UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D C 20460

AUG 25 1988

OFFICE OF WATER

MEMORANDUM

SUBJECT: Transmital of "Guide to Drinking Water Health Advisories."

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THRU: Edward V. Ohanian, Ph.D. Final Markings for Ed
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This guide has undergone extensive HEB review and is in final form al review. Your comments of May 1988 have been incorporated. A copy was requested in June by Peter Cook, but no comments have been received.

Attachment

cc: J. Orme

Draft

GUIDE TO DRINKING WATER HEALTH ADVISORIES

U.S. Environmental Protection Agency
Office of Drinking Water
Criteria and Standards Division
Health Effects Branch

August, 1988

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GUIDE TO DRINKING WATER HEALTH ADVISORIES

The US EPA Office of Drinking Water/Health Effects Branch in the Criteria and Standards Division prepared this guide to explain the Health Advisory program, the derivation of guidance values and their application to risk management decisions. "Workshops on Assessemet and Management of Drinking Water Contamination" (1987) contains a more detailed discussion of these topics (refer to Appendix II).

REGULATING DRINKING WATER CONTAMINANTS

Tap water contains many more chemicals than just water. Some chemicals occur naturally, such as the minerals which make water "hard." Other chemicals enter drinking water from human activity. Water treatment plants intentionally add some chemicals to improve water quality. Disinfectants (such as chlorine) kill bacteria and protect against disease, phosphates limit corrosion of water pipes, and coagulants remove unwanted solids from turbid waters. In addition, fluoride is often added as a health measure to prevent tooth decay. Other activities, such as industrial and municipal discharges, spills, agricultural runoff, may contaminate drinking water supplies.

Some chemicals identified in drinking water supplies are unwanted contaminants with the potential to cause adverse health risks or give water an unpleasant taste and odor. To protect the public from these risks, Congress enacted the Safe Drinking Water Act in 1974 and amended the Act in 1986. This Act mandates the EPA to identify drinking water contaminants of concern and regulate those contaminants by setting enforceable Maximum Contaminant Levels (MCLs). Waters with contaminant levels above the MCL cannot be used for public water supplies. When routine monitoring of a contaminant is not technically and economically feasible, EPA requires specific water treatment techniques as the standard.

To set MCLs, EPA evaluates information on a contaminant's potential to cause adverse health effects. A summary of this information appears in EPA's health effects criteria documents. Based on this information, the Office of Drinking Water (ODW) derives non-enforceable Maximum Contaminant Level Goals (MCLGs), which are drinking water levels that include a margin of safety to protect against any known or anticipated human health effects. MCLs are set as close to the MCLGs as feasible. Feasibility is determined by the costs and other factors. MCLs are established at "safe" levels.

PROVIDING GUIDANCE THROUGH THE HEALTH ADVISORY PROGRAM

For many contaminants not already regulated under the Safe Drinking Water Act, the ODW has prepared Health Advisories (HAs). HA documents contain information on the chemical/physical properties occurrence, environmental fate, pharmacokinetics, toxicity, and treatment/removal techniques for a given contaminant. ODW provides information to public health officials on deriving One-day, Ten-day

Longer-term and Lifetime HA values. Like MCLGs, HA values are not enforceable standards, but guidance values indicating the drinking water concentration of a contaminant that are considered protective of human health for a given duration of exposure - i.e., unlikely to result in any adverse effect on health with a margin of safety.

DERIVING HEALTH ADVISORY VALUES

To derive HA values, ODW reviews the pertinent studies describing the health effects of the contaminant. Studies are evaluated based on their overall quality, their relevance to human exposure via drinking water and their duration of exposure. Studies describing oral administration of the contaminant (especially via drinking water) for the appropriate duration of exposure (see Table 1) are the prefered basis for HA derivation. Other routes of exposure and study durations may also be considered acceptable.

Typically, ODW derives HA values from toxicity studies which describe doses to experimental animals which cause little or no adverse health effects. The HA document describes these dose levels as the Lowest-Observed-Adverse-Effect Level (LOAEL) or the No-Observed-Adverse-Effect Level (NOAEL), respectively. To estimate doses causing no adverse effects in sensitive humans, ODW divides these NOAELs/LOAELs by appropriate uncertainty factors (UFs; see Table 2).

ODW calculates HA values from a dose level (either a NOAEL or LOAEL) by making assumptions about the body weight of individuals, and their drinking water consumption rates. The final formula for calculating One-day, Ten-day and Longer-term HA values is:

$$\text{One-day, Ten-day or Longer-term HA (mg/L)} = \frac{(\text{NOAEL or LOAEL mg/kg/day})(\text{Body wt. in kg})}{(\text{Uncertainty factor})(\text{Water consumption L/day})}$$

Table 1. HA development for different durations of exposure.

Health Advisory	Preferred study duration	Protected individual/ duration of exposure
One-day HA	1 to 5 (successive) daily doses	Child exposed 1 day
Ten-day HA	7 to 30 (successive) daily doses	Child exposed < 1 month
Longer-term HA	10% of lifetime (90 days in rats/mice)	Child & adult exposed up to 7 years
Lifetime HA or MCLG	Lifetime (2 years in rats/mice); subchronic with additional UF	Adult exposed 70 yrs
Cancer risk	Lifetime (oncogenicity)	Adult exposed 70 yrs

Table 2. Uncertainty factors for HA calculation[a]

UF	HA basis	Justification
≤10	Human NOAEL	Accounts for variation within the population (intraspecies,,
100	Human LOAEL	Incorporates a factor of 10 to account for lack of a NOAEL and a factor of 10 for intraspecies variation, or,
100	Animal NOAEL	Incorporates a factor of 10 to account for interspecies differences, and 10 for intraspecies variation.
1,000	Animal LOAEL	Incorporates factors of ten for lack of a NOAEL, interspecies variation, and intraspecies variation, or,
1,000	Animal NOAEL	For Lifetime HAs, incorporating factors of ten for interspecie variation, sensitive individuals & less-than-lifetime exposure

Additional uncertainty factors, ranging from 1 to 10, may be incororated on a case-by-case basis to account for deficiencies in the data base, quality of the data, or severity of the effect.

[a]Recommended by the National Academy of Sciences (1977) and modified by ODW.

Lifetime HAs (and MCLGs) are derived using a similar equation, but the derivation is broken into a series of steps. First the Reference Dose (RfD, formerly called the ADI or Acceptable Daily Intake) is calculated:

$$\text{RfD (mg/kg/day)} = \frac{\text{(NOAEL or LOAEL mg/kg/day)}}{\text{(Uncertainty factor)}}$$

Next, the Drinking Water Equivalent Level (DWEL) is derived:

$$\text{DWEL (mg/L)} = \frac{\text{(RfD mg/kg/day)(Body weight in kg)}}{\text{(Water consumption L/day)}}$$

The Lifetime HA is the DWEL multiplied by the Relative Source Contribution (RSC), a factor to account for exposure to the contaminant from other sources such as food and air.

Table 3. Assumptions used in developing HAs/MCLGs

Body weight:	Children weigh 10 kg; Adults weigh 70 kg
Drinking water consumption:	Children drink 1 L/day; Adults drink 2 L/day
Relative source contribution: (for Lifetime HAs/MCLGs only)	20% in the absence of chemical-specific data

Lifetime HA or MCLG (mg/L) = DWEL x RSC

ODW assumes the RSC is 20% when no specific exposure data are available. Other assumptions used in deriving HAs/MCLGs are given in Table 3.

LIFETIME HAs AND MCLGs FOR CARCINOGENS

The methods for calculating Lifetime HA values and MCLGs are only followed for noncarcinogenic compounds. For probable human carcinogens, ODW does not recommend Lifetime HAs and sets MCLGs at zero (see Congressional recommendations [House Report, 1974]). Carcinogens are treated differently from other chemicals based on the nonthreshold theory that any dose of a carcinogen, no matter how small, entails some increased risk for cancer. EPA estimates this risk using statistical models. To ensure the protection of public health, these models are based on conservative assumptions, making the underestimation of risks unlikely. HA documents provide drinking water concentrations that are associated with risks of 10^{-4}, 10^{-5} and 10^{-6}; meaning that lifetime exposure to these concentrations are unlikely to cause greater than one additional case of cancer in populations of 10,000 (i.e., 10^4), 100,000 (10^5) or one million (10^6), respectively. Populations are assumed to consume 2 liters of drinking water over a lifetime. MCLs are generally set at the lowest feasible limit and usually fall within the range of 10^{-4} to 10^{-6} risk.

For compounds which may possibly be carcinogens (class C carcinogens), MCLG[illegible] based upon the DWEL and an additional UF of 10 to [illegible] uncertainties about carcinogenicity.

Table 4. Carcinogen classification and derivation of guidance values.

Classification		Evidence of Carcinogenicity: Humans	Evidence of Carcinogenicity: Animals	Derivation of Guidance Values: MCLG	Derivation of Guidance Values: Lifetime HA
A	Human carcinogen	Sufficient		zero	not recommended
B1	Probable human carcinogen	Limited		zero	not recommended
B2	Probable human carcinogen	Inadequate	Sufficient	zero	not recommended
C	Possible human carcinogen	None	Limited	(DWELxRSC) ÷ extra UF	
D	Not classified	Inadequate	Inadequate	DWEL x RSC	
E	No evidence of carcinogenicity	None	Negative	DWEL x RSC	

USING HA VALUES FOR RISK ASSESSMENT

If a contamination incident occurs, public health officials should be notified immediately. To determine the extent of the problem and its significance, public officials are advised to:

Table 5. Use of HA values for risk assessment.

Contaminant level	Recommended response	
Above One-day HA	Immediate action needed.	Reduce further exposure imme
Between One-day HA and Ten-day HA	Action needed if exposure exceeds approximately 10 days.	Exposures should be reduced wit approximately 10 days of contam tion incident.
Between Ten-day HA and Longer-term HA	Immediate action may be warranted for exposures greater than about 10 days.	Conduct a site-specific risk assessment to determine accepta levels/duration of exposure; reduce exposures accordingly.
Between Longer-term HA and Lifetime HA or 10^{-6} cancer risk level*	No immediate risk to public health.	Develop and implement strategie for reducing contaminant levels in drinking water if desirable.
Below Lifetime HA/ 10^{-6} cancer risk level	No action needed.	Protective of public health.

*See discussion of carcinogens, p. 4, and Table 4. For some compounds, levels as hi as the 10^{-4} level are considered protective of public health.

1) Determine the concentration of the contaminant in drinking water,

2) Refer to the appropriate HA document, obtained either through EPA Regional Offices (See Figure 1), or by calling the Drinkir Water Hotline (800-424-4791).

3) Compare contaminant levels to HA values to determine if actior may be needed (Refer Table 5),

4) Characterize risks to enable risk managers to take appropriate actions to ensure the safety of public water supplies.

If contaminant concentrations are below the Lifetime HA, no action is needed for protection of public health. Whenever contaminant concentrations exceed the Lifetime HA some level of action is needed. Guidance on whether immediate action is needed can be obtained by comparing drinking water levels to One-day, Ten-day and Longer-term Advisories (Table 5). Before decisions can be made on how best to manage the risks of exposure to contaminants, these risks must be clearly characterized.

ODW does not recommend Lifetime HA values for carcinogens (Class A, B1 or B2), therefore carcinogenic contaminants must be evaluated differently. Officials should compare the drinking water levels to both theoretical incremental cancer risk levels, and values for the DWEL (derived for non-carcinogenic effects). Thus, both possible carcinogenic and noncarcinogenic effects must be

evaluated when determining the risks posed by carcinogens. Drinking water concentrations ranging from the 10^{-4} to 10^{-5} risk level are generally acceptable provided these levels are also protective of other effects. Determining the acceptability of exposures at levels greater than the 10^{-4} risk level should be made on a case-by-case basis.

RISK CHARACTERIZATION

There is no sharp boundary between safe and unsafe levels of a contaminant in drinking water. When concentrations exceed HA values, risk managers must make difficult decisions. EPA advocates the use of quantitative risk assessment as a tool for this decision making process. Quantitative risk assessment involves determining:

1) Toxic effects associated with exposure (hazard identificatio

2) Dose associated with these effects (dose-response evaluatio

3) Level of human exposure (exposure assessment).

Public officials should understand the uncertainties in each of these three elements of risk characterization to effectively use HA values for the management of drinking water contaminantion. A brief description of these three elements, originally described in NAS (1983) is given below.

1) Haza... HA documents identify the likely toxic effects ass... ng water contaminants. For a few chemicals, suc... e hazards of human exposure are well characterized. For most contaminants, toxicity studies on experimental animals serve as the basis for the hazard identificatio Hazard identification is often an uncertain process because studies are not always available to assess the reproductive, developmental or carcinogenic hazards of a contaminant. In addition, variation within a population, differences between experimental animals and humans, and incomplete data on chronic health effects make it difficult to predict which effects are likely to occur in humans.

2) Dose-response evaluation: In the previous step, the adverse human health effects of most concern for a given contaminant are identified. The relationship between these toxic effects and the dose which causes them, is the dose-response relationship. If sufficient data are available, the dose-reponse evaluation yields a precise estimate of the highest dose causing no adverse health effects, i.e., ...shold dose. More often, the threshold dose cannot be estimated ...h certainty and a dose protective of public health is estimated by dividing a NOAEL/LOAEL by an uncertainty factor. The level of uncertainty in the dose-response evaluation is reflected in the magnitude of the uncertainty factor used (Table

The dose-response evaluation requires special care for two typ of contaminants:

Contaminants with steep dose-response relationships, such as organophosphate pesticides, may cause severe effects or even death at doses just slightly above those which appear safe, and may require an extra margin of safety to ensure the public is not exposed to severe hazards.

Carcinogens are evaluated differently from non-carcinogens. It is assumed that no threshold dose exists for carcinogens and any dose, no matter how small, increases the risk of cancer.

3) Exposure Assessment: Measuring the concentration of the contaminant in water is the primary focus of the exposure assessment. Often the first step in characterizing risk is to check the reported contaminant concentration to confirm that the population really is exposed. Analysis of a single sample may be sufficient to identify contamination problems, but often, multiple samples are analyzed to verify the initial findings. Analytical techniques can be quite accurate, but some variability is expected, even between identical samples. For example, for vinyl chloride, EPA considers measurements within 40% of actual concentrations acceptable for laboratory certification. In some cases, HA values may be lower than currently available analytical detection limits.

Uncertainties in exposure assessment extend beyond questions concerning chemical analysis of water samples. Exposure estimates include estimates of the drinking water consumption rate, the duration of exposure, and estimations of exposure from other sources such as food or air. Because the exposure assumptions used to develop HAs/MCLGs (see Table 3) may differ from actual conditions for a given site, site-specific exposure estimates can provide useful information for assessing and managing risks.

The final step in risk assessment is risk characterization. In this step, the hazard identification and dose-response evaluation are integrated with the exposure assessment. The risk characterization describes the estimates for the most likely outcomes from exposure to the contaminant at the levels found in drinking water, and provides the basis for informed decision making.

RISK MANAGEMENT

The risk characterization should assist the manager in deciding on both an appropriate course of action, and how fast the action must be taken. Appropriate responses to elevated contaminant levels can range from drastic immediate action (e.g., providing bottled water) to better long-range planning (e.g., adopting a monitoring program with a remedial action plan if contamination continues).

Exposures to contaminants in drinking water can be reduced using a variety of strategies. Providing bottled water or point-of-use treatment devices are primarily short-term measures

of relatively high cost. Control strategies such as reducing, eliminating contaminant sources, blending, or finding new sources will usually reduce contaminant exposures to acceptable levels. Treatment strategies including conventional treatment, aeration, absorption, biodegradation, reverse osmosis, ion exchange and electrodialysis have all been used successfully in removing various drinking water contaminants. The most appropriate actions may involve combining short-term meausures, control measures, and long-range treatment strategies to protect public health and improve water quality.

SOURCES OF INFORMATION AND ASSISTANCE

In emergency contamination incidents, the appropriate local public health official should be contacted immediately. If there is a problem locating the proper local official, the National Association of County Health Officials may be helpful (202-783-5550). For other types of inquiries, local authorities may defer questions to one of ten EPA regional offices throughout the country (FIGURE

Assistance can also be obtained from the EPA ODW Headquarters office in Washington, DC. The ODW supports a 24-hour, toll-free drinking water Hotline (800-426-4791). In addition, HA documents provide answers to many questions about drinking water contaminants. Each of the ten regional offices has a complete set of HAs, or a personal copy can be obtained by calling the Drinking Water Hotline.

REFERENCES

National Academy of Sciences, National Research Council. 1977. Drinking Water and Health, Volume 1. National Academy Press, Washington, DC.

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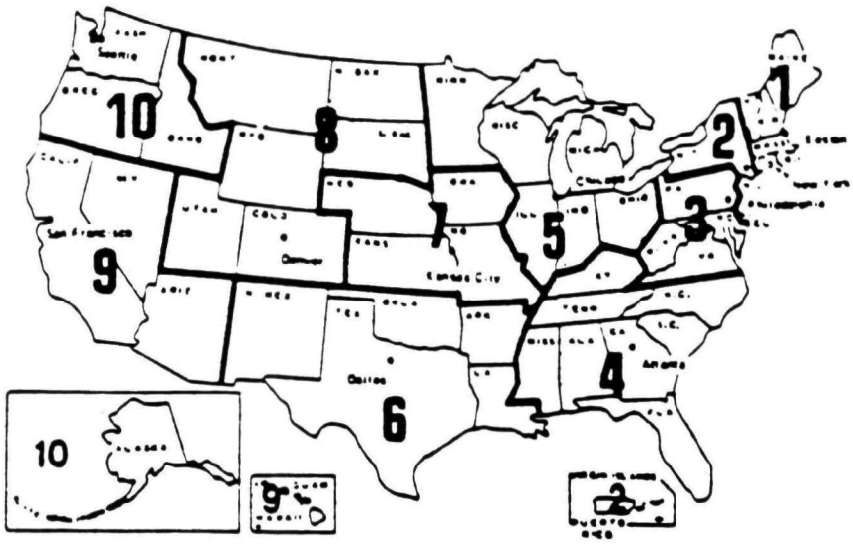
House Report. 1974. No. 93 - 185. July 10, 1974.

US EPA. Technology Transfer document "Workshops on Assessment and Management of Drinking Water Contamination." EPA/600/M-86/026. Revised March 1987.



Region 1:	(617) 565-3715	Region 6:	(214) 655-6444
Region 2:	(212) 264-2525	Region 7:	(913) 236-2800
Region 3:	(215) 597-9800	Region 8:	(303) 293-1603
Region 4:	(404) 347-4727	Region 9:	(415) 974-8071
Region 5:	(312) 353-2000	Region 10:	(206) 442-5810

FIGURE 1. EPA Regions and Regional Office Telephone Numbers.

APPENDIX I

GLOSSARY OF RISK ASSESSMENT/RISK MANAGEMENT TERMS
from US EPA (1987)

Absorbed dose. The amount of a chemical that enters the body of an exposed organism.

Absorption. The uptake of water or dissolved chemicals by a cell or an organism.

Absorption factor. The fraction of a chemical making contact with an organism that is absorbed by the organism.

Acceptable daily intake (ADI). Estimate of the largest amount of chemical to which a person can be exposed on a daily basis that is not anticipated to result in adverse effects (usually expressed in mg/kg/day). (Synonymous with RfD)

Active transport. An energy-expending mechanism by which a cell moves a chemical across the cell membrane from a point of lower concentration to a point of higher concentration, against the diffusion gradient.

Acute. Occurring over a short period of time; used to describe brief exposures and effects which appear promptly after exposure.

Additive Effect. Combined effect of two or more chemicals equal to the sum of their individual effects.

Adsorption. The process by which chemicals are held on the surface of a mineral or soil particle. Compare with absorption.

Ambient. Environmental or surrounding conditions.

Animal studies. Investigations using animals as surrogates for humans, on the expectation that results in animals are pertinent to humans.

Antagonism. Interference or inhibition of the effect of one chemical by the action of another chemical.

Assay. A test for a particular chemical or effect.

Bias. An inadequacy in experimental design that leads to results or conclusions not representative of the population under study.

Bioaccumulation. The retention and concentration of a substance by an organism.

Bioassay. Test which determines the effect of a chemical on a living organism.

Diffusion. The movement of suspended or dissolved particles from a more concentrated to a less concentrated region as a result of the random movement of individual particles; the process tends to distribute them uniformly throughout the available volume.

Dosage. The quantity of a chemical administered to an organism.

Dose. The actual quantity of a chemical to which an organism is exposed. (See absorbed dose)

Dose-response. A quantitative relationship between the dose of a chemical and an effect caused by the chemical.

Dose-response curve. A graphical presentation of the relationship between degree of exposure to a chemical (dose) and observed biological effect or response.

Dose-response evaluation. A component of risk assessment that describes the quantitative relationship between the amount of exposure to a substance and the extent of toxic injury or disease.

Dose-response relationship. The quantitative relationship between the amount of exposure to a substance and the extent of toxic injury produced.

DWEL. Drinking Water Equivalent Level -- estimated exposure (in mg/L) which is interpreted to be protective for noncarcinogenic endpoints of toxicity over a lifetime of exposure. DWEL was developed for chemicals that have a significant carcinogenic potential (Group B). Provides risk manager with evaluation on non-cancer endpoints, but infers that carcinogenicity should be considered the toxic effect of greatest concern.

Endangerment assessment. A site-specific risk assessment of the actual or potential danger to human health or welfare and the environment from the release of hazardous substances or waste. The endangerment assessment document is prepared in support of enforcement actions under CERCLA or RCRA.

Endpoint. A biological effect used as an index of the effect of a chemical on an organism.

Epidemiologic study. Study of human populations to identify causes of disease. Such studies often compare the health status of a group of persons who have been exposed to a suspect agent with that of a comparable non-exposed group.

Exposure. Contact with a chemical or physical agent.

Exposure assessment. The determination or estimation (qualitative or quantitative) of the magnitude, frequency, duration, route, and extent (number of people) of exposure to a chemical.

Human health risk. The likelihood (or probability) that a given exposure or series of exposures may have or will damage the health of individuals experiencing the exposures.

Incidence of tumors. Percentage of animals with tumors.

Ingestion. Type of exposure through the mouth.

Inhalation. Type of exposure through the lungs.

Integrated exposure assessment. A summation over time, in all media, of the magnitude of exposure to a toxic chemical.

Interspecies extrapolation model. Model used to extrapolate from results observed in laboratory animals to humans.

In vitro studies. Studies of chemical effects conducted in tissues, cells or subcellular extracts from an organism (i.e., not in the living organism).

In vivo studies. Studies of chemical effects conducted in intact living organisms.

Irreversible effect. Effect characterized by the inability of the body to partially or fully repair injury caused by a toxic agent.

Latency. Time from the first exposure to a chemical until the appearance of a toxic effect.

LC_{50}. The concentration of a chemical in air or water which is expected to cause death in 50 percent of test animals living in that air or water.

LD_{50}. The dose of a chemical taken by mouth or absorbed by the skin which is expected to cause death in 50 percent of the test animals so treated.

Lesion. A pathological or traumatic discontinuity of tissue or loss of function of a part.

Lethal. Deadly; fatal.

Lifetime exposure. Total amount of exposure to a substance that a human would receive in a lifetime (usually assumed to be seventy years).

Linearized multistage model. Derivation of the multistage model, where the data are assumed to be linear at low doses.

LOAEL. Lowest-Observed-Adverse-Effect Level; the lowest dose in an experiment which produced an observable adverse effect.

NOAEL. No-Observed-Adverse-Effect Level: the highest dose in an experiment which did not produce an observable adverse effect.

NOEL. No-Observed-Effect Level; dose level at which no effects are noted.

NTP. National Toxicology Program.

Oncology. Study of cancer.

One-hit model. Mathematical model based on the biological theory that a single "hit" of some minimum critical amount of a carcinogen at a cellular target -- namely DNA -- can initiate an irreversible sequence of events, eventually leading to a tumor.

Oral. Of the mouth; through or by the mouth.

Pathogen. Any disease-causing agent, usually applied to living agents.

Pathology. The study of disease.

Permissible dose. The dose of a chemical that may be received by an individual without the expectation of a significantly harmful result.

Pharmacokinetics. The dynamic behavior of chemicals inside biological systems; it includes the processes of uptake, distribution, metabolism, and excretion.

Population at risk. A population subgroup that is more likely to be exposed to a chemical, or is more sensitive to a chemical, than is the general population.

Potency. Amount of material necessary to produce a given level of a deleterious effect.

Potentiation. The effect of one chemical to increase the effect of another chemical.

ppb. Parts per billion.

ppm. Parts per million.

Prevalence study. An epidemiological study which examines the relationships between diseases and exposures as they exist in a defined population at a particular point in time.

Prospective study. An epidemiological study which examines the development of disease in a group of persons determined to be presently free of the disease.

Qualitative. Descriptive of kind, type or direction, as opposed to size, magnitude or degree.

Route of exposure. The avenue by which a chemical comes into contact with an organism (e.g., inhalation, ingestion, dermal contact, injection).

Safe. Condition of exposure under which there is a "practical certainty" that no harm will result in exposed individuals.

Sink. A place in the environment where a compound or material collects (see reservoir).

Sorption. a surface phenomenon which may be either absorption or adsorption, or a combination of the two; often used when the specific mechanism is not known.

Stochastic. Based on the assumption that the actions of a chemical substance results from probabilistic events.

Stratification. (1) The division of a population into subpopulations for sampling purposes; (2) the separation of environmental media into layers, as in lakes.

Subchroni- ·tion, usually used to describe studies
or l .. five and 90 days.

Synergism. ..on of two or more chemicals that results in an effect that is greater than the sum of their effects taken independently.

Systemic. Relating to whole body, rather than its individual parts.

Systemic effects. Effects observed at sites distant from the entry point of a chemical due to its absorption and distribution into the body.

Teratogenesis. The induction of structural or functional development abnormalities by exogenous factors acting during gestation; interference with normal embryonic development.

Teratogenicity. The capacity of a physical or chemical agent to cause non-hereditary congenital malformations (birth defects) in offspring.

Therapeutic Index. The ratio of the dose required to produce toxic or lethal effect to dose required to produce non-adverse or therapeutic response.

Threshold. The lowest dose of a chemical at which a specified measurable effect is observed and below which it is not observed.

Time-Weighted Average. The average value of a parameter (e.g., concentration of a chemical in air) that varies over time.

Tissue. A group of similar cells.

APPENDIX II

TOPICS COVERED IN "WORKSHOPS ONSASSESSMENT AND MANAGEMENT OF DRINKING WATER CONTAMINATION," US EPA (1987).