1-OCTENE

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I. IDENTIFICATION

Chemical Name: 1-Octene
Synonyms: *a*-Octylene; *a*-Octene; C-8 *a*-Olefin
CAS Number: 111–66–0
Molecular Formula: C_8H_{16}
Structural Formula: CH_2=CH–$(CH)_5$–CH

II. CHEMICAL AND PHYSICAL PROPERTIES[1-4]

Physical State and Appearance: Colorless liquid
Odor Description: Hydrocarbon odor
Odor Threshold: 2.0 ppm
Molecular Weight: 112.21
Conversion Factors: 1 ppm = 4.59 mg/m^3
1 mg/m^3 = 0.22 ppm
Melting Point: -102°C (-151.6°F)
Boiling Point: 121–123°C (250–253°F)
Vapor Pressure: 15mm Hg at 20°C (68°F)
Saturated Vapor Concentration: 20,000 ppm at 20°C (68°F)
Vapor Density: 3.9
Flash Point (tagged closed cup): 10°C (50°F)
Flammability Limits: LEL–0.8%; UEL–6.7%
Autoignition Temperature: 221°C (430°F)
Specific Gravity: 0.72 g/ml
Solubility in Water: 4.1 mg/1 at 25°C (78°F)
Stability: Stable
Reactivity and Incompatibilities: May be oxidized and polymerize.
Avoid contact with heat and oxidizing agents.

III. USES AND VOLUME[4-6]

Synthesis of certain intermediates that are used in the manufacture of consumer products such as synthetic lubricating oils, dielectric fluids, low density polyethylene, special detergents, and PVC plasticizer alcohols manufacture. Production, based on 1995 reporting, is greater than 50×10^6 lbs/year.

IV. ANIMAL TOXICOLOGY DATA

A. Acute Toxicity and Irritancy

1. Oral Toxicity

LD_{50} (rat) > 5 ml/kg (>3.6 g/kg)[7]

2. Eye Toxicity

Minimal irritation to the conjunctiva of rabbits was observed 1 hr following exposure to the undiluted liquid. All signs of irritation cleared within 48 hr.[8]

3. Skin Absorption

LD_{50} (rabbit) > 2.0 ml/kg (>1.4 g/kg)[9]

4. Skin Irritation

Mild to moderate irritant in rabbits with Draize scores ranging from 1.8–3.4 from exposure to the undiluted liquid.[10]

5. Skin Sensitization

Ten guinea pigs (5M/5F) were treated with 0.5 ml of 1% 1-octene in ethanol under occlusion 1 day/week for 6 hr. The treatment continued for 3 weeks. A challenge dose was applied at original and virgin sites 2 weeks following the last induction dose. Under the conditions of this test, 1-octene is not a skin sensitizer.[11]

6. Inhalation Toxicity

Nine of ten rats died following exposure for 1 hr to a nominal saturated vapor concentration (19,000 ppm). The LC_{50} (rats, 4-hr exposure, nominal concentration) is 8050 ppm. The threshold for mortality is 6050 ppm. All deaths occurred during the exposure period with the primary effect being central nervous

system (CNS) related (depression and convulsion). 1-Octene can be aspirated into the lungs upon oral exposure to the liquid with the potential for cardiac arrest, respiratory failure and asphyxia.[12,13]

B. Subacute Toxicity

The potential of 1-octene to produce irritation to the skin from repeated exposure was evaluated in rabbits. A regimen of 0.2 ml of undiluted liquid, 5 days/week for a total of 20 applications gave results similar to those observed from a single exposure, i.e., a mild to moderate irritant.[10]

C. Subchronic Toxicity[12]

Male and female rats were exposed via gavage to a 1-octene at dose levels of 5, 50, or 500 mg/kg once daily 7 days/week for 13 weeks.[14] Each treatment group and the vehicle (soya-bean oil) control group contained 40 animals (20M, 20F). Clinical signs, body weight, and food consumption were recorded at regular intervals throughout the study. Hematology, clinical chemistry, urinalysis, gross, and microscopic pathological change and organ weights were evaluated at termination of the study. Treatment-related findings occurred only at 500 mg/kg and were focused primarily on the kidneys. Urine volume was increased in males, plasma creatinine concentration was increased in the females, and while only absolute kidney and liver weight was increased in the males, relative weight of both organs was increased in both sexes. No gross pathological changes were observed in either sex. Microscopic changes were seen only in the kidneys of male animals. These changes appear characteristic of hydrocargon-induced nephropathy due to the presence of a2m-globulin, a protein not present in humans. The results of this study indicate that 1-octene produces definite treatment-related toxicity to the kidneys of male rats. Although no histopathological change was noted in the females, the increase in relative kidney weight and plasma creatinine concentration in the females indicates a slight nephrotoxic effect at the highest dosage.

D. Chronic Toxicity/Carcinogenicity

No available data.

E. Reproductive/Developmental Toxicity

No available data.

F. Genotoxicity/Mutagenicity

1-Octene was negative in the *Salmonella typhimurium*/mammalian microsomal (Ames) assay in strains TA98, TA100, TA1535, TA1537, and TA1538 and in *Escherichia coli* strain WP2urvA. In each study the testing was conducted with and without metabolic activation.[15,16]

Two studies were conducted to assess potential to produce chromosomal aberrations in Chinese hamster ovary (CHO) cells *in vitro*. In the first study, 1-octene was negative both with and without metabolic activation.[17] In the second study, it was negative without metabolic activation. With activation the aberration rate just exceeded a 2-fold increase, but a clear dose-response relationship was absent.[18] 1-Octene was negative for induction of cell transformation in mouse BALB/3T3 cells *in vitro* with and without metabolic activation.[19]

The data from the package of mutagenicity studies indicate that 1-octene does not produce site-specific damage to the DNA, structural changes to the chromosome, or morphological transformation of cells in culture into a potential malignant state. Based on the weight of evidence from the results of these studies, 1-octene is not considered to be a genotoxic agent.

G. Metabolism/Pharmacokinetics[20,21]

1-Octene is metabolized via the 1,2-eposide to the 1,2-diol. An alternate pathway has been described wherein 1-octene is metabolized first to oct-1-en-3ol and subsequently to oct-1-en-3-one.

V. HUMAN USE AND EXPERIENCE

No data was located on occupational or other human exposure to 1-octene; however, the effects to humans are expected to be similar to those observed from animal exposure. The primary effects of overexposure in humans would include irritation to the respiratory tract, drowsiness, headache, dizziness, and nausea.[1]

VI. RATIONALE

1-Octene has a low order of acute toxicity from inhalation, dermal contact and ingestion. The toxicity is centered on direct effects to the lungs and to the central nervous system. The toxicity from acute inhalation exposure is immediate with no evidence of delayed effects. The dose-response curve is relatively steep with the threshold for mortality (4-hr exposure to 6050 ppm) being near the 4-hr LC_{50} of 8050 ppm. Contact with skin or eyes are of minimal hazard in the context of workplace exposure.

Although long-term exposure studies have not been conducted, 1-octene is not expected to be a carcinogen to humans. The weight of evidence indicates it is not genotoxic. Repeated oral exposure for 13 weeks produced no unexpected effects. The top dose of 500 mg/kg caused kidney damage in the male rats that

is considered species and sex-dependent and not pertinent to human risk assessment. The toxicity to the liver of males and females and to the kidneys of females was slight and without corroborative histopathological change. The increase in plasma creatinine in the top dose females may be secondary to dehydration, increased protein catabolism or renal damage, particularly at the glomerular level. However, no additional findings support any of these effects. The no observable adverse effect level (NOAEL) in this subchronic study is judged to be near the top dose of 500 mg/kg. The concentration that would allow an equivalent dose to humans in 8 hr (55kg person; $8m^3$ inhaled air) is 750 ppm of 1-octene.

The data generated from repeated exposure to rats provides the base from which to establish an OEL for 1-octene. An added measure of confidence is gained from the exposure regimen of 7 days/week in the 13-week study compared to exposure of 5 days/week in the workplace. It is unlikely that the top dose of 1-octene produced any significant organ damage given the lack of histopathological change. There were increases in serum creatinine, but this variable is not a reliable index of renal damage until a relatively large portion of functional capacity has been damaged. Such damage should be reflected in observable pathological change. The data for the C_6–C_{10} compounds in the olefin series show similar acute toxicity qualitatively and quantitatively and 1-hexene and 1-octene exhibit similarity in threshold dosages for adverse effects from repeated exposure studies. The NOAEL for 1-hexene, from data generated in a 90-day inhalation study,[22] is similar to the NOAEL of 500 mg/kg observed for 1-octene in the 90-day gavage study when compared on a mg/kg basis. There does not appear to be unique toxicity associated with any of the chemicals in this series. The available toxicological data for the C_6–C_{10} olefins support the justification for establishing similar values for each respective OEL.

VII. RECOMMENDED OEL

8-hr time-weighted average (TWA): 75 ppm (344 mg/m^3)

VIII. REFERENCES

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