ILLINOIS POLLUTION CONTROL BOARD

IN THE MATTER OF:	)
	)
WATER QUALITY STANDARDS AND	) R08-9
EFFLUENT LIMITATIONS FOR THE	) (Rulemaking-Water)
CHICAGO AREA WATERWAY SYSTEM	)
AND LOWER DES PLAINES RIVER	)
PROPOSED AMENDMENTS TO 35 ILL.	)
ADM. CODE 301, 302, 303 and 304	)

TRANSCRIPT OF PROCEEDINGS had in the above-entitled cause on the 9th day of September, A.D. 2008, scheduled to commence at 1:20 p.m.

BEFORE: MARIA E. TIPSORD, HEARING OFFICER,
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THE HEARING OFFICER: Back on the record.

MS. ALEXANDER: I wanted to start with Gerba prefiled Question No. 3, which is referring to Page 2 of your testimony that pseudomonas was selected for study, in part, because it, quote, "Causes recreational associated, eye, skin and ear infection." And then on Page 3, the adenoviruses are a cause of ear, nose, throat and respiratory infections. Just.

To clarify, the risk assessment did not calculate quantitatively risks associated with these types of infections; is that correct?

MR. GERBA: For these two organisms, it was done qualitatively.

THE HEARING OFFICER: You have to remember to speak up.

DR. GERBA: For these two organisms, it was done qualitatively.

MS. ALEXANDER: And I also wanted to reference -- now, we were on the attachments to the May 23rd, letter from Geosyntec,

second attachment Page 3.

THE HEARING OFFICER: Excuse me, Ms. Alexander, this will be a new transcript, so that's Exhibit 73.

MS. ALEXANDER: Sorry.

Exhibit 73, the portion of that which is the letter from Geosyntec dated May 23rd, 2008. Page 3 of the second attachment, there is a statement made in the first paragraph, "Ear and eye infections associated with contact by pseudomonas contaminated water are typically associated with full immersion activities. Since these types of activities are not permitted as designated uses of the CAWS, the incidents of ear and eye exposures are expected to be low and as a result of an accidental or intnetional misuse of the waterway."

I direct this question to Dr. Petropoulou. Would I be correct in understanding that if, in fact, you fell into the water, you would be at risk of this from the pseudomonas?

MR. ANDES: Can I just -- can you

repeat the page?

MS. ALEXANDER: I'm sorry. Page 3 of the second attachment, which is entitled Review Conducted by USEPA Office of Research and Development, which is, in fact, I believe is the Tim Wade document, I identified earlier in response to that.

THE HEARING OFFICER: And that's -- what's the date on the cover letter to that?

MS. ALEXANDER: The cover letter is dated May 23rd, 2008.

DR. TOLSON: It starts with a clarification. It starts on Page 2 and it continues on Page 3.

MS. ALEXANDER: Yes. I'm sorry. I was reading from Page 3.

But the point be responded to is on Page 2. "Since pseudomonas and adenovirus were found, descriptions of non-GI illness should also be provided, to represent a clear picture of the actual risk associated with recreating in the CAWS." That's the statement being responded to.

The response begins on Page 2. I

read a portion of that response from Page 3 concerning non-GI gastrointestinal infection associated with pseudomonas.

And my question was, am I correct in understanding that this means you would be at risk of non-GI illness from pseudomonas if you actually fell into the water accidently --

DR. GERBA: Are you asking me that question?

MS. ALEXANDER: I was actually directing it, initially, to Dr. Petropoulou, because I believe she testified that she had drafted that text.

DR. PETROPOULOU: I did. With input from Dr. Tolson.

So I would defer that question to Dr. Tolson.

MS. ALEXANDER: Okay. Go ahead, either one of you.

DR. TOLSON: I won't defer it one more time. I'll go ahead and take it.

Pseudomonas can cause folliculitis. It typically requires a

pre-existing cut. So it's a very difficult thing to sort of estimate what the population pre-existing cuts would be to do that.

So there is a potential for folliculitis from pseudomonas. We have not calculated that directly within here, however, the report does go into some information on pseudomonas. Maybe we should refer to that proportion --

MS. ALEXANDER: I'm going to get there.

DR. TOLSON: Okay.

MS. ALEXANDER: I'm sorry, I didn't mean to cut you off. Were you going to continue to that --

DR. TOLSON: I was going to discuss pseudomonas and the relationship with pseudomonas between dry and wet and such. But we can -- if we can postpone that, that's fine.

MS. ALEXANDER: Yeah, I want to allow you to do that, but I actually had a few questions about it. Perhaps we can address it in that context.

Because I just wanted to keep the thread here, which is, you mentioned folliculitis. Is that a skin infection?

DR. GERBA: Excuse me. I'm more familiar with folliculitis because I've studied it.

MS. ALEXANDER: Go ahead.

DR. GERBA: Actually, it's usually infections around your hair follicles. That's where it gets the name folliculitis.

It's most commonly associated with hot tubs, particularly in the bathing suit area and around the buttocks. And that's where you most commonly see getting folliculitis.

Almost all the cases in the United States are associated with hot tubs or other type of artificial waters, generally, in the United States. Although, cases have been associated with swimming in natural waters.

And there's one study in Europe that showed a relationship between getting pseudomonas infections and swimming in lake

water.

MS. ALEXANDER: There's also a reference to eye and ear exposures, here and in the language I just quoted and in your testimony.

Are those eye and ear infections also folliculitis, or are those a different type of infection?

DR. GERBA: Those are different types of infection.

MS. ALEXANDER: Okay. Just one second. Bear with me.

All right. Now, regarding pseudomonas, I have in front of me the discussion -- at least one of the discussions of it in the risk assessment on Page 129.

THE HEARING OFFICER: One twenty-nine of --

MS. ALEXANDER: One twenty-nine of the risk assessment document, which I believe is marked as Exhibit 72.

THE HEARING OFFICER: Okay. Wait. Seventy-two is what you gave us, the Tim Wade documents.

MS. ALEXANDER: Oh, I'm sorry.

THE HEARING OFFICER: The Geosyntec report is 71.

MS. ALEXANDER: This (indicating) is 71. Okay. I apologize.

Looking at Page 129, just to read your language, it states, "Perhaps, more importantly, the outfall samples show low levels of pseudomonas -- I'm sorry, lower level pseudomonas in the corresponding wet weather samples. This suggests that the major input for pseudomonas in the waterways are sources other than the WRP effluent.

"Therefore, this infection of the WRP effluent would have minor effects on the overall loading of pseudomonas in the waterway and risks associated recreational exposure to the pathogen."

Would I be correct in summarizing that the basis for your conclusion that pseudomonas was not a risk is this the statement that wet weather levels are higher than dryer weather levels?

DR. TOLSON: I'm sorry the statement

that pseudomonas is not a risk?

MS. ALEXANDER: Basically, you have the statement -- you conclude, "Therefore, disinfection of the effluence would have minor effects on overall loading of pseudomonas." Let me ask the question more broadly.

Do you have any other basis for concluding that there was no -- that there is no significant risk from eye, skin and ear infections associated with pseudomonas?

MR. ANDES: Other than what?

MS. ALEXANDER: Other than the language I just read, stating that lower levels of pseudomonas in the dry weather samples.

I mean, is there any other basis for concluding that these types of infections from pseudomonas are not a significant risk?

DR. TOLSON: I think -- we did not quantify the exact level of pseudomonas risks -- I'm sorry, the risk, for ear or eye or skin infections. However, if you look at Page -- Table 515 in Exhibit 71, I believe,

which is the report, it lists the pseudomonas levels in the outfall effluent and also in wet weather samples.

So, for example, in the north side channel, the outfall had 1,350 CFUs per ML -- is that correct? Thirteen hundred fifty units, where the wet weather -- in other words, in the channel, had 5,427.

So it's hard to explain how the effluent disinfection would have affected something the receiving body that was, you know, a four times higher concentration. And that's the basis of my conclusion that disinfection wouldn't change the wet weather concentrations that we see.

Is that --

MS. ALEXANDER: Did you reach any conclusions as to whether it would change the dry weather concentrations?

MR. ANDES: I didn't -- whether what would change?

MS. ALEXANDER: Weather disinfection would change the dry weather concentration of pseudomonas?

DR. TOLSON: Pseudomonas is a little bit different than the other pathogens, because they have such varied sources. The concentrations of pseudomonas out of the effluent we would expect it to decrease probably quite dramatically by most of the disinfection technologies.

Our pseudomonas is one of the pathogens that comes from a lot of our sources. We talked within this testimony yesterday about trees and bushes that are nearby the shoreline, those are a significant source of pseudomonas into the waterway.

So unlike other pathogens, pseudomonas is more -- is greatly affected by these sort of nonpoint discharges or sources into the waterway.

MS. ALEXANDER: Just to be clear, however, these nonpoint discharges are wet weather discharges; is that correct?

DR. TOLSON: They can be -- for pseudomonas, they can be both wet and dry.

MS. ALEXANDER: Would you say that they are substantially or predominantly wet

weather?

DR. TOLSON: If you want pure speculation, I'd say yes.

MS. ALEXANDER: Okay.

And I get back to my question, did you specifically do any analysis of the impact of disinfection on dry weather pseudomonas levels in the waterway?

DR. TOLSON: Yes, we did. We did evaluate the dry weather days -- we did sample the waterway under dry weather conditions, and we did investigate the effectiveness of disinfection techniques on pseudomonas.

And from that conclusion, it would suggest that the effluent could be decreased quite dramatically by disinfection techniques.

MS. ALEXANDER: Okay.

Now, what -- the reason I highlighted that text on Page 129 is I was, frankly, having a little bit of difficulty finding the discussion that you have characterized elsewhere as a qualitative as

opposed to quantitative assessment of these impacts of pseudomonas. And by these impacts, I'm referring to the possible ear, eye and skin infections, there are reference to qualitative as opposed to quantitative assessment of that.

I was hoping that someone could point me to any text other than what I've highlighted on Page 129 that embodies that qualitative risk assessment of those types of illnesses.

DR. TOLSON: Well, the qualitative assessment is the comparison of pathogen concentrations under what way wet weather versus dry weather versus the outflow. So we don't know what that risk is, but we know we can measure the concentrations of wet, dry and outfall and get a sense of whether the contributions to that risk, the magnitude of that risk, who is responsible for it, what the discharges are that are responsible for it.

MS. ALEXANDER: Okay. So when you reference --

DR. TOLSON: Let me follow up --

MS. ALEXANDER: I'm sorry, I didn't mean to interrupt.

DR. TOLSON: Let me follow up with one more point.

The dose response data available to actually do that quantitative assessment is not available. There's also very scant information in the scientific literature about the concentrations in hot tubs that were responsible for outbreaks.

And I think Dr. Gerba might be able to speak to that.

DR. GERBA: Most all of the outbreaks of folliculitis are due to hot tubs, almost entirely, where there's high concentration. But nobody has ever quantified it, so you couldn't really do a risk assessment on it.

Ear infections do take immersion, I think I have to point out to when it's correlated with lake water. And eye infections, the only ones I've seen have usually been immersion, related to recreational activities.

MS. ALEXANDER: Okay. I just -- that's helpful, and I want to summarize to make sure I understand.

When you say that you've done a qualitative risk analysis of pseudomonas, what you've done, essentially, is look at the differing levels under different conditions and the impact of disinfection on the levels; is that correct?

DR. TOLSON: That is correct. We have been able to assess between wet weather versus dry weather and what are our anticipated effects would be of disinfection, absolutely.

MS. ALEXANDER: But no actual analysis of the probabilistic likelihood that anyone is going to get an ear, eye or skin infection?

DR. TOLSON: We do not have the data available, nor does anyone else, to sort of do that quantitatively.

MS. ALEXANDER: Are any of you familiar with research by lead author Fewtrell, et al., in 1992, finding that at

one of the contaminated sites studied, the relative risk of respiratory symptoms among exposed individuals was actually higher than the risk of GI symptoms?

DR. TOLSON: I'm familiar with a number of his papers, if you let me see that one?

MS. ALEXANDER: Okay. I do have something in my hand.

And I apologize that I just discovered this morning that this is actually an incomplete copy. I am happy to provide a complete copy as soon as I get my hands on it.

This, however, does have a reference to the conclusion that I'm discussing here.

MR. ANDES: Is that referenced in the questions?

MS. ALEXANDER: No, this is a follow-up to this whole discussion.

THE HEARING OFFICER: I've been handed a two-page document entitled Health Effects of White Water Canoeing, by L. Fewtrell,

F-E-W-T-R-E-L-L, et al. And it's dated June 27th, 1992 from the Lancet, L-A-N-C-E-T.

I'll mark this as Exhibit 74, if there's no objection?

Seeing none, then it's Exhibit 74.

(WHEREUPON, a certain document was marked Exhibit No. 74 for identification, as of 9/9/08.)

THE HEARING OFFICER: And Ms. Alexander, when you get a complete copy we will mark that.

MS. ALEXANDER: Okay.

And my initial question, I think, is pending to all three of you, whether any of you have seen this or are familiar with it.

DR. TOLSON: I believe I've seen it before, yes.

MS. ALEXANDER: Okay. Anybody else? Dr. Gerba?

DR. PETROPOULOU: I haven't.

MS. ALEXANDER: I'm sorry, I couldn't hear Dr. Gerba.

DR. PETROPOULOU: I haven't.

MS. ALEXANDER: I heard yours, I didn't hear Dr. Gerba.

DR. GERBA: Yes, I believe I have.

MS. ALEXANDER: You've seen it, okay.

MR. ANDES: I can ask -- actually, I think we have another witness who is very familiar with it, who -- in fact, we were going to produce this later anyway. So I don't know if you want to --

MS. ALEXANDER: Yes, I understand it was referenced in Dr. Dora Vitch's report. I just wanted to get a brief reaction from these witnesses.

Do you have any reason to doubt the accuracy of the report of the data and conclusions, in this document?

DR. TOLSON: I'm not really able to comment on that at this point. One, I haven't gone through this in great detail, and so...

MS. ALEXANDER: Understood. I just wanted to see if you had any --

DR. TOLSON: Sorry.

MS. ALEXANDER: -- immediate reaction.

Anyone else?

DR. GERBA: Yes, the thing that struck me was the very concentrations of viruses per ten liters.

MS. ALEXANDER: Very high concentrations of --

DR. GERBA: About 200 per ten liters.

MS. ALEXANDER: Two hundred what per -- I can't quite hear you.

DR. TOLSON: One hundred ninety-eight per ten liters.

THE HEARING OFFICER: Viruses, I believe he said.

DR. GERBA: One hundred ninety-eight viruses per ten liters.

MS. ALEXANDER: I also see a reference to 285 fecal colony forming units per deciliter, I believe.

Can someone do some quick math on that and translate that into what that would be per -- I think we're usually using colony forming units per 100 millimeters. That's the same thing; correct, per deciliter? Okay. Sorry.

Okay. So that 285 number for 100 milliliters, would it be fair to say that that number is lower than the fecal coliform levels generally measured in dry weather near the outfalls in the CAWS?

DR. TOLSON: I have not -- unfortunately, I don't have any reading glasses, so I can't read this at all.

As it's been characterized, it would seem, that the concentrations in the waterways that are represented by this study are very different than the concentrations that we've seen in the CAWS. That the indicator organisms are very low in this study.

The indicator organisms are very low, the pathogen organisms are very high. Compared to the CAWS, where the indicator organisms are very high and the pathogenic organisms are very low. That's probably a significant sort of input to the conclusions that they've drawn here.

The other thing that's striking is that, you know, this is a white water

canoeing. And I believe that they actually took discharge from a water treatment plant to increase the flow of a river where they had this event, if I'm characterizing it -- if I recall it correctly.

And that's more of a primary contact activity than what we have.

MS. ALEXANDER: Do you have any reason to believe that the contribution from the wastewater treatment plants of this situation would have been higher than 70 percent, as it is in the CAWS?

DR. TOLSON: Say again?

MS. ALEXANDER: Do you have any reason to believe that the percentage contribution of wastewater treatment plant effluent in this waterway was higher than 70 percent, which is the percent in the CAWS?

DR. TOLSON: I'm sorry, I'm not familiar with this study well enough from memory.

MS. ALEXANDER: I just asked if you had any reason to believe. If the answer is no, that's fine.

DR. TOLSON: No.

MS. ALEXANDER: All right. Moving on from this.

Can meningitis be caused by water born pathogens?

DR. GERBA: Yes.

MS. ALEXANDER: Can meningitis be caused by water born pathogens?

DR. TOLSON: Dr. Gerba can --

DR. GERBA: Yes.

MS. ALEXANDER: Can myocarditis?

DR. GERBA: Yes.

MS. ALEXANDER: Can encephalitis?

DR. GERBA: Yes.

MS. ALEXANDER: Okay.

And none of those, of course, are GI illnesses; correct?

DR. TOLSON: Beg your pardon?

MS. ALEXANDER: That none of those are gastrointestinal, they're all different kinds; correct?

DR. GERBA: Yes.

DR. TOLSON: I'd also like to point out that those are reportable illnesses. So

we could pole the county health records and see if there were any occurrences of those.

MS. ALEXANDER: Right.

But you did not study risks of those types of infections in the risk assessment; is that correct?

DR. GERBA: Well, we used -- again, we use infection as the limit, which could be taken into that. In other words, that's an endpoint of infection.

Your conservative things that estimate you risk by infection is what we did. That's an outcome of infection.

MS. ALEXANDER: But, in fact, only studied risk of gastrointestinal illness; is that correct?

DR. GERBA: That's right. Because, currently, that's how the U.S. Environmental Protection Agency regulates recreational waters.

MS. ALEXANDER: Bear with me one more second while I find a page number.

I apologize for the interlude. Not all the pages are -- hard to find things.

All right. I want to refer to the language on Page 95 of Exhibit 71. I got it right in time, the risk assessment.

And that language is, "Since there is a certain degree of correlation between different pathogens, indications of unacceptable levels of gastrointestinal illness may indicate a potential for other effects."

My first question is, have you quantified that correlation between GI illness and other unacceptable -- I'm sorry -- in other effects, I should say?

MR. ANDES: What page did you say that's on, I'm sorry?

MS. ALEXANDER: This is on Page 95.

DR. TOLSON: We have not undertaken that as a component of the study.

MS. ALEXANDER: Okay. And again, you state that this correlation may exist. I take it you haven't quantified the probability of such a correlation?

DR. TOLSON: No, we have not.

MS. ALEXANDER: Do you know any other

researchers that have?

DR. TOLSON: I'll refer to Dr. Gerba. I do not personally.

DR. GERBA: Say again.

MS. ALEXANDER: A quantification of the probability of the correlation between GI illness and other effects, the language used here.

DR. GERBA: You mean, in other words, the probability you have a GI was the probability of having another outcome of that?

MS. ALEXANDER: Yes. In other words, these other -- I assume it's referring to what's referred to also in the core, which is the ear, skin, eye infections that can result.

DR. GERBA: From recreational contact?

MS. ALEXANDER: Well, I mean, let me expand the question for any. Because I -- what I want to know is whether anybody that you know of has done research to quantify the probability of that correlation?

DR. GERBA: Going from GI to like,

say, meningitis?

MS. ALEXANDER: Yeah, any of these other possible effects of water born pathogens.

DR. GERBA: Not offhand, no, I can't --

MS. ALEXANDER: Okay.

DR. TOLSON: If I can add something, though.

You know, EPA's bases GI illness -- or uses GI illness for their setting acceptable limits. So I think implicit within that is the understanding that GI is the most sensitive and would be correlated to all illnesses.

MS. WILLIAMS: Is there anywhere that you can point us to where they say that?

DR. GERBA: I'm not sure what the question revolves about.

MS. WILLIAMS: Okay.

DR. TOLSON: Off the top of my head I do not know. But that is something that's potentially out there.

They had to come up with a

rationale for using GI illness, which --

MS. WILLIAMS: But you don't know what it is?

DR. TOLSON: I do not know what it is.

MS. ALEXANDER: Okay.

DR. TOLSON: And my guess is that they specified it somewhere.

MR. ANDES: I'll follow up on that.

So your understanding is the way EPA sets these standards, the sense is, if you address GI illness, you're addressing the other issues?

DR. TOLSON: That is correct.

MR. ANDES: Thank you.

MS. ALEXANDER: But you're not offering anything substantive right now to support that assumption?

DR. GERBA: I was just -- my thought was that a lot of times gastrointestinal -- I mean, we have respiratory, we have intestinal infections, also. So you can have both by the same agent is the only thought I had on that.

So in some ways that might be

covered, because you can have both diarrhea and respiratory illness from the same agent at the same time.

MS. ALEXANDER: I understand that it's possible to get really sick from multiple things at the same time. But I guess what I'm asking is whether you know of any quantification of the likelihood of that correlation.

DR. GERBA: I've answered that.

MS. ALEXANDER: And it sounds to me like the answer was no.

Let me ask another question along those lines. Is it possible in your review that there could be circumstances in which recreators would be at risk of contracting nongastrointestinal illnesses, even if they were not at significant risk of a GI illness?

DR. GERBA: There are so many caveats to that.

DR. TOLSON: There are a lot of caveats to that. There are potentials, obviously, of getting a respiratory or an ear infection and not getting GI illnesses,

that's what you're after, sure.

MS. ALEXANDER: Okay.

MR. ANDES: Can you expand on that?

DR. TOLSON: While there is that potential, we believe the predominant illness from recreational exposure to the CAWS is GI illness.

MS. ALEXANDER: I understand that's your --

DR. TOLSON: Okay.

MS. ALEXANDER: -- viewpoint.

Let me ask -- this has drawn -- sorry, I didn't mark which prefiled question this was. But I'll ask it anyway.

Approximately how many types of water born human pathogens are known to be associated with sewage overall? Just an approximation.

DR. GERBA: The number of different types?

MS. ALEXANDER: Yeah.

DR. GERBA: I'd say between 160 and 200.

MS. ALEXANDER: Okay.

Did any or all of you review the list of water born pathogens that accompanied Dr. Mary Lynn Yates' testimony submitted in this matter.

DR. GERBA: She didn't give a specific list. But she did say there were thousands, I think.

MS. ALEXANDER: There was an attached list, which I'm happy -- I mean, we're going to be marking Dr. Mary Lynn Yates' testimony. But for ease of reference, I can just have the list marked separately, if that's all right.

I do not recall, unfortunately, which document this was to the testimony, but I will represent that it was an exhibit, which I'm giving you for reference.

THE HEARING OFFICER: I've been handed Table 1, Illness Acquired By Ingestion of Water.

MS. WILLIAMS: I think it's Exhibit 6 to the testimony.

MS. ALEXANDER: Thank you.

THE HEARING OFFICER: And I believe

the title of the book is Water Born Transmissions of Infectious Agents, that's at the top.

I'm going to mark this as Exhibit 75. If there's no objection?

Seeing none, it's Exhibit 75.

(WHEREUPON, a certain document was marked Exhibit No. 75 for identification, as of 9/9/08.)

DR. GERBA: Can I make a comment right away?

This has to do with recreational water only. Many of these organisms are not -- do not occur in sewage and are not transmitted by that route.

They are -- many of these organisms are what we call water based pathogens, those that grow naturally in water. Did you want me to comment otherwise on this?

I -- again I don't see thousands of organisms listed here.

MS. ALEXANDER: Yeah. Let me just clarify your comment.

You say that they are not recreationally associated. Is that what I heard you say?

DR. GERBA: No, I said they're not sewage associated.

MS. ALEXANDER: They're not sewage associated.

But some of them are sewage associated; correct?

DR. GERBA: Oh, yes.

MS. ALEXANDER: Yes, I understand there are a few of these that are not sewage associated.

My only question to you was, with that comment in mind that you made that, you know, we all -- Dr. Yates also recognized in her testimony that not all of these are necessarily sewage related. Do you have any reason to doubt the overall accuracy of this list as your representation of human water born pathogens?

DR. GERBA: This is transmitted by recreational waters?

MS. ALEXANDER: I'm sorry?

DR. GERBA: Transmitted by recreational waters? That's what the table says.

MS. ALEXANDER: Yes, potentially transmitted by recreation, sorry.

DR. GERBA: And that's only the latter part of that. Yes.

Again, I don't see thousands of organisms listed here.

MS. ALEXANDER: Right. Okay.

So that's a subset of them.

But the only question would be is it a longer list, is it not, than the list of pathogenic organisms that were included in the risk assessment analysis? Is that correct?

MR. ANDES: Well, wait. Do we have to count up whether there's 160 to 200 here? He just testified there's 160 to 200 types.

MS. ALEXANDER: I'm sorry, say that again? You have to --

MR. ANDES: He just testified there were 160 to 200 types of pathogens.

MS. ALEXANDER: Right.

MR. ANDES: So you're questioning -- he has to count these and decide if there are that many?

MS. ALEXANDER: No, that wasn't really my question. It was more general than that.

MR. ANDES: Okay.

MS. ALEXANDER: I mean, I -- in the risk assessment you studied approximately eight, give or take; is that correct?

DR. GERBA: That's correct.

MS. ALEXANDER: And there are more listed here that are associated with recreational water use; is that correct?

DR. GERBA: That's correct.

MS. ALEXANDER: Okay. That's all I'm getting at, sorry.

DR. GERBA: But I would point out most of these are not transmitted by sewage. Of the recreational ones, that you have in recreational.

MR. ANDES: I'd like to follow up on that.

Dr. Gerba, is it accurate to say that the eight, according to your testimony,

that were chosen, were chosen to be representative of what the basic risks are from --

DR. GERBA: Sewage contaminated water. We collected the water organisms because they occurred in sewage and had the potential to be transmitted by that route.

We also selected to represent what we figured would be the ones most commonly present, ones that could be detected by methods currently available, because methods weren't available for all of these. And the ones would be there in the greatest concentration.

So they would present the greatest risk based on knowledge of dose response and the occurrence of waste water.

MR. ANDES: Thank you.

MS. ALEXANDER: And the longer list concerns illnesses acquired by ingestion of water; correct?

DR. GERBA: That's right.

MS. ALEXANDER: Okay.

And one of the exposure pathways

that you considered in your risk assessment was ingestion; correct?

DR. GERBA: That's correct.

MS. ALEXANDER: And a number of those are, in fact, transmitted fecally orally; is that correct?

DR. GERBA: That's correct.

MS. ALEXANDER: Okay.

So in other words, the list of pathogens from which one might be at risk if one fell in the water and gulped a mouthful might, in fact, be longer than, specifically, the list identified as acquired by recreational contact with water; is that correct?

DR. GERBA: That's correct.

MS. ALEXANDER: Okay.

Now, I'll address this initially to Dr. Petropoulou, it's Question No. 2 on the Petropoulou prefiled questions. And the others, if you can chime in afterwards, it's also Tolson No. 6 and Gerba No. 15.

But specifically for Dr. Petropoulou, regarding the statement at

Page 4 of your testimony in which you identify the two bases for selecting a limited subset of pathogens that you studied, these eight give or take that I referred to a moment ago. And those two bases were, one, the existence of past outbreaks caused by these viruses, and, secondly, the existence of USEPA approved SOPs for those pathogens.

Is that an accurate characterization?

DR. PETROPOULOU: Correct.

MS. ALEXANDER: Okay.

In your view, are outbreaks an accurate indicator of the actual risk of a particular pathogen?

DR. PETROPOULOU: I'll defer the first question to Dr. Gerba.

MS. ALEXANDER: Okay.

DR. GERBA: Yeah, outbreaks are one indication. But a pathogen can be transmitted by a specific route.

MS. ALEXANDER: They're one indication. But is it possible for there to be risk of a type of pathogen and no record

of outbreaks from that pathogen?

DR. GERBA: By a water route, say, or -- yes.

MS. ALEXANDER: Sure, by water route.

DR. GERBA: Yes.

MS. ALEXANDER: One second.

Now, I'd like to refer again to the second attachment to that May 23rd, 2008 letter, which was a component of Exhibit 73, Page 7 of that.

THE HEARING OFFICER: For clarification, you refer to the May 23rd letter, and it may be a copying error again, but we have the May 23rd letter attached to the May 28th letter.

MS. ALEXANDER: Yes, I'm sorry. I've been referring to May -- that is the May 23rd letter.

THE HEARING OFFICER: Okay.

MS. ALEXANDER: And this is Page 7 of the document headed Review Conducted by USEPA Office of Research and Development.

THE HEARING OFFICER: Page 7?

MS. ALEXANDER: Yes. Are you there?

Okay.

I'm referring to the statement at the bottom, that quoted language from the Office of Research and Development document. It's cited as Page 2.

They are quoting language in the document regarding, quote, "No outbreaks tradable to treated waste water." And then, the comment being responded to:

"The statement is misleading, because outbreaks are not a reliable health indicator due to problems with consistent and reliable detection. Furthermore, statements such as these require citations and peer reviewed literature, other outside sources to avoid the perception of bias."

Now, their response provided is a citation to what purports to be peer reviewed literature. Let me first ask the question, is that document cited peer reviewed, to your knowledge?

DR. TOLSON: I think that's beyond our --

MR. ANDES: Let me clarify.

Directly under that citation it points out that the statement the EPA commented on was removed from the final report.

MS. ALEXANDER: I understand that. But the subject matter is still relevant, regardless whether the statement is in the report.

MR. ANDES: But we don't have the particular statement at issue here in the final report. So you're asking them about the statements in their testimony, not the statement in the draft report.

We don't know what that statement was.

MS. ALEXANDER: Understood. But the subject matter is exactly the same, which is whether outbreaks are or are not a reliable indicator of risk.

And what I am trying to find out is the type of discussion that has taken place with EPA about that. Because EPA here has expressed a concern, and it's not obvious to me how that concern was responded to or if

it was responded to. And that's what I'm getting at with these questions.

MR. ANDES: But I guess the question that they're testifying is not whether outbreaks are a reliable health indicator, it's whether they're relevant to the choice of which particular parameters to look at in doing a study. So if you want to ask him questions about that, that's relevant, but this statement is to a different issue.

MS. ALEXANDER: It's the same issue in the sense of what does it matter whether there's been an outbreak or not. And I want to find out what kind of discussion there was with the USEPA on that topic.

And I understand that the parameters of the discussion may have changed a little, but the fact of the matter is there is extensive reference, both in the testimony and in the report itself, to the significance of outbreaks. And I want to know what the conversations were with EPA about that, and whether their concerns in any context were responded to.

DR. TOLSON: I believe the answer to your question is that, you know, we used outbreaks to identify important parameters, important pathogens to carry through the assessment.

MS. ALEXANDER: I get that.

DR. TOLSON: And that's totally it.

MS. ALEXANDER: And EPA made a comment to the effect that outbreaks are effectively of minimal significance. And then a response was provided by Dr. Petropoulou, perhaps in reliance on others, but I'm trying to understand that response.

Even though I know that particular statement that prompted the USEPA's concern is not in the record, the issue is still very much a part of this -- a part of the report and a part of discussion.

So let me return to my question, which is, there is a citation here to an MWRD paper. And my question -- I'll direct it to Dr. Petropoulou, since she drafted this -- is, was that or was that not a peer reviewed document?

DR. PETROPOULOU: Which document?

MS. ALEXANDER: I'm referring to now on Page 8, the -- on Page 8, your response says, "The report includes the following citation for the statements made." And then it cites a document.

And my question is, is that document peer reviewed? Because the question asked what about peer review.

DR. PETROPOULOU: I do not know.

MS. ALEXANDER: Okay.

MR. ANDES: I'd like to follow up.

The question for any of you is, in terms of the choice of parameters, the parameters that you chose based partly on the existence of outbreaks, did EPA finally go along with the choice of parameters?

DR. GERBA: Yes. I've been on EPA advisory committees for years in trying to get them to actually do more than just outbreaks. But EPA's position has always been the drinking water.

There hasn't been an outbreak, why are we trying to regulate it. In general,

it's the opposite thing.

What we're really finding most of the time is that they want an outbreak. And the reason you use an outbreak is because we know it can be transmitted by that route.

And there's a great deal of uncertainty whether other illnesses can be transmitted by that route if you don't have a documentation of an outbreak. That's the issue.

So it's usually the opposite problem we have with the EPA. That's why I don't really see -- usually the EPA is telling us why are you studying it if there hasn't been an outbreak.

MR. ANDES: Thank you.

MS. ALEXANDER: Beyond what's in this document, did you have any further conversations, any of you, in this meeting or otherwise, with EPA concerning the significance of outbreaks as an indicator of risk?

MR. ANDES: Ever?

MS. ALEXANDER: In the context of this

risk assessment, I should say, not ever, ever.

DR. PETROPOULOU: I have not.

MS. ALEXANDER: Okay.

DR. TOLSON: Nor I.

MS. ALEXANDER: Okay.

DR. TOLSON: I have not had any contacts with anyone.

MS. ALEXANDER: All right.

Is it possible, in your view, that a substantial number of outbreaks go undetected?

DR. GERBA: Yes.

MS. ALEXANDER: Okay.

Is it possible that pathogens that are more frequently asymptomatic, as in people aren't actually getting sick they're just getting infected, would be less likely to result in outbreaks that are actually traceable to recreation, because the people with the symptoms would not necessarily be the same people who recreated on the water?

DR. TOLSON: I don't think we have the data to really speculate on that. So I can't

address that.

MS. ALEXANDER: Dr. Gerba, can you?

DR. GERBA: It's too much speculation, I think, to tell you what the impact would be.

MS. ALEXANDER: Let me see if I can clarify my question just a little, because I think it may have sounded more speculative than it is.

Let's hypothesize a type of illness where only half the people actually exhibit symptoms. If those people don't get sick themselves but pass it onto their friends and their friends all get sick, it's going to be harder to trace those friends' illnesses to recreation on the water body than it is to trace the recreators' illnesses; is that correct?

DR. TOLSON: Again, that is still fairly speculative. But let me address the point that I think it -- the underlying point that relates our risk assessment is we do consider that only about half of the people that are ill actually -- or infected,

actually become ill. And we do consider that those people can transmit illness to their family members.

MS. ALEXANDER: I get that about the risk assessment. But my question has to do with outbreaks.

And my question is, isn't it likely that outbreaks are even less likely to be detected if the illness in question is not highly symptomatic, such as the people getting sick aren't the ones who are actually on the water?

DR. TOLSON: Our testimony here is about this risk assessment, not on public health sort of concerns about outbreaks. Outbreaks really had nothing to do with the assessment.

MS. ALEXANDER: Oh, except you did, in fact, rely on the presence or absence of outbreaks as one of the two criteria for your choice of pathogens; is that correct?

DR. TOLSON: That is correct.

MS. ALEXANDER: Okay.

DR. TOLSON: Outbreaks are a good

indicator of which ones to go after and sample. But they didn't follow through to figure out what the impacts of outbreaks were on illness rates in the Chicago population. That's not part of their assessment.

MS. ALEXANDER: You say they are a good indicator, but, in fact, they are far from a perfect indicator; is that correct?

DR. TOLSON: That is correct.

MS. ALEXANDER: Because, as Dr. Gerba has acknowledged, it's entirely impossible for outbreaks to go undetected.

MR. ANDES: Well, I could follow up. Is there a perfect indicator.

DR. GERBA: Can I follow up on that?

DR. TOLSON: No, there's no --

DR. GERBA: When you say follow up, that's largely because limitations in public health, for one thing. Every potential outbreak is not investigated or comes to the public health's attention. Or not everybody calls in every time to the public health department when they have diarrhea or other types of illnesses.

So it's more of a limitation of public health's ability to respond to that to conduct an investigation. That's probably one of the overlying factors to all of the quantifying certain sources of infection and identifying outbreaks.

MS. WILLIAMS: Can I ask a follow-up question?

MS. ALEXANDER: Sure.

MS. WILLIAMS: I think Ms. Alexander indicates she understood something that I don't think I do. So I want to go back and make sure I do.

When we talk about the risk numbers, you know, one in a thousand, two in a thousand, eight in a thousand, I always understood USEPA to use it per 1,000 swimmers. Are your numbers based on per 1,000 recreators, or do they reflect also people who have not recreated but have contacted illness from someone who did? Can you explain that?

DR. TOLSON: Right. We actually ran two sets of numbers.

One of them for the people that were actually engaged in that. And then, just sort of to be more conservative and take into consideration a potential that that disease could spread to others, we took secondary attack rates, or their family members, and considered them a second pool of people that could be infected, and presented results for those too.

MS. WILLIAMS: Are they in separate tables in your report?

DR. TOLSON: They are.

Do you want me to point to those?

MS. WILLIAMS: Yes. Just so I can take a look at them.

DR. TOLSON: Sure.

We go to Table 513 in Exhibit 71. The bottom two lines there list illnesses primary and secondary, in parentheses.

So you can see from at the North Side we have 1.55 per 1,000. And if you include the pool to include secondary -- potential secondaries, it's 2.6.

MS. WILLIAMS: Okay. Thank you.

DR. TOLSON: Okay.

MS. WILLIAMS: I'll review that. I may have some follow-up later, but thanks.

DR. TOLSON: That's fine.

MS. ALEXANDER: I'd like to turn now to the second prong of your two-pronged justification for why you picked this limited subset of eight or so pathogens, which was the presence of USEPA approved laboratory standard operating procedures, which we've been referring to as SOP for measurement of the pathogens.

And the question is, does the availability of SOPs for a particular pathogen have any relationship to the risk it poses?

DR. PETROPOULOU: The availability of EPA approved methods or standard operating procedures relates to the ability to measure the concentration of the organism. And without that concentration, we cannot quantify the risk.

So in that sense, it does relate to how we are able to measure the risk but

not the risk --

MS. ALEXANDER: Okay.

DR. PETROPOULOU: -- in general of the --

MS. ALEXANDER: Not the risk, but your ability to measure it.

And now, as we're discussed before, you did, in fact, evaluate two types of pathogens for which there was not an EPA approved SOP; is that correct?

DR. PETROPOULOU: Correct.

MS. ALEXANDER: So that was not an absolute requirement for inclusion in your subset, it was just one of the factors you considered; is that correct?

DR. PETROPOULOU: Actually, we said either EPA approved or standard operating procedures, like the ones that Dr. Gerba's laboratory is using. So we did go beyond the EPA approved methods.

We also quantified viruses that the EPA has no approved method but Dr. Gerba's lab has standard operating procedures to use for that.

MS. ALEXANDER: And, in fact, there are USEPA approved SOPs for shigella; is that correct?

DR. GERBA: Shigella, there may be, yes. I'm not familiar --

MR. ANDES: I'm sorry, SOPs --

DR. GERBA: It is a standard method, yes.

MS. ALEXANDER: SOPs.

MR. ANDES: Analytical methods, I'm not --

MS. ALEXANDER: USEPA approved SOPs.

MR. ANDES: It doesn't approve --

MS. ALEXANDER: I'm sorry, US --

MR. ANDES: It approves only analytical methods.

MS. ALEXANDER: I'm sorry, they only --

MR. ANDES: Approve analytical methods to put in 40CFR136. I'm sorry, 136.

So I'm not sure if you're referring to an approved analytical nitrogen 136 or some other EPA generated document, but I don't think it's approved.

MS. ALEXANDER: Well, I believe -- in terms of just the language of USEPA approved, I believe that is drawn directly from Dr. Petropoulou's testimony.

Did you -- I can fish through it, but did you, in fact, refer to USEPA approved SOPs?

DR. PETROPOULOU: No. EPA approved methods or standard operating procedures.

MS. ALEXANDER: Methods or --

DR. PETROPOULOU: Laboratory standard operating procedures. I was not referring to EPA SOPs.

MS. ALEXANDER: Okay.

DR. GERBA: Let me go back there.

I don't know of an EPA approved method for shigella. Usually EPA only approves methods if there is a legal requirement for monitoring in some aspect, or they're conducting a study, which requires an approval, like an information collection rule.

However, there is a standard -- it is in standard method, there is a method for

shigella.

MS. ALEXANDER: Okay.

DR. GERBA: And we did not decide not to use shigella in this study, because I had questions about how good the method really was. In all of the recreational outbreaks that were associated with, there were usually too many people in the water.

In a review for 1971 or 2000, they were all lake waters, where people were believed to be the source through accidental fecal releases -- I hope they were accidental -- into the water.

MS. ALEXANDER: I'm not going there.

DR. GERBA: And also, shigella is a weak organism, it doesn't survive very well in the environment.

MS. ALEXANDER: But, in fact, as you just referenced, there have been recreational outbreaks of shigella?

DR. GERBA: Right. Associated with, I belive, fecal releases are too much gluteal fold in the water at one time.

MS. ALEXANDER: And my next question,

which I believe was a prefiled Petropoulou, but I'll just ask it.

The question I think has been partly answered, but I want to get at the rest of the answer, which is, did the risk assessment take into account populations that that are potentially more sensitive to pathogens and may more easily become ill or suffer severe effects, such as children, pregnant women and an immunocompromised person?

Now, an answer was given earlier, if I recall correctly, that the more sensitive populations were taken into account in the secondary infection rate analysis; is that correct?

DR. TOLSON: That is correct.

MS. ALEXANDER: Is there any other manner in which sensitive populations were taken into effect?

DR. GERBA: You know, again, we determined the risk of infection, so one could always assume that that risk of infection -- and you could apply what the

outcome would be to those groups, if you wished. You know, because that's the most conservative thing you do.

What you're talking about is the result of the infections.

MS. ALEXANDER: Okay. So, in fact, your analysis really doesn't address at all whether or not we're dealing with the risk of somebody having a mild case of diarrhea and somebody having a very severe gastrointestinal illness, which might be the result if, say, the person was a young child or was on chemotherapy?

DR. TOLSON: That is correct.

MS. ALEXANDER: Okay.

MR. ANDES: I want to follow up on that.

So, as I understand what you're saying, is the risk of illness -- there's no evidence that their risk of illnesses is different for these groups, the severity of the illness?

DR. GERBA: Right. That's correct.

MR. ANDES: So your risk assessment,

in terms of how likely that the people will develop infections, does not change?

DR. GERBA: It does not change.

DR. TOLSON: Just another follow-up on that.

On the flip side of that, you know, we do not consider immunity. And I think we touched on this before, that our analysis may be biased, because there may be immunity in the population that we didn't account for.

MS. ALEXANDER: Let me -- approximately what percent of the population would you say falls into these categories of immunocompromised persons?

DR. GERBA: I -- somewhere about 25 to 35 percent of the U.S. population. It's largely represented by people -- elderly individuals over 60 years of age.

MS. ALEXANDER: And also would you say children?

DR. GERBA: And children.

MS. ALEXANDER: And --

DR. GERBA: Well, when I said

children, we usually refer to infants, small children.

MS. ALEXANDER: Okay.

Pregnant women?

DR. GERBA: And pregnant women.

MS. ALEXANDER: And people on chemo therapy?

DR. GERBA: And -- yeah, people out walking around on chemotherapy.

MS. ALEXANDER: And people on antirejection drugs for organ transplants?

DR. GERBA: Yes.

MS. ALEXANDER: And people with HIV?

DR. GERBA: Yes.

MS. ALEXANDER: This question was Petropoulou No. 3 and also is the subject matter in Gerba No. 19 and Tolson No. 10. And the question is -- specifically, first to Dr. Petropoulou, because the statement's in your testimony.

Regarding the statement at Page 5, that although the microbial analytical results were evaluated within the framework of dry and wet weather conditions, "For the

MRA risk assessments, the dry and wet weather microbial results were integrated into a comprehensive data set representative of all weather conditions on the waterway."

And my question is, does this mean -- am I correct in understanding that in assessing post-disinfection risk, you combined data from the wet and dry weather conditions?

DR. TOLSON: Yes. In fact, you have to do that in order to assess what the effect of disinfection is.

You want to figure out what the overall seasonal population of recreator risk is, you have to consider that it rains, has CSO events and there's dry weather days. We've attenuated one of those sources to the waterway by disinfecting it and then reran the calculations to figure out what the effect would be on the whole population.

MS. ALEXANDER: Would it not be possible to run separate analyses for the risk on wet weather days and the risk on dry weather days?

DR. TOLSON: Sure. In fact, we present data that shows that.

MS. ALEXANDER: Well, let's turn to Table 5-14 in Exhibit 71.

DR. TOLSON: Table 5-14 was actually my exhibit to my testimony.

MS. ALEXANDER: Right, right. It's the same thing.

DR. TOLSON: Good.

MS. ALEXANDER: So that's your overall summary. But let's turn to page -- I'm sorry Table 59.

In fact, there you've broken out wet and dry weather estimates --

MR. ANDES: We actually have this one -- that one on the chart.

MS. ALEXANDER: Okay. Yeah, for wet and dry weather.

But it's not broken out in your overall estimate; is that correct?

DR. TOLSON: It is not broken out in the overall estimates. We've presented the data in a number of different ways and tried to stratify it.

Actually that was one of the comments we got from the EPA and we tried to stratify it in the final report in various fashions. The 5.9 exhibit that you show there addresses the question what happens just under dry, what happens just under wet.

MS. ALEXANDER: But only for pre-disinfection; correct? The table is headed Total Expected Illnesses For One Thousand Exposures Using Different Estimates and Pathogen Concentrations With No Effluent Disinfection.

So this is the before; right?

DR. TOLSON: Correct.

MS. ALEXANDER: Why don't you have a comparable after table? In other words, total expected illnesses per 1,000 for wet weather and for dry weather? Or did I miss it?

MR. TOLSON: Well, if you put the dry weather in there, you can see from 5.9 that the dry weather lists are very low.

MS. ALEXANDER: That wasn't my question, though.

DR. TOLSON: It would go even lower.

MS. ALEXANDER: My question is, did you break it out, or did you not?

DR. TOLSON: It is not broken out within the report.

MS. ALEXANDER: Okay.

MS. WILLIAMS: Was it broken out and not put into the report?

DR. TOLSON: Quite possibly. We have that data, but it would be, essentially, zero for every one of those.

If you dis --

MS. ALEXANDER: Based on what? Do you have any data printed to present to support that?

DR. TOLSON: Should I go and talk from it?

MS. ALEXANDER: Go ahead.

DR. TOLSON: All right. So Table 5.9 presents a risk for dry weather, wet weather and dry and wet weather.

If you look at just the dry weather results, we have fairly low risk within the waterway. One of the other tables

that we have in our report shows that the disinfection efficiency is quite high, or some -- against some of the pathogens, maybe 99 percent.

So you would decrease the pathogens within just the dry weather by 99 percent. The risk -- corresponding risk would drop very low.

That doesn't happen to the wet weather contributions. Those would not be attenuated by the disinfection.

So the overall risks that I presented in the other table, would not change so much.

MS. ALEXANDER: Just to clarify, by the way, it's your testimony that disinfection would reduce the pathogens and not just the indicators; correct?

DR. TOLSON: That's correct.

MS. ALEXANDER: By 99 percent, approximately?

DR. TOLSON: Well, there's a table in the report that lists each individual pathogen and the full reduction within that.

It varies by pathogen and by disinfection technique.

MS. ALEXANDER: So what you're saying, essentially, is -- correct me if I'm misinterpreting you -- is that for purposes of dry weather, you would, essentially, eliminate, or largely eliminate, the pathogen risk through disinfection; correct?

DR. TOLSON: Correct.

MS. ALEXANDER: Okay.

DR. TOLSON: We've been a little bit naive about the way we constructed that, in that we consider under dry weather there are no other inputs. So if there are pseudomonas inputs from overhanging vegetation or something like that that's contributing to dry weather, we're not including that.

We're assuming that under dry weather the effluent from the waste water treatment plants are the sole contributor to the waterway.

MS. ALEXANDER: Okay.

DR. TOLSON: So that's the assumption there.

MS. ALEXANDER: So, essentially, would it be fair to say that the disinfection would not have a substantial impact, in terms of your conclusions on wet weather pathogen levels and -- although, you've just testified that dry weather disinfection would substantially eliminate that risk?

DR. TOLSON: You would take a very low risk and you would make it a much lower risk.

MS. ALEXANDER: So, essentially, what you've done in Table 14 is combine a situation in which the risk is potentially reduced -- I'm sorry -- will be reduced with one in which you say it won't be reduced; correct? You've essentially combined those two sets of data, the wet and the dry weather?

DR. TOLSON: We present the data for the wet weather and the dry weather. In order to figure out the effect of the chlorination or other disinfection techniques, there has to be some assumptions made, because we can't do the experiment out of the waterway.

The assumptions that I made is the dry weather will, just substantially from the wastewater treatment plant, if you knock that down, what's the overall effect.

THE HEARING OFFICER: And, Ms. Alexander, Table 5-14 is the one you were referring to; right? You said Table 14.

MS. ALEXANDER: I'm sorry.

THE HEARING OFFICER: That's okay.

MS. ALEXANDER: I meant 5-14.

Let me get to the point, which is, leaving aside the absolute numbers for a moment, I know that you're claiming that they're low and our people say they're higher. Leaving that aside, what you have done here in combining the wet and dry weather post-disinfection risk, would that not mean that the change in the level of risk, since that change is higher for dry weather in the case of disinfection than for wet weather, that you are reducing the level of change in risk by combining these?

In other words, the delta is going to be lower if you combine wet and dry

weather than it would be for dry weather alone?

DR. TOLSON: The risk for recreators out on the waterway, though, is affected by wet weather conditions and affected by effluent from the wastewater treatment plant.

MS. ALEXANDER: But if you're out on the --

DR. TOLSON: It's the only way of speculating the risk without considering what the true pathogen concentrations are in the waterway. We developed data that comes from a fairly extensive data set of pathogen concentrations within the waterway to develop our risk.

MS. ALEXANDER: But if I'm out there on my canoe on a dry weather day, as you've defined it, not impacted by the wet weather, I'm not going to be impacted one way or the other by these wet weather risk levels; is that correct?

DR. TOLSON: Yeah.

MS. ALEXANDER: So if you wanted to --

DR. TOLSON: Hold on, let me answer

your question.

MS. ALEXANDER: Sorry. Go ahead.

DR. TOLSON: Because I want to refer you to one other exhibit, our Table 5.8 within Exhibit 71.

You'll see that only 15 percent of the days in Chicago is it truly a dry weather day, as we've defined it within the report. So today is not a dry weather day.

MS. ALEXANDER: I understand that, and I'll have a few questions for you about that calculation later.

But my point is, is it not true that, given that you have combined a situation in which there is a significant change, leaving aside the absolute levels between pre and post, pre-disinfection and post-disinfection, and you've combined that with a situation in which there is really not, according to you, a significant change between wet weather pre-disinfection and wet weather post-disinfection, does that not mean that the -- this change between the two, between pre and post, is going to be lower

than if you broke out dry weather, the degree of change would be higher; is that correct?

DR. TOLSON: If we had calculated risks where we said, only can go out into the waterway three days after it rained and just looked at that, the risks change or the impact of disinfection would be higher. However, it would be very low chance of risk, whether it's disinfection or nondisinfection.

MS. ALEXANDER: Okay. That --

DR. TOLSON: We're talking about low numbers versus almost zero numbers. Yes, in fact, the magnitude of the change would be different.

But that's a very -- a subset of something that doesn't really -- you can't control exposure of that.

MS. ALEXANDER: Okay.

And the absolute numbers, of course, are something that will be addressed subsequently in this proceeding. This question only went to the delta --

DR. TOLSON: Okay.

MS. ALEXANDER: -- as it were.

MR. ETTINGER: Can I ask one question about this chart?

DR. TOLSON: Sure.

MR. ETTINGER: You say dry weather, and then you've go North Side, Stickney, Calumet. Where are you assuming this exposure takes place?

I assume that the level would be higher if I were to capsize my canoe directly outside the outfall than if I did it two miles downstream.

DR. TOLSON: Yeah.

MR. ETTINGER: What are you doing -- how did you work that out?

DR. TOLSON: That's a good question. And, unfortunately, the answer is going to be a little long.

But we had to make some assumptions about where the use happened within the waterway and the concentrations happened in the waterway. We don't have data that would specifically tie a person to a specific spot within the UAA.

So we collected all the data

within the segment that we defined as North Side. And we said this is the use within the North Side water -- North Side segment.

We tied all of the data that we collected from that North Side and pulled those two together to calculate risk. We don't make an assumption that a person is going to be in any one place any more often than any other place.

It may be that they're -- you know, next to the outfalls more often than are not. In which case the risk would be biased low.

It may be that they're away from the outfalls or -- in which case the risk would be biased high -- under dry weather. But under wet weather we've got inputs all along the waterway.

So the relationship between where you're recreating in the outfalls, probably much less significant.

MR. ETTINGER: Okay.

MS. ALEXANDER: This question is -- it was a Petropoulou question for a -- similar

to Gerba 17 and 18 and Tolson 8 and 9. But this is specifically for Petropoulou.

Regarding the statement at Page 5 of your testimony that the risk assessment found that downstream concentrations -- and that's concentrations of pathogens -- are consistently greater than upstream during dry weather, so within -- the context here is dry weather.

For purposes of assessing risk, did you, in fact, combine the average upstream and downstream sampling numbers?

DR. PETROPOULOU: First, I think your statement mischaracterizes my testimony.

MS. ALEXANDER: Okay.

DR. PETROPOULOU: On Page 5, I discuss downstream concentrations are consistently greater than the upstream. I am not referring to pathogens there.

The discussions for bacteria results that were analyzed with the ANOVA testing. And that was done only for indicator bacteria.

So that statement pertains only to

indicator bacteria.

MS. ALEXANDER: Okay.

DR. PETROPOULOU: And with respect to your first question, I will let Dr. Tolson explain the integration procedure for the data.

DR. TOLSON: So under dry weather we did include all of the data from each waterway segment collectively at each sampling date to put as one of the inputs into our risk assessment. That included upstream and downstream concentrations.

We believe that's probably biased high, though, since under dry weather the data was collected in close proximity to the outfall. It didn't account for the fact that very far downstream of the outfall there is probably considerable additional attenuation that is not captured.

MS. ALEXANDER: Let me just make sure I understand though.

Your pathogen concentration levels that you assumed were ultimately -- correct me if I am wrong -- an average that included

both upstream and downstream.

DR. TOLSON: That is correct. We did not, as Dr. Lanyon put so elegantly yesterday, people can go upstream or downstream. So we don't know where the exposures happened, but we considered they could go in either direction.

The exposure was averaged across the entire place where they could actually be exposed.

MS. ALEXANDER: Would it be fair to say that the large majority of the CAWS waterway reaches are downstream of one or more of the treatment plants?

DR. TOLSON: Yeah, I do not -- we do not have the data to figure out what the attenuation rate is downstream. I believe that it, obviously, goes to the Mississippi eventually.

So there's a lot more downstream than there is upstream.

MS. ALEXANDER: Do you have any data showing that most people use both the upstream and downstream portions of the CAWS,

in roughly equal measure, even though there's a lot more downstream than upstream?

DR. TOLSON: We do not have data specifically to that.

MS. ALEXANDER: Okay.

So, in other words, your assumption, if we're talking about an individual as opposed to the overall analysis, would not hold true for someone that put in their canoe or kayak, say, at a location downstream of the treatment plant outfall and continued to paddle downstream; correct?

DR. TOLSON: I believe our concentration estimates in the waterway would be conservative for that scenario.

MS. ALEXANDER: What's the basis for that statement?

DR. TOLSON: Because we incorporated the downstream concentration immediately below the outfall and we included an upstream concentration, which we'll assume to be on the other end. And we assumed a linear concentration gradient as opposed to normal

downfall.

So, most likely, the average or the mean concentration that that canoe or recreator would be exposed to would be less than the average of the upstream and downstream.

MS. ALEXANDER: But, in fact, isn't it possible, based on your results, that the upstream concentrations upstream of the outfall and dry weather were lower than the downstream concentrations?

DR. TOLSON: For the indicators, it's really the case. For the pathogens, it's really not that clearcut.

Maybe I'll let Dr. Petropoulou --

DR. PETROPOULOU: Yeah. As I mentioned in my testimony, for example, if we take cryptosporidium, there was no infectious cryptosporidium upstream or downstream. So you're comparing zero to zero.

For viruses, we found that there were many instances were there were detectible viruses upstream but not downstream. Or that the upstream

concentrations were greater than the downstream. And that was true also for dry weather.

So that is true for indicators but not really for pathogens.

MR. HARLEY: Can I ask a question?

MS. ALEXANDER: Sure. Go ahead.

MR. HARLEY: I wanted to see if I could integrate the testimony that you gave about pseudomonas with the question of impacts of disinfection, dry weather and wet weather. Now, for pseudomonas, you indicated that you measured pseudomonas concentrations at outfalls of sewage treatment plants; is that correct?

DR. TOLSON: That is correct.

MR. HARLEY: And you came up with a level of 1,350 colony forming units per milliliter, I believe you said?

DR. TOLSON: That is correct.

MR. HARLEY: And during wet weather, when you were not measuring specifically at outfalls, you were measuring 54, 27 colony forming units per milliliter.

DR. TOLSON: Actually, we have outfall data from the wet weather also. And I think that the 1,350 includes a wet weather event outfall data as well as dry weather.

MR. HARLEY: Is the outfall number affected at all by whether or not it's dry weather or wet weather, or is it relatively constant?

DR. TOLSON: I'll let Dr. Petropoulou answer that.

DR. PETROPOULOU: Is the question specifically to pseudomonas?

MR. HARLEY: Why don't we start with pseudomonas, if you please.

DR. PETROPOULOU: For example, I think it depends on the size. And by size, I mean the treatment plant.

At the North Side during dry weather, the concentration of pseudomonas at the outfall was 1,091 CFU per 100 ML. During wet weather it was 796 CFU per 100 ML.

So they are different.

MR. ANDES: Tables 3-2(a) and 3-2(b).

DR. PETROPOULOU: On the report.

And I can read the other numbers.

MR. HARLEY: You don't need to.

DR. PETROPOULOU: Okay.

MR. HARLEY: I guess my question is, by disinfection, in light of the fact that we're going to be hearing different testimony from different witnesses based on what's been prefiled about risk, but just talking in terms of affect of disinfection on levels of a pathogen, like pseudomonas, if you disinfect, you're actually getting a benefit in terms of risk reduction, both during dry weather periods and during wet weather periods; is that correct?

Because during dry weather -- I'm sorry, I should let you answer that question.

DR. TOLSON: The marginal risk reduction under wet weather, though, is not nearly as much as it would be under dry weather. So I think that's what we are getting to.

For pseudomonas, it's a little more complicated, because there's probably additional sources that have been described.

MR. HARLEY: I understand. But you would get a benefit on those dry weather days because you would be removing pseudomonas by controlling what is the clearly primary source of pseudomonas on dry weather days, which is effluent from waste water treatment plants; is that correct?

DR. TOLSON: I assume it is. The "clearly" I'm not to sure about.

It's not clearly the dominant source, we really don't know that. We made the assumption within our risk assessment that the wastewater treatment plants were the only source.

And that's just in ease of calculation of our risk estimates, that was the way that we needed to do it.

DR. PETROPOULOU: Actually, I would like to add to that. Because we did look -- pseudomonas was not frequently detected during dry weather. I believe it was 80 percent of the samples that we collected, or 73 percent of the samples that we collected that had detectable pseudomonas.

So we looked through a statistical evaluation using box plugs to see if the concentration of pseudomonas were statistically different upstream, downstream and at the outfall. And I would point out that figures 321, 322 and 323, they present those results.

And, basically, the results showed that, for example, at North Side and at Calumet, the median concentration of pseudomonas were identical virtually, or statistically the same between upstream, downstream, at the outfalls. That was not the case at Stickney, where the concentration at the outfall was greater.

The median concentration was greater than upstream and downstream. But the upstream and downstream concentrations are the same.

MR. HARLEY: Uh-huh.

DR. PETROPOULOU: So you cannot really draw a direct conclusion.

MR. HARLEY: In wet weather events, if you removed a contribution for any

pathogen -- by disinfection, that would reduce the total loading of that pathogen during the wet weather event?

DR. TOLSON: That's correct. But, as we've shown, it's just not a great contribution.

MR. HARLEY: I understand there's going to be a difference in opinion as to the relative contribution --

MR. ANDES: He didn't state there was a difference in opinion, he stated -- let him state his opinion.

MR. HARLEY: I did.

MR. ANDES: No, I think you interrupted.

MR. HARLEY: Oh, did I? I'm sorry. I didn't mean to interrupt you.

DR. TOLSON: Yes, but the relative magnitude of that is insignificant compared to the wet weather loads.

MR. HARLEY: Thank you.

THE HEARING OFFICER: On that note, let's take a ten-minute break.

(WHEREUPON, a recess was had.)

THE HEARING OFFICER: Back on the record.

Ms. Alexander, we're still with you.

MS. ALEXANDER: I'm just going to jump back in subject matter a little bit to something I missed in my earlier thread.

Which is the question, did you consider inhalation as a exposure pathway in the risk assessment? Water inhalation.

DR. TOLSON: We considered it in terms of trying to figure out what the proportion or potential ingestion component of inhalation may be to the overall dose.

MS. ALEXANDER: Explain that. What do you mean the ingestion component of inhalation?

DR. TOLSON: When you breathe in air that might have mists and things that can lodge into your mucous membranes in your mouth, in which case you could swallow it. So it's not going into your lungs, but it could be, in fact, ingested.

MS. ALEXANDER: Okay. So you did not,

in fact, take into account, if I'm understanding you correctly, the impact of inhalation -- or I should say, that exposure pathway of inhalation into the lungs, you took it into account if it goes down the other pipe?

DR. TOLSON: Right. For a respiratory illness, we did not -- as we discussed previously, we did not consider it.

MS. ALEXANDER: Okay. Now, I am turning to Page 4 of -- make sure I understand what it's Page 4 of. One moment.

This is Page 4 of the first attachment to the May 23rd letter, which is attached to the May 28th letter of Exhibit 73, Page 4. You'll see in the middle of the page there's a bullet point GI Illness is the Sole End Point of Risk.

And then, in the middle of the paragraph that follows, within the first sentence of the paragraph, "This is a major weakness in the risk assessment," there's the statement, "Pseudomonas and adenovirus were found, so the author should have explored the

inhalation route to properly examine the risk associated with recreating on this water."

MR. ANDES: I'm sorry, what page are you on?

MS. ALEXANDER: I'm on Page 4 of the first attachment, which is Review Conducted by USEPA Office of Water, Office of Science and Technology.

DR. GERBA: The one -- pseudomonas transmission by recreational water and normal healthy people by inhalation route, I wouldn't even consider that. I don't know why that's even here. I think the person is not familiar with it.

It does cause lung infections in certain groups of people, but not recreational exposure. I've never heard of that.

The -- one of the problems here you have is what's the dose from, the secondary contact-type of exposure we're looking -- what's the amount we should consider being aerosolized by that route? There is no basis to form that type of

exposure.

MS. ALEXANDER: So --

DR. GERBA: And no information on how to do that is provided.

DR. TOLSON: And I want to point out that these are the -- this is one of the comments that we had discussions with Tim Wade on --

DR. GERBA: Yeah.

DR. TOLSON: -- at the meeting.

MS. ALEXANDER: Can you please, I'm sorry, define -- describe that discussion concerning specifically respiratory?

DR. TOLSON: And at that point, there's a consideration that we would not evaluate that quantitatively within our risk assessment.

MS. ALEXANDER: You would not evaluate the inhalation pathway?

DR. TOLSON: Correct.

MS. ALEXANDER: And what was the basis for that determination that you would not evaluate it? Or your reason for the consensus, I should say.

DR. TOLSON: It was -- one, it was not the most predominant illness associated with the recreational water, that GI illness was a predominant illness. But the other one, being that there's not a mechanism by which to establish the dose or the dose response for these organisms.

MS. ALEXANDER: Okay. So, once again, with respect to the inhalation pathway, we're talking about the mechanism being the main concern --

MR. ANDES: That's not what he said.

MS. ALEXANDER: -- as opposed to the risk.

DR. TOLSON: No. I have -- there were two points there that I made, and I think both are important considerations when looking at respiratory illness and associated with recreational contact, as it were.

MS. ALEXANDER: Okay.

But the bottom line is, you didn't consider inhalation or associated respiratory illness in this analysis?

DR. TOLSON: That is correct.

DR. GERBA: Let me reiterate. That doing pseudomonas, there would be no -- there is no recreational exposure that would result in a respiratory infection for pseudomonas. I don't know why that's in there.

DR. TOLSON: And also to follow up, that we did not evaluate that quantitatively, but qualitatively in terms of the proportion of risks, we would --

MS. ALEXANDER: This also references -- I'm sorry.

DR. TOLSON: That we perceive from the various illnesses, we did consider it that way.

MS. ALEXANDER: Referring to Dr. Gerba's statement just now, I believe the text refers to pseudomonas and adenovirus.

DR. GERBA: Right.

MS. ALEXANDER: Am I correct in understanding that there are strains of adenovirus that carry with them a risk of respiratory infection?

DR. GERBA: That is correct. And the only -- and, in fact, by recreational waters.

But those are all primary contact swimming-type exposures that resulted in those types of infection, not by the inhalation route.

MS. MEYERS-GLEN: If somebody flips over in a canoe and is dumped, or in a kayak, and they get a mouthful of water, then that would be an exposure route for them?

DR. GERBA: Yeah.

MS. ALEXANDER: Bear with me just a moment.

Okay. And this is also following up on an earlier discussion, but this was Tolson Question 11, which is -- I want to clarify, regarding the statement at Page 6 of your testimony. If you'll pull that out.

Regarding the statement at Page 6 that, "Disinfection at the effluent outfall was predicted to result in a decrease in effluent pathogen loads in the water reclamation plants that have little affect on overall pathogen concentrations in the waterway."

The question is, does that

statement concern wet weather conditions?

DR. TOLSON: It concerns neither wet nor dry weather conditions. It concerns the combination of wet and dry, which I think we discussed.

MS. ALEXANDER: The combination we discussed earlier.

DR. TOLSON: Right.

MS. ALEXANDER: Would that statement apply -- well, actually, let me rephrase that.

I take it, based on your earlier testimony, that that statement would not apply specifically to dry weather conditions; is that correct?

DR. TOLSON: Your question is does disinfection affect pathogen loads in the waterway under dry weather?

MS. ALEXANDER: Yeah. Your statement is that -- yes, that disinfection of the effluent outfall --

MR. ANDES: I'm sorry, you're not talking from Question 11, though; are you? Because that's not the same as Question 11.

MS. ALEXANDER: Hold on. Let me find Question 11, just to clarify. It is one of the Tolson questions, I may have mismarked it last night.

THE HEARING OFFICER: Yeah, the second part of the question at 11 --

MR. ANDES: Okay.

THE HEARING OFFICER: -- it says it applies specifically to dry weather conditions. She did rephrase it slightly, but...

MS. ALEXANDER: Okay.

So my question is, you make the statement, Dr. Tolson, "Disinfection of the effluent outfall, to paraphrase, was predicted to result in a decrease in pathogen loads from the water reclamation plants that have little affect on overall pathogen concentrations in the waterway."

And my question is, would that statement be true, specifically for dry weather conditions? And I actually characterized it as, am I correct in understanding it would not be true in view of

your testimony that disinfection would significantly decrease pathogen loads in dry weather conditions, or overall pathogen concentration, I mean?

DR. TOLSON: Disinfection under dry weather only conditions would decrease the pathogens that come out of the waste water treatment plant. However, we can't estimate overall illness rates in the waterway without considering all the sources.

MS. ALEXANDER: I'm not talking about illness rates.

DR. TOLSON: And if you look at the pathogens, they're low to begin with, so...

MS. ALEXANDER: My question, actually, Dr. Tolson, was not about illness rates. I'm talking specifically about your testimony on Page 6 that you say, "Disinfection -- ellipsis -- would have little effect on overall pathogen concentrations in the waterway."

Do you mean that statement to apply to dry weather conditions specifically?

MR. TOLSON: I understand what your

point is. So you're -- no, under dry weather conditions --

MS. ALEXANDER: Okay.

DR. TOLSON: -- it may be different.

I understand where you're coming from now.

MS. ALEXANDER: Okay. I'm sorry, that's all I'm asking.

And I take it there are no findings in the risk assessment that would support that statement in dry weather conditions; correct?

MR. ANDES: He didn't make the statement. What statement are you asking whether it would be supported?

MS. ALEXANDER: Okay. He makes the statement here on Page 6 of his testimony that disinfection of the effluent outfall would have little effect on overall pathogen concentrations in the waterway. Now, correct me if I'm mischaracterizing, but Dr. Tolson just said that statement would not hold true for dry weather, only in the wet and combined wet and dry analysis that was done in the

risk assessment.

And I'm following up to confirm that, in fact, there are no findings that support this conclusion that Dr. Tolson is not purporting to make that this statement would apply during dry weather conditions. That's all.

DR. TOLSON: The report --

MR. ANDES: I don't know if the answer is yes or no.

MS. ALEXANDER: Got lost in that?

There's nothing in the risk assessment that supports any conclusion this would apply to dry weather; is that correct?

DR. TOLSON: I believe you're pointing out that under our Table 5-14 within the risk assessments, we did not do that for dry weather, we did it for the combined.

MS. ALEXANDER: Right.

DR. TOLSON: Yeah, we talked about that before. That is correct, we did not present risk under dry weather because we believe that the whole intent of the risk assessment was to look at overall risks,

including dry and wet weather.

And the only way to do that was to consider that it rains in Chicago.

MS. ALEXANDER: Okay. All right. I think I've covered this.

Petropoulou No. 7, regarding the statement at Page 6 of your testimony that dry weather fecal coliform concentrations upstream of the North Side and Stickney plants were greater than the effluent limit of 400 CFU per 100 milliliters proposed by IEPA.

What's your understanding of the significance of that comparison that you make?

DR. PETROPOULOU: I actually would like to point out that I also follow with the -- another statement following what you just read in my testimony. And it's the same statement for wet weather.

And looking at the result in Tables 32(a) and 32(b) and looking at the fecal coliform concentrations, in the dry weather, as I mentioned, at the North Side

and at Stickney, the concentrations are greater than the proposed effluent limit. And that is also true more so in the wet weather.

And I can read, like in the wet weather, for example, at North Side upstream of the outfall, there is 117,000 fecal coliform CFUs for 100 ML downstream. We measured a hundred thousand CFUs for a hundred ML. The outfall concentration is 22,000.

Similarly at Stickney, you can see that the upstream concentration is 172,000, the downstream concentration is 230,000. And at the outfall we measure 38,000.

The importance -- my view of that is IEPA is proposing this effluent limit to protect the users of the waterway, there are probably other sources to look into in addition to the district's effluence. Because they contribute fairly high concentrations of fecal coliform in the waterway.

That's the only significance that

I see.

MS. ALEXANDER: All right. Let me jump back.

The data you were just reading from, Table 3(b) is wet weather data; is that correct?

DR. PETROPOULOU: That is correct, yes.

MS. ALEXANDER: Okay.

And just to point out, in the dry weather data, it appears, fairly consistently, that when you're looking at fecal coliform indicators, that upstream the levels are orders of magnitude lower than downstream; is that correct?

DR. PETROPOULOU: They are great, yes.

MS. ALEXANDER: Yes, okay.

I want to get back to my original question, because you may have answered it, but I think I may have lost the thread. Which is, at Page 6 of your testimony, you state that the dry weather, as opposed to wet weather, fecal coliform concentrations upstream of the North Side and Stickney

plants, and we're looking at these numbers, for instance, 713 at North Side and that Table 3-2(a), 1,061 at Stickney -- actually the number at Calumet 170 would be lower than the 400.

But you point out that, I would say, that at least the first two are higher than the 400 fecal colony forming units per 100 milliliters that's proposed by the IEPA. And my question is, what is the significance of that comparison?

Why is it significant in your view or what -- is there a point that you're making in stating that the concentrations upstream are higher than the required effluent limit being proposed by IEPA?

DR. PETROPOULOU: And again, my statement -- I know you selected one of the two statements I made.

MS. ALEXANDER: Uh-huh.

DR. PETROPOULOU: In order for me to make my point, I would like to include both statements. And that is, look together at both the dry and wet weather conditions for

the waterway. It's the same significance.

There are probably other sources of fecal coliform in the waterway than the district's effluence. That's the significance.

MS. ALEXANDER: What levels of fecal coliform indicator bacteria are, generally, found in the effluent from these three facilities? And I mean the range, I understand that it varies.

MR. ANDES: I think we already -- that question has already been answered by Mr. Lanyon.

MS. ALEXANDER: Well, in fact, it was. I mean, I'll phrase the question differently.

In fact, didn't those levels range up to 200,000 fecal colony forming units per hundred milliliters? Is that correct?

DR. PETROPOULOU: No, that's not consistent with our findings. When we did the study in dry weather, we found concentrations that range from 42,000 to 56,000.

And during wet weather, actually,

the district's outfall contributes one-third, or 50 percent less of that, to the waterway.

If you look at the outfall concentrations during wet weather -- for example, at North Side, the dry weather concentration in the outfall was 42,000. During wet weather the district contributes 22,000 fecal coliform units. That's like 50 percent of what they contribute during dry weather.

And similar results are observed for the outfall concentration during wet weather at the district outfall.

So the concentration we measured, you can say they were from 22,000 to 38,000 in the district's outfall during wet weather and between 42,000 and 56,000 during dry weather.

MS. ALEXANDER: I think my point is a little more straightforward than that, or I should say my question is. Let's look specifically at dry weather for a moment.

And you cited the outfall numbers during dry weather that we're looking at, 42,

411, 56, 391, 56, 287, just to quote from Table 3-2(a).

Isn't it a fact that those outfall numbers, as in what the plant is discharging now during dry weather, are orders of magnitude higher than what it would be discharging under the proposed IEPA standard?

DR. PETROPOULOU: That is correct.

MS. ALEXANDER: Okay. So, in other words, a limit of 400 colony forming units per 100 milliliters, as proposed by the IEPA, might be less in some cases than ambient background levels, but it would still result in a significant reduction in the loading of at least indicator bacteria in the water body; is that correct?

DR. PETROPOULOU: I can't say that. It depends on the weather conditions.

MS. ALEXANDER: I'm talking about dry weather now exclusively, I'm sorry.

DR. PETROPOULOU: I can't make that statement. Like -- you're talking about significant and loads, I think that can be calculated, I'm just not prepared to offer an

opinion on that.

MS. ALEXANDER: Well, let me reframe my question, because I think it's a little simpler than, perhaps, how you're interpreting it.

If right now, say, as North Side, the fecal coliform level coming out of the outfall are around 42,000, would it be fair to say that they'll be significantly reduced if you put a limitation of 400 on it, and then you're going to go to 42,000 to something other than 400; correct?

DR. PETROPOULOU: That is correct.

MS. ALEXANDER: Okay. That's all I'm getting at.

I want to turn to Table 58 for a moment and just briefly revisit these issues having to do with dry and wet weather days. This is, again, in Exhibit 71.

All right. I just wanted to clarify, because this wasn't obvious to me. And that where I'm getting this from is it's Tolson Question 11 and Gerba Question 20.

I had framed the question as

"Describe how you arrived at these numbers." I think there's already been some description of that. So I'm just going to ask a few follow-up questions on that.

Was there any overlap between days that were counted as wet weather and days that were counted as post-wet weather? If that question make sense. I can rephrase it if it doesn't.

DR. TOLSON: My answer is it's fairly obvious the day after certainly has a relationship to it.

MR. ANDES: Are you asking if there's double counting?

MS. ALEXANDER: Well, let me ask it -- I just want to make sure I understand.

Let's say it rained on seven consecutive days. How many of these post-wet weather days would you assume? Would that, then, be seven wet weather days and three post-wet weather days?

DR. TOLSON: We didn't fall into that era. We actually took the meteorological data from the year and put it all out and

figured out how many multi-day bouts of rain we had, and then used the other intervening days where it was dry and calculated those intermediate weather days.

MS. WILLIAMS: When you say "meteorological data," can you elaborate a little bit?

DR. TOLSON: Yes, we collected -- we asked the district for their rain gauge data, and we used that for the basis of our establishing wet weather days within the 2006 recreational season.

MS. WILLIAMS: And what if you had rain at one gauge and not at another?

DR. TOLSON: The way we did that is we took -- I'm trying to recall exactly how we sorted this out.

I believe we had a weather station -- two weather stations, and we actually looked at the analysis for the North Shore and then we looked at the analysis for the Stickney and Calumet together. We tried to account for that within our assessment.

But, essentially, it was the same

way. We looked at all the meteorological data and did not double count days. We took into account if it rained for four days in a row, those each were rain days.

And then the days after that were the intervening days. And then days were -- had a three-day antecedent period.

MS. WILLIAMS: But did it have to rain in the area where the sample was taken to have a rain day? I'm still not sure I'm following.

If you only recorded rain at the North Side plant, was it considered a rain day at Stickney for your sampling at Stickney?

DR. TOLSON: We had to average out the meteorological data between the different weather stations we had. What we found was that about 40 percent of the days were rain days or CSO days.

Thirty percent, stationwide, were the day after it rained, 15 percent were two days after and 15 percent were kind of, at least, two days of dry weather before that

day. So it's a generalization for the entire Chicago basin, it took into account district weather data.

MS. WILLIAMS: I'm not sure I understand, but I think you answered my question.

We can go back to Ms. Alexander.

DR. TOLSON: Okay.

MS. ALEXANDER: Okay. This is Tolson Question No. 13. And there is a question to Dr. Gerba, Question 21, which is, essentially, the same. But regarding Tolson's testimony, the statement at Page 5 that "The UAA study was a primary source for exposure use data in the CAWS."

The question is, is it possible, in your view, that a water body that was perceived by the public or known to be cleaner than the CAWS, such as, for instance, Lake Michigan, might receive heavier use for activities involving substantial body contact with water? In other words, are people more likely to go kayaking and canoeing in the clean water bodies than one believed to be

contaminated?

MR. ANDES: Are we at -- let me settle that. But I think we could clarify.

Are you talking about water bodies that are simply perceived as cleaner or are actually cleaner? What's heavier use? Does that mean more people, does that mean different types of use?

I mean, what substantial body contact with water? Those are all -- I'm not sure what any of those phrases mean.

MS. ALEXANDER: I'm going to break this down a little bit.

First of all, I am talking about -- I mean, I'll say known to be cleaner in the sense that there's publicly available data out there that there is more contamination in one water body than the people who use the water may be aware of it. It's sort of the criterion and the difference I'm talking about.

And the question is -- I mean, I'll first ask the basic question. Would you agree that people are probably -- you know,

maybe more likely, potentially, to engage in incidental contact-type activities, like canoeing and kayaking in the water body known to be cleaner?

DR. TOLSON: You're venturing way off into speculation.

MS. ALEXANDER: I understand that's not -- let me move on a little bit.

DR. TOLSON: Yeah, sorry.

MS. ALEXANDER: Did you take in account in any way in this risk assessment the possibility that people might be more willing to conduct themselves in the context of their water activity, such as canoeing and kayaking, in such a way as to increase their bodily contact if they believe the water to be clean, clean? And let me just sort of clarify what I mean by that.

Were they -- for instance, did you take into account the possibility that people might be more willing to roll their kiak on Lake Michigan than in the CAWS?

DR. TOLSON: No, ma'am, we did not make any assumptions to that rolling.

MS. ALEXANDER: Or, for instance, the possibility that they might be more likely to jump off their motorboat and go swimming on a hot day in Lake Michigan than in the CAWS; did you consider that?

DR. TOLSON: All those questions are really outside of the scope of our study.

MS. ALEXANDER: Okay.

DR. TOLSON: And I can't really evaluate them.

MS. ALEXANDER: I get it. You didn't consider any of that.

This is Tolson Question 14 and Gerba Question 22. Is it your understanding that water born pathogen levels can vary with the degree of sunlight on the water, given that sunlight kills pathogens?

DR. GERBA: I think that question is misstated. You have pathogen levels, and I think you mean pathogen survival.

Because pathogen levels are independent to sunlight.

MS. ALEXANDER: So, in other words, you're including activated and deactivated

pathogens --

DR. GERBA: Right. Sunlight is one that does influence particularly the survival of bacteria in water. Not so much viruses.

Particularly like adenoviruses, which are more resistant to UV light, which is really the primary component in sunlight, that inactivates microorganisms.

Do you want me continue answering the rest of those?

MS. ALEXANDER: Yeah. You can go ahead and answer with that clarification.

DR. GERBA: Again, the issue is survival with the turbidity of the water. Generally, the more turbid the water, the longer organisms -- water born pathogens, I should say, as stated here, survive in water. And with temperature -- generally, the warmer the temperature and more rapid the organisms get inactivated, in this case, organisms meaning water born pathogens.

MS. ALEXANDER: Did your risk assessment account for these variables in any way?

DR. TOLSON: We actually measure the concentrations in the waterway, and that was the basis for the risk assessment.

MS. ALEXANDER: But in determining what -- for instance what days you were going to measure or how you were going to weight the sampling on any given day, you didn't take into account, for instance, whether there was sunlight on the water at the time or whether the water was turbid? You took the samples but you didn't weight those factors, in other words; is that correct?

DR. TOLSON: Correct. Let me think about this for a second and see whether that somehow could have biased it high or low.

I really -- I can't tell whether -- how not accounting for that would have affected the results. But I don't think it would have affected to a great extent.

DR. GERBA: Well, certainly not for adenovirus, because we didn't measure whether they were dead or alive. I would say the turbidity would have a lot to inhibit the effect of sunlight.

And I don't think the temperatures were really that warm compared to other bodies of water I studied. I don't think either of those, probably in the time span from the outfall that we looked at, would have much of a major influence, certainly for the viruses that are much more resilient and survive better in the water.

So I don't think those would be major factors, certainly, for the viruses.

MS. ALEXANDER: But temperature, turbidity and sunlight, presumably, vary from day-to-day; is that correct?

DR. GERBA: Yeah, that's right. As far as I'm concerned, this place is a cold place compared to Arizona.

MS. ALEXANDER: I'm not going to argue with that. Just one second.

MS. WILLIAMS: I feel like I want to ask a follow-up, but I'm not sure. I mean, did you look at the temperatures throughout the system?

DR. TOLSON: I don't -- actually --

MS. WILLIAMS: I mean, you're not -- I

guess when you make that last statement, you're not taking into account the power generation facility on this waterway; are you?

DR. GERBA: Temperatures were measured by the district, but that's all I can say.

DR. TOLSON: We measure the pathogen concentrations at the time that we collected those samples. And those represented what we considered to be the concentrations over that day for which that weather type that we measured them on.

So if it was a rainy day and we collected rain samples during that day, that concentration we measured was what we assumed.

MS. WILLIAMS: I guess I'm just still reacting to the idea that you said that -- the rest of us sort of haven't been at the earlier hearings -- that you felt this was a cold system. Compared to what?

DR. GERBA: Compared to like Florida. I think Dr. Joan Gross, for example, looked at die-off of clean surface waters, eventual

viruses.

In there we had really clear waters, low turbidity and higher temperatures than you have here. You get more rapid die-off of like enteroviruses in the water.

When you get the cooler --

MS. WILLIAMS: You're talking about the air temperature --

DR. GERBA: Right.

MS. WILLIAMS: Not necessarily the water temperature?

DR. GERBA: Exactly.

No, water. I'm talking water, not air.

MS. WILLIAMS: So you're talking about water --

DR. GERBA: Usually the water temperatures are related to the ambient air temperatures.

MS. WILLIAMS: And did you look at whether that's the case here?

DR. GERBA: What the temperatures were you mean?

MS. WILLIAMS: Whether there's a

natural relationship between the air temperature and the water temperature here, in this system?

DR. GERBA: It would be an unusual place that didn't, that's all I can say.

MS. WILLIAMS: Is it --

DR. GERBA: I correlated that.

MS. WILLIAMS: Is it an unusual situation to have 70 percent of the flow coming from a water treatment plant?

DR. GERBA: I've seen more, but that's a lot of -- I mean, 70 percent, I've seen 100 percent before. So it varies.

MS. WILLIAMS: Is it usual to have a system with five power generating facilities located in this proximity?

DR. GERBA: I can't comment on that.

MS. WILLIAMS: I just want to make sure that wasn't part of your -- what went into your statement.

DR. GERBA: Oh, no.

MS. WILLIAMS: I'm done.

DR. GERBA: Those are factors that influence -- I'd have to know the actual

numbers and degree to tell you how much it might influence it.

MS. ALEXANDER: All right. This is Tolson Question 15 and then Gerba Question 23.

What was the basis for using dose response data for echovirus as a surrogate for this dose response behavior for adenovirus?

DR. GERBA: Excuse me just one second.

Let me just say, we use a dose response data for echoviruses, largely because that represents a virus transmitted by the enteric route. Dose response data for that was based on ingestion.

And, of course, some of the entero adenoviruses are transmitted by the ingestion route. So we wanted to use a dose response model that included ingestion. There wasn't one available for adenoviruses.

The only one for adenovirus available was inhalation. So that was the reason we used it.

MS. ALEXANDER: Are you saying there

is dose response data connected with inhalation of adenovirus?

DR. GERBA: Yes.

MS. ALEXANDER: Given that fact, why could you not have also done the complete risk analysis of risk of respiratory inhalation-based illness from adenovirus?

MR. ANDES: He already explained why he didn't do that analysis.

Go ahead and answer.

MS. ALEXANDER: Is your answer the reason you gave before -- well, you gave a two-part answer. And one of them was that you believe GI illness is, essentially, the predominant recreation-associated illness. But the other part of the answer that you consistently gave is that there's no dose response data for these types of illnesses.

But we have one here for which, in fact, there is dose response data. And my question is, is there a reason you could not have done that analysis, given there is dose response data?

DR. GERBA: There was two organisms in

my last response, there was pseudomonas aeruginosa, there was no --

MS. ALEXANDER: Right. I understand that.

DR. GERBA: For adenoviruses, there was -- the thing I said was that there was no data to estimate what the aerosol exposure would be from secondary contact recreation.

MS. ALEXANDER: Okay. So you did have a dose response, but you didn't have the aerosol inhalation data; is what you're saying?

DR. GERBA: That's correct.

MS. ALEXANDER: Okay.

All right. This is referring to what were Tolson Questions 16 and Gerba 24. I'll represent it was a series of questions concerning the EPA ICR manual and procedures for disinfecting equipment.

I'm going to be modifying these questions, based on a document that I was handed yesterday, which I apologize, but I neglected to make copies of in my haste. But it is a letter dated September 5th, 2008 from

Marsha -- to Marsha Willhite from Coleus, transmitting a letter from Geosyntec Consultants dated August 22nd, 2008 to Thomas Granato of MWRD from Dr. Petropoulou.

And then, attached to Dr. Petropoulou's letter is an errata sheet for the risk assessment, which includes some information about Tolson 16 and Gerba 24. So --

THE HEARING OFFICER: Ms. Alexander, is that not Exhibit 59?

MS. ALEXANDER: Oh, is that -- I'm sorry. That's been introduced?

THE HEARING OFFICER: I believe so, if it talks about the same letter, yes.

MS. ALEXANDER: Never mind then. We're talking about Exhibit 59. I apologize for the excess words.

So I'm going to be asking you some questions about that. And you guys have it over there, I assume.

Am I correct in my understanding that the EPA ICR manual for disinfecting equipment to be used for virus sampling

requires that the concentration of chlorine to be used to disinfect is .1 percent, and that after chlorination the chlorine needs to be neutralized with sodium biosulphate; is that correct?

DR. GERBA: That's right.

MS. ALEXANDER: Okay.

Now, in the draft -- I shouldn't say draft -- in the final of the risk assessment that was appended to various witnesses' testimony from the district and published on the district's website, it was indicated --

THE HEARING OFFICER: Excuse me, which is Exhibit 71?

MS. ALEXANDER: Exhibit 71. I'm sorry.

THE HEARING OFFICER: Okay. For the record, it's better if we --

MS. ALEXANDER: I apologize.

THE HEARING OFFICER: That's okay.

MS. ALEXANDER: It was indicated at Page 16 of Exhibit 71 that, essentially, this procedure was not followed; is that correct?

DR. PETROPOULOU: No, that is not correct. We had a typographical error with respect to the concentration of the bleach that we used for the disinfection.

In our sampling and quality assurance plans, both for the dry and wet weather, we specified the correct cleaning and sterilization method for the equipment, that's what the district followed. We have made the correction in this errata sheet for that.

That was the purpose of this errata sheet.

THE HEARING OFFICER: The errata sheet attached to Exhibit 59?

DR. PETROPOULOU: Correct.

MS. ALEXANDER: Okay. So you characterize the change from the .1 percent solution -- I'm sorry, the .5 percent.

The .1 percent is a typographical error; is that correct?

DR. PETROPOULOU: That's correct.

MS. ALEXANDER: Are the other changes also corrections, in your view, of

typographical errors?

DR. PETROPOULOU: No, they are not. We have omitted to include how we dechlorinated the equipment, and we have added that for clarification.

I believe it was Dr. Yates. She raised that as an issue in her testimony.

So it brought it to our attention that we should include that in the report just to make sure there's no confusion about it.

MS. ALEXANDER: Do you have any lab records that are reflecting specifically this information that you, in fact, dechlorinated the equipment?

DR. PETROPOULOU: I believe Dr. Ishal from the district that was overseeing the lab, she has instructions to the lab that include an excerpt of our sampling and analysis plan. And there were instructions to the sampling staff on the boat that included information of how to disinfect equipment.

And Dr. Gerba and myself, who were

on the boat, we did the sampling during the first week. So I know that that's -- it was done properly.

MS. ALEXANDER: And just to refer to Item 1 on the errata sheet attached to Exhibit 59, you replace the reference to blue-green monkey kidney with buffalo green monkey kidney.

Am I correct in understanding that there is, in fact, no such thing as a blue-green monkey or its kidney?

DR. GERBA: No.

MS. ALEXANDER: Okay. So that's not any kind of cell culture line. The only cell culture line is buffalo green monkey kidneys used in this analysis.

Now, I'm turning next to Tolson Question 17 and Gerba Question 25. I just want to state, as an initial matter, that these questions all characterize them as having to do with sample size and proportion of the sample evaluated.

There was an indication in prefiled questions given to Dr. Yates that

this information is available in appendices to the risk assessment. We were not provided with those appendices until I saw those questions and requested the appendices from Mr. Andes. I have just been provided them.

I have not had the opportunity either to completely review those or to discuss them were my expert. I do not know whether any of that will be a problem. I simply state that as a caveat on the record, that that's an issue that might come up at some point down the road.

It is possible that some of my fact or clarification questions may be answerable by reference to that data. But we can just proceed and see how that works out.

MS. WILLIAMS: Can you just clarify for us, so the rest of us don't have them either, that it's not part of Exhibit 71?

MS. ALEXANDER: It's not currently part of Exhibit 71. I -- and I was not planning or referring in my question specifically to that data to the extent the witnesses refer to that information in their

answers, we may need to admit it into the record and mark it as an exhibit.

MR. ANDES: Appendices A, B, C and D to the report, I believe A and B I provided last week.

MS. ALEXANDER: Correct.

MR. ANDES: I've now provided A, B, C and D. If the Agency wants the whole enormous amount of information on a disk, I have another copy and I can provide that, as well. As soon as I find it under this paper.

MS. WILLIAMS: Well, first, I just wanted to understand what was in the record and what's not. So we're clear, then, that there's four appendices and two are in the record and two or not; is that correct?

THE HEARING OFFICER: No, none of them are.

MS. WILLIAMS: None of them are.

MS. ALEXANDER: There's an Attachment A that is part of the Exhibit 71 that is in the record, but there are no appendices in the record?

MR. ANDES: There are Appendices A, B,

C and D.

MS. WILLIAMS: It seem like that's something that after the hearing we can have supplemented by you guys?

MR. ANDES: That would be fine.

THE HEARING OFFICER: Yes.

MR. ANDES: That is beyond the 350 pages of the report.

MS. WILLIAMS: Which was submitted like four times; right?

MR. ANDES: Yes.

THE HEARING OFFICER: We only need it onces this time.

MS. ALEXANDER: Referring back to those questions, Tolson 17 and Gerba 25, the general question I have is, how large were the samples that you collected for virus analysis?

DR. GERBA: Near the -- by the outfall, 100 liters, and away from the outfall 300 liters.

MS. ALEXANDER: Okay.

THE HEARING OFFICER: I'm sorry?

DR. GERBA: One hundred liters by the

outfall, and then 300 liters away from the outfall.

THE HEARING OFFICER: A train went by as you finished your question, so I couldn't hear it.

DR. GERBA: To give you a perspective, that's -- 100 liters, basically, is about 25 gallons. And about 75 gallons is about 300 liters, to give you a rough idea.

MS. ALEXANDER: And what volume from these samples are, typically, analyzed for each of the viruses?

DR. GERBA: If it was divided up about -- basically -- I don't know. Do you have the actual ratio?

It's in the SOP, but I don't remember off the top of my head. I couldn't give you it right off the top of my head.

MS. ALEXANDER: Can you give me an approximation?

DR. GERBA: Well -- I tried to do at least 100 liters for each virus, when it was feasible to do that. For the Norovirus, it was not feasible, because of the analytical

method limits, you only need a few hundred microliters of a concentrate.

But we tried to do 100 liters for each of the virus groups for -- away from the outfall, and about 30 liters for each virus at the outfall.

MS. ALEXANDER: One second here.

I have in my notes -- and I haven't quite found the table yet -- that the typical volume of the sample analyzed for calcivirus was around .2 liters; is that correct?

DR. GERBA: It varied from sample to sample, something like two liters.

MS. ALEXANDER: It was about that would you say?

DR. GERBA: Yeah.

MS. ALEXANDER: And if no viruses were detected in that .2 liter sample out of the entire sample, would that have been 100 to 300 --

DR. GERBA: I'm just saying that without having looked at it. So I'm not quite sure.

MR. ANDES: Let's not make a statement without looking. Let's go back, because I heard two and I heard .2.

DR. GERBA: Yeah, I'd have to look at the exact equivalent volume. I'm not sure if you're giving me the volumes on concentrate assay or the equivalent volume of that concentrated to the water sample that was collected.

MS. ALEXANDER: All right. I found that reference.

If you turn to Table 3-7, which is Table 3-7 in Exhibit 71, Dry Weather norovirus, paren, (calcivirus results).

And then you'll see there is a column in that Equivalent Volume Assay in Liters.

DR. GERBA: Right. Right. That's right.

MS. ALEXANDER: And you see that it varies. But would I be fair in characterizing that as it all falls out to in the vicinity of .2 liters?

DR. GERBA: Yeah, about 200

milliliters, you're correct.

MS. ALEXANDER: All right. And that's out of the entire sample that was drawn, which, as I understand it, would have ranged from 100 to 300 liters?

DR. GERBA: I'm sorry, this is -- yeah, the equivalent volume of the concentrate, that was actually analyzed by the PCR method. The PCR method has very limited volume that can be assayed, where -- compared to, you know, the method of the other two viruses, almost the entire sample was assayed in 100 liter volume.

It's just the analytical method here for norovirus is limited to apparently a small sample. But still we are able to protect the virus, particularly during rainfall.

MS. ALEXANDER: Okay. But you were only -- in fact, if you only tested, assayed, the .2 liters out of your 100 to 300 liter samples, you wouldn't actually know what was in the other 99.8 percent of the sample because you didn't test it; is that correct?

DR. GERBA: Right. Let me make sure I understand.

There's concentrations that would take 300 liters and you reduce it to 20 milliliters.

MS. ALEXANDER: Right.

DR. GERBA: Is what goes on here. And then you're expanding backwards to that.

And usually your assay maybe 10 MLs for the adenovirus and ten MLs for what we call the total cultural virus. And then usually several microliters for this.

It's important here that this is 240 milliliters, by the way, which I hope is a volume nobody ever swallows in the water. The reason for the larger volumes for the other viruses is because they're in such low levels.

So, in reality, in terms of what somebody might swallow, the smallest volume that was assayed here was about 100 milliliters and the largest was about 410 milliliters. So those are relatively what somebody might have actually swallow.

Even in contact recreation, it would be greater, though, with respect to swallowing.

MS. ALEXANDER: The question I'm asking is you did not, in fact, test the entire sample you took in the case of calcivirus; is that correct?

DR. GERBA: Oh, no, it was impossible to do that.

MS. ALEXANDER: Right. And, in fact, you didn't really test anything close to the entire sample?

DR. GERBA: No, it was impossible to do that.

MS. ALEXANDER: Okay.

DR. GERBA: Not with that analytical method.

MS. ALEXANDER: Okay.

Tolson Question 18 and Gerba Question 26, what primers were used for the calcivirus analysis?

DR. GERBA: Those were primers that were developed by Jan Vanay, now with the Centers For Disease Control and Prevention.

We used these primers to investigate more than 20 outbreaks of noroviruses in the last several years.

So we know they were fairly effective in picking up all the norovirus types that were causing outbreaks, certainly on cruise ships and outbreaks in the United States.

MS. ALEXANDER: Specifically on which calciviruses are detected --

DR. GERBA: With norovirus -- the human norovirus.

MS. ALEXANDER: Okay. Just the human norovirus?

DR. GERBA: Uh-huh.

MS. ALEXANDER: Okay. Tolson 20 and Gerba 28, can you describe the method that was used to analyze the samples of -- I'm sorry -- for adenovirus?

DR. GERBA: That's in the SOP, but, basically, what you do is, again, you take part of the concentrate and we put it on a specific cell line to which adenoviruses are known to be sensitive to. The BGM cell line,

adenoviruses are not sensitive to -- they don't produce cytopathogenic effects.

But in the cell line we use, they do produce cytopathogenic effects. We expose those to cells for 14 days and then we take the negative samples and expose those to the cells for another 14 days, for a total of 28 days. And those cell lines show a cytopathogenic effects.

We use primers against the human adenoviruses to confirm there was human adenoviruses that we detected. Some human enteroviruses grow on the cell lines, too. So there was a need to confirm that they were adenoviruses.

The cell lines that we use will grow adenovirus, most of the adenoviruses, 40 and 41, which are the ones that cause gastroenteritis 2, 4, and 7 and several others. But we've been using these to grow various adenovirus serotypes in our laboratory for several years.

And we've used -- I should say -- the same procedure for detecting adenoviruses

and other studies on water -- waste water discharges, which have been published in peer reviewed scientific literature.

MS. ALEXANDER: I'm sorry, I think you just answered this question and I lost the thread. But my sub-A on that was, which specific serotypes of adenovirus are detected using the BGM cell line that you used? Could you list those for me?

DR. GERBA: We use the -- actually, PLC5 cell lines for --

MS. ALEXANDER: Oh, PLC5, okay.

DR. GERBA: -- the adenoviruses. Because they don't produce cytopathogenic effects in the buffalo green monkey cell line.

In this cell line we'll grow 40, * 41, 2, 7 and 4, to my knowledge, and probably several of the other types of it. The primers would detect, basically, any of the human adenoviruses.

MR. ANDES: In follow-up, do you consider these to be a conservative approach? And, if so, how?

DR. GERBA: Yeah, I consider -- well, the whole idea of putting adenoviruses in here was a conservative approach. Even though there was no approved EPA method for adenoviruses, the literatures indicate adenovirus were the most abundant viruses in sewage discharges. So we felt we would be neglecting the most abundant virus that could be current in sewage, and that's why this part of the study actually was done.

And then we wanted to confirm for sure that it was adenovirus that we detected, that's why we used the primers when we did it. Because we were trying to be conservative here and trying to estimate the greatest number of viruses that would be present in the sewage and in the waterway.

So that's why we felt it essential to include the adenoviruses in here. And, as you saw from the results of the study, adenoviruses were in far more abundance than the enterovirus.

And if we just used the EPA manual for the total culturable virus, we would have

missed almost the majority of the viruses we actually detected in the waterway. So I think that premise actually paid out in this study.

MS. ALEXANDER: I just -- I'm going to need to ask some follow-up on that for clarification.

Did you say that all serotypes of adenovirus are detected using the PCR -- the primers used for PCR analysis?

DR. GERBA: All the major human enteroviruses, yeah.

MS. ALEXANDER: When you say all the major human enteroviruses...

DR. GERBA: I said that, because I don't know if every human -- I'm sorry -- adenovirus has ever been tested against this set of primers. I don't know that for certain.

MS. ALEXANDER: Okay. So the PCR analysis would have detected the ones you've listed, 5 40, 41, 2, 7 and 4?

DR. GERBA: Right.

Some of those have been associated

with water born diseases, too. You know, recreational water, that's why we...

MS. ALEXANDER: There are, in fact, 51 different types of adenoviruses; correct?

DR. GERBA: Well, actually, there's a -- there may actually be 52. Some people are pushing another one, so -- I should point out too, not all the adenoviruses have been clearly associated with disease in humans, by the way, too.

Although, they've been found in human fluids and stools and infected with some people, we're not -- we're still not certain whether it involved and caused any type of particular disease in humans beings.

MS. ALEXANDER: Now, if I'm understanding you correctly, the PCR analysis that you used for the confirmation, detected more stains of adenovirus than the cell culture analysis; is that correct?

DR. GERBA: No, the -- in this case, we used PCR to confirm the presence of adenovirus growing in the cell culture. We only detected viable adenoviruses in this

study.

The PCR here was done on the cell culture as an identification step that we were finding adenovirus. For the norovirus, it was -- we need to not determine viability. We just determined the concentration of the adeno -- norovirus genome in that case.

MS. ALEXANDER: Can you just clarify what it means when you say that you confirmed, then, using the PCR analysis? You did the cell culture, you identified the sample as testing either positive or negative through the cell culture for those specific serotypes you identified; correct?

DR. GERBA: Right. What happened with the cell culture -- enteroviruses also have the capability of growing in the same culture we use to isolate adenoviruses. So we wanted to make sure we had an adequate number on the number of adenovirus growing in the cell culture.

If you look at the raw data, not all samples confirmed as adenoviruses, which were probably -- and some of these were

probably enteroviruses growing in the cell culture.

MS. ALEXANDER: Okay. So let's say you tested the sample, were using the cell culture and it was positive, but you did the PCR analysis and it was negative. That would suggest that what was growing there might have -- was probably, or perhaps, enteroviruses rather than adenoviruses; correct?

DR. GERBA: It could be. Or some other type of virus it be could. But there were not many of them, because the adenoviruses tend to grow very well in this type of cell culture, more than other virus types, apparently.

MS. ALEXANDER: When that happened, did you go back and check what it was that was growing in there that wasn't adenovirus?

DR. GERBA: You know, I think at random we did. I don't know if we did it in this study. In other studies we have done -- we've been looking at water and waste water.

And I have to go and look at the

notebooks if we looked at a few of those or not. In other studies they've always -- or not always, but some of them turned out to be enteroviruses or viruses we can't identify.

MS. ALEXANDER: Am I correct that there were at least some instances where in a sample you identified it as negative for enterovirus, when testing for enterovirus, it was positive in a cell culture for adenovirus, confirmed as negative, and, therefore, counted as negative for adenovirus, but you didn't go back to check whether there were enteroviruses in there?

DR. GERBA: We just counted -- it was viral cytopathogenic effects.

MS. ALEXANDER: Okay.

DR. GERBA: We do that as a minimum.

MS. ALEXANDER: So in other words, just to summarize, there were, at least in some cases, where you found something to be growing in the cell culture but you counted it as a negative and didn't follow up to see what exactly it was that was growing in there?

DR. GERBA: No. Because we already had an assay on BGM cells that worked well for enteroviruses, and we could be double counting the virus.

MS. ALEXANDER: But isn't it a fact that there were at least some situations where you got a negatives specifically on the enterovirus assay, but you got a positive on the cell culture for adenovirus that you confirmed it negative for adenovirus, so it could have been enterovirus instead of --

DR. GERBA: No. What we did is --

MS. ALEXANDER: -- that was going in there?

DR. GERBA: -- if there was viral cytopathogenic effects, we took that sample and then we did PCR analysis to determine whether it was an adenovirus or not.

MS. ALEXANDER: Right. And if it wasn't but there was still something growing in there, that could have been enterovirus; correct?

DR. GERBA: That is a possibility, yes.

MS. ALEXANDER: Okay.

All right. This is Tolson 21 and Gerba 29. And this refers to Tables 3-5(a) through (f) of Exhibit 71, Risk Assessment. These are the enteric virus results.

Can you please describe for me the method used to detect enteric viruses? Just summarize as you did with adenoviruses, please.

DR. GERBA: Right. Enteric viruses -- we're using that term interchangeably with total culturable viruses.

Certain enteric viruses used a lot before molecular methods came in to detect viruses in water. So now there's a tendency to total culturable viruses.

Because, basically, the EPA method we used for that, before looking for it, that's using the BGM cell line. You put your sample on the BGM cell line and then you look for the production of cytopathogenic effects that are viral, and you confirm those through another passage.

And then those are called total

culturable virus.

MS. ALEXANDER: Okay. One second.

Okay. Referring to Exhibit 71, Page 48, Section 3.3.1. That contains a description of this method that you're discussing.

In the first paragraph there you characterize Tables 3-5(d) through (f) as presenting a summary of the wet weather total enteric virus analytical results. Is the method you describe capable of detecting total enteric viruses, as in all of them?

DR. GERBA: Total culturable enteric. That's a term that's used in the literature for EPA. And EPA uses that, too.

But largely, you're really just detecting the enteroviruses. Although some real viruses and other virus types my grow in there.

But that's a terminology that's come into use.

MS. ALEXANDER: Okay. And hepatitis is an enteric virus; correct?

DR. GERBA: That's correct.

MS. ALEXANDER: And you didn't assay for that?

DR. GERBA: No, we did not.

MS. ALEXANDER: And the same with rotavirus?

DR. GERBA: No, we did not assay.

Hepatitis A we did not assay for, because the concentration would be expected to be low because the incidence is fairly low. And hepatitis A there's a vaccine now, which is also driving down the incidence of hepatitis A in the United States. The probability of finding that was pretty low.

For rotaviruses, the feeling was that the methods were not very good for looking for rotavirus. There's a cell culture method -- and I developed one of the methods -- it's been used before, and it's very difficult to use.

And the volumes you could actually assay out of it, I felt, were too small to really give us any meaningful results to actually do rotaviruses. So that's why we kind of decided against that.

MS. ALEXANDER: Okay. Turning to Tolson 22 and Gerba 30. And this is regarding the statement in Exhibit 71, the risk assessment that reverse transcription-polymerase chain reaction, RT-PCR, results were used to calculate concentrations of noroviruses in the sample.

Can you just give a brief summary of how those calculations were performed, please?

DR. GERBA: These calculations are done very similar to most probable number calculations for the coliforms, fecal coliforms, the bacteria that are often used. You take a delusion series of your sample and you look for the number of positives and negatives and then you feed that into a -- on a computer program developed by Hurley and Rosco back in 1983.

It calculates the most probable number of concentration in the sample that you are assaying. It's, basically, doing the same thing as doing a most probable number for fecal coliforms.

MS. ALEXANDER: Okay.

And just to kind of make sure I understand this properly, this RT-PCR process tells you how many copies of norovirus RNA there are in your sample; is that right?

DR. GERBA: You have to do it by a delusion series. It's a positive negative one.

You could do that by using quantitative PCR. But I felt it didn't have the sensitivity we needed, so we do the most probable number.

In other words, the sample is positive or negative as you dilute it. In other words, you take an unconcentrated sample and you dilute it one to ten, one to 100 and one 101,000.

And you are basically looking for an extinction point. You no longer find the positive PCR reaction in a sample that is diluted out far enough. And you do that usually at least in triplicate.

MS. ALEXANDER: Okay. Did this analysis involve an assumption as to the

number of copies of norovirus RNA that are associated with the presence of a certain amount of norovirus? Does that question make sense?

DR. GERBA: Yeah. Usually one genome equals one virus, it's believed.

MS. ALEXANDER: One to one?

DR. GERBA: One to one.

MS. ALEXANDER: Okay.

Help me understand the statement in the Risk Assessment, Exhibit 71, then, that the ratio of he genomes, paren, (the viron self-culture infectivity units) is one to 100 to one to 46,000.

DR. GERBA: That varies with the cell culture line you're using.

In other words, if I took -- you adapt viruses to cell culture. If I took a virus, like rotavirus in a stool sample and put in a cell culture sample, the ratio may be one to 40,000 -- 40,000 genomes to one virus.

If you adapt that to cell culture over time or use vaccine strains maybe you're

looking for, that may be down to one in a hundred. The cell culture doesn't necessarily detect all the viruses that are in the sample.

MS. ALEXANDER: I'm sorry, how does this fit in with your testimony concerning the one-to-one ratio? Am I comparing apples and oranges? Is that a different thing?

DR. GERBA: I think you are. The conservative thing would be to consider each genome one norovirus. One, because this picks up inactivated organisms.

MS. ALEXANDER: So you're saying the conservative thing would be to consider it one to one. But am I understanding correctly from the risk assessment, Exhibit 71 on Page 48, that, in fact, you used a ratio of one to 100 to one to 46,000?

DR. GERBA: The reason for that is because the only dose response data we have is for cell culture, where the ratio is one to a hundred. So, in other words, the echovirus ratio was 100 genomes to one infectivity unit. That's what was done in a

dose response curve.

So that's why it was benchmarked against that. Because we know from the echovirus data that for every hundred genomes, we would have one infected unit. And that was used to develop the dose response curve.

MS. ALEXANDER: Wouldn't it make a pretty big difference in your overall results, whether you use one to 100 or one to 46,000 -- in other words, in terms of how many virus you're assuming or correlated with the number of genomes you found?

DR. GERBA: Of course. I mean, just changing that ratio, you could make that ratio over to a wide number of things. But in this example, we had something to benchmark it again, so we were trying to bring reality into the risk assessment.

MS. ALEXANDER: So you were saying you were benchmarking it against the one-to-one ratio from the dose response data?

DR. GERBA: No, the 100. Because that was what we had based on the dose response

data which was developed in cell culture.

In other words, when they developed the dose response data, they used the infectivity in cell culture of the echovirus. And they that for every hundred genomes, approximately, they had one infectious virus in cell cultures what they did the dose response against.

So what we did is try to benchmark it against a real situation where we actually knew what the ratio was and we had a dose response curve to go with it.

MS. ALEXANDER: All right. So you opted against using the one to one because of this dose response data that you had?

DR. GERBA: Right. And we could have used the one to 40,000, for example, which could have been used, too. Because that's about what the ratio from the stool sample for, say, rotavirus is to an infectivity in a human being.

So this was the range that we picked.

MS. ALEXANDER: Okay. Not to beat the

dead horse, but just so I understand, the most conservative assumption you could of made would be one to one, the least conservative would be one to 46,000, you chose --

DR. GERBA: Right.

MS. ALEXANDER: -- the one to 100?

DR. GERBA: That's right. That's correct.

MS. ALEXANDER: Okay. Moving onto Tolson 23 and Gerba 21, they are the same question.

Did the secondary infection rates that you used in your analysis change between the interim dry weather risk assessment completed in November 2006 and the final wet and dry weather risk assessment?

DR. TOLSON: Yes, it did.

MS. ALEXANDER: Okay.

I'm going to present a document and have it marked as an exhibit, just so the rest of the room can understand what we're talking about.

(WHEREUPON, a certain document was

marked Exhibit No. 76 for identification, as of 9/9/08.)

MS. ALEXANDER: What I have here to present as the exhibit is the cover page from the interim dry weather risk assessment dated November 2006 and then the relevant table that I'll be talking about, which is Table 4-6.

THE HEARING OFFICER: I've been handed Prepared For Protecting Our Water Environment Metropolitan Water Reclamation District of Greater Chicago, Interim Dry Weather Risk Assessment and Human Health Impact Disinfection Versus No Disinfection of the Chicago Area Waterway System.

If there's no objection, I'll mark this as Exhibit 76.

Seeing none, it's Exhibit 76.

MS. ALEXANDER: Okay. Specifically I would like to compare this table -- I'm sorry -- this was marked as No. 76 to -- which is Table 4-6 in the dry weather risk assessment to table 5-6 in Exhibit 71.

DR. TOLSON: Okay. I'm with you.

MS. ALEXANDER: I'm not with you yet, hold on.

And I would point out, correct me if I'm wrong, that several -- a couple of the numbers in the interim assessment are higher than the numbers in Table 5-6. That's comparing Exhibit 76, Table 4-6 to Exhibit 71, Table 5-6. And specifically the entries for salmonella and E. Coli.

DR. TOLSON: Also total enteric viruses, yes.

MS. ALEXANDER: Now, the lower numbers that are contained in the later iteration, the wet and dry weather risk assessment, for infectivity -- or, I'm sorry, secondary attack rates, would, in fact, have the effect of lowering overall risk; is that correct?

DR. TOLSON: It is correct that if the lower the secondary attack rates, the higher the risk.

MS. ALEXANDER: Okay.

DR. TOLSON: If you'd like, I can explain the rationale --

MS. ALEXANDER: Yes.

DR. TOLSON: -- for this if you --

MS. ALEXANDER: You anticipated my next question, which is what was the basis for these changes.

DR. TOLSON: Sure. The interim report -- and it wasn't interim drafts, sort of a product here -- we assumed a 50 percent attack rate, which is a fairly conservative assumption. As we refined our estimates, we gathered additional data and took a look at what was available in the literature to sort of hone in to get a better estimate of what those would be.

For example, for total enteric viruses, we assumed 50 percent. After some additional conversations with Dr. Gerba, we settled on 25 percent as a conservative, sort of, assumption for transmission.

For adenoviruses and caliciviruses, it looked like we kept those the same from our initial assessment. The crypto and giardia results that we had in the interim are actually reversed. So they're corrected in the final.

But I'd like to point out that the -- for the giardia results, the literature reported from eight to ten percent, we actually assumed 25 percent, which is conservative beyond what the literature cites. And then, for salmonella and for E. coli, we changed our default assumption to 25 percent, which we thought was still an overly conservative estimate of the secondary attack rates for those organisms.

If you've noticed, we actually did cite some literature below. And I think in every case, the literature cited value is lower or within the range of the values that we use with our -- as our input assumptions.

MS. ALEXANDER: Okay.

Tolson 24 and Gerba 32. Did you, in fact, use a Monte Carlo simulation in quantifying risk?

DR. TOLSON: That is correct, we used the Monte Carlo simulation.

MS. ALEXANDER: Can you provide a brief description of what you did in that

simulation?

DR. TOLSON: Monte Carlo simulations are the mathematical tool to solve problems that don't have an easy analytical solution. You can't just add the numbers up and come up with the equal sign and get a final number.

It uses simulations to estimate what the final results would be. The process used here, we use Monte Carlo simulation where we selected from our data set -- and this means our data set of dry weather days, wet weather CSO days -- to represent each simulation's waterway pathogen concentrations.

And then we did simulations of a million recreational users, drawing individuals from distributions that included canoeists, fishing and boating, in relation to the proportion for which they are represented in the UAA study.

DR. GERBA: If I can point out, in microbial risk assessment, that's common practice to use Monte Carlo simulations. You get a better idea what the distribution of

risk is.

MS. WILLIAMS: Can I just ask, have you done this before though? Have you done a Monte Carlo simulation for microbial risk assessment before?

DR. TOLSON: I teach a class on probabilistic risk assessment, a graduate level class, at University of Florida. This is a component of one of the things that I teach within that class, a number of probabilistic risk assessments historically. So yes.

MS. WILLIAMS: But I'm just specifically distinguishing between microbial risk versus other types of toxic chemical risks. Was that reflected in your answer?

DR. TOLSON: The assessment, sort of, parameters are pretty much the same. My microbial risk assessment experience, I have not relied on probabilistic methods for that, but --

MS. WILLIAMS: Until now?

DR. TOLSON: That is correct.

MR. ANDES: Can you explain a little

bit more about this methodology?

DR. TOLSON: The methodology is common methodology that's employed by the agency and others to sort of assess risk. I have been involved in numerous workshops where we've discussed these, sort of, risk assessment techniques in a very fast style in sort of doing them, so...

DR. GERBA: I've been involved in a number of teams doing simulations for microbial risk assessment. It's really just a mathematical technique.

You put different numbers in is all you're doing.

MS. ALEXANDER: So just to summarize, in other words, the point of a Monte Carlo simulation is to account for a distribution spread of input variables; is that basically correct? In other words, you could account for the fact that there's no exact amount of water that every recreator is going to ingest, but it's rather a range of possibilities? Is that basically right?

DR. TOLSON: That is correct.

MS. ALEXANDER: Okay.

DR. TOLSON: The alternative is to do point estimates for all the inputs and develop one point estimate, which takes into account the averages of everything. And the way we did it takes into account the ranges and gives us sort of a range of outputs.

MS. ALEXANDER: Can we turn to figure 5-2 in Exhibit 71, the risk assessment?

THE HEARING OFFICER: Excuse me. Let's go off the record for just a second.

(WHEREUPON, discussion was had off the record.)

THE HEARING OFFICER: Back on the record.

MR. ANDES: First the appendices to the risk assessment report I have on a disk, if I could give you that right now.

THE HEARING OFFICER: Is that all four appendices?

MR. ANDES: Yes, A, B, C and D.

THE HEARING OFFICER: We'll mark that as Exhibit 77.

MR. ANDES: I also have, both paper

and on a disk, the attachments to the EPA July 31st, 2008 Melser letter.

MS. MEYERS-GLEN: I'm sorry, we didn't catch that. Could you say that again, please? Attachment what?

MR. ANDES: The attachments to the EPA letter of July 31st.

THE HEARING OFFICER: Exhibit 77 is the risk assessment appendices. If there's no objection?

Seeing none, it's Exhibit 77.

(WHEREUPON, a certain document was marked Exhibit No. 77 for identification, as of 9/9/08.)

THE HEARING OFFICER: Exhibit 78 will be the CD-ROM that is the appendices to the USEPA letter that was previously admitted as CD-ROM 73. Exhibit 73.

MR. ANDES: The last document on that CD-ROM, July 31st, 2008.

THE HEARING OFFICER: All right. So wait a minute.

Instead of -- I'm going to do something I don't normally do. I'm going to

enter this as Exhibit 73A. So that it will be clear than it goes with Exhibit 73.

And this the appendices to the letter, which was one of last documents on the CD-ROM that is Exhibit 73. So this will be Exhibit 73A. If there's no objection?

MS. WILLIAMS: I'm just trying to figure out what I have. Is that what I have? Or do I have both?

THE HEARING OFFICER: He gave you two --

MS. WILLIAMS: We have one disk, I don't know what's on it. What is this?

MR. ANDES: Those are the attachments to the EPA July 31st, 2008 letter.

MS. WILLIAMS: So that's 73A?

THE HEARING OFFICER: 73A.

MS. WILLIAMS: Thank you.

THE HEARING OFFICER: And 77 is the appendices, which he gave us both the hard copy and on CD. Or which I have both hard copy and CD.

So I'm going to mark the hard copy also, again strangely enough, as 77A.

Because 77 is the disk. There's no objection?

MR. ANDES: Let me clarify. The appendices that I gave you -- the disk marked appendices is 71; isn't it? Isn't the risk assessment report 71?

THE HEARING OFFICER: Yes, but I'm going to mark them as 77. Because I don't normally give subsets, but I also then have the same thing in hard copy, I'll call it 77A.

MR. ANDES: All right. Fine.

MS. WILLIAMS: Now, with that --

THE HEARING OFFICER: Okay. Wait a minute, I'm confused.

These are not the appendices to --

MR. ANDES: The appendices to the risk assessment report are only on that disk that says Appendices.

THE HEARING OFFICER: These (indicating) are what goes with this (indicating), are the attachments?

MR. ANDES: Yes.

THE HEARING OFFICER: Okay.

I'm not marking them as an exhibit. We have them on CD, these will be for our use.

So Exhibit 77 is the appendices to the risk assessment, and 73A is the appendices to the letter. I am thoroughly confused, but I think I've got it.

All right. No objections?

Those are entered.

(WHEREUPON, a certain document was marked Exhibit No. 73A for identification, as of 9/9/08.)

MS. WILLIAMS: So at this point, though, you have copies of -- you have a disk with appendices, Ms. Alexander has a disk with appendices. Can we just request that the Board upload this exhibit in particular, or no?

THE HEARING OFFICER: I have to be perfectly honest with you, John is out this week.

MS. WILLIAMS: No, I don't mean --

THE HEARING OFFICER: I was going to say, so I can't promise you when this would

get done.

Is it possible to get another CD burned?

MR. ANDES: Yeah. If I don't already have one, I can certainly burn another.

MS. WILLIAMS: Either one.

THE HEARING OFFICER: And we might be able to burn a CD faster than we can get it uploaded, given our staffing concerns this week.

MS. WILLIAMS: Either way.

THE HEARING OFFICER: All right. That being said, we will start again tomorrow morning with Ms. Alexander.

Drs. Gerba, Tolson, Petropoulou, thank you very much.

We're adjourned.

(WHEREUPON, the hearing was adjourned until 9/10/08 at 9:00 a.m.)

STATE OF ILLINOIS)
) SS:
COUNTY OF COOK)

I, SHARON BERKERY, a Certified Shorthand Reporter of the State of Illinois, do hereby certify that I reported in shorthand the proceedings had at the hearing aforesaid, and that the foregoing is a true, complete and correct transcript of the proceedings of said hearing as appears from my stenographic notes so taken and transcribed under my personal direction.

IN WITNESS WHEREOF, I do hereunto set my hand at Chicago, Illinois, this 18th day of September, 2008.

Certified Shorthand Reporter

C.S.R. Certificate No. 84-4327.