SAFETY DATA SHEET

GALLIUM ARSENIDE OPTICAL CRYSTAL

According to Regulation (EC) No.1907/2006 (REACH)

1. IDENTIFICATION OF THE SUBSTANCE AND THE COMPANY

CHEMICAL NAME: Gallium Arsenide
SYNONYMS, TRADE NAMES: GaAs
DESCRIPTION: Solid Inorganic Polycrystalline pieces
USAGE: Optical Material for manufacture of Optical Components.
APPEARANCE: Greyish black opaque metallic solid. No odour
SUPPLIER: CRYSTRAN LTD, 1 Broom Road Business Park, Poole, Dorset UK BH12 4PA
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2. HAZARDS IDENTIFICATION

T Toxic by ingestion and inhalation with a danger of cumulative effects.

N Dangerous for the environment.

Class 6.1 **Poison**

Signal: Danger
H301 Toxic if swallowed
H331 Toxic if inhaled

Signal: Warning
H410 Very toxic to aquatic life with long lasting effects

Prevention:
P262 Do not breathe dust/fume/gas/mist/vapours/spray.
P264 Wash thoroughly after handling.
P270 Do not eat, drink or smoke when handling this product
P273 Avoid release to the environment.

Response:
P301+P310 IF SWALLOWED: Immediately call a poison centre or doctor. Rinse mouth.
P304+P312 IF INHALED: Call a poison centre or doctor/physician if you feel unwell.

3. COMPOSITION/INFORMATION ON INGREDIENTS

COMPONENT NAME	CAS number	%	EC number (EINECS)	EU index	UN number
Gallium Arsenide	1303-00-0	100%	215-114-8	033-002-00-5	1557

4. FIRST AID MEASURES

EYES: Irrigate thoroughly with water for at least 15 minutes. Obtain medical attention.
SKIN: Wash thoroughly with soap and water. Dry area with clean towel. Remove contaminated clothing and wash clothing before re-use.
INHALATION: Remove to fresh air. Perform artificial respiration if breathing has stopped. When breathing is difficult, properly trained personnel may administer oxygen. Keep affected person warm and at rest. Obtain medical attention.
INGESTION: Do not induce vomiting. Wash out mouth thoroughly with water and give 2 cups of water to drink. Do not give carbonated drinks. NEVER MAKE UNCONSCIOUS PERSONS VOMIT OR DRINK FLUIDS. Obtain medical attention immediately!

5. FIRE FIGHTING MEASURES

FLASH POINT: Not Ignitable. Not Applicable.
AUTO IGNITION TEMP: Not Applicable.
EXTINGUISHING MEDIA: As appropriate to the environment.
UNUSUAL FIRE HAZARDS: None known.

6. ACCIDENTAL RELEASE MEASURES

CONTAMINATION CLEANUP: Wear suitable protective clothing & equipment as listed under Exposure / Personal protection. Take up and containerize for proper disposal. Avoid making dust. Containerize any cleaning materials used for proper disposal.

7. HANDLING AND STORAGE

USAGE PRECAUTIONS: Keep away from heat. Avoid skin contact. Handle Carefully. Protect against physical damage. Avoid generating dust.
STORAGE PRECAUTIONS: Keep away from foodstuffs. Keep away from acids and strong bases.

8. EXPOSURE CONTROL AND PERSONAL PROTECTION

Protective gloves made of PVA are required. Use of a laboratory coat is suggested. Safety goggles or safety glasses with side shields are required if there is any possibility of chipping or dust creation. Respirators must be worn when the threshold limit is exceeded. Provide adequate general mechanical ventilation, and local exhaust ventilation.

OCCUPATIONAL EXPOSURE LIMITS (OEL) = 0.1 mg/m^3 in 8 hour Time Weighted Average (TWA)

9. PHYSICAL AND CHEMICAL PROPERTIES

APPEARANCE :	Grey-black metallic geometric shapes, no odour.
pH IN AQUEOUS SOLUTION:	Not determined
BOILING POINT (760mm Hg)	Not determined
MELTING POINT:	1238°C
FLASH POINT:	Not Applicable
FLAMMABILITY:	Not Applicable
EXPLOSIVE PROPERTIES:	Not Applicable
SPECIFIC GRAVITY:	5.31
VAPOUR PRESSURE:	Not determined
SOLUBILITY IN WATER:	Insoluble

10. STABILITY AND REACTIVITY

STABILITY:	Stable under normal conditions of storage and use.
HAZARDOUS DECOMPOSITION:	Contact with acids releases toxic gases. Arsine and oxides of arsenic can be formed.
MATERIALS TO AVOID:	Acids and oxidising agents.

11. TOXICOLOGICAL INFORMATION

TOXIC DOSE - LD50	4700 mg/kg
CARCINOGENICITY:	Some evidence of carcinogenicity. (NTP, IARC, ACGIH, OSHA)
MUTAGENICITY/TERATOGENICITY:	Refer to attached report.
TOXICOLOGICAL FINDINGS:	Refer to attached report. Particular care should be exercised when machining and creating dust or particles.

12. ECOLOGICAL INFORMATION

Do not allow product to reach ground water, water course or sewage system, even in small quantities. Danger to drinking water. Poisonous to fish. Only release to environment with proper government permits.

13. DISPOSAL CONSIDERATIONS

DISPOSAL METHODS: Chemical residues are generally classified as special waste, and are covered by regulations which vary according to location. Contact your local waste disposal authority for advice, or pass to a chemical disposal company.

14. TRANSPORT INFORMATION

Class	UN	Packing Group	Proper Shipping Name	Special
6.1	1557	II	Arsenic Compounds, Solid, N.O.S. (Gallium Arsenide)	Marine Pollutant

15. REGULATORY INFORMATION

Hazard Symbols:	T - Toxic	N - Dangerous for the environment
Risk Phrases:	23/25	Toxic by inhalation, in contact with skin and if swallowed.
	50/53	Very toxic to aquatic organisms. May cause long-term adverse effects in aquatic environment
Safety Phrases:	20/21	When using do not eat, drink or smoke
	28	After contact with skin, wash immediately with plenty of soap and warm water.
	44	If you feel unwell, seek medical advice immediately (show label where possible)

Note that the Risk and Safety Phrases included here for completeness are being replaced with the GHS Hazard and Precautionary statements given in section 2.

REACH: Refer to restrictions on the manufacture, placing on the market and use Annex XVII/19 EC/552/200 - 19. Arsenic Compounds.

16. OTHER INFORMATION

REVISION DATE: April 2013 Supercedes issue of April 2012 (Changes in Section: 2)

The above information is believed to be correct but does not purport to be all inclusive and must be used only as a guide.

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NTP Toxicology and Carcinogenesis Studies of Gallium Arsenide (CAS No. 1303-00-0) in F344/N Rats and B6C3F1 Mice (Inhalation Studies).

US National Toxicology Program Tech Rep Ser. 2000 Sep;492:1-306

Gallium arsenide is used primarily to make light- emitting diodes, lasers, laser windows, and photodetectors and in the photoelectronic transmission of data through optical fibers. Gallium arsenide was nominated for study because of its widespread use in the microelectronics industry, the potential for worker exposure, and the absence of chronic toxicity data. Male and female F344/N rats and B6C3F1 mice were exposed to gallium arsenide particles (greater than 98% pure; mass median aerodynamic diameter = 0.8 to 1.0 mg/m^3) by inhalation for 16 days, 14 weeks, or 2 years. Genetic toxicology studies were conducted in Salmonella typhimurium, and the frequency of micronuclei was determined in the peripheral blood of mice exposed to gallium arsenide for 14 weeks.

16-DAY STUDY IN RATS: Groups of five male and five female rats were exposed to particulate aerosols of gallium arsenide with a mass median aerodynamic diameter of approximately at concentrations of 0, 1, 10, 37, 75, or 150 mg/m^3 by inhalation, 6 hours per day, 5 days per week, for 16 days. All rats survived to the end of the study. The final mean body weights of all exposed groups of males and females were similar to those of the chamber controls. Compared to chamber controls, the liver and lung weights of males exposed to 1 mg/m^3 or greater and females exposed to 10 mg/m^3 or greater were increased; the thymus weights of all exposed groups of males were decreased. Gallium arsenide particles were visible in the alveolar spaces and, to a lesser extent, within alveolar macrophages of exposed rats. Moderate proteinosis (surfactant mixed with small amounts of fibrin) and minimal histiocytic cellular infiltrate were observed in the alveoli of exposed males and females. Epithelial hyperplasia and squamous metaplasia of the larynx were observed primarily in males exposed to 150 mg/m^3.

16-DAY STUDY IN MICE: Groups of five male and four or five female mice were exposed to particulate aerosols of gallium arsenide with a mass median aerodynamic diameter of approximately 1 &mgr;m at concentrations of 0, 1, 10, 37, 75, or 150 mg/m^3 by inhalation, 6 hours per day, 5 days per week, for 16 days. The final mean body weights were similar among exposed and chamber control groups. Compared to chamber controls, the lung weights of males and females exposed to 10 mg/m^3 or greater were increased. Gallium ar senide particles were visible in alveolar spaces and macrophages in some mice exposed to 150 mg/m^3. Moderate proteinosis, mild epithelial hyperplasia, and histiocytic infiltration of the lung were observed in males and females exposed to 10 mg/m^3 or greater. In the larynx, mild squamous metaplasia was seen in mice exposed to 10 mg/m^3 or greater, and mild chronic inflammation occurred in mice exposed to 75 or 150 mg/m^3.

14-WEEK STUDY IN RATS: Groups of 10 male and 10 female rats were exposed by inhalation to gallium arsenide particulate at concentrations of 0, 0.1, 1, 10, 37, or 75 mg/m^3, 6 hours per day, 5 days per week, for 14 weeks. All rats survived until the end of the study. The final mean body weight and body weight gain of males exposed to 75 mg/m^3 were significantly less than those of the chamber controls. Hematology and clinical chemistry results indicated that exposure to gallium arsenide induced a microcytic responsive anemia with an erythrocytosis and increased zinc protoporphyrin/heme ratios in exposed groups of rats. There were also increases in platelet and neutrophil counts, a transient decrease in leukocyte counts, and increases in the serum activities of alanine aminotransferase and sorbitol dehydrogenase. These changes were of greater magnitude in male rats. The lung weights of all exposed groups of rats were increased, while testis, cauda epididymis, and epididymis weights of males exposed to 37 or 75 mg/m^3 were generally less than those of chamber controls. Total spermatid heads and spermatid counts were significantly decreased in males exposed to 75 mg/m^3, while epididymal spermatozoa motility was significantly reduced in males ees exposed to 10 mg/m^3 or greater. Gallium arsenide particles were visible in alveolar spaces and macrophages in the lungs of exposed rats. Minimal to marked proteinosis and minimal histiocytic cellular infiltration of the alveoli were observed in all exposed groups; minimal squamous metaplasia in the larynx and lymphoid cell hyperplasia of the mediastinal lymph node were observed in some males and females exposed to 37 or 75 mg/m^3. Exposure-related increases in the incidences of plasma cell hyperplasia of the mandibular lymph node, testicular atrophy, epididymal hypospermia, bone marrow hyperplasia (males), and hemosiderosis in the liver were observed in the 37 and 75 mg/m^3 groups.

14-WEEK STUDY IN MICE: Groups of 10 male and 10 female mice were exposed by inhalation to gallium arsenide particulate at concentrations of 0, 0.1, 1, 10, 37, or 75 mg/m^3, 6 hours per day, 5 days per week, for 14 weeks. One female mouse exposed to 75 mg/m^3 died before the end of the study. Final mean body weights and body weight gains of males in the 75 mg/m^3 group were significantly less than the chamber controls. Hematology and clinical chemistry results indicated that exposure to gallium arsenide

affected the circulating erythroid mass and induced a microcytic responsive anemia with an erythrocytosis and increased zinc protoporphyrin/heme ratios in male and female mice. There were also increases in platelet and neutrophil counts. Compared to the chamber controls, the lung weights of males exposed to 1 mg/m^3 or greater and females exposed to 10 mg/m^3 or greater were increased. Testis, cauda epididymis, and epididymis weights, total spermatid heads, spermatid counts, and concentration and motility of epididymal spermatozoa were generally decreased. Gallium arsenide particles were visible in alveolar spaces and macrophages in the lungs of mice exposed to 1 mg/m^3 or greater. Mild to marked proteinosis, histiocytic infiltration, and epithelial hyperplasia were observed in the alveoli of males and females exposed to 1 mg/m^3 or greater. Minimal to mild suppurative inflammation and granuloma in the lung and squamous metaplasia in the larynx were present in males and females exposed to 10 mg/m^3 or greater. Min imal hyperplasia was observed in the tracheobronchial lymph node of males exposed to 10 mg/m^3 or greater and females exposed to 37 or 75 mg/m^3. Exposure- related increases in the incidences of testicular atrophy, epididymal hypospermia, hematopoietic cell proliferation of the spleen, and hemosiderosis of the liver and spleen were observed in groups of male and female mice exposed to 10 mg/m^3 or greater.

2-YEAR STUDY IN RATS: Groups of 50 male and 50 female rats were exposed by inhalation to gallium arsenide particulate at concentrations of 0, 0.01, 0.1, or 1.0 mg/m^3, 6 hours per day, 5 days per week, for 105 weeks. Survival and Body Weights: Survival of exposed male and female rats was similar to the chamber controls. Mean body weights of males exposed to 1.0 mg/m^3 were generally less than those of the chamber controls throughout the study; females exposed to 1.0 mg/m^3 had slightly lower mean body weights during the second year. Pathology Findings: Compared to the chamber controls, the incidences of alveolar/bronchiolar neoplasms were significantly increased in females exposed to 1.0 mg/m^3 and exceeded the historical control ranges. Exposure-related nonneoplastic lesions in the lungs of male and female rats included atypical hyperplasia, alveolar epithelial hyperplasia, chronic active inflammation, proteinosis, and alveolar epithelial metaplasia. In the larynx of males exposed to 1.0 mg/m^3, the incidences of hyperplasia, chronic active inflammation, squamous metaplasia, and hyperplasia of the epiglottis were significantly increased. The incidences of benign pheochromocytoma of the adrenal medulla occurred with a positive trend in female rats, and the incidence was significantly increased in the 1.0 mg/m^3 group and exceeded the historical control range. The incidence of mononuclear cell leukemia was significantly increased in females exposed to 1.0 mg/m^3 and exceeded the historical control range.

2-YEAR STUDY IN MICE: Groups of 50 male and 50 female mice were exposed by inhalation to gallium arsenide particulate at concentrations of 0, 0.1, 0.5, or 1.0 mg/m^3, 6 hours per day, 5 days per week, for 105 (males) or 106 (females) weeks. Survival and Body Weights: Survival of male and female mice was similar to the chamber controls. Mean body weights of exposed groups of males were similar to those of the chamber controls throughout the study; mean body weights of exposed groups of females were greater than those of the chamber controls from week 13 until the end of the study. Pathology Findings: Exposure-related nonneoplastic lesions in the lung of all groups of exposed mice included suppurative focal inflammation, chronic focal inflammation, histiocyte cellular infiltration, alveolar epithelial hyperplasia, and proteinosis. Increased incidences of minimal lymphoid hyperplasia of the tracheobronchial lymph node occurred in mice exposed to 1.0 mg/m^3 and in 0.5 mg/m^{3}mg/m^3 males.

GENETIC TOXICOLOGY: Gallium arsenide was not mutagenic in several strains of Salmonella typhimurium, with or without S9 metabolic activation enzymes, and no increase in the frequency of micronucleated erythrocytes was observed in peripheral blood of male or female mice exposed to gallium arsenide by inhalation for 14 weeks.

CONCLUSIONS: Under the conditions of these 2-year inhalation studies, there was no evidence of carcinogenic activity of gallium arsenide in male F344/N rats exposed to 0.01, 0.1, or 1.0 mg/m^3. There was clear evidence of carcinogenic activity in female F344/N rats based on increased incidences of benign and malignant neoplasms in the lung. Increased incidences of benign neoplasms of the adrenal medulla and increased incidences of mononuclear cell leukemia were also considered to be exposure related. There was no evidence of carcinogenic activity in male or female B6C3F1 mice exposed to 0.1, 0.5, or 1.0 mg/m^3. Exposure to gallium arsenide caused a spectrum of nonneoplastic lesions in the lung of rats and mice, the larynx of male rats and hyperplasia of the tracheobronchial lymph node in mice.