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Asbestos (CASRN 1332-21-4)

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Human health assessment information on a chemical substance is included in the IRIS database only after a comprehensive review of toxicity data, as outlined in the IRIS assessment development process. Sections I (Health Hazard Assessments for Noncarcinogenic Effects) and II (Carcinogenicity Assessment for Lifetime Exposure) present the conclusions that were reached during the assessment development process. Supporting information and explanations of the methods used to derive the values given in IRIS are provided in the guidance documents located on the IRIS website.

STATUS OF DATA FOR Asbestos

File First On-Line 09/26/1988

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	no data	
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	07/01/1993

_I. Chronic Health Hazard Assessments for Noncarcinogenic Effects

_I.A. Reference Dose for Chronic Oral Exposure (RfD)

Substance Name — Asbestos
CASRN — 1332-21-4

Not available at this time.

_I.B. Reference Concentration for Chronic Inhalation Exposure (RfC)

Substance Name — Asbestos
CASRN — 1332-21-4

Not available at this time.

_II. Carcinogenicity Assessment for Lifetime Exposure

Substance Name — Asbestos
CASRN — 1332-21-4
Last Revised — 07/01/1993

Section II provides information on three aspects of the carcinogenic assessment for the substance in question; the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates of risk from oral exposure and from inhalation exposure. The quantitative risk estimates are presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per (mg/kg)/day. The unit risk is the quantitative estimate in terms of either risk per ug/L drinking water or risk per ug/cu.m air breathed. The third form in which risk is presented is a drinking water or air concentration providing cancer risks of 1 in 10,000, 1 in 100,000 or 1 in 1,000,000. The rationale and methods used to develop the carcinogenicity information in IRIS are described in The Risk Assessment Guidelines of 1986 (EPA/600/8-87/045) and in the IRIS Background Document. IRIS summaries developed since the publication of EPA's more recent Proposed Guidelines for Carcinogen Risk Assessment also utilize those Guidelines where indicated (Federal Register 61(79):17960-18011, April 23, 1996). Users are referred to Section I of this IRIS file for information on long-term toxic effects other than carcinogenicity.

NOTE: The carcinogen assessment summary for asbestos may change in the near future pending the outcome of a further review now being conducted by the CRAVE Work Group.

_II.A. Evidence for Human Carcinogenicity

__II.A.1. Weight-of-Evidence Characterization

Classification — A; human carcinogen

Basis — Observation of increased mortality and incidence of lung cancer, mesotheliomas and gastrointestinal cancer in occupationally exposed workers are consistent across investigators and study populations. Animal studies by inhalation in two strains of rats showed similar findings for lung cancer and mesotheliomas. Animal evidence for carcinogenicity via ingestion is limited (male rats fed intermediate-range chrysotile fibers; i.e., >10 um length, developed benign polyps), and epidemiologic data in this regard are inadequate.

__II.A.2. Human Carcinogenicity Data

Sufficient. Numerous epidemiologic studies have reported an increased incidence of deaths due to cancer, primarily lung cancer and mesotheliomas associated with exposure to inhaled asbestos. Among 170 asbestos insulation workers in North Ireland followed for up to 26 years, an increased incidence of death was seen due to all cancers (SMR=390), cancers of the lower respiratory tract and pleura (SMR=1760) (Elmes and Simpson, 1971) and mesothelioma (7 cases). Exposure was not quantified.

Selikoff (1976) reported 59 cases of lung cancer and 31 cases of mesothelioma among 1249 asbestos insulation workers followed prospectively for 11 years. Exposure was not quantified. A retrospective cohort mortality study (Selikoff et al., 1979) of 17,800 U.S. and Canadian asbestos insulation workers for a 10-year period using best available information (autopsy, surgical, clinical) reported an increased incidence of cancer at all sites (319.7 expected vs. 995 observed, SMR=311) and cancer of the lung (105.6 expected vs. 486 observed, SMR=460). A modest increase in deaths from gastrointestinal cancer was reported along with 175 deaths from mesothelioma (none expected). Years of exposure ranged from less than 10 to greater than or equal to 45. Levels of exposure were not quantified. In other epidemiologic studies, the increase for lung and pleural cancers has ranged from a low of 1.9 times the expected rate, in asbestos factory workers in England (Peto et al., 1977), to a high of 28 times the expected rate, in female asbestos textile workers in England (Newhouse et al., 1972). Other occupational studies have demonstrated asbestos exposure-related increases in lung cancer and mesothelioma in several industries including textile manufacturing, friction products manufacture, asbestos cement products, and in the mining and milling of asbestos. The studies used for the inhalation quantitative estimate of risk are listed in the table in Section II.C.2.

A case-control study (Newhouse and Thompson, 1965) of 83 patients with mesothelioma reported 52.6% had occupational exposure to asbestos or lived with asbestos workers compared with 11.8% of the controls. Of the remaining subjects, 30.6% of the mesothelioma cases lived within one-half mile of an asbestos factory compared with 7.6% of the controls.

The occurrence of pleural mesothelioma has been associated with the presence of asbestos fibers in water, fields and streets in a region of Turkey with very high environmental levels of naturally-occurring asbestos (Baris et al., 1979).

Kanarek et al. (1980) conducted an ecologic study of cancer deaths in 722 census tracts in the San Francisco Bay area, using cancer incidence data from the period of 1969-1971. Chrysotile asbestos concentrations in drinking water ranged from nondetectable to 3.6E+7 fibers/L. Statistically significant dose-related trends were reported for lung and peritoneal cancer in white males and for gall bladder, pancreatic and peritoneal cancer in white females. Weaker correlations were reported between asbestos levels and female esophageal, pleural and kidney cancer, and stomach cancer in both sexes. In an extension of this study, Conforti et al. (1981) included cancer incidence data from the period of 1969-1974. Statistically significant positive associations were found between asbestos concentration and cancer of the digestive organs in white females, cancers of the digestive tract in white males and esophageal, pancreatic and stomach cancer in both sexes. These associations appeared to be independent of socioeconomic status and occupational exposure to asbestos.

Marsh (1983) reviewed eight independent ecologic studies of asbestos in drinking water carried out in five geographic areas. It was concluded that even though one or more studies found an association between asbestos in water and cancer mortality (or incidence) due to neoplasms of various organs, no individual study or aggregation of studies exists that would establish risk levels from ingested asbestos. Factors confounding the results of these studies include the possible underestimates of occupational exposure to asbestos and the possible misclassification of peritioneal mesothelioma as GI cancer.

Polissar et al. (1984) carried out a case-control study which included better control for confounding variables at the individual level. The authors concluded that there was no convincing evidence for increased cancer risk from asbestos ingestion. At the present time,

an important limitation of both the case-control and the ecologic studies is the short follow-up time relative to the long latent period for the appearance of tumors from asbestos exposure.

__II.A.3. Animal Carcinogenicity Data

Sufficient. There have been about 20 animal bioassays of asbestos. Gross et al. (1967) exposed 61 white male rats (strain not reported) to 86 mg chrysotile asbestos dust/cu.m for 30 hours/week for 16 months. Of the 41 animals that survived the exposure period, 10 had lung cancer. No lung cancer was observed in 25 controls.

Reeves (1976) exposed 60-77 rats/group for 4 hours/day, 4 days/week for 2 years to doses of 48.7-50.2 mg/cu.m crocidolite, 48.2-48.6 mg/cu.m amosite and 47.4-47.9 mg/cu.m chrysotile. A 5-14% incidence of lung cancer was observed among concentration groups and was concentration-dependent.

Wagner et al. (1974) exposed CD Wistar rats (19-52/group) to 9.7-14.7 mg/cu.m of several types of asbestos for 1 day to 24 months for 7 hours/day, 5 days/week. A duration-dependent increased incidence of lung carcinomas and mesotheliomas was seen for all types of asbestos after 3 months of exposure compared with controls.

F344 rats (88-250/group) were exposed to intermediate range chrysotile asbestos (1291E+8 f/g) in drinking water by gavage to dams during lactation and then in diet throughout their lifetime (NTP, 1985). A statistically significant increase in incidence of benign epithelial neoplasms (adenomatous polyps in the large intestine) was observed in male rats compared with pooled controls of all NTP oral lifetime studies (3/524). In the same study, rats exposed to short range chrysotile asbestos (6081E+9 f/g) showed no significant increase in tumor incidence.

Ward et al. (1980) administered 10 mg UICC amosite asbestos 3 times/week for 10 weeks by gavage to 50 male F344 rats. The animals were observed for an additional 78-79 weeks post-treatment. A total of 17 colon carcinomas were observed. This result was statistically significant compared with historical controls; no concurrent controls were maintained.

Syrian golden hamsters (126-253/group) were exposed to short and intermediate range chrysotile asbestos at a concentration of 1% in the diet for the lifetime of the animals (NTP, 1983). An increased incidence of neoplasia of the adrenal cortex was observed in both males and females exposed to intermediate range fibers and in males exposed to short range fibers. This increase was statistically significant by comparison to pooled controls but not by comparison to concurrent controls. NTP suggested that the biologic importance of adrenal tumors in the absence of target organ (GI tract) neoplasia was questionable.

__II.A.4. Supporting Data for Carcinogenicity

Sincock (1977) reported an increased number of chromosomes and chromosome breaks after passive inclusion of asbestos with CHO-K1 cells. Chamberlain and Tarmy (1977) reported asbestos not to be mutagenic for E. coli or S. typhimurium. A positive response was unlikely, however, since prokaryotic cells do not phagocytize particles as do eukaryotic cells.

_II.B. Quantitative Estimate of Carcinogenic Risk from Oral Exposure

Not available.

_II.C. Quantitative Estimate of Carcinogenic Risk from Inhalation Exposure

__II.C.1. Summary of Risk Estimates

Inhalation Unit Risk — 2.3E-1 per (f/mL)

Extrapolation Method — Additive risk of lung cancer and mesothelioma, using relative risk model for lung cancer and absolute risk model for mesothelioma

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	4E-4 f/mL
E-5 (1 in 100,000)	4E-5 f/mL
E-6 (1 in 1,000,000)	4E-6 f/mL

__II.C.2. Dose-Response Data for Carcinogenicity, Inhalation Exposure

Human Data Occupational Group	Fiber Type	Reported Average Exposure (fiber-yr/mL)	% Increase in Cancer per fiber-yr/mL	Reference
Lung Cancer:				
Textile Products	Predominantly Chrysotile	44	2.8	Dement et al., 1983b
Textile Products	Chrysotile	31	2.5	McDonald et al., 1983a
Textile Products	Chrysotile	200	1.1	Peto, 1980
Textile Products	Chrysotile	51	1.4	McDonald et al., 1983b
Friction Products	Chrysotile	32	0.058	Berry and Newhouse, 1983
Friction Products	Chrysotile	31	0.010	McDonald et al., 1984
Insulation Products	Amosite	67	4.3	Seidman, 1984
Insulation Workers	Mixed (Chrysotile, al., 1979 Crocidolite and Amosite)	300	0.75	Selikoff et

Asbestos Products		374	0.49	Henderson and Enterline, 1979
Cement Products		89	0.53	Weill et al., 1979
		112	6.7	Finkelstein, 1983
Mesothelioma:				
Insulation workers	Mixed	375	1.5E-6	Selikoff et al., 1979; Peto et al., 1982
Insulation Products	Amosite	400	1.0E-6	Seidman et al., 1979
Textile Products Manufacturer	Chrysotile	67	3.2E-6	Peto, 1980; Peto et al., 1982
Cement Products	Mixed	108	1.2E-5	Finkelstein, 1983

__II.C.3. Additional Comments (Carcinogenicity, Inhalation Exposure)

Risks have been calculated for males and females according to smoking habits for a variety of exposure scenarios (U.S. EPA, 1986). The unit risk value is calculated for the additive combined risk of lung cancer and mesothelioma, and is calculated as a composite value for males and females. The epidemiological data show that cigarette smoking and asbestos exposure interact synergistically for production of lung cancer and do not interact with regard to mesothelioma. The unit risk value is based on risks calculated using U.S. general population cancer rates and mortality patterns without consideration of smoking habits. The risks associated with occupational exposure were adjusted to continuous exposure by applying a factor of 140 cu.m/50 cu.m based on the assumption of 20 cu.m/day for total ventilation and 10 cu.m/8-hour workday in the occupational setting.

The unit risk is based on fiber counts made by phase contrast microscopy (PCM) and should not be applied directly to measurements made by other analytical techniques. The unit risk uses PCM fibers because the measurements made in the occupational environment use this method. Many environmental monitoring measurements are reported in terms of fiber counts or mass as determined by transmission electron microscopy (TEM). PCM detects only fibers longer than 5 um and >0.4 um in diameter, while TEM can detect much smaller fibers. TEM mass units are derived from TEM fiber counts. The correlation between PCM fiber counts and TEM mass measurements is very poor. Six data sets which include both measurements show a conversion between TEM mass and PCM fiber count that range from 5-150 (ug/cu.m)/(f/mL). The geometric mean of these results, 30 (ug/cu.m)/(f/mL), was adopted as a conversion factor (U.S. EPA, 1986), but it should be realized that this value is highly uncertain. Likewise, the correlation between PCM fiber counts and TEM fiber counts is very uncertain and no generally applicable conversion factor exists for these two measurements.

In some cases TEM results are reported as numbers of fibers <5 um long and of fibers longer than 5 um. Comparison of PCM fiber counts and TEM counts of fibers >5 um show that the

fraction of fibers detected by TEM that are also >0.4 um in diameter (and detectable by PCM) varies from 22-53% (U.S. EPA, 1986).

It should be understood that while TEM can be specific for asbestos, PCM is a nonspecific technique and will measure any fibrous material. Measurements by PCM which are made in conditions where other types of fibers may be present may not be reliable.

In addition to the studies cited above, there were three studies of asbestos workers in mining and milling which showed an increase in lung cancer (McDonald et al., 1980, Nicholson et al., 1979; Rubino et al., 1979). The slope factor calculated from these studies was lower than the other studies, possibly because of a substantially different fiber size distribution, and they were not included in the calculation. The slope factor was calculated by life table methods for lung cancer using a relative risk model, and for mesothelioma using a absolute risk model. The final slope factor for lung cancer was calculated as the weighted geometric mean of estimates from the 11 studies cited in section II.C.2. The final slope factor for mesothelioma is based on the calculated values from the studies of Selikoff et al. (1979), Peto et al. (1982), Seidman et al. (1979), Peto (1980) and Finkelstein (1983) adjusted for the mesothelioma incidence from several additional studies cited previously.

There is some evidence which suggests that the different types of asbestos fibers vary in carcinogenic potency relative to one another and site specificity. It appears, for example, that the risk of mesothelioma is greater with exposure to crocidolite than with amosite or chrysotile exposure alone. This evidence is limited by the lack of information on fiber exposure by mineral type. Other data indicates that differences in fiber size distribution and other process differences may contribute at least as much to the observed variation in risk as does the fiber type itself.

The unit risk should not be used if the air concentration exceeds 4E-2 fibers/ml, since above this concentration the slope factor may differ from that stated.

__II.C.4. Discussion of Confidence (Carcinogenicity, Inhalation Exposure)

A large number of studies of occupationally-exposed workers have conclusively demonstrated the relationship between asbestos exposure and lung cancer or mesothelioma. These results have been corroborated by animal studies using adequate numbers of animals. The quantitative estimate is limited by uncertainty in the exposure estimates, which results from a lack of data on early exposure in the occupational studies and the uncertainty of conversions between various analytical measurements for asbestos.

_II.D. EPA Documentation, Review, and Contacts (Carcinogenicity Assessment)

__II.D.1. EPA Documentation

Source Document — U.S. EPA, 1985

The 1985 Drinking Water Criteria Document for Asbestos and the 1986 Airborne Asbestos Health Assessment Update have received Agency Review.

__II.D.2. EPA Review (Carcinogenicity Assessment)

Agency Work Group Review — 09/15/1987, 12/02/1987

Verification Date — 12/02/1987

__II.D.3. EPA Contacts (Carcinogenicity Assessment)

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (202)566-1676 (phone), (202)566-1749 (FAX) or hotline.iris@epa.gov (internet address).

_III. [reserved]
_IV. [reserved]
_V. [reserved]

_VI. Bibliography

Substance Name — Asbestos
CASRN — 1332-21-4
Last Revised — 07/01/1993

_VI.A. Oral RfD References

None

_VI.B. Inhalation RfC References

None

_VI.C. Carcinogenicity Assessment References

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_VII. Revision History

Substance Name — Asbestos
CASRN — 1332-21-4

Date	Section	Description
09/26/1988	II.	Carcinogen summary on-line
05/01/1989	II.	Carcinogen summary noted as pending change
12/01/1989	VI.	Bibliography on-line
03/01/1991	II.A.1.	Text revised
07/01/1991	II.C.3.	Last paragraph units changed from ug/cu.m to fibers/ml
01/01/1992	IV.	Regulatory Action section on-line
07/01/1993	II.D.1.	EPA Documentation clarified
07/01/1993	VI.C.	References alphabetized correctly
08/01/1995	II.	EPA's RfD/RfC and CRAVE workgroups were discontinued in May, 1995. Chemical substance reviews that were not completed by September 1995 were taken out of IRIS review. The IRIS Pilot Program replaced the workgroup functions beginning in September, 1995.
04/01/1997	III., IV., V.	Drinking Water Health Advisories, EPA Regulatory Actions, and Supplementary Data were removed from IRIS on or before April 1997. IRIS users were directed to the appropriate EPA Program Offices for this information.
02/22/2001	I., II.	This chemical is being reassessed under the IRIS Program.

_VIII. Synonyms

Substance Name — Asbestos
CASRN — 1332-21-4
Last Revised — 09/26/1988

- 1332-21-4
- Asbestos
- calidria-asbestos

IRIS Home

Chronic Health Hazards for Non-Carcinogenic Effects

Reference Dose for Chronic Oral Exposure (RfD)

- Oral RfD Summary
- Principal and Supporting Studies
- Uncertainty and Modifying Factors
- Additional Studies/Comments
- Confidence in the Oral RfD
- EPA Documentation and Review

Reference Concentration for Chronic Inhalation Exposure (RfC)

- Inhalation RfC Summary
- Principal and Supporting Studies
- Uncertainty and Modifying Factors
- Additional Studies/Comments
- Confidence in the Inhalation RfC
- EPA Documentation and Review

Carcinogenicity Assessment for Lifetime Exposure

Evidence for Human Carcinogenicity

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Quantitative Estimate of Carcinogenic Risk from Oral Exposure

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