Environmental Remediation of Contaminated Sites
Prof. Bhanu Prakash Vellanki
Department of Civil Engineering
Indian Institute of Technology – Roorkee

Lecture – 06
Risk Assessment
Risk Characterization

Hello everyone. So again welcome back to the latest lecture session. So we have been discussing the relevant aspects of risk assessment right. In that context, we looked at why risk assessment is relevant or necessary and we looked at a few what do we say corollaries are there I guess our analogies pardon me right and then we started looking at the risk assessment in greater detail.

So risk assessment obviously looks at 4 major aspects, so one is hazard identification and data collection. The second aspect would be toxicity assessment or looking at the dose response behaviors right and then looking at exposure assessment and then bringing all these 3 aspects together with respect to risk characterization right. So these are the 4 major aspects of risk assessment.

In that context, we were already done with you know looking at briefly the relevant aspects with respect to hazard identification right and data collection and then we moved on to what is it now toxicity assessment and dose response behaviors and such right. In that context, we looked at why we need to look at toxicity assessment in greater detail right. Though, we do have the relevant what do we say variables or you know numbers given out there in the relevant databases.

It is because there are considerable uncertainties involved so it is worthwhile obviously to look at the relevant aspects ideas right. So in that context, we moved on to what we say generic example of dose response behaviors right. So here we have that particular aspect here.

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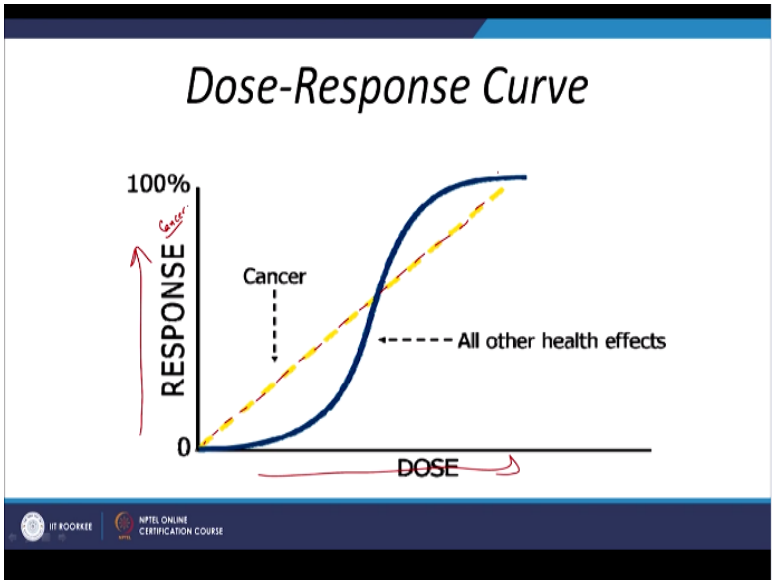

So with increasing doses as you see, you see increasing adverse responses right. It can be carcinogenic responses right or it can be any other non-carcinogenic responses. So it can be something as simple as hair fall let say right or damage to the kidney and such right not as simple as that though right. So again so cancer typically we assume that it is linear at the relatively low doses.

We are going to look at that in greater detail. So for the other aspects though this is a generic figure, this is typically not the case. We are going to again look at this in greater detail right, so again typical aspects I guess.

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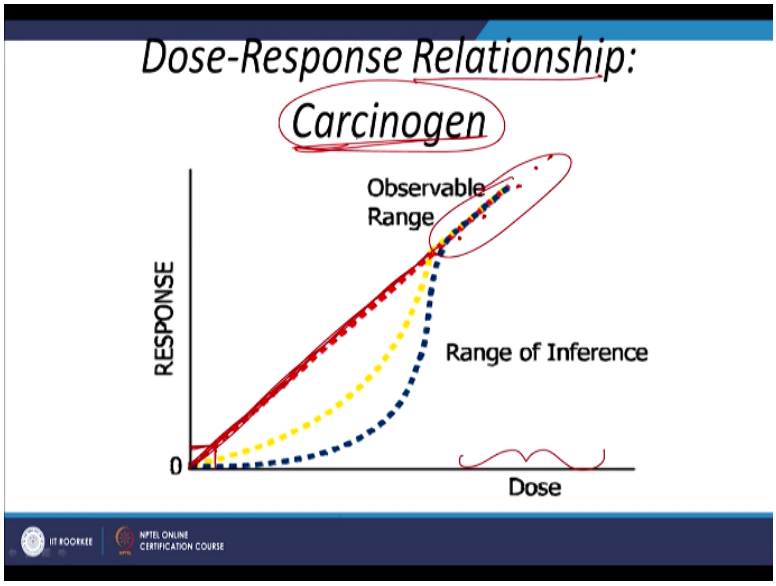

So here for carcinogens right, what are the aspects, as in I have my data only in this range I guess. So let say from my particular experiments on the animals or animal studies let say at

different doses right at different doses, these are the responses or carcinogenic responses that were observed right. So again as we looked at it earlier, understood it earlier typically right how do we go about conducting these trials let us say.

We accelerate to the trials right, that shorter periods we want to get it done within a shorter period of time not we there are when people I guess right and obviously to try to get to that relevant aspect may it increase the doses right, they increase the doses by quite few orders of magnitude from case to case I guess right. So obviously we have data only at higher doses right but obviously in general though we never we as in humans are rarely exposed to concentrations at these levels of doses that we administer to the animals right.

So how do we extrapolate the data? So in that context, let us say so here let us say there are different possible what we say patterns as in it could be this particular pattern right extrapolation by this particular model or the yellow model or the blue model right but typically though you know they go for the linearized multistage model or multistage linearized model let us say right.

And here they assume or take the most conservative estimate that even at low doses right. It is the response between what we say the behavior is linear between the dose and the response right. So even at very low doses right even at very low doses we assume that there is always going to be some carcinogenic response right. So that is the key aspect with respect to carcinogens.

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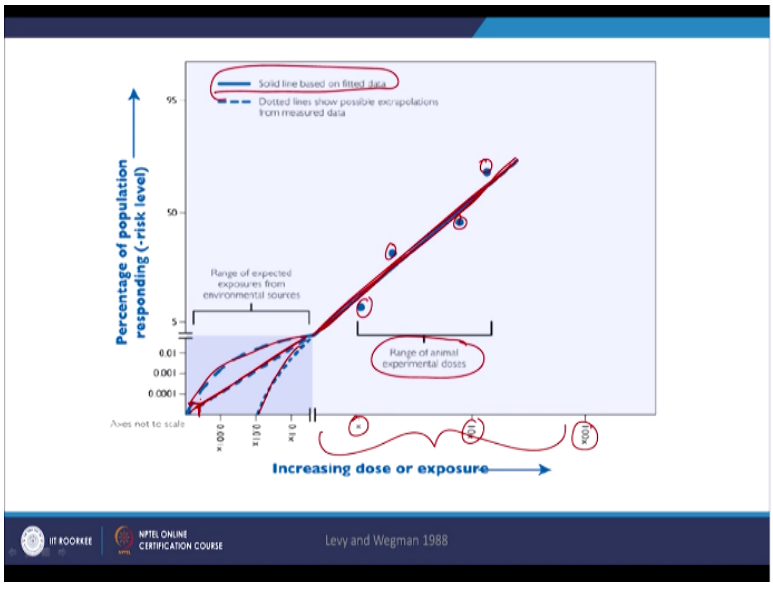

And again linearized multistage model, so here I have some actual data so looks like these are the data points on from the experimental doses or animal experimental doses right and here they came up with a particular model now right. So this is the solid line based on the fitted data, solid line based on the fitted data right but obviously they are conducted at they as in animal studies are conducted relatively high concentrations as you can see.

I believe this is logarithmic scale as you can see. So obviously there are different models right. Again, this is where some uncertainty lies but typically people assume that this linearized multistage model is what we say relatively conservative model right. Again, here the key is that even at low doses, we assume that there is going to be some adverse response as you can see out here right. So that is one particular aspect in that particular context right.

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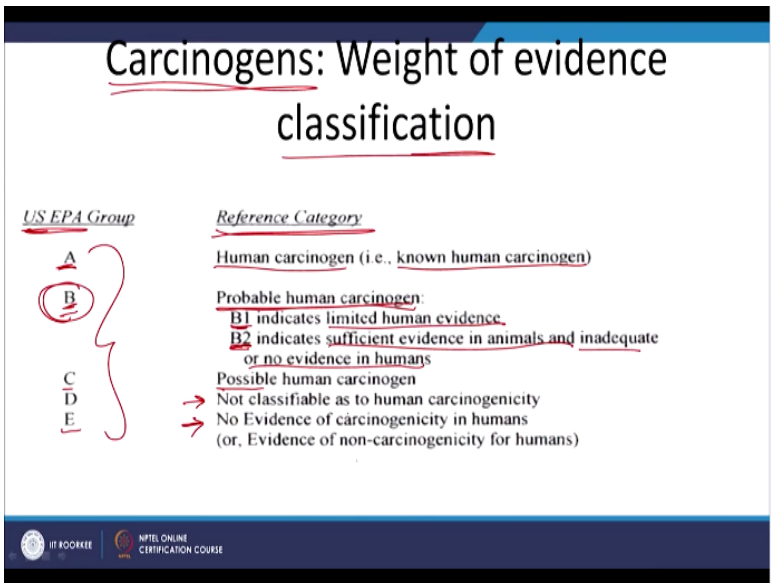

So let us move on, so here let us say carcinogens are classified right and typically we look at the US EPA classification. So here we have 5 different classifications and here is the reference category as in depends upon the level of or weight of evidence that has been gathered thus far right. So in that context, A you know the highest class human carcinogen as in it has been known to cause cancer in humans right, so that is the highest class.

In B, there are again two classes B1 and B2, so it is classified as the compounds that fall under category B or classified as probable human carcinogens right. B1 indicates there is some limited human evidence and B2 indicates sufficient evidence in animals but inadequate evidence or no evidence in humans right. So obviously B1 relatively higher and B2 relatively lower as in.

In B2, we do have what we say confirmation that the relevant compounds at the relevant exposures let us say or relevant doses cause cancer in the animals but there has been limited as in no studies might have been conducted right or studies might have been conducted and it is how there is no evidence let us say that the relevant compound has caused cancer in humans yet right, so again that is B.

And then C, possible human carcinogen, D not classifiable as to human carcinogenicity and E there is no evidence right as in after testing there is no evidence of carcinogenicity in human's right. So different classes, again this is something we need to be aware of right.

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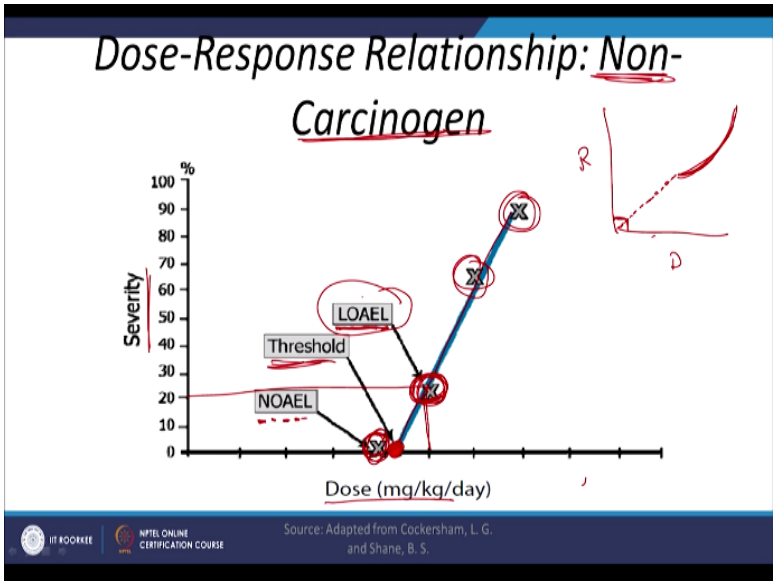

So here we come back to the non-carcinogens, earlier we looked at the carcinogens right. What did we look at there? We have the dose and response here and let us say irrespective to of what let us say for example this is the model let us say from the data that was actually observed from the animal studies but we assume that for carcinogens at the lower doses it is going to be a linearized model or linearized multistage model I guess linearized.

And we have 2 stages or different stages or multistages linearize multistage model right but for non-carcinogens as you see right there are different aspects. As in these are the data points here right. These are the data points again dose versus severity of this particular adverse response. So we have different data points from the animal studies here right. So let us look at how this is going to.

This as in the dose response behavior for non-carcinogens or just the toxic compounds is going to differ from how we look at or you know try to extrapolate the relevant effects from or for the carcinogens I guess right. So here as you see, we assume that there is a threshold level. As in, we develop the model based on the experimental data and you know that there is a threshold right.

As in below this particular threshold, you know if the dosage is below this threshold, we assume that it is relatively safe. So above that there are going to be adverse effects. So there is some sort of a reference or zero dose if you not zero pardon me a safe dose I guess right in the context of non-carcinogens. So in this context, there are 2 or 3 terms that we need to look at other than threshold.

One is the lowest observed adverse effect level or LOAEL, again I repeat it is lowest observed adverse effect level right. So obviously it is self-explanatory, what does it mean now? What is the lowest concentration as you see? This is the lowest concentration at which some adverse effect has been noticed right. So that is the lowest observed adverse effect level and then we have NOAEL which is the no observed or no observable adverse effect levels.

So what is the highest concentration right and this is the highest concentration at which no adverse effect was observed right, so NOAEL and LOAEL. So obviously you know the more the data you have, the better that you would you know the better kind of data that the better data that you would come up with, that can be used for better estimation. So depending on or we going with LOAEL or NOAEL obviously there are going to be uncertainties.

So we are going again look at these aspects. Again, what is the take-home message? For the non-carcinogens it is that we assume that there is a safe dose right unlike the cancer or carcinogens where we assume that even its low doses we assume that there is a response but we see that that is not the case out here for the non-carcinogens and then we came across 2 terms LOAEL and NOAEL right.

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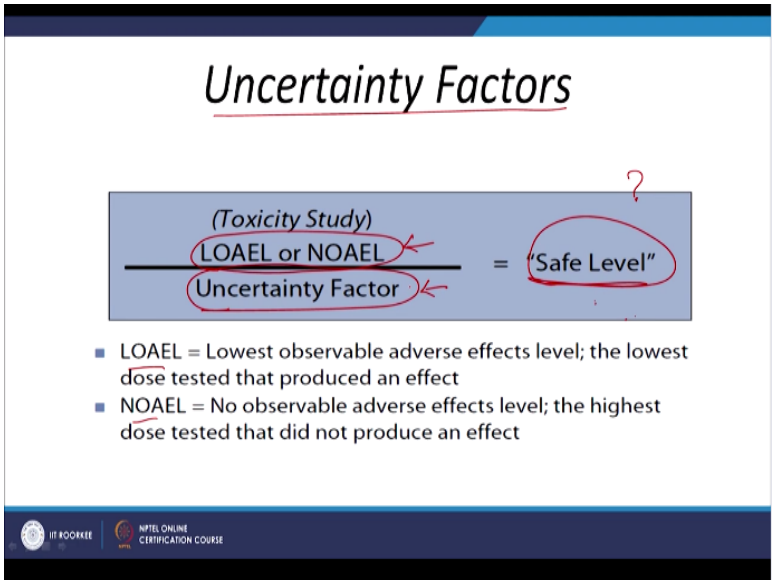

So again we are going to move on let us say, so here obviously we are need to calculate this safe level right. How do I calculate this safe level right? So it depends upon you know dividing the LOAEL or NOAEL that we just calculated and dividing that by different uncertainty factors right. Again, LOAEL lowest observable adverse effect level, no observable adverse effect level.

So we discussed this, so we are not going to go into that. So how do I so here as in safe level how do I come about that the safe level here that we are referring to refers to the safe level for the humans now. So here we are trying to what do we say estimate the safe level in the humans based on the relevant experiments or studies on the animals here. So how do I do that, dividing the LOAEL or NOAEL by some safety factors or uncertainty factors.

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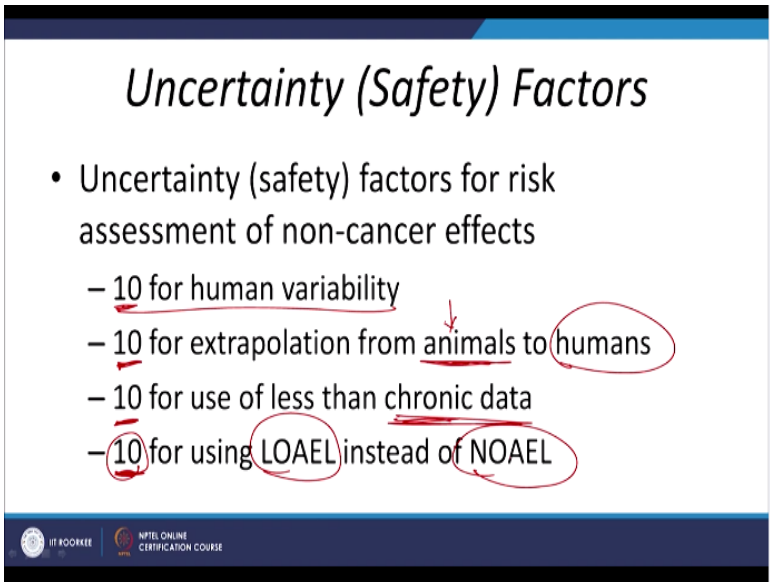

So let us look at what they are right. So 10 for human variability as in let us say my what we say response to being exposed to a particular carcinogen or toxic compound would be different to what your response would be right. So different people again let us say pregnant ladies, women, the elderly people and so on you know there will be variation from human to human. So for that particular aspect, we look at are considered 10 to be one uncertainty factor.

And certainly again 10 because obviously we are trying to what we say estimate the effects on humans based on animal studies right. So thus obviously we have another uncertainty factor that we take into account and 10 for less than chronic data right. As in as we discussed earlier right, we are trying to look at accelerated trials right. We are trying to estimate the what do we say effects based on what do we say conducting the trials at over lesser time ranges at higher doses right.

So obviously again there are going to be some such issues, so if it is not chronic data let us say and then again for that case we are going to use them and if obviously here this is under context we discussed slightly earlier, so I will use another uncertainty factor of 10 if it is I am using LOAEL instead of NOAEL right.

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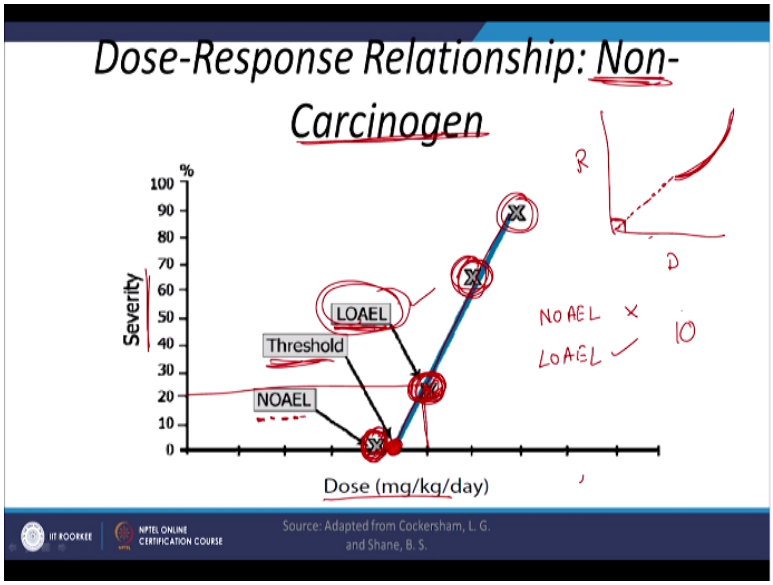

LOAEL again right if I look at this particular aspect see if I use LOAEL right, if I do not have this data point, this would have been my LOAEL right. So here it is always better to have data on NOAEL right, NOAEL rather than LOAEL because you know depending upon the limitations of your particular study, your LOAEL could be pretty high right then that would

lead to what we say under estimation of the adverse effects or you know under estimating or over estimating let us say in this context the safety level I guess right.

So obviously if I have only the LOAEL and not the NOAEL, I need to have an additional what we say safety factor or uncertainty factor right.

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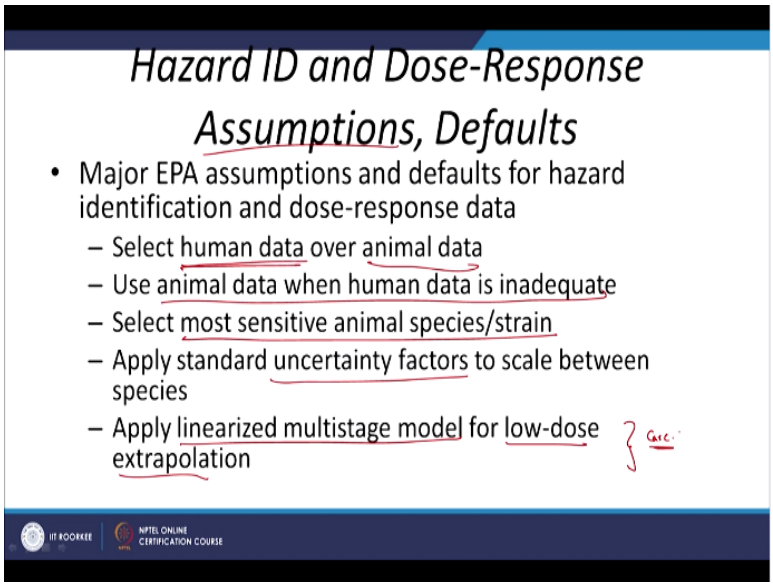

So from that I am going to be able to calculate my safe level for the humans right and some major assumptions I guess right what are they? So we are always try to select human data or animal data whenever we have such data right and use animal data obviously only when human data is inadequate and usually we look for the one that has you know we are trying to estimate something, obviously you need to have a relatively good correlation between what you observe and what you are trying to estimate right.

So obviously we are going to serve the most sensitive animal species or strain that is a different aspect obviously. So and then apply uncertainty factors to scale between species right and again linearize a multistage model for low dose extrapolation and this obviously we talked about in the context of carcinogenicity right, so linearized multistage model.

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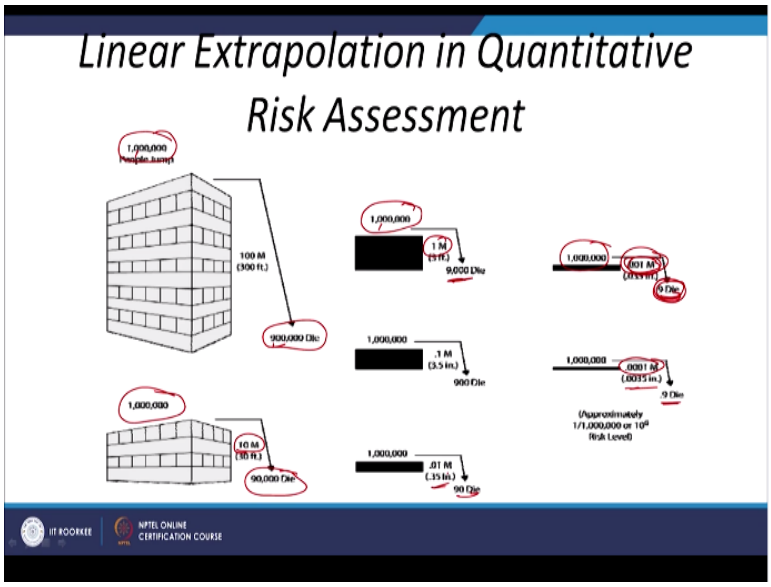

So again I mean just an example here I guess. So let us say a million people jump from a height of 100 meters, so let us say assume 900,000 or 9 lakh people die. So 10 lakh people jump 9 lakh people die let us say and let us say if people the same 10 lakh people jump from 10 meters 90,000 are going to die. Again, here we are just trying to illustrate the linearized multistage model I guess and why it is relatively conservative relatively.

So again if I then what is that you know extrapolate into so if 10 lakh people die from 1 meters, it assumes that still 9,000 people are going to die and it again assumes that even if 10 lakh people die from let us say what is it now 1 mm height I guess right, is it 1 mm, I assume so right, 9 people are going to die right. So you see that there is a built in not built in let us say this linearized multistage model what does it assume?

That there is going to be an adverse effect which is death in this case obviously I guess even at very low doses right, even at very low doses we assume that there is going to be some adverse effect right. So again some relatively conservative what do we say estimation and why is that because we are looking at cancer here right.

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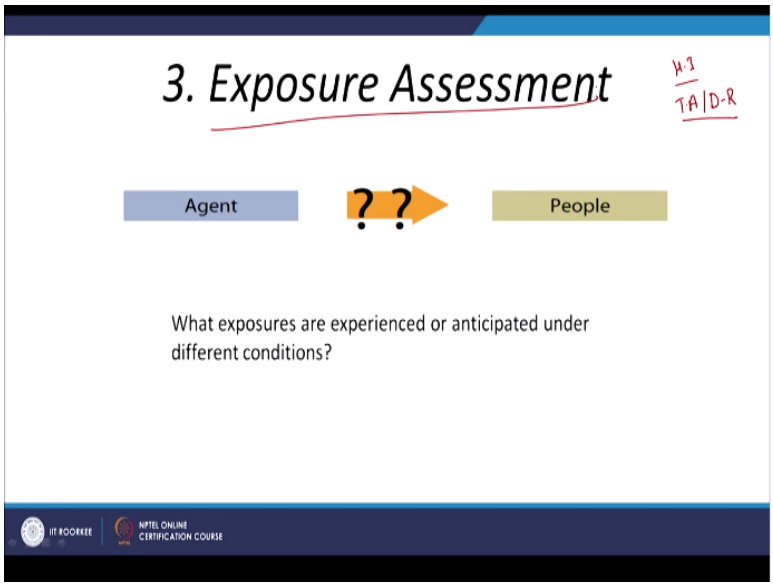

Again, moving on so we looked at hazard identification and data collection and then we looked at toxicity assessment and dose response curves or behaviors and then it is the exposure assessment I guess right. What exposures are experienced under these conditions or different conditions? As in how am I exposed to the relevant agent right. So human exposure evaluation, so what do I need to know?

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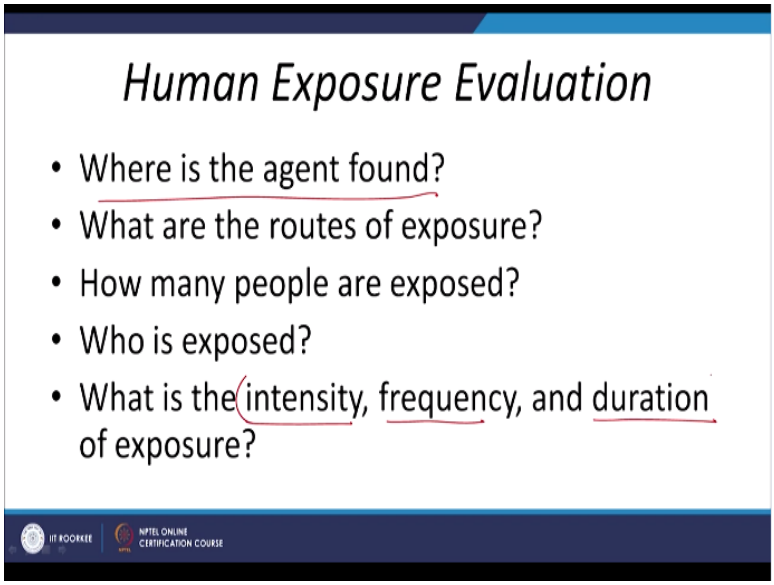

Where is the agent found right and my particular receptor I guess right? What are the routes of exposure? or am I exposed through the air pathway? and then I am going to inhale it or is it deposition on the soil and then I am going to have dermal contact with the soil or ingestion of the soil. So what are the routes of exposure? How many people or what is the level of population that is being exposed?

Who is exposed right? Is it what do we say healthy, what do we say humans or is it infants or let us say elderly people who are more susceptible, so what kind of population is being exposed right? So and then more importantly what is the intensity, frequency and duration right? So we are going to look at why this is relevant in the next slide I believe right.

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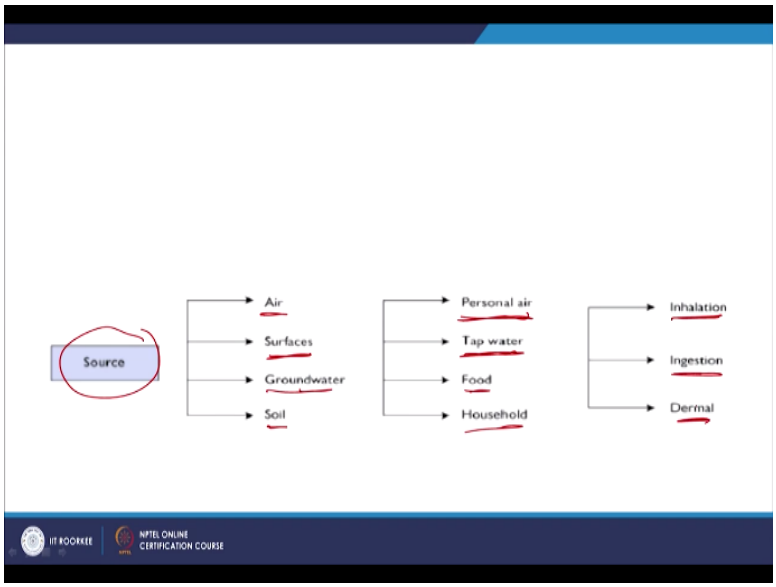

Again, I guess we have some other aspects, so again looking at the pathways, so this is the source right, different modes of transport right, through the air, surface, ground water or soil right and then while breathing personal air or from tap water food or household and then routes of exposure; inhalation, ingestion and dermal right. These are the different pathways obviously, that is an illustration out here.

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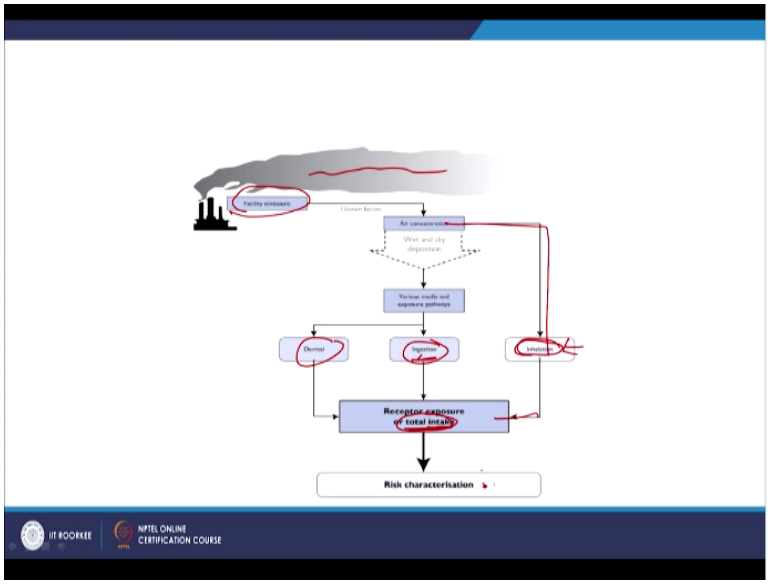

So here if we consider an example of let us say emissions from a particular factory now, what can happen now? You are going to have let us say concentrations of different contaminants in this particular plume outside in the atmosphere and then you can have wet and dry deposition that can what do we say have leads to ingestion or dermal contact or you can directly breathe the particular contaminated air and then that leads to intake right.

So we just looked at one example as in to look at or understand the different pathways at play here. So we are talking about air pollutions or emissions from particular industry and you have what we say the relevant contaminant traveling through the air or certain distance and then I end up either breathing the air directly which is inhalation directly in this case right or there can be deposition of the contaminant you know wet or dry deposition under the soil.

And different pathways let us say as in dermal contact or ingestion of that particular soil let us say or water let us say and then leading to intake of the relevant compound right and then obviously I need to look at risk characterization.

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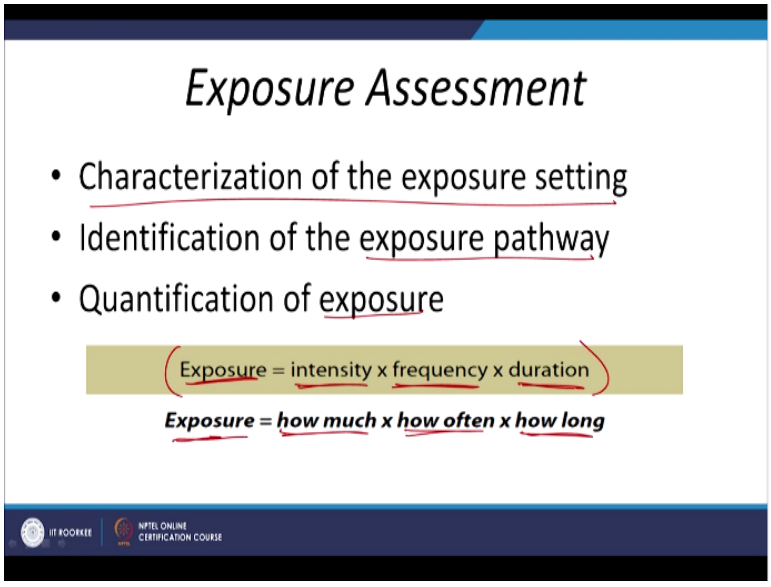

So in this context, as I talked about earlier, the exposure let us say depends upon intensity, frequency and duration or in Layman's terms how much am I being exposed to, how often am I being exposed to and how long right. So based upon all the data that we have at this until this stage we can come up with this exposure assessment right. How much am I taking in right, how often and how long?

How much, how often and how long right? Again, the uncertainties here are obviously are going to estimate some of these variables, you can have detailed studies but in general you are going to have to estimate some of these variables and again that is where the uncertainty again creeps into the picture right. Again, obviously as we talked about characterization of the exposure setting as we just looked at one example.

Identification of the exposure pathways and then quantification by this particular set of what we say formulae.

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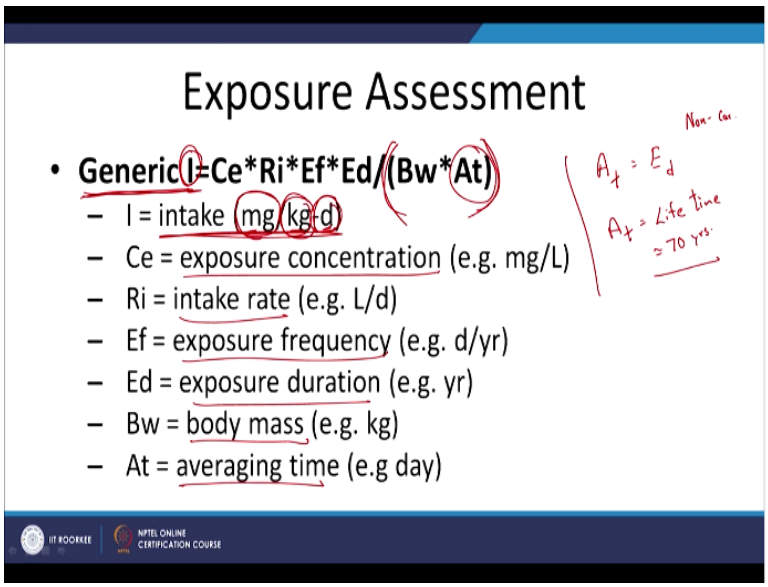

But here we have the generic formulae right where the intake is depends upon the exposure concentration, intake rate, exposure frequency, exposure duration and body mass or body weight and average time in the denominator right. Averaging time would be the exposure duration, exposure duration if it is for non-carcinogens right. For carcinogens, though the averaging time will be equal to the life time because we are looking at life time cancer risk.

And life time is typically assumed to be 70 years right. This is something that you need to keep in mind. So again as I mentioned, this is the generic formula. So for different kinds of intake as in soil ingestion, air inhalation, water ingestion, dermal contact and such, you have different other what we say variables coming into picture but here obviously you have the relevant aspects with respect to how much, how often and how long I guess right.

These are the aspects here and based on this, I am going to be able to calculate the rate of intake of that particular compound and what are the units? The mass of the contaminant per

mass of body weight or per unit mass of body weight per time per day I guess. Milligrams of the contaminant per kg of body weight per day right.

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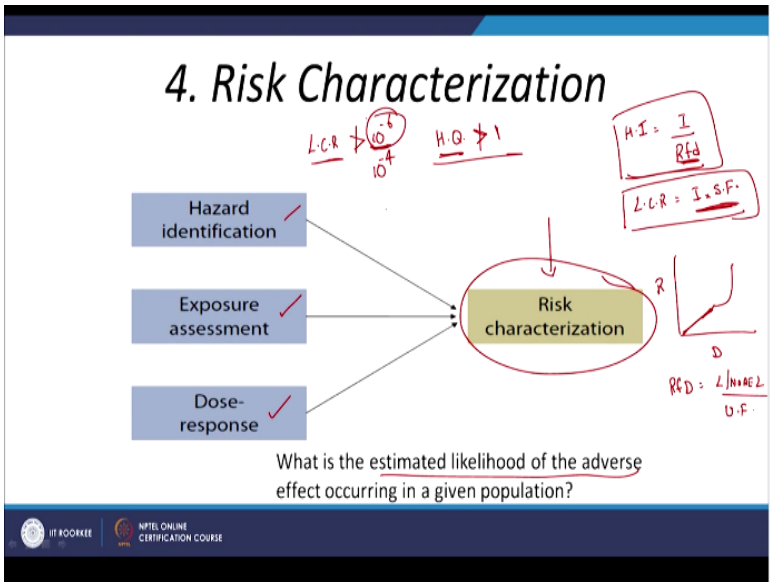

So let us move on and once I have these relevant aspects obviously as in hazard identification I am done with exposure assessment and then dose response, I can come up with risk characterization right. As in, what is the likelihood or probability of adverse effect you know or what is the likelihood or probability of this target population you know facing an adverse effect I guess right? So that is something we can get from the risk characterization right.

How do I do that again? Let us say for non-carcinogens, I can calculate the hazard index right. That is going to be equal to the intake that you calculate by the reference dose right and for carcinogens, I am going to calculate the lifetime cancer risk that is going to be equal to the intake*the slope factor right and slope factor again how do I get that as you see there from the dose response curves and such, I can get the slope factor, dose and response right.

And reference dose, how do I get the reference dose? As we talked about earlier, reference dose either LOAEL or NOAEL by the uncertainty factors right, so for non-carcinogens and for carcinogens right, intake*slope factor for lifetime cancer risk and intake/reference doses for hazard index. The summation of all the hazard index for different compounds and different pathways we look at it as hazard quotient.

So if it is >1 that means it is unacceptable risk. For lifetime cancer risk, the risk should not be >10 power -6, some cases people look at 10 power -4 but typically it is 10 power -6. Hazard

quotient should not be >1, sum of all the hazard indexes indices pardon me and lifetime cancer risks should not be >10 power -6 right.

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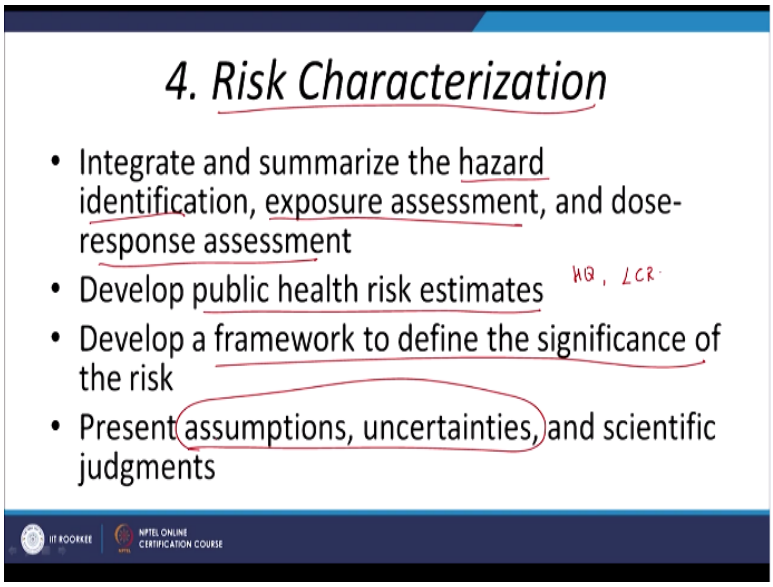

So again you know we are going to look at an example soon. So again what do we have here with respect to risk characterization, all the 3 aspects as in hazard identification, assessment and dose response, we bring all these aspects together right and then obviously we come up with risk estimates as we talked about, the hazard quotients and the lifetime cancer risks right and then we need to communicate the significance of the risk.

And more importantly though we need to give an idea about the assumptions and uncertainties here right. As in, just giving out numbers let us say right while it will serve some purpose, we always need to understand that there are considerable uncertainties you know that are at play here. So in that context, obviously when you are presenting the results, you need to present you know what are some of the gray areas out there right. So that is something that we need to look at with respect to risk characterization right.

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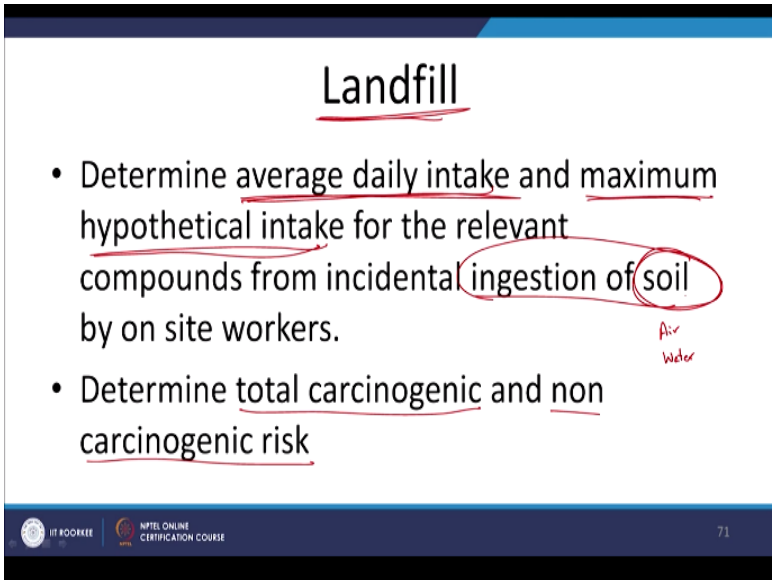

So I believe, we are now going to look at a particular example from a particular landfill site. So here we are going to present some data let us say as in a few people working at the particular landfill let us say and we are going to look at the relevant data and we are trying to going to calculate these particular variables here. So before we go further, what are we going to calculate?

We are going to calculate the average daily intake right which is relevant to exposure assessment I guess right and we are going to calculate the maximum hypothetical intake right. So these are the 2 aspects that I am going to look at obviously and also we are going to then move on to calculate the total carcinogenic and non-carcinogenic risks right but the key aspect here is that it is from ingestion of soil.

So we are not looking at all the pathways as in from inhalation or from air or from ingestion of water or such. I am only looking at soil in this particular aspect right but obviously if I am trying to calculate the hazard quotient or such, obviously I need to calculate all the different pathways for all the different compounds but for this particular example we are looking at, we are obviously only looking at the soil pathway right.

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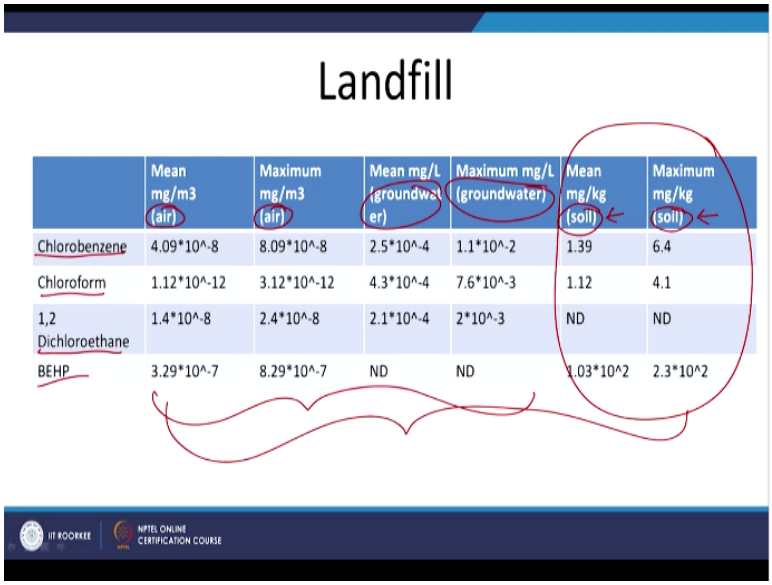

So here what are some of the data that I have? Here I have different pathways; air, ground water, soil right and I have the different compounds right. So let us say this is my hazard identification and what do we say now data collection. So let us say I have this particular set of data now. So obviously though I am not concerned with these sets of data, I am only concerned with these sets of data.

Because the question that we are looking at our example only wants to look at or understand the effects from soil ingestion I guess right. So moving on what do I need to do?

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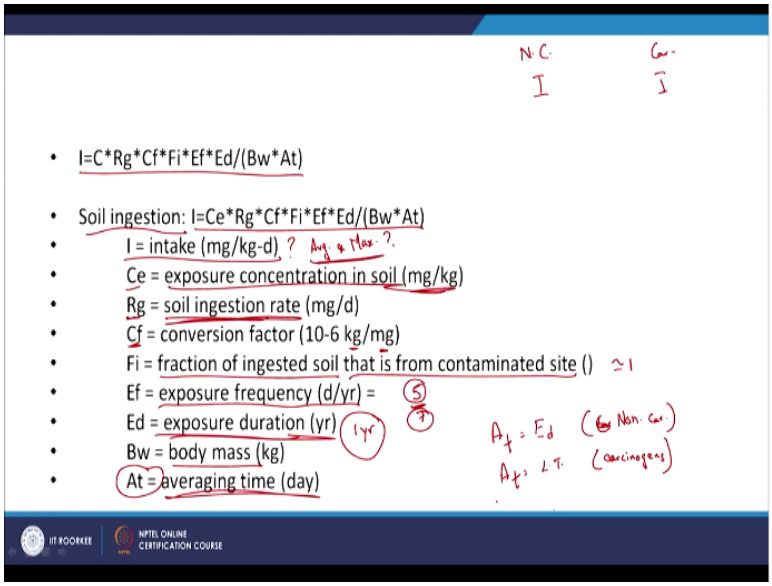

I need to come up with the relevant formula for soil ingestion. As I mentioned, it is slightly different from the generic formula right. So here we have different variables out here. Again, these are formulae that are going to be given out. So here we have intake which is what we

are going to calculate, both the average and maximum right. This is what we are trying to calculate.

So Ce is the exposure concentration in the soil milligram per kg which is what we already have out here right which is the mean and maximum we are going to consider both and then soil ingestion rate, rate at which soil is accidentally ingested, you know we are going to have some standard values here. Conversion factor from kgs to milligram 10 power -6 and fraction of ingested soil that is from contaminated site.

And here I guess we are to go with the conservative estimate and take it to be 1 and exposure frequency right. Again, it depends from case to case and here we are going to choose a particular value. We can either choose let us say people are working for what do we say 5 out of 7 and so on, 5 days out of 7 days and so on for exposure duration let us say, exposure frequency pardon me and so on.

Exposure duration, I believe we are going to look at it for over a one period one year. Body mass, we have what we say some standard values and averaging time as we mentioned averaging time is going to be equal to exposure duration for the carcinogenic pardon me for the non-carcinogenic risks and averaging time is going to be equal to life time for the carcinogens right.

So obviously what does this mean? If averaging time differs, that means we are going to have 2 different intakes, one for non-carcinogens and one for carcinogens right, so let us move on.

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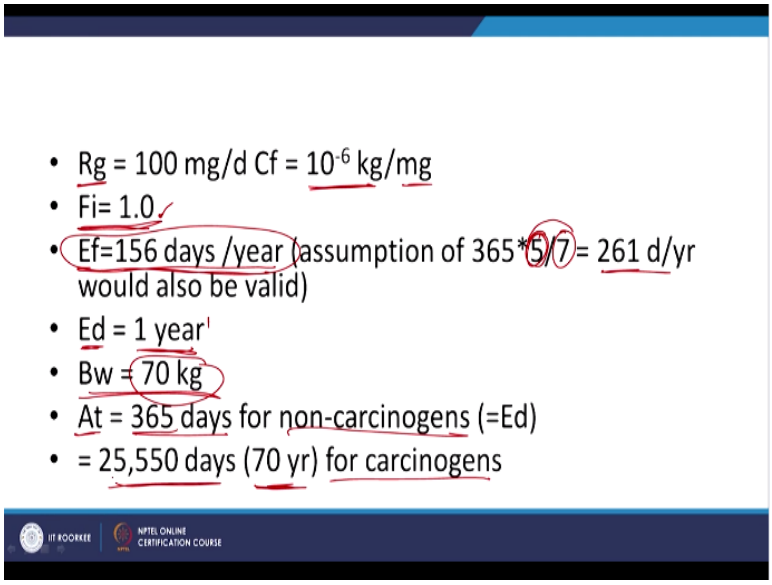

So here what are the different values? I think this is the rate of soil ingestion rate, it is 10 power -6 kg per mg. Fraction of soil that is from contaminant site, we are taking it to be 1. Based on the relevant site conditions looks like we are going with exposure frequency to be 156 days per year or as I mentioned earlier we can assume that you know out of the 7 days in a week people are going to work for 5 days and take it to be 261 days per year.

Obviously, this particular aspect would depend upon the relevant site and then we have exposure duration for that particular site we are looking at one year. Body weight from the standard table we are taking 70 kgs. So again this is where the uncertainties creep up into the relevant what we say analysis as in body weight. My body weight might be different or certainly is different from 70 kgs. Your body weight will be different and so on.

So you know how do I put a particular number on this? So in this case, obviously we are looking at the standard values and again these are the uncertainties that come into picture now. So average in time as I mentioned is equal to exposure duration for non-carcinogens right that is 365 days and for carcinogens it is equal to the lifetime which is 70 years right. So we have two different values.

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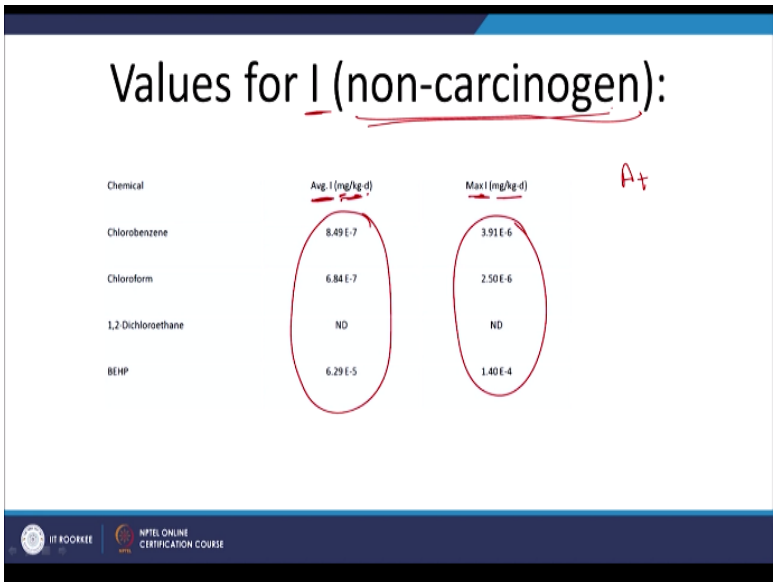

And I believe we calculate the relevant intakes right based on both the average intake or average concentration and maximum concentration. So here we have the average intakes and maximum intakes right and milligrams of compound per unit or per kg of body weight per day right. We have these 2 aspects and we calculate the intakes. Why do we say for non-carcinogen? Because the averaging time is different for non-carcinogens and carcinogens.

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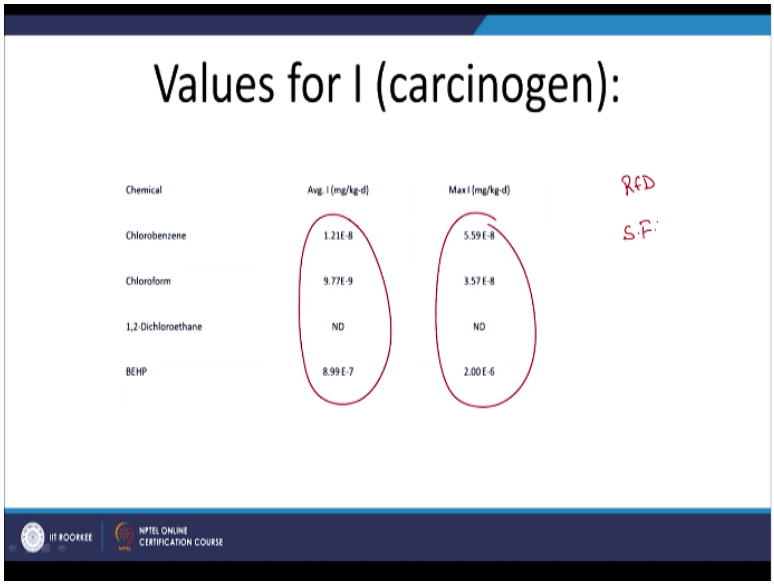

And again for carcinogens, we end up having different levels of intake for the different compounds right. So again you know just calculating the intakes obviously does not convey the relevant information to me right. So what do I need to look at or consider now? So obviously I need to consider the toxicity assessment and what is that about? That means I need to look at with the reference doses and the slope factors right.

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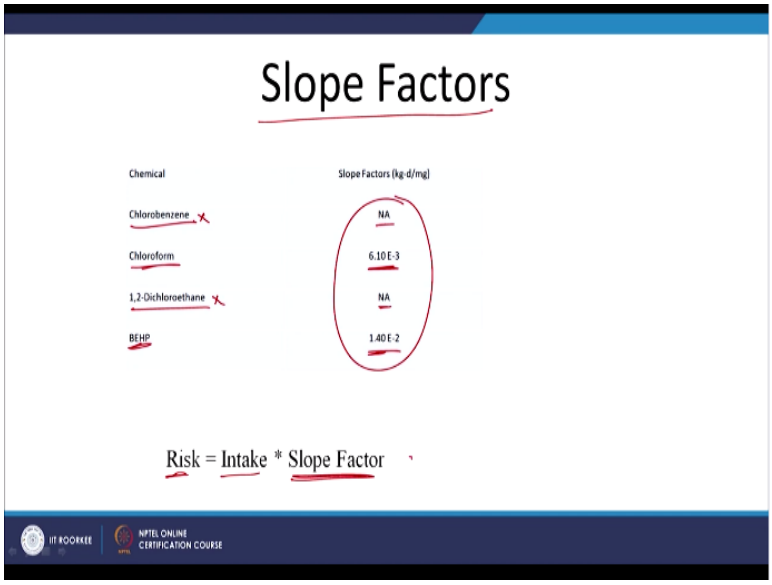

So let us look at what they are? So slope factors again from the standard data, so looks like you know these are the slope factors not available. So let us say in that case I am going to assume that chlorobenzene and 1, 2 DCA are not carcinogens right, only chloroform and BEHP are carcinogens because I have the slope factors only for those 2 particular compounds.

So then risk is=the intake*slope factor that is what we have and we are going to calculate the relevant risks.

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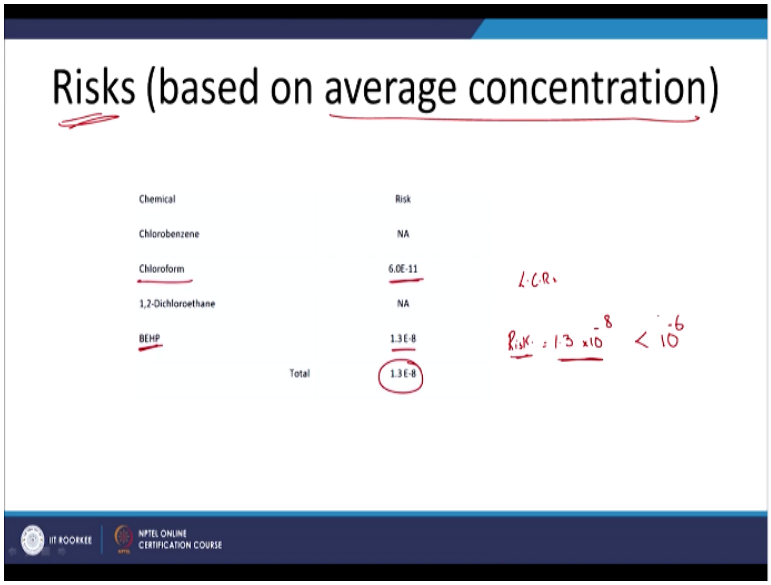

And let us see what we have here, so risks and based on the average concentration that we had in the earlier table. So for chloroform, this is the level of risk from soil ingestion and for BEHP, this is the level of risk from soil ingestion, accidental soil ingestion. So the value turns

out to be 1.3*10 power -8 right, the total risk anyway pose to the person working there for one year at a frequency of either 260 days per year or 150 days per year.

I think we took 150 days per year in this context, at the given rate of ingestion for a person of that 70 kg body weight, what we say the risk based on what is it now, the carcinogenic risk would be or the cancer risk would be what now 1.3*10 power -8 so this is <10 power -6 but obviously the conclusions need to be tempered by the fact that there are uncertainties involved.

And more importantly that we are only looking at one pathway which was soil ingestion, we have not looked at groundwater or air inhalation right. So again with respect to one pathway yes.

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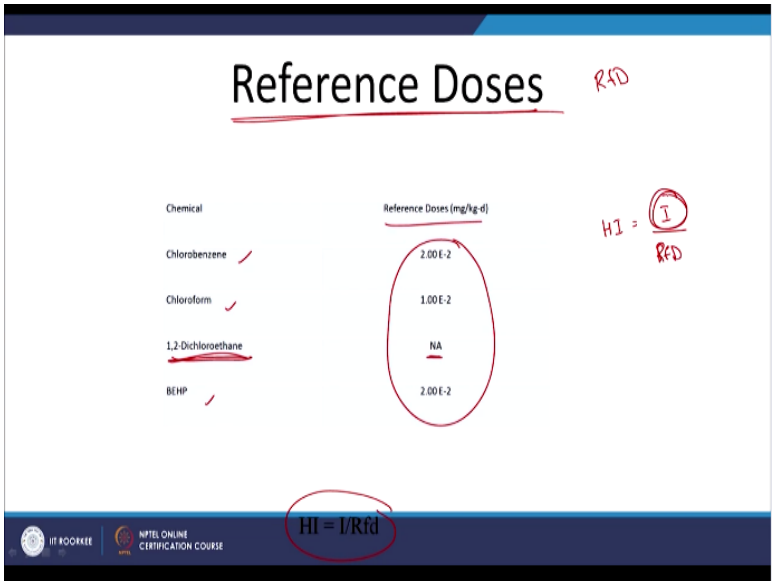

So same case we are going to move on to the non-carcinogens or the toxic compounds and these are the reference doses right, RFD as we mentioned earlier and again these are the standard values let us say and looks like 1, 2 DCA is not a toxic compound probably only what is this now carcinogen now looks like 1, 2 DCA is neither a carcinogen nor a toxic compound at least in this particular set of data that I have.

So I have the reference doses for different compounds right. Again, keep in mind that the particular compound can have both toxic effects or non-carcinogenic effects and also carcinogenic effects and that is why we have some such data here and then what do I do?

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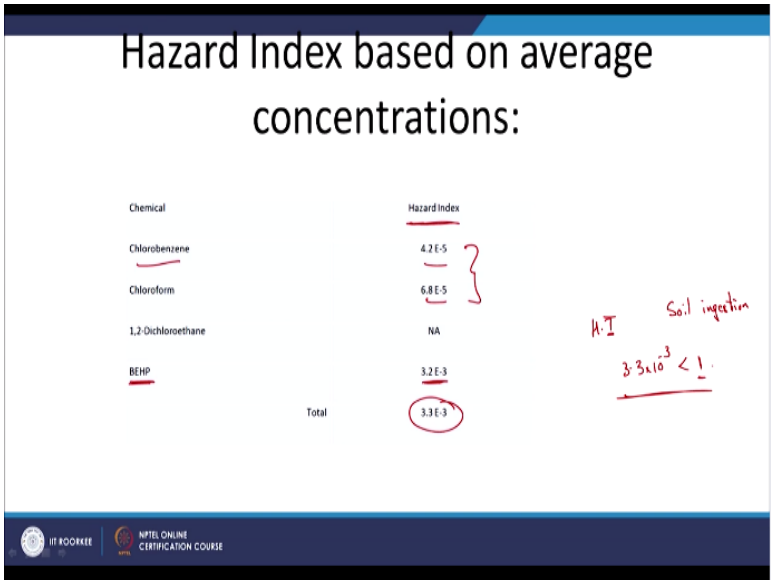

I end up calculating the hazard index now right. How do I calculate the hazard index? As I have it out here, hazard index is going to be=intake/reference dose and the intake again is for non-carcinogens right. So calculating that and how the hazard index for different particular compounds looks like there is a greater risk from BEHP compared to chlorobenzene and chloroform and the total hazard index let us say or hazard quotient let us say for soil ingestion.

Soil ingestion anyway is 3.3*10 power -3 and that is <1 right. Again, 1 is obviously the threshold we are looking at as in which is the acceptable value but obviously here we again similar to the lifetime cancer risk or cancer risks we need to consider the fact that we are only looking at the soil pathway right. So again this is how I guess this helps you understand the relevant aspects.

So maybe in the next what we say session we are going to look at the risk from air and groundwater. Those 2 pathways for both the carcinogens and non-carcinogens and then we can compare which pathway you know poses the greater risk to relevant population out there or the exposed population and then we can look at the relevant remedial measures I guess right and I guess with that I am done for today and thank you.