ionz

SAFETY DATA SHEET

Updated on 10/09/2024

1. Material Identification

Product Name : Ethanol
Catalog Number : io-5661
CAS Number : 64-17-5
Identified uses : Laboratory chemicals, manufacture of chemical compounds
Company : Ionz

>> R&D Use only

2. Hazards Identification

GHS Classification:

Flammable liquid (category 2)
Acute toxicity, oral (Category 3)
Acute toxicity, dermal (Category 3)
Acute toxicity, inhalation (Category 3)
Specific target organ toxicity, single exposure (Category 1)

Pictogram(s)

GHS Hazard Statements

>> H225 (99.99%): Highly Flammable liquid and vapor [Danger Flammable liquids]

>> H319 (37.63%): Causes serious eye irritation [Warning Serious eye damage/eye irritation]

Precautionary Statement Codes

>> P210, P233, P240, P241, P242, P243, P264+P265, P280, P303+P361+P353, P305+P351+P338, P337+P317, P370+P378, P403+P235, and P501

NFPA 704 Diamond

NFPA Health Rating

>> 2 - Materials that, under emergency conditions, can cause temporary incapacitation or residual injury.

NFPA Fire Rating

>> 3 - Liquids and solids that can be ignited under almost all ambient temperature conditions. Materials produce hazardous atmospheres with air under almost all ambient temperatures or, though unaffected by ambient temperatures, are readily ignited under almost all conditions.

NFPA Instability Rating

>> 0 - Materials that in themselves are normally stable, even under fire conditions.

EPA Safer Chemical:

EPA labels products so that consumers can easily choose ones that are safer for people and the environment. When consumers see the Safer Choice label on a product, they can be confident that the ingredients have been through a

rigorous EPA review. The label means that EPA scientists have evaluated every ingredient in the product to ensure it meets Safer Choice's stringent criteria. When people use Safer Choice products, they are protecting their families and the environment by making safer chemical choices.

EPA Safer Chemical

>> Chemical: Ethanol

>> Green circle - The chemical has been verified to be of low concern based on experimental and modeled data.

Health Hazards:

>> Excerpt from ERG Guide 127 [Flammable Liquids (Water-Miscible)]:

>> Inhalation or contact with material may irritate or burn skin and eyes. Fire may produce irritating, corrosive and/or toxic gases. Vapors may cause dizziness or asphyxiation, especially when in closed or confined areas. Runoff from fire control or dilution water may cause environmental contamination. (ERG, 2024)

ERG 2024, Guide 127 (Ethanol, solution; Ethanol)

>> Inhalation or contact with material may irritate or burn skin and eyes.

>> Fire may produce irritating, corrosive and/or toxic gases.

>> Vapors may cause dizziness or asphyxiation, especially when in closed or confined areas.

>> Runoff from fire control or dilution water may cause environmental contamination.

ERG 2024, Guide 127 (Ethyl alcohol; Ethyl alcohol, solution)

>> Inhalation or contact with material may irritate or burn skin and eyes.

>> Fire may produce irritating, corrosive and/or toxic gases.

>> Vapors may cause dizziness or asphyxiation, especially when in closed or confined areas.

>> Runoff from fire control or dilution water may cause environmental contamination.

>> Excerpt from ERG Guide 127 [Flammable Liquids (Water-Miscible)]:

>> HIGHLY FLAMMABLE: Will be easily ignited by heat, sparks or flames. CAUTION: Ethanol (UN1170) can burn with an invisible flame. Use an alternate method of detection (thermal camera, broom handle, etc.) Vapors may form explosive mixtures with air. Vapors may travel to source of ignition and flash back. Most vapors are heavier than air. They will spread along the ground and collect in low or confined areas (sewers, basements, tanks, etc.). Vapor explosion hazard indoors, outdoors or in sewers. Those substances designated with a (P) may polymerize explosively when heated or involved in a fire. Runoff to sewer may create fire or explosion hazard. Containers may explode when heated. Many liquids will float on water. (ERG, 2024)

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>> Containers may explode when heated.

>> Many liquids will float on water.

>> Highly flammable. Vapour/air mixtures are explosive. Risk of fire and explosion on contact with incompatible substances. See Chemical Dangers.

3. Composition/Information On Ingredients

Chemical name : Ethanol
CAS Number : 64-17-5
Molecular Formula : C2H6O
Molecular Weight : 46.0700 g/mol

4. First Aid Measures

First Aid:

>> Excerpt from ERG Guide 127 [Flammable Liquids (Water-Miscible)]:

>> Refer to the "General First Aid" section. Specific First Aid: Wash skin with soap and water. In case of burns, immediately cool affected skin for as long as possible with cold water. Do not remove clothing if adhering to skin. (ERG, 2024)

ERG 2024, Guide 127 (Ethanol, solution; Ethanol)

>> General First Aid:

>> Call 911 or emergency medical service.

>> Ensure that medical personnel are aware of the material(s) involved, take precautions to protect themselves and avoid contamination.

>> Move victim to fresh air if it can be done safely.

>> Administer oxygen if breathing is difficult.

>> If victim is not breathing:

>> DO NOT perform mouth-to-mouth resuscitation; the victim may have ingestedor inhaled the substance.

>> If equipped and pulse detected, wash face and mouth, then give artificial respiration using a proper respiratory medical device (bag-valve mask, pocket mask equipped with a one-way valve or other device).

>> If no pulse detected or no respiratory medical device available, provide continuouscompressions. Conduct a pulse check every two minutes or monitor for any signs of spontaneous respirations.

>> Remove and isolate contaminated clothing and shoes.

>> For minor skin contact, avoid spreading material on unaffected skin.

>> In case of contact with substance, remove immediately by flushing skin or eyes with running water for at least 20 minutes.

>> For severe burns, immediate medical attention is required.

>> Effects of exposure (inhalation, ingestion, or skin contact) to substance may be delayed.

>> Keep victim calm and warm.

>> Keep victim under observation.

>> For further assistance, contact your local Poison Control Center.

>> Note: Basic Life Support (BLS) and Advanced Life Support (ALS) should be done by trained professionals.

>> Specific First Aid:

>> Wash skin with soap and water.

>> In case of burns, immediately cool affected skin for as long as possible with cold water. Do not remove clothing if adhering to skin.

>> In Canada, an Emergency Response Assistance Plan (ERAP) may be required for this product. Please consult the shipping paper and/or the "ERAP" section.

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First Aid Measures

Inhalation First Aid

>> Fresh air, rest.

Skin First Aid

>> Remove contaminated clothes. Rinse skin with plenty of water or shower.

Eye First Aid

>> First rinse with plenty of water for several minutes (remove contact lenses if easily possible), then refer for medical attention.

Ingestion First Aid

>> Rinse mouth. Give one or two glasses of water to drink. Refer immediately for medical attention.

5. Fire Fighting Measures

>> Excerpt from ERG Guide 127 [Flammable Liquids (Water-Miscible)]:

>> CAUTION: The majority of these products have a very low flash point. Use of water spray when fighting fire may be inefficient. CAUTION: For fire involving UN1170, UN1987 or UN3475, alcohol-resistant foam should be used. CAUTION:

Ethanol (UN1170) can burn with an invisible flame. Use an alternate method of detection (thermal camera, broom handle, etc.).

>> SMALL FIRE: Dry chemical, CO2, water spray or alcohol-resistant foam.

>> LARGE FIRE: Water spray, fog or alcohol-resistant foam. Avoid aiming straight or solid streams directly onto the product. If it can be done safely, move undamaged containers away from the area around the fire.

>> FIRE INVOLVING TANKS, RAIL TANK CARS OR HIGHWAY TANKS: Fight fire from maximum distance or use unmanned master stream devices or monitor nozzles. Cool containers with flooding quantities of water until well after fire is out. Withdraw immediately in case of rising sound from venting safety devices or discoloration of tank. ALWAYS stay away from tanks in direct contact with flames. For massive fire, use unmanned master stream devices or monitor nozzles; if this is impossible, withdraw from area and let fire burn. (ERG, 2024)

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>> Use water spray, powder, alcohol-resistant foam, carbon dioxide. In case of fire: keep drums, etc., cool by spraying with water.

6. Accidental Release Measures

Isolation and Evacuation:

Isolation and evacuation measures to take when a large amount of this chemical is accidentally released in an emergency.

>> Excerpt from ERG Guide 127 [Flammable Liquids (Water-Miscible)]:

>> IMMEDIATE PRECAUTIONARY MEASURE: Isolate spill or leak area for at least 50 meters (150 feet) in all directions.

>> LARGE SPILL: Consider initial downwind evacuation for at least 300 meters (1000 feet).

>> FIRE: If tank, rail tank car or highway tank is involved in a fire, ISOLATE for 800 meters (1/2 mile) in all directions; also, consider initial evacuation for 800 meters (1/2 mile) in all directions. (ERG, 2024)

Evacuation: ERG 2024, Guide 127 (Ethanol, solution; Ethanol)

>> Immediate precautionary measure

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>> Large Spill

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Spillage Disposal:

Methods for containment and safety measures to protect workers dealing with a spillage of this chemical.

>> Remove all ignition sources. Ventilation. Do NOT wash away into sewer. Collect leaking and spilled liquid in covered containers as far as possible. Absorb remaining liquid in inert absorbent. Wash away remainder with plenty of water. Store and dispose of according to local regulations.

Accidental Release Measures

Public Safety: ERG 2024, Guide 127 (Ethanol, solution; Ethanol)

>> CALL 911. Then call emergency response telephone number on shipping paper. If shipping paper not available or no answer, refer to appropriate telephone number listed on the inside back cover.

>> Keep unauthorized personnel away.

>> Stay upwind, uphill and/or upstream.

>> Ventilate closed spaces before entering, but only if properly trained and equipped.

Spill or Leak: ERG 2024, Guide 127 (Ethanol, solution; Ethanol)

>> ELIMINATE all ignition sources (no smoking, flares, sparks or flames) from immediate area.

>> All equipment used when handling the product must be grounded.

>> Do not touch or walk through spilled material.

>> Stop leak if you can do it without risk.

>> Prevent entry into waterways, sewers, basements or confined areas.

>> A vapor-suppressing foam may be used to reduce vapors.

>> Absorb or cover with dry earth, sand or other non-combustible material and transfer to containers.

>> Use clean, non-sparking tools to collect absorbed material.

>> Large Spill

>> Dike far ahead of liquid spill for later disposal.

>> Water spray may reduce vapor, but may not prevent ignition in closed spaces.

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7. Handling And Storage

Safe Storage:

>> Fireproof. Separated from : see Chemical Dangers.

Storage Conditions:

>> Keep container tightly closed in a dry and well-ventilated place. Containers which are opened must be carefully resealed and kept upright to prevent leakage. Hygroscopic. Storage class (TRGS 510): Flammable liquids

8. Exposure Control/ Personal Protection

REL-TWA (Time Weighted Average)

>> 1000 ppm (1900 mg/m³)

>> TWA 1000 ppm (1900 mg/m3)

>> 1000.0 [ppm]

PEL-TWA (8-Hour Time Weighted Average)

>> 1000 ppm (1900 mg/m³)

TLV-STEL

>> 1000.0 [ppm]

>> 15 min Short Term Exposure Limit (STEL): 1000 ppm.

>> 1000 ppm as STEL; A3 (confirmed animal carcinogen with unknown relevance to humans).

TLV-STEL (Short Term Exposure Limit)

>> 1000 ppm [2008]

MAK (Maximale Arbeitsplatz Konzentration)

>> 380 mg/m

Emergency Response: ERG 2024, Guide 127 (Ethanol, solution; Ethanol)

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>> If it can be done safely, move undamaged containers away from the area around the fire.

>> Fire Involving Tanks, Rail Tank Cars or Highway Tanks

>> Fight fire from maximum distance or use unmanned master stream devices or monitor nozzles.

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>> ERPG-1: 1800 ppm - one hour exposure limit: 1 = mild transient health effects or objectionable odor [AIHA]

>> ERPG-2: 3300 ppm - one hour exposure limit: 2 = impaired ability to take protective action [AIHA]

>> ERPG-3: Not appropriate - one hour exposure limit: 3 = life threatening health effects [AIHA]

Inhalation Risk:

>> A harmful contamination of the air will be reached rather slowly on evaporation of this substance at 20 °C.

Effects of Short Term Exposure:

>> The substance is severely irritating to the eyes. The vapour at high levels is irritating to the eyes and respiratory tract. The substance may cause effects on the central nervous system.

Effects of Long Term Exposure:

>> The substance defats the skin, which may cause dryness or cracking. The substance may have effects on the upper respiratory tract and central nervous system. This may result in irritation, headache, fatigue and lack of concentration.

Fire Prevention

>> NO open flames, NO sparks and NO smoking. Closed system, ventilation, explosion-proof electrical equipment and lighting. Do NOT use compressed air for filling, discharging, or handling. NO contact with incompatible materials: See Chemical Dangers

Exposure Prevention

>> STRICT HYGIENE! PREVENT GENERATION OF MISTS!

Inhalation Prevention

>> Use ventilation, local exhaust or breathing protection.

Skin Prevention

>> Protective clothing. Apron. Protective gloves.

Eye Prevention

>> Wear safety goggles.

Ingestion Prevention

>> Do not eat, drink, or smoke during work.

Exposure Control and Personal Protection

Protective Clothing: ERG 2024, Guide 127 (Ethanol, solution; Ethanol)

>> Wear positive pressure self-contained breathing apparatus (SCBA).

>> Structural firefighters' protective clothing provides thermal protection but only limited chemical protection.

Protective Clothing: ERG 2024, Guide 127 (Ethyl alcohol; Ethyl alcohol, solution)

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RD50 (Exposure concentration producing a 50% respiratory rate decrease)

>> 27314.0 [mmHg]

Maximum Allowable Concentration (MAK)

>> 200.0 [ppm]

9. Physical And Chemical Properties

Molecular Weight:

>> 46.07

Exact Mass:

>> 46.041864811

Physical Description:

>> Ethanol with a small amount of an adulterant added so as to be unfit for use as a beverage.

>> COLOURLESS LIQUID WITH CHARACTERISTIC ODOUR.

Color/Form:

>> Clear, colorless, very mobile liquid

Odor:

>> Pleasant

Taste:

The sensation of flavor perceived in the mouth and throat on contact with a substance.

>> Burning

Boiling Point:

>> 173.3 °F at 760 mmHg (NTP, 1992)

>> 78 °C

Melting Point:

>> -173.4 °F (NTP, 1992)

>> -114 °C

Flash Point:

>> 55 °F (NTP, 1992)

>> 12.0 °C c.c.

Solubility:

>> greater than or equal to 100 mg/mL at 73 °F (NTP, 1992)

>> Solubility in water: miscible

Density:

>> 0.79 at 68 °F (USCG, 1999) - Less dense than water; will float

>> Relative density (water = 1): 0.79

Vapor Density:

>> 1.59 (NTP, 1992) - Heavier than air; will sink (Relative to Air)

>> Relative vapor density (air = 1): 1.6

Vapor Pressure:

>> 40 mmHg at 66 °F ; 50 mmHg at 77 °F (NTP, 1992)

>> Vapor pressure, kPa at 20 °C: 5.8

LogP:

>> -0.32

Stability/Shelf Life:

>> Stable under recommended storage conditions.

Autoignition Temperature:

>> 689 °F (USCG, 1999)

>> 400 °C

Viscosity:

>> 1.074 mPa.s at 25 °C

>> 1.074 mPa*s at 20 °C

Heat of Combustion:

>> 1336.8 kJ/mol at 25 °C

Heat of Vaporization:

>> 42.32 kJ/mol at 25 °C

Surface Tension:

>> 21.97 mN/m at 25 °C

Ionization Potential:

>> 10.47 eV

Odor Threshold:

>> Odor Threshold Low: 49.0 [mmHg]

>> Odor Threshold High: 716.0 [mmHg]

>> Detection odor threshold from AIHA (mean = 180 ppm)

Refractive Index:

>> Index of refraction: 1.3611 at 20 °C/D

Dissociation Constants:

pKa

>> 15.9 (at 25 °C)

>> pKa = 15.9 at 25 °C

10. Stability And Reactivity

>> Highly flammable. Water soluble.

CSL No

>> CSL00065

Reactants/Reagents

>> CALCIUM HYPOCHLORITE + ETHANOL

Warning Message

>> Potentially explosive

GHS Category

>> Explosive

Reference Source

>> User-Reported

Modified Date

>> 7/8/18

Create Date

>> 6/27/17

Additional Information

>> Unanticipated explosion due to formation of the side product 5-azidotetrazole

DOI Link

>> http://pubs.acs.org/cen/safety/20050725.html

Reaction Scale

>> Not Available

11. Toxicological Information

Toxicity Summary:

>> IDENTIFICATION AND USE: Ethanol is a clear, colorless, very mobile liquid. It is used in alcoholic beverages in suitable dilutions, and as a reagent in synthetic organic chemistry and chromatography, as well as industrial and laboratory organic solvent. Other uses are in manufacture of denatured alcohol, pharmaceuticals (rubbing compounds, lotions, tonics, colognes), in perfumery. Octane booster in gasoline. Pharmaceutic aid (solvent). HUMAN STUDIES: Ethanol is a central nervous system (CNS) depressant. It enhances the inhibitory effects of gamma-aminobutyric acid (GABA) at the GABA-A receptor and competitively inhibits the binding of glycine at the N-methyl-d-aspartate receptor (it disrupts excitatory glutaminergic neurotransmission). Ethanol also stimulates release of other inhibitory neurotransmitters, such as dopamine and serotonin. The most common clinical signs of ethanol toxicosis are ataxia, lethargy, vomiting, and recumbency. In more severe cases, hypothermia, disorientation, vocalization, hypotension, tremors, tachycardia, acidosis, diarrhea, respiratory depression, coma, seizures, and death may occur. Alcohol is directly irritating to the stomach and causes vomiting. High ethanol blood levels also stimulate emesis. The concern with vomiting during intoxication is that at high blood ethanol concentrations, the muscles that control the epiglottis become slow to react or even paralyzed. This increases the risk for aspiration. Ethanol intoxication reduces peripheral oxygen delivery and metabolism and causes mitochondrial oxidative dysfunction, potentially resulting in shock or hypoxia in an acutely intoxicated patient. Hypothermia may result from multiple mechanisms. Peripheral vasodilation, CNS depression, ethanol interference with the thermoregulator mechanism, and/or impaired behavioral responses to a cold environment all lead to a lowered body temperature. Moderate ethanol intake appears to reduce the risk of myocardial infarction and other heart diseases. However, high spirits consumption was associated with increased risk of cancer mortality in women. Consumption of alcoholic beverages (beer, in particular) is associated with an increased risk for rectal but not colon cancer. Beer is a commonly consumed alcoholic beverage among reproductive-age adults. Beer drinking males have an increased risk of contributing to pregnancy waste. Women consume beer before and after pregnancy recognition. Binge drinking appears to be a common drinking behavior, and those who binge drink have an increased risk of impaired fetal growth and offspring behavior. Beer consumption by lactating women might temporarily impair motor function of nursing infants. The rate of ethanol metabolism varies among individuals. Studies of twins indicate that interindividual variability in the rate of ethanol metabolism may be genetically controlled. The main pathway for ethanol oxidation in humans is to acetaldehyde via alcohol dehydrogenase pathway. Acetaldehyde is oxidized further to acetic acid by aldehyde dehydrogenase. Asians are known to be sensitive to the health effects of ethanol; the sensitivity has been attributed to different forms of the enzyme acetaldehyde dehydrogenase. Alcohol ingestion by Asians resulted in marked elevations of blood acetaldehyde levels ranging from 0.4 to 3 mg/L, and individuals developed facial flushing and tachycardia as a direct consequence of elevated blood acetaldehyde levels. ANIMAL STUDIES: A drop full-strength ethanol on rabbit eyes causes reversible injury graded only 3 on a scale of 10 after 24 hr. Application of 70% alcohol to rabbit corneas injures and temporarily loosens the corneal epithelium, but the recovery is complete. When rats were dosed with ethanol by oral gavage with 8 to 15 g/kg/day over 4 months and fed a diet containing 25% of total calories as fat, focal necrosis, inflammation, and fibrosis were observed in the liver. Nine baboons fed ethanol at 50% of total calories developed fatty liver, and four animals developed hepatitis within 9 to 12 months. Rabbits exposed to saturated vapors of ethanol for periods ranging from 25 to 365 days developed cirrhosis of the liver. Rats were given a single intraperitoneal dose of diethylnitrosamine followed by treatment with ethanol in drinking water for 12 to 18 months. Ethanol was an effective promoter of liver tumors. Cynomolgus monkeys administered up to 5 g/kg bw ethanol daily on gestation days 20-150 revealed an increase in pregnancy wastage (abortions and still births) but no structural malformation or facial change. Ethanol, and not acetaldehyde, has been implicated as the causative agent of the teratogenic effects in laboratory animals. Oral coadministration of 100 mg/kg of 4-methylpyrazole, an inhibitor of alcohol dehydrogenase, with 6 g/kg of ethanol intraperitoneally on gestation day 10 dramatically increased the embryotoxicity of ethanol in mice. Ethanol is not mutagenic in Salmonella typhimurium strains TA 97, TA 98, TA 100, TA 1535, TA 1537, or TA 1538 in the presence or absence of metabolic activation. In the presence of a metabolic activation system, ethanol is slightly mutagenic to Salmonella strain TA 102, a strain considered to respond to the presence of oxygen radicals. Ethanol did not induce mutations in mouse lymphoma L5178Y TK+/- cells and did not induce micronuclei in Chinese hamster V79 cells in the absence of metabolic activation. No chromosomal aberrations or sister chromatid exchanges were observed in Chinese hamster ovary cells treated with ethanol. ECOTOXICITY STUDIES: The

zebrafish were exposed to different concentrations (control, 0.01, 0.1, and 1%) of ethanol from blastula stage to 144 hour-post-fertilization (hpf). No effect on survival was observed except the 1% ethanol group suffered 89% mortality during 108-120 hpf. No developmental defects were observed at the 0.01 and 0.1% concentrations, but significantly higher deformity rates occurred with 1% ethanol. Hyperactivity and less tortuous swimming paths were observed in all ethanol concentrations.

Evidence for Carcinogenicity:

Evidence that this chemical does or may cause cancer. The information here is collected from various sources by the Hazardous Substances Data Bank (HSDB).

>> A3; Confirmed animal carcinogen with unknown relevance to humans.

Carcinogen Classification:

This section provides the International Agency for Research on Cancer (IARC) Carcinogenic Classification and related monograph links. In the IARC Carcinogenic classification, chemicals are categorized into four groups: Group 1 (carcinogenic to humans), Group 2A (probably carcinogenic to humans), Group 2B (possibly carcinogenic to humans), and Group 3 (not classifiable as to its carcinogenicity to humans).

IARC Carcinogenic Agent

>> Ethanol in alcoholic beverages

IARC Carcinogenic Classes

>> Group 1: Carcinogenic to humans

IARC Monographs

>> Volume 96: (2010) Alcohol Consumption and Ethyl Carbamate

>> Volume 100E: (2012) Personal Habits and Indoor Combustions

>> Ethanol in alcoholic beverages is carcinogenic to humans (Group 1). (L135)

Health Effects:

>> Acute: At 0.1% blood alcohol levels individuals experience CNS depression, nausea, possible vomiting, impaired cognition and impaired motor and sensory function. Accidents or injury can also occur due to the side effects of loss of coordination, slowed reaction time, sleepiness and impaired judgment. At >0.14% blood alcohol levels there is decreased blood flow to the brain. At greater than 0.3% blood alcohol there is a marked degree of stupefaction and possible unconsciousness. At levels greater than 0.4% there is a risk of death. Acute consumption leading to blood alcohol levels greater than 0.5% is almost universally fatal. Chronic: high levels of alcohol consumption are associated with an increased risk of alcoholism, malnutrition, chronic pancreatitis, alcoholic (fatty) liver disease, and cancer. Frequent drinking of alcoholic beverages has been shown to be a major contributing factor in cases of elevated blood levels of triglycerides. In addition, damage to the central nervous system and peripheral nervous system can occur from chronic alcohol abuse. The long-term use of alcohol is capable of damaging nearly every organ and system in the body. The developing adolescent brain is particularly vulnerable to the toxic effects of alcohol. In addition, the developing fetal brain is also vulnerable, and fetal alcohol syndrome (FAS) may result if pregnant mothers consume alcohol. The net effect of alcohol consumption on global human health is quite detrimental, with an estimated 3.8% of all global deaths and 4.6% of global disability-adjusted life-years attributable to alcohol. Ethanol is considered a teratogen (causing fetal alcohol syndrome) and a Group 1 carcinogen because of the carcinogenicity of acetaldehyde (a major metabolite of alcohol).

Effects During Pregnancy and Lactation:

Drug effects during pregnancy and lactation.

>> ◉ Summary of Use during Lactation

>> The effects of maternal alcohol (ethanol) ingestion during lactation are complex and depend on the pattern of maternal drinking. Alcohol decreases milk production, with 5 drinks or more decreasing milk letdown and disrupting nursing until maternal alcohol levels decrease. Beer may increase serum prolactin levels during nursing because of polysaccharides from barley and hops. After ingestion of nonalcoholic beer, the antioxidant capacity of milk is increased, but alcohol levels in milk are negligible. Women with a family history of alcoholism have a blunted prolactin response following breast stimulation and tend to breastfeed more frequently to compensate.

>> Breastmilk alcohol levels closely parallel blood alcohol levels. The highest alcohol levels in milk occur 30 to 60 minutes after an alcoholic beverage, but food delays the time of peak milk alcohol levels. Nursing after 1 or 2 drinks (including beer) can decrease the infant's milk intake by 20 to 23% and cause infant agitation and poor sleep patterns. Nursing or pumping within 1 hour before ingesting alcohol may slightly reduce the subsequent amounts of alcohol in breastmilk.

>> Casual use of alcohol (such as 1 glass of wine or beer per day) is unlikely to cause either short- or long-term problems in the nursing infant, especially if the mother waits 2 to 2.5 hours per drink before nursing, and does not appear to affect breastfeeding duration. Daily heavy use of alcohol (more than 2 drinks daily) appears to decrease the length of

time that mothers breastfeed their infants. The long-term effects of daily use of alcohol on the infant are unclear. Some evidence indicates that infant growth and motor function may be negatively affected by 1 drink or more daily, but other studies have not confirmed these findings. Heavy maternal use may cause excessive sedation, fluid retention, and hormone imbalances in breastfed infants. Greater or riskier alcohol consumption by nursing mothers may affect their children's academic performance negatively in school. Preliminary data failed to find an increased risk of autism spectrum disorder or attention deficit hyperactivity disorder among the infants whose mothers used alcohol during breastfeeding. The use of alcohol-based hand sanitizers do not appear to result in clinically relevant alcohol levels in breastmilk.

>> ◉ Effects in Breastfed Infants

>> A nursing mother was drinking large amounts of quinine wine, wine, champagne, beer and liquors. Her infant had been gaining 30 g of weight daily until he weighed nearly 6 kg at 5 weeks of age. The infant had been restless and sleepless for several days when he suffered from violent fits and tonic-clonic seizures that required medical treatment. After he was taken off the mother's breast and began to be nursed by a wet nurse, his weight quickly dropped by 200 g in 3 days and fell into a pattern of calm sleep.

>> A similar case of chronic heavy alcohol use by a nursing mother resulted in pseudo-Cushing syndrome in her 4-month-old breastfed infant. The infant had a bloated appearance, excessive wight gain and diminished length for age. The mother reported drinking 50 cans of beer weekly and "generous" amounts of other alcoholic beverages to increase her milk supply. The infant's symptoms resolved and growth pattern returned to normal after her mother stopped consuming alcohol.

>> A series of 23 cases of severe thrombocytopenia and bleeding were reported among 21- to 60-day-old breastfed infants of Chinese women in Singapore over a 5-year period. None of the infants had received prophylactic vitamin K at birth and all of their mothers had been taking alcohol tonics after each meal beginning at 7 to 10 days after delivery which was a common practice among only the Chinese in the mixed ethnic population delivering at the hospital. Most of the infants had also been receiving 5 to 15 mL daily of "gripe water" which had an alcohol content of about 5%. The authors attributed these cases to the lack of prophylactic vitamin K (which was common practice at the time) and increased clotting factor degradation caused by alcohol.

>> A woman who drank 750 mL of port wine in 24 hours noticed that her breastfed 8-day-old had a deep unarousable sleep, snoring, pain insensitivity, inability to suck, excessive perspiration and a feeble pulse. These symptoms were attributed to the very young age of the infant and the large amount of alcohol consumed.

>> In a series of studies, investigators measured the effect of maternal alcohol use on their breastfed infants. In one study, 12 nursing mothers with infants 25 to 216 days of age drank 0.3 grams/kg of alcohol (about 1.5 drinks for a 60 kg woman) in orange juice over 15 minutes in the morning. On a separate occasion, they drank an equal volume of orange juice. In another study, 12 nursing mothers nursing infants with a median age of 150 days drank 0.3 grams/kg of alcohol as beer or the same volume of nonalcoholic beer on a separate occasion. In a third study, 12 nursing mothers with infants averaging 3.1 months of age drank 0.3 grams/kg of alcohol in orange juice over 15 minutes in the morning. On a separate occasion, they drank an equal volume of orange juice In both studies, infants who drank milk that contained alcohol consumed 20 to 23% less milk during the 3- or 4-hour testing session, even though the time spent at the breast and number of sucks was unchanged. Mothers could perceive no difference in milk production or nursing behavior in their infants. Infants sucked more vigorously on a bottle containing their mothers' milk spiked with alcohol than on mothers' milk alone. In a study in which infants were weighed by the mothers before and after each feeding for the next 16 hours (20 hours total), infants increased the number of nursings during the period of 8 to 12 hours after the alcohol intake such that the total amount of milk consumed during the 20-hour period did not differ between the alcohol and non-alcohol days.

>> In studies that measured infant sleep, infants slept more frequently for shorter periods of time during the 3.5 to 4 hours after alcohol intake, whether it was after mothers drank 0.3 grams/kg of alcohol before breastfeeding or infants were given their mothers' milk spiked with an amount of alcohol (32 mg/100 mL) equivalent to that at 1 hour after maternal ingestion of 0.3 grams/kg of alcohol. After ingesting the alcohol-containing milk after maternal consumption of 0.3 grams/kg of alcohol, 14 infants from 4 to 11 weeks of age infants were observed for 1 hour after milk ingestion. Their behavioral state changed more frequently, they slept less, cried more and startled more than after consuming milk without alcohol. Mother-infant interactions were more conflictive after alcohol intake which may partially explain increased infant arousal after maternal and infant alcohol ingestion. A study that monitored the infants during the 24-hour period after maternal alcohol ingestion revealed that the infants compensated by spending more time in active (rapid eye movement) sleep from 3.5 hours to 24 hours with no further alcohol intake.

>> Long-term effects of alcohol ingestion during breastfeeding were studied in 2 separate populations by one group of investigators. In the first study, alcohol intake of more than 1 drink daily during nursing produced a measurable decrease in motor function development, but not mental development at 1 year of age. A later follow-up study found no decrements in performance of 18-month-old infants who were breastfed by mothers who consumed alcohol.

>> Studies have examined the effects of ingestion of pulque, an alcohol-containing drink made from agave cactus, in rural Mexican mothers. Most of the women had ingested pulque daily during pregnancy and lactation. One study found no effects on weight or length growth velocity among the 32 infants at 3 and 6 months of age whose mothers ingested an

average of about 30 g of alcohol daily compared to the infants of 62 infants who did not drink pulque. Another study compared the growth of 40 infants whose mothers ingested pulque during pregnancy and lactation and 18 whose mothers did not. Mothers who consumed pulque ingested an average of 16.3 g daily. The infants whose mothers ingested pulque regularly had poorer growth between 1 and 57 months and smaller size at 57 months.

>> A retrospective study of 222 inner city women reported only as an abstract found that 1-year-old breastfed infants scored higher on language skills and motor development and had fewer hearing problems than nonbreastfed infants. Alcohol use by the mothers did not decrease the beneficial effects of breastfeeding.

>> A subgroup analysis of a large cohort study in Norway found that the infants of mothers who drank alcohol during breastfeeding had no greater risk of asthma, allergy or lower respiratory infections at 36 months of age than infants of mothers who did not drink.

>> A study of low socioeconomic status women in South Africa evaluated development of their children at 7 years of age. Infants were grouped by whether their mothers drank alcohol during pregnancy and breastfeeding, breastfeeding only, or who abstained during breastfeeding, according to their mothers' recall at the time of the study. Compared to the infants whose mothers reported no drinking during breastfeeding (n = 64), those whose mothers reported drinking during breastfeeding only (n = 21) had lower verbal IQ, and were lower on growth charts.

>> Ileus with abdominal distension was reported in three Chinese infants, one 19 days and two 3 weeks of age. All laboratory tests were normal. Their mothers had been eating "chicken wine" (chicken cooked in Chinese rice wine), which is a postpartum custom in Chinese culture. Two infants had measurable alcohol in their blood. One had an alcohol level of 4.3 mmol/L (198 mg/L or 0.02%), 30 hours after admission and the other had a level of 4.3 mmol/L, 15 hours after admission. In the third infant, alcohol was not measured. The authors concluded that the ileus was caused by alcohol intoxication in the infants.

>> A prospective cohort study in Australia evaluated breastfed infants at 8 weeks and 12 months of age. Their mothers' alcohol use was tracked. Most mother's alcohol use was considered to be moderate and drinking was almost always timed to minimize the amount of alcohol in breastmilk. Infants' social, mental and motor development were examined with the Ages and Stages questionnaires. The infants of mothers who used alcohol postpartum had no greater risk of adverse outcomes up to 12 months of age than the infants of mothers who were alcohol abstainers.

>> A large, nested case-control study from a prospective cohort study in Australia compared infants who had been breastfed by mothers who drank alcohol during lactation to those whose mothers did not drink alcohol. The authors found that greater or riskier maternal alcohol intake determined by a maternal questionnaire was associated with decreased nonverbal reasoning at 6 to 7 years in a dose-dependent manner. This correlation was not found in children at 8 to 11 years of age. The frequency and quantity of milk consumed by infants and the timing of alcohol consumption in relation to breastfeeding were not known. In a follow-up study, a dose-dependent association was found between increased or riskier maternal alcohol consumption while breastfeeding and decreased academic scores in children in both grades 3 and 5. Another analysis of the data found that maternal alcohol consumption while breastfeeding was not associated with developmental health outcomes at 6 to 7 or at 10 to 11 years old.

>> A search was performed of the shared database of all U.S. poison control centers for the time period of 2001 to 2017 for calls regarding medications and breastfeeding. Of 2319 calls in which an infant was exposed to a substance via breastmilk, 7 were classified as resulting in a major adverse effect and one of these involved alcohol. A 16-day-old infant was exposed to alcohol and unspecified benzodiazepines in breastmilk. The infant was admitted to the intensive care unit for cardiac and respiratory arrest. The dosages and extent of breastfeeding were not reported and the infant survived.

>> In Australia, breastfed children with a language background other than English who performed better than native English-speaking students on a national standardized examination were found to have mothers who used less alcohol during pregnancy and had lower or less risky patterns of alcohol consumption while breastfeeding.

>> ◉ Effects on Lactation and Breastmilk

>> Studies in mothers who were 2 to 8 days postpartum found that acute doses of alcohol infused intravenously reduced the oxytocin-mediated milk ejection reflex following infant sucking. The effect could be overridden by administration of exogenous oxytocin, indicating that alcohol inhibits oxytocin release, not its effect on the breast. Alcohol doses of 0.5 to 0.99 grams/kg reduced oxytocin response to infants sucking by 18%; doses of 1 to 1.49 grams/kg reduced the response by 62%; and doses from 1.5 to 1.99 grams/kg reduced the response by 80%. Alcohol also increased the time for letdown to occur after nipple stimulation, from 29 seconds to 64 seconds with doses of 1 to 1.49 grams/kg and from 38 seconds to 331 seconds with doses of 1.5 to 1.99 grams/kg. Other investigators found that drinking 100 mL of whiskey containing a total of 50 mL of absolute alcohol (about 4 drinks in a 60 kg woman) abolished the rise in serum oxytocin in response to breast stimulation with a breast pump in 16 nonpregnant, nonlactating women. Pretreatment with naloxone blunted alcohol's inhibitory effect on oxytocin release.

>> Acute alcohol ingestion can either increase, decrease or have no effect on serum prolactin in nonpregnant, nonlactating women.

>> Drinking 100 mL of whiskey containing a total of 50 mL of absolute alcohol lessened the increase in serum prolactin in response to breast stimulation with a breast pump in 11 nonpregnant, nonlactating women. Serum prolactin rose by 71%

over baseline 20 minutes after stimulation without alcohol and only by 25% after alcohol consumption. Pretreatment with naloxone blunted alcohol's inhibitory effect, with the combination resulting in a 46% rise in serum prolactin over baseline. It is not clear how these finding apply to lactating women.

>> A study on 28 lactating women who were 2 to 5 months postpartum found that the normal rise in serum prolactin was enhanced when alcohol in a dose of 0.4 grams/kg was taken 35 minutes before breast stimulation with a breast pump. In subjects with a first-degree relative with a history of alcoholism, the increase in serum prolactin was blunted in magnitude, rapidity, and duration both with and without prior alcohol consumption.

>> Nursing mothers who ingested a 0.3 grams/kg dose of alcohol produced an average of 9.3% less milk 2 hours after the alcohol intake using a breast pump than they did when a nonalcoholic beverage was taken. The caloric content and composition of milk were not different during the two test periods.

>> A 1-year long survey of 587 new mothers in Australia found that women who drank more than 2 standard drinks (10 grams or 12.5 mL of absolute alcohol) daily were twice as likely to discontinue breastfeeding by 6 months postpartum than mothers who reported use below this amount.

>> Beer specifically has a reputation for increasing milk supply. A small crossover study found that ingestion of 1 liter of beer containing 6% alcohol by 11 nonpregnant, nonlactating women increased serum prolactin by nearly 2.5-fold 30 minutes after ingestion, but sparkling water with an equivalent amount of alcohol did not. In another study, 7 nonpregnant, nonlactating women were given 800 mL of beer. Six drank beer containing 4.5% alcohol and 1 woman drank nonalcoholic beer. Their average peak serum prolactin increased to 2.4 times the baseline value between 60 and 105 minutes after ingestion. The one woman who drank nonalcoholic beer had an equivalent prolactin response. Pretreatment with naloxone had no effect on the prolactin response. Animal studies indicate that a polysaccharide found in barley and malt is apparently responsible for the increase in prolactin after beer ingestion.

>> The interaction between alcohol ingestion and breast pumping was investigated in a double-blind crossover study of 13 lactating women who were exclusively nursing 2- to 5-month-old infants. Compared to placebo, ingestion of 0.4 grams/kg of alcohol increased serum prolactin during the ascending phase of blood alcohol concentrations. Pumping milk from the breasts during the ascending phase of blood alcohol enhanced the prolactin response, but pumping during the descending phase of blood alcohol blunted the prolactin increase. Milk production was lower after alcohol ingestion, but unrelated to serum prolactin or alcohol blood concentrations.

>> Twenty-three Taiwanese nursing mothers received a chicken-based soup following a cereal snack twice during the first 15 days postpartum. On one occasion the soup contained a dose of 0.3 grams/kg of alcohol (about 1.5 drinks for a 60 kg woman) and on the other occasion the soup was alcohol free. The time for the first drops of milk to be ejected after breast stimulation with a pump was longer (4.4 vs 2.9 seconds) after the alcohol-containing soup than with the nonalcoholic soup. In addition, the triacylglycerol (14.8 vs 12.3 mg/dL) and lactate (0.8 vs 0.6 mg/dL) content of breastmilk were greater at 135 minutes after ingesting the alcohol-containing soup than the nonalcoholic soup.

>> A study compared the prolactin response of 7 non-alcohol-dependent women with a family history of alcoholism to 21 women with no family history of alcoholism. Participants were given a dose of 0.4 grams/kg of alcohol or placebo in a crossover fashion on 2 days. A breast pump was used to collect breastmilk beginning 35 minutes after ingesting the test solution. Blood samples were collected for prolactin before and at various times after beverage consumption. The women with a family history of alcoholism had reduced serum prolactin responses to breast stimulation whether or not they had consumed alcohol. They tended to nurse their infants more frequently than the other mothers, apparently as a method of compensation.

>> Nursing mothers whose diets were supplemented with 660 mL daily of nonalcoholic beer had increases in the antioxidant capacity of their plasma and breastmilk. The coenzyme Q10 content of milk was higher at day 30 in the supplemented mothers. No change in oxidant markers was found in their infants' urine.

>> A prospective cohort study in Australia evaluated breastfed infants at 8 weeks and 12 months of age. Most mother's alcohol use was considered to be moderate at 14 or fewer drinks per week. Alcohol use did not adversely affect the duration of breastfeeding.

>> A study of first-time mothers in a Brazilian Baby Friendly hospital found that mothers who used alcohol during pregnancy had an increased risk of delayed lactogenesis II.

Exposure Routes:

>> The substance can be absorbed into the body by inhalation of its vapour and by ingestion.

>> inhalation, ingestion, skin and/or eye contact

Inhalation Exposure

>> Cough. Headache. Fatigue. Drowsiness.

Skin Exposure

>> Dry skin.

Eye Exposure

>> Redness. Pain. Burning sensation.

Ingestion Exposure

>> Burning sensation. Headache. Confusion. Dizziness. Unconsciousness.

>> irritation eyes, skin, nose; headache, drowsiness, lassitude (weakness, exhaustion), narcosis; cough; liver damage; anemia; reproductive, teratogenic effects

Target Organs:

Organs that are affected by exposure to this chemical. Information in this section reflects human data unless otherwise noted.

>> Eyes, skin, respiratory system, central nervous system, liver, blood, reproductive system

Adverse Effects:

An adverse effect is an undesired harmful effect resulting from a medical treatment or other intervention.

>> Neurotoxin - Acute solvent syndrome

>> Occupational hepatotoxin - Secondary hepatotoxins: the potential for toxic effect in the occupational setting is based on cases of poisoning by human ingestion or animal experimentation.

>> Reproductive Toxin - A chemical that is toxic to the reproductive system, including defects in the progeny and injury to male or female reproductive function. Reproductive toxicity includes developmental effects. See Guidelines for Reproductive Toxicity Risk Assessment.

>> ACGIH Carcinogen - Confirmed Animal.

Toxicity Data:

>> LC50 (rat) = 20,000 ppm/10H

Treatment:

Treatment when exposed to toxin

>> If you suspect someone has alcohol poisoning, call for an ambulance to take them to a hospital. While you're waiting: try to keep them sitting up and awake, give them water if they can drink it. If they've passed out, lie them on their side in the recovery position and check they're breathing properly, keep them warm and stay with them and monitor their symptoms. Acute alcohol poisoning is a medical emergency due to the risk of death from respiratory depression and/or inhalation of vomit if emesis occurs while the patient is unconscious and unresponsive. Emergency treatment for acute alcohol poisoning strives to stabilize the patient and maintain a patent airway and respiration, while waiting for the alcohol to metabolize. Emergency treatment in a hospital can involve: 1) treating hypoglycaemia (low blood sugar) with 50 ml of 50% dextrose solution and saline flush, as ethanol induced hypoglycaemia is unresponsive to glucagon; 2) Administration of the vitamin thiamine to prevent Wernicke-Korsakoff syndrome, which can cause a seizure; 3) application of haemodialysis if the blood concentration is dangerously high (>400 mg%), and especially if there is metabolic acidosis and 4) Providing oxygen therapy as needed via nasal cannula or non-rebreather mask.

Interactions:

>> Mass methanol poisonings present a serious problem for health systems worldwide, with poor outcome associated with delayed treatment. Positive pre-hospital serum ethanol concentration may have predictive value as the prognostic factor of the treatment outcome. We studied the effect of positive serum ethanol level on admission to hospital on survival in patients treated during the Czech methanol outbreak during 2012-2014. Cross-sectional cohort study was performed in 100 hospitalized patients with confirmed methanol poisoning. Pre-hospital ethanol was administered in 42 patients (by paramedic/medical staff to 30 patients and self-administered by 12 patients before admission); 58 patients did not receive pre-hospital ethanol. Forty-two patients had detectable serum ethanol concentration on admission to hospital [median 18.3 (IQR 6.6-32.2) mmol/cu dm]. Pre-hospital ethanol administration by paramedic/medical staff had a significant effect on survival without visual and CNS sequelae when adjusted for arterial blood pH on admission (OR 8.73; 95 % CI 3.57-21.34; $p < 0.001$). No patients receiving pre-hospital ethanol died compared with 21 not receiving ($p < 0.001$). Positive serum ethanol concentration on admission to hospital was a predictor for survival without health sequelae when adjusted for arterial blood pH (OR 8.10; 95 % CI 2.85-23.02; $p < 0.001$). The probability of visual and CNS sequelae in survivors reduced with increasing serum ethanol concentration on admission.

Antidote and Emergency Treatment:

>> Emergency and supportive measures: 1. Acute intoxication. Treatment is mainly supportive. a. Protect the airway to prevent aspiration and intubate and assist ventilation if needed. b. Give glucose and thiamine, and treat coma and seizures if they occur. Glucagon is not effective for alcohol-induced hypoglycemia. c. Correct hypothermia with gradual rewarming. d. Most patients will recover within 4-6 hours. Observe children until their blood alcohol level is below 50 mg/dL and there is no evidence of hypoglycemia. 2. Alcoholic ketoacidosis. Treat with volume replacement, thiamine, and supplemental glycose. Most patients recover rapidly. 3. Alcohol withdrawal. Treat with benzodiazepines.

Human Toxicity Excerpts:

>> /HUMAN EXPOSURE STUDIES/ Previous studies in animals have shown an increase of hydroxytyrosol (OHTyr), a potent phenolic antioxidant and a minor metabolite of dopamine (also called 3,4-dihydroxyphenylethanol or DOPET), after ethanol intake. The interaction between ethanol and dopamine metabolism is the probable mechanism involved. The aim of the study was to establish the contribution of the dose of ethanol on OHTyr formation. 24 healthy male volunteers were included. Subjects were distributed in three different cohorts and each volunteer received two doses of ethanol or placebo. Doses of ethanol administered were 6, 12, 18, 24, 30 and 42 g. Study design was double-blind, randomized, crossover and controlled. Hydroxytyrosol, tyrosol (Tyr), 3,4-dihydroxyphenylacetic acid (DOPAC), and homovanillic acid (HVA) urinary excretion, ethanol plasma concentrations and drunkenness were evaluated along a 6-hr period. Urinary excretion of OHTyr and Tyr increased with ethanol administered dose. A reduction in the ratio DOPAC/OHTyr from placebo to the highest dose was observed, compatible with a shift in the dopamine metabolism to preferently produce OHTyr instead of DOPAC. Also a dose-dependent increase in plasma ethanol concentrations and subjective effects was observed. This study demonstrates an endogenous production of OHTyr and Tyr in relation to ethanol administered dose in humans.

Non-Human Toxicity Excerpts:

>> /LABORATORY ANIMALS: Acute Exposure/ Clinical data indicate that cutaneous burn injuries covering greater than 10% of the total body surface area are associated with significant morbidity and mortality, in which pulmonary complications, including acute respiratory distress syndrome (ARDS), contribute to nearly half of all patient deaths. Approximately 50% of burn patients are intoxicated at the time of hospital admission, which increases days on ventilators by 3-fold, and doubles the length of hospitalization, compared to non-intoxicated burn patients. The most common drinking pattern in the United States is binge drinking, where an individual rapidly consumes alcoholic beverages (4 for women, 5 for men) in 2 hr. An estimated 38 million Americans binge drink, often several times per month. Experimental data demonstrate that a single binge-ethanol exposure, prior to scald injury, impairs innate and adaptive immune responses, thereby enhancing infection susceptibility and amplifying pulmonary inflammation, neutrophil infiltration, and edema, and is associated with increased mortality. Since these characteristics are similar to those observed in ARDS burn patients, our study objective was to determine whether ethanol intoxication and burn injury and the subsequent pulmonary congestion affect physiological parameters of lung function, using non-invasive and unrestrained plethysmography in a murine model system. Furthermore, to mirror young adult binge-drinking patterns, and to determine the effect of multiple ethanol exposures on pulmonary inflammation, we utilized an episodic binge-ethanol exposure regimen, where mice were exposed to ethanol for a total of 6 days (3 days ethanol, 4 days rest, 3 days ethanol) prior to burn injury. Our analyses demonstrate mice exposed to episodic binge ethanol and burn injury have higher mortality, increased pulmonary congestion and neutrophil infiltration, elevated neutrophil chemoattractants, and respiratory dysfunction, compared to burn or ethanol intoxication alone. Overall, our study identifies plethysmography as a useful tool for characterizing respiratory function in a murine burn model and for future identification of therapeutic compounds capable of restoring pulmonary functionality.

Non-Human Toxicity Values:

>> LD50 Mouse iv 2.0 g/L

National Toxicology Program Studies:

Reports from the National Toxicology Program, an interagency program supported by three government agencies (NIH, FDA, and CDC) within the Department of Health and Human Services. This program plays a critical role in generating, interpreting, and sharing toxicological information about chemicals of public health concerns.

>> Ethanol (EtOH) was evaluated as a known positive in the RACB protocol using CD-1 mice. A dose-range finding study provided data on water consumption, body weights, and clinical signs, which were used to select concns for the continuous cohabitation phase (Task 2) of 5, 10, and 15% w/v in distilled water. Water consumption was reduced at the middle and highest concns, by nearly equal to 9% and 25%, respectively. Interestingly, body weights remained unchanged during the course of Task 2. These concns, consumption, and body weight data produced calculated consumption estimates of nearly equal to 8.5, 16.0, and 20 g/kg/day. While the mean number of litters/pair was unchanged by EtOH consumption, the number of live pups/litter was reduced by nearly equal to 20% at the high dose. Viability, sex ratio, and pup body weight (absolute or adjusted for litter size) was unaffected by EtOH consumption. It was concluded that EtOH was causing no significant reproductive toxicity during Task 2, so a Task 3 crossover was not performed, and second generation effects (Task 4) were evaluated in the control and high dose groups only. The last litter from the control and 15% EtOH groups was nursed by the dam until weaning at /postnatal day/ 21, then provided with the same dosed water as their parents. While the viability of the F1 pups was unaffected by parental EtOH exposure, body weight was reduced by nearly equal to 25% at weaning. At the time of mating (nearly equal to /postnatal day/ 74), male and female body weights in the 15% EtOH group were 13% and 7% less than their respective controls. EtOH did not affect the proportion of F1 pairs mating or delivering live young, and the number and viability of those young were also unchanged. The weight of the F2 pups, adjusted for litter size, was reduced in the EtOH-exposed group by nearly equal to 7%. After the F2 pups were evaluated, all animals were killed, and the F1 parents were necropsied. For the EtOH-exposed group, male terminal body weight was 10% less than controls. Absolute testis weight was unchanged, while adjusted weights of liver and kidneys were increased in the EtOH-exposed mice by 11% and 12%,

respectively. Epididymal sperm motility was reduced from a control value of 80% motile, to 55% in the EtOH-consuming group; there were no changes in epididymal sperm density or morphologic abnormalities. Female mice consuming 15% EtOH weighed 8% less than controls at sacrifice, while adjusted liver weight and kidney weight was increased by 13% and 11%, respectively. In summary, ethanol, at concns sufficient to affect water consumption more than body weight, had only modest reproductive effects (reduced sperm motility) in Swiss mice. These effects mirror those found in literature reports.

Populations at Special Risk:

>> The distribution of the human liver alcohol dehydrogenase, ADH2, and aldehyde dehydrogenase, ALDH2, genotypes in 21 different populations comprising Mongoloids, Caucasoids, and Negroids was determined by hybridization of the amplified genomic DNA with allele-specific oligonucleotide probes. Whereas the frequency of the ADH1(2) allele was found to be relatively high in the Caucasoids, Mexican Mestizos, Brazilian Indios, Swedish Lapps, Papua New Guineans and Negroids, the frequency of the ADH2(2) gene was considerably higher in the Mongoloids and Australian Aborigines. The atypical ALDH2 gene (ALDH2(2)) was found to be extremely rare in Caucasoids, Negroids, Papua New Guineans, Australian Aborigines and Aurocanians (South Chile). In contrast, this mutant gene was found to be widely prevalent among the Mongoloids. Individuals possessing the abnormal ALDH2 gene show alcohol-related sensitivity responses (e.g. facial flushing), have the tendency not to be habitual drinkers, and apparently suffer less from alcoholism and alcohol-related liver disease.

12. Ecological Information

ICSC Environmental Data:

>> Environmental effects of the substance have been adequately investigated, but no significant effects have been found.

13. Disposal Considerations

Spillage Disposal

>> Remove all ignition sources. Ventilation. Do NOT wash away into sewer. Collect leaking and spilled liquid in covered containers as far as possible. Absorb remaining liquid in inert absorbent. Wash away remainder with plenty of water. Store and dispose of according to local regulations.

Disposal Methods

>> SRP: Recycle any unused portion of the material for its approved use or return it to the manufacturer or supplier. Ultimate disposal of the chemical must consider: the material's impact on air quality; potential migration in air, soil or water; effects on animal, aquatic and plant life; and conformance with environmental and public health regulations. If it is possible or reasonable use an alternative chemical product with less inherent propensity for occupational harm/injury/toxicity or environmental contamination.

>> Product: Burn in a chemical incinerator equipped with an afterburner and scrubber but exert extra care in igniting as this material is highly flammable. Offer surplus and non-recyclable solutions to a licensed disposal company. Contact a licensed professional waste disposal service to dispose of this material. Contaminated packaging: Dispose of as unused product.

>> SRP: Wastewater from contaminant suppression, cleaning of protective clothing/equipment, or contaminated sites should be contained and evaluated for subject chemical or decomposition product concentrations. Concentrations shall be lower than applicable environmental discharge or disposal criteria. Alternatively, pretreatment and/or discharge to a permitted wastewater treatment facility is acceptable only after review by the governing authority and assurance that "pass through" violations will not occur. Due consideration shall be given to remediation worker exposure (inhalation, dermal and ingestion) as well as fate during treatment, transfer and disposal. If it is not practicable to manage the chemical in this fashion, it must be evaluated in accordance with EPA 40 CFR Part 261, specifically Subpart B, in order to determine the appropriate local, state and federal requirements for disposal.

>> The following wastewater treatment technologies have been investigated for ethanol: Biological Treatment.

14. Transport Information

DOT

Ethanol
3
UN Pack Group: II

IATA

Ethanol
3,
UN Pack Group: II

15. Regulatory Information

Regulatory Information

The Australian Inventory of Industrial Chemicals

>> Chemical: Ethanol

REACH Registered Substance

>> Status: No longer Valid Update: 11-01-2016 https://echa.europa.eu/registration-dossier/-/registered-dossier/6310

>> Status: Active Update: 17-05-2023 https://echa.europa.eu/registration-dossier/-/registered-dossier/16105

New Zealand EPA Inventory of Chemical Status

>> Ethanol: HSNO Approval: HSR001144 Approved with controls

16. Other Information

Toxic Combustion Products:

Toxic products (e.g., gases and vapors) produced from the combustion of this chemical.

>> Special hazards arising from the substance or mixture: Carbon oxides

Other Safety Information

Chemical Assessment

>> IMAP assessments - Ethanol: Human health tier II assessment

>> IMAP assessments - Ethanol: Environment tier I assessment

"The information provided is believed to be accurate but is not comprehensive and should be used as a reference. It reflects our current knowledge and is intended for safety guidance related to the product. This document does not constitute a warranty of the product's properties. Ionz is not responsible for any damages resulting from handling or contact with the product incorrectly."