THE POTENTIAL ENVIRONMENTAL AND PUBLIC HEALTH EFFECTS OF CHEMICAL REGENERATION OF SPENT GRANULAR ACTIVATED CARBON

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Chemical regeneration is one method of regenerating spent granular activated carbon. The chemicals being considered for use are acetic acid, acetone, formic acid, hydrochloric acid, sodium hydroxide, methanol, ethanol, and 2-Propanol. The potential environmental and public health effects of chemical regeneration of spent granular activated carbon causes concern. Information on the eight chemicals and environmental regulations were gathered by doing a literature survey. An evaluation of these chemicals—occupational standards; acute and chronic effects; carcinogenic, reproductive, and mutagenic effects; and environmental fate—showed no anticipated hazards. With the proper preventative and mitigative measures, any unnecessary exposure and adverse effects can be prevented. Chemical regeneration is an alternative to handling spent GAC.
Introduction

Granular Activated Carbon (GAC) is a water treatment process used to remove pesticides from drinking water on O'ahu. After the GAC adsorption capacity is exhausted, it needs to be removed from the treatment facility and either disposed of or regenerated for reuse. Regeneration of spent carbon may be accomplished by thermal, chemical, hot gas, solvent, or biological methods. The City and County of Honolulu Board of Water Supply (BWS) does not currently regenerate their spent carbon and instead, sends it to a local landfill. This necessitates replacement with new "virgin" GAC approximately every 8 to 11 months. Dr. Roger Babcock from the Department of Civil Engineering at the University of Hawai'i at Manoa, is currently doing research on the use of chemicals to regenerate GAC. The chemicals being considered are acetic acid, acetone, formic acid, hydrochloric acid, sodium hydroxide, methanol, ethanol, and 2-Propanol. The use of these chemicals poses the question- what are the environmental and public health concerns regarding the use of chemical regeneration of spent granular activated carbon? This question will be answered in this paper.

Discovery of Contamination on O'ahu

Agricultural chemicals including DBCP(1,2-dibromo-3-chloropropane), EDB (1,2-Dibromomethane), and TCP (1,2,3-trichloropropane), were used for several decades by the sugarcane and pineapple industries on O'ahu. These pesticides were used to control nematodes. Over the years, these chemicals have leached through the soil and rock to our
groundwater resources, contaminating our drinking water, posing a serious public health threat.

On April 7, 1977, a spill of EDB occurred near the Del Monte Corporation's water supply well at Kunia. This spill first brought attention to the possibility of pesticide contamination of ground water in Hawai‘i. In 1979, the Environmental Protection Agency (EPA) asked Hawai‘i, along with four other states, to monitor for DBCP in ground water. The results were negative with a detection limit of 120 ng/l, at 16 sites near pineapple fields (Lau and Mink, 1987). Not until the early eighties, when more sensitive analytical techniques became available, did pesticide contamination of our groundwater become a great concern. It began with detection of DBCP and EDB at the Kunia wells. In 1980, that water supply was shut down. From 1982-83, 10 potable water wells were closed due to DBCP and EDB contamination. In 1983, pesticides were found at the Waipahu and Mililani Wells. Due to the pesticide contamination of our drinking water, water treatment plants employing GAC were constructed to clean-up water from the aquifer before public distribution.

What is GAC?

There are a number of processes that can reduce the concentration of organics in water. Adsorption by GAC beds is by far the most effective technology for the broad spectrum of organics of concern (Martin and Ng, 1984). Besides removing trace organic
contaminants such as pesticides, GAC can also remove organic compounds that cause bad taste and odor.

Activated carbon is a large-surface-area adsorbent that can be used in powdered (PAC) or granular (GAC) form. A bed of GAC is placed in a contactor column and groundwater is passed through the contactor where contaminants are adsorbed onto the carbon surface. Adsorption is the process where molecules adhere to a surface with which they come into contact, due to forces of attraction at the surface. Adsorption is a selective process because different organic compounds adhere to the carbon. Removal efficiencies of adsorbable compounds are usually 100% when the GAC is fresh. Until the adsorption capacity is reached, more water is run through the system until breakthrough has occurred. When breakthrough occurs, adsorption ceases, and the GAC is called "spent" or "exhausted" carbon. The spent GAC must be properly disposed of or regenerated on-site or off-site for reuse. The BWS uses GAC to treat groundwater that has been contaminated with pesticides.

**BWS and GAC use**

The formation of BWS in 1929, initiated the protection of our groundwater resources with monitoring programs including physical, chemical, and microbiological parameters. The BWS uses GAC as a way of treating contaminated groundwater to control the concentrations of volatile organic compounds (VOC's), such as DBCP, EDB, and TCP, in our drinking water. The Hawai'i Department of Health (DOH) maximum
contaminant limits (MCL's) for DBCP, EDB, and TCP are 0.0002, 0.00005, and 2 mg/l. The MCL's are the maximum allowable concentrations of chemicals that can be present in public drinking water supplies. Adsorption onto GAC is an effective removal method for DBCP, EDB, and TCP, as well as natural organic matter (NOM). With GAC treatment, DBCP, EDB, and TCP are removed to the limits of detection. The EPA labels GAC as the best available technology (BAT), for removing seven of the eight VOC's regulated by EPA. In 1986, GAC systems were built and put into service at Mililani and Kunia wells. Later, GAC systems were put in service at the Waipahu wells. The BWS has GAC systems at Mililani (12 contactors: 6 on-line), Kunia (8 contactors: 2 on-line), and Waipahu (18 contactors: 9 on-line). Each of these contactors has a diameter of 12 feet and filled with 20,000 pounds of carbon which has a depth of 10 feet. GAC has a high surface area and adsorbs the contaminants flowing through the GAC system. The BWS disposes of the carbon in a landfill and also sends some back to the manufacturer for regeneration (on a trial basis). The GAC is replaced every 8 to 11 months. Therefore, approximately 170 tons of carbon is being disposed of and replaced or regenerated each year. The BWS is looking towards feasible alternatives in handling the spent GAC. One of the alternatives is to possibly regenerate spent GAC in-situ by chemical regeneration.

Research with Chemical Regeneration at the University of Hawai'i

Regeneration of GAC involves removing the adsorbed compounds (pesticides) from spent activated carbon and restoring the carbon's porous surface structure, so it can be
reused. Chemical regeneration is the use of chemicals (acids and solvents) which allows for desorption of organics from GAC. Regeneration of spent carbon may be accomplished by thermal, chemical, hot gas, solvent, or biological methods.

Thermal reactivation of carbon is the industry standard method for regenerating carbon. By thermal reactivation, adsorbed organics are desorbed by volatilization and then oxidized at high temperatures. During this process, 5-10% of the carbon is lost during each cycle, which becomes a factor in the cost of GAC treatment (Cooney, Nagerl, and Hines, 1983). Biological regeneration of GAC involves chemical regeneration of compounds followed by biological degradation of the desorption fluids, outside or inside of the GAC columns.

Dr. Roger Babcock, a professor in the Department of Civil Engineering at the University of Hawai‘i at Manoa, is currently researching chemical regeneration of spent GAC, using acetic acid, acetone, formic acid, hydrochloric acid, sodium hydroxide, methanol, ethanol, and 2-Propanol. The use of chemicals in regeneration allows desorption of chemicals from GAC. The process involves passing a liquid chemical through the spent carbon, which is able to chemically desorb contaminants that were previously removed during water treatment. The liquid chemical is then drained from the carbon and treated if needed. Treatment of the desorbed fluids could destroy the desorbed contaminants and the chemicals might be recycled. The regenerated carbon would then be flushed with water to remove residual chemicals, be treated if necessary, and the GAC column would be returned to service. Studies at the University of Hawai‘i have shown
successful results, more than 50% regeneration efficiency. At this point many issues arise regarding compliance of environmental regulations, exposure pathways, populations at risk, and the potential environmental and public health effects. The environmental regulations relevant to chemical regeneration of GAC will be discussed in the following section.

**Environmental Regulations**

There are many environmental laws and regulations dedicated to protect the air, water, land, workers, and the public. Environmental regulations need to be considered since the chemicals being used could pose environmental and public health hazards. Some of them are the Safe Drinking Water Act (SDWA), the Clean Water Act (CWA), the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA), the Resource Conservation and Recovery Act (RCRA), and the Occupational Health and Safety Act (OSHA). The following is a brief description of these regulations and how they may apply to chemical regeneration.

The SDWA of 1974 was established to protect the quality of drinking water in the United States. The SDWA was enacted to ensure that public water supply facilities meet minimum national standards for the protection of public health. SDWA protects the quality of drinking water by requiring primary drinking water standards that specify maximum contaminant levels (MCL's) for chemicals that may have adverse health effects on people, and secondary drinking water regulations that specify MCL's having to do
with the aesthetic properties of drinking water. The EPA establishes safe standards for

safe standards for clean drinking water and requires all owners or operators of public water systems to comply with health related (primary) standards. Acetone is the only chemical with a MCL. Although the other chemicals used in chemical regeneration are not regulated under SDWA, they may be a potential public health concern if large amounts were to reach the public through the potable water system. This point is made, because once the GAC has been chemically desorbed, the GAC would be rinsed with water to eliminate any residual content of the chemicals used before putting the GAC column back into service. Monitoring of residual content is necessary to ensure that the concentration of the chemicals are at acceptable and safe levels for distribution. Acetone should be monitored to ensure that the concentrations do not exceed its MCL (700 µg/l). Further research would need to be done on the concentrations allowable in drinking water for the other seven chemicals.

The CWA was passed in 1972, and since then amended (1987) and reauthorized. The main objectives of CWA is to have (1) water quality that provides for the protection and propagation of fish, wildlife, and shellfish, and provides for the recreation in and on the water and (2) to prohibit the discharge of toxic pollutants in toxic amounts (Mackenthun and Bregman, 1992). CWA authorizes EPA to set effluent standards for industries and continue setting water quality standards for all contaminants in surface waters. CWA regulates conventional pollutants, toxic pollutants, and non-conventional pollutants. CWA makes it unlawful for any person/facility to discharge any pollutant from a point
source into navigable waters, unless a permit (National Pollutant Discharge Elimination System) is obtained. The NPDES enforces measures to reduce and control the discharge of pollutants into surface waters. CWA plays a role in the chemical regeneration process because after rinsing the desorbent mixture from the regenerated GAC, the desorption fluids, along with the organic compounds, will be treated if necessary, and disposed of as wastewater. The chemicals being released into the wastewater collection system eventually goes into the ocean (following treatment). The chemicals released this way (if not completely removed during treatment) may be a threat to the marine life in Mamala Bay and to the people who fish and swim in the bay. Monitoring would be necessary before the treated desorption fluid is disposed of into the wastewater system. Monitoring of the concentrations will take into account how much of the chemicals are being disposed of into the wastewater system.

CERCLA was enacted by Congress in 1980. In 1986 it was revised, and renamed Superfund Amendments Reauthorization Act (SARA). This act allows federal and state government to investigate and respond to the release of materials in the environment. CERCLA requires all facilities to respond to releases without delay. Facilities must notify the National Response Center (NRC) immediately, when there is a release of designated hazardous substances in an amount equal to or greater than its reportable quantity. Acetic acid, acetone, formic acid, hydrochloric acid, sodium hydroxide, and methanol are considered hazardous substances under CERCLA.
RCRA was passed in 1976. This law regulates land disposal of hazardous materials. Chemicals are considered hazardous based on criteria specified by EPA. These criteria are ignitability, corrosivity, reactivity, and toxicity. RCRA regulates the activities of generators, transporters, and those who treat, store, or dispose of hazardous waste. EPA issues regulations on methods of treating, storing, and disposing of waste; overseeing the location, design, and construction of facilities; mandating contingency plans to minimize negative impacts from such facilities; setting qualifications for ownership, training, and financial responsibility; and requiring permits for all such facilities (Environmental Protection Agency, 1985). Acetone, formic acid, and methanol, are considered hazardous substances under RCRA. These chemicals would need the proper permits; be transported and stored appropriately; and disposed of in a safe manner.

OSHA (Occupational Safety and Health Act) was established in 1970 to regulate toxic substances and limit human exposure to these toxins in the workplace. The primary purpose of OSHA is to protect workers from workplace hazards. To achieve this goal, the Occupational Safety and Health Administration was formed to set health and safety standards that are enforced by federal and state inspectors. These health and safety standards are studied and recommended by the National Institute of Occupational Safety and Health (NIOSH). OSHA enforces safety and health guidelines by establishing exposure limits and recommending appropriate preventative measures to reduce or eliminate the adverse health and safety effects of these hazards. The permissible exposure limit (PEL) is the time weighted average concentration for an 8 hour day and a 40 hour
workweek, to which nearly all workers may be repeatedly exposed to, day after day, without adverse effect. The short-term exposure limit (STEL) is the concentration to which workers can be exposed to for 15 minutes continuously, with at least one hour between exposure periods. The ceiling (C) is the concentration that should not be exceeded during any part of the work day. The workers at the GAC facility and the wastewater treatment plant should take precautions (personal protective equipment) when working with the chemicals being used in chemical regeneration. The chemical regeneration of GAC will require workers to monitor and control the regeneration process. OSHA will affect how the work is conducted in order to protect the health of workers from the chemicals being used for regeneration.

The relevant regulations affecting chemical regeneration have been discussed. The following section describes the potential exposure pathways and the populations that could be at risk

**Potential Exposure Pathways**

GAC, chemical regeneration, and the regulations that apply to chemical regeneration have been mentioned in the previous sections. The topic of this paper has to do with the potential environmental and public health effects of chemical regeneration and the question arises about which populations could be affected by chemical regeneration. In order to identify those populations at risk, the exposure pathways need to be identified. An exposure pathway is the course a chemical takes from a source of release to an exposed
individual or population. An exposure pathway consists of a source, transport medium, exposure point, and exposure route. There are several possible exposure pathways of chemicals from chemical regeneration.

The source of the exposure pathway would be the chemical regeneration site. If an accidental release occurs, it will occur at this site. The main transport of the chemicals would be via air, surface water and drinking water. The exposure point is the location of potential contact between the chemical and the individual or organism. The exposure points would be residential, business, and recreational areas receiving GAC treated water. Workers at the chemical regeneration facility and wastewater facility will also be considered points of exposure. Another would be the aquatic organisms of Mamala Bay and its recreational users. The last stage of the exposure pathway is the exposure route, the way a chemical comes in contact with a chemical. Exposure to the chemicals occurs via ingestion, inhalation, dermal, and eye contact.

The populations at risk would be 1) the residents and businesses obtaining drinking water from GAC treated sources, 2) the residents living adjacent to the regeneration facilities, workers transporting, storing, and overseeing the chemicals, 4) workers at the wastewater treatment facility, 5) the marine life of Mamala Bay, and 6) the people who eat the fish and shellfish that have been possibly exposed to the chemicals. Also, residents adjacent to the regeneration facilities may be exposed to chemicals in the air. These populations may be affected if the chemicals are released in amounts that could cause concern.
Future exposure pathways may exist if the right amount of chemicals are released at points of exposure: residential homes, businesses, and recreational areas. The homes and businesses most likely affected are the ones whom receive drinking water that has been GAC treated (Mililani, Waipahu, and Kunia). The main routes of exposure would be by ingestion and dermal contact. Again, residents, businesses, schools, etc. adjacent and downwind may be exposed to chemicals in the air. The main routes of exposure would be by inhalation and dermal contact. Contamination at points of exposure would be due to the chemical regeneration process at GAC plants. If the regenerated GAC isn't thoroughly rinsed after chemical regeneration and distributed to the public, residuals may reach the taps of households and businesses.

The chemicals used in chemical regeneration will need to be transported to GAC plants and stored on-site. The workers handling the chemicals can be exposed to the chemicals by inhalation or dermal contact. Occupational Health and Safety standards would need to be enforced, in order to prevent unnecessary exposure to the chemicals. Workers are not the only ones affected, nearby residents could also be affected. If the chemicals are released or a disaster occurs, residents, businesses, and schools adjacent and downwind from the GAC plant will be affected by the chemical release (inhalation).

Another exposure pathway is by wastewater. The rinse water after chemical regeneration, will most likely be disposed of into the sewer system. The rinse water will contain the chemicals used in chemical regeneration. The workers at the wastewater treatment plant may be affected if skin contact occurs or inhalation of fumes. The public
(nearby residents) could also be exposed to any fumes generated at the wastewater treatment facility during treatment. Following treatment, wastewater is ultimately disposed of into the ocean at Mamala Bay. If incomplete treatment occurs, the chemicals could be released into the ocean. Marine inhabitants, such as fishes and shellfish may be affected, and in turn humans may be affected if they eat the fish and shellfish exposed to the chemicals.

**Evaluation of Chemicals**

All contaminants which might be ingested with drinking water are potentially toxic, given a sufficient dose. Therefore, the identification of a potentially hazardous chemical in potable water is important, as is its concentration. Exposure to the eight chemicals being considered for use in regeneration could occur via inhalation, ingestion, and dermal contact. The occupational standards; acute and chronic health effects; carcinogenic, reproductive, and mutagenic effects; and environmental fate of these chemicals are reviewed in the following section.

**ACETIC ACID**

Acetic acid is a colorless liquid or crystals with a sour, vinegar like odor (National Institute of Occupational Safety and Health, 1997).

**Standards:** The PEL for acetic acid is 10 ppm (25 mg/m³), with a STEL 15 ppm (37 mg/m³) (NIOSH, 1997). No U.S. limit has been established for permissible concentration in ambient water. However, EPA has proposed an ambient environmental goal of 300

**Health Effects:** Acetic acid is a highly corrosive liquid. Acetic acid vapor can produce mild to moderate irritation of the eyes, nose, throat, and lungs. Inhalation of concentrated vapors causes damage to the respiratory tract. Ingestion of this acid may cause corrosion of the mouth and gastrointestinal tract. The acute toxic effects are diarrhea, vomiting, ulceration, or bleeding from intestines and circulatory collapse. Exposure to 50 ppm or more is intolerable to most persons and results in intensive lacrimation and irritation of the eyes, nose, and throat, with pharyngeal edema and chronic bronchitis (Hathaway, Proctor, and Hughes, 1996). Eye contact with concentrated acetic acid causes severe irritation, damage and possibly blindness (Royal Society of Chemistry, 1988b). Contact with the skin may cause dermatitis and burns. Death may occur from a dose of 20-30 ml (Patnaik, 1992).

Chronic exposure causes irritation and black coloration of the skin (RSC, 1988b). Repetitive exposure may cause erosion of dental enamel, bronchitis, and eye irritation. Workers exposed to acetic acid for 7-12 years at concentrations of 60 ppm plus one hour daily at 100-260 ppm, experienced slight irritations of the respiratory tract, stomach, and skin, bronchitis, and erosion of teeth (ACGIH, 1986).

Acetic acid is not a carcinogen. No teratogenic effects were found in rabbits following ingestion of apple cider vinegar (RSC, 1988b). It was not mutagenic in peripheral human
lymphocytes and chromosomes of hamster cells (RSC, 1988b). Skin application of acetic acid on mice did not cause local tumors (RSC, 1988b).

**Environmental Fate:** If acetic acid is released in the atmosphere, it will be degraded by reacting with photochemically produced hydroxyl radicals. The half life for this reaction in air is approximately 22 days (HSDB, 1998a). Acetic acid is also removed by wet deposition. Acetic acid has high to moderate mobility in soil. Volatilization may occur from dry soil, but not moist soil. It's main fate process in soil is biodegradation. Acetic acid biodegrades rapidly in soil and water. In aquatic environments, it will not adsorb to sediment or suspended solids. Acetic acid shows no potential for biological accumulation or food chain contamination (Prager, 1995).

**Summary:** The main threats of acetic acid will be to the regeneration workers. However, standard practices for many industries where acids are used can prevent unnecessary exposure and any harmful effects. In the unlikely event of an accidental release, acetic acid will be degraded easily and rapidly. The marine life of Mamala Bay and the people who gather food from the bay are in no threat. Therefore, no environmental and public health effects are anticipated.

**ACETONE**

Acetone is a volatile, highly flammable, colorless liquid with a fragrant, mint-like odor and a pungent, sweetish taste (NIOSH, 1997).

**Standards:** NIOSH's (1997) TWA is 250 ppm (590 mg/m³). NIOSH's recommended TWA is based on concentrations for a 10 hour workday during a 40 hour workweek.
OSHA's PEL is 750 ppm (1,780 mg/m³) with a STEL 1,000 ppm (2,380 mg/m³) (NIOSH, 1997). No U.S. limit has been established for permissible concentrations in ambient water. But, Massachusetts and Maryland has set their permissible concentrations at 250 µg/l and 3600 µg/l (Sittig, 1991a). Under CERCLA, acetone has a reportable quantity of 5,000 lb (EPA, 1990). Acetone is also considered a hazardous substance under RCRA. Acetone's MCL is 700 mg/l in drinking water.

Health Effects: Acetone is considered to be of low risk to health because few adverse health effects have been reported despite widespread use for many years (Hathaway, et al., 1996). Acetone is absorbed by all routes of exposure, although less so through the skin (Clayton and Clayton, 1993). The most likely route of exposure is inhalation of vapor.

Acute toxic effects include headaches, drowsiness, and irritation of eyes, nose, and chest. Subjects exposed to 500 ppm were aware of odor and exhibited no effects, at 1,000 ppm mild eye irritation occurred (Hathaway et al., 1996). Exposure to 12,000 ppm can cause central nervous system depression, with symptoms of headaches and drowsiness (RSC, 1988a). When ingested, acetone causes gastric irritation, pain and vomiting. Estimated minimum lethal dose by ingestion is 50 ml (Patnaik, 1992). Effects are reversible following removal from exposure.

Symptoms of chronic occupational exposure includes loss of appetite, heartburn, eye irritation, bronchitis, and gastritis. Continued dermal contact leads to dryness of the skin, producing irritation, rash, and redness. Acetone is rapidly eliminated from the body, but
slowly enough that it may accumulate over the work week and return to normal levels over the weekend (Clayton and Clayton, 1993). Several occupational studies have shown that chronic exposure to acetone appears to cause no permanent adverse health effects. Health effects from long-term exposures are known mostly from animal studies. Kidney, liver, and nerve damage, increased birth defects, and lowered ability to reproduce (males only) occurred in animals exposed long-term (Agency for Toxic Substances and Disease Registry, 1994). It is not known if people would have these same effects.

Exposure to acetone causes concern in pregnant women. Pregnant women exposed to about 30 mg/m³ and 300 mg/m³ produced embryotropic effects ranging from high lipid levels to embryotoxic effects (RSC, 1988b). There is no evidence to suggest that acetone is capable of inducing carcinogenicity in humans or laboratory animals. The Department of Health and Human Services, the International Agency for Research on Cancer, and the EPA have not classified acetone for carcinogenicity. Acetone does not cause mutagenic effects.

**Environmental Fate:** In the atmosphere, acetone will be lost by photolysis and reaction with photochemically produced hydroxy radicals (Howard, 1990). The half-life estimates from these combined processes average 22 days. It will also be washed out by rain. Acetone will volatilize and leach into the ground, if a spill was to occur. It is expected to biodegrade under both aerobic and anaerobic conditions. Acetone readily biodegrades in soil and in water. In aquatic environments, the main removal process of
acetone is volatilization, but biodegradation of the chemical is also expected to occur. Bioconcentration in aquatic organisms are low.

**Summary:** The main threats of acetone will be to the regeneration workers. However, standard practices for many industries where solvents are used can prevent unnecessary exposure and harmful effects. In the event of an accidental release, acetone will be degraded readily. If trace amounts reach aquatic environments, no harmful effects are anticipated. No environmental and public health effects are anticipated.

**FORMIC ACID**

Formic acid is a colorless liquid with a pungent and penetrating odor (NIOSH, 1997).

**Standards:** The PEL for formic acid is 5 ppm (9.4 mg/m³) (NIOSH, 1997), with a STEL 10 ppm (19 mg/m³) (Hathaway et. al, 1996). The PEL and STEL are recommended to prevent irritation of eyes and respiratory passages, and possibly of the skin. No U.S. limit has been established for permissible ambient water concentrations. But, EPA has proposed permissible ambient goal of 124 μg/l (Sittig, 1991a). Under CERCLA, formic acid has a reportable quantity of 5,000 lb (EPA, 1990). Formic acid is considered a hazardous substance under RCRA.

**Health Effects:** Formic acid is a low to moderately toxic compound. Inhalation of vapors, mists, or aerosols, may result in increased nasal discharge, cough, throat discomfort, and pulmonary edema. Formic acid is moderately toxic orally, causing salivation, vomiting, diarrhea, and gastritis. If ingested, formic acid can cause salivation, vomiting, burning sensation in the gastrointestinal tract, severe pain, and circulatory
collapse. Atmospheric concentrations as low as 32 mg/l are corrosive to the skin (RSC, 1988b). Occupational exposure to approximately 15 ppm formic acid caused nausea, but apparently no other ill effects (ACGIH, 1986). Systemic absorption of large doses of formic acid may result in damage to the liver, kidneys, and eyes. Dermal contact causes irritation, dermatitis, and ulceration of membranes.

Occupational exposure to 15 ppm of formic acid cause nausea but apparently no other ill effects (ACGIH, 1986). Formic acid is a sensitizer for some individuals, allergic contact dermatitis or possible asthma could develop in these individuals.

No information is available concerning the carcinogenicity of formic acid. There was no toxic and teratogenic effects of formic acid in fertilized chicken eggs injected with up to 20 mg (RSC, 1988b). Formic acid does not cause mutagenecity.

**Environmental Fate:** In the atmosphere, formic acid reacts with photochemically produced hydroxy radicals with a half life of 34 days (Howard, 1997). Formic acid will also be physically removed by rain and dissolve in cloud water and aerosols. If released on land, formic acid should leach into some soils where it would biodegrade or volatilize rapidly from dry soil and surfaces. Formic acid has been shown to adsorb to sediment and biodegrade in natural waters. In water, formic acid should biodegrade. It should not adsorb significantly in sediment. Bioconcentration is aquatic organisms is not an important fate process.

**Summary:** The main threats of acetic acid will be to the generation of workers. However, standard practices for many industries where acids are used can prevent unnecessary exposure and any harmful effects. In the event of an accidental release,
formic acid will be biodegraded rapidly. Therefore, no environmental and public health effects are anticipated.

**HYDROCHLORIC ACID**

Hydrochloric acid is a corrosive, colorless, non-flammable gas which fumes in air, and a pungent odor (NIOSH, 1997).

**Standards:** The ceiling value for hydrochloric acid is 5 ppm (7.5 mg/m³) (NIOSH, 1997). Ceiling limit is low to prevent toxic injury from exposure to hydrochloric acid. No U.S. limit has been established for permissible concentrations in water. Under CERCLA, hydrochloric acid has a reportable quantity of 5,000 lb (EPA, 1990).

**Health Effects:** Hydrochloric acid is an irritant. Hydrochloric acid is highly water soluble and acute exposure occurs in the upper respiratory tract. Exposure to the gas causes cough, burning of the throat, inflammation and ulceration of the nose, throat, and larynx. At low concentrations (less than 5 ppm) no harmful effects occurred. In humans, exposure to 50-100 ppm for 1 hour is barely tolerable, 35 ppm for a short while causes throat irritation, and 10 ppm was tolerable, although immediate irritation has been reported at concentrations over 5 ppm (RSC, 1988b). Ingestion of concentrated hydrochloric acid can cause severe and fatal burns to the gastrointestinal tract. Exposure to the eyes may cause burns, reduced vision or total blindness.

No serious effects have been associated with chronic exposure to low concentrations of hydrochloric acid (Finkel, 1983) Chronic exposure to higher concentrations may cause
erosion of dental enamel, nasal ulceration, dermatitis, and chronic bronchitis. Frequent contact with the dilute acid may cause dermatitis and photosensitisation.

Studies of rats showed no carcinogenic response (RCS, 1988b). No information was available concerning the mutagenecity and reproductive hazards of hydrochloric acid. Inhalation experiments in rats showed no carcinogenic response (RCS, 1988b).

**Environmental Fate:** When hydrochloric acid is spilled onto soil, it will begin to infiltrate. The amount of water in the soil will have affect the movement of the acid in the soil. During transport, the acid will dissolve some of the soil material. In the process, the acid will be neutralized, but significant amounts will remain for transport downward to the water table. In water the hydrogen chloride will dissociate almost completely (International Programme on Chemical Safety, 1982).

**Summary:** The main threats of hydrochloric acid will be to the generation of workers. However, standard practices for many industries where acids are used can prevent unnecessary exposure and any harmful effects. In the event of an accidental release, hydrochloric acid will be biodegraded rapidly. Therefore, no environmental and public health effects are anticipated.

**SODIUM HYDROXIDE**

Sodium hydroxide is a colorless to white, odorless solid (NIOSH, 1997).

**Standards:** A ceiling level for exposure to sodium hydroxide has been adopted by OSHA as 2 mg/m³ (NIOSH, 1997). This level is recommended by NIOSH and ACGIH. Sodium hydroxide is odorless, therefore no odor threshold has been established. No U.S.
limit has been set for permissible concentrations in ambient water. However, the EPA has recommended criteria for pH: to protect freshwater aquatic life, pH 6.5-9.0; to protect saltwater aquatic life, pH 6.5-8.5 and drinking water, pH 5-9 (Sittig, 1991b). Under CERCLA, sodium hydroxide has a reportable quantity of 1,000 lb (454 kg) (EPA, 1990).

**Health Effects:** Sodium hydroxide is highly corrosive to all body tissues. Direct skin and eye contact occurs most frequently. The degree of damage is proportional to concentration and length of exposure, and can range from dermatitis from chronic exposure to weak solutions, to deep serious burns with the solid material (Cook, 1989). Inhalation of the dust or concentrated mist may cause irritation of nose, sinuses, and damage to the respiratory tract. A concentration of 2 mg/m³ is reported as being noticeably but not excessively irritating (RSC, 1988b). Ingestion leads to burning of the lips, mouth, tongue, stomach, and swelling of the throat. An oral toxicity of 5-10% solution of sodium hydroxide was low in test animals, but high dosages at greater concentrations can cause vomiting, prostration, collapse, and abdominal pain following exposure (Patnaik, 1992). Contact with the eye can cause severe irritation and mild to severe corneal scarring to fluid accumulation, disintegration, and ulceration. Solutions of 25-50% cause a sensation of irritation within approximately 3 minutes, 4% solution causes irritation after several hours (Hathaway et al., 1996). If not promptly removed, severe burns with deep ulceration will occur.

Chronic exposures to high concentrations of sodium hydroxide may lead to ulcerations of nasal passages. Repeated exposure to mists or aerosols of sodium hydroxide can
produce a tolerance to its irritative effects. Repeated exposure to the skin can lead to dermatitis. Skin contact should be avoided with the chemical.

Sodium hydroxide has been found to be nonmutagenic. Sodium hydroxide was not teratogenic in mice (RSC, 1988b). In some cases, sodium hydroxide has been implicated as a possible cancer causing agent of the oesophagus 12 to 42 years after ingestion, but it may be due to tissue destruction rather than the chemical itself (RSC, 1988b).

**Environmental Fate:** No information on its environmental fate was found.

**Summary:** The main threats of sodium hydroxide will be to the regeneration workers. Occupational health and safety standards should be followed in order to prevent any unnecessary exposure to the chemical. Information on the environmental fate of sodium hydroxide is absent, therefore no environmental and public health effects are anticipated.

**METHANOL**

Methanol is a clear, water-white liquid with a mild odor at ambient temperature (NIOSH, 1997).

**Standards:** The PEL is 200 ppm (260 mg/m³) and the STEL is 250 ppm (325 mg/m³). Both ACGIH and OSHA warn against skin contact. Odor threshold has been reported at 100 ppm. No U.S. limit has been set for permissible concentrations in ambient water. But, EPA has proposed a permissible ambient goal of 3600 µg/l (Sittig, 1991b). Under CERCLA, methanol has a reportable quantity of 5,000 lb (EPA, 1990). Methanol is considered a hazardous substance under RCRA.
**Health Effects:** Methanol poisoning can be divided into three stages. First, causing narcosis, involving drowsiness, mild irritation of the eyes and mucous membranes. The second stage involves a latent period of about 10-15 hours, and then leading to more serious effects are damage to the central nervous system, resulting in nausea, vomiting, dizziness, headache, temporary or permanent blindness, acidosis, and liver and kidney damage. Inhalation of methanol at 200 ppm should not cause problems, but above that level headache, nausea, vomiting, and irritation of mucous membranes may occur (RSC, 1988a). High concentrations may damage the central nervous system and cause vision impairment. Swallowing of methanol usually results from the ingestion of adulterated methanol. An oral dose of 100-250 ml is usually fatal, but death has occurred from a dose as little as 30 ml (Sittig, 1991b). The lethal dose of methanol in untreated individuals is estimated to be approximately 70-130 ml, for the average individual (Wexler, 1998b). Liquid and vapor states of methanol are very hazardous to the eyes since methanol seems to target the optic nerve and retina.

Methanol is eliminated from the body more slowly than ethanol, resulting in cumulative toxicity from daily exposures (ACGIH, 1986). Continuous skin contact may cause dermatitis with dryness and cracking of the skin (Sax, 1984). Repeated exposures in the range of 200-375 ppm have been associated with headaches, and 1,200-8,300 ppm with damaged vision (Clayton and Clayton, 1982).

Increased congenital malformations were observed at high vapor concentrations of 10,000 ppm, but no effects were observed at 5,000 ppm (RSC, 1988a). Workers exposed
to methanol showed no increased incidence of cancer (RSC, 1988a). Methanol is not mutagenic.

**Environmental Fate:** The major environmental fate processes for methanol in water are biodegradation and volatilization. However, high concentrations will result in death or serious effects in aquatic animal life. Methanol has a high mobility in soil, therefore it's expected to biodegrade immediately under aerobic and anaerobic conditions. With a half-life of 17.8 days in ambient air, methanol will degrade by photochemical reactions (Howard, 1990). Due to its water solubility, rain is expected to physically remove some methanol from the air (IPCS, 1997).

**Summary:** The main threats of methanol will be to the regeneration workers. Standard practices in industries using alcohols can prevent unnecessary exposure and harmful effects. In the event of an accidental spill, methanol will biodegrade easily and rapidly. Therefore, no environmental and public health effects anticipated.

**ETHANOL**

Ethanol is a clear, colorless liquid with a weak, ethereal, vinous liquid (NIOSH, 1997).

**Standards:** The PEL value for ethanol is 1,000 ppm (1,900 mg/m³) (RSC, 1988a). No STEL or ceiling value has been established. The odor threshold is at 10 ppm. Ethanol is not considered a hazardous substance under CERCLA and RCRA.

**Health Effects:** The effects from inhalation and dermal contact are not serious under reasonable conditions of handling and use (Clayton and Clayton, 1982). The toxicity of ethanol is much lower than that of methanol or propanol. The major industrial exposure
to ethanol is by inhalation. The effects of ethanol if inhaled are not too serious under reasonable laboratory use. Prolonged inhalation over 5,000 ppm may cause headache, drowsiness, tremors, narcotic effects, as well as irritation to the eyes and upper respiratory tract (RCS, 1988a). High concentrations of 5,000-10,000 ppm (10-20 mg/l) can cause coughing and eye and nose irritation (Wexler, 1998a). Exposure up to 15,000 ppm (30 mg/l) causes lacrimation and coughing, and concentration up to 20,000 ppm (40 mg/l) were intolerable. Ingestion of large doses can lead to alcohol poisoning. Symptoms can be much more serious at high blood alcohol levels. Acute exposures greater than the PEL produce symptoms of central nervous system depression and eyes, nose, and throat irritation. In direct contact with skin, ethanol is not irritating.

Effects of chronic ethanol ingestion leads to dependence, tolerance, and cirrhosis of the liver. Chronic exposure can also lead to possible death.

Pregnant women should not ingest alcohol, because it may lead to congenital malformations, or fetal alcohol syndrome. There have been no reports on fetal alcohol syndrome from occupational exposure (Wexler, 1998a). Ethanol does not cause carcinogenic effects, but it may be a promoter (RSC, 1988a). Reproductive effects have been observed in experimental animals treated with high doses of ethanol during gestation (RSC, 1988a).

**Environmental Fate:** In the atmosphere, it will photodegrade in hours to an estimated 4 to 6 days. Rainout is an important transport of ethanol, due to its solubility in water. If ethanol is spilled on land, it is likely to volatilize, biodegrade, and leach into the
groundwater. If degradation isn't rapid, it will leach into the groundwater. When released into water, ethanol will volatilize (estimated half life is 6 days) and biodegrade (HSDB, 1998c). Ethanol isn't expected to be taken up by sediments or bioaccumulate in fish.

**Summary:** The main threats of ethanol will be to the main regeneration workers. The use of occupational safety guidelines can prevent unnecessary exposure and harmful effects. In the case of an accidental release, ethanol will biodegrade very rapidly. No environmental and public health effects anticipated.

**2-PROPANOL**

2-Propanol is a colorless liquid with the odor of rubbing alcohol (NIOSH, 1997).

**Standards:** The PEL is 400 ppm (980 mg/m³), with a STEL of 500 ppm (1,224 mg/m³) (NIOSH, 1997). The PEL is expected to minimize the potential for inducing narcotic effects or significant irritation of eyes and upper respiratory tract in workers. 2-Propanol is not considered a hazardous substance under CERCLA and RCRA.

**Health Effects:** Exposure to 2-Propanol, or isopropanol occurs most frequently in the occupational environment. The exposure risks are quite high, but its toxicity is comparatively low. Humans exposed to 400 ppm for 3 to 5 minutes experienced mild irritation of the eyes, nose, and throat; at 800 ppm, irritation was not severe, but the majority of subjects considered the atmosphere uncomfortable (Hathaway et al, 1996). Inhalation produces mild irritation in the eyes and nose. Ingestion causes drowsiness, dizziness, and nausea. Doses ranging from 100 to 166 ml may be fatal to humans (Patnaik, 1992). Symptoms of overexposure include headaches, drowsiness, loss of
coordination, followed by lowered blood pressure, lowered body temperature, kidney and liver dysfunction, coma, and death.

Repeated contact of 2-Propanol with the skin can cause defatting dermatitis with drying and cracking. There have been rare cases of allergic contact dermatitis.

2-Propanol has not been tested in animals to assess carcinogenicity (Hathaway et al., 1996). There were no reproductive effects observed in rats who were treated with doses of up to 1,200 mg/kg/day on gestation or in rabbits exposed to 480 mg/kg/day on gestation (Hathaway et al., 1996). No information available on mutagenic effects.

Environmental Fate: When released into the atmosphere, it will photodegrade with an estimated half-life of one to several days (Howard, 1990). Due to its high solubility in water, rainout may play a significant role in the transport of 2-Propanol from air to soil or water (HSDB, 1998d). If 2-Propanol is released on land, it is likely to evaporate and leach into the ground due to its high vapor pressure and low adsorption to soil. When released in water, 2-propanol will volatilize and biodegrade. Laboratory tests suggest that it does not have a long life in water. Isopropanol has an estimated half life of 5.4 days in water (IPCS, 1990). It is not expected to adsorb to sediment or bioconcentrate in fish. Degradation in soil and groundwater has not been determined (IPCS, 1990).

Summary: The main threats of isopropanol will be to the regeneration workers. The use of occupational health and safety guidelines can prevent unnecessary exposure and harmful effects. In the case of an accidental release, isopropanol will be degraded easily and rapidly. No environmental and public health effects anticipated.
Preventative Measures

If chemical regeneration is used, we must consider preventative measures. Preventative measures need to be applied to ensure the safety of the public and the environment. We need to ensure that the regeneration system does not release any chemicals into the groundwater, surface water, drinking water, the ocean, wastewater, the air, and the soil. In order to protect the potential populations at risk, sampling and monitoring of the GAC treated water should be tested for residual content of chemicals. Residual content should be at an acceptable level, for which it doesn't pose a public health threat. The solvents and chemicals must be compatible with the GAC system, too. The materials used to make the GAC system may not be able to withstand acetone or the acids being used. Studies on how the chemicals and materials of the GAC system react with one another should be done, before going full-scale. The acids or the solvents may cause leaching of the materials used in the GAC system. In the occupational environment, the workers should take precautions when working with or near the chemicals.

Personal protective equipment should be used, such as acid resistant clothing, gloves, eye goggles, and respirators. The proper storage and handling procedures should be utilized in order to protect the worker and the public in the surrounding environment.

Acetic acid: Acetic acid should be stored in stainless steel containers away from heat. Neoprene, butyl, or natural rubber gloves should be worn. Fires can be extinguished using carbon dioxide, dry chemical, alcohol foam, or water.
**Acetone:** Acetone should be stored away from sources of ignition or direct sunlight in a well ventilated area. Large quantities should be stored away from oxidizing material, mineral acids, and chloroform. Protective clothing should be worn especially eye protection and natural rubber or neoprene gloves. For small fires, dry powder, alcohol resistant foam, halon, or carbon dioxide extinguishers should be used. Large spills can be controlled by alcohol resistant foam. The most effective countermeasure is the natural biological biodegradation of acetone (Prager, 1995).

**Formic acid:** Formic acid should be kept in a cool, dry, well ventilated area, away from heat, sulphuric acid, and other oxidizing materials. Formic acid fire can be extinguished by carbon dioxide, dry chemical powder, alcohol foam, or water. Needs to be handled with an approved respirator, along with butyl or neoprene gloves.

**Hydrochloric acid:** should be kept in a cool, dry, well ventilated area, away from heat, sulphuric acid, and other oxidizing materials. Needs to be handled with an approved respirator, along with butyl or neoprene gloves. Hydrochloric acid is not combustible, so an extinguisher appropriate for the surrounding conditions should be used.

**Sodium hydroxide:** Sodium hydroxide should be stored in a dry place, protected against moisture, water, and away from acids, metals, explosives, organic peroxides, and easily ignitable materials. An approved respirator and butyl or neoprene gloves and safety goggles should be worn when handling sodium hydroxide. Water and halon fire extinguishers may be used as long as it doesn't come into contact with sodium hydroxide itself.
**Ethanol and Methanol:** Ethanol and methanol should be stored in clean carbon-steel tanks or drums, away from sources of ignition and oxidants. Good ventilation should prevent the formation of harmful concentrations of alcohol vapor. Gloves should be used when handling methanol and ethanol, especially if there is a possibility of prolonged skin contact. Dry chemical powder, carbon dioxide, alcohol resistant foams, or water can be used to extinguish any fires.

**2-Propanol:** 2-Propanol should not be stored in aluminum containers. It can be stored in steel drums or baked phenolic lined steel drums.

**Mitigative Measures**

Mitigative measures would need to be taken if any public threat occurred, such as an accidental release of the chemicals being used in chemical regeneration. The proper permits should be acquired as well as following procedures regulated by environmental laws, which were previously discussed. Under RCRA, acetone, formic acid, and methanol should have the proper permits for transportation, storage, and disposal. Material Safety Data Sheets (MSDS) should be provided for the workers. If an accident should occur, the public has the right to know about the problem. The Emergency Planning and Community Right to Know Act enables the public to acquire data on the chemicals being used and released into their community and environment. Under CERCLA, facilities are required to notify the National Response Center (NRC) immediately, when there is a release of designated hazardous substances in an amount equal to or greater than its
reportable quantity. Acetic acid, acetone, formic acid, hydrochloric acid, and methanol have a reportable quantity of 5,000 lb (2270 kg). Sodium hydroxide has a reportable quantity of 1,000 lb (454 kg). Emergency planning procedures prepared by the federal, state, and local governments and industry should be followed to prevent accidents from becoming a major public health hazard. Other agencies to notify are the Hazardous Materials Team (HAZMAT) and the proper water, wildlife, and health agencies.

Recommendations

It was mentioned earlier that the chemicals in the drinking water after chemical regeneration would have to be at safe and acceptable levels in order to have no adverse health effects to the public. Acetone is the only chemical with a MCL (700 µg/l) for drinking water. For the other seven chemicals, it is recommended that preliminary remediation goals (PRG) be used as a recommended MCL. The PRG combines current EPA toxicity values with "standard" exposure factors to estimate contaminant concentrations in environmental media (soil, air, and water) that are considered protective to humans, including sensitive groups, over a lifetime. The following shows the recommended levels for drinking water (EPA, 1998):

<table>
<thead>
<tr>
<th>Chemical</th>
<th>Recommended level (µg/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acetone</td>
<td>$7 \times 10^2$ (current MCL)</td>
</tr>
<tr>
<td>Acetic Acid</td>
<td>$1.28 \times 10^{-4}$ (recommended MCL)</td>
</tr>
<tr>
<td>Formic Acid</td>
<td>$7.3 \times 10^4$</td>
</tr>
</tbody>
</table>
Ethanol & $1.75 \times 10^{-3}$ (Appendix I) \\
Methanol & $1.8 \times 10^4$ \\
2-Propanol & $1.8 \times 10^4$* \\
Hydrochloric Acid & pH 6.5 - 8.5** \\
Sodium Hydroxide & pH 6.5 - 8.5** \\

*These chemicals do not have a PRG. Dr. Barbara Brooks and Dr. Leslie Au (1998) recommended using the PRG's of similar chemicals. Therefore, the recommended level for methanol is also used for 2-Propanol, due to their similar characteristics. Methanol is more toxic than 2-Propanol, so it should be safe to say that the PRG for methanol could be used for 2-Propanol (Brooks and Au, 1998).  
**Hydrochloric acid and sodium hydroxide dissociate in water, thus changing acidity of water. The pH values are secondary MCL's for drinking water. It is recommended that the desorbed fluids have a pH level 6.5-8.5 before disposing it into the wastewater system.

Monitoring of residual content is necessary to ensure that the concentration of the chemicals are at acceptable and safe levels for distribution. Before distributing the treated water into the drinking water supply and wastewater system, the concentration of the chemicals should be less than the recommended levels just mentioned. Additional research should be done on the amount of rinsing of the chemically treated GAC, to keep concentrations at the recommended levels.

**Conclusion**

Based upon the literature research, the use of chemical regeneration should be researched further. The findings have shown that the chemicals being considered for use, acetic acid, acetone, formic acid, hydrochloric acid, sodium hydroxide, methanol, ethanol, and 2-Propanol, pose little environmental and public health threats. These chemicals
biodegrade readily in the air, soil, and water. It is necessary to follow occupational safety and health standards to prevent any unnecessary exposure to the worker and the public. Monitoring of the chemicals being used is a vital part of tracking and preventing any release into the environment.
Appendix I

Safe human dose = \( \text{NOAEL} \times \text{body weight} \)

safety factor

\[
\text{Safe water concentrations (ingestion)} = \frac{\text{Safe human dose}}{2 \text{ liters/day}}
\]

\[
5 \text{ mg/dL} = \text{NOAEL (No observed adverse effects level)}
\]

\[
\frac{1 \text{ g ethanol}}{kg \text{ body weight}} (70 \text{ kg body weight}) = 70 \text{ g ethanol}
\]

\[
\frac{70 \text{ g ethanol}}{100 \text{ mg/dL} / 1 \text{ mg/dL}} = 3.5 \text{ g ethanol}
\]

\[
\frac{3.500 \text{ mg}}{2L} = 1,750 \text{ mg/L or } 1.75 \times 10^{-3} \text{ug/L}
\]

At 5 mg/dL there were mild effects such as decreased inhibition and slight incoordination (TOMES, 1997). Because there was no NOAEL for ethanol, the lowest concentration with the least effects was used to get an estimate of a recommended safe level for drinking water. Dr. Barbara Brooks and Dr. Leslie Au (1997) estimated that 1,750 mg/L would be the recommended safe level.
<table>
<thead>
<tr>
<th>Chemical Name</th>
<th>Occupational Standard</th>
<th>CERCLA</th>
<th>RCRA</th>
<th>Carcinogenecity</th>
<th>Teratogenecity</th>
<th>Mutagenecity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acetic Acid</td>
<td>PEL= 10 ppm</td>
<td>5,000 lb</td>
<td></td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Acetone</td>
<td>PEL= 750 ppm</td>
<td>5,000 lb</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Formic Acid</td>
<td>PEL= 5 ppm</td>
<td>5,000 lb</td>
<td>Yes</td>
<td>No info. available</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Hydrochloric Acid</td>
<td>C= 5 ppm</td>
<td>5,000 lb</td>
<td></td>
<td>No</td>
<td>No info. available</td>
<td>No info. available</td>
</tr>
<tr>
<td>Sodium Hydroxide</td>
<td>C= 2 mg/cu m</td>
<td>1,000 lb</td>
<td>Yes</td>
<td>Possible</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Methanol</td>
<td>PEL= 200 ppm</td>
<td></td>
<td></td>
<td>No</td>
<td>&gt;10,000 ppm</td>
<td>No</td>
</tr>
<tr>
<td>Ethanol</td>
<td>PEL= 1,000 ppm</td>
<td></td>
<td></td>
<td>Promoter</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>2-Propanol</td>
<td>PEL= 400 ppm</td>
<td></td>
<td></td>
<td>No</td>
<td>No</td>
<td>No info. available</td>
</tr>
</tbody>
</table>
References

ACGIH (1986) Documentation of the Threshold Limit Values and Biological Exposure Indicies, 5th Ed., American Conference of Governmental Industrial Hygienists, Inc., Cincinnati, OH.


Environmental Protection Agency (1985) RCRA Information on Hazardous Wastes Publicly Owned Treatment Works, Environmental Protection Agency, Washington, DC.


The Royal Society of Chemistry (1988a) Chemical Safety Data Sheets Vol. 1: Solvents

The Royal Society of Chemistry (1988b) Chemical Safety Data Sheets Vol. 3: Corrosives and Irritants


Internet references:

http://www.epa.gov/region09/waste/sfund/prg/SL-01.htm

HSDB: Hazardous Substance Data Bank- Acetic Acid

HSDB: Hazardous Substance Data Bank-Formic Acid

HSDB: Hazardous Substance Data Bank-Methanol

HSDB (1998d): Hazardous Substance Data Bank-2-Propanol
http://toxnet.nlm.nih.gov/servlets...iewers.resultPageViewer?999_1007_0.