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TOXICOLOGIC RISK ASSESSMENT FOR GROUNDWATER CONSTITUENTS

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Introduction

Groundwater throughout the United States contains both inorganic and organic nonwater chemicals (constituents). Many constituents occur in groundwater naturally, sometimes enhancing the aesthetic or nutritive properties of the resource. In some cases, however, natural constituents and those which result from human activities occur at excessive concentrations. Excessive concentrations of constituents may present a risk to the health of people who consume (or otherwise use) the groundwater. Comparison of constituent concentrations to such regulatory standards as federal Maximum Contaminant Levels (MCLs) or Maximum Contaminant Level Goals (MCLGs) or to state groundwater standards is not an adequate representation of human health risk, since these standards may not be directly tied to human health risk; standards may also consider environmental risk, aesthetic factors (e.g., taste), or feasibility and cost of treatment. Further, a generic standard cannot consider actual exposure patterns. Instead, the potential for groundwater constituents to cause adverse health effects can be evaluated using toxicologic risk assessment tools. Some basic concepts in toxicology will be reviewed and potential pathways for exposure to groundwater will be discussed.

Risk is a function of exposure to a constituent and that constituent's inherent hazard. I aspire not to explain how to evaluate human health risks associated with constituents present in groundwater, but rather to familiarize the reader with general approaches and major sources of uncertainty. Thus, the reader will be in a better position to understand the implications of toxicologic risk assessments and to make informed decisions about use or remediation of affected groundwater. This paper will address hazard first, and then exposure.

Hazard: Concepts in Toxicology

The basic premise of toxicology is the centuries-old adage that "the dose makes the poison." For each chemical, there is a dose (an exposure level) below which no harm (or no discernible harm) will occur. For instance, arsenic, that quintessential poison, may be a required nutrient at low doses. Even water, an absolute requirement for survival, can kill: although we drink an average of 1.4 liters of water each day (USEPA 1989), inhalation of only a relatively small amount of water can cause death (drowning).

In addition to illustrating that "the dose makes the poison," water demonstrates the importance of route of administration. The three environmentally relevant routes of administration are oral (ingestion), inhalation (respiratory), and dermal (skin). Each may be important for constituents in groundwater: (1) When we drink affected water we are also ingesting the constituents. Similarly, we may consume produce which contains constituents from affected irrigation water.
(2) Constituents can also volatilize (evaporate), thus moving into air which may be inhaled. (3) Constituents can be absorbed from affected water which comes into contact with the skin.

Another underlying concept in toxicology is that there are numerous different and independent toxicologic endpoints (different types of adverse

health effects). Any given chemical may cause several different adverse effects at different exposure levels or by different routes of administration. One such adverse effect is cancer. While all chemicals cause noncancer adverse health effects, only some, carcinogens, cause cancer. Other adverse health effects include skin rashes, organ damage (e.g., liver, kidney, or central nervous system), reproductive effects, blindness, birth defects, and death. Adverse health effects are typically evaluated in two groups: cancer effects and noncancer adverse health effects. This division was originally based on a belief that cancer is caused by a wholly different biological mechanism than other adverse health effects. More recent research has demonstrated that chemicals can induce cancer by a variety of means, many of them similar to mechanisms which cause noncancer adverse health effects. Retention of differential treatment of cancer and noncancer health effects probably results from habit and society's perception of cancer as a particularly bad, severe or deadly effect.

In general, many cells in a given tissue all perform the same function. Therefore injury to, or death of one or a few, cells is not biologically relevant — other cells are able to compensate. However, when enough cells of one type are damaged or killed, the organ or system may cease to function properly, or the compensatory efforts may become pathological. These are adverse effects, but they occur only after a large number of similar cells are damaged or killed. Hence, we have the concept of a "threshold" level of exposure (or damage) below which no biologically relevant adverse health effect occurs. At and above this threshold there is an adverse health effect. In practice, we cannot pinpoint the threshold, either for a population or an individual, but this concept of a threshold is basic to risk assessment for noncarcinogens.

Cancer is thought to result from damage to the genetic material of a single cell which then reproduces in an uncontrolled manner, perhaps metastasizing to other organ systems. In theory, a dose as low as a single molecule could cause cancer — it must only cause the necessary damage to the genetic material of one cell. Hence, there is no threshold below which biologically relevant damage will not occur, and cancer is known as a "nonthreshold" effect. The body, of course, has many protective mechanisms which make this one-molecule, "one-hit" scenario unlikely. For instance, the single molecule could interact with biologically irrelevant cellular molecules, or be metabolized, or cause genetic damage which kills the cell or is irrelevant, or cause genetic damage which is repaired. Also, we have learned that many chemicals cause cancer through a mechanism which does not involve direct genetic damage. Rather, these chemicals bring about an increased rate of cell production which decreases the likelihood that naturally occurring genetic damage will be properly repaired. For these "nongenotoxic" carcinogens, there is a threshold level of exposure below which the rate of cell production will not be affected.

Although the ability to cause specific adverse health effects is an inherent property of each chemical, many individual factors also affect the toxicity of chemicals. Some of these factors can be controlled, but others cannot. Among those which cannot be controlled are genetic background, previous illness, and general state of health. Other individual factors such as diet, lifestyle, exposure to pharmaceuticals, and smoking can be controlled. There is no way to account for these factors in a risk assessment except to be very conservative (risk overestimating) in risk evaluation.

There are two primary sources of information elucidating the toxic effects of environmental chemicals: studies of humans and studies of laboratory animals. There are two types of studies which provide direct information about health effects in humans. The first type, clinical studies, involves intentional exposure of humans to chemicals, typically pharmaceuticals. These are seldom an important source of information

exposed population is small, (2) the exposed population is often special (requiring some sort of medical treatment), (3) therapeutic doses are much higher than environmental exposures, and (4) most environmentally important constituents have not been used as pharmaceuticals (notable exceptions include chloroform and similar chemicals which have been used as anesthetics).

The other type of studies of humans is epidemiologic studies, which evaluate human populations that had accidental, occupational, or environmental exposure to chemicals. These are useful because they provide information about effects in humans who may have been exposed at environmentally relevant concentrations. (Of course, some accidents and some occupations result in exposure levels higher than those generally associated with environmental exposures.) However, there are many factors which make epidemiologic studies difficult to interpret. For instance, study populations are usually small, so that the frequency of an effect would have to be very high in order to be noticed. Populations can also be very difficult to follow over the course of the several decades that may be required for some effects to become noticeable. Further, the route of exposure may be different from the route of interest, and humans are constantly exposed to other chemicals (e.g., tobacco smoke, occupational constituents) which may mask the effects of the chemical of interest or make it impossible to determine the cause of a particular effect. Another concern is that the exposure dose is seldom quantified in epidemiologic studies, so a dose-response relationship cannot be established. Finally, it can be difficult to identify appropriate control (unexposed) populations.

Thus, data for use in toxicologic risk assessment most often come from studies performed in laboratory animals. In these studies, the route of administration and the dose level can be controlled, and genetically homogeneous animal populations can be used. Further, exposure to other constituents can be minimized, and it is treated animals live in the same environment. Unfortunately, in order to recognize rare effects, animal studies typically use extremely high doses of chemicals. Interpretation of these studies in light of effects that would occur at the lower levels to which humans might be exposed is complicated and uncertain. And, of course, laboratory animals are not humans.

Exposure

People can be exposed to constituents in groundwater by all three exposure routes (ingestion, inhalation, and dermal) and via many different exposure pathways. Several of these exposure pathways occur indoors (e.g., ingestion of drinking water, bathing/showering, and exposure to indoor air which is affected by groundwater constituents). Other pathways include ingestion of homegrown produce which is affected because it was irrigated with affected groundwater, ingestion of food which is prepared in affected groundwater, and dermal and inhalation exposure related to irrigation. Traditionally, toxicologic risk assessment has considered only the ingestion (drinking) pathway of exposure to water, but other pathways can also be important. This section will discuss some of the salient factors associated with evaluation of the drinking-water exposure pathway and others. The goals in evaluating exposure pathways are to identify those which are important in a particular case and to obtain estimates of constituent intake or dose. This information is then combined with hazard-related information to obtain both qualitative and quantitative evaluations of risk.

People can also be exposed to constituents present in groundwater following discharge of the groundwater to surface water. Important human health-related factors under this scenario would include dilution, attenuation of constituent concentrations, direct exposure to surface water, other uses of surface water, effects on and exposure to sediment, and resultant constituent concentrations in fish which may be consumed.

These indirect pathways of exposure to affected groundwater will not be discussed here.]

The first factor which must be considered when evaluating an exposure pathway is constituent concentration at the point of exposure. For exposure pathways which involve direct exposure to groundwater (e.g., drinking, dermal contact during bathing/showering), the concentrations which were found in the groundwater itself are typically used as exposure point concentrations. These concentrations may not perfectly represent actual concentrations at the point of exposure. For instance, constituent concentrations may be affected by beverage preparation methods (e.g., boiling of tea water increases volatilization and so decreases the concentration of volatile constituents in the water; by decreasing the amount of water without affecting the amount of metals, boiling could increase the concentration of metals in the water). Such alterations in constituent concentrations are not usually considered in exposure evaluations. When the medium of interest is not water itself (e.g., when the pathway involves exposure to air that may contain constituents which volatilized from water, as in the cases of both irrigation water and showering), we typically face additional uncertainties. For instance, unlike the concentration in groundwater (which is usually measured), concentrations in the air are seldom known because air is seldom monitored. Even if air were monitored inside or outside of representative homes, there would be no way to account for nonwater sources of constituents, such as carpet glue or the gas station down the block. In the absence of actual monitoring data, constituent concentrations in air are typically estimated using equations which include constituent concentration in groundwater as an input parameter. This contributes uncertainty. These equations generally overestimate exposure concentration, often by orders of magnitude. Available equations for estimating concentrations in irrigated produce (or livestock which consume affected groundwater or crops) contribute so much uncertainty that expensive chemical analysis of the food product is almost a necessity.

It is also important to remember that, although exposure equations typically assume that constituent concentrations remain static over time, this is not likely to be true. Rather, constituent concentrations in groundwater (and so, in other media) will change over the course of time. This is particularly true for concentrations in affected homegrown produce, because over several seasons irrigation can contribute to increased constituent concentrations in soil which may also be taken up by plants.

Another important factor is the rate of exposure to the affected medium — the amount of affected groundwater which is consumed or which comes into direct contact with the body, the amount of affected air which is inhaled or the quantity of affected produce consumed. Traditionally, it has been assumed that a person drinks 2 liters of affected water per day (USEPA 1986). A more recent estimate of daily water consumption is 1.4 liters. This includes not only water but water-based beverages (juice concentrate, coffee, tea, etc.) and represents daily consumption from all sources (e.g., at home, at work). Exposure estimates typically assume that all of the 1.4 liters of water come from the same affected source. This overestimates exposure (and so, risk). An individual's activity level greatly affects the quantity of water which is consumed.

For affected air, 23 cubic meters per day is an estimated breathing rate for an adult *(53* F.R. 148). More detailed estimates of breathing rate by gender, age, and activity levels are available (USEPA 1989). Most adults do not spend all day every day in the affected building or area (e.g., at home), and this is an important consideration in exposure estimation. In some cases it may be most effective to consider showering as a separate inhalation exposure, since concentrations during showering are higher than concentrations in general indoor air.

Dermal exposure to affected groundwater is difficult to estimate because it occurs during so many activities (bathing/showering, hand washing, dishwashing by hand, household cleaning). The key factor here is the surface area of the exposed skin. The total skin surface areas of adult males and females are about 1.94 and 1.69 square meters, respectively (USEPA 1989). Surface area estimates are also available for body parts (e.g., hands) and for children.

The quantity of affected produce consumed can be estimated based on intimate knowledge of the exposed population or can be derived from other studies (USEPA 1989). Estimates of consumption of homegrown beef and dairy products are also available and can be considered when livestock consume affected groundwater or crops irrigated with affected groundwater.

There are compilations of information about daily activities which can facilitate estimation of the duration of exposure. For instance, most Americans bathe or shower once each day, and the median duration of a shower is about 17 minutes (USEPA 1989).

Exposure estimates are typically in units of milligrams of constituent per kilogram body weight per day (mg/kg/d), thus the body weight of the exposed person has an integral role in exposure estimates. Typical assumptions include a 70-kilogram man, a 60-kilogram woman, and a 10-kilogram child.

The duration of exposure to affected groundwater or air is also an important factor. Traditionally, 70 years has been used as the life-span (USEPA 1986), but more recent evaluation of relevant data suggests that 75 years would be more appropriate (USEPA 1989). If the affected supply is to a residence, 9 to 30 years may be appropriate, since most people move from one home to another (USEPA 1989). If the affected groundwater or air is in a workplace, it might be more reasonable to consider 40 years (the entire working lifetime), but only *5* days each week.

Appropriate use and interpretation of risk assessments for groundwater which contains constituents rests on an understanding of the basic concepts of toxicology and of exposure estimation. Considerable uncertainty is inherent in the "hazard" component of risk. Data must often be extrapolated from animals to humans and from high doses to low doses. Further, there is currently no way to address chemical or biological interactions among the many constituents which may occur together in groundwater. Many other assumptions and conventions also affect the interpretation of hazard-related information.

The exposure component of risk is also fraught with uncertainty. Actual constituent concentrations in the groundwater are not static over time, and resultant concentrations in air or food are not known with certainty. It can be difficult to include all relevant exposure pathways (e.g., drinking water, indoor air, affected produce) in the risk evaluation, and, in fact, this is typically not attempted.

The assessment of toxicologic risk associated with constituents in groundwater should be undertaken with an appreciation of the science of toxicology and with respect for the many sources of uncertainty associated with both hazard and exposure. Responsible risk assessors produce meaningful risk estimates which are well documented so that the informed reader can identify and appreciate major sources of uncertainty. These uncertainties, which are typically addressed by using conservative assumptions and methods, do not detract from the usefulness of the risk evaluation. Rather, toxicologic risk assessment is a powerful tool which, when put into proper perspective, contributes to effective decision making about the use, value, or remediation of groundwater which contains constituents.

References

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