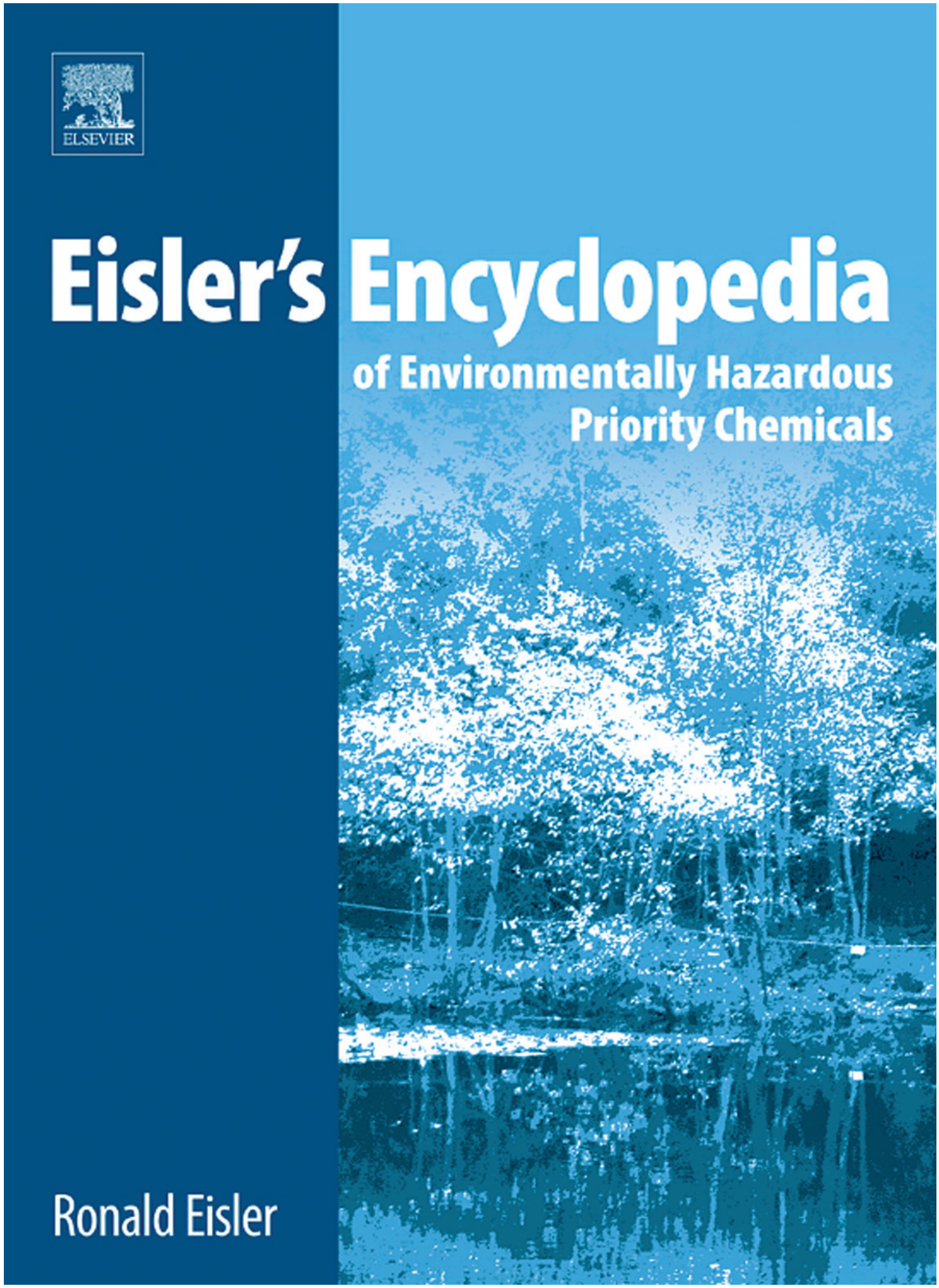
ELSEVIER
Eisler's Encyclopedia
of Environmentally Hazardous
Priority Chemicals
Ronald Eisler

EISLER'S ENCYCLOPEDIA OF

ENVIRONMENTALLY HAZARDOUS PRIORITY CHEMICALS

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EISLER'S ENCYCLOPEDIA OF

ENVIRONMENTALLY HAZARDOUS PRIORITY CHEMICALS

By

RONALD EISLER

ELSEVIER

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Dedicated to my family:

Jeannette, Renée, David, Charles, Julie, and Eb

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PREFACE

The chemicals featured in this volume are at the top of "List of Substances Discharged into the Environment as a Result of Human Activities and Considered Hazardous to Sensitive Species of Natural Resources." The List was prepared over the period 1985–2003 by environmental specialists of the U.S. Department of the Interior.

The metals, metalloids, organics, and radioactive substances chosen originated in wastes from agricultural, industrial, military, domestic, mining, and municipal sources. Some of these compounds were selected for inclusion because they had no known biological function and their presence in tissues is associated with adverse effects on growth, development, reproduction, and survival itself. Some have been incorporated into powerful biocides to control pestiferous organisms and, inadvertently, impact-desirable species of nontarget organisms. Others are highly prized by society, but the environmental consequences of extraction and refining them has adversely impacted habitats of plants and wildlife, sometimes for more than a hundred years. Several are essential to normal metabolism; however, insufficiency as well as excesses may be fatal. Most occur in a variety of chemical forms, some of which are comparatively benign and others extremely toxic. For each chemical or group of chemicals, basic information is presented on its sources, uses, properties, concentrations in living organisms, lethal and sublethal effects, identification of research opportunities, and proposed criteria to protect human health and natural resources.

It is emphasized that all proposed criteria listed were recommended by local, regional, national, and international regulatory agencies, as well as knowledgeable university and industrial researchers. In general, regulatory agencies are required to periodically update all criteria incorporating the most recent scientific findings. Unfortunately, criteria – unlike legislatively mandated standards – are not legally binding, although in certain extraordinary cases, such as massive discharge of a chemical to the biosphere, regulatory agencies are known to impose financial and other penalties. Ultimately, as chemical risk assessment predictions based on suitable databases become increasingly reliable, standards will be established for individual chemicals, together with adequate funds for enforcement, and stipulated penalties for violators.

This single volume compendium will provide a ready reference to professionals and students concerned with ecotoxicological aspects of numerous chemical wastes.

Ronald Eisler
2nd July 2007

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ABOUT THE AUTHOR

Ronald Eisler received the B.A. degree from New York University in biology and chemistry, and the M.S. and Ph.D degrees from the University of Washington in aquatic sciences and radioecology, respectively. He retired as a senior research biologist in 2004 after a 45-year career with the U.S. federal government, mainly with the U.S. Environmental Protection Agency in Rhode Island, and the U.S. Department of the Interior in the Territory of Alaska, New Jersey, Washington, D.C., and Maryland. He has held a number of special assignments and teaching appointments, including senior science advisor to the American Fisheries Society, adjunct professor of zoology at the American University in Washington, D.C., adjunct professor at the Graduate School of Oceanography of the University of Rhode Island, and visiting professor of marine biology and resident director of the Marine Biology Laboratory of Hebrew University in Eilat, Israel. Eisler is the author of approximately 150 research publications on ecotoxicological aspects of contaminants discharged into the environment as a result of human activities. In retirement, he continues to write and consult.

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BOOKS BY RONALD EISLER

Mercury Hazards to Living Organisms, 2006. CRC Press, Boca Raton, Florida, 312 pp.

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Trace Metal Concentrations in Marine Organisms, 1981. Pergamon Press, Elmsford, New York, 687 pp.

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ACROLEIN[a]

Chapter 1

1.1 Introduction

Acrolein (CH_2=CHCHO) is an aldehyde that was first isolated in 1843 from the dry distillation of fats and glycerol. It is now known that acrolein is ubiquitous in the environment; it is often present in trace amounts in foods and as a component of smog, fuel combustion products such as wood smoke, exhaust emissions from internal combustion engines, and cigarette smoke. Atmospheric concentrations of acrolein over urban areas are between 2.0 and 7.0 μg/L; cigarette smoke, however contains about 10,000 μg of acrolein/L. Acrolein is classified as a hazardous chemical because of its reactivity and flammability. At low sublethal concentrations, acrolein is widely known for its acrid pungent odor and strong irritating effects on mucous membranes of the eyes and upper respiratory tract, its toxicity to cilia in all organisms, and its interference with nucleic acid synthesis in bacteria. In bulk, acrolein during storage or transfer is potentially hazardous if it becomes overheated or contaminated with water. For example, in 1982, 17,000 residents from Toft, Louisiana, were evacuated when two large tanks of acrolein began to burn.

Acrolein enters the aquatic environment from its use as an aquatic herbicide, industrial discharges, and as a by-product of the chlorination of organic compounds in wastewater and drinking water treatment. Dilute solutions of acrolein kill undesirable plant life in irrigation streams and ditches and have been used routinely in about 4000 km of irrigation canals in southeastern Australia to control submerged weeds, including *Potamogeton tricarinatus*, *Elodea canadensis*, and *Vallisneria gigantia*. Acrolein has also been used for many years in channel maintenance in the United States (especially in western states), Canada, Egypt, Argentina, Mexico, and Turkey. Unlike most other aquatic herbicides, acrolein rapidly dissipates from water by volatilization and degradation without leaving phytotoxic residues. However, acrolein provides only temporary control of submerged weeds and also kills fish and other aquatic life at recommended treatment concentrations. In one Montana stream, acrolein killed all fish in a 4-km stretch after application to control submerged weeds; some fish deaths were recorded as far as 6.4 km downstream.

1.2 Sources and Uses

Acrolein enters the environment as a result of normal metabolic processes; incomplete combustion of coal, wood, plastics, tobacco, and oil fuels; and industrial emissions. Acrolein has been detected in smog, food, and water. It is used extensively in chemical manufacture, for control of fouling organisms, and as herbicide to control submerged weeds in irrigation canals.

1.2.1 Sources

Acrolein is ubiquitous in the environment as a result of natural and anthropogenic sources.

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[a]All information in this chapter is referenced in the following sources:

Eisler, R. 1994. Acrolein hazards to fish, wildlife, and invertebrates: a synoptic review. *U.S. Natl. Biol. Surv. Biol. Rep. 23*, 29 pp.

Eisler, R. 2000. Acrolein. Pages 739–766 in *Handbook of Chemical Risk Assessment: Health Hazards to Humans, Plants, and Animals.* Volume 2, Organics. Lewis Publishers, Boca Raton, Florida.

Sources of atmospheric acrolein include smog; incomplete combustion of coal, wood, gasoline, plastics and fats; tobacco smoke; and industrial emissions. The total amount of acrolein released into the atmosphere is unknown. In 1978, production losses of acrolein by emission from the four main U.S. plant locations were estimated at 34,682 kg; however, the gaseous emission streams are now either burned on emergence from the exhaust stack or sent to a furnace to destroy residual material. Acrolein is found in photochemical smog and contributes to the smog's irritant capacity to the eye and respiratory pathways. Recorded maximum acrolein concentrations in smog ranged from 12.0 to 14.0 μg/L (0.025–0.032 mg/m^3) in Los Angeles, between 1961 and 1963, and were 13.0 μg/L in Hudson County, New Jersey. For humans, exposure to atmospheric acrolein is greatest in the vicinity of incompletely combusted organic materials such as coal, wood, and petrol; highest acrolein concentrations are reported near forest fires and urban area fires. The burning of southern pine (*Pinus* sp.), for example, generates 22.0–121.0 mg of acrolein/kg of wood burned. Acrolein is also in the smoke of burning plastic materials. Air samples from more than 200 fires in Boston, Massachusetts, contained greater than 3000.0 μg acrolein/L (greater than 6.8 mg/m^3) in more than 10% of all samples; greater than 3000.0 μg acrolein/L air is an immediately hazardous concentration for human life and health. Cigarette smoke in some enclosed areas may account for as much as 12,400.0 μg of acrolein/L air. In the case of an enclosed room of 30 m^3 capacity, smoking 5 cigarettes raises the air concentration to about 50.0 μg acrolein/L and 30 cigarettes to 380.0 μg/L.

Acrolein is also generated when animal or vegetable fats are subjected to high temperatures. Acrolein was detected aboard submarines in trace concentrations as a degradation product during the heating of lubrication oils and edible fats. Large amounts of acrolein are generated from exhausts of internal combustion engines. Acrolein concentrations of 10,000.0 μg/L (23.0 mg/m^3) have been measured in nondiesel automobile exhausts, 2900.0 μg/L in diesel engine emissions, and 2600.0–9600.0 μg/L in other internal combustion engines. Acrolein concentrations in air from several U.S. urban areas ranged from a maximum of 10.0 μg/L in 1960 to 1.8–3.4 μg/L in 1968; during this period, the air in Tokyo had an average acrolein concentration of 7.2 μg/L. Urban acrolein pollution is derived mainly from automobile exhaust and incomplete burning of refuse. Acrolein is formed during normal metabolic degradation of spermine and spermidine, glycerol, allyl formate, allyl alcohol, and cyclophosphamide. Acrolein was also in spores from the wheat stem fungus (*Puccinia graminis*) of infected wheat (*Triticum aestivum*); acrolein was the major chemical factor that normally induced infection processes in *Puccinia*.

Acrolein has been detected in effluent water streams from industrial and municipal sources. Municipal effluents from Dayton, Ohio, for example, contained between 20.0 and 200.0 μg acrolein/L in 6 of 11 analyzed samples. Acrolein is also a component of many foods, and processing may increase the acrolein content. Acrolein has been identified in raw turkey, potatoes, onions, coffee grounds, raw cocoa beans, alcoholic beverages, hops, white bread, sugarcane molasses, souring salted pork, and cooked bluefin tuna (*Thunnus thynnus*).

Occupational exposure to acrolein may occur during its production and isolation as a chemical intermediate or during the manufacture of acrylic acid, acrylic acid esters, and methionine. Other sources of acrolein in the workplace include emissions from rubber vulcanization plants, welding of metals treated with anticorrosion primers, and pitch-cooking plants; and skin contact during herbicidal applications for aquatic weed control, and from its use as a slimicide in paper and paperboard manufacture. Acute acrolein poisoning from occupational exposure is improbable. However, the risks of poisoning are significant in certain industries including welding of fat and oil cauldrons, smelting work and foundry operations, printing plants, linoleum and oil cloth factories, manufacture of insulators, tin plating of sheet iron, and processing of linseed oil.

1.2.2 Uses

Since its discovery in 1843, acrolein has been known to polymerize readily in the presence of many chemicals, and since 1947 it has been used safely in a wide variety of commercial applications. Acrolein is presently produced by the catalytic oxidation of propylene for the manufacture of methionine, glutaraldehyde, 1,2,6-hexane thiol, and other chemicals. The largest quantity of acrolein produced by this process is converted directly to acrylic acid and acrylic acid esters. In 1975, global production of acrolein was 59,000 metric tons; in 1980, this value was 102,000 tons – including 47,600 tons produced by the United States. In 1983, about 250,000 tons (about 550 million pounds) of acrolein were produced, and 92% were converted to acrylic acid, 5% to methionine, and 3% used as an aquatic herbicide. Acrolein copolymers are used in photography, in textile treatment, in the paper industry, as builders in laundry and dishwater detergents, and as coatings for aluminum and steel panels. Acrolein is used to scavenge sulfides from oil-field floodwater systems, to cross-link protein collagen in the leather tanning industry, and to fixate tissue of histological samples. The use of acrolein as a military poison gas has been advocated because of its lacrimatory and blistering properties; during World War I (1914–18) the French used acrolein – under the name of Papite – in hand grenades because of its irritating effect on the respiratory airways and the ocular mucosae.

Acrolein has been used since 1960 to control submerged aquatic weeds in irrigation systems in the United States, Australia, and other countries where open channels distribute water for crop production. Acrolein – as Magnacide H herbicide – is applied directly into agricultural irrigation systems at 1.0–15.0 mg/L. Water in treated canals is required by the Magnacide H label to be held for 6 days before discharge into receiving waters. Acrolein is preferable to sodium arsenite for herbicidal control of submerged weeds because arsenicals are persistent (up to 1 year), and the high arsenic concentrations that are attained in water may be hazardous to humans and livestock. Acrolein is extremely effective in killing submerged weeds that are difficult to control with other herbicides. Acrolein has also been used as a herbicide in ponds, drains, and other water bodies. In Australia, the concentration of acrolein in irrigation canals to control various species of *Elodea*, *Potamogeton*, and *Vallisneria* is usually less than 15,000.0 µg/L. In general, acrolein has a low order of toxicity to terrestrial plants. Most field and garden crops can tolerate water with as much as 15,000.0 µg acrolein/L without serious adverse effects. Acrolein, as discussed later, has comparatively low persistence and low accumulation in aquatic ecosystems. One disadvantage of its use as a herbicide is its pungent, irritating odor. Also, at recommended treatment concentrations, acrolein kills fish and other aquatic organisms; therefore, acrolein should be used only in aquatic systems where these resources are considered expendable.

Acrolein has been used to control bacteria, fungi, algae, and mollusks in cooling water systems: 1500.0 µg/L killed as much as 95% of the target species in a once-through treatment. Acrolein has been applied directly to the marine environment to control the growth and settlement of mussels (*Mytilus edulis*), and other fouling organisms in cooling water systems of coastal steam electric station power plants. Mussels in marine cooling water systems are controlled with 600.0 µg acrolein/L for 8 h daily for 3 days or with 700.0 µg/L for 3 h daily for 2 weeks. Acrolein prevents growth of microorganisms in liquid fuels such as jet fuels, in feed lines of subsurface wastewater injectors, and in water conduits of paper manufacturing plants.

1.3 Environmental Chemistry

Acrolein, the simplest member of the class of unsaturated aldehydes, has a pungent, irritating odor. It is volatile, flammable, and explosive, and requires elaborate and specific conditions for storage and use. The half-time persistence of acrolein in freshwater is usually less than 50 h; in seawater it is less than 20 h, and in the atmosphere less than 3 h. Biochemical

and toxic effects of acrolein are caused by its rapid and essentially irreversible reaction with sulfhydryl compounds to form a stable thiol ether; however, many compounds can mitigate or block its toxicity. Acrolein is eventually metabolized to acrylic acid and glyceraldehyde; glycidaldehyde – an intermediate metabolite with mutagenic and carcinogenic properties – has been produced in vitro but not in vivo.

1.3.1 Chemical Properties

Acrolein is soluble in water and in many organic solvents including ethanol, acetone, and ether (Table 1.1). Acrolein is a highly reactive molecule with two reactive centers: one at the carbon–carbon double bond, and the other at the aldehydic group. Acrolein is extremely volatile, flammable, and explosive (Table 1.1), especially in sunlight or in the presence of alkali or strong acid. A potential hazard in handling acrolein is its rapid exothermic polymerization caused by the use of insufficient hydroquinone inhibitor or lack of strict control of pH. Commercial acrolein should be maintained at pH 6.0, contain less than 3% water, and 0.1–0.25% hydroquinone as a polymerization inhibitor. A typical commercial sample contains about 97% acrolein, 0.5% other carbonyls, and 2.5% water. The addition of hydroquinone (0.1–0.25%) prevents the vinyl polymerization of acrolein, and controlling the pH between 5 and 6 by acetic acid increases stability of commercial acrolein by preventing aldol condensation. Elaborate and specific conditions are now prescribed for the storage of acrolein and include vents and safety valves, construction materials, fire control, spills, and waste disposal. Commercial acrolein is stored and shipped under a blanket of oxygen-free inert gas.

Spectrophotometric determination with 4-hexyl-resorcinol and a fluorometric method with *m*-aminophenol are the most commonly

Table 1.1. Some properties of acrolein.

Variable	Datum
CHEMICAL NAME	2-Propenal
CAS NUMBER	107-02-8
STRUCTURAL FORMULA	CH_2=CHCHO
MOLECULAR WEIGHT	56.06
SPECIFIC GRAVITY	0.8427–0.8442
PHYSICAL STATE	Colorless or yellow liquid at 25°C
ODOR	Pungent, irritating
BOILING POINT	52.5–53.5°C
MELTING POINT	−86.95°C
SOLUBILITY	
Water	206.0–208.0 g/L
Organic solvents	Miscible
LOG K_{ow}	0.01
VAPOR PRESSURE	215–220 mm Hg at 20°C
EXPLOSIVE LIMITS OF VAPOR AND AIR	
Upper limit	31% acrolein
Lower limit	2.8% acrolein

used procedures for the determination of acrolein; however, gas chromatography and high-performance liquid chromatography procedures are also used. Acrolein concentrations in rainwater between 4.0 and 200.0 μg/L can be measured rapidly (less than 80 min) without interference from related compounds; the method involves acrolein bromination and analysis by gas chromatography with electron capture detection. Water samples from potential acrolein treatment systems require the use of water from that system, in preparing blanks, controls, and standards; further, acrolein measurements should be made at the anticipated use concentrations.

1.3.2 Persistence

Degradation and evaporation seem to be the major pathways for acrolein loss in water; smaller amounts are lost through absorption and uptake by aquatic organisms and sediments. The half-time persistence of acrolein in freshwater is 38 h at pH 8.6, and 50 h at pH 6.6; degradation is more rapid when initial acrolein concentrations are less than 3000.0 μg/L. Acrolein has a half-time persistence of 2.9–11.3 h at initial nominal concentrations of 20.0 μg/L, and 27.1–27.8 h at 101.0 μg/L. At pH 5, acrolein reacts by reversible hydrolysis to produce an equilibrium mixture with 92% beta-hydroxypropionaldehyde and 8% acrolein; in alkali, the primary reaction is consistent with a polycondensation reaction. Microbial degradation plays a major role in the transformation of acrolein in aquatic systems. In natural waters, acrolein degradation proceeds to carboxylic acid via a microbial pathway; beta-hydroxypropionaldehyde is readily biotransformed in about 17.4 days.

Acrolein is applied to irrigation canals to control submerged aquatic weeds at greatly different time–concentration treatments. Regardless of time–concentration regimens – which vary from 100.0 μg/L for 48 h in the United States to 15,000.0 μg/L for several hours in Australia – the daily decay-rate constants are remarkably similar, ranging from 0.14 to 0.21, and are probably affected by variations in weed density. In one case, acrolein applied to the Columbia River at an average initial concentration of 125.0 μg/L degraded to 25.0 μg/L after 48 h in samples greater than 65 km from the application point – a loss of 80%. High initial concentrations, 50,000.0–160,000.0 μg/L, of acrolein in natural waters degraded 57–80% in 192 h, suggesting that high concentrations can alter the rate of hydrolysis. In seawater, the half-time persistence of acrolein was less than 20 h. In photochemical smog, acrolein is comparatively unstable and not likely to persist; the dominant removal mechanism involves hydroxide attack on acrolein, and the atmospheric half-life persistence is 2–3 h under these conditions.

1.3.3 Metabolism

Biochemical and toxic effects of acrolein are probably caused by its reaction with critical protein and nonprotein sulfhydryl groups. The reaction of acrolein with sulfhydryl compounds is rapid and essentially irreversible, resulting in the formation of a stable thiol ether. Metabolism of acrolein is believed to result in the formation of acrylic acid and glyceraldehyde (Figure 1.1). The postulated metabolites of acrolein can be oxidized to carbon dioxide. Acrylic acid does not seem to represent a significant toxic hazard when compared to the parent acrolein because at low airborne concentrations of less than 1000.0 μg acrolein/L, the quantity of acrylic acid produced by metabolism is negligible. Thus, metabolism to acrylic acid after inhalation should be regarded as a detoxification pathway. Conjugation of acrylic acid with glutathione represents another elimination and detoxification pathway. In vitro studies of acrolein metabolism in mammals suggest that acrolein exposures may result in exposure to glycidaldehyde, an intermediate in acrolein metabolism (Figure 1.1). The major toxic effects of acrolein exposure – including irritation, ciliastasis, and hypersensitivity – are probably either due to the parent acrolein or to the reaction of glycidaldehyde with cell proteins. Glycidaldehyde is a potent mutagen and carcinogen; however, no evidence is

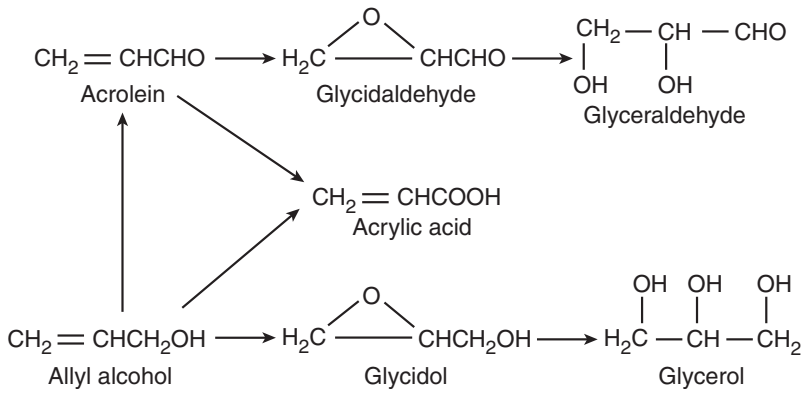

Figure 1.1.
Proposed scheme for in vitro mammalian metabolism of acrolein and allyl alcohol, a precursor of acrolein.

available showing that acrolein can produce glycidaldehyde in vivo. Acrolein is more toxic when inhaled than when taken orally. Inhalation of acrolein decreased the concentrations of protein and nonprotein sulfhydryl groups in nasal mucosal tissue. Acrolein is highly reactive towards thiol groups and rapidly conjugates with glutathione and cysteine. When glutathione is depleted, acrolein potentiates the nasal toxicity of formaldehyde to rats.

Acrolein is a metabolite of allyl alcohol and cyclophosphamide, and these compounds should be considered in acrolein metabolism schemes. Allyl alcohol in the presence of nicotinamide adenine dinucleotide phosphate (NADPH) and liver or lung microsomes degrades to acrolein, acrylic acid, and glycidol.

When added to water as an aquatic herbicide, acrolein undergoes rapid decomposition, especially in the sunlight. At the same time, it reacts rapidly with amines, alcohols, and mercaptans of aquatic plants, destroying cell structure and killing the plants. Mammals drinking acrolein-contaminated water rapidly convert acrolein to saturated alcohol compounds because of the low pH in the upper portion of their gastrointestinal tracts; the primary breakdown product is beta-propionaldehyde.

Many compounds including glutathione, 2-mercaptoethanol, beta-dimethylcysteamine, penicillamide, gamma-mercaptopropionylglycine, and *N*-acetylcysteine mitigate or block the toxic effects of acrolein. In frogs (*Rana japonica*), sulfhydryl compounds reduce the effects of acrolein on excitation–contraction uncoupling in skeletal muscle. In mice, cysteine reduced the cytotoxic effects of acrolein on tumor cells; in rabbits, cysteine mitigated acrolein-induced alveolar macrophage calcium-dependent ATP-ase, phagocytosis, and adhesiveness. In male rats, cysteine and ascorbic acid antagonized the acute lethal effects of orally administered acrolein, and 2-mercaptoethanol antagonized the inhibitory effect of acrolein on liver DNA-polymerase.

1.4 Lethal and Sublethal Effects

Acrolein degrades quickly in soils and in plant tissues regardless of mode of administration. Most terrestrial crop plants easily tolerate 25,000.0 μg of acrolein/L of irrigation water and some can tolerate 70,000.0–80,000.0 μg/L without adverse effects. Terrestrial plants were adversely affected at atmospheric concentrations of 500.0 μg acrolein/L air, but this concentration exceeds the recommended value of 110.0 μg/L (0.25 mg/m^3) air for protection of human health in occupational settings.

Adult fruit flies (*Drosophila* sp.) were comparatively resistant to acrolein and had lowered survival when reared in culture media with greater than 3,700,000.0 µg acrolein/L. At recommended concentrations for control of nuisance submerged aquatic weeds (frequently 100.0–1000.0 µg/L, often greater than 9600.0 µg/L), acrolein was lethal or harmful to almost all aquatic vertebrates and invertebrates tested in short-term exposures. The most sensitive groups of tested aquatic organisms in short-term assays were frog tadpoles (dead at 7.0 µg/L), representative species of fish (reduced survival at 14.0–62.0 µg/L), and crustaceans (death or immobilization at 34.0–80.0 µg/L). Adverse effects of acrolein on birds were observed at acute oral doses of 9100.0 µg/kg body weight (BW) (reduced survival), concentrations greater than 51.0 µg/kg egg for egg injection (abnormal development and reduced survival), and at greater than 50,000.0 µg/L air (respiratory tract histopathology). In mammals, acrolein is a strong cytotoxic and ciliostatic agent that is irritating to mucous membranes of dermal, ocular, gastrointestinal, and respiratory systems, and is systemically toxic by all routes of exposure. Adverse effects of acrolein are documented in sensitive species of mammals under the following regimens: 50.0 µg/L air for 1 min (increased blood pressure and heart rate); 300.0 µg/L air for 10 min (ocular and nasal irritation); 500.0–1000.0 µg/L air (repelled by odor); 660.0 µg/L air for 24 days (reduced survival); 8000.0–11,000.0 µg/L air for 4–6 h, or 875,000.0 µg/L air for 1 min (death); dietary concentrations equivalent to 500.0 µg/kg BW for 102 weeks (decreased survival); 850.0–6000.0 µg/kg BW by way of intravenous injection (liver necrosis, embryo resorption); and single oral doses between 4000.0 and 28,000.0 µg/kg BW (death).

Acrolein was mutagenic to certain microorganisms and to the fruit fly; mutagenicity may be due, in part, to glycidaldehyde, an acrolein metabolite. Injected into the amniotic fluid, acrolein is teratogenic to rats; teratogenicity may be due to acrylic acid, another acrolein metabolite. There is limited evidence that acrolein acts as a weak carcinogen and tumor promoter. Acrolein interacts with other chemicals, sometimes synergistically, additively, or antagonistically. Also, some chemicals normally contain acrolein as an impurity or yield acrolein as a metabolite.

1.4.1 Terrestrial Plants and Invertebrates

Most crop plants easily tolerate irrigation water with 25,000.0 µg of acrolein/L and many tolerate 70,000.0–80,000.0 µg/L without adverse effects – including corn (*Zea mays*), cotton (*Gossypium hirsutum*), milo (*Sorghum* spp.), squash (*Cucurbita* spp.), castor bean (*Ricinus communis*), tomato (*Lycopersicon esculentum*), alfalfa (*Medicago sativa*), and sugarcane (*Saccharum officinarum*). Acrolein degrades quickly in soils and plant tissues regardless of mode of administration. Atmospheric concentrations of 500.0 µg acrolein/L and higher were harmful to certain plants. Leaves of the pinto bean (*Phaseolus* spp.) and morning glory (*Ipomoea* spp.) developed brown foliar lesions after exposure to 500.0 µg/L air for 4–7 h; damage was more severe if the plants were moist during exposure. Leaves of the radish (*Raphanus* spp.) developed lesions after exposure to 1500.0 µg acrolein/L air for 6–7 h; however, leaves of the geranium (*Germanium* spp.) and the tomato showed no adverse effects after exposure to 1500.0 µg/L air for 7 h.

Acrolein inhibits DNA, RNA, and protein synthesis in the bacterium *Escherichia coli*, and this inhibition probably accounts for its cytotoxic and inhibitory effects on *E. coli* cell division. Acrolein is demonstrably mutagenic to microorganisms and to larvae of the fruit fly (*Drosophila melanogaster*). Acrolein-induced mutagenicity – including point mutations, sister chromatid exchanges, and chromosome breakages – has been observed in selected strains of bacteria (*E. coli*, *Salmonella typhimurium*), yeast (*Saccharomyces cerevisiae*), fruit fly larvae, and cultured Chinese hamster ovary cells. Acrolein's mutagenicity may be due to the metabolite glycidaldehyde: glycidaldehyde was mutagenic to bacteria and yeast under controlled conditions. Studies with

D. melanogaster show that acrolein is mutagenic in the sex-linked recessive lethal test when injected but not when fed. Acrolein caused 2.2% sex-linked mutations in *D. melanogaster* – the highest percentage recorded among several tested aldehydes. Early embryonic stages of fruit flies were most sensitive to the mutagenic properties of acrolein, and sensitivity decreased with increasing development to the point that adults showed negligible mutagenic responses. Adults of the fruit fly were generally resistant to acrolein; mortality was 25% when the culture medium contained 3,700,000.0 µg of acrolein/L, 50% at 8,600,000.0 µg/L, and 75% at 22,100,000.0 µg/L.

1.4.2 Aquatic Organisms

Adverse effects of acrolein on sensitive groups of aquatic organisms are documented at concentrations – in µg acrolein/L medium – as low as 7.0 for frog tadpoles (death), 14.0–62.0 for fish (death), 34.0–80.0 for crustaceans (death, immobilization), 50.0 for oysters (reduction in shell growth rate), 100.0–200.0 for freshwater algae (DNA and RNA reduction, photosynthesis inhibition), 151.0 for gastropods (death), >151.0 for insects (death), 500.0–2000.0 for macrophytes (leaf cell deterioration, death), 1250.0 for trematodes (death of miracidia in 20 min), and 62,000.0 for bacteria (growth reduction). Aquatic vertebrates were more sensitive than invertebrates, and younger fish were more sensitive than older fish. Aquatic insects do not avoid acrolein at concentrations that repel fish. Freshwater fishes and macroinvertebrates, when exposed under static conditions to sublethal concentrations of ^{14}C-labeled acrolein, metabolize acrolein so rapidly that neither acrolein nor its major oxidative and reductive metabolites (acrylic acid, allyl alcohol) were detected in edible tissues within 24 h after dosing.

As a herbicide, acrolein is most effective in controlling dense accumulations of submerged weeds in habitats where water flow is rapid and uniform, such as irrigation canals and rapidly flowing streams. Acrolein is lethal to various genera of submerged plants (*Hydrodictyon*, *Spirogyra*, *Potamogeton*, *Zannichellia*, *Cladophora*, *Ceratophyllum*, *Elodea*, *Chara*, *Najas*) at 1500.0–7500.0 µg/L. But some floating plants (*Pistia*, *Eichornia*, *Jussiaea*) are more resistant to acrolein than submerged plants and require concentrations that are at least double than those necessary for submerged forms. Also, acrolein has little effect on emergent aquatic macrophytes and should not be used to control emergents. Acrolein is the only herbicide now used in Australia for control of submerged aquatic weeds in larger irrigation canals; effective plant control was obtained at 9.6–28.8 mg/L for 3 h. In the United States, the U.S. Bureau of Reclamation controls aquatic algae and weeds at lower concentrations (0.1 mg/L) and longer exposures (48 h). In the Columbia River Basin in the state of Washington, acrolein is used to control submerged aquatic macrophytes at concentrations of 0.1 mg/L for 48 h or 1.0 mg/L for 4–8 h with applications every 3–5 weeks. Vegetation destruction by acrolein is maximal 1 week after application, and green filamentous algae are usually the first plants to return after 1 month. Biomass and species diversity were altered in acrolein-treated phytoplankton populations in Egyptian irrigation canals, 1 year after treatment. Although acrolein is a powerful cytotoxic agent, its inhibitory effects at sublethal concentrations on plant mitosis, nucleic acid synthesis, and protein synthesis are considered completely reversible.

Acrolein in concentrations sufficient to control nuisance submerged aquatic weeds may also kill snails, crayfish, shrimp, fish, and toads. In one case, acrolein was used to control *Potamogeton* and *Chara* in an Ohio farm pond during June. Acrolein was applied at 16,100.0 µg/L to a 0.1 ha portion of the 0.7-ha pond. Within 1 h of application, many dead amphibian tadpoles and small bluegills (*Lepomis macrochirus*) were recovered. In 24 h, *Chara* had turned white and *Potamogeton* brown; both plant species seemed dead; fish were swimming in the treated area. In 72–96 h, several large dead walleyes (*Stizostedium vitreum*) were found. One week posttreatment, all algae and weeds in the treated area were dead; weeds were