Draft Lecture Notes For a Test of Teaching

Basic Elements of Health Risk Assessments

to a lay community audience

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by

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Basic Elements of Health Risk Assessments

Introduction

- Health risk assessments (R.A.) are a valuable tool for decision making. Too often people in the lay community think this tool is impossible for those without a strong science background to understand. In some respects it is, but just as in the case of driving a car, one doesn't need to know how the engine or brakes work to be able to use the car for ones own benefit. The purpose of this training is to take some of the mystery out of understanding the methods and processes involved in R.A. so that the lay public can contribute to and get benefits out of this valuable but imperfect tool.

- Health risks can be differentiated into voluntary and involuntary health risks; these type of risks should be put in balance with other risk factors such as:
  - economic risks
  - security risks
  - environmental risks
  - social risks

- Examples of voluntary and involuntary <u>lifetime</u> death risk statistics:

activity	voluntary	involuntary
dying from riding in a car	2×10^{-2}	
dying in a tornado		4×10^{-5}
dying from cancer		2×10^{-1}
dying from cancer induced by pesticide residues on fresh foods		4×10^{-3} conserv. est.
dying from industrial accident	3×10^{-3}	
dying from working as fireman	3×10^{-2}	
dying from a boating accident	4×10^{-3}	
dying from a hunting accident	2×10^{-3}	
dying from smoking 1.4 cigarettes	1×10^{-6}	
dying from 1 chest X-ray	1×10^{-6}	
dying from eating 40 tablespoons of peanut butter	1×10^{-6}	
dying from living next to a Brownfield cleanup site		conservative est. 1×10^{-6}

- Evaluating involuntary toxicological exposure risks to the public requires an objective site specific analysis of those health risks and putting such risks in balance with other risks to arrive at acceptable level of risk. Such a risk often is defined as 1 X 10^{-6} probability of dying from cancer or non-cancerous disease as a result of a life time exposure to toxic chemicals from all routes of exposure based on a number of conservative assumptions. The community effected by this increased risk has a right to know about the risks and a right to participate in developing a consensus about how to balance those risks against other risks in the community. These rights create an obligation to try to understand basic aspects of toxicological risk assessments.

Summary of the Basic Four Step Health Risk Assessment (R.A.) Approach Process:

Hazard Identification

In this step the possible existance of toxic chemicals at a site (e.g. chromium and PCB's) are investigated by sampling the site's soil and ground water, chemically analyzing those samples, and computing the range of concentrations noted. This step involves use of leachability tests to enhance leachable extraction of chemicals to ground water, and performing a statistical analysis on the range of concentrations to estimate the upper 95% confidence level around the mean for each identified pollutant. This means there is a likelihood that 95% of the concentrations in the soil or ground water at the site are lower than the concentrations used in the risk analysis. It also assumes the chemicals that are in solid waste are all in a small size range (e.g. face powder) so they can be easily taken in by the body. Many times, however, the solid waste material is in a bulk that can not be easily absorbed by the body. The laboratory methods require such bulky waste be broken up for chemical analysis.

The community has little practicle reason for meaningful involvement in this step unless it wants to take on the expense of the sampling and chemical analysis. This may not be economically wise since the sampling is typically done by competent consultants using standard methods with the whole process overseeen by FDEP. The local community should take comfort that sampling and laboratory methods have been standardized and involve use of use of controls to assure objectivity. Also results are reviewed by FDEP or some other regulatory agencies. Due to the conservative use of leachability test data, the use of the upper 95% confidence interval, and the assumption the chemicals are all in a small physical size that can be easily taken up by the body, it is likely this step introduces an overestimate of concentrations of any toxic chemicals at a site that could be taken up by the body.

For the Chambers site in east Jacksonville, sampling and analysis has apparently identified two toxic chemicals above background levels; chromium and polychlorinated biphenyls. These two chemicals are then focused on as "compounds of potential concern".

Toxicity Assessment

The purpose of this step is to determine the relationship between the magnitude of exposure often called the "dose" and the possible occurrence of potential negative health effects to a human receptor. This health effects factor is called the "dose response". The dose response means the potential of a person getting cancer or some other noncancer disease like asthma. The dose response is typically in units of mg/kg/day. The mg is the amount of "dose" of the pollutant taken in per kg of human body weight per day that are conservatively estimated to produce a negative health effect (such as cancer) to the whole body or some critical organ.

In this step there is no potential for the public having any meaningful input to changing data used in this step. This step is one requiring health specialists in various fields working together to arrive at a common conservative consensus. EPA, OSHA, FDEP, the Florida Dept. of Public Health, the Communicable Disease Center, and the Agency for Toxic Substances and Disease Registry can all contribute their own science policy for producing dose response criteria for various toxic pollutants. Applicants and the public have little ability to influence these government science policy makers. However, applicants and the public needs to understand and appreciate the conservative way "dose response" curves are calculated.

First recognize that many risks to human health are "unprovable" because the dose is so small or the exposure time so brief to allow any measure of directly observed health effects. What happens is that effects are noted at very high dose rates over short exposure times. The ability to produce a numerical estimate in a R.A. of an unproven risk does not mean that the risk is proven. Risks are unproven because of significant gaps and uncertainties in scientific knowledge. Government policy decisions are made by health experts for purposes of R.A. to bridge the gaps and uncertainties.

For example in bridging these gaps in scientific knowledge, health experts assume there is no "safe threshold" for an exposure or dose levels. They thus construct curves that extend effects observed at high dose rates down to very low dose rates. The body, however, is known to have natural protection mechanisms that can handle some small doses for many pollutants with no apparent adverse effects.

The extent and existence of conservative science policy in R.A. are rarely fully and fairly disclosed. Yet this science policy results in regulatory decisions that the public often doesn't understand or have an ability to contribute to.

Remember that the toxicity dose response factors that consultants use in R.A. for "compounds of potential concern" come from EPA and other government science policy experts and are based on very conservative assumptions which likely over estimate dose effect by several orders of magnitude. Scientist recognize the conservative way toxicity values are derived yet resist deviating from using less conservative values until hard evidence is available on some of the missing gaps in scientific knowledge.

Exposure Assessment

This is the main step in the R.A. process that the public needs to carefully understand and possibly contribute their own thinking towards developing a consensus with the developer applicant and the FDEP about how to estimate this important parameter.

Remember, for adverse health risk to occur two factors are needed; toxicity and exposure. Toxicity can be generally inferred from the presence of toxic chemicals above background concentrations, but exposure should not be inferred it needs to be looked at on an individual site by site bases.

Exposure or dose depends primarily on the magnitude and frequency of potential human exposure to "compounds of potential concern".

Exposure is made up of direct and indirect exposure. Direct exposure is what FDEP R.A. policy assumes occurs when they derived the "Soil Cleanup Target Levels" for "compounds of potential concern". It's defined as exposure that occurs when the pollutants come into direct contact with a human. However, it is possible to have some indirect exposure which is exposure that occurs indirectly by ingestion of food that has become contaminated or taken up because the food was exposed to the "compounds of potential concern". If food crops or animals are raised in any area that is contaminated with these pollutants then this indirect exposure needs to be taken into account. It would appear that no one in the inner city is or would be allowed to grow crops or raise live stock on contaminated sites so for practical purposes indirect exposures could possibly be ignored.

Human exposure is also greatly dependent on:

1 Characteristics of the site and surrounding area,

e.g. where below the surface pollutants located and how mobile those pollutants are.

2. Physical and chemical form pollutant is in
   e.g. is the size and shape and chemical form it's in that make it more or less prone to getting out from the site and being absorbed by the body?

3. How the site is used,
   e.g. commercial, industrial, residential, institutional uses

4. The engineering and institutional safeguards that apply to cleanup and subsequent reuse of the site,
   e.g. how will dusting during remediation activities be controlled, how secure will the encapsulation material be, and how will site use be controlled and monitored.

5. The duration and frequency of human activities that occur on and around the site.
   e.g. will anyone be using local shallow ground water as a potable water source

6. The dose response sensitivity or vulnerabilities of the exposed community due to age, or other existing health conditions which tend to make the dose response greater.
   e.g. young children which tend to have greater uptake factors and ingest more dirt.

There are 3 recognized routes of exposure. Ingestion of pollutants and possible uptake through the gastro-intestional tract, inhalation of contaminated dust or gases with possible uptake in the lungs, and dermal contact with possible uptake of the pollutants through the skin.

When FDEP calculated their generic "Soil Cleanup Target Levels" they assumed all three pathways occurred simultaneously all the time. In actual site specific cases these three pathways of exposure will likely not be simultaneously all the time. If the pollutant does not exist as a gas or will not likely become airborne than the inhalation pathway may not be important, or might only be important during a short period of the remediation process. Similarly, if the people around the site could be stopped from coming into direct contact with the soil, there maybe no dermal or ingestion pathway exposure or the exposure could be for only very brief periods with frequent or infrequent repetition.

Duration and frequency of exposure is a key factor to consider when calculating exposure or total dose. Science policy requires calculation of lifetime exposures which defined as exposure over 70 years. While the FDEP assumed continuos and simultaneous exposure at a maximum exposure rate for calculating their "Soil Cleanup Target Levels", site specific R.A. can look at exposure in a less conservative way if site specific factors on exposure duration can provide justification for this. For example a site may only produce exposure during the period of construction of remediation or application of engineered safeguards. In this case the exposure duration might be only 12 months instead of 70 years.

The magnitude and even the potential for exposure existing during and following Brownfield remediation is likely to largely depend on site specific the engineered and institutional controls that are proposed and agreed to.

Exposure can also depend on "degradation, decomposition and dispersion" in calculating actual exposure concentrations as compared to concentrations measured in the subsoil or ground or surface water when any of those contaminated mediums were initially sampled compared to when exposure occurs. No adjustment was made for these factors by FDEP when they estimated 1×10^{-6} risks for their generic "Soil Cleanup Target Levels". However, in FDEP's rule (62-785.690) they recognize the potential for "natural attenuation" as an appropriate strategy for site rehabilitation. Natural attenuation recognizes and takes into account degradation, decomposition and dispersion.

In summary the important site specific variables effecting exposure are:

1. The direct or indirect (or both) nature of the exposure potential.
2. Characteristics of the site effecting pollutant mobility.
3. The physical and chemical form the pollutant is in effecting mobility and uptake.
4. Use of the site as it effects exposure pathways exposure duration and the voluntary or involuntary nature of the exposure.
5. The engineered and institutional controls as these effect exposure duration and frequency.
6. The duration and frequency of exposure as these effect overall exposure time.
7. The dose response sensitivity of the exposed population at risk.
8. The pathways of exposure that appear likely to come into play.
9. The duration decomposition and dispersion of the "compounds of potential concern".

All these exposure factors are up for intelligent but still somewhat arbitrary consensus in helping calculate the type of exposure that should apply on a site specific bases.

Understanding and contributing to a consensus agreement in this step of R.A. quantification is where the local community should concentrate it's efforts to contribute to a site specific R.A.. Here, as well as with the other R.A. steps some degree of arbitrary conservative assumptions still come into play. However, the extent of these conservative assumptions will likely effect the economic viability of developing the contaminated site.

Risk Characterization

The Risk Characterization combines the results of the Exposure Assessment with the Toxicity Assessment for each "compound of concern" to derive conservative quantitative estimates of the potential for adverse health effects as a result of a proposed Brownfields remediation effort at a specific site.

For noncarcinogenic pollutants this risk is calculated as a Hazard Quotient ratio (HQ) of the average daily exposure or dose for the pollutant in units of mg/kg-day to a reference dose response also in the same units.

$$HQ = \frac{\text{average daily dose or exposure for a specific pathway (mg/kg-day)}}{\text{reference dose response for pollutant for specific pathway (mg/kg-day)}}$$

The reference dose response is established by science experts as that dose which is conservatively estimated to cause the adverse effect at a probability level of 1×10^{-6}. All other noncarcinogenic pollutants have their HQ ratio calculated the same way and then all noncarcinogenic HQ's are summed to yield a Hazard Index (HI) for a particular exposure pathway. In the same manner all other exposure pathways for noncarcinogenic pollutants are calculated and a HI for each pathway is produced If that HI ratio for all exposure pathways does not exceed 1, then the noncarcinogenic risk assessment standard FDEP has established of not exceeding an added adverse health probability over a lifetime to any individual of 1×10^{-6} for all noncarcinogenic pollutants has been met.

The preliminary information we were given on the Chambers site indicated there were no noncarcinogenic pollutants (or compounds of potential concern) of apparent concern at that site. If this preliminary finding is true then the R.A. could ignore this area of adverse health risk and go to calculating carcinogenic risk.

The two pollutants identified as "compounds of potential concern" at the Chambers site are creosol and PCB's, which are both carcinogenic pollutants.

The purpose of carcinogenic risk characterization is to estimate the probability, over background cancer rate, that an individual will develop cancer in his/her lifetime as a result of exposure to carcinogenic "compounds of potential concern". For the Chambers site this means creosol and PCB'.

For carcinogenic risk assessment the dose response for a pollutant is calculated as a cancer slope factor (CSF) or curve put in units of 1/mg/kg-day for a given exposure pathway that is estimated to give a cancer risk of 1×10^{-6}. The exposure is calculated as an average lifetime daily dose for that exposure pathway put in units of mg/kg-day, the same exposure units used for noncarcinogenic pollutants. When the product of the CSF and the dose is less than 1×10^{-2}, which typically is the case in R.A., the equation for a particular carcinogenic pollutant for a particular pathway of exposure is:

Excess Lifetime Cancer Risk = CSF for that pathway x average lifetime daily dose for that pathway

In the same manner as with noncarcinogenic risks, all exposure pathways for all carcinogenic pollutants are summed. If the risk assessment standard FDEP has established of not exceeding an added adverse health probability over a lifetime to any individual of 1×10^{-6} for all carcinogenic pollutants is not exceeded, then the FDEP standard has been met.

Conclusion

Remember that calculating a R.A. for toxic chemicals is a very inexact and often arbitrary science. It is important that the lay public realize how in exact this science method is. This means they should try to understand and appreciate where conservative assumptions that do not necessarily match with reality were used to bridge the gap in and uncertainties in scientific knowledge, data and risk estimation methods. As pointed out at the outset risks calculated from actual statistical data on observed effects are much lower than those were one has to estimate risk not on the bases of actual observed effects but on estimates or projections based on a long list of often arbitrary and conservative assumptions. Thus one should not misunderstand that an estimated R.A. of 1×10^{-6} is not a real risk potential but only one estimated by following the rules laid down by government scientific policy makers.