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Canadian Centre for Occupational Health and Safety



CHEMINFO Chemical Profiles Created by CCOHS

CCOHS Chemical Name: Acetone

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REFRESH RECORD

SECTION 1. CHEMICAL IDENTIFICATION

CHEMINFO Record Number: 30

CCOHS Chemical Name: Acetone

Synonyms:

- Dimethyl formaldehyde
- Dimethylketal
- Dimethyl ketone
- Ketone propane
- beta-Ketopropane
- Methyl ketone

2-Propanone
Pyroacetic acid
Pyroacetic ether
Propanone

Chemical Name French: Acétone

Chemical Name Spanish: Acetona

CAS Registry Number: 67-64-1

UN/NA Number(s): 1090

RTECS Number(s): AL3150000

EU EINECS/ELINCS Number: 200-662-2

Chemical Family: Saturated aliphatic ketone / alkanone / methyl ketone / propanone

Molecular Formula: C₃-H₆-O

Structural Formula: CH₃-C(=O)-CH₃

Status of Record:

The CHEMINFO record for this chemical is complete. The full format provides a detailed evaluation of health, fire and reactivity hazards, as well as recommendations on topics such as handling and storage, personal protective equipment, accidental release and first aid.

SECTION 2. DESCRIPTION

Appearance and Odour:

Clear, colourless, volatile, free flowing liquid with a mildly pungent, characteristic sweet, slight aromatic, fruity odour.(50,51,52,53)

Odour Threshold:

Reported values vary widely; 3.6-653 ppm (geometric mean: 62 ppm) (detection); 33-699 ppm (geometric mean: 130 ppm) (recognition) (53)

Warning Properties:

NOT RELIABLE - some reported odour threshold values are about the same magnitude as the TLV. Acetone has been reported to diminish the sense of smell and adaptation to smell and irritation has occurred.(7,54)

Composition/Purity:

Available commercially in grades greater than 99% purity, with the remainder being mainly water (less than 0.5 wt%).(50,51) Other impurities present depend on the method of manufacture and may include very small amounts of phenol and benzene.

Uses and Occurrences:

Used as a solvent in formulations for surface coatings (paints, varnishes and lacquers) and related washes and thinners, mainly for acrylic and nitrocellulose lacquers and paints; as a spinning solvent in the manufacture of cellulose acetate; as a solvent in the manufacture of pharmaceuticals, vitamins and cosmetics; as a solvent for adhesives, contact cements, printing inks, gums, waxes, resins, fats, greases, oils, dyestuffs and cellulotics; for degreasing wool and degumming silk; in extraction processes; in the manufacture of smokeless powder; as a wash solvent in fibreglass boat manufacturing; as a cleaning solvent in the electronics industry; and as a carrier for acetylene in cylinders. Used as a chemical intermediate for methyl methacrylate, methacrylic acid, bisphenol A and aldol chemicals, such as diacetone alcohol, methyl isobutyl ketone, diisobutyl ketone, methylisobutylcarbinol, hexylene glycol, isophorone and phorone; also used to make functional compounds, such as antioxidants, herbicides, higher ketones, condensates with formaldehyde or diphenylamine, and vitamin intermediates.(50,51)

Acetone is a natural product of metabolism in the body and virtually every organ and tissue contains some acetone; it is a normal constituent of human blood. Other natural sources include forest fires, volcanoes and metabolism of vegetation, insects

and higher animals.(6,50)

SECTION 3. HAZARDS IDENTIFICATION

EMERGENCY OVERVIEW:

Clear, colourless, volatile liquid with a mildly pungent, characteristic sweet, slight aromatic, fruity odour. EXTREMELY FLAMMABLE LIQUID AND VAPOUR. Vapour is heavier than air and may spread long distances. Distant ignition and flashback are possible. Mild central nervous system depressant. Very high concentrations may cause headache, nausea, dizziness, drowsiness, incoordination and confusion. EYE IRRITANT. Causes severe eye irritation. Aspiration hazard. Swallowing or vomiting of the liquid may result in aspiration (breathing) into the lungs.

POTENTIAL HEALTH EFFECTS

Effects of Short-Term (Acute) Exposure

Inhalation:

In one study, volunteers exposed to concentrations up to 500 ppm reported no harmful effects.(1) In other studies, concentrations of approximately 300-500 ppm were reported to cause slight irritation of the nose and throat.(2,3,4) Exposure to 250 ppm for 4 hours has caused mild effects on performance in some behavioural tests (auditory tone discrimination and a mood test).(5) As concentrations approach 1000 ppm, noticeable irritation has occurred and some people have reported headaches, light-headedness and tiredness.(3,4,6) Inhalation of concentrations higher than 2000 ppm can cause dizziness, a feeling of drunkenness, drowsiness, nausea and vomiting. Unconsciousness may result if exposure is extremely high (greater than 10000 ppm). Intolerable nose and throat irritation would also occur at these concentrations.(6,9) Even higher concentrations can cause collapse, coma and death.

Tolerance to the effects of acetone can develop.(3,6,7) Tolerance means that, with repeated exposures, higher concentrations are required to produce symptoms which had previously been observed at lower concentrations. One case report describes two men who were working in a confined space with extremely high acetone concentrations (measured at 12000 ppm, 3 hours after the accident). Low concentrations (up to 50 ppm) of trichloroethane were also detected. After working in the area 4 hours, the men noticed irritation of the throat, headache, weakness in the legs and a feeling of drunkenness. The men then left the area for 1 hour. Upon returning, one man collapsed and the other felt faint. Rescuers, who were exposed for 2 to 3 minutes, experienced symptoms similar to the workers. The man who lost consciousness regained consciousness a short time later but was confused, drowsy, unsteady on his feet, felt nauseated and was vomiting. The other man had, at this point, also lost consciousness and was vomiting. Both men fully recovered.(8)

A single case report suggests slight kidney and liver damage may have occurred following a severe exposure to acetone.(6 unconfirmed, 9) There are insufficient details available to draw conclusions from this report.

Skin Contact:

Acetone is a non-irritant to very mild irritant, based on animal and limited human information. Application of 1 mL of acetone in a small glass tube to six male volunteers for 30 or 90 minutes resulted in only mild redness and swelling at 90 minutes.(10)

The risk of developing health effects following the absorption of acetone through unbroken skin is very slight. There are several reports of people, usually young children, becoming ill following skin exposure to acetone while lightweight casts were being put on broken limbs. The symptoms experienced were similar to those described following high inhalation exposures. In all cases, a large amount of acetone came into contact with

the skin for several hours and inhalation exposure probably also have occurred.(11) These reports are not considered relevant to people exposed to acetone at work.

Eye Contact:

Acetone is a severe irritant based on animal and limited human information. In three human cases, acetone caused corneal injury which completely healed within 48 hours.(13) In one unusual case, liquid acetone was held directly on the eye for a long time resulting in permanent damage to the eye, with clouding of the cornea.(14) Acetone vapour causes mild irritation at concentrations of around 500 ppm.(2,3) Irritation is very noticeable at 1000 ppm.(12)

Ingestion:

Ingestion is not a typical route of occupational exposure. Several studies report no effects or minor effects (slight drowsiness) in people who ingested up to 20 grams/day for several days.(6) Animal toxicity information also suggests that acetone is not very toxic following ingestion. If acetone is aspirated (breathed into the lungs during ingestion or vomiting) it can cause severe, life-threatening lung injury. Animal information suggests that acetone would be difficult to aspirate because it evaporates so quickly. Based on its physical properties, acetone can be aspirated into the lungs during ingestion or vomiting. One case report describes a man who intentionally drank 200 mL (about 7 ounces) of acetone. Within one hour, he had flushed cheeks and appeared drunk. His breathing was shallow and his throat red and swollen. He soon lapsed into coma and did not regain consciousness for 12 hours. Four weeks later, he developed symptoms similar to diabetes (increased urination, thirst and blood sugar levels). The patient fully recovered within 5 months after the incident.(15)

Effects of Long-Term (Chronic) Exposure

Although acetone has widespread industrial use there is limited information available on health effects from long-term exposure. Many occupational situations which involve acetone exposure also involve exposures to other potentially harmful chemicals.(6,23) No conclusions can be drawn from limited studies that show nervous system effects from long-term acetone exposure.

Long-term animal studies have not shown significant harmful effects from inhalation exposures. Long-term oral exposure of rats has caused kidney damage and increased liver weight at relatively high doses. Ingestion is not a typical route of occupational exposure.

Nervous System:

No conclusions can be drawn from the human information located. Studies in animals have not shown neurotoxic effects from acetone.

In a study of 110 male workers exposed for an average of 14.9 years at an acetate fibre plant, workers were divided into highly exposed (greater than 500 ppm), moderately exposed (250-500 ppm) and less exposed (less than 250 ppm) and compared with 67 unexposed male workers from the same plant. A dose-related relationship was found for a heavy, vague or faint feeling in the head, nausea and loss of weight. For workers aged 30-44, there was a significant decrease in simple reaction time and in a short-term memory test. However, this effect was not seen in workers aged less than 30 years or greater than 45 years. The authors questioned whether these findings were meaningful. Other neurobehavioural tests did not show any differences between exposed workers and controls.(16,99)

Neurotoxic effects were studied in 71 workers who were exposed to 417-892 ppm (cited as 988-2114 mg/m³) acetone while cleaning trophy medals and in 86 unexposed controls. Average exposure duration was 14 years Acetone-exposed workers had an increase in the self-reported symptoms of mood disorders, irritability, memory difficulties, sleep disturbances, numbness in hands and feet and pain in the bones, joints and muscles. No statistical analysis was reported for the incidence of these symptoms. Measurement of motor nerve conduction in the arms and legs showed some statistically

significant decreases in speed and size of the nerve impulses and an increase in the duration of the impulses. A significantly delayed reaction time in a visual test and a lower attention score were observed in psychological tests.(100,101) This study is limited by small numbers and lack of information on levels of alcohol consumption.

Early studies which showed neurotoxic effects are not reliable because of factors such as problems with experimental design and unreliable statistical analysis.(6,93)

Skin:

Prolonged or repeated contact may cause defatting of the skin and produce dermatitis (dryness, irritation, redness and cracking).

Skin Sensitization:

Acetone is not a skin sensitizer. Negative results have been obtained in tests in humans and, despite widespread industrial use, no conclusive case reports of sensitization were located. Negative results were obtained in animal tests. A negative result was obtained in 50 volunteers following 9 induction applications with 0.2 mL of 100% acetone over a 3 week period and a challenge 10-14 days later.(96) No evidence of sensitization was seen in 136 volunteers given a skin prick test with 1 or 5% acetone.(102) No conclusions can be drawn from two case reports showing sensitization to acetone. In one case, a female laboratory technician developed acute contact dermatitis while working with acetone. The acetone sensitization was believed to have developed from a previous sensitizing therapy (which involved acetone) for a skin condition.(103) In the other case, a woman working in a shoe factory tested positive with a patch test for acetone. She had developed contact dermatitis from working with epoxy resins, which are known sensitizers. No information about pre-existing allergies was reported.(104)

Heart/Blood Vessels:

No statistically significant differences in mortality from circulatory system or heart disease were observed in 948 employees exposed to up to 1070 ppm acetone for up to 23 years, when compared with the general United States population.(105,106)

Blood/Blood Forming System:

No significant changes in blood composition or chemistry were found in 60 workers who had worked at least 5 years in the acetate fibre manufacturing industry (exposures of 550-1050 ppm).(6, unconfirmed)

An early study, which reviewed 18 years of industrial experience with employees in a cellulose acetate production facility, did not show an increased incidence of illness.(6) No conclusions can be drawn from a few historical reports that described long-term exposure effects such as irritation of the airways, throat, stomach and occasionally, dizziness, attacks of giddiness and a loss of strength.(17,18)

Carcinogenicity:

Acetone is not known to be a carcinogen. Little human information was located. Animal information suggests that acetone is not carcinogenic. No statistically significant difference in mortality from cancer was observed in 948 employees exposed to up to 1070 ppm acetone for up to 23 years.(105,106) This study is limited because the acetone-exposed workers were the reference cohort for comparison to workers exposed to acetone plus methylene chloride. No unexposed controls were included in the study. In a case-control study of 10 cases of Hodgkin's disease in workers exposed to various chemicals, there was no significant association with acetone exposure.(108)

The International Agency for Research on Cancer (IARC) has not evaluated the carcinogenicity of this chemical.

The American Conference of Governmental Industrial Hygienists (ACGIH) has designated this chemical as not classifiable as a human carcinogen (A4).

The US National Toxicology Program (NTP) has not listed this chemical in its report on carcinogens.

Teratogenicity and Embryotoxicity:

The information located is not sufficient to conclude that acetone causes developmental toxicity. No conclusions can be drawn based on the limited human information available. In animal studies, inhalation of acetone caused fetotoxicity in rats and mice and embryotoxicity in mice, but only at concentrations that also caused maternal toxicity. A limited oral study in mice showed fetotoxicity and embryotoxicity at a dose that did not cause decreased maternal body weight during pregnancy.

A study of 891 women who worked or were working in the semiconductor industry showed an increased risk of miscarriages among fabrication workers. Seven chemicals were strongly associated with the increased risk of miscarriage, one of which was acetone.(20) No conclusions can be drawn from this study because of factors such as the small number of workers studied and the concurrent exposure to other potentially harmful chemicals. There is insufficient information available to evaluate a Russian study that reports increased complications of pregnancy and reduced birth weight in children of mothers exposed to acetone.(21)

Reproductive Toxicity:

The information located is not sufficient to conclude that acetone causes reproductive toxicity. No conclusions can be drawn from the limited human information available. In an oral study in rats, effects on sperm were observed at a dose that caused significant other toxicity.

A study of 25 men exposed to acetone and styrene during the manufacture of reinforced plastics showed an increased percentage of immobile sperm and abnormal sperm head shape in exposed workers compared to controls.(19) No conclusions can be drawn from this study because of factors such as the small number of workers studied and the concurrent exposure to other potentially harmful chemicals.

In a study of 735 pregnancies in 560 women working in laboratories, exposure to acetone was associated with a significant decrease in a measure of fertility (the probability of conception per menstrual cycle) in comparison to unexposed controls.(109) This study is limited by concurrent exposure to other solvents.

Mutagenicity:

Acetone is not known to be a mutagen. No human information was located. There are no confirmed studies that show mutagenicity in live animals. Negative results have been obtained in most studies with cultured mammalian cells and bacteria. Positive and negative results have been obtained in yeast. Acetone is often used as a solvent for testing the mutagenicity of other chemicals and as the solvent control in these assays.

Toxicologically Synergistic Materials:

A major effect of acetone is its enhancement of the toxicity of many other chemicals. Many occupational situations that involve acetone exposure also involve exposures to other potentially harmful chemicals. However, no human information on synergistic effects was located. In animal studies, acetone has increased the liver and/or kidney toxicity of many chemicals, including carbon tetrachloride, chloroform, trichloroethylene, bromodichloromethane, dibromochloromethane, N-nitrosodimethylamine and 1,1,2-trichloroethane. It has also enhanced the lung toxicity of styrene, the lethality of acetonitrile and the neurotoxicity of 2,5-hexanedione. Acetone appears to inhibit the metabolism and elimination of ethyl alcohol, thereby potentially increasing its toxicity. Acetone can either increase or decrease the toxicity of 1,2-dichlorobenzene, depending on the concentration of acetone used.

Potential for Accumulation:

Acetone does not accumulate. It is a normal by-product of mammalian metabolism and is found in virtually every organ and tissue, and in the blood. Acetone can enter the body by inhalation, ingestion or skin contact. It is metabolized by at least two pathways to compounds that are used by the

body to make glucose and other products of intermediary metabolism, with the generation of carbon dioxide. Acetone is excreted both unchanged, and following metabolism, mainly as carbon dioxide. The main route of excretion is in the expired air, with very little excreted in the urine. Respiratory excretion is complete within 20 hours after inhalation. The amount of unchanged acetone excreted in the urine increases with increasing exposure concentration and duration, and with exercise during exposure.(6,23,28,94)

SECTION 4. FIRST AID MEASURES

Inhalation:

This chemical is extremely flammable. Take proper precautions (e.g. remove any sources of ignition). If symptoms are experienced, remove source of contamination or have victim move to fresh air. If symptoms persist, obtain medical advice.

Skin Contact:

Remove contaminated clothing, shoes and leather goods (e.g. watchbands, belts). Flush with lukewarm, gently flowing water for 5 minutes. Obtain medical advice. Completely decontaminate clothing, shoes and leather goods before re-use or discard.

Eye Contact:

Immediately flush the contaminated eye(s) with lukewarm, gently flowing water for 15-20 minutes, while holding the eyelid(s) open. If a contact lens is present, DO NOT delay irrigation or attempt to remove the lens until flushing is done. Take care not to rinse contaminated water into the unaffected eye or onto the face. Immediately obtain medical attention.

Ingestion:

NEVER give anything by mouth if victim is rapidly losing consciousness, is unconscious or convulsing. Have victim rinse mouth thoroughly with water. DO NOT INDUCE VOMITING. If vomiting occurs naturally, have victim lean forward to reduce risk of aspiration. Have victim rinse mouth with water again. Immediately obtain medical attention.

First Aid Comments:

Consult a doctor and/or the nearest Poison Control Centre for all exposures except minor instances of inhalation or skin contact. All first aid procedures should be periodically reviewed by a doctor familiar with the material and its conditions of use in the workplace.

SECTION 5. FIRE FIGHTING MEASURES

Flash Point:

-20 deg C (-4.0 deg F) (closed cup) (55,57); -18 deg C (0 deg F) (closed cup) (50,56)

Lower Flammable (Explosive) Limit (LFL/LEL):

2.5% at 25 deg C (55,56)

Upper Flammable (Explosive) Limit (UFL/UEL):

12.8% at 25 deg C (50,55,56)

Autoignition (Ignition) Temperature:

465 deg C (869 deg F) (55,56); 538-540 deg C (1000.4-1004 deg F) (50,58)

Electrical Conductivity:

5.5-6 x 10(6) pS/m at 25 deg C (50,57,59)

Minimum Ignition Energy:

1.15 millijoules at 4.5% (59)

Flammable Properties:

EXTREMELY FLAMMABLE LIQUID. Will readily ignite at room temperature. Vapour in the flammable range may be ignited by a static charge of sufficient energy. Vapour is heavier than air and can travel a considerable distance to a source of ignition and flash back to a leak or open container. Even dilute solutions of acetone in water may be flammable.

Specific Hazards Arising from the Chemical:

During a fire, toxic gases, such as carbon monoxide, carbon dioxide and other toxic and irritating compounds, such as formaldehyde, methanol, acetic acid, hydrogen peroxide, methane and ethylene oxide may be formed, depending on fire conditions.(77,78) Vapour can accumulate in confined spaces resulting in a flammability and toxicity hazard. Closed containers may rupture violently and suddenly release large amounts of product when exposed to fire or excessive heat for a sufficient period of time.

Extinguishing Media:

Carbon dioxide, dry chemical powder, appropriate foam, water spray or fog. Water may be effective for cooling, but may not be effective for extinguishing a fire because it will not cool acetone below its flash point.(55) Fire fighting foams, such as multipurpose alcohol-resistant foams, are recommended for most flammable liquid fires.(55) Foam manufacturers should be consulted for recommendations regarding types of foams and application rates.

Fire Fighting Instructions:

Evacuate area and fight fire from a safe distance or protected location. Approach fire from upwind to avoid avoid hazardous vapours and toxic decomposition products.

Stop leak before attempting to stop the fire. If the leak cannot be stopped, and if there is no risk to the surrounding area, let the fire burn itself out. If the flames are extinguished without stopping the leak, vapours could readily form explosive mixtures with air and reignite. Water can extinguish the fire if used under favourable conditions and when hose streams are applied by experienced firefighters trained in fighting all types of flammable liquid fires.

Closed containers may rupture violently when exposed to the heat of fire and suddenly release large amounts of products. Always stay away from ends of tanks, but be aware that flying material (shrapnel) from ruptured tanks may travel in any direction. If possible, isolate materials not yet involved in the fire and move containers from fire area if this can be done without risk. Protect personnel. Otherwise, cool fire-exposed containers, tanks or equipment by applying hose streams. Cooling should begin as soon as possible (within several minutes) and should concentrate on any unwetted portions of the container. Apply water from the side and a safe distance. Cooling should continue until well after the fire is out. If this is not possible, use unmanned monitor nozzles and immediately evacuate the area.

If a leak or spill has not ignited, use water spray in large quantities to disperse the vapours and to protect personnel attempting to stop a leak. Water spray can be used to dilute spills to non-flammable mixtures and flush spills away from ignition sources. Dike fire control water for appropriate disposal. Solid streams of water may be ineffective and spread material.

For a massive fire in a large area, use unmanned hose holder or monitor nozzles; if this is not possible withdraw from fire area and allow fire to burn. Withdraw immediately in case of rising sound from venting safety device or any discolouration of tank due to fire.

After the fire has been extinguished, flammable atmospheres may remain. Before entering such an area especially confined areas, check the atmosphere with an appropriate monitoring device.

Protection of Fire Fighters:

Acetone is an eye irritant and it's thermal decomposition and combustion products are toxic/irritating by inhalation. Firefighters may enter the area if positive pressure self-contained breathing apparatus (NIOSH approved or equivalent) and full Bunker Gear is worn.

NATIONAL FIRE PROTECTION ASSOCIATION (NFPA) HAZARD IDENTIFICATION

NFPA - Health:	1 - Exposure would cause significant irritation, but only minor residual injury.
NFPA - Flammability:	3 - Liquids and solids that can be ignited under almost all ambient temperature conditions.
NFPA - Instability:	0 - Normally stable, even under fire conditions, and not reactive with water.

SECTION 6. ACCIDENTAL RELEASE MEASURES

Spill Precautions:

Restrict access to area. Ensure clean-up is conducted by trained personnel only. Wear adequate protective equipment. Extinguish or remove all ignition sources. Ventilate the area. Remove or isolate flammable and combustible materials. Notify government occupational health and safety and environmental authorities.

Clean-up:

Prevent material from entering sewers, waterways or confined spaces. Keep materials which can burn away from spilled material. Stop or reduce leak if safe to do so. Contain spill with earth, sand, or absorbent material which does not react with spilled material. Remove liquid by explosion-proof pumps or vacuum equipment. Place in suitable, covered, labelled containers. Flush area with water. Contaminated absorbent material may pose the same hazards as the spilled product. Large spills: Contact fire and emergency services and supplier for advice.

SECTION 7. HANDLING AND STORAGE

Handling:

This material is an EXTREMELY FLAMMABLE, TOXIC (eye irritant) LIQUID. Before handling, it is very important that engineering controls are operating and that personal hygiene measures are being followed. People working with this chemical should be properly trained regarding its hazards and its safe use. Eliminate all ignition sources (e.g. sparks, open flames, hot surfaces). Keep away from heat. Post "NO SMOKING" signs. It is very important to keep areas where this material is used clear of other materials which can burn (e.g., cardboard, sawdust). Use in smallest possible amounts in a well ventilated area separate from the storage area. Avoid generating vapours or mists. Prevent the release of vapours and mists into the workplace air. Do not use with incompatible materials such as strong oxidizing agents. Never return contaminated material to its original container. See Incompatibilities - Materials to Avoid section for more information. Do not dispense in storage area unless dispensing area is segregated by fire-resistant construction. To avoid splashing, carefully dispense into sturdy containers made of compatible materials. Never transfer liquids by pressurizing the original shipping containers with air or inert gas. Only use portable containers and dispensing equipment (faucet, pump, drip can) approved for flammable liquids. Ground all drums, transfer vessels, hoses and piping. Ground clips must contact bare metal. When dispensing in other than a closed system, ensure dispensing container is bonded to receiving transfer equipment and container. Label containers. Keep containers closed when not in use. Avoid damaging containers. Empty containers may contain hazardous residues. Never perform any welding, cutting, soldering, drilling or other hot work on an empty vessel, container or piping until all liquid and vapours have been cleared. To prevent

sparking, generously wet hard surfaces before they are chipped, ground, etc, in potentially hazardous areas.

For large scale operations, use non-sparking ventilation systems, approved explosion-proof equipment and intrinsically safe electrical systems in areas of use. Keep aisles and exits free of obstruction. Consider the installation of leak and fire detection equipment along with a suitable, automatic fire suppression system. To reduce the fire/explosion hazard, consider the use of an inert gas in the container or storage vessel.

Have suitable emergency equipment for fires, spills and leaks readily available. Follow handling precautions on Material Safety Data Sheet. Have suitable emergency equipment for fires, spills and leaks readily available. Practice good housekeeping. Maintain handling equipment. Comply with applicable regulations.

Storage:

Store in a cool, well-ventilated area out of direct sunlight and away from heat and ignition sources.

Inspect all incoming containers to make sure they are properly labelled and not damaged. Keep quantity stored as small as possible. Store in suitable, labelled containers (usually the shipping container). Keep containers closed. Bond and ground metal containers in storage area. Install pressure and vacuum-relief venting in all drums of flammable liquids. Make sure storage area is well ventilated. No stacking of containers. Protect from damage.

Use only approved "laboratory safe" explosion-proof refrigerator when storing small quantities.

Keep empty containers in separate storage area. Empty containers may contain hazardous residues. Keep closed. Keep storage area clear of burnable materials (e.g. old rags, cardboard). Lighted cigarettes, matches, or any other ignition sources should not be allowed around indoor or outdoor storage areas.

Store away from oxidizers and corrosives and other incompatible materials. See Incompatibilities - Materials to Avoid section for more information.

Storage area should be clearly identified, clear of obstruction and accessible only to trained and authorized personnel. Keep storage area separate from work areas.

Ground floor storage facilities are usually recommended. Store away from work process and production areas, elevators, building and room exits or main aisles leading to exits. Post warning signs. Inspect periodically for damage or leaks.

Avoid bulk storage indoors. Store in an isolated fireproof building, if possible. When storing large amounts, consider leak detection and alarm equipment for storage area.

Storage facilities should be made of fire resistant materials. Use a grounded, non-sparking ventilation system, approved explosion-proof equipment and intrinsically safe electrical systems. Store within temperature range recommended by chemical manufacturer/supplier. Alarms that warn of temperatures higher than recommended may be necessary.

Equip storage tank vents with a flame arrestor. Storage tanks should be above ground, over an area sealed on the bottom and diked to hold entire contents. To reduce the fire/explosion hazard, consider the use of an inert gas in the container or storage vessel.

Store flammable materials according to occupational health and safety regulations and fire and building codes which will describe the kind of storage area and the type of storage containers for a specified amount of the material.

Have appropriate fire extinguishers and spill clean-up equipment in or near storage area. Contain spills or leaks by storing in trays made from compatible materials. Keep absorbents for leaks and spills readily available. Provide raised sills or ramps at doorways or create a trench which drains to a safe location.

Follow any special instructions for storage on Material Safety Data Sheet (e.g. maximum storage quantities).

SECTION 8. EXPOSURE CONTROLS/PERSONAL PROTECTION

NOTE: Exposure to this material can be controlled in many ways. The measures appropriate for a particular worksite depend on how this material is used and on the extent of exposure. This general information can be used to help develop specific control measures. Ensure that control systems are properly designed and maintained. Comply with occupational, environmental, fire, and other applicable regulations.

Sampling and Analysis:

Use appropriate instrumentation and sampling strategy (location, timing, duration, frequency, and number of samples). Interpretation of the sampling results is related to these variables and the analytical method. Sampling should be carried out by trained personnel.

OSHA Analytical Methods:

OSHA METHOD NO. 69 - OSHA Analytical Methods Manual, 2nd edition. (67). Acetone. Validated method. Collection on carbosieve S-III (carbon based molecular sieve) sorbent tube. Desorption with 1% dimethylformamide in carbon disulfide (CS₂) in presence of magnesium sulfate. Analysis by gas chromatography using flame ionization detector (FID). Detection limit: 14.1 ug per sample (2.0 ppm or 4.7 mg/m³) overall.

NIOSH Analytical Methods:

*NOTE: The method described below has been reported for ketones including acetone. NIOSH METHOD 1300, Issue 2 - NIOSH Manual of Analytical Methods. 4th ed. (68). Ketones I. Fully evaluated method. Collection on coconut shell activated charcoal sorbent tube. Desorption with carbon disulfide (CS₂). Analysis by gas chromatography using flame ionization detector (FID). Estimated LOD: 0.02 mg per sample.

Direct Reading Instrumentation:

Methods of detection in commercially available devices which may be suitable: flame ionization detector, infrared photometer, photoionization analyzer, gas chromatograph analyzer.

Colorimetric Detector Tubes:

Commercially available.

Passive Sampling Devices:

Commercially available.

Engineering Controls:

Engineering control methods to reduce hazardous exposures are preferred. General methods include mechanical ventilation (dilution and local exhaust), process or personnel enclosure, control of process conditions and process modification (e.g., substitution of a less hazardous material). Administrative controls and personal protective equipment may also be required. Use a non-sparking, grounded ventilation system separate from other exhaust ventilation systems. Exhaust directly to the outside. Supply sufficient replacement air to make up for air removed by exhaust systems.

Personal Protective Equipment:

If engineering controls and work practices are not effective in controlling exposure to this material, then wear suitable personal protective equipment including approved respiratory protection. Have appropriate equipment available for use in emergencies such as spills or fire.

If respiratory protection is required, institute a complete respiratory protection program including selection, fit testing, training, maintenance and inspection. Refer to the CSA Standard Z94.4-02, "Selection, Use and Care of Respirators", available from the Canadian Standards Association.

Respiratory Protection Guidelines:

NIOSH RECOMMENDATIONS FOR ACETONE CONCENTRATIONS IN AIR (64):

UP TO 2,500 ppm: Chemical cartridge respirator with organic vapour cartridge(s); or powered air-purifying respirator with organic vapour cartridge(s); or gas mask with organic vapour canister; or SAR; or full-facepiece SCBA.

EMERGENCY OR PLANNED ENTRY INTO UNKNOWN CONCENTRATIONS OR IDLH CONDITIONS: Positive pressure, full-facepiece SCBA; or positive pressure, full-facepiece SAR with an auxiliary positive pressure SCBA.

ESCAPE: Gas mask with organic vapour canister; or escape-type SCBA.

NOTE: Substance reported to cause eye irritation or damage; may require eye protection.

NOTE: The IDLH concentration for acetone is 2,500 ppm (10% of the Lower Explosive Limit).

The purpose of establishing an IDLH value is to ensure that the worker can escape from a given contaminated environment in the event of failure of the most protective respiratory protection equipment. In the event of failure of respiratory protective equipment every effort should be made to exit immediately.

Air-purifying respirators do not protect against oxygen-deficient atmospheres.

Recommendations apply only to NIOSH approved respirators.

The respirator use limitations specified by the approving agency and the manufacturer must be observed.

ABBREVIATIONS: SAR = supplied-air respirator; SCBA = self-contained breathing apparatus; IDLH = Immediately Dangerous to Life or Health.

Eye/Face Protection:

Splash-proof chemical safety goggles and face-shield, as required.

Skin Protection:

Chemical protective gloves, coveralls, boots, and/or other chemical protective clothing to prevent repeated or prolonged contact.

Resistance of Materials for Protective Clothing:

Guidelines for acetone: (65)

RECOMMENDED (resistance to breakthrough longer than 8 hours): Butyl rubber, Barrier (PE/PA/PE), Silver Shield/4H(TM) (polyethylene/ethylene vinyl alcohol), Trelchem(TM) HPS, Trelchem(TM) VPS, Tychem(TM) CPF 3, Tychem(TM) F, Tychem(TM) BR/LV, Tychem(TM) Responder(TM), Tychem(TM) TK.

CAUTION, use for short periods only (resistance to breakthrough within 1 to 4 hours): Viton(TM)/Butyl rubber.

NOT RECOMMENDED for use (resistance to breakthrough less than 1 hour): Natural rubber, neoprene rubber, nitrile rubber, polyethylene, polyvinyl alcohol, polyvinyl chloride, Viton(TM), Tychem(TM) SL.

Recommendations are NOT valid for very thin Natural rubber, Neoprene, Nitrile and PVC gloves (0.3 mm or less).

Resistance of specific materials can vary from product to product. Breakthrough times are obtained under conditions of continuous contact, generally at room temperature. Evaluate resistance under conditions of use and maintain clothing carefully.

Personal Hygiene:

Due to the fire hazard, have a safety shower/eyewash fountain readily available in the immediate work area. Remove contaminated clothing immediately. Keep contaminated clothing in closed containers. Launder before re-wearing. Inform laundry personnel of contaminant's hazards. Do not smoke, eat or drink in work areas. Wash hands thoroughly after handling this material. Maintain good housekeeping.

EXPOSURE GUIDELINES

THRESHOLD LIMIT VALUES (TLVs) / AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH) / 2010

Time-Weighted Average (TLV-TWA):	500 ppm - Carcinogenicity Designation A4
Short-Term Exposure Limit (TLV-STEL):	750 ppm - Carcinogenicity designation A4
TLV Basis - Critical Effect(s):	Upper respiratory tract and eye Irritation CNS (Central Nervous System) impairment Hematologic (blood) effects

TLV Definitions:

CARCINOGENICITY DESIGNATION A4 - Not Classifiable as a Human Carcinogen: Inadequate data on which to classify the substance as a human and/or animal carcinogen.

TLV Comments:

BIOLOGICAL EXPOSURE INDICES (BEIs): The ACGIH has adopted a BEI for this chemical. BEIs provide an indication of worker exposure by measuring the chemical or its breakdown products in the body or by measuring biochemical changes resulting from exposure to the chemical. Consult the BEI documentation for further information.

NOTE: In many jurisdictions, exposure limits are similar to the ACGIH TLVs. Since the manner in which exposure limits are established, interpreted, and implemented can vary, obtain detailed information from the appropriate government agency in each jurisdiction.

PERMISSIBLE EXPOSURE LIMITS (PELs) / FINAL RULE LIMITS / US OCCUPATIONAL SAFETY AND HEALTH ADMINISTRATION (OSHA)

Time-Weighted Average (PEL-TWA): 750 ppm

Short-Term Exposure Limit (PEL-STEL): 1000 ppm

NOTE: The OSHA PEL Final Rule Limits are currently non-enforceable due to a court decision. The OSHA PEL Transitional Limits are now in force.

PERMISSIBLE EXPOSURE LIMITS (PELs) / TRANSITIONAL LIMITS / US OCCUPATIONAL SAFETY AND HEALTH ADMINISTRATION (OSHA)

Time-Weighted Average (PEL-TWA): 1000 ppm (2400 mg/m³)

Transitional Limit PEL Comments:

These Permissible Exposure Limits are taken from 29 CFR 1910.1000 Table Z - 1.

SECTION 9. PHYSICAL AND CHEMICAL PROPERTIES

Molecular Weight: 58.08

Conversion Factor:

1 ppm = 2.37 mg/m³; 1 mg/m³ = 0.42 ppm at 25 deg C (calculated)

Physical State: Liquid

Melting Point: -94.7 deg C (-138.5 deg F) (51,61,63)

Boiling Point: 56.2 deg C (133.2 deg F) (51,56)

Decomposition Temperature: Not available.

Relative

Density (Specific Gravity): 0.791 at 20 deg C (51,57); 0.785 at 25 deg C (61,63) (water = 1)

Solubility in Water: Soluble in all proportions (50,51,57)

Solubility in Other Liquids: Soluble in all proportions in ethanol and other lower molecular weight alcohols, diethyl ether, other ethers, benzene, chloroform, carboxylic acids, dimethylformamide, other polar organic solvents, and most oils. Miscible in limited proportions in nonpolar solvents, such as hydrocarbons.(51,57,63,70)

Coefficient of Oil/Water

Distribution (Partition Coefficient):	Log P(oct) = -0.24 (measured) (71)
pH Value:	Not available.
Acidity:	Very weak acid (60,75)
Dissociation Constant:	pKa = Approximately 19-20 (60,75)
Viscosity-Dynamic:	0.32 mPa.s (0.32 centipoises) at 20 deg C (50,51); 0.31 mPa.s (0.31 centipoises) at 25 deg C (57,63)
Viscosity-Kinematic:	0.40 mm ² /m (0.40 centistokes) at 20 deg C; 0.39 mm ² /s (0.39 centistokes) at 25 deg C (calculated)
Saybolt Universal Viscosity:	26.9 Saybolt Universal Seconds at 37.8 deg C (100 deg F) (calculated)
Surface Tension:	24.02 mN/m (24.02 dynes/cm) at 20 deg C; 23.46 mN/m (23.46 dynes/cm) at 25 deg C (calculated) (72)
Vapour Density:	2.0 (air = 1) (calculated)
Vapour Pressure:	24.7 kPa (185 mm Hg) at 20 deg C (50); 30.67-30.93 kPa (230-232 mm Hg) at 25 deg C (experimental) (73,74)
Saturation Vapour Concentration:	243800 ppm (24.38%) at 20 deg C; 302700-305300 ppm (30.53-30.27%) at 25 deg C (calculated)
Evaporation Rate:	5.6 (n-butyl acetate = 1) (58,61); 2.0 (diethyl ether = 1) (58)
Henry's Law Constant:	4.02 Pa.m ³ /mol (cited as 3.97 x 10 ⁽⁻⁵⁾ atm.m ³ /mol) at 25 deg C (73); log H = -2.79 (dimensionless constant; calculated); 3.93 Pa.m ³ /mol (cited as 3.88 x 10 ⁽⁻⁵⁾ atm.m ³ /mol) at 25 deg C (74); log H = -2.8 (dimensionless constant; calculated)

Other Physical Properties:

TRIPLE POINT: -94.7 deg C (-138.5 deg F) (50)

DIELECTRIC CONSTANT: 21.0 at 20 deg C; 20.7 at 25 deg C (57,63)

SECTION 10. STABILITY AND REACTIVITY

Stability:

Normally stable. Prolonged exposure to direct sunlight may result in the formation of carbon monoxide.(51)

Possibility of Hazardous Reactions:

None known.

Flammable Gases Released Upon Contact with Water:

None.

Incompatibility - Materials to Avoid:

NOTE: Chemical reactions that could result in a hazardous situation (e.g. generation of flammable or toxic chemicals, fire or detonation) are listed here. Many of these reactions can be done safely if specific control measures (e.g. cooling of the reaction) are in place. Although not intended to be complete, an overview of important reactions involving common chemicals is provided to assist in the development of safe work practices.

STRONG OXIDIZING AGENTS (e.g. bromine, chromium trioxide, chromyl chloride, nitric acid, nitric acid-sulfuric acid mixture, nitrosyl perchlorate or permonosulfuric acid) - may react violently or explosively, with increased risk of fire .(55,56,62,76)
HYDROGEN PEROXIDE or MIXTURES of HYDROGEN PEROXIDE and NITRIC ACID - may overheat and explode violently due to the formation of explosive dimeric and trimeric cyclic peroxides.(62)

HEXACHLOROMELAMINE or TRICHLOROMELAMINE - violent reaction with ignition. May explode if amounts are large and confined.(55)

NITRIC ACID and ACETIC ACID - mixture can explode.(55)

POTASSIUM TERT-BUTOXIDE - contact of solid butoxide with acetone vapour caused ignition after 4 minutes.(55,62)

ACTIVATED CARBON - ignites on contact.(62,76)

HALOGENATED SOLVENT/ALKALI MIXTURES (e.g. chloroform or bromoform and potassium hydroxide) - may react violently, with evolution of heat.(55,62,76)

STRONG REDUCING AGENTS (e.g. phosphorus, tin (II) chloride, metal hydrides) - may react violently. Increased risk of fire.

SULFUR DICHLORIDE - reaction may be vigorous.(62)

2-METHYL-1,3-BUTADIENE (ISOPRENE) - isoprene-acetone mixtures may form peroxides.(62,76)

Hazardous Decomposition Products:

During a fire, irritating/toxic gases, such as carbon monoxide, carbon dioxide and other toxic and irritating compounds, such as formaldehyde, methanol, acetic acid, hydrogen peroxide, methane and ethylene oxide may be formed, depending on fire conditions.(77,78)

Conditions to Avoid:

Open flames, sparks, electrostatic discharge, heat and other ignition sources; prolonged exposure to direct sunlight.

Corrosivity to Metals:

Acetone is not corrosive to the common metals at room temperature. Aluminum has shown resistance to acetone at all temperatures.(79,80) Redistilled acetone was reported to have caused mild corrosion in an aluminum storage tank.(79) Acetone is not corrosive at normal temperatures to stainless steels (e.g. types 301, 304, 316, 321, 347, 17-4PH and 400 series), carbon steel (types 1010, 1020, 1075, 1095), cast iron, ductile cast iron, high nickel cast iron (Ni-resist), high silicon cast iron (Duriron), nickel and nickel-base alloys, Monel, Hastelloy B/B-2 and C/C-22/C276, Inconel and Incoloy, Carpenter 20Cb-3, copper and alloys, such as 70-30 and 90-10 copper nickel, silicon copper, bronze, aluminum bronze, naval bronze (air free), high and low silicon bronze, brass, admiralty brass, cartridge brass, dry naval and yellow brass (air free), tantalum, titanium and zirconium.(79,80,81)

Corrosivity to Non-Metals:

Acetone attacks plastics, such as chlorinated polyvinyl chloride (CPVC), polyvinyl chloride (PVC), polyvinylidene fluoride (PVDF; Kynar), acrylonitrile-butadiene-styrene (ABS), styrene-acrylonitrile (SAN), polyurethane (rigid), high density polyethylene (HDPE), crosslinked polyethylene (XLPE), polyethylene terephthalate (PET), thermoset polyesters (bisphenol-A fumarate, halogenated, isophthalic acid and general purpose), polyphenylene oxide (Noryl), thermoset epoxy, polystyrene and ethylene vinyl acetate (EVA) (80,82); elastomers, such as nitrile Buna N (NBR), Viton A, chloroprene (neoprene), polyurethane, natural rubber, isoprene, chlorosulfonated polyethylene (CSM; Hypalon), fluorosilicone, flexible polyvinyl chloride (PVC), ethylene vinyl acetate (EVA), low-density polyethylene (LDPE) and Fluoraz (80,83); and coatings, such as coal tar epoxy, epoxy (general purpose, chemical resistant and polyamide oil base) (80). Acetone does not attack plastics, such as Teflon and other fluorocarbons like ethylene tetrafluoroethylene (EFTE; Tefzel), ethylene chlorotrifluoroethylene (ECTFE; Halar) and chlorotrifluoroethylene (CTFE; Kel-F), polyvinylidene chloride (Saran), polypropylene (PP), nylon, polyetherether ketone (PEEK) and ultra high molecular weight polyethylene (UHMWPE) (80,82); elastomers, such as ethylene propylene (EP), ethylene propylene diene (EPDM), butyl rubber (IIR; isobutylene isoprene), Teflon, Chemraz, Kalrez, chlorinated polyethylene (CM), and nylon 11 and nylon 12 (80,83); and coatings, such as polyester, urethanes, vinyls and zinc rich (80).

SECTION 11. TOXICOLOGICAL INFORMATION

LC50 (male rat): 30000 ppm (4-hour exposure); cited as 71000 mg/m³ (4-hour exposure) (29)

LC50 (female rat): 32000 ppm (4-hour exposure); cited as 76.0 mg/L (4-hour exposure) (47)

LC50 (male mouse): 18600 ppm (4-hour exposure); cited as 44000 mg/m³

(4-hour exposure) (29)

LD50 (oral, female rat): 5800 mg/kg (24)

LD50 (oral, adult male rat): 6700 mg/kg (cited as 8.5 mL/kg) (31)

LD50 (oral, newborn rat): 1750 mg/kg (cited as 2.2 mL/kg) (31)

LD50 (oral, mouse): 3000 mg/kg (32,unconfirmed)

LD50 (oral, male mouse): 5245 mg/kg (cited as 90.39 mMol/kg) (84)

LD50 (dermal, rabbit): greater than 15800 mg/kg (cited as greater than 20 mL/kg) (30)

Eye Irritation:

Acetone is a severe irritant.

Application of 0.1 mL of undiluted acetone caused severe irritation in rabbits, which completely reversed by 21 days. Average scores at 24, 48 and 72 hours for each of 4 rabbits were: corneal opacity: 2/4, 2/4, 2/4, 1.67/4; iris injury: 1.33/2, 1.33/2, 1.33/2, 1/2; redness: 3/3, 3/3, 2.67/3; 2.67/3; chemosis: 3/4, 2.67/4, 2.33/4, 2/4.(69) Application of 0.1 mL undiluted acetone caused severe irritation in rabbits (maximum average scores: redness: 2.3/3 (48 hours); iris injury: 0.5/2 (48 hours); chemosis: 0.8/4 (24 and 48 hours); corneal opacity: 1/4 (48 hours).(85) No information on reversibility was reported. Application of 0.005 mL of undiluted acetone produced severe irritation in rabbits (graded 5/10).(30) Application of 0.1 mL undiluted acetone resulted in severe irritation in rabbits (scored 66/110), while 1-30% solutions resulted in minimal irritation (scored 6/110 for 30% acetone).(33) In a modified Draize test, application of 0.1 mL undiluted acetone was reported to cause eye irritation in rabbits, which persisted for more than 21 days.(34) No scoring information was provided.

Skin Irritation:

Acetone is a non-irritant to very mild irritant.

Application of 0.01 mL undiluted acetone produced no irritation in rabbits (graded 1/10).(30) Application of 80 microL to the ears of mice caused very little swelling.(86) Application of 10 microL to intact skin, 6-8 times over 3 days, caused no irritation in guinea pigs.(35) Application of 200 microL, 4 times over 2 weeks, caused no inflammation or swelling in mice. Slight skin shedding (desquamation) was seen in 2/16 mice.(87) In an unconfirmed study, application of 500 mg for 24 hours caused mild irritation in rabbits.(32, unconfirmed) No further details were provided.

Effects of Short-Term (Acute) Exposure:

The primary effect of short-term high-level exposure is central nervous system depression.

Inhalation:

Numerous studies have evaluated the effects of acetone on the central nervous system (CNS). The degree of CNS depression depends on both the concentration of acetone and the length of exposure. Drowsiness, incoordination, loss of reflexes, unconsciousness, respiratory failure and death have been observed. In general, acetone concentrations in excess of 8000 ppm are required to produce symptoms, regardless of the exposure duration and species tested.(6) Several studies have evaluated behavioural responses in animals following acetone exposure (for example, avoidance/escape behaviours). The results of these studies have been variable and it is not possible to draw conclusions from them.(6) In one study, 10700 ppm for 30 minutes was the concentration required to reduce a behavioural response in mice by 50%. The response returned to normal 30 minutes after the exposure ended.(36) In general, the behavioural effects resulting from short exposures to high acetone concentrations are quickly and completely reversed.(88) The concentration of acetone that reduced the respiratory rate of mice by 50% (RD50) was reported to be 23480 ppm for a 5-minute exposure (37) and 77516 ppm for a 10-minute exposure.(38) The RD50 is a measure of sensory irritation (nose, throat and respiratory irritation). These results indicate that

acetone is a weak sensory irritant.

Skin Contact:

Application of 0.5 mL to the skin, 3 times a week for 3 weeks resulted in the formation of cataracts in 2/12 guinea pigs. Application of 1 mL, twice a day, 5 d/wk for 4 weeks (4 guinea pigs, 1 rabbit) or 8 weeks (4 guinea pigs, 2 rabbits) resulted in cataracts in 2 of the guinea pigs exposed for 8 weeks.(41) In a further study, 10 rabbits treated with 1 mL, 3 times a week for 3 weeks did not develop cataracts. The author suggested that the development of cataracts may be specific to guinea pigs.(89) In a more recent study, application of 0.5 mL, 5 d/wk for 6 months to guinea pigs resulted in no cataracts.(90)

Ingestion:

Oral exposure to large doses of acetone in drinking water for 14 days has produced mild toxicity in rats and mice. Compared to controls, male rats receiving approximate reported doses of 4310 or 6940 mg/kg/day and female rats given 8560 mg/kg/day had lower mean body weights. Male mice given 10310 mg/kg/day had lower body weight gain, while no decrease was observed in female mice given up to 12725 mg/kg/day. Slight liver injury was observed in female mice exposed to 8800 mg/kg/day and male mice exposed to 3900 mg/kg/day.(39) In a study designed to evaluate the aspiration risk, acetone was found to evaporate too quickly to be aspirated. However, if ingested very quickly, acetone can be an aspiration hazard.(40) Chemicals in the brain (neurotransmitters and their metabolites) were measured in male rats given a single oral dose of 2438 mg/kg. There was a significant increase in a dopamine metabolite but no changes in other neurotransmitters.(91) The toxicity of acetone to the immune system was measured in male mice given 0, 121, 621 or 1144 mg/kg/day in the drinking water for 28 days. There were no effects on antibody response, blood composition, the spleen or thymus gland.(92)

Effects of Long-Term (Chronic) Exposure:

Long-term oral exposure has caused kidney damage (degeneration of the tubules) in rats (both sexes) at 500 mg/kg/day and higher and increased liver weight at 1600 mg/kg and higher.

Inhalation:

No significant harmful effects were observed in rats exposed by inhalation to 19000 ppm (3 hr/day, 5 d/wk) for 8 weeks.(42) Male rats exposed to 0, 1000, 2000 or 4000 ppm for 13 weeks (6 hr/day, 5 d/wk) had no effects on the performance of a previously learned complex task.(88)

Ingestion:

Mild harmful effects were observed in rats and mice exposed to high oral doses for 13 weeks. Rats were given 0, 0.25, 0.5, 1, 2 or 5% acetone in the drinking water for 13 weeks. Approximate reported doses for males were 0, 200, 400, 900, 1700 and 3400 mg/kg/day, while females received 0, 300, 600, 1200, 1600 and 3100 mg/kg/day. Rats receiving the high dose had decreased body weight. Liver and kidney weights were increased in rats receiving 1600 mg/kg/day or greater. Changes consistent with macrocytic anemia were observed at 400 mg/kg/day (males) and at 1600 (males) or 1700 (females) mg/kg/day. Dose-related kidney damage was observed in males. No hyaline droplet formation was observed (an effect specific to male rats). Mice were similarly exposed. Approximate reported doses for males ranged from 380-4858 mg/kg/day for males and 892-11298 mg/kg/day for females. Liver weights were increased and spleen weights decreased in females given 11298 mg/kg/day. The volume of red blood cells was increased in females at 11298 mg/kg/day and hemoglobin was increased in females at 5945 mg/kg/day and higher and in males at 1353 mg/kg/day and higher.(39) In an unpublished study, which was conducted according to OECD guidelines, rats were given 0, 100, 500 or 2500 mg/kg/day for up to 90 days. There were no significant effects on body weights. At 2500 mg/kg/day, significant effects included increased hemoglobin and red blood cell volume, increased liver and kidney

weights and decreased brain weight (males only). Females given 500 mg/kg/day also had increased kidney weights. An increase in the severity of kidney tubule degeneration was seen in both sexes at 500 mg/kg/day and higher.(93,94-unconfirmed) Male rats given 0.5% acetone in the drinking water for 6 weeks (approximate dose 500 mg/kg/day) had no change in balance time in the rotarod test and little or no change in nerve conduction velocity.(95) In a related study, no significant changes in behaviour, balance time in the rotarod test or hind limb strength were observed in male rats administered 0.5% acetone in their drinking water for 6 weeks (approximate dose 500 mg/kg/day).(26) Neurotoxic effects (degeneration of peripheral nerves or decrease in hind limb strength) were not observed in three rats exposed to 0.5-1% acetone in their drinking water for 12 weeks (approximate dose 500-1000 mg/kg/day).(43)

Skin Sensitization:

Acetone is not a skin sensitizer.

Negative results were obtained in two mouse-ear swelling tests, a Guinea Pig Maximization Test and a Buehler test using guinea pigs.(44,96,97)

Carcinogenicity:

Acetone is not known to be a carcinogen.

It has been used as a vehicle in dermal studies using mice. Mice generally received one or two 0.2 mL applications/week for 6 months to 2 years without an increased incidence of tumours.(6,39,94) In an alternative cancer bioassay, 200 microL/day was applied for 26 weeks (7 d/wk) to the skin of mice that were genetically altered to be more sensitive to tumour-promoting chemicals. There was no increase in the incidence of skin cancers in comparison with untreated controls.(98)

Teratogenicity, Embryotoxicity and/or Fetotoxicity:

The information located is not sufficient to conclude that acetone causes developmental toxicity. Inhalation of acetone has caused fetotoxicity in rats and mice and embryotoxicity in mice, but only at concentrations that also caused maternal toxicity. A limited oral study in mice showed fetotoxicity and embryotoxicity at a dose that did not cause decreased maternal body weight during pregnancy.

Rats were exposed by inhalation to 0, 440, 2200 or 11000 ppm acetone on days 6-19 (6 hr/d) of pregnancy. Decreased body weight was observed in mothers exposed to 11000 ppm. The only statistically significant effect observed in the offspring was fetotoxicity (reduced fetal weight) at 11000 ppm. Although not significant, the percent of litters containing at least one pup with a malformation was 11.5% in the 11000 ppm group (3/26 litters) compared with 3.8% for the control group (1/26 litters).(45)

Mice were exposed by inhalation to 0, 440, 2200 or 6600 ppm acetone on days 6-17 (6 hr/d) of pregnancy. The high exposure group animals were initially exposed to 11000 ppm for one day and then the concentration was decreased to 6600 ppm because the mice experienced severe narcosis. Minimal maternal toxicity (increased liver weight) was then observed at 6600 ppm. Fetotoxicity (reduced fetal weight) and slight, but statistically significant, embryotoxicity (fetal deaths) were observed at 6600 ppm.(45) In a preliminary screening test (the Chernoff/Kavlock test), acetone was administered orally at a dose of 0 or 3500 mg/kg/day to female mice on days 6-15 of pregnancy. Acetone-exposed mothers had no significant decrease in body weight during pregnancy, however their body weight was significantly lower than controls 3 days after giving birth. In the offspring, there were significant decreases in pup weight at birth, the number of viable litters and pup survival after birth. The results indicated that acetone warranted high priority for additional developmental toxicity testing.(46) This study is limited by the use of a single dose.

Reproductive Toxicity:

The information located is not sufficient to conclude that acetone causes reproductive toxicity. Effects on sperm have been observed in rats exposed orally to a dose that caused significant other toxicity. No effects on fertility have been observed.

Rats and mice were exposed to up to 50000 ppm acetone in drinking water for 13 weeks. Sperm motility was decreased and the percentage of abnormal sperm was increased in male rats, at the high dose (reported approximate dose 3400 mg/kg/day). These same male rats had experienced kidney damage. Similar effects were not observed in the mice.(39) No testicular toxicity or effects on fertility were observed in male rats exposed to 0.5% acetone in their drinking water for 6 weeks

(approximate dose 500 mg/kg/day).(25)

Mutagenicity:

Acetone is not known to be a mutagen. There are no confirmed studies that show mutagenicity in live animals. Negative results have been obtained in most studies with cultured mammalian cells and bacteria. Positive and negative results have been obtained in yeast. Acetone is often used as a solvent for testing the mutagenicity of other chemicals and as the solvent control in these assays.(28)

In an unpublished study, negative results (micronucleus) were obtained in the peripheral blood cells of mice that received 5000-20000 ppm acetone in drinking water for 13 weeks. Approximate doses were 1000-4000 mg/kg/day.(39, unconfirmed) No conclusions can be drawn from an unconfirmed report of sex chromosome loss and nondisjunction in mice exposed by inhalation to approximately 5000 ppm (cited as 12 g/L).(32, unconfirmed) No further details were provided. Negative results (point mutations, chromosome aberrations, sister chromatid exchanges, unscheduled DNA synthesis, micronucleus) have been obtained in most studies using cultured mammalian cells, with and without metabolic activation.(6,23,28,39,48,93) Negative results (point mutations, DNA damage, DNA repair and DNA binding) have also been obtained in many studies using bacteria, with and without metabolic activation.(6,23,28,39,93) Results in yeast have been positive (aneuploidy) and negative (aneuploidy, point mutations, mitotic recombination).(6,23,28,49)

Toxicological Synergisms:

Acetone has increased the liver and/or kidney toxicity of many chemicals including carbon tetrachloride, chloroform, trichloroethylene, bromodichloromethane, dibromochloromethane, N-nitrosodimethylamine and 1,1,2-trichloroethane. It also enhances the lung toxicity of styrene, the lethality of acetonitrile and the neurotoxicity 2,5-hexanedione in laboratory animals.(6,22,23,24,25,26,27,95) It appears to inhibit the metabolism and elimination of ethyl alcohol, thereby potentially increasing its toxicity.(23) Acetone can either increase or decrease the toxicity of 1,2-dichlorobenzene, depending on the concentration of acetone used.(6)

SECTION 12. ECOLOGICAL INFORMATION

NOTE : Inclusion of Ecological Information on an MSDS is optional under the US Hazard Communication Standard and the Canadian Controlled Products Regulations (WHMIS). In other jurisdictions, inclusion of Ecological Information may be a requirement. For specific requirements, contact the relevant regulatory authorities in the jurisdiction where the MSDS is intended to be used.

The American National Standard for Hazardous Industrial Chemicals - Material Safety Data Sheets - Preparation (ANSI Z400.1-2004) provides advice on data that could be included in this section.

Databases in CCOHS's CD-ROM and Web collection which contain useful Ecological Information include [CESARS](#), [HSDB® \(Hazardous Substances Data Bank\)](#) and [CHRIS \(Chemical Hazards Response Information System\)](#).

SECTION 13. DISPOSAL CONSIDERATIONS

Review federal, provincial and local government requirements prior to disposal. Store material for disposal as indicated in Storage Conditions. Disposal by controlled incineration or secure landfill may be acceptable.

SECTION 14. TRANSPORT INFORMATION

CANADIAN TRANSPORTATION OF DANGEROUS GOODS (TDG) SHIPPING INFORMATION

Shipping Name and Description: ACETONE

UN Number: UN1090

Class: 3

Packing Group/Category: II

Special Provisions: ---

Passenger Carrying Road/Railway Vehicle Index: 5 kg or L

Marine Pollutant: ---

NOTE: This information incorporates the Transportation of Dangerous Goods Regulations SOR/2001-286, effective October 14, 2009.

US DEPARTMENT OF TRANSPORT (DOT) HAZARDOUS MATERIALS SHIPPING INFORMATION (49 CFR)

Shipping Name and Description: ACETONE

Hazard Class or Division: 3

Identification Number: UN1090

Packing Group: II

NOTE: This information was taken from the US Code of Federal Regulations Title 49 - Transportation and is effective July 1, 2009.

SECTION 15. REGULATORY INFORMATION

CANADIAN WORKPLACE HAZARDOUS MATERIALS INFORMATION SYSTEM (WHMIS)

CCOHS WHMIS Classification:

B2 - Flammable and combustible material - Flammable liquid

D2B - Poisonous and infectious material - Other effects - Toxic



B2 - Flammable Liquid



D2B - Toxic

WHMIS Health Effects Criteria Met by this Chemical:

D2B - Eye irritation - toxic - other

WHMIS Ingredient Disclosure List:

Included for disclosure at 1% or greater.

Detailed WHMIS Classification According to Criteria:

Class A - Compressed Gas:

Does not meet criteria.

Class B - Flammable and Combustible Material:

Meets criteria for "Flammable liquid".

Closed cup flash point: -20 to -18 deg C (-4 to 0 deg F).

Class C - Oxidizing Material:

Does not meet criteria.

Class D - Poisonous and Infectious Material. Division 1 - Immediate and Serious Toxic Effects:

Does not meet criteria.

Acute Lethality:

Does not meet criteria.

LC50 (male rat): 30000 ppm (4-hour exposure); cited as 71000 mg/m³ (4-hour exposure); LC50 (male mouse): 18600 ppm (4-hour exposure); cited as 44000 mg/m³ (4-hour exposure); LD50 (oral, female rat): 5800 mg/kg; LD50 (oral, mouse): 3000 mg/kg (unconfirmed); LD50 (dermal, rabbit): greater than 15800 mg/kg (cited as greater than 20 mL/kg)

Class D - Poisonous and Infectious Material. Division 2 - Other Toxic Effects:

Meets criteria for "Toxic material".

See detailed evaluation below.

Chronic Health Effects:

Does not meet criteria.

Long-term animal studies have not shown significant harmful effects from inhalation exposures. Long-term oral exposure of rats has caused kidney damage and increased liver weight at relatively high doses.

Carcinogenicity:

Does not meet criteria.

Not listed by IARC.

ACGIH A4.

Teratogenicity and Embryotoxicity:

Does not meet criteria.

No conclusions can be drawn based on the limited human information available. In animal studies, inhalation of acetone caused fetotoxicity in rats and mice and embryotoxicity in mice, but only at concentrations that also caused maternal toxicity. A limited oral study in mice showed fetotoxicity and embryotoxicity at a dose that did not cause decreased maternal body weight during pregnancy.

Reproductive Toxicity:

Does not meet criteria.

No conclusions can be drawn from the limited human information available. In an oral study in rats, effects on sperm were observed at a dose that caused significant other toxicity.

Mutagenicity:

Does not meet criteria.

No human information was located. There are no confirmed studies that show mutagenicity in live animals. Acetone is often used as a solvent for testing the mutagenicity of other chemicals and as the solvent control in these assays.

Respiratory Tract Sensitization:

Does not meet criteria.

Not reported as a human respiratory sensitizer.

Skin Irritation:

Does not meet criteria.

Non-irritant to very mild irritant based on animal and limited human information.

Eye Irritation:

"Toxic".

Severe irritant based on animal and limited human information.

Skin Sensitization:

Does not meet criteria.

Negative results have been obtained in tests in humans and, despite

widespread industrial use, no conclusive case reports of sensitization were located. Negative results were obtained in animal tests.

Class E - Corrosive Material:

Does not meet criteria.

Not corrosive to the skin. Not corrosive to aluminum or type 1020 carbon steel.

Class F - Dangerously Reactive Material:

Does not meet criteria.

US OCCUPATIONAL SAFETY AND HEALTH ADMINISTRATION (OSHA) HAZARD COMMUNICATION STANDARD (29 CFR 1910.1200)

OSHA Hazard Communication Evaluation:

Meets criteria for hazardous material, as defined by 29 CFR 1910.1200.

EUROPEAN UNION (EU) CLASSIFICATION AND LABELLING INFORMATION

This EU classification information reflects the 29th Adaptation to Technical Progress (ATP) of Council Directive 67/548/EEC. The EU has adopted the 30th ATP (2008/58/EC of 21 August 2008) and 31st ATP (2009/2/EC of 15 January 2009) of this Council Directive. See: <http://ecb.jrc.ec.europa.eu/esis> for current information.

EU Classification:

Highly flammable. Highly flammable. [F;R11] Irritant. Irritating to eyes. [Xi;R36] Repeated exposure may cause skin dryness or cracking. [R66] Vapours may cause drowsiness and dizziness. [R67] (66)

EU Risk Phrases:

Highly flammable. Irritating to the eyes. Repeated exposure may cause skin dryness or cracking. Vapours may cause drowsiness and dizziness. [R:11-36-66-67]

EU Safety Phrases:

Keep out of reach of children.* Keep container in a well ventilated place. Keep away from sources of ignition - No smoking. In case of contact with eyes, rinse immediately with plenty of water and seek medical advice. [S(2-)*9-16-26]
*This safety phrase can be omitted from the label when the substance or preparation is sold for industrial use only.

EU Comments:

NOTES RELATED TO PREPARATIONS:
Preparations containing this substance have to be assigned R67 (Vapours may cause drowsiness and dizziness) if they meet the EC criteria for volatile substances which cause clear signs of central nervous system depression and which are not already classified with respect to acute inhalation toxicity.

SECTION 16. OTHER INFORMATION

Selected Bibliography:

- (1) DiVincenzo, G.D., et al. Exposure of man and dog to low concentrations of acetone vapor. American Industrial Hygiene Association Journal. Vol. 34, no. 8 (Aug. 1973). p. 329-336
- (2) Nelson, K.W., et al. Sensory response to certain industrial solvent vapors. Journal of Industrial Hygiene and Toxicology. Vol. 25, no. 7 (Sept. 1943). p. 282-285
- (3) Matsushita, T., et al. Experimental studies for determining the MAC value of acetone: 2. Biological reactions in the "six-day exposure" to acetone. Japanese Journal of Industrial Hygiene (Sangyo Igaku). Vol. 11, no. 10 (Oct. 1969). p. 507-515

- (4) Matsushita, T., et al. Experimental studies for determining the MAC value of acetone: 1. Biological reactions in the "one-day exposure" to acetone. Japanese Journal of Industrial Hygiene (Sangyo Igaku). Vol. 11, no. 9 (Sept. 1969). p. 477-485
- (5) Dick, R.B., et al. Neurobehavioural effects of short duration exposures to acetone and methyl ethyl ketone. British Journal of Industrial Medicine. Vol. 46, no. 2 (Feb. 1989). p. 111-121
- (6) Morgott, D.A. Acetone. In: Patty's toxicology. 5th ed. Edited by E. Bingham, et al. Chpt. 74, Vol. 2. John Wiley and Sons, 2001. Article online posting date: Apr. 16, 2001
- (7) Wysocki, C.J., et al. Acetone odor and irritation thresholds obtained from acetone-exposed factory workers and from control (occupationally unexposed) subjects. American Industrial Hygiene Association Journal. Vol. 58, no. 10 (Oct. 1997). p. 704-712
- (8) Ross, D.S. Acute acetone intoxication involving eight male workers. Annals of Occupational Hygiene. Vol. 16, no. 1 (Apr. 1973). p. 73-75
- (9) Henschler, D. Acetone (Aceton). In: Gesundheitsschaedliche Arbeitstoffe, 1979. Vol. 1, 7th issue, A. p. 1-4. Verlag Chemie GmbH, 1979. (HSE translation no. 10670, Feb. 1984)
- (10) Lupulescu, A.P., et al. An electron microscopic study of human epidermis after acetone and kerosene administration. Journal of Investigative Dermatology. Vol. 60, no. 1 (1973). p. 33-45
- (11) Harris, L.C., et al. Acute acetone poisoning caused by setting fluid for immobilizing casts. British Medical Journal. (Nov. 8, 1952). p. 1024-1026
- (12) Raleigh, R.L., et al. Effects of short, high-concentration exposures to acetone as determined by observations in the work area. Journal of Occupational Medicine. Vol. 14, no. 8 (Aug. 1972). p. 607-610
- (13) McLaughlin, R.S. Chemical burns of the human cornea. American Journal of Ophthalmology. Vol. 29, no. 11 (Nov. 1946). p. 1355-1362
- (14) Grant, W.M., et al. Toxicology of the Eye. 4th ed. Charles C. Thomas, 1993. p. 55-56
- (15) Gitelson, S., et al. Coma and hyperglycemia following drinking of acetone. Diabetes. Vol. 15, no. 11 (Nov. 1966). p. 810-811
- (16) Satoh, T., et al. Cross-sectional study of effects of acetone exposure on workers' health. Proceedings of the 9th International Symposium on Epidemiology in Occupational Health, Sept. 23-25, 1992, Cincinnati, Ohio. p. 407-412. NIOSH Publication No. 94-112. International Commission on Occupational Health, National Institute for Occupational Safety and Health, 1994
- (17) Vigliana, E.C., et al. Experiences of the Clinics del Lavoro with some maximum concentrations of poisons of industry at the place of work (MAK). (English translation of: Erfahrungen der Clinica del Lavoro mit einigen maximalen Arbeitsplatzkonzentrationen (MAK) von Industriegiften. Archiv fur Gewerbepathologie und Gewerbehygiene. Vol. 13 (1955). p. 528-534
- (18) Parmeggiani, L., et al. Occupational poisoning with acetone: clinical disturbances, investigations in work rooms and physiopathological research. Medicina del Lavoro. Vol. 45, no. 8-9 (1954). p. 431-468 (English translation: NIOSHTIC Control Number: 00031405)
- (19) Jelnes, J.E. Semen quality in workers producing reinforced plastic. Reproductive Toxicology. Vol. 2, nos. 3/4 (1988). p. 209-212
- (20) Swan, S.H., et al. Historical cohort study of spontaneous abortion among fabrication workers in the semiconductor health study: agent-level analysis. American Journal of Industrial Medicine. Vol. 28, no. 6 (Dec. 1995). p. 751-769
- (21) Nizaeva, I.V. On hygienic assessment of acetone. (English translation of: K gigenicheskoi otsenke atsetona. Gigiena Truda i Professional'nye Zabolovaniya (Labor Hygiene and Occupational Diseases). Vol. 26, no. 6 (1982). p. 24-28) (HSE translation no. 13720 A, Feb. 1990)
- (22) Hewitt, W.R., et al. Nephrotoxic interactions between ketonic solvents and halogenated aliphatic chemicals. Fundamental and Applied Toxicology. Vol. 4, no. 6 (Dec. 1984). p. 902-908
- (23) International Programme on Chemical Safety (IPCS). Acetone. Environmental Health Criteria 207. World Health Organization, 1998
- (24) Freeman, J.J., et al. Acetone potentiation of acute acetonitrile toxicity in rats. Journal of Toxicology and Environmental Health. Vol. 15, no. 5 (1985). p. 609-621
- (25) Larsen, J.J., et al. Infertility in rats induced by 2,5-hexanedione in combination with acetone. Pharmacology and Toxicology. Vol. 69, no. 1 (July 1991). p. 43-46
- (26) Ladefoged, O., et al. Acetone potentiation and influence on the reversibility of 2,5-hexanedione-induced neurotoxicity studied with behavioural and morphometric methods in rats. Pharmacology and Toxicology. Vol. 74, no. 4-5 (Apr.-May 1994). p.

- (27) Charbonneau, M., et al. Influence of acetone on the severity of the liver injury induced by haloalkane mixtures. *Canadian Journal of Physiology and Pharmacology*. Vol. 69, no. 12 (Dec. 1991). p. 1901-1907
- (28) Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for acetone. TP-93/01. US Department of Health and Human Services, May 1994
- (29) Safronov, N.S., et al. Comparative acute inhalation toxicity of aliphatic aldehydes and ketones according to exposure time. *Current Toxicology*. Vol. 1, no. 1 (1993). p. 47-51
- (30) Smyth, Jr., H.F., et al. Range-finding toxicity data: list VI. *American Journal of Industrial Hygiene*. Vol. 23 (Mar.-Apr. 1962). p. 95-107
- (31) Kimura, E.T., et al. Acute toxicity and limits of solvent residues for sixteen organic solvents. *Toxicology and Applied Pharmacology*. Vol. 19, no. 4 (Aug. 1971). p. 699-704
- (32) MDL Information Systems, Inc. Acetone. Last updated: 2007-08. In: Registry of Toxic Effects of Chemical Substances (RTECS(R)). Available from: Canadian Centre for Occupational Health and Safety (CCOHS)
- (33) Kennah II, H.E., et al. An objective procedure for quantitating eye irritation based upon changes of corneal thickness. *Fundamental and Applied Toxicology*. Vol. 12, no 2 (Feb. 1989). p. 258-268
- (34) Morgan, R.L., et al. Prediction of ocular irritation by corneal pachymetry. *Food and Chemical Toxicology*. Vol. 25, no. 8 (Aug. 1987). p. 609-613
- (35) Anderson, C., et al. Animal model for assessment of skin irritancy. *Contact Dermatitis*. Vol. 15, no. 3 (Sept. 1986). p. 143-151
- (36) Glowa, J.R., et al. Behavioral toxicology of volatile organic solvents: IV. Comparisons of the rate-decreasing effects of acetone, ethyl acetate, methyl ethyl ketone, toluene, and carbon disulfide on schedule-controlled behavior of mice. *Journal of the American College of Toxicology*. Vol. 6, no. 4 (1987). p. 461-469
- (37) De Ceaurriz, J.C., et al. Sensory irritation caused by various industrial airborne chemicals. *Toxicology Letters*. Vol. 9, no. 2 (1981). p. 137-143
- (38) Kane, L.E., et al. Evaluation of sensory irritation from some common industrial chemicals. *American Industrial Hygiene Association Journal*. Vol. 41, no. 6 (June 1980). p. 451-454
- (39) Dietz, D. Toxicity studies of acetone (CAS no. 67-64-1) in F344/N rats and B6C3F1 mice (drinking water studies). NIH Publication No. 91-3122. National Toxicology Program, US Department of Health and Human Services, Jan. 1991
- (40) Panson, R.D., et al. Aspiration toxicity of ketones. *Clinical Toxicology*. Vol. 17, no. 2 (1980). p. 271-317
- (41) Rengstorff, R.H., et al. Cataracts induced in guinea pigs by acetone, cyclohexanone and dimethyl sulfoxide. Edgewood Arsenal technical report: EATR 4550. Edgewood Arsenal Research Laboratories, Department of the Army, Aug. 1971
- (42) Bruckner, J.V., et al. Evaluation of toluene and acetone inhalant abuse: II. Model development and toxicology. *Toxicology and Applied Pharmacology*. Vol. 61, no. 3 (Dec. 1981). p. 302-312
- (43) Spencer, P.S., et al. On the specific molecular configuration of neurotoxic aliphatic hexacarbon compounds caused central-peripheral distal axonopathy. *Toxicology and Applied Pharmacology*. Vol. 44, no. 1 (Apr. 1978). p. 17-28
- (44) Descotes, J. Identification of contact allergens: the mouse ear sensitization assay. *Journal of Toxicology - Cutaneous and Ocular Toxicology*. Vol. 7, no. 4 (Dec. 1988). p. 263-272
- (45) Mast, T.J., et al. Inhalation developmental toxicology studies: teratology study of acetone in mice and rats. Final report. No. NIH-Y01-ES-7- 153. Contract DE-AC06-76RLO 1830. National Toxicology Program, National Institute of Environmental Health Sciences, Nov. 1988
- (46) Environmental Health Research and Testing, Inc. Screening of priority chemicals for reproductive hazards: benzethonium chloride (CAS No. 121-54-0), 3-ethoxy-1-propanol (CAS No. 111-35-3), acetone (CAS No. 67-64-1). Final report. Contract No.: 200-85-2735. EHRT Project No.: ETOX-85-1002. National Institute for Occupational Safety and Health, Apr. 22, 1987
- (47) Pozzani, U.C., et al. The toxicological basis of threshold limit values: 5. The experimental inhalation of vapor mixtures by rats, with notes upon the relationship between single dose inhalation and single dose oral data. *American Industrial Hygiene Association Journal*. Vol. 20 (1959). p. 364-369
- (48) Von der Hude, W., et al. Genotoxicity of three-carbon compounds in the SCE test in vitro. *Environmental Mutagenesis*. Vol. 9, no. 4 (1987). p. 401-410
- (49) Zimmerman, F.K., et al. Acetone, methyl ethyl ketone, ethyl acetate, acetonitrile and other polar aprotic solvents are strong inducers of aneuploidy in *Saccharomyces*

cerevisiae. Mutation Research. Vol. 149 (1985). p. 339-351

(50) Howard, W.L. Acetone. In: Kirk-Othmer encyclopedia of chemical technology. John Wiley and Sons, 2005

(51) Sifniades, S., et al. Acetone. In: Ullmann's encyclopedia of industrial chemistry. 7th ed. John Wiley and Sons, 2005

(52) Acetone. Hawley's condensed chemical dictionary. [CD-ROM]. 15th ed. Edited by R.J. Lewis, Sr. John Wiley and Sons, Inc., 2007

(53) Odor thresholds for chemicals with established occupational health standards. American Industrial Hygiene Association, 1989. p. 12, 42-43

(54) Amoore, J.E. Effects of chemical exposure on olfaction in humans. In: Toxicology of the nasal passages. Edited by C.S. Barrow. Hemisphere Publishing Corporation, 1984. p. 155-190

(55) Fire protection guide to hazardous materials. 13th ed. Edited by A.B. Spencer, et al. National Fire Protection Association, 2002. NFPA 325; NFPA 491; NFPA 497; NFPA 77

(56) Emergency action guide for acetone. Association of American Railroads, Sept. 2006

(57) Speight, J.G. Lange's handbook of chemistry. 16th ed. McGraw-Hill, Inc., 2005. p. 2.66, 2.272, 2.352, 2.426, 2.470, 2.698

(58) Stoye, D, et al. Solvents. In: Ullmann's encyclopedia of industrial chemistry. 7th ed. John Wiley and Sons, 2005

(59) Britton, LG. Using material data in static hazard assessment. Plant/Operations Progress. Vol. 11, no. 2 (Apr. 1992). p. 56-70

(60) Roberts, J.D., et al. Basic principles of organic chemistry. 2nd ed. W.A. Benjamin, Inc., 1977. p. 736-738

(61) Riddick, J.A., et al. Organic solvents: physical properties and methods of purification. 4th ed. Techniques of organic chemistry. Vol. II. John Wiley and Sons, 1986. p. 336-337, 951-955

(62) Bretherick's reactive chemical hazards database. [CD-ROM]. 6th ed. Version 3.0. Edited by P.G. Urben. Butterworth-Heinemann Ltd., 1999

(63) CRC Handbook of chemistry and physics. 86th ed. Edited by D.R. Lide. CRC Press, 2005. p. 3-4, 6-136, 6-176

(64) Acetone. In: NIOSH pocket guide to chemical hazards. National Institute for Occupational Safety and Health

(65) Forsberg, K., et al. Quick selection guide to chemical protective clothing. 5th ed. Wiley Interscience, John Wiley and Sons, 2007

(66) European Communities. Commission Directive 98/98/EC. Dec. 15, 1998

(67) Occupational Safety and Health Administration (OSHA). Acetone. In: OSHA Analytical Methods Manual. Revision Date: Oct. 31, 2001

(68) National Institute for Occupational Safety and Health (NIOSH). Ketones I. In: NIOSH Manual of Analytical Methods (NMAM(R)). 4th ed. Edited by M.E. Cassinelli, et al. DHHS (NIOSH) Publication 94-113. Aug. 1994

(69) European Centre for Ecotoxicology and Toxicology of Chemicals (ECETOC). Eye irritation reference chemicals data bank. 2nd ed. Technical Report No. 48 (2). ECETOC, June 1998. p. 157-158

(70) Acetone. The Merck index: an encyclopedia of Chemicals, drugs and biologicals. Edited by M.J. O'Neil, et al. 14th ed. Merck and Company, 2006. p. 12

(71) Syracuse Research Corporation. Interactive LogKow (KowWin) Database Demo. Date unknown

(72) Jasper, J.J. Surface tension of pure liquid compounds. In: Compilation of data of some 2200 pure liquid compounds. Journal of Physical and Chemical Reference Data. Vol. 1, no. 4 (1972). p. 920

(73) Syracuse Research Corporation. The Physical Properties Database (PHYSPROP). Interactive PhysProp Database Demo. Date unknown

(74) Syracuse Research Corporation. Environmental Fate Database: CHEMFATE Chemical Search. Last updated: 2007-12-06

(75) Streitweiser, A., et al. Introduction to organic chemistry. 4th ed. Revised printing. Prentice Hall, Inc., 1998. p. 423, 537

(76) Armour, M.-A. Acetone. In: Hazardous laboratory chemicals disposal guide. 3rd ed. Lewis Publishers, 2003. p. 5-6

(77) Gasnot, L., et al. Experimental and kinetic analysis of the thermal degradation of the methylethylketone in methane/air flames. Combustion Science and Technology. Vol. 161 (2000). p. 1-25

(78) Hoare, D.E., et al. The combustion of simple ketones. II. Mechanism at 'high' temperatures. Combustion and Flame. Vol. 12 (Apr. 1968). p. 145-154

(79) Acetone. In: Handbook of corrosion data. 2nd ed. Edited by B.D. Craig, et al. ASM International, 1995. p. 111-115

- (80) Schweitzer, P.A. Corrosion resistance tables: metals, nonmetals, coatings, mortars, plastics, elastomers and linings, and fabrics. 4th ed. Part A, A-D. Marcel Dekker, Inc., 1995. p. 45-52
- (81) Pruet, K.M. Chemical resistance guide to metals and alloys: a guide to chemical resistance of metals and alloys. Compass Publications, 1995. p. 2-13
- (82) Pruet, K.M. Chemical resistance guide for plastics: a guide to chemical resistance of engineering thermoplastics, fluoroplastics, fibers and thermoset resins. Compass Publications, 2000. p. 2-13
- (83) Pruet, K.M. Chemical resistance guide for elastomers II: a guide to chemical resistance of rubber and elastomeric compounds. Compass Publications, 1994. p. C-2 to C-7
- (84) Tani, H., et al. Structure-toxicity relationship of monoketones. Toxicology Letters. Vol. 30 (1986). p. 13-17
- (85) Jacobs, G.A. OECD eye irritation tests on two ketones. Journal of the American College of Toxicology. Vol. 1 (1992). p. 190-191
- (86) Iyadomi, M., et al. Evaluation of skin irritants caused by organic solvents by means of the mouse ear thickness measurement method. Journal of Occupational Health. Vol. 42 (2000). p. 44-46
- (87) Dermigen Inc. Dermal irritation study in mice. Date produced: Jan. 21, 1994. American Petroleum Institute. APA/OTS 8494000005. FYI-1193-00904 A
- (88) Christoph, G.R., et al. Subchronic inhalation exposure to acetone vapor and scheduled controlled operant performance in male rats. Inhalation Toxicology. Vol. 15 (2003). p. 781-798
- (89) Rengstorff, R., et al. Attempt to induce cataracts in rabbits by cutaneous application of acetone. American Journal of Optometry and Physiological Optics. Vol. 53, no. 1 (Jan 1976). p. 41-42
- (90) Taylor, A., et al., Relationship between acetone, cataracts, and ascorbate in hairless guinea pigs. Ophthalmology Research. Vol. 25 (1993). p. 30-35
- (91) Kanada, M., et al. Neurochemical profile of effects of 28 neurotoxic chemicals on the nervous system in rats: (1). Effects of oral administration on brain contents of biogenic amines and metabolites. Industrial Health. Vol. 32 (1994). p. 145-164
- (92) Woolhiser, M.R., et al. Acetone in drinking water does not modulate humoral antibody in mice as measured by the antibody, plaque-forming cell assay. International Journal of Toxicology. Vol. 25 (2006). p. 333-339
- (93) Acetone. SIDS initial assessment report for 9th SIAM. UNEP Publications, July 1999
- (94) US Environmental Protection Agency (EPA). Acetone. Last Significant Revision: 2003-05. In: Integrated Risk Information System (IRIS)
- (95) Ladefoged, O., et al. Neurophysiological and behavioral effects of combined exposure to 2,5-hexanedione and acetone or ethanol in rats. Pharmacology and Toxicology. Vol. 65 (1989). p. 372-375
- (96) Gad, S.C., et al. Development and validation of an alternative dermal sensitization test: The mouse ear swelling test (MEST). Toxicology and Applied Pharmacology. Vol. 84 (1986). p. 93-114
- (97) Nakamura, A., et al. A new protocol and criteria for quantitative determination of sensitization potencies of chemicals by guinea pig maximization test. Contact Dermatitis. Vol. 31 (1994). p. 72-85
- (98) Holden, H.E., et al. Hemizygous Tg.AC transgenic mouse as a potential alternative to the two-year mouse carcinogenicity bioassay: Evaluation of husbandry and housing factors. Journal of Applied Toxicology. Vol. 18 (1998). p. 19-24
- (99) Satoh, T., et al. Relationship between acetone exposure concentration and health effects in acetate fiber plant workers. International Archives of Occupational and Environmental Health. Vol. 68 (1996). p. 147-153
- (100) Mitran, E., et al. Neurotoxicity associated with occupational exposure to acetone, methyl ethyl ketone, and cyclohexanone. Environmental Research. Vol. 73 (1997). p. 181-188
- (101) Mitran, E. Reply. Environmental Research Section A Vol. 82 (2000). p. 184-185
- (102) Drexler, H., et al. Skin prick tests with solutions of acid anhydrides in acetone. International Archives of Allergy and Immunology. Vol. 100, no. 3 (1993). p. 251-255
- (103) Tosti, A., et al. Unusual complication of sensitizing therapy for alopecia areata. Contact Dermatitis. Vol. 18, no. 5 (May 1988). p. 322
- (104) Koch, P., Occupational allergic contact dermatitis from epoxy resin systems and possibly acetone in a shoemaker. Contact Dermatitis. Vol. 46 (2002). p. 362-363
- (105) Ott, G.M., et al. Health evaluation of employees occupationally exposed to methylene chloride. General study design and environmental considerations. Scandinavian Journal of Work and Environmental Health. Vol. 9 (1983). p. 1-7
- (106) Ott, G.M., et al. Health evaluation of employees occupationally exposed to

methylene chloride. Mortality. Scandinavian Journal of Work and Environmental Health. Vol. 9 (1983). p. 8-16

(107) Blank

(108) Swaen, G.M.H., et al. Investigation of a cluster of ten cases of Hodgkin's disease in an occupational setting. International Archives of Environmental Health. Vol. 68 (1996). p. 224-228

(109) Wennborg, H., et al. Solvent use and time to pregnancy among female personnel in biomedical laboratories in Sweden. Occupational and Environmental Medicine. Vol. 58 (2001). p. 225-231

Information on chemicals reviewed in the CHEMINFO database is drawn from a number of publicly available sources. A list of general references used to compile CHEMINFO records is available in the database Help.

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©2010 Canadian Centre for Occupational Health & Safety
www.ccohs.ca E-mail: clientservices@ccohs.ca Fax: (905) 572-2206 Phone: (905) 572-2981
Mail: 135 Hunter Street East, Hamilton Ontario L8N 1M5