Appendix Element Five
Groundwater Protection & Management

5-A. **"Chemical Specific Consultation for Ethylene Dibromide and Dichloropropane." Prepared by Mike Allred, Ph.D., Emergency Response and Scientific Assessment Branch, Division of Toxicology, Agency for Toxic Substances and Disease Registry. July 31, 1998.**

5-B. **"Health Assessment of Well Water Contaminants in Whatcom County, Washington." November 1998.**

5-C. **GIS Map. Nitrate Concentrations in Groundwater.**

Chemical Specific Consultation for Ethylene Dibromide and Dichloropropane

July 31, 1998

Prepared by:
Mike Allred, Ph.D.
Emergency Response and Scientific Assessment Branch
Division of Toxicology
Agency for Toxic Substances and Disease Registry

I. Background and Statement of Issues

Region X of the ATSDR Office of Regional Operations (ORO) has requested the Division of Toxicology, ATSDR, to review the the toxicology of ethylene dibromide (EDB) and 1,2-dichloropropane (DCP) and discuss the potential for these chemicals to pose a health threat via inhalation and dermal contact exposures to contaminated groundwater. The exposure scenarios of concern involve the use of contaminated groundwater for domestic cooking, showering, or bathing purposes. Concentrations as high as 6.17 parts per billion (ppb or $\mu g/L$) of EDB and 24 $\mu g/L$ for 1,2-DCP have been reported (ATSDR 1998).

This consultation is not intended to predict or evaluate potential health effects associated with site-specific exposures. Such exposures can only be evaluated via thorough site-specific sampling and pathways analyses.

II. Discussion

Background

In the 1970s and early 1980s, ethylene dibromide, also known as EDB or 1,2-dibromoethane, was used as a soil fumigant to kill insects and nematodes that infested fruits, vegetables, and grain crops. EDB was also used to kill fruit flies on citrus fruits, mangoes, and papayas after they were picked. Another common use of EDB was as a "scavenger" in leaded gasolines to convert lead oxides to lead halides which were more easily released with engine exhausts and improved fuel economy. The use of EDB in the United States has decreased since the U.S. Environmental Protection Agency banned the use of EDB as a soil and grain fumigant in 1984 (ATSDR 1992; HSDB 1998) and required that lead be removed from gasoline by 1996 (EPA 1995).

1,2-dichloropropane, also known as DCP, is used as an insecticide for crops and stored grain, and as a soil fumigant to kill nematodes. Other uses of DCP are as an oil, fat, and degreasing solvent, and in the past as a scavenger in leaded gasolines (HSDB 1998). When EDB, as a commercial chemical product or a manufacturing chemical intermediate, becomes a waste, it must be managed according to Federal or State hazardous waste regulations (ATSDR 1989; HSDB 1998).

EDB and DCP are readily soluble in water (0.40 and 0.26 grams per 100 ml of water at 20 degrees Celsius, respectively; HSDB 1998), have low octanol-water partition coefficients (86 and 100, respectively; HSDB 1992), and high vapor pressures (11 and 50 mm Hg at 25 degrees Celsius, respectively; HSDB 1998). Both chemicals are readily soluble in groundwater and readily migrate as groundwater moves through the soil. With low lipid solubility, these chemicals will not sorb onto soil to any great extent; therefore losses to soil and sediment are not expected to remove large amounts of EDB and DCP from the environment. Likewise, EDB and DCP are not expected to bioconcentrate or biomagnify. As a result of their volatility, EDB and DCP rapidly volatilize from surface water into the atmosphere (ATSDR 1989, 1992). Both EDB and DCP are heavier than water; therefore, any free product is present in the groundwater will sink below groundwater to any impermeable rock or clay substrata.

EDB toxicity

In animal studies, EDB is rapidly absorbed and distributed throughout the body. It is retained to a limited extent in the kidneys, liver, and stomach, regardless of the route of exposure. EDB is metabolized to active forms capable of inducing toxic effects by either of two systems - the microsomal monooxygenase system (cytochrome P-450 oxidation) and the cytosolic activation system (glutathione conjugation). EDB is rapidly eliminated in the urine, with smaller amounts being passed in liver bile into the stool. Some EDB is eliminated via expired air (ATSDR 1992).

The pathway of biotransformation for EDB appears to be the controlling factor for its biological activity. Two reactive intermediates, 2-bromoacetaldehyde and S-(2-bromoethyl)glutathione are formed. The 2-bromoacetaldehyde is responsible for tissue damage caused by covalent binding to cellular macromolecules. S-(2-bromoethyl)glutathione is responsible for EDB's proven genotoxic effect and, perhaps, the carcinogenic effect observed for it in laboratory animals (ATSDR 1992).

Except for adverse reproductive effects (damage to sperm cells) in men after occupational exposure, chronic effects (including cancer) of EDB exposure have not been documented in humans (ATSDR 1992). There is no evidence that low level exposure of humans to EDB causes respiratory, cardiovascular, gastrointestinal, hematological, musculoskeletal, hepatic , renal, immunological, neurological, or developmental effects (ATSDR 1992). Humans exposed to low levels of EDB in contaminated water such as during bathing or swimming, are unlikely to have any local irritant effects, but may be susceptible to absorption of the compound (ATSDR 1992). Antispermatogenic effects of EDB have been observed in occupationally exposed humans. The effects included changes in sperm velocity and count. The studies did not address whether the effects were associated with reduced fertility (ATSDR 1992).

A possible interaction effect that may be seen with EDB involves interactions with disulfiram, a drug used to treat chronic alcoholism. The toxic and carcinogenic effects of EDB in animals are potentiated by disulfiram. Potentiation occurs when a chemical that is either not toxic or does not evoke a toxic effect at a given level (EDB) chemical is rendered toxic by the presence of another chemical (disulfiram). Presumably, this occurs by blocking subsequent conjugation of the aldehyde metabolite to a less toxic form. Although there is no evidence that similar effects occur in humans, the manufacturers of this drug caution that patients taking disulfiram avoid exposure to EDB (ATSDR 1992).

Populations that may be unusually susceptible to the effects of EDB includes fetuses, and premature or perinatal infants with developmentally immature P-450 (microsomal enzyme) systems, chronic alcoholics taking disulfiram, and individuals with compromised liver, renal, or pulmonary function; however, chemical-specific effects have not been seen (ATSDR 1992).

The available epidemiological studies of EDB have not identified an increased risk of cancer in people occupationally exposed by inhalation to EDB. However, in experimental animals exposed to EDB by the inhalation route, EDB is a potent carcinogen, producing cancer at the point-of-contact -- the upper respiratory tract -- as well as in numerous organs and tissues throughout the body. Although adverse reproductive effects have been documented in humans after occupational exposure to EDB, no other chronic noncancerous effects have been documented. However, data derived from animal studies, mechanisms of action, and genotoxicity studies indicate that there is a potential for certain adverse health effects in humans exposed chronically to EDB. Target organs are of two types. The first is the point of contact with the chemical, i.e., skin for dermal exposure (humans and animals), oropharynx for ingestion (humans), stomach for gavage administration (rodents), and upper respiratory tract for inhalation exposure (humans and rodents). Although there is little information on toxicity on EDB in humans after inhalation, the testis was a target organ in exposed workers; the liver and kidney have also been identified as target organs after dermal and oral exposure in humans. The liver, kidney, and testis are target organs in experimental animals, irrespective of the exposure route (ATSDR 1992).

The U.S. Department of Health and Human Services (DHHS) has concluded that EDB may reasonably be anticipated to be a carcinogen (Group 2; DHHS 1994). The U.S. Environmental Protection Agency (EPA) has classified EDB as a (B2) probable human carcinogen (IRIS 1998) and developed an inhalation unit risk for it of $0.00022/\mu g/m^3$. A corresponding air concentration of EDB that would pose a one in one million (i.e. 10^{-6}) excess cancer risk is $0.005\ \mu g/m^3$. The International Agency for Research on Cancer (IARC) has classified EDB as (2A) probably carcinogenic to humans (IARC 1987). EPA has not developed a Reference Concentration (RfC) for EDB nor has ATSDR developed a Minimal Risk Level (MRL) for EDB for noncancer endpoints.

DCP toxicity

Animal studies indicate that DCP is readily absorbed through the lungs or gastrointestinal tract (ATSDR 1989). DCP is predicted to be readily absorbed through the skin (ATSDR

1989) and this prediction appears valid since death has been observed in rabbits following dermal exposure of DCP (ATSDR 1989).

DCP has been observed to be distributed throughout the major tissues of laboratory animals with the greatest concentrations in the liver, kidney, blood, and, with inhalation exposure, the lung (ATSDR 1989). Animal studies indicate that DCP is transported in the blood and expired by the lungs. DCP is readily metabolized in the body and the metabolites are expired in air and excreted in urine and feces (ATSDR 1989).

Data regarding the toxic effects of DCP on humans result solely from case reports of people exposed by inhalation, ingestion, or dermal contact. These reports indicate that DCP primarily affects the central nervous system, liver, and kidneys, but respiratory and hematopoietic system alterations were also observed. Chronic dermal exposure to 1,2-DCP in the workplace has resulted in dermatitis (ATSDR 1989).

A limited study on the carcinogenicity of 1,2-DCP in mice after inhalation exposure suggested that 1,2-DCP was carcinogenic, but ATSDR has judged the study unreliable because of excessive mortality in the exposed group, the tumor incidence in controls was not reported, and the morphology of the observed hepatomas was not adequately characterized; therefore, no conclusions are drawn (ATSDR 1989). The central nervous system, respiratory system, liver, and kidney are the major target organs for DCP via inhalation exposure (ATSDR 1989). With ingestion exposure, the liver is the main target organ, with effects on the hematological and nervous system also being observed. An increase in the incidence of developmental effects in rats (delayed ossification of bones in the skull) has been noted. The potential for carcinogenicity in both rats and mice has been observed; there was equivocal evidence in female rats (chemically related marginal increase in adenocarcinomas of the mammary gland) and some evidence in male and female mice (chemically increased incidence of hepatocellular neoplasms (ATSDR 1989).

1,2-DCP is commonly mixed with 1,3-dichloropropene, epichlorohydrin, and related compounds in a soil fumigant known as D-D. Studies of these chemicals in combination have not identified any potentiation of effects; all effects appeared to be additive (ATSDR 1989). Other studies of DCP in combination with 1,1,2-trichloroethane, ethylene dichloride, tetrachloroethylene, 1,2-dichloropropane, and trichloropropane also observed effects to be additive with no potentiation (ATSDR 1989).

Although no populations have been identified that are unusually susceptible to DCP exposures, individuals with compromised liver, renal, or pulmonary function may be at greater risk of health effects resulting from DCP exposures (ATSDR 1992).

The DHHS has concluded that DCP is not classifiable as to its carcinogenicity to humans (DHHS 1994). Neither EPA nor IARC have classified DCP as to its carcinogenicity. The EPA has developed an RfC of 4 $\mu g/m^3$ based on a 1.3 mg/m^3 Lowest Observed Adverse Effect Level (LOAEL) for nasal epithelial hyperplasia in female rats (IRIS 1998). ATSDR has utilized the EPA RfC to develop a Chronic inhalation Reference Concentration Media Evaluation Guide (RMEG) that is also 4 $\mu g/m^3$ (ATSDR 1989).

Exposures to EDB and DCP via Cooking, Showering, or Bathing

EDB and DCP contaminants present in groundwater that is used for domestic cooking and bathing purposes have the potential to pose a threat to public health via inhalation and dermal contact exposures. Several studies have indicated that the health threat posed by inhalation and dermal contact exposures to contaminated groundwater may be as great at that posed by direct ingestion exposure. McKone (1987) modeled the mass transfer of several volatile organic compounds, including EDB, from water to air and calculated a maximum concentration of EDB in household air of $2.4x10^{-4}$ mg per liter of air, assuming a water concentration of 1 mg/L. Therefore, volatile chemicals such as EDB and DCP can be expected to be released into the ambient air of households when groundwater containing them is used for cooking, showering, or bathing. The volatilization of these chemicals will increase as the water is heated by a stove for cooking and by a water heater for showering or bathing.

The EPA has developed a model for evaluating dermal exposures from showering that concludes that the dermal dose resulting from 10-minute showers exceeds the dose associated with drinking 2 L/day for a number of volatile organic compounds including EDB and DCP (EPA 1992). The EPA notes that data to validate the model are not available and that the model may be overly conservative. However, the evidence from these two documents indicates that use of groundwater contaminated with EDB and DCP for cooking, showering, or bathing will result in inhalation and dermal contact exposures.

Cancer is the health effect of concern with respect to EDB exposure. The EPA has established a Maximum Contaminant Level Goal of 0 for EDB, with a Maximum Contaminant Level of 0.05 µg/L for regulated municipal water supply systems (IRIS 1998). The ATSDR Cancer Risk Evaluation Guideline (CREG; $10x^{-6}$ excess cancer level, assuming 2 L per day ingestion rate for a 70 kg adult) for EDB in drinking water is 0.0004 µg/L.

A Reference Concentration (RfC) for noncancer effects of 4 $\mu g/m^3$ has been developed for DCP based on findings of nasal epithelial hyperplasia in female rats.

If inhalation and dermal contact exposures to volatile organic compounds in groundwater are assumed to pose as great of a risk as direct ingestion exposure, there would be reason to be concerned if EDB were detected at 6.17 µg/L and DCP were detected at 24 µg/L in groundwater that is being used for cooking, showering, and bathing.

If there is reason to believe that inhalation and dermal contact exposures to contaminated groundwater are occurring, efforts should be undertaken to either perform sampling and pathways analysis to evaluate the nature of the potential health threat, or the households using contaminated wells should be provided with an alternative water supply to eliminate completed exposure pathways that may pose a potential health threat.

A well-use survey is needed to determine the use-status of any water wells in the area. Residences where wells are used for all domestic purposes including drinking, cooking, bathing, showering, etc. should be identified, as well as those residences where wells are used on a more limited basis for cooking, bathing, and showering only.

Once a well-use survey has been completed, a sampling program will be required to identify the nature and extent of contamination. Contaminants other than EDB and DCP may be present. The sampling program should identify all contaminated wells, identify the contaminants, and provide concentrations of the contaminants.

Once the nature and extent of groundwater contamination is known, pathways analyses should be conducted to determine where ingestion, inhalation, and dermal contact exposure pathways are complete and whether the exposures are to contaminant levels that are likely to be of public health concern.

Air sampling may be indicated to determine the concentrations of volatile organic compounds in the living areas of residences. ATSDR will assist as needed in devising sampling plans, performing pathways analyses, and determining if exposures are of public health concern.

If residences are identified where wells are contaminated with levels of contaminants that pose health threats via ingestion, inhalation, or dermal contact exposures, they should be provided with an alternative water supply that eliminates any exposure that is of health concern, whether it is via ingestion, inhalation, or dermal contact exposures.

III. Conclusions

The purpose of this consultation is to provide a focused review of the toxicology of EDB and DCP and discuss the potential for these two chemicals to pose a threat to public health via inhalation and dermal contact exposures via cooking, showering, or bathing with contaminated groundwater. This consultation is not intended to predict or evaluate potential health effects associated with site-specific exposures. Such exposures can only be evaluated via thorough site-specific sampling and pathways analyses.

EDB and DCP contaminants present in groundwater that is used for domestic cooking and bathing purposes have the potential to pose a threat to public health via inhalation and dermal contact exposures. The health threat that may be posed by such exposures may be as great at that posed by direct ingestion exposure.

If there is reason to believe that inhalation and dermal contact exposures to contaminated groundwater are occurring, efforts should be undertaken to either perform sampling and pathways analysis to evaluate the nature of the potential health threat, or provide the households using contaminated wells with an alternative water supply to eliminate all exposure pathways that pose a potential health threat.

IV. Recommendations

A well-use survey is needed to determine the use-status of water wells in the area. Residences where wells are used for all domestic purposes including drinking, cooking, bathing, showering, etc. should be identified, as well as those residences where wells are used on a more limited basis for cooking, bathing, and showering only.

Once a well-use survey has been completed, a sampling program should be conducted to identify the nature and extent of contamination. Contaminants other than EDB and DCP may be present. The sampling program should identify all contaminated wells, identify the contaminants, and provide concentrations of any contaminants.

Once the nature and extent of groundwater contamination is known, pathways analyses should be conducted to determine where ingestion, inhalation, and dermal contact exposure pathways are complete and whether the exposures are to contaminant levels that are likely to be of public health concern. The potential exists for inhalation and dermal contact exposures to EDB and DCP to be of public health concern. Air sampling may be indicated to determine the concentrations of volatile organic compounds in the living areas of residences. ATSDR will assist as needed in devising sampling plans, performing pathways analyses, and determining if exposures are of public health concern.

If residences are identified where wells are contaminated with levels of contaminants that pose health threats via ingestion, inhalation, or dermal contact exposures, they should be provided with an alternative water supply that eliminates any exposure that is of health concern, whether it is via ingestion, inhalation, or dermal contact exposures.

Anyone having questions about this consultation may contact Mike Allred, Ph.D., Emergency Response Section, Emergency Response and Scientific Assessment Branch, ATSDR, at 404/639-6360.

V. References

ATSDR. 1989. Agency for Toxic Substances and Disease Registry, Toxicological Profile for 1,2-dichloropropane. Department of Health and Human Services, Atlanta, Georgia.

ATSDR. 1992. Agency for Toxic Substances and Disease Registry, Toxicological Profile for 1,2-dibromoethane (ethylene dibromide). Department of Health and Human Services, Atlanta, Georgia.

ATSDR. 1998. Petition letter to Agency for Toxic Substances and Disease Registry from residents of Whatcom County, Washington. Department of Health and Human Services, Atlanta, Georgia.

DHHS. 1994. Seventy Annual Report on Carcinogens - Summary. U.S. Department of Health and Human Services. Research Triangle Park, NC.

EPA. 1992. Dermal Exposure and Assessment: Principles and Guidelines. EPA 600?8-91/011F. U.S. Environmental Protection Agency, Washington, D.C.

EPA. 1995. Leaded Gas Phaseout; Internet Air Quality Fact Sheet at http://www.epa.gov/r10earth/offices/air/leadedga.html. U.S. Environmental Protection Agency, Washington, D.C.

HSDB. 1998. Hazardous Substances Data Bank (database online), Micromedex, Inc.

IARC 1987. Overall Evaluations of Carcinogenicity: An Updating of IARC Monographs Volumes 1 to 42: Supplement 7. WHO, International Agency for Research of Cancer, Lyon, France.

IRIS 1998. Integrated Risk Information System, Online Database. U.S. Environmental Protection Agency, Washington, DC.

McKone TE. 1987. Human exposure to volatile organic compounds in household tap water: The inhalation pathway. Environ Sci Technol. 21:1194-1201.

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Health Assessment of Well Water Contaminants in Whatcom County, Washington

Introduction

This report is in response to a request by Whatcom County Health and Human Services, Division of Environmental Health Services, to complete a health assessment regarding the exposure of individuals to groundwater contaminated with various chemicals including 1,2-dibromoethane (EDB), 1,2-dichloropropane (1,2-DCP), nitrates, 2-(sec-butyl)-4,6-dinitrophenol (Dinoseb), 1,2,3-trichlorophenol (TCP), benzene, 2-chloro-4-ethylamino-6-isopropylamino-1,3,5-triazine (Atrazine), and 1,2-dibromo-3-chloropropane (DBCP). The request was for the Washington State Department of Health (DOH) to assess these chemicals and their impacts on the public exposed and to provide recommendations as to the best course of action to ensure protection of public health.

Organic contaminants have been detected in more than 95% of samples collected from streams and rivers and in 50% of samples collected from ground water sources throughout the U.S. Whether these contaminants can lead to health effects depends in part upon the contaminant and the extent to which an individual is exposed. We have based our assessment and recommendations on data presently available and withhold judgement on the impact to the community affected by exposure to other chemicals until the remaining data from the U. S. Environmental Protection Agency (EPA), DOH, Washington State Department of Ecology, (Ecology), the United States Geological Survey (USGS), and Whatcom County Health and Human Services become available.

In providing this response, past groundwater contamination by a particular chemical was not considered unless the contaminant persists today. Groundwater contaminant data from years past in which a chemical was once detected but not again at a later date is an issue that can not be readily addressed since length and amount of exposure can not be easily determined and since exposure has ceased. In addressing the listed contaminants, DOH adheres to and enforces the Maximum Contaminant Levels (MCLs) for groundwater that have been established by EPA unless scientific evidence has become available, since the MCL was derived, indicating that a change may be warranted. Presently, DOH does not have sufficient justification to suggest changes to the MCLs and thus considers the MCLs for these compounds to be protective. MCLs were established by EPA as a result of the passage of the 1974 Federal Safe Drinking Water Act that requires determining safe levels of chemicals in drinking water. An MCL is the maximum permissible level of a contaminant in water delivered to a public water system user. With the exception of nitrates, benzene, EDB, DBCP and 1,2-DCP, available data from Whatcom County indicate that the range of values detected in drinking water supplies for all chemicals tested were below the MCLs for the specific chemical. Benzene is not addressed in this report as the one observation was from a point source, which is being addressed independently.

Health Implications from Nitrate Exposure in Household Water

Based on concentrations observed, frequency of detection and potential health impacts, nitrate was identified as the primary chemical of concern in many of the contaminated wells. Nitrate is considered an "acute contaminant" because short-term exposure to levels above the MCL have been determined to cause deleterious effects in individuals, especially infants. DOH has a position paper on nitrates, which is attached. The recommendations provided in that document, as well as those provided in this report, should be followed by individuals exposed to nitrates through drinking water.

Health Implications from EDB Exposure in Household Water

The range of EDB found in the drinking water supplies ranged from 0.03 to approximately 6.0 µg/l. Almost all EDB found in the environment comes from human activities. Until its use was restricted in 1984 by EPA, EDB was used in soil and on crops such as citrus, vegetable, and grain to control insects and nematodes. Also, until lead was banned from gasoline, EDB was added to gasoline as a lead oxide scavenger. There have been no registered uses of EDB in this state since 1987. EDB is a volatile organic compound and can evaporate into household air during domestic use of contaminated drinking water. It can be absorbed into the body by drinking or bathing in contaminated water and by breathing contaminated air. Animal studies indicate that EDB is absorbed rapidly, distributed throughout the body and retained to a limited extent in the liver, stomach, and kidneys.

Low level EDB exposure to humans has not been associated with any deleterious outcome such as respiratory, gastrointestinal, hepatic, renal, immunological, cardiovascular, or reproductive effects. In men, occupationally exposed to EDB, the only observed effect has been on the reproductive system, specifically, damaged sperm cells. In animals, EDB has been associated with liver and kidney damage and reproductive effects, including damage to sperm. Also, EDB is a potent cancer-causing chemical in animals, specifically, rats and mice (both sexes), and although EDB has never been shown to cause cancer in humans, the animal studies have resulted in EPA considering EDB to be a probable human carcinogen.

Results from studies using animals exposed to EDB, may it be through inhalation, dermal contact or ingestion, indicate that cancer is the health effect of greatest concern. For example, male rats exposed daily for roughly one year to levels approximately two million times greater than those obtained from humans ingesting two liters of water daily contaminated with EDB at the MCL, resulted in a significant increase in the incidence of squamous cell carcinomas of the stomach and hemangiosarcomas of the circulatory system. EDB is structurally similar to DBCP and 1,2-dichlorethane, two other chemicals considered to be probable human carcinogens, and it is mutagenic in various *in vitro* and *in vivo* assays. This evidence has led EPA to establish a value of 0.05 µg/l as the MCL for regulated municipal water supply systems. This value of 0.05 µg/l has been set with the understanding that this is the lowest level to which water systems can reasonably remove EDB given present technology and resources. At present the MCL of 0.05 µg/l appears to allow for prudent protection of public health.

Health Implications from 1,2-DCP Exposure in Household Water

1,2-DCP was used commercially in agricultural soil fumigants and as a solvent in paint strippers, cleaners, and varnishes. Production of 1,2-DCP was discontinued in 1991 and its presence in soil fumigants has been phased out. It remains a health concern because past use has resulted in groundwater contamination. In Whatcom County 1,2-DCP has been detected in drinking water wells levels ranging from 1.0 approximately to 20 µg /l. 1,2-DCP is a volatile organic compound and can evaporate into household air during domestic use of contaminated drinking water. It can be absorbed into the body by drinking or bathing in contaminated water and by breathing contaminated air. Animal studies indicate that once absorbed 1,2-DCP is rapidly excreted and is not likely to bioaccumulate. Human evidence of health effects is limited to case reports of large acute exposures. The only evidence of effects in humans at repeated low level exposure are several reports of people who developed allergic contact dermatitis after a number of years working with products containing 1,2-DCP.

Most of what we know about the toxicology of 1,2-DCP comes from research in experimental animals. Animals fed DCP for prolonged periods of time in their water or diet developed anemia, central nervous system effects, liver injury, and at higher doses, kidney injury and testicular degeneration. Effects on the development of the fetus have been seen only at doses that were acutely toxic to the mothers. 1,2-DCP is a strong respiratory irritant and caused degradation of the nasal mucosa and olfactory epithelium when inhaled by animals for prolonged periods. To protect against this effect EPA recommends that, over a lifetime of exposure, air contamination by 1,2-DCP not exceed 4µg /m^3. EPA has not established a similar recommendation for oral intake of 1,2-DCP but the ATSDR has established a Minimum Risk Level of 70 µg /kg/day based on slight anemia seen in rats at 100,000 µg /kg/day over 13 weeks. EPA has, however, set a value of 5 µg/l as the MCL for regulated municipal water supply systems. The evidence for cancer is limited and marginally reliable. Neither DOH nor EPA considers the available data sufficient to classify 1,2-DCP's carcinogenic potential.

DOH does not expect any adverse health effects from use of water at the MCL of 5 µg /l. The theoretical amount absorbed through all exposure routes from routine household uses of water at the MCL is still more than 1000 times less than doses associated with even the slightest effects seen in animals. The 1000 fold safety factor considers that humans may be more sensitive than lab animals, and that there are large variations in the health status of human populations that are not well modeled by the homogeneous animals used in testing protocols. The MCL has been set with the understanding that this is the lowest level to which water systems can reasonably remove 1,2-DCP given present technology and resources. At present the MCL of 5 µg/l appears to allow for prudent protection of public health.

Health Implications from DBCP Exposure in Household Water

To date, DBCP has been found in two wells in Whatcom County, with the highest concentration being 0.3 µg/l. DBCP was used as a soil fumigant for nematodes on vegetables until 1979 when registrations of end use products were cancelled for all states with the exception of Hawaii. Hawaii stopped using DBCP in 1985. As with EDB and 1,2-DCP, DBCP is a volatile organic compound that can evaporate into household air during domestic use of contaminated drinking water. It can be absorbed into the body by drinking or bathing in contaminated water and by breathing contaminated air. Animal studies indicate that, once absorbed, DBCP is rapidly excreted and is not likely to bioaccumulate.

In laboratory animals, DBCP is a known carcinogen. Specifically, DBCP has been shown to produce tumors in the nasal cavity and on the tongues of rats and in the nasal cavity and lungs of mice when inhaled. The animal research has resulted in EPA considering this toxic compound to possibly be carcinogenic to humans. In humans, it has been shown that chronic exposure in occupational settings to DBCP can produce diffuse necrosis of seminiferous tubule cells resulting in decreased sperm count and permanent sterility. DBCP is of greater concern than EDB to individuals occupationally exposed because DBCP is much less odoriferous resulting in a greater likelihood of toxic exposure to DBCP. EPA has established a value of 0.2 μg/l as the MCL for regulated municipal water supply systems. This value of 0.2 μg/l has been set with the understanding that this is the lowest level to which water systems can reasonably remove DBCP given present technology and resources. At present the MCL of 0.2 μg/l appears to allow for prudent protection of public health.

Multiple Routes of Exposure

Exposure to volatile organic compounds found in public and private water supply systems may be greater than previously believed. Not only are individuals exposed to contaminants through ingestion, but due to the physical and chemical properties of volatile organic chemicals, individuals can also be exposed through dermal contact, and inhalation. Household uses that may result in dermal or inhalation exposure to volatile organic chemicals include: cooking, bathing, showering, washing dishes or using a Jacuzzi® or humidifier. A DOH review of available dermal and inhalation exposure models indicates that the amount of some volatile organic chemicals entering the body on a daily basis through inhalation, due to the chemical volatilizing into the air, could be equivalent to the amount of chemical entering the body daily by drinking two liters of water. Dermal exposure may also allow an equivalent amount of certain volatile organic chemicals to enter the body daily as drinking two liters of water. However, the information from these models is not precise and has not been validated. EPA currently does not have a proposed methodology for explicitly incorporating inhalation and dermal absorption exposures from household water uses in the derivation of health-based criteria.

Although precise determinations regarding total exposure from water can not yet be made from these models, sufficient information is available to suggest that precautionary measures to reduce exposure from these other routes need to be considered. Whereas DOH considers exposure to the volatile organic chemicals, EDB, 1,2-DCP, and DBCP, below their respective MCLs, to be protective, individuals should not use their well water for drinking, cooking, bathing or showering if contamination of any of these compounds exceeds their respective MCLs. Also, those individuals in households that have contaminant levels below the MCL, who are still concerned about exposure to these contaminants, can minimize exposure by taking precautionary measures such as limiting shower and bathing times, reducing the temperature of bath water, using rubber gloves when dishwashing and ensuring that bathrooms are well ventilated.

Multiple Chemical Exposure

Assessing health implications of human exposure to chemical mixtures is always a difficult task because traditional toxicology research tests examine chemicals one at a time rather than in combinations. Although this is changing somewhat, there is very little data on the toxicity of combined exposures to EDB, 1,2-DCP, DBCP and nitrates. The primary concern when chemical mixtures are present is the potential for synergism or potentiation. That is, when the presence of one co[illegible]ases the toxicity of the other so that the toxicity of the combination is greater than the effect expected by simply adding their individual toxicities.

One of the ways to look for potential interactions of chemicals is to review their absorption, metabolism, biochemical actions and excretion in experimental animals. Many chemicals to which we are voluntarily, as well as involuntarily, exposed on a daily basis interact with the microsomal monooxygenase system (cytochrome p450), a primary enzyme system that metabolizes and helps to eliminate chemicals from our bodies. For example, EDB and 1,2-DCP activate this system and are metabolized by it. However, this system is not considered to produce the reactive intermediate responsible for EDB's genotoxic effect and possibly carcinogenic effect. In contrast, DBCP has been shown to decrease microsomal enzyme activity and may preferentially affect specific isozymes of cytochrome p450. There is limited data, which addresses exposure to mixtures of these and other compounds and how they may affect the same system or target organs in our bodies. A series of experiments tested animals for effects of concomitant exposures to pesticides including aldicarb, atrazine, DBCP, 1,2-DCP, EDB, and ammonium nitrate. The results provided no evidence to suggest that a synergistic, additive, potentiated or antagonistic (chemical or functional) effect occurred with concomitant exposure to these compounds. Presently, there is not enough information on biochemical pathways and metabolism of these compounds to determine the potential of combined effects from exposure to these compounds. Based on our review, DOH did not find justification for changing MCLs based on potential chemical interactions.

Conclusions and Recommendations

Nitrate in the well water of Whatcom County residents is the primary cause for concern. Presently, Washington State guidelines recommend installing water treatment systems if nitrate concentrations exceed the MCL of 10 mg/l and that monitoring occur to ensure the MCL is not exceeded in wells with a history of contamination. DOH suggests that health officials recommend that individuals with private water systems monitor frequently and consider a new water source if well water nitrate levels exceed 5 mg/l.

Presently, DOH considers the MCLs to be protective and does not expect any deleterious health outcomes from exposure to EDB, 1,2-DCP or DBCP at their respective MCLs in drinking water supplies. Individuals with water supplies above the MCL should employ a treatment device or seek alternative water for all domestic uses. If, in the past, individuals used the water for purposes other than drinking water, such as cooking and showering, new evidence available indicates that these practices should not be recommended when contaminant levels are above their respective MCLs. Those individuals in households that have contaminant levels below the MCL, who are concerned about their water supply, can minimize exposure to these chemicals by taking precautionary measures such as limiting shower and bathing times, reducing the temperature of bath water, and ensuring that bathrooms are well ventilated. Also, exposure can be further minimized by providing or acquiring alternative drinking water supplies and by public water systems adopting new technologies as they become available, affordable, and feasible to implement.

Future Work

Several state and federal agencies are in the process of obtaining additional well water samples within the area of concern to better characterize the extent and nature of contamination. This includes ongoing sampling efforts by EPA, USGS, Ecology, DOH and Whatcom County Health and Human Services. In addition to well water testing, ATSDR will be performing an exposure investigation to measure volatile organic chemicals in indoor air. Results of this exposure investigation will be used to; validate modeling efforts, assess the health risks posed by inhalation of volatile organic chemicals during showering, and to assess the effectiveness of mitigation strategies. In addition, as part of the citizens' petition to ATSDR, these issues will also be addressed: exposure to manure fertilizer through aerial spraying, possible association of childhood leukemia with exposure to contaminated drinking water, and health impact of non-community water systems on migrant workers.

Once this sampling and exposure investigation data becomes available, DOH, in conjunction with ATSDR, will review the results, determine whether additional health concerns are present, and report the findings to Whatcom Country Health and Human Services.

References

A source for further information on these chemicals is ATSDR which has references entitled:

ATSDR's Case Studies in Environmental Medicine: Nitrate/Nitrite Toxicity,
ATSDR's Toxicological Profile for 1,2-dibromoethane;
ATSDR's Toxicological Profile for 1,2-dichloropropane, and
ATSDR's Toxicological Profile for 1,2-dibromo-3-chloropropane.

These documents can be obtained by contacting ATSDR at:

Continuing Education Coordinator
Agency for Toxic Substances and Disease Registry
Division of Health Education, E33
1600 Clifton Road NE
Atlanta, GA 30333

Also, information on these compounds can be obtained from EPA's Integrated Risk Information System on the internet at http://www.epa.gov/iris/.

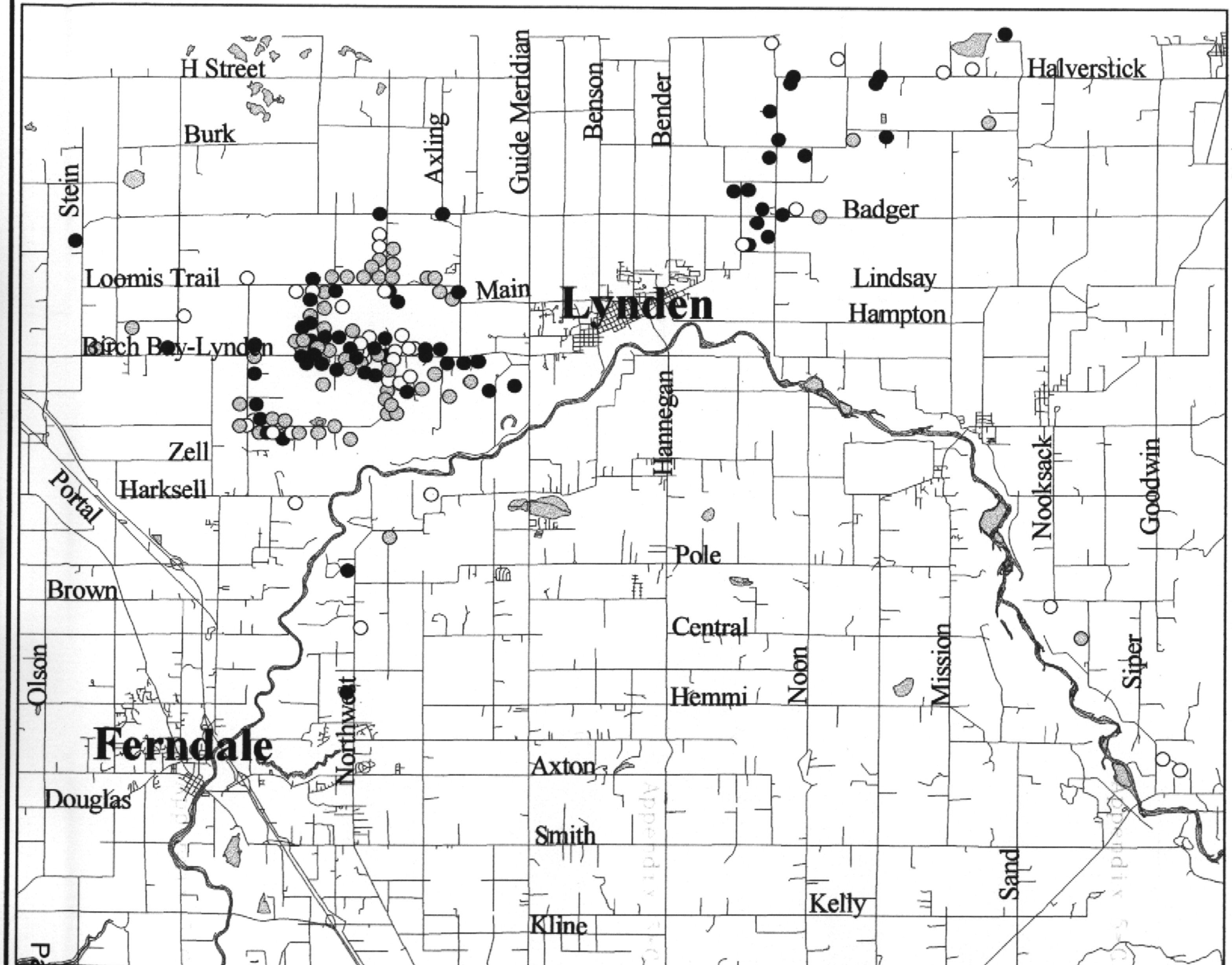
Information pertaining to ground and drinking water can be obtained from USGS's National Water Quality Assessment Program on the internet at http://wwwrvares.er.usgs.gov/nawqa/nawqa_home.html and from EPA's Office of Ground Water and Drinking Water on the internet at http://www.epa.gov/OGWDW/.

Department of Health Points of Contact:

Office of Environmental Health Assessment – Dave McBride (360) 236-3176
Division of Drinking Water – Jim Hudson (360) 236-3131
Website – http://www.doh.wa.gov/ehp

DRAFT

Whatcom County Wells Tested for Nitrate by USGS, DOH and DOE (1998)



Level of Nitrate:
- 0-3 mg/l or ppm
- Detected but less than the MCL
- >= the MCL (10 mg/l or ppm)

Well locations are approximate.
When multiple samples from the same well were available, the highest value was used.
The MCL is the Maximum Contaminant Level.
January 29, 1999 (AMK)

WHATCOM COUNTY WASHINGTON