STATE OF WEST VIRGINIA

DEPARTMENT OF ENVIRONMENTAL PROTECTION

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IN RE: HUMAN HEALTH CRITERIA WORKGROUP

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BEFORE: LAURA COOPER, Chair
SCOTT MANDIROLA
ROSS BRITTAIN
ANGIE ROSSER
LARRY HARRIS
KATHY EMERY
AUTUMN CROWE
JENNIE HENTHRON
REBECCA MCPHAIL

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Reporter: Bailey Kane

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P R O C E E D I N G S

MS. COOPER: Welcome to our December gathering of our Human Health Criteria Group. We've now gone through the Legislative Rule Making Review Committee, so that's exciting. We got one thing down, and our rule remains unchanged for the most part except for the addition of a little --- a few words to make it more clear how the workgroup is set up. So we're moving along.

And we have Ross here today who's going to do a presentation for us and I'll talk to you about it in a second and then he'll get started after that. But thank you, Ross, for working on that. That was a busy time.

Do we have anything that anybody wants to talk about before we dive into our presentations? Autumn, I did receive your email yesterday --- I think it was yesterday, additional questions for EPA and I sent those along to them and they said they would be working on a response. Okay.

So let's --- oh. And we have the court reporter from Sargent's here. Are you able to hear everything all right everything?

COURT REPORTER: Yep, I can hear everyone.

MS. COOPER: Oh, that's right. I need to start the recording again. Okay. So with that, I believe ---.

MS. MANDRIOLA: Question for you?

MS. COOPER: Go ahead, Scott.

MR. MANDRIOLA: The questions that was sent to EPA, did you share those with the rest of the group?

MS. COOPER: I didn't. I just sent them straight on to the EPA. I can forward it to everybody.

MR. MANDRIOLA: Yeah. Just curious. I just wanted to see what the questions were. Thank you.

MR. HARRIS: Is that a background or is that real?

MS. COOPER: It's my living room, but it is a background if that makes sense.

MR. HARRIS: So you're not in your living room?

MS. COOPER: I'm not in the living room. I'm in my same little corner, but I thought I'd

be more festive this morning.

MR. HARRIS: Okay. Just curious.

MS. COOPER: Thank you. Thanks for asking. All right.

So with that, I made a note to send that email and I will do so after this. We'll go ahead and get started. We'll share my screen. Do you guys hear music playing from my end? My son is in the shower and he has Alexa playing music to him like really loud, so like I have to like literally go in there and yell at him in order to make it stop because he can't hear me.

Do you all see the first slide rather than the presenter view?

MR. BRITTAIN: We see the presenter view.

MS. COOPER: Okay.

MR. BRITTAIN: Yes, we see both.

MS. COOPER: There we go.

MR. BRITTAIN: That's it.

MS. COOPER: All right. Let me hop up and close the door. Maybe that will help with the extra music. All right.

Water Quality Standards Human Health Criteria

Workgroup. Welcome to this December meeting. This is our July, August, September, October, November, December --- sixth meeting in this series. And we've come a long way. Let me get over to the agenda and we'll talk about what we're going to talk about today. Okay.

So today first we're going to quickly go talk about our next steps, where we've been, what we've done so far and what we --- how we plan to move forward with the group. And then as I mentioned, Ross Brittain is going to do a presentation for us on benzo(a)pyrene and the IRIS update, how that works and how it was --- how the change happened and what it means.

And then we're going to look at the remaining West Virginia criteria. We have a spreadsheet that we typically look at when we're looking at all the criteria where we kind of have it laid out and color coded. And I'm going to show that to you and talk about it as we move forward with the next steps in the workgroup goals. And then we're going to talk --- we're going to show and discuss the finalized workgroup goals, and then we'll plan for the next meeting.

So let's move onto the next slide, and we'll just talk about where we're going. So up to this point we've gone over in detail how EPA revised the 2015 recommended criteria. We've talked about their decision framework. We've gone through that, you know, quite extensively, looked at how they made decisions within that framework. We've gone over their equation in detail, how it's structured, you know, what the various factors in the equation do based on, you know, where they are. We've also looked at each part of the equation and how EPA decided to use each one, you know, whether they went with the mean or, you know, a specific age group and whatnot and how they did all of that.

And, of course, we also talked to EPA and we got to ask them every question that we can think of and we sent a few more like I mentioned a few minutes ago. And we gained a lot of clarity from them on a lot of the questions that we had about their approach. So --- and also, like I said earlier, we cleared that first legislative hurdle by getting through the Legislative Rulemaking Review Committee last week. That was last week. Right?

It's the just weeks are really ---.

MR. BRITTAIN: Yes.

MS. COOPER: And those 24 --- just as a reminder, those 24 recommended changes are exactly as EPA revised. I know we had some discussion in that meeting and I know some of you were there and some of you may have watched it, but you know, there were questions about the criteria and we were able to make it really clear that what we recommended was exactly precisely what EPA's 2015 criteria are for those 24 chemicals.

So now we're going to be looking onto the rest of West Virginia's criteria. As we, you know, stated in our workgroup goals from the beginning, not just the goals, but what we've put into 47 CSR 2 as to what the workgroup is established to do. We're going to look at our remaining criteria, the ones that are in West Virginia's rule. And what we're going to be doing is looking for newer toxicity data, additional bioaccumulation factor studies, any information that could better inform the relative source contribution.

And basically, we're starting that today with our presentation from Ross. He's going to talk to us today about benzo(a)pyrene and what --- the

update that happened, the 2017 I believe update to IRIS that has revised benzo(a)pyrene. So do we have any discussion on this before we move on because really the next thing we have is getting into Ross's presentation?

MS. ROSSER: This is Angie. Just to register our interest and request that the workgroup also consider the EP recommended updates that are not currently part of the West Virginia Water Quality Standards.

MS. COOPER: Right. That is duly noted, your request to do that, but what we established in a rule and is, of course, not completely establish yet because the rule hasn't, you know, completely been revised. It's still in the process, but as we --- our intent in that revision was that the state will look at the remaining West Virginia criteria, so that's what we are focused on at this point. So that's what we're going to look at. All right.

Is there any more comments, questions or thoughts? If not, we'll go ahead and move on to the next slide which is getting into Ross's presentation. All right.

Thank you. And you can take it away, Ross. Again, thank you so much for doing this for us and looking forward to it.

MR. BRITTAIN: Thank you, Laura. And you're welcome. I'm happy to help out. So I'll give, you know, kind of word of warning and apologies ahead of time. This is --- I'm going to be glancing over some fairly heavy-duty biochemistry and some statistical analysis as well, so I don't know what kind of background each of you have in those fields, but I'm trying to tailor my presentation to the layman. So --- and if you have any questions as we go along, by all means interject and I'll be happy to get into further details as you need them, as best as I can anyway.

Next slide if you would, please, Laura. So our good friend benzo(a)pyrene. It is in quick review, a five-ring polycyclic or sometimes called polynucleic aromatic hydrocarbon, collectively called the PAHs. It's a result from incomplete combustion of organic matter, typically at 300 to 600 degrees Celsius, which in English terms is about 570 degrees to about 1100 degrees Fahrenheit. It is --- the chemical formula is C20H12. You see the fire benzo

--- benzine rings over to the diagram off to the upper right there. So a benzine ring is six carbons in a ring formation. Most of them --- of the carbons are actually being shared. Uh-oh. What happened? There we go. Thank you.

So most of the carbons are actually being shared, but notice there's 12 places where the carbons are not shared with another benzine ring, and that's where the hydrogens are attached. The 12 hydrogens are attached to those locations. It is a group A known human carcinogen and it has been for quite some time. PAHs were recognized as causing chimney sweeps carcinoma in young boys who were crawling down chimneys in London and developing scrotal cancer as early as the 18th century. So --- and it's important to remember that PAHs naturally occur in mixtures. When you get that incomplete combustion in benzo(a)pyrene, it's just one of many PAHs that are created in there. You can isolate benzo(a)pyrene in the lab, but in nature you will not find benzo(a)pyrene by itself. It will be in combination with other PAHs.

There are over 40 known PAHs, but 16 of them are considered the core group that they just come

together. When you get benzo(a)pyrene, you're going to get these other 16 in varying concentrations, which is what makes risk assessment for PAHs complex and difficult. Next slide, please.

So getting into toxicology, the way we do this is we calculate a benchmark dose for noncancer hazards as we refer to them. Noncancer hazards and noncancer risk, we calculate a reference dose. And it starts with the benchmark dose that will determine the point of departure is how it's usually done these days. So if you look at the little graph off to the lower left there, what we have is the circles represent data points where they had a dose of a known compound. This is not benzo(a)pyrene by the way. This is just a demonstration, an example. So you had dose of known concentration in milligrams per kilogram in five different levels for that particular compound. And the response --- notice, that's a percent, meaning the percent of individuals that showed a particular response. And you will do this kind of a graph for every single type of response that you may get, whatever it is you're looking at. Usually there's several dozen that you're looking at in any particular study. All right.

And is typical with these types of responses, you see this kind of S shaped curve. That means it's some sort of a logistic response. And so we have multiple models --- logistic based models that we use to try to fit the data in there. And that's the way it's done now. And historically, if you would look at the POD area circled in yellow, we have what's called the NOAEL. That used to be the way that we would establish our point of departure was the NOAEL, which simply stands for the no observed adverse effect level. It's the lowest concentration in the study where there was no observed impact to it, so no toxicity responses at all. And then you can compare that one to the LOAEL which is the lowest adverse effect level. That means the lowest concentration study where an actual effect was noticed. The problem is is that you notice - for example, the NOAEL was at about 10 mg/kg and LOAEL was 18 mg/kg. The issue really stems from the fact that the real threshold where an impact occurs is somewhere between them. You don't know because that would require having doses in your study at every single level in between 10 and 18, and these studies are extremely complex, time-consuming and you just can't do that. You can't

afford to do that.

So you may also notice that LD50 that's over there as well at the inflection point where it changes from the J shape to the L shape in the curve. The LD50 represents the lethal dose 50. That's literally meaning that 50 percent of population died at that particular dose, so you don't want to use that as a point of departure unless, you know, you're Joseph Stalin or something like that.

So from a statistical standpoint, what we knew is that the real threshold we're interested in was somewhere between NOAEL and LOAEL, but we had no good way previously to do that, so people started fitting the --- taking the data and sending it to known logistic models that would allow us then to be able to estimate where that threshold is and that's what the benchmark dose is. The benchmark dose is simply a range of values with the middle value and upper and lower confidence levels. And what we typically do is we use the 90 percent of the lower confidence levels which is that PMD10, the lower 90 percent confidence level represents the 10 percent lower end of the range. That's used as a conservative estimate of where that threshold is.

The advantages of using the benchmark dose is it accounts for much more of the variability in the toxicity response and also the shape of the response curve. You can understand more accurately what's going on with this response, and it allows you to compare across other chemicals and studies as well. The disadvantage to this particular method is that it's very time-consuming. You usually have --- for each one of those doses, you have --- what you're usually looking for is 50 individuals, in this case we're talking about generally rats or mice, that are being dosed at that for each sex. So at the 10 mg/kg, for example, there would be 100 individuals, 50 males, 50 females. And same thing for 18, same thing for 25, et cetera as you go on. So you need a lot of rats to be able to do these kinds of studies. So that's generally how it works. Next slide, please.

And then what you would do is you would take that data, the raw data, enter it into EPA's aptly named benchmark dose software. It's software that is freely available on the internet. Any of you are --- can download it if you want. It's actually an Excel spreadsheet with embedded macros. That's how it works, and then the key to working this is you have to

meet the criteria. With any model, you have to have base assumptions that have to be meet. The data needs to be either in quanto or continuous form. Continuous is better. That's the way we prefer it. That's the way most of the data comes in.

You need to have a clear doses response trend, meaning that as you increase, the dose there needs to be an increase in the responses. Obviously, if something is not toxic, there won't be a response, so you won't have a clear trend there. And we also have occasionally problems where at any particular dose you get a response, it's a uniform distribution where no matter where the dose is it's pretty much the same response. Lead tends to have --- tends to operate more along that line, for example. That's why we say in toxicology there is no acceptable dose level, safe dose level for lead. But it's naturally occurring and is not safe being exposed to lead, so we do the best we can to control it.

You also have to have certain sufficient number of dose groups, at least three that were dosed with the chemical plus a control where they received a placebo, and you have to have a response in at least two of those groups and then the dose

response model should fit by some predetermined criteria. That predetermined criteria is the benchmark dose lower level. And that BMD --- or BMDL is also called the benchmark response sometimes. And that BMDL can be based on either a 95 percent lower level, which is the five percent limit, a 90 percent lower level, which is the 90 percent, or sometimes you'll use as one standard deviation. Now, one standard deviation if it's normally distributed equates to 33 percent.

The five percent is the most conservative. The one standard deviation is the least conservative. Right. So you enter the data, run it through the software and you get the output over to the right side. Notice there's seven of the logistic types of models that were used for this particular example data set. Once again, this is not benzo(a)pyrene. It does not have access to the data to be able to run for benzo(a)pyrene, but you get seven models.

And again, not knowing how many of you have a statistical background, if you have had statistics, you're used to the concept that the P value is important, right. The lower the P value, and

usually below some threshold value like .05, the lower the P value tells you that it's more significant. It's more significantly different or it's more significantly related or correlated depending on the type of analysis you're doing. But in this particular case, we want a higher P value because what that P value represents is the departure of the data from the model itself, significantly different from the model. So the higher the P value, the more closely it matches the actual model. And that's where you want to establish that threshold of five percent, ten percent or whatever.

The other thing we look at is the AIC which is Akaike information criteria, and it basically --- it's a mathematical way of determining how much mathematical --- how many mathematical loops or information did this model have to jump through in order to fit it. Right? And the more hoops you have to jump through the less confidence you have in that particular model, so we choose the model based on the highest P value and the lowest AIC, meaning you have to jump through fewer mathematical hoops to make this model. And that's why that one's highlighted there, that particular one has a BMDL of 1.976 and in that

example that would be chosen as your point of departure to establish a reference baseline. Next slide, please.

So for the non-carcinogenic effects, remember that's what --- the reference dose we're looking at, the non-carcinogenic effects for this. So what you do is you would take your point of departure and then apply uncertainty factors and/or modifying factors based on this question where the RFD, the reference dose, or the RFC, that's the reference concentration if we're looking at inhalation. You take your point of departure. You may sometimes apply some of the --- a modification factor based on bioavailability or units of exposure, respiratory volumes, things like that, but that's rare. It can be done, but it's rare.

Usually what happens is you apply the uncertainty factors to it. So the uncertainty --- UF1 is always equal to ten. And it accounts for variation in sensitivity among human populations. So think of Coronavirus and what we're doing with today as an example. We know that the elderly populations are more susceptible to problems with Coronavirus. That's part of --- as compared to younger people. That's

part of the sensitivity of human population that occur with diseases and toxic compounds as well. Every RFD will have a UF1 of 10 applied to it, it cannot be escaped.

The uncertainty factor 10 --- uncertainty factor 2 has a value of 10 as well to account for uncertainty and extrapolation between animal studies and how we're going to apply this to humans because while the biology between animals and humans is very, very similar it is not exact, right. So we account for some of that variability by applying an uncertainty factor of 10 as well, and again, unless you're working for Adolf Hitler you are not doing these kind of toxicity studies on humans. So every RFP has that particular uncertainty factor applied to it as well.

Uncertainly factor three, this is one that isn't always applied. Its values range from one to ten, but the fault is one to account for uncertainty from extrapolating from a subchronic study to a chronic study. Those, of course --- the reference dose is based on chronic exposures over a long time period, but many studies do not occur over a long time period. They're very short ranged, a few

months or a year. Anything less than two years is considered --- in a toxicity study is considered subchronic. So subchronic exposure's different than a chronic exposure, so we account for that uncertainty if you take a subchronic study and usually apply a value of 10, usually.

The uncertainty factor of 4, again, value 1 to 10. The fault is 1 to account for using a LOAEL as your point of departure. This rarely happens these days. Sometimes you're stuck with it because no matter what dose you gave them, you got a response and so you don't have a NOAEL involved. It's just that the lower you go you keep --- this is the issue with lead, no matter how low you go you keep getting this response, so you're only using the LOAEL to be able to establish that. And in that case, we apply an uncertainty factor of 10 as well.

And then there's the modifying factors which range from 1 to 10 that account for additional uncertainty due to the data quality. Maybe the study didn't do exactly what IRIS would want and so they account for some of those issues. Next slide, please. So looking specifically at the non-carcinogenic effects of benzo(a)pyrene, we've seen this both in

animals and human studies --- epidemiological human studies. This is not the --- kind of stuff. And we know that there's three types of non-carcinogenic effects. There's developmental toxicity, reproductive toxicity, and immunological toxicity.

From a developmental standpoint this is where the mother was exposed, and then in utero, the babies were exposed in utero and then it's the responses in the babies. We see neurobehavioral and cardiovascular changes that occur. The reproductive toxicity to the adults for males, we see decreased sperm count, for females we see decreased ovary weight and decreased number of follicles on the ovaries, which would then lead to decreased fecundity or fewer babies at the population level. And from an immunological standpoint, exposure to the adults decrease immunoglobulin, which is an anti-body. We're all familiar with antibodies these days thanks to the COVID pandemic going on. And also, B cell numbers. B cells actually are a white blood cell that will attack antigens such as viruses. And then you also see the associated decreased thymus. Thymus is a gland part of the lymph system gland that helps to produce the T cells, another type of white blood cell. So very

critical for your immunological responses. As it turns out, the developmental toxicity is considered the most sensitive for benzo(a)pyrene. Next slide, please.

So looking at the reference dose that was calculated specifically for benzo(a)pyrene, the benchmark doses that they found from toxicological studies, the IRIS found from toxicological studies found a range of .092 mg/kg per day of a dose to 0.16 mg/kg per day for the three different modes of toxicity, the developmental, reproductive and immunological toxicity. And the one that they considered the most sensitive because it was lowest value here was the 0.092 mg/kg per day for the developmental toxicity that came from a study by Chen and others in 2012.

And so what they did was they took that 0.092 point of departure and divided it by the uncertainty factors of 300, 10 for the required human population sensitivity, 10 for the extrapolation from a rat study in this case to humans and then an additional 3 for deficiencies in the database. That's the modifying factor at the end. So they took their 0.092, divided it by 300 and that resulted in RFD that

was in IRIS of 0.0003 mg/kg per day. And their overall confidence in this reference dose estimation is medium IRIS and that's because in the study itself this is why they applied the three-modification factor for the deficiencies in the database because they had --- the way the study was developed they may have introduced some additional maternal stress to the female adults. There was also some missing data, which means they had lower sensitivity for different developmental stages and individuals or gender specific data for all the different potential outcomes.

And not just in the Chen study, but in all studies for benzo(a)pyrene they do not have multi-generational results for this. They don't know how these impacts continue on from one generation to another. That is something they would like to see. That's a long-term study.

So what that RFD means from a risk standpoint is that if your exposure is less than the reference dose, than the non-carcinogenic effects are unlikely. If your exposure is greater than the reference dose, then non-comedogenic effects are likely. This is not an estimate or probability. It

is simply a threshold based on this benchmark dose. And what we do is we literally take the exposure that you would have, the dose and divided it by the reference dose. If it's less than one, you're okay. If it's more than one, there might be a problem. And that's the way we apply this. So ratio, you notice when the reference dose is in mg/kg/day, that's how your dose will be done as well so the units will cancel out. All right. Any --- next slide, please. So some background on what Chen did. This is to give you an idea on what we mean by developmental studies. They used an --- this is one of the studies that they did. Okay. They used an elevated plus maze. And this was the main one that they showed the impacts on. That's why I'm focusing on it. So these elevated plus maze, it's a plus because it's in the shape of a plus and it's elevated by half a meter off the ground. And two of the arms of the plus are enclosed and two of the arms of the plus are open.

The idea is that if the rodent of choice, rat or mouse, rat in this particular case, is spending time in the closed arms, that means it's feeling anxious about being so high off of the ground, whereas those on the open arm feel less anxiety.

Okay. That's where we're looking at potential developmental responses. Next slide, please. And this is figures from the actual data from Chen study. I'm not going to go into details here. I just want to have you get an idea of what's going on. So if you look at figure A on the left side along the X axis, you have male PND 35 and male PND 70 and then female PND 35 and female PND 70. The PND is short for postnatal day, how many days old it is. Right, so 35-year-old males versus 70 --- 35-day males, excuse me, versus 70-day old males and 35-day females, et cetera.

So within each group, there are four treatments. There's the control group in the black bar. Then you have .02 mg/kg treatment, the 0.2 mg/kg treatment and the 2 mg/kg treatment. All right. So you have increasing dosage as you move to the right within each group and what you generally see here is that the responses show up at day 70, the little asterisks you see there on some of the bars indicates that those treatments are significantly different than the control ones that don't have asterisks because there's little whiskers you see that bars are error bars, and they tell you that there's a lot of overlap and you can't say that they're significantly

different. But what you see is there's a delayed response. The response occurred on day 70 more so than it did on day 30 and that with increasing doses in this particular case, figure A, the latency time of the first entry in open arm decreased, meaning it took them a shorter amount of time and decided we're going to go out into the open arm. If you look at B over to the right there what you see is the same sort of thing, a delayed response. Day 70 is where most of the responses occurred and you see --- with increasing doses you see that the time spent on the open arm increases as well. What that indicates is that the rats are becoming less anxious about being half a meter off the ground. Next slide, please.

A continuation of the same data. This is actually showing data that supports the same concepts, the number of entries into the open arms. The highest doses at day 70 show that they're entering the open arms more frequently, and on the other graph to the right here, you see that this is actually the number of entries on the closed arms. So what you see is that on day 70 at the highest doses the number of entries at the closed arms is going down, so that supports that they're going out onto these open arms,

feeling less anxious. And you may say that, oh, less anxiety sounds like a good thing, right. We have a lot of stress in our culture and we want to reduce anxiety. Well. It's actually one of the reasons why people smoke cigarettes because it calms the nerves. That PAH in there calm nerves. Right? But if you are a rat or a mouse, that means you are a first order of prey item for predators, and anxiety is going to help you in that particular case because reducing anxiety puts you in exposed areas where you're much more likely to be preyed on. So the population level --- there's a reason why rats have a baseline anxiety level, right. The population level --- you're going to be losing more rats because --- exposed to benzo(a)pyrene because they're going to get predated. All right.

So note that Chen and others in their study did not calculate the benchmark dose. This is the type of data that they presented. EPA saw this study and said, oh, it looks like good data. They contacted Chen, got the raw data from Chen and they calculated the benchmark dose themselves using their software. And that's the noncarcinogenic effects of benzo(a)pyrene.

Ready to jump into the carcinogenic effects next, but before we do that, I want to see if there was any questions about any of that. Nothing? Okay. Let's go ahead to the next slide, please, if you would please.

MS. COOPER: I have a question, Ross.

MR. BRITTAIN: Sure.

MS. COOPER: So we know that benzo(a)pyrene is a carcinogen and I'll go into that in a minute, but generally, I think that I --- it's the thought that the dose for these noncarcinogen effects is just a lot higher than the dose that would be for carcinogenic effects.

MR. BRITTAIN: Yes.

MS. COOPER: So it's kind of new to this situation because this criteria is going to be more stringent than any of this data because this is the less stringent response. Right?

MR. BRITTAIN: That is correct. Any time ---.

MS. COOPER: We're going to be looking for numbers that are smaller than .02mg/kg?

MR. BRITTAIN: Exactly. Any time you're setting standards what you do is you know that

there could either be carcinogenic or noncarcinogenic effects. You calculate what your standard would be based on either type of effect. And then --- and, of course, some compounds are not carcinogenic and just have non-carcinogenic effects. Then you calculate what your standard would be for either one and then use that levels of that value to be more conservative.

MS. COOPER: Right.

MR. BRITTAIN: And in the case of benzo(a)pyrene cancer drives everything.

MS. COOPER: Right. And even though the noncancer effects aren't what's used in this one, it was interesting to see the uncertainty factors that are calculated into non-cancer effects because many of our criteria are non-cancer effects. And we saw that each reference dose is already divided by ten for correlating from animals to humans, ten for variation among humans and then another number depending on whether the study was chronic or not and another number --- another division for just random additional factors. So these numbers are already divided by ten and ten and a number lower than ten, a number lower than ten, before they ever get into that final EPA question.

MR. BRITTAIN: Yeah. And you can count on these being divided by at least 100.

MS. COOPER: Right. Okay.

MR. BRITTAN: Beyond that it depends.

MS. COOPER: Thank you.

MR. BRITTAIN: Next slide. So the carcinogenic effects, as we just mentioned, this is what really drives the risk for --- not only for benzo(a)pyrene, but the majority of PAHs. So we have evidence from numerous studies showing that benzo(a)pyrene causes cancer in animals and humans via all roots of administration, whether that be ingestion from water, ingestion of food, dermal contact or inhalation. All of the exposures are going to cause cancer.

And we see most of the tumors show up in --- this is in the case of the rats and the mice, forestomach, liver, oral cavity, the jejunum, duodenum, auditory canal, esophagus, larynx. Those are the --- where most of the tumors show up. You should know from a biological standpoint, humans do not have a forestomach. That term maybe foreign to you. Rodents have a forestomach. It's like --- it's kind of like the gizzard in --- or a crock in birds,

for example, a little pre-digestion occurs there before it goes onto the stomach.

The important thing here is that it's the metabolized benzo(a)pyrene that actually causes the cancer. They cause mutations in the genes. All right. So these metabolized benzo(a)pyrene cause DNA adducts, and adduct from a chemistry standpoint just simply means the finished product of chemical reactions, in this case biochemical reactions, and then those adducts then cause oncogene mutations. An oncogene is any gene that is capable of forming tumors. That's why you see an oncologist when you have cancer. Right. A gene has caused a tumor. So that means you have higher incidence of the tumor formation by mutating these oncogenes. These adducts also can cause tumor mutations in the tumor suppressor genes, because all biological organisms, you know, are trying to maintain genetic structure integrity as the cells undergo mitosis. And so we have it built into us, genes that are designed to suppress the oncogenes from actually creating these tumors. When you mutate those suppressor genes, that means you no longer have that safety factor built in, so you have increased tumor generation and decreased ability to be able to

fight the tumors or stop the tumors from forming. And if you want to get into the biology of apoptosis and angiogenesis, I'll be happy to get into that some other time, but you know, we'll skip that today. So benzo(a)pyrene because of that, it's mutating the oncogenes. It is mutating the tumor suppressor genes.

That's why it's called a mutagenic carcinogen, right, a mutagenic mode of action. And we do know that exposure to mutagens in early life stages, when you're a baby, an infant or a child are more likely to cause cancer than when you're exposed to them at an older state when you're an adult. So unfortunately, we don't have enough good data on impacts of mutagens at those early life stages for kids on a chemical specific basis. We have it in a general form, but not in a chemical specific basis. So what we do is apply an age dependent adjustment factor where we multiply the cancer slope factor by ten for the first two years of life, from birth to the second birthday. Then we multiply the cancer slope factor by three for years 2 through 16 and then we just use the regular cancer slope factor from age 16 through 70, which is considered the expected lifespan. So the net effect of these adjustments is that you

multiply the cancer slope factor for the entire life by 3.1 to calculate an actual risk. In this case we're actually calculating a dose based on the known risk of the Human Health Criteria, so we'll divide it by ---. Next slide, please, Laura.

So the biochemistry aspect of it, starting off with benzo(a)pyrene in the upper left corner, that should look familiar from our first slide benzo(a)pyrene, five attached benzine rings. There's a three step bioactivation process that is mediated by the cytochrome P450 enzymes, and that ends up in that lower left corner with the BPDE that has the rather lengthy name you see there. If you want, I can pronounce it, but unless you have a lot of organic chemistry, it's going to be meaningless to you. so that's why we just call it the BPDE.

Notice how the molecule has changed functionally on the kind of fringe benzo --- benzene ring that's up to the lower left there on this particular molecule. Two of the hydrogen have been replaced by hydroxyl molecules and another two of the hydrogen have been replaced by a shared oxygen atom. That's what is changed to the benzo(a)pyrene through this process. And it is that molecule, the BPDE, that

is the mutagen. It then creates the adducts on your DNA. So if you know anything about DNA, of course, it's a double helix, spiral helix, two strands that are attached together. And when it comes to our genes, there's actually only four proteins that bind our genes, the two strands together. It's either guanine, adenine, cytosine or thymine, GCAT. And it's the combinations of those four proteins that make up all of our genes. Okay.

And so what BPDE does is it replaces either the guanine or adenine in your genes with these adducts. That's the mutation that occurs. Next slide, please.

So for benzo(a)pyrene, of course, we have to develop an oral cancer slope factor. This --- generally speaking how this is done is there's a dose response, similar to what we did before for the noncarcinogenic factors. It's done in the same software. What we're looking for here is we're looking for a linear response at the low dose range. We know that the upper dose range, if it's carcinogenic. it's going to cause cancer. It's the lower dose range that we're most worried about. We have these models. If you look at that particular

graph that I have here, it looks very similar to what we saw before. It's an S shaped curve, logistic type of response, and what we look at here is our BMD, our benchmark dose, or the lowest dose that causes cancer. We're going to look at that and then on your Y axis, it's percent risk of cancer. So what we're talking about here is the percent of individuals in the study that actually got this particular type of cancer and there could be numerous types of cancer that you look for. You do one type of cancer at a time and look at the percent of individuals that developed that cancer. And you run the BMDs for all these different potentials. And you look at the lowest dose that caused that percent of cancer and then you draw a line from that lowest dose down to your origin where there's no dose and no cancer.

And it's the slope of that line that we're calculating here. Calculating a slope allows us to develop a probability of cancer within that range. That's what we're after. That's why we use one in a million for cancer and we don't use one in a million for the noncancer stuff. Because the noncancer stuff is a threshold response. The cancer stuff is a slope probability. Okay. We have several models we can ---

that they --- to use here. I will state a multi-stage Wible (phonetic) model is the most preferred. That's the best model. Next slide.

So for the benzo(a)pyrene when they developed the cancer slope model, they actually used the multistage Wible model, the best model overall, to develop this, which is good. And what it does is it predicts the probability of a carcinogenic tumor by some observation time T given the dose that was used in your toxicity study. The benchmark dose is calculated by finding the root of a nonlinear equation which involves calculus, and again, I'm not going to go into that. The benchmark dose is then the estimate of a fatal risk response in this particular case. Usually what we do for that benchmark dose is we use the lower limit once again. Often times --- or I should say most times we use the 95 percent lower confidence limit for BMD. In this particular case of benzo(a)pyrene, IRIS used 90 percent, so that's the BMD ten percent. Next slide, please.

Then what we have to do is adjust the cancer slope factor. We can do the same adjustments we did on the BMDs we did before with the noncancer stuff in terms of applying uncertainty for human

populations and animal to human studies, et cetera, but we also have to adjust for body weight. That's one of the other big adjustments.

There's several approaches. One is the direct portion reality where we make no adjustments. Another one is we take the body weight of the rats or rodents and multiply that --- or raise that to a two-thirds power. Because that is based off of the proportion of skin surface area ratios so we tend to use that for dermal type of exposures. For that type of exposures like we're dealing with here we use the body weight ratio raised to the three-quarter power because as organisms get larger and humans are considerably larger than rats, the proportion of organs the target organs within the body gets lower. So that's why you make that adjustment. And these adjustments are all used to calculate what is often called the human equivalent dose. It's the point of departure. The human equivalent dose is the point of departure. And when you use the body weight in relation to three-quarter power, the uncertainty factor from animal to human toxicity is no longer 10. It's reduced to three because we're already accounting for a lot of that uncertainty and it all kind of comes

out in the wash it turns out. Next slide, please. So benzo(a)pyrene, the cancer slope factors noted changed in 2017. And this is what's currently being used in the risk world. It's not what the old --- or the human health criteria for benzo(a)pyrene from 2015. The proposed one from 2015 EPA developed. That's not what it's about. That's not the one they're using. So the current one, cancer slope factor is 1 per mg/kg/day, which is the same thing as saying 1 mg/kg/day raised to the -1 power, which is the same thing mathematically as saying one over one mg/kg/day. The idea is that the units are on the denominator. You're going to multiply that by your dose, which is mg/kg/day, and the units will cancel out. That's why it's in the per mg/kg/day. And they did this based on Kross (phonetic) et. al and the Bellin and Kulp (phonetic) studies. Kross studied rats. Bellin and Culp studied female mice, not males. That's a limitation in that particular study, only female mice.

Both of these studies use physiological exams, which is slicing the tissues to look at the actual individual cells. They did it in many different types of tissues. They had three exposure

levels plus a control. They had about 50 animals per sex per group, which is --- that's the golden standard. And they treated it for two years, which is the minimum to be considered a chronic exposure study, right. So this is --- these are all very reasonably good studies. That's why they use these.

And interestingly the high dose treatments of all these studies --- every single rat or mouse was dead or more by the week 79 due to their exposures to benzo(a)pyrene. I'm telling you this stuff is very toxic. You know that. Next slide, please, Laura.

And this is an example of some of the output that you see in these studies. This comes from the Kross, et. al study. It's showing the probability of adenocarcinomas in the duodenum or jejunum for female rats at different doses. So if you look down at the lower left dose there, that's 2.32, that's 2.32 mg/kg that rats are getting dosed every day. And you'll see there that they have these black dots. Some circles mean that there's a tumor that developed that particular day and that's where it ended up being fatal. And you see that all of those dots are solid by the time you get to week --- just before week 80.

That's what it's saying. They'll die --- at the higher dose, they'll die by week 79. Next slide, please.

So the earlier stuff that we had on benzo(a)pyrene is cancer slope factor. It was developed --- the first cancer slope factor was developed in 1992, 25 years before the update that occurred in 2017. Benzo(a)pyrene at that point was in group B as a probable human carcinogen because they didn't have adequate data at that time, and the reason for that is because while they knew PAHs were carcinogenic, they didn't know what specific compounds within the PAH mixtures were actually causing cancer. Further studies have showed us that, yeah, benzo(a)pyrene is the big driver.

And what they did at that time was they developed four different cancer slope factors based on four different studies, either different types of tumors --- actually what you'll see here, the 11.1 mg/day from Brunn, et. al. And the other 3, the 5.9, the 9.0 and the 4.5, all came from Neil and Rigdon (phonetic) in 67. But they were at --- those were different models for different types of tumors, right, within --- within the same study.

So they had really two good studies and four different models for different types of studies. All of them had equal merit in IRIS's viewpoint and they were all within threefold of each other. So IRIS couldn't say that one was better than the other one. What they actually did is they calculated the geometric meaning of those four cancer cells to come up with the value that they use, the 7.3 mg/kg/day. That's the old value and that's the value that's used in our current human health criteria.

Note some of the issues we have with some of these studies, though. The Brunn, et. al study used only 32 rats per sex per group instead of the preferred 50. A lower sample size which means you have variability issues. And also, they had a variable dose time. They weren't dosing in every single day. And even they were inconsistent from week to week with how they were dosing, so that can create some issues too. The Neil and Rigdon study, they only studied it for a year. So this a subchronic study. It's not a chronic study. Since it was a subchronic study, they had to apply an uncertainty factor of ten. So that is part of why --- what made it higher. Next slide, please.

And this is the data --- and I'm not going to go through all of this. This is the data for the point of departure, the BMDL and the slope factors for the different types of tumors from the Kross and Bellin and Kulp study. And what you see here how you --- I'll explain how you actually calculate slope factor. Look at the BMDL, the first one, the .281 for the forstomach in male rats. That .281 remember --- I told you that they did this based on a 90 percent confidence and the lower level at 90 confidence, that means a 10 percent probability of getting the cancer. So you take that --- and, of course, the slope is simply rise over run, change in Y over change in X. So the change in Y is ten percent or .1. The change in X is the BMDL, the .281. So you take .1 divide it by .281, and if you have a calculator, you will come up with .355, which rounds off to .36. That's how the slope factor is calculated. And they do that for every single one of the different types of tumors that they had showing up for these different treatments and different sexes.

And what you should notice here is the lower that slope factor the lower the toxicity from a cariogenic standpoint for that particular tumor type

for benzo(a)pyrene. The highest one you will notice is the alimentary tract for the female mice. That's from the Bellin and Kulp study. That's the 1.4. That's the highest toxicity slope factor that came from these studies. Next slide, please.

So as with any scientific endeavor, there's always uncertainty built into this. And so the uncertainty from these particular studies I mentioned earlier, the humans do not have the forstomach which meant that these rodents, they will have a longer duration of exposure of benzo(a)pyrene within the forstomach compared to what humans would have. We would have a longer duration within the stomach itself. The rat study from Kross, they used soybean oil and gavage which is --- force-feeding is what gavage is, compared with just simple dietary exposures for the mice. And that is important here because benzo(a)pyrene is lipophilic. It's going to attach to that soybean oil. And once it's in that soybean oil, it's much more likely to go to the lymph system then through the --- to the digestive system, which changes the exposure pathways for these rats compared to what the mice would. And gavage, we also know as a prong --- gavage gives you a higher peak concentration which creates a

nonlinear response as well. So that's part of the problem with the rats' study.

The rats were dosed only five days per week as well, which means they had to use some math to adjust for that. Whereas the mice were dosed every single day, which is what you want. The alimentary tract tumors, generally speaking in both studies, had a fivefold greater cancer slope factor, so alimentary tract was conservatively chosen.

The mouse study had a threefold greater cancer slope factor compared to the rats, so they chose the mouse study as the preferred source for the cancer slope factor. They also used a bodyweight ration of three-quarter power scaling. You know, there's always some uncertainty involved with that because we don't know if in these particular rats were actually three quarters power of the humans in their bodyweight.

They did use the multistage Wible model. That's the best model overall, so that's also a good thing that reduces the uncertainty. And then there's the assumed linear low dose extrapolation for cancer. You know, there's always some uncertainty involved in that. But with mutagens, we know that

they generally follow that linear dose response pretty well, so that should be a relatively low amount of uncertainty there. And, of course, with mutagens we have to --- we use the general age dependent adjustment factors for mutagens, but the actual age adjustment that should be made specific to benzo(a)pyrene is not ---. We just use the best that we can, the knowledge we have at the moment. Next slide, please.

So they had to choose the cancer slope factor. The rat risk estimates span a fivefold range which is not good. That means that you're getting a wide degree of variability. I mentioned the issues we had with the rat studies as well previously. There was no data that they had to support any one result as most relevant to extrapolate to humans. So what they did then is they calculated --- much like they did earlier, they calculated geometric mean of all of the different variables that they had from these different studies. Getting equal weight to rats and the mice, and that came up with the 0.74, which is basically one tenth of the old version that was 7.3. And so then they realized, of course, you know, they do have the corrections for sensitive populations, but lab studies

do not really account for sensitive populations directly. So that degree of uncertainty they said that really supports the use of the highest items, that Bellin and Kulp alimentary tract value at 1.4 per mg/kg/day.

And so EPA choose that one to be the basis, but they didn't use 1.4 exactly. They rounded it off to 1, rounded it down to 1 is what they did, 1 mg/kg/day. That's the one that they developed in 2017. Next slide, please.

So the conclusions of this. Basically, IRIS kind of split the difference between the highest value in the geometric mean to hedge their bets for sensitive populations, and what you do with this cancer slope factor then is you would get --- the risk is just the toxicity times the dose. In this case, the toxicity is just the cancer slope factor. Multiply cancer slope factor times the dose, that calculates the risk, the probability of getting --- an individual getting cancer.

However, in our particular case, we actually know what the risk is. We've set it at one in a million. That's the probability of getting cancer. What we don't know is the dose of

concentration, so we then rearrange and back calculate to be able to get the dose and concentration, which will be our human health criteria.

Now, this novel --- I'll interject here as to why you see the bladder cancer and leukemia ribbons here. This is deeply personal to me. My dad died of bladder cancer. My mom died this year of leukemia. So I take this stuff very seriously to try to protect human health and environment.

And so that ends the cancer end of things. And I'll open up for questions. We have one more follow-up slide on what this means. I don't know if you wanted to answer questions here, what it means for the human health criteria. If you want to have any questions, let me know. Fire away.

MR. HARRIS: So, Ross, this is really a very complicated set of studies, and so I congratulate you on putting it together. I just wanted to correct one thing that you said with DNA, the --- those are nucleotides, not proteins.

MR. BRITTAIN: Yes.

MR. HARRIS: So the adduct that forms that the benzo(a)pyrene binds to, let's say, a guanine or adenine, so that alters that nucleotide position so

that during fetal replication or repair of DNA mistakes can be made and that's a mutation, so --- but that's the only thing I noticed. And the rest of it made me glad I didn't go to toxicology school. You did a great job.

MR. BRITTAIN: Thanks. Any other questions or issues?

MS. EMERY: Hello, Ross, this is Kathy. So if I'm understanding all of this and I'm by no means an expert on this, by the time you work all the way through the uncertainty factors and everything else, is this --- is what they've done relatively conservative?

MR. BRITTAIN: Yes. Yes, it is. It is relative to that individual compound, and one of the reasons we apply that --- as I've mentioned in previous meetings, one thing this does not account for is accumulative impacts. What are you exposed to in addition to this? And how is benzo(a)pyrene interacting with other chemicals that you are exposed to, and does that increase or decrease? Because the exposure with other chemicals may be either synergistic, meaning it increases the toxicity dramatically, or it could be antagonistic, actually

decrease the toxicity of benzo(a)pyrene.

We don't know, and the complex mixture of chemicals you have in your body --- you know, we can't model that kind of stuff. So we do the best we can. As part of why we apply these uncertainty factors so much is to try to account as much as possible for the cumulative impacts that may be occurring. Now, one of the things I can say specifically to benzo(a)pyrene is I know if you're being exposed --- unless you're working specifically in a lab where they have isolated benzo(a)pyrene, if you're being exposed to benzo(a)pyrene, you're being exposed to at least 16 other PAHs at the same. And so we know that you have the potential cumulative impacts from the PAHs.

And that's why that's one of the --- and it's hard to understand how they interact with each other. That's one of the reasons why the other PAHs that are on the human health criteria their cancer slope factors and toxicity --- actually just the cancer slope factors, are based on an equivalency factor to benzo(a)pyrene. The cancer slope factor for benzo(a)pyrene effects at least five other human health criteria because dibenz(a,h)anthracene,

benzo(a)anthracene and benzo(b)fluoranthene, et cetera, those are all based on the cancer slope factor for benzo(a)pyrene because we don't have good toxicity studies on those.

MS. COOPER: And, Ross, do we know whether they feel like benzo(a)pyrene is the most toxic of the PAHs or maybe that's just what they happened to do the study on?

MR. BRITTAIN: Yeah, it is the most toxic that we have seen thus far. Now, I will say the exposures can change, though, right, because there's differences in solubility and volatility and things like that. So while certain PAHs are maybe less toxic than benzo(a)pyrene, they may be more soluble, so you get more of it in your water. Or they may be more volatile, so you inhale more of it. So that can change the overall impacts depending on your exposure assumptions.

MS. EMERY: All right. And I have to say I would just interject that I do --- I think about this every time I get the grill out, which is for us about twice a week and throw the chicken out there, and one of the best things that we love about the chicken is the little black pieces and we eat those

everyday, so I'm not stopping doing that, but I understand to some extent.

MR. BRITTAIN: That is why the World Health Organization has declared grilled meats a carcinogenic compound. It's because they contain the PAHs and we know the PAHs are carcinogens. Actually, Laura, could you skip to the slide that has the --- go all the way to the back that has the BMD graph, the initial BMD graph? Keep going back.

MS. COOPER: The first one?

MR. BRITTAIN: Yeah, the first one. It would be like my second slide --- second or third slide, something like that. That one right there. Notice here --- getting back to the uncertainty thing, notice on the very lower left corner where the arrow is for the RFD, right, because they took the BMD as a point of departure and then applied the uncertainty factor so that RFD is telling you you're way down in a range that should be in most cases actually below your NOAEL.

MS. COOPER: No affect. Right.

MR. BRITTAIN: Yeah, no affects. And that's to account --- to conservatively account for many of the unknowns that we just don't know.

MS. EMERY: So conservatively accounted for all of the unknowns and this is impacting other constituents, then is this --- I guess what they've done here is a good starting point?

MS. COOPER: This is a good way to understand the noncancer effects because noncancer effects use this reference dose. And like Ross was saying the reference dose like you can see on this graph is way down below the no affect level. That's the NOAEL. So that's when --- that's because they put all of these uncertainty factors in there, so the reference dose that they insert into the equation for noncancer factors is below what they would expect any effect to cause --- anything to cause an effect.

MR. BRITTAIN: And recall that your cancer is a slope, so remember the RFD is a threshold. They're saying --- they're saying above this particular threshold we think that it is likely that an effect may occur. Whereas with cancer, it's an actual slope. We're calculating ---.

MS. COOPER: I'm going to forward to that --- I'm going to go forward to that slide so we can see the difference. The benzo(a)pyrene in the human health criteria calculation is using the cancer

slope factor, not the reference dose, right?

MR. BRITTAIN: Yes, because it is --- has a higher impact. A lower dose of benzo(a)pyrene is more likely to give you cancer than it is for you to have a noncancer effect. You can have both occurring --- at a high enough dose you will have both occurring.

MS. CROWE: So in their calculation of the cancer slope factor, isn't there places where they could have been more conservative, like they used the 90 percent confidence interval when they could've used 95?

MR. BRITTAIN: Yeah, that's certainly part of it.

MS. COOPER: Now, the 90 percent if you're looking at this graph here, Ross, is that --- are we saying that that's like ten percent into that line?

MR. BRITTAIN: Yeah, so remember when you calculate your slope, which is rise over run, so that benchmark dose that I showed you was .281 at the 90 percent level. So what you do is you go down here, 10 percent level on your graph and draw a line over. That's where the lowest dose that cancer caused would

be at .281 on the X axis, so that's why it's .1/.281 to get that cancer slope factor of .36 that I came up with. So if you change that to a five percent, that will also change the location on your X axis. And so the slope may not change at all. It may end up being the same or may even be lower depending on ---.

MS. COOPER: So this is the --- this is the cancer slope line, and the lowest dose that caused an effect is here, so they draw a straight line between here and zero. And, Autumn, I think you understand this, but the ten percent would be somewhere around here, 10 percent of this line, the distance in this line from here to the lowest dose that causes cancer. If you were going to go to the 95 percent, it would be more like, you know, there. You know what I mean?

MR. BRITTAIN: Yeah.

MS. COOPER: All right.

MS. CROWE: Did they give a justification why they just rounded --- I mean, that seems like that --- you know, .4 is kind of significant when we're talking about, you know, the very small amount of doses. In analytical chemistry, we're never allowed to just like drop a decimal point.

MR. BRITTAIN: Yeah, yeah. A lot of it has to do with the precision of your instruments, that kind of thing. So in analytical chemistry, you wouldn't do that, but remember, we've already applied a factor of --- a certainty factor of 100. Like, we don't have that kind of precision in what we're doing here. As a matter of fact, whenever we actually calculate actual risks off of these cancer slope factors, we rounded off to the nearest whole number because there's so much uncertainty involved in it. Anything beyond that first whole number is garbage. Right. There's too many unknowns here. So ---.

MS. CROWE: And so that's one way you can say that that's what they did, I would ---

MR. BRITTAIN: Yes.

MS. CROWE: --- I would guess, is that this is the whole number. They went down to one.

MR. BRITTAIN: Yeah. Now, we can simply choose two significant figures on several of our cancer slope factors, but a lot of that has to do with the confidence you have in your data. Remember they only have like a medium level confidence in this particular data as well. So that's part of it. If they had a high confidence, they feel better going

out to more significant digits. If you have --- the lower your confidence, the fewer significant figures you're going to put into that cancer slope ---.

MS. COOPER: So the 1.4 on this slide that I think that you're referring to, Autumn, because you said, you know, dropping .4 is a concern, so 1.4 -- I think I want to go back. That was the part of -- that was this greater ---

MR. BRITTAIN: Yeah.

MS. COOPER: --- the greatest number in this column here, which was a --- that was from alimentary tract of the female mice that gave the strongest response.

MR. BRITTAIN: Uh-huh (yes). Correct.

MR. HARRIS: So I have just a question. This is Larry. Would it be the case that for any compound known to be a mutagen or carcinogens we're going to have a better confidence in what that dose would be? In other words, you're making it even lower than you would if it was a noncarcinogen. You know what ---?

MS. COOPER: In this ---.

MR. BRITTAIN: That --- it's a good question, Larry. Generally, that really depends on

the quality of the data. The quality of the data really determines --- because there's certain uncertainty factors that are applied no matter what, right. It's the quality of the data that adds additional uncertainty to the overall impact. So, you know, you could have something that's considered a probable carcinogen versus a known carcinogen, but it has a really high-quality data set that may end up offsetting the uncertainty due to the fact that you're not sure just how carcinogenic --- you know, whether or not it truly is. Believe it or not, that actually happens. And a large reason for that is in terms of you're not sure if it's a carcinogen, but you have data over here saying you have a pretty clear high confidence in your cancer slope factor. And the reason for that is because, remember, it took 25 years for IRIS to update benzo(a)pyrene. Right. Most of the data in the assumptions that are going on in a lot of this is old and outdated unfortunately. You have a lot of chemicals that were assessed in IRIS 25, 30 years ago as probable or possible human carcinogens, but the intervening data is now telling us, oh, yes, it actually is a carcinogen. The EPA just hasn't updated

that particular --- to Group A yet, because they haven't reviewed it. So a lot of these reviews are unfortunately all been out of date.

And remember this, they've only --- IRIS has only reviewed just a --- you know, what I consider a handful of chemicals, just a few thousand chemicals. Now, there are over --- well over hundred thousand man-made chemicals in each one of our bodies/ There's at least 10,000 of them in each one of our bodies by the time we reach adulthood that are interacting in some way that, you know, we can't --- we don't know. So that's part of it, is that IRIS, if they want to go back for something like benzo(a)pyrene, that means --- to reassess benzo(a)pyrene, that means they're not doing a new chemical that probably needs to be added to their overall summary because they only have so many resources to do that kind of work. So it'll be --- at this particular pace, it will be hundreds of years before they're actually done with their reviews.

MR. HARRIS: Ross, one other thing, I'm trying to get my mind around it. Maybe we're going to get there eventually. There're these studies which are laboratory studies that give data and you

can analyze it as you explained, and then there's the DEP's ability to detect those compounds.

MR. BRITTAIN: Yeah.

MR. HARRIS: And it seems to me that you might be more careful than the laboratory and get a value that's important.

MR. BRITTAIN: Oh, we do it all the time.

MR. HARRIS: And you can't measure it in the environment.

MR. BRITTAIN: We have --- yeah. Given the current --- we have that on several of the PAHs --- 123CD pyrene and anthracene are examples where our keeper of the de minimis standards ---. Our de minimis standards are actually lower than the lowest detection level that the labs can get, and this is at a national scale. It's not just West Virginia. It's a national problem. EPA and their groups are working on trying to come up with better laboratory methods to be able to lower those detection levels to the thresholds that we're worried about. But, you know, it's the best science that we have.

That is going to be a very serious problem for P-fast compounds as they start to become regulated as well.

MR. MANDIROLA: Yeah. And this is Scott, Larry. Current PAH numbers, for instance, for benzo(a)pyrene are .0038. Okay. That --- PBD. That's for the instrument's ability to detect it, and the recommended criteria that EPA came out with is .00012. So it's a factor lower. It's more than 10 times lower than that.

And, you know, even though analytical instrumentation has excelled in its ability to reach lower detections limits over the last couple decades, some of these are not even in the area of being able to be detected. Now, how that equates to an APS permit when time comes, whatever the water quality standard is that is what's used to calculate what the discharge limit is on the end of --- and there may be a mixing zone associated with it. But you figure out what the average monthly and max daily would be in order to protect that water quality standard at .0038. That said they will get a limit that there is no way they can possibly test for. What they will end up doing, there will be a statement in that NPDES permit experiment that will indicate that they need to be none detect at the laboratories MDL and we have our Labserve (phonetic) program that makes sure they are

reaching the recommended MDLs for those compounds. So the MDL for that particular --- for benzo(a)pyrene may be .04, for instance. That's ten times higher than the water quality standard, but they need to be at the MDL to be in compliance. If they get higher than that method detection limit, then it would count towards a potential violation of your permit. But there is a distance, a void between what instruments can say and that MDL or the --- I'm sorry, what the instrument can see is the MDL. Between that level and the water quality standard there is potentially a void that at this point in time the instrumentation just doesn't have the capability. We don't have the substance to get that far.

MR. BRITTAIN: Thankfully it's a limited number.

MR. MANDIROLA: Yeah. It's almost always carcinogens.

MR. BRITTAIN: Unfortunately.

MR. MANDIROLA: Well, and there's a reason behind it. And it's simply because most of them you have to go very, very low before you come up with any no effect. Correct, Ross?

MR. BRITTAIN: Correct. Correct.

Yeah. The inherent uncertainty that makes us go more conservative than it would be if we knew it just on its own. You know, if we wanted to study humans directly, we could eliminate some of that uncertainty, but I don't think we want to do that. I wouldn't recommend it. Right.

MS. COOPER: So I think this is a good moment to move onto the next slide because it has more information on it about the criteria that --- as it stands and how this affects it. So as far as what this means for criteria, we've got here --- the 2015 EPA criteria includes a cancer slope factor of 7.3. That's based on that old data from the early --- study from the early '80s and a study from the '60s. That's what that cancer slope factor is based upon.

So with this 2017 revised cancer slope factor for benzo(a)pyrene, it would be one as we mentioned. And so this comes into the equation in the numerator here. And you'll see that this is where we have a cancer slope factor and this is where we multiply it --- or we multiply it by --- we divide ten to the minus six by this cancer slope factor. So currently, this is the equation from the benzo(a)pyrene document that EPA has on file now for their 2015 criteria. You know,

you see the 7.3 isn't here, so basically you take this 1 in a million basically, divide it by 7.3 to make it a lot smaller or --- yeah. And then --- and that comes out to .00012 that is the current recommended criteria --- EPA's 2015 criteria. So if we take that 7.3, we replace it with 1 based on this new information, which is the study that Ross just went over that was based --- this information is based on a 2001 and the 1992, I think, studies on mice and rats. And then we come up with .00091 micrograms per liter. Like Scott was mentioning a lot of times and I think in the case of this, the method --- the detection limit for this chemical is already higher than this. This is lower than the method detection limit, and many times the method detection limit is an order of magnitude higher. In this case, this change would make the recommended criteria go from 1.2 to times 10 to the minus 4, which is the same thing as this number here, to 9.1 times 10 to the minus 4, which is the same thing as this number here. So as you can see, the --- it doesn't change by an order of magnitude, but it does multiply this criteria by 7.3 basically, because you are no longer dividing it right here by 7.3. So it basically makes it 7.3 times higher, which again is

less than ten times higher which is less than an order of magnitude. Both of these numbers are below the method detection limit, I believe. Unfortunately, that's not something I put into the presentation here, but correct me if I'm wrong, the method detection limit for this chemical were already below it. So that's what would change with this criteria if we were to take this new IRIS data and incorporate it into any recommended criteria that we make.

MS. EMERY: What's our current standard again?

MS. COOPER: .003. I have that on the next slide. I think it's the next slide.

MR. BRITTAIN: .0038.

MS. EMERY: So that basically increases the standard for making that change.

MS. COOPER: Yes, it does. And instead of .0038, we would have ---.

MR. BRITTAIN: It increases the current EPA recommended ---

MS. COOPER: No, it doesn't --- it's still ---.

MR. BRITTAIN: --- from 00012 to 00091, but that's still lower than our current quality

standard ---

MS. COOPER: Yeah, there we go.

MR. BRITTAIN: --- which is one order of magnitude, one less zero. Correct?

MS. COOPER: Yeah, so --- yeah. We currently have 3.8 times 10 to the minus 3. What EPA is recommending now in their 2015 criteria is 1.2 times 10 to the minus 3 ---.

MR. BRITTAIN: Four.

MS. COOPER: --- 4, yeah. And what it would be would end up being 9.1 times 10 to the minus 4. So that's the difference. And that brings us to this next slide, which I just wanted to talk about the remaining criteria that we're looking at. This is our spreadsheet and I wanted to bring that up actually. Let me get out of this.

Sorry. I need to stop this share first. Stop share. Sorry. Kids screaming. It happens. Okay.

So I'm going to share again. And I got screen one --- there we go. Okay. So this is a spreadsheet that we are looking at when we look at our criteria right now. That's why I was on the wrong line earlier. Okay. Yeah, benzo(a)pyrene .00012 and

I was clicking before on the one above it, which was an order of magnitude different. So I just was worried that was wrong in here, but it's not. Okay. So this is just the color-coded version of the criteria that we look at. If I scroll up, you'll see the 24 criteria that we've already recommended. So I'm scrolling down to below those because we're not really talking about those now. We're talking about the remaining and criteria. You'll see that there are --- there are 36 of these. These are the criteria that either are in --- that we have in our criteria now, in our standards, but it also has the folates (phonetic), busted out to the various folates. We currently have, you know, folates all combined together, and so this ends up being 36 criteria. But I have highlighted in yellow here the PAHs that we're talking about today. Of course, we're talking about benzo(a)pyrene, but benzo(a)pyrene numbers are used to d form the criteria of all these other yellow highlighted chemicals. So these are basically the ones that we're starting with because we talked about them today, and we'll also be talking about PAHs in the January meeting. I've asked Jenny if she can bring some information based on PAHs. I think she's

going to mainly focus on bioaccumulation factors. Any studies that have --- that are --- that we know of that have come out that may better inform bioaccumulation factors for these PAHs.

And I have these --- I just wanted to mention --- show that these are color --- I've coded color --- color coded these to show, like, the differences in opinion basically. You'll see that the blue ones are EPA criteria --- are where EPA criteria are more stringent than West Virginia's current criteria. So those are the ones that Angie wants --- you know, is happy with, because they become more stringent if we recommend --- if we adopted the EPA recommended criteria. And the red --- the pink --- the salmon colored one are criteria that are recommended by West Virginia manufacturers that are actually less stringent than that EPA criteria. and if I scroll back up, you'll see that the green ones are the ones that were recommended by manufacturers that are either more stringent than or very close to the EPA recommended criteria.

So these are the ones we recommended that are in a rule now and this is the --- this is where they overlap with what had been suggested, that

we only adopt criteria that become more stringent. And again, the highlighted in orange ones are the ones that are currently in our proposed rule.

MS. ROSSER: The yellow 9 through 12 rows should --- should column G be salmon on those? Oh, wait. I'm sorry. No, I'm sorry, different row. They are renumbering for me, below 24.

MS. COOPER: Right. And that's just because I was counting them making sure that, okay, so this is the 24.

MS. ROSSER: Yeah, yeah, yeah. Okay.

MS. COOPER: They're in the rule. And I restarted the count.

MS. ROSSER: So 38 through 41, those ---?

MS. COOPER: Yes. Okay. So the other thing is I updated the slide this morning because I've made sort of a mistake there. That the Manufacturers Association actually --- what they recommended in their --- when they submitted to us their --- the letter in 2019 --- in the fall of 2019 when we received those recommendations, they had put values in here, but what they also said was they recommend that we keep the West Virginia current criteria because

these values were --- ended up being higher and they didn't see a reason to --- you know, to recommend higher limits for --- for the PAHs. They wanted to just go ahead and stick with what we have. So these that are in white here are --- because the manufacturer's recommended that we actually just keep the criteria the same for PAHs. That was their recommendation.

MS. ROSSER: Right. My question is why aren't those coded salmon? Because the .0038 are less stringent than the EPA criteria.

MS. COOPER: Oh, well, yeah, that's true. I just made that adjustment just this morning and I was kind of confused about what color I should make them.

MS. ROSSER: I love those colors. This is great.

MS. COOPER: Right. Technically they are less stringent than what's recommended by EPA. And then that also becomes more confusing because now we have this new IRIS update to benzo(a)pyrene which would affect all the PAHs, which would make them --- basically this is that calculation that I did here just to see it. It would be like that would be the

IRIS number that was recommended. .0009, I need to make that a little more viewable. Yeah.
So that's the other thing, and of course, I have it in the wrong column now because now it looks like it's an MCL. But you get the idea, that these are the ones that we're looking at because there is new information and because they all get lumped together as PAHs. So when you do a study on one, you can correlate that study to the others as they do with the cancer slope factor for those. So that's what we're going to focus on --- we're going to continue to focus on PAHs next month and we'll move on to something else after that. But ---.

MS. ROSSER: Can you explain why you have decided we're focusing on PAHs?

MS. COOPER: Well, because there's --- because --- well, today because IRIS updated this information, so we really wanted to explain that because that is something that EPA would certainly take into consideration and accept if we were to adopt numbers based on that new information.
Beyond that, it's because they are a group that when you study one, you can correlate it to the others or many studies do --- they combine all of these

chemicals into one study, so it would be likely --- we'd be likely to find a study that would include --- that would be discussing all of these together just because they're a group that can --- it's --- instead of looking for just like altering, which we may find information on altering, or we're going to be looking, but we know that we have studies out there that are for the PAHs.

MS. ROSSER: Okay. That's helpful to understand the rationale behind that. I guess as a workgroup member I would offer that, you know, from our perspective, we would like to prioritize those chemicals, A, that we're seeing the biggest difference between current standards and EPA recommendations in terms of that needs to become more stringent, and those that are --- I don't know if Ross can explain, like the most toxic or most dangerous --- they're all dangerous, and those that we know are in use in West Virginia. So it's like trying to create some criteria for what we prioritize trying to reach consensus on.

MR. BRITTAIN: Angie, I'll intervene there. That's one --- that's another one of the reasons to focus on these PAHs because they are ubiquitous throughout West Virginia because it's in

coal, it's in petroleum' it's in our fuels, you know, diesel fuel in particular, so --- and, of course, also the byproducts of the use of these. So it is everywhere.

So focusing on --- a PAH focus has a lot of merit just from that standpoint as well. But yeah, I'll be happy to --- Laura, if you want --- or whoever, if you want me to or you can do it yourself, to go through and, like, look at, you know, what --- I don't actually know what's actual --- I know what's going to be the most toxic. Dioxins are the most toxic. But what's actually in use in West Virginia is another issue.

MR. MANDIROLA: We find that pretty much in any petroleum product that you deal with. As well as the industrial processes, they use it in the industrial processes as well.

MS. HENTHRON: And keep in mind that PAHs are anytime you burn something, so forest fires, residential wood burning, cigarette smoke. Yeah, I mean, it's not just industrial sources for these.

MR. BRITTAIN: Oh, no. That's why I was trying to get the point across. It's ---.

MS. HENTHRON: Yes.

MR. BRITTAIN: It's when you go fill up your gas --- your lawnmower. It's --- it's, you know --- they are very pervasive not just in West Virginia, but in the entire country.

MS. COOPER: What was the first thing that you mentioned, Angie, that you want to focus --- you want to look at these based on whether they became more stringent than EPA's recommended criteria or whether they became less stringent than EPA's recommended criteria? Which one would you think would be beneficial to focus on if we were looking at it in that way?

MS. ROSSER: Those where we're seeing --- EPA is recommending something more stringent than what we have. And if we, you know, build up a criteria on top of that is where there is the biggest gaps or biggest differences. I mean, I'm looking at --- as we look down through some of it, there are orders of magnitude, so ---.

MR. BRITTAIN: Uh-huh (yes).

MS. COOPER: Right.

MS. ROSSER: To me, there's a sense of, like, you know, we got a lot of --- we got ground to make up and we need to do it sooner than later.

MR. BRITTAIN: Uh-huh (yes).

MS. COOPER: Okay. So I mean, as you can see, we can add more --- I'd like us to be familiar with this. This is the most simplified version that we've put together, and again, this is only category A criteria as you can see at the top of the columns here. Just so we can --- it's harder when you add another whole set of columns to each one. But when we add --- we can look at this with more detail like in the ways that you're suggesting and kind of mark them or, you know, list the ways that people are exposed to these or if they're in West Virginia, if they're being used and then, you know, we can look at them in this way, too, the biggest gap between what EPA recommends and what West Virginia has and just kind of mark them that way. I mean, we have them marked already whether they are either less or more, but I don't have them marked as to whether the biggest gaps are between what's recommended by EPA and what we have in our criteria. So we can certainly do that and bring this --- bring this back next time to look at it again.

MR. HARRIS: So this is Larry. I've been on this council since it began, and I was always

under the impression that our water quality rules were adopting EPA standards as they promulgated them. And now I'm looking at this and I'm seeing it's not that way at all. And it's the first time I realized it is during these sessions that we're having.

MS. COOPER: The current we have were adopted as recommended by EPA when they were recommended. We just haven't updated them as for the 2015 recommendations yet.

MR. HARRIS: The white columns is the latest ones?

MS. COOPER: Yes.

MR. HARRIS: That's what I'm not clear on. Okay.

MS. COOPER: Yep. There we go. I added 2015 there to make that clearer. So ---.

MS. ROSS: Say that again, Larry. Say that again.

MR. HARRIS: Why don't we just adopt all of those? It would be a lot simpler and more probably --- if I understood your presentation, Ross, it looks like even the new ones maybe made it less stringent for carcinogen, but still way below what we can detect, so you know, it's probably ---.

MS. COOPER: If we --- if we propose to adopt all of them as is, we would have --- Angie would have issues with it and the industry would have issues --- might have issues with it. But I know that Angie would. She's made that really clear that if they become less --- if EPA recommends something that's less stringent as they have here and here and here and any of these that are white in the blue column, then we would have opposition to that. So there's potential opposition to whatever we recommend, so that's why we're going through this process to try to at least understand it the best way we possibly can and see if we can come to a consensus on what we --- what we can recommend.

MR. HARRIS: You know, the question that I have then is why did EPA's data become less stringent? Is it just in the last four years due to the influence of ---?

MS. COOPER: So the exercise that we went through on benzo(a)pyrene right now is an example of why some of these criteria have become less stringent, because as you saw with all of that information that we went through today, those data would become less stringent if EPA were to reevaluate

them right now because they've got new data. So that's why any of these would have become less stringent because in the time between when they very first recommended criteria in 2015, in that gap of time, there was studies that came out or new information was gathered that better informed the criteria, and sometimes they became less stringent as the case with benzo(a)pyrene.

MR. BRITTAIN: And usually the reason it became less stringent, Larry, is because they not only had better confidence in the dataset, they had better studies that fit what IRIS was looking more than the older studies, so they were able to remove some of the uncertainty factors. That's usually what inform those differences. They became more certain, so you didn't have to apply a factor of --- uncertainty factor of ten or three or something like that.

MR. MANDIROLA: And the more certain you are that --- you can change the cancer slope. Correct?

MS. COOPER: I mean, there are a lot of uncertainty factors in there, and if any new studies eliminated any uncertainty whatsoever, which

you know there are many ways that they can do that by doing a different study and planning a study in a certain way, then it could eliminate some of that uncertainty and come up with a more accurate number, whether it's more or less stringent.

MR. HARRIS: You know, I don't have a graph to show this, but I think I used to have one that showed over the years from 1900 up to the present the number of new compounds that were giving permits for is increasing and the cancer is increasing at about the same amount, so I think it's one of the things that this group should do is identify every carcinogen and then make sure it's more stringent than the most stringent protection in every carcinogen and then go on to the other things.

MS. COOPER: Well, there are a lot of things that changed between 1900 and now. I mean, just causation doesn't mean --- correlation doesn't necessarily mean causation. For instance, I grill that chicken twice a week on my grill. I might not of had that back in 1900. I don't know. There are a lot of --- a lot of changes that can change, you know ---.

MR. HARRIS: Probably not a good example because I think people cooked over fires for a

long time. But I understand what you're saying.

MS. COOPER: Well, I also eat a lot more Cheetos than I did back then. I don't know what it is.

MR. BRITTAIN: And I can add to that discussion. I've looked at it because I've had similar concerns myself, Larry. I've looked at the data since the beginning of when we really started regulating this stuff, which is in the 1970s. If you look at the cancer rates and deaths by cancer and that kind of stuff from 1975 to the present, the cancer rates increased for the first 20 years through like the mid '90s. Then it leveled off and the cancer rates have actually gone down very slightly, but the cancer rates from --- no, the cancer rates today are slightly higher than they were in 1975. The death by cancer has gone down dramatically, though, because we're better at fighting cancer --- detecting and fighting cancer. And that's one of the issues we don't know. We are actually a lot better at detecting cancer now than we were in 1975.

MR. HARRIS: That's another factor. You're right.

MR. BRITTAIN: Yeah, that plays into

quite a bit. And one of the questions I always have is, like, why haven't we changed that baseline cancer rate because what I want to know is, like, are we having an impact by the amount of remediation that we're doing? It could be that the cancer hasn't changed just because of the fact that human lifestyle, like Laura was talking about, that may trump everything we're doing from a remediation standpoint. You know, that may be the overriding factor. It may be that people are just living longer and you're getting --- you're just --- because you're living longer, you're just going to get cancer because that's the biology. We did not evolve to be able to deal with cancer. Our life span was too short at that particular time.

So there's lots of factors that play into this. We're not going to be able to answer that particular question. But it's a very good question and an important question, Larry. And I'm right with you on it.

MR. HARRIS: Yeah. And you know, I mean, some --- like colorectal cancer is the result of seven separate mutations in these various either suppressor genes or growth control genes, and so the

more contact you have with mutagens, that's going to develop --- now, people quit smoking, so there's probably less lung cancer. Well, no, there's still a lot of lung cancer, but that could make things better by fewer people smoking. Anyway, yeah. Okay.

MR. BRITTAIN: Very complex. We haven't teased out that information yet.

MS. COOPER: All right. I'm going to move on to the next slide which is just looking at our workgroup goals again. We've gone through these each time for several meetings. We've discussed them in detail here. We've discussed them among ourselves at DEP, and we feel like this is the final version of our workgroup goals. We're going to try and develop reasonable standards. We're going to get protective regulations as, you know, we talked about all day here and we're trying to learn and broaden our horizons, which again I said, you know, we're definitely doing that and our ultimate goals is to reach a consensus on what to be able to propose to the secretary in --- I believe it's May to recommend additional criteria revisions for the coming year. So those are our workgroup goals, and after that, we ---.

MS. EMERY: You changed it back to

approvable, though.

MS. COOPER: Yes. Yes, I did.

MR. HARRIS: All right. Well, we oppose.

MS. COOPER: Okay. I'm sorry about that, but that's ---.

MR. HARRIS: I'm also opposed to having that first bullet being one of the goals.

MS. COOPER: Right. Well, one of the --- I mean, we've been through this --- we've been over this discussion a few more --- a few times.

MS. ROSSER: How does this workgroup make decisions? I mean, do we vote on these goals?

MS. COOPER: We're chaired by myself and the group at DEP. Ultimately, it's our decision to propose criteria. The workgroup will be able to come to consensus hopefully to propose to Secretary what to recommend, but no, we don't --- we're not --- we don't plan --- we want to reach a consensus, not have votes because we know that we won't be able to reach a consensus if we are --- if we're all voting on various things. And we've been through these goals several times and had several discussions about them and we feel --- DEP, I mean, we feel that settling on

reasonable standards that are approvable by the legislature and EPA, I mean, it's just an absolute must. We must have standards that will be able to be approved by them because that's the process. We can't change ---.

MR. HARRIS: How do you know what will be approvable?

MS. ROSSER: How do you know what's going to be approvable by the legislature? That should not be a criteria. Again, we oppose and it's disconcerting to put this much time into a workgroup where our input is not ---.

MR. HARRIS: I'm with ---.

MR. MANDIROLA: The one thing I would add is you got to remember these are goals. They're aspirational goals. Nothing would get sent to EPA for approval if we can't get it through the legislator, so any --- I mean, my goal working with water quality standards whenever an update is done is to get something that can be both approved by the legislature and EPA, because if we can't get it through the legislature, it never gets to the EPA. Nothing would ever change.

So I'm not --- I don't think --- folks

may be reading more into that then what our intention is with approvable. I mean, we will never go to EPA or --- with a standard update if we can't get it through the legislator first because that's the process we have to deal with in West Virginia. It has to go through our legislature before we can get it there.

MS. ROSSER: What is your problem with the word --- what is your problem ---?

MR. MANDIROLA: No, it's not --- I mean, this is aspiration.

MS. ROSSER: What's the problem --- what's your problem with the word defensible?

MR. MANDIROLA: I can defend something all day long. That doesn't mean --- that doesn't get it to EPA.

MS. ROSSER: But if it's the science that you think is right and the policy you want to defend, why not stand up for it?

MR. MANDIROLA: Again, that would be fine. We could stand up for it. We could fight all day long, but inevitably it will never get to EPA if it doesn't get through our legislative process first. That's --- I mean, like I said, I don't think --- I

think folks may be reading more into that then what our intention is. Our intention is to get it through the process. That's what we mean by that statement is to get it through the process that we have to deal with. And the process we have to deal is first to get it approved by our legislature, then to get it approved by EPA.

MS. COOPER: That's the reality of what we need to do, and in order to that, we'll have to have standards that will be able to do both of those things.

MR. HARRIS: I just don't --- I guess I just don't get why the legislature would not approve something that was science based and that we --- the DEP, whose very name is environmental protection, has brought the standard to them. They should just accept that.

MS. HENTHRON: This is Jennie. And I've tried to remain silent on this issue, but you just said you wanted to do something that was supported by science and Angie had said she doesn't want to lower any of the --- or increase any of the criteria even if the science supports that. So that's, I think, the reason that we're at an impasse

here is for that reason, because science may say one thing, but some of the members of this group may not be able to support that based on their position. So I'm just hearing DEP trying to recognize that dichotomy with this.

MS. COOPER: And having this group is us being as transparent as we possibly can be and allowing every concerned party to have input on this process. So we're doing --- we're going --- we're going pretty out there on this limb of transparency. We want to involve you guys in every possible way in this. And, in fact, our goal is to reach a consensus in this group, and we sincerely hope to be able to do that.

MS. HENTHRON: This is just a way to maybe try to get this off of dead center by having a discussion on something, and I don't know if it'll work, but I've just been thinking about it. You know, one of the things that the Manufacturers Association has said in these discussions is for the IRIS component, the cancer slope factor, we would advocate the use of whatever the current cancer slope factor is from IRIS. And that would mean for that particular example that is done here, we would advocate the use

of the new number for benzo(a)pyrene. Some of these have gotten lower over the years and we didn't quarrel with those, so would that be something that we could try to reach consensus on, that that component of the calculation should be the IRIS recommended cancer slope factor?

And maybe those would be ways that we could work towards reaching consensus. Instead of looking at it number by number, maybe try to figure out where we have differences and resolve that.

MR. BRITTAIN: This is Ross. I'll also state that there should be --- not all chemicals are carcinogenic, so it should also be the IRIS reference dose. It's either one depending on what the chemical is that operates by non-cancerous and cancerous type of toxicity. And the only other question --- or concern I have there --- and I can't remember this off the top of my head is IRIS does not have RFDs or cancer slope factors for every single compound, right. That's important to know because they --- it takes --- because they move at less than glacial speed. And so there may --- and I can't remember if all --- if they have that toxicity data on all of the compounds that we have water quality

standards for or not. That's something we'd have to review real quick.

And then the other question is if IRIS doesn't have one, like, in my particular silo, we have the next level down, which is the provisionally peer review toxicity values, PPRTV, or we can go to tier 3, which is places like the California EPA or ATSTR (phonetic) as alternate sources if there is not an IRIS guide. And we must use those alternate sources when IRIS does not have the value. So that's another consideration if there are any that IRIS doesn't have any to use as well.

Not to throw a monkey wrench into it. Personally, I agree, it should be based on science. And we should be using everything IRIS has. But IRIS doesn't always have everything depending on the comment.

MS. ROSSER: Think for the ones with human health criteria --- and that's why I did say carcinogens. I do think that all of the cancer slope factors were from IRIS. That's why I did that one, that merited. I couldn't remember, Ross, on reference doses whether that was accurate or not.

MS. COOPER: Yeah. And I agree that I

--- I do believe that we ought to use the latest IRIS numbers, and even in the case of the PAHs, if it's going to make the criteria slightly larger, that's the latest science. That's what water quality standards are supposed to be, the latest science that are --- that best protect --- that is able to protect the use. So I mean, I would recommend that we do that. Would we be able to have consensus on that?

MS. MCPHAIL: I guess so. I know I'm in a different place on this. This is Rebecca.

MS. COOPER: Yeah, so that's something we'll talk about as we move forward, too. And I think it's --- we're just about at noon now. So let's move on and just quickly plan when the January meeting will be.

As I mentioned I've asked Jennie to present at that meeting regarding the PAHs. So especially --- well, I would like to make sure everybody can be there, but tentatively I have it as January 27th on a Wednesday, towards the end of January. So if we don't have any objection to that date, then I'll send out a meeting invite for that later today. Do we have a ---?

MR. BRITTAIN: Laura, I can't attend

that day. I don't know how much you need me, but I can't attend that day. I have a DLR retreat that day.

MS. COOPER: Okay. So we'll look at that and see what we can put together. Do we have anything else before we conclude today?

All right. Thank you all for being here I really appreciate your involvement. Thank you, Ross, for presenting to us. That was really helpful. I can't wait to watch the video again so I can learn it all over again. And you all take care and have a happy holiday.

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HEARING CONCLUDED AT 12:00 P.M.

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CERTIFICATE

I hereby certify, as the stenographic reporter, that the foregoing proceedings were taken stenographically by me, and thereafter reduced to typewriting by me or under my direction; and that this transcript is a true and accurate record to the best of my ability.

I certify that the attached transcript meets the requirements set forth within article twenty-seven, chapter forty-seven of the West Virginia Code.

Bailey Kane,

Court Reporter