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UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

DATE: **June 10, 1998**

MEMORANDUM

OFFICE OF
PREVENTION, PESTICIDES AND
TOXIC SUBSTANCES

SUBJECT: ***ALUMINUM/MAGNESIUM PHOSPHIDE*** **- REVISED REPORT** of the Hazard Identification Assessment Review Committee.

FROM: Jess Rowland, Executive Secretary 6/10/98
Hazard Identification Assessment Review Committee
Health Effects Division (7509C)

THROUGH: K. Clark Swentzel, Chairman, K. Clark Swentzel 6/10/98
Hazard Identification Assessment Review Committee
Health Effects Division (7509C)

TO: Paul Lewis, Risk Assessor
Risk Characterization and Analysis Branch
Health Effects Division (7509C)

PC Code 066501

On April 16, 1988 the Health Effects Division's Hazard Identification Assessment Review Committee (HIARC) reviewed the toxicology database and selected the toxicological endpoints for occupational exposure risk assessments for aluminum/magnesium phosphide. HIARC's conclusions were presented in the Committee's report dated May 4, 1998 (HED Doc. No.012601).

On June 2, 1998 the Committee met again selected doses and endpoints for acute and chronic dietary risk assessments as requested by the Chemistry Science Advisory Council. In addition, the dose and endpoints selected for Occupational exposure (Short-Term inhalation) was revised.

This report includes the decisions made at both meetings and supersedes the previous report.

Committee Members in Attendance at the April 16, 1998 Meeting:

Members present were: Karl Baetcke, William Burnam, Karen Hamernik, Mike Metzger (Co-Chairman, John Redden, Jess Rowland, (Executive Secretary) and Clark Swentzel (Chairman). Member(s) in absentia: Robert Frick, Susan Makris and Melba Morrow. Data was presented by Stanley Gross of the Toxicology Branch 2.

HED staff participating at the meeting were: Sanju Diwan, John Whalan, P. Lewis, D. Hrdy and Tracy Keigwin.

Committee Members in Attendance at the June 2, 1998 Meeting:

Members present were: William Burnam, Susan Makris, John Redden, Jess Rowland (Executive Secretary), Melba Morrow and Clark Swentzel (Chairman). Members in absentia were: Robert Fricke and Karen Hamernik.

HED staff participating at this meeting were: Stan Gross (Toxicologist), Paul Lewis (Risk Assessor), and Steve Knizner (Branch Senior Scientist, RCAB).

Data Presentation: _____
Stan Gross, Ph.D

Report Preparation: _____
Jess Rowland
Executive Secretary

I. INTRODUCTION

On April 16, 1998 the Health Effects Division's Hazard Identification Assessment Review Committee (HIARC) reviewed the toxicology database and selected the toxicological endpoints for occupational risk assessments only. Dose levels and endpoints were not selected for establishing the acute and chronic Reference Doses (RfDs) since the current use pattern does not indicate the need for dietary risk assessments and the lack of oral toxicity studies.

Following the HIARC meeting, the Chemistry Science Advisory Council met on April 27, 1998 and determined that dose levels must be selected for establishing acute and chronic Reference Doses since tolerances (40 CFR.180.225) are established for residues of the fumigant phosphine in/on raw agricultural commodities from postharvest treatment with aluminum phosphide

Consequently, the HIARC met on June 2, 1998 and selected the doses and endpoints for acute and chronic dietary risk assessments. The HIARC acknowledges that inhalation studies are not appropriate for oral (dietary) risk assessments. However, inhalation studies were used in hazard identification because: 1) the toxicology database for this chemical was limited to studies conducted via the inhalation route, 2) these are the only studies in which the Committee can quantitate the dosage of phosphine exposed to laboratory animals, and 3) use of an inhalation "dose" provides a conservative approach for oral risk assessments.

During this evaluation, the HIARC also revised the toxicological endpoint selected for Short-Term occupational exposure (inhalation) risk assessment.

This report includes the decisions made at both meetings and supersedes the previous report.

II. HAZARD IDENTIFICATION

A. Acute Reference Dose (RfD)

Study Selected: 90-Day Inhalation Toxicity Study §82-4

MRID No.: 41413101

Executive Summary: A 90-day inhalation study was conducted with male and female Fischer 344 rats exposed via inhalation to phosphine using three different exposure regimens as follows: I) at 0, 0.3, 1.0 or 3.0 ppm, 6 hours/day, 5 days/week for 13 weeks; II) a second exposure regimen initiated on study Day 48 at 0 or 10 ppm. After 3 days of exposure 4 of 10 females died and therefore this group was terminated; and III) a third exposure regimen initiated on study Day 75 with additional groups of rats at 0 or 5 ppm for 15 days; exposure was terminated on study Day 90. For each exposure regimen, recovery groups were included in the study and these groups were sacrificed after 4 weeks of post-exposure observations.

In the 5 ppm for 15-day exposure regimen, there were no treatment-related effects on survival, body weight, food consumption, ophthalmological or hematological parameters. No treatment-related histopathological lesions were seen in either sex. Males exhibited statistically significant increases in alkaline phosphatase activity and blood urea nitrogen.

These increases, however, were not considered to be biologically significant since: 1) similar increases were not seen in females ; 2) there was no corroborative histo-pathological lesions in the kidneys; and 3) the effect did not persist after recovery.

In rats exposed at 10 ppm for 3 days, there was 80% mortality in females but no mortality in males. Both sexes of rats exhibited coagulative necrosis in the tubules of the kidneys and pulmonary congestion was observed in the females that died.

In the 13-week exposure regimen, there was no mortality in either sex at any concentration tested. There was a transient decrease in body weight gain accompanied by decreased food consumption. Red blood cell counts, hemoglobin concentration, and hematocrit values were slightly decreased in males at 3.0 ppm (at 4 weeks only), but no effects were observed in these males at 13 weeks or in females at either interval. No exposure-related gross or histologic findings were observed at levels up to and including 3.0 ppm. Under the conditions of this study, for subchronic inhalation toxicity, the NOEL was 3 ppm (HDT); a LOEL was not established.

Dose and Endpoint for Risk Assessment: 5 ppm = 0.007 mg/L= 1.8 mg/kg/day based on lack of treatment-related effects following 15 days of exposure.

Route-To-Route Extrapolation: Since an inhalation concentration was selected for oral dietary risk assessment, the following route-to route extrapolation (i.e., inhalation to oral) was used for establishing the acute RfD in mg/kg/day:

To convert the ppm to mg/L/day:

$$\text{mg/L/day @ 25 C/101 kPa} = \frac{\text{(ppm) x Molecular Weight}}{\text{24,450 (Boyle's gas law)}}$$

$$\text{mg/L} = \frac{\text{5 ppm x 34 (MW)}}{\text{24,450}} = 0.007 \text{ mg/L/day}$$

To convert mg/L/day to mg/kg/day:

mg/kg/day = Concentration (mg/L/day) x Absorption x Conversion Factor x Duration of Exposure x Activity factor

mg/kg/day = 0.007 mg/L x 1 x 47.0 x 6 hours x 1.0

Where:

0.0063 mg/L = Concentration
1 = absorption factor (100%, default)
47.0=Conversion Factor [Respiratory Volume (7.15 L/hr/kg) ÷ Body Weight (0.152 kg)].
1 = Activity Factor (Animal default is 1).

Uncertainty Factor:= 100 which includes 10 x for intra-species variation and 10 x for inter-species extrapolation.

Acute RfD = 1.8 mg/kg/day = **0.018 mg/kg**
100 (UF)

Comments about Study/Endpoint: The Committee determined that the 5 ppm concentration is appropriated for this (acute dietary) risk assessment, because: 1) no treatment-related effects were seen at this concentration after 15 exposures; 2) no treatment-related effects were seen at a lower concentration (3 ppm) after a longer (13 weeks) duration of exposure; and 3) an oral study was not available in the database.

In addition, this concentration (5 ppm) is comparable to the concentration of 6.7 ppm derived by using the LOEL of 20 ppm established in an acute neurotoxicity study in rats and an Uncertainty Factor of 3 for the lack of a NOEL (i.e., 20 ppm ÷3 = 6.7 ppm). The Committee did not elect to use the acute neurotoxicity study since a NOEL was not established in the study; instead the acute neurotoxicity study was used as a "co-critical" or "support" study. In the acute study, (43903801), Crl:CD rats (11/sex/concentration) were exposed to phosphine at 0, 20, 30 or 40 ppm (1% a.i. nitrogen) for 4 hours. The LOEL for neurobehavioral effects was 20 ppm (the lowest concentration tested) based on decreased body temperature and decreased motor activity in both sexes; a NOEL was not established.

This risk assessment is required.

B. Chronic Dietary [Reference Dose (RfD)]

Study Selected: Chronic Inhalation Toxicity/Carcinogenicity - Rat § 83-5.

MRID No. 44415101

Executive Summary: In a chronic/oncogenicity study, Charles River Fischer CDF Rats (60/Sex/Group) were exposed to phosphine under dynamic chamber conditions to 0, 0.3, 1 and 3 ppm of phosphine for 52 weeks of a two year study. The rats were kept under standard laboratory conditions, observed twice daily and sacrificed (10/sex/group) during week 52 of the study.

There were no adverse effects observed for the initial 12 month period. Body weights (taken weekly); food consumption (weekly); routine hematologic, serum biochemical and urinary analyses were all comparable to control animals. Ophthalmological observations, gross pathology, organ weights and histopathology indicated no adverse effects from the PH3 exposures. The NOEL was 3.0 ppm (HDT); a LOEL was not established. This NOEL is based on the results of the study reported in a 52 week interim report. The final report is due to be submitted to the Agency in November, 1998.

Dose and Endpoint for Risk Assessment: NOEL = 3 ppm = 0.004 mg/L = 1.13 mg/kg/day. The NOEL is based on the results of a 52-week Interim Report. The final report is due November, 1998.

Route-To-Route Extrapolation: Since an inhalation concentration was selected for oral dietary risk assessment, the following route-to route extrapolation (i.e., inhalation to oral) was used for establishing the chronic RfD in mg/kg/day:

To convert the NOEL of 3 ppm to mg/L/day:

$$\text{mg/L/day @ 25 C/101 kPa} = \frac{\text{ppm x Molecular Weight}}{\text{24,450 (Boyle's gas law)}}$$

$$\text{mg/L} = \frac{3 \text{ ppm} \times 34 \text{ (MW)}}{24{,}450} = 0.004 \text{ mg/L/day}$$

To convert mg/L/day to mg/kg/day:

mg/kg/day = Concentration (mg/L/day) x Absorption x Conversion Factor x Duration of Exposure x Activity factor

mg/kg/day = .004 mg/L x 1 x 47.0 x 6 hours x 1.0

Where:

0.004 mg/L = Concentration (NOEL)
1 = absorption factor (100%, default)
47.0=Conversion Factor [Respiratory Volume (7.15 L/hr/kg) ÷ Body Weight (0.152 kg)].
1 = Activity Factor (Animal default is 1).

Uncertainty Factor:= 100 which includes 10 x for intra-species variation and 10 x for inter-species extrapolation.

Chronic RfD = $\frac{1.13 \text{ mg/kg/day}}{100 \text{ (UF)}} = 0.0113 \text{ mg/kg/day}$

Comments about Study/Endpoint: The dose recommended for oral risk assessment is based on an inhalation NOEL. Phosphine has been shown to be toxic via the inhalation route. However, since a dose is needed for risk assessment (due to the existence of tolerances), HIARC has recommended an inhalation NOEL. It is noted that the "dose" recommended is overly conservative and is recommended as a worst case scenario

In a "safety study" published in the open literature (Hackenberg, 1972), diets were treated with Phostoxin pellets at high dosage at rates of 48 and 90 gm/metric ton, fumigated for 48 hours and 72 hours, mixed for 2 hours, and then aerated for one hour. The feed was then stored frozen in small sealed containers until used as laboratory rat feed. Sixteen separate batches of feed were treated this way over the two year period. Samples of diet were taken for analytically determined levels for phosphine at the time the feed was removed from the freezer. Phosphine levels ranged from 0.2 to 7.5 ppm and averaged approximately 1 ppm. The amounts of phosphine that remained in the feed offered to the rats as food was not measured (but would be expected to dissipate). Therefore the actual dosages in this study are unknown. Rats were fed the "treated" pellets for two years. Two groups of 60 rats each (30 males and 30 females) were used, one as treatment group and other as controls. The rats were observed for the effects on growth, food consumption, survival, morbidity, hematology, blood chemistry and gross and microscopic pathology. No differences were seen between the controls and the treated animals for any toxicity parameter. No increased oncogenicity resulted from fumigation residues (Accession Nos. 26937, 2693. 6000).

This is not a guideline study (toxicity secondary to phosphine residues is not possible when aeration is adequate), however the study is useful as a "safety study" for residues from excessive treatment levels (fumigated at 10 times the level of phosphine normally used for commodity fumigation). The study is acceptable for showing that the LEL for toxicity from residues was not achieved with the excessive fumigation treatment rates. The amount of phosphate residues from the fumigation were not determined. The phosphine sealed in the stored feed after inadequate (one hour) post-fumigation aeration is expected to have dissipated from the feed placed in the cages before the rats could ingest the feed. Therefore the 2 year NOEL or LEL for PH3 trapped in the feed was not determined. The 2 year NOEL for the ingestion of phosphine fumigation residues was greater than 10 times the fumigation levels normally used.

This risk assessment is required

C. Occupational/Residential Exposure

There are no registered residential uses at the present time. Based on the use pattern, the route of exposure of concern is inhalation and not dermal. Consequently doses and endpoints were selected only for occupational inhalation exposure risk assessments only.

1. Short-Term Inhalation - (1-7 days)

Study Selected: 90 day rat inhalation study. §82-4

MRID No. 41413101

Executive Summary: See Acute Dietary

Dose and Endpoint for Risk Assessment: 5 ppm (0.007 mg/L) based on lack of treatment-related effects following 15 days of exposure.

Comments about Study and Endpoint: This concentration is appropriate for this exposure period of concern (i.e., 1- 7 days) since the treatment was for 15 days and no treatment-related effects were observed at this concentration.

This risk assessment is required.

2. Intermediate-Term (7 Days to Several Months)

Study Selected: 90 day rat inhalation study. §82-4

MRID No. 41413101

Executive Summary: See Acute Dietary

Dose and Endpoint for Risk Assessment: NOEL= 3 ppm (0.004 mg/L) based on lack of treatment-related effects at the highest concentration tested; a LOEL was not established

Comments about Study and Endpoint: This study is appropriate for the exposure period of concern because of the duration of exposure (i.e., 90 days) and also the NOEL of this study is supported by a similar NOEL established in a 90-day neurotoxicity study in rats (MRID No. 4421040). In that study, no treatment-related effects were observed in survival, clinical signs, body weights, neurobehavioral effects or gross and histopathology in male and female Crl:CD rats exposed to phosphine (1% a.i in nitrogen) at 0, 0.3, 1 or 3 ppm, 6 hours/day, 5 days/week for approximately 90 days. The NOEL was 3 ppm (HDT); a LOEL was not established.

This risk assessment is required.

3. Long-Term Inhalation (Several Months to Life-Time)

Study Selected: Chronic Toxicity/Carcinogenicity - Rat § 83-5.

MRID No. 44415101

Executive Summary: See Chronic Dietary

Dose and Endpoint for Risk Assessment: NOEL = 3 ppm (0.004 mg/L) the highest concentration tested.

Comments about Study/Endpoint:. The NOEL is based on the results of a 52-week Interim Report. The final report is due November, 1998

This risk assessment is required.

D. Recommendation for Aggregate Exposure Risk Assessments

Not applicable; there are no registered residential uses at the present time.

E. Margins of Exposure For Occupational Exposure Risk Assessments

A Margin of Exposure (MOE) of 100 (10 x for intra-species variation and 10 x for inter-species extrapolation) is adequate for occupational exposure via the inhalation routes. A MOE is not required for residential exposure since there are no registered residential uses at the present time.

III. CLASSIFICATION OF CARCINOGENIC POTENTIAL

1. Carcinogenicity in Rats.

No studies are available to assess the carcinogenic potential of aluminum/magnesium phosphide in rats.

2. Carcinogenicity Study in Mice

No studies are available to assess the carcinogenic potential of aluminum/magnesium phosphide in rats.

3. Classification of Carcinogenic Potential: The carcinogenic potential of aluminum/magnesium phosphide has not evaluated by the HED Cancer Assessment Review Committee since there are no tumor data to evaluate.

IV. MUTAGENICITY

1. Gene Mutations

Salmonella typhimurium reverse gene mutation assay (MRID No. 41434301): The test is negative with hydrogen phosphide (PH_3) in all strains up to cytotoxic concentrations (≥488 ppm/plate +/-S9).

2. Chromosome Aberrations

In vitro cytogenetic assay with Chinese hamster ovary (CHO) cells) (MRID No. 41434302): The test is positive, PH_3 at 2500 and 5000 ppm without S9 activation caused a significant but not dose-related increase in the frequency of cells with structural chromosome aberrations. Significant clastogenic effects were also noted at 2500 ppm with S9 activation but not at the highest dose tested (5000 ppm). This assay is currently listed as Unacceptable, because a repeat assay was not performed. The study should be upgraded, however, because the positive findings from a mouse lymphoma assay (MRID No. 42987302) conducted with zinc phosphide (Zn_3P_2)favor chromosomal damage rather than a true point mutational event and can be considered independent confirmation of *in vitro* clastogenic activity for PH_3.

3. Other Genotoxic Mechanisms

In vivo unscheduled DNA Synthesis (UDS) in primary rat hepatocytes (MRID No. 42788101). The test is negative in male Fischer rats exposed via inhalation to PH_3 doses of 0, 4.8, 13, 18 or 23 ppm (equiv. to 0, 11.4, 30.8, 42.6 or 54.5 mg/m^3, respectively) for 6 hours. Overt toxicity (i.e., difficulty in breathing) but no target cell cytotoxicity was observed at the highest dose tested.

4. Non-guideline studies

Based on the findings reported by Garry et al., (1989) that pesticide applicators exposed to PH_3 had increased levels of chromosome damage, the USEPA sponsored a series of acute (Kligerman et al.,1994a) and subacute (Kligerman et al., 1994b) inhalation cytogenetic studies with PH_3. Summaries of these studies are as follows:

(i). PH_3 was negative for the induction of micronucleated polychromatic erythrocytes (MPE) in bone marrow cells and splenocytes and negative for the induction of sister chromatid exchange or chromosomal aberrations in splenocytes of CD-1 male mice exposed by inhalation to 0, 5, 10 or 15 ppm for 6 hours. Overt toxicity, manifested as lethargy and shallow breathing was seen at the highest dose tested. There was a dose-related and significant reduction of splenocyte cell cycling at all levels, which indicates that PH_3 was cytotoxic to splenocytes. There was, however, no adverse effect on bone marrow cells (Kligerman, et al., 1994a; MRID No. 43315103).

(ii). As part of the NTP-sponsored studies, male B6C3F1 mice and male F344 rats were exposed by inhalation to 0, 1.25, 2.5 or 5.0 ppm PH_3, 6 hours/day, 5 days/week over an 11-day period. Bone marrow cells and/or peripheral blood lymphocytes were harvested and examined for sister chromatid exchanges and chromosomal aberrations (mouse and rat peripheral blood lymphocytes) and for MPEs (rat bone marrow and mouse bone marrow and peripheral blood lymphocytes). In addition, B6C3F1 males were exposed via inhalation to 0 or 5 ppm as above over a 12-day period and mated with untreated females in a dominant lethal assay. Results show that PH_3 was not genotoxic at any endpoint.

While there was no evidence that the test material reached the target sites in potentially genotoxic concentrations, dosing was considered adequate based on the data from other submitted guideline studies (Kligerman, et al., 1994b; MRID No. 43315101).

(iii). Following subchronic inhalation exposure (0, 0.3, 1.0 or 4.5 ppm, 6 hours/ day, 5 days/week for 13 weeks) but not acute inhalation exposure (0 or 5.5 ppm, 2 weeks, 6 hours/day, 5 days/week for 2 weeks), PH_3 at 4.5 ppm caused a statistically significant increase in MN induction in the spleen lymphocytes and bone marrow cells of Balb-c male and female mice. There was, however, no increase in gene mutations at the HPRT locus in the recovered spleen lymphocytes (MRID No. 43315102).

(iv). After 6 hours or inhalation exposure, PH_3, at the highest dose tested (19 ppm) induced a significant increase in chromosomal aberrations in the bone marrow of Sprague Dawley male rats but not in the female rats. The effect is considered equivocal because increased chromosomal aberration frequencies were only seen in high-dose males with severely reduced mitotic indices (MIs). Females did not show increased chromosome aberrations and did not have decreased MIs. There was also no effect on peripheral lymphocytes (MRID No. Not assigned).

(v). No significant differences in the frequency of micronuclei in the peripheral lymphocytes of 31 PH_3 fumigators compared to control (21) was observed in the evaluation of PH_3 at occupational exposure levels (Barbosa, et al., 1993).

5. Conclusions:

PH_3 is not mutagenic in bacteria but is clastogenic *in vitro*. Both the negative Ames test and the positive Chinese hamster ovary cell chromosome assay are consistent with the *in vitro* test results for Zn_3P_2. Studies conducted *in vivo* indicate that PH_3 is not clastogenic in mice or rats or cause dominant lethal mutations in mice following acute exposures for up to 2 weeks. There is, however, evidence that inhalation exposures of PH_3 for up to 13 weeks induced significant clastogenic and/or aneuploidogenic effects in male and female mice. The biological relevance of this finding can not be fully ascertained until the results of the 2-year bioassay currently underway are submitted and reviewed.

The acceptable studies satisfy the pre-1991 mutagenicity initial testing battery guidelines. No further testing is required at this time.

V. FQPA CONSIDERATIONS

The database included a prenatal inhalation developmental toxicity study in rats. In that study, pregnant CD rats were exposed to aerosol concentrations of phosphine (1%a.i. nitrogen) at 0, 0.03, 0.3,3.0, 5.0 or 7.5 ppm, 6 hours/day during gestation days 6 through 15, inclusive. Maternal toxicity was manifested as mortality in 14/19 dams at 7.5 ppm during the exposure period. These dams received 3 to 10 exposures; the remaining 5 dams that had not been exposure to phosphide were sacrificed and the entire dosing group was removed from the study. No mortality occurred at the other dose groups. No treatment-related effects were seen in maternal body weight ,body weight gain, food consumption and gross pathology. For maternal toxicity, the NOEL was 5 ppm and the LOEL was 7.5 ppm based on mortality. No developmental toxicity was seen. For developmental toxicity, the NOEL was 5 ppm (MRID No. 41377002).

VI. DATA GAPS

None. The toxicology data requirements for a food-use chemical is not required for aluminum/magnesium phosphide since no phosphine exposure is expected from the use pattern (fumigant). Any phosphine left in fumigated commodities would be expected to be removed by adequate aeration of the commodities. Bound reaction products formed by reactions with phosphine and biological materials form innocuous phosphates. Therefore, the Committee determined that no additional toxicology studies are required for this chemical.

VII. ACUTE TOXICITY

Acute Toxicity of Aluminum/Magnesium Phosphide

Guideline No.	Study Type	MRID #(S).	Results	Toxicity Category
81-1	Acute Oral	No study	--	NA
81-2	Acute Dermal	No study	--	NA
81-3	Acute Inhalation	41377001	LC_{50} = >11 ppm (HDT) 0.014 mg/L	I
81-4	Primary Eye Irritation	No study	--	NA
81-5	Primary Skin Irritation	No study	--	NA
81-6	Dermal Sensitization	No study	--	NA

VIII. SUMMARY OF TOXICOLOGY ENDPOINT SELECTION

The doses and toxicological endpoints selected for various exposure scenarios are summarized below.

EXPOSURE SCENARIO	Concentration	ENDPOINT	STUDY
Acute Dietary	1.18 mg/kg/day converted from 5 ppm	No treatment-related effects after exposure for 15 days.	15- Day exposure regiment in a 90-day inhalation - Rat.
	UF=100	**Acute RfD =0.018 mg/kg/day**	
Chronic Dietary	1.13 mg/kg/day converted from 3 ppm	No treatment-related effects after chronic (52 weeks) inhalation exposure.	Chronic Toxicity Inhalation-Rat
	UF=100	**Chronic RfD =0.0113 mg/kg/day**	
Short-Intermediate or Long-Term (Dermal)	None	The use pattern does not indicate potential exposure via the dermal route. Therefore, dermal risk assessments are not required.	
Short Term (Inhalation)	0.007 mg/L	No treatment-related effects after exposure for 15 days.	15- Day exposure regiment in a 90-day inhalation - Rat.
	UF=100		
Intermediate (Inhalation)	NOEL= 0.004 mg/L	No evidence of toxicity at the highest tested concentration.	90-Day Inhalation - Rat
	UF=100		
Long-Term (Inhalation)	NOEL= 0.004 mg/L	No evidence of toxicity at the highest tested concentration.	Chronic Toxicity Inhalation - Rat
	0.004 mg/L		

IX. REFERENCES

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