Electronic Waste Management – Issues and Challenges

Prof. Brajesh Kumar Dubey
Department of Civil Engineering
Indian Institute of Technology, Kharagpur

Lecture – 08
E-Waste Health Risk Assessment

So, let us get started with the third module of the second week. So, in this week if you remember the first 2 modules, the first 2 videos we try to look at different contaminants, where they are used in electronics, why they are used in electronics what are the health impact, environmental impact then we also looked at some of the case studies one from china, and then I showed you the case will like a very quickly we showed that we china one we went into detail in terms of the data. And then we looked at quickly some of the from a photographic toward you can you may want to call it in terms of the impact in khana. As well as in Nigeria. So, which was (Refer Time: 00:59) also. So, just to put, things in perspective, then now we will start talking about more in say we looked at these are bad. We what we are calling these are bad, these are harmful, but how we calculate that, how we calculate that at what concentration they are really bad.

So, that is called a concept of health risk assessment we talked about the exposure scenario as well, in the previous one of the previous video in this week. So, in terms of exposure scenario and the concentration level, how we do this risk assessment calculation. So, how that math is done will try to try to answer it over here. So, we will go over some of the basics of the methodology and then we will try to have some examples.

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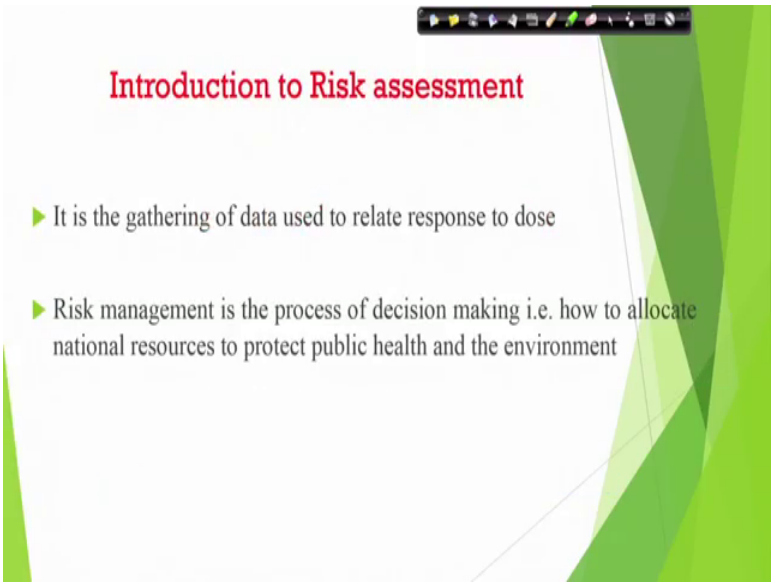

So, in terms of the risk assessment what is we basically is trying to get generate data so that we can relate response to dose. As a dose response curve which you may have seen it is very similar to even if when you take the medicine, isn't it? You take a certain dose, and based on the dose there is a response, the body gets the response the body gives in terms of something getting better, and if you do not get better response what doctor will do he will change the medicine and give you some other medicine. So, any dose you give there is a response.

So, that is so, based on similarly for the contaminants to for the toxicants as well, that if there is a certain dose of course, the body will have certain response. So, the risk management or the risk assessment and the risk management in the process of the decision making. So, we have to say everything has a risk. While crossing a road has a risk as well. While driving from point a to point b has a risk. Even swimming in a swimming pool is a risk, going to the beach has a risk. So, everything has certain amount of risk what we need to do is to manage that risk, we have to we have to make the decision in a certain way so that we can allocate our natural resource allocate the natural resource so that we can minimize this risk. Some of some material may have a based on their toxicity level other stuff has higher risk versus the other. So, we can so, risk management or first for the once the risk assessment is done then the risk has to be managed, where we have the we make certain decision to how to allocate natural resource to protect the public health, and the environment associated with that.

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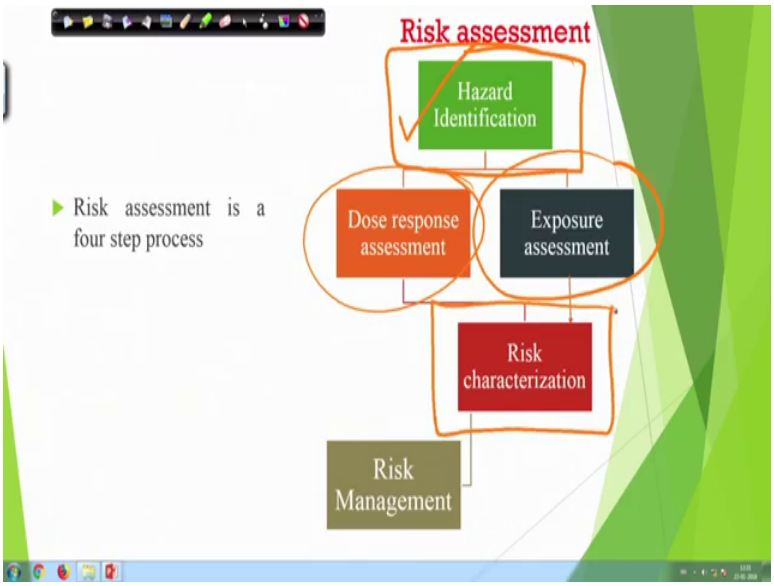

So, it is a it is a 4-step process, it is; let me try to move this on that side I can if I make sorry, yeah.

So, we can see risk assessment is a 4-step process, the first thing is we need to find out the hazard identification. So, as you can see the top tip top box over there that is the hazard identification. Now what does that mean? We have already done that. So, from the last few videos, that what we have been trying to do? We have been trying to identify that different chemicals which are present in the electronic waste stream. So, those are the hazard and I am talking now I am kind of discussing a just from the from e waste perspective there are that hazard identification in a transportation sector will be defined in a slightly different way. But for the electronic waste those contaminants, those elements, that we already talked about in the last couple of videos, those are our hazard. Then we have to we and then we need to know their concentration. So, that is the identification of the hazard. So, that part we kind of already looked at. Now the next step will be like a at what level it is becoming to becoming available to the public, or what level we are getting exposed to. That you saw in that if you remember from the china case study which we talked about the different levels of presence in ppm 2.5 different element.

So, those are, we had a base when a other city which was very far from the US processing facility and then we have a small we had 2, 2 sample set one was near the US

processing fellows facility one was away from US processing facility and we compared the numbers. So, that is and the exposure we already talked about inhalation dermal, and injection. So, that is your exposure, that is how it will get exposed. Then we need to do that dose response assessment. How that particular dose is what kind of effect we get for that we do AC 50, LC 50, IC 50 those kind of calculation, and based on hazard then exposure, as well as the dose based on these 3 aspect we do risk characterization. We try to identify what is a real risk? Yes, the risk is there. Something may be very toxic, but if it is not we are not exposed to it. Say if you have a cyanide in a small bottle kept somewhere in a lab in a corner, I am not going to touch that.

So, I am not going to expose to it. So, unless I get exposed then only there is a risk if I am not exposed to it there is no risk that is a hazardous material, that is a hazardous chemical just sitting there, but I am not I am not touching it. I am (Refer Time: 06:10) for some it is not getting into my body either through my nose or through my food or through my skin. So, if it is not it is there is our risk is not there. So, that is what we need to look at the exposure assessment, and then exposure based on that exposure what is the dose response and then based on that we can characterize what is the risk. And once we know the risk we have to manage that risk. Even when you talked about those of you pretty sure that all of you must have taken a watercourse.

So, when we talk about this water quality standard, when you have 10 microgram per liter of arsenic as a drinking water standard; that also, has a one in a one in a million-cancer risk. So, out of a million people we are taking that out of a million people, let us take that one person will it still have cancer at the level of 10 micrograms per liter. Ideally, we should try to bring it as close to 0 as possible. But as you go to closer to 0, you need more advanced technologies, more; that means, more cost. So, at some point of time you need to manage that risk. Because you may have to see how to better use your resources the economic resources that the country has. So, it is a 4-step process again, risk assessment hazard identification exposure scenario dose response risk characterization and finally, we have to manage that risk.

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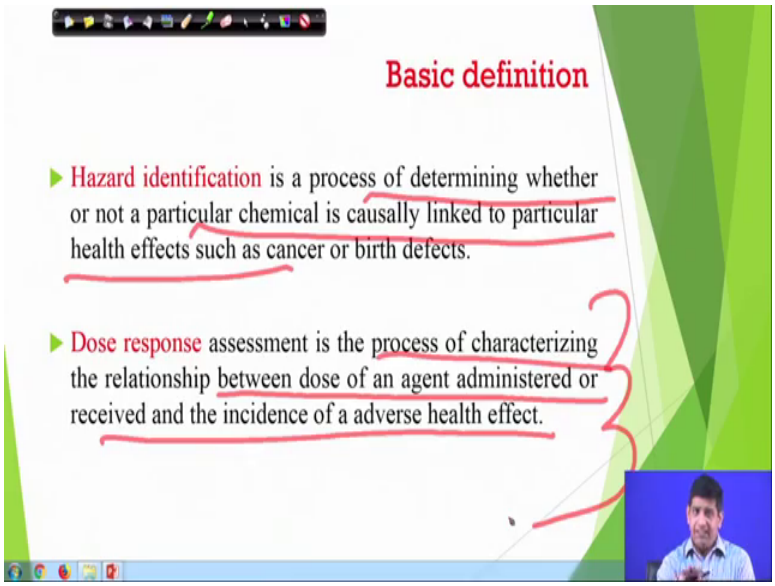

So, that is a so, that is the risk management part. So, in terms look at some of this basic definition, hazard identification is where you have to process of determining whether or not a particular chemical is so, you are looking at whether or not a particular chemicalize is casually linked to particular health effects we already talked about that. Those responses you try to characterize the relationship between dose of an agent, and received the incidence of adverse health effect that you do mostly in the lab setting, in the lab setting you feed these chemicals to guinea pig mice and other species, and you start seeing that impact. And that is how you get dose data and in terms of the dose response curves.

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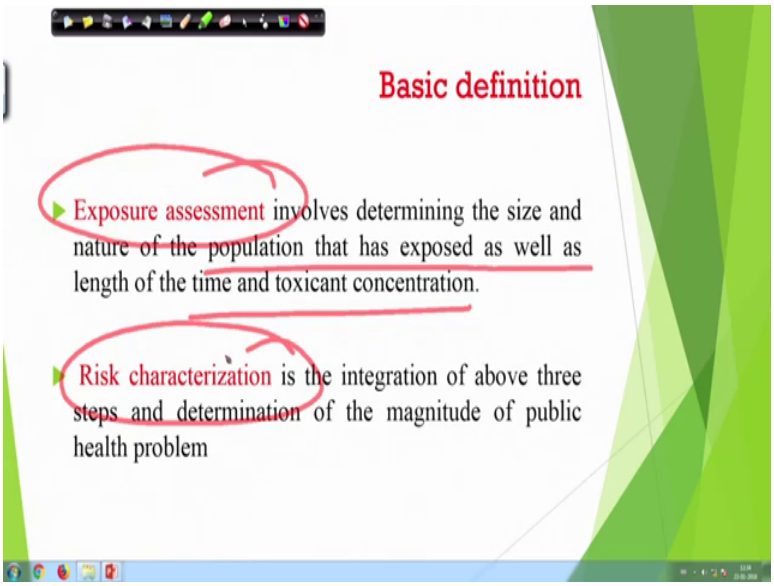

So, that is done for that aspect. Then you do the exposure assessment, which involves reminding the size and nature of population which is exposed, you look at the size of the population you look at the nature of the population. And then you try to find out as well as the length of time and the toxic and consideration. So, that is basically what kind of exposure they are getting and based on these integration of above 3 steps we try to find out what is the actual risk what is the risk, and then we try to characterize that risk from there.

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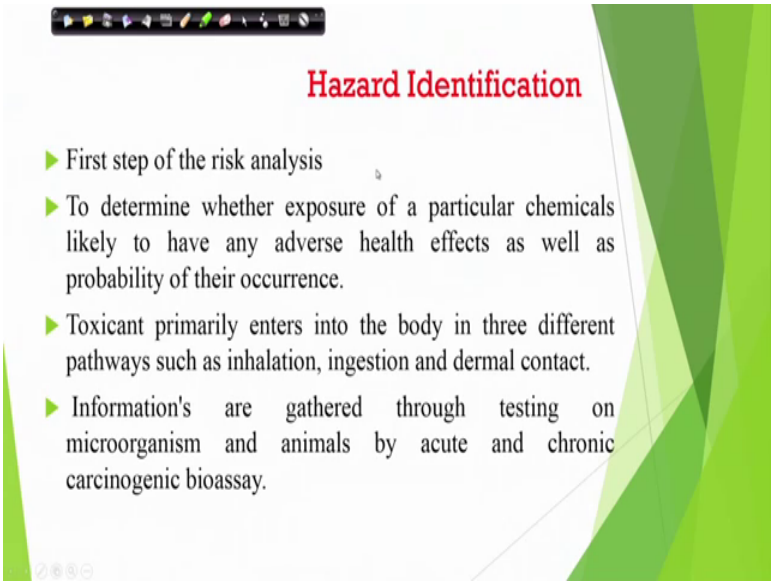

So, hazard identification is the first step. Whether the exposure of a particular chemical likely to have an adverse effect or not, and toxicant will primarily enter the body in 3 different pathway inhalation ingestion and dermal contact. Then you did generate the information through testing on microorganisms, and animals by acute or chronic carcinogenic a chronic bioassays. So, that is what you try to find out there hazard.

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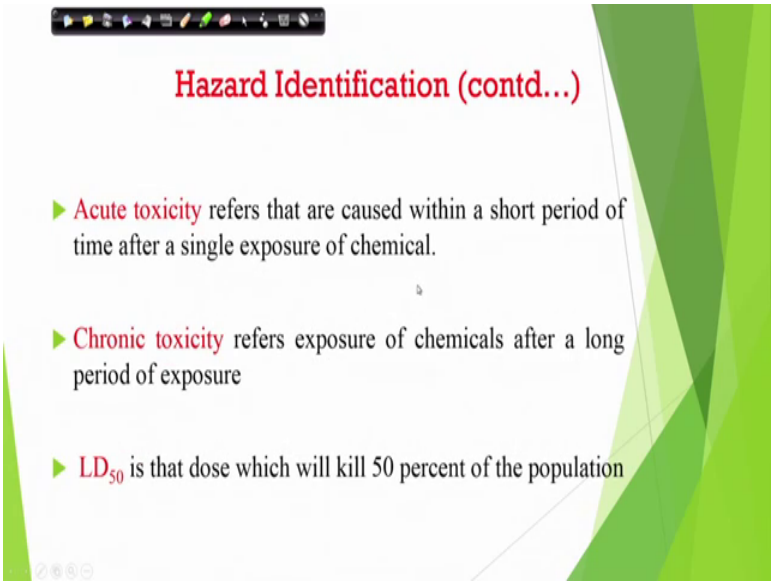

Then acute toxicity chronic toxicity you can go for LD 50 acute toxicity is a short period chronic is a long period. So, that is the photochemical. So, a long-term exposure LD 50 which kills 50 percent of the population. So, is the dose at which kill 50 percent of the population. So, we need all those we have to do all those kind of calculation based on the data that we collect using those toxicity testing on different animal's different species.

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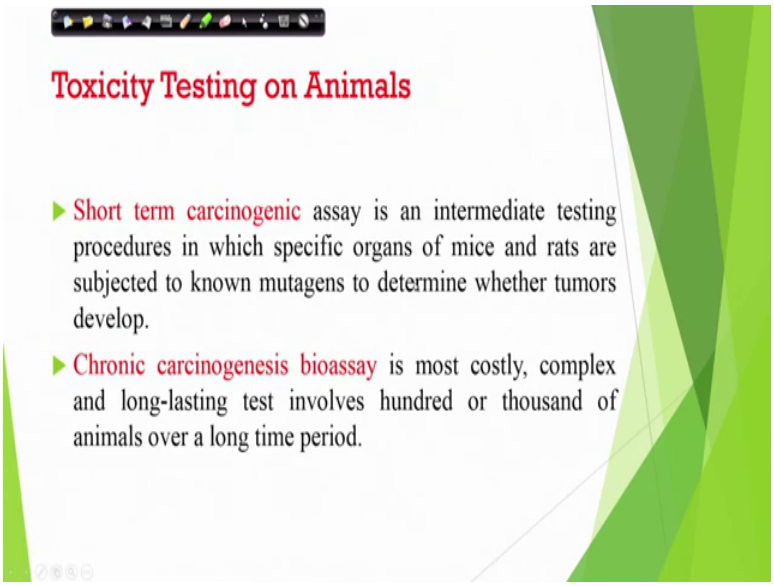

So, for short term carcinogens assay is we try to look at this specific procedures specific organ.

So, mice or rats are subjected to non-mutagens to find out whether the tumor will develop, chronic is mostly costly complex long lasting test, involves 100s and 1000's of animals over a long period of time.

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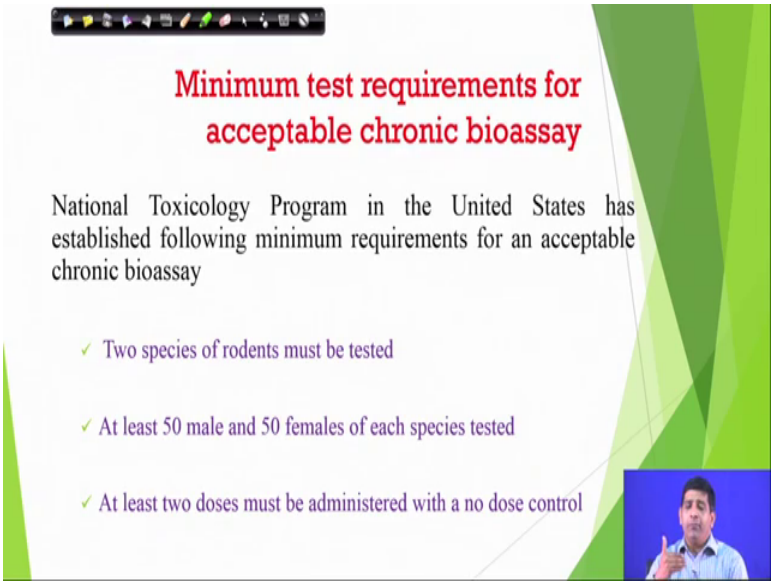

So, both things are looked at. Minimum test requirement they say even when you do these kind of testing, when you try to come up with these dose response. You have to do

is testicle relevant datasets. So, there is a national toxicology program in the US has established the following minimum requirement for a acceptable chronic bioassay. So, you need to test at least 2 species of rodents. At least 50 male and 50 female species has to be tested. At least 2 doses might be administered with no dose control. So, that is you have these are the bare minimum that you need to do.

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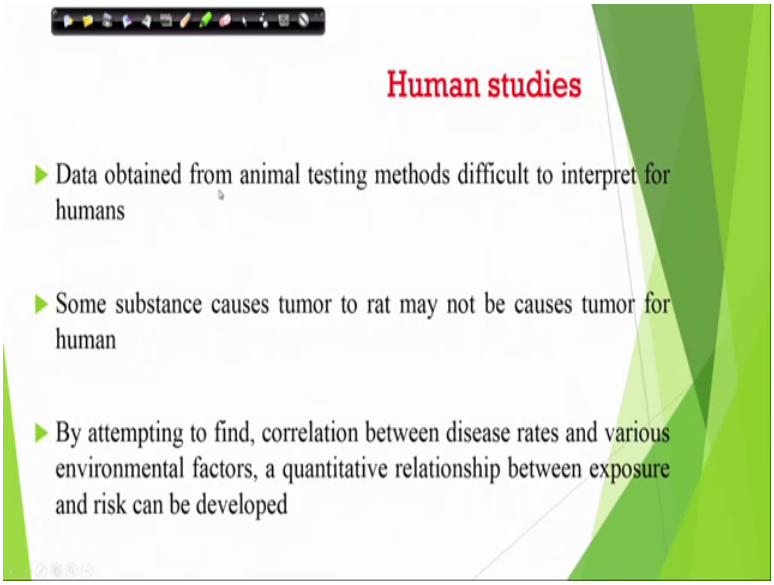

Then on human studies data obtained from animal studies of method is difficult to interpret for humans. So, some substance may cause 10 cause tumor to rat, but may not cause occur to human.

So, what by attempting to find correlation between disease rate environmental factors, a quantitative relationship between exposure and risk can be developed. So, we try to do some like a model some extrapolation is testical model to come up with that.

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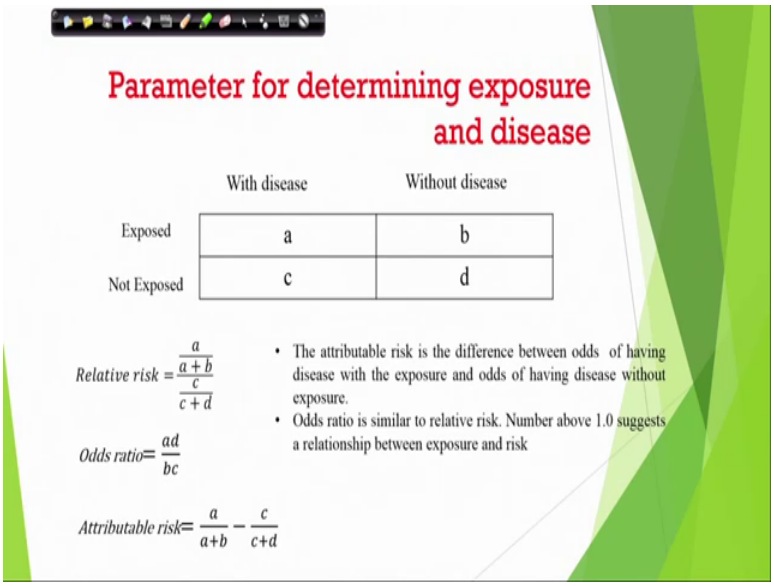

So, in terms of; if you look at the parameter for determining exposure and disease, if you have exposed you had exposed and not exposed, with disease and without disease. So, exposed and with diseases a, without diseases b and that not exposed c and d. So, the attributable risk is the difference between the odds of having disease with the exposure and odds of having disease without exposure. So, it is exposed and having the diseases a not exposed, but it still have the disease of b so that if you look at relative risk. So, relative risk becomes what, a divided by a plus b is what? With disease you are exposed. So, that is your where the you are supposed to get it.

So, it that is the attributable difference and then c is when that c numbers were not exposed, but it still got the disease. So, it is a c divided by c plus d. So, that is the fraction kind of gives you the value where you can get the disease even without being exposed to that. So, we look at that is a relative risk we define that as a relative risk, then odds ratio is where you have a times d divided by b times c; where you have there with disease you have exposed with disease not exposed without disease. So, that is kind of that is the that is what you expect, isn't it? You are exposed it should get the disease not exposed should not get the disease. So, that is again it is similar to relative risk. So, number 1, if it is number is above one it will suggest that there is a relationship between exposure and risk. And b and c not exposed and getting the disease exposed not getting exposed, but with does not get disease.

So, that is the b and c. So, that is a b a multiplied by d divided by b multiplied by c. So, that gives us the odds ratio. And if it is greater than 1 it gives some indication that yes exposure and risk is related. Attributable risk is where you have a divided by a plus b, where you get the disease because again a with exposure. And minus c divided by c plus d, because you are getting their disease even without being exposed. So, that is so, that is your attributable risk attributable risk is the difference between the odds of having the disease with the exposure and odds of having disease without exposure. So, these are the different terminology used in terms of the risk assessment when we are trying to point that exposure and disease different parameters that we use.

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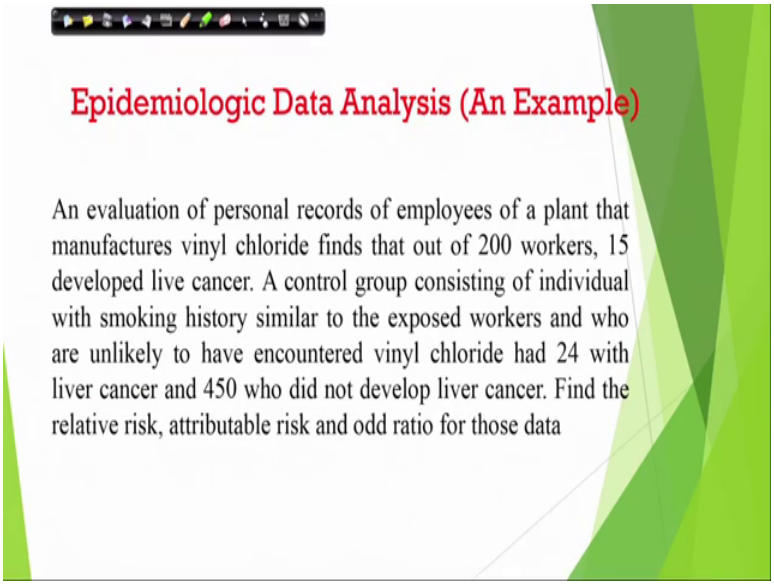

And then there is a epidemiological data analysis. Epidemiological data is when you have a population which got exposed to certain contaminants and then got sick. So, the data collected from them it is not the rat and mice data it is just it is a data all the humans. So, epidemiology is the huge area there are if there are certain departments of epidemiology as a part of college of public health.

So, you there is a huge area of research and teaching and all that, and we do not see that much popular in Indian context. In fact, the whole public health education unfortunately after our independence this public health became part of they went to the like to the doctors and it is not really a doctor it is kind of between the doctors and the engineers. So, that is a environmental engineer and the medical doctors, something in between will

be the public health professional environmental health professional. And it is environmental health is one aspect of public health. Public health is a huge program. And it is a it needs to be developed, it is a actually it is a good bridging point between engineers and medical medicine field; where we start looking at the interaction. Because ultimately what is the goal. Goal is to better human our humans should be more protective, there should be less sickness, people should be we should we need to be proactive in terms of the preventing the health. Because that is a proactiveness of health prevention is what is public health, but unfortunately since.

Now, it is reactive our health sector is becoming reactive rather than proactive. And that proactive comes in the public health aspect. So, let us look at this epidemiological data study. So, in evaluation of this personal records of an employees of a plant. It the plant is manufacturing vinyl chloride 200 workers 50 developed cancer. So, and liver cancer. I am sorry the r is missing here. A control group consisting of individual with a smoking history similar to the exposed workers and who are unlikely to encountered vinyl chloride had 24 with live cancer, and 450 who did not develop liver cancer. So, find out the relative risk attributable risk and odd ratio for those data. So, again what we tell the previous slide we can set up the table here as well.

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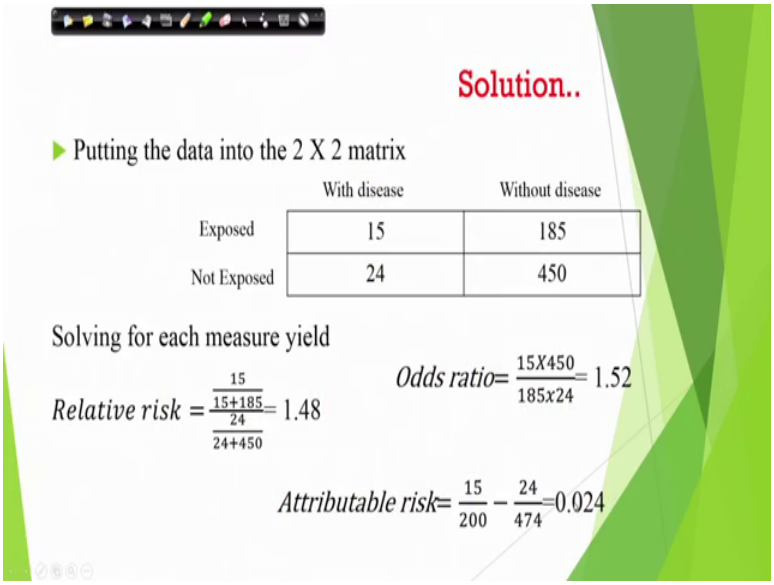

So, let us look at the 2 by 2 matrix. So, we have a with disease without disease. So, some people got exposed to vinyl chloride and we had 15 people to which you got liver cancer.

And then other we had 24 people, which were here we had not never encountered a vinyl chloride, but had 24 with liver cancer. And 450 who did not develop liver cancer. So, we had 450 who were not exposed in develop liver cancer. Here they are exposed, but 15 people got liver cancer and 185 did not get liver cancer.

So, again remembering the previous slide the 2 slides before relative risk, we can divide this is now a yeah what we have a b c and d. So, we had what a divided by. So, this plus this. So, that is the relative risk in terms of exposed, and disease divided by not exposed to getting disease. So, it is more than one odds ratio is 1.52 which is 15 times 450 this times this. Attributable risk is 15 times 200 if minus 24. So, you get 0.024. So, that is the risk in terms of where if you are exposed you will get more chances of getting it. So, that is a multiplied by 2.4 percent or something like that. So, that is kind of how we do some of these data analysis in terms of what whether the risk is there or risk is not there. So, this this kind of calculations are useful in terms of finding out like a human health risk assessment part of that.

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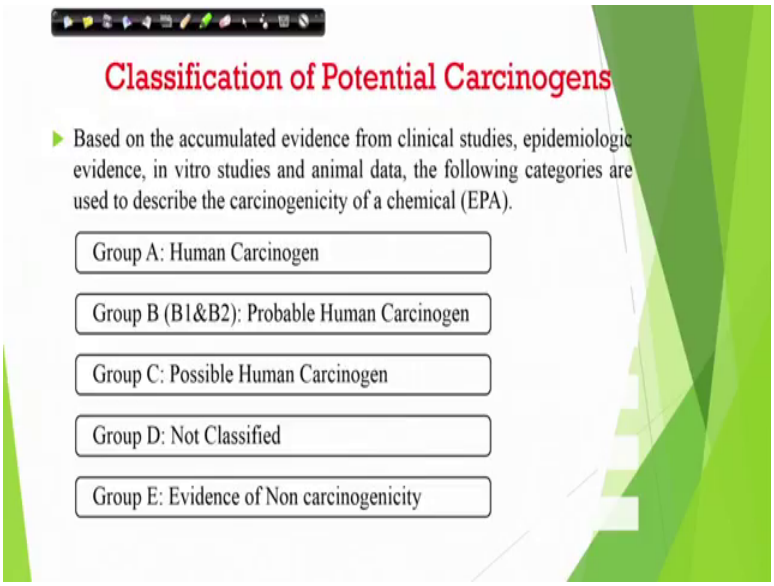

Then in terms of the carcinogens when we look at there are evidence from clinical study epidemiologic evidence there are different groups group a group b c and d, and e is a human carcinogen B1 and B2 probable human carcinogen. C is possible human carcinogen based on it is we do not know for sure. B is a is sure shot that it is a human carcinogen there has been enough data to prove that b lab data is kind of shows that yes

it could be human carcinogen, but we are and there are some anecdotal evidence from epidemiological data as well.

But we are not really 100 percent sure as such. Possible human carcinogen is based on the rat and my study we see that yes there are some indication that yes it could be human carcinogen, but we haven't reached the surety stage, yet we need we do not have a epidemiological and other data to support that. Group d is not classified group is evidence of non-carcinogenicity. So, it is not we do not it is not carcinogen.

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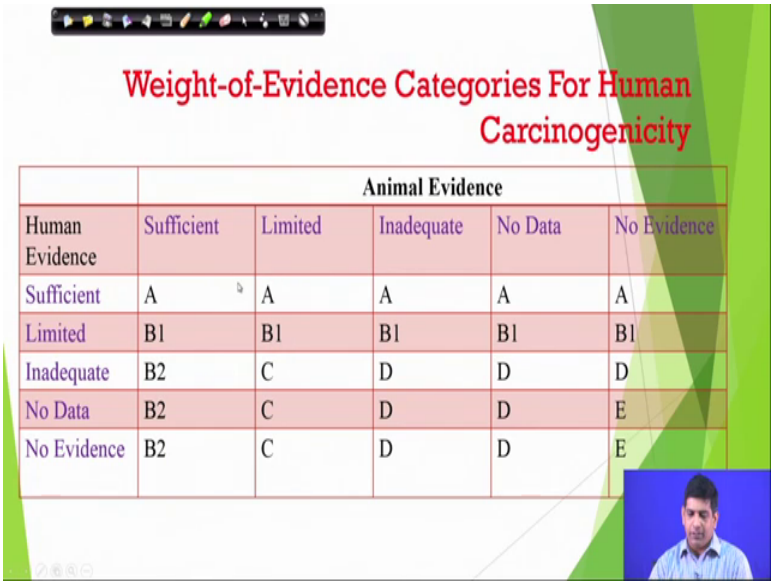

So, these are the classification that you see. And weight or evidence categories for human carcinogenicity. So, we look at human evidence if you have sufficient this a limited evidence is there inadequate. So, sufficient of course, limited evidence inadequate evidence no data or no evidence. So, based on that as you can see. So, animal evidence and human evidence. So, in if resource like A with where you have human evidence as well as the animal evidence this is limited.

So, if it is limited it is not sufficient it is kind of inadequate no data no evidence. So, based on weight of evidence category for human carcinogenicity based on the lab data, we can try to look at the human evidence and try to correlate which one kind of works out aware.

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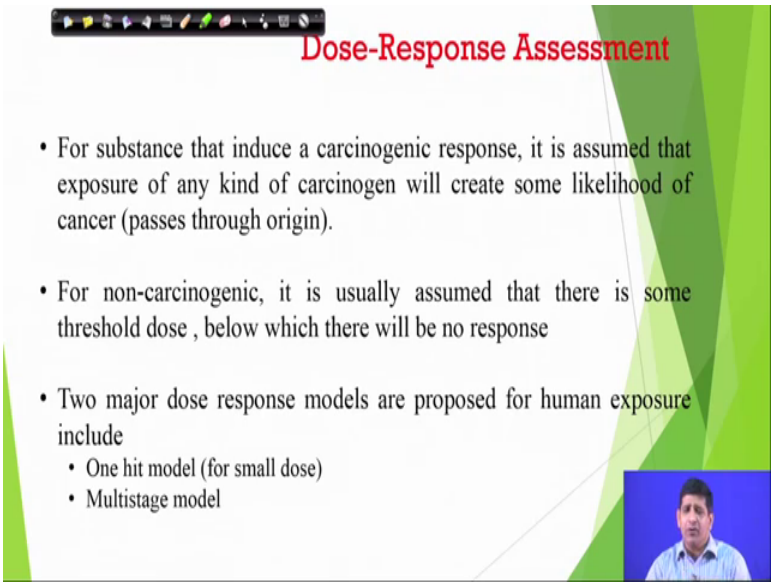

So, based on all these data we try to generate this dose response curve. The dose response curve is you do a certain exposure, and for exposure you look at what is the corresponding impact. So, for substance that induces a carcinogen response, assume that exposure of any kind will create some likelihood of cancer. For non-carcinogen, we assume that there is some threshold dose below which there will be no response. 2 major dose proposed like human hit model and multistage model.

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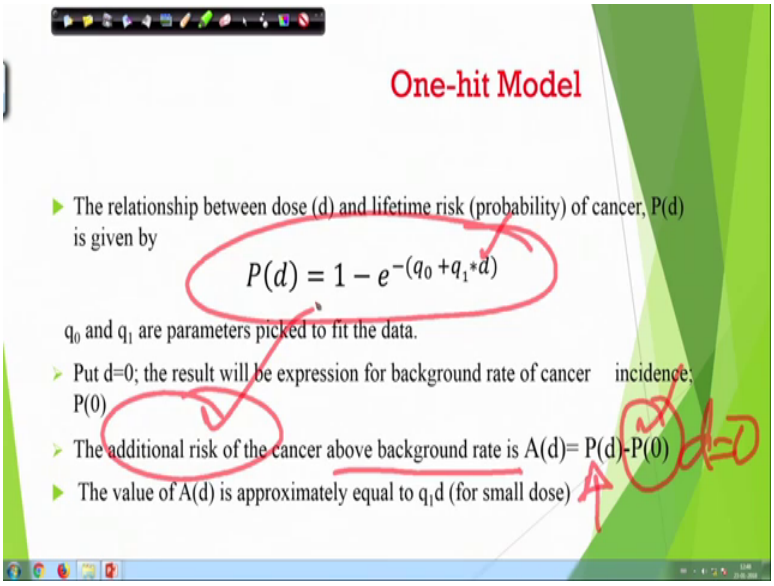

So, one hit model is the relationship between the dose and the lifetime risk probability we can put it in this kind of equation. Where you are like a dose and that and lifetime probability of cancer which is P d can be expressed as 1 minus e to the power of minus q 0 plus q 1 times t. Now what is this q 0? Q 0 and q 1 the parameters pick to fit the data. And so, when d is equal to 0, result will be expressed as the background rate of cancer.

So, there is no dose. So, there is no dose. So, this is the data that we will get that is the background. That is a background data. So now, as you increase d values as the dose goes up, you will see the your probability also will keep on changing. So, additional risk of cancer ever background which is A d is P d minus P 0. P d is the this value at for any particular just to explain that just to get you very clear. P d is with this is the dose. Q 1 and q 2 are the parameters picked to fit the data. So, they are basically constant. So now, as you increase the d value, you will see that changes in the probability. Now d is equal to 0; that means, no additional dose that is the background probability for having that particular cancer. Now as we increase the d the numbers will change. So, to find out the additional risk of the cancer above the background, background was when P 0 when d is was d is equal to 0. So, that is the that is we are calling it P 0. Now when d has a certain value we are calling it P d. So, P d minus P 0 is giving us what is the additional risk. So, value of A d is approximately equal to q and d for a small doses. At a small doses this is actually equal to probably to a closer to q and times d.

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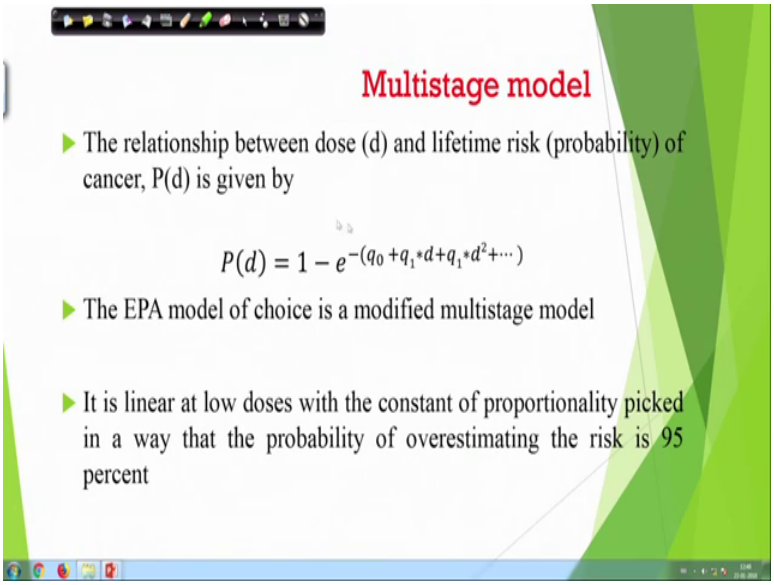

So, that is this so, that is we that is the one hit model. Now the multistage model is where you take the dose and the lifetime risk probability of cancer P d is divided by one e to the power of minus q 0 q 1 times d q and times d square, and then the list goes on. So, this is the e P a choices multistage model. They go for this multistage model in terms of within the probability it is the linear. At low doses, if you have a low dose it is linear with constant of proportionality picked in a way that the probability of overestimating the risk is 95 percent. So, you try to always try to over estimate rather than under estimate. So, here you can again d is equal to 0 will give you background, and then when as the d changes you will have that P d giving you the what is the probability of that cancer. So, that is this is called multistage model. Now we can calculate the potential factor for carcinogen.

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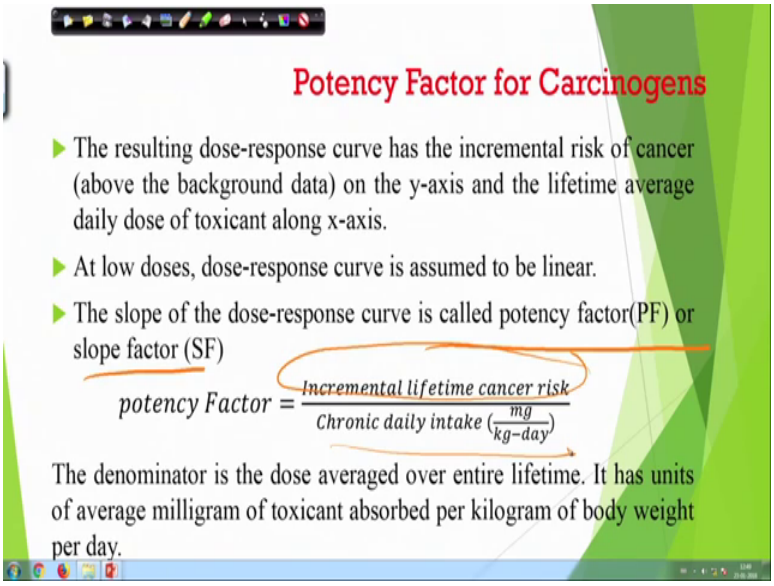

So, resulting those response curve as the incremental risk of cancer above the background data. On the y axis in the life time cancer toxicant along the x axis. At low doses the dose response curve is assumed to be linear, but the slope of the dose response curve is called the potential factor or the slope factor. That is some people call it a slow factor some people call potency factor. So, that is the slope of slope. So, this is potency factor is the incremental lifetime cancer risk divided by chronic daily intake. So, that is how we define a potential factor. Whatever is the life time cancer is divided by the daily intake, which is a milligram per kilogram per day and again. So, that is the denominator is the dose averaged over the entire lifetime. It has units of average milligram of toxicant

absorbed per kilogram of body weight per day. So, that is the denominator. And so, that is how we calculate potency factor or the slope factor.

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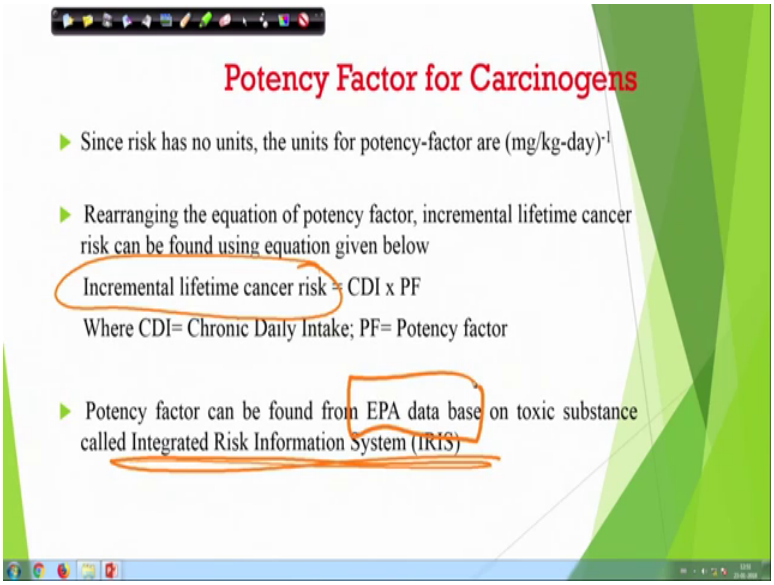

Now, since risk has no unit the units of potency factor as milligram per kilogram day as a inverse of that. Now the we can rearrange that equation and then we can find out the incremental lifetime cancer risk.

So, if you put that incremental if you from the previous one if you go back to the previous slide.

You can see potency factor is incremental lifetime cancer risk divided by the chronic daily intake. So, this way we can call it CDI this is the potential factor. So, if you want to find out this value over here, if you want to if you are interested in this thing over here, just a minute let me grab that. So, if you are interested in incremental lifetime cancer risk, which is being tried to do in the next slide, and let us get it of that over here. So, in terms of incremental lifetime cancer risk which you see over here. So, that is what it is CDI which was a chronic daily intake times the potency factor. So, we just rearrange that equation. Now potency factor is there, potency factor of the slow factor is available in the integrated risk is a risk information system. So, IRIS database the EPA database has that potency factor. So, we can get the data from there.

So now incremental lifetime cancer risk is CDI which is a chronic daily intake. So, we can find this particular value. Once we know the potency factor. So, we can calculate what is the incremental lifetime cancer risk. And as a set potency factor or the slop factor as we want to call it is available in the EPA database IRIS database for most of the most of the contaminants, most of the chemicals.

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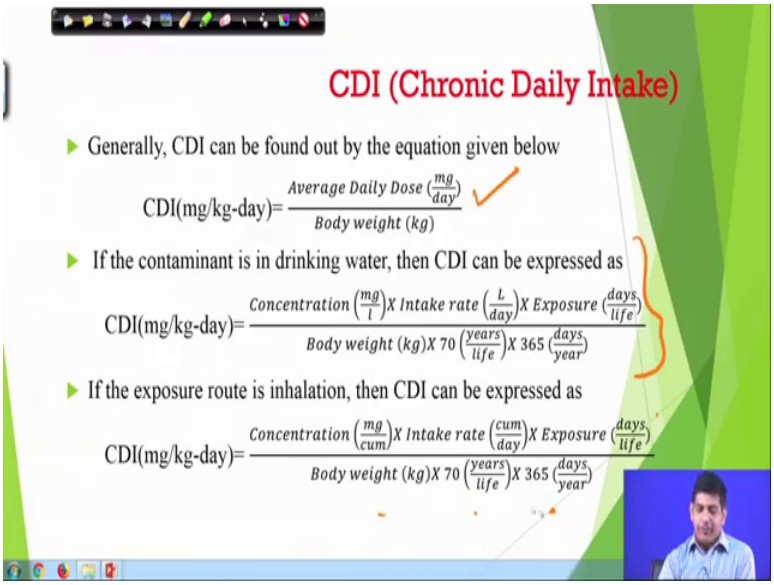

So, being said that if we can calculate generally CDI can be found out by the equation given below, we can calculate the CDI which is the what we call was that chronic daily intake. And what that would be? You can take the average daily dose. So, average daily dose how much milligram per day and then the body weight. So, that is kind of gives you milligram per kilogram per day. So, that is CDI values. Now if the contaminant is in drinking water, then CDI can be expressed like concentration in milligrams per liter times integrate how much liters per day.

So, if you say. So, you have a certain contaminant x milligram per liter. Typically, people consume 2 liters of water per day. So, intake rate is around 2 liters per day, exposure is how much days per life. Then you divided it by body weight in kg average body weight. 70 years say the typical life 365 days per year. So, that if the exposure route that is can be written up for the ingestion route. For the ingestion route this is how the equations can be written up. If it is inhalation route. Again, you have milligram per cubic meter. And how much cubic meter of air we take per day in our body exposure days per life body weight

years of life have how many days per year. So, express both the ingestion as well as the inhalation route. So, just to give you some examples there will be some math problem associated with that in your exam as well. So, make sure that you understand this concept you if you watch the video again and again if you need to, but make sure you understand if you do not have if you do not understand.

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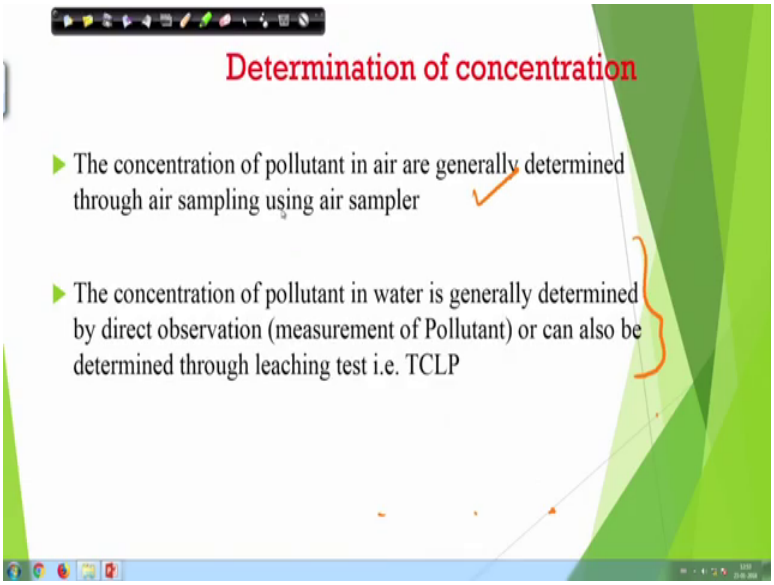

Let us know we will be happy to answer questions for you, but do let us if you are if you need help feel free to send us in question on the discussion forum will be more than happy.

So, and then so now, we need to know the concentration, isn't it? It says that what is the concentration in there. So, we to we need to know the concentration values. So, how we can how we get the concentration values? There are different ways of determining, generally determined through air sampling using air sampler, for the air for the water we can do direct observation we can measurement of pollutants, for a how much potentially it may leach out there are different t leaching test can be done TCLP is one test which is done which those of you have taken solid waste class before we have talked about there. In TCLP a few have taken solid waste class from anywhere, you may have talked about TCLP as well. So, it is one of the it is basically to find out how much contaminant will leach in AMSW landfill scenario. So, let us on that part, but if you are in the body we also have we can use the test of simulating gastric acid of the body. So, our gastric acid

of the body could be simulated in we can try to find out different things gets ingested into the body how much it will be absorbed. So, there are different ways of doing that. We will talk about it is I think, we can talk about this TCLP in in the next class because it is longer in the next video. It is a little bit of longer discussion on that. So, what we have done in this particular video? Again, as the in the issue area I am trying to say that what we are going to learn, and then towards the end I am trying to summarize what we have learnt. So, that you can relate to it and you that is how what was the learning objective and what was actually what we achieved. So, in this particular video the thing was we were more concerned looking at the risk assessment and risk management.

So, we I tried to give you some example first of all we looked at into what are the different components of risk assessment, hazard identification, those response exposure scenario, risk characterization, and then the risk management. Now in terms of the risk, we also looked at different carcinogen non-carcinogen, how we calculate these hazard. So, like a slow factor, or potency factor, in chronic daily intake rate, how those numbers are got. So, we kind of talked about that. And as part of the process of those calculation now we are at this stage where we need to know the concentration of these chemicals getting say in this case getting from electronic waste into the soil. Or into the water. So, how we do that calculation? We will continue that in the next video. So, again look at the videos look at the reading material, any question put it on the discussion board. We will be more than happy to answer. And if I hope that you keep enjoying this course, we are this is we have finished now the third video of the second week, and with 4 will again I will see you in the next video.

Thank you.