

1           BEFORE THE ILLINOIS POLLUTION CONTROL BOARD

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3       IN THE MATTER OF:

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5       WATER QUALITY STANDARDS AND    )

6       EFFLUENT LIMITATIONS FOR       )

7       THE CHICAGO AREA WATERWAY     )

8       SYSTEM AND THE LOWER           )

9       DES PLAINES RIVER:             ) No. R08-9

10      PROPOSED AMENDMENTS TO        )

11      35 Ill. Adm. Code Parts         )

12      301, 302, 303 and 304         )

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15                   REPORT OF PROCEEDINGS had before the  
16       ILLINOIS POLLUTION CONTROL BOARD held on  
17       September 10, 2008, at 9:00 o'clock a.m. at the  
18       Thompson Center, Room-9-040, Chicago, Illinois.

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1 A P P E A R A N C E S:

2 ILLINOIS POLLUTION CONTROL BOARD:

3 MS. MARIE TIPSORD, Hearing Officer

4 MR. TANNER GIRARD, Member

5 MR. ANAD RAO, Senior Environmental Scientist

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7 ILLINOIS ENVIRONMENTAL PROTECTION AGENCY:

8 Ms. Stefanie Diers

9 Ms. Deborah Williams

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1                   CHAIRMAN TIPSORD: Good morning. We're  
2 back again. My name is Marie Tipsord. And I'm  
3 not going to go through the whole spiel, but this  
4 is the Water Quality Standards and Effluent  
5 Limitations for the Chicago Area Waterways System  
6 and Lower Des Plaines River, proposed amendments  
7 to 35 Il. Admin Code 301, 302, 303, and 304 docket  
8 number R08-9.

9                   To my right is Dr. Tanner  
10 Girard. He is the presiding Board member in this  
11 matter. To my left is Dr. Anand Rao, from our  
12 technical staff. Board members Nicolas Melas and  
13 Andrew Moore will be joining us later today. Both  
14 had emergencies arise.

15                   Which brings us to, at the close  
16 of yesterday we were still with the Natural  
17 Resource Defense Counsel, Ms. Ann Alexander, who  
18 also has had an emergency and will join us later.  
19 So rather than finishing her questions this  
20 morning, we'll go to the IEPA. So that leaves us  
21 with our panel, which is Dr. Petropoulou,  
22 Dr. Tolson, Dr. Gerba. I'll remind you all, you  
23 were sworn in yesterday, and you are still sworn  
24 in today. I apologize for the out of order

1 nature, but we're going to go to the IEPA who is  
2 going to ask you some questions.

3 MS. DIERS: I'm going to begin with  
4 my pre-filed questions for Dr. Petropoulou. I'm  
5 going to start with number one.

6 You state in your pre-filed  
7 testimony the following: "For the last three  
8 years I have been the project manager for the  
9 Metropolitan Water Reclamation District of Greater  
10 Chicago's Microbial Risk Assessment Study. I have  
11 been intimately involved in every aspect of the  
12 MRA study." When you state, "The main objective  
13 of the MRA study was to evaluate the human health  
14 impact of continuing the current practice of not  
15 disinfecting the effluents from the North Side,  
16 Stickney and Calumet water reclamation plants  
17 versus initiating disinfection of the effluents at  
18 these three plants"; did you formulate that  
19 objective?

20 DR. PETROPOULOU: I did not.

21 MS. DIERS: Who did formulate that  
22 objective?

23 DR. PETROPOULOU: That objective,  
24 the way you stated your question, was formulated

1 in the request for the proposal that the District  
2 should conduct a study.

3 MS. DIERS: Do you know when that  
4 was put together?

5 DR. PETROPOULOU: I believe it was  
6 issued in January of 2005. I don't recall the  
7 exact date that they issued the RFP.

8 MS. DIERS: If I understand, you  
9 said the district formulated the objective; is  
10 that correct?

11 DR. PETROPOULOU: I don't know if  
12 the district alone formulated the objective or  
13 they had a panel that worked preparing the request  
14 for the proposal, but it was in the request for  
15 the proposal.

16 MS. DIERS: So as you are sitting  
17 here today, you don't know what individuals were  
18 involved in that?

19 DR. PETROPOULOU: No.

20 MS. DIERS: I'm going to skip to  
21 question eight. And ask about how the sampling  
22 locations were chosen for this study? Could you  
23 just explain that?

24 DR. PETROPOULOU: Yes. We selected

1 a subset of the District's ambient water quality  
2 stations for the sampling that we did during dry  
3 and wet weather for this study, and we also  
4 collected samples at the outfalls and at the  
5 pumping stations at the each of the Calumet, North  
6 Side and Stickney waterway segments as well.

7 MS. DIERS: When you were talking  
8 about sampling at the pumping stations during the  
9 wet weather events, can you explain was the  
10 sampling performed at the outfall itself or was it  
11 near the waterway near the stations?

12 DR. PETROPOULOU: Okay. It was at  
13 the North Side -- because the sampling crew for  
14 safety reasons, they could not go close to the  
15 outfall of the pumping station. Based on the  
16 boat's captain, they made the decision that the  
17 safest location to anchor the boat and collect the  
18 samples was at the Wilson Avenue ambient water  
19 quality station. At the Stickney waterway segment  
20 upstream of Stickney actually at the Racine Avenue  
21 pumping station, I believe the sample was  
22 collected at the south fork or public station at  
23 35th Avenue. So that was the most approximate  
24 location they could collect that samples. And for

1 the 125th Street pumping station, the sample was  
2 collected downstream. I believe it was Halsted  
3 Avenue.

4 MS. DIERS: On page 2 and 3 of your  
5 pre-filed testimony you list three specific  
6 objectives of the 2005 dry weather samplings. Can  
7 you explain when these objectives were formulated,  
8 and were you involved in formulating those  
9 objectives?

10 DR. PETROPOULOU: These objectives  
11 were formulated at the kick-off meeting for the  
12 project. After we were awarded the project we met  
13 with the District and we formulated the  
14 objectives.

15 MS. DIERS: Do you recall what  
16 individuals were involved in formulating the  
17 objective?

18 DR. PETROPOULOU: It was the  
19 GeoSyntec team, which includes, as I defined  
20 yesterday in my testimony, GeoSyntec, our experts  
21 and also the District. Like we met with the  
22 District and we discussed the objectives of the  
23 study.

24 MS. DIERS: And would that be the

1 same for the 2006 wet weather sampling?

2 DR. PETROPOULOU: It's very  
3 similar. The objectives for the 2006 study were  
4 actually not different at the beginning. At the  
5 beginning we set the same objectives. The idea  
6 was that in 2005 we would collect samples, and we  
7 would -- we anticipated that we would account for  
8 both dry and wet weather, but when we did the  
9 sampling, we realized that we didn't capture wet  
10 weather events, and the district decided to extend  
11 the study in 2006. So the objectives for the most  
12 part remained the same except we added an  
13 additional set of sampling events to capture  
14 specifically wet weather in 2006?

15 MS. MEYERS-GLEN: May I actually ask  
16 a follow-up question.

17 CHAIRMAN TIPSORD: You need to speak  
18 up. They can't hear you in the back of the room.

19 MS. MEYERS-GLEN: With the pumping  
20 stations I had a quick question. At 125th Street,  
21 the pumping station -- actually it's two. You  
22 said the sample was collected downstream at  
23 Halsted Avenue. How far approximately is that,  
24 the location it was sampled from, from the outfall

1       itself?

2                       DR. PETROPOULOU: I don't know the  
3       exact mileage from that.

4                       MS. MEYERS-GLEN: Approximately.

5                       DR. PETROPOULOU: But I can tell  
6       you -- I know the distance from the Calumet  
7       outfall to Halsted, which is about 1.1 miles. I  
8       can't give you a number. I don't know.

9                       MS. MEYERS-GLEN: Do you know  
10      approximately? I know you can't give me by feet,  
11      but is it like a mile, two miles, a quarter of a  
12      mile? I'm just trying to get a sense of how far  
13      away from the outfalls this actually was sampled  
14      for the purpose of outfalls.

15                      MR. ANDES: I'm sure we can provide  
16      that answer.

17                      DR. PETROPOULOU: Right. I just  
18      don't have that number. Again, just to clarify,  
19      we wanted to capture the effect that the pumping  
20      stations had in the waterway. So we went  
21      approximately as close as we could to the  
22      outfalls, but it wasn't the idea to capture the  
23      outfall itself for the risk assessment. Because  
24      what really matters is what goes in the waterway.

1 That's what the recreational users are exposed to.

2 MS. MEYERS-GLEN: The highest  
3 concentration though, if you are going to be  
4 capturing it, would actually com from the outflow  
5 itself, correct?

6 DR. PETROPOULOU: Right. Assuming  
7 that the recreational user would be exposed to the  
8 outflow of the pumping station discharge, which is  
9 not very likely.

10 MS. MEYERS-GLEN: But it is  
11 possible?

12 DR. PETROPOULOU: I don't know if  
13 it's possible. I know that the sampling crew  
14 could not get very close to collect the samples  
15 for safety, so based on that I assume it's  
16 unlikely.

17 MS. MEYERS-GLEN: There are 230  
18 CSOs, correct?

19 DR. PETROPOULOU: I'm talking about  
20 the pumping stations. I'm not talking about the  
21 CSO outflows.

22 MR. ANDES: Yes, I think we might  
23 want to clarify. Which outfalls are we talking  
24 about?

1 DR. PETROPOULOU: Just pumping  
2 stations.

3 MS. MEYER-GLEN: If it's possible my  
4 two questions were going to be in the proximity of  
5 the other two points, so instead of doing that, if  
6 you were going to provide me with that in the  
7 first one, if you could provide me with all three  
8 instead of going through those questions?

9 DR. PETROPOULOU: All right.

10 MS. DIERS: I want to go back and  
11 clarify when we were talking about the objective  
12 from 2005 to 2006. When I'm looking at your  
13 pre-filed testimony you had three objectives for  
14 the dry, and then there were four for the wet  
15 weather. So in 2006 did you add the objective  
16 that's number four on page 3 of your pre-filed  
17 testimony, does it quantify any reduction of risk  
18 that would result from disinfecting plant  
19 effluents during wet weather?

20 CHAIRMAN TIPSORD: For the record  
21 that's Exhibit 68.

22 DR. PETROPOULOU: That objective was  
23 the same both in dry and wet weather.

24 MS. DIERS: Could you explain what

1 objective changed in 2006 --

2 DR. PETROPOULOU: We added an  
3 additional objective, which is objective number  
4 one under wet weather, and that was to account for  
5 any influx of the increased flow, the wet weather  
6 flow from the treatment plants would have on the  
7 microbial flow from the waterway.

8 MR. ANDES: If I could just  
9 follow-up. Two questions. First, am I correct to  
10 say that the 2005 initial objective included  
11 looking at wet versus dry, there simply weren't  
12 wet weather events in 2005; is that correct?

13 DR. PETROPOULOU: Yes.

14 MR. ANDES: And then the additional  
15 objective that was added of wet weather, was to  
16 consider the additional wet weather flow through  
17 the treatment plants?

18 DR. PETROPOULOU: Correct.

19 MR. ANDES: Okay, thank you.

20 MEMBER RAO: Just as a follow-up to  
21 that. This additional objective that you added,  
22 it states, "To evaluate the impact of reclamation  
23 plant wet weather flow on microbial quality of the  
24 plants outfall." So did you sample the outfall or

1 it was still in the waterway?

2 DR. PETROPOULOU: We collected  
3 samples in the outfalls both during dry and wet  
4 weather.

5 MEMBER RAO: For all three plants?

6 DR. PETROPOULOU: Correct.

7 MS. DIERS: I'm on question 16. Why  
8 did you take dry weather measurements at the  
9 surface at one meter depth, but not take wet  
10 weather measurements at the same depth?

11 DR. PETROPOULOU: We collected  
12 samples at the surface at one meter depth during  
13 dry weather and we looked at the results to see if  
14 there was a statistical difference between the one  
15 meter depth surface, and we found there was not.  
16 So during wet weather we collected samples at the  
17 surface.

18 MS. MEYERS-GLEN: Can I ask one  
19 follow-up? You guys testified yesterday that you  
20 didn't take into account temperature, is that  
21 correct?

22 CHAIRMAN TIPSORD: The trains go by  
23 and we can't hear you at all up here.

24 MS. MEYERS-GLEN: You stated

1 yesterday, however, that in the study you did not  
2 take into account temperature as one of your  
3 parameters, is that correct?

4 DR. PETROPOULOU: We didn't  
5 correlate the results to temperature, but we did  
6 measure the results of the temperatures to the  
7 waterways.

8 MS. MEYER-GLEN: But that wasn't a  
9 factor in the risk assessment?

10 DR. PETROPOULOU: No, it was not.

11 MS. DIERS: I'm on question 19 now.  
12 Explain what you mean by the statement on page 6  
13 of your pre-filed testimony that states, "The  
14 results indicate that there are no significant  
15 correlations between dry weather fecal chloroform,  
16 indicator bacteria and pathogens. The wet weather  
17 results indicate that there is a better  
18 correlation between fecal chloroforms and other  
19 indicator bacteria and pathogens.

20 DR. PETROPOULOU: We looked at the  
21 correlation statistics between the different  
22 bacteria types, which included the three  
23 indicators that we did, E. Coli, fecal chloroform  
24 and enterococci with pseudomonas and Salmonella.

1 The only other two bacteria that we analyzed which  
2 are pathogens, and we found there was no  
3 statistical correlation. There was no good  
4 statistical correlation between the indicators and  
5 the pathogens, and that included indicators such  
6 as fecal chloroform, enterococci and E. Coli lie  
7 with the pathogens.

8 MS. DIERS: Do you know why you were  
9 seeing that type of -- I guess, do you know why  
10 you were seeing that type of statistical analysis  
11 when you did your study?

12 DR. PETROPOULOU: You mean for the  
13 wet weather?

14 MS. DIERS: Yes.

15 DR. PETROPOULOU: Or for the dry?

16 MS. DIERS: For both. Can you  
17 explain wet and then dry?

18 DR. PETROPOULOU: For the wet  
19 weather we found that there was actually better  
20 correlation between the indicators and between the  
21 indicators and the pathogens. And when I'm  
22 talking about pathogens, I'm referring again to  
23 bacteria data. We did this analysis only for  
24 bacteria. Now, why we find the better correlation

1 between -- during wet weather samples, I can't  
2 tell you why. I didn't research that subject.  
3 Perhaps Dr. Gerba can speculate why the wet  
4 weather data correlates better than the dry  
5 weather.

6 DR. GERBA: Might be fresher  
7 materials. It's probably not treated sewage.  
8 When you go through sewage treatment and waste  
9 water treatment, the ratios between the indicators  
10 and pathogens may change because there's  
11 differences in removal rates by the waste water  
12 treatment. When you are doing wet water flows,  
13 basically from the surface sources, you are  
14 getting raw waste water. So there's probably  
15 going to be better correlation. There's no  
16 differential for the survival of that process. I  
17 would expect a much better correlation through the  
18 wet weather for that reason because it's fresher  
19 stuff and not treated.

20 MS. DIERS: Can you explain why you  
21 noticed significant differences in the E. Coli and  
22 enterococci results by site during wet weather?

23 DR. PETROPOULOU: Again, that was a  
24 factual finding in our statistical analysis. We

1 found that when we tested the data with the Nova,  
2 the tests -- whether the data are the same or they  
3 are not, we found that the set that describes E.  
4 Coli and enterococci are different by site, which  
5 means they are different statistically between  
6 North Side, Stickney and Calumet.

7 MS. DIERS: But you had no  
8 indication as to why?

9 DR. PETROPOULOU: No, that was  
10 outside our study. We didn't do forensics to find  
11 out why.

12 MS. DIERS: Did you look at whether  
13 or not indicator organisms other than fecal  
14 chloroforms had better correlations with the  
15 pathogens during dry weather?

16 DR. PETROPOULOU: We did. We looked  
17 at correlations between all bacteria types.

18 MS. DIERS: What are the results of  
19 those?

20 DR. PETROPOULOU: Similar to what we  
21 found between E. Coli. There were other  
22 indicators between the pathogen types that we  
23 looked at.

24 MS. DIERS: I'm on 23. On page 5 of

1 your pre-filed testimony you state, "Results  
2 indicate the concentrations of bacteria, viruses  
3 and protozoa in the waterway increase during wet  
4 weather conditions." Will the bacteria, viruses  
5 and protozoa that are present and due to CSOs  
6 decrease as CSO flows are decreased or eliminated  
7 with a completion of TARP?

8 DR. PETROPOULOU: Is this a  
9 hypothetical question?

10 MR. ANDES: Her study didn't really  
11 deal with TARP in any way.

12 MS. DIERS: Do you have an opinion?

13 DR. PETROPOULOU: I don't know  
14 enough about the TARP to express an opinion.

15 MS. DIERS: Is that the same for the  
16 other individuals sitting on the panel; do you  
17 have an opinion about the impact with TARP?

18 DR. TOLSON: I don't know what  
19 frequency CSOs would occur from the TARP.

20 DR. GERBA: I don't have enough data  
21 to do that anyway to make a judgment.

22 MS. WILLIAMS: I'm going to ask a  
23 couple follow-up on the sampling since these are  
24 the questions for Dr. Petropoulou.

1 I'm looking at pages 12, 13, and  
2 14 of the report. Maybe if you turn to that, it  
3 would be --

4 CHAIRMAN TIPSORD: That's  
5 Exhibit 71?

6 MS. WILLIAMS: Exhibit 71 --  
7 -- it would be more helpful.

8 I'm trying to understand some of  
9 the differences here about how the sampling  
10 stations are defined. First on page 13, under  
11 upstream sampling locations, number one, it says,  
12 "NSC Oakton Avenue also known as WW102 sampling  
13 location 3," and then it says, "8200 feet or  
14 1.6 miles." Do you see that?

15 Then if you turn to page 15,  
16 under "Upstream of North Side Water Reclamation  
17 Plant at the NSC, it says NSC Oakton Avenue, again  
18 also known as WW102, sampling location three. And  
19 then it says, "2800 feet or .5 miles from the  
20 Water Reclamation" -- can you explain that?

21 DR. PETROPOULOU: It's probably the  
22 number was transposed instead of 8200 feet.  
23 Obviously it's an inconsistency. I would have to  
24 verify which one is the distance.

1 MS. WILLIAMS: You don't know  
2 whether that distance was 8200 or 2800 feet  
3 upstream?

4 DR. PETROPOULOU: I don't know.

5 MS. WILLIAMS: Will the District be  
6 able to supplement that after the hearing into the  
7 record?

8 DR. PETROPOULOU: Yes.

9 MS. WILLIAMS: I think I have the  
10 same issue -- look at page 13 again. Let's look  
11 at number 3, "CSC Indiana Avenue, also known as  
12 WW56, sampling location 29, that has the same  
13 value there, 2800 feet or .53 miles." Then when  
14 you turn to page 15, near the top, "Upstream of  
15 the Calumet Water Reclamation plant at LCR, that  
16 says, "WW 56 is 6300 feet or 1.2 miles." Do you  
17 know as you sit here today whether that station is  
18 2800 feet or 6300 feet upstream of the plant?

19 DR. PETROPOULOU: I would have to  
20 verify that.

21 MS. WILLIAMS: You would agree,  
22 those are a big difference?

23 DR. PETROPOULOU: Right, right.

24 MS. WILLIAMS: Would that difference

1 effect whether it was representative of an  
2 upstream location or not?

3 DR. PETROPOULOU: We went to the  
4 closest upstream and downstream location during  
5 dry weather.

6 MS. WILLIAMS: The closest one?

7 DR. PETROPOULOU: Yes.

8 MS. WILLIAMS: So then how did you  
9 determine that the station was not too close so  
10 that it was impacted by the plant itself?

11 DR. PETROPOULOU: I think we had  
12 criteria that we used for the location of the  
13 stations, and that included a distance of about 10  
14 to 15 waterway widths. I know that when we  
15 calculated those distances, we verified them with  
16 the District's sampling staff with the GPS units  
17 and the distances checked out. I can see the  
18 discrepancies and the way the distances are  
19 important, but when we planned the closest  
20 locations from the station, we had the District  
21 sampling crew verify that they were 10 to 15  
22 waterway widths from the outfalls.

23 MS. WILLIAMS: But you didn't  
24 compare that to any modeling that's been done to

1 determine the length of any upstream impacts from  
2 the plant effluent itself, correct?

3 DR. PETROPOULOU: We based that on  
4 practical information that we had.

5 MS. WILLIAMS: I'd like to point out  
6 one more of the descriptions that I think has an  
7 error in it.

8 MR. ANDES: I'm sorry, can I  
9 follow-up on the questions that you just asked?

10 MS. WILLIAMS: Well, this is the  
11 last one for this. I want to get out on the  
12 record where I think there are errors so you can  
13 fix them. Then you can follow-up. Is that okay?

14 On the bottom of page 13, the last  
15 station that you've listed, CSE Halsted, it states  
16 that that station is 5800 feet or 11 miles --

17 DR. PETROPOULOU: It's 1.1 miles.

18 MS. WILLIAMS: So your testimony is  
19 this should be 1.1; you don't need to look into  
20 that one? Okay, thank you.

21 MR. ANDES: First, I will just state  
22 that we will certainly in one form or another get  
23 back to you to address those typos.

24 MS. WILLIAMS: What, I'm sorry?

1                   MR. ANDES: First I can state that  
2 we will get back to you promptly to clarify the  
3 numbers. I wanted to ask Dr. Tolson what effect  
4 this might have on any estimates of risk, these  
5 issues?

6                   DR. TOLSON: Because these are,  
7 these sampling locations are in close proximity to  
8 the outfall, we feel that they are probably  
9 conservative if you are estimating the  
10 concentrations, as we discussed yesterday, we  
11 didn't have upstream and downstream's. If there's  
12 any impact from the upstream location of the  
13 outfall, then we overestimated the concentrations  
14 in the waterway.

15                   MS. WILLIAMS: But wouldn't it  
16 change at all your opinion of what impact  
17 disinfection would have on the instream values if  
18 you were using upstream, un-impacted numbers that  
19 were impacted, wouldn't that effect the  
20 conclusions about the impact of disinfection?

21                   DR. TOLSON: Yes, it would tend to  
22 diminish the impact of disinfection -- it would  
23 strengthen the argument that disinfection would  
24 have a lower impact on the overall risk of the

1 waterway.

2 MS. WILLIAMS: I have one more  
3 follow-up regarding sampling for Dr. Petropoulou.  
4 Yesterday we talked about the differences or the  
5 different definitions, I guess, of wet weather.  
6 You for sampling purposes, had a definition of wet  
7 weather and then Dr. Tolson went and made some  
8 extrapolations about days that were impacted after  
9 a rain event. So when I was asking him questions  
10 yesterday about how he used meteorological data, I  
11 wanted to follow-up with you and understand for  
12 purposes of sampling, when you detected rain at  
13 one station but not another, how was that  
14 interpreted for the samplers?

15 DR. PETROPOULOU: Actually the  
16 sampling protocol included input from the  
17 District's waterway weather center. So if we were  
18 planning to do something in Calumet, those were  
19 the gauges that were used to trigger the sampling.  
20 If we were planning something for Stickney, the  
21 same thing, or North Side. And actually we would  
22 send the boat where the rainy event was predicted  
23 to occur.

24 MS. WILLIAMS: So would it be

1 possible that you would have sent a boat to the  
2 North Side where a rain event was predicted to  
3 occur, but at the same time someone would be  
4 taking a dry weather sample at Stickney on that  
5 day?

6 DR. PETROPOULOU: No, absolutely,  
7 no, because we didn't do the dry and wet weather  
8 sampling at the same time. One was done in 2005  
9 and the other 2006.

10 MS. WILLIAMS: Okay. In 2005 --  
11 that the dry year, right -- how did you determine  
12 that the dry weather samples were not being  
13 impacted at rain events at a different station?

14 DR. PETROPOULOU: Because, as I  
15 said, originally the study was to capture randomly  
16 dry or wet weather events, but the reason the  
17 District decided to expand the study is because  
18 when we look at the data, all the sampling took  
19 place without rain. Like there was at least three  
20 days of dry weather --

21 MS. WILLIAMS: Throughout the  
22 system?

23 DR. PETROPOULOU: It was at three of  
24 the segments where we did the sampling.

1 MS. WILLIAMS: So you did not look  
2 at for sure whether, for example, a Stickney dry  
3 weather sample, there were three days without rain  
4 at Stickney, but you did not look at for sure  
5 whether there had been rain at North Side?

6 DR. PETROPOULOU: We actually did.  
7 I think for the 2005 season it was a dry season.  
8 We didn't get rain events. I know that it rained  
9 a couple times after we completed the sampling,  
10 but not before the sampling.

11 MS. WILLIAMS: So it just didn't  
12 rain in 2005, so it wasn't an issue?

13 DR. PETROPOULOU: I didn't say it  
14 didn't rain in 2005. I said in 2005 when we did  
15 the sampling, we didn't capture those weather  
16 events, and that's what made the District decide  
17 to expand the study in 2006.

18 MS. WILLIAMS: Can we find this in  
19 the report? Does it explain this issue?

20 DR. PETROPOULOU: We have that data,  
21 and we can compile and provide that data.

22 MS. WILLIAMS: That would be  
23 helpful. Thank you.

24 MR. ANDES: And would that data be

1 in the appendices in that report?

2 DR. PETROPOULOU: So we have used  
3 that data for modeling purposes to calculate dry  
4 and wet weather data. So from the District we  
5 have.

6 MR. ANDES: So what specific data  
7 are we talking about?

8 DR. PETROPOULOU: Rain gauge data.

9 MS. WILLIAMS: Rain gauge data would  
10 be helpful for 2005 and 2006.

11 MR. ANDES: Sure.

12 MS. MEYER-GLEN: Can I ask a few  
13 follow-up?

14 MS. WILLIAMS: Yes, I think we are  
15 done with Dr. Petropoulou.

16 MS. MEYER-GLEN: We were talking  
17 about whether it was raining at North Side and not  
18 at Stickney, I was wondering about the converse.  
19 If you had a rain event triggered at Stickney and  
20 not at North Side, which is upstream, how would  
21 that have been handled?

22 MS. ANDES: Is that a hypothetical?  
23 I think they already said it wasn't.

24 MS. MEYERS-GLEN: Well, did it

1     happen?  If it did happen, how was it handled?

2                     DR. PETROPOULOU:  Was that the  
3     question?

4                     MS. MEYER-GLEN:  And would you know?

5                     DR. TOLSON:  Under wet weather  
6     sampling?

7                     MS. MEYERS-GLEN:  Wet and  
8     potentially dry.  I guess I'm asking how would you  
9     have characterized it.  If you would have  
10    characterized it as wet weather for the entire  
11    system or only at Stickney which is downstream,  
12    and if so, then how would the North Side plant,  
13    which would not have been wet, have been handled?

14                    DR. PETROPOULOU:  We didn't -- I  
15    believe we didn't sample, unless it rained in the  
16    entire waterway.  I know there was one sampling  
17    wet weather day where we sent two sets of boats,  
18    one at North Side and one at Stickney on the same  
19    date because there was rain events at both  
20    waterways.  So we have captured a situation like  
21    that, where it rains in both waterways.  The way  
22    we account are sampling measured what was in the  
23    waterway in that particular segment of the  
24    waterway when we had the significant rain event.

1 So we didn't account specifically for what was  
2 like at North Side or Calumet except for the  
3 measured concentrations in the waterway.

4 MS. MEYERS-GLEN: I think that  
5 answers it.

6 DR. TOLSON: I have one more thing.  
7 For our Stickney dry water samples, I don't  
8 believe there were any days where there was rain  
9 at North Side that would have drifted into --

10 MS. MEYER-GLEN: I'm asking the  
11 converse. If you have downstream, if you have  
12 rain at Stickney, and yet you don't have the  
13 alarms go off or you don't have enough prediction  
14 over .5 at North Side, how would that be handled  
15 because then you have rain downstream but not  
16 upstream, potentially attributed to the waterways  
17 in general, and I was wondering how that would  
18 have been handled?

19 MR. ANDES: I guess the first  
20 question is whether in fact that's just a  
21 hypothetical or whether that -- do you know of any  
22 situation that would have happened?

23 DR. PETROPOULOU: How would that be  
24 captured in North Side or at Stickney?

1 MS. MEYERS-GLEN: I was wondering  
2 how that would be characterized with both, because  
3 if you have dry on top and wet on bottom, would  
4 that be considered a wet day for the system as a  
5 whole or would it be then accounted for that it  
6 was dry at North Side?

7 DR. TOLSON: So there would be no  
8 samplings taking place at the north side?

9 MS. MEYERS-GLEN: Correct.

10 DR. TOLSON: Because it was wet  
11 somewhere.

12 MS. MEYERS-GLEN: Correct.

13 DR. TOLSON: So that is off the  
14 table. For Stickney it would be considered a wet  
15 weather day for Stickney, but that would be for  
16 sampling purposes only. I don't know if that  
17 situation actually occurred. I don't know if we  
18 had a situation where it occurred there.

19 MS. MEYERS-GLEN: That answers half  
20 my question, and that's helpful. But then what  
21 would happen at North Side if it's not wet --

22 CHAIRMAN TIPSORD: He answered. I  
23 believe he did answer, there be wouldn't be  
24 sampling at the North Side.

1 MS. MEYERS-GLEN: But would it be  
2 characterized as a wet day through the system,  
3 even though there was not enough rain to actually  
4 technically have it be a wet weather day.

5 CHAIRMAN TIPSORD: Again, what he's  
6 answered is it would be a wet weather day for  
7 Stickney. Perhaps this is some of the confusion,  
8 when you classified a wet weather event, it was  
9 for that segment of the stream; it is not a  
10 systemwide weather event; is that correct?

11 DR. TOLSON: There's two different  
12 situations here, sampling where we took the wet  
13 weather and dry weather, and there is the analysis  
14 for risk, where it's more nebulous where we  
15 couldn't say it's wet or dry and we had to take  
16 into account that it was wet two days ago. So  
17 there is a difference there. For the sampling we  
18 had to be very sure that we captured wet days and  
19 dry days, that's why we had the antecedent periods  
20 of dry weather before we considered it a dry day.

21 MS. MEYERS-GLEN: So that wouldn't  
22 be effected then when you averaged it throughout  
23 the system, correct?

24 CHAIRMAN TIPSORD: I couldn't hear

1 you.

2 MS. MEYERS-GLEN: It wouldn't have  
3 an effect then when it was averaged throughout the  
4 system for risk?

5 DR. TOLSON: It is not averaged.  
6 That's a different calculation. The sampling and  
7 the risk assessment components here on dry and  
8 wet, we have to think about them differently.

9 MS. MEYERS-GLEN: Thank you.

10 CHAIRMAN TIPSORD: Did the IEPA have  
11 anything?

12 MS. WILLIAMS: I just want to ask  
13 one clarifying point about the additional  
14 information. So you are going to provide us  
15 information on the rain gauge data. And can you  
16 point me to where, like in the appendices we would  
17 look if we wanted to match that up with the  
18 sampling data; is the sampling results --

19 DR. PETROPOULOU: It's the dates.  
20 It's the sampling dates.

21 MS. WILLIAMS: But are the actual  
22 sampling results, like the raw data included in  
23 the appendices at all?

24 MR. ANDES: I believe the raw data

1 is not included in appendices A through D. We can  
2 provide that. We have that electronically. We  
3 can provide it. It's voluminous even on a disk,  
4 but we can certainly provide all of the raw data  
5 sheets.

6 MS. WILLIAMS: I think we would like  
7 that.

8 MR. ANDES: That's fine.

9 MS. WILLIAMS: That's all. So if  
10 you want us to move on to Dr. Gerba --

11 CHAIRMAN TIPSORD: No, let's go off  
12 the record.

13 (Brief recess taken, after  
14 which the following  
15 proceedings were had:)

16 CHAIRMAN TIPSORD: Back on the  
17 record. Ms. Alexander, we'll return to you and  
18 let you finish with this panel.

19 MS. ALEXANDER: I'm Ann Alexander  
20 from Natural Resources Defense Counsel for the  
21 record. I'm going to pick up where I left off  
22 which is at Tolson 24 and Gerba question 32. The  
23 question, that pre-filed question was asked and  
24 answered, which was did you use a Monte Carlo

1 simulation in quantifying risk? Please describe  
2 how that was done. Dr. Tolson responded to that  
3 question.

4 To follow-up on that, I would  
5 like to turn to Figure 5.2 in Exhibit 71, if you  
6 will. My question regarding Figure 5.2 is, would  
7 that be an example of range of values for one  
8 parameter of Monte Carlo analysis, which would be  
9 the incidental ingestion rate?

10 DR. TOLSON: Correct. That is a  
11 truncated distribution for ingestion of  
12 paracetamol.

13 MS. ALEXANDER: Just to understand  
14 the graph, it would look to me that the percent  
15 probability of ingesting 4.18 ml's per hour of  
16 water is around .15; if I'm understanding it  
17 correctly?

18 DR. TOLSON: That is correct.

19 MS. ALEXANDER: So in other words,  
20 it's an illustration of percent probability of  
21 this range of events?

22 DR. TOLSON: Probability,  
23 distribution, function, yes.

24 MS. ALEXANDER: Am I correct that

1 you also used a probabilistic range of values in  
2 your calculation for the other input variables  
3 instead of just one value?

4 DR. TOLSON: That is correct. Those  
5 input variables that had some variability we  
6 captured the variability with a range similar to  
7 what we've shown here for the ingestion.

8 MS. ALEXANDER: That would include,  
9 for example, the exposure duration, water  
10 consumption, pathogen concentration, all of those  
11 would have ranges?

12 DR. TOLSON: Pathogen concentration  
13 is a little bit different. That was actually  
14 developed from a Monte Carlo boot strap resampling  
15 from the data set. But the other ones, yes, those  
16 were all input ranges or distributions that were  
17 used in the model.

18 MS. ALEXANDER: Okay. And you used  
19 a probability density function or PDF for those,  
20 correct?

21 DR. TOLSON: You got it.

22 MS. ALEXANDER: My question is, then  
23 given that you did PDF for those different input  
24 variables except in pathogen concentration, why

1 did you not include an illustration of a  
2 probabilistic spread similar to what's in  
3 Figure 5.2 for each of those input variables?  
4 Why just one of them?

5 DR. TOLSON: Well, we could have.  
6 We could have made illustrations for each one, but  
7 the long normal parameters for the other ones are  
8 listed in the text.

9 MS. ALEXANDER: But am I correct  
10 there's no illustration for the others, you just  
11 did one illustration?

12 DR. TOLSON: The model input that  
13 would create that under any statistical programs  
14 are listed in the text.

15 MS. ALEXANDER: Okay. But there's  
16 no pictures is my question; I'm just confirming.

17 DR. TOLSON: I like pictures too,  
18 but I only included a couple pictures in here,  
19 illustrations.

20 MS. ALEXANDER: Now turning to table  
21 5.13 --

22 MR. ANDES: I'm sorry, table 5.13?

23 MS. ALEXANDER: 5.13. My question  
24 on that is, why did you present your risk numbers

1 in that table as a single number rather than a  
2 probabilistic spread given that the inputs were  
3 probabilistic?

4 DR. TOLSON: In order to have a  
5 comparison point for the U.S. EPA acceptable risk  
6 number of eight per thousand, we actually computed  
7 the estimated point value from the probabilistic  
8 distribution. In other words, for each individual  
9 within the distribution of ingestion rates that  
10 had a chance of getting sick, we rolled the dice  
11 to see whether they were actually sick or not  
12 sick, took the total number of those per thousand  
13 and presented the results.

14 MS. ALEXANDER: So in other words,  
15 you did a probabilistic spread but did not present  
16 it here, but rather presented a point data as  
17 opposed to a spread?

18 DR. TOLSON: For ease of  
19 presentation of the results, we presented a single  
20 point number so we could basically compare it  
21 against the EPA numbers. It's also much easier to  
22 interpret a number versus a number,  
23 disinfection-not disinfection versus a range  
24 versus a range.

1           Q.       Did the Monte Carlo Analysis  
2       conclude that there was a one hundred or lower  
3       probability of the risk identified in table 5.13?  
4       I mean using the language of these probabilistic  
5       spreads, was your conclusion that there was a one  
6       hundred percent or lower probability of either  
7       risk numbers listed in table 5.15?

8                   MR. ANDES:  Isn't every probability  
9       100 percent or lower?

10                  MS. ALEXANDER:  No.  In the context  
11       of the Monte Carlo Analysis, if you are presenting  
12       point data like this, you might also be presenting  
13       median probability.  In other words, a 50 percent  
14       or lower probability of these numbers, where there  
15       may actually be a possibility of a higher risk;  
16       that's how the Monte Carlo Analysis works,  
17       correct?

18                  DR. TOLSON:  I'm sorry, it's not  
19       quite that way.  The way that we've developed  
20       these numbers here, the numbers presented in table  
21       5.13 is actually to create an outcome from each  
22       one of the simulations and produce the approximate  
23       frequency of the outcome per thousand as shown in  
24       this table.

1 MS. ALEXANDER: Are these numbers  
2 derived from a probabilistic spread that you did?  
3 Derived from something like what's illustrated  
4 from one of the input variables?

5 DR. TOLSON: Yes, probabilistic  
6 techniques went into deriving these numbers, that  
7 is correct.

8 MS. ALEXANDER: So is it your  
9 testimony, correct me if it's not, that there is a  
10 100 percent or lower probability of the risks  
11 presented in this table?

12 DR. TOLSON: The probability of a  
13 person getting ill is somewhere between 0 and 100  
14 percent. The lower probability that we've  
15 estimated on this table is certainly between 0 and  
16 100 percent. I'm really not sure how to  
17 characterize that.

18 MS. ALEXANDER: Let me rephrase the  
19 question. I'm not sure if it's lack of clarity on  
20 my part or -- but, for instance, for pathogenic E.  
21 Coli -- actually let me look at this a little  
22 differently.

23 For total illness, including  
24 secondary, you presented a number associated with

1 North Side of 4.15. Is it your conclusion that  
2 there is a hundred percent or lower probability  
3 that 4.15 is in fact the number of anticipated  
4 illnesses?

5 DR. TOLSON: That's not the way that  
6 this number should be interpreted.

7 MS. ALEXANDER: Okay. What is the  
8 way it should be interpreted?

9 DR. TOLSON: This number is our  
10 estimate of the illnesses, including secondary  
11 illnesses, that would result from one thousand  
12 exposures to the North Side segment.

13 MS. ALEXANDER: When you say it's  
14 your estimate, presumably estimates have  
15 uncertainty that surround them. In other words,  
16 there are bounds on that. You have confidence  
17 bounds. Is that accurate?

18 DR. TOLSON: We did not do a  
19 quantitative uncertainty analysis on this data.  
20 Our probabilistic assessment was through the  
21 dimension of variability. To do a certainty  
22 analysis, it would be a two dimensional Monte  
23 Carlo, which is a little trickier sort of device  
24 to construct and do, and to my knowledge -- do you

1 know of any two dimensional Monte Carlo  
2 probabilistic -- I don't not know of any two  
3 microbial two dimensional Monte Carlo analysis  
4 that have been done.

5 MS. ALEXANDER: So this is a single  
6 dimensional analysis.

7 DR. TOLSON: Correct. I'm impressed  
8 with your knowledge of probabilistic analysis.

9 MS. ALEXANDER: I still flunked  
10 math.

11 MR. ANDES: If I can follow-up for a  
12 moment.

13 Was it your testimony earlier  
14 that these numbers in fact were overestimates of  
15 risk?

16 DR. TOLSON: That is correct, a  
17 number of the input that went in here were  
18 conservatively developed, and I believe this  
19 number is a conservative estimate of the risk.

20 MS. ALEXANDER: Referring back to  
21 the documents marked yesterday, which were  
22 exhibits, the EPA documents or the exchanges of  
23 correspondence, Exhibit 73 is the correspondence  
24 of EPA. I'm going to refer back to that again. I

1 want to ask you as a general question, would it be  
2 fair in your view to say that EPA was consistently  
3 critical of your failure to include the full  
4 results of your Monte Carlo analysis and to  
5 essentially show your work, present your graphs  
6 and data?

7 CHAIRMAN TIPSORD: Excuse me, if I  
8 may too, that's U.S. EPA. Yesterday we did refer  
9 them to as U.S. EPA, since this is a new  
10 transcript we better be clear that it's U.S. EPA  
11 or the confluence.

12 MS. ALEXANDER: Yes, U.S. EPA.

13 DR. TOLSON: We've had numerous  
14 correspondence with the EPA, and I don't think  
15 that that characterization is accurate. In fact,  
16 I think up until the final EPA letter, it would  
17 have been very complimentary to the approach.  
18 There were some comments within in it suggesting  
19 ways we could add transparency, and I believe we  
20 addressed those in responses back to the EPA.

21 MS. ALEXANDER: Let's dig into that  
22 just a little bit. Can we turn to the documents  
23 under the May 28, 2008 cover letter from the Water  
24 Reclamation District to Allen Melzer. The

1 document I'm referring to is the first attachment,  
2 which is a review conducted by U.S. EPA's Office  
3 of Research and Development, page 5, toward the  
4 bottom. There's a bullet point. Do you see that?  
5 That states, "Inadequate reporting of risk  
6 assessments and methods," and then there is the  
7 statement that they make -- again, I'm reading  
8 from the EPA's transcribed text that you are  
9 responding to -- "The actual risk assessment is  
10 brief and contains no graphs and few brief  
11 tables." Would you consider that to be a  
12 statement critical of your presentation?

13 DR. TOLSON: I'm going to need more  
14 help. There is a four-page and then there's like  
15 one page. Are you on the five-page?

16 MS. ALEXANDER: I'm on the  
17 five-page, so it's the last page on the bottom.

18 DR. TOLSON: The last page, it  
19 starts with "Overall, the risk assessment" at the  
20 top?

21 MS. ALEXANDER: No, the top of the  
22 page 5 I'm looking at is a sentence that includes  
23 the words "Enteric viruses" -- and the first page  
24 is "Review conducted by U.S. EPA Office of

1 Development" -- in other words it's the first  
2 attached to the May 23rd.

3 CHAIRMAN TIPSORD: Actually it's the  
4 last attachment to the May 23rd.

5 MS. ALEXANDER: I stamped it wrong.

6 CHAIRMAN TIPSORD: At least on our  
7 copy it's the last attachment.

8 MS. ALEXANDER: Okay, no more late  
9 night stapling. So you found it. Let me restate  
10 my question.

11 I'm referring to the bottom of  
12 the page. You see the bullet point that says,  
13 "Inadequate reporting of risk assessment results  
14 and methods," and then there is the statement they  
15 make, "The actual risk assessment is brief and  
16 contains no graphs and few brief tables." My  
17 question is, would you consider that to be a  
18 criticism of the amount of data presented and your  
19 failure to show your work?

20 DR. TOLSON: It was clear that they  
21 didn't understand it. You know, we've tried to  
22 correct that through additions to the text, and we  
23 also have a response here that tries to provide  
24 some clarity for the Agency.

1                   MS. ALEXANDER: But my original  
2 question went to whether they were consistently  
3 critical, and I just want to establish whether we  
4 have the -- whether we can agree as it were on  
5 what is a criticism.

6                   MR. ANDES: This is one instance.  
7 Consistent would imply a series.

8                   MS. ALEXANDER: I'm aware it's one  
9 instance. Let me continue.

10                   Moving on to what I've just been  
11 informed was the first attachment, which is  
12 entitled "Review Conducted by US EPA Office Of  
13 Water, Office of Science and Technology," turn to  
14 page 6.

15                   MR. ANDES: Can I clarify one thing  
16 first?

17                   MS. ALEXANDER: Certainly.

18                   MR. ANDES: These issues raised by  
19 EPA were in the interim report.

20                   MS. ALEXANDER: I understand that.

21                   MR. ANDES: And the letter, the  
22 subsequent letter from EPA indicating that most of  
23 their concerns had been addressed regarded the  
24 interim report, so as long as we have that on the

1 record as well.

2 MS. ALEXANDER: Yes, that is  
3 understood. And my question went to whether they  
4 were consistently critical on this issue, and I'd  
5 like to establish that. So going to page 6, you  
6 see the bullet that says, "Interval estimates were  
7 not recorded." And then the text under that,  
8 "This is a major failing since only one estimate  
9 of the risk was reported with a significant amount  
10 of assumptions and uncertainty bounds on these  
11 estimates must be provided. 95 percent bounds.  
12 Complete details of the Monte Carlo analysis  
13 should be provided so that the distribution of  
14 risk can be visualized." Do you see that?

15 DR. TOLSON: Yes, I'm with you.  
16 These are concerns that are raised by the Agency  
17 on the interim report. You know, we responded to  
18 them. We've had discussions with them based on  
19 the correspondence subsequent to this, they  
20 indicated that those were addressed.

21 MS. WILLIAMS: I need to interrupt  
22 at this point. We let kind of let this go  
23 yesterday, but just to be clear in line of the  
24 questioning, the EPA is U.S. EPA. I think when

1 you went to Agency --

2 CHAIRMAN TIPSORD: We can ask them  
3 every time. I think I made that clear at the  
4 beginning this morning. If you are concerned, I  
5 can have them do it every time.

6 MS. WILLIAMS: As he started saying  
7 the Agency, I just want to have it clear on the  
8 record that the Agency is not us. Sorry to  
9 interrupt.

10 DR. TOLSON: Can I add one more  
11 thing to that? Just based on this comment, I  
12 think it is really a misunderstanding on the U.S.  
13 EPA'S part about how we conducted the risk  
14 assessment, because the context of what we are  
15 asking here does not fit with the context for  
16 which we were presenting the results. So I really  
17 believe it's a misunderstanding on the U.S. EPA'S  
18 part on how we conducted the risk assessment and  
19 what the numbers we presented mean.

20 MS. ALEXANDER: Can you clarify the  
21 statement you just made about the different  
22 context about why this does not reflect the  
23 context in which you presented your results?

24 DR. TOLSON: Sure. Often in a

1 probabilistic risk assessment one would produce a  
2 distribution of outcomes, but those are exposure  
3 outcomes. And within the context of the way that  
4 we've conducted the risk assessments, there are  
5 within the model, those distribution of exposure  
6 outcomes. But for each one of those exposures,  
7 the total dose, we actually calculated whether  
8 that person got sick or not, and then counted  
9 those people. So we used those intermediate sort  
10 of distributions to do sensitivity analysis and do  
11 other things that you are allowed to do with a  
12 probabilistic risk assessment. We can tell which  
13 of the input models had the most effect on the  
14 risk. We presented that into the report. We  
15 could look at the effects of changing input  
16 parameters, increasing them or decreasing them.  
17 How that would change the overall effect of risk.  
18 Those are in the report. In fact, those are in  
19 the report based on comments by U.S. EPA, and I  
20 think those are good comments, and we responded to  
21 those by including the information. But the end  
22 risk numbers that we presented in the report are  
23 misinterpreted by EPA as opposed to what we were  
24 really doing.

1 MS. ALEXANDER: Let me just ask a  
2 couple follow-ups to that. Referring to the  
3 statement under the bullet point, "Interval  
4 estimates were not reported" they make the  
5 statement "with significant amount of assumptions  
6 and uncertainty bounds on these estimates must be  
7 provided (95 percent bound)." Is the reference to  
8 uncertainty and 95 bounds to the two dimensional  
9 analysis?

10 DR. TOLSON: That would be correct.  
11 That's one of the reasons that I'm fairly certain  
12 that this was misinterpreted by U.S. EPA.

13 MS. ALEXANDER: Isn't it possible  
14 that in fact here they were asking, recommending  
15 that you perform the two dimensional analysis  
16 which you then did not do?

17 DR. TOLSON: I don't believe so. I  
18 don't think they understood exactly the  
19 implications of what they were asking.

20 MR. ANDES: Just to follow up, did  
21 the EPA ever in any of its correspondence or  
22 report ever suggest you do a two dimensional  
23 model?

24 DR. TOLSON: No, they did not.

1 MS. ALEXANDER: Isn't it a fact they  
2 repeatedly asked you to do uncertainty analysis?  
3 They used the term "uncertainty" as they did here?

4 DR. TOLSON: I'm unaware that they  
5 repeatedly asked. They asked in the context of  
6 these questions. We had a meeting with the  
7 Agency. We responded to those from the interim  
8 draft. We got some concurrence on the final draft  
9 that we've addressed those questions.

10 MS. ALEXANDER: Just with reference  
11 to uncertainty, I would call your attention to the  
12 text under the bullet point that says,  
13 "Variability and Uncertainty were not discussed,  
14 evaluated or quantified." The text is, "Each step  
15 of risk assessment contains variability and  
16 Uncertainty. Uncertainty could be considered in  
17 the dose response parameters or in microbial  
18 densities." Would the method by which you would  
19 consider uncertainty be a two dimensional  
20 analysis?

21 DR. TOLSON: Not necessarily. And  
22 in fact, if you look at Exhibit 71, page 130 to  
23 132, we provide several pages of text describing  
24 sensitivity and uncertainty analysis, and some of

1 this we included after we got the comments from  
2 the Agency just to be more responsive to their  
3 desires to see some of the information in there,  
4 information about uncertainty in the report.

5 MS. ALEXANDER: And just so I  
6 understand, I'm looking at your text at the bottom  
7 of 130 of Exhibit 71, which state a probabilistic  
8 assessment of uncertainty combined with  
9 variability data could be used to create a two  
10 dimensional probabilistic input, however such  
11 assessment was outside the scope of the study due  
12 to logistical constraints." Am I correct in  
13 understanding here that you were essentially  
14 saying that you were declining to consider  
15 uncertainty as we've used the term here to create  
16 a two dimensional probabilistic output?

17 DR. TOLSON: We did not include  
18 uncertainty.

19 MR. ANDES: To follow-up in  
20 discussing logistical constraints, it talks about  
21 boundary conditions. Can you explain?

22 DR. TOLSON: Yes. For the  
23 uncertainty analysis -- uncertainty is different  
24 than variability. Maybe I should go back to that

1 definition. Variability has to do with sort of  
2 the natural differences between input models, the  
3 nature difference between or the differences  
4 between ingestion rates. There's some  
5 variability, not every person goes out. With  
6 additional information, one can reduce or one  
7 cannot reduce that variability. Some people are  
8 going to capsize and some people are going to not  
9 get wet at all. That's the natural condition of  
10 things. Uncertainty has to do with something that  
11 you can't measure or can't provide additional  
12 information to, to reduce within the analysis.

13 MS. ALEXANDER: Moving on to page 7,  
14 same document. I call your attention to what's  
15 the first nonitalicized text on that page, which  
16 begins "In summary" -- which states, "In summary,  
17 while the QMRA methodology is appropriate, many  
18 assumptions are questionable and important details  
19 are left out. There's no evaluation of the  
20 potential range of risk and no sensitivity  
21 analysis, therefore the QMRA does not provide  
22 sufficient information to support the assertion  
23 that there is minimal risk of the current state of  
24 no disinfection. These details should be provided

1 to support the claims made or another independent  
2 risk assessment should be conducted." And my only  
3 question there is, would you consider that to be a  
4 criticism of the level of data actually presented  
5 in the report?

6 DR. TOLSON: I wish I could give you  
7 a yes or no answer, but I'm going to expand on it  
8 slightly, as you knew I would.

9 Yes, we took into consideration  
10 this comment. However, this is a comment on the  
11 interim report. We've included, for example,  
12 table 5.17 within Exhibit 71 that takes into  
13 account sensitivity of these parameters to see  
14 what the effect the ingestion rate or duration of  
15 weather type, this is actually kind of an  
16 uncertainty analysis that was conducted here that  
17 gets to that point. This says, what if we're off,  
18 what if we move the whole distribution one way or  
19 another, how would that effect the outcome? And  
20 this was provided in response to the Agency  
21 comments. We had discussions with the U.S. EPA,  
22 and it would appear from their response letter  
23 from this, that they have some concurrence that  
24 we've addressed those concerns.

1                   CHAIRMAN TIPSORD: Excuse me,  
2 Dr. Tolson, for the record before we get too far  
3 way, can you please tell us what QMRA stands for?

4                   DR. TOLSON: Quantitative microbial  
5 risk assessment.

6                   MS. ALEXANDER: But in fact the  
7 response given to this statement does not contain  
8 the information that you just provided; it simply  
9 points to a section of the report; is that  
10 correct?

11                  DR. TOLSON: Yes. It appears that  
12 we went over and above the initial response. With  
13 our addressing this comment, we went over and  
14 above the response.

15                  MS. ALEXANDER: And I would point  
16 out in your response, you state, "In addition,  
17 uncertainties associated with risk assessments are  
18 also discussed in this section." Just to clarify,  
19 when you in fact in Exhibit 71, the risk  
20 assessment, the only way in which you addressed  
21 uncertainties as you've used that term in the  
22 context of two dimensional analysis is to say that  
23 you weren't going to do it; is that correct?

24                  DR. TOLSON: That's not true. We

1 actually discuss uncertainties associated with the  
2 number of input parameters and their biases and  
3 whether their biases were conservative or  
4 anti-conservative.

5 One other point is, the section  
6 that you've referenced here includes pointing to  
7 table 5.17 in the Exhibit 71, which is an  
8 uncertainty analysis associated with this  
9 assessment.

10 MS. ALEXANDER: But in fact there is  
11 one meaning to the term uncertainty which you  
12 assign it, if I'm correct, in Section 5.4.7, which  
13 is the type of uncertainty that would be  
14 associated with two dimensional analyses, correct?  
15 I mean, you use that term in a very specific way,  
16 do you not, at the bottom of page 130, "A  
17 probabilistic assessment of uncertainty combined  
18 with variability could be used to create a  
19 two-dimensional probabilistic input," and then you  
20 proceed to decline to perform that kind of  
21 analysis; is that correct?

22 DR. TOLSON: That's not the only  
23 uncertainty. We have developed some uncertainty  
24 estimates within the response to the Agency's

1       comments.

2                       MS. ALEXANDER:  Turning to page 12,  
3       same document.  At page -- there is, at the very  
4       top, there is a reference to page 111, and they  
5       state "Since Monte Carlo analysis was used, why  
6       wasn't a risk distribution e.g., 50th percentile,  
7       et cetera, generated?"  Do you understand that --  
8       I mean, perhaps this is a better presentation of  
9       the question I was trying to ask about your final  
10      risk results -- Do I understand correctly that  
11      your response is as stated here, that you wanted  
12      to simplify the presentation?

13                      DR. TOLSON:  I think my response to  
14      that comment is it speaks for itself.  That was  
15      one of the reasons that are listed here.  If you  
16      want, I could read that response into the record.

17                      MS. ALEXANDER:  No, that's not  
18      necessary.  My question there would be, did you in  
19      fact generate a risk distribution along the lines  
20      of the example given here?  For example,  
21      50th percentile, 90th percentile, et cetera, is  
22      that something that you generated?

23                      DR. TOLSON:  No.  A risk  
24      distribution does not come out, an exposure

1 distribution comes out.

2 MR. ANDES: If I can follow-up.  
3 Your understanding was, as with the previous  
4 issues on sensitivity and uncertainty, EPA raised  
5 these questions as to the interim report and you  
6 addressed the issues with the Agency. Your  
7 understanding was by, you told them that these  
8 additional analyses would be performed and they  
9 indicated those would address their concerns; is  
10 that correct?

11 DR. TOLSON: That is correct. In  
12 addition to that, we've described in a little more  
13 detail exactly what we had done with the Agency so  
14 they would understand why the results looked like  
15 they did.

16 MR. ANDES: Thank you.

17 CHAIRMAN TIPSORD: Excuse me,  
18 Ms. Alexander, I have a question as well.

19 We are talking about a lot about  
20 the Agency's comments and correspondence on the  
21 interim report. Has the U.S. EPA seen the final  
22 report? And are we expecting them to comment on  
23 that?

24 MR. ANDES: I believe the July 31,

1 2008 document from U.S. EPA concerns the final  
2 report.

3 CHAIRMAN TIPSORD: Okay.

4 MS. ALEXANDER: Well, this is a good  
5 segue to another one of the documents contained in  
6 73, which is the May 31, 2007, letter to U.S.  
7 EPA from the District. And this attaches what  
8 appears to be, and was discussed yesterday, a  
9 summary of responses to meeting discussions, which  
10 is a follow-up on the April 10th meeting; is that  
11 correct?

12 DR. TOLSON: Yes, according to the  
13 cover, that's what this is.

14 MS. ALEXANDER: Now, just to  
15 summarize, is it your testimony that all of the  
16 issues identified in bullet points here were  
17 resolved to the satisfaction of the U.S. EPA?

18 DR. TOLSON: I don't know that.

19 MS. ALEXANDER: So they may or may  
20 not have been?

21 MR. ANDES: The documents speak for  
22 themselves.

23 MS. ALEXANDER: Well, the documents  
24 are the documents, but there are conversations

1 that happened outside the documents. I'm not  
2 asking about the documents. I'm asking about his  
3 understanding as to whether these issues were  
4 resolved. People can pick up phones.

5 MR. ANDES: Were there other  
6 conversations you had with them besides the April  
7 10, 2007 conference call?

8 DR. TOLSON: I did not have any  
9 additional conversations with them.

10 MS. ALEXANDER: And do you have any  
11 knowledge whether anybody else at GeoSyntec did?

12 DR. TOLSON: I do not believe that  
13 anyone else did, but I don't have knowledge of  
14 anyone else having discussions.

15 MS. ALEXANDER: Well, let me ask  
16 about a couple specifics.

17 Can we please turn to the last  
18 page of that document. The one which text fills  
19 half the page. The second to the last bullet  
20 states, "The U.S. EPA requested that the report  
21 also examine strep either as to determine illness  
22 rates associated with specific secondary contact  
23 activities such as canoeing and fishing. In the  
24 final report we will include a summary of the

1 proportion of the overall illnesses that were  
2 attributed as identified uses (canoeing, fishing  
3 and recreational boating)."

4 DR. TOLSON: Yes I see that.

5 MS. ALEXANDER: Is it your belief  
6 that this response was satisfactory to the U.S.  
7 EPA, that that was resolved?

8 DR. TOLSON: I haven't had  
9 additional comments with them. Although, we did  
10 do what we stated that we would do here in table  
11 5.11 of Exhibit 71, there's a proportion of  
12 recreational -- I'm sorry, it was table 5.12 on  
13 Exhibit 71, there's stratified risk estimates,  
14 still estimated illness, rates assuming single  
15 recreation use with no effluent disinfection.

16 MS. ALEXANDER: And then moving to  
17 the last bullet point, that one reads, "U.S. EPA  
18 asked if fish consumption, (particularly by  
19 sensitive population such as women and children  
20 was considered in the risk assumption.) We  
21 explained that the fish consumption was not  
22 included in the subject design. We added that the  
23 states usually issue fish advisories to protect  
24 sensitive populations." I believe -- I'm not sure

1 I'm confident to say it was yours -- but it was  
2 one of your testimony that this issue was in fact  
3 resolved to the satisfaction of the EPA; is that  
4 your recollection?

5 DR. TOLSON: When we left the  
6 meeting, it was my impression that we had resolved  
7 that but --

8 DR. GERBA: I think the issue here  
9 is -- if the issue you are using ineffectivity as  
10 your -- oh, the fish you mean?

11 MS. ALEXANDER: I'm talking about  
12 the fish.

13 DR. GERBA: Yes, I've studied fish  
14 around the sewage ponds before in the United  
15 States and different parts of the world. And  
16 other investigators have studied it, actually  
17 growing in completely raw sewage. You can  
18 actually grow fish. They get pretty big and  
19 tough, but you can grow them and harvest them.  
20 But they don't take up these pathogens quite  
21 interestingly if you clean them and process them  
22 correctly. Even fish growing in the sewage ponds,  
23 like Tilapia. If you've ever been to Israel and  
24 eaten Tilapia in Israel, it was probably grown in

1 a waste toxication pond, that you are being served  
2 in a restaurant. So you can actually produce  
3 fish. So I didn't think that, considering the  
4 quality of this water compared to other fish are  
5 grown in the lack of any, health risks, I kind of  
6 neglected that as being really significant.

7 MS. ALEXANDER: One follow-up to  
8 that, aside from the fact that I'll never eat  
9 Tilapia again, do you know of any research to the  
10 contrary, in other words research that found that  
11 fish grown in contaminated waters are an exposure  
12 to for pathogens?

13 DR. GERBA: I would imagine in  
14 certain types of environments you would have that  
15 possibility with certain types of pathogens in  
16 different parts of the world. If you have abraded  
17 hands and that, certainly.

18 MS. ALEXANDER: But you don't know  
19 of specific research to the contrary?

20 DR. GERBA: Talking about fresh  
21 water environments now, right?

22 MS. ALEXANDER: Well, let me first  
23 limit it to fresh water.

24 DR. GERBA: Not off the top of my

1 hand -- the top of my head, no.

2 DR. TOLSON: I would add, I do  
3 recall that there has been a study done on  
4 handling fish and they did washing, and they were  
5 looking for cryptosporidia, which I believe they  
6 found, but considering the pathogen had dry  
7 weather and zero viable in the waterway, I'm not  
8 too sure of the relevance of that fact.

9 MS. ALEXANDER: Dr. Gerba, the same  
10 question for salt water since you drew that  
11 distinction?

12 DR. GERBA: Salt water, you can get  
13 infections. I mean, possibly if you are -- I  
14 would think there's a possibility with maybe  
15 cholera, vibrio-cholera may cause skin infections  
16 on non-homeland types. May be related to fish  
17 handling and may be related to sewage polluted  
18 waters in the developing world too.

19 MR. ANDES: A follow up. Is cholera  
20 common in Illinois?

21 DR. GERBA: Not for over a hundred  
22 years.

23 CHAIRMAN TIPSORD: I'd like to  
24 follow-up too. Any of you know of the fish

1 advisories issued by the State of Illinois for the  
2 CAWS system?

3 DR. TOLSON: I haven't paid  
4 attention to those, so I do not know.

5 DR. PETROPOULOU: I do not know.

6 CHAIRMAN TIPSORD: Thank you.

7 MS. ALEXANDER: Okay, I would like  
8 to turn next to the portion of Exhibit 73, which  
9 is the letter dated July 12, 2007, from U.S. EPA  
10 to the District. And I'd like to specifically  
11 call your attention to the text toward the bottom.  
12 The paragraph that begins, "We believe." Do you  
13 see where I'm looking?

14 DR. TOLSON: Yes, I do.

15 MS. ALEXANDER: And that text reads  
16 in full "We believe it would be helpful to also  
17 include a discussion of how only including certain  
18 pathogens effected, and in all likelihood resulted  
19 in an underestimation of the risk estimates." The  
20 first question, I'm sorry I should ask for  
21 background, is it your understanding that this  
22 letter -- this letter appears to be a response to  
23 the summary of issues that was provided in the May  
24 31, 2007 letter; is that your understanding as

1 well?

2 DR. TOLSON: Based on the dates,  
3 that seems reasonable.

4 MS. ALEXANDER: And I'd call your  
5 attention to the very first paragraph just to  
6 establish this.

7 DR. TOLSON: Yes.

8 MS. ALEXANDER: All right. So  
9 referring back to the text that I just read, where  
10 they state, "It would be helpful to include a  
11 discussion of how only including certain pathogens  
12 effected and all likelihood resulted in an  
13 underestimation of the risk estimate," did you in  
14 fact include a discussion, not of your  
15 justification for selecting the pathogens, but of  
16 the impact of that decision in terms of creating a  
17 likely underestimation of the risk?

18 DR. TOLSON: I would characterize it  
19 as, yes, there is some underestimation of risk.  
20 We just don't think it's a very important  
21 underestimation of risk. In fact, within the  
22 uncertainty and sensitivity section, we highlight  
23 out a bullet that this study did not account for  
24 all pathogens that might be present. However, the

1 pathogens that were selected include those that we  
2 think are most responsible for illness in the  
3 Chicago Area Waterways.

4 MS. ALEXANDER: I'm aware that you  
5 included a section that states that you did not  
6 study all pathogens. But my question is, I was  
7 not able to find a discussion specifically of the  
8 fact or the fact as expressed by U.S. EPA that  
9 this will result in an underestimation of risk.  
10 Did you include such a discussion in the document?  
11 I could not find it.

12 MR. ANDES: That's what he just  
13 referred to.

14 MS. ALEXANDER: Well, what he  
15 referred to is something different from my  
16 question, which is the fact that not all pathogens  
17 were studied. But the statement here by U.S. EPA  
18 is what they say, it would be helpful to include a  
19 discussion of how only including certain pathogens  
20 effected and in all likelihood resulted in an  
21 underestimation of the risk estimate. So they are  
22 not just asking it would appear for a discussion  
23 of what pathogens you included and why, but are  
24 they not asking specifically for a discussion of

1 the impact of that choice on underestimation of  
2 the risk?

3 MR. ANDES: You are asking for his  
4 speculation as to the EPA's state of mind?

5 MS. ALEXANDER: I'm asking for his  
6 understanding of the statement here.

7 DR. TOLSON: Yes, I can give you the  
8 specifics that we have in the report. It says,  
9 this is Exhibit 71, page 131, it says, "The  
10 following factors may lead to an overestimation or  
11 underestimation of risk. We did not quantify  
12 obviously the" --

13 MS. ALEXANDER: I'm sorry, what page  
14 did you say?

15 DR. TOLSON: 131, this is the  
16 sensitivity and uncertainty analysis on  
17 Exhibit 71. They read you the bullet that's  
18 there. It says, "This study did not account for  
19 all pathogens that may be present in the CAWS,  
20 recreational water, however the pathogens that  
21 were selected for inclusion in the study include  
22 regulatory indicators and those that can be  
23 measured by EPA approved methods and were judged  
24 most likely through gastrointestinal illness. See

1 Section 2.1 for more complete rationale of  
2 pathogen selections."

3 MS. ALEXANDER: So am I correct in  
4 understanding that there is no further discussion  
5 beyond the text you just read and the text  
6 referenced by it, which is presumably roughly  
7 summarized by it, of the way in which your choice  
8 of pathogens to select may have resulted in an  
9 underestimation of the risk; is that correct?  
10 That's it?

11 DR. TOLSON: I have no clue about  
12 the magnitude of that underestimation  
13 quantitatively. But we believe, you know, the  
14 best scientific evidence would suggest we've  
15 captured, most, if not all of the risk, associated  
16 with illness because we've captured pathogens that  
17 are representative of what we would expect to find  
18 in the waterway when we found them. We picked the  
19 ones that we expected there to be in high  
20 concentrations. And for some we didn't detect  
21 those.

22 MS. ALEXANDER: And isn't it a fact  
23 that early on in reviewing the interim dry weather  
24 risk assessment, EPA also expressed the concern

1 that due to failure to consider risks from  
2 hepatitis A, Shigella --

3 MR. ANDES: Are we on a particular  
4 document?

5 MS. ALEXANDER: Yes, I can refer you  
6 to this is the review conducted by U.S. EPA Office  
7 of Research and Development, page 2, this is an  
8 attachment to Exhibit 73, under cover of the May  
9 28, 2008 letter, top of page 2. You see the first  
10 bullet is, "No justification" -- My only question  
11 is, the question I was starting to ask is, isn't  
12 it a fact that early on, U.S. EPA expressed the  
13 concern that given that the risks presented are  
14 only for a few gastrointestinal pathogens and  
15 risks were not presented for Hepatitis A,  
16 Shigella, campylobacter, to name a few. The risks  
17 presented will be biased low."

18 DR. GERBA: I think when we  
19 discussed this yesterday. Again, if you want me  
20 to go through the list, hepatitis A, there is a  
21 vaccine available for that. The incidence is  
22 declining. The exact amount of Hepatitis A is  
23 very small so the risk is going to be much smaller  
24 than the other risks we have. We picked the

1 organisms and viruses that would be in the  
2 greatest concentration, and therefore present the  
3 greatest risk. Shigella, there have been no  
4 recreational outbreaks in at least the -- at least  
5 from 1971 associated with sewage discharges. It  
6 was only with bathers in the water where there was  
7 accident fecal discharges in the water. Also the  
8 organism does not survive well in the aquatic  
9 environment and methodologies for it.

10 Campylobacter is another one  
11 that does not survive well in the environment.  
12 There are many sources in the environment, birds  
13 excrete this and seagulls and that, so the amount  
14 of risk to the other water borne pathogens would  
15 be low. And the methodology for getting it out of  
16 waste waters is inadequate and would underestimate  
17 the true concentration. So we went through that  
18 with the EPA and discussed those, and as far as I  
19 know there was agreement to that. Salmonella was  
20 the one selected because it's usually always found  
21 in waste water, certainly raw waste water it can  
22 be detected fairly earlier and easier. EPA has  
23 developed regulations using Salmonellas.  
24 Salmonella has been used as an indicator of

1 recreational water quality in Europe.

2 MS. ALEXANDER: Dr. Gerba, are you  
3 disagreeing with the concern expressed by U.S. EPA  
4 here and in their subsequent correspondence that  
5 the fact that the analysis includes only a few  
6 gastrointestinal pathogens will bias the risk as  
7 low?

8 MR. ANDES: I'm going to object to  
9 the characterization of subsequent correspondence.  
10 You can ask him about that particular  
11 correspondence. Correspondence were different for  
12 different statements.

13 MS. ALEXANDER: I'll limit it as to  
14 this correspondence.

15 My question is, are you disagreeing  
16 with the statement here that choice, including  
17 only a few gastrointestinal pathogens, will bias  
18 the risk low?

19 DR. GERBA: I couldn't say that. I  
20 think we went to the high side because I think the  
21 risk from these would be insignificant compared to  
22 Salmonella, for example, for all the reasons I  
23 just gave you. So, no, I don't think we -- you  
24 might move it up in insignificant notch by

1 including one. But saying it's low, low that's a  
2 relative term.

3 MS. ALEXANDER: So there is a  
4 possibility it could be biased low, but you are  
5 not quantifying that?

6 DR. GERBA: Yes, I'm saying  
7 statistically it probably wouldn't be any  
8 different, if you give the variations of the  
9 limitations that I just did for what we used with  
10 Salmonella. The data and estimate would be  
11 statistically different.

12 MR. ANDES: If I can follow-up. Is  
13 it accurate in terms of what you just said that  
14 overall you believe the risk assessment was biased  
15 high?

16 DR. GERBA: Yes, I believe it was on  
17 the high side actually.

18 MR. ANDES: In terms of the -- back  
19 to Dr. Tolson -- back to the July 12, 2007 letter,  
20 is it accurate for me to read that in the  
21 discussion about how, concerning a discussion of  
22 only including certain pathogens, the first  
23 statement by EPA is "We believe it would be  
24 helpful to also include...?"

1 DR. TOLSON: That is correct, that's  
2 the characterization by the EPA.

3 MR. ANDES: Thank you.

4 CHAIRMAN TIPSORD: Ms. Alexander, do  
5 you have more questions along this line or can we  
6 take a short break?

7 MS. ALEXANDER: I do, but I don't  
8 mind taking a break. That's fine.

9 CHAIRMAN TIPSORD: Let's take a  
10 ten-minute break.

11 (Whereupon, a break was taken  
12 after which the following  
13 proceedings were had.)

14 CHAIRMAN TIPSORD: It's 11:11.  
15 Let's continue with Ms. Alexander.

16 MS. ALEXANDER: Okay, I'm continuing  
17 with questions on the document we were on before  
18 the break, which is the July 12, 2007 letter, a  
19 portion of Exhibit 73.

20 I want to call your attention at  
21 the bottom of the first page to the quotation of  
22 text from the document we were previously  
23 referring to on the cover of the May 31, 2007  
24 letter.

1                   MR. ANDES: I'm sorry, where are we  
2 again?

3                   MS. ALEXANDER: We are now on the  
4 July 12, 2007 letter.

5                   MR. ANDES: Okay.

6                   MS. ALEXANDER: Bottom of the page,  
7 the fourth bullet on the page states, the  
8 quotation states, and then there's a quotation of  
9 text from the May 31, 2007 letter which we've  
10 established is a summary of the April 10th meeting  
11 and the quoted language reads: "There were  
12 comments regarding the use of risk model pathogen  
13 and analytical data. Please note that the  
14 microbial concentrations were not estimated. They  
15 were based on actual measured concentrations in  
16 the samples collected from the waterways." And  
17 then I would call your attention on the following  
18 page to the comment on that text made by U.S. EPA  
19 in the letter which is, "But actual samples are  
20 only an estimate of the range of pathogens that  
21 can occur. The observed data can be used to  
22 estimate a distribution of pathogen exposure."  
23 And my question is, in the risk assessment did you  
24 do this? Did you use the observed data to

1 estimate a distribution of pathogen exposure?

2 DR. TOLSON: No, we did not. We did  
3 a boot strap resampling within our Monte Carlo  
4 analysis.

5 MS. ALEXANDER: So that was the boot  
6 strap analysis?

7 DR. TOLSON: Yes.

8 MS. ALEXANDER: Moving down to the  
9 text that begins, the second bullet on page 3 --  
10 do you see that on page 2, and then they quote the  
11 language from the attachment to the May 31st  
12 letter which stated, "The U.S. EPA requested that  
13 the report also examine stratified risk to  
14 determine illness rates associated with specific  
15 secondary contact activities, such as canoeing and  
16 fishing." In the final report, we will include a  
17 summary of the portion of the overall illness that  
18 were attributed to the identified uses (canoeing,  
19 fishing and recreational boating)." And then I  
20 call your attention to the U.S. EPA's response to  
21 that quote which states, "The approach described  
22 above while useful is not the same as reporting  
23 stratified estimates. Stratified estimates should  
24 include illness rates for each activity, not just

1 a portion of illness attributed to that activity.  
2 For example, what would the risk be for one  
3 thousand canoeists on the Chicago area waterways.  
4 Since such competitive activities take place in  
5 the waterway, this is a relevant question." Now  
6 my question to you is, did you in fact do the  
7 analysis recommended here?

8 DR. TOLSON: I believe so. I  
9 believe we've discussed that. Maybe that was in  
10 my testimony.

11 MS. ALEXANDER: You've included  
12 stratified estimates for illness rates for each  
13 activity, not just the proportion of illness  
14 attributed to that activity?

15 DR. TOLSON: That is correct. If  
16 you go to Exhibit 71, table 5.12 you'll see that  
17 individual activity and their risks are listed on  
18 table 5.11, a proportion of recreational use  
19 attributed to gastrointestinal illnesses due to  
20 effluent disinfection, we did what we said we did  
21 and we carried it a step further and produced the  
22 actual numbers. And we broke that down not only  
23 by recreational activity but by waterway stretch  
24 that we evaluated.

1 MS. ALEXANDER: I'm going to call  
2 your attention to the text beginning the last  
3 bullet on page 3, it states and then the quoted  
4 text from the earlier document is "U.S. EPA fish  
5 consumed by sensitive population, we explained  
6 that the fish consumption was not included in the  
7 study design. We added that states typically  
8 issue fish advisories to protect sensitive  
9 populations." And then I call your attention to  
10 the response which follows, "Our concern on this  
11 issue is not what is the risk of fish consumption  
12 in and of itself, it is that people engage in  
13 fishing and incidental contact activity have a  
14 likelihood of consuming fish they catch in the  
15 waterways, which will lead to an overall higher  
16 risk for that group, even though they are engaging  
17 in an incidental contact activity, the true total  
18 risk for appreciable percentage of anglers is the  
19 risk of secondary exposure to the water, plus the  
20 risk due to fish consumption since fishing is  
21 being actively promoted on several portions of the  
22 waterways, the is studies should calculate the  
23 total risk to this group."

24 Now, Dr. Gerba, I'm aware of

1 your testimony regarding your views on that  
2 exposure pathway, but my only question is, is it  
3 your understanding that in fact this issue of  
4 whether or not fish consumption is an exposure  
5 pathway was never resolved to the satisfaction of  
6 U.S. EPA?

7 DR. GERBA: As far as I know it was.  
8 We discussed it.

9 MS. ALEXANDER: Does the language  
10 that I just read in your understanding reflect a  
11 resolution of that issue to their satisfaction?

12 DR. GERBA: I don't even understand  
13 the type -- if they are talking about  
14 microorganisms if you processed the fish and  
15 cooked it, there is no risk. So I'm not sure what  
16 the issue is here. As far as I'm aware it was  
17 resolved.

18 MS. ALEXANDER: I'm aware of your  
19 testimony, that there has been some discussion  
20 previously in the record as to whether or not this  
21 issue was resolved to the satisfaction of the EPA.  
22 And my question is, does it not appear from this  
23 that U.S. EPA in fact was not satisfied with your  
24 response and still wanted you to, as of the date

1 of this letter, to include that information in  
2 your risk assumption, in your risk assessment?

3 DR. GERBA: I don't know. I really  
4 couldn't say. As far as I know, it was  
5 satisfactory. I have nothing I can really add to  
6 that except for what I've already stated.

7 CHAIRMAN TIPSORD: You have to speak  
8 up.

9 DR. GERBA: I don't know what I can  
10 add to that except for what I've already stated.  
11 I don't know if it was the same individual that  
12 responded. As far as I know, it wasn't.

13 DR. TOLSON: I'd like to point out  
14 that people who are engaged in fishing have  
15 contact and ingestion of the water, and that  
16 ingestion is about half of what canoeing is. It's  
17 fairly appreciable ingestion.

18 MS. ALEXANDER: That's a different  
19 exposure pathway than eating the fish that comes  
20 from the water, correct?

21 DR. TOLSON: That is correct. We  
22 did not evaluate eating the fish, but we did  
23 evaluate people licking their hands after they  
24 were playing with the fish.

1 MS. ALEXANDER: But this reference I  
2 just read concerns fishing consumption, is that  
3 correct, as opposed to the other pathway you just  
4 referenced?

5 DR. TOLSON: That is correct.

6 DR. PETROPOULOU: That is what was  
7 discussed at the meeting when they brought up the  
8 issue at the April 10th meeting. They were  
9 talking about eating the fish.

10 MS. ALEXANDER: Correct. And that's  
11 what is referenced here, fish consumption, eating  
12 the fish.

13 DR. PETROPOULOU: Right, this is a  
14 new comment. I concur that the reviewer who wrote  
15 this comment expresses a new concern about fish.

16 MS. ALEXANDER: Was Mr. Melzer, the  
17 signatory of this letter, at that meeting? We can  
18 probably answer that from the document. I would  
19 call your attention to the attachment to the May  
20 31, 2007 letter, page 1.

21 MR. ANDES: Mr. Melzer is the active  
22 chief of the branch. Do we have any reason to  
23 believe these are his comments?

24 MS. ALEXANDER: I would call to your

1 attention that Mr. Melzer was at the meeting.

2 MR. ANDES: We have no foundation  
3 for this because we don't know who wrote this  
4 letter from the EPA, do we?

5 MS. ALEXANDER: I call your  
6 attention to page 3, isn't it a fact that Allen  
7 Melzer is the person who signed this letter?

8 DR. TOLSON: His signature is on the  
9 letter, that's correct.

10 MS. ALEXANDER: Do you have any  
11 reason, one way or the other, to believe that he  
12 didn't write this letter?

13 DR. TOLSON: I don't know that.

14 MS. ALEXANDER: Lastly, I call to  
15 your attention the only line on page 3, "Please  
16 call me at" -- and he provides a phone number --  
17 "if you'd like to discuss these further." Am I  
18 correct from your previous testimony that you did  
19 not in fact -- neither you or nor anyone else from  
20 GeoSyntec didn't call?

21 MR. ANDES: The letter wasn't  
22 addressed to them.

23 MS. ALEXANDER: I'm sorry, you are  
24 right. It was addressed to MWRD. I assume you

1 did not have further contact with Dr. Melzer after  
2 this based on your earlier testimony?

3 DR. TOLSON: I did not.

4 MS. ALEXANDER: Do you have any  
5 knowledge as to if anyone from MWRD had any  
6 further knowledge?

7 MR. ANDES: I don't know if he is a  
8 doctor. I don't know anything about his  
9 scientific background.

10 MS. ALEXANDER: I'm sorry, I should  
11 have said Mr. Melzer. I don't know his  
12 background. Let me ask the same question of  
13 Dr. Gerba and Petropoulou. Do you have any  
14 knowledge as to anyone from MWRD ever contacted  
15 Mr. Melzer after this letter?

16 DR. PETROPOULOU: In general?

17 MS. ALEXANDER: No, following the  
18 receipt of this letter at the investigation to  
19 call him if necessary.

20 DR. PETROPOULOU: I don't know.

21 DR. GERBA: I don't know.

22 MS. ALEXANDER: Okay.

23 MR. ANDES: Can I follow-up for a  
24 moment? In terms of the EPA's request to add the

1 risk due to fish consumption to the risk of  
2 secondary exposure, you've discussed in the report  
3 the risk of secondary exposure?

4 DR. TOLSON: Yes, and is it your  
5 testimony that the fish consumption would be  
6 negligible?

7 DR. GERBA: Yes.

8 MR. ANDES: So it would add a  
9 negligible amount to the risk?

10 DR. GERBA: Yes.

11 MS. WILLIAMS: Can I follow-up?  
12 When you use the term secondary exposure, did you  
13 mean to say secondary contact exposure as opposed  
14 to -- isn't secondary exposure a term you use when  
15 referring to people who weren't recreating?

16 MR. ANDES: I'm using the EPA's term  
17 in the letter.

18 DR. GERBA: I'd have to look at the  
19 letter. Even though they are engaging in  
20 incidental contact, the two total risks for a  
21 appreciable anglers is it's a risk of secondary  
22 exposure to the water plus the risk due to  
23 consumption.

24 MS. WILLIAMS: So what does

1 secondary mean as it is used in there?

2 DR. GERBA: Well, I didn't write it,  
3 but he refers to angling as a secondary exposure,  
4 meaning there's no direct link or contact with the  
5 water is what I assume.

6 MR. ANDES: It's not like swimming.

7 DR. GERBA: Not like swimming. So  
8 in other words, fishing here is considered a  
9 secondary exposure.

10 MS. WILLIAMS: Thank you.

11 MS. ALEXANDER: I have what I  
12 believe is my last question or set of questions on  
13 Exhibit 73, which concerns page 14 of the 15 page  
14 document, whichever one that is.

15 CHAIRMAN TIPSORD: Which is the  
16 attachment to the May 28th letter?

17 MS. ALEXANDER: Yes, I'm sorry,  
18 attachment to the May 28th letter, and I would  
19 call your attention -- you'll see that there is a  
20 discussion of a text on pages 115 to 116 of the  
21 report, and then there's a response. And I'm  
22 going to call your attention to the second to the  
23 last sentence which states -- of the response --  
24 which states, "Therefore the proposed dynamic

1 model" -- and this is referring to secondary  
2 transmission" -- considers a stated estimated  
3 level of immunity and estimated disease incidence  
4 only in the recreational population and their  
5 immediate family." Do you see that?

6 DR. TOLSON: Yes -- I don't have it  
7 in front of me.

8 MS. ALEXANDER: Just to clarify,  
9 that means that this disease model did not  
10 consider anyone -- let me ask the question. Who  
11 was included in immediate family?

12 DR. TOLSON: Say that again.

13 MS. ALEXANDER: Who is included in  
14 the immediate family?

15 DR. TOLSON: For that input we  
16 looked at Cook County census records to figure out  
17 the number of people living within one household  
18 and there's a distribution obviously, so it's  
19 somewhere between one and it was eight or so  
20 individuals in the house.

21 MS. ALEXANDER: So in other words  
22 your disease transmission model did not consider  
23 secondary transmission by, you know, any family  
24 members not living in the household or friends?

1 DR. TOLSON: That is correct. It is  
2 limited as it says in the report to the immediate  
3 family.

4 MS. ALEXANDER: Okay.

5 MR. ANDES: Follow-up. Could you  
6 read the last sentence of that paragraph, your  
7 response on that issue?

8 DR. TOLSON: The last sentence is,  
9 "This approach addresses the important dynamic  
10 aspects of disease transmission from CAWS exposure  
11 and the population most at risk."

12 MR. ETTINGER: I'm confused. You  
13 looked at secondary diseases within a family, is  
14 that what you did?

15 DR. TOLSON: That is correct.

16 MR. ETTINGER: How do these diseases  
17 spread? You don't have to get too graphic.

18 DR. TOLSON: Dr. Gerba will give you  
19 the more interesting explanation, so I'm going to  
20 defer to him.

21 MR. ETTINGER: Just in general.

22 DR. GERBA: Most of these diseases  
23 are transmitted by the fecal-oral route. So they  
24 could be spread in the family by touching food

1 surfaces. You completely wash your hands in the  
2 restroom, but you've always got a little bit of  
3 fecal material on your hands. So you can spread  
4 it from one location to another, and somebody puts  
5 their fingers in the mouth, for example, which  
6 children do at a much more frequency than adults,  
7 that's been quantitated in the risk models.

8 MR. AL: Is that why we have waiters  
9 wash their hands after they use the restrooms?

10 DR. GERBA: That's one of the  
11 reasons why they do.

12 MR. ETTINGER: So adults can  
13 potentially spread this disease to unrelated  
14 persons if they don't wash their hands in the  
15 restroom?

16 DR. GERBA: That's correct.

17 MR. ETTINGER: And they might maybe  
18 go into a Subway if they hadn't washed their hands  
19 and put their hand on a rail --

20 DR. GERBA: That's correct.

21 MR. ETTINGER: Thank you.

22 MR. ANDES: Please expand on that.

23 DR. TOLSON: If you refer to  
24 Exhibit 71 on the uncertainty and sensitivity

1 analysis on page 131, we clearly indicate that  
2 that's a limitation of our study, that there's a  
3 potential that this may have underestimated total  
4 population risk. We don't think that  
5 underestimate is due to a significant degree given  
6 the conservative nature to which we described  
7 secondary attack rates.

8 MR. ANDES: Can you state the last  
9 sentence of that paragraph, explain why that's  
10 likely very low? Do you want to read that?

11 DR. TOLSON: "Due to the small  
12 recreational population compared to the total  
13 metropolitan population and the endemic nature of  
14 pathogens on the population, this essential  
15 underestimation of risk and the effects of  
16 recreational illness is based on population  
17 illness rate is likely very low."

18 MR. ANDES: Isn't it also accurate  
19 in the previous bullet, that your assumptions on  
20 the secondary transmission rates are actually  
21 biased high?

22 DR. TOLSON: Correct. As we stated,  
23 secondary transmission rates are generally at the  
24 high end of those reported in the technical

1 literature, therefore the assumptions of secondary  
2 transmissions are conservative and as a result the  
3 rates are biased high.

4 MR. ETTINGER: How is that  
5 literature developed? What did they do to  
6 determine the secondary rates?

7 DR. GERBA: A lot of these studies  
8 took place in households Rotavirus, Salmonella or  
9 E. Coli. They look at the number of individuals  
10 who became ill after the index case in the house.

11 MR. ETTINGER: Did they look at the  
12 population size of the area in which the people  
13 were living in conducting these studies?

14 DR. GERBA: To go beyond that, they  
15 used dynamic models, secondary transmission in  
16 epidemiology. That's another approach for  
17 microbial risk assessment is to put this in a  
18 dynamic model to determine the effect on the  
19 entire community. It takes a lot of work, but  
20 generally I don't like that approach because it  
21 minimizes the risk here that we saw. You can see  
22 that most of these illnesses for example, like  
23 Rotavirus are being spread by people who have  
24 developed foodborne illnesses who go to a show or

1 a hospital or some other case, and I think using  
2 that approach would minimize the risk that we see  
3 here because it makes the newer risk look totally  
4 insignificant compared to the enteric viral  
5 infections going on in Chicago at one time.

6 MR. ETTINGER: It would minimize it  
7 in terms of percentages, but if I were to look at  
8 the number of total cases, that would be something  
9 I would want to look at, isn't it?

10 DR. GERBA: It gets much more  
11 difficult once you move beyond the household  
12 because then how many times do you touch the  
13 Subway handle, and how many times are Nora virus  
14 there in that -- your speculation and assumptions  
15 become gigantic after that point after. You are  
16 doing it. Usually people default into using  
17 epidemiological models to go beyond that, but that  
18 takes a great, a lot of assumptions. And, again,  
19 it would minimize, if I wanted to show that was no  
20 impact on this community, that's what I would use,  
21 the dynamic models. We wanted to be more  
22 conservative than that and look at who is going to  
23 become ill. And I think the uncertainty would  
24 become huge at that point. That's one of the

1 reasons I think that approach has a lot of  
2 limitations to it.

3 MR. ETTINGER: Would it make a  
4 difference to your model whether there were a  
5 hundred canoers a year or a million canoers a  
6 year?

7 MR. ANDES: In what respect? Would  
8 it make a difference to his model?

9 MR. ETTINGER: Would it make any  
10 difference to the conclusions if there were a  
11 hundred canoers or a million?

12 DR. TOLSON: If you were to go all  
13 the way up to a million, then I might consider  
14 changing the way that we view our models and make  
15 them population based as opposed to the way we've  
16 done it here because at that point pretty much  
17 everybody is a recreater, and the dynamics would  
18 change in the population. But I don't anticipate  
19 having a million -- I don't anticipate having the  
20 entire community of Chicago on the waterway. If  
21 that were the case, I may change the fundamental  
22 structure of the way we did our model.

23 MR. ETTINGER: Somewhere between a  
24 hundred and a million would you start thinking

1 about it or it's only a million --

2 MR. ANDES: A hundred thousand. We  
3 don't have testimony in terms of the actual number  
4 of recreaters. And if it did, it would be more  
5 than a few thousand, not a hundred thousand.

6 MR. ETTINGER: Well, we don't know  
7 what's going to happen. I'm asking a hypothetical  
8 question if the number of canoers were to increase  
9 a great deal, would that effect the relevance of  
10 your study?

11 DR. TOLSON: If a large portion of  
12 the entire metropolitan area, Chicago area were  
13 being engaged into recreating on the waterway,  
14 then it might make sense to evaluate it on a  
15 population level. In that case we would tend to  
16 dilute the effect of the waterway because you'd  
17 have to take into account the endemic illness  
18 rates for all these different pathogens that are  
19 spread around through ways other than contacting  
20 through the waterway, and those would need to be  
21 integrated through the model.

22 MR. ETTINGER: Now, just as a matter  
23 of arithmetic, if I've got two cases per a  
24 thousand, I'm going to have more total cases if

1 I've got a hundred thousand users than one  
2 thousand users, right?

3 DR. TOLSON: Your arithmetic is  
4 exactly right.

5 MR. ETTINGER: And nothing you did  
6 looks at that issue?

7 MR. ANDES: Looks at that issue? He  
8 laid out risk numbers.

9 MR. ETTINGER: That's it. That's  
10 what I'm asking. You did not -- none of your  
11 conclusions are effected by how many users there  
12 are?

13 DR. TOLSON: We selected the models  
14 and the methodologies that we think would be the  
15 best to capture potential risk from the waterway.  
16 Those tend to overestimate the risk and the  
17 contributions from the waterway, did not include  
18 dynamic models that were population based. Had we  
19 gone to population based models, we would have  
20 arrived at different conclusions, but we would  
21 have had another four days of testimony on all  
22 those additional assumptions that we would have  
23 made about whether somebody was ill because of the  
24 tomato that they had on their sandwich rather than

1 the waterway.

2 MR. ETTINGER: If you were going to  
3 build a dynamite factory that had a one in a  
4 thousand chance of exploding, but you were going  
5 to put it in a remote location or across the  
6 street from a school --

7 MR. ANDES: That is just not  
8 relevant. It's hypothetical. We are not talking  
9 about a dynamite plants.

10 CHAIRMAN TIPSORD: Is that an  
11 objection?

12 MR. ANDES: It absolutely is.

13 CHAIRMAN TIPSORD: Sustained. Let's  
14 move on.

15 MS. MEYERS-GLEN: I'll just ask one  
16 quick follow-up to that. On page 7 -- well,  
17 looking at, Dr. Tolson, attachment 3, page 120 of  
18 your testimony, just following up on a quick  
19 secondary attack rates question that was asked,  
20 you confine your analysis of secondary attack  
21 rates to immediate family and you don't go expand  
22 beyond that is what I'm hearing; is that correct?

23 DR. TOLSON: That's correct. We may  
24 have underestimated the total illness rates that

1 could be contributed to that.

2 MS. MEYERS-GLEN: And in other parts  
3 of your testimony, in fact you talk about how --  
4 I'm trying to remember -- yes, viruses,  
5 cryptosporidium in common settings are commonly  
6 spread in daycare centers and schools; is that  
7 correct?

8 DR. TOLSON: Correct.

9 MS. MEYERS-GLEN: So if a child or  
10 youth is paddling on the waterway and is  
11 asymptomatic, but then goes to daycare, that was  
12 not accounted in your study, even though you  
13 listed that as being one of the places where  
14 studies occur predominantly for this type of  
15 virus; is that correct? That's where you get a  
16 lot of your data as far as endemic behavior for  
17 cryptosporidium; is that correct?

18 DR. GERBA: Can I make a correction.  
19 It's a parasite, not a virus.

20 MS. MEYERS-GLEN: Sorry about that.

21 CHAIRMAN TIPSORD: Ms. Meyers-Glen,  
22 you've already asked him two questions and now you  
23 are getting ready to ask a third. Can he answer  
24 the first two before you ask the third?

1 DR. TOLSON: Going back to the first  
2 question, did we account for the potential for a  
3 child recreating on there to spread that to a  
4 school, to his class at school?

5 MS. MEYERS-GLEN: That's correct.

6 DR. TOLSON: No, we did not account  
7 for that specific pathway.

8 MS. MEYERS-GLEN: Your data, whether  
9 you are talking about cryptosporidium -- you use  
10 that very situation because in your data you  
11 talked about how cryptosporidium through crowded  
12 settings is spread through daycare centers and  
13 schools; is that correct?

14 DR. TOLSON: That's correct. I  
15 recall for dry weather or upstream or downstream  
16 there was no cryptosporidium, no infections.

17 MS. MEYER-GLEN: That you detected?

18 MR. ANDES: They can detect it.

19 MS. MEYERS-GLEN: They did detect it  
20 in wet weather. So it was detected in the study?

21 DR. GERBA: Some of the secondary  
22 attack rates we discussed were developed from  
23 daycare centers too, just to mention that.

24 MS. MEYERS-GLEN: The reason being,

1     there are hot beds for that kind of an illness,  
2     correct?

3                     DR. GERBA: I hope they are not hot  
4     beds, but secondary transmission occurs in there.  
5     I think situations are better than they used to be  
6     because of laws in certain states requiring them  
7     to use disinfectants and cleaning, but usually you  
8     get a greater secondary spread because children  
9     don't have great sanitary habits as adults do.

10                    MR. ANDES: So those rates were  
11     considered in this calculation?

12                    DR. GERBA: So, yes, those rates  
13     were considered.

14                    MR. ANDES: And the higher attack  
15     rates you see are because they come from daycare  
16     centers.

17                    MS. MEYERS-GLEN: But then that  
18     wasn't applied as far as outside the family?

19                    DR. GERBA: Attack rates were.

20                    MR. ANDES: You need to explain.  
21     Would you explain rates secondary transmission?

22                    DR. GERBA: One is tax rates, a tax  
23     rate, a lot of them have been developed in daycare  
24     centers or institutions where you have a large

1 number of people?

2 MR. ANDES: We use those rates?

3 DR. GERBA: We use those rates  
4 because they tend to be higher because small  
5 children with poor sanitary habits, so it spreads  
6 easier with a lot of those infections.

7 DR. TOLSON: I think -- what you are  
8 getting at is, we don't have a separate subgroup  
9 that looks at how many children are actually on  
10 the waterway and how many of those could possibly  
11 be transmitted to a larger than their family size,  
12 which would be class size.

13 MS. MEYERS-GLEN: This is not only  
14 for daycare centers, but for crowded centers like  
15 nursing homes, correct?

16 DR. TOLSON: Correct.

17 MR. ANDES: Do you expect a lot  
18 people in nursing homes to kayaking on the  
19 waterways.

20 DR. TOLSON: I don't have any data  
21 to support that that happens.

22 MS. MEYERS-GLEN: Or summer camp?

23 DR. TOLSON: We don't have data from  
24 the UAA on specific age ranges of the individuals

1 that were participating.

2 DR. GERBA: That would take a lot --  
3 that kind of information would take a lot of  
4 speculation to do that. We'd have to figure out  
5 how often some of these people might go to a  
6 daycare center or children who are not preschool  
7 children. So to try to do that, I think, is a  
8 little bit unrealistic. You have to make so many  
9 assumptions that the uncertainty would be  
10 increased to a great degree. If you wanted to  
11 look at specific groups of people, like how often  
12 does a child get infected and how often do they go  
13 to a nursing home after a period of time in which  
14 they are in infected, which may only last a week  
15 to five days, so the amount of uncertainty you are  
16 creating becomes greater and greater. Especially  
17 when you don't have data to back up that type of  
18 assumption with the frequency of occurrence.

19 CHAIRMAN TIPSORD: Ms. Alexander?

20 MS. ALEXANDER: I'm going to  
21 continue with questions that were posed in the  
22 pre-filed questions specifically to Dr. Gerba.  
23 The first question is number four to Dr. Gerba,  
24 which is regarding the statement on page 5 of your

1 testimony that disinfection "is warranted in  
2 situations where direct human contact in the  
3 immediate vicinity of an outfall is possible," and  
4 the question is, do you have any basis to believe  
5 that recreation on the CAWS does not occur in the  
6 immediate vicinity of the water reclamation  
7 district outflow?

8 MR. ANDES: I believe we answered  
9 that yesterday that specific question.

10 MS. ALEXANDER: I don't have a  
11 recollection that that specific question was  
12 answered, and it's pretty much a yes or a no.

13 MR. ANDES: I think because I  
14 objected and asked for clarification of it. So  
15 I'd rather not go back over that question.

16 MS. ALEXANDER: My recollection is  
17 that he did not have any basis to believe. Do you  
18 have any different recollection?

19 MR. ANDES: Any basis to believe?

20 MS. ALEXANDER: Any basis to believe  
21 the recreation does not occur in the immediate  
22 vicinity of the water reclamation district  
23 outflow.

24 MR. ANDES: We had an extended

1 conversation of what direct human contact meant.

2 CHAIRMAN TIPSORD: My recall is this  
3 was specifically about Stickney and North -- it  
4 was only about two, not the more general, all of  
5 the outfalls. And he did specifically discuss  
6 there not being Stickney, and also because you had  
7 not taken samples at the one site close to the  
8 outfall for safety reasons and this is only based  
9 on the captain -- is this bringing it back to you,  
10 Ms. Alexander?

11 MS. ALEXANDER: Yes. This is a very  
12 general question as to whether you have any basis  
13 to believe generally that recreation does not  
14 occur in the immediate vicinity of any of the  
15 reclamation district outfalls, any of the three  
16 here? Do you have any knowledge yourself or any  
17 basis?

18 CHAIRMAN TIPSORD: We've already  
19 discussed Stickney and North Side. So could you  
20 discuss the other ones.

21 DR. GERBA: What do you mean  
22 vicinity?

23 MS. ALEXANDER: Vicinity is your  
24 term, Dr. Gerba, so I would put that back to you.

1 Your statement was, "Disinfection is warranted in  
2 situations where direct human contact in the  
3 immediate vicinity of an outfall is possible.

4 DR. GERBA: What was your question  
5 again?

6 MS. ALEXANDER: My question is do  
7 you have any basis to believe that recreation on  
8 the CAWS does not occur in the immediate vicinity  
9 of the water reclamation outfalls?

10 DR. GERBA: No.

11 MR. ETTINGER: I think yesterday I  
12 did ask about that quote, and I think I also asked  
13 you what you meant by immediate vicinity, and I  
14 think you said something like it depends or that  
15 you have to look at different factors. Do you  
16 recall that?

17 DR. GERBA: Yes, I do. I said it's  
18 a site specific situation.

19 MR. ETTINGER: Yes. If I were --

20 MR. ANDES: I think he already  
21 answered it.

22 MR. ETTINGER: I'm going to ask a  
23 new question based on that. What factors would I  
24 use then to decide what the immediate vicinity is?

1 DR. GERBA: Well, I think it depends  
2 on a lot of factors. Probably a lot to do with  
3 hydrology, dilution rates, the volume of the waste  
4 water relative to the volume that it's being  
5 released in. The type of treatment, degree of  
6 treatment the waste water may receive. The types  
7 of flows or CSOs involved in that type of  
8 treatment or are they contained in that process.  
9 Water use in the community. And a lot of it has  
10 to do with how, where the outfall might be located  
11 in that area. There's a lot of factors, rainfall  
12 events and other things that might be considered.

13 MR. ETTINGER: Well, so, if I were  
14 trying to decide whether or not to disinfect at a  
15 plant that was some distance from a beach, I would  
16 look at all of those factors?

17 DR. GERBA: I think I would put all  
18 of those into consideration in the water quality  
19 on the beach and the occurrence and concentration  
20 of pathogens in the water too, because a lot of  
21 those factors, without actual data on the  
22 occurrence of pathogens and indicators might be  
23 difficult to sort out, so you would take a whole  
24 range of factors in there.

1                   MR. ANDES: And this really has been  
2 addressed. This is pretty much a repeat of his  
3 answer from yesterday.

4                   MR. ETTINGER: No, it's not.

5                   DR. GERBA: Excuse me, I thought it  
6 was because I just listed the factors I listed  
7 yesterday.

8                   MR. ETTINGER: How many miles could  
9 the pathogens continue downstream from the plant?

10                  DR. GERBA: How many miles?

11                  MR. ETTINGER: Of pathogens.

12                  DR. GERBA: Well, one, right a way,  
13 it depends on the pathogen. It depends on the  
14 flow rate. It depends on the climate. There are  
15 a large number of factors that have to be  
16 considered.

17                  MR. ETTINGER: Well, your study  
18 assumes that some of these pathogens hang around  
19 for three or four days after a weather even occur.

20                  DR. GERBA: I assumed that it did --

21                  MR. ANDES: You asked that it  
22 assume. Did you assume anything or measure?

23                  DR. GERBA: (Response inaudible.)

24                  CHAIRMAN TIPSORD: She's right, I've

1 got four people talking at once. We all need to  
2 talk at once. We're going to ask these questions  
3 and we're going to answer them. If some  
4 repetition occurs, I apologize, but it's already  
5 been a long couple of days.

6 Dr. Gerba, do you two need to  
7 confer before I ask this?

8 Dr. Gerba, the question was, did you  
9 assume that the pathogens did or did you actually  
10 measure? And your answer is.

11 DR. GERBA: Measured the pathogens.

12 CHAIRMAN TIPSORD: Mr. Ettinger then  
13 had a follow-up question based on that.

14 MR. ETTINGER: You found through  
15 measurements that some pathogens can live up to  
16 three days in ambient rivers, correct?

17 DR. GERBA: Let me qualify that,  
18 when you say live, they may be decreasing in  
19 concentrations. Usually once you discharge a  
20 pathogen, they will be decreasing in population.  
21 So we usually refer to things like, okay, in  
22 24 hours you get 90 percent reduction, you get  
23 99 percent reduction. So there's no like one  
24 individual lives and dies, so they decrease in

1 concentration over time. Once they leave -- well,  
2 actually once they leave the human body, it's a  
3 rough world out there so they decrease.  
4 Particularly in waterways. It's not as we talked  
5 about yesterday things like sunlight will  
6 inactivate organisms is one factor, antagonistic  
7 organisms, which eat the organisms you put out  
8 there. That's why you can't answer that  
9 generically. They will be decreasing over time is  
10 the best answer. But usually from a discharge you  
11 might find these organisms three days later.

12 MR. ETTINGER: So depending on flow  
13 conditions, you might be concerned about a beach  
14 that was three days below a sewage discharge  
15 plant?

16 DR. GERBA: That's always a  
17 possibility, but finding a pathogen doesn't mean  
18 there's a significant risk or finding an indicator  
19 there isn't a significant risk. In other words, I  
20 could start out with a hundred pathogens per  
21 hundred meters, and by the time it gets to the  
22 beach, because of inactivating factors such as  
23 sunlight. Basically that level of risk is one and  
24 becomes insignificant or meets the requirements

1 that might be set by the regulatory agency for the  
2 risk.

3 MR. ETTINGER: Thank you.

4 MS. ALEXANDER: Continuing with  
5 Dr. Gerba. Question number five, and that is  
6 regarding the discussion on page 5 of your  
7 testimony concerning disinfections byproduct,  
8 which I may refer to as DBPs. Are DBPs produced  
9 as a byproduct of chlorination?

10 DR. GERBA: Yes.

11 MS. ALEXANDER: Does UV, ultraviolet  
12 disinfection create the same type and level of  
13 DBPs as chlorination?

14 DR. GERBA: Repeat, DBPs?

15 MS. ALEXANDER: DBPs, disinfection  
16 byproducts.

17 DR. GERBA: With UV light there is a  
18 lot of uncertainty about potential of disinfection  
19 byproducts because it hasn't been studied that  
20 much. I was on U.S. EPA's advisory committee for  
21 five years, and I've attended workshops on UV  
22 light. And one of the things that comes through  
23 is they really haven't been studied very  
24 thoroughly. There have been fewer byproducts, if

1     there are any byproducts.  But questions have been  
2     raised about potential production of byproducts  
3     with UV light, particularly going to medium  
4     pressure of vapor lamps, which have a big receptor  
5     of light and it effects more molecules in the  
6     environment.  To say that there might be none, I  
7     think there probably haven't been enough studies.  
8     A lot of people feel there are probably lower  
9     levels of disinfection byproducts, but a lot of  
10    committees I've been on there have been concerns  
11    voiced that we haven't really studied the range of  
12    disinfection byproducts, particularly when we are  
13    looking at the sewage influence which have a large  
14    variety of organic matter that may be effected by  
15    ultraviolet light processes.

16                   MS. ALEXANDER:  I'd like to call  
17    your attention in Exhibit 71 to page 66, if I may.  
18    Let me first ask you, would it be fair in your  
19    view to characterize the level of disinfection  
20    byproducts generated by UV conventional doses as  
21    negligible?

22                   DR. GERBA:  I think -- I didn't  
23    write this section for one thing.  Let me just say  
24    that right off the bat.

1 MS. ALEXANDER: Are you disagreeing  
2 with the statement in this section that "The  
3 formation of harmful byproducts by UV is  
4 negligible at conventional UV doses?"

5 DR. TOLSON: Can you point to us  
6 where in the document?

7 MS. ALEXANDER: That's what I was  
8 searching for. I have it in my notes. It's on  
9 this page, but I may have gotten the page wrong.

10 MEMBER RAO: It's on page 67, third  
11 paragraph.

12 MS. ALEXANDER: Okay, yes, that's  
13 one reference. I believe there is another. But  
14 there's a statement made, and I quote on page 67,  
15 "UV disinfection results in DBPs and is not  
16 discussed further." Do you agree with that  
17 statement?

18 DR. GERBA: Based on the current  
19 state of knowledge for low vapor pressure lamps  
20 and drinking water, yes, I think you could say  
21 it's negligible but it hasn't been studied in  
22 great detail. That's based on the current state  
23 of knowledge.

24 MR. ANDES: I think for the record

1 we have other witnesses later who can testify  
2 further about that issue.

3 MS. WILLIAMS: Who?

4 MR. ANDES: Possibly Dr. Blanchly  
5 and I believe Dr. Hass.

6 MR. ETTINGER: Have you looked at,  
7 on the next page of your testimony, on page 68,  
8 you discuss other disinfectants in addition to  
9 chlorine.

10 CHAIRMAN TIPSORD: Just for the  
11 record, Mr. Ettinger, you say he discusses, he  
12 said he didn't write the report.

13 MR. ANDES: What page, I'm sorry?

14 MR. ETTINGER: Page 68. The EPA  
15 found use of disinfectants other than chlorination  
16 does not necessarily eliminate the use of  
17 halogenated DP -- whatever it is -- disinfection  
18 byproducts is easier than the letters for me. Did  
19 you look at other forms of disinfection?

20 DR. PETROPOULOU: This section  
21 compiles information from other forms.

22 MR. ETTINGER: Did you look at  
23 boron?

24 DR. PETROPOULOU: I don't believe

1 so, no.

2 MR. ETTINGER: Are you aware of  
3 disinfection byproducts from boron?

4 DR. GERBA: Can I ask a question  
5 bromine or boron?

6 MR. ETTINGER: Bromine or boron,  
7 what is that?

8 DR. GERBA: The question is, I think  
9 you are confusing bromine with boron because I've  
10 never heard of boron being used as a disinfectant  
11 before whereas bromine is used as a disinfectant.

12 MR. ETTINGER: I believe I have, but  
13 you are saying boron you have not heard of being  
14 used as --

15 DR. GERBA: No, I have not.

16 MR. ETTINGER: And bromine --

17 DR. GERBA: Has been used as a  
18 disinfectant.

19 MR. ETTINGER: And does it have  
20 disinfection byproduct that could effect aquatic  
21 life?

22 DR. GERBA: Bromine does produce  
23 disinfectant byproducts to my knowledge. Its  
24 effects on aquatic life, I don't know.

1                   MR. ANDES: I may have other  
2 witnesses that can answer that.

3                   MS. ALEXANDER: I wanted to clarify  
4 for the record, I quoted some language a moment  
5 ago which I could not find. It was defined on  
6 page 66. It is in fact on page 64. This is  
7 two-thirds down the page, there is a statement,  
8 "UV disinfectant is reportedly characterized by  
9 the following advantages over chlorine and then a  
10 study is cited from 2004," and the third bullet is  
11 the language I quoted, "The formation of harmful  
12 byproduct by UV is negligible at conventional UV  
13 doses." I offer that as clarification of my  
14 question.

15                   My question is, Dr. Gerba,  
16 turning to your question number six to you, what  
17 is the most common method of disinfection  
18 currently used in waste water treatment?

19                   DR. GERBA: I don't have any  
20 statistics I could quote, but from personal  
21 experience chlorination in the United States.

22                   MS. ALEXANDER: And are you familiar  
23 generally with U.S. EPA health criteria governing  
24 disinfection byproducts?

1 DR. GERBA: No.

2 MS. ALEXANDER: Would you be aware  
3 generally that those criteria are set based on  
4 assumptions of long-term chronic exposure?

5 DR. GERBA: Yes.

6 MS. ALEXANDER: Were you aware that  
7 the maximum containment -- the MCLG -- now I'm  
8 blanking on what exactly that stands for --  
9 maximum contaminant level goal for trichlormethane  
10 was set based on an assumption of studies. The  
11 consumption is two liters per day for a 150 pound  
12 adult over a period of seven years?

13 MR. ANDES: We will have someone  
14 offer testimony on this later.

15 MS. ALEXANDER: I'm just asking if  
16 you are familiar with that or have any  
17 disagreement with that?

18 DR. GERBA: Yes, I'm aware of it  
19 because I served on the EPA's drinking water  
20 advisory committee. For drinking water I should  
21 say all those things are related to what we've  
22 been talking about.

23 MS. ALEXANDER: Are you aware of any  
24 health data or standards that have been set

1 concerning occasional exposure as opposed to  
2 chronic exposure to chlorination disinfection  
3 byproducts in a recreational context?

4 DR. GERBA: Repeat the one part.

5 MS. ALEXANDER: Are you aware of  
6 either any health data that have been generated or  
7 standards promulgated concerning occasional  
8 exposure to chlorination disinfection byproducts  
9 in the recreational context?

10 DR. GERBA: Yes.

11 MS. ALEXANDER: Can you explain?

12 DR. GERBA: There have been various  
13 studies about, particularly in swimming pools,  
14 exposure to disinfection byproducts being inhaled  
15 or absorbed through the skin from chlorination of  
16 swimming pool waters and even hot tubs.

17 MS. ALEXANDER: So am I correct in  
18 concluding from that, that there is current  
19 ongoing exposure to trichlormethane associated  
20 with use of swimming pools that are disinfected  
21 with chlorine?

22 DR. GERBA: I believe there are,  
23 yes.

24 MS. ALEXANDER: This is Gerba

1 question number eight, which is slightly  
2 different. Have there been any studies to your  
3 knowledge of the impact of these byproducts on  
4 recreational users as opposed to their presence in  
5 water?

6 DR. GERBA: I believe there have  
7 been, yes.

8 MS. ALEXANDER: Can you identify any  
9 of those studies or describe them?

10 DR. GERBA: I can't describe the  
11 studies. There have been studies on chlorination  
12 means, for example, causing respiratory problems  
13 among people using swimming pools.

14 MS. ALEXANDER: Based on this  
15 knowledge of any of these studies that you just  
16 referenced, do you have any basis to believe that  
17 the effects of disinfection byproducts on  
18 recreational users would be comparable to the  
19 effects from chronic injection?

20 DR. GERBA: I don't have any basis.  
21 That would be too much speculation.

22 MS. ALEXANDER: Okay. Let me turn  
23 to question nine, which is how do you believe that  
24 the risks from recreational exposure to

1 microorganisms, and I'm asking that question  
2 generally not specific to the CAWS, would compare  
3 to the risks from disinfection byproducts.

4 DR. GERBA: That would be too much  
5 speculation. I would have to sit down and do a  
6 calculation to know that one. But usually risks  
7 are greater from the microorganisms.

8 MR. ANDES: You can expand on that.

9 DR. GERBA: I would point on that  
10 the way you do risk for chemicals is so much  
11 different -- lot of ways much more conservative to  
12 the microbial risks that we actually perform in  
13 that. So that should be pointed out.

14 MR. ETTINGER: Can you read that  
15 back.

16 (Record read as requested.)

17 DR. GERBA: Maybe I should  
18 extrapolate. In the chemical risk assessment, a  
19 lot of times they add a lot of factors that  
20 make -- maybe I want to mention that.

21 DR. TOLSON: Sure. We usually do  
22 that to provide a greater protection because the  
23 end points tend to be very severe end points.  
24 Cancer would be one of them. So there is a lot of

1     uncertainty factors built in. In other words,  
2     there's a desire to err on the side of safety.  
3     For microbial risk assessments we really try to  
4     focus in on what we predict would be the actual  
5     number of events. So it's very difficult to  
6     compare chemical versus microbial.

7                     DR. GERBA: To give you maybe one  
8     example right away. The dose response data we are  
9     talking about is developed in human beings, the  
10    dose response for chemicals is developed in rats  
11    and mice, so they add extra safety factors in  
12    there for that reason. That's why we're fairly  
13    sure about our risk models because we actually  
14    develop those in human beings. We have ways of  
15    validating our models because of outbreak data.  
16    We know often times how much people ingested in  
17    food and water from that. We can look at our dose  
18    response models and risk models and validate it.  
19    And that's why we feel very confident in microbial  
20    risk models because we have the ability to  
21    validate it, and we don't really, usually have  
22    that ability in the chemical risk assessment  
23    models. So we feel much more confident because we  
24    have the ability to validate from outbreak data or

1 exposure data.

2 MS. ALEXANDER: Just to follow-up on  
3 the comparison. Dr. Gerba, did you write a  
4 chapter entitled "Risk Assessment" for a book  
5 entitled "Environmental Microbiology" that was  
6 published by Academic Press?

7 DR. GERBA: Yes, I did.

8 MR. ANDES: Is this a follow-up  
9 question? We haven't seen this?

10 MS. ALEXANDER: Yes, this is a  
11 comparison to microorganisms as opposed to DBPs,  
12 and I'd like to discuss this. I'm presenting as  
13 an exhibit the title page from the book, and on  
14 the back side is a page from the chapter that I  
15 just referenced.

16 CHAIRMAN TIPSORD: If there's no  
17 objection, I will mark this as Exhibit 78. Seeing  
18 none, it's mark as Exhibit 78.

19 (Document marked as  
20 Exhibit No. 78 for  
21 identification.)

22 MS. ALEXANDER: And I call your  
23 attention in the text on the back side under the  
24 heading 24.4, "Microbial Risk Assessment" in the

1 second paragraph, the language beginning, this is  
2 starting with the second sentence, "The trouble is  
3 that the risks posed to the community by these low  
4 levels of pathogens in the water supply over time  
5 are not like those posed by low levels of chemical  
6 toxins or carcinogens. For example, it takes just  
7 one amoeba in the wrong place at the wrong time to  
8 effect one individual, whereas the same individual  
9 would have to consume some quantity of a toxic  
10 chemical to be comparably harmed." My question  
11 is, do you still believe that statement to be  
12 accurate?

13 DR. GERBA: Oh, yes. Can I follow  
14 up? Of course it refers to drinking water.

15 MR. ANDES: Are you trying to  
16 compare the risk from these two types of exposure?

17 DR. GERBA: No, it's just a  
18 statement of fact.

19 MR. ANDES: Are they two different  
20 kinds of risks?

21 DR. GERBA: Yes.

22 MS. ALEXANDER: And in fact the  
23 point is they are two different kinds of risks,  
24 correct?

1 DR. GERBA: Yes.

2 MS. ALEXANDER: With that, I have no  
3 further questions for this panel.

4 THE COURT: Thank you. It's about  
5 ten after 12:00. Why don't we go ahead and take a  
6 lunch break then and come back with the IEPA.

7 (Whereupon the hearing was adjourned  
8 and a lunch recess was taken.)

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1 STATE OF ILLINOIS )  
 ) SS.  
2 COUNTY OF COOK )

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4 I, DENISE A. ANDRAS, being a Certified  
5 Shorthand Reporter doing business in the City of  
6 Des Plaines, Illinois, County of Cook, certify  
7 that I reported in shorthand the proceedings had  
8 at the foregoing hearing of the above-entitled  
9 cause. And I certify that the foregoing is a true  
10 and correct transcript of all my shorthand notes  
11 so taken as aforesaid and contains all the  
12 proceedings had at the said meeting of the  
13 above-entitled cause.

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DENISE A. ANDRAS, CSR  
CSR NO. 084-003592

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