Toxicologic agents of concern for search-and-rescue dogs responding to urban disasters

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Highly trained **search-and-rescue (SAR)** dogs are indispensable members of urban SAR teams responding to a variety of natural and man-made disasters. A large-scale disaster could result in the release and mixing of dozens or even hundreds of chemical agents. Therefore, urban disaster sites should be considered potentially contaminated with many dangerous chemicals and substances.

In a previous report,[1] general toxicologic hazards for SAR dogs responding to urban disasters were discussed. The purpose of the present article is to raise awareness of some of the specific common substances that may be found at urban disaster sites and their potential for short- and long-term health effects.

Hydrocarbons

The term hydrocarbon refers to any compound consisting of carbon and hydrogen. The most common classes of hydrocarbons include petroleum distillates (eg, diesel fuel and exhaust, gasoline, naphtha [lighter fluid, varnish, and dry-cleaning products], Stoddard solvents [dry-cleaning solvents, general cleaners and degreasers, paint thinners, photocopier toners, printing inks, and adhesives], mineral spirits, mineral seal oil [furniture polish], kerosene, jet fuel, fuel oil, lubricating oils, petroleum jelly, paraffin wax, and asphalt), aliphatic hydrocarbons (1,3-butadiene, butane, ethane, hexane, methane, and propane), aromatic hydrocarbons (benzene, toluene, and xylene), terpenes (turpentine and pine oil), chlorinated hydrocarbons (carbon tetrachloride, chlorinated hydrocarbon insecticides, chloroform, methyl chloride, methylene chloride, tetrachloroethylene, trichloroethane, vinyl chloride, and trichloroethylene), and others (automatic transmission fluid, coal pitch, cutting fluids, dielectric fluids, liquefied hydrocarbon gas, naphthalene, machining fluids, pressurized paints, thermoplastic road paint, and waste oils).[2]

The large number of motor vehicles typically found in an urban disaster site can be a source of many hydrocarbon chemicals, including gasoline, motor oil, and transmission fluid. Many of these substances are a mixture of hydrocarbons. For instance, gasoline may typically contain 150 hydrocarbon compounds in addition to various additives and blending agents.[3] Depending on the nature of the disaster, specific hydrocarbons may be of greater or lesser concern. For instance, at the World Trade Center and the Pentagon disaster sites in September 2001, jet fuels were involved. On the other hand, a study[4] of volatile organic compounds at municipal structural fires revealed that benzene dominated, along with toluene and naphthalene.

Hydrocarbons can be absorbed following respiratory, oral, or dermal exposure. Inhalation may occur as a result of exposure to dust, particles, vapors, or aerosols. Absorption following dermal exposure is slower and less extensive than that following respiratory or oral exposure. Preferential distribution to fatty tissues occurs, especially with the aliphatic hydrocarbons, and neurotoxicoses are thought to occur when lipophilic hydrocarbons dissolve in nerve cell membranes and disrupt membrane protein function. Metabolism of hydrocarbons is predominantly through oxidation by cytochrome P-450 isoenzymes and conjugation to more water-soluble compounds. Metabolism in the liver may represent an activation pathway for some hydrocarbons and a detoxification pathway for others. Most hydrocarbons are excreted in urine as metabolites; exhalation of unchanged parent compounds and biliary excretion are other potential routes of elimination.[3] The risk and extent of adverse effects are related to the types and amounts of chemicals involved and the duration of the exposure.[3]

Toxicosis resulting from exposure to hydrocarbons has been associated with a variety of clinical signs, including dermal (dermatitis, skin eruptions, burns, epidermal necrosis), ocular (conjunctivitis, corneal irritation, corneal necrosis), gastrointestinal (nausea, vomiting, diarrhea, abdominal pain), pulmonary (vascular epithelium damage, petechiation, hemorrhage, atelectasis, respiratory distress), and CNS (euphoria, seizures, coma) disorders.[3,5,6] In addition, particular effects have been associated with exposure to specific hydrocarbon compounds (**Appendix 1**). Hepatotoxicosis has been associated with chloroform, carbon tetrachloride, and trichloroethylene. Renal toxicosis characterized by glomerular injury and proximal tubular damage may also be associated with severe systemic exposures to these agents.[5] Individuals with cardiac disease or chronic lung disease may be more susceptible to a solvent's arrhythmogenic actions; arrhythmias reported in people have included premature atrial or ventricular contractions, atrial fibrillation, ventricular tachycardia or fibrillation, and asystole.[7] There is limited evidence that most hydrocarbons are carcinogenic in both humans and animals.[3] People with subclinical or clinical epilepsy are considered to be at increased risk of seizures from exposure to xylene because of its CNS effects. In addition, those who take haloperidol, acetaminophen, or aspirin or have a nutritionally inad-

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equate diet may also be more susceptible to the toxic effects of hydrocarbons.[3] Thus, SAR dogs with a history of epilepsy, cardiopulmonary disease, or other medical conditions or dogs taking certain medications could be at an increased risk for health problems associated with hydrocarbon exposure.

Polychlorinated Biphenyls

Polychlorinated biphenyls (PCBs) are a group of synthetic chlorinated organic compounds manufactured in the United States from 1929 to 1977. Formulations vary by isomer composition and chlorine weight. These compounds were widely used as coolants for electrical transformers and capacitors, as extenders in paints and pesticides, as lubricants in gas turbines, and in hydraulic systems, textiles, sealants, carbonless copy paper, fluorescent light ballasts, air conditioners, television sets, and other products. Use of PCBs in open systems was banned in 1976, and use in closed systems was banned in 1979. However, older buildings may still contain PCBs that could be released during a fire or other disaster.[8] Polychlorinated biphenyls are oily liquids or solids that are colorless to light yellow, odorless, and tasteless. They can be found in air as vapors or aerosols.[9]

Polychlorinated biphenyls can be absorbed following dermal, oral, or respiratory exposure.[9] At disaster sites contaminated with PCBs, SAR dogs could be at risk of exposure as a result of breathing contaminated air, walking across contaminated substrates, and licking contaminated substrates or their feet. Dermal absorption is the major route of exposure in people working around PCBs. Following absorption, PCBs are transported to liver and muscle and then redistributed to adipose tissue. In the liver, PCBs are metabolized to hydroxylated phenolic compounds that can be excreted or further metabolized to methylsulfinyl metabolites. Excretion is through the urine and feces.[8] Unchanged PCBs may be stored for years in the liver or body fat and can potentially be passed in the milk during lactation.[9]

Evaluation of the health effects of PCBs is complicated, because the toxicity of a PCB mixture depends on the toxicity of the individual congeners in the mixture, the effects of their interactions, and the effects of any impurities, particularly chlorinated dibenzofurans, that may be present. In general, toxic effects following oral exposure to PCBs include hepatic damage (hepatomegaly, lipid accumulation, fibrosis, and necrosis), gastric injury (gastritis, ulceration, and hemorrhage), thyroid suppression, anemia, dermal effects (facial edema, acne, folliculitis, and alopecia), reproductive damage (suppressed lactation, low testosterone concentration, reduced conception rate, and low birth weight), neurobehavioral abnormalities, hepatocellular carcinoma, pericardial edema, weight loss, renal tubular damage, and death.[9] Toxic effects following dermal exposure include hepatic damage, renal damage, hyperplasia, hyperkeratosis, reduced weight gain, thymic atrophy, and death. Toxic effects following inhalation include hepatic damage and renal damage but typically occur only following exposure to high concentrations. Animal studies[9] also show that PCBs containing 60% chlorine by weight are clearly carcinogenic, with the target organ being the liver. Polychlorinated biphenyls may also act as modifying agents following exposure to other carcinogens, potentially acting as tumor promoters or inhibitors.[8]

Hazardous Metals

Metals are present in virtually every man-made structure found in an urban environment, including buildings, furniture, office equipment, appliances, and supplies used in manufacturing. Although the most commonly used metals and alloys are of relatively low toxicity, highly toxic metals are still in use in a wide range of items to which SAR dogs may be exposed at urban disaster sites. In most cases, hazardous metals are contained in such a way as to minimize human or animal exposure, but when the structural integrity of buildings or equipment is compromised, containment may break down and exposure to hazardous metals may occur. Fires, explosions, and compressive forces that often accompany building damage may substantially increase the potential for exposure to toxic concentrations of metals as a result of release from complexes (eg, release of arsenic during burning of pressure-treated lumber) or formation of particulates or fumes (eg, release of lead fumes from lead piping or shielding). Many toxic metals are relatively poorly absorbed following oral exposure but readily absorbed following inhalation of particulates.[10] Additionally, chemical reactions resulting from the combination of metals with acids or other agents may result in the formation of toxic products (eg, arsenic reacts with acids to form highly toxic arsine gas).[11] Because SAR dogs generally work without the same respiratory protection used by their human counterparts and search low to the ground where dusts and gases may accumulate, they may be at higher risk of developing toxicoses, compared with human SAR workers. Some of the more important hazardous metals likely to be encountered by SAR dogs include antimony, arsenic, beryllium, cadmium, chromium, cobalt, lead, mercury, nickel, thallium, and zinc (**Appendix 2**).

Asbestos

The term asbestos is used to refer to any of six different hydrated magnesium silicates that exist as mineral fibers: amosite, chrysotile, crocidolite, tremolite, actinolite, and anthophyllite[12,13] (tremolite, actinolite, and anthophyllite also have nonfibrous forms that are not considered to be asbestos). Asbestos is further divided into serpentine (chrysotile) and amphibole (amosite, crocidolite, tremolite, actinolite, anthophyllite) groups. Chrysotile, the sole serpentine asbestos fiber, is flexible and curly and often occurs in intertwined bundles of fibrils. Amphiboles have straight, brittle, needle-like shapes and are found stacked in parallel rows.[13,14] In nature, asbestos particles consist of parallel aggregations of long crystalline fibrils that may be up to several centimeters long. Milling of asbestos is accomplished by mechanically crushing and cleaning the fibers, producing fibers that range in length from < 1 μm to approximately 50 μm.[13] Because asbestos fibers are strong and highly resistant to heat and chem-

ical degradation, asbestos was used in a wide variety of products where these features were desirable, including fireproof fabrics and insulation. The largest single use of asbestos has been as a binding agent in cement panels and pipes.[13] Because of health concerns, the widespread use of asbestos has declined since the 1970s,[13] but asbestos is still present in the concrete and insulation of many existing urban structures. Under normal circumstances, the matrix in which asbestos is bound prevents substantial human or animal exposure to asbestos fibers. During an urban disaster, however, compressive and explosive forces may result in the release of asbestos and pose a hazard to rescue workers at disaster sites.[13] SAR dogs may be at increased risk of asbestos exposure, as they actively sniff areas in which dusts that might contain asbestos fibers have settled.

Asbestos is predominantly an inhalation concern; no firm evidence exists to indicate that asbestos poses a clinically important hazard when ingested.[12] Most epidemiologic and experimental research on asbestos-related lung disease has focused on chronic low-level exposure, as this is the primary means of human exposure. Less information is available on the effects expected from short-term, high-level asbestos exposure, as might occur at a large, heavily contaminated urban disaster site. A few studies[12] in rodents have demonstrated that a single exposure to concentrated asbestos may be associated with development of pulmonary fibrosis, and acute exposure of SAR dogs to urban disaster sites may result in development of acute airway disease, such as bronchitis or bronchiolitis.[12,15] However, the long-term effects of short-term, high-level exposure to asbestos are as yet not known.

Inhaled asbestos fibers have been associated with pulmonary inflammation and fibrosis (asbestosis), lung cancer, and mesothelioma in humans, with fiber length determining the type of disease that develops.[12,15] Studies[16,17] on long-term asbestos exposure in dogs have shown that the pulmonary response to inhaled asbestos is similar to that of humans, although asbestos-related mesotheliomas in dogs are associated primarily with chrysotile fibers,[18] whereas mesotheliomas in humans are almost always associated with amphibole fibers.[14] Asbestos-related disease appears to be less common in dogs than in humans,[12,19] possibly because of the shorter life spans of dogs compared with humans, in whom asbestos-related disease may take years to decades to develop.[12]

The finding of asbestos in some canine mesotheliomas has led some to propose that dogs may be useful as sentinels for environmental contamination with asbestos.[18,20] More investigation is needed to determine whether monitoring the pulmonary health of SAR dogs may serve to help predict the potential for development of asbestos-related pulmonary disease in rescue workers and others exposed to urban disaster sites.

Gases

Previous reports[21-23] have indicated that firefighters may be exposed to a variety of potentially harmful gases, including hydrogen cyanide (from combustion of wool, silk, nylon, paper, and polyurethane and polyurethane foam), nitrogen dioxide (from combustion of fabrics, cellulose nitrate, and celluloid), hydrogen chloride (from combustion of polyvinyl chloride and some fire-retardant materials), various halogen acid gases (from the combustion of films, fluorinated resins, and some fire-retardant materials containing bromine), sulfur dioxide (from the combustion of sulfur-containing materials), and carbon monoxide (from incomplete combustion of hydrocarbon fuels and some methylene chloride-containing paint strippers and aerosol products). Because of the wide variety of situations in which SAR dogs might be used, these gases should also be considered when planning for the health and safety of these dogs. It is impossible to adequately discuss all gaseous toxicants in this section, but a few of the most important will be discussed.

Hydrogen cyanide—Although other forms of cyanide exist, cyanide in the air is mainly hydrogen cyanide. Hydrogen cyanide is not easily removed from the air by settling, rain, or snow. As a gas, it is explosive and colorless and has a faint odor recognized by some people as a bitter almond smell.[24] The primary method used to manufacture hydrogen cyanide is through the reaction of ammonia with methane, but hydrogen cyanide can be formed in other ways, including the reaction of ammonia with air and natural gas and the combustion of various materials. Additionally, adding cyanide salts to water will produce large quantities of hydrogen cyanide.[21,24]

Exposure to even small amounts of hydrogen cyanide is extremely dangerous. Cyanide blocks electron transport in cytochrome aa_3, inhibiting oxidative phosphorylation and aerobic metabolism. The partial pressure of oxygen in the peripheral tissues begins to rise, resulting in decreased unloading of oxygen from hemoglobin. Oxyhemoglobin concentrations in venous blood therefore increase, causing a flush to the skin and mucous membranes. This is the cause of the cherry-red mucous membrane color often associated with cyanide toxicosis.[24-26] A brief period of hyperpnea and cardiac arrhythmias can be identified, but central respiratory arrest is the cause of death. Death can occur within seconds or minutes following inhalation of large amounts of hydrogen cyanide. Other signs associated with acute cyanide poisoning include lethargy, confusion, hypotension, nausea, and vomiting. Long-term cyanide exposure may cause weakness, nausea, and abdominal pain and has been linked with several debilitating neurologic disorders.[24]

Hydrogen sulfide—Under normal conditions, hydrogen sulfide exists as a gas. It is referred to as sewer gas because it smells like rotten eggs. Humans can smell hydrogen sulfide at concentrations between 0.0005 ppm (0.00055 mg/m^3) and 100 ppm (110.55 mg/m^3). At concentrations > 100 ppm, hydrogen sulfide can no longer be smelled by humans because of olfactory paralysis.[27] Hydrogen sulfide is a flammable gas produced from human and animal waste and by petroleum refineries, natural gas plants, paper mills, iron smelters, food processing plants, and tanneries.[28] Heavier than air, hydrogen sulfide accumulates in low-lying areas[27,28] and, therefore, may present a risk of exposure to SAR dogs working close to the ground,

even when clinically important exposures might not be expected for their human counterparts.

The physiologic effects of hydrogen sulfide depend on its concentration in inspired air. Hydrogen sulfide is a potent inhibitor of cytochrome oxidase, similar to hydrogen cyanide,[28] but has a greater tendency to produce local tissue reactions such as conjunctivitis and pulmonary edema.[26] The main targets in hydrogen sulfide toxicosis are the eyes, the olfactory apparatus, and the nervous and respiratory systems. Health officials are still unsure whether long-term exposure to low doses of hydrogen sulfide causes any long-term health effects. Importantly, the odor of hydrogen sulfide is an unreliable warning signal because of rapid paralysis of the olfactory apparatus and rapid development of other signs, including death, at high concentrations.[28] There are no published reports of carcinogenesis, mutagenesis, or teratogenesis caused by hydrogen sulfide.[27,28]

Freon—Freon is a term for any 1 of several chemical compounds used as refrigerants, aerosol propellants, and solvents. The most commonly used Freon is Freon-12, or dichlorodifluoromethane. Freon-12 is a gas at ordinary temperatures. Typically, Freons are colorless, odorless, and considered to be chemically unreactive. Therefore, the toxicity of Freon is generally low, unless exposure to massive quantities takes place. Freon-12 was given to rats and mice 24 hours a day for 1 month at concentrations as high as 500 mg/m^3, and no significant changes were identified.[29] However, when the rats and mice were exposed to the same concentrations of Freon-12 and the products of its pyrolysis, they had reduced gas exchange, retarded reflex development, and inhibited activity compared with control animals.[29] In another study,[30] inhalation of 2% Freon-113 in dogs caused a reduction in nicotinic transmission, similar to inhalation of 2% halothane. However, exposure to small amounts of Freon-113, even for up to 14 days, did not cause any long-term gross or histologic changes.[30] In outdoor environments, Freon is expected to dissipate rapidly; accumulation is not likely to occur except in confined spaces.

Halogenated gases—The most common gases in this group are chlorine, bromine, and fluorine. These gases have largely industrial uses.

Chlorine gas is used in the paper, chemical, and textile industries and for sewage treatment. It is irritating to the eyes and upper respiratory tract and can cause production of oxygen radicals, which then lead to further damage to epithelial cells in these areas.[31] Pulmonary edema can occur within 6 to 24 hours after exposure to dangerous concentrations of chlorine gas. Long-term respiratory difficulties can develop following halogenated gas exposure, although these signs may eventually resolve in some individuals. Chlorine gas has a very strong and irritating odor; often a greenish cloud is visible, allowing exposed individuals to smell and see the gas.[31]

Bromine gas is used in the petrochemical and sanitation industries, as a fire retardant, and as an agricultural chemical. Exposure to bromine gas has resulted in many human fatalities. Even brief exposures to bromine fumes cause coughing, epistaxis, gastrointestinal irritation, and diffuse dermatitis in people. Animals exposed to bromine gas at a concentration of 3 ppm have developed pulmonary edema and damage to the respiratory epithelia.[31]

Fluorine gas is used in the petrochemical and aluminum manufacturing industries; it is also found in dyes, ceramics, agricultural chemicals, and flux and is used for etching glass. It is very irritating, causing both upper and lower respiratory tract damage.[31] Ocular and nasal irritation occur following exposure to a fluorine concentration of 5 to 10 ppm. Diffuse damage to respiratory airways and parenchyma in humans has occurred following exposure to a fluorine concentration of 100 ppm.[31]

Carbon monoxide—Carbon monoxide is a colorless, odorless, nonirritating gas produced by incomplete oxidation of hydrocarbon-based fuels. Because carbon monoxide is slightly lighter than air, it is unlikely to pose a major risk to SAR dogs, unless they are entering confined, poorly ventilated spaces containing motor vehicle exhaust or smoke. Another potential source of exposure is methylene chloride, found in some industrial paint products, because it is metabolized to carbon monoxide.[22]

Carbon monoxide is rapidly absorbed by the lungs, diffusing across the alveolar-capillary membranes and attaching to hemoglobin with an affinity 250 times that of oxygen. The uptake of carbon monoxide is influenced by the concentration of carbon monoxide to which an individual is exposed, the individual's ventilatory rate, and the duration of the exposure to the gas. The lungs are also the site of excretion of carbon monoxide; its elimination seems to be complex, depending on duration of exposure and whether exposure was continuous.[23]

Three forms of carbon monoxide toxicosis have been described: acute intoxication, recurrent symptom syndrome, and delayed neuropsychiatric sequelae.[22] Acute intoxication occurs immediately, but once the exposure is terminated, the patient usually improves. The patient may then deteriorate 1 to 2 weeks later with recurrent symptom syndrome before recovering a second time. Other patients develop more serious signs such as delayed neuropsychiatric sequelae and may never fully recover. Signs of mild intoxication include headache, nausea, dizziness, and vomiting. Signs of moderate intoxication include confusion, dyspnea, weakness, tachycardia, tachypnea, and ataxia. Signs of severe intoxication include severe lethargy, disorientation, dysrhythmias, hypotension, syncope, lactic acidosis, seizures, pulmonary edema, and coma. Cognitive function deficits have been reported in people occupationally exposed to low doses of carbon monoxide,[23] but it is unknown at what point prolonged low-dose exposure to carbon monoxide might affect an SAR dog's cognition and ability to reliably perform in a field situation.

Soaps, Detergents, Acids, and Alkalis

In an urban disaster setting, soaps, detergents, and various cleaning products are commonly encountered, but they are rarely found in large amounts, unless the

area involved is highly industrialized or fire suppression foams have been used. Soaps are generally low in oral and dermal toxicity and typically cause only mild gastrointestinal and mucous membrane irritation.[32,33] Detergents are nonsoap surfactants (a substance capable of reducing the surface tension of a liquid in which it is dissolved) in combination with inorganic ingredients such as phosphates, silicates, or carbonates. Detergents are classified according to their charge in solution as nonionic, anionic, and cationic.[32] Nonionic and anionic detergents are ocular, dermal, and gastrointestinal irritants, with nonionic detergents being the less irritating of the two.[32-34] Cationic detergents are generally more toxic than the others.[32-34] Dermal and ocular exposure to cationic detergents can result in erythema, edema, pain, and ulceration. Oral ingestion of cationic detergents can result in corrosive burns and may result in systemic toxicosis as well. Cationic detergents are hypothesized to possess ganglionic blocking effects and a curare-like action, causing paralysis of the neuromuscular junction of striated muscles.[35]

Acids or alkalis may be found in small amounts in cleaning products and may be present in large amounts in industrial settings. Some of the more common acid-containing substances include toilet bowl cleaners, drain openers, metal cleaners, antirust compounds, automobile battery fluid, and pool sanitizers.[35] Some of the more common alkali-containing substances include drain openers, oven cleaners, bleaches, industrial pipeline cleaners, bathroom and household cleaners, electric dishwasher soaps, and low-phosphate detergents.[36] Acids and alkalis are both corrosive but produce their effects in different ways. Acids cause a coagulation-type necrosis, with destruction of surface epithelium and submucosa and some involvement of blood vessels and lymphatics. Alkalis cause liquefaction necrosis, allowing deep penetration into mucosal tissues as cells are destroyed.[33-36] Oral, corneal, or dermal contact with acids or alkalis may cause mild to moderately severe burns.[35,36] Initial signs may not reliably predict the extent of injury. Exposure to acid vapors, mists, or aerosols may result in dyspnea, pulmonary edema, hypoxemia, bronchospasm, pneumonitis, tracheobronchitis, and ocular irritation.[35]

Ethylene Glycol

Ethylene glycol is an aliphatic polyalcohol. It is colorless, odorless, clear, and slightly viscous. Ethylene glycol has a slightly sweet to bitter taste and is highly flammable.[37] It is found in many items commonly kept in home and industrial settings, including antifreeze, coolants, deicing solutions, solvents used in the paint and plastics industries, photographic developing solutions, hydraulic brake fluids, inks, condensers and heat exchangers, home solar units, polishes, and cosmetics. Most exposures to ethylene glycol are oral. Because dogs that have been deprived of water are more likely to drink ethylene glycol,[38] SAR dogs may be at an increased risk of exposure if they become dehydrated while working. Ethylene glycol's vapors are heavier than air, and ethylene glycol is not likely to exist in large amounts as an aerosol.[37]

The minimum oral toxic dose of ethylene glycol in dogs has not been determined. However, because the lethal dose can be as low as 6.6 mL/kg (3 mL/lb), any suspected oral exposure to ethylene glycol should warrant evaluation.[39,40] Ethylene glycol is quickly absorbed from the gastrointestinal tract and may be detected in the blood within 30 minutes after a clinically significant exposure. Ethylene glycol undergoes metabolism to more toxic acidic metabolites in the liver within the first 4 to 8 hours after exposure.

Early neurologic signs of ethylene glycol toxicosis (ataxia, depression) can progress to renal failure and more severe neurologic signs (coma, seizures). Dogs can become polyuric and polydipsic within 1 to 2 hours.[41,42] Within 6 to 12 hours, dogs develop metabolic acidosis from the acid and aldehyde metabolites and then become progressively more lethargic. They may eventually develop seizures. Most dogs that survive this early phase develop oliguric renal failure 24 to 72 hours after ingestion as a result of progressive loss of nephron integrity.[43]

Propylene Glycol

Propylene glycol is a dihydroxy alcohol made from glycerol. It is a clear, colorless, odorless, tasteless, and slightly syrupy liquid at room temperature. Propylene glycol must be heated or briskly shaken to produce a vapor.[44] It has many uses in home and industrial settings, and can be found in liquid pharmaceuticals, inks, artificial smoke, antifreeze, deicing solutions, lubricants, plasticizers, and resins. As with other short-chain alcohols, absorption is rapid after ingestion, with detectable concentrations in blood within 30 minutes after ingestion and peak concentrations within 2 hours.[45] Propylene glycol is approximately a third as toxic as ethylene glycol in dogs but should not be considered nontoxic. Propylene glycol is quickly metabolized to lactate, and in large quantities may cause lactic acidosis, liver damage, and renal insufficiency. The most common clinical signs of propylene glycol intoxication include CNS depression, weakness, and ataxia. Hypernatremia, seizures, vomiting, recumbency, coma, polyuria, polydipsia, hemolysis, Heinz body anemia, and increased kidney and liver enzyme activities have also been reported.[45,46]

Phenol

Phenol is a hydrolyzed form of benzene and is a colorless to white crystalline solid when pure. The commercial product, which contains some water, is a liquid.[47] Phenol is flammable and has a distinct sweet and tarry disinfectant odor.[48] Large amounts of phenol may be present in industrial sites, as it is used in phenolic resins, caprolactam (a nylon precursor), nonionic detergents, dyes, indicators, medical and veterinary antiseptics, disinfectants, preservatives, and slimicides.

In dilute solution (0.1 to 4.5%), phenol is an irritant, but in concentrations of 5% or more, phenol is caustic. Phenol is quickly absorbed across the skin, and large doses can lead to muscle tremors, convulsions, coma, and death.[49] If phenol is ingested, it may cause oral and esophageal burns, along with panting, profuse vomiting, diarrhea, salivation, ataxia, gastric ulcers, muscle fasciculations, and methemoglobinemia. Large

doses can cause seizures, coma, arrhythmias, hypotension, pulmonary edema, and severe metabolic acidosis.[50,51] Hepatic and renal damage may occur within 12 to 24 hours.[52] The oral LD_{50} in dogs is approximately 300 to 500 mg/kg (136 to 227 mg/lb).[53]

Phenol does not remain in the air long, with an aerosol half-life of < 24 hours.[48] Exposure to aerosolized phenol can cause respiratory irritation, and repeated exposure to high concentrations over several days can cause muscle tremors and loss of coordination.[48,54,55] Continued exposure to high concentrations of aerosolized phenol for several weeks can cause paralysis, along with severe injury to the heart, kidneys, liver, and lungs, followed by death in some cases.[54]

Alcohols

Alcohols are found in many substances, but unless ingested in large quantities, they generally do not cause severe problems. Alcohols are primarily used as solvents or as intermediate chemicals in the synthesis of other compounds.[56] Ethanol, isopropanol, and methanol are the most commonly encountered alcohols.

The effects of alcohols vary, and alcohols appear to affect cell membranes, rather than specific receptor sites.[57] The basic mechanism of action of alcohols is thought to be dissolution of lipid membranes, thereby affecting ion channels and their proteins and leading to depressant effects on the CNS. Ethanol has also been shown to augment γ-aminobutyric acid-mediated synaptic inhibition.[58] All alcohols can be absorbed following dermal, oral, or respiratory exposure,[59-61] and clinical effects are dose dependent. Central nervous system depression and ataxia are the most common signs, but gastritis, hypotensive shock, acidosis, dyspnea, and tremors may also develop. Methanol causes blindness only in humans and primates.

Conclusion

It is not possible to anticipate all of the potential toxicologic hazards that SAR dogs may encounter while searching urban disaster sites, but this article is meant to raise awareness of some of the common toxic substances and the means by which SAR dogs might be exposed to them. Awareness and planning are critical to minimizing the chances of a dangerous exposure in the first place, but will also help facilitate prompt evaluation and initiation of appropriate treatment if problems do occur while SAR dogs are in the field. Additionally, knowledge of potential long-term health problems is important for veterinary practitioners responsible for the regular health care of these dogs once they return home.

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Appendix 1

Effects associated with exposure to specific hydrocarbons[3]

Hydrocarbon	Effects
Gasoline[a]	Respiratory, oral, and dermal irritation; transient to permanent neurologic impairment; respiratory effects secondary to aspiration
Jet fuel[b]	Hepatic damage following inhalation of intermediate duration; skin cancer following long-term dermal exposure
n-hexane[c]	Peripheral neuropathy; dermal and ocular irritation
Toluene[d]	Fatigue, headache, nausea, and drowsiness; permanent CNS damage, including ototoxicosis
Benzene	Nonlymphocytic leukemia in humans; additional hematologic, immunologic, and lymphoreticular effects in humans and animals

[a]Degree to which additives such as lead contribute to adverse effects is unknown. [b]Composition is typically more uniform than composition of other hydrocarbons. [c]Commercial hexane is a mixture of *n*-hexane and other hydrocarbons and has not been shown to cause peripheral neuropathy following equivalent exposure. [d]Toxic only at concentrations > 100 ppm.

Appendix 2

Sources, kinetics, and toxic effects of selected hazardous metals

Metal	Source	Kinetics	Toxic effects
Antimony	Fireproofing chemicals, manufacture of glassware and ceramics, pigments, insecticides, rodenticides, and antibilharzial medications[10]	ADME: Slow GI absorption and poor absorption via inhalation; stibine gas[a] is readily absorbed via inhalation[10] Toxicity: Low systemic toxicity; trivalent forms are more toxic than pentavalent forms; stibine gas is highly toxic following inhalation	GI: Severe vomiting, oral mucosal ulceration, hemorrhagic gastroenteritis, watery diarrhea[10] Respiratory: Metal fume fever, pulmonary interstitial fibrosis[10] Hematologic: Hemolysis (stibine gas)[62] Other: Hypovolemia, shock, anemia, myocardial degeneration, proximal renal tubular degeneration, cochlear damage, hepatic injury (stibine gas)[10,62]
Arsenic	Ground water, pesticides and other agricultural products, chemical warfare agents, microelectronics manufacturing, fossil fuel manufacturing[11]	ADME: Well absorbed orally and via inhalation; substantial dermal absorption with prolonged contact or compromised dermal integrity; arsine gas[b] is readily absorbed via inhalation; arsenic crosses placenta and can deposit in bones; primarily excreted via the urine; other routes of excretion include sweat, saliva, milk, and incorporation into hair, epithelium, and nails[10] Toxicity: Trivalent forms are more toxic than pentavalent forms; arsine gas is highly toxic following inhalation[10]	General: Lethargy, death[10] GI: Severe vomiting, oral mucosal ulceration, hemorrhagic gastroenteritis, watery diarrhea, abdominal pain[10] Respiratory: Metal fume fever, pulmonary interstitial fibrosis[10] Hematologic: Hemolysis (arsine gas)[10] Other: Endothelial damage, myocardial degeneration, proximal renal tubular degeneration, pulmonary edema, cardiac arrhythmias, hypovolemia, shock, anemia[10]
Beryllium	Nuclear reactors, x-ray windows, aerospace equipment and fuels, automotive parts, computers and other electronics, dental supplies, telecommunications equipment, welding materials[63]	ADME: Poor oral absorption, some dermal absorption; inhalation is main route of exposure; minimal absorption from lungs, as most inhaled beryllium is sequestered in fibrotic granulomata within the lungs and pulmonary lymph nodes[10] Toxicity: GI, dermal, and pulmonary irritant; minimal systemic toxicity; humans, but no other species, develop pulmonary delayed hypersensitivity (chronic berylliosis)[10]	GI: Erosive gastroenteritis; small intestinal mucosal necrosis[10] Respiratory: Erosive tracheobronchitis, pulmonary fibrosis with granulomata[10] Other: Bone marrow erythroid hypoplasia[10]
Cadmium	Paints and pigments, electroplating, galvanizing, NiCad batteries, jewelry manufacturing, shielding for nuclear reactor cores, fossil fuel combustion[64]	ADME: Poor oral absorption; up to 30% of inhaled cadmium is absorbed; binds to metallothionein in blood and cells; stored in kidney, liver, lung, and pancreas; excreted through urine[10] Toxicity: GI and pulmonary irritant, binds macromolecules of renal tubular epithelium, and interferes with vitamin D and calcium metabolism[10]	GI: Mild gastritis to severe hemorrhagic gastroenteritis, vomiting, diarrhea, oral and esophageal ulceration[10] Respiratory: Tracheobronchitis, pneumonitis, metal fume fever, pulmonary edema[10] Renal: Proximal renal tubular degeneration and necrosis, β_2-microglobulinuria, glucosuria, aminoaciduria[10] Other: Corneal ulceration, ovarian and testicular necrosis[10]

Continued on next page.

Appendix 2 Continued

Metal	Source	Kinetics	Toxic effects
Chromium[c]	Leather tanning materials, pressure-treated lumber, anticorrosive agent for boilers, metal plating, lithography and photography materials, textile manufacturing, welding materials, glass manufacturing, television picture tubes[65]	ADME: Poor oral absorption of trivalent salts; hexavalent salts more readily absorbed orally; up to 85% of inhaled hexavalent chromiumis absorbed via lungs; carried in blood by transferrin or RBCs; crosses placenta; 80% excreted through urine[65] Toxicity: GI and pulmonary irritant; binds macromolecules of renal tubular epithelium; interferes with vitamin D and calcium metabolism[65]	GI: Vomiting; diarrhea; oral, esophageal, and gastric corrosive injury with or without perforation[65] Respiratory: Tracheobronchitis, pneumonitis, metal fume fever, pulmonary edema[65] Renal: Renal tubular degeneration and necrosis[65] Other: Hypovolemia, circulatory failure, hepatitis and hepatic necrosis, metabolic acidosis, methemoglobinemia, thrombocytopenia, anemia, dermal hypersensitivity[65]
Cobalt	Aircraft engine manufacturing, mining equipment and cutting tools, tire manufacturing, paints and pigments, pottery production, diamond polishing, jewelry manufacturing[66]	ADME: Absorbed via skin, ingestion and inhalation; binds to albumin; accumulates in liver and adipose tissue; excreted primarily through urine[66] Toxicity: Acute toxicity uncommon, primarily a chronic disease; oxidative injury to myocardium and hepatocytes; alters calcium channels in cells and interferes with cellular respiration at mitochondrial level[66]	Respiratory: Hard metal disease (pulmonary interstitial fibrosis), cough, dyspnea, asthma[66] Cardiovascular: Enlarged heart, left ventricular failure, pericardial effusion[66,67] GI: Vomiting, diarrhea[66] Other: Polycythemia, hepatocellular necrosis, corneal injury, acute hypersensitivity reactions[66]
Lead	Batteries, welding materials, solders, plastic and rubber manufacturing, leaded gasoline, paints and pigments, ammunition, electrical and radiologic shieldings, radiator repair products, copper and zinc smelting[10]	ADME: Poor oral absorption in adults; inhaled lead is readily absorbed from the lungs; inorganic lead not appreciably absorbed dermally; organic leads well absorbed dermally, orally, and by inhalation[10] Toxicity: Impairs heme synthesis, inpedes vitamin D metabolism, competes with calcium ions, inhibits membrane-associated enzymes[10]	GI: Vomiting, diarrhea, anorexia (signs may be intermittent)[10,68] Neurologic: Ataxia, behavior changes, lethargy, seizures (signs may be intermittent)[10,68] Other: Anemia, weight loss, renal insufficiency, decreased fertility[10,68]
Mercury	Dental amalgams, batteries, instrumentation (eg, thermometers, barometers, calibration instruments), electroplating, jewelry, paints and pigments, photographic materials, semiconductor solar cells, paper pulp manufacturing[69]	ADME: Elemental mercury not absorbed orally; fumes from elemental mercury are absorbed via inhalation; mercury salts have low GI absorption; organic mercurials well absorbed orally; inhaled mercury vapor readily crosses lungs, placenta, and blood-brain barrier; highest levels in kidney; excretion primarily via urine[10] Toxicity: Ingested elemental mercury has low oral toxicity; inhaled mercury vapor causes respiratory irritation; absorbed mercury is damaging to renal tubular epithelium; myocardial degeneration; organic mercury may cause neuronal necrosis and axonal degeneration[10]	GI: Inorganic salts may cause severe oral, esophageal, and gastric corrosive injury; vomiting; diarrhea; abdominal pain[10,69] Respiratory: Metal fume fever, tracheobronchitis, pneumonitis, pulmonary edema, dyspnea[10,17] Neurologic: Organic mercury may cause ataxia, gait abnormalities, visual disturbances, behavior changes, muscle tremor and movement disorders[10,17] Other: Renal insufficiency, glomerulonephritis, cardiac arrhythmias, anemia (blood loss), myocardial failure, abortion, fetal cerebellar and cerebral deformities (organic mercury)[10,17]
Nickel	Electroplating, NiCad batteries, glass, jewelry, coins, cutlery, dental and medical implant manufacturing, pigments, magnetic tape manufacturing, computer components, fossil fuel combustion[70]	ADME: Relatively well absorbed orally; 35% of inhaled nickel is absorbed; distributed to lungs, kidney, and skin; 90% excreted in urine[70] Toxicity: Generally only dermal and inhalation exposures are associated with significant signs; nickel carbonyl inhalation has highest potential to cause serious signs; severe respiratory irritant; myocardial damage reported experimentally[70]	Respiratory: Cough, dyspnea, cyanosis, pulmonary edema, interstitial paralysis[16,71] GI: Vomiting, diarrhea[70] Other: Lethargy, fever, ataxia, seizures, dermatitis[70]
Thallium	Rodenticides (banned in the United States), photoelectric cells, lamps, semiconductors[72]	ADME: Well absorbed orally, dermally, and by inhalation; distributed widely throughout body; 60% excreted via feces, remainder in urine; extensive enterohepatic recirculation results in long half-life[10,73] Toxicity: Replaces potassium in metabolic reactions; cumulative toxicosis possible owing to long half-life; alters cell membrane function and mitochondrial activity; results in dysfunction of a variety of systems, most notably GI, neurologic, and dermatologic[10,73]	GI: Nausea, vomiting, diarrhea, hemorrhagic gastroenteritis, abdominal pain; onset within hours after exposure[74] Neurologic: Disorientation, seizures, behavioral alterations, coma, peripheral neuropathy; onset within 1 week after exposure[74] Dermatologic: Erythema (within hours to days), alopecia initially at areas of friction (eg, axilla, commissures of lips), dermal necrosis, epidermal slough (within days to weeks)[74] Other: Necrotizing pneumonia, renal or hepatic injury, anemia, cardiac dysrhythmias[74]
Zinc	Galvanizing, dyes and pigments, wood preservatives, medicinal agents, televisions, x-ray and computer monitor screens; pesticides, cosmetics manufacturing, dental cements, electroplating, paper manufacturing[75]	ADME: Poor oral absorption; persistence of zinc objects in acidic stomach environment may allow for enhanced absorption; excreted primarily via urine[16,74] Toxicity: Irritant, oxidative damage to RBCs, hemolysis, nephrotoxicosis[16,74]	GI: Vomiting, anorexia, diarrhea[16] Hematologic: Intravascular hemolysis, anemia, icterus, hemoglobinemia[16] Other: Azotemia, hemoglobinuria, proteinuria[16]

[a]Stibine gas is the toxic gas formed when acidic antimony compounds react with hydrogen gas. [b]Arsine gas is the toxic gas formed when arsenic reacts with acids; it is used in the manufacturing of microchips.[11] [c]Trivalent and hexavalent salts.

ADME = Absorption, distribution, metabolism, and excretion. GI = Gastrointestinal.