Ingredient name: Acetaldehyde

CAS No: 75-07-0

Datasheet No: 1333

OCCUPATIONAL SAFETY AND HEALTH GUIDELINE FOR ACETALDEHYDE

INTRODUCTION

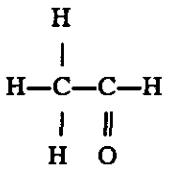
This guideline summarizes pertinent information about acetaldehyde for workers and employers as well as for physicians, industrial hygienists, and other occupational safety and health professionals who may need such information to conduct effective occupational safety and health programs. Recommendations may be superseded by new developments in these fields; readers are therefore advised to regard these recommendations as general guidelines and to determine periodically whether new information is available.

SUBSTANCE IDENTIFICATION

- **Formula**

CH_3CHO

- **Structure**



- **Synonyms**

Acetic aldehyde, ethanal, acetylaldehyde, ethyl aldehyde

- **Identifiers**

1. CAS No.: 75-07-0
2. RTECS No.: AB1925000
3. DOT UN: 1089 26
4. DOT label: Flammable Liquid

- **Appearance and odor**

Acetaldehyde is a colorless, flammable, volatile liquid or gas (above 69°F) with a pungent, fruity odor detectable at low concentrations. The odor threshold is reported to be between 0.05 and 2.3 parts per million (ppm) parts of air.

CHEMICAL AND PHYSICAL PROPERTIES

- **Physical data**

1. Molecular weight: 44.1
2. Boiling point (at 760 mm Hg): 21°C (69.8°F)
3. Specific gravity (water = 1): 0.79 at 20°C (68°F)
4. Vapor density (air = 1 at boiling point of acetaldehyde): 1.5
5. Melting point: -123.5°C (-190.3°F)
6. Vapor pressure at 20°C (68°F): 740 mm Hg
7. Solubility: Miscible with water, alcohol, ether, acetone, benzene, gasoline, solvent naphtha, toluene, turpentine, and xylene
8. Evaporation rate (ether = 1): 3

- **Reactivity**

1. Conditions contributing to instability: Contact of acetaldehyde with air may cause the formation of explosive peroxides, and contact of this substance with heat or flame may cause fires or explosions. Contact with trace metals or alkaline materials may cause acetaldehyde to undergo hazardous polymerization.

2. Incompatibilities: Fire and explosion may result from contact of acetaldehyde with strong oxidizers. Acetaldehyde reacts vigorously with acid anhydrides, alcohols, anhydrous ammonia, amines, ketones, phenols, hydrogen cyanide, hydrogen sulfide, halogens, phosphorus, isocyanates, and strong alkalies.

3. Hazardous decomposition products: Toxic gases (such as carbon monoxide and methane) may be released in a fire involving acetaldehyde.

4. Special precautions: Liquid acetaldehyde attacks some coatings and some forms of plastic and rubber.

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service Centers for Disease Control
National Institute for Occupational Safety and Health
Division of Standards Development and Technology Transfer

U.S. DEPARTMENT OF LABOR
Occupational Safety and Health Administration

• **Flammability**

The National Fire Protection Association has assigned a flammability rating of 4 (extreme fire hazard) to acetaldehyde.

1. Flash point: −37.8°C (−36°F) (closed cup)

2. Autoignition temperature: 175°C (347°F)

3. Flammable limits in air (% by volume): Lower, 4.0; upper, 60.0

4. Extinguishant: Use dry chemical, carbon dioxide, or alcohol foam to fight fires involving acetaldehyde. Water may be ineffective, but it may be used to keep fire-exposed containers cool and to protect persons attempting to stop the leak. If a leak or spill of acetaldehyde has not ignited, water spray may be used to disperse vapors.

Fires involving acetaldehyde should be fought upwind and from the maximum distance possible. Isolate the hazard area and deny access to unnecessary personnel. Emergency personnel should stay out of low areas and ventilate closed spaces before entering. Vapor explosion and poison hazards may occur indoors, outdoors, or in sewers. Vapors may travel to a source of ignition and flash back. Containers of acetaldehyde may explode in the heat of the fire and should be moved from the fire area if it is possible to do so safely. If this is not possible, cool containers from the sides with water until well after the fire is out. Stay away from the ends of containers. Personnel should withdraw immediately if they hear a rising sound from a venting safety device or if a container becomes discolored as a result of fire. Dikes should be used to contain fire-control water for later disposal. If a tank car or truck is involved in a fire, personnel should isolate an area of a half mile in all directions. Firefighters should wear a full set of protective clothing (including a self-contained breathing apparatus) when fighting fires involving acetaldehyde. Firefighters' protective clothing may provide limited protection against fires involving acetaldehyde.

EXPOSURE LIMITS

• **OSHA PEL**

The current Occupational Safety and Health Adminstration (OSHA) permissible exposure limit (PEL) for acetaldehyde is 100 ppm (180 mg/m^3) as an 8-hr TWA concentration and 150 ppm (270 mg/m^3) as a short-term exposure limit (STEL). A STEL is a 15-min TWA exposure which should not be exceeded at any time during the working day [29 CFR 1910.1000, Table Z-1-A].

• **NIOSH REL**

The National Institute for Occupational Safety and Health (NIOSH) considers acetaldehyde to be a potential occupational carcinogen and recommends exposures be controlled to the lowest feasible limit [NIOSH 1992].

• **ACGIH TLV®**

The American Conference of Governmental Industrial Hygienists (ACGIH) has assigned an acetaldehyde threshold limit value (TLV) of 100 ppm (180 mg/m^3) as a TWA for a normal 8-hr workday and a 40-hr workweek and a STEL of 150 ppm (270 mg/m^3) for periods not to exceed 15 min [ACGIH 1991b].

• **Rationale for limits**

The OSHA and ACGIH limits are based on the risk of conjunctivitis and sensory irritation of the respiratory tract associated with exposure to acetaldehyde. The NIOSH limit is based on positive carcinogenic results in animal studies.

HEALTH HAZARD INFORMATION

• **Routes of exposure**

Exposure to acetaldehyde can occur through inhalation, ingestion, or contact with the eyes, skin, or mucous membranes.

• **Summary of toxicology**

1. *Effects on Animals*: Acetaldehyde is an irritant of the eyes, mucous membranes, and upper respiratory tract in animals; at high concentrations, it is a central nervous system depressant. Acetaldehyde has caused squamous cell carcinomas and adenocarcinomas of the nasal cavity in rats and hamsters; it is also embryotoxic and teratogenic in several species of animals. Acetaldehyde causes severe irritation when applied to the eyes of rabbits; it causes mild irritation in contact with skin [NIOSH 1991]. In rats, the oral LD_{50} is 661 mg/kg, and the LC_{50} is 37 g/m^3 (20,550 ppm) for 30 min [NIOSH 1991]. Cats exposed to 380 ppm for 7 hr showed no effects; but increasing the concentration to 1,520 ppm caused signs of respiratory tract irritation [ACGIH 1991a]. Rats exposed to concentrations ranging from 400 to 5,000 ppm for 6 hr/day, 5 days/week for 4 weeks showed slight degeneration of the nasal epithelium at 400 ppm, and growth retardation, increased urinary output (in males only), and slight-to-moderate degeneration (with or without hyperplasia and metaplasia) of the nasal epithelium at 1,000 or 2,200 ppm [IARC 1985]. The rats exposed to 5,000 ppm showed severe growth retardation, increased neutrophil counts, reduced urine volumes, increased lung weights, and severe degenerative hyperplasia and metaplasia of the nasal, laryngeal, and tracheal epithelium [NLM 1992]. Hamsters were exposed to acetaldehyde concentrations ranging from 390 to 4,560 ppm for 6 hr/day, 5 days/week for 90 days. At the lowest concentration, these animals showed no toxic effects; at the highest concentration, however, they showed signs of eye and nose irritation, growth retardation, and erythrocytosis. At autopsy, these high-dose animals showed

increased kidney and heart weights and severe histopathologic changes of the respiratory epithelium [IARC 1985]. Fetal malformations (facial and cranial), digital anomalies, and embryonic deaths (resorptions) occurred in the offspring of rats and mice from dams treated with acetaldehyde during pregnancy [IARC 1985]. Acetaldehyde has been tested for carcinogenicity in rats by inhalation and in hamsters by inhalation and intratracheal administration. In rats, inhalation of acetaldehyde caused a statistically significant increase in the incidence of nasal adenocarcinomas and squamous cell carcinomas of the lungs. In hamsters, inhalation caused a significant increase in the incidence of laryngeal carcinoma, and intratracheal injections resulted in the induction of "peribronchiolar adenomated lesions," which were apparently not classified as tumors. The International Agency for Research on Cancer (IARC) has concluded that there is sufficient evidence for the carcinogenicity of acetaldehyde in animals [IARC 1987]. Acetaldehyde is mutagenic in bacterial and mammalian test systems with and without activation [NIOSH 1991].

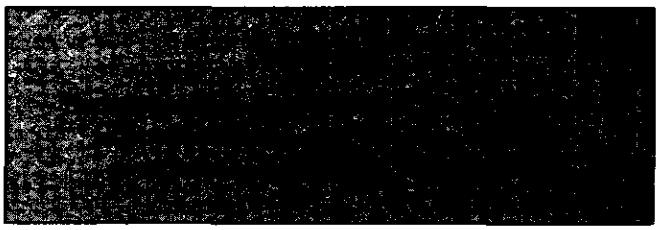
2. *Effects on Humans*: Acetaldehyde is an irritant of the eyes, mucous membranes, skin, and upper respiratory tract, and it is a central nervous system depressant in humans. On the basis of effects seen in animals, acetaldehyde is a potential carcinogen in humans. Although sensitive individuals experienced irritation when exposed to 25 ppm for 15 min, most unacclimated human volunteers exposed to 50 ppm experienced mild eye irritation, and all subjects exposed to 200 ppm developed conjunctivitis [Proctor et al. 1988]. Upper respiratory tract irritation was reported at a vapor concentration of 134 ppm [Proctor et al. 1988]. Eye contact with liquid acetaldehyde causes a burning sensation and superficial corneal injury; exposure to concentrations above 50 ppm may cause persistent tearing, photophobia, and injury to the corneal epithelium [Grant 1986]. Acetaldehyde causes erythema when splashed on the skin; if contact is repeated or prolonged, this substance may cause dermatitis or skin burns [Proctor et al. 1988]. A study of East German workers exposed to acetaldehyde and other chemicals in a chemical factory showed an increase in the number of cancers of the bronchial tubes and oral cavity. However, IARC has concluded that the results of this study are inconclusive because the workers were exposed to other chemicals and only a small, poorly defined population was involved [IARC 1987].

- **Signs and symptoms of exposure**

1. *Acute exposure*: Acute exposure to acetaldehyde can cause irritation of the eyes with burning, conjunctivitis, tearing, blurred vision, and photophobia; irritation and burning of the nose with rhinorrhea; and irritation of the upper respiratory tract with pain and coughing. Exposure to high levels of this substance may cause headache, drowsiness, dizziness, excitement, and agitation, followed by narcosis or stupor, pulmonary edema, and possibly death resulting from respiratory failure; however, ingestion can also induce nausea, vomiting, and diarrhea. Skin contact may cause dermatitis and burns of the exposed area.

2. *Chronic exposure*: Chronic exposure to acetaldehyde can cause conjunctivitis, coughing, difficult breathing, and dermatitis. On the basis of effects seen in animals, chronic exposure to acetaldehyde may cause heart and kidney damage, embryotoxicity, teratogenic effects, and possibly cancer in humans.

- **Emergency procedures**



Keep unconscious victims warm and on their sides to avoid choking if vomiting occurs. Initiate the following emergency procedures:

1. *Eye exposure*: Tissue irritation may result from exposure to concentrated solutions, vapors, mists, or aerosols of acetaldehyde. ***Immediately and thoroughly*** flush eyes with large amounts of water, occasionally lifting the upper and lower eyelids.

2. *Skin exposure*: Skin irritation may result. ***Immediately*** remove contaminated clothing and ***thoroughly*** wash contaminated skin with soap and water.

3. *Inhalation exposure*: If vapors, mists, or aerosols of acetaldehyde are inhaled, move the victim to fresh air ***immediately.***

If the victim is not breathing, clean any chemical contamination from the victim's lips and perform cardiopulmonary resuscitation (CPR); if breathing is difficult, give oxygen.

4. *Ingestion exposure*: Take the following steps if acetaldehyde or a solution containing it is ingested:

—Have the victim rinse the contaminated mouth cavity several times with a fluid such as water.

—Have the victim drink a glass (8 oz) of fluid such as water.

—Induce vomiting by giving syrup of ipecac as directed on the package. If ipecac is unavailable, have the victim touch the back of the throat with a finger until productive vomiting ceases.

—Do ***not*** force an unconscious or convulsing person to drink fluid or to vomit.

5. *Rescue*: Remove an incapacitated worker from further exposure and implement appropriate emergency procedures (e.g., those listed on the material safety data sheet required by OSHA's hazard communication standard [29 CFR 1910.1200]). All workers should be familiar with emergency procedures and the location and proper use of emergency equipment.

EXPOSURE SOURCES AND CONTROL METHODS

The following operations may involve acetaldehyde and may result in worker exposures to this substance:

—Synthesis of acetic acid, acetic anhydride, acrolein, aldol, butanol, butylene glycol, chloral, crotonaldehyde, 2-ethylhexanol, metaldehyde, paraldehyde, pentaerythritol, peracetic acid, pyridines, and trimethylolpropane

—Manufacture of synthetic resins, aniline dyes, herbicides, fungicides, other pesticides, explosives, and pharmaceuticals

—Synthesis of rubber processing chemicals, disinfectants, cosmetics, and perfumes

—Use of acetaldehyde in silvering mirrors and as an alcohol denaturant

—Use of acetaldehyde as a hardening agent in photography and in the manufacture of gelatin, glue, lacquers, varnishes, and casein products

—Use of acetaldehyde as a flavoring agent and additive in milk products and candies and as a preservative for food and leather

The following methods are effective in controlling worker exposures to acetaldehyde, depending on the feasibility of implementation:

—Process enclosure

—Local exhaust ventilation

—General dilution ventilation

—Personal protective equipment

Good sources of information about control methods are as follows:

1. ACGIH [1992]. Industrial ventilation—a manual of recommended practice. 21st ed. Cincinnati, OH: American Conference of Governmental Industrial Hygienists.

2. Burton DJ [1986]. Industrial ventilation—a self study companion. Cincinnati, OH: American Conference of Governmental Industrial Hygienists.

3. Alden JL, Kane JM [1982]. Design of industrial ventilation systems. New York, NY: Industrial Press, Inc.

4. Wadden RA, Scheff PA [1987]. Engineering design for control of workplace hazards. New York, NY: McGraw-Hill.

5. Plog BA [1988]. Fundamentals of industrial hygiene. Chicago, IL: National Safety Council.

MEDICAL MONITORING

Workers who may be exposed to chemical hazards should be monitored in a systematic program of medical surveillance that is intended to prevent occupational injury and disease. The program should include education of employers and workers about work-related hazards, placement of workers in jobs that do not jeopardize their safety or health, early detection of adverse health effects, and referral of workers for diagnosis and treatment. The occurrence of disease or other work-related adverse health effects should prompt immediate evaluation of primary preventive measures (e.g., industrial hygiene monitoring, engineering controls, and personal protective equipment). A medical monitoring program is intended to supplement, not replace, such measures. To place workers effectively and to detect and control work-related health effects, medical evaluations should be performed (1) before job placement, (2) periodically during the term of employment, and (3) at the time of job transfer or termination.

• Preplacement medical evaluation

Before a worker is placed in a job with a potential for exposure to acetaldehyde, a licensed health care professional should evaluate and document the worker's baseline health status with thorough medical, environmental, and occupational histories, a physical examination, and physiologic and laboratory tests appropriate for the anticipated occupational risks. These should concentrate on the function and integrity of the eyes, skin, and respiratory tract. Medical monitoring for respiratory disease should be conducted using the principles and methods recommended by the American Thoracic Society [ATS 1987].

A preplacement medical evaluation is recommended to assess an individual's suitability for employment at a specific job and to detect and assess medical conditions that may be aggravated or may result in increased risk when a worker is exposed to acetaldehyde at or below the prescribed exposure limit. The licensed health care professional should consider the probable frequency, intensity, and duration of exposure as well as the nature and degree of any applicable medical condition. Such conditions (which should not be regarded as absolute contraindications to job placement) include a history and other findings consistent with eye, skin, and respiratory tract diseases.

- **Periodic medical examinations and biological monitoring**

Occupational health interviews and physical examinations should be performed at regular intervals during the employment period, as mandated by any applicable Federal, State, or local standard. Where no standard exists and the hazard is minimal, evaluations should be conducted every 3 to 5 years or as frequently as recommended by an experienced occupational health physician. Additional examinations may be necessary if a worker develops symptoms attributable to acetaldehyde exposure. The interviews, examinations, and medical screening tests should focus on identifying the adverse effects of acetaldehyde on the eyes, skin, and respiratory tract. Current health status should be compared with the baseline health status of the individual worker or with expected values for a suitable reference population.

Biological monitoring involves sampling and analyzing body tissue or fluids to provide an index of exposure to a toxic substance or metabolite. Acetaldehyde can be detected in the blood, urine, and breath of exposed individuals. However, aldehyde concentrations in these biological specimens have not been correlated with airborne concentrations of this substance. Therefore, no biological monitoring method acceptable for routine use has yet been developed for acetaldehyde.

- **Medical examinations recommended at the time of job transfer or termination**

The medical, environmental, and occupational history interviews, the physical examination, and selected physiologic or laboratory tests that were conducted at the time of job placement should be repeated at the time of job transfer or termination. Any changes in the worker's health status should be compared with those expected for a suitable reference population. Because occupational exposure to acetaldehyde may cause diseases with prolonged latent periods, the need for medical monitoring may extend well beyond the termination of employment.

WORKPLACE MONITORING AND MEASUREMENT

A worker's exposure to airborne acetaldehyde is determined by using a silane-treated glass tube that is packed with a 225-mg backup section and a 450-mg sampling section of pretreated XAD-2 adsorbent coated with 10% (by weight) 2-(hydroxymethyl)piperidine. Samples are collected at a maximum flow rate of 0.05 liter/min until a maximum air volume of 3 liters is collected (8-hr TWA), or they are collected at a maximum flow rate of 0.05 liter/min until a maximum air volume of 0.75 liter is collected (STEL). Analysis is conducted by gas chromatography using a nitrogen/phosphorus detector. The limit of detection for this procedure is 580 parts per billion. This method is described in the OSHA Computerized Information System [OSHA 1990]. A similar method for sampling and analyzing acetaldehyde is included in Method 2538 of the *NIOSH Manual of Analytical Methods* [NIOSH 1984].

PERSONAL HYGIENE

If acetaldehyde contacts the skin, workers should immediately flush the affected areas with large amounts of water and then wash with soap and water.

Clothing and shoes contaminated with acetaldehyde should be removed immediately, and provisions should be made for safely removing this chemical from these articles. Persons laundering contaminated clothing should be informed of the hazardous properties of acetaldehyde, particularly its potential for causing skin or eye burns on prolonged contact.

A worker who handles acetaldehyde should thoroughly wash hands, forearms, and face with soap and water before eating, using tobacco products, or using toilet facilities.

Workers should not eat, drink, or use tobacco products in areas where acetaldehyde or a solution containing acetaldehyde is handled, processed, or stored.

STORAGE

Acetaldehyde should be stored in a cool, dry, well-ventilated area in tightly sealed containers that are labeled in accordance with OSHA's hazard communication standard [29 CFR 1910.1200]. Bulk quantities of acetaldehyde should be stored outside in detached, refrigerated tanks that conform to the requirements of the hazard communication standard. Storage in an inert gas atmosphere is recommended. Only nonsparking tools and equipment should be used to handle this OSHA Class IA flammable liquid. Containers of acetaldehyde should be protected from physical damage and should be stored separately from alkaline materials, acids, halogens, alcohols, ammonia, amines, ketones, hydrogen sulfide, hydrogen cyanide, acid anhydrides, phenols, oxidizing agents, and all ignition sources. All electrical service in storage areas should be of explosionproof design. To prevent static sparks, containers and equipment used to transfer acetaldehyde should be electrically grounded and bonded. Because empty containers may contain acetaldehyde residues, they should be handled appropriately.

SPILLS AND LEAKS

In the event of a spill or leak involving acetaldehyde, persons not wearing protective equipment and clothing should be restricted from contaminated areas until cleanup is complete.

The following steps should be undertaken following a spill or leak:

1. Do not touch the spilled material; stop the leak if it is possible to do so without risk.

2. Notify safety personnel.

3. Remove all sources of heat and ignition.

4. Provide maximum explosionproof ventilation to ventilate area of leak or spill.

5. Use nonsparking tools during cleanup.

6. Use water spray to flush spills away from workers and to dilute the spill.

7. Absorb small liquid spills with paper towels, vermiculite, or sand and place the material in a covered container for later disposal.

8. For large liquid spills, build dikes far ahead of the spill to contain the acetaldehyde for later reclamation or disposal.

SPECIAL REQUIREMENTS

U.S. Environmental Protection Agency (EPA) requirements for emergency planning, reportable quantities of hazardous releases, community right-to-know, and hazardous waste management may change over time. Users are therefore advised to determine periodically whether new information is available.

- **Emergency planning requirements**

Acetaldehyde is not subject to EPA emergency planning requirements under the Superfund Amendments and Reauthorization Act (SARA) [42 USC 11022].

- **Reportable quantity requirements for hazardous releases**

A hazardous substance release is defined by EPA as any spilling, pumping, pouring, emitting, emptying, discharging, injecting, escaping, leaching, dumping, or disposing of hazardous substances into the environment (including the abandonment or discarding of contaminated containers). In the event of a release that is above the reportable quantity for that chemical, employers are required by the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) [40 CFR 355.40] to notify the proper Federal authorities.

The reportable quantity for acetaldehyde is 1,000 lb. If an amount equal to or greater than this quantity is released within a 24-hr period in a manner that will expose persons outside the facility, employers are required to do the following:

—Notify the National Response Center ***immediately*** at (800) 424-8802 or at (202) 426-2675 in Washington, D.C. [40 CFR 302.6].

—Notify the emergency response commission of the State likely to be affected by the release [40 CFR 355.40].

—Notify the community emergency coordinator of the local emergency planning committee (or relevant local emergency response personnel) of any area likely to be affected by the release [40 CFR 355.40].

- **Community right-to-know requirements**

Employers who own or operate facilities in SIC codes 20 to 39, who employ 10 or more workers, and who manufacture 25,000 lb or more or otherwise use 10,000 lb or more of acetaldehyde per calendar year are required by EPA [49 CFR 372.30] to submit a Toxic Chemical Release Inventory Form (Form R) to EPA reporting the amount of acetaldehyde emitted or released from their facility annually.

- ***Hazardous waste management requirements***

EPA considers a waste to be hazardous if it exhibits any of the following characteristics: ignitability, corrosivity, reactivity, or toxicity as defined in 40 CFR 261.21-261.24. Acetaldehyde is listed as a hazardous waste under the Resource Conservation and Recovery Act (RCRA) [42 USC 6901 et seq.] and has been assigned EPA Hazardous Waste No. U001. This substance has been banned from land disposal and may be treated by fuel substitution or incineration. Acetaldehyde may also be disposed of in an organometallic or organic lab pack that meets the requirements of 40 CFR 264.316 or 265.316.

Providing detailed information about the removal and disposal of specific chemicals is beyond the scope of this guideline. The U.S. Department of Transportation, EPA, and State and local regulations should be followed to ensure that removal, transport, and disposal of acetaldehyde are conducted in accordance with existing regulations. To be certain that chemical waste disposal meets EPA regulatory requirements, employers should address any questions to the RCRA hotline at (800) 424-9346 or at (202) 382-3000 in Washington, D.C. In addition, relevant State and local authorities should be contacted for information about their requirements for waste removal and disposal.

RESPIRATORY PROTECTION

- **Conditions for respirator use**

Good industrial hygiene practice requires that engineering controls be used where feasible to reduce workplace concentrations of hazardous materials to the prescribed exposure limit. However, some situations may require the use of respirators to control exposure. Respirators must be worn if the ambient concentration of acetaldehyde exceeds

prescribed exposure limits. Respirators may be used (1) before engineering controls have been installed, (2) during work operations such as maintenance or repair activities that involve unknown exposures, (3) during operations that require entry into tanks or closed vessels, and (4) during emergencies. Workers should use only respirators that have been approved by NIOSH and the Mine Safety and Health Administration (MSHA).

- **Respiratory protection program**

Employers should institute a complete respiratory protection program that, at a minimum, complies with the requirements of OSHA's respiratory protection standard [29 CFR 1910.134]. Such a program must include respirator selection, an evaluation of the worker's ability to perform the work while wearing a respirator, the regular training of personnel, fit testing, periodic workplace monitoring, and regular respirator maintenance, inspection, and cleaning. The implementation of an adequate respiratory protection program (including selection of the correct respirator) requires that a knowledgeable person be in charge of the program and that the program be evaluated regularly. For additional information on the selection and use of respirators and on the medical screening of respirator users, consult the *NIOSH Respirator Decision Logic* [NIOSH 1987b] and the *NIOSH Guide to Industrial Respiratory Protection* [NIOSH 1987a].

PERSONAL PROTECTIVE EQUIPMENT

Gloves and protective clothing should be worn to prevent skin contact with acetaldehyde. Chemical protective clothing should be selected on the basis of available performance data, manufacturers' recommendations, and evaluation of the clothing under actual conditions of use. The following materials have been tested against permeation by acetaldehyde and have demonstrated good-to-excellent resistance: Teflon®, butyl rubber, and polyethylene/ethylene vinyl alcohol. Butyl rubber may provide more than 8 hr of resistance to permeation by acetaldehyde. Natural rubber, neoprene, nitrile rubber, polyethylene, polyvinyl alcohol, polyvinyl chloride, and Viton® have demonstrated poor resistance to permeation by acetaldehyde.

If acetaldehyde is dissolved in water or an organic solvent, the permeation properties of both the solvent and the mixture must be considered when selecting personal protective equipment and clothing.

Splashproof safety glasses, goggles, or face shields should be worn during operations in which there is any possibility of eye contact with acetaldehyde. Eyewash fountains and emergency showers should be available within the immediate work area whenever the potential exists for eye or skin contact with this substance. Contact lenses should not be worn if the potential exists for acetaldehyde exposure.

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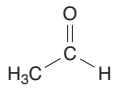
Acetaldehyde

CAS No. 75-07-0

Reasonably anticipated to be a human carcinogen

First listed in the *Sixth Annual Report on Carcinogens* (1991)

Also known as ethanal



Carcinogenicity

Acetaldehyde is *reasonably anticipated to be a human carcinogen* based on sufficient evidence of carcinogenicity from studies in experimental animals.

Cancer Studies in Experimental Animals

Exposure to acetaldehyde by inhalation caused tumors in two rodent species and at two different tissue sites. In rats of both sexes, it caused cancer of the nasal mucosa (squamous-cell carcinoma and adenocarcinoma), and in hamsters of both sexes, it caused cancer of the larynx (carcinoma) (IARC 1985, 1987). Inhalation of acetaldehyde also promoted the induction of respiratory-tract tumors by intratracheal instillation of the known carcinogen benzo[*a*]pyrene in hamsters of both sexes.

Since acetaldehyde was listed in the *Sixth Annual Report on Carcinogens,* an additional study in rats has been identified. Administration of acetaldehyde in drinking water increased the incidences of hemolymphoreticular cancer (leukemia and lymphoma combined), benign tumors of the pancreas (islet-cell adenoma), and cancer of the bone (osteosarcoma) and nasal cavity (carcinoma) in males and benign mammary-gland tumors (fibroma or fibroadenoma) in females (Soffritti *et al.* 2002). Increased incidences of tumors observed at other sites occurred only at one of the lower doses tested.

Cancer Studies in Humans

The data available from epidemiological studies are inadequate to evaluate the relationship between human cancer and exposure specifically to acetaldehyde. A survey of workers producing acetaldehyde and other aldehydes in Germany reported 9 cases of cancer, including 5 of lung cancer and 2 of oral-cavity cancer, among an unspecified number of workers; these incidences reportedly were higher than expected, but the observations were confounded by the fact that all cases of cancer occurred in tobacco smokers (IARC 1985, 1987).

Since acetaldehyde was listed in the *Sixth Annual Report on Carcinogens,* additional epidemiological studies have been identified, primarily case-control studies of populations exposed to acetaldehyde (the main initial metabolite of alcohol) following consumption of alcoholic beverages. Alcoholic beverage consumption is listed in the Report on Carcinogens as *known to be a human carcinogen*. In its 1999 review, the International Agency for Research on Cancer noted that three small case-control studies found increased risks of alcohol-related cancer (of the oral cavity, pharynx, larynx, and esophagus) among individuals with genetic variations (polymorphisms) that result in increased levels of acetaldehyde after alcohol consumption. However, IARC concluded that the data available were inadequate to evaluate the carcinogenicity of acetaldehyde (IARC 1999). Since then, a number of review articles and meta-analyses have summarized the results of subsequent studies that found dose-response relationships between alcohol consumption and cancer of the oral cavity, pharynx, larynx, and esophagus, and possibly the stomach and colorectum, among individuals with genetic polymorphisms that increase blood or salivary levels of acetaldehyde (Bagnardi *et al.* 2001, Zeka *et al.* 2003, Boffetta and Hashibe 2006, Baan *et al.* 2007, Boccia *et al.* 2009, Salaspuro 2009). In 2009, IARC concluded that acetaldehyde associated with alcohol consumption was carcinogenic to humans (Secretan *et al.* 2009). Few studies have been conducted on the association of these polymorphisms with cancer at other tissue sites, and the role of acetaldehyde in pancreatic, liver, bladder, or breast cancer is not clear (van Dijk *et al.* 2001, Terry *et al.* 2006, Seitz and Becker 2007, Visvanathan *et al.* 2007, Druesne-Pecollo *et al.* 2009).

Studies on Mechanisms of Carcinogenesis

Alcohol is metabolized to acetaldehyde by alcohol dehydrogenases (ADH), and acetaldehyde is metabolized to acetic acid by aldehyde dehydrogenases (ALDH). In some individuals, genetic polymorphisms in these enzymes can result in either higher rates of acetaldehyde production from alcohol or lower rates of acetaldehyde metabolism to acetic acid, resulting in higher blood acetaldehyde levels after a given level of alcohol intake than in individuals without these polymorphisms. Five ADH genes have been identified in humans, two of which have been shown to be polymorphic. The variant allele of the *ALDH2* gene, which is prevalent in Asians, encodes an enzyme that has almost no ability to detoxify acetaldehyde (IARC 1999).

Properties

Acetaldehyde is an aliphatic aldehyde that exists at room temperature as a colorless gas with a fruity, pungent odor. It is miscible with water, ether, benzene, gasoline, solvent naphtha, toluene, xylene, turpentine, and acetone. It is very flammable and is unstable in air (Akron 2009, HSDB 2009). Physical and chemical properties of acetaldehyde are listed in the following table.

Property	Information
Molecular weight	44.0[a]
Specific gravity	0.79 at 16°C/4°C[a]
Melting point	–124°C[a]
Boiling point	21°C[a]
Log K_{ow}	–0.34[b]
Water solubility	1,000 g/L at 25°C[a]
Vapor pressure	902 mm Hg at 25°C[a]
Vapor density relative to air	1.5[a]
Dissociation constant (pK_a)	13.6 at 25°C[a]

Sources: [a]HSDB 2009, [b]ChemIDplus 2009.

Use

Acetaldehyde is used primarily as a chemical intermediate in the production of acetic acid, pyridine and pyridine bases, peracetic acid, pentaerythritol, butylene glycol, and chloral. It is also used in the synthesis of crotonaldehyde, flavor and fragrance acetals, acetaldehyde 1,1-dimethylhydrazone, acetaldehyde cyanohydrin, acetaldehyde oxime, various acetic acid esters, paraldehyde, metaldehyde (a molluscicide widely used to kill slugs and snails), polymers, and various halogenated derivatives (IARC 1985, 1999). Acetaldehyde has been used in the manufacture of aniline dyes, plastics, and synthetic rubber, to silver mirrors, and to harden gelatin fibers. It has also been used in the production of polyvinyl acetal resins, in fuel compositions, to inhibit mold growth on leather, and in the manufacture of disinfectants, pesticides, drugs, explosives, lacquers and varnishes, photographic chemicals, phenolic and urea resins, and rubber accelerators and antioxidants (EPA 1994).

Acetaldehyde is considered by the U.S. Food and Drug Administration to be generally recognized as safe for use as a flavoring agent and adjuvant (Furia and Bellanca 1975, HSDB 2009). It is an important component of food flavorings and is added to milk products, baked

goods, fruit juices, candy, desserts, and soft drinks; it is especially useful for imparting orange, apple, and butter flavors. The concentration of acetaldehyde in food generally is up to 0.047%. In 1976, about 8,600 kg (19,000 lb) of acetaldehyde was used as food additives. Acetaldehyde is also used in the manufacture of vinegar and as a fruit and fish preservative. It is approved for use in phenolic resins in molded containers for contact with non-acidic foods. Acetaldehyde is no longer registered as an active ingredient in any pesticide. When it was used as a fumigant for storage of apples and strawberries, it was exempted from a residue tolerance (IARC 1985, EPA 1994, HSDB 2009).

Production

Acetaldehyde was first produced commercially in 1916, and its U.S. production peaked at 1.65 billion pounds in 1969 (IARC 1985). In 2015, combined U.S. production and imports were in the range of 250 million to 500 million pounds (EPA 2016), similar to the range of 100 million to 500 million pounds reported from 1994 to 2002 (EPA 2004). Data on U.S. imports and exports of acetaldehyde indicated that although exports have decreased substantially from the 42.6 million pounds reported in 1989 (USITC 2009), they have continued to greatly exceed imports (as shown in the table below). In 2009, acetaldehyde was available from 49 suppliers, including 21 U.S. suppliers (ChemSources 2009).

Category	Year	Quantity (lb)
Production + imports[a]	2015	250 million to 500 million
U.S. imports[b]	2017	177,000
U.S. exports[b]	2017	4.5 million

Sources: [a]EPA 2016. [b]USITC 2018.

Exposure

There is high potential for exposure of the general population to acetaldehyde through ingestion, inhalation, and dermal contact and of workers through inhalation and dermal contact. The main source of exposure of the general population is through consumption of alcoholic beverages and the subsequent metabolism of alcohol to form acetaldehyde (HSDB 2009). Because acetaldehyde may form in wine and other alcoholic beverages after exposure to air (Hagemeyer 2002), alcoholic beverages (including wines, beer, and spirits) also frequently contain acetaldehyde as a volatile component (HSDB 2009).

Acetaldehyde is a product of most hydrocarbon oxidation reactions and is a normal intermediate in the respiration of most higher plants. It is found in trace amounts in many plant products, including apples, broccoli, coffee, grapefruit, grapes, lemons, mushrooms, onions, oranges, peaches, nectarines, pears, pineapples, raspberries, strawberries, cranberries, sour cherries, and mango. It has been detected in the essential oils of alfalfa, rosemary, balm, clary sage, daffodil, bitter orange, camphor, angelica, fennel, mustard, peppermint, and lychee, and in oak and tobacco leaves and cotton leaves and blossoms (IARC 1985, Burdon *et al.* 1996, Gorny *et al.* 1999, Gunes *et al.* 2002, Bonerz *et al.* 2007, Mahattanatawee *et al.* 2007). Acetaldehyde has also been detected in breast milk. Consumers may be exposed to acetaldehyde in many milk products, including all types of cheese, yogurt, and milk of varying fat content (Mistry and Hassan 1992, Barbieri *et al.* 1994, Jandal 1996, Beshkova *et al.* 1998, Van Aardt *et al.* 2001, Kondyli *et al.* 2002, Boscaini *et al.* 2003, Di Cagno *et al.* 2004, Fernandez-Garcia *et al.* 2004, Blagden and Gilliland 2005, Gadaga *et al.* 2007, Kaminarides *et al.* 2007). Acetaldehyde has also been detected in cooked beef, chicken, and fish (HSDB 2009, Yasuhara and Shibamoto 1995) and is used as a synthetic flavoring ingredient in processed foods, especially margarine (HSDB 2009).

According to EPA's Toxics Release Inventory, environmental releases of acetaldehyde have increased slightly since 1988, when 9.5 million pounds was released, 73% to air, 23% to underground injection wells, and the remainder to surface water and landfills. Since then, releases to underground injection wells have decreased, and releases to surface water have increased. In 2007, 11.4 million pounds of acetaldehyde was released from 336 facilities that processed, produced, or used the chemical; 29 facilities each released more than 100,000 lb. Of the total amount, 94% was released to air, 3.1% to underground injection wells, and 2.8% to water (TRI 2009). Acetaldehyde will volatilize rapidly from water or land, and it will leach into the ground, where it will biodegrade (HSDB 2009). Acetaldehyde is also degraded readily in soil, sewage, and natural waters by microorganisms (EPA 1987).

Acetaldehyde is a natural product of photooxidation of hydrocarbons commonly found in the atmosphere and occurs naturally as emissions from forest fires, volcanoes, and animal wastes. In the 1990s, annual emissions of acetaldehyde from all sources in the United States were estimated at 12.1 million kilograms (27 million pounds) (IPCS 1995). Burning wood produces acetaldehyde at approximately 0.7 g/kg of wood, and fireplace emissions range from 0.083 to 0.20 g/kg of wood burned (HSDB 2009). In the 1990s, annual emissions from residential burning in the United States were estimated at 5,000 metric tons (11 million pounds) (IPCS 1995). Acetaldehyde is also a combustion product of some plastics (e.g., polycarbonate) and some hard and soft polyurethane foams. It also occurs in gasoline exhaust (1.4 to 8.8 mg/m^3) and diesel exhaust (0.05 to 6.4 mg/m^3); however, very little is emitted from small engines such as lawn mowers or leaf blowers (IARC 1985, Baldauf *et al.* 2006).

Many individuals are exposed to acetaldehyde by inhalation. The highest ambient-air concentrations of acetaldehyde were reported for urban or suburban areas or near sources of combustion (HSDB 2009). In ambient air, concentrations of acetaldehyde generally averaged 5 μg/m^3. Indoor air concentrations were higher than ambient concentrations in all locations where acetaldehyde air concentrations were measured, both in the United States and in other countries (Miguel *et al.* 1995, Mukund *et al.* 1996, Brickus *et al.* 1998, MacIntosh *et al.* 2000, Possanzini *et al.* 2002, Baez *et al.* 2003, Hellen *et al.* 2004, Hodgson *et al.* 2004, Park and Ikeda 2004, Saijo *et al.* 2004, Sax *et al.* 2004, Shendell *et al.* 2004, Gilbert *et al.* 2005, Cavalcante *et al.* 2006, Ohura *et al.* 2006, Pang and Mu 2006, Sax *et al.* 2006, Hodgson *et al.* 2007, Possanzini *et al.* 2007). Acetaldehyde is also found in tobacco and marijuana cigarette smoke (1,220 μg per cigarette) and tobacco cigarettes (980 to 1,370 μg per cigarette).

In 1988–89, acetaldehyde was detected in 4 of 10 surveyed water supplies (EPA 1987). In surface water, concentrations generally are less than 0.1 μg/L, and the contribution from drinking water to human exposure is considered negligible (IPCS 1995).

The National Occupational Exposure Survey (conducted from 1981 to 1983) estimated that 216,533 workers, including 97,770 women, potentially were exposed to acetaldehyde (NIOSH 1990). Workers potentially exposed include those involved in the manufacture or use of industrial organic chemicals, dyes, fabricated rubber, plastics, urea-formaldehyde foam insulation, fuels, drugs, explosives, varnishes, pesticides, food additives, leather goods, and mirrors (IARC 1985, EPA 1994).

Regulations

Coast Guard, Department of Homeland Security

Minimum requirements have been established for safe transport of acetaldehyde on ships and barges.

Department of Transportation (DOT)

Acetaldehyde is considered a hazardous material, and special requirements have been set for marking, labeling, and transporting this material.

Environmental Protection Agency (EPA)

Clean Air Act

Mobile Source Air Toxics: Listed as a mobile source air toxic for which regulations are to be developed.
National Emission Standards for Hazardous Air Pollutants: Listed as a hazardous air pollutant.
New Source Performance Standards: Manufacture of acetaldehyde is subject to certain provisions for the control of volatile organic compound emissions.
Prevention of Accidental Release: Threshold quantity (TQ) = 10,000 lb.
Urban Air Toxics Strategy: Identified as one of 33 hazardous air pollutants that present the greatest threat to public health in urban areas.

Clean Water Act

Designated a hazardous substance.

Comprehensive Environmental Response, Compensation, and Liability Act

Reportable quantity (RQ) = 1,000 lb.

Emergency Planning and Community Right-To-Know Act

Toxics Release Inventory: Listed substance subject to reporting requirements.

Resource Conservation and Recovery Act

Listed Hazardous Waste: Waste code for which the listing is based wholly or partly on the presence of acetaldehyde = U001.

Occupational Safety and Health Administration (OSHA, Dept. of Labor)

While this section accurately identifies OSHA's legally enforceable PELs for this substance in 2018, specific PELs may not reflect the more current studies and may not adequately protect workers.
Permissible exposure limit (PEL) = 200 ppm (360 mg/m^3).
Considered a highly hazardous chemical: Threshold quantity (TQ) = 2,500 lb.

Guidelines

American Conference of Governmental Industrial Hygienists (ACGIH)

Threshold limit value – ceiling (TLV-C) = 25 ppm.

National Institute for Occupational Safety and Health (NIOSH, CDC, HHS)

Immediately dangerous to life and health (IDLH) limit = 2,000 ppm.
Listed as a potential occupational carcinogen.

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Acetaldehyde; CASRN 75-07-0

Human health assessment information on a chemical substance is included in the IRIS database only after a comprehensive review of toxicity data, as outlined in the IRIS assessment development process. Sections I (Health Hazard Assessments for Noncarcinogenic Effects) and II (Carcinogenicity Assessment for Lifetime Exposure) present the conclusions that were reached during the assessment development process. Supporting information and explanations of the methods used to derive the values given in IRIS are provided in the guidance documents located on the IRIS website.

STATUS OF DATA FOR Acetaldehyde

File First On-Line 06/30/1988

Category (section)	Assessment Available?	Last Revised
Oral RfD (I.A.)	not evaluated	
Inhalation RfC (I.B.)	yes	10/01/1991
Carcinogenicity Assessment (II.)	yes	06/30/1988

I. Chronic Health Hazard Assessments for Noncarcinogenic Effects

I.A. Reference Dose for Chronic Oral Exposure (RfD)

Substance Name — Acetaldehyde
CASRN — 75-07-0

Not available at this time.

I.B. Reference Concentration for Chronic Inhalation Exposure (RfC)

Substance Name — Acetaldehyde
CASRN — 75-07-0
Last Revised — 10/01/1991

The inhalation Reference Concentration (RfC) is analogous to the oral RfD and is likewise based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. The inhalation RfC considers toxic effects for both the respiratory system (portal-of-entry) and for effects peripheral to the respiratory system (extrarespiratory effects). It is expressed in units of mg/cu.m. In general, the RfC is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily inhalation exposure of the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Inhalation RfCs were derived according to the Interim Methods for Development of Inhalation Reference Doses (EPA/600/8-88/066F August 1989) and subsequently, according to Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry (EPA/600/8-90/066F October 1994). RfCs can also be derived for the noncarcinogenic health effects of substances that are carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

I.B.1. Inhalation RfC Summary

Critical Effect	Exposures*	UF	MF	RfC
Degenration of olfactory epithelium **Short-term Rat Inhalation Studies** **Appleman et al., 1986;1982**	NOAEL: 273 mg/cu.m (150 ppm) NOAEL:(ADJ): 48.75 mg/cu.m NOAEL(HEC): 8.7 mg/cu.m LOAEL: 728 mg/cu.m (400 ppm) LOAEL(ADJ): 130 mg/cu.m LOAEL(HEC): 16.9 mg/cu.m	1000	1	9E-3 mg/cu.m

*Conversion Factors -- MW = 44.5. Appleman et al., 1986: Assuming 25C and 760 mmHg, NOAEL(mg/cu.m) = 150 ppm x 44.5/24.45 = 273. NOAEL(ADJ) = 273 mg/cu.m x 6 hours/day x 5 days/7 days = 48.75 mg/cu.m. The NOAEL(HEC) was calculated for a gas:respiratory effect

in the ExtraThoracic region. MVa = 0.23 cu.m/day, MVh = 20 cu.m/day, Sa(ET) = 11.6 sq. cm, Sh(ET) = 177 sq. cm. RGDR(ET) = (MVa/Sa) / (MVh/Sh) = 0.18. NOAEL(HEC) = NOAEL(ADJ) x RGDR = 8.7 mg/cu.m.

Appleman et al., 1982: Assuming 25C and 760 mmHg, LOAEL(mg/cu.m) = 400 ppm x 44.5/24.45 = 130. LOAEL(ADJ) = 728 mg/cu. m x 6 hours/day x 5 days/7days = 130 mg/cu.m. The LOAEL(HEC) was calculated for a gas:respiratory effect in the ExtraThoracic region. MVa = 0.17 cu.m/day, MVh = 20 cu.m/day, Sa(ET) = 11.6 sq. cm., Sh(ET) = 177 sq.cm. RGDR(ET) = (MVa/Sa) / (MVh/Sh) = 0.13. LOAEL(HEC) = LOAEL(ADJ) x RGDR = 16.9 mg/cu.m.

I.B.2. Principal and Supporting Studies (Inhalation RfC)

Appleman, L.M., R.A. Woutersen, V.J. Feron, R.N. Hooftman and W.R.F. Notten. 1986. Effect of variable versus fixed exposure levels on the toxicity of acetaldehyde in rats. J. Appl. Toxicol. 6(5): 331-336.

Appleman, L.M., R.A. Woutersen, and V.J. Feron. 1982. Inhalation toxicity of acetaldehyde in rats. I. Acute and subacute studies. Toxicology. 23: 293-297.

Two short-term studies conducted by the same research group are the principal studies used. While these studies are short-term in duration, together they establish a concentration-response for lesions after only 4 weeks of exposure. These same types of lesions appear at longer exposure times and higher exposure levels in chronic studies (Wouterson et al., 1986; Wouterson and Feron, 1987; Kruysse et al., 1975). Under other circumstances, studies of short duration may not be considered appropriate, but for this chemical the observed effects are consistent with pathology seen in long-term studies. The 150-ppm exposure level was therefore established as the NOAEL from the Appleman et al. (1986) study and the LOAEL from the Appleman et al. (1982) study.

Appleman et al. (1986) conducted two inhalation studies on male Wistar rats (10/group) exposing them 6 hours/day, 5 days/week for 4 weeks to 0, 150, and 500 ppm (0, 273 and 910 mg/cu.m, respectively). Duration-adjusted concentrations are 0, 48.75, and 162.5 mg/cu.m, respectively. One group was exposed without interruption, a second group was interrupted for 1.5 hours between the first and second 3-hour period, and a third group was interrupted as described with a superimposed peak exposure profile of 4 peaks at 6-fold the basic concentration per 3-hour period. The purpose was to test intermittent and peak exposure effects. Urine samples were collected from all rats and lung lavage performed on 4-5 per group at the end of the experiment. Cell density, viability, number of phagocytosing cells, and phagocytic index were determined on the lavage fluid. Microscopic examination was performed on the nasal cavity, larynx, trachea with bifurcation and pulmonary lobes of all rats of all groups.

Continuous and interrupted exposure to 500 ppm did not induce any visible effect on general condition or behavior, but peak exposures at this level caused irritation. No behavioral differences were noted in the other groups. Mean body weights of the group exposed to 500 ppm with interruption and with peak exposures were statistically significantly lower than those of the controls. Body weights were similar to controls in the other exposure groups. Mean cell density and cell viability were significantly decreased in the group exposed to 500 ppm with or without peak exposures. The mean percentage of phagocytosing cells and the phagocytic index were significantly lower than controls in all groups exposed to 500 ppm, especially the group exposed to superimposed peaks. Histopathological changes attributable to exposure were found only in the nasal cavity. Degeneration of the olfactory epithelium was observed in rats exposed to 500 ppm. Interruption of the exposure or interruption combined with peak exposure did not visibly influence this adverse effect. No compound-related effects were observed in rats interruptedly or uninterruptedly exposed to 150 ppm during the 4-week exposure period; therefore, the NOAEL is 150 ppm. The NOAEL(HEC) based on effects on the olfactory epithelium in the extrathoracic region is 8.7 mg/cu.m.

Appelman et al. (1982) exposed Wistar rats (10/sex/group) for 6 hours/day, 5 days/week for 4 weeks to 0, 400, 1000, 2200, or 5000 ppm acetaldehyde (0, 728, 1820, 4004 and 9100 mg/cu.m, respectively). Duration-adjusted concentrations are 0, 130, 325, 715 and 1625 mg/cu.m, respectively. The general condition and behavior of the rats were checked daily. Blood picture (Hb, Hct, RBC, total and differential WBC, and plasma protein) and chemistry were examined at the end of the treatment period. Activities of plasma glutamic-oxalacetic transaminase, glutamic-pyruvic transaminase, and alkaline phosphatase were also determined. Urine was analyzed for density, volume, pH, protein, glucose, occult blood, ketones, and appearance. The kidneys, lungs, liver, and spleen were weighed. Microscopic examination was performed on the lungs, trachea, larynx, and nasal cavity (3 transverse sections) of all animals and on the kidneys, liver, and spleen of all control and high- concentration groups.

During the first 30 minutes of each exposure at the 5000-ppm level, rats exhibited severe dyspnea that gradually became less severe during the subsequent exposure period. Two animals died at this level (1 female, 1 male) and one male died at the 2200-ppm level, but the cause of death could not be determined due to autolysis or cannibalism. Growth was retarded in males at the three highest exposure concentrations and in females at the 5000-ppm level. The percentage of lymphocytes in the blood was lower and the percentage of neutrophilic leukocytes higher in males and females of the 5000-ppm group than in controls. There were a few statistically significant differences in several blood chemistry parameters between the exposure groups and the control group but none of them were concentration-related. Statistically significant changes in organ-to-body weight ratios included decreased liver weights in both sexes and increased lung weights in males at the 5000-ppm level. Males in the 5000-ppm level produced less urine, but it was of higher density. Compound-related histopathological changes were observed only in the

respiratory system. The nasal cavity was most severely affected and exhibited a concentration-response relationship. At the 400-ppm level, compound-related changes included: slight to severe degeneration of the nasal olfactory epithelium, without hyper- and metaplasia, and disarrangement of epithelial cells. At the 1000- and 2200-ppm levels, more severe degenerative changes occurred, with hyperplastic and metaplastic changes in the olfactory and respiratory epithelium of the nasal cavity. Degeneration with hyperplasia/metaplasia also occurred in the laryngeal and tracheal epithelium at these levels. At 5000 ppm changes included severe degenerative hyperplastic and metaplastic changes of the nasal, laryngeal, and tracheal epithelium. Based on the degenerative changes observed in the olfactory epithelium, the 400-ppm level is designated as a LOAEL. The LOAEL(HEC), based on the ventilation rates for female rats, is 16.9 mg/cu.m. No NOAEL was identified.

Woutersen et al.(1986) exposed Wistar rats (105/sex/group) for 6 hours/day, 5 days/week for up to 28 months to 0, 750, 1500 and 3000/1000 ppm (0, 1365, 2730, 5460/1820 mg/cu.m, respectively). The highest concentration was gradually decreased because of severe growth retardation, occasional loss of body weight, and early mortality in this group. The duration-adjusted concentrations are 0, 244, 488, and 975/325 mg/cu.m, respectively. The general condition and behavior of the rats were checked daily. Samples of a wide range (otherwise not specified) of tissues, including the nasal cavity, trachea with main bronchi, and lungs were examined by light microscopy. The rats in the high-exposure concentration showed excessive salivation, labored respiration, and mouth breathing. The respiratory distress was still observed when the concentration was reduced to 1000 ppm, although fewer were dyspneic. Only a few rats died during the first 6 months of the study but thereafter a sharp increase in the numbers of deaths occurred in the high-concentration group. All top concentration rats had died by 25 months. When the study was terminated, only a few animals remained alive in the mid-concentration group. The cause of early death or moribund condition was nearly always partial or complete occlusion of the nose by excessive amounts of keratin and inflammatory exudate. Several showed acute bronchopneumonia occasionally accompanied by tracheitis. Growth retardation occurred in males of each test group and in females of the two highest concentrations. The only exposure- related histopathology occurred in the respiratory system and showed a concentration-response relationship. The most severe abnormalities were found in the nasal cavity. Basal cell hyperplasia of the olfactory epithelium was seen in the low- and mid-concentration rats. The decrease in these changes in the olfactory epithelium was attributed to the incidence of adenocarcinomas at the higher levels. The respiratory epithelium of the nasal cavity was involved (hyperplasia and squamous metaplasia with keratinization) at the mid and high concentrations. Hyperplasia and squamous metaplasia, occasionally accompanied by keratinization, occurred in the larynx of rats exposed at the mid and high concentrations. The tracheal epithelium was not visibly affected at any exposure level. Adenocarcinomas occurred at all exposure concentrations and squamous cell carcinoma at the mid and high concentrations only. It thus appeared that the nasal tumors could be distinguished into two major types:

adenocarcinomas from olfactory epithelium, and squamous cell carcinoma from the respiratory epithelium. The lowest exposure concentration, 750 ppm, is clearly a LOAEL based on the above changes in the olfactory epithelium. The LOAEL(HEC) is 56 mg/cu.m. No NOAEL was identified.

Woutersen and Feron (1987) conducted an inhalation study in which Wistar rats (30 rats/sex/group) were exposed to 0, 750, 1500, or 3000/1500 ppm acetaldehyde (0, 1365, 2730, 5460/2730 mg/cu.m, respectively) for 6 hours/day, 5 days/week for 52 weeks with a 26- or 52-week recovery period. The highest concentration was gradually decreased because of severe growth retardation, occasional loss of body weight, and early mortality. Duration-adjusted concentrations are 0, 244, 488, and 975/488 mg/cu.m, respectively. The general condition and behavior of the rats were checked daily. Histopathology was performed as described for Wouterson et al. (1986).

At the end of the 52-week exposure period, most of the animals in the high-concentration group exhibited labored respiration and mouth breathing. The respiratory distress diminished during the recovery period but did not disappear completely. Adenocarcinoma and squamous cell carcinoma occurred at the mid and high concentrations. Degeneration of the olfactory epithelium was similar in rats terminated after 26 weeks of recovery and rats killed immediately after exposure termination. Histopathological changes found in the respiratory epithelium were comparable with, but less severe than, those observed immediately after exposure termination. After 52 weeks of recovery, the degeneration of the olfactory epithelium was still visible to a slight degree in animals from all exposure groups. Animals in the high-concentration group did not show restoration of the olfactory epithelium. At the low concentration, normal olfactory epithelium was present in some animals but replacement of olfactory epithelium by respiratory epithelium was frequently seen. Histopathological changes in the respiratory epithelium of the two females of the high-concentration group examined were essentially comparable with those found in rats terminated after 26 weeks of recovery. These data suggest that there is incomplete recovery of olfactory and respiratory epithelium changes induced at all exposure concentrations for periods as long as 52 weeks after exposure termination.

Kruysse et al. (1975) conducted a 90-day inhalation study in hamsters (10/sex/concentration). The hamsters were exposed to acetaldehyde vapor at concentrations of 0, 390, 1340, or 4560 ppm (0, 127, 435.5 or 1482 mg/cu.m, adjusted for duration, respectively), for 6 hours/day, 5 days/week for 90 days. Histopathological changes attributable to exposure were observed only in the respiratory tract. At 4560 ppm, body weights were significantly reduced and the relative weights of heart, kidney, brain, testicle, and lung were significantly increased. Histopathological changes of the nasal cavity, larynx, trachea, and bronchi included necrosis, inflammatory changes, and hyperplasia and metaplasia of the epithelium. Mild effects observed at 1340 ppm consisted of statistically significant increased kidney weight in males, and small areas of

stratified epithelium in the trachea in both sexes (30% of the animals). At 390 ppm, with the exception of a tiny focus of metaplastic epithelium in the trachea of 1 out of the 20 animals examined, no adverse effects were observed. The 390-ppm concentration was identified by the authors as a NOAEL. The study by Appelman et al. (1982) identified a similar level (400 ppm) as a LOAEL [LOAEL(HEC) = 16.9 mg/cu.m] for Wistar rats, but surface area values in hamsters are not available so that a comparison on HEC values could not be made to determine the relative sensitivities of the species to acetaldehyde. The LOAEL for the extrarespiratory effects (effect on kidney weight) is 1340 ppm and the NOAEL also at 390 ppm. The NOAEL(HEC) for extrarespiratory effects is 127 mg/cu.m.

I.B.3. Uncertainty and Modifying Factors (Inhalation RfC)

UF — An uncertainty factor of 10 was applied to account for sensitive human populations. A factor of 10 was applied for both uncertainty in the interspecies extrapolation using dosimetric adjustments and to account for the incompleteness of the database. A factor of 10 was applied to account for subchronic to chronic extrapolation.

MF — None

I.B.4. Additional Studies/Comments (Inhalation RfC)

Saldiva et al. (1985) exposed male Wistar rats (12/group) to 0 or 243 ppm (442 mg/cu.m) of acetaldehyde 8 hours/day, 5 days/week for weeks. Duration- adjusted values are and 105/cu.m., respectively. The animals were evaluated pulmonary mechanics before after exposure period, gross paraffin-embedded sample observations made exposure, especially respiratory system. Increases in RF, FRC, RV, TLC significantly different from control values. Damage distal airways was suggested since functional tests elasticity severe obstruction not demonstrated. Histopathological investigation showed an intense inflammatory reaction with olfactory epithelium hyperplasia polymorphonuclear mononuclear infiltration submucosa. Cannulation precluded evaluation tracheal effects no differences between observed lower tract. Although this study presents pathology data only a descriptive fashion, it identifies LOAEL nasal/cu.m (HEC = 13.7 that is consistent principal studies. LOAEL(HEC) thoracic on function 220.5/cu.m.

Saldiva et al. (1985) exposed male Wistar rats (12/group) to 0 or 243 ppm (442 mg/cu.m) of acetaldehyde 8 hours/day, 5 days/week for 5 weeks. Duration- adjusted values are 0 and 105 mg/cu.m., respectively. The animals were evaluated for pulmonary mechanics before and after the exposure period, and gross and paraffin-embedded sample observations were made after exposure, especially of the respiratory system. Increases in RF, FRC, RV, and TLC were significantly different from control values. Damage to distal airways was suggested since functional tests for damage to elasticity or for severe obstruction were not demonstrated.

Histopathological investigation showed an intense inflammatory reaction with olfactory epithelium hyperplasia and polymorphonuclear and mononuclear infiltration of the submucosa. Cannulation precluded evaluation of tracheal effects and no differences between the control and exposed animals were observed for the lower respiratory tract. Although this study presents the pathology data in only a descriptive fashion, it identifies a LOAEL for nasal effects of 105 mg/cu.m (HEC = 13.7 mg/cu.m) that is consistent with the principal studies. The LOAEL(HEC) for thoracic effects on pulmonary function is 220.5 mg/cu.m.

Feron (1979) exposed Syrian golden hamsters (35 males/group) by inhalation to 1500 ppm acetaldehyde 7 hours/day, 5 days/week for 52 weeks. The duration- adjusted concentration is 487.5 mg/cu.m. Exposure to acetaldehyde vapor resulted in epithelial hyperplasia and metaplasia, accompanied by inflammation in the nasal cavity and trachea. No evidence of carcinogenicity was observed.

In an inhalation study Feron et al. (1982) exposed Syrian golden hamsters to 2500 ppm (948 mg/cu.m adjusted for duration) for the first 9 weeks, 2250 ppm, (853 mg/cu.m adjusted for duration) for weeks 10-20, 2000 ppm (758 mg/cu.m adjusted for duration) for weeks 21-29, 1800 ppm (682.5 mg/cu.m adjusted for duration) for weeks 30-44, and 1650 ppm (626 mg/cu.m adjusted for duration) for weeks 42-52, for 7 hours/day, 5 days/week for a total of 52 weeks. Compound-related changes included rhinitis, hyperplasia, and metaplasia of the nasal, laryngeal, and tracheal epithelium, and nasal and laryngeal carcinomas. No LOAEL was identified.

No inhalation studies for reproductive or developmental effects have been performed. No oral or inhalation developmental studies, nor any reproductive studies, exist.

Zorzano and Herrera (1989) studied the pattern of acetaldehyde appearance in maternal and fetal blood, maternal and fetal liver and placenta after oral ethanol administration or intravenous acetaldehyde administration (10 mg/kg) to pregnant Wistar rats. The study demonstrated that acetaldehyde was able to cross the placental barrier at high concentrations (fetal blood concentrations were only detectable when maternal blood concentrations were greater than 80 uM). The fetal oxidation capacity in liver and placenta was shown to be lower than that of the maternal liver. A threshold above which the removal capacity of acetaldehyde metabolism by the fetoplacental unit would be surpassed was estimated to be 80 uM (maternal blood concentration) in the 21-day pregnant rat and possibly lower at early pregnancy when aldehyde dehydrogenase is absent from fetal liver.

Retention of acetaldehyde in humans under "physiologic conditions" of breathing rate and tidal volume has been shown to be approximately 60% between 100 and 200 mg/cu.m for a few minutes (Egle, 1970), and retention was shown to decrease slightly at higher concentrations. Breathing rate and volume and exposure concentration were shown to influence retention.

Retention has not been determined at lower concentrations comparable with the HEC estimates derived here, however. Retention of acetaldehyde from cigarette smoke was shown to be 99% (Dalhamn et al., 1968). Acetaldehyde has been shown to be absorbed via inhalation at high concentrations (9000-10,000) for 1 hour (Watanabe et al., 1986). Binding and metabolism in blood and rat nasal mucosa have been demonstrated (Hagihara et al., 1981; Casanova-Schmitz et al., 1984). Casanova-Schmitz et al. (1984) observed that rats exposed to 700 ppm for 2 hours demonstrated only 0.7 mM in circulating blood 5 minutes after exposure termination, suggesting that binding in the respiratory tract and rapid metabolism significantly reduces systemic circulation at steady state.

I.B.5. Confidence in the Inhalation RfC

Study — Medium
Database — Low
RfC -- Low

Confidence in the principal studies is medium since appropriate histopathology was performed on an adequate number of animals and a NOAEL and LOAEL were identified, but the duration was short and only one species was tested. Confidence in the database is low due to the lack of chronic data establishing NOAELs and due to the lack of reproductive and developmental toxicity data. Low confidence in the RfC results.

I.B.6. EPA Documentation and Review of the Inhalation RfC

Source Document — This assessment is not presented in any existing U.S. EPA document.

Other EPA documentation -- U.S. EPA, 1991

Agency Work Group Review — 05/18/1989, 04/25/1991

Verification Date — 04/25/1991

I.B.7. EPA Contacts (Inhalation RfC)

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (202)566-1676 (phone), (202)566-1749 (FAX) or hotline.iris@epa.gov (internet address).

II. Carcinogenicity Assessment for Lifetime Exposure

Substance Name — Acetaldehyde
CASRN — 75-07-0
Last Revised — 06/30/1988

Section II provides information on three aspects of the carcinogenic assessment for the substance in question; the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates of risk from oral exposure and from inhalation exposure. The quantitative risk estimates are presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per (mg/kg)/day. The unit risk is the quantitative estimate in terms of either risk per ug/L drinking water or risk per ug/cu.m air breathed. The third form in which risk is presented is a drinking water or air concentration providing cancer risks of 1 in 10,000, 1 in 100,000 or 1 in 1,000,000. The rationale and methods used to develop the carcinogenicity information in IRIS are described in The Risk Assessment Guidelines of 1986 (EPA/600/8-87/045) and in the IRIS Background Document. IRIS summaries developed since the publication of EPA's more recent Proposed Guidelines for Carcinogen Risk Assessment also utilize those Guidelines where indicated (Federal Register 61(79):17960-18011, April 23, 1996). Users are referred to Section I of this IRIS file for information on long-term toxic effects other than carcinogenicity.

II.A. Evidence for Human Carcinogenicity

II.A.1. Weight-of-Evidence Characterization

Classification — B2; probable human carcinogen

Basis — Based on increased incidence of nasal tumors in male and female rats and laryngeal tumors in male and female hamsters after inhalation exposure.

II.A.2. Human Carcinogenicity Data

Inadequate. The only epidemiological study involving acetaldehyde exposure showed an increased crude incidence rate of total cancer in acetaldehyde production workers as compared with the general population (Bittersohl, 1974). Because the incidence rate was not age adjusted, and because this study has several other major methodological limitations (including concurrent exposure to other chemicals and cigarette exposure, short duration, small number of subjects, and lack of information on subject selection, age and sex distribution) it is considered inadequate to evaluate the carcinogenicity of acetaldehyde.

II.A.3. Animal Carcinogenicity Data

Sufficient. Feron (1979) exposed groups of 35 male Syrian Golden hamsters to 0 or 1500 ppm acetaldehyde by inhalation 7 hours/day, 5 days/week, for 52 weeks. These animals were also exposed weekly by intratracheal instillation to increasing doses of benzo(a)pyrene (BaP) in 0.2 mL of 0.9% NaCl, or to NaCl alone. Animals were killed and autopsied after exposure and 26 weeks of recovery in air. No neoplastic effects due to acetaldehyde alone were found. The highest BaP dose (1 mg/week for 52 weeks) combined with acetaldehyde exposure produced twice the incidence of squamous cell carcinomas compared with the same dose of BaP alone. In the second part of this study, no respiratory tract tumors were found in groups of 25 male hamsters which were intratracheally instilled once a week with 0.2 mL of 2% or 4% acetaldehyde in 0.9% NaCl for 52 weeks.

Feron et al. (1982) studied male and female hamsters exposed by inhalation to acetaldehyde alone or in combination with intratracheally administered BaP or diethylnitrosamine. The animals were exposed for 7 hours/day, 5 days/week, for 52 weeks to a time weighted average concentration of 2028 ppm. They were killed and autopsied after a 29-week recovery period; that is, at week 81. A slight increase in nasal tumors and a significantly increased incidence of laryngeal tumors was observed in both male and female hamsters exposed to acetaldehyde alone. This study supported the observation of Feron (1979) that acetaldehyde treatment enhanced tumorigenicity (production of tracheobronchial carcinomas) of BaP.

The carcinogenicity of acetaldehyde was studied in 420 male and 420 female albino SPF Wistar rats (Woutersen and Appelman, 1984; Woutersen et al., 1985). After an acclimatization period of 3 weeks, these animals were randomly assigned to four groups of 105 males and 105 females each. The animals were then exposed by inhalation to atmospheres containing 0, 750, 1500, or 3000 ppm acetaldehyde for 6 hours/day, 5 days/week, for 27 months. The concentration in the highest dose group was gradually reduced from 3000 to 1000 ppm because of severe growth retardation, occasional loss of body weight and early mortality in this group. Interim sacrifices were carried out at 13, 26, and 52 weeks. One tumor was observed in the 52 week sacrifice group and none at earlier times. Exposure to acetaldehyde increased the incidence of tumors in an exposure-related manner in both male and female rats. In addition, there were exposure-related increases in the incidences of multiple respiratory tract tumors. Adenocarcinomas were increased significantly in both male and female rats at all exposure levels, whereas squamous cell carcinomas were increased significantly in male rats at middle and high doses and in female rats only at the high dose. The squamous cell carcinoma incidences showed a clear dose-response relationship. The incidence of adenocarcinoma was highest in the mid-exposure group (1500 ppm) in both male and female rats, but this was probably due to the high mortality and competing squamous cell carcinomas at the highest exposure level. In the low-exposure group, the adenocarcinoma incidence was higher in males than in females.

In a concurrent study, 30 animals of each sex were exposed to the same concentrations of acetaldehyde for 52 weeks followed by a recovery period of 26 weeks (10 animals) or 52 weeks (20 animals). Significant increases in nasal tumors were observed in male and female rats, including adenocarcinomas and squamous cell carcinomas, in both recovery groups. These findings indicate that after 52 weeks of exposure to acetaldehyde, proliferative epithelial lesions of the nose may develop into tumors even without continued exposure.

II.A.4. Supporting Data for Carcinogenicity

Acetaldehyde has been shown by several laboratories to induce sister chromatid exchange (SCE) in cultured mammalian cells (Obe and Ristow, 1977; Obe and Beer, 1979; deRaat et al., 1983; Bohlke et al., 1983; Ristow and Obe, 1978; Jansson, 1982; Norrpa et al., 1985). A recent study provided evidence that SCE-inducing lesions may be persistent for several cell generations (He and Lambert, 1985). The in vitro SCE response did not require metabolic activation. The induction of SCE by acetaldehyde has also been detected in bone marrow cells of mice and hamsters in vivo (Obe et al., 1979; Korte and Obe, 1981). Acetaldehyde caused chromosomal aberrations in mammalian cell culture (Bird et al., 1981; Bohlke et al., 1983) and plants (Rieger and Michaelis, 1960), but not in Drosophila (Woodruff et al., 1985). Chromosome gaps and breaks were found in rat embryos after a single intraamniotic injection on day 13 of gestation (Barilyak and Kozachuk, 1983). Acetaldehyde produced sex-linked recessive lethal gene mutations after injection in Drosophila (Woodruff et al., 1985), but has been negative in testing in Salmonella (Commoner, 1976, Laumbach et al., 1976; Pool and Wiesler, 1981., Marnett et al., 1985, Mortelmans et al., 1986). Acetaldehyde has been shown to produce crosslinks between protein and DNA in the nasal respiratory mucosa (Lam et al., 1986).

Acetaldehyde is similar in stucture to formaldehyde (classified B1) which also produces nasal tumors in animals exposed by inhalation.

II.B. Quantitative Estimate of Carcinogenic Risk from Oral Exposure

Not available.

II.C. Quantitative Estimate of Carcinogenic Risk from Inhalation Exposure

II.C.1. Summary of Risk Estimates

Inhalation Unit Risk — 2.2E-6 per (ug/cu.m)

Extrapolation Method — Linearized multistage-variable exposure input form (extra risk)

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	5E+1 ug/cu.m
E-5 (1 in 100,000)	5E+0 ug/cu.m
E-6 (1 in 1,000,000)	5E-1 ug/cu.m

II.C.2. Dose-Response Data for Carcinogenicity, Inhalation Exposure

Tumor Type: nasal squamous cell carcinoma or adenocarcinoma
Test animals: rat/SPF Wistar, male
Route: inhalation
Reference: Woutersen and Appleman, 1984

Dose		Tumor Incidence
Lifetime Average Exposure		
Administered (ppm)	**Human Equivalent (ppm)**	
0	0	1/94
750	130	20/95
1500	255	49/95
1540	279	47/92

II.C.3. Additional Comments (Carcinogenicity, Inhalation Exposure)

Actual measured exposures on two occasions for the low and medium dose groups were 727/735 and 1438/1412 ppm, respectively. The highest dose administered is given as TWA. Low-dose extrapolation was performed using two forms of the linearized multistage model, the quantal model (Crump et al., 1977) and a form which allows analysis for a variable dose pattern, adjusts for intercurrent mortality, and is capable of estimating risk at any time from any dosing pattern (Crump and Howe, 1984). The latter model is referred to as the variable exposure form. Comparison of the results from the two models showed very little difference in the unit risk estimates. The variable exposure form was selected for the final unit risk estimate because it allows the combination of the lifetime study and the recovery study for risk estimation. The above estimates are from male rats; the unit risk calculated from data on female rats at 18 months was 1.6E-6 per (mg/cu.m). No difference was found in tumor incidence between animals exposed for a full lifetime and those exposed for 12 months and allowed to recover. At the end of 24 months, however, the tumor incidences in the recovery group were less than those in the lifetime exposure group.

The unit risk should not be used if the air concentration exceeds 5E+3 ug/cu.m, since above this concentration the unit risk may not be appropriate.

II.C.4. Discussion of Confidence (Carcinogenicity, Inhalation Exposure)

An adequate number of animals was observed in a lifetime study. Increases in nasal tumors were observed in both male and female rats, and similar unit risks were obtained using these data.

II.D. EPA Documentation, Review, and Contacts (Carcinogenicity Assessment)

II.D.1. EPA Documentation

Source Document — U.S. EPA, 1987

The 1987 Health Assessment Document is a final draft which has received both agency and external review.

II.D.2. EPA Review (Carcinogenicity Assessment)

Agency Work Group Review — 01/13/1988

Verification Date — 01/13/1988

II.D.3. EPA Contacts (Carcinogenicity Assessment)

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (202)566-1676 (phone), (202)566-1749 (FAX) or hotline.iris@epa.gov (internet address).

III. [reserved]
IV. [reserved]
V. [reserved]

VI. Bibliography

Substance Name — Acetaldehyde
CASRN — 75-07-0

VI.A. Oral RfD References

None

VI.B. Inhalation RfD References

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VII. Revision History

Substance Name — Acetaldehyde
CASRN — 75-07-0

Date	Section	Description
10/01/1991	I.B.	Inhalation RfC summary on-line

VIII. Synonyms

Substance Name — Acetaldehyde
CASRN — 75-07-0
Last Revised — 06/30/1988

- 75-07-0
- ACETALDEHYD
- Acetaldehyde
- ACETIC ALDEHYDE
- ACETYLALDEHYDE
- ALDEHYDE ACETIQUE
- ALDEIDE ACETICA
- ETHANAL
- ETHYL ALDEHYDE
- NCI-C56326
- OCTOWY ALDEHYD
- RCRA WASTE NUMBER U001
- UN 1089