



Chemical Watch Factsheet

A Beyond Pesticides/ NCAMP Factsheet

Metam Sodium

Sodium methylthiocarbamate, usually called metam sodium, is a potent biocidal fumigant. Metam sodium causes birth defects and fetal death, and is a mutagen. In addition, it is highly toxic to fish and aquatic organisms. Marketed by ICI Americas (Vapam™), HASP (Metam-Fluid™), Amvac, and a host of others, metam sodium is used to sterilize soil before planting a variety of crops. It was virtually unheard of until the July 14, 1991 spill of 19,500 gallons into California's Sacramento River.

The pesticide decomposes in water to form a volatile compound, methyl isothiocyanate (MITC), which is extremely irritating to respiratory mucous membranes, eyes and lungs. Stringent precautions must be taken to avoid inhalation of the evolved gas. Metam sodium is quite acutely toxic, with an oral LD₅₀ of 146-280 mg/kg for albino mice and 450-820 mg/kg for albino rats.^{1,2} Metam sodium can react with sweating skin to form MITC. Prolonged or repeated contact may cause serious burns and intoxication, as seen in exposure to mustard gas.³ Speed is of the essence in removing material

from skin.

At least one researcher noted neurological effects. The clinical picture of poisoning in mice, rats and rabbits consisted of reduced motor activity, tremors and incoordination, while larger doses produced spasmodic twitching of the limbs.² Profuse salivation was the dominant symptom in cats. Yet, EPA does not consider metam sodium neurotoxic and

present both at high doses and at the lowest doses tested.

In the first rabbit teratogenicity study, doses of 0, 50, 100 or 200 mg/kg were administered⁵. There was a dose related increase in dead implantations reaching statistical significance at the 100 and 200 mg/kg dose levels.⁵

In the second rabbit teratogenicity study, doses of 0, 10, 30 or 100 mg/kg were administered.^{5,6} Developmental toxicity was apparent at the mid and high doses; exposed females had only 46% live fetuses in comparison to unexposed females with an average of 96% live fetuses.^{5,7} These results were statistically significant ($p < .01$).

chemicalWATCH Stats:

CAS Registry Number: 137-42-8

Chemical Class: Dithiocarbamate

Use: Non selective soil fumigant

Toxicity rating: Toxic

Signal Words: Caution, Warning, Danger

Health Effects: Listed as a carcinogen and developmental toxicant. It can cause birth defects and fetal death. Respiratory, eye and throat irritation, diarrhea, and rash are some symptoms of exposure to metam sodium.

Environmental Effects: Toxic to fish and other aquatic organisms. It has a high mobility in soil and rapidly degrades. It is not expected to bioconcentrate nor adsorb to sediment.

requires no further data.

Little is known about biological metabolism of metam sodium. But, labeled metam sodium was found in kidney, lung and bone marrow and not in tumor tissue in mice with lymphomas.⁴

Metam sodium is teratogenic, causing birth defects in the nervous system of rats and rabbits, and is probably teratogenic for the skeleton and other organs as well. The pesticide also is a reproductive toxin, drastically reducing the number of surviving fetuses that survive to birth in both rats and rabbits. Abnormalities were

The investigators noted that although the incidences of meningocele (incompletely formed skull) and spina bifida were low, with only two malformed fetuses found. These observations were also seen in the rat studies at higher levels, and there were none in either concurrent or historical controls. Other malformations were also present.

In the first Wistar rat teratogenicity study, doses of 0, 60, 120 or 240 mg/kg were administered.⁸

There were an increased number of dead implantations in the treatment groups. Teratogenic effects consisted of meningocele in 12 out of 291 fetuses (4.21%) in the 240 mg/kg group, compared to zero in the historical or concurrent control animals.

In the second rat teratogenicity study, with doses of 0, 10, 40 or 120 mg/kg, there was a significantly dose related increase in anomalies including skeletal variations related of the ribs, sternum and vertebral column as well as two cases of meningocele.^{8,9,10} There was a significant increase in the percent of live fetuses/dam at the 10 and 120 mg/kg level.

These results were statistically significant ($p < .05$).

It is important to note that the data was submitted to EPA in 1987, but was not reviewed until late in 1991 when the EPA was informed of the studies by California officials (PAY, Oct. '91).

Metam sodium is a mutagen. In cultures of human lymphocytes metam sodium caused increased frequencies of chromosomal aberrations with and without activation.^{11,12} In a second study the pesticide caused increased frequency of chromosome aberrations and of polyploidy in Chinese hamster bone marrow cells.^{11,13}

Metam sodium is highly toxic to fish.¹⁴ The Sacramento River spill killed hundreds of thousands of fish, and steril-

ized 45 miles of river. It is said not to be toxic to birds, LD50 > 5000 mg/kg.

Metam sodium is expected to display high mobility in soil and has the potential to leach into ground water.¹⁵ It is not expected to bioconcentrate in fish.¹⁶ It was detected in the water of 2 of 91 farms in an agricultural area of Canada.¹⁷

Breakdown of metam sodium and its metabolites is variable. One researcher found decomposition to MITC within 1-5 hours,¹⁸ but planting instructions give pre-plant intervals as long as 60 days.¹⁹

UPDATE: *September 2007*

Metam sodium is the third most commonly used agricultural pesticide (by weight) in the U.S.²⁰ It is a broad-spectrum soil fumigant that can be used to control plant parasitic nematodes, weeds, germinating weed seeds, and soil-borne plant pathogenic fungi affecting a variety of economically important fruit and vegetable crops. Metam sodium has been cited as a potential alternative to methyl bromide fumigation.

In one study, treatment of mice with metam sodium decreased survival following exposure to non-pathogenic *Escherichia coli* within 24-48 h, demonstrating that metam sodium may lead to a suppression of innate immunity²¹. In Earlimart, CA on November 13, 1999, a metam-sodium sprinkler application to a potato field led to a series of illnesses in the community. A temperature inversion and a shift in the prevailing winds resulted in 173 subjects being ill as a result of MITC exposure. Of the 173 subjects, 170 had MITC-compatible symptoms, including eye or upper respiratory irritation (77.6%), non-specific systemic symptoms (64.7%), and lower respiratory symptoms (20.0%)²². In another study, it was suggested that MITC was probably responsible for some of the immunological changes noted in metam sodium treated mice. It is also likely that the parent compound (metam sodium) or a synergistic action of the parent compound with one or more of the decomposition products (MITC, methylamine and carbon disulfide) is responsible for these remaining changes.

There is evidence that metam sodium can act as a contact sensitizer in humans, inducing allergic dermatitis. It also may exacerbate or induce respiratory allergy (asthma)²⁰. The EPA is expected to complete the Reregistration Eligibility Decision (RED) for metam sodium in September 2007.

Metam Sodium chemicalWATCH Factsheet Bibliography

- 1 Gosselin, R.E., Smith, R.P. and Hodge, H.C. 1984. Clinical toxicology of commercial products. Fifth ed. Williams & Wilkins, Baltimore, MD.
- 2 Nesterova, M.F. 1969. Standards for carbathion [methyl dithiocarbamate] in working zone air. Hygiene & Sanitation 34:33-37.
- 3 Schubert, H. 1978. Contact Dermatitis. 4:370.
- 4 Evans, R.G. 1982. Europ. Soc. Therap. Radiol. Oncol.132: June 28.
- 5 Dapson, S.C. and Ioannou, Y.M.,EPA/ OPP Toxicology Branch. 1991. Teratology - developmental toxicity species rabbit. Data evaluation record. Metam sodium (aqueous solution). Aug.
- 6 Hellwig, J. 1987. Report on the study of the prenatal toxicity of metam sodium (aqueous solution) in rabbits after oral administration (gavage). July 15.
- 7 Kieczka, H.1990. Metam-sodiuml teratogenicity in rabbits, summary. BASF Corp.
- 8 Whitby, K.E. and Swentzel, C. 1991. Teratology - developmental toxicity . species rat. Data evaluation record. Metam sodium (aqueous solution). Aug. .
- 9 Hellwig, J. and Hildebrand, B. 1987. Report on the study of the prenatal toxicity of metam sodium in rats after oral administration (gavage). March.
- 10 Stauber, F.1990. Metam sodium/teratogenicity in rats, summary. BASF Corp.
- 11 CDFA.1989. Metamsodium. Toxicology one-liners and conclusions.
- 12 BASF. 1987. Report on the in vitro cytogenetic investigations in human lymphocytes with metam sodium. FRG.
- 13 BASF. 1987a. Cytogenetic study in vivo of metam-sodium in Chinese hamsters bone marrow chromosome analysis. FRG.
- 14 Hartley, 1987. Agrochem. Handbook, 2nd ed
- 15 Swan, R.L. 1983. Res. Rev. 85:17-28.
- 16 Kenega, E.E. 1980. Ecotox. Env. Safety. 4:26-38.
- 17 Frank, R. et al. 1987. Arch. Environ. Contam. Toxicol. 16:1-8.
- 18 White-Stevens, R. 1971. Pesticides in the Environment, vol. 1, p. 224.
- 19 Thompson, W.T. 1983. Agricultural chemicals. III. Fumigants, Growth Regulators, Repellents, and Rodenticides.
- 20 Pruett SB., et al., 2001. Toxicology of metam sodium. J Toxicol Environ Health B Crit Rev. Apr-Jun;4(2):207-22.
- 21 Pruett SB. et al., 2005. Sodium methyldithiocarbamate inhibits MAP kinase activation through toll-like receptor 4, alters cytokine production by mouse peritoneal macrophages, and suppresses innate immunity. Toxicol. Sci. 87(1):75-85.
- 22 O'Malley M., et al. 2004. Modeling of methyl isothiocyanate air concentrations associated with community illnesses following a metam-sodium sprinkler application. Am J Ind Med. 46(1):1-15