FOREWORD INTRODUCTION

ACETONE

CAS N°: 67-64-1

SIDS Initial Assessment Report (SIAR) for the 9th SIAM

Place: Paris, France Date: June 29-30, 1999
July 1, 1999

Chemical Name: Acetone

CAS No: 67-64-1

Sponsor Country: USA

National SIDS Contact Point in Sponsor Country:

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HISTORY:

This high production volume (HPV) chemical was assigned to the USA in Phase 4 of the OECD HPV voluntary testing program. A SIDS Dossier was prepared by the Chemical Manufacturer's Association and submitted to the National SIDS Contact Point (USA) on March 14, 1997. The draft SIAR was reviewed on March 7, 1998 at SIAM 7 and the conclusions on the environment were accepted. Modifications to the health part were made in accordance with the comments received from the participants.

COMMENTS:

Deadline for Circulation:

Date of Circulation:

SIDS INITIAL ASSESSMENT PROFILE

CAS No.	67-64-1
CHEMICAL NAME	Acetone
STRUCTURAL FORMULA	CH_3-CO-CH_3

RECOMMENDATIONS

The chemical is currently of low priority for further work

SHORT SUMMARY OF CONCLUSIONS OF INITIAL ASSESSMENT WHICH SUPPORTS THE RECOMMENDATIONS

Summary of hazard assessment

The acute toxicity is low. Acetone is not a skin irritant or sensitiser but is a defatting agent to the skin. Acetone is an eye irritant. The subchronic toxicity of acetone has been examined in mice and rats that were administered acetone in the drinking water and again in rats treated by oral gavage. Acetone-induced increases in relative kidney weight changes were observed in male and female rats used in the oral 13-week study. Acetone treatment caused increases in the relative liver weight in male and female rats that were not associated with histopathologic effects and the effects may have been associated with microsomal enzyme induction. Hematologic effects consistent with macrocytic anemia were also noted in male rats along with hyperpigmentation in the spleen. The most notable findings in the mice were increased liver and decreased spleen weights. Overall, the no-observed-effect-levels in the drinking water study were 1% for male rats (900 mg/kg/d) and male mice (2258 mg/kg/d), 2% for female mice (5945 mg/kg/d), and 5% for female rats (3100 mg/kg/d). For developmental effects, a statistically significant reduction in foetal weight, and a slight, but statistically significant increase in the percent incidence of later resorptions were seen in mice at 15,665 mg/m^3 and in rats at 26,100 mg/m^3. The no-observable-effect level for developmental toxicity was determined to be 5220 mg/m^3 for both rats and mice. Teratogenic effects were not observed in rats and mice tested at 26,110 and 15,665 mg/m^3, respectively. Lifetime dermal carcinogenicity studies in mice treated with up to 0.2 mL of acetone did not reveal any increase in organ tumor incidence relative to untreated control animals.

The scientific literature contains eight different studies that have measured either the neuro-behavioural performance or neurophysiological response of humans exposed to acetone. Effect levels ranging from about 600 to greater than 2375 mg/m^3 have been reported. Neurobehavioral studies with acetone-exposed employees have recently shown that 8-hr exposures in excess of 2375 mg/m^3 were not associated with any dose-related changes in response time, vigilance, or digit span scores. Clinical case studies, controlled human volunteer studies, animal research, and occupational field evaluations all indicate that the NOAEL for this effect is 2375 mg/m^3 or greater.

Acetone has been tested in a wide variety of aquatic and terrestrial species. Acute toxicity to fish

ranges from an LC_{50} of 6,070 mg/L for Brook trout to 15,000 mg/l for Fathead minnow. The lowest LC_{50} for aquatic invertebrates is 2,100 mg/L, ranging to 16,700 mg/L. The NOEC's for toxicity to aquatic plants range from 5,400-7,500 mg/L. The chronic NOEC for Daphnia is 1,660 mg/L. Tests using Ring-neck pheasant and Japanese quail produced no adverse effects at 40,000 mg/kg. In summary, ecotoxicity testing shows that acetone exhibits a low order of toxicity.

An assessment factor of 100 was used to calculate a predicted no effect concentration (PNEC) for acetone in an aqueous environment, because acute toxicity data were available for algae, crustaceans, and fish. The lowest PNEC value for these species was calculated to be 21 mg/L when using the LC_{50} value of 2100 mg/L for marine brine shrimp.

Summary of general exposure information

Worldwide production capacity of acetone was 3.8 million tonnes in 1995 with the actual volume produced being somewhat less at 3.7 million tonnes. Production capacity in the United States constituted about 33% (1.3 million tonnes) of the global capacity, while Western Europe and Asia (including Japan) were about 31% (1.2 million tonnes) and 19% (0.7 million tonnes), respectively. Major end uses of acetone can be divided into three separate categories as: i) a chemical feedstock, ii) a formulating solvent for commercial products, and iii) an industrial process solvent. Acetone can be found in wide variety of consumer and commercial products but only a few are known to contain high concentrations. These include paints and paint-related products, such as paint thinners, finger nail polish removers, automotive waxes and tar removers.

PECs have been derived based on the results from air and water monitoring data. The PEC_{local} (2500 μg/L [water], 10,000 $\mu g/m^3$ [air]) and PEC_{global} (50 μg/L [water], 10 $\mu g/m^3$ [air]) values are intended to represent plausible worst case environment concentrations on a global and regional scale.

High concentrations of acetone can be detected in a variety of occupational environments (up to 2876 mg/m^3 at cellulose acetate factory). The predominant route of both occupational and consumer exposure is through vapor inhalation. The estimated human exposure (EHE) value for workplace employees is 1780 mg/m^3. Using a USEPA modelling programme entitled SCIES (Screening Consumers Inhalation Exposure Software), a scenario intended to represent a likely indoor consumer use of a product (45 min application of a spray contact adhesive that contained 21% acetone) predicted a short-term exposure (EHE) value of 900 mg/m^3 for the consumer use of the product.

IF FURTHER WORK IS RECOMMENDED – INDICATE ITS NATURE

None recommended

FULL SIDS SUMMARY

CAS NO: 67-64-1		SPECIES	PROTOCOL	RESULTS
PHYSICAL-CHEMICAL				
2.1	Freezing. Point			-94.6 °C
2.2	Boiling Point			56.1 °C at 760 mm Hg
2.3	Vapor Pressure			182 mm Hg at 20 °C 400 mm Hg at 39.5 °C
2.4	Partition Coefficient			-0.24 (Log Kow)
2.5	Water Solubility			100% at 20 °C
2.6	Flash Point			Cleveland open cup: -9 °C Tag closed cup: -17 °C
2.7	Flammability			Lower limit : 2.2% (v/v) at 25 °C Upper limit : 13.0% (v/v) at 25 °C
2.8	Autoignition Temperature			465 °C
2.9	Specific Gravity			0.791 at 20 °C
ENVIRONMENTAL FATE/BIODEGRADATION				
3.1.1	Photodegradation		 Calculated Calculated	Undergoes slow photolysis Water: $T_{1/2}$ >43 hr Air : $T_{1/2}$ = 80 hr
3.1.2	Stability in Water		SRC Program	Does not hydrolyze
3.1.3	Stability in Soil		SRC Program	Log K_{OC} = 0.30 (Calculated)
3.2	Monitoring Data			Water (µg/L): residential well water : 2 - 7 sea water : 5 - 53 ground water : 12 - 25 lake water : 1 - 50 storm water runoff : 0 - 100 cloud water : 0 - 17,300 industrial wastewater : 138 - 37,709 landfill leachate : 50 - 62,000 Air (µg/m^3): inside office building : 7.1 - 28.5 inside home : 9.5 - 81 urban street : 2.4 - 306 nonsmoking workplace : 4.7 - 415 inside aircraft cabin : 7.1 - 560 human breath : 230 - 11,285 smoking workplace : 9.5 - 21,085
3.3	Transport/Distribution		Fugacity Level 1 Calculated Measured	Distribution: Air :71.0% Water : 28.6% Soil : 0.0% Partition Coefficients: Octanol/Water : 0.58 Water/Air: 334

3.4	Type of Biodegradation			aerobic anaerobic
3.5	Biodegradation		OECD 301D	Freshwater: BOD_5 : 14% BOD_{15} : 74% BOD_{28} : 74% Seawater : BOD_5 : 38% BOD_{10} : 67% BOD_{15} : 69% BOD_{20} : 76%
3.6	Oxygen Demand			Theoretical (ThOD): 2.20 g O_2/g Chemical (COD) : 2.00 g O_2/g
3.7	Bioconcentration			Atlantic Cod BCF : 0.65
ECOTOXICOLOGY				
4.1	Acute/Prolonged Toxicity to Fish			LC_{50} (mg/L) : Fathead minnow : 15,000 Japanese medaka : 14,300 Mosquito fish : 13,000 Goldfish : >5000 Golden orfe : 9880 Bluegill sunfish : 8300 Rainbow trout : 7400 Brook trout : 6070
4.2	Acute Toxicity to Aquatic Invertebrates			LC_{50} (mg/L) : *Nitocra spinipes* : 16,700 *Daphnia magna* : 15,800 *Daphnia pulex* : 8800 *Daphnia cucullata*: 7635 *Artemia salina* : 2100
4.3	Toxicity to Aquatic Plants			NOEC (mg/L) : *Scenedesmus quadricauda* : 7500 *Selenastrum capricornutum* : 7000 *Chlorella pyrenoidosa* : 3400 *Scenedesmus pannonicus* : 4740 *Lemna gibba*: 5400 *Lemna minor*: 5400
4.4	Toxicity to Bacteria, Diatoms, and Protozoa			NOEC (mg/L) : *Escherichia coli* : 25,000 *Nitzschia linearis* : 11,610 *Skeletonema costatum* : 6000 *Chilomonas paramecium* : 3520 *Uronema parduczi* : 1710 *Pseudomonas putida* : 1700 *Microcystis aeruginosa* : 530 *Entosiphon sulcatum* : 28
4.5.2	Chronic Toxicity to Aquatic Invertebrates			NOEC (mg/L) : *Ceriodaphnia dubia* : 1866 *Daphnia magna* : 1660
4.6.1	Toxicity to Soil Dwelling Organisms		Predicted	NOEC (mg/L) : *Lumbricus terrestris* : >1000
4.6.2	Toxicity to Terrestrial Plants			NOEC (mg/L) : Ryegrass : >80 Radish : >80 Lettuce : >80 Corn : >80

4.6.3	Toxicity to Other Non-Mammalian Terrestrial Species			NOEC (mg/kg) : Japanese quail : >40,000 ring-neck pheasant : >40,000
TOXICOLOGY				
5.1.1	Acute Oral Toxicity	rat mouse rabbit		LD_{50} : 8400 mg/kg LD_{50} : 5250 mg/kg LD_{50} : 5300 mg/kg
5.1.2	Acute Inhal. Toxicity	rat		LC_{50} : 50,000 mg/m^3
5.1.3	Acute Dermal Toxicity	rabbit		LD_{50} : >15,700 mg/kg
5.2.1	Skin Irritation	rabbit		not irritating
5.2.2	Eye Irritation	rabbit	Draize	highly irritating
5.2.3	Respiratory Irritation	mouse	RD_{50}	weakly irritating
5.3	Sensitization	mouse	ear swelling	not sensitizing
5.4	Repeated Dose Toxicity	mice : male mice : female	OECD 408	NOEL : 1% (2258 mg/kg/day) 2% (5945 mg/kg/day)
		rat : male rat : female	OECD 408	NOEL : 1% (900 mg/kg/day) 5% (3100 mg/kg/day)
5.5	Genetic Toxicity In Vitro		OECD 471	bacterial test: reverse mutation : neg. At 10 mg yeast gene mutation : neg. at 5% forward mutation : neg. At 500 mM prophage induction : neg. at 10% non-bacterial test : chromosomal aberration : neg. at 5 mg/mL sister chromatid exchange : neg. at 5 mg/mL cell transformation : neg. at 0.5% alkaline elution : neg. at 1% mouse lymphoma : neg. At 30 mg/mL chromosomal malsegregation : pos. at 6.8%
5.6	Genetic Toxicity In Vivo	rat mouse hamster	OECD 474	embryo cell transformation assay : rat : negative at 0.1% mouse : negative at 0.01% micronucleus assay : negative at 865 mg/kg
5.7	Carcinogenicity	mouse		NOEL : 0.2 mL (dermal)
5.8	Toxicity to Reproduction	rat		NOEL : 0.5% (drinking water)
5.9	Developmental Toxicity/ Teratogenicity	rat mouse	OECD 414	NOEL : teratogenicity : >26,110 mg/m^3 developmental : 5220 mg/m^3
			OECD 414	NOEL : teratogenicity : >15,665 mg/m^3 developmental : 5220 mg/m^3
5.11	Experience with Human Exposure			see SIAR text

1. IDENTITY

Acetone is a clear colorless liquid that is highly flammable and infinitely soluble with water. Reagent grade acetone can contain up to 0.5% water as well as small amounts of other polar solvents. Acetone vapors have a characteristic sweet and fruity odor at low concentrations. The odor threshold for humans has been reported at values ranging from about 24 to 1615 mg/m^3, with 235 to 339 mg/m^3 being the range of odor recognition thresholds for most people and 95 mg/m^3 being the odor detection threshold for unadapted individuals (Devos *et al*., 1990; Leonardos *et al*., 1969).

Virtually every organ and tissue within the human body contains some acetone which is one of three biochemicals collectively referred to as ketone bodies. Acetone is produced within the body as a result of the breakdown and utilization of stored fats and lipids as a source of energy (Wieland, 1968). Consequently, conditions such as strenuous physical exercise and prolonged dieting, which lead to a break-down of fat within the body, may result in higher than average amounts of acetone in the bloodstream (Williamson and Whitelaw, 1978). Measurable amounts of acetone are continuously being excreted in the breath and urine of humans as a result of its high volatility and solubility in water (Wigaeus *et al*., 1981).

2. GENERAL INFORMATION ON EXPOSURE

Worldwide production capacity of acetone was 3.8 million tonnes in 1995 with the actual volume produced being somewhat less at 3.7 million tonnes (Bizzari, 1996). Production capacity in the United States constituted about 33% (1.3 million tonnes) of the global capacity, while the capacity in Western Europe and Asia (including Japan) was about 31% (1.2 million tonnes) and 19% (0.7 million tonnes), respectively. The average annual production of acetone is expected to rise at a global rate of 3.3% until the year 2000.

Major end uses of acetone can be divided into three separate categories. These include use as: i) a chemical feedstock, ii) a formulating solvent for commercial products, and iii) an industrial process solvent. The majority of worldwide production is used as a feed-stock to prepare methyl methacrylate/methacrylic acid and Bisphenol A (Bizzari, 1996). Several aldol chemicals, such as methyl isobutyl ketone, methyl isobutyl carbinol, isophorone, and diacetone alcohol, are also prepared directly from nascent acetone. Acetone has many favorable properties that make it useful as a formulating solvent for a variety of paints, inks, resins, varnishes, lacquers, surface coatings, paint removers, and automotive care products. As an industrial process solvent, acetone is used to manufacture cellulose acetate yarn, polyurethane foam, vitamin C, and smokeless gun powder. At least 75% of the acetone consumed in 1995 was used in captive processes for the preparation of downstream chemicals, while only about 12% was used as a formulating solvent for commercial products.

Large-scale commercial production of acetone is generally accomplished by one of two processes. The first, and by far the most common, is through the acid catalyzed hydro-lytic cleavage of cumene hydroperoxide (Bizzari, 1996). Acetone and phenol are formed as co-products in this reaction at a ratio of 0.61 to 1.00. The second process, catalytic dehydrogenation of isopropyl alcohol, accounted for about 6% of the US production in 1995. Other methods, such as biofermentation, propylene oxidation, and diisopropyl-benzene oxidation, are either experimental in nature or account for a very small percentage of worldwide production.

The release of acetone by chemical manufacturers' and end users accounts for a very small percentage (1%) of the estimated 40 million tonnes that are annually released to the environment (Table 1). Vegetative releases, forest fires, and other natural events account for nearly half (47%) of the estimated annual emissions of acetone, with another 50% resulting from the tropospheric photooxidation of propane and other alkanes and alkenes (Singh *et al*., 1995). Since 1993, US industries have not been required to report their TRI (Toxic Release Inventory) emissions of acetone as required under SARA Title III, Section 313. In 1992, 2548 facilities reported a total environmental release of 60,904 tonnes of acetone with 60,904 tonnes emitted to the air, 454 tonnes to water, 254 tonnes to land, and 1446 tonnes injected underground (USEPA, 1994).

Table 1
Estimated average annual emissions of acetone from different sources

Acetone Source	Global Annual Emissions (tonnes x 10^{-6}) Average	Range
Primary Anthropogenic		
stationary sources	0.5	0.4 - 0.7
mobile sources	0.3	0.2 - 0.3
Primary Biogenic		
Vegetation	9	4 - 18
Secondary Anthropogenic		
propane oxidation	17	15 - 20
isobutane & isopropane oxidation	2	1 - 3
isobutene & isopropene oxidation	1	1 - 2
myrcene oxidation	0.2	0.2 - 0.3
Biomass Burning	10	8 - 12
Total	40	30 - 46

Acetone can be found as an ingredient in a variety of consumer products ranging from cosmetics to processed and unprocessed foods. Acetone has been rated as a GRAS (Generally Recognized as Safe) substance when present in beverages, baked goods, deserts, and preserves at concentrations ranging from 5 to 8 mg/L (Oser and Ford, 1973). It can also be detected in measurable amounts in onions, grapes, cauliflower, tomatoes, milk, cheese, beans, peas, and other natural foods. Milk from dairy cattle may contain very high levels of acetone, ranging as high as 225 mg/L for the milk from hyperketo-nemic cows (Andersson and Lundström, 1984). Acetone has also been identified, but not quantified, in air samples from numerous plants and microorganisms. In addition to its elimination in the expired air of all mammals, acetone is excreted as a metabolic end-product by some bacteria (*Clostridium butylicium*), molds, fungi (*Paecilaomyces variotii*), and algae (*Cryptomonas ovata palustris*) (George *et al*., 1983; Sunnesson *et al*., 1996; Collins and Kalnins, 1966).

Acetone is often detected as an end product of thermal combustion and biological decom-position. Except for tree foliage, the release of acetone from living vegetation has been poorly quantified (Khalil and Rasmussen, 1992). Emissions from poultry manure (530 g/kg), backyard waste incinerators (4.0 g/kg), pine wood combustion (2.8 g/kg), neoprene combustion (990 mg/kg), and wood burning stoves (145 mg/kg) have all been measured and reported (Smith *et al*., 1977; Yocum *et al*., 1956; Hartstein and Forshey, 1974; Lipari *et al*., 1984).

3. ENVIRONMENT

3.1 Environmental Exposure

3.1.1 *General Discussion*

A level I fugacity analysis revealed that acetone preferentially locates in the air compart-ment when released to the environment (Table 2). The fugacity analysis was based on the equilibrium established after the release of 100 moles (5.8 kg) of acetone into the envi-ronment (Mackay and Paterson, 1981). A substantial amount of acetone can also be found in water, which is consistent with the high water to air partition coefficient and its small, but detectable, presence in rain water, sea water, and lake water samples. Very little acetone is expected to reside in soil, biota, or suspended solids. This is entirely consistent with the physical and chemical properties of acetone and with measurements showing a low propensity for soil absorption and a high preference for moving through the soil and into the ground water (Steinberg and Kreamer, 1993).

Table 2
State-state distribution of acetone in the environment

Environmental Compartment	Mass Distribution (%)
Air	71.00
Water	28.58
Sediment	0.01
Soil	0.00
Biota	0.00
suspended solids	0.00

Acetone meets the OECD definition of readily biodegradable which requires that the biological oxygen demand (BOD) is at least 70% of the theoretical oxygen demand (THOD) within the 28-day test period (Table 3). Studies by the standard dilution method have shown greater than 75% of the acetone is biodegraded when using non-acclimated sewage seed from either a freshwater or a sea water sanitary waste treatment plant. These results compare favorably with the values from biodegradability tests performed according to OECD 301D guidelines. Using the OECD method, the BOD_5, BOD_{15}, and BOD_{28} for acetone were found to be 14%, 74%, and 74%, respectively (Waggy *et al*., 1994). The BOD_5 of acetone has been measured by numerous investigators and produced values ranging from about 23% to 83% depending on the test and the type of sewage seed. The THOD of 2.20 g O_2/g of acetone has been found to be only slightly greater than the measured chemical oxygen demand (COD) value of 2.00 g O_2/g of acetone (Price *et al*., 1974).

Studies with several different strains of anaerobic bacteria from municipal waste water treatment plants have shown that acetone is completely degraded to CO_2 following aceto-acetate formation through an initial carboxylation reaction and incorporated into the carbon cycle (Platen and Schink, 1989). Soil bacteria have also been shown to biode-grade acetone to CO_2 (Taylor *et al*., 1980).

Table 3
The biological oxygen demand from acetone in water samples

Sample Type	Biological Oxygen Demand (g)				Author(s) (year)
	5 days	10 days	15 days	20 days	
freshwater	55	79	78	78	Lamb & Jenkins, 1952
freshwater	56	76	83	84	Price *et al*., 1974
saltwater	38	67	69	76	Price *et al*., 1974

Table 4
Comparison of the environmental fate and removal processes for acetone

Acetone Removal Process	Approximate Half-life (days)	Author(s) (year)
aqueous biodegradation	0.6	Rathbun *et al*., 1993
volatilization river	6	Howard *et al*., 1990
soil biodegradation	7	Sanders, 1995
total tropospheric removal	22	Meyrahn *et al.*, 1986
hydroxyl radical reaction	31	Meyrahn *et al.*, 1986
aqueous photolysis	40	Betterton, 1991
atmospheric photolysis	80	Meyrahn *et al.*, 1986
volatilization lake	100	Howard *et al*., 1990

Two processes govern the photochemical removal of acetone from the troposphere: reaction with hydroxyl radicals and photolysis. The two processes occur at about equal rates in clear unpolluted skies yielding a total tropospheric lifetime of about 32 days (Meyrahn *et al*., 1986). The reaction with hydroxyl radicals will predominate over photo-lysis in urban areas where hydroxyl radical concentrations are greater, and during cloudy winter-time conditions where photodecomposition is minimal. Rain out and other forms of wet deposition are considered to be minor tropospheric removal processes (Chatfield *et al*., 1987). Calculated and measured rate constants have been used to estimate the elimination half-life ($t_{½}$= 0.693/k_{calc}) of acetone through various environmental processes (Table 4). These data show that acetone is rapidly biodegraded in water and that this is the dominant removal process in the environment. The slow removal of acetone from the troposphere indicates that it is relatively non-reactive and a minor contributor to urban ozone and peroxyacyl nitrate (PAN) concentrations (Derwent *et al*., 1996).

3.1.2 *Predicted Environmental Concentration*

Measurable amounts of acetone can be found in both mobile and stationary emission sources (Table 5). The levels of acetone in the air from municipal landfills and cigarette smoke can be relatively high, but they are minor contributors to the total global mass. The direct release of acetone from vegetation is an important emission source that is often overlooked. In a qualitative evaluation, acetone was found to be emitted from all 22 of the forest plant species examined (Isidorov *et al*., 1985).

Background levels of acetone in the atmosphere have been assessed from both ground level and airborne monitoring stations located throughout the world. The average acetone concentrations at rural ground level sites are generally lower than the values reported for urban areas (Table 6). The concentration of acetone in urban areas can show large unpre-dictable variations that are likely related to the amount of vehicle traffic and to the emis-sion of precursor alkanes and alkenes (Zweidinger *et al*., 1988; Chatfield *et al*., 1987). Airborne measurements of acetone in the upper troposphere and lower stratosphere reveal an average concentration of 190 to 285 ng/m^3 in these regions (Singh *et al*., 1995).

Table 5
Mobile and stationary emissions of acetone

Emission Source	Airborne Concentration (mg/m^3)	Author(s) (year)
fuel or crude oil fire	0.02 - 0.16	Booher & Janke, 1997
automobile exhaust	0.09 - 4.50	Grimaldi *et al*., 1996
factory fence line	1.9 - 9.7	Hoshika *et al*., 1981
tree foliage	7.8 - 12.6	Khalil & Rasmussen, 1992
municipal landfill	15.7 - 77.1	Brosseau & Heitz, 1994
cigarette smoke	498 - 869	Euler *et al*., 1996

Acetone has routinely been detected in the expired air of humans and in the air samples from many different occupied environments (Table 7). The levels in these samples can vary greatly, ranging from a few µg/m^3 to nearly 25 mg/m^3. Cigarette smoking, emissions from furnishings and construction materials, and excretion by the lung are perhaps the greatest contributors to indoor acetone levels. The acetone levels in indoor air are generally higher than those found outdoors (Jarke *et al*., 1981).

Table 6
Background levels of acetone in urban and rural air samples

Location	Background Concentration (µg/m^3)	Range (µg/m^3)	Author(s) (year)
Smoky Mts, Tennessee	---	1.7 - 9.5	Arnts & Meeks, 1981
Copenhagen, Denmark	---	0.5 - 5.2	Granby *et al*., 1997
Point Barrow, Alaska	2.4	0.7 - 6.9	Cavanagh *et al*., 1969
Waldhof, Germany	3.8	---	Solberg *et al*., 1996
Central Ontario	4.0	---	Shepson *et al*., 1991
Eastern Georgia	4.3	0.0 - 15.9	Lee *et al*., 1995
Los Angeles, California	3.8	0.2 - 15.2	Grosjean *et al*., 1996
Ispra, Italy	4.7	---	Solberg *et al*., 1996
Donan, France	4.7	---	Solberg *et al*., 1996
Athens, Greece	---	1.7 - 18.3	Kalabokas *et al*., 1997
Columbus, Ohio	5.0	0.0 - 21.8	Spicer *et al*., 1996

Southern Germany	6.2	0.5 - 11.4	Slemr *et al*., 1996
Western Colorado	---	2.4 - 8.3	Goldan *et al*., 1995
Western Alabama	10.0	0.7 - 5.2	Goldan *et al.*, 1997
Sao Paulo, Brazil	---	0.5 - 7.4	Grosjean *et al.*, 1990
Rome, Italy	16.1	10.0 - 21.8	Possanzini *et al.*, 1996
Stockholm, Sweden	9.5	1.7 - 24.2	Jonsson *et al.*, 1985
Vancouver, Canada	19.2	8.3 - 30.9	Li *et al.*, 1997
Boston, Massachusetts	32.0	9.7 - 64.0	Kelly *et al*., 1993
Houston, Texas	81.9	29.4 - 223.1	Kelly *et al*., 1993

Table 7
Acetone concentration range in various airborne samples

Sample Type	Airborne Concentration (µg/m^3)	Author(s) (year)
inside office building	7.1 - 28.5	Daisey *et al*., 1994
inside home	9.5 - 81	Lewis & Zweidinger, 1992
urban street	2.4 - 306	Jonsson *et al.*, 1985
nonsmoking workplace	4.7 - 415	Heavner *et al*., 1996
inside aircraft cabin	7.1 - 560	Dechow *et al*., 1997
human breath	230 - 11,285	Crofford *et al.*, 1977
smoking workplace	9.5 - 21,085	Heavner *et al*., 1996

Fugitive stack emissions have been used to estimate fence line concentrations of acetone at three industrial sites. Airborne emissions reported under USEPA SARA Title III section 313 for the year 1989 or 1990 were used in conjunction with the USEPAs ISCST (Industrial Source Complex Short Term) dispersion model to calculate the highest 24-hr concentration and the highest average annual concentration of acetone at property sites beyond the fence line (Table 8). The highest average annual concentration at the three industrial sites ranged from 4.3 to 9.3 mg/m^3. Actual fence line measurements of acetone at five locations outside of the Eastman Chemical Company site in Kingsport, Tennessee showed that the average concentration ranged from 0.05 to 0.50 mg/m^3 which were notably lower than the predicted 24-hr average.

Table 8
Fugitive emissions of acetone and the resulting maximum predicted off-property concentrations

Company & Location	Fence Line Concentration (mg/m^3)	
	24-hr average	annual average
Eastman Chemical Co. Kingsport, TN	0.9	9.3
Hoechst-Celanese Corp. Narrows, WV	2.6	8.3
Hoechst-Celanese Corp. Rock Hill, SC	0.1	4.3

Acetone has been found in surface and ground water samples at concentrations that were highly dependent on the type of sample (Table 9). Ambient background levels of acetone are the result of both natural and commercial releases and are generally reflective of the physical processes affecting absorption from the air, movement through soil, and micro-bial biodegradation. A search of the open literature and the nearly 2000 entries in USEPAs STORET database revealed that acetone levels in natural water and industrial monitoring wells rarely exceeded 1 mg/L.

A USEPA-sponsored survey has determined the acetone concentrations in the discharge from 4000 industrial and publicly owned wastewater treatment plants (Table 10). The highest recorded individual concentration of 37.7 mg/L was found in the discharge from a paint and ink industry facility; whereas, the highest median concentration of 2.5 mg/L was associated with printing and publishing plants (Howard *et al*., 1990). The highest reported aqueous acetone concentration was found in the wastewater from a specialty chemical manufacturing plant. Although wastewater acetone levels of about 200 mg/L were found in water samples from the primary influent at the wastewater treatment plant serving this manufacturing site, the levels in the receiving river water and sediment beyond the treatment plant were below the analytical detection limit (Jungclaus *et al*., 1978). These results are in agreement with data showing that 94% of the acetone removed by a pilot-scaled wastewater facility occurs during secondary treatment (Bhattacharya *et al*., 1996).

Table 9
Acetone concentration range in different water samples

Sample Type	Aqueous Concentration (µg/L)	Author(s) (year)
residential well water	2 - 7	Dewalle & Chain, 1981
sea water	5 - 53	Corwin, 1969
ground water	12 - 25	Sabel & Clark, 1984
lake water	1 - 50	Jungclaus *et al*., 1978
storm water runoff	0 - 100	Line *et al*., 1997
cloud water	0 - 17,300	Aneja, 1993
industrial wastewater	138 - 37,709	Howard *et al*., 1990
landfill leachate	50 - 62,000	Brown & Donnelly, 1988

Predicted environmental concentrations (PECs) of acetone have been derived from the air and water monitoring data described above. The values listed in Table 11 have been taken from the published report that best provide a plausible worst case environmental concentration on both a global and regional scale. The $PEC_{(local)}$ and $PEC_{(global)}$ air concen-trations of 10,000 and 10 µg/m^3 were based on factory fence line concentrations (Table 5; Hoshika *et al*., 1981) and ambient air concentrations for a remote region in the western US (Table 6; Golden *et al*., 1995), respectively. The $PEC_{(local)}$ and $PEC_{(global)}$ water con-centrations of 2500 and 50 µg/L represent the highest median acetone concentration from an industrial wastewater treatment plant (Table 10; Howard *et al*., 1990) and the highest reported natural water concentration of acetone from seawater (Table 9; Corwin, 1969).

3.2 Effects on the Environment

3.2.1 *Aquatic Effects*

As shown in Tables 12 and 13, acetone is minimally toxic to freshwater and marine or-ganisms exposed for 1 to 10 days. Acute NOEC for vertebrate and invertebrate organ-isms were greater than 3500 mg/L and the LC_{50} values were generally greater than 10,000 mg/L. The marine brine shrimp (*Artemia salina*) showed the greatest sensitivity to acetone with a 1-day LC_{50} value of 2100 mg/L.

When examined at a seawater concentration of 1.52%, acetone did not bioconcentrate in the tissues or organs of the Atlantic cod (*Gadus morhua*) (Rustung *et al*., 1931). The 7-day EC_{50} values of greater than 10,000 mg/L and no observable effect levels of 5400 mg/L were similar for two species of aquatic duckweed, *Lemna gibba* and *Lemna minor* (Cowgill *et al*., 1991). The 10-day LC_{50} values for acetone in the 3-brood test with *Daphnia magna* and *Ceriodaphnia dubia* were 4068 mg/L and 6693 mg/L, respectively (Cowgill and Millazo, 1991). The maximum acceptable concentration of acetone that did not affect the survival of *Daphnia magna* exposed for 28 days was approximately 2100 µL/L (1660 mg/L) (LeBlanc and Surprenant, 1983).

Table 10
Acetone concentrations in the discharge water from industrial and public wastewater treatment plants

Industrial Category	Number of Positive Occurrences	Median Acetone Concentration (µg/L)
nonferrous metal	2	6.6
textile mills	4	11.0
inorganic chemicals	8	13.8
porcelain/enameling	4	14.7
pesticide manufacturing	7	52.7
oil and gas extraction	5	59.2
pulp and paper	6	59.8
leather tanning	4	74.7
pharmaceuticals	6	75.4
mechanical products	6	84.4
photographic industries	1	94.9
publicly owned treatment works	40	96.8
organic chemicals	1	113.9
plastics and synthetics	10	164.1
petroleum refining	14	166.9
organics and plastics	24	374.4
explosives	23	388.0
auto and other laundries	2	437.5
electronics	12	441.2
rubber processing	1	604.4
transportation equipment	6	616.7
paint and ink	22	894.9
coal mining	1	2260.8
printing and publishing	7	2501.2

Table 11
Predicted environmental acetone concentrations

Area	Concentration Air (µg/m^3)	Concentration Water (µg/L)
$PEC_{(local)}$	10,000	2500
$PEC_{(global)}$	10	50

3.2.2 *Terrestrial Effects*

The 5-day LC_{50} of acetone for Japanese quail (*Coturnix coturnix japonica*) and ring-neck pheasants (*Phasianus colchicus*) was greater than 40,000 mg/kg (Hill *et al*., 1975). The EPAs ECOSAR program predicted a 14-day earthworm (*Lumbricus terrestris*) LC_{50} value of greater than 1000 mg/L (Meylan and Howard, 1998). Acetone vapors were shown to be relatively toxic to two types insects and their eggs. The time to 50% lethality (LT_{50}) was found to be 51.2 hr and 67.9 hr when the flour beetle (*Tribolium confusum*) and the flour moth (*Ephestia kuehniella*) were exposed to an airborne acetone concentration of 61.5 mg/m^3 (Tunç *et al*., 1997). The LT_{50} values for the eggs were 30-50% lower than for the adult. The direct application of acetone liquid to the body of the insects or surface of the eggs did not, however, cause any mortality.

The effects of acetone on the growth and germination of terrestrial plants and seeds has also been examined (Gorsuch *et al*., 1990). A 168-hr exposure of ryegrass (*Lolium perenne*), radish (*Raphanus sativus*), and lettuce (*Lactuca sativa*) to acetone concen-trations as high as 80 mg/L did not cause any effects. The IC_{50} value obtained when tobacco pollen (*Nicotiana sylvestris*) was incubated with acetone for 18 hr was 20,500 mg/L (Kristen *et al*., 1994). This value, however, conflicts with the 2-hr NOEC of 12 mg/L for the germination of another tobacco plant species, *Nicotiana tabacum* (Schubert *et al*., 1995).

Table 12
Acute and chronic toxicity of acetone to aquatic invertebrates

Species	Duration (hr)	NOEC (mg/L)	LC_{50} (mg/L)	Author(s) (year)
Freshwater Organisms				
Water flea *Daphnia magna*	240	---	4068	Cowgill & Milazzo,
Water flea *Ceriodaphnia dubia*	240	1866	6693	Cowgill & Milazzo,
Water flea *Daphnia magna*	48	8500	15,800	Sloof *et al*., 1983
Water flea *Daphnia pulex*	48	5800	8800	Canton & Adema,
Water flea *Daphnia cucullata*	48	---	7635	Canton & Adema,
Snail *Planorbella trivolvis*	96	≥ 100	---	Ewell *et al*., 1986
Aquatic earthworm *Lumbriculus*	96	≥ 100	---	Ewell *et al*., 1986
Sideswimmer *Gammarus fasciatus*	96	≥ 100	---	Ewell *et al*., 1986

Pillbug *Gaecidotea*	96	≥ 100	---	Ewell *et al*., 1986
Flatworm *Dugesia*	96	≥ 100	---	Ewell *et al*., 1986
Marine Organism				
Harpacticoids *Nitocra spinipes*	96	---	16,700	Lindén *et al.*, 1979
King crab *Lithodes antarcticus*	168	750	---	Lombardo *et al*., 1991
Grass shrimp *Palaemonetes pugio*	288	---	69,400	Rayburn & Fisher,
Brine shrimp *Artemia salina*	24	---	2100	Price *et al*., 1974

Table 13
Acute toxicity of acetone to aquatic vertebrates

Species	Duration (hr)	NOEC (mg/L)	LC_{50} (mg/L)	Author(s) (year)
Freshwater Fish				
Fathead minnow *Pimephales*	48	12,000	15,000	Sloof *et al*., 1983
Fathead minnow *Pimephales*	96	---	9100	Cardwell *et al*., 1974
Japanese medaka *Oryzias latipes*	48	9500	14,300	Sloof *et al*., 1983
Mosquito fish *Gambusia affinis*	96	10,000	13,000	Wallen *et al*., 1957
Goldfish *Carassius auratus*	24	5000	---	Bridié *et al.*, 1979
Brook trout *Salvelinus fontinalis*	96	---	6070	Cardwell *et al*., 1974
Golden Orfe *Leuciscus idus*	48	---	9880	Juhnke & Lüdemann,
Bluegill sunfish *Lepomis*	96	3700	8300	Cairns & Scheier,
Rainbow trout *Salmo gairdneri*	48	5700	7400	Sloof *et al*., 1983
Bleak *Alburnus alburnus*	96	---	11,000	Lindén *et al.*, 1979
Guppy *Poecilia reticulata*	48	6700	9600	Sloof *et al*., 1983
Hydra *Hydra oligactis*	48	11,500	13,500	Sloof *et al*., 1983
Pond snail *Lymnaea stagnalis*	48	3500	7000	Sloof *et al*., 1983
Freshwater Amphibians				
Mexican axolotl *Ambystoma*	48	12,000	20,000	Sloof & Baerselman,
African clawed toad *Xenopus laevis*	48	20,000	24,000	Sloof & Baerselman,
Insects				
Mosquito *Aedes aegypti*	48	3500	15,000	Sloof *et al*., 1983
Mosquito *Culex pipens*	48	8000	17,000	Sloof *et al*., 1983

3.2.3 *Other Effects*

The ability of acetone to inhibit cell multiplication has been examined in a wide variety of microorganisms (Table 14). The results have generally indicated mild to minimal toxicity with NOECs greater than 1700 mg/L for exposures lasting from 6 hr to 4 days. Longer exposure periods of 7 to 8 days with bacteria produced mixed results; but overall the data indicate a low degree of toxicity for acetone. The only exception to these findings were the results obtained with the flagellated protozoa (*Entosiphon sulcatum*) which yielded a 3-day NOEC of 28 mg/L. This was likely a spurious value, however, and the result could not be verified from the tests with other species of protozoa.

The four species of green algae examined in the multiplication inhibition test were relatively insensitive to the effects of acetone treatment. The lowest NOEC of 3400 mg/L was obtained following the 48-hr treatment of *Chlorella pyrenoidosa*. The lowest NOEC for bacteria, in contrast, was found to be 530 mg/L following the 192-hr treatment of *Microcystis aeruginosa*. The IC_{50} values for acetone have also been measured and compared using commercial and natural bacterial test cultures. The IC_{50} value of 48,000 mg/L obtained using the Polytox™ test system was found to compare favorably with the IC_{50} of 48,619 mg/L for an activated sludge test culture (Nirmalakhandan *et al*., 1994). The EC_{50} value for acetone in the Microtox™ test using the bacteria *Photobacterium phosphoreum* was found to be about 14,000 mg/L (Chen and Que Hee, 1995).

Table 14
Acetone toxicity thresholds in the cell multiplication inhibition test

Species	Duration (hr)	NOEC (mg/L)	Author(s) (year)
Flagellated protozoa *Entosiphon sulcatum*	72	28	Bringmann & Kühn,
Bacteria *Microcystis aeruginosa*	192	530	Bringmann & Kühn,
Bacteria *Pseudomonas putida*	16	1700	Bringmann & Kühn,
Ciliated protozoa *Uronema parduczi*	20	1710	Bringmann & Kühn,
Green algae *Chlorella pyrenoidosa*	48	3400	Sloof *et al*., 1983
Flagellated protozoa *Chilomonas*	48	3520	Bringmann & Kühn,
Green algae *Scenedesmus*	48	4740	Sloof *et al*., 1983
Marine diatom *Skeletonema costatum*	120	6000	Cowgill *et al*., 1989
Green algae *Selenastrum*	96	7000	Sloof *et al*., 1983
Green algae *Scenedesmus*	168	7500	Bringmann & Kühn,
Freshwater diatom *Nitzschia linearis*	120	11,610	Patrick *et al*., 1968
Bacteria *Escherichia coli*	1.5	25,000	Reinhartz *et al*., 1987

3.3 Initial Assessment for the Environment

Considering the availability of acute data for algae, crustaceans, and fish an assessment factor of 100 was used to calculate a predicted no effect concentration (PNEC) for acetone in an aqueous environment. Using the LC_{50} value of 2100 mg/L obtained with the marine brine shrimp (*Artemia salina*), the lowest PNEC value for acetone was calculated to be 21 mg/L.

The lowest PNEC was compared to the $PEC_{(local)}$ and $PEC_{(global)}$ values for water (Table 11) to calculate PEC/PNEC ratios. The $PEC_{(global)}$ of 50 µg/L produced a PEC/PNEC ratio of 0.002; whereas, the $PEC_{(local)}$ value of 2500 µg/L yielded a ratio of 0.12. These margins of exposure are each less than one; acetone was therefore judged to have low environmental risk potential.

4. HUMAN HEALTH

4.1 Human Exposure

Virtually every organ and tissue within the human body contains some acetone, which is one of three biochemicals collectively referred to as ketone bodies. Measurable amounts of acetone are continuously being excreted in the breath and urine of humans as a result of its high volatility and solubility in water (Brega *et al*., 1991). The acetone found in the body is produced in the liver following the utilization of stored fats and lipids as a source of energy (Landau and Brunengraber, 1987). The ability of humans to naturally produce and dispose of acetone may to a large degree explain its relatively low toxicity following external exposure to moderate amounts of the vapor or liquid (Wigaeus *et al*., 1981; Haggard *et al*., 1944). The background levels of acetone in blood and urine can vary widely but tend to average 1 to 2 mg/L. The levels in expired alveolar air are, however, about 1000-fold lower at 1 µg/L (Morgott, 1993).

Exogenous exposures to acetone typically occur by the pulmonary route. The high blood- to-air partition coefficient suggests that a large percentage of inhaled acetone will be absorbed into the body; the occurrence, however, of a peculiar wash-in/wash-out effect effectively reduces the uptake to about 50% (Johanson, 1991). The miscibility of acetone in the fluid layers lining the lung appears to be responsible for the wash-in/wash-out phenomenon. Under normal conditions acetone is efficiently and effectively metabolized to a variety of products that are used as building blocks for the synthesis of glucose, amino acids, and other more complex biochemicals (Argilés, 1986). Sustained high blood levels of acetone can result in the induction of enzymes responsible for its own metabolism and the metabolism of other chemicals (Koop and Casazza, 1985; Forkert *et al*., 1994). This compensatory response to high blood levels is responsible for the ability of acetone to potentiate the hepato- and nephrotoxicity of chemicals that undergo metabolic activation by microsomal enzymes to form toxic metabolites.

4.1.1 *Occupational Exposure*

High airborne concentrations of acetone have been found in a variety of occupational environments (Table 15). These levels reflect the high volatility and low intrinsic toxicity which combine to make acetone an attractive industrial process solvent. The predominant route of both occupational and consumer exposure to acetone is through vapor inhalation. Oral and dermal uptake can occur, but the body burden from these exposure routes is relatively small compared to respiratory absorption. Impermeable gloves should be worn together with a supplied air respirator when working with liquid acetone or when the vapor concentration exceeds the occupational exposure limit.

Table 15
Exposure to acetone in various occupations

Factory Type	8-Hour TWA Concentration (mg/m^3)	Author(s) (year)
automotive repair shop	12 - 77	Winder & Turner, 1993
print shop	6 - 235	Nasterlack *et al*., 1994
electronics plant	2 - 648	Hallock *et al*., 1993
fiberglass fabrication	40 - 1580	DeRosa *et al*., 1996
varnish production	5 - 1448	Franco *et al*., 1986
cellulose acetate factory	12 - 2876	Satoh *et al*., 1996

The estimated human exposure (EHE) value for workplace employees has been set at 1780 mg/m^3 based on an examination of the data in Table 15. This exposure value for acetone also agrees well with the occupational exposure limits established in many countries and provides some assurance that it represents a plausible worst case concentration.

4.1.2 C*onsumer Exposure*

Acetone can be found in wide variety of consumer and commercial products but only a few are known to contain high concentrations (Sack *et al*., 1992). These include paints and paint-related products, such as paint thinners, finger nail polish removers, automotive waxes and tar removers (Table 16). Consumer exposures will most likely occur by the inhalation route and will be the greatest for those using adhesives, automotive products, and paint-related products that contain a high percentage of acetone.

Table 16
Average acetone concentration in various consumer product categories

Product Category	Number Products Assayed	Product Prevalence (%)	Average Concentration (%)
oils, greases & lubricants	71	5.3	0.2
cleaners for electronic equipment	111	16.1	0.3
household cleaners & polishers	463	10.8	0.3
miscellaneous products	76	17.2	7.4
fabric & leather treatments	91	14.6	12.9
adhesive-related products	69	24.3	18.8
automotive products	111	22.7	28.1
paint-related products	167	51.5	29.3

Using a USEPA modelling program entitled SCIES (Screening Consumers Inhalation Exposure Software), a 45-min exposure model was created for the application of a spray contact adhesive that contained 21% acetone. This scenario was selected because it depicts a realistic short duration exposure that involves the direct indoor air release of large amounts of acetone. Although consumer products such as nail polish removers can contain 70 to 80% acetone, the resulting air acetone concentrations are generally lower than those described in the following scenario because

of the small volumes of liquid typically applied. The spray contact adhesive scenario describes a plausible worst case consumer application where respirators may not be worn because of the short task duration and relatively low VOC content of the product.

SPRAY CONTACT ADHESIVE SCENARIO

Input Parameters

Use Rate	:	1 event/year
Mass of Product	:	225.0 g
Duration of Use	:	0.66 hr
Zone 1 Volume	:	40.0 m^3
Whole House Volume	:	292.0 m^3
House Air Exchange Rate	:	0.20 room air exchanges/hr
User Inhalation Rate	:	1.20 m^3/hr (during use)
User Inhalation Rate	:	1.10 m^3/hr (after use)
Molecular Weight	:	58.08 g/mole
Vapor Pressure	:	182 torr
Weight Fraction	:	0.210
Starting Time	:	9:00 AM

Output Summary

Evaporation Time	:	0.021 hr
Release Time	:	0.66 hr (duration of exposure)
Duration Following Use	:	8759.34 hr
Interval Between Uses	:	8760.00 hr

User Potential Dose Rate From Inhalation	:	1264.3 mg/yr
Non-User Potential Dose Rate From Inhalation	:	561.6 mg/yr

	Average (mg/m^3)	Peak (mg/m^3)
Concentration in Zone of Release :		
During period of use	556.03	907.19
During period after use	0.18	847.86
Concentration in Zone 2 :		
During period of use	10.90	27.75
During period after use	0.07	82.90

The modelling results shown above indicate average and peak exposures to acetone of 556 and 907 mg/m^3, respectively. The estimated short-term human exposure (EHE) value associated with the use of consumer products was therefore set at the peak exposure concentration of 900 mg/m^3 that was predicted in this scenario.

4.1.3 *Indirect Exposure*

Acetone levels in the body at any point in time are reflective of free fatty acid utilization and acetoacetate production by the liver. Consequently, many normal and abnormal physiological states can appreciably increase the body burden of acetone through the process of ketogenesis. Children and adolescents typically have higher acetone blood levels than adults due to their higher energy

expenditure. In fact, 2 to 5 day old infants have been found to have acetone blood levels ranging as high as 140 mg/L (Peden, 1964). Furthermore, vigorous exercise and the resulting utilization of fatty acids as a fuel source can lead to a condition commonly called post-exercise ketosis that results in a dramatic increase in blood ketone body concentrations. In addition to these normal physiological conditions, there are a number of clinical states that can result in human ketosis. In each of these conditions, the ketosis can be traced to the increased mobilization and utilization of free fatty acids by the liver. These conditions include pregnancy, fasting, prolonged vomiting, and alcoholism (Morgott, 1993).

Other clinical conditions, such as diabetic ketoacidosis and starvation, can lead to much larger increases in blood acetone levels (Table 17). In each of these situations, the elevations in blood acetone are typically accompanied by even larger increases in the remaining two ketone bodies, acetoacetate and β-hydroxybutyrate (Sulway *et al.*, 1971). Unlike acetone, however, these two ketone bodies disrupt normal acid-base balance and cause many of the acute symptoms of diabetes due to their ionization (Winek, 1976). Acetone, in contrast, is non-ionic and is produced together with carbonic acid during the breakdown of acetoacetate (Koorevaar and Van Stekelenburg, 1976). Because acetone has a normal physiological role in the body, the estimated short-term human exposure (EHE) value for endogenous acetone was set at 10 mg/L, which represents the upper limit for blood acetone in healthy individuals.

Table 17
Human plasma acetone concentrations expected under various exposure and health conditions

Physiological State or Condition	Plasma Concentration Range		
	(mg/L)	(mg %)	(mM)
healthy	< 10	< 1.0	< 0.17
occupational exposure	< 100	< 10.0	< 1.72
diabetic ketoacidosis	100 - 700	10.0 - 70.0	1.72 - 12.04
toxic exposure	> 200	> 20.0	> 3.44

4.2 Effects on Human Health

About twenty separate instances of human acetone poisoning have been reported in the medical literature. Many of these case reports have involved patients seen in hospital emergency wards following either accidental or intentional ingestion of acetone. The case reports provide a clear picture of the signs, symptoms, and prognosis that accompany acute acetone intoxication. The most noticeable features of high exposures to acetone vapor are irritation to the eyes, nose, and throat. If the exposure is extremely large, as in cases of accidental ingestion of liquid acetone, fatigue, irritability, dizziness, and breathing irregularities may occur. When the poisoning is severe, these symptoms may precede the development of gastrointestinal disturbances and a temporary loss of consciousness. While many reports of severe acetone poisoning have been reported in the literature, no deaths have ever been recorded.

The following three methods have been used to study the sensory irritation potential of acetone for the eyes, nose, and throat: physiological techniques, psychophysical methods, and subjective ques-

tionnaires. It is important to understand the differences between sensory irritation and both sensitization and chemical irritation. Sensory irritation, known also as the "common chemical sense" or chemesthesis, occurs when a vapor or gas interacts with trigeminal nerve receptors in the ocular or nasal mucosa. Sensory irritation often occurs as a physical sensation that is described using a variety of terms including: pungency, piquancy, stinging, burning, and tickling. Sensitization, in contrast, is an allergic reaction that is manifested through a either a cell-mediated (dermal sensitization) or a humoral response (pulmonary sensitization) by the immune system. Chemical or primary irritation denotes an inflammatory reaction with localized redness and swelling. This type of irritation is found when a chemical solid or liquid makes direct contact with the skin or eyes. Sensory irritation is a generally milder effect than either sensitization or chemical irritation.

The studies listed in Table 18 were conducted both in the workplace using acetone-exposed employees and in the laboratory using naive volunteers exposed to acetone in an inhalation chamber. The studies using objective physiological and psychophysical tech-niques showed acetone to be an extremely weak sensory irritant. Subjective symptom questionnaires, in contrast, indicated that acetone was a sensory irritant at much lower vapor concentrations. Recent research indicates that the irritancy responses observed using subjective symptom questionnaires are likely caused by the odor of acetone (Dalton *et al*., 1997). Investigators have shown that both acetone and phenyl ethyl alcohol, a known non-irritant with a strong odor, produced subjective irritancy responses in humans following a 20-min inhalation exposure at 1900 mg/m^3. Objective psychophysical methods, in contrast, showed little if any irritancy effect in humans exposed under the same conditions.

The scientific literature contains eight different studies that have measured either the neurobehavioral performance or neurophysiological response of humans exposed to acetone. Many of the early neurotoxicity studies with acetone were not amenable to reliable statistical analysis because of the variability in the data and the inability to reproduce the results. A close inspection of these early investigations also reveals many problems with design, conduct, or interpretation that hinder their use.

Among more recent studies with acetone, NOAELs ranging from vapor concentrations of 600 mg/m^3 to greater than 2375 mg/m^3 have been reported. The wide range in effect levels are likely due to statistical errors caused by large numbers of independent variables, analytical problems, and the failure to use multiple concentrations to evaluate dose-response characteristics. Neurobehavioral studies with acetone-exposed employees have recently shown that 8-hr exposures up to 2375 mg/m^3 were not associated with any dose-related changes in reaction time, vigilance, or digit span scores (Satoh *et al*., 1996). When the test subjects were divided into three age groups, a statistically significant decrease in simple reaction time and digit span scores was observed in one of the groups 30 to 44 years of age, but not in the older or younger age groups.

Table 18
Reported cases of human sensory irritation from acetone vapors

Test Method	Type of Subjects	No Effect Level (mg/m^3)	Author(s) (year)
Subjective			
questionnaire	naive	475	Nelson *et al*., 1943
questionnaire	workers	< 595	Satoh *et al*., 1996
questionnaire	naive	595	Matsushita *et al*., 1969

questionnaire	naive	1185	DiVincenzo *et al*., 1973
questionnaire	workers	1900	Raleigh & McGee, 1972
questionnaire	both	2375	Seeber *et al*., 1992
questionnaire	naive	2850	Stewart *et al*., 1975
questionnaire	workers	3560	Oglesby *et al*., 1949
Objective			
acoustic	naive	7120	Roberts *et al*., 1996
spirometry	naive	18,985	Douglas, 1974)
psychophysics	naive	> 23,730	Cometto-Muñiz *et al*.,
psychophysics	naive	> 23,730	Cometto-Muñiz *et al*.,
laterialization	workers	> 35,600	Wysocki *et al*., 1997
laterialization	naive	> 83,070	Wysocki *et al*., 1997

4.2.1 *Acute Toxicity*

The acute effects of a single exposure to acetone vapor have been examined in mice, rats, guinea pigs, and cats. The adverse effects observed in laboratory animals are generally similar to the signs of central nervous system depression seen in cases of human intoxication. Vapor concentrations in excess of 24,000 mg/m^3 are generally required to elicit any sign of acute acetone intoxication in laboratory animals. Animal studies have demonstrated that the acute narcotic effects of acetone are strongly dependent upon both the length and magnitude of the exposure (Flury and Wirth, 1934; Haggard *et al*., 1944; Kagen, 1924; Specht *et al*., 1939). Regardless of the species examined, the narcotic effects of acetone tend to proceed through several distinct phases that can be described as follows: drowsiness, lack of coordination, loss of autonomic reflexes, narcosis, respira-tory failure, and death.

The hallmark of animal studies with acetone is the extremely high vapor concentrations or long exposure duration needed to produce an adverse effect. An 8-hr inhalation LC_{50} value of 50,100 mg/m^3 was reported for female rats (Pozzani *et al*., 1959). Single-dose oral lethality studies have also been performed in rats, mice, and rabbits. The oral LD_{50} was found to be 10.7 mL/kg (8.5 g/kg) in rats, 90.4 mmol/kg (5.25 g/kg) in mice, and greater than 5.3 g/kg in rabbits (Smyth *et al*., 1962; Tanii *et al*., 1986; Krasavage *et al*., 1982). An examination of the oral LD_{50} values for male and female rats from different age groups reveals that acetone is more acutely toxic for newborn rats than for adults (Table 19). The LD_{50} values for rats aged 14 days and older were not, however, substantially different (Kimura *et al*., 1971).

Table 19
Acute lethality of acetone to Sprague-Dawley rats from different age groups

Age Group	Weight Range (g)	LD_{50} (g/kg)	95% Confidence Limits (g/kg)
newborn (24-48 hr)	5 - 8	2.8	2.1 - 4.8
immature (14 day)	16 - 50	7.1	4.9 - 10.1
young adult	80 - 160	11.5	8.6 - 15.3
old adult	300 - 470	10.7	9.8 - 11.8

The ability of acetone to dehydrate and delipidate unprotected skin is well known from industrial and laboratory experience. Laboratory animal studies have confirmed this ob-servation and also shown a low potential for systemic toxicity following exposure by the dermal route. The 24-hr dermal LD_{50} was found to be greater than 20 mL/kg (15.7 g/kg) in rabbits (Smyth *et al*., 1969).

4.2.2 *Irritation/Sensitization*

Acetone did not cause contact hypersensitization in the mouse ear swelling test or the guinea pig maximization test (Descotes, 1988; Nakamura *et al*., 1994). The sensory irritation potential for acetone vapors was determined by measuring the concentration-related decline in the respiration rate of mice. The RD_{50} values for acetone were found to be 183,970 mg/m^3 and 55,725 mg/m^3 in two separate studies (Kane *et al*., 1980; De Ceaurriz *et al*., 1981).

Studies conducted in rabbits have generally shown that acetone can be a severe eye irritant when applied undiluted and left in contact with the cornea. Dilute aqueous solutions, however, are minimally irritating. Corneal thickness measurements three days after the treatment of rabbits with 0.1 mL of undiluted acetone produced severe eye irritation (Morgan *et al*., 1987). An acetone concentration of 3.9 M (225 g/L) was found to cause a 50% increase in ocular edema after a 1-hr exposure. Acetone treatment for up to several minutes was shown to destroy the corneal epithelium, but not the corneal stroma. All injury to the corneal epithelium was reversible within 4 to 6 days. Acetone was not found to be a corrosive eye irritant (Märtins *et al*., 1992).

4.2.3 *Repeated Dose Toxicity*

The subchronic toxicity of acetone has been examined in rats following oral gavage and drinking water consumption. In the gavage study, acetone was administered in water to male and female rats for 90 consecutive days at dose levels of 100, 500, and 2500 mg/kg (Mayhew and Morrow, 1988). The rats showed an increase in several hematological parameters and an increase in the serum activity of three enzymes. Increases in the absolute liver and kidney weight were observed for female rats at the two highest dose levels. Increases in organ-to-body weight ratios were also observed, but only at the highest dose level tested. Male rats administered 2500 mg/kg showed an increase in organ-to-body weight ratios for the liver and kidney, but the absolute weights of the organs were unaffected. No liver pathology was observed, however some histopatho-logical abnormalities were observed in the renal tubular cells of male and female rats treated at the high dose.

In a more relevant study, acetone was administered in the drinking water of mice and rats for either 14 days or 13 weeks. The drinking water concentrations and calculated average daily doses of acetone are presented in Table 20 (Dietz *et al*., 1991). No mouse or rat mortality was observed in either the 14-day or the 13-week study. Overt clinical signs of toxicity were only observed in the rats treated at the 10% level in the 14-day study. Acetone-induced increases in relative kidney weight were observed in the male and female rats treated for 13 weeks. The kidney weight changes were reportedly associated with a nephropathy that occurred spontaneously in untreated control rats. The increases in the relative liver weight of male and female rats were not associated with histopatho-logic changes and may have been caused by microsomal enzyme induction. Hematologic effects consistent with macrocytic anemia were noted in male rats along with hyperpig-mentation in the spleen. The most notable findings in mice were increased liver and decreased spleen weights, which were confined exclusively to female mice administered a 5% concentration of acetone (Dietz, 1991). The authors concluded that the no-observed-effect-level was 1% for male rats and male mice, 2% for female mice, and 5% for female rats.

Table 20
Time-weighted-average dose for male and female Fisher 344 rats and B6C3/F_1 mice exposed to acetone in their drinking water

Water Concentration (%)	14-Day Average Dose (mg/kg/day)				13-Week Average Dose (mg/kg/day)			
	Rats		Mice		Rats		Mice	
	male	female	male	female	male	female	male	female
0.125	-	-	-	-	-	-	380	-
0.25	-	-	-	-	200	200	611	892
0.5	714	751	965	1569	400	600	1353	2007
1.0	1616	1485	1579	3023	900	1200	2258	4156
2.0	2559	2328	3896	5481	1700	1600	4858	5945
5.0	4312	4350	6348	8804	3400	3100	-	11,298
10.0	6942	8560	10,314	12,725	-	-	-	-

4.2.4 *Reproduction/Devlopmental Toxicity*

Acetone showed minimal reproductive and developmental effects in animals exposed either by inhalation or via drinking water. No reproductive performance changes or testicular histopathological effects were noted in male rats treated with 0.5% acetone in their drinking water for 6 weeks (Larsen *et al*., 1991). In another study, however, an acetone drinking water concentration of 5% caused a mild decrease in testicular weight, a moderate increase in the incidence of abnormal sperm, and depressed sperm motility after 13 weeks of treatment (Dietz *et al*., 1991). These findings indicate that high concentrations of acetone can have a mild effect on rat spermatogenesis.

The potential for acetone vapors to cause developmental effects was examined in virgin and pregnant rats and mice (Mast *et al*., 1988). Mated rats were exposed by inhalation to 1045, 5220, or 26110 mg/m^3 of acetone on days 6 through 19 of gestation. Mice were ex-posed at concentrations of 1045, 5220, or 15665 mg/m^3 of acetone on days 6 through 17 of gestation. No effects were seen in the mean liver or kidney weights of pregnant dams, the organ-to-body weight ratios, the number of implantations, the mean percentage of live pups per litter, the mean percentage of resorptions per litter, or the fetal sex ratio. No treatment-related effects were seen in maternal or virgin body weight, or the maternal uterine weight of the treated mice. A treatment-related increase was observed in the liver-to-body weight ratios for pregnant dams. A statistically significant reduction in fetal weight, and a slight, but statistically significant increase in the incidence of late resorptions was also seen in mice exposed to 15,665 mg/m^3 of acetone. The incidence of fetal malformations in mice was not altered by gestational exposure to acetone at any exposure concentration. The no-observed-effect level for developmental toxicity was found to be 5220 mg/m^3 for both rats and mice. Acetone did not produce any teratogenic effects at any of the exposure concentrations tested. The no-observed-effect level for teratogenicity was, therefore, greater than or equal to 15,665 mg/m^3 for mice and 26,110 mg/m^3 for rats.

4.2.5 *Neurotoxicity*

Mild neurobehavioral changes have been observed in rats repeatedly exposed to high vapor concentrations of acetone. Female rats were exposed 4 hr/day for 2 weeks at acetone concentrations of 7120, 14240, 28480, and 37975 mg/m^3 were examined for their response to avoidance and escape stimuli before and after each exposure (Goldberg *et al*., 1964). Repeated daily exposures to

14,240 mg/m^3 of acetone produced an inhibition of avoidance behavior but did not produce any signs of motor imbalance. Acetone concentrations of 28,480 and 37,975 mg/m^3 produced ataxia in several animals after a single exposure, however, a rapid tolerance developed and ataxia was not seen on subsequent days. In a recent schedule controlled operant performance study, acetone did not cause any permanent effects in rats exposed to the vapor for 13 weeks at 2375, 4750, and 9495 mg/m^3 (Christoph and Stadler, 1997).

4.2.6 *Carcinogenicity*

Information on the carcinogenicity of acetone is available from dermal studies performed in mice. In each of these studies, acetone was used as the vehicle to evaluate the effects of a test chemical. The test design therefore included untreated and vehicle-treated study groups. The carcinogenicity of acetone was evaluated in a group of 29 female ICR/Ha Swiss mice treated topically with 0.1 mL of acetone or 0.1 mL of an acetone-water mixture (9:1) three times per week for up to 424 days (Van Duuren *et al*., 1978). Histopathological analysis of all major organs revealed a total of 14 lung tumors, one liver tumor, one forestomach tumor, and no skin tumors in the acetone and acetone/water treatment groups. Lung papillary tumors were seen in 37% of the untreated mice and 24% of the acetone or acetone-water treated mice. The incidence of forestomach tumors in acetone or acetone-water treated mice was comparable to untreated mice. Except for one undifferentiated malignant liver tumor, which was not cited as a remarkable finding, the incidence of systemic tumors in the acetone and acetone-water treated mice was not different from the background incidence in untreated mice. In another study, the application of 0.2 mL of acetone to the shaved dorsal skin of male and female CF1 mice once per week for two years had no effect on the survival of the 300 animals tested (Zakova *et al*., 1985). Dermal inflammatory reactions (focal acanthosis, dermal fibrosis) were seen in 6% of the animals and a fibrosarcoma was seen in one male mouse. An historical analysis of the organ pathology observed in two previous dermal carcinogenicity studies showed no evidence of a treatment-related increase in tumors or organ lesions from acetone (Ward *et al*., 1986). Sixty female SENCAR mice received 0.2 mL of acetone once or twice per week for up to 92 weeks. The major organs and tissues from all of the animals were examined both macroscopically and microscopically following necropsy. Fifty percent of the animals survived past 96 weeks of age with 15 of the mice dying due to neoplastic lesions and 27 due to non-neoplastic lesions.

4.2.7 *Genotoxicity*

Acetone has been repeatedly tested in a variety of prokaryotic and eukaryotic test systems without causing genotoxic effects. Studies in the *Salmonella* assay have shown acetone to be non-mutagenic and to be an acceptable vehicle for dissolving and delivering water- insoluble chemicals to the tester strains (Anderson and MacGregor, 1980). EPA-spon-sored studies have shown acetone to be negative in *Salmonella* strains TA97, TA98, TA100, and TA1535 at levels up to 1 mg/plate (NTP, 1987). Subsequent studies then found that acetone was negative in strains TA92, TA94, TA98, TA100, TA1535, and TA1537 at a concentration of 10 mg/plate (Ishidate *et al*., 1984). Acetone was not geno-toxic to *Schizosaccharomyces pombe* either with or without metabolic activation (Abbondandolo *et al*., 1980). Acetone induced aneuploidy, but not mitotic recombination or point mutations, in *Saccharomyces cerevisiae* when tested at concentrations greater than 40 mg/mL using a cold-interruption procedure (Zimmermann *et al*., 1985). These effects were not observed, however, when *Saccharomyces cerevisiae* was tested according to the standard overnight incubation procedure (Albertini, 1991).

Acetone did not produce genotoxic effects in an embryo cell transformation assay per-formed in rats and mice, and was also negative in a micronucleus assay using hamsters (Rhim *et al*., 1974;

Basler, 1986). Acetone did not cause chromosomal aberrations or sister chromatid exchanges in Chinese hamster ovary cells treated at concentrations up to 5 mg/mL (Loveday *et al*., 1990). Acetone concentrations ranging from 10.5 to 20.9 mM (0.6 to 1.2 mg/mL) also did not cause chromosomal aberrations or sister chromatid exchanges in cultured human lymphocytes (Norppa, 1981). Acetone did not cause point mutations at the thymidine kinase locus in L5178Y mouse lymphoma cells treated at a level of 10 mg/mL (Amacher *et al*., 1980).

4.2.8 *Epidemiology*

An epidemiological evaluation of mortality and clinical laboratory data for 948 employees in a fiber production plant exposed to 8-hr average acetone concentrations of 900, 1830, and 2540 mg/m^3 over 23 years produced no unusual findings (Table 21). The liver enzymes, clinical chemistry values, and hematological parameters were all within normal range (Ott *et al*., 1983a,b,c). Standard mortality ratios for death from all causes, cardio-vascular disease, and malignant neoplasms were below expectations by 55%, 61%, and 43%, respectively.

Table 21
Observed and expected mortality rates for men and women occupationally exposed to acetone

Cause of Death	Male Mortality Ratio		Female Mortality Ratio	
	observed	expected	observed	expected
all causes	24	53.8	3	6.7
malignant neoplasm	5	10.0	2	2.3
cardiovascular disease	15	40.4	2	2.8

Four health surveillance studies have been conducted on acetone-exposed employees from cellulose acetate facilities located worldwide. The studies did not reveal any evidence of systemic toxicity or dose-related adverse heath effects based on the results obtained from a wide variety of biochemical and hematological tests (Table 22).

Table 22
Occupational health surveys with acetone exposed workers

Factory Location	Number Examined	Employed (years)	Exposure (mg/m^3)	Clinical Measurements	Author (year)
United	800	Unknown	1425 - 5100	hematology & urinalysis	Oglesby *et al*.,
United	948	< 23	900 - 2540	Hematology, urinalysis, & mortality	Ott *et al*., 1983
Italy	60	> 5	1305 - 2490	Hematology, urinalysis, & clinical chemistry	Grampella *et al*., 1987
Japan	110	15	48 - 2415	Hematology, immunology,	Satoh *et al.*, 1996

4.3 Initial Assessment for Human Health

The inhalation EHE values for occupational and consumer groups have been set at 1780 and 900 mg/m^3, respectively. The most critical effect of acetone inhalation for both industrial and consumer contact is central nervous system depression. This endpoint was selected over the more commonly reported sensory irritation effects based on the findings from a recently completed comprehensive review of the odor and irritancy potential of acetone (Arts *et al*., 1998). The authors of this review concluded that subjective reports of acetone's irritancy were unreliable and likely related to its distinctive odor. Furthermore, the authors determined that the true irritancy threshold for acetone vapors was very high, ranging somewhere between 23,730 and 94,930 mg/m^3. Clinical case studies, controlled human volunteer studies, animal research, and occupational field evaluations all indicate that the NOAEL for the CNS-related effects of acetone is about 2375 mg/m^3. Acetone is therefore considered to have a low potential for neurological risk to humans.

In a subchronic drinking water study, renal toxicity and increased liver and decreased spleen weights were observed. The reported NOAEL's were 900mg/kg/d and 3,100 mg/kg/d for male and female rats, and 2,258 mg/kg/d and 5,945 mg/kg/d for male and female mice. Worst-case EHE's on a body weight basis for occupational and consumer exposures are 254 mg/kg/d and 16 mg/kg/d, respectively. Developmental toxicity and teratogenicity of acetone were measured in rats and mice. For developmental endpoints the NOAEL in rats and mice is 5,220 mg/m^3, while no teratogenic effects were observed at the highest doses tested of 26,111 mg/ m^3 in rats and 15,665 mg/m^3 in mice. Acetone is therefore considered to have a low potential for renal damage and developmental effects in humans.

The unconsciousness, respiratory distress, and vomiting associated with cases of accidental or intentional exposure to acetone appear to occur when the blood levels are in excess of 1000 mg/L. Likewise, the drowsiness observed in patients with uncontrolled diabetes mellitus has been associated with acetone blood levels in excess of 150 mg/L. By com-parison, an 8-hr occupational exposure to 1780 mg/m^3 of acetone is expected to result in an acetone blood level of about 60 mg/L. This shows that the blood levels associated with occupational exposures to acetone are below those causing central nervous system depression.

5. CONCLUSIONS AND RECOMMENDATIONS

5.1 Conclusions

An examination of all available information on the biological activity of acetone indicates that the vapors are mildly toxic after both direct contact or systemic absorption. The primary effect of acute high-level exposure appears to be central nervous system depres-sion. Comparative studies with other solvents have shown that the irritative properties of acetone vapor are extremely mild and are often confused with its odor. Although many cases of accidental or intentional human acetone poisoning have occurred, no instances of death or permanent injury have been recorded. Appreciable quantities of acetone are continually being produced and eliminated in the body as a result of energy needs. Normal background levels in the blood can, therefore, dramatically fluctuate depending upon age, eating habits, and level of physical fitness.

The data indicate that acetone does not appear to pose a neurotoxic, carcinogenic, or reproductive health hazard at the concentrations reported to be found in the environment. Information obtained from occupationally exposed individuals, animal feeding studies, and *in vitro* screening assays support this conclusion. The kidney appeared to be the most sensitive target tissue in the animal studies. Acetone has also been tested in a wide variety of aquatic and terrestrial organisms and

produced minimal to mild effects in every instance. The mild effects have allowed acetone to be used as a carrier solvent for dissolving and testing less soluble chemicals. The preceding analysis shows that acetone has a low potential for harming both human health and the environment.

5.2 Recommendations

Acetone has a low priority for further work. The health and environmental effects of acetone have both been well studied.

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H E D S E T

D a t a S h e e t

CAS-No.:	67-64-1
EINECS-No.:	200-662-2
IUPAC-Name:	Acetone

1.03 Submitter Identification

Company	Environmental Protection Agency
Street	401 M Street, SW
Date	02/20/97
Postal Code	20460
Town	Washington, DC
Country	United States
Phone	202-260-3749
Telefax	202-260-8168
Telex	N/A

1.04 OECD and Company Information

Type	lead organization
Name	Environmental Protection Agency
Partner	Chemical Manufacturers Association
Date	02/20/97
Street	401 M Street, SW
Postal Code	20460
Town	Washington DC
Country	United States
Phone	202-260-3749
Telefax	202-260-8168
Telex	N/A
Other Manufacturer	no

1.1 Substance Information

Molecular Formula:	C3H6O
Molecular Weight:	58.08
Smiles Code:	CC(=O)C
Substance Type	organic
Physical Status	liquid
Purity	99.5-99.8% (w/w)

1.2 Synonyms

Remark	2-Propanone Beta-Ketopropane Acetone

Dimethyl Ketone
Methyl Ketone
Propanone
Ketone Propane
Ketone, Dimethyl

1.3 Impurities

Remark	Water, not more than 0.5 wt % (ASTM D1364); acidity (as free acetic acid), not more than 0.002 wt %, equivalent to 0.019 mg of KOH per gram of sample (ASTM D1613); water miscibility, no turbidity or cloudiness at 1:10 dilution with water (ASTM D1722); alkalinity (as ammonia), not more than 0.001 wt % (ASTM D1614); and permangamate time, color of added $KMnO_4$ must be retained at least 30 min at 25 C in the dark (ASTM D1363).
Remark	Other impurities that have been identified include: benzene (0-50 ppm), acetaldehyde (0-70 ppm), methanol (0-500 ppm), diacetone alcohol (0-300 ppm), mesityl oxide (0-10 ppm), formaldehyde (0-1 ppm), isopropanol (0-100 ppm).
Reference	Kirk-Othmer. 1991. Encyclopedia of Chemical Technology, Fourth Edition. Volume 1. John Wiley & Sons. New York.
	Gerlich, O. (1995). Euclid data sheet: Acetone. Existing Substance Dossier. Phenolchemie GmbH. Gladbeck, Germany.

1.5 Quantity

Quantity Produced or Imported	>1,000,000 tons (1993)
Produced 12 mo After Regulation	yes
Imported 12 mo After Regulation	yes
Remark	11 Producers in United States, global production.
Information Source	Chemical Manufacturers Association

1.6.1 Labelling

Labelling	As in Directive 67/548/EEC
Specific Limits	no
Symbols F	Nota
R Phrases	11
S Phrases	9-16-23-33
Text	Keep container in a well-ventilated place--Keep away from sources of ignition--No smoking--Do not breathe vapors--Take precautionary measures against static discharges. Separate the phrases with '-' and the text for S-phrases with '--'.

1.6.2 Classification

Classification	as in Directive 67/548/EEC
Class of Danger	highly flammable
R Phrases	11

1.7 Use Pattern

Type	industrial
Category	chemical industry: used in synthesis
Remark	bisphenol-A, isophorone, methyl isobutyl ketone, other chemical intermediates

Type	industrial
Category	basic industry: basic chemicals
Remark	major use as solvent for fats, oils, waxes, resins, plastics, lacquers, paints, inks, varnishes, rubber cements

Type	industrial
Category	chemical industry: used in synthesis
Remark	methyl methacrylate, methacrylic acid and higher methacrylates (33%)

Type	industrial
Category	process solvent: used in manufacturing
Remark	smokeless gunpowder, cellulose acetate yarn, vitamin intermediates

Type	industrial
Category	other
Remark	antiseptic solution, cleaning and drying agent, pharmaceutical aid

1.8 Occupational Exposure Limit Values

Type of Limit	8-h TWA PEL (OSHA)
Value	2400 mg/m^3 (1000 ppm)
Country	United States
Reference	Code of Federal Regulations 41:50-204.50, 1994.

Type of Limit	8-h TWA
Value	1185 mg/m^3 (500 ppm)
Country	Australia
Remark	Short-Term Exposure Limit 2400 mg/m^3 (1000 ppm)

Type of Limit	8-h MAK (DE)
Value	1200 mg/m^3 (500 ppm)
Country	Austria, Germany, Switzerland (DFG-MAK/DFG-Peak)
Remark	Short-Term Exposure Limit 6000 mg/m^3 (2500 ppm)

Type of Limit	8-h TWA TLV
Value	1780 mg/m^3 (750 ppm)

Country	Belgium, Luxembourg: ARAB-TWA/ARAB-STEL Ireland, Italy: ACGIH-TWA/ACGIH-STEL Portugal, Spain: ACGIH-TWA/ACGIH-STEL
Remark	Short-Term Exposure Limit 2400 mg/m^3 (1000 ppm)
Type of Limit	8-h TWA OEL
Value	800 mg/m^3 (330 ppm)
Country	Czechoslovakia
Remark	Short-Term Exposure Limit 4000 mg/m^3 (1660 ppm)
Type of Limit	8-h TWA (AGSM)
Value	600 mg/m^3 (250 ppm)
Country	Denmark
Type of Limit	8-h TWA
Value	200 mg/m^3 (84 ppm)
Country	China
Type of Limit	8-h TWA OEL
Value	1200 mg/m^3 (500 ppm)
Country	Finland
Remark	Short Term Exposure Limit 1500 mg/m^3 (625 ppm)
Type of Limit	8-h TWA OEL
Value	1800 mg/m^3 (750 ppm)
Country	France
Type of Limit	8-h TWA OEL
Value	600 mg/m^3 (250 ppm)
Country	Hungary
Remark	Short Term Exposure Limit 1200 mg/m^3 (500 ppm)
Type of Limit	8-h TWA OEL
Value	1780 mg/m^3 (750 ppm)
Country	India
Remark	Short Term Exposure Limit 2375 mg/m^3 (1000 ppm)
Type of Limit	MAC (Japan)
Value	470 mg/m^3 (200 ppm)
Country	Japan
Type of Limit	MAC (NL) 8-h TWA
Value	1780 mg/m^3 (750 ppm)
Country	The Netherlands
Type of Limit	8-h TWA OEL
Value	2400 mg/m^3 (1000 ppm)
Country	The Philippines
Type of Limit	8-h TWA OEL
Value	200 mg/m^3 (84 ppm)

Country	Poland
Type of Limit	8-h TWA
Value	200 mg/m^3 (84 ppm)
Country	Russia
Remark	Short Term Exposure Limit 200 mg/m^3 (84 ppm)
Type of Limit	8-h TWA OEL
Value	600 mg/m^3 (250 ppm)
Country	Sweden
Remark	Short Term Exposure Limit 1200 mg/m^3 (500 ppm)
Type of Limit	8-h TWA OEL
Value	2400 mg/m^3 (1000 ppm)
Country	Turkey
Type of Limit	8-h TWA (EH40)
Value	1780 mg/m^3 (750 ppm)
Country	United Kingdom
Remark	Short Term Exposure Limit 3560 mg/m^3 (1500 ppm)
Type of Limit	8-h TLV TWA (ACGIH)
Value	1780 mg/m^3 (750 ppm)
Country	United States
Remark	Short-Term Exposure Limit 2375 mg/m^3 (1000 ppm)
Remark	Exposures above the TLV-TWA up to the STEL should not be longer than 15 minutes and should not occur more than four times per day.

1.9 Source of Exposure

Remark	Acetone is a product of the photooxidation of some alkane and alkene compounds that are found in urban air and is also a by-product resulting from oxidation of humic substances. In addition, natural sources of acetone include by-products from forest fires, volcanoes, and metabolism of insects and higher animals.
Remark	Acetone is a normal constituent of human blood and is a component of human breath (of metabolic origin).
Remark	Acetone may be released to the environment as stack emissions, fugitive emissions, and in waste water in its production and use in the manufacture of methacrylates, as a solvent, and as a chemical intermediate in the manufacture of methyl isobutyl ketone and other chemicals.
Remark	Acetone has also been identified in wastewater from industrial and municipal treatment plants.

Remark	Acetone does not appear to be persistent in the environment due to its biodegradability, despite its widespread presence in the environment.

2. Physico-chemical Data

2.1 Melting Point

Value	-94.6 °C
GLP	no data
Reference	Handbook of Chemistry and Physics (1986). R.C. Weast (ed.), 67th Ed., p. C51. CRC Press Inc. Boca Raton, FL.

2.2 Boiling Point

Value	56.1 °C at 760 mm Hg
GLP	no data
Reference	Handbook of Chemistry and Physics (1986). R.C. Weast (ed.), 67th Ed., p. C51. CRC Press Inc. Boca Raton, FL.

2.3 Density

Value	0.791 g/mL at 20 °C
GLP	no data
Reference	Handbook of Chemistry and Physics (1986). R.C. Weast (ed.), 67th Ed., p. C51. CRC Press Inc. Boca Raton, FL.

2.4 Vapour Pressure

Value	182 mm Hg at 20 °C
GLP	no data
Reference	Kirk-Othmer Encyclopedia of Chemical Technology (1991). 4th Ed. Volume 1. John Wiley & Sons, New York, NY.

Value	230 mm Hg at 25 °C
Method	other (calculated)
GLP	no data
Reference	NOMO5 Program. Syracuse Research Corp.,Syracuse, NY.

2.5 Partition Coefficient

Value	-0.24
Type	Log P_{ow}
GLP	no
Reference	Hansch, C. and Leo, A. (1979). Substituent Constants for Correlation Analysis in Chemistry and Biology, p. 179. John Wiley & Sons, New York, NY.

2.6 Water Solubility

Description	miscible
GLP	no data
Remark	Miscible with water, alcohol, dimethylformamide, ether.
Reference	The Merck Index (1983). M. Windholz (ed.), 10th Ed., p. 57. Merck & Co., Rahway, NJ.

2.7 Flash Point

Value	-17 °C
Type	closed cup
GLP	no data
Reference	Fire Hazard Properties of Flammable Liquids, Gases, and Volatile Solids (1991). National Fire Protection Association, NFPA 325M, 10th Ed. Quincy, MA.

2.8 Auto Flammability

Value	465 °C (autoignition temperature)
GLP	no data
Reference	Fire Hazard Properties of Flammable Liquids, Gases, and Volatile Solids (1991). National Fire Protection Association, NFPA 325M, 10th Ed. Quincy, MA.

2.9 Flammability

Result	highly flammable
GLP	no data
Reference	Fire Hazard Properties of Flammable Liquids, Gases, and Volatile Solids (1991). National Fire Protection Association, NFPA 325M, 10th Ed. Quincy, MA.

2.10 Explosive Properties

Result	not explosive
GLP	no data
Reference	Fire Hazard Properties of Flammable Liquids, Gases, and Volatile Solids (1991). National Fire Protection Association, NFPA 325M, 10th Ed. Quincy, MA.

3. Environmental Fate and Pathways

3.1 Stability

3.1.1 Photodegradation

Type	air
Light Source	xenon lamp
Light Spect.	250-330 nm
Rel. Intens.	Based on intensity of sunlight
Spectrum	lambda (max) >295 nm epsilon (max) 295 nm

Concentration	200 mg/L
GLP	no data
Test substance	no data
Result	Quantum yield varied with wavelength from 1.59 to 0.27 for CO_2 production. Direct photolysis half-life was 32 days. The half-life reported is the annual average in the lower troposphere at 40 degrees northern latitude. Indirect photolysis rate constant estimated to be 0.00000026 cm^3/mol/sec based on OH sensitizer concentration of 1,180,000 mol/cm^3.
Test condition	Temperature for direct photolysis test equaled room temperature
Reference	Meyrahn, H., Pauly, J., Schneider, W., and Warneck, P. (1986). Quantum yields for the photodissociation of acetone in air and an estimate for the lifetime of acetone in the lower troposphere. J. Atmos. Chem. 4:277-291.

Type	air
Light Spect.	279-313 nm
Rel. Intensity	based on intensity of sunlight
Spectrum	lambda (max) >295 nm epsilon (max) 295 nm
GLP	no data
Test substance	no data
Result	Quantum yield: 0.15 (25 torr); 0.08 (> 400 torr) Photolysis half-life is 40 days near the earth surface to 10 days at 200 mbar pressure. Attack by hydroxyl radicals with half-life of 20 days near earth surface to 100 days at 200 mbar pressure.
Reference	Gardner, E.P. (1984). The primary quantum yields of photodecomposition of acetone in air under tropospheric conditions. J. Phys. Chem. 88:5069-5076. Chatfield, R.B., Gardner, E.P., and Calvert, J.G. (1987). Sources and sinks of acetone in the troposphere: Behavior of reactive hydrocarbons and a stable product. J. Geophys. Res. 92:4208-4216.

3.2 Monitoring Data (Environment)

Type	background concentration
Media	air
Remark	Acetone detected at 1.6-4 part per billion by volume (ppbv), 4.8-12 ppbC, average concentration over a 1-yr period in Denver, Colorado, USA.
Reference	Anderson, L.G., Lanning, J.A., and Wolfe, P. (1994). Acetone in the urban atmosphere: A case study in Denver, Colorado. Israel J. Chem. 34:341-353.

Type	background concentration
Media	air
Remark	1.6 ppb (4.8 ppbC) and 1.8 ppb (5.4 ppbC) found in two rural sites in Ontario, Canada, 1988.

Reference	Shepson, P.B., Hastie, D.R., Schiff, H.I., Polizzi, M., Bottenheim, J.W., Anlauf, K., Mackay, G.I., and Karecki, D.R. (1991). Atmospheric concentrations and temporal variations of C_1-C_3 carbonyl compounds at two rural sites in central Ontario. Atmos. Environ. 25A:2001-2015.
Type	background concentration
Media	air
Remark	12 ppb (36 ppbC) in troposphere above Tucson, Arizona; 2.8 ppb (8.4 ppbC) at two rural sites 40 km away.
Reference	Snider, J.R. and Dawson, G.A. (1985). Tropospheric light alcohols, carbonyls, and acetonitrile: Concentrations in the southwestern United States and Henry's Law data. J. Geophys. Res. 90:3797-3805.
Type	background concentration
Media	air
Remark	Range of 4.1-94 part per billion by volume (ppbv), 12.3-282 ppbC, at two urban sites in USA. Additionally, a range of 19.5-89.6 ppbv, (58.5-268.8 ppbC) was reported in a variety of work settings, including indoor air.
Reference	Kelly, T.J., Callahan, P.J., Piell, J., and Evans, G.F. (1993). Method development and field measurements for polar volatile organic compounds in ambient air. Environ. Sci. Technol. 27:1146-1153.
Type	background concentration
Media	air
Remark	Qualitative detection in volcanic gas from Guatemala.
Reference	Stoiber, R.E., Leggett, R.E., Jenkins, T.F., Murrmann, R.P., and Rose, W.I. (1971). Organic compounds in volcanic gas from Santiaguito volcano, Guatemala. Am. Geolog. Soc. Bull. 82:2299-2302.
Type	contaminated site
Media	air
Remark	Acetone detected at 770-4100 parts per billion by volume (ppbv) 2310-12,300 ppbC, around several different manufacturing sites.
Reference	Hoshitia, Y., Nihei, Y., Muto, G. (1981). Pattern display for characterization of trace amounts of odorants discharged from nine odor sources. Analyst 106:1187-1202.
Type	background concentration
Media	air
Remark	6.7-32.3 parts per billion as carbon (ppbC) was detected in seven Florida (USA) sites.
Reference	Lonneman, W.E., Sella, R.L., and Bufalini, J.J. (1978). Ambient air hydrocarbon concentrations in Florida. Env. Sci. Technol. 12:459-463.

Type	background concentration
Media	air
Remark	0.5-20.6 parts per billion as carbon (ppbC) was detected in USA continental and marine areas.
Reference	Duce, R.A., Mohnen, V.A., Zimmerman, P.R., Grosjean, D., Cautreels, W., Chatfield, R., Jaenicke, R., Ogren, J.A., Pelliari, E.D., and Wallace, G.T. (1983). Organic material in the global troposphere. Rev. Geophys. Space Phys. 21:921-952.
Type	background concentration
Media	air
Remark	An average of 470 parts per trillion by volume (pptv) (1410 pptC) of acetone at ground level to 120 pptv (360 pptC) in the upper troposphere was detected.
Reference	Arnold, F., Knop, G., and Ziereis, H. (1986). Acetone measurements in the upper troposphere and lower stratosphere- implications for hydroxyl radical abundances. Nature 321:505-507.
Type	background concentration
Media	air
Remark	4-52 part per billion as carbon (ppbC) was detected at three sites in the USA.
Reference	Arnts, R.R. and Meeks, S.A. (1981). Biogenic hydrocarbon contribution to the ambient air of selected areas. Atmos. Environ. 15:1643-1651.
Type	contaminated site
Media	ground water
Remark	A concentration of 43,700 µg/L was detected onsite at a contaminated landfill; 0.2-0.7 µg/L acetone was found in wells adjacent to the landfill.
Reference	DeWalle, F.B. and Chien, E.S.K. (1981). Detection of trace organics in well water near a solid waste landfill. J. Am. Water Works Assoc. 73:206-211.
Type	contaminated site
Media	air
Remark	20-250 part per billion by volume (ppbv) (60-750 ppbC) was detected in a house near a contaminated landfill.
Reference	Hodgson, A.T., Garbesi, K., Sextro, R.G., and Daisey, J.M. (1992). Soil-gas contamination and entry of volatile organic compounds into a house near a landfill. J. Air Waste Manage. Assoc. 42:277-283.
Type	other
Media	air
Remark	Acetone was detected in seven different product categories. The percentage of products with acetone at the average concentration (w/w%) are as follows:

	23% automotive - 28.1 11% household cleaners - 0.3 51% paints - 29.3 15% fabric & leather - 12.9 16% electronic equipment - 0.3 5% oils, greases, lubricants - 0.2 24% adhesives - 18.8
Reference	Sack, T.M., Steele, D.H., Hammerstrom, K., and Remmers, J. (1992). A survey of household products for volatile organic compounds. Atmos. Environ. 26A:1063-1070.
Type	other
Media	air
Remark	Acetone was found in the homes of smoking and non-smoking adults at average concentrations of 71 and 50 $\mu g/m^3$, respectively.
Reference	Heavner, D.L., Morgan, W.T., and Ogden, M.W. (1996). Determination of volatile organic compounds and respirable suspended particulate matter in New Jersey and Pennsylvania homes and workplaces. Environ. Int. 22:159-183.
Type	other
Media	air
Remark	Acetone was emitted from particle board at rate ranging from 37- 41 $\mu g/m^2/h$.
Reference	Tichenor, B.A. and Mason, M.A. (1988). Organic emissions from consumer products and building materials to the indoor environment. J. Air Pollut Control Assoc. 38:264-268.
Type	other
Media	air
Remark	78.8 ppm (236.4 ppmC) found in smoke from polypropylene burning.
Reference	Woolley, W.D. (1982). Smoke and toxic gas production from burning polymers. J. Macromol. Sci. Chem. A17:1-33.
Type	background concentration
Media	air
Remark	14-66 $\mu g/m^3$ (6-30 ppb) (18-120 ppbC) acetone was detected in a new office building over a period of one year.
Reference	Hodgson, A.T., Daisey, J.M., and Grot, R.A. (1991). Sources and source strengths of volatile organic compounds in a new office building. J. Air Waste Manage. Assoc. 41:1461-1468.
Type	contaminated site
Media	air
Remark	6838-32,500 part per billion by volume (ppbv) (20,514-97,500 ppbC) was detected in the air at municipal landfill sites.

Reference	Brosseau, J. and Heitz, M. (1994). Trace gas compound emissions from municipal landfill sanitary sites. Atmos. Environ. 28:285-293.
Type	contaminated site
Media	water
Remark	Acetone ranged from 9 ppb influent to 41 ppb effluent in a textile finishing plant.
Reference	Gordon, A.W. and Gordon, M. (1981). Analysis of volatile organic compounds in a textile finishing plant effluent. Trans. Ky. Acad. Sci. 42:149-157.
Type	background concentration
Media	water
Remark	0-41 ng/mL acetone was detected in cloud water at a remote continental (USA) site.
Reference	Aneja, V.P. (1993). Organic compounds in cloud water and their deposition at a remote continental site. J. Air Waste Manage. Assoc. 43:1239-1244.
Type	background concentration
Media	water
Remark	0-0.052 mg/L acetone was detected in seawater samples from Florida and the Eastern Mediterranean.
Reference	Corwin, J.F. (1969). Volatile oxygen-containing organic compounds in sea water: Determination. Bull. Marine Sci. 19:504-509.
Type	background concentration
Media	biota
Remark	Acetone is a normal endogenous biochemical that can be routinely detected and measured in body fluids. Detectable amounts of acetone have been found in a variety of biological specimens including whole blood (fetal through adult), cerebrospinal fluid, urine, exhaled air, and breast milk.
Reference	Dowty, B.J., Laseter, J.L., and Storer, J. (1976). The transplacental migration and accumulation in blood of volatile organic compounds. Pediatr. Res. 10:696-701. Sulway, M.J., Trotter, M.D., Trotter, E., and Malins, J.M. (1971). Acetone in uncontrolled diabetes. Postgrad. Med. J. 47(Suppl.):383-387. Zlatkis, A., Bertsch, W., Lichtenstein, H.A., Tishbee, A., Shunbo, F., Liebich, H.M., Coscia, A.M., and Fleischer, N. (1973). Profile of volatile metabolites in urine by gas chromatography-mass chromatography. Anal. Chem. 45:763-767. Pellizzari, E.D., Hartwell, T.D., Harris, B.S.H., Waddell, R.D., Whitaker, D.A., and Erickson, M.D. (1982). Purgeable

organic compounds in mother's milk. Bull. Environ. Contam. Toxicol. 28:322-328.

Type	background concentration
Media	biota
Remark	The normal limit for blood, serum, and plasma acetone in non-fasting adults has been shown to range from 0.8-4.4 mg/L depending on the analytical method applied. The acetone concentration in plasma can be 8-11% greater than the level in whole blood. Infants, pregnant women, and training athletes can have ketone body levels that are elevated 2 to 20-fold above normal due to the ketogenesis resulting from their higher energy requirements.
Reference	Paterson, P., Sheath, J., Taft, P., and Wood, C. (1967). Maternal and foetal ketone concentration in plasma and urine. Lancet II:862-865.

Koeslag, J.H., Noakes, T.D., and Sloan, A.W. (1980). Post-exercise ketosis. J. Physiol. 301:79-90.

Ashley, D.L., Bonin, M.A., Cardinali, F.L. McCraw, J.M., and Wooten, J.V. (1994). Blood concentrations of volatile organic compounds in a nonoccupationally exposed US population and in groups with suspected exposure. Clin. Chem. 40:1401-1404.

Trotter, M.D., Sulway, M.J., and Trotter, E. (1971). The rapid determination of acetone in breath and plasma. Clin. Chem. Acta 35:137-143.

Kimura, M., Kobayashi, K., Matsuoka, A., Hayashi, K., and Kimura, Y. (1985). Head-space gas-chromatographic determination of 3-hydroxybutyrate in plasma after enzymic reactions, and the relationship among the three ketone bodies. Clin. Chem. 31:596-598.

Brega, A., Villa, P., Quadrini, G., Quadri, A., and Lucarelli, C. (1991). High-performance liquid chrom-otographic determination of acetone in blood and urine in the clinical diagnostic laboratory. J. Chromatogr. 553:249-254.

Gavino, V.C., Vinet, B., David, F, Garneau, M., and Brunengraber, H. (1986). Determination of the concen-tration and specific activity of acetone in biological fluids. Anal. Biochem. 152:256-261.

Wang, G., Maranelli, G., Perbellini, L., Raineri, E., and Brugnone, F. (1994). Blood acetone concentration in "normal people" and in exposed workers 16 h after the end of the workshift. Int. Arch. Occup. Environ. Health65:285-289.

Type	background concentration
Media	biota
Remark	Endogenous acetone concentrations in normal human spot urine specimens have been shown to range from 0.3-3.0 mg/L. The urinary concentration of acetone was not found to increase appreciably when test subjects performed light physical exercise. A consistent diurnal trend was observed, however, with higher urine acetone concentrations found in the late evening and early morning than during the day.
Reference	Brega, A., Villa, P., Quadrini, G., Quadri, A., and Lucarelli, C. (1991). High-performance liquid chrom-atographic determination of acetone in blood and urine in the clinical diagnostic laboratory. J. Chromatogr. 553:249-254. Kobayashi, K., Okada, M., Yasuda, Y., and Kawai, S. (1983). A gas chromatographic method for the determination of acetone and acetoacetic acid in urine. Clin. Chem. Acta 133:223-226. Levey, S., Balchum, O.J., Medrano, V., and Jung, R. (1964). Studies of metabolic products in expired air. II. Acetone. J. Lab. Clin. Med. 63:574-584. Pezzagno, G., Imbriani, M., Ghittori, S., and Capodaglio, E. (1988). Urinary concentration, environ-mental concentration, and respiratory uptake of some solvents: Effect of the work load. Am. Ind. Hyg. Assoc. J. 49:546-552. Wang, G., Maranelli, G., Perbellini, L., Raineri, E., and Brugnone, F. (1994). Blood acetone concentration in "normal people" and in exposed workers 16 h after the end of the workshift. Int. Arch. Occup. Environ. Health 65:285-289.

Type	background concentration
Media	biota
Remark	The normal value for endogenous acetone in expired air specimens from adult humans was found to average between 0.7-1.6 mg/L, regardless of whether the subjects were fed or fasted overnight.
Reference	Rooth, G. and Tibbling, G. (1968). Free fatty acids, glycerol and alveolar acetone in obese women during phenformin treatment. Acta Med. Scand. 184:263-267. Rooth, G. and Östenson, S. (1966). Acetone in alveolar air, and the control of diabetes. Lancet II:1102-1105. Levey, S., Balchum, O.J., Medrano, V., and Jung, R. (1964). Studies of metabolic products in expired air. II. Acetone. J. Lab. Clin. Med. 63:574-584.

Crofford, O.B., Mallard, R.E., Winton, R.E., Rogers, N.L., Jackson, J.C., and Keller, U. (1977). Acetone in breath and blood. Trans. Am. Clin. Climatol. Assoc. 88:128-139.

Trotter, M.D., Sulway, M.J., and Trotter, E. (1971). The rapid determination of acetone in breath and plasma. Clin. Chem. Acta 35:137-143.

Jansson, B.O. and Larsson, B.T. (1969). Analysis of organic compounds in human breath by gas chromatography-mass spectrometry. J. Lab. Clin. Med. 74:961-966.

Stewart, R.D. and Boettner, E.A. (1964). Expired-air acetone in diabetes mellitus. New Eng. J. Med. 270:1035-1038.

Tassopoulos, C.N., Barnett, D., and Fraser, T.R. (1969) Breath-acetone and blood-sugar measurements in diabetes. Lancet II:1282-1286.

Phillips, M. and Greenberg, J. (1987). Detection of endogenous acetone in normal human breath. J. Chromatogr. 422:235-238.

Wang, G., Maranelli, G., Perbellini, L., Raineri, E., and Brugnone, F. (1994). Blood acetone concentration in "normal people" and in exposed workers 16 h after the end of the workshift. Int. Arch. Occup. Environ. Health 65:285-289.

Type	other
Media	biota
Remark	Four workers exposed to 30 ppm (71.1 mg/m^3) of acetone for 2 h were found to retain about 80% of the inhaled acetone. The concentration of acetone in the urine increased from about 0.75 mg/L at the beginning of the workshift to about 2.0 mg/L by the end of the shift. The acetone in venous blood increased from 1.0 mg/L at the start of the shift to 3.3 mg/L by the end. Urine and blood acetone levels returned to normal within 24 h.
Reference	Baumann, K. and Angerer, J. (1979). Untersuchungen zur Frage der beruflichen Lösungsmittelbelastung mit Aceton. Krebsgefaehrdung Arbeitsplatz Arbeitsmed. 19:403-408.

Type	other
Media	biota
Remark	Biological monitoring of styrene exposure in the workplace was not affected by co-exposures to acetone. Styrene metabolite concentrations in the urine of 22 workers exposed to styrene and acetone were not affected by 8-h TWA acetone exposures that ranged from about 10-210 ppm (25 to 498 mg/m^3).

Reference	DeRosa, E., Cellini, M., Sessa, G., Saletti, C., Rausa, G., Marcuzzo, G., and Bartolucci, G.B. (1993). Bio-logical monitoring of workers exposed to styrene and acetone. Int. Arch. Occup. Environ. Health 65:S107-S110.

3.3 Transport and Distribution in Environmental Compartments

3.3.1 Transport

Type	volatility
Media	water-air
Method	mass-transfer coefficients measurement
Result	The liquid film mass-transfer coefficient K_L ranged from 0.28-0.54 m/day.
Reference	Rathbun, R.E. and Tai, D.Y. (1982). Volatilization of ketones from water. Water Air Soil Pollut. 17:281-293.

Type	volatility
Media	water-air
Method	acetone measured in model stream
Result	Volatilization coefficient ranged from 82,300-111,000 min^{-1}.
Reference	Rathbun, R.E., Stephans, D.W., and Tai, D.Y. (1991). Fate of acetone in an outdoor model stream with a nitrate supplement, southern Mississippi, U.S.A. J. Hydrol. 123:225-242.

3.3.2 Distribution

Media	water-air
Method	other (measurement)
Remark	Partition between air and seawater at a variety of temperatures was measured and calculated.
Result	Partition coefficient K (m/atm) was 14.8-71.3.
Reference	Zhou, X. and Mopper, K. (1990). Apparent partition coefficients of 15 carbonyl compounds between air and seawater and between air and freshwater: Implications for air-sea exchange. Environ. Sci. Technol. 24:1864-1869.

Media	water sediment
Method	other (measurement)
Result	200-230 ppm acetone was detected in wastewater; acetone was not detected in river water or sediment.
Reference	Jungclaus, G.A., Lopez-Avila, V., and Hites, R.A. (1978). Organic compounds in an industrial wastewater: A case study of their environmental impact. Environ. Sci. Technol.12:88-96.

Media	water-air
Method	other (measurement)
Result	Henry's law constant was 25.6-27.0 m/atm at 25°C.
Reference	Betterton, E.A. (1991). The partitioning of ketones between the gas and aqueous phases. Atmos. Environ. 25A:1473-1477.

3.4 Mode of Degradation

Remark	biological oxidation
Reference	Rathbun, R.E., Stephens, D.W., and Tai, D.Y. (1993). Bacterial degradation of acetone in an outdoor model stream. Environ. Pollut. 79,153-162. Rathbun, R.E., Stephans, D.W., and Tai, D.Y. (1991). Fate of acetone in an outdoor model stream with a nitrate supplement, southern Mississippi, U.S.A. J. Hydrol. 123,225-242. Taylor, D.G., Trudgill, P.W., Cripps, R.E., and Harris, P.R. (1980). The microbial metabolism of acetone. J. Gen. Microbiol. 118,159-170.

3.5 Biodegradation

Type	aerobic
Inoculum	activated sludge, domestic
Degradation	78% after 28 days
Results	readily biodegradable
Method	OECD Guideline 301 D
GLP	no data
Test substance	no data
Reference	Waggy, G.T., Conway, R.A., Hansen, J.L., and Lessing, R.L. (1994). Comparison of 20-d BOD and OECD closed-bottle biodegradation tests. Environ. Toxicol. Chem. 13:1277-1280.

Type	aerobic
Inoculum	activated sludge, domestic
Concentration	100 mg/L
Degradation	42% after 155 h
Method	other
GLP	no data
Test substance	no data
Reference	Urano, K. and Kato, Z. (1986). A method to classify biodegradabilities of organic compounds. J. Hazard. Materials 3:147-159.

Type	aerobic
Inoculum	activated sludge, domestic
Concentration	500 mg/L
Degradation	0% after 24 h
Results	Under test conditions no biodegradation observed
Method	other
GLP	no
Test substance	no data
Remark	This study used a quite high substrate concentration for a limited period of time (24 h), when contrasted to more current methods.

Reference	Gerhold, R.M. and Malaney, G.W. (1966). Structural determinants in the oxidation of aliphatic compounds by activated sludge. J. Water Pollut. Control Fed. 38:562-579.
Type	aerobic
Inoculum	activated sludge, domestic
Concentration	2.5 mg/L
Degradation	78.2%
Results	readily biodegradable
Method	other
GLP	no
Test substance	no data
Remark	Results based on BOD.
Reference	Lamb, C.B. and Jenkins, G.F. (1952). B.O.D. of synthetic organic chemicals. Proc. Ind. Waste Conf. 36:326-339.
Type	aerobic
Inoculum	activated sludge, domestic, adapted
Concentration	333 mg/L
Degradation	86% after 8 h
Results	readily biodegradable
Method	other
GLP	no
Test substance	no data
Reference	Hatfield, R. (1957). Biological oxidation of some organic compounds. Ind. Eng. Chem. 49:192.
Type	aerobic
Inoculum	activated sludge, domestic, adapted
Degradation	47% after 10 days
Method	other
GLP	no
Test substance	no data
Remark	Early study of a wastewater treatment plant.
Test concentration	250-1000 mg/L.
Reference	Mills, E.J. and Stack, V.T. (1954). Biological oxidation of synthetic organic chemicals. Proc. Ind. Waste. Conf. 38:492-517.
Type	aerobic
Inoculum	activated sludge, domestic, adapted
Degradation	38% after 5 days
GLP	no data
Test substance	no data
Remark	Results based on BOD measurement.
Test concentration	0.4-3.2 mg/L
Reference	Babeu, L. and Vaishnav, D.D. (1987). Prediction of biodegradability for selected organic chemicals. J. Ind. Microbiol. 2:107-115.
Type	anaerobic

Inoculum	inoculum from sediment and groundwater
Concentration	50 mg/L
Degradation	100% after 244 days
GLP	no data
Test substance	no data
Remark	Test concentration reported as ppm carbon.
Remark	Results were comparable in sulfite and nitrate-reducing systems.
Reference	Mormile, M.R., Liu, S., and Suflita, J.M. (1994). Anaerobic biodegradation of gasoline oxygenates: Extrapolation of information to multiple sites and redox conditions. Environ. Sci. Technol. 28:1727-1732.

Type	aerobic
Inoculum	activated sludge, domestic
Concentration	10 mg/L
Degradation	81% after 20 days
GLP	no data
Test substance	no data
Remark	BOD/ThOD ratio.
Reference	Young, R.H.F., Ryckman, D.W., and Buzzell, J.C. (1968). An improved tool for measuring biodegradability. J. Water Pollut. Control Fed. 40:R354-R368.

Type	aerobic
Inoculum	activated sludge, domestic
Concentration	3.2 mg/L
Degradation	38% after 5 days
GLP	no data
Test substance	no data
Remark	results based on BOD.
Reference	Vaishnav, D.D., Boethling, R.S., and Babeu, L. (1987). Quantitative structure-biodegradability relationships for alcohols, ketones and alicyclic compounds. Chemosphere 16:695-703.

Type	aerobic
Inoculum	lab-generated organisms seeded from domestic sludge.
Degradation	100%
GLP	no data
Test substance	no data
Remark	Removal rate was 125 mg/L/day after a 5-day lag.
Concentration	166-500 mg/L.
Reference	Chou, W.L., Speece, R.E., and Siddiqi, R.H. (1978). Acclimation and degradation of petrochemical wastewater components by methane fermentation. In: Biotechnology and Bioengineering Symposium No. 8., C.D. Scott, ed., pp. 391-414. John Wiley and Sons, New York, NY.

3.6 BOD_5, COD or BOD_5/COD Ratio

Method	other
Year	1979
BOD_5	1.85 g/g
COD	1.92 g/g
GLP	no data

BOD_5/COD Ratio	0.96
Method	APHA "Standard Methods" 1989.
Concentrations	3, 7, and 10 mg/L were used.
Remark	In additional testing, BOD_{10}, BOD_{15}, and BOD_{20} were determined (Birdie et. al., 1979). ThOD - 2.21 (based on calculation). BOD_{10} - 76% of ThOD BOD_{15} - 83% of ThOD BOD_{20} - 84% of ThOD
Test condition	COD Method = ASTM D1252-67 (reapproved 1974). BOD_5 Method = APHA Standard Methods No. 219,1971
Reference	Birdie, A.L., Wolff, C.J.M., and Winter, M. (1979). BOD and COD of some petrochemicals. Water Res. 13:627-630.

BOD_5/COD Ratio	no data
BOD_5	56% of ThOD
Concentrations	3, 7, 10 mg/L
Method	APHA Standard Methods 1989.
Reference	Waggy, G.T., Conway, R.A., Hansen, J.L., and Blessing, R.L. (1994). Comparison of 20-d BOD and OECD closed-bottle biodegradation tests. Environ. Toxicol. Chem. 13:1277-1280.

3.7 Bioaccumulation

Species	haddock (adult)
Temperature	7 °C
BCF	0.69
Year	1931
GLP	no
Test condition	static
Reference	Rustung, E., Koren, F., and Föyen, A. (1931). Über Aufnahme und von Aceton im Organismus von Kaltblütern. Biochem. Z. 242:366-376.

4. Ecotoxicity

4.1 Acute/Prolonged Toxicity to Fish

Type	flow through
Species	Salvelinus fontinalis
Exposure Period	96 h
LC_{50}	6070 mg/L
Analyt. Monitoring	no data
GLP	no data

Test Substance	no data
Remark	The exposure process is described in U.S. EPA: Methods for Acute Toxicity Tests with Fish, Macro-invertebrates, and Amphibians. EPA-660/3-75-009. Committee on Methods for Toxicity Tests with Aquatic Organisms (1975). The methods used by Cardwell et.al. (1974) are similar in duration of exposure, type of test vessel, physical/chemical parameters monitored, selection of dilution water, and selection of test species.
Reference	Cardwell, R.D., Foreman, D.G., Payne, T.R., and Wilber, D.J. (1974). Acute and chronic toxicity of four organic chemicals to fish. Project Report to C.E. Stephen, U.S. EPA, Environmental Research Laboratory - Duluth. Duluth, MN.

Type	flow through
Species	Lepomis macrochirus
Exposure Period	96 h
LC_{50}	7300 mg/L
Analyt. Monitoring	no data
GLP	no data
Test Substance	no data
Remark	Test Method similar to U.S. EPA: Methods for Acute Toxicity Tests with Fish, Macroinvertebrates, and Amphibians. EPA-660/3-75-009. Committee on Methods for Toxicity Tests with Aquatic Organisms, 1975.
Reference	Cardwell, R.D., Foreman, D.G., Payne, T.R., and Wilber, D.J. (1974). Acute and chronic toxicity of four organic chemicals to fish. Project Report to C.E. Stephen, U.S. EPA, Environmental Research Laboratory - Duluth. Duluth, MN.

Type	flow through
Species	Pimephales promelas
Exposure Period	96 h
LC_{50}	9100 mg/L
Analyt. Monitoring	no data
GLP	no data
Test Substance	no data
Remark	Test method similar to U.S. EPA: Methods for Acute Toxicity Tests with Fish, Macroinvertebrates, and Amphibians. EPA-660/3-75-009. Committee on Methods for Toxicity Test with Aquatic Organisms, 1975.
Reference	Cardwell, R.D., Foreman, D.G., Payne, T.R., and Wilber, D.J.(1974). Acute and chronic toxicity of four organic chemicals to fish. Project Report to C.E. Stephen, U.S. EPA - Environmental Research Laboratory - Duluth. Duluth, MN.

Type	static
Species	Gambusia affinis
Exposure Period	72 h
LC_{50}	13,000 mg/L
Analyt. Monitoring	no data

GLP	no data
Test substance	no data
Results	24-h LC_{50} = 13,500 mg/L 48-h LC_{50} = 13,000 mg/L Below 11,500 mg/L, the fish showed no permanent distress.
Remark	Method similar to Doudoroff et al., Bioassay methods for the evaluation of acute toxicity of industrial wastes to fish. Sewage Ind. Wastes 23:1380-1397, 1951.
Reference	Wallen, I.E., Greer, W.C., and Lasater, R. (1957). Toxicity to Gambusia affinis of certain pure chemicals in turbid waters. Sewage Ind. Wastes 29:695-711.

Type	flow through
Species	Pimephales promelas
Exposure Period	96 h
LC_{50}	8120 mg/L
Analyt. Monitoring	yes
GLP	no data
Test Substance	no data
Remark	Method similar to: Methods for Measuring the Acute Toxicity of Effluents to Aquatic Organisms. W. Piltier, Bioassay Subcommittee. EPA Biological Advisory Committee, Ecology Branch. EPA-600/4-28-012, 1978.
Reference	Veith, G. (1983). Structure-toxicity relationships for the fathead minnow, Pimaphales promelas: Narcotic industrial chemicals. Can. J. Fish Aquat. Sci. 40:743-748.

Type	static
Species	Oncorhynchus mykiss
Exposure Period	96 h
LC_{50}	5540 mg/L
Analyt. Monitoring	no data
GLP	no data
Test Substance	prescribed by 1.1-1.4
Remark	Method similar to: Methods for Measuring the Acute Toxicity of Effluents to Aquatic Organisms. W. Piltier, Bioassay Subcommittee. EPA Biological Advisory Committee, Ecology Branch, EPA-600/4-28-012, 1978.
Reference	Johnson, W.W. and Finley, M.T. (1980). Handbook of Acute Toxicity of Chemicals to Fish and Aquatic Inver-tebrates. Department of the Interior Fish and Wildlife Service. Resource Publication 137. Washington, DC.

Type	flow through
Species	Pimephales promelas
Exposure Period	96 h
LC_{50}	6210-8120 mg/L
Analyt. Monitoring	yes
GLP	no data
Test substance	no data
Test method	similar to OECD Guideline 204.

Remark	Results from 3 test runs (LC_{50} in mg/L): 24-h: 8830, 9400, 8030 72-h: 8120, 7940, 6400 96-h: 8120, 7280, 6210
Reference	Brooke, L.T., Call, D.J., Geiger, D.L., and Northcott, C.E. (1984). Acute Toxicities of Organic Chemicals to Fathead Minnows (Pimephales promelas). Center for Lake Superior Environmental Studies.

Type	static
Species	Poecilia reticulata
Exposure Period	14 day
LC_{50}	6400 mg/L
Analyt. Monitoring	no data
GLP	no data
Test substance	no data
Test method	similar to U.S. EPA: Methods for Acute Toxicity Tests with Fish, Macroinvertebrates, and Amphibians. EPA-660/3-75-009. Committee on Methods for Toxicity Tests with Aquatic Organisms, 1975.
Reference	Konemann, H. (1981). Quantitative structure-activity relationships in fish toxicity studies. Part 1: Relationship for 50 industrial pollutants. Toxicology 9:209-221.

Type	flow through
Species	Salmo gairdneri
Exposure Period	24 h
LC_{50}	6100 mg/L
Analyt. Monitoring	no data
GLP	no data
Test substance	no data
Remark	Acetone (2930 mg/L) produced an increase in ventilation rate, reaching a maximum of 158% of controls at 21 hours for the duration of the exposure period.
Remark	Method similar to that contained in: Sprague, J.B. (1969). Measurement of pollutant toxicity to fish. I. Bioassay methods for acute toxicity. Water Res. 3:793-821.
Reference	Majewski, H.S., Klaverkamp, J.F., and Scott, D.P. (1978). Acute lethality and sub-lethal effects of acetone, ethanol, and propylene glycol on the cardiovascular and respiratory systems of rainbow trout (Salmo gairdneri). Water Res. 13:217-221.

Type	static
Species	Lepomis macrochirus
Exposure Period	96 h
LC_{50}	8300 mg/L
Analyt. Monitoring	no data
GLP	no
Test substance	no data

Remark	Test method similar to Doudoroff, P. (1951). Bioassay methods for the evaluation of acute toxicity of industrial wastes to fish. Sewage Ind. Wastes 23:1380-1397.
Reference	Cairns, J. and Scheier, A. (1968). A comparison of the toxicity of some of the common industrial waste components tested individually and combined. Progressive Fish Culturist 30:3-8.

Type	static
Species	Carassius auratus
Exposure Period	24 h
LC_{50}	>5000 mg/L
Analyt. Monitoring	no data
GLP	no data
Test substance	no data
Remark	Method similar to that described in: American Public Health Association. Review papers on measurement of pollutant toxicity to fish. Sprague, J.B. (1969). Bioassay methods for acute toxicity. Water Res. 3:793-821.
Reference	Birdie, A.L., Wolff, C.J.M., and Winter, M. (1979). The acute toxicity of some petrochemicals to goldfish. Water Res.13:623-626.

Type	static
Species	Leuciscus idus
Exposure Period	48 h
LC_{50}	7505-11,300 mg/L
Analyt. Monitoring	no data
GLP	no data
Test substance	no data
Remark	Test method similar to: U.S. EPA: Methods for acute toxicity tests with fish, macroinvertebrates, and amphibians. EPA-660/3-75-009. Committee on methods for toxicity tests with aquatic organisms, 1975.
Reference	Juhuke, I. and Luedemann, D. (1978). Results of the study of 200 chemical compounds on acute toxicity using the golden orfe test. Z. Wasser Abwasser Forsch. 11:161-164.

Type	flow through
Species	Pimephales promelas
Exposure Period	1 h
LC_{50}	6210-8030 mg/L
Analyt. Monitoring	yes
GLP	no data
Test substance	no data
Remark	Test method similar to U.S. EPA: Methods for acute toxicity test with fish, macroinvertebrates, and amphibians. EPA-660/3-75-009. Committee on Methods for Toxicity Test with Aquatic Organisms, 1975.
Result	Results of 3 test runs are as follows (LC_{50} in mg/L): 24-h: 8830, 9400, 8030 48-h: 8290, 8880, 7940

	72-h: 8120, 7940, 6400 96-h: 8120, 7280, 6210
Test substance	minimum purity 90%; analysis of test article in water from fish exposure tanks.
Reference	Brooke, L.T., Call, D.J., Geiger, D.L., and Northcott, C.E. (1984). Acute toxicities of organic chemicals to fathead minnows (Pimephales promelas). Center for Lake Superior Environmental Studies. University of Wisconsin - Superior. pp. 319.

4.2 Acute Toxicity - Aquatic Invertebrates

Species	Daphnia magna
Exposure Period	48 h
LC_{50}	12,600 & 12,700 mg/L (two laboratories)
Analyt. Monitoring	no data
GLP	no data
Test substance	no data
Remark	Tests conducted according to a protocol from the Dutch Standard Institute (Adema, 1978).
Reference	Canton, J.H. and Adema, D.M.M. (1978). Repro-ducibility of short-term and reproduction toxicity experiments with Daphnia magna and comparison of the sensitivity of Daphnia magna with Daphnia pulex and Daphnia cucullata in short-term experiments. Hydrobiologia 59:135-140.

Species	Daphnia pulex
Exposure Period	48 h
LC_{50}	8800 mg/L
Analyt. Monitoring	no data
GLP	no data
Test substance	no data
Remark	Tests conducted according to a protocol from the Dutch Standard Institute (Adema, 1978).
Reference	Canton, J.H. and Adema, D.M.M. (1978). Repro-ducibility of short-term and reproduction toxicity experiments with Daphnia magna and comparison of the sensitivity of Daphnia magna with Daphnia pulex and Daphnia cucullata in short-term experiments. Hydrobiologia 59:135-140.

Species	Daphnia cucullata
Exposure Period	48 h
LC_{50}	7635 mg/L
Analyt. Monitoring	no data
GLP	no data
Test substance	no data
Remark	Tests conducted according to a protocol from the Dutch Standard Institute (Adema, 1978).
Reference	Canton, J.H. and Adema, D.M.M. (1978). Repro-ducibility of short-term and reproduction toxicity experiments with Daphnia magna and comparison of the sensitivity of Daphnia magna

	with Daphnia pulex and Daphnia cucullata in short-term experiments. Hydrobiologia 59:135-140.
Species	Daphnia magna
Exposure Period	48 h
LC_{50}	13,500 mg/L
Analyt. Monitoring	no data
GLP	no data
Test substance	no data
Remark	Procedure used individuals 12-hours old. The test water was from a local spring-fed pond with an average hard-ness 154.5 mg/L, pH of 7.7, and temperature of 22°C.
Reference	Randall, T.L. and Knopp, P.V. (1980). Detoxification of specific organic substances by wet oxidation. J. Water Pollut. Control Fed. 52:2117-2130.
Species	Daphnia magna
Exposure Period	24 h
LC_{50}	>10,000 mg/L
Analyt. Monitoring	no data
GLP	no data
Test substance	no data
Remark	Procedure used individuals 24-hours old. Test used tap water free of chlorine, saturated with oxygen, hardness 16 (German), pH 7.6-7.7, temperature 20-22°C.
Reference	Bringmann, V.G. and Kuhn, R. (1977). Results of the damaging effect of water pollutants on Daphnia magna. Z. Wasser Abwasser Forsch. 10:161-166.
Species	Daphnia pulex
Exposure Period	18 h
LC_{50}	1550 mg/L
Analyt. Monitoring	no data
GLP	no data
Test substance	no data
Remark	Test containers selected for compatibility with the size of the test organism. Duplicate test chambers with 10-12 concentrations. Test duration was 18 hours of which 16 hours were fluorescent illumination. Water temperature 23°C plus or minus 2°C. No supplemental food or air.
Reference	Bowman, M.C., Oller, W.L., and Cairns, T. (1981). Stressed bioassay systems for rapid screening of pesticide residues. Part I: Evaluation of bioassay systems. Arch. Environ. Contam. Toxicol. 10:9-24.
Species	Culex restuans (white-dotted mosquito)
Exposure Period	18 h
LC_{50}	7840 mg/L
Analyt. Monitoring	no data
GLP	no data

Test substance	no data
Remark	Test containers selected for compatibility with the size of the test organism. Duplicate test chambers with 10-12 concentrations. Test duration was 18 hours of which 16 hours were fluorescent illumination. Water temperature was 23°C plus or minus 2°C. No food or air added.
Reference	Bowman, M.C., Oller, W.L., and Cairns, T. (1981). Stressed bioassay systems for rapid screening of pesticide residues. Part I: Evaluation of bioassays systems. Arch. Environ. Contam. Toxicol. 10:9-24.

Species	Hyalella azteca
Exposure Period	18 h
LC_{50}	3520 mg/L
Analyt. Monitoring	no data
GLP	no data
Test substance	no data
Remark	Test containers selected for compatibility with the size of the test organism. Duplicate test chambers with 10-12 concentrations. Test duration was 18 hours of which 16 hours were fluorescent illumination. Water temperature was 23°C plus or minus 2°C. No food or air added.
Reference	Bowman, M.C., Oller, W.L., and Cairns, T. (1981). Stressed bioassay systems for rapid screening of pesticide residues. Part I: Evaluation of bioassay systems. Arch. Environ. Contam. Toxicol. 10:9-24.

Species	Lithodes antarcticus (southern king crab, larval stage)
Exposure Period	120-192 h.
EC_{50}	1010-4660 mg/L
Analyt. Monitoring	no data
GLP	no data
Test substance	as prescribed by 1.1-1.4
Test method	American Public Health Association for Static Bioassay Procedures (APHA, AWWA, WPCF) 1976.
Remark	The mortality curve of larvae exposed to 7500 mg/L acetone (acetone controls) did not differ from that of seawater controls.
Result	Results as LC_{50} in mg/L are as follows: 120-h: 4660 144-h: 3880 168-h: 2330 192-h: 1010
Reference	Lombardo, R.J., Ferrari, L., and Vinuesa, J.H. (1991). Effects of lindane and acetone on the development of larvae of the southern King Crab (Lithodes antarcticus). Bull. Environ. Contam. Toxicol. 46:185-192.

Species	Streptocephalus rubricaudatus
Exposure Period	24 h
LC_{50}	64,300 mg/L

Analyt. Monitoring	no data
GLP	no data
Test substance	no data
Remark	The hatching and 24-h toxicity test procedure used dry-stored cysts of S. rubricaudatus (originating from Algeria). Hatching was obtained by hydrating dried cysts in a petri dish in U.S. EPA freshwater medium (1985). After 18 hours incubation (at 25°C), the free-swimming larvae were pipet-transferred into a second petri dish for a supplemental period of 6 h. The test endpoint was death, defined by the complete lack of movement during 10 seconds of observation under a dissection microscope.
Reference	Crisinel, A., Delaunay, L., Rossel, D., and Tanadellas, J. (1994). Cyst-based ecotoxicological tests using Anostracans: comparison of two species of Streptocephalus. Environ. Toxicol. Water Qual. 9:317-326.

Species	Daphnia magna
Exposure Period	48 h
LC_{50}	104,712 mol/L
Analyt. Monitoring	no data
GLP	no data
Test substance	no data
Remark	Age of test organism was less than 2 days; number of test organisms per group was 25; test volume was 1 L; temperature was 22°C plus or minus 1°C; hardness was approximately equal to one.
Reference	Hermens, J., Cantor, H., Janssen, P., and DeJong, R. (1984). Quantitative structure-activity relationships and toxicity studies of mixtures of chemicals with anesthetic potency: acute lethal and sublethal toxicity to Daphnia magna. Aquatic Toxicol. 5:143-154.

4.3 Toxicity to Aquatic Plants e.g. Algae

Species	Chlorella pyrenoidosa
Endpoint	see below
Analyt. Monitoring	no data
GLP	no data
Test substance	no data
Remark	Also tested was the green algae, Scenedesmus quadricauda. Photosynthesis was used as the test criterion and was quantified by monitoring the uptake of $^{14}CO_2$ from $NaH^{14}CO_3$, as previously described by Stratton et al. (1980). Acetone alone was not inhibitory to either S. quadricauda or C. pyrenoidosa. Photosynthetic activity in these species was stimulated above 0.2% acetone while stimulatory activity increased 30-40% at an acetone concentration of 1.0%.
Method	Method similar to: Stratton, G.W. et al. (1980). Bull. Environ. Contam. Toxicol. 24:562.

Reference	Stratton, G.W. and Corke, C.T. (1981). Interactions between acetone and two pesticides toward unicellular green algae. Bull. Environ. Contam. Toxicol. 27:13-16.
Species	Chlorella pyrenoidosa
Endpoint	growth rate
Exposure Period	14 day
EC_{50}	3020 mg/L
Analyt. Monitoring	no data
GLP	no data
Test substance	no data
Exposure Period	10-14 days.
Remark	Growth was monitored by following the increase in optical density over time for 10-14 days using a spectrophotometer equipped with a universal test tube adapter and appropriate filters. Effects of acetone were assayed against the growth of C. pyrenoidosa at five to ten concentrations ranging from 0.1% to 6.0%.
Reference	Stratton, W.S. and Smith, T.M. (1988). Interaction of organic solvents with the green alga Chlorella pyrenoidosa. Bull. Environ. Contam. Toxicol. 40:736-742.
Species	Chlorella pyrenoidosa
Endpoint	Effects on membrane integrity and cell leakage
Analyt. Monitoring	no data
GLP	no data
Test substance	no data
Remark	Acetone-induced leakage from C. pyrenoidosa was monitored by following the loss of carbon compounds from cells using radioisotopic techniques. The cells were radiolabeled photosynthetically using ^{14}C-sodium bicarbonate. Significant leakage occurred at 1.5% and lower (depending on the exposure period (i.e., 24, 48, or 96 h).
eference	Stratton, G.W. (1989). Effect of the solvent acetone on membrane integrity in the green alga, Chlorella pyrenoidosa. Bull. Environ. Contam. Toxicol. 42:754-760.
Species	Anabaena inaequalis
Endpoint	photosynthetic ability
Analyt. Monitoring	no data
GLP	no data
Test substance	no data
Method	Cells were incubated for 2 h and harvested by filtration through 0.45 m membrane filters. Photosynthetic changes were noted by monitoring the uptake of $^{14}CO_2$ from $NaH^{14}CO_3$. The amount of radioactivity incorpor-ated into the cells was determined using a liquid scintillation counter. Percent inhibition was calculated. Anabaena cylindrica and Anabaena variabilis also examined.
Remark	A. inaequalis photosynthetic activity was significantly altered at acetone concentrations of 1000 mg/L and 4000 mg/L, where

	stimulation was observed. A. variabilis photosynthesis was significantly stimulated by acetone concentrations below 10,000 mg/L. No significant stimulation of $^{14}CO_2$ uptake occurred with A. cylindrica, although inhibition was observed above 6000 mg/L acetone. Inhibition was 75% at 8000 mg/L and 95% at 10,000 mg/L.
Reference	Stratton, G.W., Burrell, R.E., Krup, M.L., and Corke, C.T. (1980). Interactions between the solvent acetone and pyrethroid insecticide permethrin on activities of the blue-green alga Anabaena. Bull. Environ. Contam. Toxicol. 24:562-569.
Species	Anabaena inaequalis
Endpoint	nitrogen fixation ability
Analyt. Monitoring	no data
GLP	no data
Test substance	no data
Method	Assayed using the acetylene reduction technique. After the addition of a 10% atmosphere of acetylene, the cells were incubated for 5 h and the ethylene produced was assayed by gas chromatography. A. variabilis was not included in these studies due to its inability to fix nitrogen. Anabaena cylindrica and Anabaena variabilis were also examined
Remark	A. inaequalis activity was stimulated by all acetone concentrations from 1000 mg/L to 10,000 mg/L. The degree of stimulation was greater than that observed in photosynthetic studies. A. cylindrica exhibited significantly increased acetylene reduction at levels of acetone less than 4000 mg/L and decreased significantly at levels greater than 5000 mg/L.
Reference	Stratton, G.W., Burrell, R.E., Krup, M.L., and Corke, C.T. (1980). Interactions between the solvent acetone and pyrethroid insecticide permethrin on activities of the blue-green alga Anabaena. Bull. Environ. Contam. Toxicol. 24:562-569.
Species	Skeletonema costatum
Endpoint	growth sensitivity
Analyt. Monitoring	no data
Year	1988
GLP	no data
Test substance	no data
Remark	S. costatum was cultured in growth medium to achieve the selected density of 100,000 cells/mL. Total cell count and total cell volume were measured by use of a Coulter counter.
Result	Classified as practically nontoxic (> 100 mg/L).
Reference	Cowgill, U.M., Milazzo, D.P., and Landenberger, B.D. (1989). Toxicity of nine benchmark chemicals to Skeletonema costatum, a marine diatom. Environ. Toxicol. Chem. 8:451-455.
Species	Scenedesmus quadricauda

Endpoint	toxicity threshold
Analyt. Monitoring	no data
GLP	no data
Test substance	no data
Remark	Additional Species tested was Microcystis aeruginosa. Test cultures prepared from the dilution series and the control cultures were kept under standardized conditions for 8 days with constant lighting at 27 °C. Cultures were shaken daily and the concentration of the algal suspen-sions of each test culture was measured turbidimetrically.
Result	The chemical concentration causing the onset of cell multiplication inhibition was defined as the toxicity threshold. The toxicity threshold was 7500 mg/L for S. quadricauda and 530 mg/L for M. aeruginosa.
Reference	Bringmann, G. and Kuhn, R. (1978). Testing of substances for their toxicity threshold: model organisms Microcystis (Diplocystis) aeruginosa and Scenedesmus quadricauda. Mitt. Internat. Verein. Limnol. 21:275-284.

4.4 Toxicity to Bacteria

Type	aquatic
Species	Paramaecium caudatum
Exposure Period	4 h
LC_{50}	6800 mg/L
Analyt. Monitoring	no data
GLP	no data
Test substance	no data
Remark	Method described in: Stressed bioassay systems for rapid screening of pesticide residues. I. Evaluation of bioassay systems. Environ. Contam. Toxicol. 10:9-24. (1981).
Reference	Rajini, P.S., Krishnakumare, M.K., and Majunder, S.K. (1989). Cytotoxicity of certain organic solvents and organophosphorus insecticides to the ciliated protozoan Paramecium caudatum. Microbios 59:157-163.

Type	other
Species	Uronema parduzci
Endpoint	toxicity threshold
Exposure Period	20 h
Analyt. Monitoring	no data
GLP	no data
Test substance	no data
Remark	The protozoan test Species was fed with pure inactive cultures of E. coli to avoid metabolism of the test article by the bacteria. The test period for determination of a toxicity threshold was 20 h. Quantification of bacteria (food) and protozoa (test species) was done by cell counter. A 5% difference in protozoan cell count between test article and control was used to determine the toxicity threshold.
Result	Result is given as a toxicity threshold of 1710 mg/L.

Reference	Bringmann, G. and Kuhn, R. (1980). Determination of the harmful effect of water pollutants on protozoa. II. Bacteriovorous ciliates. Z. Wasser Abwasser Forsch. 13:26-31.
Type	other
Species	Chilomonas paramecium
Endpoint	toxicity threshold
Exposure Period	48 h
Analyt. Monitoring	no data
GLP	no data
Test substance	no data
Remark	The flagellate saprozoic protozoan test species was fed pure inactive cultures of E. coli to avoid metabolism of the test article by the bacteria. The test period for determination of a toxicity threshold was 48 h. Quantification of bacteria(food) and protozoa (test species) was by electronic cell counter. A 5% difference in protozoan cell count between test Species and controls was used to determine the toxicity threshold.
Result	Result is reported as a toxicity threshold of 3516 mg/L.
Reference	Bringmann, G. and Kuhn, R. (1980). Determination of biological damage from water pollutants to protozoa. III. Saprozoic flagellates. Z. Wasser Abwasser Forsch. 13:170-173.
Type	other
Species	Entosiphon sulcatum
Exposure Period	72 h
Analyt. Monitoring	no data
GLP	no data
Test substance	no data
Remark	The protozoan test Species was fed pure inactive cultures of E. coli to avoid metabolism of the test article by the bacteria. The test period for determination of a toxicity threshold was 72 h. Quantification of bacteria (food) and flagellates (test species) was performed by electronic cell counter. A 5% difference in protozoan cell count between test species and controls was used to determine the toxicity threshold.
Result	Result is reported as a toxicity threshold of 28 mg/L.
Reference	Bringmann, G. and Kuhn, R. (1978). Determination of the biological toxicity of water-bound substances towards protozoa. I. Bacteriovorous flagellates (model organism: Entosiphon sulcatum). Z. Wasser Abwasser Forsch. 11:210-215.
Type	aquatic
Species	Pseudomonas putida
Endpoint	oxygen uptake
Analyt. Monitoring	no data
GLP	no data
Test substance	as prescribed by 1.1-1.4

Remark	Oxygen uptake was measured over a 10-min. period at 27°C before, during, and after sample addition. Growth was determined by inoculating P. putida into medical flats and incubating at 27°C. Thirty minutes before inoculation with acetone, the test cultures were diluted with fresh medium to a density with an absorption of approximately 0.8 at 600 m measured spectrophotometrically. The test solutions were redistributed to medical flats, acetone added, and incubated for 6 hours at 27°C. Growth was terminated by formalin addition and immediately followed by density measurements.
Result	Oxygen uptake over 10 min (EC_{10}) was 1380 mg/L. Growth inhibition over 7 h (EC_{10}) was 540 mg/L.
Reference	Slabbert, J.L. and Grabow, W.O.K. (1986). A rapid water toxicity screening test based on oxygen uptake of Pseudomonas putida. Toxicity Assess. 1:13-26.

Type	aquatic
Species	Escherichia coli
Endpoint	minimal inhibitory concentrations (MIC)
Analyt. Monitoring	no data
GLP	no data
Test substance	no data
Remark	Test Species was a mutant strain with enhanced sensitivity to a wide spectrum of toxic substances. The assay is based on the ability of toxicants to inhibit the de novo synthesis of an inducible enzyme, e.g., β-galactosidase, by a rough mutant of E. coli, which is highly sensitive to a wide spectrum of toxic substances.
Result	The minimal inhibitory concentration (MIC) was 25,000 mg/L (defined as the concentration causing 20% toxicity).
Reference	Reinhartz, A., Lampert, I., Herzberg, M., and Fish, F. (1987). A new short-term sensitive bacterial assay kit for the detection of toxicants. Toxicity Assess. 2:193-206.

Type	aquatic
Species	Polytox (proprietary blend of 12 aerobic bacteria strains)
Exposure Period	6 h
IC_{50}	48,000 mg/L
Analyt. Monitoring	no data
GLP	no data
Test substance	no data
Remark	The percent inhibition at different concentrations of acetone was based on the reduction in oxygen uptake rate of spiked reactors compared to that of the control reactor. Plotted against the respective concentrations, the concentration causing 50% inhibition or IC_{50} was determined.
Reference	Nirmalakhandan, N., Arulgnanendran, V., Mohsin, M., Sun, B., and Cadena, F. (1994). Toxicity of mixtures or organic chemicals to microorganisms. Water Res. 28:543-551.

Type	aquatic
Species	activated sludge of a predominantly domestic sewage
EC_{50}	77.4 mg/L
Analyt. Monitoring	no data
Method	ISO 8192
Year	1991
GLP	no data
Test substance	as prescribed by 1.1-1.4
Remark	Activated sludge of a predominantly industrial sewage was also tested.
Result	EC_{50} for the industrial/synthetic sewage was 59.4 mg/L.
Reference	Kilroy, A.C. and Gray, N.F. (1992). The toxicity of four organic solvents commonly used in the pharmaceutical industry to activated sludge. Water Res. 26:887-892.

Type	aquatic
Species	activated sludge
Exposure Period	16 h
EC_{50}	>5000 mg/L
Analyt. Monitoring	no data
Method	OECD Guideline 209
GLP	no data
Test substance	no data
Reference	Alsop, G.M., Waggy, G.T., and Conway, R.A. (1980). Bacterial growth inhibition test. J. Water Pollut. Control Fed. 52:2452-2456.

Type	aquatic
Species	activated sludge of a predominantly domestic sewage
Exposure Period	3 h
EC_{50}	>1000
Analyt. Monitoring	no data
Method	OECD Guideline 209
GLP	no data
Test substance	as prescribed by 1.1-1.4
Reference	Klecka, G.M. and Landi, L.P. (1985). Evaluation of the OECD activated sludge respiration inhibition test. Chemosphere 14:1239-1251.

4.5.2 Chronic Toxicity to Aquatic Invertebrates

Species	Lithodes antarcticus
Endpoint	mortality
Exposure	7 day
EC_{50}	>0.75 g/L
Analyt. Monitoring	no data
GLP	no data
Test substance	as prescribed by 1.1-1.4
Method	The experiments were conducted following the recommendations of the APHA, AWWA, WPCF Standard Methods for the examination of water and wastewater, 14th

	ed., Am. Pub. Health Assoc., Washington, D.C. 1976, i.e. 7-day, 48-h static renewal. 8°C and 35 parts per thousand salinity.
Remark	Mortality in the seawater controls was lower than 10% during the first seven days of culture and the acetone controls (0.75 g/L) did not show mortality above that of the seawater controls during this period.
Reference	Lombardo, R.J., Ferrari, L., and Vinuesa, J.H. (1991). Effects of lindane and acetone on the development of larvae of the Southern King Crab (Lithodes antarcticus Jaquinot). Bull. Environ. Contam. Toxicol. 46:185-192.

4.6 Toxicity to Terrestrial Organisms

4.6.2 Toxicity to Terrestrial Plants

Species	Raphanus sativus L. var. Champion 708 (radish)
Endpoint	emergence and growth
Exposure Period	7 day
NOEC	100 mg/L
GLP	no
Test substance	no data
Remark	Also tested were Lactuca sativa L. var. 525 Ithaca M.T.O. (lettuce) and Lolium perenne L. var. Manhattan (rye grass).
Method	The bioassay was most similar to the blotter-sandwich technique, and was designed to determine the dose-response characteristics of acetone on the germination and early growth of three representative terrestrial plants during a 7-day exposure period.
Result	7-day NOEC for all three Species was 100 mg/L.
Reference	Gorsuch, J.W., Kringle, R.O., and Robillard, K.A. Chemical effects on the germination and early growth of terrestrial plants (1990). In: Plants for Toxicity Assessment, ASTM STP 1091. W. Wang, J.W. Gorsuch, and W.R. Lower, eds., pp. 49-58. American Society for Testing and Materials. Philadelphia, PA.

Species	Zea mays L. var. rugosa Bouaf
Endpoint	Total germination and percentage of normal seedlings
Exposure Period	5 sec., 0.25, 0.50, 1.0, 2.0, 4.0, or 8.0 h; immersion in 100% acetone.
GLP	no data
Test substance	as prescribed by 1.1-1.4
Method	The organic solvent infusion technique has been used successfully to improve germination.
Remark	Total germination and percentage of normal seedlings in both cultivars (Florida Staysweet and Crisp-n-Sweet 710) were significantly decreased after 8 h of immersion in acetone. Average seedling dry weight, however, did not decrease. Results indicate that acetone could be used as an infusion agent for fungicides in the seed of some sweet corn cultivars without compromising seed germination or vigor.

Reference	Hung, P.E. (1992). Infusion of shrunken-2 sweet corn seed with organic solvents: effects on germination and vigor. Horticult. Sci. 27:467-470.
Species	Cucumis sativus (long green cucumber)
Endpoint	active dormancy - breaking factor
Exposure Period	various
Year	1993
GLP	no data
Test substance	as prescribed by 1.1-1.4
Method	Dormant and non-dormant seeds were immersed in acetone in glass-stoppered containers at 10°C for various time periods. After treatment the seeds were allowed to air-dry for 24 h in open petri dishes and then used in germination experiments.
Remark	Acetone was found not only to break the dormancy in cucumber seeds, but also to prevent its induction by far-red light. The data also show that prevention of dorm-ancy development as well as breakage of dormancy by acetone are accompanied by a change in the permeability of the cell membrane of the perisperm-endosperm envelope around the embryo.
Reference	Amritphale, D., Dixit, S., and Singh, B. (1993). Effect of acetone on the induction and breakage of secondary dormancy in seeds of cucumber. J. Exp. Botany. 44:1621-1626.

4.6.3 Toxicity Non-Mammalian Terrestrial Species

Species	Coturnix coturnix japonica
Endpoint	mortality
Exposure Period	5 days
LC_{50}	>20,000 ppm
GLP	no data
Test substance	as prescribed by 1.1-1.4
Method	5-day dietary trial with 14-day old coturnix quail.
Remark	Total mortality was 0/45 at 5 days.
Reference	Hill, E. F. and Carmardese, M.B. (1986). Lethal dietary toxicities of environmental contaminants and pesticides to Coturnix. Patuxent Wildlife Research Center. Laurel, MD. pp. 22-23.

4.7 Biological Effects Monitoring

Remark	The bioaccumulation potential of a chemical in muscle tissue from rainbow trout has been shown to be related to the octanol water partition coefficient. The partition coefficient for acetone of -0.24 indicates a high degree of water solubility and low potential to bioaccumulate or biomagnify in the environment.
Reference	Paterson, S. and Mackay, D. (1989). Correlation of tissue, blood and air partition coefficients of volatile organic chemicals. Br. J. Ind. Med. 46:321-328.

Neely, W.B., Branson, D.R., and Blau, G.E. (1974). Partition coefficient to measure bioconcentration potential of organic chemicals in fish. Environ. Sci. Technol. 8:1113-1115.

4.8 Biotransformation and Kinetics

Type	plant
Remark	The objective of the experiment was to determine if acetone inhibits the mutagenic activity of promutagenic dimethylnitrosamine (DMN) and methylbutylnitrosamine in a higher plant, Arabidopsis thaliana. Seeds were immersed for 3 hours at 25°C in 1 mL of acetone mixed with buffer for pretreatment. They were then immersed for 3 hours at 25°C in 2 mL of the mixture containing the mutagens and acetone for treatment. Following treatment, the seeds were rinsed for 30 min in distilled water and sown on soil in a greenhouse.
Result	The frequency of mutations and the degree of sterility induced by DMN was markedly reduced in the presence of acetone.
Reference	Gichner, T. and Veleminsky, J. (1986). Organic solvents inhibit the mutagenicity of promutagens dimethyl-nitrosamine and methylbutylnitrosamine in a higher plant Arabidopsis thaliana. Mutagenesis 1:107-109.

Type	animal (Daphnia magna)
Remark	The hypothesis of constancy of the tissue residues in animals treated with narcotic organic chemicals was tested by determining the effect of body length, time, and ambient concentration on tissue concentration in Daphnia magna narcotized by exposure to toxic levels of acetone.
Result	The lower than expected toxicity of acetone may be due to the degradation of this chemical by Daphnia. Acetone, a simple organic compound, may be readily metabolized by Daphnia. As a result, some of the radioactivity in Daphnia tissues would be associated with accumulated metabolites rather than the original compound, and the narcotizing body burdens of acetone would be over-estimated. Acetone did not exert a significant negative influence on the effective internal concentration. When predicted body burdens for acetone were calculated using mean body sizes, exposure concentrations, and exposure durations, body burden acetone residues of 115 mmole/kg were more than an order of magnitude from the overall mean for all narcotics tested.
Reference	Pawlisz, A.V. and Peters, R.H. (1993). A test of the equipotency of internal burdens of nine narcotic chemicals using Daphnia magna. Environ. Sci. Technol. 27:2801-2806.

Type	other
Remark	This paper reports the results of a research program concerned with the analyses and explanation of differences in sensitivity of species to toxic substances using biological properties of the species. The project aims at the development of predictive

	models, which, in analogy to QSARs, are called Quantitative Species Sensitivity Relationships. The distributions of acute toxicity data of different Species were studied for 26 chemicals.
Result	Chemicals with a specific mode of action have large sensitivity ratios whereas inert chemicals with lower toxicity have lower ratios. Acetone had the lowest ratio of all twenty-six chemicals studies.
Reference	Hoekstra, J.A., Vaal, M.A., Notenboom, J., and Sloof, W. (1994). Variations in the sensitivity of aquatic species to toxicants. Bull. Environ. Contam. Toxicol. 53:98-105.
Type	plant (various species)
Remark	This paper describes experiments conducted to test the effects of volatiles including (acetone) on seed deterioration during seed storage. Seeds tested were lettuce, soybean, sunflower, carrot, and rice. It has been shown that the yields of volatiles such as acetone in soybean seeds increase during seed development and decrease to trace levels after reaching yellow maturation. The authors showed in a preliminary study that the evolution of volatiles, such as acetone, is a widespread phenomenon occurring in stored seeds. Many types of dry seeds that were tested continued to evolve volatiles and accumulate them during storage. Acetone was found to have only slight deleterious effects on some species.
Reference	Zhang, M., Maeda, Y., Furihata, Y., Nakamaru, Y., and Esashi, Y. (1994). A mechanism of seed deterioration in relation to the volatile compounds evolved by dry seeds themselves. Seed Sci. Res. 4:49-56.
Type	aquatic (Daphnia magna)
Remark	This work examines the hypothesis that exposure of Daphnia magna to sublethal levels of narcotic contam-inants including acetone may affect subsequent sensitivity of animals. Prior exposure (24 h) of Daphnia to sublethal levels of acetone had no effect on their sensitivity to effective levels of these chemicals. Effective burdens (24-h acute exposure) were independent of the sublethal body burdens (24-h sublethal exposure) and of the sublethal water concentrations ($p < 0.025$). These results imply that animals from polluted sites should be no more resistant to high body residues of pollutants than those from clean sites and that the toxicity of narcotic organic compounds like acetone may be independent of the time course of uptake.
Reference	Pawlisz, A.V. and Peters, R.H. (1995). Effects of sublethal exposure on lethal body burdens of narcotic organic chemicals in Daphnia magna. Environ. Sci. Technol. 29:613-621.

4.9 Additional Reports

Remark	The objective of this paper is to compare the usefulness of a representative of the Urodela (Ambystoma mexicanum) and Anura (Xenopus laevis) species as biological indicators in toxicological bioassays. Toxicity test conditions were as follows: static, 1-L size, 20°C plus or minus 1°C, circadian light and dark schedule, 48-h exposure for acetone. The 48-h LC_{50} for A. mexicanum was 20,000 mg/L and the over 48-h LC_{50} for A. laevis was 24,000 mg/L.
Reference	Sloaff, W. and Baesselman, R. (1980). Comparison of the usefulness of Mexican Axolotl (Ambystoma mexicanum) and the clawed toad (Xenopus laevis) in toxicological bioassays. Bull. Environ. Contam. Toxicol. 24:439-443.
Remark	The effects of acetone on the growth of four fungi were determined to be as follows: EC_{50} for Polyporous hirsutus was greater than 2.0%, Pestalotia sp. was 1.25%, Sclerotinia homeocarpa was 0.88%, and Fusarium oxysporum was 1.8%. It was concluded that acetone was a moderately fungitoxic compound, but the specific mode of action was not elucidated.
Reference	Burrell, R.E. and Corke, C.T. (1980). Interactions of the solvent acetone with the fungicides benomyl and captan in fungal assays. Bull. Environ. Contam. Toxicol. 25:554-561.
Remark	This paper provides the 96-h TL_m (50% survival) for Lepomis macrochirus (bluegill sunfish) of 8300 ppm and the 120-h TL_m (50% reduction in number of cells produced) for the diatom Nitzschia linearis (widely distributed in unpolluted soft fresh waters of the U.S.) of 11,493-11,727 ppm acetone.
Reference	Patrick, R., Cairns, J., and Scheir, A. (1968). The relative sensitivity of diatoms, snails, and fish to twenty common constituents of industrial wastes. Progressive Fish Culturist 30:137-140.
Remark	Acetone is often used as a carrier solvent in aquatic bioassays at 100 ppm without affecting the evaluation of the test article. This paper provides comparative chronic data for Daphnia magna and Pimephales promelas. Endpoints evaluated include: survival of adults, number of young per adult, primiparous instar, days to primiparous instar, and total number of broods for the daphnid. Fish endpoints included: embryo survival, hatching rate, larval survival, length and weight. Differences between the solvent control (acetone and dilution water) and control dilution water were minimal.
Reference	McCarthy, J.F. and Whitmore, D.K. (1985). Chronic toxicity of di-n-butyl and di-n-octyl phthalate to Daphnia magna and the fathead minnow. Environ. Toxicol. Chem. 4:167-179.
Remark	Static acute and flow-through toxicity tests were performed with Daphnia magna. The 48-h LC_{50} value for acetone was 39,000 µL/L. The maximum acceptable toxicant concentrations determined during the chronic toxicity test with

acetone were between 1400 and 2800 µL/L. Acetone was sufficiently low in toxicity to suggest that the recommended usage limits for acetone as a co-solvent (500 µL/L during acute toxicity tests; 100 µL/L during chronic toxicity tests).

Reference LeBlanc, G.A. and Surprenant, D.C. (1983). The acute and chronic toxicity of acetone, dimethylformamide, and triethylene glycol to Daphnia magna (Straus). Arch. Environ. Contam. Toxicol. 12:305-310.

Remark A multi-species test procedure was used to measure the acute aquatic effects of acetone on seven aquatic species simultaneously: Asellus intermedius (pillbug), Daphnia magna (water flea), Dugesia tigrina (flatworm), Gammarus fasciatus (sideswimmer), Helisoma trivolvis (snail), Lumbriculus variegatus (segmented worm) and Pimephales promelas (fathead minnow). These species were chosen because of their ecological importance diversity, and amenability to laboratory culturing. The 96-h static LC_{50} for all species was > 100 mg/L.

Reference Ewell, W.S., Gorsuch, J.W., Kringle, R.O., Robillard, K.A., and Spiegel, R.C. (1986). Simultaneous evolution of the acute effects of chemicals on seven aquatic species. Environ. Toxicol. Chem. 5:831-840.

Remark The test species was Xenopus laevis and the endpoint was the minimum concentration inhibiting growth. The method was the frog embryo teratogenesis assay Xenopus (FETAX), as described by Damont et al. (1983). The 96-h bioassay determines the relative teratogenic potential. The purpose of this experiment was to determine whether carrier solvents interacted with the teratogens t-retinoic acid and 6-aminonicotinamide to affect survival, development, and growth of Xenopus embryos.

Result The 96-h minimum concentrations that inhibited growth were: 18,000 mg/L for trial 1, 15,000 mg/L for trial 2, and 10,000 mg/L for trial 3.

Reference Rayburn, J.R. Fort, D.J., McNew, R., and Bantel, J.A. (1991). Synergism and antagonism induced by three carrier solvents with t-retinoic acid and 6-aminonico-tinamide using FETAX. Bull Environ. Contam. Toxicol. 46:625-632.

Remark The test species was Xenopus laevis and the endpoint was the reproduction rate for 12 weeks post-hatch at 0.10% acetone. The method uses groups of eggs that were put either in 800-mL jars or 3-L glass containers and maintained in aerated tap water at 22°C (plus or minus 1°C) under 16-h photoperiod conditions. According to the volume of water the eggs were reared in groups of 10 or 25. After hatching, tadpoles were fed Infusyl tablets. Each jar or tank was covered with a glass plate in order to limit evaporation. Water was changed weekly. Daily monitoring of egg and tadpole mortality was conducted

	throughout the first week of treatment. The metamorphosis pattern was investigated on surviving tadpoles.
Result	Growth by weight and development were slightly delayed in animals at the beginning of treatment (premetamor-phosis). After metamorphosis, the weight of juvenile Xenopus was higher than that of the water controls. It was speculated that acetone might first delay develop-ment; then because of feeding habits or other reasons, tadpoles could regain normal weight gain and even show a tendency for increased growth.
Reference	Marchal-Segault, D. and Tamade, F. (1981). The effects of lindane, an insecticide, on hatching and postembryonic development of Xenopus laevis (Daudin) Anauran Amphibian. Environ. Res. 24:250-258.

5. Toxicity

5.1 Acute Toxicity

5.1.1 Acute Oral Toxicity

Type	LD_{50}
Species	rat
Value	ca. 5800 mg/kg
GLP	no data
Test substance	no data
Reference	Freeman, J.J. and Hayes, E.P. (1985). Acetone potentiation of acute acetonitrile toxicity in rats. J. Toxicol. Environ. Health 15:609-621.

Type	LD_{50}
Species	rat
Value	ca. 8400 mg/kg
GLP	no
Test substance	no data
Reference	Smyth, H.F., Carpenter, C.P., Weil, C.S., Pozzani, U.C., and Striegel, J.A. (1962). Range-finding toxicity data: List VI. Am. Ind. Hyg. Assoc. J. 23:95-107.

Type	LD_{50}
Species	rat
GLP	no
Test Substance	analytical grade acetone (ACS specifications).
Remark	Groups of 6-12 male and female Sprague-Dawley rats of various ages were intubated with neat acetone. They were observed for 1 week. LD_{50} values in g/kg (95% confidence limits) were: newborn, 1.7 (1.3-3.0), 14-day-old, 4.4 (3.1-6.3), young adults [80-160 g], 7.2 (5.4-9.5), older adults [300-470 g], 6.7 (6.1-7.3).
Reference	Kimura, E.T., Ebert, D.M., and Dodge, P.W. (1971). Acute toxicity and limits of solvent residue for sixteen organic solvents. Toxicol. Appl. Pharmacol. 19:699-704.

Type	LD_{50}
Species	mouse
Value	ca. 5250 mg/kg
GLP	no data
Test substance	no data
Remark	Male ddY mice weighing 24-27 g were intubated with acetone following ip injection of 0.16 mL of olive oil/g. LD_{50} value of 5250 mg/kg was reported with a 95% confidence range of 3580-7700 mg/kg.
Reference	Tanii, H., Tsuji, H., and Hashimoto, K. (1986). Structure-toxicity relationship of monoketones. Toxicol. Lett. 30:13-17.

5.1.2 Acute Inhalation Toxicity

Type	LC_0
Species	rat
Exposure Time	30 minute
Value	16,000 ppm
GLP	no
Test substance	no data
Remark	Female rats were exposed (whole body exposure) to acetone at nominal air concentrations of the following: 6/6 rats died at 32,000 ppm; 1/6 animals exposed to 16,000 ppm acetone for 4 hours also died.
Reference	Smyth, H.F., Carpenter, C.P., Weil, C.S., Pozzani, U.C., and Striegel J.A. (1962). Range-finding toxicity data: List VI. Am. Ind. Hyg. Assoc. J. 23:95-107.

Type	LC_{50}
Species	rat
GLP	no
Test substance	no data
Remark	LC_{50} values with 95% confidence intervals for 4-hr and 8-hr exposures were 32,000 ppm (27,400-37,200) and 21,000 ppm (17,900-24,800). Exposure was to female Carworth Farms-Nelson rats.
Reference	Pozzani, U.C., Weil, C.S., and Carpenter, C.P. (1959). 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. Am. Ind. Hyg. Assoc. J. 20:364-369.

5.1.3 Acute Dermal Toxicity

Type	LD_0
Species	rabbit
Value	>7400 mg/kg
GLP	no
Test substance	no data

Remark	Exposure time was 24 hours. Both sexes were used; skin was abraded. Test substance was "practical" grade.
Reference	Roudabush, R.L., Terhaar, C.J., Fassett, D.W., and Dziuba, S.P. (1965). Comparative acute effects of some chemicals on the skin of rabbits and guinea pigs. Toxicol. Appl. Pharmacol. 7:559-565.

Type	LD_0
Species	guinea pig
Value	> 7400 mg/kg
Method	other
GLP	no
Test substance	no data
Remark	Male Hartley-derived guinea pigs were used; abraded and intact skin was exposed for 24 h to a "practical" grade of acetone.
Reference	Roudabush, R.L., Terhaar, C.J., Fassett, D.W., and Dziuba, S.P. (1965). Comparative acute effects of some chemicals on the skin of rabbits and guinea pigs. Toxicol. Appl. Pharmacol. 7:559-565.

Type	LD_{50}
Species	rabbit
Value	>15,700 mg/kg
GLP	no
Test substance	no data
Remark	Exposure was for a 24-h period. The hair was completely clipped from the trunk of four male albino rabbits. The dose was injected under an impervious plastic film (method of Draize et al., J. Pharmacol. Exp. Therap. 82:377, 1944). Animals were observed for 14 days.
Reference	Smyth, H.F., Carpenter, C.P., Weil, C.S. (1962). Range-finding toxicity data: List VI. Am. Ind. Hyg. Assoc. J. 23:95-107.

5.2. Corrosiveness and Irritation

5.2.1 Skin Irritation

Species	rabbit
Result	not irritating
Classification	not irritating
GLP	no
Test substance	no data
Remark	Exposure time was 24 h. Acetone, 0.01 mL, was applied to the shaved stomach of 5 rabbits.
Reference	Smyth, H.F., Carpenter, C.P., and Weil, C.S. (1962). Range-finding toxicity data: List VI. Am. Ind. Hyg. Assoc. J. 23:95-107.

5.2.2 Eye Irritation

Species	rabbit
Result	highly irritating
Classification	irritating
GLP	no
Test substance	no data
Method	20 µL of acetone was added to the center of cornea and the eye was read 18-24 h later and scored after staining with fluorescein.
Results	The dose administered was 15.8 mg. Acetone was assigned a rating of Grade 5 in system with maximum of Grade 10. The 10-grade ordinal series is based upon the degree of corneal necrosis that results from instillation of various volumes and concentrations of a chemical. Grade 1 indicates at most a very small area of necrosis resulting from 0.5 mL of undiluted chemical in the eye. Grade 5 indicates a severe burn from 0.005 mL, and grade 10 indicates a severe burn from 0.5 mL of a 1% solution in water or propylene glycol.
Reference	Carpenter, C.P. and Smyth, H.F. (1946). Chemical burns of the rabbit cornea. Am. J. Ophthamol. 29:1363-1372. Smyth, H.F., Carpenter, C.P., and Weil, C.S. (1962). Range-finding toxicity data: List VI. Am. Ind. Hyg. Assoc. J. 23:95-107.

Species	rabbit
Result	highly irritating
Classification	irritating
Method	Draize Test
GLP	no data
Test substance	no data
Remark	0.1 mL of acetone was placed in the conjunctival sac and the eye was scored at 24 h. The data from this study indicate that corneal thickening is directly related to eye irritation and damage (r=0.86). Acetone eye swelling (215%) was rated as severe. Irritancy ratings for aqueous solutions were: 3, 10, and 30% acetone, mild irritation; 1% acetone, mild/slight irritation; corneal thickening ratings for 1, 3, 10, and 30% aqueous acetone solutions were all mild.
Reference	Kennah, H.E.. Hignet, S., Laux, P.E., Dorko, J.D., and Barrow, C.S. (1989). An objective procedure for quantifying eye irritation based upon changes of corneal thickness. Fund. Appl. Toxicol. 12:258-268.

5.3 Sensitization

Type	Mouse ear swelling test
Species	mouse
Result	not sensitizing
Classification	not sensitizing
GLP	no data

Test substance	no data
Method	Following removal of hair with clippers, mice are injected twice intradermally in the test area with Freund's complete adjuvant. The mice are tape stripped in the application area, and the chemical or solution (0.1 mL) is applied topically. Stripping and application of the Test substance is repeated on three additional consecutive days. Seven days later, 20 µL of test compound or solution is applied to the left ear and 20 µL of the vehicle (if any) is applied to the right ear. Twenty-four and 48-h later, the ear thicknesses are measured while the animals are under light ether anesthesia.
Remark	This test was reported to have correctly identified 48/49 known human sensitizers and 23/23 known human nonsensitizers. The missed compound was a weak human sensitizer. Acetone was also not a sensitizer in a modified MEST that used a patch-test procedure for the sensitization step.
Result	Acetone was not a sensitizer in a similar mouse ear sensitization test (Descotes, 1988) or in a modification of the guinea pig maximization test of Magnusson and Kligman (Nakamura et al., 1994).
Reference	Gad, S.C., Dunn, B.J., Dobbs, D.W., Reilly, C., and Walsh, R.D.(1986). Development and validation of an alternative dermal sensitization test: The mouse ear swelling test (MEST). Toxicol. Appl. Toxicol. 84:93-114. Descotes, J. (1988). Identification of contact allergens: The mouse ear sensitization assay. J. Toxicol. Cutaneous Ocular Toxicol. 7:262-272. Nakamura, A., Momma, J., Sekiguchi, H., Noda, T., Yamano, T., Kaniwa, M., Kojima, S., Tsuda, M., and Kurokawa, Y. (1994). A new protocol and criteria for quantitative determination of sensitization potencies of chemicals by guinea pig maximization test. Contact Dermatitis 31:72-85.

5.4 Repeated Dose Toxicity

Species	mouse
Strain	B6C3F1
Sex	male/female
Route of Administration	drinking water
Exposure Period	14 days and 13 weeks
Frequency of Treatment	ad libitum
Post Exposure Observation Period	none
Doses	14 days: 0.5, 1.0, 2.0, 5.0, and 10.0%; 5 mice/sex. 13-week females: 0.25, 0.5, 1.0, 2.0, and 5.0%; 10 mice each. 13-week males: 0.125, 0.25, 0.5, 1.0, and 2.0%; 10 mice each.
Control Group	Yes
Method	OECD Guideline 407 OECD Guideline 408 was used for the 13-week studies.

GLP	yes
Test substance	as prescribed by 1.1-1.4
Remark	NOEL: 1% (males: 14 days, 1579 mg/kg; 13 weeks, 2258 mg/kg; females: 14 days, 3023 mg/kg; 13 weeks, 4156 mg/kg.
Remark	LOEL: 2% (males: 14 days, 3896 mg/kg; 13 weeks, 4858 mg/kg; females: 14 days, 5481 mg/kg; 13 weeks, 5945 mg/kg.
Remark	Mice, 6-7 weeks old at start of the study, were housed individually. Drinking water containing acetone and NIH 07 feed were provided ad libitum. The time-weighted average dosages were: 14-day males, 965, 1579, 3896, 6348, 10,314 mg/kg; 14-day females, 1569, 3023, 5481, 8804, 12,725 mg/kg; 13-week males, 380, 611, 1353, 2258, 4858 mg/kg; 13-week females, 892, 2007, 4156, 5945, 11,298 mg/kg. Body weights were determined weekly and water consumption twice weekly. At necropsy, liver, right kidney, right testis, heart, thymus, brain, lungs, and, at 13 weeks only, spleen were taken for determination of weights and histopathology. Blood samples were obtained before the 13-week sacrifice for measurement of hematological indices. Male reproductive endpoints were assessed and stage and length of the estrous cycle were evaluated in females.
Result	Water consumption, and thus acetone dose, was reduced at acetone concentrations of 5% and above. There were no deaths during the studies. Body weight gain was depressed in mice given 10% acetone in the 14-day study only. There were no treatment-related clinical signs of toxicity. Absolute and relative liver weights in female mice only were significantly elevated in the 13-week 5% group; similar increases were seen in the 14-day animals. Hematological changes observed in the 13-week animals were increased hematocrit in 5% females ($p < 0.01$), increased hemoglobin in 2% ($p < 0.05$) and 5% ($p < 0.01$) females and 0.5, 1.0, and 2% males ($p < 0.05$). Histopathological alterations were seen only in mice during the 14-day studies; these included centrilobular hepatocellular hypertrophy in 5 of 5 male mice in each of the 2, 5, and 10% groups, 2 of 5 females in the 5% group, and 5 of 5 females in the 10% group. There were no changes in male or female reproductive indices.
Reference	Dietz, D.D., Leininger, J.R., Rauckman, E.J., Thompson, M.B., Chapin, R.E., Morrissey, R.L., and Levine, B.S. (1991). Toxicity studies of acetone administered in the drinking water of rodents. Fund. Appl. Toxicol. 17:347-360.

Species	rat
Strain	Fischer 344
Sex	male/female
Route of Administration	drinking water
Exposure Period	14 days and 13 weeks
Frequency of Treatment	ad libitum
Post Exposure	

Observation Period	none
Doses	14-day: 0.5, 1.0, 2.0, 5.0, 10%; 5/sex/dose level. 13-week: 0.25, 0.5, 1.0, 2.0, 5.0%; 10/sex/dose level.
Control Group	Yes
Method	OECD Guideline 407 OECD Guideline 408 was used for the 13-week studies.
GLP	yes
Test substance	as prescribed by 1.1-1.4
Remark	Rats, 6-7 weeks old at start of the study, were housed 5 per cage. Drinking water containing acetone and NIH 07 feed were provided ad libitum. The time-weighted average doses were: 14-day males, 714, 1616, 2559, 4312, and 6942 mg/kg; 14-day females, 751, 1485, 2328, 4350, 8560 mg/kg; 13-week males, 200, 400, 900, 1700, and 3400 mg/kg; 13-week females, 300, 600, 1200, 1600, and 3100 mg/kg. Body weights were determined weekly and water consumption twice weekly. At necropsy, liver, right kidney, right testis, heart, thymus, brain, lungs, and, at 13 weeks only, spleen were taken for determination of weights and histopathology. Blood samples were obtained before the 13-week sacrifice for measurement of hematological indices. Male reproductive endpoints were assessed, and stage and length of the estrous cycle were evaluated in females.
Remark	NOEL was 2% for 14-day (males: 2%, 2559 mg/kg; females: 5%, 4350 mg/kg); 1% for 13-week (males: 1%, 900 mg/kg; females: 5%, 3100 mg/kg). LOEL was 5% for 14-day (males: 5%, 4312 mg/kg; females: 10%, 8560 mg/kg); 2% for 13-week (males: 2%, 1700 mg/kg).
Result	No deaths were seen during the study. Water consumption, and thus the acetone dose, was reduced in rats given 5% or greater level of acetone. Body weights were depressed in male and female rats given 5 or 10% acetone in both the 14-day and 13-week studies. There were no treatment-related clinical toxic signs during the studies. During the 13-week study, relative kidney (both sexes), liver (both sexes), and testis weights were found in the 2 and 5% groups. Similar increases were reported to have occurred in the 14-day study at the same or lower doses (numbers not given). Hematological effects included mild lymphocytosis in male rats at 2% and male and males at 5%, decreased erythrocyte counts and hemoglobin levels at 2 and 5% and reticulocyte counts at 0.5% in male rats, and increased mean corpuscular hemoglobin and mean cell volume at 1% and higher in males and in 5% females. Platelet counts were mildly depressed in males and females in the 5% dose groups. Histopathologic lesions included bone marrow hypoplasia in 5 of 5 male rats given 10% acetone in the 14-day study. Dose-related increases in the incidence and severity of nephropathy, similar to that seen in aging rats, were seen in male rats. Minimal-to-mild splenic pigmentation was seen in male rats at the 2% and 5% dose levels in the 13-week studies. Acetone exposure of male rats for 13 weeks resulted in

	depressed sperm motility, cauda epididymal weight, and epididymal weight and an increased incidence of abnormal sperm. There was no indication of changes in vaginal cytology suggestive of changes in the estrous cycle.
Reference	Dietz, D.D., Leininger, J.R., Rauckman, E.J., Thompson, M.B., Chapin, R.E., Morrissey, R.L., and Levine, B.S. (1991). Toxicity studies of acetone administered in the drinking water of rodents. Fund. Appl. Toxicol. 17:347-360.
Species	rat
Strain	Sprague-Dawley
Sex	male/female
Route of Administration	gavage
Exposure Period	93, 94, or 95 days (interim sacrifice at 46 or 47 days)
Frequency of Treatment	once/day
Post Exposure Observation Period	1 day
Doses	100, 500, 2500 mg/kg; 30 M/30 F per dose levelControl Groupyes
Method	OECD Guideline 408
GLP	yes
Test substance	as prescribed by 1.1-1.4
Remark	Thirty male and 30 female 31-day-old rats were housed individually. Animals were dosed once/day by oral gavage with solutions of 0, 1.0, 5.0, or 25% acetone in reagent grade water. Dosing volumes were adjusted weekly for body-weight changes. Animals were dosed for 46-47 days (interim sacrifice) or 93-95 days (final sacrifice). Retroorbital blood samples and urine were collected prior to interim sacrifice of 10 males and 10 females from each group at 46-47 days and 20 males and 20 females from each group at 94-96 days (one day after end of dosing period). Ophthalmic examinations were conducted prior to study termination. Extensive gross pathological examination was performed at necropsy at which time organs were removed for determination of weights at final sacrifice. Approximately 26 organs or tissues and all tissue masses were removed at final necropsy and prepared for histological examination.
Result	One control female (day 85), one 100 mg/kg female (day 3), two 2500 mg/kg males (days 6 and 36), and three 2500 mg/kg females (days 3, 3, and 56) died during the study; deaths of 5 of the 6 were ascribed to dosing errors. No toxicologically significant effects on body weight or food intake were seen. Clear salivation and clear salivation prior to dosing were seen in both sexes in the 2500 mg/kg group. Hemoglobin, hematocrit, and mean cell volume were significantly increased in males of the 2500 mg/kg group at the interim sacrifice. At the final sacrifice, hemoglobin, hematocrit, mean cell volume, and mean cell hemoglobin were significantly elevated in 2500 mg/kg males and hemoglobin and hematocrit in 2500 mg/kg females. Statistically sig-nificant differences at final sacrifice

	included decreased platelet count in 2500 mg/kg males, increased mean cell volume in 500 mg/kg females, increased alanine amino-transferase in 2500 mg/kg females at the interim sacrifice and in males at the final sacrifice, depressed glucose and potassium levels in 2500 mg/kg males at the final sacrifice. Other statistically significant and nonsignificant changes were reported in 2500 mg/kg males and females at the final sacrifice, but these were not considered toxi-cologically significant. Statistically significant organ weight changes included increased kidney weights in 500 and 2500 mg/kg females, increased kidney-to-body and -brain weight ratios for males and females in the 2500 mg/kg group, increased liver/body weight ratio in 2500 mg/kg males, increased liver weights, and liver-to-body and -brain ratios in 2500 mg/kg females, depressed brain weights in 2500 mg/kg males, and increased heart/brain weight ratio in 2500 mg/kg females. Histopathological findings included renal proximal tubule degeneration in control and exposed animals of both sexes and intracyto-plasmic droplets or granules (hyaline droplets) in the proximal tubular epithelium in control and exposed animals, predominantly in males. (Kidney lesions are expected components preceding the development of chronic progressive glomerulonephtopathy, a common aging syndrome in Sprague-Dawley rats.) Although the incidence levels for both of these lesions were similar in control and exposed animals, the severity of distribution was markedly altered with increasing dose. In male rats, testicular interstitial edema was seen in both control and test animals with similar incidence and severity. Reactive hyperplasia of the mesenteric and mandibular lymph nodes and splenic granular pigmentation was seen more commonly in 2500 mg/kg male rats; these increases were not statistically or biologically significant.
Reference	Mayhew, D.A. and Morrow, L.D. (1988). Ninety-day gavage study in albino rats using acetone. United States Environmental Protection Agency Contract No. 68-01-7075. American Biogenic Corporation Study 410-2313.
Species	rat
Strain	Sprague-Dawley
Sex	male
Route of Administration	inhalation
Exposure Period	2, 4, and 8 weeks
Frequency of Treatment	3 h/day, 5 days/wk
Post Exposure Observation. Period	2 weeks (following 8-week exposure only)
Doses	19,000 ppm; 9 animals (total)/time-of-exposure group
Control Group	yes
GLP	no data
Test substance	ACS Grade, Instr-Analyzed (J.T. Baker)
Remark	Groups of rats were exposed to 19,000 ppm of acetone for 3 h per day. Exposures were repeated 5 times per week for 2, 4, or

	8 weeks. At 2, 4, and 8 weeks of exposure and 2 weeks postexposure, groups of 5 exposed animals and 5 controls were weighed and anesthetized (pentobarbital), and blood was withdrawn for deter-mination of serum glutamic-oxaloacetic transaminase (SGOT, lactic dehydrogenase (LDH), and blood urea nitrogen (BUN). The rats were killed, and the whole brain, lungs, kidneys, and liver were removed and weighed. Lungs were also weighed dry to determine fluid content, and triglyceride was determined in liver. At each time interval, 4 exposed rats and 4 controls were killed, and liver, heart, lung, kidney, and brain were taken for histopathological examination.
Result	Body weight gain was slightly, but nonsignificantly ($p>0.05$), depressed throughout the exposure period and 2 weeks postexposure. Brain and kidney weights were depressed during the exposure period only. Nonsignif-icant increases in SGOT (AST) were seen at 2, 4, and 8 weeks. No other effects were seen. Although body, brain, and kidney weights were depressed and SGOT was slightly elevated, there were no statistically significant findings with respect to any toxicological index meas-ured. There were no untoward histopathological findings.
Reference	Bruckner, J.V. and Peterson, R.G. (1981). Evaluation of toluene and acetone inhalant abuse. II. Model develop-ment and toxicology. Toxicol. Appl. Pharmacol. 61:302-312.

5.5 Genetic Toxicity in Vitro

Type	chromosomal aberration
System of Testing	Chinese hamster lung fibroblast cell line CHL (Cancer Research Institute: Tokyo)
Concentration	40 mg/mL
Metabolic Activation	with and without
Result	positive
GLP	no data
Test substance	no data
Remark	Cells were exposed to chemical for 24 or 48 h. Colcemid added 2 h before harvesting cells, which were trypsin-ized, suspended in hypotonic KCl for 13 min, and separated by centrifuging. The cells were fixed with acetic acid-methanol and fixed on glass slides, which were air dried. The cells were stained with Giemsa, and 100 metaphases were scored for polyploid cells and structural chromosomal aberrations.
Result	Acetone produced 6.0% polyploid cells at 48 h, and 28.0% cells with structural aberrations were at 24 h. The authors consider an incidence of less than 4.9% aberrations to be negative and greater than 10% to be positive. The dose at which structural aberrations were detected in 20% of the metaphases observed (D20) was 36.9 mg/mL. The authors noted that the test was positive at 48 h also, but negative in the presence of S9 mix. Control and solvent-control (saline,

	DMSO, ethanol, sodium carboxymethyl cellulose) incidences of aberrations were said to be 3% or less.
Reference	Ishidate, M., Jr., Sofuni, T., Yoshikawa, K., Hayashi, M., Nohmi, T., Sawada, M., and Matsuoka, A. (1984). Primary mutagenicity screening of food additives currently used in Japan. Food Chem. Toxicol. 22:623-636.
Type	chromosomal aberration
System of Testing	Chinese hamster ovary cells
Concentration	0.5-5.0 mg/mL
Metabolic Activation	with and without
Result negative	
GLP	no data
Test substance	as prescribed by 1.1-1.4
Remark	Cells were exposed to chemical for 8 h, washed to remove the test chemical, and treated with colcemid for 2.0-2.5 h before cell harvest. The method of Galloway et al., Environ. Mutagen. 7,1985 was followed except that the total duration of 10-12 h. The cells were fixed with 3:1 acetic acid-methanol and stained with 5% Giemsa on glass slides. Simple, complex, and "other" aberrations were determined on 100-200 cells. Chromatid and chromosome gaps were recorded but were not used in the analysis.
Result	Acetone produced 0-3.5% simple aberations and 0-2% complex aberations, and a total percentage of 1.5-4.0% for the three dose levels tested. The results were equal to or less than the values observed with untreated control cells.
Reference	Loveday, K.S., Anderson, B.E., Resnick, M.A., and Zeiger, E. (1990). Chromosome aberration and sister chromatid exchange tests in Chinese hamster ovary cells in vitro. V: Results with 46 chemicals. Environ. Mol. Mutagen. 16:272-303.
Type	sister chromatid exchange
System of Testing	Chinese hamster ovary cells
Concentration	0.05-5.0 mg/mL
Metabolic Activation	with and without
Result negative	
GLP	no data
Test substance	as prescribed by 1.1-1.4
Remark	Cells were exposed to chemical for 2 h before adding bromodeoxyuridine (BrdUrd), which was incubated for 24 h. After 26 h fresh medium with BrdUrd and colcemid was added for an additional 2-2.5 h at 37°C. Cells were examined for signs of toxicity (confluence in the monolayer) before harvesting. Cells were separated by centrifugation, fixed with 3:1 acetic acid-methanol, fixed on glass slides, and stained with Hoechst 33258 and then 5% Giemsa. Fifty (50) second division metaphase cells were scored for sister chromatid exchanges (SCEs).
Result	Acetone produced 8.5-8.7 SCEs per cell when tested without activation at the three dose levels examined. When tested with

	activation 6.4-7.5 SCEs per cell were observed. The results were equal to or less than the values observed with untreated control cells. A postive trend test with at 20% increase in chromatid exchanges with at least one dose was required for a positive response.
Reference	Loveday, K.S., Anderson, B.E., Resnick, M.A., and Zeiger, E. (1990). Chromosome aberration and sister chromatid exchange tests in Chinese hamster ovary cells in vitro. V: Results with 46 chemicals. Environ. Mol. Mutagen. 16:272-303.
Type	two-stage cell transformation assay
System of Testing	BALB/3T3 clone A31-1-1 (JCRB0601)
Concentration.	0.5%
Metabolic Activation	without
Result negative	
GLP	no data
Test substance	no data
Method	BALB/3T3 cells in culture were treated with test chem-ical (but not acetone) for 72 h. The chemical was removed, and the cells were grown in medium for 3 days. The promoter 12-O-tetradecanoylphorbol 13-acetate (TPA) or 0.5% acetone was added. After two weeks, the promoter was removed, and the cells were grown for 3 weeks at which time they were collected and stained with Giemsa.
Remark	Acetone caused no transformation when applied during the promotion phase to cells treated with DMSO. It is not clear that cells were treated with acetone alone or with acetone followed by TPA. TPA was, however, applied to the cells in acetone solution.
Reference	Sakai, A. and Sato, M. (1989). Improvement of carcin-ogen identification in BALB/3T3 cell transformation by application of a 2-stage method. Mutat. Res. 214:285-296.
Type	minimal inhibitory concentration
System of Testing	trp- E. coli, 3 strains: WP2 (wild-type, repair proficient), WP67 (uvr- polA-), and CM871 (uvrA- recA- lexA-).
Concentration.	Up to 40 mg/well
Metabolic Activation	with and without
Result	negative
GLP	no data
Test substance	no data
Method	Six replicates (rows) of eight twofold dilutions of each compound were prepared in Microtiter plates. Three rows were filled with phosphate-buffered saline and three with S9 mix. One strain of each of the three bacteria was added to each of the eight wells in one of the rows. The plates were incubated at 37°C and observed for increases in turbidity or the formation of a pellet of settled cells. Apparently positive results were confirmed by subculture on agar plates. Method is liquid micromethod modification of the rec-assay system with B.

	subtilis (Kada et al., 1981) and the E. coli system of McCarroll et al. (1981).
Remark	Method results in a minimal inhibitory concentration (MIC). The MIC for acetone under each condition of strain and activation (six values) was > 40 mg/well. A ratio between the MICs in repair-proficient (WP2) and repair-deficient (WP67 and CM871) strains greater than 2 was considered to be significant in the test. Although these ratios could not be obtained for acetone (since all values were "> 40 mg"), the values suggest that acetone would be an extremely weak DNA-damaging agent if it were positive. The overall accuracy for predicting car-cinogenicity for the DNA-repair test was 72.4% for a battery of 75 of the 135 compounds for which clear carcinogenicity data were available and that included several compounds reported to be nonmutagenic carcin-ogens or noncarcinogenic mutagens.
Reference	De Flora, S., Zanacchi, P., Camoirano, A., Bennicelli, C., and Badolati, G.S. (1984). Genotoxic activity and potency of 135 compounds in the Ames reversion test and in a bacterial DNA-repair test. Mutat. Res. 133:161-198. Kada, T., Hirano, K., and Shirasu, Y. (1980). Screening of environmental chemical mutagens by the Rec-assay system with Bacillus subtilis. In: De Serres, F.J. and Hollaender, A. (Eds.). Chemical Mutagens, Vol. 6, Plenum, New York, 149-173. McCarroll, N.E., Piper, C.E., and Keech, B.H. (1981). An E. coli microsuspension assay for the detection of DNA damage induced by direct-acting agents and promutagens. Environ. Mutagen. 3:429-444.
Type	mitotic chromosomal malsegregation, mitotic recombination, and point mutations.
System of Testing	Saccharomyces cerevisiae diploid strain D61.M
Concentration.	6.82-7.83%
GLP	no data
Test substance	no data
Remark	Chemicals were at least 97%
Results	Positive for aneuploidy; negative for mitotic recombination and point mutations.
Method	Chemicals were pipetted directly into growing cultures in peptone-glucose-yeast extract (YEPD) medium and incubated at 28°C for 4 h, placed in an ice bath for < 16 h, and then incubated at 28°C on a shaker for 4 h (cold-interruption procedure). Samples of cultures were plated on a selective cyclohexamide medium. After 6-7 days, the plates were scored for colony color and numbers. Red colonies reflect cumulative effects of events like point mutations, mitotic recombinations, and deletion of chromosomal fragments. White colonies

	contain presumptive monosomics; these are confirmed by establishment of a requirement for leucine.
Remark	Acetone gave inconsistent results with the original protocol, which did not have the ice-storage step. The authors found that storage in ice for 16 h or more fol-lowing the initial incubation gave repeatable positive results (Zimmermann et al. 1984). Most of the cyclohex- amide-resistant colonies were white and almost all of these were leucine requiring, indicating that these colonies were monosomics. Red resistant colonies did not increase and were not significantly leucine requiring, indicating that acetone did not induce point mutations or recombinations under the test conditions.
Remark	Using the method of Zimmermann et al. (1985), Mayer and Goins (1994) reported that concentrations of acetone up to 459 mM (2.7%) did not cause chromosome loss or mutations in S. cerevisiae D61.M. In an interlaboratory comparison of mitotic chromosome loss in S. cerevisiae, acetone was positive in one laboratory at levels of ca. 45-55 mg/mL using the cold-interruption procedure of Zimmermann et al. (1985) but negative in a second lab-oratory. Both laboratories reported acetone negative using the standard procedure with overnight incubation at 28°C (Whittaker et al., 1989). Acetone was positive for production of aneuploidy in S. cerevisiae using the cold-interruption procedure of Zimmermann et al. (1985) at levels > 40 mg/mL. It was negative using the standard procedure and did not produce other genetic effects (gene mutation, mitotic recombination, etc.) with either protocol (Albertini, 1991). The merokinetic effect (multipolarity) of acetone on chromosome division of human leukocytes was reported by Kabarity (1969). Acetone caused the formation of multiple-spindle apparatus leading to the movement of each part of the centrosome to one pole. The author concluded that lymphoma TK+/- 3.7.2C cells
Concentration.	10-30 mg/mL
Metabolic Activation	without
	Resul0 Reference EHRT (1987). Screening of Priority Chemicals for Reproductive Hazards. Environmental Health Research and Testing, Inc. Cincinnati, OH. Project No. ETOX-85-

EXTRACT FROM IRPTC LEGAL FILES

File: 17.01 LEGAL **rn : 3971**

systematic name:2-Propanone
common name :acetone
reported name :ACETONE
cas no :67-64-1 rtecs no :AL3150000
area : AUS type : REC

subject	specification	descriptor
AIR	OCC	TLV

TWA: 1780MG/M3 (750PPM) STEL: 2375MG/M3 (1000PPM)
entry date: MCH 1985

original : ILO , , , , ,
amendment: AOHGN*, APPROVED OCCUPATIONAL HEALTH GUIDE THRESHOLD LIMIT VALUES, , , , 1983

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File: 17.01 LEGAL **rn : 14731**

systematic name:2-Propanone
common name :acetone
reported name :ACETONE
cas no :67-64-1 rtecs no :AL3150000
area : BEL type : REC

subject	specification	descriptor
AIR	OCC	TLV

TWA: 1780MG/M3 (750PPM); STEL: 2375MG/M3 (1000PPM)
entry date: JUL 1987

original : ILO , , , , ,
amendment: TLVBE*, THRESHOLD LIMIT VALUES(TOLERABLE LIMIT VALUES), , , , 1984

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File: 17.01 LEGAL **rn : 15424**

systematic name:2-Propanone
common name :acetone
reported name :ACETONE
cas no :67-64-1 rtecs no :AL3150000
area : FIN type : REC

subject	specification	descriptor
AIR	OCC	MPC

TWA: 1200MG/M3 (500PPM) STEL: 1500MG/M3 (625PPM)
entry date: MAY 1989

original : ILO , , , , ,
amendment: APWFI*, HTP-ARVOT (LIST OF LIMIT VALUES FOR CONCENTRATIONS OF TOXIC SUBSTANCES KNOWN TO BE HARMFUL TO HEALTH), 25 , , 10 ,

1988

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File: 17.01 LEGAL **rn : 16007**

systematic name:2-Propanone
common name :acetone
reported name :ACETONE
cas no :67-64-1 rtecs no :AL3150000
area : HUN type : REG

subject	specification	descriptor
AIR	OCC	MAC

TWA: 200MN/M3; STEL(30 MIN): 1000MG/M3
entry date: MCH 1985

original : ILO , , , , ,
amendment: HSMSZ*, HUNGARIAN STANDARD MSZ NO., 21461-78 , , , 1978

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File: 17.01 LEGAL **rn : 16192**

systematic name:2-Propanone
common name :acetone
reported name :ACETONE
cas no :67-64-1 rtecs no :AL3150000
area : ITA type : REC

subject	specification	descriptor
AIR	OCC	TLV

1000MG/M3 (420PPM)
entry date: MCH 1985

original : ILO , , , , ,
amendment: TLVIT*, VALORI LIMITE PONDERATI(APPRAISED LIMIT VALUES), , ,

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File: 17.01 LEGAL **rn : 16428**

systematic name:2-Propanone
common name :acetone
reported name :ACETONE
cas no :67-64-1 rtecs no :AL3150000
area : NLD type : REC

subject	specification	descriptor
AIR	OCC	MXL

TWA: 1780MG/M3 (750PPM)
entry date: JUN 1987

original : ILO , , , , ,
amendment: NMACN*, NATIONALE MAC-LIST(NATIONAL MAC-LIST), , , , 1986

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File: 17.01 LEGAL **rn : 16943**

systematic name:2-Propanone
common name :acetone
reported name :ACETONE
cas no :67-64-1 rtecs no :AL3150000
area : POL type : REG

subject	specification	descriptor
AIR	OCC	MPC

TWA: 200MG/M3
entry date: MCH 1985

original : ILO , , , , ,
amendment: OMLWS*, ORDINANCE OF THE MINISTER OF LABOUR, WAGES AND SOCIAL AFFAIRS, 22DEC , , , 1982

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File: 17.01 LEGAL **rn : 17169**

systematic name:2-Propanone
common name :acetone
reported name :ACETONE
cas no :67-64-1 rtecs no :AL3150000
area : ROM type : REG

subject	specification	descriptor
AIR	OCC	MPC

TWA: 1000MG/M3; CLV: 1500MG/M3
entry date: MCH 1985

original : ILO , , , , ,
amendment: OMHRO*, ORDINANCE OF THE MINISTRY OF HEALTH, 60 , , , 1975

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File: 17.01 LEGAL **rn : 17543**

systematic name:2-Propanone
common name :acetone
reported name :ACETONE
cas no :67-64-1 rtecs no :AL3150000
area : CHE type : REG

subject	specification	descriptor
AIR	OCC	MAK

TWA: 1780MG/M3 (750PPM)
entry date: DEC 1987

original : ILO , , , , ,
amendment: ZWACH*, ZULAESSIGE WERTE AM ARBEITSPLATZ(PERMITTED VALUES IN THE WORKPLACE), , , , 1987

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File: 17.01 LEGAL **rn : 18086**

systematic name:2-Propanone
common name :acetone
reported name :ACETONE
cas no :67-64-1 rtecs no :AL3150000
area : YUG type : REG

subject	specification	descriptor
AIR	OCC	MAC

TWA: 800MG/M3 (336PPM)
entry date: MCH 1985

original : ILO , , , , ,
amendment: ORYUG*, ORDINANCE, 24-3698/1 , , , 1971

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File: 17.01 LEGAL **rn : 50877**

systematic name:2-Propanone
common name :acetone
reported name :ACETONE
cas no :67-64-1 rtecs no :AL3150000
area : IMO type : REG

subject	specification	descriptor
AQ	EMI	PRMT
AQ	MARIN	PRMT

This substance is presently considered to present no harm to human health, marine re sources, amenities or other legitimats uses of the sea when discharged into the sea from tank cleaning or deballasting operations
entry date: APR 1993

original : IMODC*, , , , , 1992

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File: 17.01 LEGAL **rn : 100031**

systematic name:2-Propanone
common name :acetone
reported name :ACETONE
cas no :67-64-1 rtecs no :AL3150000
area : ARG type : REG

subject	specification	descriptor
AIR	OCC	MPC

8H-TWA : 1780 MG/M3 (750 PPM), 15MIN-STEL : 2375 MG/M3 (1000 PPM)
(MAXIMUM 4 TIMES/DAY WITH INTERVALS OF A LEAST 60 MINUTES)
entry date: OCT 1991 effective date: 29MAY1991

title: LIMIT VALUES FOR CHEMICAL SUBSTANCES IN THE WORKING ENVIRONMENT-RESOLUTION NO. 444/1991 OF THE MINISTRY OF WORK AND SOCIAL SECURITY (AMENDING REGULATION DECREE NO. 351/1979 UNDER LAW NO. 19587/1972: HYGIENE AND SAFETY AT WORK)
original : ARGOB*, Boletin Oficial de la Republica Argentina(Argentinian Official Bulletin), 24170 , I , 1 , 1979
amendment: ARGOB*, Boletin Oficial de la Republica Argentina(Argentinian Official Bulletin), 27145 , I , 4 , 1991

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File: 17.01 LEGAL **rn : 300477**

systematic name:2-Propanone
common name :acetone
reported name :ACETONE
cas no :67-64-1 rtecs no :AL3150000
area : CAN type : REG

subject	specification	descriptor
AIR	OCC	TLV

TWA: 750 PPM, 1,780 MG/M3; STEL: 1,000 PPM, 2,375 MG/M3. PRESCRIBED BY THE CANADA OCCUPATIONAL SAFETY AND HEALTH REGULATIONS, UNDER THE CANADA LABOUR CODE (ADMINISTERED BYTHE DEPARTMENT OF LABOUR). THE REGULATIONS STATE THAT NO EMPLOYEE SHALL BE EXPOSED TO A CONCENTRATION OF AN AIRBORNE CHEMICAL AGENT IN EXCESS OF THE VALUE FOR THAT CHEMICAL AGENT ADOPTED BY ACGIH (AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS) IN ITSPUBLICATION ENTITLED: "THRESHOLD LIMIT VALUE AND BIOLOGICAL EXPOSURE INDICES FOR 1985-86".
entry date: MCH 1991 effective date: 13MCH1986

amendment: CAGAAK, Canada Gazette Part II, 120 , 6 , 1105 ,

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File: 17.01 LEGAL **rn : 301601**

systematic name:2-Propanone
common name :acetone
reported name :ACETONE
cas no :67-64-1 rtecs no :AL3150000
area : CAN type : REG

subject	specification	descriptor
TRNSP LABEL PACK		CLASS RQR

PIN (PRODUCT IDENTIFICATION NO.): UN1090. CLASS (3.1): FLAMMABLE LIQUID. SPECIAL PROVISIONS: 99. PACKING GROUP II, (I=GREAT DANGER, III=MINOR DANGER). MAXIMUM AMOUNT PER PACKAGE THAT MAY BE TRANSPORTED ON PASSENGER AIRCRAFT OR VEHICLE: 5 L. MAXIMUM AMOUNT PER PACKAGE THAT MAY BE TRANSPORTED ONA CARGO AIRCRAFT: 60 L. PRESCRIBED BY THE TRANSPORTATION OF DANGEROUS GOODS REGULATIONS, UNDER THE TRANSPORTATION OF DANGEROUS GOODS ACT (ADMINISTERED BY THE DEPARTMENT OF TRANSPORT). THE ACT AND REGULATIONS ARE INTENDED TO PROMOTE SAFETY INTHE TRANSPORTATION OF DANGEROUS GOODS IN CANADA, AS WELL AS PROVIDE ONE COMPREHENSIVE SET OF RULES APPLICABLE TO ALL MODES OF TRANSPORT ACCROSS CANADA. THESE ARE BASED ONUNITED NATIONS RECOMMENDATIONS. THE ACT AND REGULATIONS SHOULD BE CONSULTED FOR DETAILS. RECORDS ARE ENTERED UNDER THE PROPER SHIPPINGNAME FOUND IN THE REGULATIONS; THIS MAY INCLUDE VERY GENERAL GROUPS OF CHEMICAL SUBSTANCES.
entry date: OCT 1991 effective date: 06DEC1990

amendment: CAGAAK, Canada Gazette Part II, 124 , 26 , 5523 ,

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File: 17.01 LEGAL **rn : 302345**

systematic name:2-Propanone
common name :acetone
reported name :ACETONE
cas no :67-64-1 rtecs no :AL3150000
area : CAN type : REG

subject	specification	descriptor
GOODS LABEL SALE IMPRT	CONSM	RQR PRO

IT IS PROHIBITED TO SELL, ADVERTISE OR IMPORTINTO CANADA ADHESIVES, CLEANING SOLVENTS, THINNING AGENTS AND DYES CONTAINING ACETONE, WHEN PACKAGED AS CONSUMER PRODUCTS, UNLESS DETAILED LABELLING REQUIREMENTS ARE MET. THIS PROHIBITION IS PRESCRIBED BY SCHEDULE I OF THE HAZARDOUS PRODUCTS ACT (HPA), ADMINISTERED BY THE DEPARTMENT OF CONSUMER AND CORPORATE AFFAIRS. IT AUTHORIZES THE PROHIBITION OF PRODUCTS THAT ARE LIKELY TO BEOF DANGER TO THE HEALTH AND SAFETY OF THE PUBLIC.
entry date: MAY 1991 effective date: 01NOV1988

amendment: CAGAAK, Canada Gazette Part II, 122 , 24 , 4625 ,

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File: 17.01 LEGAL **rn : 302508**

systematic name:2-Propanone
common name :acetone
reported name :ACETONE
cas no :67-64-1 rtecs no :AL3150000
area : CAN type : REG

subject	specification	descriptor
USE STORE LABEL	OCC	RQR

INGREDIENT DISCLOSURE LIST CONCENTRATION 1% WEIGHT/WEIGHT. THE WORKPLACE HAZARDOUS MATERIALS INFORMATION SYSTEM (WHMIS) IS A NATIONAL SYSTEM TO PROVIDE INFORMATION ON HAZARDOUS MATERIALS USED IN THE WORKPLACE. WHMIS IS IMPLEMENTED BY THE HAZARDOUS PRODUCTS ACT AND THE CONTROLLED PRODUCTS REGULATIONS (ADMINISTERED BY THE DEPARTMENT OF CONSUMER AND CORPORATE AFFAIRS). THE REGULATIONS IMPOSE STANDARDS ON EMPLOYERS FORTHE USE, STORAGE AND HANDLING OF CONTROLLED PRODUCTS AND ADDRESS LABELLING AND IDENTIFICATION, EMPLOYEE INSTRUCTION AND TRAINING, AS WELL AS THE UPKEEP OF A MATERIALS SAFETY DATA SHEET (MSDS). THE PRESENCE IN A CONTROLLED PRODUCT OF AN INGREDIENT IN A CONCENTRATION EQUAL TO OR GREATER THAN SPECIFIED IN THE INGREDIENT DISCLOSURE LIST MUST BE DISCLOSED IN THE SAFETY DATA SHEET.
entry date: APR 1991 effective date: 31DEC1987

amendment: CAGAAK, Canada Gazette Part II, 122 , 2 , 551 ,

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File: 17.01 LEGAL **rn : 400270**

systematic name:2-Propanone
common name :acetone
reported name :ACETONE
cas no :67-64-1 rtecs no :AL3150000
area : CSK type : REG

subject	specification	descriptor
AIR	AMBI	CLASS

THE SUBSTANCE IS CLASSIFIED IN THE FOURTH GROUP OF AIR POLLUTANTS (ORGANIC GASES AND VAPOURS)
entry date: JAN 1992 effective date: 1OCT1991

title: PROVISION OF FEDERAL COMMITTEE FOR ENVIRONMENT TO ACT NO. 309 FROM 9 JULY 1991 ON AIR PROTECTION AGAINST AIR POLLUTANTS
original : SZCSR*, Sbirka Zakonu Ceskoslovenske Socialisticke Republiky(Collection of the Law of Czechoslovak Socialist Republic), , 84 , 2061 , 1991

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File: 17.01 LEGAL **rn : 400406**

systematic name:2-Propanone
common name :acetone
reported name :ACETONE
cas no :67-64-1 rtecs no :AL3150000
area : CSK type : REG

subject	specification	descriptor
WASTE	INDST	CLASS
		RQR

THE SUBSTANCE IS CLASSIFIED AS HAZARDOUS WASTE COMPONENT. IT IS OR CAN BE DANGEROUS TO HUMAN HEALTH OR ENVIRONMENT. QUANTITY, SPECIFICATION, USE OR DISPOSAL OF THE WASTE MUST BE REPORTED TO AUTHORITIES. TRANSPORT AND DISPOSAL OF THE WASTE MUST BE PERFORMED IN ACCORDANCE WITH SPECIAL DIRECTIVE
entry date: JAN 1992 effective date: 1AUG1991

title: PROVISION OF FEDERAL COMMITTEE FOR ENVIRONMENT WHICH DECLARES

WASTE CLASSIFICATION AND CATALOGUE
original : SZCSR*, Sbirka Zakonu Ceskoslovenske Socialisticke Republiky(Collection of the Law of Czechoslovak Socialist Republic), , 69 , 1650 , 1991

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File: 17.01 LEGAL **rn : 400540**

systematic name:2-Propanone
common name :acetone
reported name :ACETONE
cas no :67-64-1 rtecs no :AL3150000
area : CSK type : REG

subject	specification	descriptor
AIR	OCC	MAC

TWA: 800.0MG/M3; CLV: 4000.0MG/M3
entry date: DEC 1991 effective date: MCH1985

title: DIRECTIVE NO. 46/1978 ON HYGIENIC REQUIREMENTS ON OCCUPATIONAL ENVIRONMENT
original : HPMZC*, HYGIENICKE PREDPISY MINISTERSTVA ZDRAVOTNICTVI CSR(HYGIENIC REGULATIONS OF MINISTRY OF HEALTH OF CSR), 39 , , , 1978
amendment: HPMZC*, HYGIENICKE PREDPISY MINISTERSTVA ZDRAVOTNICTVI CSR(HYGIENIC REGULATIONS OF MINISTRY OF HEALTH OF CSR), 58 , , , 1985

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File: 17.01 LEGAL **rn : 401111**

systematic name:2-Propanone
common name :acetone
reported name :ACETONE
cas no :67-64-1 rtecs no :AL3150000
area : CSK type : REG

subject	specification	descriptor
FOOD		MPC

LIMIT OF ADDITIVE PRESENT DUE TO PRODUCTION, PACKING, TRANSPORT AND STORAGE OF FOOD PRODUCTS: 5G/KG.
entry date: DEC 1991 effective date: 1JUL1986

title: DIRECTIVE NO. 50/1978 ON FOREIGN SUBSTANCES IN FOODSTUFFS
original : HPMZC*, HYGIENICKE PREDPISY MINISTERSTVA ZDRAVOTNICTVI CSR(HYGIENIC REGULATIONS OF MINISTRY OF HEALTH OF CSR), 43 , , , 1978
amendment: HPMZC*, HYGIENICKE PREDPISY MINISTERSTVA ZDRAVOTNICTVI CSR(HYGIENIC REGULATIONS OF MINISTRY OF HEALTH OF CSR), 61 , , , 1986

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File: 17.01 LEGAL **rn : 500483**

systematic name:2-Propanone
common name :acetone
reported name :ACETONE
cas no :67-64-1 rtecs no :AL3150000
area : DEU type : REC

subject	specification	descriptor
AQ USE	INDST	CLASS RQR

THIS SUBSTANCE IS CLASSIFIED AS IN GENERAL NOT HAZARDOUS TO WATER (WATER-HAZARD CLASS: WGK 0). (THE DIFFERENT CLASSES ARE: WGK 3 = VERY HAZARDOUS; WGK 2 = HAZARDOUS; WGK 1 = SLIGHTLY HAZARDOUS; WGK 0 = IN GENERAL NOT HAZARDOUS.) THE CLASSIFICATION FORMS THE BASIS FOR WATER-PROTECTION REQUIREMENTS FOR INDUSTRIAL PLANTS IN WHICH WATER-HAZARDOUS SUBSTANCES ARE HANDLED.
entry date: DEC 1991

title: ADMINISTRATIVE RULES CONCERNING WATER-HAZARDOUS SUBSTANCES (VERWALTUNGSVORSCHRIFT WASSERGEFAEHRDENDE STOFFE)
original : GMSMA6, Gemeinsames Ministerialblatt. Joint Ministerial Papers, , 8 , 114 , 1990

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File: 17.01 LEGAL **rn : 502155**

systematic name:2-Propanone
common name :acetone
reported name :ACETONE
cas no :67-64-1 rtecs no :AL3150000
area : DEU type : REG

subject	specification	descriptor
AIR	EMI	MPC

THIS SUBSTANCE BELONGS TO CLASS III. THE AIR EMISSIONS OF ORGANIC COMPOUNDS MUST NOT EXCEED (AS THE SUM OF ALL COMPOUNDS IN ONE CLASS) THE FOLLOWING MASS CONCENTRATIONS: CLASS I - 20 MG/M3 AT A MASS FLOW OF >= 0.1 KG/H; CLASS II - 100 MG/M3 AT A MASS FLOW OF >= 2 KG/H; CLASS III - 150 MG/M3 AT A MASS FLOW OF >= 3 KG/H. IF COMPOUNDS FROM DIFFERENT CLASSES ARE PRESENT, THE MASS CONCENTRATION MUST NOT EXCEED 150 MG/M3 AT A TOTAL MASS FLOW OF >= 3 KG/H.
entry date: JAN 1992 effective date: 01MCH1986

title: TECHNICAL GUIDELINES FOR AIR POLLUTION CONTROL (TECHNISCHE ANLEITUNG ZUR REINHALTUNG DER LUFT)
original : GMSMA6, Gemeinsames Ministerialblatt. Joint Ministerial Papers, , 7 , 93 , 1986

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File: 17.01 LEGAL **rn : 502438**

systematic name:2-Propanone
common name :acetone
reported name :ACETONE
cas no :67-64-1 rtecs no :AL3150000

area : DEU type : REC

subject	specification	descriptor
AIR	OCC	MAK

8H-TWA: 1000 ML/M3 (PPM); 2400 MG/M3 (20C, 101.3 KPA). SUBSTANCE ELICITING VERY WEAK EFFECTS. 60MIN-STEL: 2000 ML/M3 (PPM); 4800 MG/M3; CEILING VALUE; 3X/SHIFT. VAPOUR PRESSURE: 24 KPA AT 20 C.
entry date: JAN 1992

title: MAXIMUM CONCENTRATIONS AT THE WORKPLACE AND BIOLOGICAL TOLERANCE VALUES FOR WORKING MATERIALS (MAXIMALE ARBEITSPLATZKONZENTRATIONEN UND BIOLOGISCHE ARBEITSSTOFFTOLERANZWERTE)
original : MPGFDF, MITTEILUNG DER SENATSKOMMISSION ZUR PRUEFUNG GESUNDHEITSSCHAEDLICHER ARBEITSSTOFFE (DEUTSCHE FORSCHUNGSGEMEINSCHAFT), XXVII , , 17 , 1991

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File: 17.01 LEGAL **rn : 510565**

systematic name:2-Propanone
common name :acetone
reported name :ACETONE
cas no :67-64-1 rtecs no :AL3150000
area : DEU type : REG

subject	specification	descriptor
CLASS LABEL		CLASS
PACK		RQR

CLASSIFICATION AND LABELLING IN GERMANY IS GENERALLY THE SAME AS FOR THE EEC (SEE OJEC** L180, 1991). HOWEVER, SLIGHT MODIFICATIONS MAY BE INTRODUCED FOR SOME SUBSTANCES IN THE GERMAN LEGISLATION.
entry date: APR 1992 effective date: 15JUN1991

title: ORDINANCE ON HAZARDOUS SUBSTANCES. (GEFAHRSTOFFVERORDNUNG)
original : BGZBAD, Bundesgesetzblatt (Federal Law Gazette), , I , 1931 , 1991

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File: 17.01 LEGAL **rn : 612864**

systematic name:2-Propanone
common name :acetone
reported name :ACETONE
cas no :67-64-1 rtecs no :AL3150000
area : GBR type : REG

subject	specification	descriptor
TRNSP LABEL		CLASS
		RQR

LABELLING OF ROAD TANKERS: FLAMMABLE LIQUID. EMERGENCY ACTION CODE: 2(Y)E

entry date: JAN 1983 effective date: 28MCH1979

title: HAZARDOUS SUBSTANCES (LABELLING OF ROAD TANKERS) REGULATIONS 1978
original : GBRSI*, STATUTORY INSTRUMENTS, 1702 , , , 1978

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File: 17.01 LEGAL **rn : 650642**

systematic name:2-Propanone
common name :acetone
reported name :ACETONE
cas no :67-64-1 rtecs no :AL3150000
area : GBR type : REG

subject	specification	descriptor
TRNSP	MARIN	RQR
AQ	MARIN	RQR
AQ	EMI	RQR

CLASSIFIED AS A NON-POLLUTING LIQUID SUBSTANCE. DOCUMENTARY EVIDENCE OF ASSESSMENT AND APPROVAL REQUIRED BY A CARRIER. DISCHARGE INTO THE SEA IS NOT PROHIBITED.
entry date: 1992 effective date: 06APR1987

title: THE MERCHANT SHIPPING (CONTROL OF POLLUTION BY NOXIOUS LIQUID SUBSTANCES IN BULK) REGULATIONS 1987, SCHEDULE 2
original : GBRSI*, STATUTORY INSTRUMENTS, 551 , , 15 , 1987
amendment: GBRSI*, STATUTORY INSTRUMENTS, 2604 , , 2 , 1990

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File: 17.01 LEGAL **rn : 665433**

systematic name:2-Propanone
common name :acetone
reported name :ACETONE
cas no :67-64-1 rtecs no :AL3150000
area : GBR type : REG

subject	specification	descriptor
AIR	OCC	OES

8H-TWA: 1780MG/M3 (75PPM); STEL(10MIN-TWA): 3560MG/M3 (3560PPM)
entry date: 1992 effective date: 01JAN1992

title: EH40 OCCUPATIONAL EXPOSURE LIMITS FOR USE WITH THE CONTROL OF SUBSTANCES HAZARDOUS TO HEALTH REGULATIONS
original : GBRSI*, STATUTORY INSTRUMENTS, 1657 , , 10 , 1988
amendment: GNHSE*, GUIDANCE NOTE FROM THE HEALTH AND SAFETY EXECUTIVE, EH40 , , 11 , 1992

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File: 17.01 LEGAL **rn : 762000**

systematic name:2-Propanone
common name :acetone
reported name :ACETONE
cas no :67-64-1 rtecs no :AL3150000
area : IND type : REG

subject	specification	descriptor
MANUF		RQR
SAFTY		RQR
STORE		RQR
IMPRT		RQR

These rules define the responsabilities of occupiers of any industrial activity in which this toxic and hazardous substance may be involved. These responsabilities encompass: (a) assessment of major hazards (causes, occurrence, frequency); (b) measures to prevent accidents and limit eventual impairment to human health and pollution of the environment; (c) provision of relevant factual knowledge and skills to workers in order to ensure health and environmental safety when handling equipments and the foregoing chemical; (d) notification of the competent authorities in case of major accidents; (e) notification of sites to the competent authorities 3 months before commencing; (f)preparation of an on-site emergency plan as to how major accidents should be coped with; (g) provision of competent authorities with information and means to respond quickly and efficiently to any off-site emergency; (h) provision of information to persons outside the site, liable to be affected by a major accident; (i) labelling of containers as to clearly identify contents, manufacturers, physical, chemical and toxicological data; (j) preparation of a safety data sheet including any significant information regarding hazard of this substance and submission of safety reports to the competent authorities; (k) for the import of a hazardous chemical to India, importers must supply the competent authorities with specified information regarding the shipment.
entry date: SEP 1992 effective date: 27NOV1989

title: THE MANUFACTURE, STORAGE AND IMPORT OF HAZARDOUS CHEMICALS RULES. 1989
original : GAZIN*, THE GAZETTE OF INDIA, 787 , , , 1989

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File: 17.01 LEGAL **rn : 800148**

systematic name:2-Propanone
common name :acetone
reported name :ACETONE
cas no :67-64-1 rtecs no :AL3150000
area : JPN type : REC

subject	specification	descriptor
AIR	OCC	MAC

TWA: 470MG/M3 (200PPM)
entry date: DEC 1991

title: MAXIMUM ALLOWABLE CONCENTRATIONS RECOMMENDED BY THE JAPANESE ASSOCIATION OF INDUSTRIAL HEALTH.
original : SAIGBL, Sangyo Igalu (Japanese Journal of Industrial Health), 33 , 4 , 277-287 , 1991

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File: 17.01 LEGAL **rn : 1010048**

systematic name:2-Propanone
common name :acetone
reported name :ACETONE
cas no :67-64-1 rtecs no :AL3150000
area : MEX type : REG

subject	specification	descriptor
AIR	OCC	MXL

AT ANY WORKPLACE WHERE THIS SUBSTANCE IS PRODUCED, STORED OR HANDLED A MAXIMUM PERMISSIBLE LEVEL OF 2400MG/M3 (1000PPM) MUST BE OBSERVED FOR A PERIOD OF 8 HOURS OR 3000MG/M3 (1260PPM) FOR 15 MINUTES FOUR TIMES A DAY WITH INTERVALS OF AT LEAST 1 HOUR.
entry date: DEC 1991 effective date: 28MAY1984

title: INSTRUCTION NO.10 RELATED TO SECURITY AND HYGIENIC CONDITIONS AT WORKPLACES. (INSTRUCTIVO NO. 10, RELATIVO A LAS CONDICIONES DE SEGURIDAD E HIGIENE DE LOS CENTROS DE TRABAJO).
original : DOMEX*, Diario Oficial, , , , 1984
amendment: DOMEX*, Diario Oficial, , , , 1989

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File: 17.01 LEGAL **rn : 1120809**

systematic name:2-Propanone
common name :acetone
reported name :ACETONE
cas no :67-64-1 rtecs no :AL3150000
area : RUS type : REG

subject	specification	descriptor
AIR	OCC	MAC CLASS

CLV: 200.0MG/M3 (VAPOUR) HAZARD CLASS: IV
entry date: MAY 1990 effective date: 01JAN1989

amendment: GOSTS*, GOSUDARSTVENNYI STANDART SSSR(STATE STANDARD OF USSR), 12.1.005 , , , 1988

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File: 17.01 LEGAL **rn : 1122198**

systematic name:2-Propanone
common name :acetone
reported name :ACETONE
cas no :67-64-1 rtecs no :AL3150000
area : RUS type : REG

subject	specification	descriptor
AIR	AMBI	MAC

0.35MG/M3 1X/D, 0.35MG/M3 AV/D.
entry date: SEP 1985 effective date: AUG1984

amendment: PDKAV*, PREDELNO DOPUSTIMYE KONTSENTRATSII (PDK) ZAGRYAZNYAYUSHCHIKH VESHCHESTV V ATMOSFERNOM VOZDUKHE NASELENNYKH MEST (MAXIMUM ALLOWABLE CONCENTRATIONS (MAC) OF CONTAMINANTS IN THE AMBIENT AIR OF RESIDENTIAL AREAS), 3086-84 , , , 1984

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File: 17.01 LEGAL **rn : 1122704**

systematic name:2-Propanone
common name :acetone
reported name :ACETONE
cas no :67-64-1 rtecs no :AL3150000
area : RUS type : REG

subject	specification	descriptor
AQ	SURF	MAC CLASS

2.2MG/L HAZARD CLASS: III
entry date: JUL 1990 effective date: 1JAN1989

amendment: SPNPV*, SANITARNYE PRAVILA I NORMY OKHRANY POVERKHNOSTNYKH VOD OT ZAGRIAZNENIA (HEALTH REGULATION AND STANDARDS OF SURFACE WATER PROTECTION FROM CONTAMINATION), 4630-88 , , , 1988

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File: 17.01 LEGAL **rn : 1200096**

systematic name:2-Propanone
common name :acetone
reported name :ACETONE
cas no :67-64-1 rtecs no :AL3150000
area : SWE type : REG

subject	specification	descriptor
AIR	OCC	HLV

1D-TWA: 600MG/M3 (250PPM). 15MIN-STEL: 1200MG/M3 (500 PPM)
entry date: 1992 effective date: 01JUL1991

title: HYGIENIC LIMIT VALUES.
original : AFS***, ARBETARSKYDDSSTYRELSENS FOERFATTNINGSSAMLING, 1990:13 , , 5-64 , 1990

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File: 17.01 LEGAL **rn : 1302002**

systematic name:2-Propanone
common name :acetone

reported name :ACETONE
cas no :67-64-1 rtecs no :AL3150000
area : USA type : REG

subject	specification	descriptor
FOOD	ADDIT	RSTR
TRANS		RSTR
STORE		RSTR
PACK		RSTR

; Summary - THIS SUBSTANCE IS INCLUDED ON A LIST OF SUBSTANCES USED TO PREPARE ADHESIVES WHICH MAY BE SAFELY USED AS COMPONENTS OF ARTICLES INTENDED FOR USE IN PACKAGING, TRANSPORTATION, OR HOLDING FOOD IN ACCORDANCE WITH THE FOLLOWING PRESCRIBED CONDITIONS: SUBSTA NCE MUST BE SEPARATED FROM THE FOOD BY A FUNCTIONAL BARRIER, MUST NOT EXCEED LIMITS OF GOOD MANUFACTURING PRACTICE USED WITH DRY FOODS, OR NOT EXCEED TRACE AMOUNTS AT SEAMS AND EDGE EXPOSURES WHEN USED WITH FATTY AND AQUEOUS FOODS. ALSO REGULATED BY SEA M INTEGRITY, LABELING STANDARDS, AND ANY PROVISION UNDER 21 CFR 175
entry date: NOV 1991 effective date: 1977

title: SUBSTANCES FOR USE ONLY AS COMPONENTS OF ADHESIVES
original : FEREAC, Federal Register, 42 , , 14534 , 1977
amendment: CFRUS*, Code of Federal Regulations, 21 , 175 , 105 , 1988

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File: 17.01 LEGAL **rn : 1309525**

systematic name:2-Propanone
common name :acetone
reported name :2-PROPANONE
cas no :67-64-1 rtecs no :AL3150000
area : USA type : REG

subject	specification	descriptor
CLASS	INDST	RQR
AIR	EMI	RQR
AQ	EMI	RQR

5000 (2270); Summary - RELEASES OF THIS HAZARDOUS SUBSTANCE, IN QUANTITIES EQUAL TO OR GREATER THAN ITS REPORTABLE QUANTITY (RQ), REPORTED AS [LBS (KG)!, ARE SUBJECT TO REPORTING TO THE NATIONAL RESPONSE CENTER UNDER THE COMPREHENSIVE ENVIRONMENTAL RESPONSE, COMPENSATION, AND LIABILITY ACT. (#)- RQ IS SUBJECT TO CHANGE
entry date: SEP 1991 effective date: 1990

title: CERCLA: LIST OF HAZARDOUS SUBSTANCES AND REPORTABLE QUANTITIES
original : CFRUS*, Code of Federal Regulations, 40 , 302 , 4 , 1990
amendment: CFRUS*, Code of Federal Regulations, 40 , 302 , 4 , 1990

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File: 17.01 LEGAL **rn : 1323003**

systematic name:2-Propanone
common name :acetone
reported name :ACETONE

cas no :67-64-1 rtecs no :AL3150000
area : USA type : REG

subject	specification	descriptor
CLASS	PESTI	RQR
MANUF	PESTI	PRMT
FOOD	ADDIT	RQR

CASE NAME ACETONE; Summary - THIS SUBSTANCE IS INCLUDED ON A LIST OF ACTIVE INGREDIENTS CONTAINED IN A PRODUCT FIRST REGISTERED BEFORE NOVEMBER 1, 1984, FOR WHICH A REGISTRATION STANDARD HAS NOT BEEN ISSUED. PUBLICATION OF THIS LIST INITIATES AN ACCELERATED REREGISTRATION AND DATA C ALL-IN FOR PRODUCTS CONTAINING THE LISTED ACTIVE INGREDIENTS.
entry date: JAN 1992 effective date: 1989

title: FEDERAL INSECTICIDE, FUNGICIDE, AND RODENTICIDE ACT PESTICIDES REQUIRED TO BE REREGISTERED; LIST D
original : FEREAC, Federal Register, 54 , 204 , 43388 , 1989
amendment: FEREAC, Federal Register, 54 , 204 , 43388 , 1989

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File: 17.01 LEGAL **rn : 1324017**

systematic name:2-Propanone
common name :acetone
reported name :ACETONE
cas no :67-64-1 rtecs no :AL3150000
area : USA type : REG

subject	specification	descriptor
AQ	GRND	MONIT
AQ	GRND	MXL

; Summary - THIS LIST IS REQUIRED ONLY FOR GROUND-WATER MONITORING AT RCRA LAND BASED HAZARDOUS WASTE DISPOSAL UNITS. THIS FINAL RULE WILL REQUIRE THAT AN ANALYSIS OF ALL THE CONSTITUENTS OF THIS LIST BE PERFORMED ON THE GROUND WATER TAKEN FROM WELLS SURROUNDING TH OSE UNITS. THIS ANALYSIS TAKES PLACE WHEN GROUND-WATER CONTAMINATION IS FIRST DETECTED, AND THEN AGAIN ONCE PER YEAR 40 CFR 264. WHEN A LISTED CONSTITUENT IS FOUND TO BE PRESENT A BACKGROUND VALUE MUST BE SET IN COMPLIANCE WITH 40 CFR 264.98(H)(2) UNLE SS OTHERWISE STATED.
entry date: SEP 1991 effective date: 1987

title: LIST (PHASE 1) OF HAZARDOUS CONSTITUENTS FOR GROUND-WATER MONITORING FINAL RULE: INCLUDING MAXIMUM CONCENTRATION OF CONSTITUENT: FOR GROUNDWATER PROTECTION.
original : FEREAC, Federal Register, 52 , , 25947 , 1987
amendment: CFRUS*, Code of Federal Regulations, 40 , 264 , , 1990

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File: 17.01 LEGAL **rn : 1325006**

systematic name:2-Propanone
common name :acetone
reported name :ACETONE

cas no :67-64-1 rtecs no :AL3150000
area : USA type : REC

subject	specification	descriptor
SAFTY	OCC	MXL
USE	OCC	MXL

20000 PPM
entry date: OCT 1991 effective date: JUN1990

title: POCKET GUIDE TO CHEMICAL HAZARDS
original : XPHPAW, US PUBLIC HEALTH SERVICE PUBLICATION, 90 , 117 , 30 , 1990
amendment: XPHPAW, US PUBLIC HEALTH SERVICE PUBLICATION, 90 , 117 , 30 , 1990

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File: 17.01 LEGAL **rn : 1332196**

systematic name:2-Propanone
common name :acetone
reported name :ACETONE
cas no :67-64-1 rtecs no :AL3150000
area : USA type : REG

subject	specification	descriptor
WASTE	INDST	CLASS
STORE		RQR
TRNSP	REMOV	RQR

IGNITABLE; Summary - THIS CHEMICAL, IF DISCARDED, MUST BE TREATED AS AN ACUTE HAZARDOUS WASTE. ACUTE HAZARDOUS WASTES REGULATIONS ARE MORE RESTRICTIVE FOR EXCLUSION. ANY RESIDUE OF THIS CHEMICAL LABELED AS ACUTELY HAZARDOUS AND REMAINING IN A CONTAINER, OR AN INNER LINER R EMOVED FROM A CONTAINER, IS CONSIDERED A HAZARDOUS WASTE IF DISCARDED UNLESS TRIPLE RINSING OR OTHER CLEANING MEASURES ARE TAKEN (40 CFR 261.33E).
entry date: JAN 1992 effective date: 1980

title: RCRA-RESOURCE AND CONSERVATION RECOVERY ACT: DISCARDED COMMERCIAL CHEMICAL PRODUCTS, OFF-SPECIFICATION SPECIES, CONTAINER RESIDUES, AND SPILL RESIDUES THEREOF.
original : FEREAC, Federal Register, 45 , , 78541 , 1980
amendment: CFRUS*, Code of Federal Regulations, 40 , 261 , 33 , 1990

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File: 17.01 LEGAL **rn : 1332565**

systematic name:2-Propanone
common name :acetone
reported name :2-PROPANONE
cas no :67-64-1 rtecs no :AL3150000
area : USA type : REG

subject	specification	descriptor
WASTE	INDST	CLASS
STORE		RQR
TRNSP	REMOV	RQR

IGNITABLE; Summary - THIS CHEMICAL, IF DISCARDED, MUST BE TREATED AS AN ACUTE HAZARDOUS WASTE. ACUTE HAZARDOUS WASTES REGULATIONS ARE MORE RESTRICTIVE FOR EXCLUSION. ANY RESIDUE OF THIS CHEMICAL LABELED AS ACUTELY HAZARDOUS AND REMAINING IN A CONTAINER, OR AN INNER LINER R EMOVED FROM A CONTAINER, IS CONSIDERED A HAZARDOUS WASTE IF DISCARDED UNLESS TRIPLE RINSING OR OTHER CLEANING MEASURES ARE TAKEN (40 CFR 261.33E).
entry date: JAN 1992 effective date: 1980

title: RCRA-RESOURCE AND CONSERVATION RECOVERY ACT: DISCARDED COMMERCIAL CHEMICAL PRODUCTS, OFF-SPECIFICATION SPECIES, CONTAINER RESIDUES, AND SPILL RESIDUES THEREOF.
original : FEREAC, Federal Register, 45 , , 78541 , 1980
amendment: CFRUS*, Code of Federal Regulations, 40 , 261 , 33 , 1990

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File: 17.01 LEGAL **rn : 1334044**

systematic name:2-Propanone
common name :acetone
reported name :ACETONE
cas no :67-64-1 rtecs no :AL3150000
area : USA type : REG

subject	specification	descriptor
USE		RQR
PACK		RQR

THIS SUBSTANCE IS LISTED AS AN ADJUVANT OF RELEASE AGENTS, WAXES, AND DISPERSANTS.; Summary - THIS SUBSTANCE IS INCLUDED IN A LIST OF RESINOUS AND POLYMERIC COATINGS WHICH MAY BE USED AS THE FOOD CONTACT SURFACE OF ARTICLES IF THE COATING IS APPLIED AS A CONTINUOUS FILM PRODUCED FROM ANY BASIC OLEFIN POLYMER LISTED IN 21 CFR 177.1520 1988 AND FOR MULATED FROM OPTIONAL SUBSTANCES WHICH ARE RECOGNIZED AS SAFE FOR USE IN OR ON FOOD AND FROM SUBSTANCES SUBJECT TO LIMITATIONS AS DESCRIBED HERE.
entry date: NOV 1991 effective date: 1977

title: INDIRECT FOOD ADDITIVES: ADHESIVES AND COMPONENTS OF COATINGS FOR POLYOLEFIN FILMS
original : FEREAC, Federal Register, 42 , , 14534 , 1977
amendment: CFRUS*, Code of Federal Regulations, 21 , 175 , 320 , 1988

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File: 17.01 LEGAL **rn : 1336032**

systematic name:2-Propanone
common name :acetone
reported name :2-PROPANONE
cas no :67-64-1 rtecs no :AL3150000
area : USA type : REG

subject	specification	descriptor
AIR	EMI	RQR
SOIL	EMI	RQR
AQ	EMI	RQR
MANUF	EMI	RQR

; Summary - FACILITIES THAT EXCEEDED A MANUFACTURING, IMPORTATION, OR PROCESSING THRESHOLD OF 25,000 LBS OR THE USE OF 10,000 LBS FOR THIS CHEMICAL MUST REPORT TO EPA ANY RELEASES OF THE CHEMICAL (OR CATEGORY CHEMICAL) TO AIR, LAND, WATER, POTW, UNDERGROUND INJECTIO N, OR OFF SITE TRANSFER. THIS REGULATION COVERS STANDARD INDUSTRIAL CLASSIFICATION (SIC) CODES 20-39 ONLY).
entry date: OCT 1991 effective date: 1987

title: SUPERFUND AMENDMENTS AND REAUTHORIZATION ACT, TITLE III. EPCRA SECTION 313 LIST OF TOXIC SUBSTANCES
original : CFRUS*, Code of Federal Regulations, 40 , 372 , 65 , 1988
amendment: CFRUS*, Code of Federal Regulations, 40 , 372 , 65 , 1988

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File: 17.01 LEGAL **rn : 1340604**

systematic name:2-Propanone
common name :acetone
reported name :ACETONE
cas no :67-64-1 rtecs no :AL3150000
area : USA type : REC

subject	specification	descriptor
AIR	OCC	TLV

Time Weighted Avg (TWA) 750 ppm, 1780 MG/M3, skin; Short Term Exposure Limit (STEL) 1000 ppm, 2380 MG/M3; Summary - THIS THRESHOLD LIMIT VALUE IS INTENDED FOR USE IN THE PRACTICE OF INDUSTRIAL HYGIENE AS A GUIDELINE OR RECOMMENDATION IN THE CONTROL OF POTENTIAL HEALTH HAZARDS.
entry date: DEC 1991 effective date: 1989

title: THRESHOLD LIMIT VALUES
original : ACGIH*, Threshold Limit Values and Biological Exposure Indices, , , 11 , 1989
amendment: ACGIH*, Threshold Limit Values and Biological Exposure Indices, , , 11 , 1991

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File: 17.01 LEGAL **rn : 1402094**

systematic name:2-Propanone
common name :acetone
reported name :ACETONE
cas no :67-64-1 rtecs no :AL3150000
area : EEC type : REG

subject	specification	descriptor
FOOD		RQR
FOOD		MXL
FOOD		RSTR

THE SUBSTANCE MAY BE USED FOR THE MANUFACTURE OF REGENERATED CELLULOSE FILM WHICH IS INTENDED TO OR DOES COME INTO CONTACT WITH FOODSTUFFS. IT MAY BY USED AS SOLVENT; MAXIMUM TOTAL QUANTITY OF ALL SOLVENTS: 0.6MG/DM2 ON THE SIDE IN CONTACT WITH FOODSTUFFS.
entry date: OCT 1987 effective date: 01APR1987

title: COUNCIL DIRECTIVE OF 25 APRIL 1983 ON THE APPROXIMATION OF THE LAWS OF THE MEMBER STATES RELATING TO MATERIALS AND ARTICLES MADE OF REGENERATED CELLULOSE FILM INTENDED TO COME INTO CONTACT WITH FOODSTUFFS. (83/229/EEC).
original : OJEC**, Official Journal of the European (Communities)/Union, L123 , , 31 , 1983
amendment: OJEC**, Official Journal of the European (Communities)/Union, L228 , , 32 , 1986

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File: 17.01 LEGAL **rn : 1402327**

systematic name:2-Propanone
common name :acetone
reported name :ACETONE
cas no :67-64-1 rtecs no :AL3150000
area : EEC type : REG

subject	specification	descriptor
FOOD	INDST	PRMT

THIS SUBSTANCE, PROVIDED IT SATISFIES THE PURITY CRITERIA LAID DOWN, MAY BE USED AS AN EXTRACTION SOLVENT DURING THE PROCESSING OF RAW MATERIALS, OF FOODSTUFFS, OF FOOD COMPONENTS, OR OF FOOD INGREDIENTS. IT SHOULD BE USED IN COMPLIANCE WITH GOOD MANUFACTURING PRACTICE FOR ALL USES: I.E. ITS USE SHOULD RESULT IN THE PRESENCE OF RESIDUES OR DERIVATIVES IN TECHNICALLY UNAVOIDABLE QUANTITIES PRESENTING NO DANGER TO HUMAN HEALTH.
entry date: 1991 effective date: 13JUN1991

title: COUNCIL DIRECTIVE OF 13 JUNE 1988 ON THE APPROXIMATION OF THE LAWS OF THE MEMBER STATES ON EXTRACTION SOLVENTS USED IN THE PRODUCTION OF FOODSTUFFS AND FOOD INGREDIENTS. (88/344/EEC).
original : OJEC**, Official Journal of the European (Communities)/Union,

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File: 17.01 LEGAL **rn : 1421907**

systematic name:2-Propanone
common name :acetone
reported name :ACETONE
cas no :67-64-1 rtecs no :AL3150000
area : EEC type : REG

subject	specification	descriptor
CLASS		CLASS
LABEL		RQR
PACK		RQR

CLASS: F - HIGHLY FLAMMABLE; HIGHLY FLAMMABLE (R 11). LABEL: F - HIGHLY FLAMMABLE; HIGHLY FLAMMABLE (R 11); KEEP CONTAINER IN A WELL-VENTILATED PLACE (S 9); KEEP AWAY FROM SOURCES OF IGNITION - NO SMOKING (S 16); DO NOT BREATH GAS/FUMES/VAPOUR/SPRAY (APPROPRIATE WORDING TO BE SPECIFIED

BY THE MANUFACTURER) (S 23); TAKE PRECAUTIONARY MEASURES AGAINST STATIC DISCHARGES (S 33).
entry date: APR 1992 effective date: 1JUL1992

title: COUNCIL DIRECTIVE 67/548/EEC OF 27 JUNE 1967 ON THE APROXIMATION OF THE LAWS, REGULATIONS AND ADMINISTRATIVE PROVISIONS RELATING TO THE CLASSIFICATION, PACKAGING AND LABELLING OF DANGEROUS SUBSTANCES
original : OJEC**, Official Journal of the European (Communities)/Union, 196 , , 1 , 1967
amendment: OJEC**, Official Journal of the European (Communities)/Union, L 180 , , 79 , 1991

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File: 17.01 LEGAL **rn : 1645330**

systematic name:2-Propanone
common name :acetone
reported name :ACETONE
cas no :67-64-1 rtecs no :AL3150000
area : IMO type : REC

subject	specification	descriptor
TRNSP LABEL PACK	MARIN	CLASS

HAZARD CLASS: 3 = INFLAMMABLE LIQUID. PACKING GROUP: II = MEDIUM DANGER (I=GREAT DANGER - III=MINOR DANGER). UN NO. 1090
entry date: JAN 1991

amendment: !IMCOC*, International Maritime Dangerous Goods Code, , , 10004 , 1990

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File: 17.01 LEGAL **rn : 1744374**

systematic name:2-Propanone
common name :acetone
reported name :ACETONE
cas no :67-64-1 rtecs no :AL3150000
area : UN type : REC

subject	specification	descriptor
TRNSP LABEL PACK		CLASS

HAZARD CLASS: 3 = INFLAMMABLE LIQUID. PACKING GROUP: II = MEDIUM DANGER (I=GREAT DANGER - III=MINOR DANGER). UN NO. 1090
entry date: AUG 1990

amendment: !UNTDG*, UN Transport of Dangerous Goods, Recommendation prepared by theCommittee of Experts on the Transport of Dangerous Goods, , , 15 , 1989