

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

No. R08-9

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 STATE OF ILLINOIS
 Pollution Control Board

TRANSCRIPT OF PROCEEDINGS held in the
 above-entitled cause before Hearing Officer Marie
 Tipsord, taken before Tamara Manganiello, RPR, at
 160 North LaSalle Street, Room N-502, Chicago,
 Illinois, on the 28th day of July, A.D., 2009,
 commencing at 9:06 a.m.

1 APPEARANCES

2 ILLINOIS POLLUTION CONTROL BOARD
Ms. Marie Tipsord, Hearing Officer
3 Mr. G. Tanner Girard, Acting Chairman
Ms. Andrea S. Moore, Board Member
4 Mr. Thomas E. Johnson, Board Member
Mr. Shundar Lin, Board Member
5 Mr. Gary L. Blankenship, Board Member
Ms. Alisa Liu, Environmental Scientist

6
7 ILLINOIS ENVIRONMENTAL PROTECTION AGENCY
Ms. Stefanie Diers
8 Ms. Deborah Williams

9 NATURAL RESOURCES DEFENSE COUNCIL
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BY: MS. ANN ALEXANDER

12 BARNES & THORNBURG, L.L.P.
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15 BY: MR. FREDERIC P. ANDES,

16 Appeared on behalf of the Metropolitan
17 Water Reclamation District of Greater
Chicago.

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1 HEARING OFFICER TIPSORD: Good
2 morning, everyone. My name is Marie Tipsord.
3 I've been appointed by the Board to serve as
4 hearing officer in this proceeding entitled
5 Water Quality Standards and Effluent
6 Limitations for the Chicago Area Waterway
7 System and Lower Des Plaines River, Proposed
8 Amendments to 35 Ill. Admin. Code 301, 302,
9 303 and 304. This is docket number R08-9.

10 With me today to my immediate left
11 is acting chairman, G. Tanner Girard,
12 presiding Board member, to his immediate left
13 is Board member Gary Blankenship and then to
14 Mr. Blankenship's left is Board member
15 Shundar Lin. To my far right is Board member
16 Thomas Johnson and to my immediate right is
17 Alisa Liu from our technical staff.

18 A couple of things I want to note.
19 First of all, for those keeping track, I
20 believe this is day 29 on my count. Also, I
21 received an e-mail this week from -- or last
22 week from Tom Diamond, who's been in contact
23 with the Exxon Mobil attorneys about the
24 schedule of the hearing on August 13th. Both

1 Robin Garibay and Carl Adams -- I believe I'm
2 pronouncing those correctly -- are flying in
3 from out of state and so they would like to
4 start with them to ensure that they hopefully
5 are done. As you know, that's consistent
6 with my attempts to try and keep people from
7 coming back, except for Dr. Yates who has at
8 our pleasure come back today. So we will
9 begin with the Stephan (phonetic) witnesses
10 on August 13th and then go to Exxon Mobil. I
11 think we should still be able to do that in
12 that time frame we have.

13 Also, just as a minor note, I had
14 in the past been at the close of each hearing
15 putting in cumulative exhibit lists and since
16 the exhibit list is now very lengthy after
17 each hearing from now on I will only be
18 adding to that exhibit list. I won't be
19 printing out the whole cumulative exhibit
20 list to be included in the docket just to try
21 and save some paper.

22 With that, I think today we are
23 going to begin with Dr. Yates, Marylynn
24 Yates, who was with us at the end of -- in

1 May. And if we have time this afternoon,
2 which I think I've heard that we are going
3 to, we will hopefully begin with Corn.
4 Products.

5 We will begin with the questions
6 from the Metropolitan Water Reclamation
7 District of Greater Chicago. Anyone may ask
8 a follow-up question. You need not wait
9 until your turn to ask a question. I do ask
10 that you raise your hand, wait for me to
11 acknowledge you, after I have acknowledged
12 you, please state your name, whom you
13 represent before you begin your questions.

14 Please speak one at a time. If
15 you speak over each other, the court reporter
16 will not be able to get your questions on the
17 record. Please note that any questions asked
18 by a Board member or staff are intended to
19 help build a complete record for the Board's
20 decision and not express any preconceived
21 notion or bias.

22 We will go until around 5:00
23 today. We will have a lunch break. And with
24 that, Dr. Girard.

1 DR. GIRARD: Good morning. On behalf
2 of the Board, I welcome everyone to hearing
3 day 29 in this rulemaking. Thank you for all
4 the extraordinary time and effort that has
5 been invested in helping the Board get a
6 complete record for our decision. We look
7 forward to your testimony and questions
8 today. Thank you.

9 HEARING OFFICER TIPSORD: And with
10 that, I believe we're ready to begin.

11 Dr. Yates, please remember you are
12 under oath having been sworn in in May. We
13 won't do that again.

14 THE WITNESS: Thank you.

15 MS. ALEXANDER: And we have one small
16 housekeeping matter. Last time during
17 Dr. Yates' testimony she referenced a CDC
18 Morbidity and Mortality Report that
19 documented a schistosoma outbreak. We
20 referenced it having pulled it up on the
21 internet and promised the Board that we would
22 provide it as an exhibit. I have it today.
23 It can be marked at the convenience of Board
24 and counsel.

1 HEARING OFFICER TIPSORD: Let's go
2 ahead and get that marked now and take care
3 of it.

4 MS. ALEXANDER: Okay. So this will be
5 Exhibit 286?

6 HEARING OFFICER TIPSORD: Actually,
7 it's Exhibit 301.

8 MS. ALEXANDER: I'm sorry. Exhibit
9 289?

10 HEARING OFFICER TIPSORD: 301.

11 MS. ALEXANDER: I guess I missed some
12 days.

13 HEARING OFFICER TIPSORD: It may be
14 that you didn't print off the most recent
15 ones since I started doing the different
16 placement.

17 MS. ALEXANDER: How many copies do you
18 need?

19 HEARING OFFICER TIPSORD: Can we get
20 at least three?

21 (Document tendered.)

22 HEARING OFFICER TIPSORD: I have
23 marked the CDC Morbidity and Mortality Report
24 marked May 26th, 2000, Surveillance For

1 Waterborne Disease Outbreaks, United States,
2 1997 to 1998. If there's no objection, we'll
3 mark it as Exhibit 301. Seeing none, it's
4 Exhibit 301.

5 MR. ANDES: First, one scheduling
6 question. After we're complete with the
7 questioning of Dr. Yates, the plan was to
8 move on to Corn Products witnesses. And as I
9 mentioned to Ms. Alexander, we have trimmed
10 down our questions somewhat for Dr. Yates so
11 we could be completed with that fairly soon.
12 I don't know if the Corn Products people are
13 here and when they would be ready to go
14 but...

15 HEARING OFFICER TIPSORD: Actually, I
16 believe Mr. Reed indicated to me that they'll
17 be available around the noon hour; is that
18 correct?

19 MR. REED: That's right. 12:30.

20 MR. ANDES: Okay. Well, we could be
21 done before then.

22 HEARING OFFICER TIPSORD: We'll take
23 an early lunch.

24 MR. ANDES: Okay.

1 WHEREUPON:

2 MARYLYNN V. YATES, Ph.D.

3 called as a witness herein, having been previously
4 duly sworn, was examined and testified as follows:

5 EXAMINATION

6 BY MR. ANDES:

7 Q. Good morning, Dr. Yates.

8 A. Good morning.

9 Q. We have to cover a few issues that
10 were raised in your previous testimony. One of them
11 was a statement that you made concerning the
12 upstream and downstream sampling. The statement
13 that you made was that in the risk assessments we
14 had assumed there was equal use of upstream and
15 downstream locations when it was your understanding
16 more miles of the CAWS were below or downstream of
17 the treatment plants.

18 I want to start on that issue by
19 looking at a map, which I know I have here
20 somewhere. This is a figure that's already an
21 exhibit in one of the exhibits, I believe the dry
22 and wet weather risk assessment.

23 HEARING OFFICER TIPSORD: So this is
24 already an exhibit, Mr. Andes?

1 MR. ANDES: Yes.

2 HEARING OFFICER TIPSORD: Then we
3 won't enter it again. And I have additional
4 copies if anyone needs one. And before we go
5 any further, I just want to note for the
6 record that Dr. Yates' initial testimony was
7 Exhibit 249 for purposes of the record to try
8 and keep things straight.

9 BY MR. ANDES:

10 Q. Dr. Yates, this is a figure that shows
11 the sample locations that were used for the dry
12 weather risk assessment. Let's contrast this for a
13 moment and just confirm your understanding. This
14 shows the samples where they were taken for the dry
15 weather assessment upstream, downstream and at the
16 outfalls. The wet weather assessment actually
17 looked at locations all throughout the system. So
18 your discussion was specifically about the dry
19 weather sampling locations; am I right?

20 A. Correct.

21 Q. Now is it your understanding that
22 these sampling locations were fairly close to each
23 of the treatment plants, upstream and downstream?

24 A. I have to admit I don't recall exactly

1 how far from the treatment plants each of the
2 sampling locations was.

3 Q. Now those sampling locations in the
4 vicinity of the plants were then -- and confirming
5 your understanding, these samples in the vicinity of
6 the plants were then used to analogize and make
7 conclusions for the rest of the system, correct?

8 A. I'm really not sure what it is that
9 you're saying.

10 Q. Well, in determining the risks
11 throughout the system including, say, areas
12 significantly downstream, these were the data points
13 that were used?

14 A. That's my understanding, yes.

15 Q. Okay. Now since the bacteria levels
16 would tend to attenuate particularly as you go
17 significantly downstream on the Ship Canal or on the
18 Cal-Sag, for example, your understanding is that
19 that attenuation was not -- or decay was not
20 factored in at all; am I right?

21 A. That's my understanding, yes.

22 Q. So then that would tend to
23 overestimate what the risks are downstream?

24 A. If the concentrations of the pathogens

1 decreased significantly, which we do not know
2 because they were not measured, but if the
3 concentrations of the pathogens decreased
4 significantly as you move downstream, then the risk
5 that would be calculated based on that specific
6 number would tend to be overestimated, yes.

7 Q. And you would expect ordinarily that
8 those levels would decrease as you go downstream; am
9 I right?

10 A. It would depend on a number of
11 factors. The length of time that these -- that some
12 of these pathogens can remain infectious can be
13 weeks depending on the environmental conditions. So
14 since those concentrations downstream were not
15 measured, I really can't say as to whether they were
16 lower than the concentrations at the sites that you
17 measured.

18 Q. But they certainly wouldn't increase,
19 right? The main sources that we're talking about
20 here during dry weather are the treatment plants.
21 You wouldn't expect -- there are not other sources
22 coming in significantly, so you wouldn't expect the
23 numbers to go up, they would only go down?

24 A. In general, that's correct, yes.

1 Q. Now in terms of the issue of sort of
2 what's upstream and downstream, when we're taking
3 samples at Stickney, for example, the upstream
4 sample at Stickney, that's actually downstream of
5 Northside, correct?

6 A. Not -- well, assuming, looking at
7 this. I have not looked at, you know, the flows,
8 but looking at this picture it certainly appears
9 that that's the case, yes.

10 Q. Okay. So by including that as an
11 upstream sample, it's not an upstream sample absent
12 any contributions, it's an upstream sample that
13 would take into account the contributions coming
14 down there from Northside?

15 A. It appears that that would be the
16 case, yes.

17 Q. Also, when you questioned whether the
18 risk assessment assumed there was equal recreation
19 both up and downstream, did you look at whether
20 there are, for example, some high recreation areas
21 in the Lake Calumet system that are upstream of the
22 Calumet plant or Upper North Shore Channel
23 activities that are upstream of the Northside plant?
24 Did you look at the extent to which, in fact, there

1 might be some high recreation areas that were
2 upstream?

3 A. I did not look at the amount of
4 recreation that might be upstream or downstream.
5 One of the points I was trying to make is that
6 there's no justification in the report for the fact
7 that you used both downstream and upstream pathogen
8 concentrations in doing the risk assessment. The
9 report does not justify in any way why that was
10 done.

11 Q. Well, since the assessment looked
12 separately -- and we can provide the table -- at
13 what the risks are upstream, downstream and at the
14 outfall, doesn't that show in some respects
15 particularly a worst case because it's actually
16 looking at what the risk assessment is for actually
17 at the outfall?

18 A. I don't believe that the -- any
19 overestimation of risk that might have been provided
20 by looking upstream versus downstream outweighs the
21 other numerous ways that we talked about when I was
22 here last that the risks tended to be
23 underestimated, for example, by looking at
24 extraordinarily small sample volumes and

1 extrapolating to the entire sample. I believe those
2 risks -- that the lack of consideration of those
3 risks far underestimates the risk and that these
4 risks, while they may tend in some places to
5 overestimate risk, I think that when you combine the
6 two you're still far underestimating risk.

7 Q. But as to this -- let's take one issue
8 at a time. As to this issue in particular where we
9 look separately at upstream, downstream and actually
10 risks at the outfall -- and you would agree that
11 people are probably not going to be canoeing for
12 long periods of time directly at the outfalls of
13 these plants, right?

14 A. I really couldn't speculate on that,
15 sir.

16 Q. Well, let's look at the -- and this is
17 not in this form. This table is not in this form in
18 the record.

19 HEARING OFFICER TIPSORD: Let's enter
20 it as an exhibit then.

21 MR. ANDES: It's entitled Illness
22 Rates For All Pathogens. It's a summary of
23 information in Exhibit 71.

24 HEARING OFFICER TIPSORD: If there is

1 no objection, we'll mark Illness Rates For
2 All Pathogens as Exhibit 302. Seeing none,
3 it's Exhibit 302. And there are more copies
4 up here if anyone needs one.

5 BY THE WITNESS:

6 A. Could you please tell us which tables
7 that are in the Exhibit 71 these data are from?

8 BY MR. ANDES:

9 Q. It is taken from table 4-7 of the dry
10 weather report and I believe -- so not the main
11 report in Exhibit 71, but I do believe that we've
12 introduced the dry weather report.

13 HEARING OFFICER TIPSORD: Is that
14 Exhibit 72, Fred, dry weather risk assessment
15 of human health impacts?

16 MR. ANDES: That would be it, table
17 4-7 of Exhibit 72. And I'll note for the
18 record that in reviewing that table we found
19 a typo which actually increases the number.
20 In the Stickney downstream sample, the table
21 4-7 reflected as .022 should have been .220,
22 so the table we've just provided reflects the
23 higher, corrected number.

24 MS. WILLIAMS: Are we going to have

1 testimony, Fred, on the -- I mean, is there
2 any way to authenticate that that -- is there
3 elsewhere in the document that this is just a
4 typo?

5 MR. ANDES: Yes. We could certainly
6 provide testimony to that. But if you go
7 back into the dry weather report, .22 is
8 reflected. That's the real number. We
9 just -- it was reflected improperly in that
10 table.

11 MS. WILLIAMS: Thank you.

12 BY MR. ANDES:

13 Q. So, again, these are dry weather
14 risks, Dr. Yates, and you'll note that the analysis
15 looks separately at upstream risk, downstream risk,
16 combined upstream and downstream and actually at the
17 outfall and the highest number here is about one
18 illness rate per thousand and that compares, am I
19 right, to the EPA primary contact criteria of eight
20 per thousand?

21 A. I'm not sure what the question is.

22 Q. So the risk at the outfall -- and
23 correct me if I'm wrong -- here is assessed as about
24 one illness per thousand and that compares to the

1 EPA recommended primary contact criterion of eight
2 per thousand?

3 A. If you're asking if the primary
4 contact number that EPA uses is eight per thousand,
5 yes, that's correct.

6 Q. Okay. And this is one per thousand.
7 And that's at the outfall, correct, so you would
8 agree that that would probably be the maximum risk
9 that we'd be dealing with in a dry weather
10 situation?

11 A. I really couldn't speculate on that.

12 Q. Okay. Let me move on to another
13 issue, and you referred to it previously concerning
14 the use of what you characterize as small samples.
15 And I believe that our initial discussion was
16 concerning the use of the equivalent of .2 liters to
17 test out of a total of 300 liters and let's talk
18 about that issue for a minute. To clarify, my
19 understanding is you were specifically talking about
20 norovirus?

21 A. I was specifically talking about
22 norovirus because norovirus is the only virus for
23 which you actually listed the equivalent volume of
24 sample that was tested. I don't have the

1 information on the equivalent volume of sample that
2 was tested for the other organisms -- the other
3 viruses specifically.

4 Q. Okay. Well, let me try to lay out my
5 understanding of how the process worked. And
6 correct me if I'm wrong or if there's anything here
7 that's inconsistent with your understanding.

8 MR. ANDES: And I would also say to
9 the Board that if we have any questions or
10 need any testimony, Dr. Gerba is here, whose
11 lab conducted the test. He can certainly
12 provide some clarification.

13 BY MR. ANDES:

14 Q. So let's start with the 300 liters of
15 water. My understanding, explaining it as best a
16 layman can -- and since we have you and Dr. Gerba
17 here you can correct me if I'm wrong --

18 A. Don't worry, we will.

19 Q. -- is that you start out with 300
20 liters of water with bacteria in it. I'm sorry, not
21 bacteria. I keep getting corrected on that.
22 Viruses. And then that is filtered -- that is run
23 through a filter so all of the viruses in that
24 300 liters of water are left on filter. That

1 there's then a process to separate the viruses off
2 the filter and they're put into a one-and-a-half
3 liter container.

4 Then there's another process by
5 which that's filtered again and all of the viruses
6 then are put in a 30 milliliter container of water.
7 So all of the viruses that started in the 300 liters
8 have now been put in a much smaller volume of water
9 to make the analysis easier to do. Is that fair to
10 characterize it that way?

11 A. First of all, it sounds like you've
12 been to my class, so, very good, you would have
13 passed that question on the exam.

14 The only point I would raise is
15 that I was unable to find anywhere in any of the
16 documentation what volume of final concentrate --
17 that 30 mls that you mentioned, I was unable to find
18 that information anywhere in any of the
19 documentation. So I could not tell what volume that
20 300 liters or in some cases 125 or whatever, I could
21 not find out what final volume that original sample
22 was concentrated to. You've said 30 milliliters. I
23 couldn't find that anywhere in the documentation,
24 which is one of the reasons I've been saying what

1 I've been saying all along is that I cannot go back
2 and calculate the equivalent volume.

3 Q. Okay.

4 A. It's nowhere in the documentation.

5 Q. We can certainly have Dr. Gerba
6 clarify that at this point. He's already been sworn
7 in.

8 A. I'll accept that that's the case, sir.
9 I'm just saying it's not in the documentation. I'll
10 accept that it's 30 mls. That's fine. That would
11 be a very typical volume. I'm not going to argue
12 with it.

13 Q. Okay. And I believe that was in the
14 appendix to the report.

15 A. I have read all of the appendices,
16 sir, and I cannot find that information.

17 Q. Okay. Well, we can certainly have
18 testimony as to it and then we can provide
19 documentation at a later point if that can't be
20 located. If that's not available, we can provide
21 laboratory information.

22 A. Again, it doesn't matter. I accept
23 that it was 30 mls. As I said, that's a very
24 typical volume and it's going to be within a couple

1 of mls of that. That's fine. My point is that I
2 could not find it documented anywhere.

3 Q. And so we may have this issue with
4 regard to the next step as well, so let's take it to
5 the next step. And if we need to fill in the gaps
6 either with a document or with Dr. Gerba's
7 testimony, we can.

8 But my understanding, again,
9 speaking as a layman, is that that 30 was then split
10 into two samples of 15 milliliters, one of which was
11 sent off site to one lab and another was sent to --
12 was used by Dr. Gerba's lab specifically, then most
13 of that 15 milliliters was used to test for
14 adenovirus?

15 A. Actually, this information was in the
16 appendices and it states that 10 milliliters were
17 sent to Dr. Gerba's laboratory and that 8.3 of them
18 were analyzed for adenoviruses.

19 Q. Okay. So 8.3 of the ten were --

20 A. Right.

21 Q. -- tested for adenovirus?

22 A. Uh-huh.

23 Q. Then a small amount -- a much smaller
24 amount, the equivalent of .2 liters of the larger

1 sampling was tested for norovirus?

2 A. Approximately, right. That
3 information is provided in the tables in Exhibit 71,
4 the exact volume, but it's around 200 milliliters,
5 yes.

6 Q. Okay. So of this concentrated sample,
7 we end up with most of what was at Dr. Gerba's lab
8 to be tested for adenovirus, a small amount being
9 used to test for norovirus. Now that was the
10 equivalent of .2 liters or 200 milliliters, right?

11 A. That's according to the tables in the
12 document, yes.

13 Q. Now the normal assumption you've used
14 in terms of intake -- ingestion of water in
15 recreation is 30 milliliters, right?

16 A. In studies that I have done, yes,
17 we've assumed 30 milliliters of water being ingested
18 during recreation.

19 Q. So this 200 milliliter equivalent
20 actually represents a much larger amount of water
21 relative to virus concentrations -- virus amounts
22 than you ordinarily assume in a study?

23 A. Two hundred milliliters, of course, is
24 more than 30. The point is that you only tested

1 200 milliliters of water and if you didn't find
2 anything in that particular 200 milliliters volume,
3 you assumed the entire rest of the sample contained
4 no noroviruses.

5 So while the 200 milliliters that
6 you looked at that some people might have swallowed
7 didn't contain any noroviruses, the other 299.8
8 liters of water may have contained millions of
9 noroviruses, therefore, people exposed to that 99.9
10 plus percent of the sample that you didn't analyze,
11 somebody could have swallowed 30 mls of that and it
12 could have contained numerous noroviruses sufficient
13 to cause them to become infected and potentially
14 ill.

15 Q. Now --

16 A. So I don't believe there's any
17 relevance to the volume that a person might ingest
18 and the volume that you actually analyzed. They're
19 two totally separate issue.

20 Q. Now when you do a norovirus test --
21 which is a DNA-based test; am I right?

22 A. RNA.

23 Q. RNA. Thank you. That's usually done
24 on a small sample; am I right?

1 A. That's correct, yes.

2 Q. So the sample size used here was
3 actually fairly typical of what you would ordinarily
4 do in -- rather than testing on large samples? In
5 fact, it would be a lot of effort to have to sort of
6 keep testing over and over on small samples?

7 A. That's correct.

8 Q. And the number of samples taken will
9 also affect -- you're talking about a risk that
10 there's a false negative. So to take 125 samples as
11 was done here would actually be something to reduce
12 the risk of not getting a norovirus in the sampling,
13 correct?

14 A. Not really, no. I think you're
15 combining two totally different issues here.

16 Q. The more samples you take, don't you
17 reduce the risk of getting one where, you know,
18 there's a lot of noroviruses floating around but you
19 just didn't happen to get it in your sample? The
20 more samples you take, the less risk there is of
21 that?

22 A. I guess. But still I don't agree that
23 it's going to overcome the fact that you looked at
24 such a tiny, tiny, tiny fraction of the sample. You

1 are trying to make a determination as to whether or
2 not there's a health risk associated with recreating
3 in this particular water body. And to analyze such
4 a tiny, tiny, tiny amount of a sample, less than a
5 tenth of a percent of a sample, and then extrapolate
6 that to the rest of the sample and say, oh, okay,
7 we're good, this water is not -- it doesn't pose a
8 health risk to me is just not the appropriate way to
9 do it.

10 There should have been much more
11 effort taken to analyze more of the sample if you're
12 going to base such a huge decision as to whether or
13 not to disinfect the water and thereby reduce the
14 concentrations of pathogens and protect public
15 health.

16 So I just think that when the
17 stakes -- if you're doing a research study, it might
18 be different. But the stakes here, you're setting
19 policy decisions that are going to have a huge
20 impact on the health of the people in this community
21 and it just to me is irresponsible to look at such a
22 small fraction of a sampling and extrapolate to the
23 rest and say we're fine. It's just too important.

24 Q. You don't have -- so, first, you don't

1 have that issue as to what we did, say, for
2 adenovirus or bacteria, you're just speaking
3 specifically here about the norovirus, correct?

4 A. The norovirus is probably the best
5 example of where you had the potential to
6 underestimate because you looked at such a tiny,
7 tiny fraction.

8 With the adenoviruses, we had this
9 discussion last time, where, okay, with the
10 adenovirus you analyzed about a quarter of the
11 sample, right?

12 Q. Let me interrupt for a moment because
13 we'll get to that issue with the adeno. Let's take
14 one issue at a time. Your issue is that --

15 A. The norovirus is the place where you
16 analyzed the tiniest fraction of the sampling,
17 that's correct.

18 Q. So that would mean for the other
19 viruses we analyzed a pretty large fraction of the
20 sample, right?

21 A. You analyzed more of the sample,
22 that's correct.

23 Q. Well, if it was tiny for one, then it
24 was probably all the rest for the others, right,

1 because it didn't just -- logic would say --

2 A. I don't know. I don't know. You said
3 you ended up with 30 mls of concentrate.
4 Dr. Gerba's lab analyzed less than 10 mls of it for
5 the adenos and the noros. I assume -- I don't know
6 how much was analyzed at HML for enteros and I don't
7 know what happened to the rest of the sample. So I
8 can account for about -- in Dr. Gerba's lab for less
9 than a third of the sample, a third of the sample,
10 essentially.

11 Q. But taking a third of the sample, the
12 chance that you're going to get a non-detect when
13 there's a lot of viruses floating around is not
14 appreciable, correct?

15 A. I wouldn't say not appreciable. I
16 would say that the risk of not -- I shouldn't use
17 the word risk, should I?

18 Q. It can be a false negative, right?

19 A. The chances are much less if you're
20 analyzing a third of the sample than if you're
21 analyzing a tenth of a percent of the sample.

22 Q. Thank you. We're going to use a table
23 and this is in Exhibit 71.

24 HEARING OFFICER TIPSORD: This is

1 table 3-9, summary of dry weather virus
2 detection, and it's already also table 3-10
3 from Exhibit 71, correct?

4 MR. ANDES: Yes. And there are no
5 changes in these tables.

6 HEARING OFFICER TIPSORD: And we have
7 extra copies if anyone needs one.

8 BY MR. ANDES:

9 Q. So, Dr. Yates, what we're talking
10 about here as to norovirus is specifically table 3-9
11 where you can see that the norovirus levels are one
12 detect out of 25, three detects out of 25 and one
13 detect out of 25?

14 A. Uh-huh.

15 Q. Now it would be relevant, wouldn't it,
16 to think about, well, what generally is the risk
17 when we're sampling for norovirus that we're going
18 to get non-detects when there really are significant
19 levels there? Let me refer you to answer that
20 question.

21 Let's look at the wet weather
22 samples because on table 3-10 we see that in wet
23 weather we do have a lot of detects of norovirus.
24 Same method used, same issues you would have in

1 terms of small samples, but we find the norovirus in
2 a fair number of samples, low levels, but it's
3 detected which tends to indicate -- at least it
4 would seem to indicate that you do have a fair
5 chance of picking up noroviruses using this method
6 when there are noroviruses actually there. And if
7 you compare the wet weather samples where you did
8 find noroviruses even in these small samples to the
9 dry weather samples where you really didn't find it,
10 it would be logical to assume that there is a lot
11 less norovirus in dry weather than there is in wet
12 weather, correct, wouldn't that be a reasonable
13 conclusion?

14 A. I don't think so.

15 Q. Really? So --

16 A. I wouldn't look at it that way.

17 Q. So since you're finding norovirus in
18 wet weather, 44 percent, 63 percent, 17 percent, so
19 even with this small sample size you're finding
20 plenty of norovirus, you're capturing it and you
21 can't capture much more than 63 percent, but yet
22 when you look at dry whether you don't find it.
23 You're saying that's because you took two small
24 samples.

1 But when we took those same small
2 samples on wet weather we found it. So one could
3 say, well, okay, so more likely it's there in wet
4 weather and not there in dry weather. There's no
5 difference in the sampling methods or the sample
6 size, it's really a difference in what's in the
7 water?

8 MS. ALEXANDER: I think this was asked
9 and answered. The witness has said that she
10 wouldn't look at it that way. Perhaps you
11 need to allow the witness an opportunity to
12 explain why.

13 BY MR. ANDES:

14 Q. Sure.

15 A. Well, there's a couple of different
16 things here. Again, if you're looking at wet
17 weather one could assume that if you'd actually
18 analyzed a larger portion of the sample, two things
19 would have happened, A, you would have had much
20 higher percentages of the samples being positive
21 and, B, the concentrations you would have measured
22 would have been higher.

23 You have an issue when you're
24 looking at a small sample not just of whether or not

1 the organism is there, but you have a detection
2 limit issue. You can only detect a certain -- down
3 to a certain level. So I think that there are -- I
4 don't agree with your characterization that looking
5 at that tiny, tiny sample volume was justified.

6 Q. Well, but your point was focused on
7 detection and saying the fact that we found -- we
8 detected norovirus very rarely in dry weather and
9 you're saying that's because of the sample size?

10 A. Uh-huh.

11 Q. We're saying, well, but we have the
12 same sample size in wet weather and we found it all
13 over the place so --

14 A. Well, I wouldn't say 17 percent is all
15 over the place.

16 Q. But 63 percent would be?

17 A. Not really.

18 Q. Really?

19 A. Not really.

20 Q. So ten out of 16 samples of it
21 detected, that's pretty widespread, isn't it?

22 A. Well, it depends on how you
23 characterize widespread. I would say that if you
24 really have a good method and if you really do have

1 viruses all over the place, then you would have
2 detected them in closer to 100 percent of the
3 samples. I don't characterize 63 percent as that
4 huge and I certainly don't characterize 17 or
5 44 percent as that high either.

6 Q. There's a significant difference
7 between 63 percent and 12 percent, right?

8 A. I haven't done a statistic, sir. I
9 couldn't say if it's significant.

10 Q. We're going to move on to the next
11 issue. Let's talk about the issue of adeno and
12 enteroviruses. As I understand your issue here, Dr.
13 Yates, the question -- and I'll pull some tables and
14 we can start talking about this. Let's get all of
15 our tables straight here. Let me make sure I have
16 all my copies. This table, as well, is from
17 Exhibit 71, it's table 3-6.

18 A. I don't need it. I have it right
19 here.

20 Q. Let's walk through this again and I'll
21 try to provide a layman's perspective. We have a
22 number of samples here, and this is dry weather,
23 where there was a test for viruses, the total levels
24 found are reflected in the total MPN per 100 liter

1 column. Then there was a PCR confirmation. And if
2 PCR was positive for adenovirus, then the people
3 doing the study assumed that that entire
4 concentration of virus detected was all adenovirus,
5 correct?

6 A. That is my understanding, yes, sir.

7 Q. So, for example, at Calumet outfall
8 72605 where there was a 7.52 count and a positive
9 confirmation, it was assumed then that the entire
10 amount was all adenovirus, 7.52, correct?

11 A. That is my understanding, yes.

12 Q. And, in fact, that's a conservative
13 assumption, am I right, because that's not
14 necessarily all adenovirus and doesn't necessarily
15 all have that level of risk posed by adenovirus?

16 MS. ALEXANDER: Is that a question?

17 BY MR. ANDES:

18 Q. Am I right, that that's a conservative
19 assumption that it's all 100 percent adenovirus?

20 A. I couldn't say whether or not that's a
21 conservative assumption.

22 Q. Well, if -- you're assuming that it
23 all contributes to risk at an adenovirus level,
24 correct?

1 A. That's what you assumed, yes.

2 Q. And that's not necessarily true,
3 right, because it's been --

4 A. Correct.

5 Q. -- PCR confirmed? Okay.

6 And we'll see later how that
7 contributes to the total risk.

8 Now in a situation such as
9 Northside where you had a sample of 13.9 and
10 negative on PCR, that was not felt to contain
11 adenovirus so wasn't counted toward adenovirus and
12 you're contention is -- well, let's stop for a
13 minute.

14 So there are approximately 11
15 samples on this table out of 42 -- I'm sorry. Out
16 of 75 samples there were 42 that had detectable
17 virus. Some of them had less than one, meaning not
18 detect. Thirty-one of them had PCR positive, so
19 we're down to 11 where there was PCR negative, so no
20 adeno, so those were sort of put aside.

21 Your contention is -- correct me
22 if I'm wrong -- that we should have looked at them
23 for enterovirus?

24 A. The point that I made last time was

1 that you went to great lengths to say how wonderful
2 this cell line was because it enabled you to detect
3 both enteroviruses and adenoviruses, therefore, when
4 you got a cell culture positive result you knew that
5 the virus -- first of all, there were viruses in
6 that sample and that those viruses were either
7 entero or adenoviruses.

8 You then did a test looking to
9 determine whether or not the viruses that were
10 present were adenoviruses. If they were not
11 adenoviruses, you counted the sample as being
12 negative when indeed you already had proof that
13 there were viruses present in that sample.

14 And according to the work that has
15 been done by Dr. Gerba, you know that that cell line
16 allows enteroviruses to grow in it. And if it
17 wasn't adenovirus positive, you just ignored the
18 possibility that indeed those were enteroviruses.
19 You never analyzed the sample for enteroviruses.
20 You just counted that sample as being negative when
21 indeed you knew there were viruses in it.

22 Q. Now, first, when you say that we took
23 great pains to use that method because it would
24 detect adeno and entero --

1 down a little bit.

2 BY THE WITNESS:

3 A. Question 31A: Are you aware that the
4 cell line used is not designed to be specific for
5 adenoviruses as the cell line was selected because
6 it will detect both adenoviruses and enteroviruses.

7 BY MR. ANDES:

8 Q. And your concern laid out in the
9 testimony that we were responding to was that you
10 actually thought that cell culture analysis for
11 adeno appears to produce a relatively large number
12 of false positive results. Would that be false
13 positive for adeno and for entero?

14 A. No. False positive for adeno.

15 Q. So you didn't -- so your concern was
16 that it wasn't doing a good job of detecting adeno.
17 And isn't it logical to say this question then was
18 directed to saying, yes, it is designed to address
19 adeno; the issue at hand in your bullet and then in
20 our question was really whether it was going to do a
21 good job of detecting adeno?

22 MS. ALEXANDER: You're asking her what
23 you meant by your question?

24

1 BY MR. ANDES:

2 Q. Wasn't your point specific to adeno?

3 A. I'm making two points. Point number
4 one is that obviously this analysis is not specific
5 for adeno because you detected virus signal, you had
6 infective, growing, living virus -- not living, but
7 infective viruses present in the samples and then
8 you looked to see whether they were adenoviruses and
9 they were not, so it was not specific for adeno.
10 That is one concern.

11 The bigger concern is that you
12 ignored samples that had viruses in them but were
13 not adenoviruses and you did that with the full
14 knowledge that that cell line enabled both
15 adenoviruses and enteroviruses to grow.

16 Q. And it also -- now is that human and
17 non-human enterovirus?

18 A. I really do not know, sir.

19 Q. And does it detect other kinds of
20 viruses, as well?

21 A. I do not know, sir.

22 Q. So it's possible there are other types
23 of viruses that are also detected. Do you have any
24 information indicating that it only detects adeno

1 and entero?

2 A. I'm just going by what you have
3 indicated in here. I have not done studies with
4 this particular cell line myself.

5 Q. And you're aware that the other
6 portion of those samples was sent off to be tested
7 with another method for enteroviruses, correct?

8 A. I am aware that another fraction of
9 the sample was tested for enteroviruses, yes.

10 Q. So it's not that we didn't test for
11 enterovirus, you're saying we should have also
12 looked at these samples, as well, to see if there
13 was entero?

14 A. That is exactly my point --

15 Q. Now --

16 A. -- because you have said that this
17 cell line enables enteroviruses and adenoviruses to
18 grow in the sample. And when you did not find
19 adenoviruses, you just ignored the fact that you had
20 other viruses growing.

21 I am aware that you had another
22 fraction of the sample tested for enteroviruses.
23 There were other samples -- the samples that you had
24 tested for enteroviruses, there were times when

1 those samples were negative for enteroviruses.
2 However, the other portion of the sample that came
3 up -- that was tested in Dr. Gerba's laboratory that
4 was positive for viruses but negative for
5 adenoviruses, you have already said that that cell
6 line allows the enteroviruses to grow.

7 You did not test that sample to
8 determine whether or not those samples indeed
9 contained enteroviruses. They were negative by the
10 test at HML for enterovirus and yet you know that
11 they were positive for something in the samples
12 tested in Dr. Gerba's laboratory and you didn't
13 check to see whether there were enterovirus knowing
14 full well that enteroviruses could grow in that cell
15 line. So you said that sample was negative for
16 enteroviruses. You completely ignored it.

17 Q. Well, let me clarify. The issue
18 wasn't whether it was negative for enteroviruses.
19 The study used a different --

20 MS. WILLIAMS: I think he's trying to
21 testify.

22 MS. ALEXANDER: Same objection.

23 BY MR. ANDES:

24 Q. Let me clarify. The study used

1 another method to test for enteroviruses, correct?

2 A. Are you talking -- I want to make sure
3 I know what study.

4 Q. This risk assessment -- in this risk
5 assessment another method with the other half of the
6 sample was used specifically to test for
7 enteroviruses, correct?

8 A. I don't know if half of the sample was
9 tested for enteroviruses, but a fraction of the
10 sample was sent to another laboratory and tested
11 using a different method for enteroviruses, yes.

12 Q. Is that a method that is accepted by
13 EPA?

14 A. My understanding is that the method
15 that was used by HML was EPA's standard method for
16 enteroviruses. However, as you have said, the test
17 that Dr. Gerba used to detect the adenoviruses also
18 detects enteroviruses. You had a positive virus
19 sample, it was not adenoviruses and therefore you
20 ignored the fact that it could be enteroviruses.

21 The fact that some other portion
22 of the sample that was analyzed by HML was negative
23 for enteroviruses does not mean that these were not
24 enteroviruses.

1 Again, I pointed out last time
2 that's one of the issues that you face when you take
3 a sample and split it into different portions. You
4 are assuming that the viruses are uniformly
5 distributed throughout the sample and that may not
6 indeed be the case and this is a perfect
7 illustration of that.

8 You have taken a whole sample,
9 taken a part of it, analyzed it, found it to be
10 negative, taken another part of that sample and
11 analyzed it using a different method but a method
12 which is published in the peer-reviewed literature
13 and shown to be able to detect that enterovirus and
14 you did not go the extra step necessary to determine
15 indeed whether or not it did contain those
16 enteroviruses knowing full well that the decision
17 that you're making at the end of the day has huge
18 implications for public health.

19 Q. So you're saying that even though we
20 used an EPA approved method for detecting
21 enteroviruses and we found certain results, we
22 should have also done something with this other test
23 that detects some virus, we don't know what they
24 were, and we should have tried to figure out if

1 there were enteroviruses in there too even though we
2 had already used the approved method? And by the
3 way, the method we're talking about that detects
4 both is not an EPA approved method for detecting
5 enteroviruses; am I right?

6 A. It's not an EPA approved method for
7 detecting adenoviruses either.

8 Q. So wouldn't it make sense to use the
9 EPA approved method for detecting enteroviruses in
10 this study? Wouldn't you have --

11 HEARING OFFICER TIPSORD: Mr. Andes,
12 we have beat this horse to death. It's time
13 to move on. We covered this at the last
14 hearing and we're doing it again. It's time
15 to move on.

16 BY MR. ANDES:

17 Q. I believe one of the statements you
18 just made was that this was of enormous consequence.
19 I don't remember the exact words you used. I will
20 refer you to a table. I thought I had a copy of
21 this table, but I don't seem to be able to locate
22 them. But this is table 5-13 in the report in
23 Exhibit 71.

24 A. Yes.

1 Q. Let me read from this and then I'll
2 provide it. Dr. Yates, this table presents a
3 breakdown of the illnesses per thousand exposures
4 due to various pathogens. And the total illnesses,
5 if I can summarize, for the three different areas of
6 the Waterway were 4.15, 5.67 and 0.41 illnesses per
7 thousand.

8 Now when we look at the enteric
9 virus part of it, which is the part we're talking
10 about here, the numbers are .002, .002 and .001 out
11 of 4, 5 and .41. So it's a small percentage of the
12 total illnesses due to enteric virus; am I right?

13 A. If you're asking if .002 is a small
14 fraction 4.15, yes, I would agree that it is.

15 Q. So even if you took those 11 samples
16 out of 75 and found there were detectable levels of
17 enterovirus and even if that, say, doubled the
18 amount of illness attributable to enterovirus, we
19 would be up to .004, .004 and .002. That would
20 still be a fairly small percentage of the total
21 contribution toward illnesses, correct?

22 A. It would. But, again, this just is
23 one example of where you have made an error -- in my
24 opinion, an error in the manner in which you

1 calculated the risks. This is one example and there
2 are numerous others throughout.

3 But the very specific issue that
4 you asked, if you would like me to answer, is .004
5 still a small fraction of 4.15, yes, it is.

6 Q. Thank you. We'll move on from that
7 issue. Let's go toward one of the other issues we
8 talked about a little bit the last time was the
9 amount of water ingested typically. And I
10 believe -- I'm just looking for some of my charts
11 here. I believe that the amount that you've usually
12 used in tables -- I'm sorry, in studies has been
13 30 milliliters ingested, correct?

14 A. That is correct.

15 Q. In a primary contact situation, right?

16 A. Again, we're getting to this issue of
17 how you define primary contact. If you're defining
18 primary contact as swimming, no, this was not
19 swimming.

20 Q. Okay.

21 A. The context in which I used it was not
22 swimming.

23 Q. Okay. And that's not material to the
24 issue at hand.

1 A. Okay.

2 Q. We're just talking recreation
3 generally for now.

4 A. Okay.

5 Q. Let me provide you with some tables
6 again, tables and figures. These are figure 5-3 and
7 table 5-4 from Exhibit 71.

8 A. I have it.

9 Q. Okay. So in this study when we look
10 at table 5-4 and we look at the high exposure
11 scenario, which was canoeing, you'll see that the
12 90th percentile it was assumed that people are
13 ingesting about 14 milliliters an hour?

14 A. Uh-huh.

15 Q. So that would mean that -- correct me
16 if I'm wrong -- it was assumed that 10 percent of
17 the people recreating would be ingesting about 14
18 milliliters an hour in that exposure group, am I
19 right, 14 or more?

20 A. Just a second. I'm thinking of --

21 Q. It's not a trick question. I'm just
22 trying to --

23 A. I know. And it's taking me a minute
24 because you're asking it in the opposite way. So my

1 understanding is, yes, if the 90th percentile is 14
2 mls per hour then there would be ten out of 100
3 ingesting 14 or more per hour, yes.

4 Q. And there are smaller amounts of
5 people, 5 percent, two-and-a-half percent, et
6 cetera, that would actually be assumed to be
7 ingesting more than 14 milliliters an hour, at the
8 95th percentile level and above they'd be ingesting
9 17 or 22, correct?

10 A. Correct.

11 Q. Okay. Now if we go to figure 5-3,
12 which shows the duration distribution for canoeists
13 in the study, this showed the duration of their
14 canoeing experience and the mean number of hours
15 that they're assumed to be on the water body as
16 2.67; am I correct?

17 A. Yes.

18 Q. And, in fact, a fair number of people
19 are assumed to be on the water three, four, even
20 almost five hours in this distribution, right?

21 A. Correct.

22 Q. Okay. So then those people, if we do
23 a very simple math and say, well, you had 10 percent
24 of the people with at least 14 milliliters an hour

1 of exposure -- I'm sorry, 14 milliliters an hour of
2 ingestion of water and about two-and-a-half hours
3 average on the water, that would put them at
4 something like 30 some milliliters of water?

5 A. Sure, roughly.

6 Q. Okay. So that would actually be
7 fairly consistent with the kind of ingestion
8 scenarios you've used in other studies with 30
9 milliliters, actually even higher than 30?

10 A. Okay.

11 MS. ALEXANDER: Is that a question?

12 BY MR. ANDES:

13 Q. I'm just confirming that the ingestion
14 scenarios used here were at least as conservative in
15 that regard as have been used in other studies. I
16 think, in fact, 37 something if we just multiply 14
17 by 2.67.

18 A. I'll believe you. I don't have my
19 calculator here so I'll believe the math. That
20 would be about right.

21 Q. Okay. Let me move on to another
22 issue. And I don't want to go over ground that
23 we've covered before, so if I stray into that I
24 trust I'll hear about it.

1 MS. ALEXANDER: You can bet on it.

2 BY MR. ANDES:

3 Q. Dr. Yates, we talked at the last
4 hearing about -- and I don't think this will be
5 repetitive. There was a study provided by
6 Ms. Alexander at the last hearing, a study by Teunis
7 and Moe, Norwalk Virus: How Infectious is It?

8 A. Uh-huh.

9 Q. And I had a few questions to ask about
10 that. I believe you were using this study to
11 indicate -- correct me if I'm wrong -- that if you
12 were to ingest a single norwalk virus that the risk
13 was 50 percent that you would get ill; am I right?

14 A. The point -- can you refer to which
15 exhibit that was?

16 HEARING OFFICER TIPSORD: I believe
17 it's Exhibit 255, Norwalk Virus: How
18 Infectious is It by Peter Teunis and
19 Christine L. Moe, et al.

20 THE WITNESS: Thank you.

21 BY THE WITNESS:

22 A. So your question?

23 BY MR. ANDES:

24 Q. Well, I wanted to confirm, first,

1 that's what you're using this study in support of
2 that?

3 A. My point in raising this study was to
4 indicate that the conclusion of Drs. Teunis and Moe
5 was that the average probability of infection for a
6 single norovirus particle would be close to .5,
7 which is higher than reported for any other virus
8 that had been studied to that point in time.

9 Q. Now the study on page -- I want to ask
10 a couple of questions about the study itself. One
11 aspect on Page 1469 of the exhibit mentions that the
12 8F2A inoculum in the first column has been stored in
13 a stock suspension for more than 25 years and that
14 this suspension of veal infusion broth with a half a
15 percent bovine serum albumin contains high
16 concentrations of protein acting as a sticky matrix
17 resulting in considerable aggregation of suspended
18 virus. It then points out that, therefore, the low
19 dosage administered in this challenge study
20 represents virus clumps rather than single viruses.

21 Do you have any information as to
22 how representative that is of a way one would
23 encounter viruses ordinarily?

24 A. My knowledge of the way that most

1 viruses might be present in fecal material would be
2 that they would be present as aggregates or clumps
3 of more than a single virus particle at a time.

4 Q. In fecal material?

5 A. Correct.

6 Q. Are most studies done on actually
7 looking at the risk from ingestion of a single
8 particle?

9 A. Different studies are done in
10 different ways. Typically, you would feed people
11 more than a single particle.

12 Q. So there's nothing about this -- and I
13 thought this is, in fact, pointed out that there's
14 some unique aspects of this study because it looks
15 at the viruses in clumps rather than in single
16 particles?

17 A. I'm not sure that that was a question.

18 Q. Well, for example, let me point you to
19 Page 1471. This says in the first column,
20 aggregation of infectious particles changes the dose
21 response relation in several ways including the
22 parent does may be lower than the number of viruses
23 that was actually present. There may be other
24 effects associated with being part of an aggregate.

1 We have no data in these areas.

2 So the single-hit model of
3 infection was used in this study even though the
4 information was actually in virus clumps rather than
5 in single particles and I'm wondering how that
6 shifts the nature of the risk assessment and it
7 sounds like they don't have data on that.

8 A. Again, these two -- the authors of
9 this manuscript are world-renown experts in this
10 area. Dr. Teunis has literally an international
11 reputation specifically in dose response modeling
12 and, therefore, I have every confidence that when he
13 states that to the best of his ability to determine
14 the infectious dose when he says that the
15 probability from a single particle would be .5, I
16 have absolutely every confidence that he is in a
17 position to make that judgment knowing as he did all
18 of the peculiarities of the particular system within
19 which he was working.

20 So I have every confidence that
21 what he has stated -- what he and Dr. Moe have
22 stated in this article are the best information that
23 they could possibly get knowing all of the caveats
24 that are associated with the samples that they had

1 to work with.

2 Q. Well, on Page 1475 it talks about the
3 differences in the infectivity of individual
4 sporozoites causes -- this is getting very
5 technical -- heterogeneity inactivity infectivity
6 and influences the parameter estimates in the shape
7 of the dose-response relation.

8 I guess what I'm asking is isn't
9 it possible that there are differences here between
10 the nature of the assessment because they're using
11 these clumps from this 25-year old sticky particle
12 sample and then using a single-hit model for looking
13 at what the risks are from a single particle?
14 There's some uncertainty involved in that and this
15 seems to note that there could be some changes in
16 the dose-response relation simply because of the
17 difference between the clumps and a more
18 heterogeneous sample.

19 A. I didn't hear a question. I think
20 you're just trying to -- you're restating what he
21 has said and I --

22 Q. You don't disagree with any of that? -

23 A. What I have already said is that
24 Dr. Teunis is a world-renowned expert, he

1 understands the pitfalls, he understands all of the
2 potential assumptions that are being made as he does
3 his mathematical modeling to determine what the
4 infective dose is and he understands the assumptions
5 that are going into it and, therefore, when he
6 states that to the best of his ability to calculate
7 it the median -- the mean infectious dose is .5 --
8 or there's a probability of infection from being
9 exposed to a single particle is .5, I have
10 absolutely no reason to doubt him.

11 The important point that's being
12 made here is that the infectious dose for norovirus
13 is extraordinarily low and the probability of
14 becoming infected from an extremely small dose, as
15 low as one, maybe a clump of ten, who knows, is
16 very, very, very high, 50 percent in this particular
17 case.

18 Q. And to clarify, when we talk about
19 50 percent or some other number, that's livery of a
20 particle for directly to the target. In a risk
21 assessment, that's not looking at what's the
22 probability of exposure, what's the probability that
23 you're going to ingest a particle, how much water
24 are you going to be drinking, any of those issues?

1 A. No. That probability is specifically
2 saying if you ingest some number of particles, what
3 is your probability of becoming infected as a result
4 of being exposed to that number of particles. It's
5 irrelevant what -- you know, it doesn't take into
6 account the volume of water, anything like that,
7 it's how many particles got into your body.

8 Q. Okay. So it's one portion of a larger
9 risk assessment? You're not contending that it's
10 the whole picture?

11 A. Correct. It's one step in the risk
12 assessment process, yes.

13 Q. And have you looked at how that issue
14 was dealt with in the risk assessment done here in
15 Exhibit 71?

16 A. I'm not sure what --

17 Q. Have you looked at how that issue, the
18 risk of infection from a norovirus particle was
19 dealt with in the risk assessment?

20 A. In the risk assessment my
21 understanding is that you used a particular dose
22 response that followed a beta poisson for the
23 norovirus; is that what you're asking?

24 Q. Right.

1 A. Yes.

2 Q. And that was around 26 percent. So a
3 significant number was used, as well, in this report
4 to show the chance of being infected by one
5 norovirus particle?

6 MS. ALEXANDER: Objection.

7 BY THE WITNESS:

8 A. I don't know where you're coming up
9 with 26 percent.

10 MS. ALEXANDER: Objection. I don't
11 think that was a question and I object to the
12 term significant as vague. I think we need
13 to define terms like that.

14 BY MR. ANDES:

15 Q. Okay. So in table 5-5 -- and this is
16 adapted from Haas and Rose studies -- the N50 was
17 6.17 indicating that six particles would give you a
18 50 percent chance of infection, correct?

19 A. That's what it says here.

20 Q. And do you have any reason to believe
21 that's not straight from the literature?

22 A. No.

23 Q. Okay. So your main point is simply
24 the norovirus is very infectious?

1 HEARING OFFICER TIPSORD: Okay. In
2 that case, the IEPA has a few questions for
3 Dr. Yates.

4 EXAMINATION

5 BY MS. WILLIAMS:

6 Q. Dr. Yates, I'm going to ask some of my
7 pre-filed questions. Some of them have already been
8 answered and even a couple of these that I'm asking
9 you've gone into great detail already, but I'm
10 looking for just a broad overview in my answer; does
11 that make sense?

12 A. Yes. But if I get too specific,
13 please remind me and I will try to generalize.

14 Q. Pre-filed question number two states,
15 in your opinion, what were the analytical errors you
16 found with the microbial risk assessment study
17 conducted by MWRDGC? I know we've spent many --
18 you've answered many, many detailed questions, but
19 just in a broad overview sense could you summarize
20 that for us?

21 A. My overview answer -- I hope this is
22 an overview -- would be in the fact that such small
23 sample volumes were analyzed that, especially in the
24 case of norovirus, that there was an underestimate

1 of the exposure from noroviruses. And the other big
2 issue with respect to the analytical methods was the
3 ignoring of the potential enterovirus positive
4 samples.

5 So in all, I believe that the
6 biggest flaw in the analytical portion of the sample
7 analysis portion of the risk assessment was that
8 there would be an underestimate of the magnitude of
9 the exposure to human pathogens in the water and
10 therefore the risks would be biased low.

11 Q. Question three asks, in your opinion,
12 why is MWRDGC's epidemiological study not a
13 sufficient tool to assess the needs for
14 disinfection?

15 A. First, let me say that I believe that
16 the epidemiological study in general is being
17 conducted in a very thorough way and I have
18 absolutely no reason to doubt that the information
19 that comes out of that study will be extremely
20 useful especially as it relates to the secondary
21 recreational activities.

22 I do believe, though, that there
23 are some things that are not going to be determined
24 through that study, one of them is the risk of

1 secondary spread. So in other words the people who
2 are exposed to the water may become infected but not
3 ill, they can pass that infection onto others
4 outside of their family and those risks are not
5 being taken into consideration in this particular
6 study. So, again, you could be biasing the risk
7 low.

8 My main issue with this is that
9 this is only one piece of information that one
10 should consider when determining whether or not we
11 need to disinfect. Even though, like I said, the
12 study is being conducted in a very thorough manner
13 with a couple of exceptions, this is only one piece
14 of information. It's studying a relatively small
15 portion of people over a small -- a short time
16 period and I think there are a lot of other pieces
17 of information that need to go into doing -- into
18 making such a huge decision as to whether or not to
19 disinfect the effluent.

20 And so while this is one piece of
21 information that obviously should be considered, I
22 don't think it should be the only piece.

23 MR. ANDES: If I can follow-up with
24 that? Have you heard anyone say it should be

1 the only piece of information considered?

2 THE WITNESS: I would have not been
3 privy to all of the discussions. However,
4 again, this is one epidemiological study
5 that's being conducted. And as a scientist
6 an NF1 is never sufficient, especially not
7 when we're making a decision as large as the
8 decision that's being considered here, do we
9 disinfect or do we not disinfect, okay?

10 The other issue -- another issue
11 that is not being considered in this
12 particular epidemiological study or I don't
13 know how it's being considered is the issue
14 of the susceptibility to infection and
15 illness of at-risk -- at higher-risk
16 populations. I have no idea how they're
17 going to be -- or whether they're going to
18 have sufficient numbers of very young
19 children, elderly, other immunocompromised or
20 immunoincompetent if you prefer that term,
21 other individuals who are going to be at
22 higher risks.

23 So, again, this is one study and
24 for what they're doing I believe they're

1 doing a good job, but there are certain
2 things that they're just not going to be able
3 to take into consideration and it's just
4 impossible at this point for me to say that
5 this particular study is going to be
6 sufficient to make a decision of this nature.

7 It's one study. There are issues
8 they're not considering and therefore a lot
9 of other issues need to be considered when
10 making a decision about whether or not to
11 disinfect.

12 MR. ANDES: And, Dr. Yates, you talk
13 about things that are not being considered,
14 but as to sensitive populations you said you
15 just have no idea how those are being
16 addressed?

17 THE WITNESS: I don't know whether --
18 I don't have information on whether or not
19 they have sufficient numbers of individuals
20 enrolled in the study that belong -- that are
21 members of those categories, so I do not know
22 whether they will have a sufficient sample
23 size to do statistically meaningful analyses
24 of those populations.

1 MR. ANDES: So that's something we
2 won't know until we actually see the results,
3 correct, until we see results from the study?

4 THE WITNESS: That certainly is
5 correct. Until you know whether or not you
6 have sufficient numbers of people in those
7 categories, you couldn't calculate the
8 statistical power that you would have with
9 that.

10 MR. ANDES: And, in general, in terms
11 of the overall study, I think you're probably
12 aware, but we recently filed papers
13 indicating that over 9,000 people who already
14 have been enrolled in the study is a fairly
15 significant amount of people.

16 THE WITNESS: The total number of
17 individuals that have been enrolled -- I did
18 read the latest information from
19 Dr. Dorevitch and the total number of
20 individuals who have -- who are enrolled in
21 the study appears that it's going to be --
22 he's going to exceed -- probably exceed his
23 original goals. So as I said, in general, I
24 believe that this study is -- I have no real

1 issues with it.

2 MR. ANDES: And this is the first
3 study done of this sort, epidemiological
4 study on secondary contact in this country;
5 am I correct?

6 THE WITNESS: To my knowledge, it is.

7 MR. ANDES: And the EPA effort
8 currently to develop primary contact
9 recreational criteria, it's my
10 understanding -- correct me if I'm wrong --
11 that EPA is using or intending to use both
12 quantitative risk assessments and
13 epidemiological studies in a process where
14 they will reach ultimate conclusions about
15 safe bacteria levels; am I right?

16 THE WITNESS: I have not spoken with
17 anyone at the -- and we're talking about the
18 US EPA here?

19 MR. ANDES: Yes.

20 THE WITNESS: My understanding is that
21 the United States Environmental Protection
22 Agency is using a variety of sorts of
23 information in determining what types of
24 standards they are going to set for

1 recreational water quality.

2 But, again, the types of -- the
3 situation with the EPA I believe is a very
4 different situation from what -- the US EPA.
5 It's a very different situation from what
6 we're dealing with here in that the
7 epidemiological studies that are being
8 conducted by the United States Environmental
9 Protection Agency are intended specifically
10 to look at associations between health
11 effects and levels of different
12 microorganisms and try to determine
13 relationships between those and then come up
14 with a standard that would be used to
15 determine whether or not we're going to be
16 closing a beach on a particular day.

17 That's a very different kind of a
18 decision than what we're talking about here
19 where we are looking at using a single
20 epidemiological study and a risk assessment
21 that has numerous flaws, as we've discussed,
22 and using that information to decide do we
23 disinfect this effluent or do we not
24 disinfect this effluent when we have full

1 knowledge that this effluent contains
2 disease-causing microorganisms and that --
3 and knowing that if we disinfect this
4 effluent we will decrease the concentrations
5 of those microorganisms thereby decreasing
6 the risks. We're just talking apples and
7 oranges here.

8 MR. ANDES: Let me clarify, Dr. Yates.
9 Aren't the EPA bacteria criteria used not
10 only for beach closures but, in fact, to
11 determine control requirements for sewage
12 treatment plants, combined sewer overflows,
13 the same issues concerning disinfection that
14 we're talking about here?

15 THE WITNESS: The EPA standards -- my
16 understanding of the criteria that EPA is
17 developing, they will be used for beach
18 closures, they will also be used for such
19 things as total maximum daily load, TMDL,
20 determinations and some other uses.

21 MS. WILLIAMS: I would like to
22 follow-up on your follow-up, please.

23 MR. ANDES: Feel free.

24 BY MS. WILLIAMS:

1 Q. Do you have any reason to believe that
2 US EPA is looking at establishing technology-based
3 effluent requirements for sewage treatment plants as
4 part of this analysis?

5 A. I do not have that understanding, no.
6 The other thing I would say is that I do not believe
7 based on every conversation I've had and every
8 meeting and every expert workshop that I've been
9 associated with related to this particular issue I
10 have no reason to believe that EPA is going to
11 establish water quality criteria that are less
12 stringent than the ones that are currently in place.

13 MS. WILLIAMS: Are you done with your
14 following up because that's one of my
15 pre-filed questions?

16 MR. ANDES: No. First, the numbers
17 we're talking about for EPA, you don't know
18 yet so nobody at EPA knows yet what the
19 numbers are going to be, correct, because the
20 studies have not been done that are going to
21 help establish those criteria?

22 THE WITNESS: When you say the
23 numbers, you mean the numerical values that
24 the criteria are going to be?

1 MR. ANDES: Yes.

2 THE WITNESS: I do not know what the
3 numerical values of the criteria are going to
4 be, no, because the studies are still
5 ongoing.

6 MR. ANDES: And, in fact, those
7 studies include, as EPA has recently
8 announced, specifically that one of the
9 important things is doing -- under a consent
10 decree is to do epidemiological studies that
11 will be used in establishing what those
12 numeric criteria are going to be, correct?

13 THE WITNESS: Under the consent decree
14 my understanding is that the EPA will be
15 conducting additional epidemiological studies
16 in order to help form the basis for the
17 establishment of the new criteria, yes.

18 MR. ANDES: And those studies are just
19 going to be starting soon?

20 THE WITNESS: They have already
21 conducted some studies. They are -- I don't
22 know exactly when the new studies are going
23 to start, but they are going to be doing
24 additional studies.

1 MR. ANDES: And those are all primary
2 contact studies, correct?

3 THE WITNESS: My understanding, yes,
4 is that those are primary contact studies.

5 MR. ANDES: Okay.

6 BY MS. WILLIAMS:

7 Q. Dr. Yates, question six asked why you
8 believe that US EPA's revised bacteria criteria will
9 be more stringent than the current criteria rather
10 than either less stringent or simply more targeted?

11 A. It's well documented in a number of
12 documents in the literature, some of which have been
13 talked about during these very hearings, that the
14 organisms that are typically used as indicators, the
15 coliform bacteria, the fecal coliform bacteria,
16 E. coli, enterococci, et cetera, that those
17 organisms -- the use of those organisms tend to
18 underpredict risk and EPA is very concerned that
19 they establish criteria that are going to be
20 adequately protective of public health at whatever
21 risk level they deem to be appropriate.

22 Knowing that the current
23 indicators that are used will underpredict risk,
24 especially from organisms such as viruses and

1 protozoan parasites like cryptosporidium and
2 giardia, EPA is making special efforts to look for
3 either better indicators or to make sure that
4 whatever levels they establish of the indicators
5 will be better predictors of risk.

6 And if you look at the -- if you
7 look at waterborne disease outbreaks, for example,
8 what you'll see is that the number of outbreaks --
9 the trends in outbreaks between recreational
10 outbreaks and drinking water outbreaks has reversed
11 itself in recent years. In other words, in the past
12 more of the outbreaks were associated with drinking
13 water. Now we've moved to the point where more of
14 our outbreaks are associated with recreational
15 water.

16 And I believe that EPA would point
17 to the revisions and increasing their attention to
18 the microbiological standards for our drinking water
19 as resulting in higher quality drinking water in the
20 United States and thus better public health
21 protection of our drinking water that people
22 consume.

23 Now they're turning their
24 attention to the recreational waters. And knowing

1 that increasing the stringency of the
2 microbiological quality standards for drinking water
3 has resulted in what EPA would point to as better
4 public health protection, there's absolutely no
5 reason to believe they wouldn't do exactly the same
6 for our recreational water quality.

7 They know that if they come up
8 with indicators that are better predictors of human
9 health risks, we will end up with better protection
10 of public health from recreational sources.

11 Q. Do you have any opinion as you sit
12 here today of what indicators might be better or
13 does that remain to be seen?

14 A. That really does remain to be seen. I
15 have not seen the outcome of all of the studies that
16 the EPA has already conducted, much less the ones
17 that they're planning to conduct so I really
18 couldn't speculate.

19 Q. Thank you. I'm going on to question
20 nine. On Page 13 you state, quote, US EPA has in
21 recent years informally applied a standard of five
22 times the primary contact standard, paren, sometimes
23 as high as ten times, closed paren, or a 1000 CFU
24 per 100 milliliters in evaluating proposed state

1 standards for recreational waters in which
2 non-primary contact recreation takes place.

3 Do you have an opinion on whether
4 this informally applied standard is appropriate or
5 based in the scientific literature?

6 MR. ANDES: If I can first ask --
7 that's clearly evidence being provided -- is
8 there any document indicating that that's an
9 EPA number?

10 MS. WILLIAMS: I'm quoting from the
11 pre-filed testimony. That was a quote from
12 the pre-filed testimony.

13 MR. ANDES: Okay.

14 BY THE WITNESS:

15 A. If you're asking whether this
16 informally applied standard for non-primary contact
17 recreation has a basis in the scientific literature,
18 I am not aware that it does. I am not aware of what
19 the basis for that was, no.

20 MS. WILLIAMS: Did you have any
21 follow-up, Fred?

22 MR. ANDES: No.

23 BY MS. WILLIAMS:

24 Q. I'm moving on to question 11. On Page

1 20 you state, quote, the process of disinfection
2 itself is not susceptible to fine tuning, it's
3 impact is binary, closed quote. Please explain this
4 statement.

5 A. The point that I was making with that
6 statement was that if you disinfect, you reduce the
7 concentration of organisms, if you don't disinfect,
8 you don't. It's as simple as that.

9 So by disinfecting the water, you
10 reduce the concentration of pathogens thereby
11 decreasing the risk of exposure to pathogens and
12 improving public health. If you don't disinfect,
13 you don't.

14 MR. ANDES: Can I follow up on that?

15 I guess my first question about that is that
16 I believe you testified earlier -- correct me
17 if I'm wrong -- that some methods of
18 disinfection reduce some pathogens and other
19 methods of disinfection reduce other
20 pathogens; am I right?

21 THE WITNESS: I would characterize it
22 slightly differently. I would say that
23 different disinfectants have different
24 degrees of efficacy against different

1 pathogens.

2 MR. ANDES: So you're not saying that
3 disinfection will reduce all pathogens? Any
4 particular disinfection technique would not
5 reduce all pathogens, correct?

6 THE WITNESS: No, that's not what I'm
7 saying at all, sir. I'm saying that for a
8 given disinfectant the degree to which it
9 would reduce different pathogens may be
10 different, but it would be highly unusual for
11 a particular disinfectant to have absolutely
12 no effect against any given pathogen --
13 against a particular pathogen.

14 MR. ANDES: So it might have some
15 effect against some pathogens?

16 THE WITNESS: Correct. It's going to
17 be different.

18 MR. ANDES: And it won't eliminate the
19 pathogens, correct?

20 THE WITNESS: Again, it's going to
21 depend greatly on how we define our universe.
22 But you could potentially find a situation
23 where for a finite volume of water and for a
24 given number of pathogens that you could

1 completely decrease the concentration of
2 those pathogens -- that particular pathogen
3 to non-detectable levels.

4 MR. ANDES: And have you reviewed the
5 testimony of Dr. Blaxley on that topic where
6 he contrasts conventional disinfection, which
7 clearly doesn't do that, versus extreme
8 disinfection that would cost a lot more but
9 achieve a higher level of removal?

10 THE WITNESS: I'm not sure what you're
11 referring to by extreme disinfection.

12 MR. ANDES: Such as using California
13 with gray water.

14 THE WITNESS: I don't know what
15 California's disinfection requirements are
16 for gray water.

17 MR. ANDES: You haven't looked at what
18 level of disinfection would be required to
19 achieve various levels of removal; am I
20 right?

21 THE WITNESS: I am not a wastewater
22 treatment engineer, as I've mentioned before.
23 All I know is that depending on the type of
24 disinfectants that you use you can achieve

1 different levels of removal of different
2 pathogens.

3 MR. ANDES: And when you testified
4 that there's sort of an either/or here, that
5 either you disinfect or you don't, you may
6 have reviewed testimony by -- I can't
7 remember if it was Dr. Oris or Dr. Gorlick
8 (phonetic) indicating that, in fact,
9 secondary treatment removes up to -- and I
10 believe that actually we talked about this --
11 that secondary treatment removes up to
12 99 percent of pathogens. I think you said
13 that wasn't enough. But that's not zero.

14 So when you say either remove
15 pathogens or you don't, you're saying once
16 you go on to secondary treatment, which could
17 remove a lot of pathogens, then you either
18 disinfect or you don't? You're not saying
19 there's zero removal of pathogen if you don't
20 disinfect because you admitted --

21 THE WITNESS: No. I've never said
22 that there's zero removal if you don't
23 disinfect.

24 MR. ANDES: Because you had conceded

1 that secondary treatment could remove in the
2 90s, just in terms of percentages you just
3 felt that wasn't enough; am I right?

4 THE WITNESS: I was disagreeing with
5 your characterization of 99 percent or
6 90 percent as being a lot. But certainly it
7 is documented that secondary treatment will
8 reduce the concentrations of different
9 pathogens to different degrees.

10 MR. ANDES: Okay.

11 BY MS. WILLIAMS:

12 Q. There was one follow-up question I
13 wanted to ask last time you were here but didn't get
14 a chance.

15 A. And you still remember it?

16 Q. Yeah. I wrote it down. So I'd like
17 to just quote a couple sentences from the transcript
18 if that's okay. I'm on Page 18 of the morning
19 transcript, Line 15. You state, the other thing I'd
20 like to point out is we use the term indicator for a
21 variety of purposes and so we need to be very clear
22 that we understand the context in which we're using
23 that term. We can use indicators to indicate how
24 well a treatment process is working, we can also use

1 them to indicate potential risks, so we need to be
2 clear exactly what context we're talking about
3 indicators in. Do you recall that?

4 A. I do.

5 Q. And the follow-up I wanted to ask at
6 that point was in this proceeding in which we are
7 holding hearings on the Illinois EPA's proposal to
8 the Pollution Control Board, which context of the
9 term indicator are we looking at?

10 A. I believe that the main
11 characterization of indicators in this particular
12 context would be using the indicator organisms to
13 tell you something about the overall microbiological
14 quality of the water with respect to its potential
15 to have a public health impact.

16 Q. That's fine if that's your answer. I
17 don't know if that answer is based at all on a
18 review of the Agency's proposal to the Pollution
19 Control Board in this proceeding.

20 A. I mean, at other points during the
21 time that I've been here in some of these other
22 documents we have used indicators in other ways
23 talking about how well a particular wastewater
24 treatment process will indicate a particular -- how

1 well a particular wastewater treatment process will
2 inactivate an indicator organism and what does that
3 tell you about how well it inactivates a particular
4 pathogen, which is a somewhat different way of using
5 indicators.

6 But overall I think what you're
7 concern is is using the indicators as a way to tell
8 you something about the microbiological quality of
9 that treated water to which people are being
10 exposed.

11 Q. Do you know if the Agency is proposing
12 an indicator organism that is reflective of the
13 quality?

14 A. I do not.

15 MS. WILLIAMS: That's all I have for
16 this witness. Thank you.

17 HEARING OFFICER TIPSORD: Any other
18 questions for Dr. Yates?

19 Dr. Yates, thank you so much for
20 coming back. We appreciate your testimony
21 and I hope you got your pizza.

22 THE WITNESS: I did. I can't believe
23 you remember that, Marie, but I did get my
24 pizza. It is fabulous. Thank you.

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HEARING OFFICER TIPSORD: On that
note, a couple of things -- off the record.
(Which were all the
proceedings had in the
above-entitled cause
on this date.)

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

3

4 I, Tamara Manganiello, CSR, RPR, do hereby
5 certify that I reported in shorthand the proceedings
6 held in the foregoing cause, and that the foregoing
7 is a true, complete and correct transcript of the
8 proceedings as appears from my stenographic notes so
9 taken and transcribed under my personal direction.

10

11

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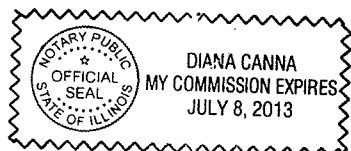
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before me this 7th day
of August, A.D., 2009.

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D. Canna
Notary Public

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24



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