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RESULTS OF A WORKSHOP ON HEALTH EFFECTS OF CRUDE OIL EXPOSURES RELATED TO OPERATION DESERT STORM

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The experiments reported herein were conducted according to the principles set forth in the current edition of the "Guide for the Care and Use of Laboratory Animals," Institute of Laboratory Animal Resources, National Research Council.

This technical report has been reviewed by the NMRI scientific and public affairs staff and is approved for publication. It is releasable to the National Technical Information Service where it will be available to the general public, including foreign nations.

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13. ABSTRACT (Maximum 200 words)

On 14 and 15 February 1991, a workshop was held to identify the potential health effects of crude oil exposures as they might occur in the field under combat situations. The most significant findings and recommendations of the workshop were: If at all possible, stay out of refineries, petrochemical plants, terminal facilities, desalinization plants and other related facilities due to the extremely high inherent hazards presented by the chemicals that may be present. No significant acute medical problems were identified with skin contact with crude oil, nor were problems foreseen with most aspects of casualty care. Prolonged contact may, however, result in an impairment of physical performance due to the weight of soaked clothing, the odor (nauseating to some), increased heat stress from decreased sweating, and such. Troops should be warned not to use solvents such as gasoline, diesel fuel, or kerosene to remove the oil, because the resulting defatting of the skin will make them more prone to dermatitis, secondary skin infections, and increased absorption of chemical warfare (CW) agents. Exposure to the plumes from burning oil fields was judged to be at least as hazardous as similar exposures from other fossil fuel fires or forest fires, in that the primary effects would be eye and nasal irritation and smoke inhalation. The standard issue gas mask provides significant protection from the irritant effects of the smoke particles and from exposure to H_2S and SO_2. A simple sensing device for H_2S gas, available off-the-shelf, should be obtained for use in and around oil fields. In concentrations that can be achieved in the field, H_2S is lethal. If potable water becomes contaminated with crude oil, it probably remains safe to drink up to the point that personnel will reject it due to odor or taste.

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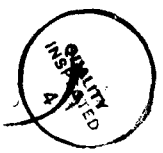

PREFACE

This document, which summarizes the findings and recommendations of the "Workshop on Health Effects of Crude Oil Exposures Related to Operation Desert Storm" held in Dayton, Ohio 14-15 February 1991, was prepared by members of the Toxic Hazards Research Unit, ManTech Environmental Technology, Inc., under Department of the Air Force Contract NO. F33615-90-C-0532 (Task No. N07). James N. McDougal, LtCol, USAF, BSC, served as Contract Technical Monitor.

This workshop was sponsored by the U. S. Navy under the direction of the Officer-in-Charge of the Toxicology Detachment, Naval Medical Research Institute, CAPT David A. Macys, MSC, USN, and was supported by the Naval Medical Research and Development Command, Task No. M0096.004.0006. Specific medical and industrial hygiene support was provided by the staff of the Navy Environmental Health Center.

The intent of this workshop was to quickly ascertain the seriousness of the potential threat posed by crude oil to the casualty care and medical support system for Operation Desert Storm, and to provide a synopsis of the relevant information to the medical personnel in the field as rapidly as possible. This publication documents the findings and conclusions of the workshop, and will be useful as a resource for future concerns regarding warfare in or near industrial facilities where those facilities can be used as weapons. To that end, the sponsoring activity will update this document as new information becomes available, such information is actively solicited.

The findings of this workshop, where consensus existed, represent the collective experience of the participants. Where experiences differed, we have attempted to circumscribe the differences. The conclusions and recommendations expressed herein are those of the staffs of the sponsoring Navy organizations. As such, these recommendations may differ from those of the workshop's civilian participants. They are based on the information provided and the recommendations made by the participants at the workshop, but any errors of interpretation, omission, or otherwise are the responsibility of the sponsor's staffs. They are not to be construed as official or reflecting the views of the Navy Department or the Naval Services at large.

Appendix E, Summary of Toxicological Data Pertaining to Petroleum and Crude Oil Constituents, was prepared by John F. Risher, CAPT, USNR-R.

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SECTION I

EXECUTIVE SUMMARY

1 The most significant findings and recommendations of the workshop include the following:

1.1 If at all possible, stay out of refineries, petrochemical plants, terminal facilities, desalinization plants and other related facilities due to the extremely high inherent hazards presented by the chemicals that may be present. The greatest concern is for spherically shaped pressure vessels which could explode violently, and for several specialty chemicals whose containers could be easily ruptured or detonated. Smoke from fires at these facilities should also be avoided if at all possible due to the toxicity of the chemicals and their combustion products.

1.2 No significant acute medical problems were identified with skin contact with crude oil, nor were problems foreseen with most aspects of casualty care. Prolonged contact may, however, result in an impairment of physical performance due to the weight of soaked clothing, the odor (nauseating to some), increased heat stress from decreased sweating, and such.

1.3 Troops should be cautioned to remove the excess oil from their skin and clothing when possible, but that residual oil appears to pose no significant problems. Troops should be warned not to use solvents such as gasoline, diesel fuel, or kerosene to remove the oil, because the resulting defatting of the skin will make them more prone to dermatitis, secondary skin infections, and increased absorption of chemical warfare (CW) agents.

1.4 Exposure to the smoke plumes from burning oil fields was judged to be at least as hazardous as similar exposures from other fossil fuel fires or forest fires, in that the primary effects would be eye and nasal irritation, and smoke inhalation. Because most oil in the theater is contaminated with H_2S, the burning oil will release SO_2 in concentrations that could be quite high. Ground-hugging plumes should be avoided, especially if the smoke is gray or white. The standard issue gas mask provides significant protection from the irritant effects of the smoke particles and from exposure to H_2S and SO_2 (see Appendix E for further information regarding gas masks).

1.5 A simple sensing device for H_2S gas, available off-the-shelf, should be obtained for use in and around oil fields. In concentrations that can be achieved in the field, H_2S is lethal. The sensor could be attached to the existing CW agent detectors, or used separately.

1.6 If potable water becomes contaminated with crude oil, it probably remains safe to drink up to the point that personnel will reject it due to odor or taste. Boiling or aerating are simple and effective means of removing the contaminants.

SECTION II

SUMMARY OF FINDINGS AND RECOMMENDATIONS

1 INTRODUCTION

1.1 This document summarizes the findings and recommendations of a workshop on the acute health effects which might occur in troops exposed to crude oil during the course of Operation Desert Storm. Participants in the workshop were personnel from oil companies with working knowledge of (a) conditions in oil fields in Saudi Arabia, Kuwait and/or Iraq, or (b) the toxicity of crude oil. Also present and contributing were personnel from the Navy and Air Force with some familiarity with the operational questions of concern. This workshop was deemed necessary to validate and expand on preliminary responses given to these questions by the staffs of the Naval Medical Research Institute's Toxicology Detachment (NMRI TOX DET) and the Navy Environmental Health Center (NEHC), because none of these personnel had any relevant direct experience. ManTech Environmental Technology, Inc., the contractor operating the Toxic Hazards Research Unit (THRU) for the Air Force and Navy at Wright-Patterson Air Force Base (WPAFB), coordinated the workshop. This included both recording and transcribing the proceedings. The transcript was used to prepare this document that addresses all aspects of potential health effects discussed during the meetings, as well as other factors the participants deemed to be of concern to operations in this situation.

1.2 A list of participants from the private sector is attached as Appendix B. Although not representing their organizations formally, the participants have access to the experience embodied in those organizations and were willing to share it freely. Attendees from military organizations (including the contractor) are also listed in Appendix B. These personnel were invaluable in focusing on the specific operationally related concerns regarding the prevention and treatment of casualties, and pragmatic concerns for operating in the desert environment. Appendix B lists sources of expertise and points of contact on specific matters.

1.3 For the purposes of discussion, three exposure scenarios were described to the participants in general terms: (1) an amphibious assault that must cross oil-slick-covered water and/or oil-soaked sand beaches; (2) a land attack that might have to contend with oil-filled trenches as part of fixed defensive fortifications; and (3) a land attack through or past oil fields. This document will focus on the common elements of exposure, effect, and recommendation. The primary concern was to identify what addition these exposures might make to the disease and non-battle injury (DNBI) load on the casualty care and medical treatment capabilities supporting the operation, and whether treatment of battle wounds/injuries might be complicated. To simplify the discussion, complications introduced by operations near or in refineries and other facilities that can be treated as "point sources," and by the introduction of CW agents, are dealt with as add-ons to the primary discussion.

They are likely to be inherently not only the most hazardous, but also the most limited in scope. Only the most significant of those hazards will be mentioned here; the bulk of them will be discussed in Section III.

1.4 The findings of this workshop, where consensus existed, represent the collective opinions of the individuals invited to participate. Where consensus on an issue was less evident, we have attempted to circumscribe the differences in opinion. The conclusions and recommendations expressed herein are those of the staffs of the sponsoring activities, the NMRI TOX DET and the NEHC. As such, these recommendations may differ from those of the workshop's civilian participants. They are based on the information provided and the recommendations made by the participants at the workshop, but any errors of interpretation or omission are the responsibility of the sponsors' staffs.

2 FINDINGS

2.1 AGENTS OF CONCERN AND EFFECTS

2.1.1 Crude Oil. For these purposes, crude oil will be separately examined both as the oil itself and its volatile components. The volatiles, in turn, will be separated into the volatile hydrocarbons and H_2S. This distinction is of greatest importance when the crude is relatively fresh; a light crude will lose up to 10 to 15% of its volume immediately, and up to 25% of its volume within 24 h, due to evaporation of volatiles. This process is highly dependent on the surface-to-volume ratio of the bulk oil, and any actions that tend to disperse the oil (e.g., well blowout); thus, crude in a pool or tank will retain more of its volatile components than crude in an oil slick.

2.1.1.1 With a few exceptions, whole crude oil does not pose an acute hazard due to skin contact. Some personnel may be, or may become, sensitive to the crude, and it may be mildly to moderately irritating, particularly in contact with the eyes. Ingestion of crude, unless in massive quantities (e.g., > eight ounces) is unlikely to result in more than transient nausea, possibly vomiting, gastrointestinal tract disturbances, and self-limiting diarrhea. Inhalation exposures occur with the volatile components only and are dealt with below.

2.1.1.2 Although skin irritation is a possibility as a result of skin contact with crude oil, experience is very limited with contact that is prolonged over several days, as may be the case in some combat situations. An additional complication is the abrasion possible with the addition of sand to the crude oil on skin and in clothing. The possibility that severe abrasion may occur in areas such as the armpits, groin, and feet is significant. Folliculitis and other problems due to the occlusion of the skin by oil can be expected to be common among those personnel who experience prolonged skin contact.

2.1.1.3 Prolonged contact with crude oil, either on the skin or with contaminated clothing, may result in an impairment of performance. This may be due to the extra weight of contaminated clothing and other materials; to the constant odor of contaminated skin or clothing, which some may find nauseating; to the decrease in evaporative heat loss as a result of decreased sweating if the skin is covered with an oil layer or if clothing cannot breathe due to oil saturation; or to other factors not considered here.

2.1.2 **Volatile Hydrocarbon Compounds (VHCs).** The presence of VHCs increases the hazard presented by crude oil as mentioned above. Because the degree to which they may be present cannot be well predicted, they should be assumed to be present unless otherwise indicated.

2.1.2.1 Volatile Hydrocarbon Componds increase the irritancy of crude to the skin and eyes. Skin contact will result in defatting of the skin, increasing the potential for dermatitis and secondary skin infections.

2.1.2.2 The presence of VHCs may complicate ingestion because they can be aspirated if the crude is vomited. Induction of vomiting is not necessary due to the low toxicity of crude oil, and is contra-indicated for crudes containing a significant concentration of VHCs because of the danger of possible aspiration of those materials.

2.1.2.3 Volatile Hydrocarbon Compounds pose a significant inhalation hazard. In very high concentrations they can be life threatening due to central nervous system (CNS) depression. Irritation of the eyes, nose and throat, dizziness, nausea, lack of coordination, drowsiness, tremors, and unconsciousness are the progressive signs of high concentrations. It should be noted that these concentrations also pose a flammability and explosion hazard.

2.1.3 **Hydrogen Sulfide (H_2S).** This volatile gaseous compound is evolved from sour crudes that are present in most if not all oil fields in the area of concern. This gas is of particular concern because deaths have occurred in the Kuwaiti oil fields due to H_2S exposures.

2.1.3.1 Hydrogen sulfide is a colorless gas which is heavier than air. It tends to collect in "pockets" or form clouds near the surface before slowly dissipating. The characteristic odor is that of rotten eggs. The sense of smell quickly fatigues, however, and odor cannot be relied upon for adequate warning of either the presence of H_2S or relative concentration. The standard issue gas mask provides significant protection from exposure to H_2S and SO_2 (see Appendix E for further information regarding gas masks).

2.1.3.2 With or without the odor being detected, effects from exposures up to about 50 ppm range from mucus membrane irritation, to nausea, to drowsiness. Delayed pulmonary edema can result from relatively low exposures; exercise potentiates this effect. Sudden unconsciousness

without any warning symptoms has been reported in personnel entering an area of high concentration. Concentrations of 250 ppm or greater can be lethal; such concentrations can easily be achieved around well heads.

2.1.4 Combustion Products. Burning crude oil has been used as a threat; the magnitude of this threat, insofar as the smoke poses an inhalation hazard, is at least as great and possibly greater than the hazard posed by a forest fire or most other fossil fuel fires due to the components of the complex mixture which is crude oil.

2.1.4.1 The particles that compose the smoke itself are likely to be acutely irritating to the eyes and respiratory tract, but the standard issue gas mask provides significant protection. It will also offer some protection from higher molecular weight hydrocarbons which may volatilize due to the heat.

2.1.4.2 Of greater concern is the presence of uncombusted H_2S gas, SO_2, and other toxic and irritant gases. The VHCs will mostly be consumed in the fire to form soot and some irritant aldehyde gases; H_2S and other sulfur compounds will be oxidized to SO_2 gas and a sulfuric acid aerosol; and the nitrogen in the air will be oxidized to NO_X. The standard issue gas mask provides significant protection against H_2S and SO_2 (see Appendix E for further information regarding gas masks).

2.1.4.3 In general, the concentrations of hazardous combustion products will be greatest at locations close to the combustion source; concentrations will remain high in the smoke plume emanating from the source, with dilution due to mixing with the atmosphere occuring with increasing distance and increasing winds. Particular concern should be paid to ground-hugging plumes. It should be noted that certain weather conditions can magnify this problem; this is discussed in paragraph 2.4.3.

2.2 CASUALTY CARE ISSUES

2.2.1 Skin Contamination. Generally, the presence of crude oil on the patient should present no major complications; it can be wiped off whenever convenient in the treatment process. Two caveats should be noted, however.

2.2.1.1 Oil and oxygen-enriched atmospheres are potentially explosive. The level of contamination of hands oily from removing oily clothing from patients can be sufficient to contaminate fittings on O_2-using treatment equipment, with potentially disastrous consequences. Oil on the face of a patient does not contraindicate O_2 treatment, but it is important to remove oil-contaminated clothing and wipe the oil from the head, neck, and chest of the pateint before placing the O_2 mask. Because an oxygen-enriched atmosphere may exist for up to one foot from a nasal cannula or face mask, sources of ignition should not be used in proximity to oil-contaminated personnel or clothing. In particular, before using a defibrillator, excess oil should be wiped off of the

patient, particularly from the trunk and neck areas. Also see the Note in paragraph 2.2.2, and consult Appendix C for details.

2.2.1.2 Oil contaminated clothing removed from patients, and oily cloths or rags used to wipe off patients, represent a potential fire hazard due to spontaneous combustion. Such material should not be left lying around where they could cause additional problems, but rather they should be stored in a metal container with a tight lid. The contaminated clothing and rags should not be left to accumulate but should be disposed of frequently.

2.2.2 **Wound Contamination.** Concern with oil in wounds and the methods for debridement were raised by staff aboard the USS Comfort. Current industrial practice for external and superficial wound cleaning is being modified to include use of waterless hand cleaners (e.g., GOJO®, Goop®), white petrolatum, mineral oil, corn oil, and neosporin ointment. These agents must be removed as completely as possible from within the wound after efforts to remove the crude oil have been completed.

> (**NOTE:** The precautions mentioned above for oxygen therapy also apply to any oil-based cleaning agent mentioned here, such as the waterless hand cleaners – they contain petroleum distillates.)

Puri-clens® wound deodorizer and cleanser, and Betadine® solution were the only cleaners identified during this assessment that are approved by the USFDA for direct cleaning within wounds. Body cavities can be irrigated with normal saline or a normal saline-Betadine solution mixture. All surfactants evaluated were unsuitable for wound decontamination due to toxicity of ingredients.

2.3 POTABLE WATER CONTAMINATION

2.3.1 The potential for crude oil getting into ships' potable water sources had been previously identified as a problem of concern. Participants' experience, although limited, indicated that it should not pose a significant problem because simple treatment was possible. Moreover, acute toxic effects generally do not occur until contamination levels significantly exceed objectionable taste and odor levels.

2.3.2 Sources of water in recaptured facilities should not be assumed to be potable. Clear liquids may not be water, or the water may have anticorrosion or other additives in it. Finally, potable water sources in refineries and other fixed sites are easy to contaminate; this could be an attractive act of sabotage due to its simplicity.

2.4 OTHER

2.4.1 **Refineries and Other Facilities.** Treating and refining crude oil and developing various chemical products from crude make use of many different chemicals, most of them flammable, toxic,

or both. The refining and storage of some of these chemicals takes place under pressurized conditions, and some of the storage vessels are maintained at high pressure. There are a multitude of health, fire, and explosion hazards that can very easily be triggered by the unwary or by simple sabotage. Participants were unanimous in their concern that operations in and around these facilities could be extremely dangerous.

2.4.1.1 Many of the Kuwaiti personnel who operated the facilities are now in Saudi Arabia. They should be available to be contacted and should have knowledge of the specific chemicals used and stored in each facility. Particular concern was expressed about the presence of hydrogen fluoride (HF) and the organo-lead compounds tetramethyl and tetraethyl lead (TML, TEL), all of which are highly toxic. Hydrogen fluoride is also extremely corrosive; the TML and TEL are explosive.

2.4.1.2 Spherical storage vessels are under considerable pressure and contain liquified volatiles which are flammable and explosive. Fire, blast, and shrapnel effects could be expected to a distance of at least one thousand meters. Retinal burns are possible if personnel were looking at the fireball.

2.4.1.3 Large storage tanks generally have a sufficiently concentrated layer of volatiles over the surface of the liquid to be easily ignitable. After burning for a number of hours, the flaming oil may erupt out of the tank as a result of a heat wave contacting and instantly boiling the water in the tank bottom. This poses a threat to personnel within several hundred meters of the tank.

2.4.1.4 Adjacent to many refineries may be large yellow or yellow-brown mounds. These are piles of elemental sulfur, a by-product of the refining process. Dust formation should be minimal (fortunately, because the dust is explosive); however, it is a flammable solid, producing clouds of SO_2, an upper respiratory tract and mucus membrane irritant, which in high concentrations can induce respiratory paralysis.

2.4.2 **Chemical Warfare (CW) Agent Considerations.** The effect that having a layer of oil on the skin may have on the absorption of CW agents is unknown. Concern was high due to the use of petroleum distillates in commercially available insecticides to accelerate the absorption of the agent through the skin.. Additional concern was focused on the impact of an oil layer on the effectiveness of decontamination solutions. The use of solvents such as gasoline, diesel fuel, and kerosene to remove the oil was considered undesirable because they defat the skin, rendering it more permeable to various agents.

2.4.3 **Weather.** Although winds in the area are common, inversions and coastal fogs are known to occur. These conditions can significantly enhance the hazards presented by irritant and toxic smoke and gas plumes from fires and explosions (see section 2.1.4 and paragraph 2.4.1), particularly if they result in the plumes remaining on the ground, rather than rising and dissipating. The history of air pollution incidents in Europe and the USA indicates that such weather conditions

can concentrate pollutants which would otherwise dissipate, creating significant respiratory and eye irritation problems for even healthy young people.

3 RECOMMENDATIONS

(NOTE: Not all of these recommendations may be practical under every circumstance; they are made with the expectation that even the impractical may inspire a useful alternative.)

3.1 CASUALTY PREVENTION MEASURES

3.1.1 Reemphasize personal hygiene requirements, including removing excess oil from skin and clothing when possible. Pay particular attention to friction points such as the armpits, groin, and feet. A change of clothing would be ideal; dry socks would probably be the single most useful change.

3.1.2 Do not use solvents such as gasoline, diesel fuel, or kerosene to remove oil from the skin. Note that the solvents and the oil are flammable – NO SMOKING!

3.1.3 Obtain disposable goggles for eye protection of Marines riding ashore through oil-covered water to avoid eye irritation from spray. Using the standard issue gas mask without cartridge is an acceptable substitute for protection, but the contaminated spray is likely to be difficult to clean off the lenses.

3.1.4 Pay close attention to wind direction, the intent being to stay upwind (whenever possible) of potential sources of toxic and irritant materials (e.g., plumes of smoke from burning oil wells, venting oil wells, clouds from refineries). Should a shift of wind occur causing a ground-hugging plume to advance toward troops, move troops in a direction perpendicular to the advancing plume front, when possible.

3.1.5 Use the standard issue gas mask for protection from irritant smoke from burning oil (see Appendix E for further information regarding gas masks).

3.1.6 Obtain H_2S sensors for use near oil fields and refineries. The odor provides inadequate warning. An alarm setting of 15 ppm would provide plenty of warning; 100 ppm should be the maximum setting, but would provide little advance warning and little margin for error.

3.1.7 Make gas masks available for use in rescuing personnel overcome by H_2S. Attempting a rescue without one will result in more casualties.

3.2 CASUALTY TREATMENT MEASURES/CONCERNS

3.2.1 Do not induce vomiting if crude oil is ingested. If vomiting occurs, watch for aspiration pneumonia. This is of particular concern if the crude has not weathered, or if a refined product was ingested. A transient diarrhea will probably occur.

3.2.2 Clean wounds externally with a waterless hand cleaner, such as GOJO®, if wounds are contaminated by crude oil. Remove all such cleaners from within the wound prior to dressing. The wound itself may be gently cleaned with gauze pads saturated with Puri- Clens® or Betadine® solution. Deep internal wounds and body cavities can be irrigated with normal saline or normal saline-Betadine solution mixture which poses no unusual wound treatment issues beyond those normally encountered.

3.2.3 There is no immediate need to remove oil from the skin. When oil is removed, dry wiping and/or soap and water, a waterless hand cream, white petrolatum, neosporin ointment, or similar emollient should be used rather than a solvent.

3.2.4 Remove oil-contaminated clothing and wipe off the head, neck and chest before placing the oxygen mask on if casualties requiring O_2 are oil-contaminated. Before handling O_2 fittings or bottles, wipe off any oil from the hands, ensuring that they are oil-free.

3.2.5 Remove from the body oil contamination before initiating any medical procedure that could produce a spark (e.g., defibrillation).

3.2.6 Ensure that all accumulated oil-contaminated clothing or wipes are stored in tightly-lidded metal drums or well away from any combustibles. Empty containers frequently and dispose of the contaminated materials.

3.2.7 Anticipate an increase in the number of blisters and skin abrasions/irritations among personnel whose boots or clothing become contaminated due to the penetrating and abrasive nature of the mixture of oil and sand. The introduction of new boots may exacerbate this if the boots are not broken in quickly.

3.2.8 Be aware that if vehicle and other hard surfaces become contaminated with oil, an increase in injuries resulting from slips, trips, and falls may be anticipated.

3.3 POTABLE WATER

3.3.1 Consider extending ship's water intake by hose to 20 feet below the hull when in oil-contaminated waters if feasible, to minimize amount of oil drawn into the intake.

3.3.2 Water remains potable, if not palatable, when contaminated by crude oil. Toxic levels are not generally reached until after taste and odor become intolerable.

3.3.3 Decontaminating unpalatable water requires boiling to evaporate the volatiles, probably for 5 to 15 minutes. An alternate method is to aerate the water to evaporate the volatiles. This could involve pouring from one vessel to another repeatedly, or bubbling air through the water.

3.3.4 Use contaminated water for showering but not for cooking.

3.3.5 Consider water sources ashore at any recaptured facility to be non-potable (and possibly sabotaged).

3.4. OTHER

3.4.1 Due to the ease with which well heads can be wired for sabotage, and the fact that both VHCs and H_2S released from open well heads can form colorless, surface-hugging clouds at concentrations which are both lethal and explosive, participants recommended the following pragmatic approach: if a well can be seen or heard (a whistling sound) to be venting, use a remote incendiary device (e.g., white phosphorus grenade, mine-clearing device) from upwind to ignite it; in any case, prior to entering an oil well field, an incendiary should be fired in ahead of the advance from upwind, and repeated from time to time during the advance through the field. It should be noted that this will not necessarily dissipate lower but still lethal concentrations of H_2S, for which a detector is the only reliable means of identification.

4 ADDITIONAL ISSUES

4.1 REFINERIES

4.1.1 If operating in or around fixed facilities, approach from and remain upwind whenever possible, and avoid low-lying areas and confined or constricted spaces including bermed tank farms or other bermed areas. The volatiles from the crude, and the refined products, will tend to collect in such areas. Concentrations can be high enough to produce serious CNS effects, respiratory depression, and asphyxia. These concentrations are also flammable and explosive.

4.1.2 In addition to avoiding spheres and large storage tanks, small white-painted tanks should be avoided. These likely contain organo-lead compounds. These are neurotoxicants, but more importantly in this context, these are explosive and can detonate with disastrous results.

4.1.3 All refineries utilize cooling water; cooling this water for reuse requires large cooling towers. The refineries in the area use wooden structures which can be 4-5 stories tall. Concern here is for the chemicals used to prevent corrosion in the piping, which are usually chromate salts. If the cooling tower is ignited, clouds of chromate salt-bearing smoke will be released, which will be even more irritating than those discussed previously.

4.2 MISCELLANEOUS

4.2.1 The specific gravity of crude oil is below 1.0 -- it floats on water. What is the significance? You can't swim in it, you'll sink. This may be of concern if pools or trenches of oil are encountered and someone decides that going through is faster than going around.

4.2.2 Oil-saturated sand acts like quicksand. Traction becomes negligible and vehicles both wheeled and tracked become mired in it.

4.2.3 Crude oil is a lubricant which can penetrate into machinery and produce undesired effects, particularly in contact with plastic or rubber seals, gaskets, etc, where it can act as a solvent. When combined with sand, it makes a penetrating abrasive.

SECTION III

REFERENCE DOCUMENT

1 PROBLEM

1.1 Navy and Marine personnel may be exposed to large pools of crude oil and other environments associated with the production and processing of crude oil. Operational imperatives may require that operations continue in these environments to the extent possible. The purpose of this workshop was to define hazards, and toxic effects of hazardous materials associated with crude oil, and crude oil storage and processing, that might be encountered by military personnel during the conduct of military operations. Hazards to be addressed included, but were not limited to, toxic chemicals, physical hazards, and fire and explosion hazards that might result in injuries or casualties. The assessment of hazards was to emphasize hazards that might hamper or prevent military operations or result in casualties, to describe expected toxic effects (particularly acute effects), and to describe unusual or subtle hazards or toxic effects. Products of this workshop were to be a description of crude oil and crude oil product hazards, a description of adverse health effects associated with exposure to the hazards, and recommendations for preventing or alleviating the adverse health effects.

2 *OIL; OIL CHARACTERIZATION, PRODUCTION, PROCESSING, AND HANDLING HAZARDS IN THE PERSIAN GULF REGION*

2.1 The workshop primarily focused on hazards that might be associated with military operations within or near Kuwait. In order to facilitate the association of hazards with various operating conditions, the document focuses initially on identifying hazards in different oil industry environments and within sites of unintentional or deliberate environmental contamination.

2.2 Figure 1 demonstrates the distribution of the major oil formations within Kuwait, the Arabian (Persian) Gulf, and nearby areas of Saudia Arabia, Iraq, and Iran. Although both gas and oil fields are depicted, they are primarily oil fields. The predominant field in southern Kuwait is the Burgan Field, 15 miles south of Kuwait City. This field produces intermediate crude, API gravity from the high teens to the low 30's, and has a subsurface pressure of 3200 psi. The gas composition of the field is 70% methane, 14% ethane, 6% propane, 3% butane, and impurities to include approximately 0.1% (1000 ppm) hydrogen sulfide (H_2S) gas at wellheads. Based on the H_2S concentration, the field is considered "sour." Oil wells in the Burgan Field are approximately one mile apart and produce approximately 10,000 to 20,000 barrels per day. Oil wells do not have derricks to identify their location. Most wells are covered by sand, but have a flag that mark their location. The Burgan Field is considered very prolific; however, most wells are believed to be shut down at this time. Very few gas wells are within the Burgan Field. The Wafra Field to the south of the Burgan Field has oil wells that

are approximately 2000 feet apart. These wells have a production rate of several hundred barrels per day. The field should be considered sour. Most wells in the Wafra Field have been ignited. Several fields near the Burgan Field (Umm Gudair, Fuwaris, Minagish, Magwa, and Ahmadi) contain oil that is similar to that produced by the Burgan Field and are also considered sour. Hazards within well fields, and particularly at well heads, include crude oil, flammable volatile hydrocarbon gases, and hydrogen sulfide gas, which may be concentrated in low-lying areas. Hydrogen sulfide gas has the smell of rotten eggs, but high-concentration exposures may inhibit the sense of smell.

2.3 Crude oil is usually pumped from several wells through a series of pipelines to gathering facilities (tank batteries) to include gas – oil separating plants (GOSPs) in some oil fields. The pipelines are usually buried at a very shallow depth beneath oil-covered roadways that meander through the oil fields. GOSPs, which are often rather large facilities within the oil fields, contain storage tanks for crude oil and gas separating facilities. The basic field separation of crude oil in GOSPs include collection and storage of volatile gases (e.g., methane, ethane, propane, butane) as liquid petroleum gases (LPGs) in pressurized spheres, or the gases are pumped to gas plants; gravity separation of water, which may be reinjected or drained to a collection point; and pumping of the separated oil to storage tanks. The shape of some structures provides a clue of the types of materials that may be contained within. Metal spheres are usually associated with storage of LPGs under pressure. Storage tanks with floating roofs are used to store volatile liquid fuels such as crude oils, kerosene, diesel, and gasoline at essentially atmospheric pressure. Insulated dome-roofed tanks may contain liquefied natural gas (LNG) which is a cryogenic liquid. Horizontal cylindrical tanks with spherical ends also may contain LPGs. All pressure vessels are of concern.

2.4 Hazards at gathering points include crude oil exposure, exposure to LPGs, hydrocarbon vapors, and hydrogen sulfide gas. As will be emphasized later, LPG storage spheres and domed LNG storage tanks are significant explosion and fire hazards that may be the source of heat and toxic combustion products. Active open well heads from wells with high subsurface pressures may also pose an injurious noise hazard.

2.5 Crude oil or field-separated oil may also be pumped to refineries for further processing or be pumped to offshore tanker loading facilities. Due to their abundance, the primary hazards in refineries are crude oil, LNGs in domed storage tanks and LPGs in pressurized spheres, hydrocarbon vapors, and hydrogen sulfide gas (particularly in hydrogen sulfide recovery and processing units). Refineries, due to their structure and operations, pose several additional hazards including harmful process chemicals and the potential for injuries due to collapsing structures. Asbestos, an irritant and carcinogen following chronic exposure, may have been used in some refineries for insulation of pipes and other fire resistance applications such as fireproofing cements on structural steel. More acute hazards are associated with process chemicals, which may or may not be present due to the type of

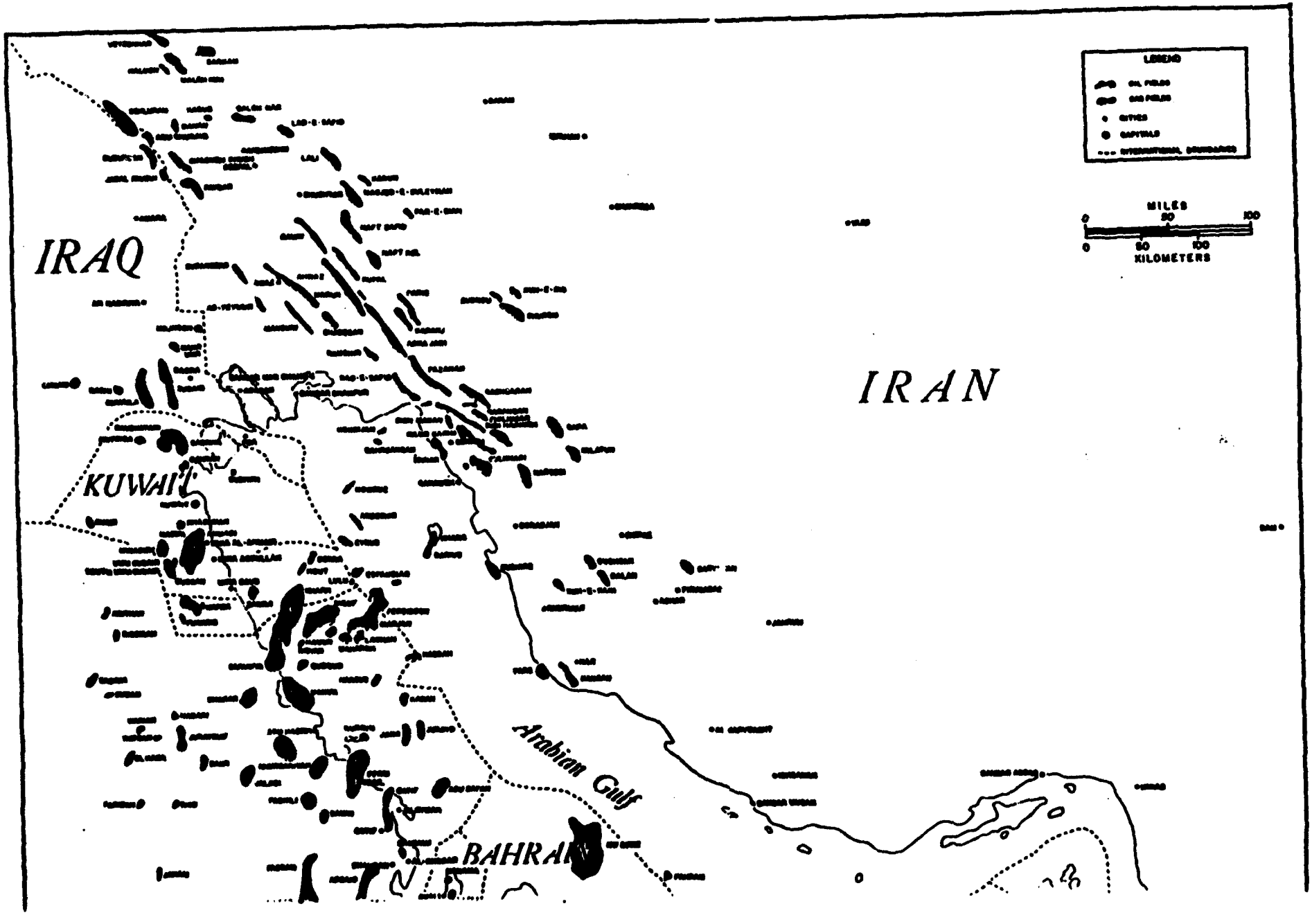


Figure 1. Oil and Gas Fields in Kuwait and Neighboring Persian Gulf Countries. The fields depicted are primarily oil fields.

technology used in the refining process. Crude oil dewaxing operations would require anhydrous ammonia, an irritant gas, for the operation of chillers. The operation of alkylation units would involve use of concentrated sulfuric acid or hydrofluoric acid. Other acid or solid catalysts are employed in the various refinery process units. Tetraethyl lead or tetramethyl lead may be found in refineries that produce leaded gasolines. Water treatment facilites within refineries use chlorine gas, a respiratory irritant. The use of hydrogen fluoride alkylation units may not be widespread in Kuwait.

2.6 Offshore terminals are the primary facilities for loading crude oil and petroleum products for export. An unsophisticated loading facility might be a flexible hose tied to a buoy. Crude oil and liquefied products may be fire hazards. Petroleum gases under pressure would be both fire and explosion hazards.

2.7 Crude oil or its separated or refined components could also occur as environmental contaminants either through accidental releases of materials as a result of combat operations or as part of deliberate obstacles. The hazards could include oil or liquid fuel contaminated surface water, beaches, and other sands and roadways. Military personnel operating in these environments may be subject to direct skin exposure and skin exposure after permeation through contaminated clothing and footgear. Volatile components may be inhaled. During marine or amphibious operations in contaminated waters there is also a possibility for accidental oral ingestion to occur. Pools of crude oil or crude oil liquid products used as deliberate obstacles may also be a source for accidental ingestion exposure. Food consumption in contaminated areas may also contribute to oral ingestion. As in most environments containing crude oil or crude oil products, there is also high potential for a concurrent fire and smoke hazard. These hazards and other associated hazards are further detailed in Section 3.5. A recommendation of several key workshop participants was that military planners contact the Oil Ministry of the Kuwaiti government in exile or the Oil Ministry of the Kingdom of Saudi Arabia to obtain more specific technical information about oil production, gathering, refining, and loading facilities within intended areas of operation. A second recommendation was to use commercially available PC-based computer software to estimate fire, explosion, and toxic gas dispersion hazards; and distances between oil producing or refinery equipment and/or facilities.

2.8 Complicating environmental factors contributing to the hazard potential of crude oil, crude products, or oil processing and storage facilities would be temperature, wind, fog, sandstorms, and terrain features. Warm temperatures may approach the flash point and increase the explosion potential of gases. Dependent on where one is positioned, wind may be helpful for the dispersion of noxious gases. However, a downwind position is disadvantageous. The absence of wind currents may prolong exposure to heavy gases such as hydrogen sulfide. Fog may lead to increased formation and persistence of toxic aerosolized products and inhalable particulates during fires. Terrain features

such as depressions or low lying terrain features may be the sites for collection and concentration of toxic heavy gases like hydrogen sulfide.

2.9 Although the workshop participants were charged to consider the conduct of necessary operations in or near environments containing crude oil or crude oil products, the consensus of the participants was that facilities associated with the production, collection, refining, and handling of crude oil and its associated products, as well as contaminated areas, posed a multiplicity of health hazards to personnel. The collective opinion of the participants was that if the choice could be made, the best choice would be to avoid operations in these threatening environments. The detailed information that follows in Section 3 provides the basis for this overall recommendation and provides specific information regarding health risks and the management of health risks and toxic health effects associated with crude oil or crude oil product hazards that might be encountered in the Persian Gulf Region. Although exact exposure scenarios were unknown, the workshop participants considered a wide variety of reality-based situations to define crude oil and crude oil product hazards, their harmful health effects, and methods of alleviating their harmful effects.

3 SPECIFIC HAZARDS, EFFECTS, PREVENTION, AND ALLEVIATION

3.1 CRUDE OIL HAZARDS

3.1.1 There are several settings that may result in the exposure of military personnel to crude oil. The possibilities include, but are not limited to: (1) oil fields at and around well heads; (2) ruptured pipelines and storage tanks; (3) crude oil-contaminated bodies of water, beaches, sands and roadways; and (4) pooled crude oil within containment berms around storage tanks or within deliberate obstacles placed in operational terrain. Personnel exposures could be whole body as might happen when one is in a contaminated body of water or in a pool of crude oil. In the event of a whole body immersion, routes of exposure may be dermal, ocular, oral (to include aspiration), and inhalation. Exposures to crude oil such as in the spray from well heads, ruptured tanks and pipes, and contaminated waters could result in dermal and ocular exposures. The possibility also exists that aerosolized crude oil in these sprays, as well as gases and vapors evolved from crude oil, could be inhaled. The toxic effects described hereafter are focused on the potential routes of exposure. Acute toxic effects that would result in immediate casualties or hamper military operations are emphasized.

3.1.2 Physical Effects. Because of its lubricity, crude oil may result in falls and associated physical injuries to limbs or other portions of the body. Physical injuries could also result from the abrasive effects of sand that may become intermixed with crude oil that is in contact with the skin. In physically active personnel, interactions of crude oil with skin in high-friction areas may lead to irritation of the skin and blister formation (soles of feet and groin) thereby hampering mobility.

3.1.3 Dermal Exposure. Dermal exposure can result in the clogging of pores and hair follicles thereby compromising body heat loss. Of less immediate consequence, dermal exposure can result in acne and folliculitis. The defatting action of lighter crude oil components can result in acute redness and itching or dermatitis.

3.1.3.1 To the extent practical, remove as much of the crude oil from the skin as possible. Sorbent materials should be used if water and soap or non-petroleum solvents are not available. Oil-soiled socks and clothing should be changed at the earliest opportunity.

3.1.4 Ocular Exposure. Ocular exposure can result in slight stinging and a transient redness. No permanent damage should result. The immediate treatment is to flush the eye with water.

3.1.5 Ingestion Exposure. The effects following ingestion of a small amount of crude oil are not expected to result in clinical signs of toxicity other than mild gastrointestinal disturbances. The primary debilitating effects of an ingestion exposure are associated with aspiration of crude oil into the respiratory tract. The low boiling point components of crude oil are primarily responsible for the chemical pneumonitis and pulmonary edema associated with crude oil aspiration.

3.1.5.1 To treat individuals exposed via ingestion, ***do not*** induce vomiting as this may lead to aspiration of the crude oil into the lung. Trained medical personnel may elect to remove ingested crude oil via gastric lavage. Trained medical personnel should provide symptomatic treatment.

3.1.6 Inhalation Exposure. The gas composition of crude oil largely determines the effects of inhalation exposure. Hydrocarbon vapors evolved from crude oil may cause narcosis, and high environmental concentrations of hydrogen sulfide evolved from sour crude oils may result in asphyxiation. Details on the effects and treatment of hydrocarbon vapor and hydrogen sulfide exposed personnel are presented below.

3.2 HYDROGEN SULFIDE (H_2S) GAS

3.2.1 The effects of H_2S have been recognized in the petroleum industry for almost 60 years. Hydrogen sulfide exposure in the petroleum industry was one of the major industrial hazards in the U.S.A. in the 1930s. Men have been found dead on derrick floors, apparently overwhelmed by the rush of gas when they first drilled into a pocket. Before the danger became known, exposures to large quantities of H_2S occurred without respiratory protection. In some oil fields, gases were found to contain 10 to 12% of H_2S by volume.

3.2.2 Average concentration of H_2S at the well head in Kuwait is 0.1% (1000 ppm). Concentrations may range from being negligible to 15% (150,000 ppm). The potentially worst point sources of H_2S are the well heads. All well heads in the theatre of action should be considered to be potentially sour (i.e., containing H_2S). The odor threshold for H_2S is low (.005 to 0.13 ppm). At

concentrations up to 30 ppm, the odor is that of rotten eggs. The sense of smell fatigues quickly at very low concentrations, and the odor goes undetected even after prolonged exposure. Although the odor is detectable at low concentrations which are not life threatening, the discomfort and possible nausea may be distracting and reduce an individual's efficiency of operation. Above 100 ppm, H_2S rapidly abolishes the sense of smell. Exposures to 250 ppm for 120 min or less is considered potentially life threatening and serious injury is likely. Exposures to 500 to 700 ppm could result in a loss of consciousness, with death possible in 30 minutes to an hour. Exposure to 700 to 1000 ppm results in rapid loss of consciousness, respiratory failure, and death.

(**NOTE:** Inhalation of concentrations in excess of 250 ppm of hydrogen sulfide can be lethal. The probability that these conditions could be created from a crude oil source in a nonconfined area is low but not negligible: the potential for lethal concentrations in the vicinity of a nonburning sour well, or in the head space above sour crude oil tanks, in deep trenches/pits and such requires caution.)

3.2.3 Inhalation Exposure. Exposure to low concentrations can cause irritation of the nose (rhinitis) and throat, headache, dizziness, nausea, and nervousness. High concentrations may cause bronchitis, pulmonary edema, and paralysis of the respiratory system resulting in asphyxiation. Stupor or unconsciousness may be the first signs of excessive exposure because of effects on the CNS (refer to Appendix A).

3.2.3.1 For treatment of inhalation exposure, immediately remove the patient from the exposure area. Resuscitate and administer oxygen as necessary. Continue to monitor for pulmonary edema or excessive tracheobronchial secretions. In severe poisonings, inhalation of amyl nitrite followed by sodium nitrite injections (10 ml of a 3% solution) iv over a period of 2 to 4 min for adults has been suggested, repeating at one-half dose if and when signs and symptoms reappear (refer to Appendix A).

3.2.4 Ocular Exposure. The first signs of exposure to low concentrations (i.e., 4 to 15 ppm) are irritation, tearing, and photophobia. Hydrogen sulfide gas can also cause painful conjunctivitis (which may progress to keratoconjunctivitis), sometimes with corneal erosion and spasm of the eye lids. Lasting eye damage is unlikely to occur.

3.2.4.1 For treatment, hold the eyelids open and flush with water for 15 minutes. Eye irritation usually disappears without any treatment; however, local administration of steroids has been reported to be an effective treatment, especially in preventing progression of conjunctivitis to keratitis.

3.2.5 Dermal Exposure. H_2S Gas can cause irritation but it is not of major concern.

3.2.6 Ingestion. Exposure to H_2S gas by ingestion is not of concern.

3.3 VOLATILE HYDROCARBONS

3.3.1 There is some concern about exposure to volatile aromatic components of fresh crude oil such as benzene, xylene, toluene, and hexane. Exposure to pure solvents are of particular concern in refineries and storage facilities. Solvents have a defatting action that makes the skin more prone to absorbing other chemical agents and to secondary infections. The degree of acute health hazard for exposure to any one of these volatiles varies. Cardiac sensitization to the effects of epinephrine was raised as a concern, although there was some feeling that the levels to produce this effect would be quite high (greater than 50,000 ppm). Because the effects of these compounds are similar, differing only in degree, and treatments are similar, information will be given collectively. The specific effects of the volatile hydrocarbons benzene, xylene, toluene, hexane, propane, and butane are further described in Appendix A.

3.3.2 Aliphatic components such as methane, ethane, propane, and butane are of concern as asphyxiants. They are also of concern when in pressurized containers for their potential as "pressure bombs" as described in the section on explosion, fire, and smoke hazards (Section 3.5).

3.3.3 Dermal Exposure. The aromatic components may be absorbed through the skin. Dermal contact may cause irritation (viz. erythema). Repeated or prolonged skin contact has a defatting action, causing drying, fissuring, and dermatitis.

3.3.3.1 For treatment, contaminated personnel should wash with soap and copius amounts of water after removing their contaminated clothing.

3.3.4 Inhalation exposure. The principal action of volatile hydrocarbons is predominantly upon the CNS as a depressant causing effects ranging from fatigue, headache, nausea, and dizziness, to convulsions, paralysis, and loss of consciousness. Toxic manifestations resulting from a single exposure will vary with the atmospheric concentration. Death due to respiratory collapse can occur in 5 to 10 min at extremely high concentrations (i.e., 3,000 to 20,000 ppm). Inhalation of lower concentrations (approximately 250 to 500 ppm) will only cause vertigo, drowsiness, headache, and nausea.

3.3.4.1 For protection, rescuers should wear a standard issue gas mask (however, efficacy against volatile hydrocarbons will be diminished after 15 minutes). They should remove the victim from the exposure area and resuscitate and administer oxygen as needed.

3.3.5 Ocular Exposure. Vapors may be slightly irritating to the eyes. Pain may occur upon contact, and dulling of the cornea may occur after significant eye contact with the liquid. Damage is not extensive or long-lasting following a splash.

3.3.5.1 For treatment, irrigate with water.

3.3.6 Ingestion Exposure. Effects are similar to those described under inhalation exposure (3.3.4).

3.3.6.1 For treatment, careful gastric lavage using care to prevent aspiration into the lungs is recommended. Do not induce vomiting.

3.4 ASPHALT, DIESEL FUEL, GASOLINE, JET ENGINE FUELS, AND KEROSENE HAZARDS

3.4.1 Asphalt, diesel fuel, gasoline, jet engine fuels, and kerosene are fire hazards. Additionally, they produce toxic fumes. The absorption of fuels following dermal exposure can lead to multisystemic effects following dermal contact. A primary injurious effect of asphalt exposure is thermal burns. Additional information on the health effects and methods of treatment for exposure to these materials are addressed in Appendix A.

3.5 EXPLOSION, FIRE, AND SMOKE HAZARDS

3.5.1 Disruptions in an oil production facility, either accidental or deliberate, are prone to catastrophic explosions and fires. The major factors which contribute to fire and explosion hazards in these sites include the flammable nature and voluminous inventory of crude oil and oil products, the high-pressurized storage condition of petroleum gases, the continuous processing and transferring of petroleum products, and the close proximity of processing and transfering units and storage tanks. Apart from the oil production facilities, the oil fields in the Persian Gulf Region may have oil wells located only a few thousand feet apart. Sabotage efforts to explode oil wells or processing/storage units in GOSPs and refineries or to ignite oil pool traps, oil-laden sand, or water are likely events and therefore military personnel will find themselves in an immediately hazardous situation. The effects of heat, smoke, combustion products, and fire gases are of major concern.

3.5.2 Explosion, Fire, and Heat. Explosion of flammable materials in an oil-producing facility produces a shock wave that will immediately knock down individuals standing within a few hundred meters. Injury from the fall or from flying objects will require medical attention. The largest explosions are likely to result from the rupturing of domed storage tanks and pressurized metal spheres where the LNGs and LPGs are kept. From a design standpoint, LPG spheres are pressurized to their maximum allowable servicing load. Added stress from an impact may crack the metal allowing the sudden release of the explosive gas into the oxygenated atmosphere. The explosive force of these "fire bombs" propagates the initiation of more explosions and fires. Steel structures under heat exposure will lose their strength over time, and a lot of equipment, including columns, will be collapsing. Flames from burning refinery equipment may reach 50 to 100 feet in height causing heat to be radiated for several thousand feet. Skin and retinal burns will result among individuals

standing close to the fire. Every effort should be made to shield individuals from the heat. Flames from an exploding LPG sphere can result in an explosive fireball a few thousand feet in diameter and height. Without protective equipment, a distance of up to 3000 feet may be necessary to avoid heat radiation. Recommended stand-off distances are given in Appendix D.

3.5.2.1 Refinery and storage fires may burn for a considerable amount of time, possibly up to two weeks. Well fires may burn for months. Due to the unpredictable nature of a fire in a petroleum facility, the workshop participants advised that fire fighting efforts be avoided. However, if military operations necessitate stopping a fire in a facility, the general guidelines to follow are: (1) contain the fire by stopping the flow of fuel or draining the fuel; (2) depressurize high-pressure vessels; and (3) cool the equipment. Water spray is the basic procedure for cooling equipment and structures coupled with draining fuels into closed process sewers. Water sources may be limited unless the facility is located along the coast and/or the facility firepumps are in service. Water pumping capacity of up to 10,000 gallons per minute may be needed to control fires and up to 5,000 to 10,000 gallons of firefighting foam concentrate may be needed for each storage tank to be extinguished. Attempts to extinguish well fires should not be made until control measures to shut off the flow are prepared and are in place.

3.5.2.2 A potential problem emerges due to the use of water spraying procedures. Water is more dense than oil and will settle to the bottom of a storage vessel. When the heatwave in a burning storage tank reaches the water at the tank bottom, the water may instantly boil causing flaming oil to erupt from the tank. Individuals within several hundred feet of the tank will be at risk from the burning oil. Additional considerations which may become problems for fighting fires are: (1) limitation of the amount of fire fighting equipment and foam concentrates on site to handle more than one fire at a time, (2) stripping of essential components of fire fighting equipment by the opposing military forces, and (3) time to extinguish a fire. One large (approximately 200 foot) crude oil tank may take 4 to 6 h to extinguish. During that time, the equipment to deliver several thousand gallons of foam, to lay the foam streams, and several hundred thousand gallons of water must be in operation.

3.5.3 **Smoke and Combustion Products Hazards.** The burning of crude oil is similar to the burning of other natural resources, such as fossil fuels. The components of the smoke emitted from a crude oil fire include carbon monoxide, carbon dioxide, and the products of combustion or pyrolysis. These latter products include the oxides of sulfur and nitrogen, aldehydes, and acrolein. Sulfur dioxide results from the combustion of hydrogen sulfide. Crude oil fires also produce several unburned products due to inefficient combustion. The solid particulates of smoke may be coated with polycyclic aromatic hydrocarbons and other hydrocarbons, which may pose additional hazards.

3.5.3.1 The components of smoke are irritating when inhaled and, at high concentrations, may be fatal. High concentrations of carbon monoxide produce death in a few minutes. Asphyxiation may result from high concentrations of carbon dioxide or from oxygen-depleted air. The irritant effects of smoke usually involve the mucosal surface, in particular the eyes and the upper respiratory tract (nostrils to larynx). Very low airborne concentrations (few ppm) of acrolein, aldehydes, and acids (e.g., sulfuric) will produce immediate irritation.

3.5.3.2 In addition to a burning sensation of the eyes, nose, and throat, symptoms may include headaches, dizziness, confusion, nausea, and vomiting. Eye redness, tearing and coughing may also occur. A transient decrement in performance is likely. The degree of irritancy is associated with the concentration of the individual components in the smoke cloud. There are several noteworthy conditions which affect smoke concentrations. In general, the greater the distance from the source of the smoke, the greater the dilution of air contaminants. However, air movement is important. Low-lying areas may be subject to stagnant air and therefore should be avoided. Buildings and other enclosures also are static air environments.

3.5.3.3 Respiratory tract toxicity, including lung damage, may result from short-term exposure of high concentrations of smoke. Pulmonary edema, a consequence of inhalation of oxides of nitrogen, may develop more rapidly in individuals under increased exertion.

3.5.3.4 Unburned volatile hydrocarbons, such as hexane, benzene and toluene are not likely to be at high concentrations in the smoke cloud. For low concentration acute exposures, these hydrocarbons are not toxic. Higher concentrations may cause drowiness and other CNS complications (refer to section 3.3 on volatile hydrocarbons).

3.5.4 **Alleviations and Personal Protection.** Fires and smoke should be avoided. If eye irritation occurs, wash with water. For burns or respiratory effects, supportive treatment is recommended, possibly including oxygen and resuscitation. Suggestions for immediate medical treatment for exposure to carbon monoxide or oxides of nitrogen are given in Appendix A. Following the subsidence of symptoms of minor eye or respiratory tract irritation, individuals should be able to return to duty.

3.5.4.1 Individuals directed to extinguish fires should don appropriate fire fighting gear. During military operations when unexpected smoke clouds appear, standard military gas masks may have to be donned. Safe distances will have to be defined. Monitors for H_2S should be considered.

3.6 MINOR HAZARDS

3.6.1 The hazardous materials identified below may be encountered, however, they were not considered major hazards either as a consequence of their effects, minimal acute effect, or infrequent presence in oil industry facilities in the Persian Gulf region.

3.6.2 Ammonia. Ammonia may be present in the cooling units in refineries at LNG facilities. Therefore the possibility of an ammonia line being broken accidentally or intentionally exists. Ammonia has a very penetrating, distinctly pungent suffocating odor. Odor threshold is probably between 25 and 50 ppm although it has been reported as being as low as 1 ppm. Immediate health effects, treatment, and other relevant information concerning ammonia exposure are described in Appendix A.

3.6.3 Hydrogen Fluoride. When alkylation units are present, the potential exists for exposure to HF. This gas has a sharp and penetrating odor with a threshold ranging from 0.04 to 0.13 ppm. Immediate health effects, treatment, and other relevant information concerning HF exposure are described in Appendix A.

3.6.4 Sulfuric Acid. When alkylation units are present, the potential exists for exposure to sulfuric acid (H_2SO_4). Potential exists for exposure to H_2SO_4 as a result of exposure to combustion products of a refinery or oil field. Sulfuric acid is also routinely used for controlling the pH of cooling water. It may be stored in a variety of vessels or barrels around cooling towers. Immediate health effects, treatment, and other relevant information concerning H_2SO_4 exposure are described in Appendix A.

3.6.5 Sulfur Dioxide. Potential exists for exposure to SO_2 as a result of exposure to combustion products of a refinery or oil field. Immediate health effects, treatment, and other relevant information concerning SO_2 exposure are described in Appendix A.

3.6.6 Tetraethyl- and tetramethyl Lead. Potential for exposure to tetraethyl- and tetramethyl lead exists if blending facilities for the production of gasoline are in the vicinity of explosions from any deliberate or accidental source. Immediate health effects, treatment, and other relevant information concerning tetraethyl- and tetramethyl lead exposure are described in Appendix A (Alkyl Lead Compounds).

3.6.7 Radiation Sources. Potential exists for exposure to 137Ce and other radiation sources, which may be used in liquid level detectors in various parts of the plant. Explosions may result in unpredictable scatter of these radioactive sources. Other locations where radiation sources may be present are laboratories and maintenance shops. The health effects of exposure to radiation include various effects on the blood, and potential cancer. High exposures can also result in acute symptoms

and radiation sickness. Treatment includes removing the source of radiation through decontamination.

3.6.8 Chlorine. Chlorine is used for water treatment and in cooling towers. Immediate health effects, treatment, and other relevant information concerning chlorine exposure are described in Appendix A.

3.6.9 Asbestos. Asbestos is used in structural fire-proofing. It is not an acute toxic hazard, but is likely to be spread in the case of explosions. See relevant information concerning health effects, treatment, and such in Appendix A.

4 MAJOR CONCLUSIONS AND RECOMMENDATIONS OF WORKSHOP PARTICIPANTS

4.1 MAJOR CONCLUSIONS

4.1.1 Oil fields in the neutral zone (southern part of Kuwait) should be considered "sour" (containing H_2S). H_2S gas is considered the major health hazard from crude or burning crude, followed by SO_2.

4.1.2 The most serious hazard is fire and the potential for explosions propagating from fires. Explosion of vessels containing specialty chemicals poses serious injury consequences as well.

4.1.3 Opportunities for mischief include detonating sour oil wells and releasing oil onto sand, water, or roads.

4.1.4 Oil soaked terrain and/or equipment will increase the number of slips, trips, and falls for personnel attempting to maneuver under these conditions.

4.1.5 Health effects related to crude oil and crude oil products are associated with the source of exposure (i.e., focal areas may have potentially high concentrations of toxic or irritating chemicals). These areas may include refineries, tank farms, or erupted well heads. Enclosed spaces also pose a higher hazard due to stagnant air conditions resulting in failure to dilute toxic concentrations of airborne chemicals.

4.2 MAJOR RECOMMENDATIONS

4.2.1 The Kuwaiti government should be contacted to identify and describe layouts of refineries and other petroleum facilities. The chemicals present in the oil-producing facilities should be identified to evaluate chemical hazards. In addition, the use of commercially available PC-based computer software to estimate fire, explosion, and toxic gas dispersion hazards should be considered.

4.2.2 Remain upwind of open wells.

4.2.3 Avoid oil gathering facilities, refineries, and terminals if at all possible due to the fire and explosion hazards inherent in these facilities.

4.2.4 There should be no smoking, flames, or open lights in all oil-producing facilities.

4.2.5 If military operations necessitate entering a refinery, then limit the number of personnel allowed to enter, and provide standard issue gas masks (see Appendix E for further information regarding gas masks).

> [**NOTE:** The only source of breathing air considered totally acceptable for use under these conditions is a self-contained breathing apparatus (SCBA). In the unlikely event an oxygen breathing apparatus (OBA) were available, it would be of limited use because of the problems of canister life and surface temperatures of canisters that may exceed the flash point of materials in the area.]

4.2.6 The loss of odor detection for H_2S due to olfactory fatigue or adaptation is an important concern. The standard issue gas mask provides significant protection against H_2S (see Appendix E for further information regarding gas masks). The use of H_2S monitors by individuals in forward areas was recommended. A suggested set point for the alarm is the current TLV of 10 ppm. However, a concentration of 10 ppm only indicates a potential for hazard. Performance should not be affected.

4.2.7 When attempting to rescue personnel overcome by H_2S, smoke, or other airborne chemical contaminants, don an SCBA.

4.2.8 Protective eye wear is recommended for military personnel coming in contact with mists or fogs positioned over oil-ladened sea water.

4.2.9 Avoid exposure to crude oil and crude oil products. For skin contact, dry wipe. Do not use fuels such as gasoline or kerosene to remove excess oil. At the earliest opportunity, use waterless hand cleaners followed by soap and water to remove the crude oil. Oil-soiled socks and clothing should be changed at the earliest opportunity. Treat affected areas as needed.

4.2.10 Eye contact with crude oil may cause irritation and discomfort. Treat by flushing the eyes with water.

4.2.11 Ingestion of small amounts of crude oil is not considered to be a serious problem. Do not induce vomiting for alleviation of symptoms.

4.2.12 Inhalation of fresh crude oil poses a concern for inhalation of volatile hydrocarbons. Symptoms include headache, dizziness, confusion, nausea, or vomiting. Inhalation of weathered crude oil is of less concern because of the loss of volatile hydrocarbons. Central nervous system symptoms may be more severe for crude oil vapor containing H_2S. Inhalation of high concentrations

of H_2S may be lethal. Individuals may rapidly lose their ability to smell even potentially fatal concentrations of hydrogen sulfide, due to olfactory fatigue. Performance may decrease. Supportive treatment, including oxygen, is recommended for alleviation of symptoms. Smoke inhalation is potentially life threatening, especially in confined quarters. Coughing and eye irritation are associated with smoke inhalation.

4.2.13 Cardiac sensitization to epinephrine following crude oil vapor exposure is unlikely due to the expected low concentrations of volatile hydrocarbons present in the vapor phase.

4.2.14 For wounded individuals contaminated with crude oil, normal decontamination procedures should be followed with the advisory of keeping oxygen treatment areas free of oil and disposing of oil-contaminated clothing as soon as possible to avoid spontaneous combustion. Avoid the use of solvents and fuels to remove oil from the skin because these petroleum products may increase the absorption of chemical or biological warfare agents.

4.2.15 Oil and an oxygen-enriched atmosphere are potentially explosive (refer to Appendix C). Prior to oxygen treatment, oil should be wiped from the victim's face. Decontamination procedures may have already taken place by the aid station personnel prior to oxygen treatment. Equipment involving the use of oxygen must not become contaminated with oil.

4.2.16 Designate a contact point within the military that will provide professional advice to the questions asked regarding health effects and treatment. Additionally, knowledge of oil-producing facilities and fire fighting measures is needed.

APPENDIX A

EMERGENCY HEALTH INFORMATION SHEETS

TABLE OF CONTENTS

This appendix contains summaries of toxicity and emergency action information obtained from a number of sources, including several oil companies and the American Petroleum Institute. These documents represent toxicity and hazard risk as applied to civilian medical and community emergencies. Because they are collected from several sources, they may not be entirely consistent in their approach to a specific problem, but the differences are small. This list is not all inclusive of chemicals found in the petroleum industry, but supplies data on the materials most likely to be found in oil refineries and oil fields. More extensive information is available from the NMRI TOX DET by calling (513) 255-6058 or AV 785-6058, FAX (513) 476-7094.

EXXON EMERGENCY HEALTH INFORMATION SHEET

ALKYL LEAD COMPOUNDS

FORMULA: $Pb(C_2H_5)_4$ $Pb(CH_3)_4$

SYNONYMS: TEL; TETRAETHYL LEAD TML; TETRAMETHYL LEAD
TETRAETHYLPLUMBANE TETRAMETHYLPLUMBANE

NFPA CODE©

UN NO.
1649

CAS NO.
78-00-2 (TEL)
75-74-1 (TML)

HAZCHEM NO.
2PE

(OTHER I.D. NO.)

PROPERTIES

- Colorless oily liquid
- May be dyed with color
- Heavier than water
- Insoluble in water
- Vapor density: heavier than air

	TEL	TML	
• Molecular weight:	323.44	267.33	
• Boiling Point (°C):	200	110	
(°F):	392	230	
• Vapor pressure:	0.2	0.22	(ATMOS)
@ 22°C	152	167	(mmHg)

KEY HAZARDS

- HIGHLY TOXIC WHEN INHALED
- READILY ABSORBED THROUGH THE SKIN
- IRRITATING and CORROSIVE to SKIN, EYES, AND RESPIRATORY TRACT
- REACTS with OXIDIZING MATERIALS — SUCH AS PERCHLORATES
- UNSTABLE — MAY EXPLODE; FLAMMABLE
- KEEP AWAY FROM _____
  (Specify for each site)

WARNING PROPERTIES

- PLEASANT, fruity or musty ODOR — BUT intensity is NOT ADEQUATE WARNING of HAZARDOUS concentrations
- Liquid is OFTEN DYED (red, blue, orange) for COLOR IDENTIFICATION
- METALLIC TASTE may result from exposure

FIRE/ EXPLOSION

- FLAMMABLE LIMITS: 1.6 - 8.6%
- MAY EXPLODE; — with sudden SHOCK, PRESSURE, or HIGH TEMPERATURE
- To EXTINGUISH SHUT OFF FLOW OF MATERIAL — use DRY CHEMICAL, FOAM, CARBON DIOXIDE; WATER SPRAY or MIST may be used to PROTECT FIREFIGHTERS.
- Combat fire from BEHIND EXPLOSION RESISTANT or UNMANNED LOCATIONS
- FIREFIGHTERS should APPROACH FROM UPWIND and WEAR SELF CONTAINED BREATHING APPARATUS, FIRE may produce LEAD FUMES and HYDROCARBONS

HEALTH HAZARDS

- INHALATION and SKIN ABSORPTION — produces central nervous system effects; anxiety, insomnia, tremor, delusion, coma, — EFFECTS MAY BE DELAYED AND MAY BE FATAL
- SKIN IRRITATION may result in first or second degree CHEMICAL BURNS
- EYES — can cause SEVERE EYE DAMAGE and IRRITATION

EMERGENCY MEDICAL TREATMENT

- INHALATION — REMOVE FROM CONTAMINATED AREA, resuscitate and administer oxygen if necessary; FOLLOW-UP — treat symptomatically.
- SKIN CONTACT — CONTINUOUSLY FLUSH WITH SOAP AND WATER while removing contaminated clothing. Consider the use of WHITE GASOLINE or KEROSINE to DECONTAMINATE SKIN.
- EYE CONTACT — hold eyelids open and FLUSH WITH WATER FOR 15 MINUTES; FOLLOW-UP — refer to ophthalmologist.
- CENTRAL NERVOUS SYSTEM EFFECTS ARE OFTEN DELAYED; keep victim under close observation with sedation if necessary. Measure baseline and subsequent LEAD in URINE.
- IN ALL CASES, GET MEDICAL ATTENTION. • CALL POISON CONTROL CENTER

READ OTHER SIDE

ALKYL LEAD COMPOUNDS

PERSONAL PROTECTIVE EQUIPMENT

- When responding to a **LEAK/SPILL, OR WHILE ATTEMPTING RESCUE, USE SELF-CONTAINED BREATHING APPARATUS (SCBA)** with a full facepiece in the pressure demand (positive pressure) mode.
- **MAJOR LEAK/SPILL**, use a **FULLY ENCAPSULATED NEOPRENE** or **POLYVINYL CHLORIDE (PVC) SUIT.**
- **MINOR SPILL**, use a **CHEMICAL PROTECTIVE SUIT, BOOTS, and GLOVES** of the same materials as noted above and goggles.
- **FULL FACEPIECE CANISTER RESPIRATORS** *may be suitable* for situations of low concentrations and short time frame.

COMMUNITY ACTIONS

- **SMALL LEAK/SPILL** (single drum, cylinder, small container) — **ISOLATE 35 METERS** (115 feet) in all directions; restrict entry to emergency response personnel.
- **LARGE LEAK/SPILL** (many drums, cylinders, tanks) — **ISOLATE 100 METERS** (250 feet) in all directions; restrict entry to emergency response personnel, then **EVACUATE IN A DOWNWIND DIRECTION AN AREA OF 1 KILOMETER** (0.6 mile) **WIDE AND 1.5 KILOMETERS** (1 mile) **LONG.**
- These are approximations; conditions change requiring constant reassessment and good judgment.

CRITICAL EXPOSURE LEVELS

Concentration/Time	Effect
0.10 mg (lead)/m^3-TEL 0.15 mg (lead)/m^3-TML	ACGIH Threshold Limit Value (TLV)
1.0 mg/m^3 for 120 minutes or less	Extreme Respiratory Irritation
100 mg/m^3 for 15 minutes or less	**FATAL FOR SHORT EXPOSURES**

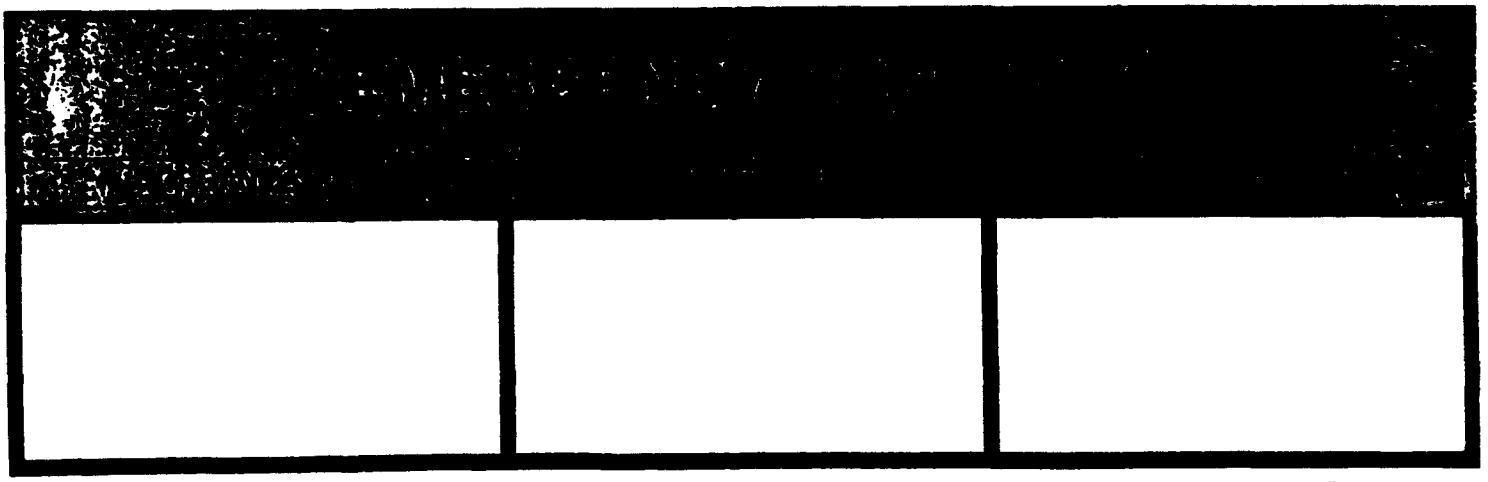
- The above concentrations represent average values; effects due to exposures vary among individuals.

EXPOSURE MEASUREMENT

- Colorimetric procedure (Ethyl Corp., ***LEAD IN AIR TEST KIT*** or equivalent)
- Direct reading instruments — (Vinten Instruments, Ltd., Lead Alkyl Analyzer — Model No. 705; Telmatric Air Analyzer or equivalent)

SPILL CONTROL

- **ABSORB** liquid with vermiculite, sand, or other commercial sorbents.
- Prevent entry into sewers and water courses.
- Remove ***ALL*** ignition sources.
- **LEAD DEPOSITED ON SURFACES** must be washed off with water, collected and disposed. Contaminated soil must be removed.

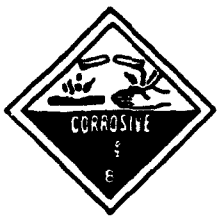


Exxon Biomedical Sciences Inc.

December 1988

READ OTHER SIDE

EXXON EMERGENCY HEALTH INFORMATION SHEET



AMMONIA

NFPA CODES©

FORMULA: NH_3
SYNONYMS: AMMONIA GAS; ANHYDROUS AMMONIA; LIQUID AMMONIA

UN NO.	CAS NO.	HAZCHEM NO.	(OTHER I.D. NO.)
1005	7664-41-7	2PE	

PROPERTIES

- Colorless gas
- Highly soluble in water
- Lighter than air as a gas
- Heavier than air as a cold vapor cloud from liquid source (-33°C)
- Molecular weight: 17.03
- Vapor pressure: 10 ATMOS @ 25.7°C
- Boiling point: -33.35°C

KEY HAZARDS

- IRRITATING AND CORROSIVE TO SKIN, EYES AND RESPIRATORY TRACT
- REACTS VIOLENTLY WITH OXIDIZING AGENTS, HALOGENS AND ALKALI METALS
- KEEP AWAY FROM _____
  (Specify for each site)

WARNING PROPERTIES

- PENETRATING, PUNGENT ODOR at lower concentrations
- IRRITATING to eyes and respiratory tract at higher concentrations
- Tends to form WHITE VAPOR CLOUD at liquid source

FIRE/ EXPLOSION

- FLAMMABLE LIMITS 16%-25% (NH_3 in air); auto-ignition temperature 651°C; will burn only under extreme fire conditions
- MAY EXPLODE when in contact with strong oxidizers, halogens and alkali metals
- In case of fire, use water spray to cool exposed containers and to protect personnel effecting shutoff.
- TO EXTINGUISH, SHUT OFF FLOW OF MATERIAL; water spray and foam can be used.

HEALTH HAZARDS

- INHALATION — can produce pulmonary IRRITATION, chest pain, bronchospasms, pneumonia and pulmonary edema—MAY BE FATAL
- SKIN — can cause IRRITATION, severe burns and blister formation upon contact with gas; liquid contact can cause severe burns
- EYES — can cause SEVERE IRRITATION, burning and tearing upon exposure to gas; liquid contact can cause severe burn

EMERGENCY MEDICAL TREATMENT

- INHALATION — REMOVE FROM CONTAMINATED AREA, resuscitate and administer oxygen if necessary; FOLLOWUP — treat symptomatically.
- SKIN CONTACT — FLUSH WITH WATER FOR 15 MINUTES while removing contaminated clothing; FOLLOWUP — treat as burn.
- EYE CONTACT — hold eyelids open and FLUSH WITH WATER FOR 15 MINUTES; 2-3 drops of 0.5% tetracaine solution or an equally effective aqueous topical solution may be used; FOLLOWUP — refer to ophthalmologist.
- EFFECTS MAY BE DELAYED; keep victim under observation.
- IN ALL CASES, SEEK MEDICAL ATTENTION.

READ OTHER SIDE

AMMONIA

PERSONAL PROTECTIVE EQUIPMENT

- When responding to a LEAK/SPILL, OR WHILE ATTEMPTING RESCUE, USE SELF-CONTAINED BREATHING APPARATUS with a full facepiece operated in the pressure demand (positive pressure) mode.
- For a MAJOR LEAK/SPILL, use a FULLY ENCAPSULATED SUIT of nitrile, butyl, neoprene or polyvinyl chloride (PVC)
- For a MINOR SPILL, use a CHEMICAL PROTECTIVE SUIT, boots and gloves of the same materials as noted above.

COMMUNITY ACTIONS

- SMALL LEAK/SPILL (single drum, cylinder, small containers) — ISOLATE 25 METERS in all directions and restrict entry to emergency response personnel.
- LARGE LEAK/SPILL (many drums, cylinders, or large tank) — ISOLATE 50 METERS in all directions and restrict entry to emergency response personnel, then EVACUATE IN A DOWNWIND DIRECTION AN AREA OF .5 KILOMETER WIDE AND 1 KILOMETER LONG.
- These are approximations; conditions change, requiring constant reassessment and good judgment.

CRITICAL EXPOSURE LEVELS

- (ppm = parts per million; 10,000 ppm = 1% in air)

Concentration/Time	Effect
1-5 ppm	Odor threshold
25 ppm	ACGIH threshold limit value (TLV); generally irritating to unconditioned persons
100 ppm for 120 min or less	Extreme respiratory discomfort
1,000 ppm for 120 min or less	Life threatening or serious injury likely
5,000-10,000 ppm	IMMEDIATELY FATAL

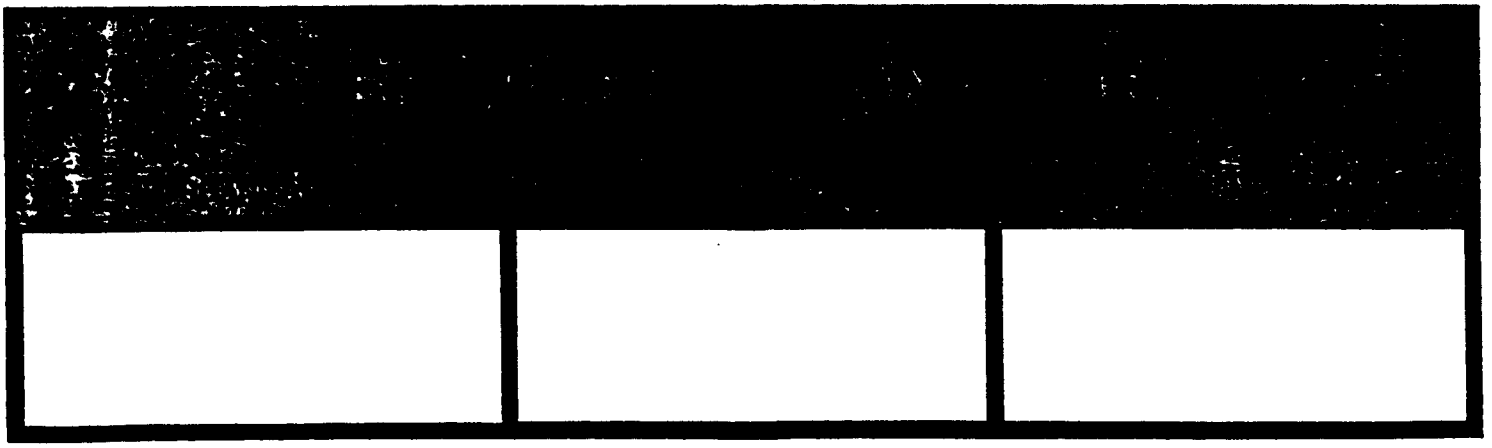
- The above concentrations represent average values; effects due to exposures vary among individuals

EXPOSURE MEASUREMENT

- Colorimetric detector tubes (i.e. Dräger, etc.)
- Direct reading instruments

SPILL CONTROL

- Prevent entry to sewers and water courses.
- Cover liquid spill with chemical hazard foam to minimize gas release.
- Use water spray downwind of spill to reduce gas.
- Water should not contact pools of liquid NH_3 as this will increase gas evolution.



Exxon Biomedical Sciences Inc.

February 1988

READ OTHER SIDE

From: Medical Management of Chemical Exposures in the Petroleum Industry, American Petroleum Institute.

ASBESTOS

Physical Data[6,8]

Chemical Family: Mineral dusts

Chemical Formula: Asbestos is a generic term that applies to a number of naturally occurring hydrated mineral silicates of differing chemical compositions. Such minerals are divided into two groups:

1) Pyroxenes, which include chrysotile ($3MgO \cdot 2SiO_2 \cdot 2H_2O$), the type most widely used in industry in the United States; and
2) amphiboles, including amosite ($FeMg \cdot SiO_3$), crocidolite ($NaFe(SiO_3)_2 \cdot FeSiO_3 \cdot H_2O$), tremolite ($Ca_2 Mg_5 Si_8 O_{22} (OH)_2$), anthopyllite ($(MgFe)_7 Si_8 O_{22} (OH)_2$), and actinolite ($CaO \cdot 3(MgFe)O \cdot 4SiO_2$).

Normal Physical State: Asbestos fibers are characterized by high tensile strength, flexibility, heat and chemical resistance, and favorable frictional properties.

Synonyms[11]

Actinolite, amosite, amphibole, anthopyllite, ascarite, chrysotile, crocidolite, tremolite.

Degree of Health Hazard

Highly toxic for chronic exposure.

Exposure Limits

ACGIH-TLV: Eight-hour, time-weighted average exposure, as follows:

Amosite	0.5 fiber/cc	> 5 μm
Chrysotile	2 fibers/cc	> 5 μm
Crocidolite	0.2 fiber/cc	> 5 μm
Other forms	2 fibers/cc	> 5 μm

Recognized to have carcinogenic or co-carcinogenic potential for humans associated with industrial processes.

OSHA
(1976):[10] 2 fibers/cc, > 5 μm in length (eight-hour, time-weighted average)
10 fibers/cc, > 5 μm in length (ceiling concentration)

NIOSH
(1976):[10] 0.1 fibers/cc, > 5 μm in length, (eight-hour, time-weighted average)
0.5 fibers/cc, > 5 μm in length (peak concentration based on a 15-minute sample period)

Toxicity

Asbestosis is a chronic lung disease due to the inhalation of asbestos fibers and is characterized by diffuse interstitial fibrosis, frequently associated with pleural fibrosis (thickening) or pleural calcification.

The characteristic x-ray changes of asbestosis are small irregular opacities in the lower and middle lung fields, often accompanied by pleural thickening and pleural calcifications.

The pulmonary fibrotic changes develop slowly over the years—often progressively even without further exposure—and their radiographic detection is a direct correlate of their extent and profusion. In some cases, minor fibrosis with considerable respiratory impairment and disability can be present without equivalent x-ray changes. Conversely, extensive pleural radiographic findings may be present with little functional impairment.

Commonly found in advanced cases of asbestosis are pulmonary rales, dyspnea, finger clubbing, and cyanosis, but any or all can be absent in any one case.

Pulmonary hypertension is frequently associated with advanced asbestosis and the resultant cor pulmonale may be the cause of death.

Exposure to airborne asbestos fiber also has been associated with bronchogenic carcinoma, mesothelioma, and cancer of the gastrointestinal tract.

Asbestosis and asbestos-related cancers usually take many years to develop. However, prolonged exposure to the material is not necessary in order to develop such diseases; short-term or intermittent exposures may lead to the same results. Once the inhaled fiber is trapped in the lung tissue, it continues its biological activities regardless of further exposure. Cigarette smoking produces a statistically significant increase in bronchial tumors in those exposed to asbestos.[13] Mesothelioma may also be produced, independent of cigarette smoking.

Spicules of asbestos easily penetrate the skin, especially the fingers in those hagging the fiber. Chronic irritation of the dermis occurs with the formation of corns which may have to be excised.[4]

Medical Treatment[6]

Immediate

Inhalation: None.

Asbestos *continued*

Ingestion: Not applicable.
Skin Contact: Not applicable.
Eye Contact: Not applicable.

Follow-up

Inhalation: No satisfactory treatment other than removal from any further exposure and therapy for any complicating infection.
Ingestion: Not applicable.
Skin Contact: Not applicable.
Eye Contact: Not applicable.

Comments or Discussion

The only satisfactory means of management is prevention of exposure; cigarette smoking is known to increase the risk of cancer. Monitoring procedures may include chest x-ray, pulmonary function studies, and sputum cytology.

Biological Monitoring Procedures

None.

From: *Medical Management of Chemical Exposures in the Petroleum Industry*, American Petroleum Institute.

ASPHALT

Physical Data

Chemical Family: Main constituent is bitumen with small amounts of sulfur, oxygen, nitrogen, and other minerals.

Normal Physical State: Blackish-brown solid or semi-solid material.

Melting Point: Softens to viscous liquid above 90°C.

Odor and Warning Properties: Characteristic.

Synonyms

Petroleum asphalt, bitumen (not to be confused with coal tar).

Degree of Health Hazard

Low for both acute and chronic exposures.

Exposure Limits

ACGIH-TLV: 5 mg/m^3 (eight-hour, time-weighted average)

NIOSH: 5 mg/m^3, determined during any 15-minute period.

Toxicity

Effects on one group of mice inhaling an aerosol of petroleum asphalt, and on another group exposed to smoke from heated petroleum asphalt, included congestion, acute bronchitis, pneumonitis, bronchial dilation, some peribronchiolar round-cell infiltration, abscess formation, loss of cilia, epithelial atrophy and necrosis.*

Mice injected subcutaneously with a mixture of the aromatic and saturated fractions of asphalt produced both benign and malignant tumors. This route of exposure is inappropriate for human cancer evaluation. Both malignant and benign tumors were observed in experimental animals after dermal application of asphalt mixed with solvents. The lack of quantitative dose-response data in animal experiments indicates that intimate contact must extend over a long period before neoplasia become manifest. The NIOSH Criteria Document on Occupational Exposure to Asphalt Fumes reported no data indicating a cancer risk in man.*

Medical Treatment

Immediate

Inhalation: No special treatment indicated. Treat symptomatically.

Ingestion: Not applicable.

Skin Contact: Reported medical treatment deals mainly with thermal burns from contact with molten asphalt. Do not use toxic solvents to remove asphalt from the skin. So-called waterless cleansers may aid in removal. Treat as thermal burns.

Eye Contact: Flush with water. Refer to an ophthalmologist.

Follow-up

Inhalation: No special treatment indicated. Treat symptomatically.

Ingestion: Not applicable.

Skin Contact: Treat as thermal burn.

Eye Contact: No specific treatment indicated.

Comments or Discussion

None.

Biological Monitoring

None.

*Criteria for a Recommended Standard . . . Occupational Exposure to Asphalt Fumes (1977).

From: *Medical Management of Chemical Exposures in the Petroleum Industry*, American Petroleum Institute.

BENZENE

Physical Data

Chemical Family: Aromatic hydrocarbons
Chemical Formula: C_6H_6
Normal Physical State: Clear colorless liquid
Boiling Point: 80.1°C
Melting Point: 5.5°C
Flammable Limits: 1.3–8.0%
Concentration of Chemical in Saturated Air (at 6°C): 13.15%
Odor & Warning Properties: Characteristic odor, not useful for warning purposes.

Synonyms

Benzol; cyclohexatriene

Degree of Health Hazard

Moderate for acute and high for chronic overexposures.

Exposure Limits

ACGIH-TLV: 10 ppm (eight-hour, time-weighted average). In addition, the ACGIH lists benzene as an industrial substance suspect of carcinogenic potential for humans.

OSHA: 10 ppm (eight-hour time-weighted average)
25 ppm ceiling concentration
50 ppm maximum peak above ceiling

NIOSH: 1 ppm as determined by an air sample collected at one liter/minute for two hours.*

Toxicity—Acute

Benzene acts predominantly upon the central nervous system as a depressant causing effects ranging from fatigue, headache, nausea, dizziness, to convulsions, paralysis and loss of consciousness. Toxic manifestations resulting from a single exposure will vary with the atmospheric concentration. Death due to respiratory collapse can occur almost immediately, or may be delayed up to several days. See table.

*Revised Criteria for a Recommended Standard ... Occupational Exposure to Benzene (August 19, 1976).

Benzene

Benzene Vapor Concentration	Exposure	Probable Response
25 ppm	8 hrs.	None.
50–100 ppm	6 hrs.	Slight drowsiness and possibly slight headache.
500 ppm	1 hr.	Symptoms of acute toxicity may occur.
7,500 ppm	30 min.	Life threatening depressant effects.
20,000 ppm	5 min.	Probably fatal.

Acute exposure to high concentrations may also result in chronic toxicity. Benzene may also be absorbed through the skin. Dermal contact may cause erythema and irritation.

Toxicity—Chronic

Benzene produces chronic effects on the bone marrow and blood which may lead to leukopenia, aplastic anemia and leukemia. Prolonged overexposure to benzene may also cause fatigue, nausea, loss of appetite, vertigo, headache, irritability, nervousness, nosebleed, and other hemorrhagic signs.

Repeated or prolonged skin contact has a defatting action, causing drying, fissuring, and dermatitis.

Studies on laboratory animals have raised the possibility that chronic exposure to benzene may have adverse effects on reproductive function or performance. No adverse reproductive effects in humans have been determined.

Medical Treatment[7,48,49]

Immediate

Inhalation: Rescuers should wear respiratory equipment. Remove from exposure. Resuscitate and administer oxygen as needed.

Ingestion: *Do not induce vomiting.* Gastric lavage, if indicated, using care to prevent aspiration into the lungs.

Skin Contact: Wash with soap and plenty of water after removing contaminated clothing.

Eye Contact: Irrigate with water.

Follow up

Inhalation: Treat symptomatically.

December 1980

Benzene *continued*

Ingestion: Treat symptomatically. Observe for any abnormal signs due to aspirated benzene.

Skin Contact: Treat symptomatically. Emollients may be useful.

Eye Contact: None.

Comments or Discussion

None.

Biological Monitoring

Phenol is the major metabolite eliminated in the urine after inhalation, ingestion, or absorption via the skin. While urinary phenol levels are not a measure of damage to the individual, they can be used as an indication of recent exposure to benzene.

"Normal" values for phenol in urine have been reported by various investigators to run from about 5 mg/liter to as much as 42 mg/liter, with an average around 30 mg/liter. NIOSH* considers a urinary phenol level of 75 mg/liter as indicative of a time-weighted average exposure to benzene of 10 parts per million.

Certain foods and medications may result in increased urinary phenols. These include, but are not limited to, bananas, smoked meats, coffee, aspirin, bismuth, and Chloroseptic lozenges. Any increase above the "normal" urinary phenol level, or above 75 mg/liter, should be viewed as possibly due to exposure to benzene.

*NIOSH Criteria Document, "Occupational Exposure to Benzene" (1974)

From: *Medical Management of Chemical Exposures in the Petroleum Industry*, American Petroleum Institute.

BUTANE, ISOBUTANE

Physical Data

Chemical Family: Aliphatic hydrocarbon
Chemical Formula: C_4H_{10}
Normal Physical State: Colorless gas
Boiling Point: – 11.7°C
Flammable Limits: 1.8–8.4%

Synonyms

2-methyl propane, trimethyl methane

Degree of Health Hazard

Very weak CNS depressant and asphyxiant.

Exposure Limits

ACGIH-TLV: 600 ppm (A limit of 800 ppm is being considered by the ACGIH.)

Medical Treatment

Immediate

Inhalation: Remove from exposure. Apply artificial respiration as necessary.
Ingestion: Not applicable.
Skin Contact: For exposure to liquid, treat as a cold burn.
Eye Contact: For exposure to liquid, treat as a cold burn.

Follow-up

Inhalation: None.
Ingestion: None.
Skin Contact: None.
Eye Contact: None.

Comments or Discussion

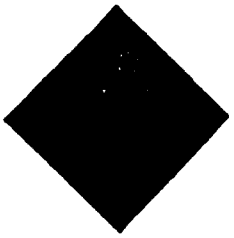
None

Biological Monitoring

None.

EXXON EMERGENCY HEALTH INFORMATION SHEET

CARBON MONOXIDE



FORMULA: CO
SYNONYMS: CARBON OXIDE

NFPA CODE©

UN NO.	CAS NO.	HAZCHEM NO.	
1016	630-08-0	2SE	(OTHER I.D. NO.)

PROPERTIES

- Colorless gas
- Vapor density: lighter than air
- Slightly soluble in water
- Molecular weight: 28.01
- Vapor pressure @ 20°C: > 760 mmHg
  > 1 ATMOS
- Boiling point: -192°C (-314°F)

KEY HAZARDS

- **FLAMMABLE**
- **CHEMICAL ASPHYXIANT**
- **KEEP AWAY FROM** _____
  (specify for each site)

WARNING PROPERTIES

- **ODORLESS** — *provides no warning*
- **TASTELESS**
- A victim may notice the onset of a headache; a severe prolonged case may exhibit cherry red skin discoloration and/or blue lips.

FIRE/ EXPLOSION

- **FLAMMABLE LIMITS** — 12.5% to 74% (CO in air);
  Autoignition temperature — 609°C (1128°F).
- **SHUT OFF** all ignition sources; **SHUT OFF FLOW OF MATERIAL.**
- Gas may travel to ignition source and flash back.
- **FIREFIGHTERS** should wear **SELF-CONTAINED BREATHING APPARATUS; APPROACH FROM UPWIND.**
- **TO EXTINGUISH** — use water, foam, carbon dioxide or dry chemical.

HEALTH HAZARDS

- **INHALATION — CHEMICAL ASPHYXIANT**, producing central nervous system depression, myocardial ischemia — **MAY BE FATAL.**
- **SKIN — CRYOGENIC (cold) BURNS — FROSTBITE** may result if exposed to the liquid

EMERGENCY MEDICAL TREATMENT

- **INHALATION — REMOVE FROM CONTAMINATED AREA, ADMINISTER OXYGEN** and resuscitate if necessary; **FOLLOW-UP** — treat symptomatically.
- **EYES** — systemic poisoning may cause blindness; **FOLLOW-UP** — refer to ophthalmologist.
- **SKIN CONTACT** — in case of **FROSTBITE**, warm affected areas slowly, with clothing or with room temperature water; **FOLLOW-UP** — treat as a burn.
- **EFFECTS MAY BE DELAYED**; keep victim under sustained observation for up to eight hours.
- **IN ALL CASES, GET MEDICAL ATTENTION**
- **CALL POISON CONTROL CENTER**

READ OTHER SIDE

CARBON MONOXIDE

PERSONAL PROTECTIVE EQUIPMENT

- When responding to a LEAK/SPILL, OR WHILE ATTEMPTING RESCUE, USE A SELF-CONTAINED BREATHING APPARATUS with a facepiece operated in the pressure demand (positive pressure) mode.
- DO NOT USE AIR PURIFYING RESPIRATOR
- HEAVY CLOTHING, GLOVES (LEATHER), AND BOOTS will provide protection against direct contact (frostbite).

COMMUNITY ACTIONS

- SMALL LEAK/SPILL (single cylinder, small vessel) — ISOLATE 15 METERS (50 feet) in all directions and restrict entry to emergency response personnel.
- LARGE LEAK/SPILL (many cylinders, large vessels or containers) — ISOLATE 30 METERS (100 feet) in all directions and restrict entry to emergency response personnel, then EVACUATE IN ALL DIRECTIONS A DISTANCE OF 0.8 KILOMETER (0.5 mile).
- These are conservative approximations; conditions change, requiring constant reassessment and good judgement.

CRITICAL EXPOSURE LEVELS

- (ppm = parts per million; 10,000 ppm = 1% in air)

Concentration/Time		Effect
50 ppm	55 mg/m^3	ACGIH Threshold Limit Value (TLV)
1500 ppm for 15 minutes or	1714 mg/m^3	Extreme respiratory discomfort
750 ppm for 120 minutes	857 mg/m^3	
3500 ppm for 15 minutes	4000 mg/m^3	LIFE THREATENING or serious injury possible
1650 ppm for 120 minutes	4000 mg/m^3	
5000 ppm	5716 mg/m^3	IMMEDIATELY FATAL

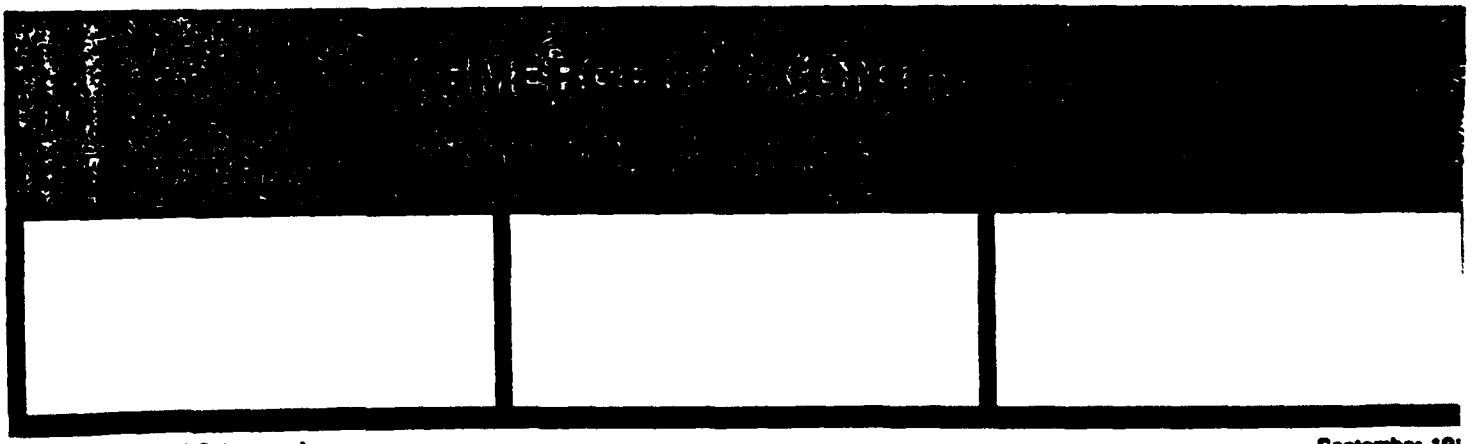
- The above concentrations represent average values; effects due to exposures vary among individuals.

EXPOSURE MEASUREMENT

- Colorimetric detector tubes (i.e., Dräger, etc.).
- Direct reading instruments.

SPILL CONTROL

- ISOLATE the area until gas has dispersed.
- ELIMINATE all sources of ignition in the area.
- VENTILATE closed spaces; check the CO and oxygen concentration before entering.



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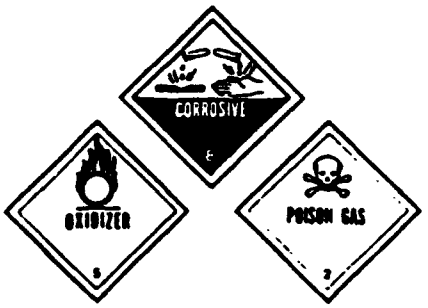
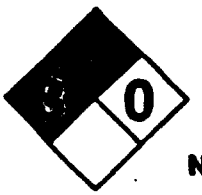
EXXON EMERGENCY HEALTH INFORMATION SHEET



CHLORINE

FORMULA: Cl_2



NFPA CODE©

UN NO.	CAS NO.	HAZCHEM NO.	
1017	7782-50-5	2XE	(OTHER I.D. NO.)

PROPERTIES

- Amber compressed liquid or greenish yellow gas
- Slightly water soluble
- Heavier than air
- Molecular weight: 70.9
- Vapor pressure: 10 ATMOS @ 35.6°C
- Boiling point: -34.6°C

KEY HAZARDS

- **HIGH ACUTE TOXICITY BY INHALATION**
- **CORROSIVE TO SKIN AND EYES**
- **STRONG OXIDIZER; COMBUSTIBLE MATERIALS WILL BURN IN CHLORINE**
- **REACTS EXPLOSIVELY WITH FLAMMABLE GASES, VAPORS, LIQUIDS AND FINELY DIVIDED METALS**
- **KEEP AWAY FROM** _____
  (Specify for each site)

WARNING PROPERTIES

- **IRRITATING ODOR — SMELLS LIKE BLEACH**

FIRE/ EXPLOSION

- **NON-FLAMMABLE**
- **MAY EXPLODE** in contact with flammable gases, vapors, liquids or finely divided metals
- Oxidizer, may cause fire on contact with combustible materials
- Water may increase gas generation from liquid source.
- In case of fire, use water spray to cool exposed containers and to protect personnel effecting shutoff.
- **TO EXTINGUISH, SHUT OFF FLOW OF MATERIAL;** water spray or foam can be used.

HEALTH HAZARDS

- **INHALATION** — can produce pulmonary **IRRITATION**, chest pains, pneumonia, bronchitis and pulmonary edema — **MAY BE FATAL**
- **SKIN** — can cause **IRRITATION**, burning, inflammation and blister formation upon contact with gas; liquid contact can cause chemical burns
- **EYES** — can cause **IRRITATION** upon contact with gas; liquid contact can cause chemical burns

EMERGENCY MEDICAL TREATMENT

- **INHALATION — REMOVE FROM CONTAMINATED AREA,** resuscitate and administer oxygen if necessary. **FOLLOW-UP** — treat symptomatically.
- **SKIN CONTACT — WASH WITH SOAP/WATER AND FLUSH WITH WATER FOR 15 MINUTES** while removing contaminated clothing; **FOLLOW-UP** — treat as chemical burn.
- **EYE CONTACT** — hold eyelids open and **FLUSH WITH WATER FOR 15 MINUTES; FOLLOW-UP** — treat as chemical burn; refer to ophthalmologist.
- **EFFECTS MAY BE DELAYED;** keep victim under observation.
- **IN ALL CASES, GET MEDICAL ATTENTION.**
- **CALL POISON CONTROL CENTER.**

READ OTHER SIDE

CHLORINE

PERSONAL PROTECTIVE EQUIPMENT

- When responding to LEAK/SPILL, OR WHILE ATTEMPTING RESCUE, USE SELF-CONTAINED BREATHING APPARATUS with a full facepiece operated in the pressure demand (positive pressure) mode.
- For a MAJOR LEAK/SPILL, use a FULLY ENCAPSULATED SUIT of Tyvek/Saranex, nitrile, butyl, or Viton.
- For a MINOR LEAK/SPILL, use a CHEMICAL PROTECTIVE SUIT, boots and gloves of the *same materials as noted above.*

COMMUNITY ACTIONS

- SMALL LEAK/SPILL (single drum, cylinder, small containers) — ISOLATE 45 METERS (150 feet) in all directions and restrict entry to emergency response personnel.
- LARGE LEAK/SPILL (many drums, cylinders, tank) — ISOLATE 90 METERS (295 feet) in all directions and restrict entry to emergency response personnel, then EVACUATE IN A DOWNWIND DIRECTION AN AREA OF 1.0 KILOMETER (0.6 mile) WIDE AND 1.5 *KILOMETERS (1 mile)* LONG.
- These are approximations; conditions change, requiring constant reassessment and good judgment.

CRITICAL EXPOSURE LEVELS

- (ppm = parts per million; 10,000 ppm = 1% in air)

Concentration/Time	Effect
0.01 - 0.03 ppm	*Odor threshold*
1 ppm	ACGIH threshold limit value (TLV)
3 ppm for 15 min. or less or 2 ppm for up to 120 min.	*Extreme respiratory discomfort*
10 ppm for 15 min. or less or 5 ppm for up to 120 min.	Life threatening or serious injury likely
500-1000 ppm	FATAL FOR SHORT EXPOSURES

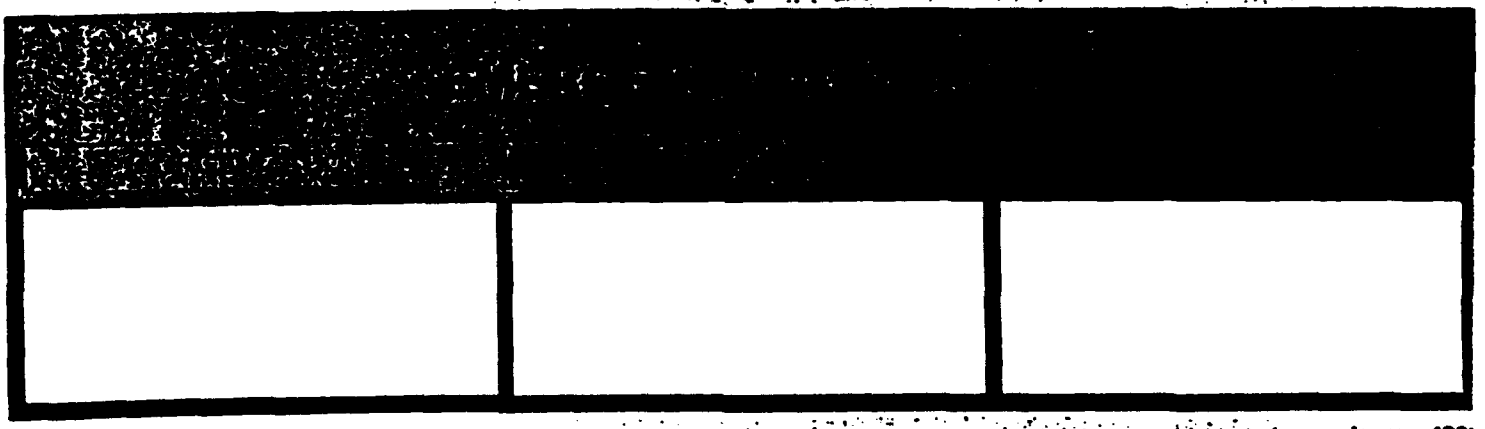
- The above concentrations represent average values; effects due to exposures vary among individuals.

EXPOSURE MEASUREMENT

- Colorimetric detector tubes (i.e. Dräger, etc.)
- Direct reading instruments

SPILL CONTROL

- Prevent entry to sewers and water courses.
- Cover liquid spill with protein foam to minimize vapor release.
- Use water spray downwind from spill to reduce gas concentrations.
- Keep water away from leak source; acid formation/corrosion can make leak worse.



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READ OTHER SIDE

From: *Medical Management of Chemical Exposures in the Petroleum Industry*, American Petroleum Institute.

CRUDE OILS

Physical Data

Chemical Family: Complex hydrocarbon mixture

Chemical Composition: Nonuniform, highly complex mixtures of paraffinic, naphthenic, and aromatic hydrocarbons. Small amounts of sulfur and even smaller amounts of nitrogen and oxygen compounds usually are present.

Boiling Point: Dependent upon composition of crude oil

Normal Physical State: Crude oil may contain, or be composed of, compounds in the gaseous, liquid, and/or solid state, depending on the nature of these compounds and the existent conditions of temperature and pressure.

Flammable Limits: May be highly flammable. Because crude oils have widely variable compositions, specific flammable limits cannot be determined.

Odor & Warning Properties: Some crude oils contain sulfur and have an unpleasant, sometimes sickening, odor of garlic or rotten eggs. The odorous sulfur usually exists in the form of mercaptans or hydrogen sulfide.

Synonyms

Petroleum, rock oil

Degree of Health Hazard

Dependent on composition of crude oil. Some crude oils and crude oil fractions have recently been reported to produce benign and malignant tumors in mice after prolonged exposure by skin application.

Exposure Limits

Composition varies greatly, thus a single limit is not applicable.

Toxicity

The varying content of crude oil does not permit definite toxicological conclusions. Crude oil contains sulfur in various forms and concentrations including sulfide and mercaptans. The gaseous petroleum fractions and the more highly volatile products have a mild anesthetic action. The gases may exist in sufficiently high concentrations to produce narcosis and usually only occur as a result of accidental exposures in enclosed or confined spaces.

The lower boiling point products produce severe chemical pneumonitis if aspirated into the lungs. Dermatitis from lower boiling point fractions may develop, usually as the result of their defatting effect on the skin. Heavier, more viscous fractions may plug skin follicles and lead to dermatitis. A small percentage of people may develop a skin sensitivity to petroleum. Skin cancer from chronic exposure must be considered a possibility.

Medical Treatment

Immediate

Inhalation: Remove to uncontaminated area. Resuscitate and administer oxygen as needed.

Ingestion: Treat symptomatically.

Skin Contact: Wash with soap and water.

Eye Contact: Flush with water.

Follow-up

Inhalation: Treat symptomatically.

Ingestion: Treat symptomatically.

Skin Contact: Any suspicious skin lesions should be surgically excised and examined pathologically.

Eye Contact: None indicated.

Comments or Discussion

None.

Biological Monitoring

No biological monitoring procedures available.

From: *Medical Management of Chemical Exposures in the Petroleum Industry*, American Petroleum Institute.

DIESEL FUEL

Physical Data[16,17]

Chemical Family: Mixed petroleum hydrocarbons

Chemical Composition: Usually that fraction which distills after kerosine, consisting of various paraffins, naphthenes, simple aromatics, and condensed ring aromatics. All diesel fuels have varying amounts of sulfur and additives.

Diesel Fuels	Flash °C	Boiling Range °C	Percent Sulfur
No. 1 Fuel Oil (Premium)	57	180–300	0.2
No. 2 Fuel Oil (Regular)	60	190–430	0.3
Economy Fuel Oil	77	220–370	1.0

The additives for diesel fuel are corrosion inhibitors, anti-oxidants, dispersants, metal deactivators, bactericides, and flow and viscosity improvers.

Corrosion inhibitors and anti-oxidants:
1) Zinc alkyl or aryl dithiophosphates
2) Methyl ditertiary butylphenol
3) Terpene-phosphorus pentasulfide addition products
4) p-phenylenediamine
5) Sulfurized esters
6) Terpenes
7) Olefins

Dispersants:
1) Alkyl polyamide

Metal Deactivators:
1) N,N-disalicylidene-1,2-diaminopropane

Bactericides and Fungicides:
None cited.

Flow & Viscosity Improvers:
1) Isobutylene methacrylate polymers

Boiling Point: 180–370° C.
Normal Physical State: Liquid
Flammable Limits: Flammable
Odor & Warning Properties: Typical.

Synonyms

No. 1 fuel oil; No. 2 fuel oil; economy fuel oil; distillate fuel.

Degree of Health Hazard

Acute: Low by all routes (note: aspiration hazard).
Chronic: Moderate by all routes.

Exposure Limits

Composition varies greatly, thus a single limit is not applicable.

Toxicity[8]

The varying composition of diesel fuels does not permit definite toxicological conclusions. The principal acute toxic effect of inhalation overexposure to diesel fuel is CNS depression. Fuels containing significant quantities of highly cracked stocks may contain polynuclear aromatic hydrocarbons.

While concentrated additives used in diesel fuels may require stringent control, the dilute concentrations in final commercial use generally present no significant toxicological danger. The presence of sulfur or chlorinated additives can have a directly irritating effect on the skin. Zinc alkyl dithiophosphates are slightly toxic orally and may be mildly irritating to the skin and highly irritating to the eyes. Methyl ditertiary butylphenol is moderately toxic on ingestion and irritating upon skin contact. p-Phenylenediamine derivatives are potent irritants and sensitizers. Alkyl polyamide has low systemic toxicity and slightly irritating properties. N,N-disalicylidene-1,2-diaminopropane exhibits moderate systemic toxicity on ingestion and inhalation, and is mildly irritating to skin and eyes. The listed viscosity improvers are physiologically inert.

Medical Treatment

Immediate[48,49]

Inhalation: Remove from further exposure. Resuscitate and administer oxygen as needed.

Ingestion: Treat symptomatically. Do not induce vomiting. Gastric lavage, being careful to avoid aspiration into the lungs.

Skin Contact: Wash with soap and water.

Eye Contact: Flush with water.

Follow-up

Inhalation: None.

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Diesel Fuel *continued*

Ingestion: Treat symptomatically. Observe for any signs of lung damage from aspiration of the diesel fuel.

Skin Contact: Any suspicious skin lesions should be surgically excised and examined pathologically.

Eye Contact: None.

Comments or Discussion

None.

Biological Monitoring

None.

From: *Medical Management of Chemical Exposures in the Petroleum Industry*, American Petroleum Institute.

GASOLINE

Physical Data

Chemical Family: Mixture of C_4 to C_{12} hydrocarbons
Chemical Composition: Commercial grades of motor gasoline contain highly branched paraffins ("alkylate"), branched and internally unsaturated cyclic olefins, naphthenes and aromatics. Benzene in concentrations of less than 0.5 to perhaps as much as 3.0% can be found in U.S. gasolines.
Normal Physical State: Liquid
Melting Point: −90.5 to −95.4°C.
Boiling Point: 39–200°C
Flammable Limits: 1.4–7.4%
Concentration in Saturated Air (at 25° C): Variable, depending on composition.
Odor & Warning Properties: Distinct odor.

Degree of Health Hazard

Moderate to mild for inhalation and skin contact. High for ingestion.

Exposure Limits

The composition varies greatly and thus a single exposure limit is not applicable. In general, the aromatic hydrocarbon content will determine what limit applies. Consequently, the content of benzene, other aromatics, and additives should be determined to arrive at the appropriate limit.

Toxicity—Acute

Gasoline is a central nervous system depressant; its effects progress from confusion through incoordination, unconsciousness, coma, and death as severity of exposure increases. Following severe acute overexposure, degenerative changes may occur in the liver, kidneys, and other organs, but in most cases recovery in humans is clinically complete.

Ingestion of gasoline is occasionally reported as a result of siphoning attempts. In this case, the principal risk is chemical pneumonitis, lung edema, and hemorrhage resulting from aspiration. While swallowing small amounts of gasoline may result in gastrointestinal distress and central nervous system depression, recovery is usually complete in the absence of complications resulting from aspiration into the lungs.

Toxicity—Chronic

There is no conclusive evidence that repeated exposures to relative low concentrations of gasoline vapor in air have any deleterious effects. Gasolines containing significant amounts of benzene may present a risk of benzene-associated blood disorders. Pure n-hexane, a minor constituent of gasoline, is known to cause peripheral nervous system disease. Potential overexposure to these two constituents in gasoline must, therefore, be considered. Generally, no excessive exposure to benzene has been reported from the usual handling of gasolines. No studies on n-hexane exposures from gasoline have been reported. If gasolines are handled indoors, in unventilated areas, potential overexposure to n-hexane and benzene should be evaluated. For the compound specific diseases, see Benzene and Hexane data sheets for additional information.

Medical Treatment[7,23,48,49]

Immediate

Inhalation: Remove from exposure. Resuscitate and administer oxygen as necessary.

Ingestion: Do not induce vomiting. Gastric lavage, taking care to avoid aspiration into the lungs.

Skin Contact: Wash with water.

Eye Contact: Wash with water.

Follow-up

Inhalation: Observe for possible sequelae, central nervous system depression, hypotension and coma.

Ingestion: Treat symptomatically. Observe for chemical pneumonitis.

Skin Contact: None.

Eye Contact: None

Comments or Discussion

The presence of benzene, n-hexane, alkyl lead compounds, or other special additives may be a complicating factor.

Biological Monitoring

None.

From: *Medical Management of Chemical Exposures in the Petroleum Industry*, American Petroleum Institute.

HEXANE

Physical Data

Chemical Family: Aliphatic paraffin
Chemical Formula: C_6H_{14}
Normal Physical State: Volatile liquid
Boiling Point: 68.95°C (156.1°F)
Melting Point: −95°C (−139.0°F)
Flammable Limits: 1.18–7.40% vapor by volume in air
Concentration of Chemical in Saturated Air (at 25°C): 19.94%

Synonyms

Dipropyl; n-hexane.

Degree of Health Hazard

Low for acute exposure; moderate for chronic exposure.

Exposure Limits

ACGIH-TLV: 100 ppm (eight-hour, time-weighted average). (A limit of 50 ppm is being considered by the ACGIH.)
OSHA: 500 ppm (eight-hour, time-weighted average).

Toxicity

Hexanes are weak anesthetic agents. Unacclimatized individuals found no irritation at 500 ppm but encountered nausea, headache, eye and throat irritation at 1,400–1,500 ppm. The health hazard varies depending on the grade of hexane being used. The grade can vary from pure (100% n-hexane) to the technical or commercial grade (45–86% n-hexane with isohexanes and cyclopentanes as the remaining constituents). The technical or commercial grade of hexane may contain up to six percent benzene. Exposure to grades of hexane containing appreciable amounts of benzene should be treated as a benzene exposure.

Hexane has been associated with reports of polyneuropathy in Japan, America, and France. These effects have been associated with exposure to prolonged and excessive solvent vapors in the workplace. The disorder is characterized by degeneration of peripheral nerves. The clinical signs may progress for as long as several months following termination of exposure. Confirmatory studies in animals have been conducted identifying n-hexane as the etiologic agent.

Medical Treatment[48]

Immediate

Inhalation: Remove from exposure. Resuscitate and administer oxygen as necessary.
Ingestion: *Do not* induce vomiting. Gastric lavage being careful to avoid lung aspiration.
Skin Contact: Wash with soap and water.
Eye Contact: Wash with water.

Follow-up

Inhalation: Treat symptomatically.
Ingestion: Treat symptomatically. Observe for chemical pneumonitis.
Skin Contact: None.
Eye Contact: None.

Comments or Discussion

Neurological function tests should be included in the periodic medical surveillance of potentially exposed asymptomatic personnel.

Biological Monitoring

None.

EXXON EMERGENCY HEALTH INFORMATION SHEET

HYDROGEN FLUORIDE

FORMULA: HF
SYNONYMS: ANHYDROUS HYDROFLUORIC ACID
HYDROFLUORIC ACID
HF-A

NFPA CODE©

UN NO.	CAS NO.	HAZCHEM NO.	(OTHER I.D. NO.)
1775	7664-39-3		

PROPERTIES

- Sharp, penetrating odor
- Highly soluble in water
- Lighter than air as a gas, but often behaves as if it is heavier than air
- Molecular weight: 20.01
- Vapor pressure @ 20°C: 650 mmHg 0.86 ATMOS
- Boiling point: 19.4°C (67°F)

KEY HAZARDS

- EXTREMELY IRRITATING AND CORROSIVE TO SKIN, EYES AND RESPIRATORY TRACT
- KEEP AWAY FROM _____
  (Specify for each site)

WARNING PROPERTIES

- SHARP, PENETRATING ODOR at low concentrations
- IRRITATING to eyes and respiratory tract at high concentrations

FIRE/ EXPLOSION

- NOT COMBUSTIBLE OR FLAMMABLE.
- REACTIVE with concrete, glass and ceramics.
- CORROSIVE TO MOST METAL — reaction may form FLAMMABLE GAS (hydrogen).
- FIREFIGHTERS SHOULD WEAR SELF-CONTAINED BREATHING APPARATUS; APPROACH FROM UPWIND.
- To EXTINGUISH fire in area of this material remove/shut off fuel source(s), use water spray, dry chemical or foam.

HEALTH HAZARDS

- INHALATION — can produce SEVERE PULMONARY IRRITATION, chest pain, bronchospasms and pulmonary edema — MAY BE FATAL
- SKIN — can cause SEVERE/BURNS and PERMANENT SKIN DAMAGE — MAY BE FATAL
- EYES — can cause SEVERE IRRITATION and PERMANENT VISUAL DAMAGE
- BONE — can cause SEVERE and PERMANENT BONE DAMAGE

EMERGENCY MEDICAL TREATMENT

- INHALATION — REMOVE FROM CONTAMINATED AREA, *resuscitate and administer oxygen if necessary, Pulmonary Edema will require* RESPIRATORY SUPPORT; FOLLOW-UP — treat symptomatically.
- SKIN CONTACT — FLUSH CONTINUOUSLY WITH WATER FOR 15 MINUTES while removing contaminated clothing, apply iced 0.13-2% BENZALKONIUM CHLORIDE (ZEPHIRAN) and/or 2.5-10% calcium gluconate gel. Monitor the patient's EKG. For severe burns, administer *INTRAVENOUS CALCIUM GLUCONATE* (10%) — MAY BE LIFE SAVING. If subungual tissues are involved, perform EVULSION under local anesthesia; FOLLOW-UP — treat as burn aimed at preventing infection by applying topical antibiotics.
- EYE CONTACT — HOLD EYELIDS OPEN and FLUSH CONTINUOUSLY WITH WATER FOR 15 MINUTES; until reaching HOSPITAL . Consider applying a 1% aqueous calcium gluconate solution, 2-3 drops of 0.5% tetracaine may also be used; FOLLOW-UP — refer to ophthalmologist.
- EFFECTS MAY BE DELAYED; keep victim under observation.
- IN ALL CASES, GET MEDICAL ATTENTION. • CALL POISON CONTROL CENTER

READ OTHER SIDE

HYDROGEN FLUORIDE

PERSONAL PROTECTIVE EQUIPMENT

- When responding to a **LEAK/SPILL, OR WHILE ATTEMPTING RESCUE, USE SELF-CONTAINED BREATHING APPARATUS** with a full facepiece operated in the pressure demand (positive pressure) mode.
- For a **MAJOR LEAK/SPILL**, use a **FULLY ENCAPSULATED SUIT** of vinyl or chlorinated polyethylene material.
- For a **MINOR SPILL**, use **CHEMICAL PROTECTIVE SUITS, BOOTS, and GLOVES** of the same material as noted above.

COMMUNITY ACTIONS

- **SMALL LEAK/SPILL** (single drum, small container) — **ISOLATE 60 METERS** (200 feet) in all directions and restrict entry to emergency response personnel.
- **LARGE LEAK/SPILL** (many drums, large tank) — **ISOLATE 90 METERS** (300 feet) in all directions and restrict entry to emergency response personnel, then **EVACUATE IN A DOWNWIND DIRECTION AN AREA OF 1 KILOMETER** (0.6 mile) **WIDE AND 2 KILOMETERS** (1.3 miles) **LONG.**
- These are approximations; conditions change, requiring constant reassessment and good judgment.

CRITICAL EXPOSURE LEVELS

- (ppm = parts per million; 10,000 ppm = 1% in air)

Concentration/Time	Effect
0.04 — 0.13 ppm 0.03 — 0.11 mg/m³	Odor threshold
3.0 ppm; 2.5 mg/m³	ACGIH Threshold Limit Value (TLV)
10 ppm; 8 mg/m³ for 15 minutes or less	Extreme respiratory discomfort
30 ppm; 25 mg/m³	**LIFE THREATENING** or Serious Injury Likely
120 ppm; 100 mg/m³	**IMMEDIATELY FATAL**

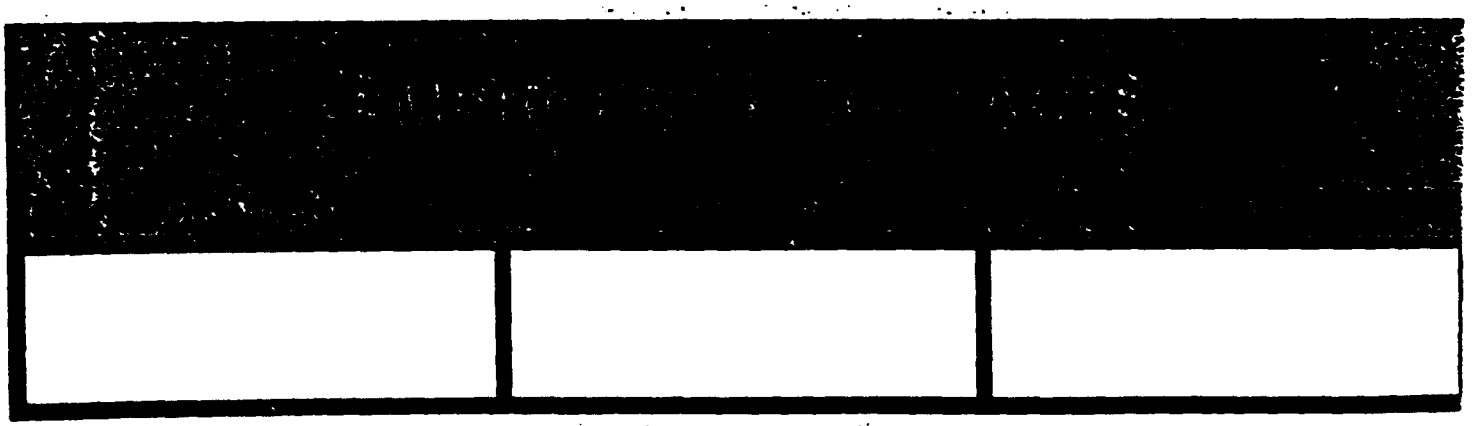
- The above concentrations represent average values; effects due to exposures vary among individuals.

EXPOSURE MEASUREMENT

- Colorimetric detector tubes (i.e. Dräger, etc.)
- Direct reading instruments

SPILL CONTROL

- Prevent entry to sewers and water courses, dike with sandbags or foam barrier.
- Cover liquid spill with chemical hazard foam to minimize gas release. **NEUTRALIZE SPILL** with slaked lime.
- Use water spray downwind of spill to reduce gas concentrations. Knocked down water is hazardous (highly acidic) and should be contained.



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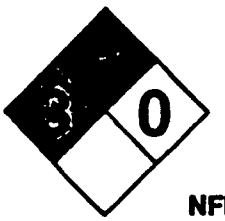
EXXON EMERGENCY HEALTH INFORMATION SHEET

HYDROGEN SULFIDE

FORMULA: H_2S
SYNONYMS: SULFURETTED HYDROGEN; HYDROSULFURIC ACID; SOUR GAS



NFPA CODE©

UN NO.	CAS NO.	HAZCHEM NO.	(OTHER I.D. NO.)
1053	7783-06-4	2WE	

PROPERTIES

- Colorless gas
- Slightly soluble in water
- Slightly heavier than air
- Molecular weight: 34.08
- Vapor pressure: 20 ATMOS @ 25.5°C
- Boiling point: -60.7°C

KEY HAZARDS

- **HIGH ACUTE TOXICITY BY INHALATION**
- **CAN DEADEN THE SENSE OF SMELL**
- **FLAMMABLE**
- **REACTS VIOLENTLY WITH OXIDIZING AGENTS, (E.G., PEROXIDES, NITRATES)**
- **KEEP AWAY FROM** _____
  (Specify for each site)

WARNING PROPERTIES

- Unpleasant **ROTTEN EGG ODOR** at low concentrations
- Sweet odor and possible ***IRRITATION*** *of eyes and respiratory tract at higher concentrations*
- **ODOR IS UNRELIABLE AS A WARNING;** can deaden the sense of smell

FIRE/ EXPLOSION

- **FLAMMABLE LIMITS** 4.3% to 46% (H_2S in air), auto-ignition temperature 260°C
- Dangerously reactive with strong oxidizers
- Shut off all ignition sources.
- Toxic gases are produced in fires (oxides of sulfur).
- *In case of fire, use* water spray to cool exposed containers and to protect personnel effecting shutoff.
- **TO EXTINGUISH, SHUT OFF FLOW OF MATERIAL;** water spray or foam can be used.

HEALTH HAZARDS

- **INHALATION — can produce IRRITATION,** bronchitis, pulmonary edema and paralysis of the respiratory system causing asphyxiation — **MAY BE FATAL**
- **SKIN — can cause IRRITATION;** can produce cryogenic (cold) burns if contact with liquid
- **EYES — can cause IRRITATION,** tearing, severe pain

EMERGENCY MEDICAL TREATMENT

- **INHALATION — REMOVE FROM CONTAMINATED AREA,** resuscitate and administer oxygen if necessary. **FOLLOW-UP** — observation for delayed onset of pulmonary edema and/or infection. Treat symptomatically.
- **EYE CONTACT — hold eyelids open and FLUSH WITH WATER FOR 15 MINUTES.** Treat symptomatically. **FOLLOW-UP** — refer to ophthalmologist.
- **EFFECTS MAY BE DELAYED:** keep victim under observation.
- **IN ALL CASES, GET MEDICAL ATTENTION.**
- **CALL POISON CONTROL CENTER.**

READ OTHER SIDE

PERSONAL PROTECTIVE EQUIPMENT

- When responding to LEAK/SPILL, OR WHILE ATTEMPTING RESCUE, USE SELF-CONTAINED BREATHING APPARATUS with a full facepiece operated in the pressure demand (positive pressure) mode.
- If skin contact with liquid is possible, CHEMICAL PROTECTIVE CLOTHING should be worn to prevent cryogenic burns.

COMMUNITY ACTIONS

- SMALL LEAK/SPILL (single drum, cylinder, small containers) — ISOLATE 35 METERS (115 feet) in all directions and restrict entry to emergency response personnel.
- LARGE LEAK/SPILL (many drums, cylinders, or large tank) — ISOLATE 75 METERS (250 feet) in all directions and restrict entry to emergency response personnel, then EVACUATE IN A DOWNWIND DIRECTION AN AREA OF 1 KILOMETER (0.6 mile) WIDE AND 1.5 KILOMETERS (1 mile) LONG.
- These are approximations; conditions change, requiring constant reassessment and good judgment.

CRITICAL EXPOSURE LEVELS

- (ppm = parts per million; 10,000 ppm = 1% in air)

Concentration/Time	Effect
.005 - 0.13 ppm	Odor threshold
10 ppm	Threshold of irritation; ACGIH threshold limit value (TLV)
50 ppm for 120 min or less	Extreme respiratory discomfort
250 ppm for 120 min or less	Life threatening or serious injury likely
700-1000 ppm	Rapid loss of consciousness; DEATH

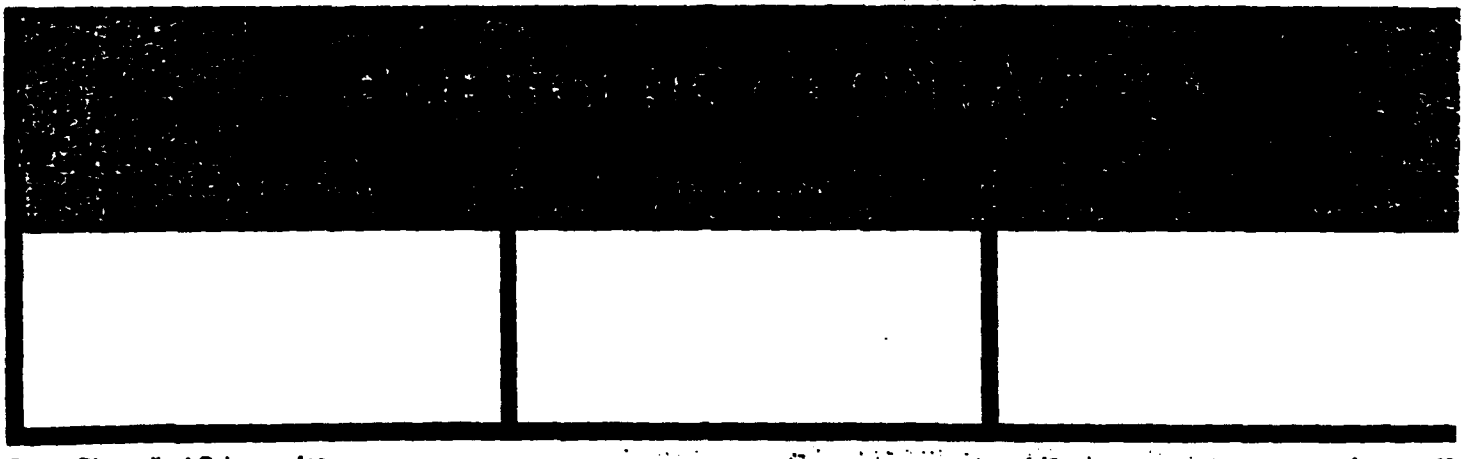
- The above concentrations represent average values; effects due to exposures vary among individuals.

EXPOSURE MEASUREMENT

- Colorimetric detector tubes (i.e. Dräger, etc.)
- Direct reading instruments
- Coexisting hydrogen may give incorrect low readings on direct reading instruments.

SPILL CONTROL

- Remove all ignition sources.
- Prevent entry to sewers and water courses.
- Cover liquid spill with foam to minimize gas release.
- Keep water away from leak source; acid formation/corrosion can make leak worse.
- If water mixes with H_2S, use caustic to neutralize.



From: *Medical Management of Chemical Exposures in the Petroleum Industry*, American Petroleum Institute.

KEROSENE OR JET ENGINE FUELS

Physicial Data[2,3]

Chemcial Family: Petroleum hydrocarbon mixtures.

Chemical Composition: These hydrocarbon mixtures are similar chemically, consisting of aliphatic, olefinic, naphthenic (cycloparaffinic), and aromatic hydrocarbons. The principal components of kerosene are aliphatics ranging from C_{10} to C_{16}. Jet engine fuels are usually composed of gasoline and kerosene, predominately of molecular species ranging from C_5 to C_{16}.

Normal Physical State: Non-viscous oil liquid.

Boiling Range: 175–325°C. (kerosene); 40–325°C. (jet fuel)

Flammable Limits: 0.7–5.0% (kerosene)

Odor & Warning Properties; Characteristics, not altogether disagreeable odor.[3]

Synomyns

Kerosine, astral oil, coal oil, No. 1 fuel oil.

Degree of Health Hazard[7]

Low for both acute and chronic inhalation and skin contact. High for ingestion.

Exposure Limits[22]

For Kerosene:

NIOSH (1977): 100 mg/m³ (time-weighted average for up to a ten-hour work shift, 40-hour work week).

For Jet Fuel: If benzene is found to be present in this material, benzene levels should apply.

Toxicity

Exposure to mists may cause eye and respiratory mucous membrane irritation. Contact with lung tissue by aspiration will cause chemical pneumonitis. Prolonged or related contact with skin may result in irritation, drying and dermatitis.[3]

Polyneuropathy and neurologic dysfunction were reported to develop in workers exposed to jet fuel. A long-term exposure estimated from a one-time measurement to range from 500 to 3,000 ppm of jet fuel (3,476 to 20,859 mg/m³, assuming a molecular weight of 170) has resulted in other non-specific signs and symptoms.[22] These materials can enter the lungs upon aspiration following ingestion and spontaneous or induced vomiting. Aspiration of kerosene results in a progression of events, marked by chemical pneumonitis with prominent endothelial damage and pulmonary hemorrhage and edema. Central nervous system depression may follow systemic absorption.

Medical Treatment[7,23]

Immediate

Inhalation: Not applicable.

Ingestion: Do not induce vomiting. A person who has ingested kerosine should be given olive oil or some other vegetable oil orally to retard absorption of kerosine. Gastric lavage and the induction of vomiting are not advisable because of the possibility of the development of chemical pneumonia from aspiration of kerosine. See "Comments or Discussion."

Skin Contact: Wash with soap and water.

Eye Contact: Wash with water.

Follow-up

Inhalation: Not applicable.

Ingestion: Treat symptomatically. Observe for chemical pneumonitis.

Skin Contact: Treat for any dermatitis resulting from prolonged exposure.

Eye Contact: None.

Comments or Discussion[23]

Emetics are definitely contraindicated. In general, the potential benefits of gastric lavage do not justify the risk in case of kerosene ingestion. If and when warranted by specific circumstances, the procedure should be performed cautiously. Copious amount of water or three per cent sodium bicarbonate may be used. Instill 30 to 60 ml. of olive oil in the stomach at the conclusion of lavage and follow this with a saline cathartic in water (e.g., sodium sulfate). Parenteral antibiotic therapy as prophylaxis against bacterial invasion of the lungs and supportive treatment of pulmonary edema by the use of positive pressure oxygen therapy are recommended. Avoid epinephrine because of possible adverse effects on the sensitized myocardium. A cleansing enema may help remove unabsorbed kerosene.[23]

Biological Monitoring

No specific monitoring procedures available correlating exposure with possible effects.

December 1980

EXXON EMERGENCY HEALTH INFORMATION SHEET

OXIDES OF NITROGEN

NITROGEN DIOXIDE
FORMULA: NO_2
DINITROGEN TETRAOXIDE
FORMULA: N_2O_4
SYNONYMS: NOX
NITROGEN PEROXIDE

NFPA CODE©

UN NO.	CAS NO.	HAZCHEM NO.	(OTHER I.D. NO.)
1067	10102-44-0	2RE	

PROPERTIES

- Reddish-Brown as a gas
- Yellowish-Brown as a liquid
- Decomposes in water to form acid
- Molecular weight: 46.01(NO_2) 92.02(N_2O_4)
- Vapor pressure @ 20°C: 720 mmHg 0.95 ATMOS
- Boiling point: 21°C (70°F)

KEY HAZARDS

- **ACUTE TOXICITY BY INHALATION**
- **CORROSIVE TO SKIN, EYES AND RESPIRATORY TRACT**
- **EXTREMELY STRONG OXIDIZER**
- **KEEP AWAY FROM** _____
  (specify for each site)

WARNING PROPERTIES

- **IRRITATING ODOR** — but **UNRELIABLE AS A WARNING** of hazardous concentrations
- **IRRITATING TO EYES, SKIN AND RESPIRATORY TRACT**

FIRE/ EXPLOSION

- **NON-FLAMMABLE**
- **DOES NOT** present an explosive hazard itself, however, **REACTIONS WITH FUELS** and **CHLORINATED HYDROCARBONS MAY BE VIOLENT.**
- **OXIDIZER** may cause fire on contact with combustible materials.
- **FIREFIGHTERS SHOULD WEAR SELF-CONTAINED BREATHING APPARATUS; *APPROACH FROM UPWIND.***
- In case of fire, use water spray to cool exposed containers and to protect personnel effecting shutoff.
- **TO EXTINGUISH** fire in the area of this material remove/shut off fuel source(s), use dry chemical, carbon dioxide or halon.

HEALTH HAZARDS

- **INHALATION** — can produce **SEVERE PULMONARY IRRITATION,** pulmonary edema may be delayed for up to 72 hours including fibrosis and **PERMANENT LUNG DAMAGE** may occur — **MAY BE FATAL.**
- **SKIN** — may be **CORROSIVE** causing severe irritation or **CHEMICAL BURNS;** can produce cryogenic (cold) burns on contact with liquid.
- **EYES** — can cause **SEVERE IRRITATION,** burning and tearing upon exposure to the gas; liquid exposure can produce frostbite and **PERMANENT EYE DAMAGE.**

EMERGENCY MEDICAL TREATMENT

- **INHALATION — REMOVE FROM CONTAMINATED AREA,** resuscitate and administer oxygen if necessary; **FOLLOW-UP** — treat symptomatically.
- **SKIN CONTACT — FLUSH WITH WATER FOR AT LEAST 15 MINUTES** while removing contaminated clothing; **FOLLOW-UP** — treat as a burn.
- **EYE CONTACT** — hold eyelids open and **FLUSH CONTINUOUSLY WITH WATER FOR 15 MINUTES; FOLLOW-UP** — refer to ophthalmologist.
- **EFFECTS MAY BE DELAYED** for many hours; keep victim under observation.
- **IN ALL CASES, GET MEDICAL ATTENTION.**
- **CALL POISON CONTROL CENTER.**

READ OTHER SIDE

OXIDES OF NITROGEN

PERSONAL PROTECTIVE EQUIPMENT

- When responding to a LEAK/SPILL, OR WHILE ATTEMPTING RESCUE, USE A SELF-CONTAINED BREATHING APPARATUS with a facepiece operated in the pressure demand (positive pressure) mode.
- For a MAJOR LEAK/SPILL, USE A FULLY ENCAPSULATED SUIT of chlorinated polyethylene (PVC) or Viton material.
- For a MINOR LEAK/SPILL, use a CHEMICAL PROTECTIVE SUIT, boots and gloves of the same material as noted above.

COMMUNITY ACTIONS

- SMALL LEAK/SPILL (single drum, cylinder, small containers) — ISOLATE 45 METERS (150 feet) in all directions and restrict entry to emergency response personnel.
- LARGE LEAK/SPILL (many drums, cylinders, or large tank) — ISOLATE 90 METERS (300 feet) in all directions and restrict entry to emergency response personnel, then EVACUATE A DOWNWIND AREA OF 0.6 KILOMETER (0.4 mile) WIDE AND 1.3 KILOMETERS (0.8 mile) LONG.
- These are conservative approximations; conditions change, requiring constant reassessment and good judgement.

CRITICAL EXPOSURE LEVELS

- (ppm = parts per million; 10,000 ppm = 1% in air)

Concentration/Time		Effect
0.39 ppm	0.78 mg/m^3	ODOR Threshold
3.0 ppm	6.0 mg/m^3	ACGIH Threshold Limit Value (TLV); generally irritating to unconditioned persons.
50 ppm for 15 minutes or	100 mg/m^3	Extreme respiratory discomfort
25 ppm for up to 120 minutes	50 mg/m^3	
100 ppm for 15 minutes or	200 mg/m^3	LIFE THREATENING or serious injury likely
50 ppm for up to 120 minutes	100 mg/m^3	

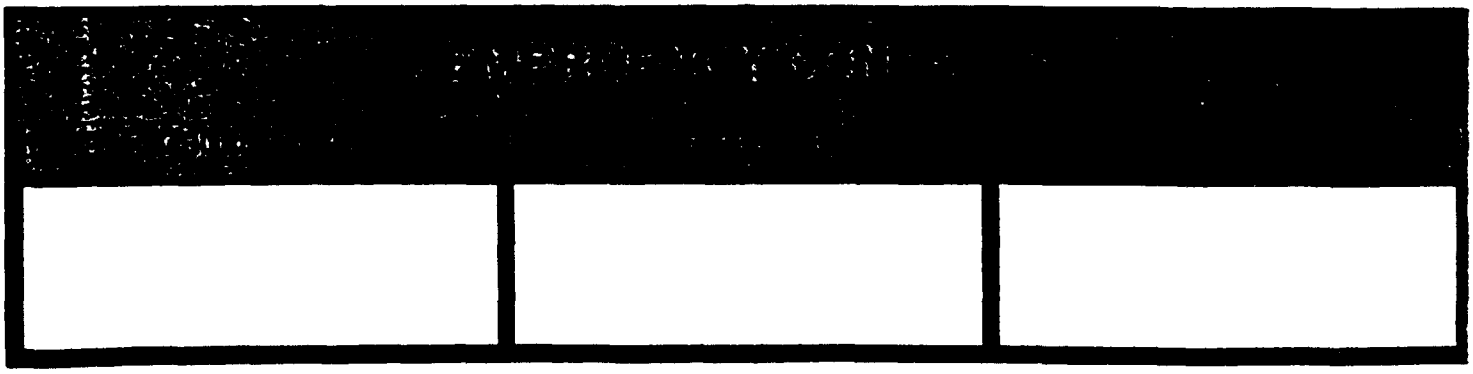
- The above concentrations represent average values; effects due to exposures vary among individuals.

EXPOSURE MEASUREMENT

- Colorimetric detector tubes (i.e., Dräger, etc.).
- Direct reading instruments.

SPILL CONTROL

- DIKE the spill to prevent entry into sewers and water courses.
- USE WATER SPRAY DOWNWIND from spill to reduce gas concentrations. DO NOT put water directly on the spill. Knocked down water is CORROSIVE. Dike the contaminated runoff.
- NEUTRALIZE spill with slaked lime, sodium bicarbonate or crushed limestone.
- ABSORB liquid with fly ash or cement powder.



Exxon Biomedical Sciences, Inc.

September 198

READ OTHER SIDE

From: *Medical Management of Chemical Exposures in the Petroleum Industry*, American Petroleum Institute.

PROPANE

Physical Data

Chemical Family: Aliphatic paraffin
Chemical Formula: C_3H_8
Normal Physical State: Gas
Boiling Point: −42.1°C
Melting Point: −189.69°C
Flammable Limits: 2.4–9.5% vapor by volume in air
Odor & Warning Properties: Pure propane is odorless but commercial grades may have a natural gas odor.

Synonyms

Dimethylmethane, propyl hydride. Not to be confused with LPG (Liquefied Petroleum Gas) often referred to as "propane."

Degree of Health Hazard

Low for both acute and chronic exposures.

Exposure Limits

ACGIH: *A simple asphyxiant.*
OSHA: 1,000 ppm (eight-hour, time-weighted average).

Toxicity

Very little toxicological work has been done on propane. Guinea pigs exposed to concentrations from 2.4 to 2.9% and 4.7 to 5.5% propane by volume for periods of five minutes, 30 minutes, and one to two hours, showed signs of irregular breathing at the lower concentrations; tremors at high concentrations during the first five minutes; and nausea, retching, and a state of stupefication during longer periods of exposure. Human cases of overexposure were not found in the literature.

Medical Treatment[48,50]

Immediate

Inhalation: Remove from exposure. Resuscitate and administer oxygen as needed.
Ingestion: Not applicable.
Skin Contact: Not applicable.
Eye Contact: Not applicable.

Follow-up

Inhalation: None.
Ingestion: None.
Skin Contact: None.
Eye Contact: None.

Comments or Discussion

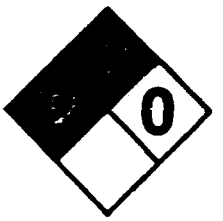
None.

Biological Monitoring

None indicated.

SULFUR DIOXIDE

FORMULA: SO_2
SYNONYMS: SULFUR OXIDE
SULFUROUS ACID ANHYDRIDE
SULFUROUS OXIDE



NFPA CODE©

UN NO.	CAS NO.	HAZCHEM NO.	
1079	7449-09-5	2RE	(OTHER I.D. NO.)

PROPERTIES

- Colorless gas or liquid under pressure
- Soluble in cold water
- Evaporates rapidly from liquid
- Vapor density: heavier than air
- Molecular weight: 64.07
- Vapor pressure: 2432 (mmHg) @ 20°C
  3.2 (ATMOS)
- Boiling point: -10°C (14°F)

KEY HAZARDS

- IRRITATING TO EYES, SKIN AND RESPIRATORY TRACT
- KEEP AWAY FROM _____
  (Specify for each site)

WARNING PROPERTIES

- STRONG SULFUROUS, SUFFOCATING ODOR at high concentrations
- IRRITATING TO EYES AND RESPIRATORY TRACT at high concentrations

FIRE/ EXPLOSION

- NOT COMBUSTIBLE OR FLAMMABLE
- Reacts with water to form CORROSIVE ACID
- To EXTINGUISH fire in area of this material remove/shut off fuel source(s). Use water spray or foam.

HEALTH HAZARDS

- INHALATION — can produce SEVERE PULMONARY IRRITATION, chest pain, severe breathing difficulty, pulmonary edema — MAY BE FATAL
- SKIN — can produce IRRITATION; liquid exposure can produce FROSTBITE and CRYOGENIC (COLD) BURNS
- EYES — can produce SEVERE IRRITATION, burning, and tearing upon exposure to gas; liquid exposure can produce frostbite and PERMANENT EYE DAMAGE

EMERGENCY MEDICAL TREATMENT

- INHALATION — REMOVE FROM CONTAMINATED AREA, resuscitate and administer oxygen if necessary; FOLLOW-UP — treat symptomatically for possible lung damage.
- SKIN CONTACT — FLUSH WITH WATER FOR 15 MINUTES while removing contaminated clothing; FOLLOW-UP — treat as chemical burn.
- EYE CONTACT — hold eyelids open and FLUSH WITH WATER FOR 15 MINUTES; two or three drops of 0.5 percent tetracaine solution or an equally effective aqueous topical solution (Lavoptika) may be used; FOLLOW-UP — refer to ophthalmologist.
- EFFECTS MAY BE DELAYED by several hours; keep victim under observation for infection or scarring.
- IN ALL CASES, GET MEDICAL ATTENTION. • CALL POISON CONTROL CENTER

READ OTHER SIDE

SULFUR DIOXIDE

PERSONAL PROTECTIVE EQUIPMENT

- When responding to a LEAK/SPILL, or WHILE ATTEMPTING RESCUE, USE SELF-CONTAINED BREATHING APPARATUS with a full facepiece operated in the pressure demand (positive pressure) mode.
- For a MAJOR LEAK/SPILL, use a FULLY ENCAPSULATED SUIT of vinyl, Viton, flame retardant urethane or chlorinated polyethylene material.
- For a MINOR SPILL, use a CHEMICAL PROTECTIVE SUIT, boots and gloves of the same material as noted above.

COMMUNITY ACTIONS

- SMALL LEAK/SPILL (small container) — ISOLATE 37 METERS (120 feet) in all directions and restrict entry to emergency response personnel.
- LARGE LEAK/SPILL (many drums, large tank) — ISOLATE 75 METERS (250 feet) in all directions and restrict entry to emergency response personnel, then EVACUATE IN A DOWNWIND DIRECTION OF 1 KILOMETER (0.6 mile) WIDE AND 1.5 KILOMETER (1 mile) LONG.
- These are approximations; conditions change, requiring constant reassessment and good judgment.

CRITICAL EXPOSURE LEVELS

- (ppm = parts per million; 10,000 ppm = 1% in air)

Concentration/Time		Effect
2 ppm;	5.2 mg/m^3	ACGIH Threshold Limit Value (TLV)
3-5 ppm;	8 - 13 mg/m^3	Odor threshold
20 ppm; for 15 minutes or	52 mg/m^3	Extreme respiratory discomfort
10 ppm; for 120 minutes	26 mg/m^3	
100 ppm; for 15 minutes or	260 mg/m^3	Life threatening or serious injury likely
50 ppm; for 120 minutes	130 mg/m^3	
1000 ppm;	2600 mg/m^3	IMMEDIATELY FATAL

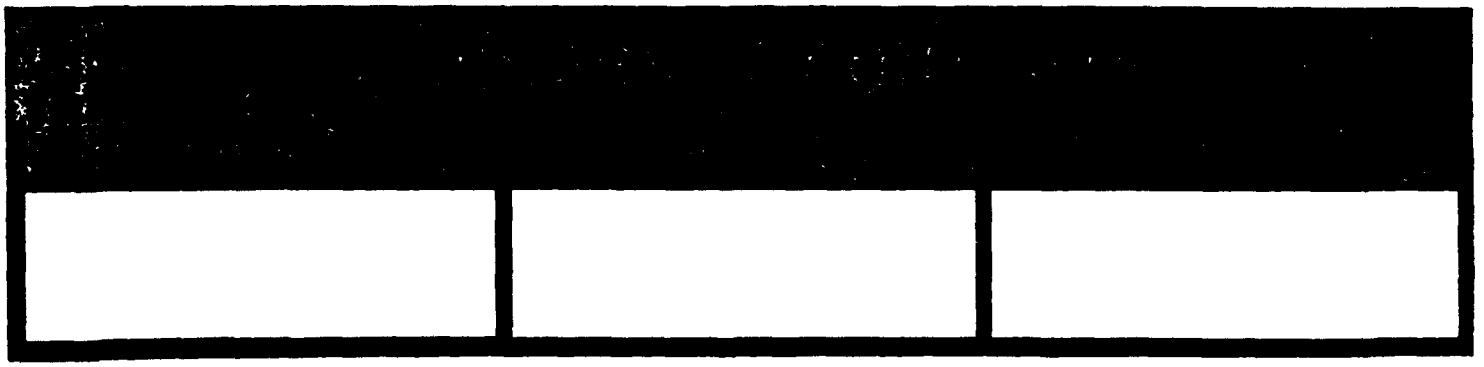
- The above concentrations represent average values; effects due to exposures vary among individuals.

EXPOSURE MEASUREMENT

- Colorimetric detector tubes (i.e. Dräger, etc.)
- Direct reading instruments

SPILL CONTROL

- Use water spray downwind to reduce GAS but DO NOT PUT WATER ON LEAK OR SPILL AREA.
- Isolate area until gas has dispersed.
- Prevent entry to sewers and water courses.



Exxon Biomedical Sciences Inc.

November 1988

READ OTHER SIDE

From: *Medical Management of Chemical Exposures in the Petroleum Industry*, American Petroleum Institute.

SULFURIC ACID

Physical Data

Chemical Family: Inorganic acid
Chemical Formula: H_2SO_4
Normal Physical State: Liquid
Boiling Point: 330°C
Melting Point: 10.5°C
Flammable Limits: Not applicable.

Synonyms

Oil of vitriol, battery acid.

Degree of Health Hazard

Moderate for acute and chronic overexposure.

Exposure Limits

ACGIH-TLV:	1 mg/m³ (eight-hour, time-weighted average)
OSHA:	1 mg/m³ (eight-hour, time-weighted average)
NIOSH:	1 mg/m³ (ten-hour workday, 40-hour workweek, time-weighted average)

Toxicity[3,6,13,26]

Concentrated sulfuric acid is extremely irritating and corrosive to the skin and mucous membranes primarily due to its affinity for water. As sulfuric acid is diluted with water, the intensity of its dehydration/charring action gradually diminishes to that of irritation of the skin and mucous membranes. Eye contact with sulfuric acid is capable of causing irreparable damage to the cornea resulting in blindness.

Sulfuric acid mist is a strong irritant to eye and respiratory mucous membranes. Inhalation can cause tickling of the nose and throat, sneezing and coughing, and reflex increase in the rate of respiration, which decreases with the depth of penetration of the mist into the lungs. Due to these factors, it is rare that anyone who is able to escape would be exposed to high concentrations.

Persons may become acclimatized to sulfuric acid mist, tolerating three to four times higher concentrations than are tolerated by unacclimatized individuals.

Fuming sulfuric acid, or oleum, is a solution of sulfur trioxide in anhydrous sulfuric acid. The "fumes" of oleum are initially composed of sulfur trioxide which will combine with water, present either in the air or on the mucous membranes of exposed persons, to form sulfuric acid. Fuming sulfuric acid, because of its higher vapor pressure and attendant release of sulfur trioxide is intensely irritating to the respiratory tract.

Medical Treatment[6,48]

Immediate

Inhalation: Remove from exposure. Resuscitate and administer oxygen as needed.

Ingestion: Do not induce vomiting. Careful gastric lavage.

Skin Contact: Wash thoroughly with water.

Eye Contact: Flush thoroughly with water for at least 15 minutes. Consult an ophthalmologist.

Follow-up

Inhalation: Treat symptomatically.
Ingestion: Treat symptomatically.
Skin Contact: Treat as chemical burn.
Eye Contact: Follow up with ophthalmologist.

Comments or Discussion

Individuals with any respiratory or cardiac disease or a preexisting asthma must be carefully evaluated in regard to job placement.

NIOSH Criteria Document "Sulfuric Acid" (June, 1974) available.

Biological Monitoring

Periodic evaluation of pulmonary function is advisable.

From: *Medical Management of Chemical Exposures in the Petroleum Industry*, American Petroleum Institute.

TOLUENE

Physical Data

Chemical Family: Aromatic Hydrocarbons
Chemical Formula: $C_6H_5CH_3$
Normal Physical State: Colorless liquid
Boiling Point: 110.6°C
Melting Point: −95°C
Explosive Limits: 1.27–7.0%
Concentration of Chemical in Saturated Air (at 25°C): 3.75%
Odor & Warning Properties: Toluene has an aromatic odor. The odor of toluene is detectable by most people at concentrations ranging from 10 ppm to 15 ppm. Odor has little value as a warning property.

Synonyms

Toluol, methylbenzene, phenylmethane, methacide.

Degree of Health Hazard

Slight for acute and moderate for chronic overexposures.

Exposure Limits

ACGIH-TLV:	100 ppm—Skin
OSHA:	220 ppm (eight-hour, time-weighted average)
	300 ppm (acceptable ceiling concentration)
	500 ppm (acceptable maximum peak above the acceptable ceiling concentration for an eight-hourshift, maximum duration 10 minutes)
ANSI:	(Z37.12-1974)
	100 ppm (eight-hour, time-weighted average)
	200 ppm (acceptable ceiling concentration)
	500 ppm (acceptable maximum peak above the acceptable ceiling concentration for an eight-hour shift, maximum duration 10 minutes)
NIOSH:	100 ppm (eight-hour, time-weighted average)
	200 ppm (acceptable ceiling concentration)

Toxicity—Acute

Toluene acts predominantly upon the central nervous system as a depressant causing fatigue, headache, confusion, paresthesia, dizziness, collapse, and muscular incoordination. Toxic manifestations resulting from a single exposure will vary with the atmospheric concentration. See Table.

Acute exposure of short duration and at infrequent intervals can be tolerated without irreversible toxic effect. However, sustained exposure to concentrations of toluene which produce narcosis may result in fatal paralysis of the respiratory center. Toluene can be absorbed through the skin.

Toxicity—Chronic

Benzene-free toluene does not produce chronic effects on the bone marrow and blood. Prolonged overexposure to toluene may cause fatigue, nausea, loss of appetite, vertigo, headache, and alcohol intolerance.

Wilson (23)* in 1943 reported on the effects of exposure of 100 workers out of a total of 1,000 employees exposed to vapor of commercial toluene who presented themselves to the hospital for examination. The patients were classified into 3 groups: Group 1—those patients exposed to toluene vapor from 50 up to 200 ppm; Group 2—those persons exposed to vapor from 200 to 500 ppm; and Group 3—those workers exposed to vapor from 500 to 1,500 ppm. Exposures were from 6 to 8 hours daily for periods of 1 to 3 weeks.

The following effects were reported at:

50 to 200 ppm (approximately 60 percent of the patients)—headache, lassitude, and loss of appetite.

200 to 500 ppm (approximately 30 percent of the patients)—headache, nausea, bad taste in the mouth, anorexia, lassitude, slight but definite impairment of coordination and reaction time, and momentary loss of memory. Complaints were more numerous and more pronounced than at lower exposure levels.

500 to 1,500 ppm (approximately 10 percent of the patients)—headache, nausea, dizziness, anorexia, palpitation and extreme weakness. Loss of coordination was pronounced and reaction time was definitely impaired.

Repeated or prolonged skin contact has a defatting action, causing drying, fissuring, and dermatitis.

*NIOSH Criteria Document Ref. (23): Wilson, R. H.: Toluene Poisoning. JAMA 123:1106–08 (1943).

Toluene *continued*

Toluene Vapor Concentration	Time of Exposure	Probable response	Probable After Effects
50–100 ppm	8 hrs.	No serious effects. Slight drowsiness and possibly slight headache in the unconditioned workers.	None.
200 ppm (MAC)	8 hrs.	Unconditioned workers may complain of fatigue, some muscular weakness and burning, itching, or "crawling" skin. There may be complaints of headache and some nausea.	Unconditioned workers may complain of fatigue of short duration and a few may suffer restless sleep.
300–400 ppm	8 hrs.	Varying degrees of fatigue and headache. Varying degrees of muscular weakness, mental confusion and slight incoordination.	Fatigue lasting several hours and insomnia.
600 ppm	3 hrs.	Marked fatigue and mental confusion, exhilaration, headache, and dizziness.	Fatigue and weakness lasts several hours. There may be complaints of nausea and nervousness. Many suffer headache.
	8 hrs.	Definite mental confusion, considerable incoordination and staggering gait.	
800 ppm	3 hrs.	Nausea and pronounced confusion. Considerable incoordination and staggering gait.	Nervousness and fatigue may last several days. There may be marked insomnia.

Medical Treatment[6,48]

Immediate

Inhalation: Remove from exposure. Resuscitate and administer oxygen as needed.

Ingestion: Do not induce vomiting. Careful gastric lavage.

Skin Contact: Wash with soap and water after removing contaminated clothing.

Eye Contact: Flush with water.

Follow-up

Inhalation: Treat symptomatically.

Ingestion: Treat symptomatically. Observe for any signs of pneumonitis from aspirated toluene.

Skin Contact: Treat symptomatically. Emollients may be useful.

Eye Contact: None.

Comments or Discussion

None.

Biological Monitoring

On the basis of various studies, a level of 5 g/l of hippuric acid in urine has been shown to correlate with a time-weighted average exposure of 200 ppm of toluene vapor. This level of urinary hippuric acid, if from toluene alone, represents an unacceptable absorption of toluene posing and possible risk of toluene poisoning. It should be noted, however, that normal dietary constituents such as certain fruits and vegetables raise the normal level of hippuric acid excretion. Methods based on colorimetry, fluorimetry, and ultraviolet spectrophotometry are used to determine hippuric acid in urine.

From: *Medical Management of Chemical Exposures in the Petroleum Industry*, American Petroleum Institute.

XYLENE

Physical Data

Chemical Family: Aromatic hydrocarbons
Chemical Formula: $C_6H_4(CH_3)_2$
Normal Physical State: Liquid

	o-Xylene	m-Xylene	p-Xylene
Boiling Point:	144.4°C	193.1°C	138.4°C
Melting Point:	− 25.2°C	− 47.9°C	13.3°C
Flammable Limits (% in air):	1.1–6.4	1.1–6.4	1.1–6.6
Concentration of Chemical in Saturated Air (at 25°C):	0.68%	1.09%	1.13%
Synonyms:	o-xylol 1,2-Dimethyl benzene	m-xylol 1,3-Dimethyl benzene	p-xylol 1,4-Dimethyl benzene

Odor and Warning Properties: May be recognized by its characteristic aromatic odor. For xylene, the odor threshold was reported at 20 ppm, with the odor of 40 ppm reported as distinctly noticeable.

Degree of Health Hazard

Moderate for both acute and chronic overexposures.

Exposure Limits

ACGIH-TLV: 100 ppm—Skin
OSHA: 100 ppm—Skin
NIOSH: 100 ppm (time-weighted average up to a ten-hour workday, forty-hour workweek)
200 ppm (acceptable ceiling concentration for a sampling period of ten minutes)

Toxicity—Acute

Inhalation is the primary exposure of xylene. Xylene can be absorbed through the skin. Acute exposures to high concentrations may result in central nervous system depression which can lead to unconsciousness. Characteristic indications of acute overexposure include giddiness, fatigue, palpitation, dyspnea, anxiety, and numbness of hands and feet.

The narcotic and other effects of xylene at high concentrations were indicated in a case where three painters were using a solvent of 90% xylene in a confined space. The estimated xylene concentration reached 10,000 ppm. One fatality occurred from pulmonary edema, the other two men survived but showed temporary hepatic and renal impairment.

Toxicity—Chronic

No confirmed cases of chronic effects of overexposure by inhalation to xylene. Repeated or prolonged skin contact has a defatting action, causing drying, fissuring, and dermatitis.

Medical Treatment[40,49]

Immediate

Inhalation: Remove from exposure. Resuscitate and administer oxygen as needed.

Ingestion: *DO NOT* induce vomiting. Careful gastric lavage.

Skin Contact: Wash with soap and water after removing contaminated clothing.

Eye Contact: Flush with water.

Follow-up

Inhalation: Treat symptomatically.

December 1980

Xylene *continued*

Ingestion: Treat symptomatically. Observe for any signs of pneumonitis from aspirated xylene.

Skin Contact: Treat symptomatically. Emollients may be helpful.

Eye Contact: None.

Comments or Discussion

NIOSH Criteria Document "Xylene" (May, 1975) available.

Biological Monitoring

Breath analysis for xylene exposures is feasible but has not been reported.

m-Methylhippuric acid concentration in urine has been used as an index of exposure to xylene. Since at least 75% of technical xylene consists of the m-isomer, this metabolite predominates in urine and its concentration appears to be well correlated with the atmospheric xylene level and the total dose of xylene absorbed by an individual.[4a]

APPENDIX B

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APPENDIX C

HAZARDS OF MEDICAL OXYGEN IN PETROLEUM-RICH ENVIRONMENTS

Workshop discussions resulted in a difference in opinion between practicing physicians and hazard assessment/fire fighters with respect to the hazards of using medical oxygen around oil-contaminated patients, clothing, and equipment. Consensus opinion was that oxygen administration should **NEVER** be withheld from a patient because of the presence of oil, but that it is necessary to remove excess oil from contaminated patients. Because medical facilities may be handling large numbers of oil-contaminated individuals, the following information is offered to aid in dealing with the combination of crude oil and medical oxygen treatment.

The strictures against using oil in and around oxygen equipment are well known and generally followed. However, medical facilities and other operations using gaseous or liquid oxygen may be forced to operate on patients or in areas heavily contaminated with crude oil. Increasing the oxygen content of atmospheric air results in four major changes with respect to fire hazard. The energy needed to start a fire/explosion decreases to the point where small sparks of static electricity or the rapid opening of an oxygen cylinder valve is sufficient to ignite the oxygen/oil mixture at 100% oxygen. The rate at which flames spread after ignition increases to the extent of explosion at high oxygen concentrations. The temperature needed for spontaneous ignition decreases significantly. The range of fuel-to-oxygen mixtures that are flammable increases and the maximum temperature of the fire also increases.

The National Fire Protection Association (NFPA) defines an oxygen-rich atmosphere as containing 23.5% or more by volume. This includes the oxygen mixture often used for respiratory support. The use of oxygen for respiratory support or sustained breathing operations does not result in a generalized oxygen-rich atmosphere. However, increasing caution in the immediate vicinity of the oxygen equipment is necessary.

Oxygen equipment must be kept scrupulously free of oil. Individuals working with oxygen equipment must remain oil-free. This requires the removal of crude oil contamination and other petroleum-based materials.

Rapid opening of cylinder valves may bring combustible materials downstream of the valve in contact with hot oxygen. Open valves slowly.

Oxygen-rich atmospheres exist in the immediate vicinity of all oxygen administration equipment, oxygen tents, incubators, and similar equipment. Oxygen-rich atmospheres exist at the site of intentional release of gases from such equipment (within 1 ft of a nasal cannula or face mask, oxygen tent openings, the venting portion of a ventilator) and in the vicinity of the discharge ports of liquid oxygen equipment.

Normal static electrical sparks will **NOT** constitute an ignition hazard during normal operations in the absence of ether, alcohols, acetone, oils, greases, or lotions. To minimize hazard, oil-contaminated patients should be wiped free of readily removed oil.

Waterless hand cleaners and other petroleum-based materials should also be removed from patients prior to administration of oxygen.

Human skin is difficult to ignite in low pressure oxygen. However, it will burn readily in the presence of grease that can act as an ignition source.

Some relevant examples of accidents from the NFPA manual are instructive.

> A pressure regulator that had been in use for some time was disconnected from an oxygen cylinder supplying an incubator and reconnected to a new one. When the valve was opened, the regulator components ignited. The infant was killed and five others were injured in the flash fire.
>
> A gunsmith with greasy hands loosened the fittings on a welding oxygen cylinder with a greasy wrench. A spark ignited the grease, burning the gunsmith and resulting in $10,000 in damage.

APPENDIX D

APPROXIMATE SAFE STAND-OFF DISTANCES FOR PETROCHEMICAL EQUIPMENT

The following table represents an estimate of stand-off distances at which minimal damage to equipment or personnel will occur. This table is currently an estimate based on the personal experience of individuals who have been involved in fighting fires and controlling hazards within oil refineries. It should be noted that this experience is based on dealing with nonmilitary refinery and oil field accidents and explosions. If the initiating event leading to explosion and fire is more catastrophic (artillery fire as opposed to valve failure or pipe rupture), the ensuing fire and explosion may be more violent. Thus the table represents a guide to those in the vicinity of oil production or refining equipment. Those calculations are crude and the actual stand-off distance for propane spheres may have to be increased by a factor of at least two.

EQUIPMENT ITEM	STAND OFF DISTANCE	COMMENTS
OIL WELL HEADS AND REFINERY EQUIPMENT NOT LISTED BELOW	200 feet	Based on blast and shrapnel effects. Heat is a major factor in ability to approach fires.
PETROLEUM STORAGE TANKS	500 to 1000 feet	Petroleum storage tanks often have water in the bottom of the tank. When on fire, the contents will transfer heat to this water causing it to boil and expel the tank contents with considerable speed.
PROPANE SPHERES	1500 to 3000 feet	Blast overpressure and schrapnel are the determining factors. Note that fireball brightness can cause eye damage.

APPENDIX E

MILITARY ISSUE GAS MASK EFFECTIVENESS

During the workshop several individuals had expressed concern that general military issue gas masks might be ineffective in protecting against exposure to hydrogen sulfide and/or sulfur dioxide. These concerns were based on experience with commercially available respirators and a lack of information on the equipment issued to coalition forces. Further investigation into the effectiveness of military issue masks revealed the following information which positively supports their efficacy (information provided by U.S. Army sources).

Tests of the C2 canister as used with the M40 mask with a challenge concentration of 5000 ppm of hydrogen sulfide at a flowrate of 32 liters per minute provided about one hour of protection. The M13A2 filter element as used with the M17 mask provided about one-half hour of protection under very dry conditions (Benton, 1991).

Performance of the C2 canister in filtering sulfur dioxide with a challenge concentration of 700 ppm at a flowrate of 30 liters per minute maintained protective ability for about an hour. Filtering ability increased with increasing humidity (Harrison and Poirier, 1983). The M13A2 filter element while not tested under the same conditions, should afford equal protection based on its relative performance in removing other acid gases.

Because workshop participants were working under the assumption of gas mask ineffectiveness, self-contained breathing apparatus (SCBA) and oxygen breathing apparatus (OBA) were proposed as alternatives. These two measures are still effective alternatives to gas masks with the caveat that the OBA would be of limited use because of the problems of canister life and surface temperatures of canisters which may exceed the flash point of materials in the area.

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APPENDIX F

CONTACTS FOR HEALTH ISSUES FOR CRUDE OIL AND PYROLYSIS PRODUCTS

I. Key U.S. Government Contacts

The following individuals have been identified as the key contacts within their respective departments/agencies for issues dealing with the oil and smoke problems in the Persian Gulf.

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APPENDIX G

SUMMARY OF TOXICOLOGICAL DATA PERTAINING TO PETROLEUM PRODUCTS AND CRUDE OIL CONSTITUENTS

TABLE OF CONTENTS

CHEMICAL: ACETALDEHYDE

Existing Regulations, Guidelines, and Standards:

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Rat	Inhalation	400 ppm (728 mg/m^3)	4 weeks (6 hr./day; 5 days/wk.)	Slight-to-moderate degeneration of nasal olfactory epithelium; some nasal epithelial hyperplasia and metaplasia; disarrangement of nasal epithelial cells.	Appleman et al., 1982
		1000 ppm (1,820 mg/m^3)		Slight degenerative, hyperplastic, and metaplastic changes in nasal, larynageal, and tracheal epithelium; growth retardation; increased urine production in males in two middle dosage groups.	
		2,200 ppm (4,004 mg/m^3)			
		5,000 ppm (9,100 mg/m^3)		Severe degenerative hyperplastic and metaplastic changes of nasal, laryngeal, and tracheal epithelium; severe growth retardation; elevated blood neutrophil and reduced lymphocyte count; reduced production of high density urine; increased lung weights.	

CHEMICAL: ACETALDEHYDE CONTINUES....
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Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Rat	Inhalation	750 ppm (1,365 mg/m^3)	Up to 28 mos. (6 hr./day; 5 days/wk.)	Growth retardation; degeneration, hyperplasia, metaplasia, and adenocarcinomas of nasal olfactory epithelium.	Woutersen et al., 1986
		1,500 ppm (2,730 mg/m^3)		Growth retardation; degeneration, hyperplasia, metaplasia, and adenocarcinomas of nasal olfactory epithelium; squamous metaplasia, accompanied by slight to severe keratinization and squamous cell carcinomas of the respiratory epithelium.	
		3,000 ppm (5,460 mg/m^3)		Growth retardation; increased mortality (requiring reducing conc. to 1,000 ppm over time); degeneration, hyperplasia, metaplasia, and adenocarcinomas of nasal olfactory epithelium; squamous metaplasia, accompanied by slight to severe keratinization and squamous cell carcinomas of the respiratory epithelium.	

CHEMICAL: ACETALDEHYDE CONTINUES....
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Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Rat	Inhalation	750 ppm (1,365 mg/m^3)	52 weeks (6 hr./day; 5 days/wk.)	Thinning of olfactory epithelium; focal basal cell hyperplasia; in some animals, focal swelling in nasal cavity, suggestive of a neoplastic process at 26 weeks, progressing to adenocarcinomas during a 26-52 week recovery period; normal olfactory epithelium still absent after 52-week post-exposure recovery period.	Woutersen & Feron, 1987
		1,500 ppm (2,730 mg/m^3)		In addition to effects seen at low exposure concentration, hyperplasia and metaplasia of respiratory epithelium, frequently accompanied by keratinization and rhinitis.	
		3,000 (later reduced to 1,500 ppm) (5,460 mg/m^3)		In addition to all effects seen at 1,500 ppm, no regeneration of respiratory epithelium was observed.	

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Hamster	Inhalation	390 ppm (710 mg/m^3)	90 days (6 hr./day; 5 days/wk.)	None	Kruysse et al., 1975
		1340 ppm (2,439 mg/m^3)		Significantly increased kidney weights in males; small areas of stratified epithelium in trachea.	
		4,560 ppm (8,299 mg/m^3)		Significantly reduced body weight; significantly increased relative heart, kidney, brain, testicle and lung weights; necrosis, inflammatory changes, hyperplasia, and metaplasia of epithelium in nasal cavity, larynx, trachea, and bronchi.	

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Hamster	Inhalation	1,500 ppm (2,730 mg/m^3)	52 weeks (7 hr./day; 5 days/wk.)	Epithelial hyperplasia and metaplasia; inflammation in nasal cavity & trachea.	Feron, 1979
Hamster	Inhalation	2,500 ppm (1st 9 weeks) 2,250 ppm (weeks 11-20) 200 ppm (weeks 21-29) 1,800 ppm (weeks 30-44) 1,650 ppm (weeks 45-52)	52 weeks (7 hr./day; 5 days/wk.)	Rhinitis; hyperplasia and metaplasia in nasal, laryngeal, and tracheal epithelium; nasal and laryngeal carcinomas.	Feron et al. 1982

Appleman, L.M., R. A. Woutersen, and V. J Feron. 1982. Inhalation toxicity of acetaldehyde in rats. I. Acute and subacute studies. *Toxicology* 23: 293-307.

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REVIEW ARTICLES AVAILABLE

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CHEMICAL: ACROLEIN

Existing Regulations, Guidelines, and Standards:

OSHA Permissible Exposure Limit (PEL):	0.1 ppm (0.25 mg/m^3)
ACGIH TLV TWA:	0.1 ppm (0.25 mg/m^3)
STEL:	0.3 ppm (0.8 mg/m^3)
U.S. EPA (OWRS) Ambient Water Quality Criteria:	0.32 mg/l
U. S. EPA Carcinogenicity Classification:	Group C (possible human carcinogen)

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Rat	Inhalation	0.4 ppm (0.82 mg/m^3)	62 days (6hr./day; 5 days/wk.)	Restrictive lung lesion.	Kutzman, 1981; Kutzman et al., 1985; Costa et al., 1986
		1.4 ppm (3.21 mg/m^3)		More pronounced decrement in pulmonary function.	
		4.0 ppm (9.17 mg/m^3)		Obstructive lung disease; high mortality in males (32/57), but none in females; significantly reduced body weight gain; bronchiolar epithelial necrosis and sloughing; bronchiolar edema; focal pulmonary edema; some instance of edema of trachea and peribronchial lymph nodes, and acute rhinitis of upper respiratory tract; increased lung weight, connective tissue, elastin, and hydroxyproline.	

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Rat	Inhalation	0.4 ppm (0.82 mg/m^3)	13 Weeks (6 hr./day; 5 days/wk.)	None	Feron et al., 1978
		1.4 ppm (3.21 mg/m^3)		Hyperactivity; decreased mean body weight and brain weight; squamous metaplasia and neutrophilic infiltration in nasal cavities.	
		4.9 ppm (11.23 mg/m^3)		Increased mortality; depressed body weight; treatment-related increase in relative lung weight; hyperplasia & metaplasia of tracheal epithelium; necrotizing rhinitis & keratinization of squamous nasal epithelium; lung lesions characterized by nyperplasia & metaplasia of bronchial & bronchiolar epithelium, hemorrhaging, perivascular & alveolar edema, bronchiolitis, bronchopneumonia, & macrophage aggregation.	

CHEMICAL: ACROLEIN CONTINUES....
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Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Hamster	Inhalation	0.4 ppm (0.82 mg/m^3)	13 Weeks (6 hr./day; 5 days/wk.)	None	Feron et al., 1978
		1.4 ppm (3.21 mg/m^3)		Hyperactivity; slight inflammation in nasal cavity.	
		4.9 ppm (11.23 mg/m^3)		Depressed body weight; treatment-related increase in relative lung weight; hyperplasia & metaplasia of tracheal epithelium; necrotizing rhinitis and keratinization of nasal squamous epithelium.	
Rabbit	Inhalation	0.4 ppm (0.82 mg/m^3)	13 Weeks (6 hr./day; 5 days/wk.)	None	Feron et al., 1978
		1.4 ppm (3.21 mg/m^3)		Nasal membrane irritation, sneezing.	

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
		4.9 ppm (11.23 mg/m^3)		Depressed body weight; treatment-related increase in relative lung weight; hyperplasia & metaplasia of tracheal epithelium; necrotizing rhinitis; keratinization of nasal squamous epithelium; hyperplasia & metaplasia of bronchial & bronchiolar epithelium; lung hemorrhaging; perivascular and alveolar edema: bronchiolitis; bronchopneumonia; macrophage aggregation in lung.	
Rat Guinea Pig Dog Monkey	Inhalation	0.22 ppm (0.50 mg/m^3)	90 Days (continuous)	Decreased body weight; clinical symptoms; histological changes in lung & liver.	Lyon et al., 1970
Hamster	Inhalation	4.0 ppm (9.17 mg/m^3)	1 Year	Reduced body weight; histological changes in nasal cavity; increased relative lung weight.	Feron and Kruysse, 1977

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U.S. EPA. 1986. Health Assessment Document for Acrolein. U. S. Environmental Protection Agency, Office of Research and Development, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Research Triangle Park, NC. EPA-600/8-86-014A.

CHEMICAL: AMMONIA

Existing Regulations, Guidelines, and Standards:

OSHA Permissible Exposure Limit:	**50 ppm**
ACGIH TLV/TWA:	**25 ppm**
STEL:	**35 ppm**
NIOSH 5-min. Ceiling REL:	**50 ppm**
U.S. EPA Reference Concentration (RfC):	**0.1 mg/m^3**

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Human	Inhalation	9.2 PPM (T.W.A.) (6.4 mg/m3)	12.2 Year (mean Exposure)	None (in pulmonary function or subjective symptomatology compared with controls).	Holness et at., 1989
Human	Inhalation		6 Weeks (5 days/wk)		Ferguson et al., 1977
		25 ppm	2 hr./day	None	
		50 ppm	4 hr./day	Transient irritation of nose and throat.	
		50 ppm	6 hr./day	Transient irritation of nose and throat.	
		100 ppm	6 hr./day	Transient irritation of nose and throat (No effects on respiratory, cardiac, or neurological parameters).	
Human	Inhalation	110 ppm (76 mg/m3)	2 hr. (two exposures one week apart)	Throat irritation.	Verberk, 1977
		140 ppm (97 mg/m3)		Subjects found concentration to be intolerable.	

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Human	Inhalation	21 mg/m^3	10 min.	Faint sensory irritation.	MacEwen et al., 1970
		35 mg/m^3	10 min.	Moderate sensory irritation.	
Human	Inhalation	500 ppm (348 mg/m^3)	30 min.	Nasal & throat irritation; increase in respiratory minute volume.	Silverman et al., 1949
Human	Inhalation	0.36 mg/m^3	Continuous	Odor threshold.	Carson et al., 1981
Human	Oral	34 mg/l (in drinking water)	Continuous	Taste threshold.	Campbell et al., 1958
Rat	Inhalation	25 ppm (17.4 mg/m^3) 50ppm (34.8 mg/m^3) 150 ppm (104.4 mg/m^3) 250 ppm (174 mg/m^3)	5-7 Weeks (continuously)	Increased severity of rhinitis, otitis, tracheitis, and pneumonia in animals innoculated with *M. pulmonalis* at all concentrations. Concentration-dependent increase in severity of respiratory lesions. At 250 ppm, thickening and hyperplasia of nasal epithelium in rats not exposed to *M. pulmonalis*.	Broderson et al., 1976
Mouse	Inhalation	303 ppm		RD_{50} value (exposure concentration to evoke a 50% decrease in respiratory rate).	Kane et al., 1979
Rat	Inhalation	3, 6. 5, 10, 20, 45, & 90 ppm	8 min.	Cessation of tracheal ciliary activity in concentration-dependent manner (7-8 minutes at 3 ppm vs. 5 seconds at 90 ppm).	Dahlman, 1956

CHEMICAL: AMMONIA CONTINUES....
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Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Rat	Inhalation	200 (± 50) ppm	4, 8, or 12 days	Hyperplasia of tracheal epithelium (time- & concentration-dependent); loss of cilia and change in type and thickness of epithelium over time at 200 ppm.	Gambel and Clough, 1976
		435 ppm (± 135)	7 days	Acute inflammatory reaction in trachea, with infiltration of neutrophils, large mononucleated cells, monocytes, and immature fibroblasts; evidence of necrotic changes at luminal surface.	
Mouse	Inhalation	20 ppm (13.9 mg/m^3)	6 Weeks	Time- & concentration-dependent effects: Darkening/reddening, edema, congestion, and hemorrhage of lungs after 6 wks exposure (but not after 1, 2, 3, or 4 wks).	Andersen et al., 1964
Guinea Pig	Inhalation	50 ppm (35 mg/m^3)	6 Weeks	Same as mice; also, grossly enlarged & congested spleens; congested livers & lungs; pulmonary edema.	Andersen et al., 1964
Chicken	Inhalation	20 ppm (13.9 mg.m^3)	12 Weeks	Same as mice.	Andersen et al., 1964
		200 ppm (139 mg/m^3)	3 Weeks	Same as 20 ppm chickens; also, liver congestion & slight clouding of cornea.	
		1000 ppm (695 mg/m^3)	2 Weeks	Same as 200 ppm chickens; also, congestion of spleen and corneal opacities (after 8 days).	

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Guinea Pig	Inhalation	170 ppm (118 mg/m^3)	18 Weeks	No effects after 12 wks; mild changes in spleen, kidney, suprarenal glands, and livers at 18 wks.	Weatherby, 1952
Mouse	Inhalation	305 ppm (212 mg/m^3)	5 days (6hr./day)	Nasal lesions.	Buckley et al., 1984
Rat	Inhalation	40 mg/m^3	90-114 days (continuous)	No effect.	Coon et al., 1970
		127 mg/m^3		No effect.	
		262 mg/m^3		Nasal discharge; non-specific circulatory & degenerative changes in lungs and kidneys (difficult to directly relate to ammonia exposure).	
		455 mg/m^3 and 470 mg/m^3	Mortality (90-98%)		
Pig (Duroc)	Inhalation	10, 50, 100, or 150 ppm		Decrease in food intake and body weight gain; nasal, lacrimal, and mouth secretions at higher concentrations.	Stombaugh et al., 1969
Rat, Guinea Pig, Rabbit, Dog, Monkey	Inhalation	155 mg/m^3	6 Weeks (5 days/wk; 8hr./day)	None	Coon et al., 1970
		770 mg/m^3		Lung irritation in rats and guinea pigs; occular and nasal irritation in rabbits and dogs.	

AMMONIA REFERENCES

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CHEMICAL: BENZENE

Existing Regulations, Guidelines, and Standards:

OSHA Permissible Exposure Limit (PEL):

8-hr.TWA:	1 ppm
STEL:	5 ppm
10-min. maximum ceiling limit:	50 ppm

U.S. EPA (ODW) Maximum Contaminant Level: 0.005 mg/l
U. S. EPA Carcinogenicity Classification: Group A (Human Carcinogen)

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Human	Inhalation		Occupational	Leukemia	Ott et al., 1978; Rinsky et al., 1981; Wong et al., 1983
	Oral		Occupational	Leukemia	
Mouse	Inhalation	10 ppm (31.9 mg/m^3)	178 days (6 hr./day; 5 days/wk.)	Reduced ability of marrow progenitor cells to form colonies; significantly reduced numbers of circulating erythrocytes and lymphocytes.	Baarson et al., 1984
Mouse	Inhalation	301 ppm	115 days	Marked increase in number of cells in marrow and thymus; progressive decline in mitogen-induced B- and T-cell proliferation.	Rozen et al., 1985

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Mouse	Inhalation	5 ppm (15.9 mg/m^3)	Days 6-15 of gestation	Non-significant decrease in early nucleated red cell numbers.	Keller and Snyder, 1988
		10 ppm (31.9 mg/m^3)		Greater decrease (still non-significant) in early nucleated red cell numbers.	
		20 ppm (63.9 mg/m^3)		Decrease in early nucleated marrow red cells, and increases in splenic blasts and dividing and nondividing granulocytes in 6-wk old adult offspring (similar pattern observed earlier in 2-day neonates upon examination of hepatic hematopoeitic precursor cell types).	
Mouse	Inhalation	10 ppm (31.9 mg/m^3)	2 Weeks	Males previously exposed to the same concentration in utero exhibited decreases in GM-CFU-C more severe than those in control group (not pre-exposed in utero).	Keller and Synder, 1986
Mouse	Inhalation	1 ppm	13 Weeks (6 hr./day; 5 days/wk.)	None	Ward et al., 1985
		10 ppm		None	

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
		30 ppm		None	
		300 ppm		Bilateral testicular atrophy; abnormal sperm; severe decrease in spermatozoa in epididymal ducts; lesions of bone marrow, mesenteric and mandibular lymph nodes, thymus, spleen, and ovaries; decrease in myeloid/erythroid ratios; anemia; thrombocytpenia; leucopenia; decreased hematocrit and total hemoglobin. (Lesions more severe in males.)	
Rat	Inhalation	1 ppm	13 Weeks (6 hr./day; 5 days/wk.)	None	Ward et al., 1985
		10 ppm		None	
		30 ppm		None	
		300 ppm		Decreased lymphocyte count; increase in neutrophils; slightly decreased femoral marrow cellularity (as early as 7 days of exposure).	

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Mouse	Inhalation	10 ppm	6 days (6 hr./day)	Depressed peripheral blood B- and T-lymphocyte counts; marrow B-lymphocyte colony-forming ability reduced 70% (compared with controls); reduced PHA-stimulated blastogenesis in splenic T-lymphocytes.	Rozen et al., 1984
		31 ppm		Depressed peripheral blood B- and T-lymphocyte counts; marrow B-lymphocyte colony-forming ability reduced 70% (compared with controls); reduced PHA-stimulated blastogenesis in splenic T-lymphocytes.	

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
		100 ppm		Depressed peripheral blood B-andT-lymphocyte counts; depressed erythrocyte count; marrow B-lymphocyte colony forming ability reduced 70% (compared with controls); decreased femoral B-lymphocyte numbers; reduced PHA-stimulated blastogenesis in splenic T-lymphocytes; depressed splenic T-lymphocyte count.	
		301 ppm		Depressed peripheral blood B- and T-lymphocyte counts; depressed erythrocyte count; marrow B-lymphocyte colony forming ability reduced 70% (compared with controls); decreased femoral B-lymphocyte numbers; reduced splenic B-lymphocyte colony forming ability; reduced PHA-stimulated blastogenesis in splenic T-lymphocytes; depressed splenic T-lymphocyte count.	

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Mouse	Inhalation	1 ppm	10 days (continuous)	None	Toft et al., 1982
		10 ppm		None	
		21 ppm 50 ppm 95 ppm		Depression of colony forming ability of marrow cells from the tibia; decreases in number of nucleated cells in tibia; increase in micronuclei in polychromatic erythrocytes. (All effects severe after just 96 hr. of exposure.)	
Mouse	Inhalation	Second group of mice: 1, 10, 21, 50, 95 ppm	8 hr./day	No effects at any exposure concentration after 96 hr.	Toft et al., 1982

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Rat	Inhalation	10 ppm	Days 6-15 of gestation (7 hr./day)	None	Kuna and Kapp, 1981
		50 ppm		Decreased fetal body weight.	
		500 ppm		Decreased fetal body weight; dilated lateral and third ventricles of the brain in some fetuses; maternal toxicity.	
Rat	Inhalation	10 ppm	Days 6-15 of gestation (6 hr./day)	None	Coate et al., 1984
		40 ppm		None	
		100 ppm		Reduced body weight.	
Mouse	Inhalation	157 ppm	Days 6-15 of gestation (continuous)	None	Ungvary and Tatrai, 1985
		313 ppm		None	

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Rabbit	Inhalation	157 ppm	Days 7-20 of gestation (continuous)	Reduced fetal body weight (no maternal toxicity).	Ungvary and Tatrai, 1985
		313 ppm (1,000 mg/m^3)		Reduced fetal body weight (no maternal toxicity).	
Rat	Inhalation	313 ppm (1,000 mg/m^3)	Days 9-14 of gestation (continuous)	Reduced fetal body weight.	Hudak and Ungvary, 1978
Mouse	Inhalation	500 ppm	Days 6-15 of gestation (continuous)	Decreased fetal body weight; minor skeletal variations, but no malformations.	Murray et al., 1979
Rabbit	Inhalation	500 ppm	Days 6-18 of gestation (continuous)	Minor skeletal variations, but no malformations.	Murray et al., 1979
Rat	Inhalation	100 ppm	Days 6-15 of gestation (6 hr./day)	None	Green et al., 1978
		300 ppm		None	
		2200 ppm		Decreased fetal body weight; no other fetotoxic or teratogenic effects.	

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CHEMICAL: CARBON MONOXIDE

Existing Regulations, Guidelines, and Standards:

U.S. EPA Standard — 9 ppm (10 mg/m^3): 8 hr. exposure
35 ppm (40 mg/m^3): 1 hr. exposure

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Human	Inhalation	4,300 ppm	3-5 hrs.	Elevated (23%) COHb; increased transcapillary permeability to ^{131}I-labled human serum albumin.	Parving, 1972
Human	Inhalation	5,000 ppm	2-3 min.	Decreased inspiratory capacity and total lung capacity.	Chevalier, et al., 1966
Human	Inhalation	50 ppm	4 hr.	COHb elevated to 2.17%	Brinkhouse, 1977
		100 ppm		COHb elevated to 4.15%; mean treadmill exercise duration (at 85% maximum heart rate) decreased.	
Human	Inhalation	50 ppm	20 min.	Exercise duration (treadmill to exhaustion) decreased in non-smokers (COHb 2.5%); change in respiratory pattern in both smokers (COHb = 4.1%) and non-smokers (COHb = 2.5%).	Drinkwater, et al, 1974

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Human	Inhalation	100 ppm	1 hr.	Mean treadmill exercise time until exhaustion significantly decreased (COHb level = 3.95%)	Aronow and Cassidy, 1975.
Human	Inhalation	50 ppm	4 hr.	Stroke volume (during exercise @ 35% VO_2 max) decreased with higher ambient temperature; heart rate increased, w/o change in cardiac output or stroke volume. (COHb level = 4.5-6.8%).	Gliner et al., 1975
Human	Inhalation	30 ppm	5 hr.	Decreased VO_2 max; increased V_E and HR during treadmill exercise (to exhaustion).	Klein et al., 1980
Human	Inhalation	500 ppm (0.05%)	5 min.	Increased HR; decreased VO_2 max (15.4% COHb)	Pirnay et al., 1971
Human	Inhalation	225 ppm	1 hr.	Decreased VO_2 max; increased H during submaximal exercise.	Pirnay et al., 1971
Human	Inhalation	6-12% COHb	3.5 hr.	Decreased nerve conduction velocity.	Groll-Knapp et al., 1978

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Human	Inhalation	3.0-17.6% COHb	2 hr.	Decreased nerve conduction velocity at all COHb concentrations.	Groll-Knapp et al., 1972
Human (pediatric)	Inhalation	15% COHb	Various	Asymptomatic	Crocker and Walker, 1985
		16.7% COHb		Nausea; headache	
		19.8% COHb		Vomiting	
		18.6% COHb		Lethargy	
		24.5% COHb		Visual symptoms; syncope	
		36.9% COHb		Seizures	
Human (13-wks. old)	Inhalation	60% COHb	Unknown	Convulsions; unconsciousness (recovery of minor neurologic deficits w/in 6 weeks)	Venning et al., 1982

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Human (pediatric)	Inhalation	(est. 27-37% COHb)	Unknown	Persistent symptoms after hyperbaric O_2 and normobaric O_2 are: anxiety or emotional instability; memory impairment; spatial/temporal disorganization; perceptual problems; auditory and visual memory impairment.	Klees et al., 1985
Dog, Rabbit, Rat	Inhalation	50 ppm	3 mos. (continuous)	None (normal EKG & HR)	Musselman et al., 1959
Dog	Inhalation	50-100 ppm	6 Wks.	AbnormalEKG; heart dilation; myocardial thinning; some scarring & degeneration in heart muscle (COHb 2.6-12%)	Preziosi et al., 1970
Monkey	Inhalation	100 ppm	24 Wks. (23 hr./day)	Abnormal EKG; increased sensitivity to fibrillation voltage.	DeBias et al., 1973
Rat	Inhalation	100 ppm	46 days	9.2% COHb	Penny et al., 1974 a,b
		200 ppm	30 days	15.8 COHb; cardiac hypertrophy	
		500 ppm	20-42 days	41.12% COHb; hypertrophy of both left & right ventricles; increased LDH levels.	

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Rat	Inhalation	50 ppm	3 mos.	1.8% COHb; no signs of toxicity.	Musselman et al., 1959
Rabbit				Increased hemoglobin, hematocrit, & red blood cells; 3.2% COHb; no signs of toxicity.	
Dog				Increased hemoglobin, hematocrit, & RBC's; 7.3% COHb; no EKG changes; no signs of toxicity.	
Monkey (cholesterol-fed)	Inhalation	100-300 ppm	7 mos. (4 hr./day; 5 days/wk.)	Increased coronery atherosclerosis; 9-26% COHb	Webster et al., 1970
Monkey	Inhalation	250 ppm	2 weeks (continuously)	Coronary artery subendothelial edema; gaps between endothelial cells; infiltration cells containing lipid droplets; 20.6% COHb.	Thomsen, 1974
Monkey	Inhalation	50-500 ppm	14 mos (12 hr./day)	No evidence of aortic or coronary atherosclerosis in high- or low-cholesterol diets.	Malinow et al., 1976

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Cat	Inhalation	7.5-42.0% COHb	2 hr.	Decreased ERG β-wave amplitude (dose-related effect beginning at 7.5% COHb).	Ingenito and Durlacher, 1979
Rat	Inhalation	12.2-54.9% COHb	4 hr.	Decreased performance on conditioned avoidance test at 12.2% COHb; decrements in reflex, grasping, and conditioned avoidance seen at higher COHb levels.	Mullin and Krivanek, 1982
Rabbit	Inhalation	90 ppm	Mating to day before parturition	Maternal COHb 8-9% increase in neonatal mortality; decrease in birth weight.	Astrup et al., 1972
		180 ppm		Maternal COHb 16-18%; 35% mortality of neonates; decreased birth weight; increased incidence of malformations.	
Mouse	Inhalation	250 ppm	Days 6-15 of gestation (7 or 24 hr./day)	7 hr./day: increase in number of resorptions. 24 hr./day: decrease in fetal body weight and crown-rump length.	Schwetz et al., 1979
Rabbit	Inhalation	250 ppm	Days 6-18 of gestation (7 or 24 hr./day)	Increase in body weight and crown rump length at 7 hr./day exposure.	Schwetz et al., 1979

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Rat	Inhalation	30 ppm	Days 3-20 of gestation	4.8% maternal COHb; decrease in successful pregnancies.	Garvey and Longo, 1978
		90 ppm		8.8% maternal COHb; decrease in successful pregnancies; 14% increase in fetal brain weight; decreases in fetal lung weight (24 %) and brain seratonin concentration.	
Rat	Inhalation	150 ppm	Throughout gestation	12.2-14% peak maternal COHb levels; decreased birth & pre-weaning weights; decreased response of offspring in behavioral open field test; increased rate of habituation in offspring.	Fechter and Annau, 1976
Rat	Inhalation	COHb 15.6%	Throughout gestation	Decreased acquisition and retention of 2-way active avoidance in offspring.	Mactutus & Fechter, 1984

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CHEMICAL: CHLORINE

Existing Regulations, Guidelines, and Standards:

OSHA TWA = 0.5 ppm
STEL = 1 ppm

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Human	Inhalation	0.5 ppm (1.4 mg/m^3)	2 days (8 hr./day)	None	Rottman et al., 1983
		1 ppm (2.9 mg/m^3)		Throat irritation; itchiness of eyes; decrease in performance on pulmonary function tests.	
Monkey (Rhesus)		0.1 ppm (0.3 mg/m^3)	1 year (6 hr./day; 5 days/wk.)	None	Klonne et al., 1987
		0.5 ppm (1.4 mg/m^3)		None	
		2.3 ppm (6.7 mg/m^3)		Mild trachael lesions, consisting of focal epithelial hyperplasia with loss of cilia & decreased numbers of goblet cells.	
Rat	Inhalation	0.5 ppm (1.4 mg/m^3)	62 days (6 hr./day; 5 days/wk)	Some areas of focal erosion of epithelia and cilia loss in trachea; increase in pulmonary resistance to tidal airflow.	Kutzman, 1983

CHEMICAL: CHLORINE CONTINUES....
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Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
		1.5 ppm (4.3 mg/m^3)		Occasional signs of ocular and upper respiratory tract irritation; significant increase in lung collagen.	
Rat	Inhalation	5.0 ppm (14.5 mg/m^3)		Severe ocular and upper respiratory tract irritation; areas of focal erosion of epithelia and loss of cilia in trachea; increase in pulmonary resistance to tidal airflow (concentration-dependent); significant increase in lung collagen.	
Rat	Inhalation	0.5 ppm (1.4 mg/m^3) 1.5 ppm, (4.3 mg/m^3) 5.0 ppm (14.5 mg/m^3)	62 days (6 hr./day; 5 days/wk)	No effect on male or female reproductive parameters at any chlorine concentration	Kutzman, 1983
Rat	Inhalation	1 ppm (2.9 mg/m^3) 3 ppm (8.7 mg/m^3)	6 weeks (6 hr./day; 5 days/wk.)	Focal irritation around respiratory bronchioles & alveolar ducts. Focal irritation around respiratory bronchioles & alveolar ducts; increased hepatocellelar cytoplasmic vacuolization; significantly elevated alkaline phosphatase levels.	Barrow et al., 1979

CHEMICAL: CHLORINE CONTINUES...
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Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
		9 ppm (257.4 mg/m^3)		Marked inflammation throughout respiratory tract; necrotic lesions in nasal turbinates; increase in alveolar macrophages; hyperplasia & hypertrophy of bronchiolar and alveolar duct epithelia; increased hepatocellular cytoplasmic vacuolization; significantly elevated alkaline phosphatase, gamma-glutamyl transpeptidase, and SGPT levels; epithelial cell changes in the kidney proximal convoluted tubules	

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CHEMICAL: n-HEXANE

Existing Regulations, Guidelines, and Standards:

OSHA Permissible Exposure Limit: 500 ppm (1,800 mg/m^3)
ACGIH TLV/TWA: 50 ppm (180 mg/m^3)
NIOSH TWA: 100 ppm (360 mg/m^3)
15 min. ceiling: 510 ppm
U.S. EPA Inhalation Reference Concentration: (RfC): 0.2 mg/m^3

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Human	Inhalation	58 ppm (204 mg/m^3)	6.2 years avg. (1-12 yrs, actually)	Decrease in motor nerve conduction velocity (compared with controls); other neurophysiological signs of peripheral nueropathy.	Sanagi et al., 1980
	Inhalation	500-1,000 ppm (630-1,260 mg/m^3)	Occupational exposure (possible multi-chemical exposure)	Polyneuropathy, with subsequent development of muscular atrophy and paresthesia in the distal extremeties.	Yamada, 1967
	Inhalation	500-2,500 ppm commercial hexane (60-70% n-hexane)	Occupational exposure (8-14 hr./day) (possible multi-chemical exposure)	Polyneuropathy. Initially, numbness of distal portion of extremeties, followed by abnormal electromyography, decreased conduction velocity, giant axonal degeneration, and paranodal & internodal swelling of axons.	Yamamura, 1969; Inoue et al. 1970; Iida, 1982; Sobue et al., 1978
Mouse	Inhalation	500 ppm (1,762 mg/m^3) 1,000 ppm (3,525 mg/m^3)	13 weeks (6 hr./day; 5 days/week)	None. (Not examined for neuropathological alterations.) Mild lesions of nasal turbinates (not examined for neuropathological alterations).	Dunnick et al., 1989

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
		4,000 ppm (14,099 mg/m^3)		Histopathologic changes (mild inflammatory, erosive, & regenerative lesions) in olfactory and respiratory epithelium.	
		10,000 ppm (35,247 mg/m^3)		Modest paranodal axonal swelling in tibial nerve; histopathologic changes (more severe inflammatory, erosive, & regenerative lesions) in olfactory and respiratory epithelium.	
		1,000 ppm (3,525 mg/m^3)	13 weeks (22 hr./day; 5 days/wk.)	Depression of final body weight (relative to controls); histopathologic changes (mild inflammatory, erosive, & regenerative lesions) in olfactory & respiratory epithelium; modest paranodal swelling in tibial nerve.	
Mouse	Inhalation	100 ppm (353 mg/m^3)	1 year (24 hr./day; 6 days/wk.) (commercial grade hexane; 65-70% n-hexane)	None	Miyagaki, 1967

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
		250 ppm (881 mg/m^3)		Electromyographic changes, increasing in incidence and severity with increasing concentrations; abnormal posture and muscle atrophy in dose-dependent manner.	
		500 ppm (1,762 mg/m^3)			
		1,000 ppm (3,525 mg/m^3)			
		2,000 ppm (7,050 mg/m^3)			
Rat	Inhalation	6, 26, & 129 ppm (18, 95 & 444 mg/m^3)	26 weeks (6 hr./day; 5 days/wk.)	No evidence of neuropathological changes associated with nervous system degeneration.	Bio/Dynamics, Inc., 1978
Rat	Inhalation	126 ppm (444 mg/m^3)	6 months (22 hr./day; 7 days/wk.)	None	IRDC, 1981
		502 ppm (1,769 mg/m^3)		Evidence of neurotoxicity, including abnormal gait, axonal degeneration, and myelin vacuolization.	

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Rat	Inhalation	500 ppm (1,762 mg/m^3)	16 weeks (12 hr./day; 7 days/wk.)	Slight decreases in body weight gain and motor nerve conduction velocity.	Huang et al., 1989
		1,200 ppm (4,230 mg/m^3)		Significant decreases in body weight gain and motor nerve conduction velocity; degeneration of peripheral nerves (paranodal swellings and demyelination/remyelination of myelinated nerve fibers).	
		3,000 ppm (10, 574 mg/m^3)			
Rat	Inhalation	500 ppm (1,762 mg/m^3)	14-30 weeks (9 hr./day; 5 days/wk.)	Significantly decreased weight gain.	Frontali et al., 1981
		1,500 ppm (5,286 mg/m^3)		None	
		5,000 ppm (17, 624 mg//m^3)		Significantly decreased weight gain; giant axonal degeneration; paranodal and internodal swelling of axons.	
		2,500 ppm (8,812 mg/m^3)	14-30 weeks (10 hr./day; 6 days/wk.)	Giant axonal degeneration; paranodal and internodal swelling of axons.	

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Rabbit	Inhalation	3,000 ppm (10,574 mg/m^3)	24 weeks (8 hr./day; 5 dyas/wk.)	Clinical signs of ocular and upper respiratory tract irritation and respiratory difficulties (gasping, lung rales, mouth breathing) seen throughout the study.	Lungarella et al., 1984
Rat	Inhalation	500 ppm (1,762 mg/m^3)	9 weeks (22 hrs.day; 7 days/wk.)	Hind limb paralysis and related neuropathology (giant axonal swelling in both central & peripheral sites) after 9 weeks of exposure.	Altenkirch et al., 1982
		700 ppm (2,467) mg/m^3)		Same as 500 ppm.	
		700	40 weeks (8 hr./day; 7 days/wk.)	No neuropathies resulted from this discontinuous exposure regimen.	
Rat	Inhalation	1,000 ppm (3,525 mg/m^3)	61 days (18 hr./day; 7 days/wk.)	Testicular damage; total loss of germ cell line in some animals up to 14-mos. post-exposure.	Nylen et al., 1989
Rat	Inhalation	1,000 ppm (3,489 mg/m^3)	Days 8-16 of gestation (6 hr./day)	Depressed post natal growth for up to 3 wks. after birth; no teratogenic effects.	Bus et al., 1979

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Mouse	Oral (gavage)	7.92 g/kg/day	Days 6-15 of gestation	Reduction in fetal weight.	Marks et al., 1980
		9.9 g/kg/day		Reduction in fetal weight; no teratogenic effects, even at maternally toxic doses.	
Rat	Inhalation	200 ppm (705 mg/m^3)	Days 6-19 of gestation	None	Mast et al., 1987
		1,000 ppm (3,525 mg/m^3)		Significantly reduced fetal weight	
		5,000 (17,623 mg/m^3)		Significantly reduced maternal weight gain; significantly reduced fetal weight. No teratogenic or other developmental effects.	
Rat	Inhalation	100 or 400 ppm (352 or 1,410 mg/m^3)	Days 6-15 of gestation (6 hr./day)	No teratogenic effects.	Litton Bionetics, 1979, 19
Mouse	Inhalation	200 ppm (705 mg/m^3) 1,000 ppm (3,525 mg/m^3) 5,000 ppm (10,484 mg/m^3)	5 days	Negative dominant lethal study; no effects on sperm viability.	Mast et al., 1989[a]
Mouse	Inhalation	200 ppm (705 mg/m^3) 1,000 ppm (3,525 mg/m^3) 5,000 ppm (10,484 mg/m^3)	5 days	No effect on sperm morphology.	Mast et al., 1989[b]

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CHEMICAL: HYDROGEN SULFIDE

Existing Regulations, Guidelines, and Standards:

World Health Organization Standard: 0.1 ppm (0.15 mg/m^3): 24-hr. Exposure
U.S. EPA Reference Concentration: 0.0009 mg/m^3 for Lifetime Exposure

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Human	Inhalation	25 ppb (0.35 mg/m^3)		Odor threshold.	NIOSH, 1977
		3-5 ppm		Offensive odor.	
		Approximately 100 ppm		Loss of olfactory ability to detect odor.	
Human	Inhalation	>1,000 ppm	Single exposure (1-2 breaths)	Systemic intoxication.	Deng and Chang, 1987; Spolyar, 1951
Human	Inhalation	? (high levels)	Acute	Respiratory paralysis (w/consequent asphxyia).	Anon 1986; Milby, 1962; Haggard, 1925; Adelson e al 1966.
Human	Inhalation	500-1,000 ppm	Acute	Sudden fatique, headache, dizziness, intense anxiety, loss of olfactory function, nausea, abrupt loss of consciousness, disturbance of optic nerve, hypertension, insomnia , mental disturbances, pulmonary edema, coma, convulsions, and respiratory (followed by cardiac) arrest.	Burnett et al., 1977; Frank, 1986; Anon, 1986; Thoman, 1969

CHEMICAL: HYDROGEN SULFIDE CONTINUES....
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Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Human	Inhalation	250 ppm	Approximately 3 min.	Unconsciousness	McDonald and McIntosh, 1951
Human	Inhalation	10 ppm	6-7 hr	Inflammation of the cornea.	Frank, 1986; Milby, 1962
		50-200 ppm	Acute	Eye and mucous membrane irritation; lacrimation; loss of coronary reflex; changes in visual acuity and perception, progressing to inflammation and ulceration, with scarring of the cornea, in severe cases.	
Rat	Inhalation	10 ppm ($14\ mg/m^3$)	4 hours	None	Lopez etal., 1987
		200 ppm ($279\ mg/m^3$)		None	
		400 ppm ($557\ mg/m^3$)		Increased number of cells in post-exposure nasal lavage; increased protein and lactate dehydrogenase in both broncho-alveolar and nasal lavage fluid.	

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Rat	Inhalation	116 mg/m^3	4 hours	Mild perivascular edema.	Lopez et al., 1988a
		615 mg/m^3		Marked perivascular and alveolar edema; PMN, proteinaceous fluid, fibrin, and exfoliated cells in bronchioles; necrosis of bronchiolar cilated cells; hyperplasia of Type II alveolar cells.	
Rat	Inhalation	14 mg/m^3	4 hours	None	Lopez et al., 1988b
		280 mg/m^3		None	
		560 mg/m^3		Necrosis and exfoliation of respiratory and olfactory mucosal cells.	
Mouse	Inhalation	10.1 ppm (14.1 mg/m^3)	90 days (6 hr./day; 5 days/wk.)	Significant, but intermittent, reduction in body weight gain;	CIIT, 1983a
		30.5 ppm (42.5 mg/m^3)		Significant, but intermittent, reduction in body weight gain.	
		80 ppm (110 mg/m^3)		Significant, continuous reduction in body weight gain; inflammation of the nasal mucosa.	

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Rat	Inhalation	10.1 ppm (14.1 mg/m^3)	≥ 90 days (6 hr./day; 5 days/wk.)	Intermittent reduction in body weight gain.	CIIT, 1983 b,c
		30.5 ppm (42.5 mg/m^3)		Intermittent reduction in body weight gain.	
		80 ppm (110 mg/m^3)		Significant, constant reduction in body weight gain; significantly reduced brain weight in males.	
Rat	Inhalation	50 ppm (69.7 mg/m^3)	Days 6-20 of gestation (6 hr./day)	Slight reduction in fetal body weight.	Saillenfait et al., 1989
		100 ppm (139 mg/m^3)		Slight reduction in fetal body weight. No terata were observed.	
		150 ppm (209 mg/m^3)		Slight reduction in fetal body weight; significant reduction in maternal body weight; reduced absolute weight gain.	
Pig	Oral	3.1 mg/kg/day in food (dried greens)	105 days	No G. I. disturbance.	CIIT, 1983d

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CHEMICAL: LEAD AND COMPOUNDS

Existing Regulations, Guidelines, and Standards:

U.S National Ambient Air Quality Standard: 1.5 ug/m^3 (average over calendar quarter)
OSHA Action Level: 30 ug/m^3
Permissible Exposure Limit (PEL): 50 ug/m^3 (average over 8-hr.work period)
U.S. EPA (ODW) Maximum Contaminant Level: 0.005 mg/l
MCLG: 0 mg/l (due concern of blood levels ≥ 10 ug/dl)
U.S.EPA and IARC: Group B2 Carcinogen (Kidney tumors in rats & mice; probable human carcinogen)- (U.S. EPA, 1989)

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Human (adult)	Various	blood Pb conc. ≥ 100 ug/dl	Various	Lead encephalopathy (severe, irreversible brain damage; onset may be rapid, with convulsions, coma, and death w/in 48 hrs. in otherwise asymptomatic individuals.)	U S EPA, 1986
		blood Pb conc. 40-60 ug/dl		Sub-encephalopathic CNS and peripheral nerve damage.	
		blood Pb conc. 30-50 ug/dl		Peripheral nerve dysfunction; slowing of nerve conduction velocities.	
Human (child)	Various	blood Pb conc. 80-100 ug/dl	Various	Encephalopathic symptoms and death; possible permanent severe mental retardation and other marked neurologic deficiencies in non-fatal cases.	U.S. EPA (1986)
		blood Pb conc. 40-60 ug/dl		Peripheral nerve damage.	

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
		(blood Pb conc. ≤ 60 ug/dl)		Peripheral nerve dysfunction; slowed conduction velocity; cognitive (IQ), electrophysiologic, and neuropsychologic deficits.	
Human (child)	Various	Avg. blood Pb conc. 21 ug/dl	Various	Highly significant linear relationship between IQ scores and current blood levels in 3-7 year old children.	Schroeder and Hawk, 1986
		(blood Pb conc. 50-70, ug/dl)		IQ score deficit of approx. 5 pts.	
		(blood Pb conc. 30-50 ug/dl)		IQ score deficit of approx. 4 pts.	
		(blood Pb conc. 15-30 ug/dl)		IQ score deficit of approx. 1-2 pts.	
Human (child)	Various	(blood Pb conc. 30-50 ug/l)	Various	Changes in EEG pattern; IQ deficit.	Burchfiel et al., 1980
Human (child)	Various	blood lead 6-59 ug/dl (mean = 32.5, ug/dl)	Various	Linear relationship between EEG slow wave voltage changes and blood lead levels.	Otto et al., 1981.

CHEMICAL: LEAD AND COMPOUNDS CONTINUES....
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Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Human	Various	< 10 ug/dl blood Pb level		Inhibition of erythrocyte ALAD activity (indicator of lead exposure).	U.S. EPA, 1986.
Human	Various	Approx 40 ug/dl blood Pb conc.	Various	Reduced hemoglobin production in children.	U. S. EPA, 1986.
		50 ug/dl blood Pb conc.		Reduced hemoglobin production in adults.	
		70 ug/dl blood Pb conc.		In children, frank anemia of the hypochromic & normocytic type, associated with reticulocytosis and variable basophilic stippling.	
		80 ug/dl blood Pb conc.		In adults, frank anemia of the hypochromic & normocytic type, associated with reticulocytosis and variable basophilic stippling.	
Human	Various	12-120 ug/dl blood Pb conc.	Various	Reduced vitamin D metabolism, leading to altered calcium homeostasis and immunoregulatory activities.	U.S.EPA, 1986

CHEMICAL: LEAD AND COMPOUNDS CONTINUES....
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Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Human	Various	blood Pb conc. 40-50 ug/dl	Various	Male reproductive effects.	U. S. EPA, 1986
Human	Various	blood Pb conc. 60 ug/dl	Various	Female reproductive effects (effects on CNS of fetus, manifested postnatally as mental retardation and other neurologic deficits).	U.S. EPA, 1986
Human	Various	blood Pb conc. 30-40 ug/dl	≤9 mos.	Hypertension in white male.	U.S. EPA, 1986
Human	Maternal blood	blood lead nitrate conc. 40 ug/dl		Methemoglobinemia in infants.	Walton, 1951.
Monkey	Oral (gavage)	6 ug/kg/day (tetramethyl-lead)	6 mos.	No clinical or neurologic signs (mean blood level did not exceed 6.26, ug/dl)	Heywood, et al., 1979
Monkey	Oral (gavage)	6 ug/kg/day (tetraethyl-lead	6 mos.	No clinical or neurologic signs (mean blood level did not exceed 2.69 ug/dl)	Heywood, et al., 1979

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Rat	Oral	25 ppm (lead acetate) in drinking water plus ≤ 0.22 ppm Pb in food (total daily intake was: males: 0.512 mg/kg females: 0.394 mg/kg)	Lifetime	Shortening of lifespan (median age at death); systemic uptake of lead by liver, kideny & spleen (w/o symptoms).	Schroeder et al., 1965
Rat	Oral	25 ppm (lead nitrate) in drinking water; 0.2 ug/g food.	Lifetime	Increased incidence of glycosuria; decreased fasting glucose levels; no shortening of lifespan.	Shroeder et al., 1970
Rat & Mouse	Oral	25 ppm lead in drinking water; 0.2 ppm Pb in food		Reproductive/developmental toxicity (failure to breed, death of pups, and runting) in both species.	Schroeder and Mitchner, 1971
Rat	Oral	0.1% diet	29 mos	Reduced body weight gain; polychromasia; anisocytosis.	Van Esch et al., 1962
		1.0% diet	24 mos.	Reduced body weight gain, resulting in 35% lower body weight than controls; anemia; leucocytosis; basophilic stippling in RBC's; polychromasia; anisocytosis; presence of target cells.	

CHEMICAL: LEAD AND COMPOUNDS CONTINUES....
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Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Rat	Oral (as lead acetate in diet)	18 ppm Pb	2 years	Stippling of RBC's;	Azar et al., 1973
		62 ppm Pb		Stippling of RBC's; decreased ALAD activity	
		141 ppm Pb		Stippling of RBC's decreased ALAD activity; elevated blood lead level (18.5 ug/dl).	
		548 ppm Pb		Increased mortality, stippling of RBC's,;decreased ALAD activity; elevated urinary ALA levles; elevated blood lead level.	
		1130 ppm Pb		Stippling of RBC's; decreased ALAD activity; elevated blood lead level.	
		2102 ppm pb		Increased mortality; stippling of RBC's; decreased ALAD activity; elevated blood lead level (98.4 ug/dl)	

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Dog	Oral (as lead acetate in diet)	16 ppm Pb	2 Years	Significant elevation in blood lead (16.6 ug/dl)	Azar et al., 1973
		57 ppm Pb		Signifiant elevation in blood lead.	
		155 ppm Pb		Significant elevation in blood lead; decreased ALAD activity.	
		576 ppm Pb		Significant elevation in blood lead (75.8 ug/dl); increased stippling of RBC's; decreased ALAD activity.	
Rat	Oral (diet)	10, 50, 100, 500, 1,000 and 2,000 ppm	3-generation reproductive study	No effect on any reproductive parameter.	Azar et al., 1973
Rat	Oral (lead acetate in diet)	10 ppm	22 mos.	None	API, 1971
		50 ppm		None	
		100 ppm		Decreased food consumption & growth; decreased glucose-6-phosphatase activity at 12 mos. (but not at 22 mos)	

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
		1,000 ppm		Decreased food consumption & growth; decreased glucose-6-phosphatase activity at 12 (but not 22) mos; elevated urinary ALA levels at 6, 12, 18, & 22 mos; elevated urinary coproporphyrin levels at 22 mos.	
Dog	Oral (as lead acetate in diet)	10 ppm	22 mos.	None	API, 1971
		50 ppm		None	
		100 ppm		None	
		1,000 ppm		Decreased "hemograms"; slightly elevated urinary ALA levels.	
Monkey	Oral	1.25 mg/kg	22 mos. (7 days/wk.)	None	API, 1971
	(stomach intubation; lead acetate in distilled H_2O)	25 mg/kg		Decreased bone marrow activity; no behavioral changes seen.	

CHEMICAL: LEAD AND COMPOUNDS CONTINUES....
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Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Rat	Oral (in diet, as lead arsenate)	276 ppm Pb	120 Weeks (lactation thru post-weaning)	None	Kroes et al., 1974
		1,104 ppm Pb		Significantly reduced body weight & food consumption; increased mortality after week 26; reduced hemoglobin & packed cell volume in males; enlargement dilitation, and abscesses of bile duct; marked bile duct proliferation, pericholangitis, and cholangiofibrosis (Note: bile duct effects may be result of As (vs. Pb)-See Byron et al., 1967).	
Rat	Oral (as lead acetate in diet)	10 ppm Pb	3-generation reproductive study	No effects on any reproductive parameter.	API, 1971
		100 ppm Pb		No effects on any reproductive parameter.	
		1,000 ppm Pb		No effects on any reproductive parameter.	

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Rabbit	Oral (as lead acetate in diet)	54.6 ppm Pb	Days 7-16 of gestation	No effects on reproductive parameters or malformations at either dosage.	API, 1971
		546 ppm Pb			
Mouse	Oral (as tetraethyl lead, by intubation	0.01 mg/kg	Days 5-15 of gestation	Increased number of resorptions.	Kennedy et al., 1975
		0.1 mg/kg		Increased number of resorptions.	
		1.0 mg/kg		Reduced maternal body weight gain; retardation of fetal growth; delayed skeletal ossification; increased number of resorptions.	
		10 mg/kg		Maternal CNS toxicity & reduced body weight gain; retardation of fetal growth; delayed skeletal ossification; increased number of resorptions.	

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Rat	Oral (as tetraethyl lead, by intubation	0.01 mg/kg	Days 6-16 of gestation	None	Kennedy et al. 1975
		0.1 mg/kg		None	
		1.0 mg/kg		Decreased maternal body weight gain (70% less than controls); retarded fetal growth; increased number of resorptions.	
		10.0 mg/kg		Maternal CNS toxicity and deceased body weight gain; retarded fetal growth; increased number of resorptions; delayed skeletal ossification.	
Mouse & Rat	Oral (as lead acetate, by intubation	7.14 mg/kg			Kennedy et al., 1975
		71.4 mg/kg			
		714 mg/kg		Maternal CNS toxicity; delayed skeletal ossification; reduced fetal weight.	

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Rat	Oral (gavage, as tetraethyl lead)	7.5 mg/kg 15 mg/kg	3 Days (days 9, 10, 11 or 12, 13, 14 of gestation)	Fetal growth retardation; delayed skeletal ossification; dose-dependent decrease in maternal body weight at two lower dosages.	McClain & Becker, 1972
		30 mg/kg		CNS toxicity; 100% maternal mortality.	
Rat	Oral (gavage, as tetramethyl lead)	40 mg/kg 80 mg/kg 112 mg/kg	3 Days (days 9, 10, 11 or 12, 13, 14 of gestation)	Fetal growth retardation; delayed skeletal ossification; dose-dependent decrease in maternal body weight at three lowest dosages.	McClain & Becker, 1972
		160 mg/kg		CNS toxicity; 100% maternal mortality.	
Rat	Oral (gavage, as trimethyl lead)	15 mg/kg 30 mg/kg	3 Days (days 9, 10, 11 or 12, 13, 14 of gestation)	Fetal growth retardation; delayed skeletal ossification; dose-dependent decrease in maternal body weight at two lower dosages.	McClain & Becker, 1972
		38 mg/kg		50% maternal mortality; CNS toxicity.	

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CHEMICAL: NITROGEN DIOXIDE

Existing Regulations, Guidelines, and Standards:

U.S. National Ambient Air Quality Standard: 0.1 mg/m^3

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Human	Inhalation	< 1 ppm (1.9 mg/m^3)	Not Reported	Neglible short-term effect on respiratory tract in health humans.	U.S. EPA, 1982
	Inhalation	0.1 ppm (0.2 mg/m^3)		Impairment of respiratory tract function in asthmatic humans.	Bauer et al., 1986
	Oral	10 ppm (nitrate-N in water; 1 mg/kg/day)		Methemoglobinemia	Walton, 1951
Rat	Inhalation	0.04 (0.075 mg/m^3) 0.4 (0.753 mg/m^3) 4.0 ppm (7.53 mg/m^3)	Up to 27 mos.	Thickenen air-blood barrier and increased lipid peroxidation at all concentrations; progressive hypertrophy & hyperplasia of bronchial epithelium, thickened walls, cellular infiltration & fibrotic organization at 4.0 ppm, with severity of lesions increasing with increasing duration of exposure; less severe lesions at 0.4 ppm, seen only at 27 months.	Kubota et al., 1987; Sagai and Ichinose, 1987; Sagai et al., 1984
Rat	Inhalation	Approximately 16 ppm (30.1 mg/m^3)	≤ 17 mos. (continuous)	Emphysematous lesions in terminal bronchioles.	Juhos et al., 1987
Rat	Inhalation	0.8 ppm (1.5 mg/m^3)	≤ 813 days (continuous)	Equivocal evidence of emphysema.	Haydon et al., 1965
		12 ppm (22.6 mg/m^3)		Emphysematous lesions.	

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Rat	Inhalation	0.8 ppm (1.5 mg/m^3)	Continuous	Equivocal evidence of emphysema.	Freeman et al., 1966
Rat	Inhalation	2 ppm (3.8 mg/m^3)	2 years	Loss of respiratory tract cilia; ultra-structural changes in epithelium of terminal bronchioles.	Stephens et al., 1971
Rat	Inhalation	0.5 ppm (0.9 mg/m^3)	4 weeks (continuous)	None	Rombout et al., 1986
		1.3 ppm (2.45 mg/m^3)		None	
		2.7 ppm (5.1 mg/m^3)		Appearance of lung lesions.	
		10.6 ppm (19.9 mg/m^3)		Loss of respiratory tract cilia; influx of alveolar macrophages; hypertrophy and hyperplasia of bronchiolar epithelium.	
Rat	Inhalation	1 ppm (1.9 mg/m^3)	≤ 15 weeks (7 hr./day; 5 days/wk)	Biochemical alterations in lung.	Gregory et al., 1983
		5 ppm (9.4 mg/m^3)		Lung lesions, including accumulation of sub-pleural macrophages and focal hyperinflation.	

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Rat	Inhalation	0.1 ppm (0.2 mg/m^3)	1 month (continuous)	None	Sherwin & Richters, 1982
		0.5 ppm (0.9 mg/m^3)		None	
		3 ppm (5.7 mg/m^3)		Morphometric changes in lung.	
		10 ppm (18.8 mg/m^3)		Morphometric changes in lung.	
Mouse		0.3 ppm (0.6 mg/m^3)	6 weeks (6 hrs./day; 5 days/wk)	Increased numbers of Type II pneumocytes.	Sherwin et al., 1985

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COMMENTS ON POLYCYCLIC AROMATIC HYDROCARBONS (PAH) SECTION OF APPENDIX

A number of PAHs including pyrene, fluoranthene, benz[a]anthracene, chyrysene, triphenylene, benzo[a]pyrene and benzo[e]pyrene have been identified in crude oil from both south Louisiana and Kuwait, and it is reasonable to assume that they are associated with crude oil from other locations, as well. Some of these PAHs have been identified by the U.S. Environmental Protection Agency (U.S. EPA) as probable human carcinogens, while others are either classified as having insufficient information upon which to base a carcinogenicity classification or have not yet been tested for tumorigenic potential.

The burning of crude oil may have varying effects on the PAH concentration. While for some PAHs burning of crude oil results in less total PAHs than were in the original crude, the concentration of PAHs with five or more rings has been reported to be 10 to 20 times greater in the smoke than in the oil (Benner et al., 1990). Incineration of crude might also produce small oil droplets of varying size, which might further complicate any human exposure scenario.

In the PAH section of the appendix to this technical report, information is presented on all PAH chemicals for which information could be found, whether or not they have been specifically identified as contaminants of crude oil. While structural similarities among compounds often result in similar toxicologic manifestations, the reader is cautioned concerning the use of structural relationships to develop quantitative estimates of health risk.

POTENTIAL CARCINOGENICITY OF PAHs

PAH	U.S. EPA Carcinogenicity Classification	Basis of Classification
Acenaphthene	Not yet evaluated by the U.S. EPA for evidence of human carcinogenic potential.	
Acenaphthylene	D (not classifiable as to human carcinogenicity)	No human data; inadequate data from animal bioassays.
Anthracene	D (not classifiable as to human carcinogenicity)	No human data; inadequate data from animal bioassays.
Benz[a]anthracene	B2 (probable human carcinogen)	No human data, but sufficient data from animal bioassays. (Produced tumors in mice exposed by gavage; intraperitoneal, subcutaneous, or intramuscular injection; and topical application Produced mutations in bacteria and in mammalian cells, and transformed mammalian cells in culture.)
Benzo[a]pyrene (BaP)	B2 (probable human carcinogen)	No direct human data, but sufficient data from animal bioassays. (Evidence of carcinogenicity in multiple rodent and non-rodent species following oral, intratracheal, inhalation, and dermal administration; tested positive in several *in vitro* bacterial and mammalian genetic toxicology assays.)
Benzo(b)fluoranthene	B2 (probable human carcinogen)	No human data, but sufficient data from animal bioassays. (Produced tumors in mice after lung implantation, skin painting, and intraperitoneal or subcutaneous injection.)

POTENTIAL CARCINOGENICITY OF PAHs

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PAH	U.S. EPA Carcinogenicity Classification	Basis of Classification
Chrysene	B2 (probable human carcinogen)	No human data; but sufficient data from animal bioassays. (Produced carcinomas and malignant lymphoma in mice following intraperitoneal injection, and skin carcinomas in mice exposed dermally. Produced chromosomal abnormalities in hamsters and mouse germ cells after gavage exposure; tested positive in bacterial gene mutation assays and transformed mammalian cells exposed in culture.)
Fluoranthene	D (not classifiable as to human carcinogenicity)	No human data; inadequate data from animal bioassays.
Fluorene	D (not classifiable as to human carcinogenicity)	No human data; inadequate data from animal bioassays.
Naphthalene	D (not classifiable as to human carcinogenicity)	No human data; inadequate data from animal bioassays.
Phenanthrene	D (not classifiable as to human carcinogenicity)	No human data; inadequate data from animal bioassays.
Pyrene	D (not classifiable as to human carcinogenicity)	No human data; inadequate data from animal bioassays.

CHEMICAL: POLYCYCLIC AROMATIC HYDROCARBONS (PAHs)

Existing Regulations, Guidelines, and Standards:

Acenaphthene:	U.S. EPA oral reference dose (RfD):	0.06 mg/kg/day
Anthracene:	U.S. EPA oral reference dose (RfD):	0.3 mg/kg/day
Fluorene:	U.S. EPA oral reference dose (RfD):	0.0002 mg/kg/day
Fluoranthene:	U.S. EPA oral reference dose (RfD):	0.0002 mg/kg/day
Pyrene:	U.S. EPA oral reference dose (RfD):	0.03 mg/kg/day

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
TOXICITY OF ACENAPHTHENE					
Mouse	Oral (gavage)	175 mg/kg/day	90 days	Increased liver weight (considered *adaptive change since not* accompanied by histopathologic alterations).	U.S. EPA, 1989a
		350 mg/kg/day	90 days	Increased liver weight; hepatic cellular hypertrophy; significant increase in cholesterol levels (females, only).	
		700 mg/kg/day	90 days	Increased liver weight; hepatic cellular hypertrophy; significant increase in cholesterol levels.	
Rat	Intratracheal; intraperitoneal injection		Single exposure	Inflammatory changes in lung; vascular disorders; degeneration of internal organs and central nervous system; splenic degeneration among unscheduled deaths.	Reshetyuk et al., 1970

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Rat (partially hepatectomized)	Oral	15 mg/kg in diet	7 days	Increased liver regeneration.	Gershbein, 1975
TOXICITY OF ACENAPHTHYLENE					
Mouse	Oral (gavage)	100 mg/kg day	90 days	15% mortality in females (vs. none in males and control females); decreased erythrocyte counts in males; significantly increased cholesterol and albumin in females; increased liver weight; centrilobular hepatocellular hypertrophy; enlarged, dark, mottled, prominent reticular pattern or pale areas in liver; granular, pitted, rough, mottled appearance of kidney; nephropathy, increased incidence and severity of renal tubular dilitation, epithelial hyperplasia of the collecting ducts; slight hyperplasia of the transitional epithelium in the renal pelvis, and renal tubular microconcretions in females.	Hazleton Laboratories America, Inc., 1988

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
		200 mg/kg/day		25% mortality in females; in addition to other effects seen, at 100 ppm, also had increased hemoglobin, hematocrit, and platelet counts; increased total protein in males; decreased ovary weights.	
		400 mg/kg/day		40% mortality in females; in addition to effects seen at 100 and 200 mg/kg/day, also saw centrilobular hepatocellular hypertrophy in males; increased incidence of renal tubular regeneration; decreased erythrocyte count, hematocrit, and hemoglobin in females; increased leucocyte and segmented neutrophil counts in females; decreased number, smaller, and less active corpora lutea.	

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Rat	Oral (gavage)	176 mg/kg/day	2 months (every other day)	Decreased weight gain; histological alterations in liver; focal bronchial pneumonia; lung hemorrhages	Rotenberg and Mashbitis, 1965
Rat	Inhalation	Unknown	5 months	Alveolar desquamation; bronchitis; hyperplasia and metaplasia of bronchial epithelium.	Reshetiuk et al., 1970
Rat	Inhalation		4 months	Bronchitis; hyperplasia and metaplasia of bronchial epithelium.	Rotenberg and Mashbits, 1965
Mouse	Dermal	0.25%	Lifetime (frequency of administration not specified)	No tumors; 65% survival at 6 mos.; 35% survival at 1 yr.	Cook, 1932
		Other notes on Acenaphthylene		Positive in Salmonella typhimurium forward mutation assay.	Kaden et al., 1979.
				Negative in Salmonella typhimurium TA 98 and TA 100 test in presence of hepatic homogenates..	Bos et al., 1988

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
TOXICITY OF ANTHRACENE					
Mouse	Oral (gavage)	250 mg/kg/day 500 mg/kg/day 1000 mg/kg/day	90 days	None None None (full range of clinical and toxicologic/pathologic parameters examined).	U.S. EPA, 1989b
Rat	Oral (in oil in diet)	≈ 28 mg/kg/day (total dose ≈ 4.5g)	78 weeks (6 days/wk.)	No tumors, when observed up to ≈ 200 days.	Schmahl, 1955
Rat (3- to 6-month old females)	Lung implant	0.5 mg/rat (≈ 2 mg/kg)	Single/continuous	No tumors during 55-81 week observation period.	Stanton et al., 1972
Rat	Oral (in oil in diet)	4.5g (total dose)	78 weeks (6 days/wk.)	No treatment-related tumors.	Druckrey and Schmahl, 1955
		Other notes on Anthracene		Negative in tests for DNA damage in primary rat hepatocytes (1µg/ml), chinese hamster ovary cells (1,000 µg/ml), and HeLa cells (100 µg/ml).	Williams, 1977; Probst et al., 1977; Tong et al., 1981.

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
				Negative in cell transformation (morphologic change) tests up to 30 µg/ml using mouse BALB/3T3 cells, guinea pig fetal cells, Syrian hamster embryo cells, and mouse embryo CH310T1/2 cells.	DiPaolo et al., 1972; Evans and DiPaolo, 1975; Pienta et al., 1977; Lubet et al., 1983
				Negative in unscheduled DNA synthesis, sister chromatid exchange, chromosomal aberration, and gene mutation tests.	Brookes and Preston, 1981
TOXICITY OF BENZ[A] ANTHRACENE:					
Mouse (males)	Oral (gavage)	500 mg/kg/day (≈ 225 mg total/mouse)	5 weeks (3 doses/wk.)	Increased incidence of pulmonary adenoma and hepatoma (437-444 days after treatment initiated).	Klein, 1963
Mouse	Oral (gavage)	0.5 mg/mouse (≈ 17 mg/kg)	Single exposure	No tumors at 16 mos. post-exposure.	Bock and King, 1959
			16 months (8 or 16 treatments at 3-7 day intervals)	Stomach papillomas in 2/27 (vs. 0/16 controls) mice.	

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Mouse	Intraperitoneal (in DMSO)	638 µg/mouse	Days 1, 8, and 15 of age	Statistically significant increased incidence of liver adenomas and carcinomas in males, only (31/39 treated vs. 2/28 controls); significantly increased incidence of pulmonary ademonas or carciinomas.	Wislocki et al., 1986
		Other notes on benz[a]anthracene		Positive results in tests for complete carcinogenicity and initiating activity in skin painting assays in 2 strains of mice.	IARC, 1973
				Subcutaneous injection produced injection site sarcomas in mice 9 mos. after treatment.	Steiner and Falk, 1951; Steiner and Edgecomb, 1952
				Tested positive for DNA damage in primary rat hepatocytes and HeLa cells.	Probst et al., 1981; Martin et al., 1978
				Positive for forward mutation in Chinese hamster cells, V79 cells, mouse lymphoma L5178Y cells, and rat liver epithelial cells.	Slaga et al., 1978; Krahn and Heidelberger, 1977; Amacher et al., 1980; Amacher and Turner, 1980; Tong et al., 1981
				Positive tests for cell transformation with hamster embryo cells and mouse prostate C3HG23 cells.	Pienta et al., 1977; DiPaolo et al., 1969; Marguardt and Heidelberger, 1972

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
TOXICITY OF BENZO[A]PYRENE (BAP)					
Mouse	Oral (diet)	0, 1, 10, 20, 30, 40, 45, 50, 100, & 250 ppm	1-197 days	Stomach tumors at dietary concentrations of 20 ppm and above (incidence related to both dosage and number of administered doses).	Neal and Rigdon, 1967
Mouse	Oral (diet)	250 ppm 1,000 ppm		Apparent increased incidences of leukemia and lung adenomas at both dosages.	Rigdon and Neal, 1966, 1969
Hamsters	Inhalation	2.2 mg/m^3	10 wks. (4-5 hr./day, followed by ≤ 675 days at 3 hr./day, 7 days/wk)	None	Thyssen et al., 1981
		9.5 mg/m^3		Tumors of the nasal cavity, larynx, trachea, and pharynx.	
		45 mg/m^3		Respiratory tract tumors similar to those in mid-dosage group; also, neoplasms of upper digestive tract.	

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
		Other notes on Benzo[a]pyrene		Positive in *in vitro* mammalian sister chromatid exchange, chromosomal aberration, sperm abnormality, and *mouse specific locus tests; also,* positive in *in vitro* bacterial DNA repair, bacteriophage induction, point mutation, sister chromatid exchange, chromosomal aberration, and tranaformation of mammalian cultured cell tests.	IARC, 1973, 1983; Santodonato et al., 1981
TOXICITY OF BENZO(B)FLUORANTHENE					
Rat (female)	Lung implant	0.1 mg (0.4 mg/kg) 0.3 mg (1.2 mg/kg), and 1.0 mg (4.1 mg/kg)	Lifetime (beginning at 3 months)	*Epidermoid* carcinomas; pleomorphic sarcomas in lung and thorax (statistically significant dose-response relationship).	Deutch-Wenzel et al., 1983
Mouse	Intraperitoneal injection (in DMSO)	126 µg/mouse total dose	3 injections (days 1, 8, and 15 of age; sacrificed at 52 weeks)	Statistically significant increase in incidence of liver adenomas and hepatomas (combined) in males, only.	LaVoie et al., 1987

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Mouse	Subcutaneous injection	2.6 mg/mouse total dose	3 injections (over 2 months)	Injection site sarcomas in 18/24 survivors (of 30 at start of study).	Lacassagne et al., 1963
Mouse (female)	Dermal (skin painting)	0.01% soln.	(3 times/wk.) 14 mos.	Single papilloma in 1 of 10 surviving mice.	Wynder and Hoffman, 1959
		0.1%	12 mos.	Papillomas (65% of mice); carcinomas (85% of mice).	
		0.5%	8 mos.	Papillomas (100% of mice); carcinomas (90% of mice).	
		Other notes on benzo[b]fluoranthene		Positive in reverse mutation assay in Salmonella TA98; mixed results with Salmonella TA100.	Mossanda et al., 1979; LaVoie et al., 1979; Hermann, 1981; Amin et al., 1985 a,b.

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
TOXICITY OF CHRYSENE					
Mouse	Intraperitoneal injection (in DMSO)	160 µg/mouse (total dose)	3 injections (days 1, 8, and 15 of age; ≤ 1 yr. observation period)	Liver adenomas (predominantly) and carcinomas; malignant lymphoma; (all tumors statistically significant in males; no tumors in females)	Wislocki et al., 1986
		640 µg/mouse (total dose)		*Liver adenomas and carcinomas* (predominantly); lung adenomas; no malignant lymphomas as seen in low-dose males; (all tumors statistically significant in males; no tumors in females).	
Mouse	Intraperitoneal injection (in DMSO)	320 µg/mouse (total dose)	3 injections (days 1, 8, and 15 of age; observation period thru 38-42 weeks of age)	Hepatic tumors (statistically significant) in males, only.	Buening et al., 1979

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Mouse	Intraperitoneal injection (in DMSO)	320 µg/mouse (total dose)	3 injections (days 1, 8 and 15 of age; observation thru 38-42 weeks of age)	Hepatic tumors (statistically significant) in males; no liver tumors seen in females.	Chang et al., 1983
		Other notes on Chrysene		Shown to be complete carcinogen in mouse skin painting assay.	Wynder and Hoffmann, 1959
				Produced chromosomal effects in chinese hamster cells, mouse oocytes, and hamster spermatogonia *following 450 or 900 mg/kg gavage* doses.	Basler et al., 1977; Roszinsky-Kocher et. al., 1979.
				Positive in cell transformation tests using Syrian hamster embryo cells at 10 µg/ml.	Pienta et al., 1977
				Negative for cell transformation in mouse prostate C3HG23 cells.	Marguardt and Heidelberger, 1972

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
TOXICITY OF FLUORANTHENE					
Mouse	Oral (gavage)	125 mg/kg/day	13 weeks	Nephropathy; increased salivation; liver enzyme effects (but not considered significant or adverse by authors at this dosage level).	U.S. EPA, 1988
		250 mg/kg/day		Nephropathy; increased salivation; elevated SGPT; increased liver weights (relative and absolute); microscopic liver lesions; hematolagical alterations.	
		500 mg/kg/day		Nephropathy; increased salivation; elevated SGPT; increased liver weights (relative and absolute); microscopic liver lesions; hematological alterations.	

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Mouse	Intraperitoneal injection (in DMSO)	700 µg total dose (163 mg/kg)	3 injections (days 1, 8 and 15 of age; necropsy at 24 wks. of age)	No significant incidence of lung tumors.	Busby et al., 1984
		3,500 µg total dose (815 mg/kg)		Statistically significant combined incidence of adenomas and adenocarcinomas (males & females combined); 80% of tumors were adenomas; 20% adenocarcinomas.	
Mouse	Dermal	10% solution (in acetone)	13 months (3 times/wk.)	No tumors.	Suntzeff et al., 1957
Mouse	Dermal	0.1% solution (in acetone)	Lifetime (3 times/wk.)	No tumors.	Wynder and Hoffman, 1959
Mouse	Dermal	1% solution (50 µl dosages)	12 months (3 times/wk.)	No tumors.	Hoffman et al., 1972
Mouse	Dermal	50 mg (in decalin or 50:50 decalin: dodecane mixture)	82 weeks (2 times/wk.)	No tumors.	Horton and Christian, 1974

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
		Other notes on Fluoranthene		Positive in mouse co-carcinogen skin painting assays with benzo[a]pyrene (due increased formation of BaP adducts).	Van Duuren and Goldschmidt, 1976; Rice et al., 1988
				Equivocal (both positive and negative) results in chromosal effects tests in Chinese hamster cells.	Palitti et al., 1986 (positive); DeSaliva et al., 1988 (negative)
				Negative in test for gene mutations in human lymphoblast cells.	Crespi and Thilly, 1984
				Equivocal (both positive and negative) results in gene mutation tests using mutant Chinese hamster ovary cell lines.	Hoy et al., 1984 (positive); Li, 1984 (positive); Hoy et al., 1984 (negative)
				Equivocal results (both positive and negative) in mutagenicity assays in several strains of Salmonella typhimurium.	Positive: Kayden et al., 1979; Kinae et al., 1981; LaVoie et al., 1982; Babson et al., 1986; Bos et al., 1988 Negative: Tokiwa et al., 1977; Kinae et al., 1981; Bos et al., 1987

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
TOXICITY OF FLUORENE					
Mouse	Oral (gavage)	125 mg/kg/day	13 weeks	No dose-related statistically significant effects.	U.S. EPA, 1989c
		250 mg/kg/day		Decreased erythrocyte count and packed cell volume in females; increased liver weight (relative and absolute); hypoactivity; significant increase in spleen and kidney weights (relative and absolute) in males.	
		500 mg/kg/day		Hypoactivity; labored respiration; ptosis; unkempt appearance; decreased erythrocyte count, packed cell volume, and hemoglobin (both sexes); increased serum bilirubin; decreased BUN; increased liver, spleen, and kidney weights (both sexes); histopathological increases in amounts of hemosiderin in spleen and Kupffer cells in liver.	

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Rat	Oral (diet)	0.62-1% fluorene	104 days; then maintained on 0.5% or 1% fluorene drets.	Decreased growth rate.	Wilson et al., 1947
	Oral (diet)	0.125, 0.25, or 0.5% fluorene	453 days	No tumors or other effects reported.	
Mouse	Subcutaneous injection (in glycol)	10 mg/injection	Seven injections; then observed for 18 mos.	No injection site tumors.	Shear, 1938
		Other notes on Fluorene		Negative in unscheduled DNA synthesis assay using primary rat hepatocytes (at 10 nmol and 100 nmol/ml).	Probst et al., 1981; Williams et al., 1989
				Positive in DNA damage (strand-break) assay using L5178Y mouse lymphoma cells at 0.15 µm in presence of hepatic homogenates and at 0.5 µm in absence of hepatic homogenates.	Garberg et al., 1988

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
				Negative in forward mutation assays in L5178Y mouse lymphoma cells at up to 30 µg/ml in the presence of hepatic homogenates, and up to 60 µg/ml *w/o* hepatic homogenates.	Amacher et al., 1981; Oberly et al., 1984
				Negative in DNA damage assay using primary rat hepatocytes (max. concentration 3 mM).	Sina et. al., 1983
				Negative in reverse mutation assays in five strains of Salmonella typhimurium (1,000 µg/plate) and in forward mutation assays in Salmonella strain TM677 (50 µg/ml).	McCann et al., 1975; LaVoie et al., 1979, 1981; Sakai et al., 1985; Bos et al., 1988; Kaden et al., 1979; Mamber et al., 1983
				Negative in DNA damage assay using S. typhimurium TA 1535 (16.7 µg/ml).	Nakamura et al., 1987
				Negative in DNA damage assay in Escherichia coli at 2 mg/ml.	Mamber et al., 1983

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
TOXICITY OF NAPHTHALENE					
Rat	Oral (diet)	10 g/rat total (≈ 30-60 mg/kg/day)	6 times/week, until 10g total dose acheived; observation period 700-800 days.	No carcinogenic response.	Schmahl, 1955
Mouse (females)	Inhalation	0, 10, or 30 ppm	6 months (6 hr./day; 5 days/wk.)	Statistically significant increase in number of adenomas per mouse lung (but no apparent dose-response).	Adkins, 1986
Rat	Intraperitoneal injection	20 mg/injection	40 weeks (once/wk.)	No carcinogenic response.	Schmahl, 1955
Rat	Subcutaneous injection (in sesame oil)	500 mg/kg/dose (coal-tar derived naphthalene; 10% unidentified impurities).	Seven injections at 2-week intervals; study length 18 mos.	Lymphosarcomas in 14.7% (5/34) of surviving rats at 18 mos. (vs. 2% of controls). Note: presence of impurities and previous dermal application of carbofuchsin to injection site obscure significance of those results.	Knake, 1956

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
		Other notes on Naphthalene		Negative in DNA damage assays using primary rat hepatocytes.	Sina et al., 1983
				Negative in transformation assays using rat and mouse embryo cells.	Freeman et al., 1973; Rhim et al., 1974
				Negative in DNA damage assays using Salmonella typhimurium and Escherechia coli.	Nakamura et al., 1987; Mamber et al., 1983
				Negative in reverse mutation assays in a variety of strains of Salmonella typhimurium at up to 2.5 mg/plate (w/ or w/o hepatic homogenates).	McCann et al., 1975; Anderson and Styles, 1978; Florin et al., 1980; Gatehouse, 1980; Connor et al., 1985; Ho et al., 1981; Sakai et al., 1985; Mortelmans et al., 1986; Bos et al., 1988

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
TOXICITY OF PHENANTHRENE					
Mouse	Intraperitoneal injection (in DMSO)	0.25 mg total dose	Days 1, 8 and 15 of age; observed to 38-42 wks of age.	No increase in incidence of pulmonary tumors over controls; no hepatic tumors; one treated female (of 18 examined) developed malignant lymphoma.	Buening et al., 1979
Mouse	Subcutaneous injection (in acetone/ gelatin vehicle)	40 µg	Single injection; observed for 52-62 wks.	No increase in pulmonary, hepatic, or other tumors over controls.	Grant and Roe, 1963
Mouse	Subcutaneous injection (in tricaprylin	5 mg	Single injection; observed for 4 mos.	No tumors reported.	Steiner, 1988
		Other notes on Phenanthrene		Negative for DNA damage in tests with several mammalian cell cultures.	Lake et al., 1978; Probst et al., 1981; Rice et al., 1984
				Negative in f orward mutation assay using Chinese hamster ovary cells (at 1 µg/ml).	Huberman and Sachs, 1976

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
				Positive for forward mutation in test with human lymphoblast TK6 cells incubated with rat liver S9 (Arochlor) and 9 µg/ml phenanthrene.	Barfknecht et al., 1981
				Negative in sister chromatid exchange and chromosome aberration assays in mammalian cell cultures.	Popescu et al., 1977
				Negative in cell transformation assays in several types of mammalian cells (5-40 µg/ml).	Marguardt and Heidelberger, 1972; Kakunaga, 1973; Evans and DiPaolo, 1975; Pienta et al., 1977.

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
TOXICITY OF PYRENE					
Mouse	Oral (gavage)	75 mg/kg/day	13 wks.	None	U.S. EPA, 1989d
		125		Nephropathy, characterized by the presence of multiple foci of renal tubular regeneration, often accompanied by lymphocytic infiltrates and/or foci of interstitial fibrosis (all kidney lesions evaluated as minimal or mild); decreased kidney weights (relative and absolute).	
		250		Same as mid-dose.	
Rat (male)		2000 mg/kg diet	40 days	Decreased body weight gain in 2 of 6 rats; enlarged livers and increased hepatic lipid content.	White and White, 1939

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Mouse	Intraperitoneal injection (in DMSO)	40 µg total dose	3 injections (days 1, 8 and 15 of age; observation period of 1 year)	No increased incidence of liver or lung tumors or malignant lymphoma (compared with controls)	Wislocki et al., 1986
		141 µg total dose		Same as low dose.	
		466 µg total dose		No increase in tumor incidence (same as two lower dosages); decreased survival rate (75% decrease in males; 65% decrease in females), occurring primarily between last injection and weaning.	
		Other notes on Pyrene		Negative as complete carcinogen or as initiator in mouse skin-painting assays.	Badger et al., 1940; Horton and Christian, 1974; VanDuuren and Goldschmidt, 1976; Salaman and Roe, 1956; Scribner, 1973
				Did not produce tumors in s.c. injected mice (single injection) observed for 18 mos.	Shear and Leiter, 1941

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
				Positive for sister chromatid exchange in CHO mammalian cells in one study, but negative when concentration increased 10-fold.	Evans and Mitchell, 1981
				Negative for sister chromatid exchange and chromosome aberrations in CHO cells.	Brooks and Preston, 1981
				Negative for sister chromatid exchange in rat liver epithelial cell system.	Tong et al., 1981
				Positive in L51784 mouse lmphoma gene mutation assay.	Jotz and Mitchell, 1981
				Negative for chromosome aberrations and sister chromatid exchange in bone marrow of several mouse strains injected intraperitoneally.	Purchase and Ray, 1981

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
				Negative in cell transformation assays in a variety of mammalian cell types.	DiPaolo et al., 1969; Pienta et al., 1977; Casto, 1979; Chen and Heidelberger 1969; DiPaolo et al., 1972; Kakunaga, 1973; Evans and DiPaolo, 1975.
				Negative in DNA damage assays in Escherechia coli and Bacillus subtilis.	Ashby and Kilbey, 1981
				Equivocal results (both positive and negative) in a variety of bacterial gene mutation tests.	Positive: Kinae et al., 1981; Bridges et al., 1981; Matijasevic and Zeiger, 1985; Sakai et al., 1985; Kaden et al., 1979; Bos et al., 1988 Negative: McCann et al., 1975; LaVoie et al., 1979; Ho et al., 1981; Bos et al., 1988

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CHEMICAL: REFINED PETROLEUM PRODUCTS

Existing Regulations, Guidelines, and Standards:

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Human: Acute	Inhalation	Direct sniffing of vapors (gasoline)	15-20 breaths	Intoxication similar to ETOH (lasting 5-6 hrs).	Edminster and Bayer, 1985; Polkis and Burkett, 1977
	Inhalation	5,000-16,000 ppm (AVGAS)	unknown	Death	Wang and Irons, 1961
	Inhalation	1,000 ppm 7,000 ppm (gasoline)	15 min. 5 min.	Drowsiness, numbness, intoxication.	Fielder et al., 1921
	Inhalation	140-270 ppm (gasoline)	8 hrs.	Slight eye irritation; GI disturbance.	Drinker et al., 1943
		1,000 ppm (volatile fraction distilled below 110°C)	1 hr.	Slight dizziness; nausea; headache.	
		2,600 ppm (distillate fraction)	1 hr.	Intoxication; mild anesthesia.	
		10,000 ppm (distillate fraction)	approximately 5 min.	Dizziness and drunkenness.	

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
	Inhalation	200, 300, & 1,000 ppm (unleaded gasoline)	30 min.	Eye irritation (number of reportings increasing with increasing concentration).	Davies et al., 1960
	Oral	Approximately 7.5 g/kg* (gasoline)	single dose	Death	Machle, 1941
	Dermal	complete immersion (gasoline)		Cutaneous injury (full thickness skin loss).	Hansbrough et al., 1985
Human: Chronic	Inhalation	unknown (gasoline vapor from spills; individual case study)	12 months	Hemolytic anemia and myelofibrosis.	McLean, 1960
	Inhalation/ Dermal	unknown (use of gasoline to clean metal parts)	2 years	Thrombocytopenia	Machele, 1941
	Inhalation/ Dermal	unknown (51 retail filling station employees)	N.R.	Headaches, fatigue, sleep disturbances, memory loss, giddiness, & generalized weakness.	Pandya et al., 1975
	Inhalation	unknown (workers chronically exposed) (gasoline)	reported only as chronic	Decrease in phagocytic activity of peripheral blood granulocytes; decrease in total blood protein & globulin levels; increases in blood properdin levels (compared with controls).	Przybylowski et al., 1976 (abstract)

*Usually fatal, although may vary somewhat with aromatic content of gasoline (increasing aromatics increases toxicity)

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Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Human: Chronic	Multiple (primarily inhalation)	Unknown (petroleum products)	Various (chronic)	18 of 50 patients in epidemiological study with acute nonlymphocytic leukemia (ANLL) had been occupationally exposed to petroleum products or their combustion residues (significantly different from multiple control groups).	Brandt et al., 1978
	Multiple	Unknown (petroleum, tar, or pitch)	≥ 20 yrs.	Renal cell carcinoma incidence *increased among males* occupationally exposed to petroleum, tar, or pitch products.	McLaughlin et al., 1984
	Multiple (primarily inhalation)	Unknown (crude petroleum or refinery products)	Various (chronic)	Male refinery employees had 3 x risk of esophageal and stomach cancer and ≈ 2 x expected risk of lung cancer; all risks increased with increasing duration of employment.	Hanis et al., 1979
	Multiple (primarily inhalation)	Unknown (petroleum and petrochemical products)	Various (chronic)	Significant increases in the incidence of: lymphocytic leukemia in refinery workers; multiple myeloma in petrochemical workers; cutaneous melanoma in middle Atlantic region petroleum industry plants.	Schottenfeld et al., 1981

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Human: Chronic (Cont.)	Multiple (primarily inhalation)	Unknown (crude petroleum and refinery products)	Various (chronic)	Statistically significant excesses in incidences of stomach, pancreatic, skin, and prostate cancer; also, increases in cancer of brain, multiple myelomas, and leukemia in white males, only.	Thomas et al., 1982
	Multiple (primarily inhalation)	Unknown (crude oil and refinery products)	Various (chronic)	Significant increase in brain cancer in young refinery workers who died <20 years from start of exposure.	Theriault and Goulet, 1979
	Multiple (primarily inhalation)	Unknown (crude oil and refinery products)	Various (chronic)	No significant excess in brain cancer deaths, but incidence did increase in refinery workers employed >20 years.	Wen et al., 1982
	Multiple (primarily inhalation)	Unknown (gasoline)	Various (chronic)	Slight upward trend in incidence of renal cell cancer among gasoline station attendants with increasing duration of employment; not seen in other petroleum exposure occupations.	McLaughlin et al., 1985
	Multiple (primarily inhalation)	Unknown (crude petroleum and refinery products)	Various (chronic)	In review of methology of 8 epidemiologic studies of cancer risk among oil refinery workers, concluded that the evidence for association with brain cancer is reasonably consistent.	Savitz and Moure, 1984

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Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Human: Chronic (Cont.)	Multiple (primarily inhalation)	Unknown (gasoline)	Various (chronic)	Increased incidence of primary liver cancer in service station employees.	Stemhagen et al., 1983
	Multiple (primarily Inhalation)	Unknown (crude petroleum and refinery products)	Various (chronic)	Judged collectively, data from 25 studies provide suggestive evidence of an association between employment in petroleum refineries and risk of stomach cancer, respiratory system cancer (lung, pleura, nasal cavity, and sinuses), and cancer of the lymphatic and hematopoietic tissues.	U.S. EPA, 1987
	Multiple (primarily inhalation)	Unknown (petroleum and refinery products)	Various (chronic)	Increased incidence of bladder cancer in petroleum industry employees).	Howe et al., 1980
	Inhalation (abusers)	Unknown (gasoline)	Intermittent (during pregnancy)	Profound retardation, initial hypotonia progressing to hypertonia, scaphocephaly, prominent occiput, poor post-natal head growth, and additional minor anomalies.	Hunter et al., 1979
	Unspecified	Unknown (gasoline and mixtures of gasoline and chlorinated hydrocarbons)	Various (occupational)	Gynecological disorders (infantilism and chronic inflammation of the uterus).	Vozovaya, 1974

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Human: Chronic (Cont.)	Inhalation	Unknown (gasoline vapors)	Various (occupational)	Menstrual disorders.	Zhibura, 1974
	Inhalation	"Low concentrations" (gasoline vapors)	5 years	Increased incidence of amenorrhea.	Mirinski, 1979
	Inhalation	Unknown (diesel and exhaust fumes)	Various (occupational)	Significantly greater probability ($p<0.01$) of having chromosome breaks in diesel vehicle drivers.	Fredga et al., 1982
Rat: Acute/Subchronic	Oral (gavage)	10 ml/kg-25 ml/kg (PS-6 unleaded gasoline)	Single dose	Dose-dependent increase in severity of GI irritation, diarrhea, and bleeding around eyes, nose, and mouth; lung irritation and congestion.	Elars Bioresearch Laboratories, 1982
Rat	Oral (gavage)	0.5 g/kg 2.0 g/kg (PS-6 unleaded gasoline)	4 weeks	Onset of glomerulonephritis; neuropathy.	Borriston Labs, 1983
Rabbit	Dermal	0.5 ml (PS-6 unleaded gasoline) on patch	24 hrs.	Edema (within 24 hrs) and erythema (within 72 hrs.) (both gone within 14 days).	Elars Bioresearch Laboratories, 1982

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Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Guinea Pig	Dermal	0.5 ml (PS-6 unleaded gasoline) on patch for initial dose; then diluted 50% for remaining 9 treatments.	3 weeks (6 hr/test; 3 times/week; 10 total treatments)	Edema and erythema (both within 24 hrs.)	Elars Bioresearch Laboratories, 1982
Rabbit	Dermal	2.5 ml/kg body wgt. (PS-6 unleaded gasoline)	2 weeks (24 hr./day by patch; 5 days/wk.)	Progressive deterioration of test site (w/skin becoming necrotic, thickened, cracked, green in color, and odiferous); some bleeding & sloughing of skin; evidence of kidney congestion.	Elars Bioresearch Laboratories, 1982
		5 ml/kg		Same as low-dose group.	
		8 ml/kg		Same as low-dose group; also, decreased appetite and weight loss.	
Rat	Inhalation	Unknown (gasoline exhaust fumes)	5 weeks	Significant reduction in peak expiratory flow and forced expiratory mean flow (suggestive of diffuse bronchial damage).	Saldiva et al., 1985

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Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Rat	Inhalation	100 ppm ("super" grade gasoline vapor; 98 octane; 0.45g TEL/l)	12 weeks (8 hr./day; 5 days/wk.)	Pathologic changes in lung (ranging from minor scattered foci of interstitial fibrosis to widespread sclerosis) after 6-12 wks. of exposure, increasing w/time; irregular alveolar collapse, associated w/overdistension of uninvolved alveoli after 6 wks; effects on type 2 pneumocytes ranging from early hypertrophy and hyperplasia to reduction in number and vacuolization and degeneration of surfactant-producing organelles; prostration and tachypnea after 8 wks.	Lykke and Stewart, 1978
Rats & Mice	Inhalation	Unknown (gasoline exhaust)	5 weeks	Obstructive airway pattern seen in pulmonary function tests; positive micronucleous (mutagenicity) test; pathological respiratory lesions.	Massad et al., 1986

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Rats (females only)	Inhalation	100 ppm ("super" grade gasoline; 98 octane; 0.45g TEL/l)	12 weeks (8 hr./day; 5 days/wk.)	Increasing signs of distress after 6 weeks; pathologic changes in lung ultrastructure, increasing in severity w/length of exposure (degenerative changes in interstitial fibroblasts and vascular endothelium by 5 weeks; hypertrophic and hyperplastic changes in type 2 pneumocytes between weeks 6-10; end-stage interstitial sclerosis; irregular alveolar collapse and overdistension; vacuolar cytoplasmic degeneration of surfactant-producing organelles in type 2 pneumocytes between weeks 9-12).	Lykke et al., 1979
Rabbit	Inhalation	60 mg/l (gasoline - leaded and unleaded)	6 months (4 hr./day; 6 days/wk.)	Decreased serum and liver monamine oxidase (MAO) activity.	Przybylowski et al., 1977
Rabbit	Inhalation	Unspecified (gasoline vapors - leaded and unleaded	Unspecified (chronic)	Decreased blood protein, total globulin, and gamma globulin levels (more pronounced w/leaded gasoline); decreased blood albumin and ceruloplasmin levels (leaded gasoline, only).	Przybylowski et al., 1974

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Rabbit	Inhalation	Unspecified (gasoline vapors - leaded and unleaded	Unspecified (chronic)	Increased urinary excretion of 17-ketosteroids; decreased excretion of 17-hydroxycortico steroids (suggestive of pituitary-adrenal changes) (effects of unleaded gasoline more pronounced).	Przybylowski et al., 1978
Rat	Inhalation	1,500 ppm (unleaded gasoline)	18 months (6 hr./day; 5 days/wk.)	Scattered axonal degeneration in gracile tracts of spinal cord, and increased incidence of abnormalities of anterior horn cells.	Spencer, 1983
Rat	Inhalation	1,552 ppm (unleaded gasoline)	13 weeks (6 hr./day; 5 days/wk.)	Increased respiratory rate; increased platelet count; regenerative epithelium and dilated tubules in kidney.	McFarland, 1983
		384 ppm		Increased platelet count; slight increase in liver weight.	
Rat	Inhalation	374 ppm (leaded gasoline; 1.94g Pb/gal./0.72 ppb)		Increased urinary lead excretion and tissue lead levels.	McFarland, 1983
		103 ppm (0.19 ppb lead)		Slight increase in liver weight; increased urinary lead excretion and tissue lead levels.	

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Monkey	Inhalation	384 ppm or 1, 552 ppm (unleaded gasoline)	13 weeks (6 hr./day; 5 days/wk.)	None	McFarland, 1983
		103 or 374 ppm (leaded gasoline; 1.94g/gal./0.19 or 0.72 ppb of lead)	13 weeks (6 hr./day; 5 days/wk.)	None	
Rat	Inhalation	67 ppm (PS-6 unleaded gasoline)	103-113 weeks (6 hr./day; 5 days/wk.)	Renal carcinomas in males.	McFarland, 1982
		292 ppm		Renal carcinomas in males; 1 malignant renal tumor (mixed) in females.	
		2,056 ppm		Renal carcinomas in males.	
Mouse	Inhalation	67 ppm (PS-6 unleaded gasoline)	103-113 weeks (6 hr./day; 5 days/wk.)	None	McFarland, 1982
		292 ppm		Increased incidence of liver nodules and masses in females.	
		2,056 ppm		Increased incidence of liver nodules and masses in females; decreased body weight.	

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Rat	Inhalation	40 ppm (unleaded gasoline-blend)	90 days	Renal lesion in 1/10 males.	Halder et al., 1983
		370 ppm		Renal lesion in 50% of males.	
		3,866 ppm		Renal hyaline droplet accumulation; proximal convoluted tubular degeneration and regeneration; corticomedullary tubular dilitation and necrosis.	
				No effects in females at any dosage; renal effects observed may be peculiar to males rats (i.e., α-2 μglobulin-dependent).	
Rat (male, only)	Inhalation	300 mg/m^3 (gasoline - grade BR-1)	2.5 months	General intoxication; decreased spermatogenesis; no dominant lethal mutations.	Feller, 1972
Rat	Inhalation	300 mg/m^3 (gasoline fumes)	30-45 days	Accelerated rate of uterine myometrial contractions.	Lipovskii, 1978

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CHEMICAL: SULFUR DIOXIDE

Existing Regulations, Guidelines, and Standards:

U.S. EPA Standard for Oxides of Sulfur:

1 year exposure: 0.03 ppm (80 ug/m^3)
24 hr. expousre: 0.14 ppm (365 ug//^{3})
3 hr. exposure: 0.5 ppm (1,300 ug/m^3)

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Human	Inhalation	40 ppb (0.104 mg/m^3)	≥ 11 years	None (absence of symptoms of chronic obstructive pulmonary disease).	Euler et al., 1987
Mouse	Inhalation	0.05 ppm (0.13 mg/m^3)	3 mos. (continuously)	None	Oshima et al., 1969
		0.15 ppm (0.39 mg/m^3)		Increased mortality; lung congestion, with redness on exterior surface; hemorrage, infiltration of inflammatory cells, and abscesses inside lungs; thickened smooth muscle in bronchi.	
		0.23 ppm (0.60 mg/m^3)		Same as above.	
Dog	Inhalation	15 ppm (39 mg/m^3)	5 mos. (2 hr./day; 4-5 days/wk)	None	Scanlon et al; 1984, 1987
		50 ppm (131 mg/m^3)	10-11 mos. (2 hr./day; 4-5 days /wk)	Coughing; mucous hypersecretion; increased pulmonary airway resistance; histological changes in respiratory tract.	

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CHEMICAL: TOLUENE

Existing Regulations, Guidelines, and Standards:

U.S. EPA Inhalation Reference Concentration: 1.5 mg/m^3 for Lifetime Exposure
OSHA Permissible Exposure Limit (PEL): 100 ppm (air)
Time Weighted Avg. (TWA) STEL: 150 ppm (air)
NIOSH Recommended Exposure Limits (REL) for Occupational Exposure:
TWA: 100 ppm (375 mg/m^3)
10-Minute Ceiling: 200 ppm (750 mg/m^3)
U.S. EPA (ODW) Maximum Contaminant Level: 2.0 mg/l
Health Advisories:
1 day: 21.5 mg/l
10 day: 3.46 mg/l

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Human	Inhalation	10 ppm (37.7 mg/m^3)	6 hr. (single exposure)	None	Andersen et al., 1983
		40 ppm (151 mg/m^3)		None	
		100 ppm (377 mg/m^3)		Dizziness; headache; feeling of intoxication; eye and nose irritation.	
Human	Inhalation	100 ppm (377 mg/m^3)	6.5 hr. (single exposure)	Nasal and eye irritation; moderate fatigue; sleepiness; headaches; feeling of intoxication; decrease in psychometric performance (esp. visual perception and accuracy, visual perserverance, and color discrimination).	Baelum et al., 1985

CHEMICAL: TOLUENE CONTINUES....
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Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Human	Inhalation	200 ppm (754 mg/m^3)	7 hr. (single exposure)	Prolongation of reaction time; decrease in pulse rate and systolic pressure.	Ogata et al., 1970
Human	Inhalation	100 ppm (377 mg/m^3)	8 hr. (single exposure)	Moderate fatigue; sleepiness; headache.	von Oettingen et al., 1942
		200 ppm (754 mg/m^3)		Muscular weakness; confusion; impaired coordination; dilated pupils; aftereffects (fatigue, general confusion; moderate insomnia).	
Human	Inhalation	50 ppm (189 mg/m^3)	1-3 Weeks	Frequent headaches and lassitude.	Wilson, 1943
		100 ppm (377 mg/m^3)		Frequent headaches and lassitude	
Rat	Inhalation	30 ppm (113 mg/m^3)	106 Weeks	None	CIIT, 1980
		100 ppm (377 mg/m^3)		None	
		300 ppm (1,130 mg/m^3)		None	

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Rat	Oral (gavage)	312 mg/kg/day	13 Weeks (5 days/wk.)	None	NTP, 1989
		625 mg/kg		Increase in absolute and relative kidney & liver weights.	
		1,250 mg/kg		Increase in absolute and relative kidney & liver weights; mineralized foci and necrosis of neuronal cells in brain.	
		2,500 mg/kg		Increased mortality; increase in absolute and relative kidney weights; hepatocellular hypertrophy; nephrosis; damage to tubular epithelium; mineralized foci and necrosis of neuronal cells in brain; hemorrhage of bladder muscularis; prostration, ataxia, hypoactivity, piloerection; lacrimation; excessive salivation; tremors; decreased body weight in males.	
		5,000 mg/kg		100% mortality in first week.	

CHEMICAL: TOLUENE CONTINUES....
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Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Mice	Oral (gavage)	312 mg/kg/day	13 Weeks (5 days /wk.)	None	NTP, 1989
		625 mg/kg		None	
		1,250 mg/kg		None	
		2,500 mg/kg		Subconvulsive jerking; prostration; impaired grasping reflex, bradypnea, hypothermia, ataxia, and hypoactivity. Reduced mean body weight in males; increased mortality (8/20).	
		5,000 mg/kg		100% mortality.	
Rat	Oral (gavage)	118 mg/kg	193 Days (138 total doses)	None	Wolf et al., 1956
		354 mg/kg		None	
		590 mg/kg		None	

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Rat	Inhalation	600 ppm (2,262 mg/m^3)	2 Years (6.5 hr./day; 5 days/wk.)	Non-neoplastic lesions of nasal cavity at 15 mos.; changes in olfactory & respiratory epithelia.	NTP, 1989
		1,200 ppm (4,524 mg/m^3)		Increased incidence and severity of non-neoplastic lesions of nasal cavity at 15 mos.; changes in olfactory & respiratory epithelium.	
Mouse	Inhalation	120 ppm (453 mg/m^3)	2 Years (6.5 hr./day; 5 days/wk.)	None	NTP, 1989
		600 ppm (2,262 mg/m^3)		None	
		1,200 ppm (4,524 mg/m^3)		Slight hyperplasia of bronchial epithelium.	
Rat	Inhalation	100 ppm (37.7 mg/m^3)	26 Weeks (6 hr./day; 5 days/wk.)	Significantly increased serum SGPT in females.	API, 1980
		1,500 ppm (5,655 mg/m^3)		Slight (but not statistically significant) increase in serum SGPT in females.	

CHEMICAL: TOLUENE CONTINUES....
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Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Rat	Inhalation	30 ppm (113 mg/m^3)	Up to 24 mos.	None	Gibson and Hardisty, 1983
		100 ppm (377 mg/m^3)		Slight, but significant, reduction in hematocrit in females.	
		300 ppm (1,131 mg/m^3)		Slight, but significant, reduction in hematocrit and increase in mean corpuscular hemoglobin.	
Rat	Inhalation	320 ppm (1,206 mg/m^3)	30 Days (continuously)	Decreased weight of cerebral cortex and overall brain weight; reduction of total phospholipids in cerebral cortex.	Kyrklund et al., 1987
Rat	Inhalation	200 ppm (754 mg/kg)	30 Days (continuously)	None	Ikeda et al, 1986
		400 ppm (1,508 mg/m^3)		Significant decrease in norepinephrine in olfactory cortex of brain; significant decrease in dopamine in the striatum (brain).	

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Rat	Inhalation	500 ppm (1,885 mg/m^3)	12 Weeks (5 days/wk., 8 or 16 hr./day)	Alteration in CNS excitability, as indicated by disruptions & frequency changes in hippocampal theta-wave activity (seen at 10 days in 16 hr./day exposed animals, but only after 40 days in 8 hr./day animals). No increase toward normal theta-wave activity after 1 month of recovery.	Naalsund, 1986
Guinea Pig, Dog, Monkey	Inhalation	389 mg/kg/day	90 Days	None	Jenkins et al., 1920
Mouse	Oral (drinking water)	16 ppm (60 mg/m^3) 80 ppm (302 mg/m^3) 400 ppm (1,508 mg/m^3)	Pre- and post-natal	Impairment of rotorod performance at 45-55 days of age in all exposure groups, but dose-response relationship was inverted.	Kostas and Hotchin, 1981
Mouse	Inhalation	200 ppm (754 mg/m^3) 400 ppm (1,508 mg/m^3)	Days 7-16 of gestation (7 hr./day)	Significantly increased incidence of dilated renal pelvises in pups. Significantly increased LDH activity in brains of dams; significantly increased liver & kidney LDH activity in exposed, but non-pregnant, females.	Courtney et al., 1986

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Rat	Inahalation	500 ppm (1,885 mg/m^3)	2-generation	No effects.	API, 1985
		2,000 ppm		Significant inhibition of growth of offspring over both generations	
Mouse	Inhalation	500 mg/m^3	Days 6-15 of gestation (12 hr./day)	None	Ungvary and Tatrai, 1985
		1,000 mg/m^3		Significant retardation of fetal weight.	
Rabbit	Inhalation	500 mg/m^3	Days 7-20 of gestation (continuously)	None	Ungvary and Tatrai, 1985.
		1,000 mg/m^3		Retardation of maternal body weight gain; change in relative liver weight; increased incidence of spontaneous abortion.	

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CHEMICAL: XYLENE

Existing Regulations, Guidelines, and Standards:

OSHA Permissible Exposure Limit (PEL): 100 ppm
Time-weighted Avg. (TWA) STEL: 150 ppm
NIOSH Recommended Exposure Limit (REL): 10-hour: 100 ppm
10-min.: 200 ppm

U.S. EPA (ODW) Health Advisories:
1 day (10 kg child): 12 mg/l
10 day (10 kg child): 7.8 mg/l
Longer Term (70 kg adult): 27.3 mg/l
Longer Term (10 kg child): 7.8 mg/l
Lifetime 0.4 mg/l

U.S. EPA Inhalation Reference Concentration (RfC): 0.27 mg/m^3 for Lifetime Exposure
U.S. EPA Oral Reference Dose (RfD): 2.0 mg/kg/day for Lifetime Exposure

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Human	Inhalation (*p-xylene*)	20 ppm (87 mg/m^3)	5 days (7.5 hr./day)	None	Hake et al., 1981
		100 ppm (434 mg/m^3)		Headache; nose and throat irritation in females.	
		150 ppm (651 mg/m^3)		Decrease in performance on Flanagan Coordination Test. (Females not exposed to this concentration).	
Human	Inhalation	100 ppm (*m-xylene*)	2 weeks (6 hr./day; 5 days/wk.)	Impaired reaction time and equilibrium (however, some tolerance developed)	Salvolainen et al., 1979

CHEMICAL: XYLENE CONTINUES....
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Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Human	Inhalation	435 mg/m^3 (mixed xylenes)	70 min.	No effects on 5 neurological performance tests.	Gamberale et al., 1978
		1,300 mg/m^3 (mixed xylenes)		No effects on 5 neurological peformance tests.	
		1,300 mg/m^3 (mixed xylenes) re-exposure	70 min. (exercise during first 30 min.)	Decreased performance on all 5 tests (addition, sample & choice reaction times, short-term memory, critical flicker fusion frequency).	
Human	Inhalation	392 mg/m^3 (m-xylene)	1-8 days	Neurotoxic symptoms, when accompanied by exercise.	Salvolainen et al., 1980
Human	Inhalation	868 mg/m^3 (m-xylene)		Impaired balance.	Salvolainen and Linnoila, 1979
Rat	Inhalation (o-xylene)	4,750 mg/m^3	1 year (8 hr./day; 7 days/wk.)	Depressed weight gain; hepatomegaly.	Tatrai et al., 1981
Rat & Dog	Inhalation (mixed xylenes)	180 pppm (782 mg/m^3)	65 days (6 hr./day; 5 days/wk.)	No effects in either species at any concentration.	Carpenter et al., 1975
		460 ppm (1,997 mg/m^3)			
		810 ppm (3,517 mg/m^3)			

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Rat	Oral (gavage) (Technical grade xylene)	250 mg/kg/day	2 years (5 days/wk. for 103 weeks)	Increase in mortality in males.	NTP, 1986
		500 mg/kg/day		Increase in mortality in males; slightly reduced body weights (compared with controls).	
Mouse	Oral (gavage) (technical grade xylene)	500 mg/kg/day	2 years (5 days/wk. for 103 weeks)	None	NTP, 1986
		1,000 mg/kg/day		Hyperactivity (lasting 5-30 min. after each dosing).	
Rat	Oral (gavage) (technical grade xylene)	62.5 mg/kg/day	13 weeks	None	NTP, 1986
		125 mg/kg/day		None	
		250 mg/kg/day		None	
		500 mg/kg/day		None	
		1,000 mg/kg/day		None	
		2,000 mg/kg/day		Decreased body weights in both sexes (compared w/controls).	

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Mouse	Oral (gavage) (technical grade xylene)	125 mg/kg/day	13 weeks	None	NTP, 1986
		250 mg/kg/day		None	
		500 mg/kg/day		None	
		1,000 mg/kg/day		None	
		2,000 mg/kg/day		Increased mortality in females; lethargy, short& shallow breathing, unsteadiness, tremors, and paresis in both sexes.	
Rat	Inhalation (o-, m-, or p-xylene)	150 mg/m^3	Days 7-14 of gestation	Skeletal retardation (p-xylene, only)	Ungvary et al., 1980
		1,500 mg/m^3		Maternal toxicity (ortho-only); decreased fetal weight (o-xylene, only); skeletal retardation (p-xylene, only);	

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
		3,000 mg/m^3		Maternal toxicity (all isomers); increased maternal mortality for o-xylene; decreased fetal weight for all isomers; skeletal retardation (o-and p-xylene, only); development of extra rib in fetuses (m- and p-xylene, only).	
Rat	Inhalation (mixed xylenes and isomers)	250 mg/m^3	Days 7-15 of gestation (continuous)	Skeletal retardation in fetuses (mixed xylenes & all isomers).	*Ungvary and Tatrai, 1985*
		1,900 mg/m^3		Skeletal retardation in fetuses (mixed xylenes & all isomers).	
		3,400 mg/m^3		Skeletal retardation in fetuses (mixed xylenes & all isomers); increased incidence of extra ribs, retardation of fetal weight, and increased incidence of dead or resorbed fetuses.	
Mouse	Inhalation (mixed xylenes & all isomers)	500 mg/m^3	Days 6-15 of gestation (12 hr./day)	Fetal weight and skeletal retardation (o-, m-, and p-xylene).	Ungvary and Tatrai, 1985
		1,000 mg/m^3		Fetal weight and skeletal retardation (mixed xylenes and all isomers).	

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Rabbit	Inhalation (mixed xylenes & all isomers)	500 mg/m^3	Days 7-20 of gestation (continuous)	Increased incidence of dead or resorbed fetuses (m-xylene, only); reduction in mean fetal body weight in females.	Ungvary and Tatrai, 1985
		1,000 mg/m^3		Decreased maternal weight gain; increased number of aborted fetuses (data provided for mixed & p-xylene, only).	
Rat	Inhalation (mixed xylenes)	60 ppm (261 mg/m^3)	131 days before mating; 20 day mating period; Days 1-20 of gestation; Days 5-20 of lactation (6 hr./day)	None	Bio/Dynamics Inc., 1982
		250 ppm (1,085 mg/m^3)		None	
		500 ppm (2,170 mg/m^3)		Delayed skeletal ossification; reduced fetal weight (females, only).	
Rat	Inhalation (mixed xylenes)	1,000 mg/m^3	Days 9-14 of gestation (continuous)	Increased incidence of retarded skeletal development and structural anomalies.	Hudak and Ungvary, 1978

Toxicity:

Species	Route of Exposure	Dose/ Concentration	Duration of Exposure	Effects	Reference
Rat	Inhalation (p-xylene)	3,500 mg/m^3	Days 7-16 of gestation (6 hr./day)	None	Rosen et al., 1986
		7,000 mg/m^3		Decreased maternal body weight gain; no fetal effects.	
Mouse	Inhalation (o-, m-, or p-xylene	2,171-30,973 mg/m^3	30 min. (single exposure)		Moser et al., 1985
		6,078 mg/m^3		Disruption of operant behavior (differential reinforcement of low rates).	
		8,684 mg/m^3		Motor performance decrements (inverted screen test).	
Rat	Inhalation (p-xylene)	6,947 mg/m^3	4 hrs. (single exposure)	Depression in flash-evoked potential amplitude recorded in visual cortex.	Dyer et al., 1987
Rat	Inhalation	320 ppm (1,389 mg/m^3)	30 or 90 days	Increased liver-to-body weight ratio and decreased linoleic acid in cerebral cortex ethanolamine phosphoglyceride after 30 days (but no longer seen at 90 days).	Kyrklund et al., 1987

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