

● FUMIGANT FACT SHEET

METAM SODIUM

Metam sodium is the most widely used soil fumigant, and the third most widely used pesticide in U.S. agriculture. Half of its use is in potato production, and 90 percent of its use is in Idaho, Washington, Oregon, and California.

Metam sodium acts as a fumigant by breaking down into methylisothiocyanate (MITC).

Symptoms of metam sodium poisoning in exposed people include burns, eye irritation, difficulty breathing, nausea, diarrhea, anxiety, and blurry vision. Poisonings have occurred as far as a mile from the application site.

In laboratory animals, metam sodium caused a wide variety of health effects. These include a reduction in the activity of immune system cells, a reduction in the levels of the hormone that triggers ovulation, a reduction in leg strength, a reduction in activity, anemia, damage to the lungs, and damage to the liver.

Both the U.S. Environmental Protection Agency (EPA) and the California EPA classify metam sodium as a carcinogen (a compound that causes cancer). These classifications are based on laboratory studies in which metam sodium exposure caused malignant tumors. California EPA also classifies metam sodium as a reproductive toxicant because it has caused pregnancy loss in laboratory studies.

Metam sodium commonly contaminates air in areas where it is used. A model developed by the California Department of Health Services estimated that almost 100,000 people in California are exposed to potentially damaging amounts of metam sodium in air.

Millions of fish were killed by a metam sodium spill in California. Low levels of metam sodium cause malformations in fish.

Metam sodium kills beneficial soil fungi and soil bacteria that cycle nitrogen, an important nutrient.

BY CAROLINE COX

Metam sodium (see Figure 1) is a soil fumigant that was developed in the 1950s.¹ It is “active against all living matter in the soil” and therefore acts as a fungicide, herbicide, insecticide, and nematocide simultaneously.² It is sold under a variety of brand names, including Vapam, Sectagon, and Sanafoam.³

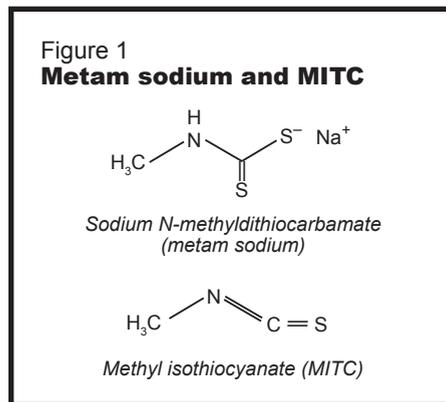
Use

Metam sodium is the most widely used soil fumigant in the U.S.⁴ and the third most widely used agricultural pesticide. Based on 2002 estimates, about 55 million pounds are used annually in the U.S. Use is increasing as metam sodium replaces the ozone-depleting fumigant methyl bromide.⁵

About half of the metam sodium



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used in the U.S. is used in potato production. An additional twenty percent is used in tomato production.² Almost 90 percent of U.S. metam sodium use occurs in Idaho, Oregon, Washington, and California.⁵

Metam sodium also has a few urban uses, including root control in sewer lines and treatment of utility poles.⁴

Metam sodium is applied at high rates; typical application rates in western

states are 150 to 300 pounds per acre.⁴

How Does Metam Sodium Kill Living Things?

According to the U.S. Environmental Protection Agency (EPA), metam sodium breaks down quickly to a molecule called MITC.⁶ (See Figure 1.) MITC is “responsible for the fumigant properties of metam sodium.”⁶ MITC inactivates certain parts of amino acids, the molecular building blocks from which proteins are made.²

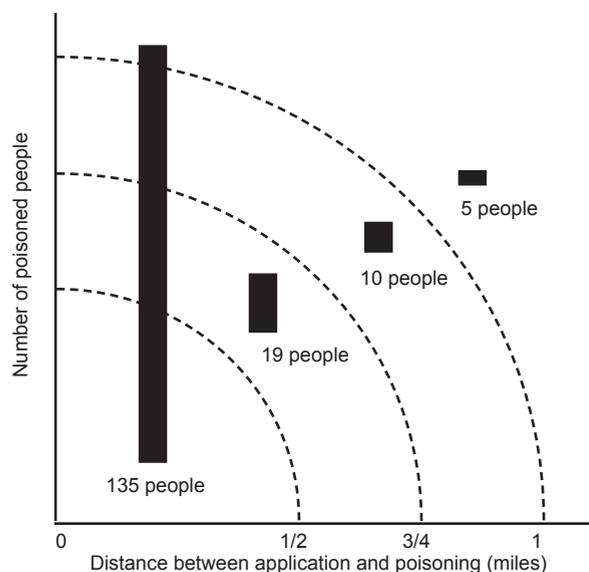
Breakdown Products

In addition to MITC, metam sodium breaks down into methyl isocyanate, carbon disulfide, and hydrogen sulfide.⁷ Some hazards of these compounds are discussed in “Effects on Pregnancy” and “Effects on Behavior,” p. 14.

Inert Ingredients

Most commercial metam sodium fumigants contain ingredients other than metam sodium. According to U.S.

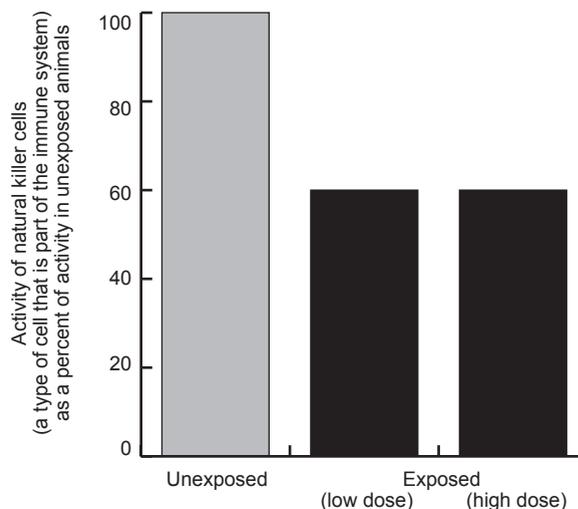
Figure 2
1999 Metam Poisoning Incident in California



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.* 46:1-15.

Metam sodium poisoning incidents have occurred more than a mile from the site where the fumigant was applied.

Figure 3
Effects of Metam Sodium on the Immune System



Padgett, E.L., D.B. Barnes, and S.B. Pruett. 1992. Disparate effects of representative dithiocarbamates on selected immunological parameters in vivo and cell survival in vitro in female B6C3F1 mice. *J. Toxicol. Environ. Health.* 37:559-571.

In laboratory animals, metam sodium exposure reduced activity of immune system cells called natural killer cells.

pesticide law, many of these ingredients are called “inert.”⁸

There is not much public information about the identity of these ingredients. In 2000, NCAP asked for information about inert ingredients in metam sodium products through the Freedom of Information Act. Manufacturers of most metam sodium products claimed that this information was confidential.⁹

Most studies conducted to satisfy registration requirements at EPA use metam sodium or MITC alone.¹⁰

Poisoning Symptoms

Based on calls to U.S. poison control centers, common symptoms of metam sodium poisoning include burns (both superficial and severe), eye irritation, headache, nausea, difficulty breathing, and vomiting. Other symptoms reported to EPA include sore throat, diarrhea, blisters, anxiety, blurry vision, and persistent breathing problems.¹¹

EPA has reports of over 700 metam sodium poisoning incidents.¹¹ Poisoning symptoms have been reported in people as far away as a mile from metam sodium

applications, and one incident in 1999 involved over 150 people.¹² (See Figure 2.) Poisoning symptoms occur at concentrations of metam sodium that are too low to have a noticeable odor.¹²

Effects on the Immune System

The immune system is a complex system that protects an individual from bacteria, viruses, and foreign substances.¹³ One important part of the immune system is the thymus, an organ that produces some immune system cells.¹⁴ A series of laboratory studies dating back more than a decade has shown that metam sodium and MITC have serious effects on the thymus and other parts of the immune system.

Led by a cellular biologist who has worked both at Mississippi State and Louisiana State Universities, the first studies showed that both oral and skin exposure to metam sodium reduced the size of the thymus and the activity of immune system cells called natural killer cells. The decrease in immune system activity occurred at all dose levels tested.^{15,16} (See Figure 3.)

Subsequent experiments showed that MITC reduced thymus size.¹⁷

Recent research showed that both metam sodium and MITC reduced the production of immune system compounds called cytokines. The researchers calculated that the effects of MITC in this experiment were caused by amounts that would be breathed in by a child near a metam sodium application.¹⁸ The researchers also identified the molecular mechanism through which metam sodium has an impact on cytokines.¹⁹

In EPA’s recent evaluation of metam sodium’s human health risks, the only discussion of immune system toxicity is one sentence stating that there is “some evidence that MITC may cause immunotoxicity at high oral and dermal doses.”²⁰

Effects on Hormones

Hormones are chemical messengers. The hormone system (also called the endocrine system) regulates all biological processes in humans and many other animals.²¹

Metam sodium’s effects on hormones

were first described in 1994 when EPA toxicologists working with laboratory animals showed that metam sodium stops the normal “surge” of a hormone that triggers ovulation. MITC had similar effects. Higher doses of metam sodium produce a greater reduction in the surge than lower levels.²²

Ignoring this research, EPA wrote in its recent evaluation of metam sodium’s toxicity that “it is notable that based on the available toxicology studies in metam sodium and MITC, there is no indication of endocrine disruption.”²³

Recent research from Louisiana State University showed an impact of metam sodium exposure on an additional hormone. In these laboratory studies, metam sodium caused an increase in the blood levels of a stress hormone. The increased levels of this hormone then caused atrophy of the thymus.²⁴

Carcinogenicity (Ability to Cause Cancer)

In laboratory studies sponsored by metam sodium manufacturers, exposure to this fumigant caused malignant

blood vessel tumors.^{14,25} (See Figure 4.) Based on these studies, EPA classified metam sodium as a “probable human carcinogen.”²⁵

EPA’s recent assessment of metam sodium risks concluded that most scenarios involving agricultural workers who apply metam sodium exceed the agency’s cancer risk guidelines. This is true even with maximum use of protective equipment and engineering controls to minimize exposure.²⁶

The California Environmental Protection Agency (CAL/EPA) also classifies metam sodium as a chemical known to cause cancer.²⁷

Mutagenicity (Ability to Cause Genetic Damage)

MITC caused abnormal chromosomes in a laboratory study sponsored by a metam sodium manufacturer.²⁸

Effects on Pregnancy

Laboratory studies sponsored by metam sodium manufacturers show that exposure to this pesticide can reduce pregnancy success.

One study showed that litters produced by exposed pregnant animals had fewer live offspring than litters produced by unexposed animals. This reduction occurred at all but the lowest dose level used in this experiment.²⁹

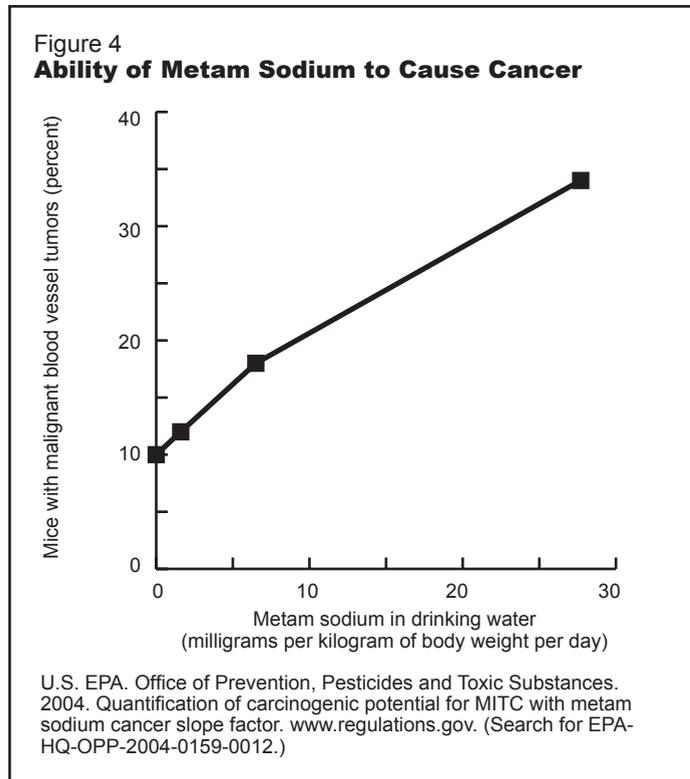
Another study showed that exposed pregnant animals had more early pregnancy failures than unexposed animals. The increase in pregnancy loss occurred at all but the lowest dose level tested in this experiment.³⁰ (See Figure 5.)

According to EPA, the metam sodium breakdown product carbon disulfide causes fetal loss. In addition, EPA reports that women exposed to methyl isocyanate (another breakdown product of metam sodium) following the notorious Bhopal, India pesticide accident had more spontaneous abortions than normally expected.³¹

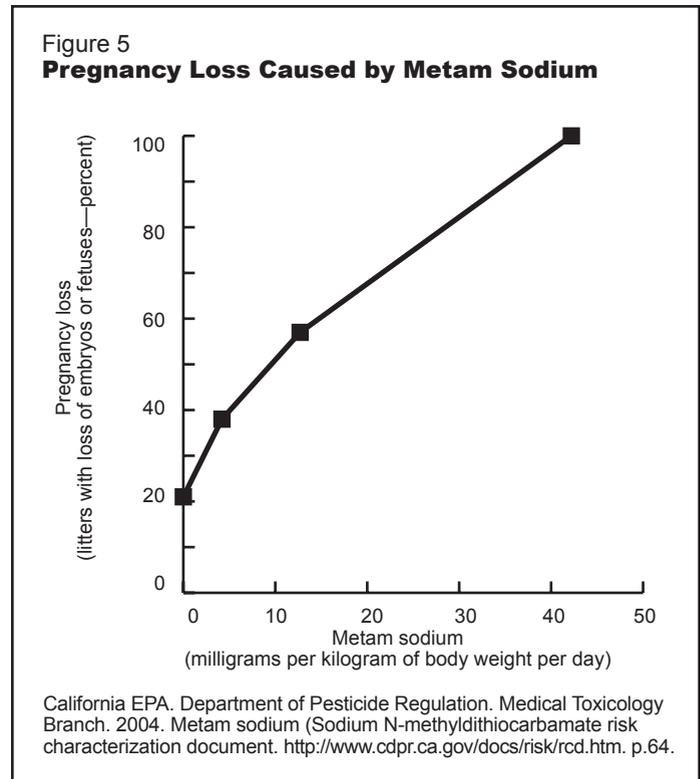
CAL/EPA classifies metam sodium as a chemical known to cause reproductive toxicity.²⁷

Effects on Behavior

A laboratory study sponsored by a

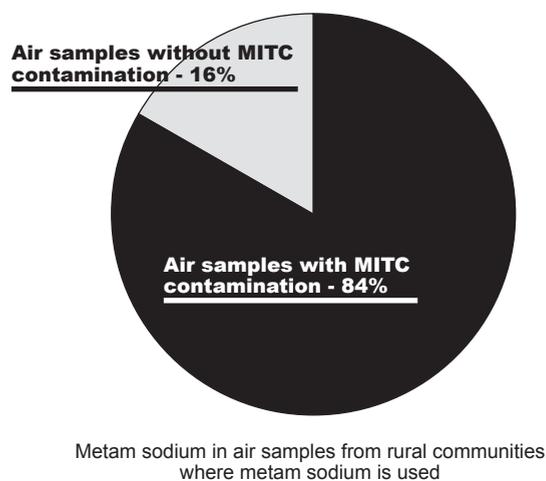


In laboratory animals, metam sodium exposure caused malignant tumors. Both U.S. EPA and California EPA classify it as a carcinogen.



Metam sodium caused pregnancy loss in exposed laboratory rabbits. California EPA classifies metam sodium as a reproductive toxicant.

Figure 6
Air Contamination by MITC



Lee, S. et al. 2002. Community exposures to airborne agricultural pesticides in California: Ranking of inhalation risks. *Environ. Health Perspect.* 110: 1175-1184.

In California, metam sodium frequently contaminates the air in rural communities where it is used.

metam sodium manufacturer showed that exposure to this fumigant affects behavior. An eclectic collection of behaviors were impacted: breathing, response to an approaching object, leg strength, walking, and motor activity.³²

The metam sodium breakdown product hydrogen sulfide causes other behavior changes: convulsions, dizziness, weakness, and irritability.³³

Anemia

Exposure to metam sodium can cause anemia. In laboratory studies sponsored by a metam sodium manufacturer, exposure caused a decrease in the numbers of red blood cells and in the oxygen-carrying molecule found in these cells. In one experiment this occurred at all dose levels tested.³⁴

Asthma

According to EPA, a metam sodium spill in California in 1991 resulted in both the development of new asthma cases and the worsening of existing asthma in people who lived or worked near the spill.³⁵

In addition, laboratory studies spon-

sored by a metam sodium manufacturer showed that inhaling metam sodium or MITC damages the respiratory system.³⁶

Liver damage

Metam sodium can damage the liver. Pathologists at Vanderbilt University Medical Center showed that a single oral dose of metam sodium caused liver injury and inflammation.³⁷

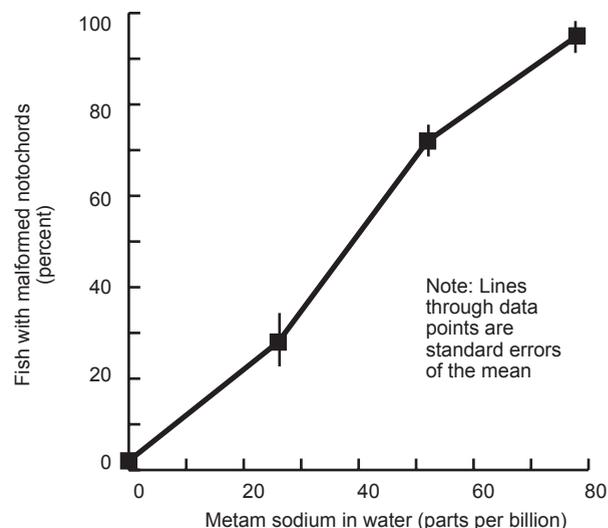
Liver damage also occurred in a test sponsored by metam sodium's manufacturer. In this study, hepatitis occurred at the two highest dose levels tested.³⁸

Contamination of Air

MITC is highly volatile³⁹ (easily turns into a gas) so it often contaminates air.

Recently, the California Department of Health Services assessed MITC air contamination by developing a model based on samples collected in areas where metam sodium has been used. (See Figure 6.) The agency's model estimated that over 50 percent of the people in these areas were exposed to levels of MITC above health guidelines. Almost 100,000 people in California

Figure 7
Ability of Metam Sodium to Cause Malformations in Fish



Haendel, M.A. et al. 2004. Developmental toxicity of the dithiocarbamate pesticide sodium metam in zebrafish. *Toxicol. Sci.* 81:390-400.

Low concentrations of metam sodium caused a nervous system malformation in zebrafish.

are exposed to potentially damaging amounts of MITC.⁴⁰

Water Contamination

Neither MITC nor metam sodium is included in the U.S. Geological Survey's national water quality monitoring program.⁴¹ This means that there is no systematic information about water contamination. However, EPA notes that metam sodium and MITC are "readily soluble in water and have low absorption into soil, thus these compounds can potentially leach into shallow ground water and leaky aquifers."⁴²

Effects on Fish

Recent research shows that metam sodium causes developmental toxicity in fish. (This means that exposed fish do not develop normally.) Scientists at Oregon State University showed that zebrafish embryos exposed to metam sodium developed nervous system malformations when exposed to concentrations as low as 26 parts per billion.⁴³ (See Figure 7.)

In addition, MITC causes death of fish at concentrations below 100 parts

per billion.⁴⁴

A 1991 metam sodium spill in California killed over a million fish in the Sacramento River.⁴⁵

A recent assessment done by fisheries biologists at the Institute of Ocean Sciences in British Columbia (Canada) looked at potential causes for a dramatic decrease in survival of sockeye salmon in the Fraser River. Three pesticides, including metam sodium, were classified as "high risk" in this analysis, along with a variety of other pollutants.⁴⁶

Effects on Other Animals

Metam sodium is toxic to a wide variety of animals. Examples include the following:

- Concentrations of a few parts per million of metam sodium kill oysters and shrimp.⁴⁷
- Concentrations of less than a hundred parts per billion of MITC kill water fleas.⁴⁷
- The 1991 Sacramento River spill reduced the number of salamanders in and around the river by more than 90 percent for at least three years.⁴⁸

Effects on Soils

Recent studies suggest that applications of metam sodium can impact many of the living organisms that are necessary for a healthy soil.

For example, a 2004 study from China Agricultural University looked at free-living, beneficial nematodes in soil. The researchers found that metam sodium fumigation reduced the numbers of free-living nematodes in tomato fields by as much as 80 percent.⁴⁹

Metam sodium can also inhibit mycorrhizal fungi (a kind of beneficial fungi) in soil. Research by the U.S. Dept. of Agriculture showed that fumigation reduced mycorrhizal fungi on sorghum. The reduction was as much as 98 percent in some of these experiments.⁵⁰ Impacts on mycorrhizal fungi were also found by scientists from Leiden University (The Netherlands).⁵¹

Fumigation with metam sodium also can change the bacterial community in soils, with persistent impacts on bacteria that cycle nitrogen, an important plant nutrient. In a study from Japan, the numbers of these bacteria were reduced by over 99 percent.⁵² ♣

References

1. Metam Sodium Task Force. 2000. Metam-sodium information sheet. www.metampsc.com/Documents/info.pdf.
2. U.S. EPA. Office of Prevention, Pesticides and Toxic Substances. 2004. Metam sodium/metam potassium: The HED chapter of the reregistration eligibility decision document (RED). www.regulations.gov. (Search for EPA-HQ-OPP-2005-0125-0003), p. 11
3. Washington State Univ. 2006. Pesticide Information Center OnLine. Query for metam sodium products. <http://picol.cahe.wsu.edu/labels/Labels.php>.
4. U.S. EPA. Office of Prevention, Pesticides and Toxic Substances. 2005. Overview of the use and usage of soil fumigants. www.epa.gov/oppsrrd1/reregistration/soil_fumigants/soil_fumigant_use.pdf, pp. 17-19.
5. U.S. EPA. Office of Prevention, Pesticides and Toxic Substances. 2005. Overview of the preliminary metam sodium risk assessment. www.regulations.gov (Search for EPA-HQ-OPP-2005-0125-0002.), p. 3.
6. Ref. # 5, p. 2.
7. Ref. # 2, p. 14.
8. Federal Insecticide, Fungicide, and Rodenticide Act § 2(a) and 2(m).
9. U.S. EPA. Office of Prevention, Pesticides and Toxic Substances. 2000. Response to Freedom of Information Act Request RIN-0961-00. (Nov. 2.) 40 Code of Federal Regulations §158.340.
10. U.S. EPA. Office of Prevention, Pesticides and Toxic Substances. 2003. Review of metam sodium incident reports. www.regulations.gov. (Search for EPA-HQ-OPP-2004-0159-0009.)
11. 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.* 46:1-15.
12. U.S. National Library of Medicine. National Institutes of Health. 2005. MedlinePlus medical encyclopedia: Immune response. www.nlm.nih.gov/medlineplus/ency/article/000821.htm.
13. Merriam-Webster. 2005. MedlinePlus medical dictionary. www.nlm.nih.gov/medlineplus/mplu-dictionary.html
14. Pruet, S.B. et al. 1992. Immunotoxicological characteristics of sodium methylthiocarbamate. *Fund. Appl. Toxicol.* 18:40-47.
15. Padgett, E.L., D.B. Barnes, and S.B. Pruet. 1992. Disparate effects of representative dithiocarbamates on selected immunological parameters in vivo and cell survival in vitro in female B6C3F1 mice. *J. Toxicol. Environ. Health.* 37:559-571.
16. Keil, D.E. et al. 1996. Role of decomposition products in sodium methylthiocarbamate-induced immunotoxicity. *J. Toxicol. Environ. Health* 47:479-492.
17. Pruet, S.B. et al. 2005. Sodium methylthiocarbamate inhibits MAP kinase activation through Toll-like Receptor 4, alters cytokine production by mouse peritoneal macrophages, and suppresses innate immunity. *Toxicol. Sci.* 87:75-85.
18. Pruet, S.B., R. Fan, and Q. Zheng. 2006. Involvement of three mechanisms in the alteration of cytokine responses by sodium methylthiocarbamates. *Toxicol. Appl. Pharmacol.* In press.
19. Ref. #2, p. 16.
20. U.S. EPA. 2006. Endocrine primer. www.epa.gov/scipoly/ospendo/edspoverview/primer.htm.
21. Goldman, J.M. et al. 1994. Blockade of ovulation in the rat by the fungicide sodium N-methylthiocarbamate: Relationship between effects on the luteinizing hormone surge and alterations in hypothalamic catecholamines. *Neurotoxicol. Teratol.* 16:257-268.
22. Ref. # 2, p. 31.
23. Myers, L.P. et al. 2005. Sodium methylthiocarbamate causes thymic atrophy by an indirect mechanism of corticosterone up-regulation. *J. Immunotoxicol.* 2:97-106.
24. Ref. # 2, p. 28.
25. Ref. # 2, p. 48-49.
26. California EPA. Office of Environmental Health Hazard Assessment. 2006. Chemicals known to the state to cause cancer or reproductive toxicity. February 3, 2006. www.oehha.ca.gov/prop65/prop65_list/files/P65single20306.pdf.
27. U.S. EPA. Office of Prevention, Pesticides and Toxic Substances. 2005. 4th revised toxicology disciplinary chapter for: Metam sodium (PC Code 039003) and methyl isothiocyanate (MITC, PC Code 068103). www.regulations.gov. (Search for EPA-HQ-OPP-2005-0125-0004.), pp. 59-60.
28. Ref. # 28, p. 37-38.
29. Ref. # 28, p. 38-40.
30. Ref. # 28, p. 65-69.
31. Ref. # 28, p. 60-62.
32. National Institute for Occupational Safety and Health. 2005. NIOSH pocket guide to chemical hazards: Hydrogen sulfide. www.cdc.gov/niosh/npg/npgd0337.html.
33. Ref. # 28, p. 23-27.
34. Ref. # 2, p. 31-34.
35. Ref. # 28, p. 30-31, 33-35.
36. Thompson, R.W., H.L. Valentine, and W.M. Valentine. 2002. In vivo and in vitro hepatotoxicity and glutathione interactions of N-methylthiocarbamate and N,N-dimethylthiocarbamate in the rat. *Toxicol. Sci.* 70:269-280.
37. California EPA. Department of Pesticide Regulation. Medical Toxicology Branch. 2004. Metam sodium (Sodium N-methylthiocarbamate risk characterization document.) <http://www.cdpr.ca.gov/docs/risk/rod.htm>, p.40.
38. U.S. EPA. Office of Prevention, Pesticides and Toxic Substances. 2004. Environmental fate and ecological risk assessment for the existing uses of metam-sodium. www.regulations.gov. (Search for EPA-HQ-OPP-2004-0159-0118.), p.1.
39. Lee, S. et al. 2002. Community exposures to airborne agricultural pesticides in California: Ranking of inhalation risks. *Environ. Health Perspect.* 110: 1175-1184.
40. U.S. Geological Survey. National Water-Quality Assessment (NAWQA) Program. 2003. USGS NAWQA constituents - pesticides. <http://water.usgs.gov/navqa/constituents/pesticides.html>.
41. Ref. # 39, p. 21.
42. Haendel, M.A. et al. 2004. Developmental toxicity of the dithiocarbamate pesticide sodium metam in zebrafish. *Toxicol. Sci.* 81:390-400.
43. Ref. # 39, p. 40-41.
44. Hankin, D.G. and D. McCanne. 2000. Estimating the number of fish and crayfish killed and the proportions of wild and hatchery rainbow trout in the Cantara spill. *Calif. Fish Game* 86:4-20.
45. Johannessen, D.I. and P.S. Ross. 2002. Late-run sockeye at risk: An overview of environmental contaminants in Fraser River salmon habitat. Canadian Tech. Rep. Fisheries Aquat. Sci. 2429. Fisheries and Oceans Canada. Institute of Ocean Sciences. p.81.
46. Ref. # 39, p. 41-44.
47. Luke, C. and D. Sterner. 2000. Possible effects of the Cantara spill on amphibian populations of the upper Sacramento River. *Calif. Fish Game* 86:41-60.
48. Zhi-Ping, C. et al. 2004. Impact of soil fumigation practices on soil nematodes and microbial biomass. *Pedosphere* 14:387-393.
49. Schreiner, R.P., K.L. Ivors, and J.N. Pinkerton. 2001. Soil solarization reduces arbuscular mycorrhizal fungi as a consequence of weed suppression. *Mycorrhiza* 11:273-277.
50. de Jong, F.M.W, E. van der Voet, and K.J. Canters. 1995. Possible side effects of airborne pesticides on fungi and vascular plants in The Netherlands. *Ecotoxicol. Environ. Safe.* 30:77-84.
51. Toyota, K. et al. 1999. Impact of fumigation with metam sodium upon soil microbial community structure in two Japanese soils. *Soil Sci. Plant Nutr.* 45(1):207-223.