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TOXICOLOGY OF INGESTED ASBESTOS
LITERATURE SURVEY

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INFORMATIVE SUMMARY WITH CONCLUSIONS BASED ON THE SAMPLE RECEIVED. ADDITIONAL INFORMATION INCLUDING THE EFFECTS OF REPEATED EXPOSURE MAY BE REQUIRED AS SPECIFIC USES AND FORMULATIONS ARE DEVELOPED OR IF PROCESS CHANGES OCCUR.

A survey of the literature that is pertinent to the ingestion of asbestos was made.

Asbestos is a ubiquitous mineral appearing everywhere - air, snow, rivers, drinking water, beverages, and drugs. Even control animals in one feeding study had asbestos particles in the several organs examined.

Since it must be assumed that during inhalation, some portion of the material must be swallowed, the incidence of gastrointestinal cancer in asbestos workers is pertinent to the ingestion of asbestos. Although the incidence of GI cancer in asbestos workers is relatively low in relation to the incidence of lung cancer, studies have consistently shown GI cancer incidences of 2-3 times that expected in the general population. The incidence appears to increase with the duration of exposure and there is a long latent period for development.

Feeding asbestos to animals has not increased the incidence of tumors.

A study designed to determine the carcinogenicity of various sizes of asbestos and fiberglass particles when applied to the pleura of rats showed a definite relationship between carcinogenicity and size. -Fibers < 3 microns in diameter and > 20 microns in length are more carcinogenic than fibers with diameters > 3 microns regardless of length or less than 20 microns in length regardless of diameter. Most fibers found in water or beverages are individual fibers less than 20 microns in length.

In 1973 The Advisory Committee on Asbestos Cancer reported to the Director of the International Agency for Research on Cancer (a division of the World Health Organization). As part of their report they stated that, "such evidence as there is does not indicate any risk from asbestos fibers present in water, food, beverage, or drugs".

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TOXICOLOGY OF INGESTED ASBESTOS

LITERATURE SURVEY

Charles T. Lichy

Toxicology Research Laboratory

October 22, 1974

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TOXICOLOGY OF INGESTED ASBESTOS

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TOXICOLOGY OF INGESTED ASBESTOS

Introduction

Although there is a large body of literature on the hazard of inhalation of asbestos, there is a dearth of definitive information on the hazard of asbestos ingestion. This report ignores literature on the inhalation hazard and focuses only on the data, theory, and conjecture dealing with the toxicology of ingested asbestos. However, since workers exposed to atmospheres containing asbestos ingest the material as well as inhale it, the literature relating to gastrointestinal cancer in asbestos workers is reviewed.

Part of the difficulty in performing definitive ingestion studies has been the difficulty of asbestos analyses. The analysis for asbestos requires an electron microscope (at least for fibers less than about .3 microns in diameter) and the method usually requires a tedious fiber count from electron micrograph plates. In addition, the reproducibility from sample to sample is usually poor. The time required is about 11 man-hours per sample. Standard samples do not exist. This means that it is difficult to adequately characterize the samples of asbestos fed to animals and makes comparisons of studies difficult.

Asbestos in Environment

"Asbestos" is a generic term for a number of hydrated silicates that, when crushed or processed, separate into flexible fibers made up of fibrils. There are many asbestos minerals, although only six are of commercial importance. Chrysotile, a

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serpentine mineral, accounts for 95% of world's production. The other minerals are all amphiboles and include amosite, crocidolite, anthophyllite, tremolite, and actinolite. These asbestos minerals differ in chemical and physical characteristics which may affect their biological reactivity.

Rock outcrops of asbestos minerals and soils derived from asbestos minerals as well as commercial uses result in the material being ubiquitous. Water flowing over rock outcrops or soils can pick up the asbestos. The Ottawa river in Canada, for example, was found to contain asbestos particles (Table 1). Cunningham and Pontefract, (1971) examined twelve samples of Canadian beer and wine, six samples of U.S. beer, six samples of wine from around the world, and several Canadian soft drinks. All were found to contain asbestos fibers in the range of 1.1 to 12.2 million fibers per liter (mfl). Samples of drinking water from Canadian cities which use surface water were also found to contain asbestos. Most of the fibers found in Canadian beer and filtered city water were less than 1 micron in length whereas most samples of wine, some American beer and unfiltered water contained fibers ranging from 1-15 microns in length. Filtering seems to help. The Ottawa river before filtering contained 9.5 mfl while the city of Ottawa tap water contained 2.0 mfl. The Detroit river contained 18-25 mfl while the city of Windsor tap water contained about 1.8 mfl (Kay, 1973). Samples of melted snow contained asbestos indicating asbestos in water may originate from dust. Wehman and Plantholt (1974) found asbestos fibrils ranging from 2.18 to 23.98 million fibers per liter of gin. Biles and Emerson (1968) also found asbestos fibers in beer.

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TABLE 1

Table 1 Asbestos Fibres in Beverages and Water		
Sample	Source *	No. of fibres $\times 10^6 \text{ l.}^{-1}$
Beer	Canadian 1	4.3
Beer	Canadian 2	6.6
Beer	USA 1	2.0
Beer	USA 2	1.1
Sherry	Canadian	4.1
Sherry	Spanish	2.0
Sherry	South African	2.6
Port	Canadian	2.1
Vermouth	French	1.8
Vermouth	Italian	11.7
Soft drink	Ginger ale	12.2
Soft drink	Tonic water I	1.7
Soft drink	Tonic water II	1.7
Soft drink	Orange	2.5
Tap water	Ottawa, Ottawa river (F)	2.0
Tap water	Toronto, Lake Ontario (F)	4.4
Tap water	Montreal, St Lawrence river (F)	2.4
Tap water	Hull, Quebec, Ottawa river (NF)	9.5
Tap water	Beauport, Quebec, St Lawrence river (6 km below Quebec City) (NF)	8.1
Tap water	Drummondville, Eastern Townships, Quebec, St Francois river (F)	2.9
Tap water	Asbestos, Eastern Townships, Quebec, Nicolet river (F)	5.9
Tap water	Thetford Mines, Eastern Townships, Quebec, Lac à la Truite (NF)	172.7
Melted snow	Ottawa, top 30 cm (2-3 weeks precipitation)	33.5
River water	Ottawa river, at Ottawa	9.5

* F, Filtration plant used; NF, no filtration plant used.

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Nicholson, Maggiore, and Selikoff (1972) studied a number of samples of parenteral drugs and found asbestos fibers (Table 2). Based on their report, a study was undertaken by the FDA. Although the data are still preliminary (Fed Reg 38 (188) 9-28-73 pp 27077-27081), 11 of 13 samples of parenteral drugs had clear-cut evidence of asbestos, one sample was questionable, and one was negative. Although these drugs are not ingested, they are examples of the widespread occurrence of asbestos.

Perhaps the greatest concern of asbestos ingestion is that from drinking water. Studies have been made by several workers. Asbestos fibers have been found in practically every locale where sampling programs have been conducted and regardless whether the water source was surface or deep well. The levels seem to be higher in the coastal and mountainous regions where serpentine rock is indigenous, but asbestos has been found throughout the midwest as well. Fiber levels range generally from a few thousand fibers per liter to about ten million fibers per liter. For example, Sargent (1973) reported levels of asbestos in drinking water from Vermont which ranged from less than one thousand to 247 thousand fibers per liter (Table 3). Cunningham and Pontefract (1971) reported asbestos levels of 2-10 million fibers per liter except for an asbestos mining town in Quebec Province which registered 172.7 million fibers per liter (Table 1). Cook, Glass, and Tucker (1974) reported levels of asbestiform amphibole (not chrysotile) minerals of 1-30 million fibers per liter in the Duluth water supply. Kay (1973) reported levels of asbestos in

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TABLE 2

Fibrous particulates seen in samples of parenteral drugs obtained during two periods of time. Observations were made by optical microscope (O) or electron microscope (E). The optical determinations were made on one quarter of each sample, the electron-microscope determinations on 10^{-3} of each sample. All drugs were labeled for intravenous use except those identified by IM for intramuscular or IP for intraperitoneal. Amounts of samples are in grams unless specified otherwise. "Yes" after optical data indicates that some fibers in the sample were identified as chrysotile by optical microscopy; while many fibers seen in all samples could have been asbestos, the majority were not. All values of 27×10^{-6} g or less for the estimated mass of asbestos could have resulted from the analysis background; N, number.

Drug	Amount (g)	Fibers by O, 1969			Asbestos	Chrysotile fibers by E		Asbestos, estimated mass in total sample (10^{-6} g)
		5-20 μ m (N)	20-100 μ m (N)	> 100 μ m (N)		1969 (N)	1970-71 (N)	
Sodium acetazolamide	1	52	35	7			> 100	1100
Sodium ampicillin	1	10	13	3	Yes	32	2	7.6
Cephaloridine	2	38	28	4			85	210
Sodium cephalothin	2	44	22	5		8	6	7.7
Chloramphenicol	2						8	14
Cortisone (IM)	0.25	6	2	1			2	4.3
Lincomycin HCl	1.2	2	4	0			4	10
Sodium methicillin	2	91	51	10		6	22	42
Neomycin sulfate* (IM)	1	28	6	1			2	1.7
Neomycin sulfate* (IP)	5	101	57	38	Yes	79	1	3.9
Sodium oxacillin	2	24	35	18			> 100	410
Streptomycin sulfate* (IM)	1	15	0	0			4	7.5
Streptomycin sulfate* (IM)	2	15	8	1				
Sulfisoxazole diolamine	4	24	7	1			8	15
Tetracycline HCl†	1	26	16	7	Yes	50	35	86
Tetracycline HCl†	0.5					13		
Vancomycin HCl	1	16	7	1		26	30	78
Potassium penicillin G	10^6 units	14	4	1			12	14
Sodium penicillin G	2×10^6 units						9	27
Water control	20 cm ³	6	0	0		3	7	101

* Duplicate analyses of samples from different manufacturers of the same drug. † Duplicate analyses of samples from the same lot of a single manufacturer. ‡ Average of seven samples ranging from 1.2×10^{-6} to 21×10^{-6} g.

Nicholson, Maggiore, and Selikoff - 1972

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the drinking water of Canadian cities (Ontario Province) ranging from 2-4 million fibers per liter. He reported that the Ontario Ministry of Health has indicated that "ingestion of asbestos at these levels does not appear to present a health hazard at this time."

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Ingestion During Inhalation

Although this review does not deal with inhalation of asbestos fibers, the nose, throat, and trachea of humans is an efficient particle trap and any particle that is caught in the mucosa of these areas will pass through the gastrointestinal tract. Studies have shown that only a relatively narrow range of particles are inhaled and most efficiently at 0.25-0.50 microns (Hilding, 1963). The efficiency, of course, varies with the shape and density of the particles. Although asbestos, being fibrous, may be quite different in the size ranges, the majority of the particles must be swallowed.

Selikoff and co-workers began in the early 1960's to study asbestos workers. On January 1, 1943, there were 632 members in the New York and New Jersey union local of the International Association of Heat and Frost Insulators and Asbestos Workers, AFL-CIO (IAHFIAW). Nine had died before reaching twenty years from first employment. The fate of the remainder through 1971 are shown in Table 4. The study was then expanded to include all of the workers in the IAHFIAW in the United States and Canada. These results are shown in Table 5. The results indicate an increase of GI cancer of 2-3 times that expected from national averages. It is, however, responsible for only about 5% of the total deaths.

In a paper given in Lyon, France in 1973, Selikoff and co-workers reported on more studies. An entire work force of an amosite factory, which manufactured asbestos insulating

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materials, was studied. The group studied was selected from those first employed during 1941 - 1945 and consisted of 933 men who worked a range of one day to 20 years. These results are shown in Table 6. The incidence of cancer of the stomach, colon, and rectum was increased 2-3 times.

Although the deaths from GI cancer were relatively few in relation to lung cancer, the consistency of the results suggest that the increase is real. Of course, the exposure to asbestos of the insulation workers is much higher than the ordinary population. In the United States, as a whole, the incidence of GI cancer has diminished from 5.3% of all deaths in 1960 to 4.9% of all deaths in 1970.

The Advisory Committee on Asbestos Cancers reported in 1973 to the Director of the International Agency for Research on Cancer (part of the World Health Organization) and stated that a small excess risk of gastrointestinal cancer existed from asbestos inhalation, but was relatively small compared to bronchial cancer.

McDonald, et al (1971) studied the mortality in workers from chrysotile mines in Quebec, Canada and found that although the respiratory cancer was higher than normal, the rate of gastrointestinal cancer was unrelated to the dust levels that the workers were exposed to.

Enterline and Henderson (1973) reported a study of retirees from the asbestos industry. They found that men exposed only to

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chrysotile asbestos had a respiratory cancer mortality rate 2.4 times the expected; whereas men exposed to both chrysotile and crocidolite had a respiratory cancer mortality rate 5.6 times the expected. In no case, however, was the gastrointestinal cancer rate significantly higher than expected.

There seemed to be a trend toward increasingly higher incidence among older people (75 + years) but the values are not statistically significant.

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TABLE 4

Expected^a and observed number of deaths among 623 New York-New Jersey asbestos insulation workers 1 January 1943-31 December 1971, twenty or more years after onset of first exposure to asbestos

	1943-51		1952-61		1962-71		Total, 1943-71	
	Expected	Observed	Expected	Observed	Expected	Observed	Expected	Observed
Total cancer: all sites	11.3	28	18.3	57	17.6	104	47.2	189
Lung cancer	1.5	13	3.5	23	5.1	48	10.1	84
Pleural mesothelioma	n.a. ^c	1	n.a.	2	n.a.	5	n.a.	8
Peritoneal mesothelioma	n.a.	1	n.a.	3	n.a.	20	n.a.	24
Cancer of stomach, colon, rectum, oesophagus	4.1	7	5.0	18	3.9	16	13.0	41
Cancer all other sites	5.7	6	9.8	11	8.6	15	24.1	32
Asbestosis	n.a.	1	n.a.	10	n.a.	22	n.a.	33
All other causes	65.1	44	89.4	94	78.3	61	232.8	199
Total all causes	76.4	73	107.7	161	95.9	187	280.0	421
Person-years of observation	3726		4406		2898		11,030	

^a 632 members were on the union's rolls on 1 January 1943. Nine died before reaching 20 years from first employment. All others entered these calculations upon reaching the 20-year from onset of first exposure point.

^b Expected rates are based upon age-specific death rate data of the US National Office of Vital Statistics from 1949-67. Rates were extrapolated 1943-48 from rates for 1949-55, and for 1968-71 from rates for 1961-67.

^c US death rates not available, but these are rare causes of death in the general population.

Selikoff, Hammond, and Seidman - 1973

TABLE 5

Expected^a and observed deaths among 17,800 asbestos insulation workers in the United States and Canada 1 January 1967-31 December 1971

	Total		Distribution by duration from onset of exposure			
			Less than 20 years		20 years and more	
	Expected	Observed	Expected	Observed	Expected	Observed
Total deaths	805.63	1092	178.94	211	626.69	881
Cancer: all sites	144.09	459	26.31	51	117.78	408
Lung cancer	44.42	213	7.03	22	37.39	191
Pleural mesothelioma	n.a. ^b	26	n.a.	2	n.a.	24
Peritoneal mesothelioma	n.a.	51	n.a.	3	n.a.	48
Cancer of stomach	6.62	16	0.97	1	5.65	15
Cancer of colon, rectum	17.51	26	2.51	3	15.00	23
Cancer of oesophagus	3.21	13	0.44	1	2.77	12
All other cancers	72.33	114	15.36	19	56.97	95
Asbestosis	n.a.	78	n.a.	5	n.a.	73
All other causes	661.54	555	152.63	155	508.91	400
Number of men	17,800		12,681		5,119	
Person-years of observation	86,300		62,673		23,627	

^a Expected deaths are based upon age-specific death rate data of the US National Office of Vital Statistics. Rates for 1968-71 were extrapolated from rates for 1961-67.

^b US death rates not available, but these are rare causes of death in the general population.

Selikoff, Hammond, and Seidman - 1973

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TABLE 6

Expected^a and observed deaths among 933^b amosite asbestos factory workers first employed 1941-45, and observed to 31 December 1971

	Before 1952		1952-61		1962-71		Total, 1941-71	
	Expected	Observed	Expected	Observed	Expected	Observed	Expected	Observed
Total deaths	69.98	88	108.27	146	121.24	250	299.49	484
Total cancer: all sites	9.81	15	18.72	44	21.63	84	50.16	143
Lung cancer	1.49	3	3.71	23	6.21	47	11.41	73
Pleural mesothelioma	n.a. ^c	1	n.a.	0	n.a.	2	n.a.	3
Peritoneal mesothelioma	n.a.	0	n.a.	0	n.a.	4	n.a.	4
Cancer of stomach	1.45	3	1.84	4	1.29	4	4.58	11
Cancer of colon, rectum	1.56	2	2.61	5	2.88	8	7.05	15
Cancer of oesophagus	0.27	0	0.45	0	0.51	0	1.23	0
All other cancers	5.04	6	10.11	12	10.74	19	25.89	37
Asbestosis	n.a.	3	n.a.	7	n.a.	17	n.a.	27
All other causes	60.17	70	89.55	95	99.61	149	249.33	314

^a Expected rates are based upon age-specific death rate data of the US National Office of Vital Statistics from 1949-67. Rates were extrapolated for 1941-48 from rates for 1949-55, and for 1968-71 from rates for 1961-67.

^b 933 men were employed. In 5 cases, ages were not known and these men have been excluded from these calculations. 877 men were traced to death or to 31 December 1971. 51 men were partially traced and remain in the calculations until lost to observation.

^c US death rates not available, but these are rare causes of death in the general population.

Selikoff, Hammond, and Seidman - 1973

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Ingestion Studies

In a study by Bonser and Clayson (1967) forty rats were maintained on a diet for 75 weeks which contained 1.5 grams of asbestos per Kg of food. No tumors of any kind resulted nor were there any lesions on the intestinal wall which might have suggested a previous absorption of asbestos fibers. There was, however, a high mortality rate in the study: only 38% of the controls and 30% of the treated rats survived the 75 weeks.

In an unpublished study by L.M. Swinburn (personal communication to FDA) asbestos particles were fed once a week to an unspecified number of SPF Wistar rats for 16 to 18 weeks, administered in butter. Although the asbestos particles were of the size range known to produce tumors by other modes of administration, no tumorigenic effect was noted. This was, however, a very short study. With a single large dose, the fibers were totally cleared by the gastrointestinal tract in 48 hours and no asbestos was detected in the animal tissues at the end of one week. The gastrointestinal tract of the rat seemed to provide an effective barrier to penetration.

In another study, W.E. Smith, et al (1965), reported that hamsters maintained on a diet of one percent chrysotile or amosite throughout their life had no gastrointestinal tumors.

Harley, Gross, and Greene reported at the 1974 Annual Meeting of the International Academy of Pathology (US-Canada Div.) on rat feeding studies. In one study ten rats were fed 5% asbestos for

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21 months. In another study, ten rats were given intragastric injections of asbestos and taconite (an iron ore containing asbestos). In a third study, 20 rats were fed asbestos and taconite mixed in butter and were examined at weekly intervals for five weeks. Although occasional mineral fibers were found in the tissue digests of seven animals from all of the studies, only one of the fibers was consistent with the ingested test material. Also similar fibers were found in the control animals. Electron microscopic examination of the gut wall disclosed no evidence of tissue invasion by fibers except for the presence of a fiber in a vacuole of a phagocytic cell in the intestinal submucosa of one animal.

Pontefract and Cunningham (1973) reported intragastric injections of asbestos resulted in asbestos being found in the blood. Rats given 9.4×10^9 fibers had $4.65 \times 10^6/\text{g}$ in the blood two days later (Table 7). Four days after treatment with 94×10^9 (10 x the first dose) fibers the blood levels were $1.19 \times 10^6/\text{g}$. This indicates that there is a mechanism for clearance of fibers from blood. Assays of other tissues revealed asbestos fibers but in numbers similar to the controls. Apparently, the body contains asbestos particles from air, food and water contamination. Most of the fibers found were 0.2-2.0 micrometers in length.

In early 1974, as a result of a water pollution suit brought by the Environmental Protection Agency (EPA) against the Reserve Mining Corporation, the water of Duluth, Minnesota, was found to

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TABLE 7

Numbers of Asbestos Fibres Recovered from Treated and Non-Treated Rats						
	Controls mean *		Group 1 † mean *		Group 2 ‡ mean *	
Blood	0.00	—	4.65 §	1.02	1.19	0.77
Spleen	2.33	0.67 §	4.11	0.49	3.45	1.20
Omentum	2.46	0.46	2.74	0.87	18.25	7.79
Heart	2.09	0.41	—	—	2.28	0.40
Brain	0.05	0.02	0.31	0.19	0.29 **	0.11
Lungs	1.06	0.26	1.80	0.54	1.74	0.26

* Average of five rats in all cases except where noted. † Animals injected with 9.4×10^8 fibres and tissues examined 2 d later. ‡ Animals injected with 9.4×10^8 fibres and tissues examined 4 d later. § Standard error of the mean. || Four rats only. ¶ $P < 0.01$; ** $P < 0.05$.
(The figures in the table are fibres $g^{-1} \times 10^{-6}$.)

Pontefract and Cunningham - 1973

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contain asbestos fibers. Although the EPA felt that asbestos fibers constituted a hazard to health and the U.S. District Court in Minnesota concurred, most of the evidence appeared to be based on inhalation studies. Masson, McKay, and Miller (1974) studied the patterns of cancer in the Duluth, Minnesota, area and found no increase in the incidence of cancer among persons of all ages including children, but noted that the period of observation (15 years) was short relative to the latent period for asbestos-induced carcinogenesis.

In 1971, Merliss suggested that the high rate of stomach cancer in Japan was a result of using talc, which may be contaminated with asbestos, as a coating on rice. The rate of stomach cancer appears to follow roughly the rice-eating habits of people in various areas within Japan. Japanese who migrated to the U.S. and do not routinely eat talc-coated rice do not have the high incidence of stomach cancer. Other Asians who do not eat talc-treated rice also do not have high incidence of stomach cancer. Smith, 1973, reported a study in which hamsters were fed talc containing fibrous materials. No tumors were found and Smith suggested nitrosamines as the causative agent for stomach cancer in Japan rather than talc-treated rice.

The Advisory Committee on Asbestos Cancer reported in 1973 to the Director of the International Agency for Research on Cancer (a Division of the World Health Organization). As part of their report they stated that, "such evidence as there is does not indicate any risk from asbestos fibers present in water, food, beverages, or drugs".

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Mechanics of Asbestos Toxicity

A theory has been postulated in recent years that the principle mechanism of action of asbestos is mechanical rather than chemical and depends on its fibrous character (length to diameter ratio). Stanton and Wrench of the National Cancer Institute reported in 1972 on an extensive study comparing various types and sizes of asbestos and fiberglass when applied to the pleura of rats. Asbestos, inducing a high incidence of mesotheliomas, caused fewer mesotheliomas when reduced to sub-microscopic fibrils by milling. Fiberglass did not cause mesotheliomas except when reduced to sizes resembling asbestos fibers. Metals and hydrocarbons have been postulated to cause tumors or at least enhance the activity of asbestos. (Harington,-1965, Harington and Roe,-1965). Neither fine particulate nickel or nickel-chrome steel or oil showed any tumorigenic effects. In an editorial in the Journal of the National Cancer Institute in 1974, Stanton postulated that fibers < 3 microns in diameter and > 20 microns in length are more carcinogenic than fibers with diameters larger than 3 microns regardless of length or with lengths less than 20 microns regardless of diameter. Short fibers are probably not carcinogenic because they are effectively disposed of by overt or intracellular phagocytic activity. Since asbestos found in liquids are short individual fibers less than 20 microns in length, their presence may constitute a minimum hazard if the studies of Stanton and Wrench are reliable.

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One report (Cunningham and Pontefract - 1973) comments that the more time that fibers remain in water, the more their magnesium is leached out and the central canal begins to disappear. The electron diffraction pattern also begins to fade. This may indicate a slow solubilization of the fibers in water. However, no other report makes this comment.

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