000) and the assumptions in the previous paragraph, i.e., 12,000 water systems monitoring for asbestos, the total annual analytic cost (in 1979 dollars) of using commercial laboratories would be $420 million.

A substantial portion of the high costs of analysis doubtless results from the time-consuming process of sample preparation and fiber counting/determination. The costly,

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115/ Stewart, supra note 99.

116/ Logsdon, G. S., "Water Filtration for Asbestos Fiber Removal," 124, EPA-600/2-79-206 (December 1979).

laborious nature of this process has been documented in EPA's own research:[117/]

> Sample analysis generally takes more than one working day, including all preparation steps, although analysis of a single sample usually does not require more than eight hours of an analyst's time .... Because of the work load, most electron microscope laboratories (in 1979) are not able to provide results in less than three or four weeks after receipt of samples. Thus electron microscope results can not be used to monitor on-going plant performance.

The technical and economic infeasibility of using TEM, "the only method that yields actual asbestos fibers counts in water samples,"[118/] is crucial to the issue of whether a treatment technique should be proposed -- if an MCL is infeasible. As previously mentioned, no surrogate measures exist for determining asbestos levels in drinking water. As a practical matter, therefore, there exist no economically or technically feasible analytical methods to determine the efficacy of treatment techniques.

B. Asbestos Treatment and Removal Is Also Unduly Costly.

Prior to mandating a treatment technique to reduce contaminant levels in drinking water, the Administrator must also determine that means to accomplish concentration reductions are feasible, i.e., "generally available (taking costs

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117/ Id. at 87.

118/ Id. at 124.

into consideration)." As used in its everyday sense, the term means "widely used for accomplishment of the intended purposes."[119/] Because their use is so limited, it is difficult, if not impossible, to conclude that asbestos treatment and removal techniques are "generally available." Only six water systems in the U.S. specifically treat water supplies to remove asbestos fibers.

As the most recent studies demonstrate, the technological feasibility of asbestos treatment and removal techniques is far from established. Filtration plants receiving identical asbestos-containing influent waters, identically designed and optimized for fiber treatment, exhibit widely variable removal performance.[120/]

Determination of the feasibility of asbestos removal also must involve practical considerations such as whether water system operators have adequate engineering and operating experience. There is little data and experience available to support a finding of "generally available" in this regard, as well. Expert engineering opinion has cautioned, for example, that due to the requirements to produce consistently high quality waters to accomplish asbestos removal, ". . . a need

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119/ "Generally is defined to mean 'universally' or 'in a general manner.'" Webster's Third New International Dictionary 945 (1976). "General," itself, is defined as a characteristic of the majority or "widespread." Id. at 944. "Available" is defined as "capable of use for the accomplishment of a purpose." Id. at 150.

120/ McGuire, supra note 103, at 366-367.

may arise for more highly skilled operators. Such additional cost would also have to be charged to asbestos removal."[121/]

The limited data available on use of asbestos removal techniques indicates clearly that the costs of treatment operations are substantial. First, pilot studies must be undertaken. In Seattle, the pilot phase alone of an engineering feasibility study on asbestos removal cost approximately $200,000. Capital costs for the modification of existing treatment and filtration plants, or for the construction of new plants to comply with an MCL or treatment technique, would be enormous. According to EPA data,[122/] modifications to a 2.3 million gallon per day (mgd) plant cost $1.1 million. New construction was even more expensive: $7.7 million for a 36 mgd plant and $25 million for a 100 mgd facility (in 1979 cost adjusted dollars). Assuming again that 12,000 water systems might be required to undertake the least burdensome treatment technique -- modification of existing facilities -- a modest $1 million in construction costs per plant balloons to $12 billion in total costs. This estimate obviously understates the magnitude of the financial impact that would result.

121/ Gumerman, R.C., et al., "Estimating Costs for Water Treatment As a Function of Size and Treatment Efficiency," EPA-600/2-78-182. (1978).

122/ Logsdon, supra note 116, at 120.

Operational costs would be additive to such pilot phase, modification, and new construction costs. There would be the expenses of labor, chemicals, power, maintenance and repairs. For a major water utility -- the 100 mgd plant previously cited -- annual operational costs have been estimated as $1.2 million (1979 dollars);[123/] costs in 1984 would be substantially greater. A more current (1981-82) cost model for chemical costs alone shows that optimization of treatment plants for asbestos removal "results in large increases in operating costs over and above standard treatment conditions" -- $2.84 million for five treatment plants.[124/] Using these costs ($568,000 per plant) as a yardstick, the 12,000 utilities with asbestos in their water supplies would face a collective economic impact exceeding $6.8 billion per year in extra operating costs.

EPA must also take in account the potential adverse health effects of asbestos treatment and removal techniques. The two water filtration processes shown to reduce asbestos levels in drinking water -- granular media filtration and diatomaceous earth filtration -- normally use alum (aluminum sulfate) as a primary coagulant. Under optimum treatment conditions, alum concentration levels of 2.5 to 10.5 mg/liter

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123/ "Seattle Tolt Water Supply Mixed Asbestiform Removal Study," EPA-600/2-79-153, Appendices B and C, pp. 101-103 (Dec. 1979).

124/ McGuire, supra note 103, at 370.

are common,[125] resulting in an aluminum residual in treated waters from .05 to 0.5 mg/liter. The ANPR, 48 Fed. Reg. at 45514-15, notes that the relationship between senile demential and dialysis encephalopathy "and other ailments has not been correlated with aluminum ingestion but . . . has become a cause for concern." A recent study reaffirms EPA's concern about the potential neurotoxicity of aluminum:[126]

> The incidence of both senile and dialysis dementia has been linked to levels of aluminium in the environment. Exposure to aluminium could increase the permeability of the BBB [blood-brain barrier] to small behaviourally active peptides or other substances. Repeated insults by aluminium might be involved in dementia or other forms of CNS [central nervous system] disease.

Consideration of an MCL or treatment techniques for asbestos must therefore include full consideration of the potential adverse effects of aluminum, as well as all other chemicals used in the treatment and removal process.

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In sum, very expensive and quite inaccurate and imprecise monitoring capabilities exist for asbestos at the levels found in U.S. water supplies. Moreover, any treatment of such waters to reduce asbestos concentrations would

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125/ Id. at 367.

126/ Banks, W.A., and A.J. Kastin, "Aluminium Increases Permeability of the Blood-Brain Barrier to Labelled DSIP and B-Endorphin: Possible Implications for Senile and Dialysis Dementia," The Lancet 1228-29 (Nov. 26, 1983).

require reliance on relatively new and potentially very expensive technology. It is, therefore, not "economically and technically feasible" to monitor asbestos in drinking water; "quality control and testing procedures to insure compliance" with either an MCL or a treatment technique requirement are not available; and treatment techniques for asbestos are not "generally available." No primary drinking water regulation for asbestos is therefore possible under the SDWA.

CONCLUSION

The time for tentativeness about asbestos in water has passed. More than sufficient first quality data have been developed, to a large extent due to an extensive EPA research program, to close the book on asbestos. As Dr. Commins concluded in his lengthy review of the ingestion evidence:[127/]

> A great deal of time and money has been spent in the last 10 years or so in evaluating the subject, and now it would seem the controversy has for all practical purposes ended, and maybe the issue can be regarded as essentially a non-problem. Perhaps research effort should now be sensibly diverted into various other environmental issues.

When EPA issues its promised next set of proposals for national revised primary drinking water standards, no proposal for a recommended maximum contaminant level for

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127/ Commins, supra note 13, at 11.

asbestos should be included. Instead, EPA should in that notice explain that "in the judgment of the Administrator," asbestos is not a substance in drinking water that "may have any adverse effect on the health of persons."128/ Moreover, EPA should fulfill its proper role of assuring the public, based on its comprehensive decade-plus program to explore all aspects of the asbestos in water issue, that it need not fear any health effects from asbestos in the nation's water supplies.

Respectfully sumbitted,

Timothy S. Hardy
Kirkland & Ellis
1776 K Street, N.W.
Washington, D.C. 20006
(202) 857-5042

Special Counsel to the
Association of Asbestos/
Cement Pipe Producers

John F. Welch
Association of Asbestos/
Cement Pipe Producers
1600 Wilson Boulevard
Suite 1008
Arlington, VA 22209
(703) 841-1556

January 3, 1984

128/ SDWA § 1401(1)(B), 42 U.S.C. § 300f(1)(B).