Diallylamine
[124-02-7]

Review of Toxicological Literature

Prepared for

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EXECUTIVE SUMMARY

Diallylamine [124-02-7] is a flammable liquid and a dangerous fire hazard when exposed to heat or flame. It is prepared by refluxing diallylcyanamide with diluted sulfuric acid. It has also been prepared by treating allylamine with allyl bromide or allyl chloride. Diallylamine is currently produced by the Hoechst Celanese Corporation. Production and import volumes were not available.

Diallylamine was used earlier in the production of the herbicide Randox® which was discontinued by Monsanto in 1984. It is currently used in the production of *N*,*N*-diallyl-2,2-dichloroacetamide [37764-25-3].

No data or reports on environmental occurrence or human exposure were found for diallylamine. Diallylamine is regulated by the U.S. Department of Transportation as a hazardous material for the purpose of transportation.

In humans exposed to diallylamine via inhalation for 5 minutes, the lowest toxic concentration (TC_{Lo}) observed was 5 ppm (20 mg/m^3). However, diallyamine is not considered intolerable to humans at 70 ppm (280 mg/m^3). Following acute or repeated exposure to mono-, di-, and tri-allylamines for several years, six male workers experienced symptoms including mouth, jaw, and tooth pain; chest pain/tightness; bloodshot eyes; sore throat; runny nose and eyes; neck pain; dizziness; nausea and vomiting; breathing difficulty; and headache.

Based on the American National Standards Institute's (ANSI) toxicity classification for rat acute oral LD_{50} and rabbit acute dermal LD_{50} data, diallylamine with reported lowest values of 316 mg/kg (3.25 mmol/kg) and 280 mg/kg (2.9 mmol/kg), respectively, is classified as toxic. The 4-hour and 8-hour inhalation LD_{50} values for rats were 2755 ppm (10950 mg/m^3) and 795 ppm (3160 mg/m^3), respectively. The acute intraperitoneal LD_{50} for mice was 187 mg/kg (1.92 mmol/kg). Diallylamine was found to be a potent respiratory irritant in male OF_1 mice, with a RD_{50} (50% reduction in respiratory frequency) of 4 ppm (16 mg/m^3) for the upper respiratory tract and 56 ppm (220 mg/m^3) for the lower respiratory tract.

Diallylamine did not induce *his* gene mutations in *Salmonella typhimurium* in the presence or absence of rat/hamster liver S9 metabolic activation. No other genotoxicity data were available.

No chronic, reproductive, carcinogenicity, or immunotoxicity data of diallylamine were available.

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TABLES

1.0 BASIS OF NOMINATION TO THE ICCEC

The nomination of diallylamine [124-02-7] to the ICCEC is based on high production volumes and the lack of toxicity data.

2.0 IDENTIFICATION AND CLASSIFICATION

Diallylamine
[124-02-7]

$H_2C{=}CH{-}CH_2{-}NH{-}CH_2{-}CH{=}CH_2$

2.1 Chemical Identification

Diallylamine ($C_6H_{11}N$, mol. wt. = 97.16) is also called:

2-Propen-1-amine, *N*-2-propenyl- (9CI)

Di-2-propenylamine

N-2-Propenyl-2-propen-1-amine

Diallylamine has the designation for shipping UN 2359.

2.2 Physical-Chemical Properties

Property	Information	Reference
Physical State	Liquid	Budavari (1996)
Boiling Point, °C	111	Weast and Astle (1980)
Melting Point, °C	-100	Lewis (1992)
Specific Gravity at 20°C	0.7889	Lewis (1992)
Odor	Recognizable but refreshing odor at 4-9 ppm (16-36 mg/m^3)	HSDB (1996)
Odor Threshold	2-9 ppm (8-36 mg/m^3)	HSDB (1996)
Solubility:		

Water	Soluble in water	Lewis (1992)
Organic Solvents	Soluble in ethyl alcohol and diethyl ether	Weast and Astle (1980)

Diallylamine is a flammable liquid and a dangerous fire hazard when exposed to heat or flame. Vapors may travel to a source of ignition and flash back, while containers may explode in the heat of a fire. The compound may present a vapor explosion hazard indoors, outdoors, or in sewers. When heated to decomposition, diallylamine emits toxic fumes of NO_x (Lewis, 1992).

2.3 Purity and Commercial Availability

Diallylamine is available from Hoechst Celanese Corp., Chemical Group Specialty Chemicals. Information on purity was not available.

3.0 PRODUCTION PROCESS ANALYSIS

Diallylamine has been prepared by treating allylamine with allyl bromide or allyl chloride or by refluxing diallylcyanamide with diluted sulfuric acid (Budavari, 1996).

A mixture of monoallylamine, diallylamine, and triallylamine is produced commercially by a reaction of allyl alcohol and ammonia with palladium acetylacetonate at 110°C for 4 hours, using 1,2-bis(diphenylphosphono)propane as the catalyst and propylene glycol as the solvent. Reaction of allyl alcohol with acetylacetone at 85°C for 3 hours gives 70% monoallylamine and 26% diallylamine (Schweizer et al., 1978).

4.0 PRODUCTION AND IMPORT VOLUMES

Circa 1977, Shell Company was listed as a producer of diallylamine (TSCAPP, 1983). Production volume was not reported. In 1991, 2 million pounds (900,000 kg) of diallylamine was used to produce *N*,*N*-diallyl-2,2-dichloroacetamide (see **Section 5.0**). Diallylamine is listed

as being produced currently by Hoechst Celanese Corporation; production volume data was not reported (SRI Int., 1996).

5.0 USES

Diallylamine was used in the reaction with chloroacetyl chloride to produce the herbicide *N*,*N*-diallyl-2-chloroacetamide (allidochlor; Randox®); production was discontinued by Monsanto in 1984 (Sine, 1991; Budavari, 1996). Diallylamine is currently used to produce *N*,*N*-diallyl-2,2-dichloroacetamide [37764-25-3], which is used as a safener (a safener prevents objectionable changes in mixtures of two or more substances that would otherwise be incompatible) in the pre-emergence herbicide EPTC (*S*-ethyl di-*N*,*N*-propylthiocarbamate; EPTAM®) (Sine, 1991; SRI Int., 1997).

6.0 ENVIRONMENTAL OCCURRENCE

No data or reports on environmental occurrence were found.

7.0 HUMAN EXPOSURE

NIOSH has not conducted a survey on diallylamine (RTECS, 1996). The only human exposure data found are presented in **Section 9.1**.

8.0 REGULATORY STATUS

	Regulatory Action	Effect of Regulation/Other Comments
DO	46 CFR 172. Subpart B. Table of Hazardous Material and Special	Diallylamine is listed as a hazardous material for the

	Regulatory Action	Effect of Regulation/Other Comments
T	Provisions.	purpose of transportation. Labeling must indicate that it is a flammable liquid, a poison, and corrosive. Quantity limitations are as follows: Passenger aircraft or railcar, 1 L; cargo aircraft, 5 L.

9.0 TOXICOLOGICAL DATA

Summary: In humans exposed to diallylamine via inhalation for 5 minutes, the lowest toxic concentration (TC_{Lo}) observed was 5 ppm (20 mg/m^3). Diallylamine is not intolerable to humans at 70 ppm (280 mg/m^3). Following acute or repeated exposure to mono-, di-, and tri-allylamines for several years, six male workers experienced symptoms including mouth, jaw, and tooth pain; chest pain/tightness; bloodshot eyes; sore throat; runny nose and eyes; neck pain; dizziness; nausea and vomiting; breathing difficulty; and headache. Based on the American National Standards Institute's (ANSI) toxicity classification for rat acute oral LD_{50} and rabbit acute dermal LD_{50} data, diallylamine is classified as a toxic compound. The lowest reported LD_{50} values for rat acute oral and rabbit acute dermal exposures were 316 mg/kg (3.25 mmol/kg) and 280 mg/kg (2.9 mmol/kg), respectively. The 4-hour and 8-hour inhalation LD_{50} values for rats were 2755 ppm (10950 mg/m^3) and 795 ppm (3160 mg/m^3), respectively. The acute intraperitoneal LD_{50} for mice was 187 mg/kg (1.92 mmol/kg). Diallylamine exhibited a high respiratory irritant potency in male OF_1 mice, with a RD_{50} (50% reduction in respiratory frequency) of 4 ppm. Diallylamine did not induce *his* gene mutations in *Salmonella typhimurium* in the presence or absence of rat or hamster liver S9 metabolic activation.

9.1 Human Data

The lowest toxic concentration (TC_{Lo}) in humans (sex not provided) exposed to diallylamine for 5 minutes (dose range not provided) was 5 ppm (20 mg/m^3) (Archiv. Env. Health, 1960; cited by RTECS, 1996).

The smell of diallylamine at concentrations of 2-9 ppm (8-36 mg/m^3) is recognizable, but not unpleasant, to human subjects. At exposure to 22 ppm (87 mg/m^3), some subjects develop

mucous membrane irritation and chest discomfort. However, diallylamine is not intolerable at 70 ppm (280 mg/m^3) (Hine et al., 1960; cited by Beard and Noe, 1981, and by HSDB, 1996).

Six male workers (ages 33-49) were exposed, either acutely or repeatedly over several years, to mono-, di-, and tri-allylamines. The workers experienced symptoms (which appeared rapidly after exposure) including mouth, jaw, and tooth pain; chest pain/tightness; bloodshot eyes; sore throat; runny nose and eyes; neck pain; dizziness; nausea and vomiting; breathing difficulty; and headache (Shell Oil Co., 1982).

9.2 General Toxicology

9.2.1 Chemical Disposition, Metabolism, and Toxicokinetics

No data were found on the chemical disposition, metabolism, and toxicokinetics of diallylamine.

9.2.2 Acute Exposure

LD_{50}/LC_{50} data are represented in **Table 1**. Studies describing acute toxicity effects other than LD_{50}/LC_{50} are presented in **Table 2.**

9.2.2.1 Dermal Exposure

In rats (strain and age not provided), application of 0.1 mL diallylamine to a 1 cm^2 area of shaved abdominal skin caused severe necrosis and death (Patty, 1963; cited by HSDB, 1996).

Diallylamine was corrosive on intact rabbit skin (strain, age, and dose not provided) exposed for 4 hours and observed for 4, 24, and 48 hours after treatment (ICI Americas, 1975). Severe necrosis was present at all observation times.

Diallylamine was applied for 24 hours to the intact skin of New Zealand white rabbits, at doses of 215, 464, 1000 or 2150 mg/kg (2.21, 4.78, 10.29 or 22.13 mmol/kg) (ICI Americas,

1975). Necrosis, erythema, and edema were observed. All deaths occurred within 24 hours of dosing, most in less than 4 hours.

Table 1. LD_{50}/LC_{50} Data for Diallylamine

Route	Species/sex	LD_{50} Value (range) mg/kg	mmol/kg	Reference
dermal	rabbit	280	2.9	Am. Ind. Hyg. Assoc. J., 1962; cited by RTECS, 1996
	rabbit	562 (383-825)	5.8 (3.94-8.49)	ICI Americas, 1975
intraperitoneal	mouse	187	1.92	Archiv. Env. Health, 1960; cited by RTECS, 1996
oral (gavage)	rat (female)	501 (344-730)	5.16 (3.54-7.51)	ICI Americas, 1975
	(male)	316 (205-488)	3.25 (2.11-5.02)	
oral	rat	578	5.95	Stamathis, 1985; cited by Boor and Hysmith, 1987
	mouse	355	3.65	Izmerov et al., 1982; cited by RTECS, 1996
		LC_{50} Data ppm	mg/m^3	
inhalation (4h)	rat	2755	10950	Stamathis, 1985; cited by Boor and Hysmith, 1987
inhalation (8h)		795	3160	

Table 2. Acute Toxicity of Diallylamine (continued)

Species, Strain, Age	Animals	Chemical Form, Purity	Dose	Exposure/ Observation Period	Results/Comments	Reference
9.2.2.1 Dermal Exposure						
rat (strain and age n.p.)	number and sex n.p.	diallylamine, purity n.p.	0.1 mL applied to 1 cm^2 area of shaved abdominal skin	n.p.	Exposure caused severe necrosis and death, but the incidence of mortality was not provided.	HSDB (1996; citing Patty, 1963)
rabbit (strain and age n.p.)	exposed: 6 (sex n.p.) controls: none	diallylamine, purity n.p.	n.p. (dose was applied to intact skin)	4 h; observations were made 4, 24, and 48 h after treatment	Diallylamine was corrosive and caused severe necrosis in the skin of all rabbits at all observation times. Mortality data were not provided	ICI Americas (1975)
rabbit, New Zealand white albino (age n.p.)	exposed: 2 M, 2 F per dose controls: n.p.	diallylamine, purity n.p.	215, 464, 1000, or 2150 mg/kg (2.21, 4.78, 10.29, or 22.13 mmol/kg) applied to intact abdominal skin	24 h; 14 days	Necrosis, erythema, and edema were observed. All deaths occurred within 24 hours of dosing; most in less than 4 hours.	ICI Americas (1975)
9.2.2.2. Oral Exposure						
rat, Sprague-Dawley (age n.p.)	exposed: 5 M, 5 F per dose controls: none	diallylamine, purity n.p.	F: 215, 464, 1000, or 4640 mg/kg (2.21, 4.78, 10.29, or 47.76 mmol/kg) by gavage M: 215, 464, 1000 or 2150 mg/kg (2.21, 4.78, 10.29, or 22.13 mmol/kg) by gavage	single dose; 14 days	F: No observable effects occurred at 215 mg/kg (2.21 mmol/kg), while at higher doses, deaths occurred within 20 hours after dosing. M: Dose-related depression (lethargy) occurred in all exposed groups. At the lower doses, death sometimes occurred 1 week or more after dosing, while at 1000 mg/kg (10.29 mmol/kg) and higher, all male rats died within 2 hours.	ICI Americas (1975)
9.2.2.3 Ocular Exposure						
rabbit (strain and age n.p.)	n.p.	diallylamine, purity n.p.	50 mg (0.52 mmol); eyes were rinsed for 20 sec. after dosing	n.p.	Diallylamine was severely irritating.	RTECS (1996; citing Archiv. Env. Health, 1960)
9.2.2.3 Ocular Exposure (continued)						

Table 2. Acute Toxicity of Diallylamine (continued)

Species, Strain, Age	Animals	Chemical Form, Purity	Dose	Exposure/ Observation Period	Results/Comments	Reference
rabbit, New Zealand (age n.p.)	exposed: 6 (sex not given) controls: untreated eye served as control	diallylamine, purity n.p.	0.1 mL in 1 eye of each rabbit	single dose; observations were made 1, 2, 3, and 7 days after dosing	Severe corneal damage was observed. In the conjunctivae, severe erythema, chemosis (edema of the bulbar conjunctiva), and discharge were observed. There were no signs of remission 7 days after treatment.	ICI Americas (1975)
9.2.2.4 Inhalation Exposure						
mouse (strain and age n.p.)	n.p.	diallylamine, purity n.p.	0.88 mM (1.9%)	n.p.	Almost all mice died within 10 minutes. No other details were provided.	Beard and Noe (1981)
mouse, OF_1 (age n.p.)	exposed: 6 M	diallylamine, purity n.p.	23-133 ppm (91-528 mg/m^3)	1 h exposure period (15 min measurement period) via head only for upper respiratory tract; 2 h exposure via tracheal cannulation for lower respiratory tract.	Classified as a potent upper respiratory tract irritant with a RD_{50} (the concentration of a sensory irritant responsible for a decrease of 50% in the respiratory frequency) of 4 ppm (16 mg/m^3), and a potent pulmonary irritant with a RD_{50} of 56 ppm (224 mg/m^3).	Gagnaire et al. (1993)
rat, Sprague-Dawley albino (age n.p.)	exposed: 5 M, 5 F controls: none	diallylamine, purity n.p.	> 6.3 mg/L/h (> 0.065 mmol/L/h)	1 h; 14 days	Moderate eye irritation and depression (lethargy) were observed during exposure and persisted after cessation of treatment, but the rats appeared normal after 24 hours. No deaths occurred.	ICI Americas (1975)

Abbreviations: f= female; m = male ; n.p. = not provided

9.2.2.2 Oral Exposure

Female Sprague-Dawley rats were administered, by gavage, a single dose of diallylamine at 215, 464, 1000, and 4640 mg/kg (2.21, 4.78, 10.29, and 47.76 mmol/kg) (ICI Americas, 1975). No observable effects occurred at the lowest dose tested, while mortality was induced at higher doses within 20 hours after dosing. In the same study, male Sprague-Dawley rats administered diallylamine at 215, 464, 1000, and 2150 mg/kg (2.21, 4.78, 10.29, and 22.13 mmol/kg) exhibited a dose-dependent depression (lethargy) in all exposed groups. At lower doses, death sometimes occurred a week or later after dosing, while at 1000 mg/kg (10.29 mmol/kg) and higher, all male rats died within 2 hours.

9.2.2.3 Ocular Exposure

In rabbits (strain and age not specified), application of 50 mg (0.52 mmol) diallylamine to the eyes was severely irritating (Archiv. Env. Health, 1960; cited by RTECS, 1996). Eyes were rinsed for 20 seconds following exposure, but the duration of exposure was not specified.

In New Zealand White rabbits, severe corneal damage, severe erythema, chemosis (edema of the bulbar conjunctiva), and discharge of the conjunctivae were observed one, two, three, and seven days after application of 0.1 mL diallylamine into one eye (ICI Americas, 1975). It was not specified at what time which damage occurred, but there were no signs of remission at seven days after treatment.

9.2.2.4 Inhalation Exposure

Almost all mice (strain and age not provided) exposed to 0.88 mM (1.9%) diallylamine died after 10 minutes (Beard and Noe, 1981).

The ability of diallylamine (23-133 ppm; 91-528 mg/m^3) to induce expiratory bradypnea indicative of upper airway irritation was evaluated in male OF_1 mice during a 15-minutes oronasal exposure (Gagnaire et al., 1993). The RD_{50} (the concentration of a sensory irritant responsible

for a decrease of 50% in the respiratory frequency) for diallyamine was 4 ppm (16 mg/m^3), indicative of strong irritancy. In the same study, diayllamine was evaluated also for pulmonary irritation by measuring the decrease in respiratory rate of non anesthetized tracheally cannulated mice during a 2-hour exposure period. Under these conditions, diayllamine was highly irritating with a RD_{50} of was 56 ppm (220 mg/m^3).

Moderate eye irritation and depression (lethargy) were observed in Sprague-Dawley albino rats during a 1-hour inhalation exposure to greater than 6.3 mg/L/h (> 0.065 mmol/L/h) diallylamine (ICI Americas, 1975). Symptoms persisted after cessation of treatment, but the rats appeared normal after 24 hours.

9.2.3 Short-term and Subchronic Exposures

The study described in this section is presented in **Table 3.**

Repeated exposure of rats (strain and age not specified) to diallylamine [i.e., fifty 7-hour exposures to 200 ppm (800 mg/m^3)] caused changes in liver and kidney weights, a reduction in growth, and mortality (Beard and Noe, 1981). Chemical pneumonia and myocarditis were also present in some rats.

9.2.4 Chronic Exposures

No data on chronic exposures were found.

9.3 Reproductive Effects

No data on reproductive effects were found.

9.4 Carcinogenicity

No data on carcinogenicity were found.

9.5 Genotoxicity

The study described in this section is presented in **Table 4.**

As reported by Zeiger et al. (1987), diallylamine did not induce *his* gene mutations in *Salmonella typhimurium.* Strains TA98, TA100, TA1535, and TA1537 were exposed to doses ranging from 33 to 10,000 µg/plate (0.4 to 103 µmol/plate) using the pre-incubation method in either the presence or absence of 10% rat or hamster liver S9 metabolic activation. No increase in the number of revertants was observed in any strain under any S9 condition.

Table 3. Short-term and Subchronic Toxicity of Diallylamine

Species, Strain, Age	Animals	Chemical Form, Purity	Dose	Exposure/Observation Period	Results/Comments	Reference
rat (strain and age n.p.)	n.p.	diallylamine, purity n.p.	Fifty 7-hour exposures to 200 ppm (800 mg/m^3)	n.p.	Exposure caused changes in liver and kidney weights, and in growth. Chemical pneumonia and myocarditis were also present in some rats. Deaths occurred, but the incidence of mortality was n.p.	Beard and Noe (1981)

Abbreviations: n.p. = not provided

Table 4. Genotoxicity of Diallylamine

Test System	Biological Endpoint	S9 Metabolic Activation	Chemical Form, Purity	Dose	Endpoint Response	Comments	Reference
S. typhimurium strains TA98, TA100, TA1535, TA1537	*his* reverse gene mutations	± 10% rat or hamster S9	diallylamine, purity n.p.	33, 100, 333, 1000, 3333, and 10,000 µg/plate (0.34, 1.03, 3.40, 10.29, 34.30, and 102.92 µmol/plate)	negative/negative	Pre-incubation method was used.	Zeiger et al. (1987)

Abbreviations: n.p. = not provided

9.6 Immunotoxicity

No data on immunotoxicity were found.

10.0 STRUCTURE-ACTIVITY RELATIONSHIPS

The ability of 16 aliphatic amines to induce upper airway irritation and pulmonary toxicity in male OF_1 mice was evaluated (see **Section 9.2.2.4**) (Gagnaire et al., 1993). The tested compounds were isopropylamine, *n*-propylamine, isobutylamine, diisopropylamine, *n*-butylamine, cyclohexylamine, di-*n*-propylamine, *n*-pentylamine, *tert*-octylamine, *n*-hexylamine, diisobutylamine, di-*n*-butylamine, *n*-heptylamine, *n*-octylamine, allylamine, and diallylamine. For each chemical, the RD_{50} (the concentration of a sensory irritant responsible for a decrease of 50% in the respiratory frequency) was determined for both types of exposures. Diallyamine was a highly potent irritant, and in contrast to the saturated amines, this potency was much greater than that predicted from the log P of the n-octonal/water partition coefficient.

11.0 ONLINE DATABASES AND SECONDARY REFERENCES

11.1 Online Databases

Chemical Information System Files

SANSS (Structure and Nomenclature Search System)
TSCAPP (Toxic Substances Control Act Plant and Production)
TSCATS (Toxic Substances Control Act Test Submissions)

DIALOG Files

359 Chemical Economics Handbook
302 Kirk-Othmer Encyclopedia of Chemical Technology

Internet Databases

Code of Federal Regulations full text. 1996 versions of various titles via GPO Gate, a gateway

by the Libraries of the University of California to the GPO Access service of the Government Printing Office, Washington, DC. Internet URL http://www.gpo.ucop.edu/

National Library of Medicine Databases

EMIC and EMICBACK (Environmental Mutagen Information Center)

STN International Files

BIOSIS (Biological Abstracts)
CA File (Chemical Abstracts)
CANCERLIT
CEN (Chemical & Engineering News)
CIN (Chemical Industry Notes)
CSNB (Chemical Safety News Base)
EMBASE (Excerpta Medica)
HSDB (Hazardous Substances Data Bank)
IPA (International Pharmaceutical Abstracts)
MEDLINE (Index Medicus)
PROMT (Predicasts Overview of Markets and Technology)
RTECS (Registry of Toxic Effects of Chemical Substances)
TOXLINE
TOXLIT

TOXLINE includes the following subfiles:

Toxicity Bibliography	TOXBIB
International Labor Office	CIS
Hazardous Materials Technical Center	HMTC
Environmental Mutagen Information Center File	EMIC
Environmental Teratology Information Center File (continued after 1989 by DART)	ETIC
Toxicology Document and Data Depository	NTIS
Toxicology Research Projects	CRISP
NIOSHTIC7	NIOSH

Pesticides Abstracts	PESTAB
Poisonous Plants Bibliography	PPBIB
Aneuploidy	ANEUPL
Epidemiology Information System	EPIDEM
Toxic Substances Control Act Test Submissions	TSCATS
Toxicological Aspects of Environmental Health	BIOSIS
International Pharmaceutical Abstracts	IPA
Federal Research in Progress	FEDRIP
Developmental and Reproductive Toxicology	DART

11.2 Secondary References

CRC Handbook of Toxicology, M.J. Derelanko, Ph.D. and M.A. Hollinger, Ph.D., Eds. 1995. CRC Press, Boca Raton, FL. Listed in Section 12 as Derelanko and Hollinger, Eds. (1995).

Farm Chemicals Handbook, C. Sine, Ed. 1991, Meister Publishing Company, Willoughby OH. Listed in Section 12 as Sine (1991).

The Federal Environmental & Safety Authority (FESA), CD-ROM with quarterly updates of the Federal Guidelines. CPI Electronic Publishing, Scottsdale, AZ. Last updated February 1997.

Kirk-Othmer Encyclopedia of Chemical Technology, 2nd ed., Vol. 2, A.E. Schweizer, R.L. Fowlkes, J.H. McMakin, and T.E. Whyte, Jr. Eds. 1978. A Wiley-Interscience Publication, John Wiley & Sons, New York, NY. Listed in Section 12 as Schweizer et al. (1978).

The Merck Index, 12th ed., S. Budavari, Ed. 1996. Merck Research Laboratories, Merck & Co., Inc., Whitehouse Station, NJ. Listed in Section 12 as Budavari (1996).

Patty's Industrial Hygiene and Toxicology, 3rd ed., Vol. 2B, D.H. Clayton and F.E. Clayton, Eds. 1981. A Wiley-Interscience Publication, John Wiley & Sons, Inc., New York, NY. Listed in Section 12 as Beard and Noe (1981).

The Pesticide Manual, 9th ed., C.R. Worthing and R.J. Hance, Eds. 1991, The British Crop Protection Council. Listed in Section 12 as Worthing and Hance (1991).

Sax's Dangerous Properties of Industrial Materials, 8th ed. Vol. 11. Lewis, R.J., Jr. 1992. Van Nostrand Reinhold, New York, NY. Listed in Section 12 as Lewis (1992).

SRI Directory of Chemical Producers, SRI International, Menlo Park, CA, 1996. Listed in Section 12 as SRI International (1996).

12.0 REFERENCES

Beard, R.R., and J.T. Noe. 1981. Aliphatic and Alicyclic Amines. In: G.D. Clayton and F.E. Clayton, eds. Patty's Industrial Hygiene and Toxicology, 3rd ed. Vol. 2B., Chapter 44, John Wiley and Sons, New York, NY. pp. 3135-3173.

Boor, P.J., and R.M. Hysmith. 1987. Allylamine Cardiovascular Toxicity. Toxicology 44:129-145.

Budavari, S. Ed. 1996. The Merck Index, 12th ed. Merck Research Laboratories, Merck and Co., Inc., Whitehouse Station, NJ. p. 504.

Gagnaire, F., S. Azim, P. Simon, B. Cossec, P. Bonnet, and J. De Ceaurriz. 1993. Sensory and Pulmonary Irritation of Aliphatic Amines in Mice: A Structure-Activity Relationship Study. J. Appl. Toxicol. 13(2):129-135.

HSDB. 1996. The Hazardous Substances Data Bank. Online database produced by the National Library of Medicine.

ICI Americas, Inc. 1975. Initial Submission: Skin Irritation Classification with Diallylamine in Rabbits with Cover Letter Dated 082892. EPA TSCA Section 8ECP Test Submission. Doc. No. 88-920006947. Fiche No. OTS0545350.

Lewis, R.J., Jr. 1992. Sax's Dangerous Properties of Industrial Materials, 8th ed. Vol. 11. Van Nostrand Reinhold. New York, NY. p. 1062.

RTECS. 1996. Registry of Toxic Effects of Chemical Substances. Data base produced by the National Library of Medicine.

Schweizer, A.E., R.L. Fowlkes, J.H. McMakin, and T.E. Whyte, Jr. 1978. Amines. In: Kirk-Othmer Encyclopedia of Chemical Technology, 2nd ed., Vol 2. p.272-283.

Shell Oil Co. 1982. Initial Submission: Letter Submitting Enclosed Information on Exposure of Workers to Mono-allylamine, Di-allylamine, and Tri-allylamine. EPA TSCA Section 8ECP Test Submission. Doc. No. 88-920002051. Fiche No. OTS0539146.

Sine, C., Ed. 1991. Farm Chemicals Handbook. Meister Publishing Company, Willoughby OH. p. C 126, C 261.

SRI Int. 1996. Directory of Chemical Producers, United States. SRI International, Menlo Park, CA. From Online Database DIALOG File 359.

SRI Int. 1997. Chemical Economics Handbook. SRI Int. Menlo Park, CA.

TSCAPP. 1983. Online database produced by Chemical Information Systems.

Weast, R.C., and M.J. Astle, Eds. 1980. CRC Handbook of Chemistry and Physics. CRC Press, Inc. Boca Raton, FL.

Zeiger, E., B. Anderson, S. Haworth, T. Lawlor, K. Mortelmans, and W. Speck. 1987. Salmonella Mutagenicity Tests: III. Results From the Testing of 255 Chemicals. Environ. Mutagen. 9:1-110.

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