CAS Registry Number: 102-71-6 Toxicity Effects

Selected toxicity information from HSDB, one of the National Library of Medicine's databases. 1

Names (NTP)
- Triethanolamine
- 2,2',2''-NITRILOTRIETHANOL

Human Toxicity Excerpts

- **HUMAN EXPOSURE STUDIES:** ... Considered to have low acute and chronic toxicity. ... If deleterious effects were to occur in man ... These would probably be acute in nature and due to its alkalinity rather than its inherent toxicity. [Bingham, E.; Cohrsen, B.; Powell, C.H.; Patty's Toxicology Volumes 1-9 5th ed. John Wiley & Sons. New York, N.Y. (2001)., p. V4 785]**PEER REVIEWED**
- **HUMAN EXPOSURE STUDIES:** Triethanolamine and diethanolamine produce mild skin irritation only in concentrations above 5%; little skin sensitization develops. [Ellenhorn, M.J. and D.G. Barceloux. Medical Toxicology - Diagnosis and Treatment of Human Poisoning. New York, NY: Elsevier Science Publishing Co., Inc. 1988., p. 907]**PEER REVIEWED**
- **HUMAN EXPOSURE STUDIES:** Triethanolamine has been identified as causing allergic contact dermatitis, erythematous vesicular lesions, eczema, contact dermatitis, and irritation in workers exposed to triethanolamine in their occupations. [American Conference of Governmental Industrial Hygienists. Documentation of Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices for 2001. Cincinnati, OH. 2001., p. 3]**PEER REVIEWED**
- **HUMAN EXPOSURE STUDIES:** ...A total of 1,357 patients suspected of having allergic eczematous contact dermatitis were patch-tested with triethanolamine. Positive tests were obtained in 41 of these 1,357 patients. [Scheuer B; Hautarzt 34: 126-9 (1983) ]**PEER REVIEWED**
- **SIGNS AND SYMPTOMS:** The ingestion of several ounces can probably be tolerated by man, but unless the liquid is partly neutralized with acid, alkali burns of the mouth, pharynx and esophagus are likely. [Gosselin, R.E., R.P. Smith, H.C. Hodge. Clinical Toxicology of Commercial Products. 5th ed. Baltimore: Williams and Wilkins, 1984., p. II-106]**PEER REVIEWED**
- **CASE REPORTS:** A case of allergic contact dermatitis induced by occupational exposure to monoethanolamine, diethanolamine, and triethanolamine in cutting oils was examined. ...Monoethanolamine, diethanolamine, and triethanolamine occur in many products. Each of the three can cause contact dermatitis. [Blum A, Lischka G; Contact Dermatitis 36 (3): 166 (1997) ]**PEER REVIEWED**
- **CASE REPORTS:** Three cases of occupational asthma caused by ethanolamines were summarized. ...The three cases share one common feature: exposure to triethanolamines occurred at temperatures higher than that of the ambient air. This agrees with the view that significant inhalation exposure to ethanolamines does not occur when the compounds are used under ambient conditions. [Savonius B et al; Allergy 49 (10): 877-881 (1994) ]**PEER REVIEWED**
- **CASE REPORTS:** ...We report a case of a woman with a contact allergy to triethanolamine inadvertently discovered when she had a reaction to a triethanolamine-containing fluorescent marking pen. [Hamilton TK, Zug KA; Am J Contact Dermat 7 (3): 164-5 (1996) ]**PEER REVIEWED**

Non-Human Toxicity Excerpts

- **LABORATORY ANIMALS: Acute Exposure:** The principal toxic effect in animals has been
ascribed to alkalinization (systemic alkalosis), ... /and/ functional signs of transient liver injury have been described in animals after sublethal doses. Gross pathology has been limited to the GI tract in fatal oral poisonings in rats and guinea pigs. [Gosselin, R.E., R.P. Smith, H.C. Hodge. Clinical Toxicology of Commercial Products. 5th ed. Baltimore: Williams and Wilkins, 1984., p. II-106]**PEER REVIEWED**

**LABORATORY ANIMALS: Acute Exposure:** Tested by application of a drop to rabbit eyes, ... It caused moderate, presumably transient injury, graded 5 on a scale of 1 to 10 after 24 hr, and in another test caused negligible irritation. [Grant, W.M. Toxicology of the Eye. 3rd ed. Springfield, IL: Charles C. Thomas Publisher, 1986., p. 944]**PEER REVIEWED**

**LABORATORY ANIMALS: Acute Exposure:** When tested on rabbit eyes in 0.023 molar aqueous solutions by continuous application for 15 min after removal of corneal epithelium to facilitate penetration ... solutions adjusted to pH 10 was essentially noninjurious. Same solution adjusted to pH 11 caused moderate corneal swelling and hyperemia of iris and conjunctiva .... [Grant, W. M. Toxicology of the Eye. 3rd ed. Springfield, Illinois: Charles C. Thomas, 1974., p. 1050]**PEER REVIEWED**

**LABORATORY ANIMALS: Acute Exposure:** The effects observed in rats and guinea pigs in an acute oral study/ were confined to the GI tract. ... Toxic effects were probably from the alkaline irritation, because larger doses of the neutralized material produced no symptoms at levels where the free base would cause 100% mortality. [Bingham, E.; Cohrssen, B.; Powell, C.H.; Patty's Toxicology Volumes 1-9 5th ed. John Wiley & Sons. New York, N.Y. (2001)., p. V4 786]**PEER REVIEWED**

**LABORATORY ANIMALS: Acute Exposure:** The effects observed in rats and guinea pigs in an acute oral study/ were confined to the GI tract. ... Toxic effects were probably from the alkaline irritation, because larger doses of the neutralized material produced no symptoms at levels where the free base would cause 100% mortality. [Bingham, E.; Cohrssen, B.; Powell, C.H.; Patty's Toxicology Volumes 1-9 5th ed. John Wiley & Sons. New York, N.Y. (2001)., p. V4 786]**PEER REVIEWED**


**LABORATORY ANIMALS: Subchronic or Prechronic Exposure:** In a 90-day subacute feeding /study/ with rats, the max dose producing no effect was 0.08 g/kg. Microscopic lesions and deaths occurred at 0.73 g/kg, and 0.17 g/kg produced alterations in liver and kidney weights. [Bingham, E.; Cohrssen, B.; Powell, C.H.; Patty's Toxicology Volumes 1-9 5th ed. John Wiley & Sons. New York, N.Y. (2001)., p. V4 786]**PEER REVIEWED**

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**LABORATORY ANIMALS: Subchronic or Prechronic Exposure:** Fourteen day repeated dose studies of triethanolamine in F344 rats and B6C3F1 mice were performed by inhalation, drinking water, or dermal routes of exposure. Exposures for both species in the inhalation study were 0, 0.125, 0.25, 0.5, 1.0 or 2.0 g/cu m, 6 hr/day, 5 days/wk, for 2 wk (10 exposures). The only histopathologic observation was a minimal acute inflammation of the laryngeal submucosa in rats and mice. In the oral study, concentrations of triethanolamine in drinking water (adjusted to pH 7.4) were 0, 0.5, 1.0, 2.0, 4.0, and 8.0 g/100 ml. Water consumption was significantly reduced in the 4 and 8% dose groups of rats and mice. No compound-related gross or microscopic lesions were observed in the liver or kidneys of rats; cytoplasmic vacuolization of hepatocytes was observed in the high dose groups of male and female mice. Dose levels of triethanolamine in the dermal study were 0, 0.14, 0.28, 0.56, 1.13 and 2.25 g/kg for rats and 0, 0.21, 0.43, 0.84, 1.69, and 3.37 g/kg for mice. Triethanolamine was applied as the undiluted compound, 5 days/wk for 2 wk. Chronic active necrotizing inflammation of the skin at the application site was observed at a greater frequency and severity in dosed rats than in dosed mice. [Snyder, R. (ed.). Ethyl Browning's Toxicity and Metabolism of Industrial Solvents. 2nd ed. Volume II: Nitrogen and Phosphorus Solvents. Amsterdam-New York-Oxford: Elsevier, 1990., p. 445]**PEER REVIEWED**

**LABORATORY ANIMALS: Subchronic or Prechronic Exposure:** Repeated exposure revealed cumulative properties. Retardation of body weight gain, excitation, aggression, and changes in the blood morphology were noted in rats given 520 mg /2,2',2''-nitrilotriethanol/kg
bw for 2 months. Mortality reached 75% in this study. [Sheftel, V.O.; Indirect Food Additives and Polymers. Migration and Toxicology. Lewis Publishers, Boca Raton, FL. 2000., p. 617] **PEER REVIEWED**

- **LABORATORY ANIMALS: Subchronic or Prechronic Exposure:** When guinea pigs were administered 8 g/kg/day, 5 days a week of either commercial grade or high purity grade triethanolamine to the shaved and subsequently bandaged skin, the guinea pigs dies between the second and the seventeenth application. Necrosis of the epithelium was observed kidneys and liver showed cloudy swelling and congestion; fatty changes were seen in the central ascini of the liver, and lung and adrenal congestion were observed. [American Conference of Governmental Industrial Hygienists. Documentation of Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices for 2001. Cincinnati, OH. 2001., p. 1]**PEER REVIEWED**

- **LABORATORY ANIMALS: Subchronic or Prechronic Exposure:** Triethanolamine (TEA) was applied to the skin of male and female C3H mice (15/sex/dose group) three times weekly for 95 days (37 applications). ...The results indicate that TEA caused a mild local reaction at all concentrations tested, but did not cause systemic toxicity under these conditions. [DePass LR et al; Food Chem Toxicol 33 (8): 675-80 (1995)]**PEER REVIEWED**

- **LABORATORY ANIMALS: Chronic Exposure or Carcinogenicity:** ...An increased incidence of malignant lymphomas, particularly thymic lymphomas, developed in female but not male ICR-JCL mice fed diets containing 0.03 or 0.3% (w/w) triethanolamine throughout their lifespan compared to controls. The triethanolamine containing diets were prepared by heating mixtures of mouse feed plus triethanolamine for 40 min at 100 deg C. The incidences of malignant tumors in lymphoid tissues were 2.8% (1/36), 19% (7/37), and 25% (9/36), respectively. [Snyder, R. (ed.). Ethyl Browning's Toxicity and Metabolism of Industrial Solvents. 2nd ed. Volume II: Nitrogen and Phosphorus Solvents. Amsterdam-New York-Oxford: Elsevier, 1990., p. 447]**PEER REVIEWED**

- **LABORATORY ANIMALS: Chronic Exposure or Carcinogenicity:** ...Triethanolamine /was administered/ in drinking water at concentrations of 0 (control), 1% or 2% to groups of 50 male and female F344 rats for two years. Due to increased mortality and loss of body weight gain in the female 2% group, the concentration of triethanolamine in the drinking water was reduced by half for females after week 68 of the study. There were no significant increases in incidences of primary tumors in treated groups compared to controls when analyzed by chi-square test; However, positive trends were noted for hepatic neoplasms in males and for uterine endometrial sarcomas and renal cell adenomas in females by survival-adjusted analysis. Kidney toxicity (acceleration of chronic nephropathy, mineralization of the renal papilla, and nodular hyperplasia of the pelvic mucosa) was also observed in this study. [Snyder, R. (ed.). Ethyl Browning's Toxicity and Metabolism of Industrial Solvents. 2nd ed. Volume II: Nitrogen and Phosphorus Solvents. Amsterdam-New York-Oxford: Elsevier, 1990., p. 447]**PEER REVIEWED**

- **LABORATORY ANIMALS: Chronic Exposure or Carcinogenicity:** The carcinogenic potential of triethanolamine was examined in B6C3Fl-mice. The mice were exposed to drinking water containing 0, 1 or 2% triethanolamine. After 82 weeks surviving mice were sacrificed. Body weights in animals receiving 2% ethanolamine were lower than controls by week 60 for females and during the first 20 weeks for male mice. Water consumption of male mice receiving 1% triethanolamine was slightly higher than that of female mice. Total intakes of triethanolamine for 82 weeks were 26.9 and 37.3 grams/mouse in the low dose group and 62.8 and 63.6 grams/mouse in the high dose group for females and males, respectively. No significant increases were noted in any organ weights. Tumors developed in the liver, lung, hematopoietic system, Harderian gland, mammary gland, kidney, spleen, subcutis, thyroid gland, adrenal gland, pituitary gland, and uterus in the control and in each treatment group. No dose related increase of the incidence of any tumor was seen. /It was/ concluded that the chronic toxicity study documented a lack of carcinogenic activity of triethanolamine in B6C3Fl-mice. [Konishi Y et al; Fundam and Appl Toxicol 18 (1): 25-9 (1992)]**PEER REVIEWED**

- **LABORATORY ANIMALS: Chronic Exposure or Carcinogenicity:** The carcinogenic potential of triethanolamine given in the drinking water over a two year period was investigated.
using F344-rats. There appeared to be no increase in the incidence of any specific tumors over the corresponding control group. However, a positive trend was noted in the occurrence hepatic tumors (neoplastic nodule or hepatocellular carcinoma) in males and of uterine endometrial sarcomas and renal cell adenomas in females by trend analysis tests. Histologically, all tumors observed in this study were similar to the spontaneous tumors observed in earlier studies. Toxic lesions of the liver associated with triethanolamine administration were not observed, although nonneoplastic lesions, which are common in aging F344-rats, were noted in the liver of both control and experimental groups. The absolute and relative kidney weights, however, increased significantly and dose dependently in the treated groups of both sexes. Macroscopically, kidneys were enlarged, granular on the surface, and anemic in color. In addition, mineralization of the renal papilla, nodular hyperplasia of the pelvic mucosa, and pyelonephritis with or without papillary necrosis were observed more frequently and dose dependently in the treated groups than in controls, while there were no tumors in the renal pelvis of any of the groups. It was concluded that triethanolamine is not carcinogenic in F344-rats when given continuously in the drinking water for 2 years, although it is toxic to the kidneys, especially in females. [Maekawa A et al; J of Toxicol and Environ Health 19 (3): 345-57 (1986)]**PEER REVIEWED**

**LABORATORY ANIMALS: Chronic Exposure or Carcinogenicity:** Groups of 60 male and 60 female Fischer 344/N rats, six weeks of age, were administered triethanolamine (purity, 99%) topically in acetone on five days per week for 103 weeks. Male rats received 0, 32, 63 or 125 mg/kg bw and females received 0, 63, 125 or 250 mg/kg bw triethanolamine. The survival rates of males were 21/50, 11/50, 18/49 and 19/50 and of females were 25/50, 29/50 and 18/50 in the control, low-, mid- and high-dose rats respectively. The mean body weight of females receiving 250 mg/kg bw ranged from 9% to 12% lower than that of the vehicle controls from weeks 73 to 93, and by the end of the study, was 7% lower than that of the vehicle control group. There was no significant increase in the incidence of tumors at any site. [IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer, 1972-PRESENT. (Multivolume work)., p. V77 388 (2000)]**PEER REVIEWED**

**LABORATORY ANIMALS: Chronic Exposure or Carcinogenicity:** Mice fed a diet containing 0.3 or 0.03% triethanolamine developed malignant tumors. ... [Hoshino H, Tanooka H; Cancer Res 38 (11): 3918-21 (1978)]**PEER REVIEWED**

**LABORATORY ANIMALS: Chronic Exposure or Carcinogenicity:** Under these experimental conditions triethanolamine is not carcinogenic in F344 rats but is toxic to the kidneys. [Maekawa A et al; J Toxicol Environ Health 19 (3): 345-57 (1986)]**PEER REVIEWED**

**LABORATORY ANIMALS: Chronic Exposure or Carcinogenicity:** Groups of 40 male and 40 female mice were fed triethanolamine in the diet at 0% (control), 0.03%, or 0.3% for their life span. ...The total incidence of malignant tumors in females at the 0.03% and 0.3% dosings was significantly higher than that observed for the control animals. Based upon these data, a dose response relationship existed. The higher tumor incidence was due to tumors in the lymphoid tissues, primarily thymic. At 0% ,0.03%, and 0.3% the numbers of thymic and nonthymic lymphomas were 1, 7, and 9, respectively. When lymphomas were combined with other types of tumors of other tissues (renal, mammary, genitals, lung, ovary and stomach), the numbers of total tumors at 0%, 0.03%, and 0.3% were respectively 1, 10, and 13 for females and 1, 3, and 1 for males. The total malignant tumor incidence when summed up for both sexes was significantly higher that that of the controls. [American Conference of Governmental Industrial Hygienists. Documentation of Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices for 2001. Cincinnati, OH. 2001., p. 2]**PEER REVIEWED**

**LABORATORY ANIMALS: Chronic Exposure or Carcinogenicity:** Administration of monomethanolamine by the IV route in dogs produced increased blood pressure, diuresis, salivation, and pupillary dilatation...; monoethanolamine was more active than triethanolamine. [Bingham, E.; Cohrssen, B.; Powell, C.H.; Patty's Toxicology Volumes 1-9 5th ed. John Wiley & Sons. New York, N.Y. (2001)., p. V4 782]**PEER REVIEWED**

**LABORATORY ANIMALS: Chronic Exposure or Carcinogenicity:** Groups of 50 6-wk-old
F344/DUCRJ rats were given triethanolamine (TEA) dissolved in distilled drinking water at dose levels of 0 (controls), 1, or 2% ad libitum for 104 weeks. The dose levels in females were reduced by half from week 69 because of associated nephrotoxicity. Moribund, dead, and surviving rats, terminated at week 113, were autopsied and the following organs examined histopathologically: brain, spinal cord, nerves, pituitary, thyroid, parathyroid, thymus, lung, trachea, heart, liver, spleen, pancreas, adrenals, kidney, bladder, salivary glands, tongue, esophagus, stomach, small and large intestine, rectum, gonads, accessory sex organs, mammary, lymph nodes, skin, muscle, sternum, bone, eyes, and nasal cavity. Tumor varieties and incidence in all groups were similar to those spontaneously occurring in this strain, without statistically significant increases. Renal damage was examined in the treated groups, and nodular hyperplasia, pyelonephritis, and papillary necrosis were observed. Under the conditions of this study, triethanolamine was not carcinogenic in F344 rats but was toxic to the kidneys.

**PEER REVIEWED**

**LABORATORY ANIMALS: Developmental or Reproductive Toxicity:** Triethanolamine was embryotoxic when injected into three day chick embryos. Triethanolamine did not produce a significant increase in the incidence of malformations (3 malformations per 110 treated eggs compared to 1 malformation per 100 acetone control eggs). [Snyder, R. (ed.). Ethyl Browning's Toxicity and Metabolism of Industrial Solvents. 2nd ed. Volume II: Nitrogen and Phosphorus Solvents. Amsterdam-New York-Oxford: Elsevier, 1990., p. 445]**PEER REVIEWED**

**GENOTOXICITY:** In a battery of short-term tests, triethanolamine did not induce mutations in bacteria (Salmonella typhimurium strains TA98, TA100, TA1535, TA1537, and TA1538 or Escherichia coli strains WP2 and WP2 uvrA in the presence or absence of S-9 fractions prepared from livers of Aroclor-induced rats), mitotic gene conversion in Saccharomyces cerevisiae JD1 cells, or chromosomal damage in cultured rat liver RAL4 cells. Triethanolamine was inactive in inducing revertants to histidine prototrophy in the excision repair deficient Bacillus subtilis strain TKJ5211 with or without rat liver S-9 preparations. [Snyder, R. (ed.). Ethyl Browning's Toxicity and Metabolism of Industrial Solvents. 2nd ed. Volume II: Nitrogen and Phosphorus Solvents. Amsterdam-New York-Oxford: Elsevier, 1990., p. 446]**PEER REVIEWED**

**GENOTOXICITY:** ...N-nitrosodiethanolamine, known carcinogen and mutagen, ...may not be the main mutagenic product. [Hoshino H, Tanooka H; Cancer Res 38 (11): 3918-21 (1978) ] **PEER REVIEWED**

**GENOTOXICITY:** Triethanolamine did not produce any morphological transformation in Chinese hamster embryo cells at concentrations of 25-500 mg/mL and was also inactive in the Ames S.typhimurium and E.coli tests, as well as in the chromosomal aberration test in Chinese hamster cells; however the assay was not carried out with microsomal metabolic activation. [Bingham, E.; Cohrssen, B.; Powell, C.H.; Patty's Toxicology Volumes 1-9 5th ed. John Wiley & Sons. New York, N.Y. (2001)., p. V4 787]**PEER REVIEWED**

**Human Toxicity Values**

- None found

**Non-Human Toxicity Values**


Absorption, Distribution and Excretion

- The elimination of 14(C)triethanolamine from the blood of mice administered 1.0 mg/kg bw iv showed first-order biphasic kinetics with a rapid (0.58-hr half-life) and a slow phase (10.2-hr half-life). The slow phase half-lives for elimination of triethanolamine in mice after dermal exposure to 1000 and 2000 mg/kg bw in acetone were 9.7 hr and 18.6 hr. Skin absorption rates (as blood concentration-time curves) after dermal application of aqueous and neat 14(C) triethanolamine to mouse skin (2000 mg/kg bw, enclosed by a glass ring) showed no significant change with the use of water as the vehicle. [IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer, 1972-PRESENT. (Multivolume work)., p. V77 389 (2000)] **PEER REVIEWED**

- In a dermal pharmacokinetic study, (14)C-triethanolamine was absorbed more slowly and less extensively in F344 rats than in C3H/HeJ mice. 48 hr after dermal application of (14)C-triethanolamine to mice (1,000 mg/kg dose), about 60% of the radioactivity was recovered from the urine and about 20% was recovered in the feces; less than 10% of the radioactivity was found in skin at the site of application. It was concluded that triethanolamine does not undergo extensive biotransformation in mice, since greater than 95% of the radioactivity recovered from the urine was identified as the parent compound. [Snyder, R. (ed.). Ethyl Browning's Toxicity and Metabolism of Industrial Solvents. 2nd ed. Volume II: Nitrogen and Phosphorus Solvents. Amsterdam-New York-Oxford: Elsevier, 1990., p. 443]**PEER REVIEWED**

- Triethanolamine was rapidly absorbed in orally dosed rats, and subsequently excreted mainly as unchanged parent compound in the urine. 24 hr after oral administration of triethanolamine (single dose of 2-3 mg/kg), 53% and 20% of the administered dose was recovered as the parent compound in the urine and feces, respectively. [Snyder, R. (ed.). Ethyl Browning's Toxicity and Metabolism of Industrial Solvents. 2nd ed. Volume II: Nitrogen and Phosphorus Solvents. Amsterdam-New York-Oxford: Elsevier, 1990., p. 443]**PEER REVIEWED**

- After single oral administration to male rats, the excretion ratios of unchanged /triethanolamine/ in the urine and feces for one day were 53% and 20% of the dose, respectively. [Kohri N et al; Arch Pract Pharm 42: 342-348 (1982)] **PEER REVIEWED**

- The half-life for dermal absorption of radioactivity was estimated to be 1.3 hours. ...TEA is absorbed extensively following dermal application to mice at dosages relevant to toxicity testing. [Stott WT et al; Food Chem Toxicol 38 (11): 1043-51 (2000)] **PEER REVIEWED**

- Absorption in the gastrointestinal tract of triethanolamine administered orally to Wistar rats is rapid; 63% of the dose disappeared from intestines within 65 min. In dermal toxicity studies, the peak blood levels of 14(C)triethanolamine were observed 2 hr after its application in C3H/HeJ mice (2000 mg/kg bw), whereas in Fischer 344 rats (1000 mg/kg bw), the blood levels (expressed as radioactivity) indicated that triethanolamine was absorbed less rapidly than by mice. Data from various studies in mice and rats (1000-2000 mg/kg bw) suggest that absorption of dermally administered triethanolamine is almost complete in 24 hr. [IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer, 1972-PRESENT. (Multivolume work)., p. V77 389 (2000)] **PEER REVIEWED**

- About 60% of the radioactivity in 14(C)triethanolamine applied to mouse skin (1000 mg/kg bw) was excreted in 48 hr in urine and 20% in feces, with less than 10% found in the skin at the site of application. [IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer, 1972-PRESENT. (Multivolume work)., p. V77 389 (2000)] **PEER REVIEWED**
Metabolism/Metabolites

- After multiple oral administration to male and female rats, triethanolamine was mainly excreted unchanged. The urinary and fecal excretion ratio of unchanged triethanolamine remained constant throughout the treatment period (for five to six days) in both males and females. A small amount of triethanolamine (1.4-2.7%) was excreted as glucuronide conjugates. [IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer, 1972-PRESENT. (Multivolume work)., p. V77 390 (2000)]**PEER REVIEWED**

- The biotransformation of 14(C)triethanolamine to monoethanolamine and diethanolamine was specifically investigated in mice after both intravenous and dermal treatments. Neither of the hypothetical metabolites was detected in urine (by mass spectral analysis), whereas more than 95% of the radioactivity detected in urine was identified as unchanged triethanolamine. In vitro, triethanolamine had an inhibitory effect on the incorporation of 32(P)phosphate into phospholipids from rabbit and human tissues. Cytochrome P450 monooxygenase-dependent oxidative N-dealkylation of triethanolamine does occur in microorganisms, with formation of diethanolamine, ethanolamine and glyoxylate as reaction products. [IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer, 1972-PRESENT. (Multivolume work)., p. V77 389 (2000)]**PEER REVIEWED**

TSCA Test Submissions

- None found

Footnotes

1 Source: the National Library of Medicine's Hazardous Substance Database, 10/28/2007.