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Best Sections

Non-Human Toxicity Excerpts :

Chronic toxicity studies in mice with **beechwood creosote** caused some animal deaths (bronchopneumonia with pulmonary abscess), but showed no dose-lethal effect relation. The spleen of the male mice showed a wt decr. The creosote caused no significant histopathological changes in the organs and tissues. Deviations in the hematological and clin indicators were within physiological deviations. The beechwood creosote was not carcinogenic.

[Miyazato T et al; Oyo Yakuri 28 (5): 909-24 (1984)] **PEER REVIEWED**

Probable Routes of Human Exposure :

Cancer incidence was studied among 922 **creosote** exposed impregnators at 13 plants in Sweden and Norway. The subjects had been impregnating wood (eg, railroad cross-ties and telegraph poles), but no data on individual exposures were available. The study population was restricted to men employed during the period 1950-1975, and their cancer morbidity was checked through the cancer registries. The total cancer incidence was somewhat lower than expected, 129 cases versus 137 expected (standardize incidence ratio 0.94). Increased risks in both countries combined were observed for lip cancer (standardised incidence ratio 2.50, 95% confidence interval (95% confidence interval) 0.81-5.83), skin cancer (standardised incidence ratio 2.37, 95% confidence interval 1.08-4.50), and malignant lymphoma (standardised incidence ratio 1.9, 95% confidence interval 0.83-3.78). Exposure to sunlight may have contributed to the risk of lip and skin cancer. The small number of cancer cases does not permit valid conclusions. The findings indicate that impregnating **wood with creosote** in earlier decades increased the risk of skin cancer.

[Karlehagen S et al; Scand J Work Environ Health 18 (1): 26-9 (1992)] **PEER REVIEWED** [PubMed Abstract](#)

Synonyms :

BEECHWOOD CREOSOTE

PEER REVIEWED

Non-Human Toxicity Excerpts :

Chronic toxicity studies in rats with **beechwood creosote** (312.5 mg/kg/day for females and 394.4 mg/kg/day for males) caused some animal deaths (bronchopneumonia with pulmonary abscess and leukemia). There was no dose lethal effect relation, however, the mortality in the male rats was higher. In addn, the male rats showed toxic renal effects, and elevated blood urea nitrogen and serum inorg phosphorus. A number of benign and malignant tumors were observed, of which myelogenous leukemia, adenoma of the hypophysis, and interstitial cell tumor of testes occurred more frequently. There was no carcinogenic effect in rats in dietary levels up to 1.2% for 96 wk.

[Miyazato T et al; Oyo Yakuri 28 (5): 925-47 (1984)] **PEER REVIEWED**

Synonyms :

WOOD CREOSOTE

PEER REVIEWED

General Manufacturing Information :

IN THE PAST ... **WOOD CREOSOTE** ENJOYED ... USE IN TREATMENT OF PULMONARY TUBERCULOSIS & ABSCESES OF LUNG. HOWEVER, MODERN THERAPY HAS REMOVED THIS MATERIAL FROM MOST TREATMENT REGIMES.

[International Labour Office. Encyclopedia of Occupational Health and Safety. Vols. I&II. Geneva, Switzerland: International Labour Office, 1983., p. 570]

PEER REVIEWED

Human Toxicity Excerpts :

... **WOOD CREOSOTE** (ESPECIALLY BEECHWOOD) ... /HAS TOXICITY/ SIMILAR TO BUT LESS THAN PHENOL.

[Gosselin, R.E., R.P. Smith, H.C. Hodge. Clinical Toxicology of Commercial Products. 5th ed. Baltimore: Williams and Wilkins, 1984., p. II-192] **PEER REVIEWED**

Antidote and Emergency Treatment :

1. Stringent measures should be taken to avoid contamination of skin or eyes and inhalation of vapor. Skin contamination should be promptly washed off with soap and water. Contamination of the eyes should be removed by washing with copious amounts of water, then specialized medical attention should be obtained promptly because corneal injury may be severe. 2. If a significant amount of **creosote** has been ingested and the patient is alert and able to swallow, immediately administer a slurry of activated charcoal by mouth Further efforts to limit absorption depend on whether there has been corrosive injury to the esophagus. If pharyngeal redness and swelling are evident, neither induced emesis or gastric lavage are advisable: emesis will re-expose the esophagus to the **creosote**, and a gastric tube may perforate the esophagus. If there is minimal evidence of pharyngeal injury, careful gastric intubation and lavage with activated charcoal may be undertaken after placement of a cuffed endotracheal tube to protect the airway. Sorbitol should be administered if diarrhea has not already developed in response to the

creosote. Whether gastric lavage is accomplished or not, repeated administration of activated charcoal by mouth, at half or more the initial dose every 2-4 hours, may well be beneficial. 3. Maintain pulmonary ventilation mechanically with oxygen, if necessary. 4. Draw a blood sample to test for methemoglobinemia, to measure BUN and blood electrolytes, and to check for signs of liver injury (bilirubin, GOT, LDH, ALT, AST, and alkaline phosphatase). Test the urine for protein and cells, and for "smoky" phenolic excretion products. 5. Give fluids intravenously to correct dehydration and electrolyte disturbances. Include glucose to protect the liver and bicarbonate to relieve metabolic acidosis, as necessary. Monitor fluid balance carefully to signal discontinuation of intravenous fluids if renal failure occurs. Plasma or blood transfusion may be needed to overcome shock. 6. Monitor ECG to detect arrhythmias and/or conduction defects that may appear as manifestations of a toxic cardiomyopathy. 7. Diazepam may be needed to control tremors or seizures. ... 8. Hemodialysis is not effective in accelerating disposition of phenol (or, presumably, **creosote**), but HEMOPERFUSION over charcoal probably is effective. This should be instituted in severe **creosote** poisonings. 9. Methemoglobinemia is rarely, but intravenous administration of 1% methylene blue may be considered if 25-30% of hemoglobin is converted. Dose is 0.1 ml of 1% solution per kg body weight, given over no less than ten minutes. Nausea, dizziness, and a transient increase in blood pressure may occur. /**Creosote**/

[Morgan DP; Recognition and Management of Pesticide Poisonings. 4th ed. p.153 EPA 540/9-88-001. Washington, DC: U.S. Government Printing Office, March 1989] **PEER REVIEWED**

Non-Human Toxicity Excerpts :

The acute poisoning symptoms produced by oral admin of **creosote** to rats (LD50 870 mg/kg, females; 885 mg/kg, males) and mice (LD50 433 mg/kg, females; 525 mg/kg, males) were mainly characterized by a marked convulsion followed by asphyxia and coma. The animals died from resp or cardiac failure. In the subacute toxicity test, **creosote** was added to powd diet and fed to mice and rats for 3 mo. The concn of **creosote** in test diets were 0.15-1.8% in mice and 0.3-2.5% in rats. Incr in body wt were slightly inhibited in mice of higher dose groups and in both female and male rats of the highest dose group. Food intakes were also smaller in these groups of animals as compared with control and the lower dose groups. A slight decr in the wt of several organs was observed in mice of higher dose groups. Slight incr in the wt of the liver and kidney in female rats, and the liver of male rats were observed in the treated groups. These changes were not attributable to the toxicity of **creosote** since no significant changes were found in blood and pathological exam in these groups of animals.

[Miyazato T et al; Oyo Yakuri 21 (6): 899-919 (1981)] **PEER REVIEWED**

General Manufacturing Information :

The methods used for reclaiming **creosote** ... from /wood/ treatment soln The soln are conducted into tanks or ponds, after which the **creosote** ... settles to the bottom ... /&/ can then be recovered ... with submerged pumps. /**Creosote**/

[Parr, J.F., P.B. Marsh, and J.M. Kila (eds.). Land Treatment of Hazardous Wastes. Park Ridge, New Jersey: Noyes Data Corporation, 1983., p. 400] **PEER REVIEWED**

Disposal Methods :

Incineration: **Creosote** contains more than 200 separate constituents, most of which are polynuclear aromatic hydrocarbons. The best disposal method for **creosote** is incineration at high temp. Empty containers should be thoroughly drained before disposal. Water will not be effective for container rinsing. **Creosote** is normally used without dilution, so rinsing with kerosene or other hydrocarbon solvent would leave the rinsate as a disposal problem.

****PEER REVIEWED****

Human Toxicity Excerpts :

FATALITIES HAVE OCCURRED 14-36 HR AFTER THE INGESTION OF ABOUT 7 G BY ADULTS OR 1-2 G BY CHILDREN. THE SYMPTOMS ... INCLUDED SALIVATION, VOMITING, RESPIRATORY DIFFICULTIES, THREADY PULSE, VERTIGO, HEADACHE, LOSS OF PUPILLARY REFLEXES, HYPOTHERMIA, CYANOSIS, & MILD CONVULSIONS. CONTACT OF **CREOSOTE** WITH THE SKIN OR CONDENSATION OF VAPORS OF **CREOSOTE** UPON THE SKIN OR MUCOUS MEMBRANES MAY INDUCE AN INTENSE BURNING & ITCHING WITH LOCAL ERYTHEMA, GRAYISH YELLOW TO BRONZE PIGMENTATION, PAPULAR & VESICULAR ERUPTIONS, GANGRENE, & IN ISOLATED INSTANCES CANCER. ... HEINZ BODIES HAVE BEEN NOTED IN THE BLOOD OF A PATIENT 1 YR AFTER HIS EXPOSURE TO **CREOSOTE**. ... SIMILAR OBSERVATIONS /WERE MADE/ FOLLOWING PERCUTANEOUS ABSORPTION

[Clayton, G. D. and F. E. Clayton (eds.). Patty's Industrial Hygiene and Toxicology: Volume 2A, 2B, 2C: Toxicology. 3rd ed. New York: John Wiley Sons, 1981-1982., p. 2603] ****PEER REVIEWED****

Non-Human Toxicity Excerpts :

ACUTE FATAL DOSE FOR SHEEP IS 4-6 G/KG BODY WT & FOR CALVES OVER 4 G/KG. LETHAL DOSE PRODUCED AN INDEFINITE SYNDROME OF APATHY, WEAKNESS & DEATH AFTER A FEW DAYS. **CREOSOTE** SHOWED MARKED CUMULATIVE EFFECTS IN BOTH SHEEP & CALVES. THE DAILY INGESTION OF 10-15% OF THE ACUTE LETHAL DOSE CAUSED DEGENERATIVE CHANGES IN THE ORGANS WHICH EVENTUALLY CAUSED DEATH. CATTLE HAVE BEEN POISONED IN A Paddock CONTAINING NEWLY CREOSOTED POLES, FROM DRINKING **CREOSOTE** FROM AN OPEN DRUM ... & LIBERAL APPLICATION OF MIXT OF **CREOSOTE** & FUEL OIL USED AS CURE FOR RINGWORM. ANIMALS SHOWED ABDOMINAL PAIN, THIRST, DYSPNEA & UNWILLINGNESS TO MOVE, ... WIDESPREAD CONGESTION & HEMORRHAGES.

[Clarke, M. L., D. G. Harvey and D. J. Humphreys. Veterinary Toxicology. 2nd ed. London: Bailliere Tindall, 1981., p. 104] ****PEER REVIEWED****

Drug Warnings :

WHEREVER **CREOSOTE** IS INDICATED FOR INTERNAL MEDICATION **CREOSOTE** FROM WOOD TAR SHOULD BE DISPENSED & UNDER NO CIRCUMSTANCES SHOULD **CREOSOTE** FROM COAL TAR BE GIVEN, UNLESS EXPLICITLY SO DIRECTED.

[The Merck Index. 10th ed. Rahway, New Jersey: Merck Co., Inc., 1983., p. 368] ****PEER REVIEWED****

Probable Routes of Human Exposure :

A study was conducted to identify the major components in **creosote** vapors, and to measure the concn of polycyclic aromatic hydrocarbons in particulate matter, in **creosote** impregnation facilities (SIC-2491) and in the handling of impregnated wood. Analysis of **creosote** vapors by collection on XAD2 resin followed by gas chromatography and mass spectrometry resulted in identification and quantitation of 12 main components. These included phenols, cresols, xlenols, methylstyrene, indene, naphthalene, diphenyl, dibenzofurane, benzothiophene, quinoline, isoquinoline and fluorene. Concn of vapor and polycyclic aromatic hydrocarbons were measured at various work locations in two impregnation facilities, as well as during the repair of old rails for which molded ties were changed, during the replacement of rails in a railway yard, during the welding of switches, during the making of switch elements in a hall and during the loading of poles on board ship. Naphthalene was the major component of the vapors, its proportion in the total vapors averaging 52% in the impregnation facilities and 32% in the handling of treated wood. Mean vapor concn ranged from 0.1 to 11 mg/cu m and from 0.5 to 37 mg/cu m for the handling of impregnated wood and for the impregnation facilities, respectively. The concn of polycyclic aromatic hydrocarbons in particulate matter ranged from 0.2 to 0.46 ug/cu m). The main components of the polycyclic aromatic hydrocarbons were phenanthrene, anthracene and pyrene. Benzo(a)pyrene concn were under 0.03 ug/cu m, except in the cases of boring of railroad ties and manual metal arc welding where they ranged from 0.24 to 0.89 ug/cu m.

[Heikkila PR et al; Scand J Work Environ Health 13 (5): 431-437 (1987)]

PEER REVIEWED [PubMed Abstract](#)

Major Uses :

Creosote is extensively used as a wood preservative, usually by high-pressure impregnation of lumber. /**Creosote**/

[Morgan DP; Recognition and Management of Pesticide Poisonings. 4th ed. p.152 EPA 540/9-88-001. Washington, DC: U.S. Government Printing Office, March 1989] **QC REVIEWED**

Cleanup Methods :

Information is presented for quantitative evaluation of treatment potential for **creosote** and pentachlorophenol wood treating contaminants in soil systems. The study was conducted in three phases: (1) Characterization, (2) treatability screening and (3) field evaluation. Data generated in phases 1 and 2 were discussed in a previous EPA Report (EPA/-600/2-88-055). The report provides review of data generated during phases 1 and 2 plus discussion of data generated during the two-year field evaluation study. Results from the three-phase study indicated that **creosote** contaminants, i.e., polycyclic aromatic hydrocarbon compounds, and pentachlorophenol are subject to degradation in soil systems; loading rates and previous exposure of site soil to particular contaminants were identified as important factors in determining rates of transformation for a particular site. Although populations of polycyclic aromatic hydrocarbons and pentachlorophenol acclimated organisms increased markedly when these compounds were applied to test soils, no correlation was found between microbial population levels and

transformation rates for specific compounds of concern. Migration of compounds of interest was negligible except in a highly sandy soil from one of the eight sites for which column leaching studies were conducted.

[McGinnis GD et al; Govt Reports Announcements & Index (GRA&I), Issue 21 (1991)] **PEER REVIEWED**

Non-Human Toxicity Excerpts :

Daphnia pulex were acutely and chronically exposed to water-soluble fractions of hydrocarbons. In acute studies, the most to least toxic were coal-tar **creosote**, No 2 fuel oil, naphthalene and phenanthrene. During chronic studies, marked redn occurred in growth rates, number of broods, and impairment of molting and an incr occurred in abortion rates after admin of **creosote** and phenanthrene.

[Geiger JG, Buikema AL Jr; Can J Fish Aquat Sci 39 (6): 830-6 (1982)] **PEER REVIEWED**

Environmental Bioconcentration :

The transport and effects of (14)carbon labeled wood preservatives (**creosote** with labeled phenanthrene or acenaphthene, pentachlorophenol and bis(tri-n-butyltin oxide) impregnated in wood posts were examined in a terrestrial microcosm chamber. Of the chemicals tested, **creosote** accumulated in the vole to the greatest extent (eg, whole body concn of 7.2 and 37.0 ppm for phenanthrene and acenaphthene, respectively).

[Gile JD et al; J Agric Food Chem 30 (2): 295-301 (1982)] **PEER REVIEWED**

Probable Routes of Human Exposure :

In a small wood-preserving industry, spot samples were taken from surfaces contaminated with **creosote** at several places and tested for mutagenicity. The application of a wipe test can give a first indication of occupational exposure to mutagenic and carcinogenic substances, particularly when exposure occurs more from skin contact than from inhalation. Mutagens appeared in the urine of rats after ip admin. Despite these results, no incr in mutagenicity was detected in the urine of **creosote** workers in relation to their work.

[Bos RP et al; Br J Ind Med 41 (2): 260-2 (1984)] **PEER REVIEWED** [PubMed Abstract](#)

Synonyms :

CREOSOTE

PEER REVIEWED

Synonyms :

CREOSOTE BEECHWOOD

PEER REVIEWED

Formulations/Preparations :

/CALCIUM CREOSOTATE OR CALCREOSE IS/ A MIXT OF CALCIUM CMPD OF **CREOSOTE** CONSTITUENTS.

[Budavari, S. (ed.). The Merck Index - Encyclopedia of Chemicals, Drugs and Biologicals. Rahway, NJ: Merck and Co., Inc., 1989., p. 403] **PEER REVIEWED**

General Manufacturing Information :

CREOSOTE IS AN OILY DISTILLATE FROM WOOD TARs, CONSISTING /MAINLY/ OF PHENOLIC CMPD, GUAIACOL, CRESOL, & METHYLCRESOL

[Grant, W. M. Toxicology of the Eye. 2nd ed. Springfield, Illinois: Charles C. Thomas, 1974., p. 328] **PEER REVIEWED**

General Manufacturing Information :

(VET) GUAIACOL, NATIONAL FORMULARY , IS THE CHIEF PHENOLIC CONSTITUENT OF **CREOSOTE** AND IS GENERALLY AVAILABLE IN LIQUID FORM, ALTHOUGH IT OCCURS AS A CRYSTALLINE SOLID ALSO. GUAIACOL HAS BEEN EMPLOYED AS GUAIACOL CARBONATE, NATIONAL FORMULARY , & AS THE WATER SOLUBLE POTASSIUM GUAIACOL SULFONATE, NATIONAL FORMULARY (SOLUBLE GUAIACOL), FOR EXPECTORANT AND ANTITUSSIVE ACTIVITY.

[Booth, N.H., L.E. McDonald (eds.). Veterinary Pharmacology and Therapeutics. 5th ed. Ames, Iowa: Iowa State University Press, 1982., p. 709] **PEER REVIEWED**

General Manufacturing Information :

Wood tar **creosote** /is/ a mixture of phenolic cmpd, being almost completely composed of guaiacol and cresol, which is used in medicine as an expectorant. Its antibacterial potency varies with different samples, but in general is about two to three times that of phenol.

[International Labour Office. Encyclopedia of Occupational Health and Safety. Vols. I&II. Geneva, Switzerland: International Labour Office, 1983., p. 569] **PEER REVIEWED**

Major Uses :

Creosote is applied to wood to be used in marine environments infested with teredine borers.

[Kirk-Othmer Encyclopedia of Chemical Technology. 3rd ed., Volumes 1-26. New York, NY: John Wiley and Sons, 1978-1984., p. V24 597 (1984)] **PEER REVIEWED**

Other Chemical/Physical Properties :

DARK-BROWN POWDER; **CREOSOTE** ODOR; SHARP PHENOLIC TASTE; PARTLY SOL IN WATER /CALCIUM DERIVATIVE/

[Budavari, S. (ed.). The Merck Index - Encyclopedia of Chemicals, Drugs and Biologicals. Rahway, NJ: Merck and Co., Inc., 1989., p. 403] **PEER REVIEWED**

NFPA Hazard Classification :

Health: 2. 2= Materials that, on intense or continued (but not chronic) exposure, could cause temporary incapacitation or possible residual injury, including those requiring the use of respiratory protective equipment that has an independent air supply. These materials are hazardous to health, but areas may be entered freely if personnel are provided with full-face mask self-contained breathing apparatus that provides complete eye protection. /**Creosote** oil/
[Fire Protection Guide to Hazardous Materials. 12 ed. Quincy, MA: National Fire Protection Association, 1997., p. 325-28] **QC REVIEWED**

NFPA Hazard Classification :

Flammability: 2. 2= This degree includes materials that must be moderately heated before ignition will occur and includes Class II and IIIA combustible liquids and solids and semi-solids that readily give off ignitable vapors. Water spray may be used to extinguish fires in these materials because the materials can be cooled below their flash points. /**Creosote** oil/
[Fire Protection Guide to Hazardous Materials. 12 ed. Quincy, MA: National Fire Protection Association, 1997., p. 325-28] **QC REVIEWED**

NFPA Hazard Classification :

Reactivity: 0. 0= This degree includes materials that are normally stable, even under fire exposure conditions, and that do not react with water. Normal fire fighting procedures may be used. /**Creosote** oil/
[Fire Protection Guide to Hazardous Materials. 12 ed. Quincy, MA: National Fire Protection Association, 1997., p. 325-28] **QC REVIEWED**

Autoignition Temperature :

637 DEG F /**CREOSOTE** OIL/

[Fire Protection Guide to Hazardous Materials. 12 ed. Quincy, MA: National Fire Protection Association, 1997., p. 325-28] **QC REVIEWED**

Fire Fighting Procedures :

WATER MAY BE USED TO BLANKET FIRE. /**CREOSOTE** OIL/

[Fire Protection Guide to Hazardous Materials. 12 ed. Quincy, MA: National Fire Protection Association, 1997., p. 325-28] **QC REVIEWED**

Skin, Eye and Respiratory Irritations :

Creosote is irritating to skin, eyes, and mucous membrane.

[Morgan DP; Recognition and Management of Pesticide Poisonings. 4th ed. p.152 EPA 540/9-88-001. Washington, DC: U.S. Government Printing Office, March 1989] **PEER REVIEWED**

Disposal Methods :

Creosote is a waste chemical stream constituent which may be subjected to ultimate disposal by controlled incineration.

[USEPA; Engineering Handbook for Hazardous Waste Incineration p.2-5 (1981)
EPA 68-03-3025] **PEER REVIEWED**

Evidence for Carcinogenicity :

Classification of carcinogenicity: 1) evidence in humans: limited; 2) evidence in animals: sufficient. Overall summary evaluation of carcinogenic risk to humans is Group 2A: The agent is probably carcinogenic to humans. /**Creosote**, from table/

[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). Available at:
<http://monographs.iarc.fr/index.php> p. S7 177 (1987)] **PEER REVIEWED**

Antidote and Emergency Treatment :

Conservative therapy and prevention of secondary skin infection ... for **creosote** irritation.

[Zenz, C. Occupational Medicine-Principles and Practical Applications. 2nd ed. St. Louis, MO: Mosby-Yearbook, Inc, 1988., p. 682] **PEER REVIEWED**

Human Toxicity Excerpts :

CONTACT OF LIQ **CREOSOTE** WITH EYE CAUSED PAINFUL PROTRACTED KERATOCONJUNCTIVITIS, ... INVOLVED LOSS OF CORNEAL EPITHELIUM, CLOUDING OF CORNEA, MIOSIS, & LONG-LASTING IRRITABILITY & PHOTOPHOBIA.

[Grant, W.M. Toxicology of the Eye. 3rd ed. Springfield, IL: Charles C. Thomas Publisher, 1986., p. 283] **PEER REVIEWED**

Human Toxicity Excerpts :

A fatal case of **creosote** poisoning is described. On presentation, extensive oropharyngeal ulceration was noted and gastric lavage withheld. Post-mortem exam showed an intact esophagus and stomach.

[Bowman CE et al; Postgrad Med J 60 (705): 499-500 (1984)] **PEER REVIEWED**
[PubMed Abstract](#)

Human Toxicity Excerpts :

Workers in contact with technical **creosote** or with treated timbers sometimes develop skin irritation, vesicular or papular eruptions, dermal pigmentation, and occasionally gangrene and skin cancer. Photosensitization has been reported. Eye contamination has resulted in conjunctivitis and keratitis, sometimes resulting in corneal scarring.

[Morgan DP; Recognition and Management of Pesticide Poisonings. 4th ed. p.152
EPA 540/9-88-001. Washington, DC: U.S. Government Printing Office, March 1989] **PEER REVIEWED**

Non-Human Toxicity Excerpts :

WHEN ADMIN ORALLY ... THE APPROX LETHAL DOSE ... /IS/ 0.1 G/KG FOR PIGEONS

... . THE CARCINOGENICITY OF **CREOSOTE** OILS HAS BEEN STUDIED QUITE THOROUGHLY USING MICE. ... THE HIGH CARCINOGENIC POTENCY COULD NOT BE CORRELATED WITH THE CONTENT OF BENZOPYRENE. ... SOMETHING OTHER THAN (OR IN CONJUNCTION WITH) BENZOPYRENE CAUSED THE SKIN CANCER. FOLLOWING ORAL INGESTION THE RESULTANT LESIONS ARE THOSE OF INTENSE IRRITATION & CONGESTION OF THE ENTIRE GASTROENTERIC TRACT.

[Clayton, G. D. and F. E. Clayton (eds.). Patty's Industrial Hygiene and Toxicology: Volume 2A, 2B, 2C: Toxicology. 3rd ed. New York: John Wiley Sons, 1981-1982., p. 2602] **PEER REVIEWED**

Non-Human Toxicity Excerpts :

Several fractions of **creosote** P1 separated by TLC showed mutagenicity toward Salmonella typhimurium TA98. Thus mutagenicity is probably caused by the presence of mutagenic aromatic hydrocarbons. The mutagenic polycyclic aromatic hydrocarbons benzo(a)pyrene and benz(a)anthracene were detected in concn of 0.18 and 1.1% respectively.

[Bos RP et al; Mutat Res 130 (3): 153-8 (1984)] **PEER REVIEWED** [PubMed Abstract](#)

Non-Human Toxicity Excerpts :

Creosote (type P1) was assayed for mutagenicity in the Salmonella/microsome assay. It showed mutagenic properties towards Salmonella typhimurium TA1537, TA1538, TA98 and TA100 in the presence of S9 mix. This mutagenicity is probably caused by the presence of mutagenic aromatic hydrocarbons.

[Bos RP et al; Mutat Res 119 (1): 21-6 (1983)] **PEER REVIEWED** [PubMed Abstract](#)

Ecotoxicity Values :

LC50 Mysidopsis bahia 0.018 mg/l/96 hr, marine-grade **creosote** /Conditions of bioassay not specified/

[Borthwick PW, Patrick JM Jr; Environ Toxicol Chem 1 (4): 281-8 (1982)]
PEER REVIEWED

Ecotoxicity Values :

LC50 pink shrimp 0.24 mg/l/96 hr, marine-grade **creosote** /Conditions of bioassay not specified/

[Borthwick PW, Patrick JM Jr; Environ Toxicol Chem 1 (4): 281-8 (1982)]
PEER REVIEWED

Ecotoxicity Values :

LC50 Sheepshead minnows 0.72 mg/l/96 hr, marine-grade **creosote** /Conditions of bioassay not specified/

[Borthwick PW, Patrick JM Jr; Environ Toxicol Chem 1 (4): 281-8 (1982)]
PEER REVIEWED

Ecotoxicity Values :

EC50 Eastern oysters (shell deposition) 0.71 mg/l/96 hr, marine-grade **creosote** /Conditions of bioassay not specified/

[Borthwick PW, Patrick JM Jr; Environ Toxicol Chem 1 (4): 281-8 (1982)]

PEER REVIEWED

Absorption, Distribution & Excretion :

CREOSOTE IS RAPIDLY ABSORBED FROM THE GASTROENTERIC TRACT AND THROUGH THE SKIN. IT APPEARS TO BE EXCRETED IN THE URINE MAINLY IN CONJUGATION WITH SULFURIC, HEXURONIC, & OTHER ACIDS. OXIDATION ALSO OCCURS WITH THE FORMATION OF CMPD THAT IMPART A SMOKY APPEARANCE TO THE URINE. TRACES ARE EXCRETED BY WAY OF THE LUNGS.

[Clayton, G. D. and F. E. Clayton (eds.). Patty's Industrial Hygiene and Toxicology: Volume 2A, 2B, 2C: Toxicology. 3rd ed. New York: John Wiley Sons, 1981-1982., p. 2603] **PEER REVIEWED**

Environmental Fate :

AQUATIC FATE: Mysid bioassays and chem analyses estimated the half-life (less than 1 wk) for marine-grade **creosote** in seawater.

[Borthwick PW, Patrick JM Jr; Environ Toxicol Chem 1 (4): 281-8 (1982)]

PEER REVIEWED

Environmental Fate :

Groundwater contamination in the shallow aquifer beneath the site of an abandoned **creosote** facility in Conroe, TX, was characterized by sampling soils and water quality at 14 monitoring wells and 35 boreholes. Results from 6 sampling trips over 2 yr for inorg and org chem concn in the groundwater showed wells around the site were contaminated to levels greater than 800 ug/l for naphthalene, 400 ug/l for methyl naphthalene and 150 ug/l for dibenzofuran. Conservative constituents, traced by chloride concn up to 75 mg/l, had migrated 300 ft (90 meters) downgradient of the site. Org contaminants had been adsorbed and microbially degraded in their migration from the waste source as evidenced by their attenuated concn.

[Bedient PB et al; Ground Water 22 (3): 318-29 (1984)] **PEER REVIEWED**

Environmental Fate :

TERRESTRIAL FATE: The transport and effects of (14)carbon labeled wood preservatives (**creosote** with labeled phenanthrene or acenaphthene, pentachlorophenol and bis(tri-n-butyltin oxide) impregnated in wood posts were examined in a terrestrial microcosm chamber. The terrestrial microcosm chamber contained a Willamette Valley topsoil, ryegrass, invertebrates and a gravid gray-tailed vole (*Microtus canicaudus*). Approx 2.5 mo after introduction of the posts, 95% of the chemicals remained in the posts. Of the material released into the ecosystem, most remained in the upper soil layer immediately surrounding the posts.

[Gile JD et al; J Agric Food Chem 30 (2): 295-301 (1982)] **PEER REVIEWED**

Fish/Seafood Concentrations :

Chem analyses of hepatopancreas and tail muscle tissues from freshly caught lobsters from 19 areas around the Maritime Provinces of Canada and samples of lobsters stored in crates, cars and tidal pounds for various periods provided information about the accumulation of polycyclic aromatic hydrocarbons in lobsters. When exposed to **creosote**, accumulation of polycyclic aromatic hydrocarbons occurred in lobsters held in all types of storage units. The main possibility for appreciable concn of polycyclic aromatic hydrocarbons was among lobsters stored for 2-3 mo in tidal pounds constructed with creosoted materials. The accumulation was greater in summer than during winter storage in the pounds. Generally, fewer lobsters were stored in tidal pounds in summer than in winter.

[McLeese DW; Can Tech Rep Fish Aquat Sci 0 (1203): I-IV, 1-28 (1983)] **PEER REVIEWED**

RCRA Requirements :

U051; As stipulated in 40 CFR 261.33, when **creosote**, as a commercial chemical product or manufacturing chemical intermediate or an off-specification commercial chemical product or a manufacturing chemical intermediate, becomes a waste, it must be managed according to Federal and/or State hazardous waste regulations. Also defined as a hazardous waste is any residue, contaminated soil, water, or other debris resulting from the cleanup of a spill, into water or on dry land, of this waste. Generators of small quantities of this waste may qualify for partial exclusion from hazardous waste regulations (40 CFR 261.5).

[40 CFR 261.33 (7/1/91)] **PEER REVIEWED**

FIFRA Requirements :

A Registration Standard was issued April, 1986 for **creosote** used as a wood preservative.

[USEPA; Report on the Status of Chemicals in the Special Review Program and Registration Standards in the Reregistration Program p.2-5 (1989)] **PEER REVIEWED**

FIFRA Requirements :

Criteria of concern: oncogenicity, mutagenicity, and teratogenicity. Action/Use affected: In order to avoid cancellation, registrants must adhere to the terms and conditions of the Federal Register notices cited for **creosote** ... wood uses only. Reference: 49 FR 28666 (7/13/84); 51 FR 1334 (1/10/86)

[Environmental Protection Agency/OPTS. Suspended, Cancelled, and Restricted Pesticides. 5th Ed. Washington, DC: Environmental Protection Agency, February 1990.] **PEER REVIEWED**

Special Reports :

DHHS/ATSDR; Toxicological Profile for **Creosote** (1990) ATSDR/TP-90/09

Emergency Medical Treatment :

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The following Overview, *** PHENOL AND RELATED AGENTS ***, is relevant for this HSDB record chemical.

Life Support:

- o This overview assumes that basic life support measures have been instituted.

Clinical Effects:

0.2.1 SUMMARY OF EXPOSURE

0.2.1.1 ACUTE EXPOSURE

- A) USES: Phenol (also known as carbolic acid and phenic acid) is used in the treatment of localized skin disorders and as a local anesthetic. Dilute phenol solutions have been injected for celiac plexus nerve blocks. It is also used extensively in the manufacture of many other chemicals and drugs, as a dye and indicator, antiseptic, disinfectant, a reagent in chemical analysis, and a preservative for pharmaceuticals.
- B) PHARMACOLOGY: A phenol achieves its affect via several mechanisms.
- C) TOXICOLOGY: In concentrations of 5% or greater, it rapidly denatures all proteins it contacts. Some phenols, notably dinitrophenol or hydroquinone, will cause methemoglobinemia. There is also some thought that it may cause increased acetylcholine release at the neuromuscular junction causing CNS stimulatory effects.
- D) EPIDEMIOLOGY: Calls to poison centers concerning phenol are relatively rare, but many workers in various industries may be exposed to low levels of phenol. Severe manifestations and deaths are very rare.
- E) WITH POISONING/EXPOSURE
 - 1) MILD TO MODERATE TOXICITY: Exposure causes irritation to the affected tissue (eg, skin, mucous membranes) and discoloration.
 - 2) SEVERE TOXICITY: Phenol toxicity occurs most frequently after acute ingestion or chronic dermal

application. However, systemic toxicity can also result from inhalation of vapors.

- 3) DERMAL: The major hazard of phenol is its ability to penetrate the skin rapidly, especially in its liquid form. Its strong corrosive effect on body tissue can cause severe chemical burns. However, due to its local anesthetizing properties, skin burns may be painless. Skin absorption can cause systemic symptoms and even death. Chronic exposure may lead to symptoms described for acute poisoning as well as eye and skin discoloration.
- 4) INGESTION: Phenol ingestion may cause oral, esophageal, and gastric burns. Systemic symptoms of toxicity include nausea, vomiting, diarrhea, dyspnea, tachypnea, pallor, profuse sweating, hypotension, dysrhythmias, acute lung injury, methemoglobinemia, hemolytic anemia, elevated anion gap metabolic acidosis, agitation, lethargy, seizures and coma.
- 5) PULMONARY: Inhalational exposures can cause digestive disturbances (vomiting, dysphagia, diarrhea, anorexia) and can irritate and even burn the respiratory tract. Signs and symptoms of chronic inhalation exposure may include headache, cough, weakness, fatigue, anorexia, nausea, vomiting, insomnia, nervousness, weight loss, paresthesias, ochronosis, and albuminuria.
- 6) OCULAR: Direct contact to the eyes may result in symptoms ranging from redness, pain, and blurred vision to severe burns that may lead to partial or even complete loss of vision.

0.2.1.2 CHRONIC EXPOSURE

- A) Chronic exposures have been reported to cause death from liver and kidney injuries. It may also affect the pancreas and heart muscle.
- B) Other signs and symptoms of chronic exposures include: headache, vertigo, fainting, cough, fatigue, muscle aches and pain, lack of appetite, difficulty swallowing, excess salivation, diarrhea, nausea, vomiting, insomnia, nervousness, weight loss, pallor, partial paralysis, ochronosis, albuminuria, and dark urine.
- C) Phenol is not considered a serious respiratory hazard in the workplace because of its low volatility.
- D) Skin the primary route of entry (for vapor, liquid, and solid forms of phenol). Skin absorption can occur even at low vapor concentration and without significant discomfort. Prolonged contact with the skin may cause dermatitis.

0.2.3 VITAL SIGNS

0.2.3.1 ACUTE EXPOSURE

- A) Hypotension, hypothermia, tachypnea, and tachycardia may develop with severe toxicity.

0.2.4 HEENT

0.2.4.1 ACUTE EXPOSURE

- A) Direct contact irritates eyes, nose and throat. Ingestion of a solution greater than 5% may result in oral burns. The affected area generally turns white and

is without pain but may become necrotic several days (ie, coagulation necrosis). Eye exposure may result in severe burns. Partial or complete loss of vision may occur.

0.2.5 CARDIOVASCULAR

0.2.5.1 ACUTE EXPOSURE

- A) Hypotension and tachycardia are commonly reported. Dysrhythmias have developed in patients following ingestion, while undergoing chemical face-peels with phenol, and after phenol injection. Cardiovascular collapse has occurred following injection of phenol.

0.2.6 RESPIRATORY

0.2.6.1 ACUTE EXPOSURE

- A) Tachypnea is commonly reported; acute lung injury and bronchospasm may also occur. Stridor has been reported from exposure to high concentrations of phenol. Respiratory arrest occurred 30 minutes post ingestion of 26.7 grams of phenol in one case.
- B) Phenol is not considered a serious respiratory hazard in the workplace because of its low volatility. However, systemic absorption can damage lungs and lead to cardiorespiratory collapse.

0.2.7 NEUROLOGIC

0.2.7.1 ACUTE EXPOSURE

- A) Initial CNS excitation, including seizures, is commonly followed by CNS depression ranging from lethargy to coma and death.

0.2.8 GASTROINTESTINAL

0.2.8.1 ACUTE EXPOSURE

- A) Phenol is extremely corrosive and may cause oral and esophageal burns and abdominal pain following ingestion.
 - 1) Contamination of chlorinated water supplies with phenol may lead to the production of chlorinated phenols and subsequent gastrointestinal disorders.

0.2.9 HEPATIC

0.2.9.1 ACUTE EXPOSURE

- A) Exposure may result in hepatic injury, which may result in death.

0.2.9.2 CHRONIC EXPOSURE

- A) Chronic exposures have been reported to cause death from liver damage.

0.2.10 GENITOURINARY

0.2.10.1 ACUTE EXPOSURE

- A) Renal toxicity associated with oliguria or anuria may occur. Exposure has resulted in acute tubular necrosis.

0.2.11 ACID-BASE

0.2.11.1 ACUTE EXPOSURE

- A) Metabolic acidosis may occur following ingestion.

0.2.13 HEMATOLOGIC

0.2.13.1 ACUTE EXPOSURE

- A) Methemoglobinemia may occur following exposure to some phenols, most notably dinitrophenol or hydroquinone. Deep venous thrombosis was reported following intravenous injection of phenol. Hemolysis, thrombocytopenia and mild coagulopathy are reported.

0.2.14 DERMATOLOGIC

0.2.14.1 ACUTE EXPOSURE

- A) Phenol is corrosive to the skin, but because of anesthetic qualities, it will numb rather than causing a burning pain on contact. Skin becomes red and swollen, then white and opaque (ie, coagulation necrosis). Deep burns result that may become gangrenous.
- B) Poisoning is usually through skin absorption; lethal quantities can be absorbed.
- C) Dermal contact with phenolic compounds may result in irritation, dermatitis, abnormal pigmentation, and burns. Dermatitis and depigmentation appear to be the most common adverse effects. Diaphoresis may develop with systemic toxicity.

0.2.14.2 CHRONIC EXPOSURE

- A) Dermatitis and leukoderma resulting from workplace contact with phenol or phenol containing products has been reported.

0.2.15 MUSCULOSKELETAL

0.2.15.2 CHRONIC EXPOSURE

- A) Chronic systemic absorption has caused blue or brown discoloration of the tendons over the knuckles of the hands.

0.2.16 ENDOCRINE

0.2.16.1 ACUTE EXPOSURE

- A) In experimental animal studies, resorcinol (ie, meta-hydroxyphenol) has been shown to be goitrogenic and to have antithyroid activity.

0.2.19 IMMUNOLOGIC

0.2.19.1 ACUTE EXPOSURE

- A) Phenol has been shown to be potentially immunotoxic in experimental animal studies.

0.2.20 REPRODUCTIVE HAZARDS

- A) A 27-year-old woman at 30 weeks of pregnancy unintentionally ingested 50 g of resorcinol, and developed unconsciousness, drowsiness, tonic-clonic seizures, hypothermia, and respiratory failure. Approximately 24 hours after delivery, the newborn was pronounced dead. Following supportive therapy, the mother was discharged home on day 15.
- B) Fetotoxicity and skeletal abnormalities have been reported in animal experiments.

0.2.21 CARCINOGENICITY

0.2.21.1 IARC CATEGORY

- A) IARC Carcinogenicity Ratings for CAS108-95-2 (IARC Working Group on the Evaluation of Carcinogenic Risks to Humans, 2006; IARC Working Group on the Evaluation of Carcinogenic Risks to Humans, 2007; IARC Working Group on the Evaluation of Carcinogenic Risks to Humans, 2010; IARC Working Group on the Evaluation of Carcinogenic Risks to Humans, 2010a; IARC Working Group on the Evaluation of Carcinogenic Risks to Humans, 2008; IARC, 2004):
 - 1) IARC Classification
 - a) Listed as: Phenol

b) Carcinogen Rating: 3

- 1) The agent (mixture or exposure circumstance) is not classifiable as to its carcinogenicity to humans. This category is used most commonly for agents, mixtures and exposure circumstances for which the evidence of carcinogenicity is inadequate in humans and inadequate or limited in experimental animals. Exceptionally, agents (mixtures) for which the evidence of carcinogenicity is inadequate in humans but sufficient in experimental animals may be placed in this category when there is strong evidence that the mechanism of carcinogenicity in experimental animals does not operate in humans. Agents, mixtures and exposure circumstances that do not fall into any other group are also placed in this category.

0.2.21.2 HUMAN OVERVIEW

- A) Although one study found a high risk of lung cancer among woodworkers exposed to phenol, subsequent studies have not demonstrated an increased risk of cancer. There is, however, a report of squamous cell cancer in situ related to **creosote** exposure.

0.2.21.3 ANIMAL OVERVIEW

- A) Phenol was not considered carcinogenic to rats or mice after oral exposure in drinking water. It was a promoter of skin cancer in mice.

0.2.22 GENOTOXICITY

- A) Phenol has caused DNA damage, mutations, chromosomal aberrations, unscheduled DNA synthesis, DNA inhibition and micronuclei in experimental animals and cultured cells.

0.2.23 OTHER

0.2.23.1 ACUTE EXPOSURE

- A) Facial peels with phenol have resulted in toxic shock syndrome.

Laboratory:

- A) Depending on the exposure, different laboratory studies are recommended. For patients with systemic or moderate to severe symptoms, obtain a complete blood count, electrolytes, urinalysis, and baseline renal function and liver enzyme measurements.
- B) Monitor methemoglobin concentration in patients with cyanosis or symptoms.
- C) In addition, careful monitoring of the patient's acid-base balance and continuous cardiac monitoring may be necessary.
- D) Chest radiographs are recommended in patients with inhalational exposure or respiratory symptoms.
- E) Phenol levels are not readily available or useful in exposed patients.

Treatment Overview:

0.4.2 ORAL EXPOSURE

A) MANAGEMENT OF MILD TO MODERATE TOXICITY

- 1) For mild to moderate toxicity, the mainstay of treatment is decontamination and supportive care. Rinse the mouth, and dilute with small amounts (up to 4

ounces for a child and 8 ounces for an adult) of water if the patient can tolerate it. Treat respiratory irritation with oxygen, administer inhaled beta agonists if bronchospasm develops.

B) MANAGEMENT OF SEVERE TOXICITY

- 1) For patients with severe toxicity, the mainstay of treatment continues to be decontamination and good supportive care. Endoscopy should be performed, ideally within 12 hours, after a significant ingestion to evaluate for GI burns. Treat seizures with benzodiazepines, add barbiturates or propofol if seizures persist. Treat symptomatic methemoglobinemia with methylene blue. Treat dysrhythmias with standard ACLS antidysrhythmics. DERMAL: For dermal burns apply antibiotic ointment, cover with a sterile dressing and for severe extensive burns consult a burn surgeon. INHALATION: Administer oxygen, inhaled beta agonists for bronchospasm. Early airway management if there is evidence of upper airway burns or swelling or severe respiratory distress. OCULAR: After irrigation evaluate visual acuity and perform a slit lamp exam. Consult an ophthalmologist if ocular burns are present, antibiotics and mydriatics may be indicated along with close follow-up. PARENTERAL: Supportive care for symptoms. There is one case report for the use of charcoal hemoperfusion improving clinical status and decreased total and free phenol injections in an adult.

C) DECONTAMINATION

- 1) PREHOSPITAL: Rinse the mouth, and dilute with small amounts (up to 4 ounces for a child and 8 ounces for an adult) of water if the patient is awake and alert.
- 2) HOSPITAL: Rinse the mouth, and dilute with small amounts (up to 4 ounces for a child and 8 ounces for an adult) of water if the patient is awake and alert. For large recent ingestions, consider inserting a small flexible nasogastric tube and aspirating stomach contents. The risk of traumatic injury should be weighed against the potential benefit.
 - a) INHALATION: Remove patient to fresh air, and support patient with supplementary oxygen and ventilation as needed.
 - b) DERMAL: Immediately flush skin with large amounts of water and remove contaminated clothing as soon as possible.
 - c) OCULAR: Immediately flush eye(s) with water for at least 15 minutes.

D) AIRWAY MANAGEMENT

- 1) Patients with severe respiratory symptoms, upper airway injury, or critically ill with CNS depression or seizures may require early intubation.

E) ANTIDOTE

- 1) There is no specific antidote for phenol exposures. Although some MSDS information will mention a phenol antidote kit with castor oil or other vegetable oils or polyethylene glycol, there is no evidence that use of such kits are effective.

F) ENHANCED ELIMINATION

- 1) There is one case report of the use of charcoal hemoperfusion for a phenol overdose from accidental IV injection.

G) PATIENT DISPOSITION

- 1) HOME CRITERIA: Patients who are asymptomatic or with minimal symptoms after inadvertent exposure to low concentration products and are otherwise improving may be managed at home.
- 2) OBSERVATION CRITERIA: Patients with deliberate ingestions, eye exposure, or symptoms should be sent to a health care facility for observation until they are clearly improving and clinically stable.
- 3) ADMISSION CRITERIA: Patients with worsening symptoms or severe systemic symptoms should be admitted to the hospital for further evaluation. Patients with severe grade II or grade III GI burns on endoscopy, seizures, mental status changes, dysrhythmias, or severe respiratory distress require ICU admission. Patients should remain admitted until they are clearly improving and clinically stable.
- 4) CONSULT CRITERIA: Consult a medical toxicologist or poison center for any patient with systemic symptoms, severe exposure, or in whom the diagnosis is unclear. Consult a gastroenterologist to perform endoscopy in any patient drooling, stridor, persistent vomiting or dysphagia or a significant ingestion. Patients with severe or extensive dermal burns should be evaluated by a burn specialist. Patients with eye burns should be evaluated by an ophthalmologist.

H) PITFALLS

- 1) There is no evidence for the use of phenol antidote kits. The mainstay of initial treatment should be decontamination and/or removal from phenol exposure. Patients with GI burns on endoscopy are at risk for stricture formation and should be followed up in 2 to 3 weeks for barium swallow or repeat endoscopy.

I) PHARMACOKINETICS

- 1) Pheno vapor is rapidly absorbed after ingestion, dermal exposure, and inhalation. Peak plasma levels in patients receiving lumbar or thoracic sympathetic blocks with unconjugated and conjugated phenols occurred roughly at 20 minutes and 1 hour, respectively. Elimination half-life in the cases of these blocks was approximately 30 minutes for unconjugated phenols and 1 hour for conjugated phenols. Dermal exposures to the skin has a peak blood phenol concentration in 1 hour. Phenol is mostly metabolically converted into water-soluble sulfates and glucuronides, small amounts are excreted unchanged. Excretion is primarily renal.

J) TOXICOKINETICS

- 1) In once case of dermal exposure, an adult male with a 90% phenol exposure had an elimination half-life of approximately 14 hours.

K) DIFFERENTIAL DIAGNOSIS

- 1) The differential diagnosis to phenol exposures includes other caustics that can cause respiratory irritation or topical burns or other caustics that can cause respiratory irritation or topical burns or other drug substances that can cause methemoglobinemia.
- 0.4.3 INHALATION EXPOSURE
- A) INHALATION: Move patient to fresh air. Monitor for respiratory distress. If cough or difficulty breathing develops, evaluate for respiratory tract irritation, bronchitis, or pneumonitis. Administer oxygen and assist ventilation as required. Treat bronchospasm with inhaled beta2 agonist and oral or parenteral corticosteroids.
 - B) ACUTE LUNG INJURY: Maintain ventilation and oxygenation and evaluate with frequent arterial blood gas or pulse oximetry monitoring. Early use of PEEP and mechanical ventilation may be needed.
- 0.4.4 EYE EXPOSURE
- A) DECONTAMINATION: Irrigate exposed eyes with copious amounts of room temperature water for at least 15 minutes. If irritation, pain, swelling, lacrimation, or photophobia persist, the patient should be seen in a health care facility.
- 0.4.5 DERMAL EXPOSURE
- A) OVERVIEW
 - 1) Remove phenol with undiluted polyethylene glycol 300 to 400 or isopropyl alcohol prior to washing, if readily available. Wash exposed areas twice or for at least 10 minutes with large quantities of SOAPY water. Water alone may be harmful. A physician may need to examine the exposed area if irritation or pain persist after the area is washed.

Range of Toxicity:

- A) TOXIC DOSE: ADULT: Acute ingestion of as little as 1 g of phenol in adults has resulted in death, but toxicity has been noted at significantly lower doses. The minimum toxic dose of phenol and related compounds is not well established in the literature, which consists mostly of case reports. PEDIATRIC: Reportedly, ingestions of 50 to 500 mg in infants have been lethal.
- B) THERAPEUTIC DOSE: ADULT: A typical oral dose in lozenges for oral use is 32.5 mg phenol per lozenge every 2 hours as needed. In adults, 0.6 to 1.3 g phenol daily has been used therapeutically. PEDIATRIC: In children over the age of 3, phenol lozenges containing 10 to 500 mg may be given every 2 hours. Chloraseptic liquids and sprays have also been used in children and adults.

[Rumack BH POISINDEX(R) Information System Micromedex, Inc., Englewood, CO, 2013; CCIS Volume 158, edition expires Nov, 2013. Hall AH & Rumack BH (Eds): TOMES(R) Information System Micromedex, Inc., Englewood, CO, 2013; CCIS Volume 158, edition expires Nov, 2013.] **PEER REVIEWED**