

**In The Matter Of:**  
*Paul Halderson, et al., v.*  
*Star Blends, et al.,*

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*Lewis G. Sheffield*  
*May 9, 2014*  
*Volume 2*

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*Metropolitan Court Reporters, Inc.*  
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*Apple Valley, Minnesota 55124*

1 STATE OF WISCONSIN CIRCUIT COURT TREMPLEALEAU COUNTY  
 2 -----  
 3 Paul Halderson and Case No. 12-CV-74  
 4 Lyn M. Halderson, Code Nos: 30303 & 30201  
 5 N17388 County Road T  
 6 Galesville, Wisconsin 54630  
 7 and  
 8 Arctic View Farms, LLC  
 9 N17388 County Road T  
 10 Galesville, Wisconsin 54630,  
 11 Plaintiffs,  
 12 vs.  
 13 Star Blends LLC  
 14 1919 Riley Rd.  
 15 Sparta, Wisconsin 54656  
 16 and  
 17 ABC Insurance Company,  
 18 a fictitious company  
 19 and  
 20 Northern States Power Company  
 21 d/b/a Xcel Energy Services Inc.  
 22 1414 W. Hamlin Avenue  
 23 Eau Claire, WI 54702,  
 24 Defendants.  
 25 -----  
 26 STATE OF MINNESOTA IN DISTRICT COURT  
 27 COUNTY OF CASS NINTH JUDICIAL DISTRICT  
 28 11-CV-12-1670  
 29 -----  
 30 Randall and Peggy Norman,  
 Plaintiffs,  
 vs.  
 Crow Wing Cooperative Power & Light Company,  
 Defendant.

VOLUME II

Continuing Deposition of LEWIS G. SHEFFIELD, PhD, taken pursuant to Notice of Taking Deposition, and taken before John T. Kirby, a Notary Public in and for the County of Dakota, State of Minnesota, on the 9th day of May, 2014, at 1 South Pinckney Street, Madison, Wisconsin, commencing at approximately 9:22 a.m.

1 APPEARANCES:  
 2 Scott Lawrence, Esquire, of the LAWRENCE  
 3 LAW OFFICE, S.C., 403 South Fourth Avenue, P.O. Box 117,  
 4 Saint Nazianz, Wisconsin 54232-0117, 920-773-2811,  
 5 ATTORNEYS@LDLAWSTN.COM, appeared representing the  
 6 Plaintiffs, both captions.  
 7  
 8 Timothy R. Thornton, Esquire, of the firm  
 9 of BRIGGS & MORGAN, 2400 IDS Center, Minneapolis,  
 10 Minnesota 55402, 612-977-8400, tthornton@briggs.com,  
 11 appeared representing Defendant NSP/Xcel Energy.  
 12  
 13 Paul F. Carlson, Esquire, of the firm of  
 14 KENNEDY, CARLSON & VAN BRUGGEN, LLP, 116 Ash Avenue NW,  
 15 P.O. Box 647, 218-631-2505, pcarlson@kevblaw.com, Wadena,  
 16 Minnesota 56482-0647, appeared representing Crow Wing  
 17 Cooperative Power & Light Company.  
 18 ALSO PRESENT:  
 19 Theresa A. Peterson, DVM.  
 20  
 21 VIDEOGRAPHER:  
 22 Mark C. Haskins, HASKINS MEDIA SERVICES,  
 23 1071 Whitney Drive, Apple Valley, Minnesota 55124,  
 24 952-997-6455, mark@haskinsmediaservices.com.  
 25 EXHIBIT INDEX  
 26 275 90-28 282 150-6 PREVIOUSLY MARKED  
 27 276 112-17 283 151-11 249 164-14  
 28 277 88-29 284 153-15 250 138-18  
 29 278 135-22 285 155-17 251 157-19  
 30 279 141-17 286 156-29 252 173-27  
 280 149-16 287 165-27 253 (No Ref)  
 281 149-26 288 167-23 254 84-29  
 289 172-20

EXAMINATIONS

1  
 2  
 3 By Mr. Lawrence: 84.  
 4 By Mr. Carlson: 176.  
 5 By Mr. Thornton: 182.  
 6  
 7 \* \* \* \*  
 8  
 9 WHEREUPON, the following proceedings were duly had:  
 10  
 11 \* \* \* \*  
 12  
 13 MR. HASKINS: Today is Friday, May 9, 2014.  
 14 The time is approximately 9:22 a.m. This is Volume II,  
 15 Tape Number 1, of the continuing video deposition of Dr.  
 16 Lewis G. Sheffield, taken by Defendant Northern States  
 17 Power Company in the matter of Paul Halderson, et al,  
 18 versus Star Blends LLC, et al, State of Wisconsin,  
 19 Circuit Court, Trempealeau County, Case Number 12-CV-74.  
 20 This deposition is being held at the Law Firm of Boardman  
 21 and Clark, Madison, Wisconsin.  
 22 My name is Mark C. Haskins, I'm the video  
 23 technician of Haskins Media Services, Apple Valley,  
 24 Minnesota 55124.  
 25 Will counsel please note their appearances,  
 26 after which the court reporter will swear in the witness.  
 27 MR. LAWRENCE: The Plaintiffs appear by  
 28 attorney Scott Lawrence. Also present is their  
 29 consultant, Dr. Theresa Peterson.  
 30 MR. THORNTON: Tim Thornton for NSP.

1 MR. LAWRENCE: Then, I think you ought to  
 2 put the Minnesota case into the record also, please.  
 3 MR. HASKINS: Sure. Again, this matter has  
 4 also been noticed in the matter of Randall and Peggy  
 5 Norman versus Crow Wing Cooperative Power and Light  
 6 Company. Court File Number 11-CV-12-1670, in the State  
 7 of Minnesota, County of Cass. I think that covers it  
 8 then?  
 9 MR. LAWRENCE: Sure. And the Plaintiffs in  
 10 that case, at least for the time being, appear by Scott  
 11 Lawrence, and also Charles Bird may appear by phone later  
 12 this morning.  
 13 MR. CARLSON: Paul Carlson representing  
 14 Crow Wing Power.  
 15 MR. HASKINS: Okay.  
 16  
 17 LEWIS G. SHEFFIELD, PhD,  
 18 an expert witness in the above matter,  
 19 after having been first duly sworn,  
 20 testified under oath as follows:  
 21  
 22 CONTINUING CROSS EXAMINATION  
 23  
 24 BY MR. LAWRENCE:  
 25  
 26 Q Good morning, Dr. Sheffield.  
 27 A Good morning.  
 28 Q Good to see you again. I am going to, in a few moments  
 29 anyway, continue asking you questions about exhibit 254,  
 30 that long spreadsheet we've talked about the last time,

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1 and the other documents you have in front of you which I  
2 will get to in a minute.  
3 Before I do that, with respect to the  
4 second study, the later one in time that we were  
5 discussing in the first round of your deposition that  
6 involved measurements of involving Messenger RNA, could  
7 you describe how that study came about and how it was  
8 funded, please?  
9 A You're asking some very long ago - -  
10 Q Understood. And if you don't remember, you don't  
11 remember.  
12 A Well, I can sort of, I think, give a general gist of  
13 this. I had been part of an earlier study with Douglas  
14 Reinemann, in which we did a lot of functional measure-  
15 ments. And, I am sorry, I am not going to remember years  
16 from this, but there was a request for proposals through  
17 the College of Agriculture, I believe that came about  
18 from a line in the state budget. But this is, again,  
19 very old memories.  
20 I responded to that and this project was  
21 selected for that. And it was designed to be, in some  
22 respects, a follow-up to the previous Reinemann study  
23 looking at some broader ideas.  
24 Q So the funding basically came out of the Wisconsin state  
25 government?  
26 A I believe that is correct.  
27 Q And if I understood what you just said correctly, you  
28 believe it came through the Department of Agriculture, is  
29 that right?  
30 A Well, it came through the - it was funneled to the UW

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1 through the College of Agriculture.  
2 Q Oh, the College of Agriculture. Excuse me.  
3 A Yes. I don't know about state departments that would be  
4 involved. I may have known that ten years ago, but I  
5 don't recall that.  
6 Q Sure. All right. And can you describe with any more  
7 specificity than that, than what you just did, what the  
8 object was in follow-up to the earlier study?  
9 A Yes. The objective was to first develop some tools  
10 to assess gene expression in cattle. At that time, this  
11 technology was rather poorly developed, particularly in  
12 cattle, it was beginning to be developed in humans and  
13 model species, like mice.  
14 Our objective was to try to get as broad a  
15 spectrum as we could of things that might be relevant to  
16 immune function. And, of course, along the way, include  
17 some things that might be either general of some genes  
18 that might be part of the immune function, but part of  
19 bigger things as well. And as you might notice in here  
20 some things that we used as controls that you wouldn't  
21 expect to see in immune function cells, and then use this  
22 to determine whether exposure to very low voltages in  
23 dairy cattle affected any of these potential measures.  
24 Q And we discussed before, a little bit anyway, about  
25 exhibit 254, which is a four-page spreadsheet, and I'll  
26 represent to you that that spreadsheet was printed off of  
27 materials that were subpoenaed through the University and  
28 provided in April of 2008. They were copied by a copy  
29 shop here in Madison directly from the University  
30 Counsel's office and provided to us. And that spread-

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1 sheet appeared on a disk that was - probably you have  
2 never seen this disk cover before, but it's got your name  
3 on it and it talks about SV ARRAY. And there's all kinds  
4 of files on there, one of which contains that spread-  
5 sheet.  
6 Do you recall seeing that spreadsheet in  
7 the past, back when you were doing - after you were doing  
8 - -  
9 A I don't recall whether these - all the last rows of the  
10 means were in the spreadsheet or not, but it looks very  
11 much like this, and I do seem to, my suspicion - well,  
12 let me say, I didn't realize that it would have also been  
13 sorted by treatment group, whether they have treatments  
14 at random. But it does look very much like the spread-  
15 sheet that I would have generated. And I don't know - I  
16 don't recall whether those last rows were something I had  
17 generated in there or not. The numbers in them look like  
18 the numbers that I would have generated, had I done it.  
19 So.  
20 Q Okay. And there very well may be a copy of this - -  
21 A I'm not saying it was on the original, I'm just saying I  
22 don't recall that it looked exactly like that. But the  
23 data looks right.  
24 Q There may be a copy of that spreadsheet on that disk  
25 without the means and so forth, the last four rows also,  
26 I'm not sure. But that one does appear - -  
27 A This does look like something I would have generated.  
28 Like I said, I don't recall that being on there, but I'm  
29 not saying it wasn't. It was like ten years ago that I  
30 generated this.

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1 Q Sure. And on the disk, as we received it from UW, there  
2 was the - well, the data can be opened in EXCEL, what  
3 seems to take a few steps to do that with software that's  
4 available these days. Can you describe for me what  
5 software you were using back then?  
6 A I believe that was done in the program called Minitab.  
7 Q Can you spell that for John?  
8 A M-i-n-i-t-a-b.  
9 Q All right. Go ahead.  
10 A That is a fairly standard statistics program. It's  
11 commercially available, and reasonably widely used.  
12 Q And I take it from your earlier testimony, you do not  
13 recall asking the program to calculate the means or P  
14 values in this case, is that correct?  
15 A Oh, I would have done that.  
16 Q Okay.  
17 A I just don't recall putting them on this spreadsheet. I  
18 certainly would have asked the program to calculate  
19 means, standard errors and P values.  
20 Q What statistical test would you have used initially to do  
21 that on this particular data?  
22 A The initial test would have been a t-test. We have two  
23 treatment groups, and so there would have been a - what's  
24 often called a Student's t-test done on each piece.  
25 Q Then, is there such a thing as a two sample t-test?  
26 A That's what the Student's t-test is. That's what I would  
27 have done.  
28 Q Just so that we can get the mathematics straight, let me  
29 mark another exhibit, please. This will be 277.  
30 MR. THORNTON: I thought we were on 276.

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1 Q That's the disk.  
 2 MR. THORNTON: Oh, okay.  
 3 Q I'll just give you a moment to look at 277. I believe  
 4 this just reflects the basic statistical computation that  
 5 would be done to accomplish a two sample treatment and  
 6 control t-test in these circumstances. Would you take a  
 7 look at that and tell me if you agree?  
 8 A That looks like the t-test, yes.  
 9 Q All right. So, in this particular case, given that we  
 10 have ten treatment cows and ten control cows, I believe  
 11 we'd be looking at a t-test with - or a pool t-test with  
 12 18 degrees of freedom, is that correct?  
 13 A I think that's correct.  
 14 Q And so, assuming the mathematics to be correct as at the  
 15 bottom of exhibit 254, let's just talk about what those  
 16 numbers are. We will take the first variable, ACK2 as an  
 17 example. The C mean would be what?  
 18 A That would be the mean of the control group. That is the  
 19 cows that were not treated with voltage.  
 20 Q Sure. And that's just a simple average - -  
 21 A That's just an arithmetic average in this case, yes.  
 22 Q And the T Mean would be what?  
 23 A That would be the arithmetic average of the animals, the  
 24 treated animals, that is, exposed to the current.  
 25 Q And the Fold (T/C) is what?  
 26 A A common way of expressing gene expression is to look at  
 27 how much it changed as a relative. So, basically, that  
 28 is taking the T Mean divided by the C Mean.  
 29 Q And then the P value at the bottom is what?  
 30 A The last row is what is called a P value. And that is

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1 taken from the t-test, as described here, comparing the  
 2 treatment and control mean. And the smaller the number,  
 3 the higher the level of significance we put on it.  
 4 Q So, mechanically, to arrive at the P value, one would  
 5 take the data, compute the means, compute the T value in  
 6 accordance with exhibit 277 in front of you, and then go  
 7 to either tables, or there are many online calculators  
 8 that will do this for you these days also, and the tables  
 9 or the calculator will give you the P value from the T  
 10 number, correct?  
 11 A Essentially. Most standard statistics programs,  
 12 including Minitab, will compute that. I don't know the  
 13 algorithm it uses, but that's correct.  
 14 Q Back in the days before software did this for us, we  
 15 probably looked it up in a table at the back of the book  
 16 that had many pages in it.  
 17 A That is how I learned to do it many years ago.  
 18 Q And essentially, the P value shows you how far out you  
 19 are on the bell shape curve away from the mean, correct?  
 20 A Essentially, yes. Actually, let me correct that  
 21 slightly.  
 22 Q Sure. Please do.  
 23 A What it actually shows you is how far away from zero your  
 24 effect of your treatment gets. And the smaller the  
 25 number, the further away from no treatment effect you  
 26 are.  
 27 Q Then, I've also marked as an exhibit this morning,  
 28 exhibit 275, and I believe you had at least 10 or maybe  
 29 20 minutes to look at that before we got going this  
 30 morning, is that correct?

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1 A That's correct.  
 2 Q And I will represent to you what that document is, in the  
 3 interest of getting through this testimony today in less  
 4 than three day's time, and in the interest of not driving  
 5 Mr. Kirby too nuts with long words. I asked Dr. Chris  
 6 Chase, who is a veterinarian and a professor at South  
 7 Dakota State University, and a past president of the  
 8 American Association of Veterinary Immunologists, to look  
 9 at a copy of exhibit 254 and summarize his understanding  
 10 of what those variables mean, where he could. I think  
 11 there's a couple where he couldn't come up with the  
 12 meaning, and I'm going to ask you about that. All right.  
 13 I also asked him to indicate his opinion on  
 14 whether or not a change in each variable is indicative of  
 15 an immune system specific effect, as he characterizes it,  
 16 or, in other words, if there's a change in that variable,  
 17 is that more likely than not indicative of a change in  
 18 immune function as opposed to something else. And he has  
 19 given his opinion on that.  
 20 I want to ask you those questions, too.  
 21 And you may not agree with him on all of them, obviously.  
 22 But this is going to permit us to do this in shorthand.  
 23 MR. THORNTON: I object. Exhibit 275 is  
 24 hearsay, and is the work and opinion of an expert that  
 25 was not timely identified in this litigation.  
 26 Q With that in mind, Professor Sheffield, let's go back to  
 27 ACK2, which we talked about a little bit in the last  
 28 deposition. But in shorthand, would you agree with  
 29 Professor Chase's characterization of that variable on  
 30 exhibit 275?

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1 A Yes, I'll say that's a - in all of these cases, obviously  
 2 there's far more one could say about it, but that's a  
 3 fair assessment.  
 4 Q All right. And if you agree with his comment in the  
 5 immune system specific effect column that he says, yes,  
 6 and in particular, it has an effect on mast cells?  
 7 A Yes, it does.  
 8 Q You told me in the first portion of your deposition that  
 9 there were variables in this study that you did not  
 10 expect to see an effect on. Would this be one of them or  
 11 not?  
 12 A This one you might see an effect on or might not. It  
 13 depends on what effect you have.  
 14 Q Well, certainly wasn't one of the variables you put in  
 15 here where you did not expect to see an effect?  
 16 A I would have thought that neither result would have  
 17 surprised me very much.  
 18 Q I'm contemplating asking you which of these variables you  
 19 did not expect to see an effect on and which variables  
 20 you should not have seen in the cells at all, because you  
 21 told me that was true of some of them. And I take it  
 22 there probably are relatively few of those in this study,  
 23 is that correct?  
 24 A There are not many.  
 25 Q Why don't we do that first then, and I won't have to ask  
 26 you that about each individual one. Okay? First of all,  
 27 those that you did not expect to see an effect on. If  
 28 you want to just scan the list and tell us which ones  
 29 those are as we go.  
 30 A Yes. Before that, may I define what I mean by expect?

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1 Q Please.  
 2 A Because I think this is an important thing. Let's  
 3 assume, if the voltage exposure was having some  
 4 measurable effect on the immune function, would you  
 5 expect to see these changed? So, I'm not - when I say  
 6 expect, I'm not trying to imply that I expected voltage  
 7 to either have or not have an effect. It's if it had an  
 8 effect, would this be something that it might have  
 9 affected? Is that clear and fair?  
 10 Q I think so. Mr. Thornton, any problems with that?  
 11 MR. THORNTON: Can you try it again?  
 12 A I'll try it. Okay. When you say expect, there's two  
 13 aspects. Am I expecting that voltage has an effect, or  
 14 am I expecting that if something has an effect, would it  
 15 affect that measurement? So, whatever we use as our  
 16 treatment, if it affected immune function, would this be  
 17 something you might expect it to see changing. As  
 18 opposed to a different question, which is, did I expect  
 19 going in, because I try to approach things scientific-  
 20 ally as I don't know what the answer is before, in terms  
 21 of whether my treatment was having an effect. It's a  
 22 different aspect. Am I expecting the treatment effect or  
 23 am I expecting this measurement to reflect any possible  
 24 treatment effect?  
 25 Q So, perhaps stated another way, if we find the relatively  
 26 few variables where you did not expect to see an effect,  
 27 that's another way of saying there's simply not - those  
 28 variables aren't at play with the change in immune  
 29 function. Would that be fair?  
 30 A I think that's fair.

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1 Q Okay. Which ones are those then?  
 2 A Okay. The ones that I would expect not too see an  
 3 effect?  
 4 Q Correct. Before you even start.  
 5 A Adenyl cyclase. ATP Synthase. The CaATPase; calcium  
 6 ATPase. Casein Kinase. CleavagePolyA. I guess I was  
 7 unsure about CREP1 and CREP2. GAP. The column labeled  
 8 Glu TransV, which is Roman numeral 5. Stands for Glucose  
 9 Transporter 5.  
 10 Q I'm sorry. Let me see if I can find that one.  
 11 MR. THORNTON: Bottom of page 2.  
 12 Q Or 254, would that be the one on the far right column or  
 13 has a Roman I, not V.  
 14 A Oh, yes. That is an error on exhibit 275. That should  
 15 be Glu Trans IV, not V. So, the spreadsheet here is  
 16 Glucose Transporter 5. On this - get my numbers right -  
 17 exhibit 275, it is typed as GluTransV. That actually  
 18 should be an IV in there.  
 19 Q And on 254 it says GluTransI, I think there should be a  
 20 V.  
 21 A That's Glucose Transporter 4. That is the most common of  
 22 the Glucose Transporter proteins in those.  
 23 Q So that would be one of these variables?  
 24 A Yes. And that's one I would not expect to change in this  
 25 situation.  
 26 Q Okay. Please continue.  
 27 A Hexo Kin 1. Which stands for Hexokinase 1. I would be  
 28 unsure about IGF1Rb, insulin-like growth factor 1  
 29 receptor beta form. And similarly IGR1R. I would be  
 30 unsure about Leptin.

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1 MR. THORNTON: You say what?  
 2 A Leptin.  
 3 Q What page are you on?  
 4 A Bottom, near the last on Page 6. The various  
 5 collagenases, mmp1, 3 and 9, I think would be unlikely.  
 6 I wouldn't say it's impossible, but it's, I would say  
 7 unlikely. PLC and PLCa, although I would expect activity  
 8 of those to go up, I'm not sure I would expect the  
 9 expression level to change.  
 10 I would say the same thing about the next  
 11 several genes, PKACAlpha 1, PKACAlpha, PKACDelta,  
 12 PKCalpha, PKG1beta, RASGAP, RhoGDI. GDK is an  
 13 interesting one. Classically, we don't think of this as  
 14 even being present in things other than in the illo  
 15 cells.  
 16 So, at first glance, you don't expect to  
 17 see very much of it, if any, in this. Although it turns  
 18 out there are a few studies that have shown TEK to be  
 19 involved in lymphocyte proliferation and activity. And  
 20 we did see this gene in this study. We didn't see a  
 21 change in it, but we did see it expressed at the above  
 22 basil levels.  
 23 Q Sure. You came up with a P value of about .13, it looks  
 24 like, from the spreadsheet.  
 25 A I don't remember that. I would have to look at the  
 26 spreadsheet to see.  
 27 Q Sure.  
 28 A But I'll take your word for that for now.  
 29 Q Okay. So that would be a question mark, perhaps?  
 30 A That was one I - there's not that much known about it in

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1 lymphocytes, but it's kind of - was unexpected to me to  
 2 even find any substantial amounts of it.  
 3 Q So, I guess it's neither a yes or a no, but it's  
 4 interesting?  
 5 A It's interesting, and that's how I would describe that.  
 6 On Page 11, Casein we wouldn't expect to see at all.  
 7 That's only present in mammary tissue. The Klebella,  
 8 that is actually a gene from a bacterium, (negative  
 9 batgerium), Klebella pneumonia. I don't remember exactly  
 10 which gene, off the top of my head. It's one of the  
 11 lymphocyto (ph) genes of the bacteria. And that is so  
 12 different in the lymphocyto RNA genes, if you carry out,  
 13 you would expect to see a signal there. And we didn't.  
 14 Q Sure. And I'm sorry, plus I take it would be one you  
 15 shouldn't even see it - -  
 16 A Shouldn't even see it. You'd be watching an empty well,  
 17 or very close to it. That's what we call a specificity  
 18 control. Same thing with pGEM. This is a plasmin that  
 19 is present in certain bacteria, not in the geriotics (ph).  
 20 And GAPDH is another one that is - is very commonly used  
 21 as what's called a housekeeping gene. It's something  
 22 that you rarely see changing.  
 23 And then the Empty, that is his assumption  
 24 here that nothing was added to that well, that's correct  
 25 for that.  
 26 Q So, because you jumped around a little bit there, I would  
 27 like to just summarize then. The items that you should  
 28 not have seen in the cells at all, I believe would be -  
 29 well, on exhibit 254, it would be on the last page, right  
 30 and the end, I think, if I understood you correctly?

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1 A That's correct.  
 2 Q And they would include casein, Klebella, pGEM, and Empty,  
 3 is that correct?  
 4 A That's correct.  
 5 Q Did I miss any? Are there any others that you shouldn't  
 6 have seen at all?  
 7 A I think that's correct.  
 8 Q And then starting on, let's stick to 254 for a moment.  
 9 You described PLC as being one that you did not expect to  
 10 see a change, at least in gene expression, is that  
 11 correct?  
 12 A That's correct.  
 13 Q And then the same comment, this is where you jumped  
 14 around a little bit, so I would just like to clarify it  
 15 for the record, if we may. The same would be true of  
 16 PLCa, next to it, is that - -  
 17 A That's correct. PLCa is what we call an isoform. It has  
 18 essentially the same activity, but it's a slightly  
 19 different sequence of the gene.  
 20 Q If my notes are correct, the next one going to the right  
 21 on 254 would be PKACAlpha. I'm not sure I got that right  
 22 though.  
 23 A PKA - I may have skipped over one. There's a column  
 24 here, says, PKAbCat. That I wouldn't expect to change  
 25 either.  
 26 Q And, again, - -  
 27 A PKCAAlpha, PKARIIB.  
 28 Q I think the next one in that category was RASGap, GAD, is  
 29 that correct?  
 30 A Actually, I'm not sure - I don't think I would have

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1 expected PKG1Alpha, and PKG1Beta. And then we go to the  
 2 RASGAP.  
 3 Q That, also, you did not expect a change, correct?  
 4 A I didn't expect that to change.  
 5 Q And the RhoGDI also, is that correct?  
 6 A I didn't expect to see it, no.  
 7 Q The rest of the sheet going to the right on the third  
 8 page of exhibit 254, none of those fall in the category  
 9 of did not expect a change, correct?  
 10 A All of them fall into the category of, you may see a  
 11 change.  
 12 Q On the last page, just as the first one we covered at  
 13 length, was interesting. After that, the first one I  
 14 noted as you did not expect a change was PKCDelta, about  
 15 halfway across the sheet. Did I miss any or did we miss  
 16 any?  
 17 A There is another one in here called PKAR2Alpha. I may  
 18 not have mentioned that one. But that is one I wouldn't  
 19 have expected.  
 20 Q I think you did say that before, at least I wrote it down  
 21 anyway.  
 22 A Is that - - there's a - - these abbreviations start  
 23 looking very similar. The PKA stands for protein kinase  
 24 8. And the C and R stands for catalytic or regulatory  
 25 sub-unit, and then we have 1 and 2 alpha and beta for  
 26 each of those. So, it's easy to let them run together  
 27 for a while. But that is one I would not expect to see  
 28 changing. I may have mentioned it earlier. I think  
 29 that's correct.  
 30 Q All right. And then I think that is the last one that

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1 falls into the category of did not expect to change until  
 2 we get to casein, and we covered those last several a few  
 3 moments ago.  
 4 A I think that's correct.  
 5 Q All right. I think we probably have them all then. I  
 6 would then like to go through, I think we will go through  
 7 the ones that you didn't name as falling in that  
 8 category, and I will ask you for each of those variables,  
 9 whether you would agree with Dr. Chase's description of  
 10 the variable, and his conclusion regarding whether that  
 11 variable is associated with an immune system specific  
 12 effect.  
 13 I think the first of those that we - well,  
 14 go ahead.  
 15 MR. THORNTON: I'm going to object again  
 16 based upon an opinion of an expert that hasn't been  
 17 identified in a timely fashion or hasn't produced a  
 18 report.  
 19 Q We've talked about ACK2 already, I believe. I don't  
 20 think we need to cover that again.  
 21 The next one is cFos. Do you agree with  
 22 Dr. Chase's characterization of that item, and whether or  
 23 not there is an immune system specific effect?  
 24 MR. THORNTON: I don't want to continue to  
 25 interrupt you, but can I have a continuing objection?  
 26 Q Certainly.  
 27 MR. THORNTON: All right. Go ahead.  
 28 A I will agree with his assessment. I do not know exactly  
 29 what is meant by immune system specific effect. There  
 30 are two possibilities that come to my mind. I am

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1 assuming what he means here is that the effect it has is  
 2 very specific to the immune system, it is not something  
 3 you see throughout the body. That's what I assumed when  
 4 I first saw that column.  
 5 Q I think that's a fair characterization.  
 6 A Okay. I want to make sure that what I'm saying lines up  
 7 with what everyone is. I think the question I'm  
 8 answering is the question that is intended to be  
 9 answered.  
 10 There is another aspect, and cFos is a very  
 11 good chain to describe this with. CFos and cJun and some  
 12 of the others on here are very common genes of the body.  
 13 Almost every cell in the body has cFos in it. It's  
 14 normally expressed at a very low level, and certain  
 15 activators of the cell change the expression level of  
 16 cFos.  
 17 For example, if you take a cell and add a  
 18 growth promoting agent to it, one of the very early  
 19 effects is that the expression level of cFos goes very  
 20 high. So, things that would affect the immune system  
 21 might well affect cFos even though it is not an immune  
 22 specific event, which is why I put it in the category of  
 23 maybe you would see an effect on it. I would say many of  
 24 the effects on cFos are very transient, it goes up and it  
 25 comes back down very quickly.  
 26 Q The next one in the list is cJun. Same question for each  
 27 one. Do you agree with Dr. Chase's description as set  
 28 forth on exhibit 275 and his conclusion about immune  
 29 system specific effect?  
 30 A I will agree with that, and I will say the same thing I

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1 did about cFos. It's another of these genes that is  
 2 activated by a lot of different things, and present in  
 3 most cells in the body.  
 4 Q For each one we come to, I'm just going to ask the same  
 5 question so we don't make a lot of transcript here. The  
 6 next one - -  
 7 MR. CARLSON: To the extent that Mr.  
 8 Thornton objects, I'm going to join in those objections  
 9 rather than making similar objections. So, both cases,  
 10 okay?  
 11 MR. LAWRENCE: Understood. I don't have  
 12 any concern about opinion objections.  
 13 MR. CARLSON: All right. But to the extent  
 14 he makes an objection, I don't want to waste time voicing  
 15 the same objection because I'm here on a different case.  
 16 MR. LAWRENCE: Understood.  
 17 MR. CARLSON: Okay.  
 18 Q The next one that was not a yes to, did not expect to see  
 19 an effect, is CasKin1. At least that's the abbreviation  
 20 on the spreadsheet.  
 21 A Yes. His assessment of this is correct. It's one you  
 22 might see. Because this is a very general enzyme in  
 23 cells, you might expect to see some increase, but not  
 24 seeing an increase would also not be too surprising.  
 25 Q You can skip CasKin2 and go to CD14.  
 26 A I will agree with his assessment of CD14.  
 27 Q And he does assess that as to be something to the immune  
 28 system, and you would agree with that?  
 29 A Yes, definitely. Same with CD23. Is that okay, if I  
 30 just go down the list of these?

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1 Q Please.  
 2 A Same with CD23. Same with CD8. Same with CD3. These  
 3 are all very common proteins on the immune system cells  
 4 and they have major roles in the immune function.  
 5 Q Cdk1?  
 6 A Cdk1, his assessment of it is correct. Cyclin dependent  
 7 kinase is expressed during what we call the cell cycle.  
 8 That is, cells are proliferated - or stimulated to  
 9 proliferate, so it is present in most cells. So we  
 10 wouldn't expect it specific to the immune system, unlike  
 11 the CD genes, but if cells are being stimulated to  
 12 proliferate, you might see it increased.  
 13 Q I think we can skip ClevPolyA, and go - well, we probably  
 14 should cover CREB1 and CREB2, because you were unsure,  
 15 shall we say, on those with respect to - -  
 16 A Yes. Correct. His description of what they do is, I  
 17 will agree with. These are cyclic A and P. To put this  
 18 in some context, is a very common substance we call a  
 19 second Messenger. It has a wide variety of effects on  
 20 activity of many enzymes and expression of many genes.  
 21 And it's used as a second Messenger in many cells in the  
 22 body, including some in the immune system.  
 23 CREB1 is a protein that is activated by  
 24 cyclin A and P. So I actually would say it probably  
 25 wouldn't see the expression change a lot, but there are  
 26 some cases when you do see changes in CREB1, and the same  
 27 thing with KREB2, expression levels. So that's why I  
 28 said I was unsure about it. It is not something that's  
 29 specific to the immune system, I would say a very, very  
 30 common protein in many cells in the body.

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1 Q Both KREB1 and 2?  
 2 A KREB1 and 2, yes.  
 3 Q Then I think the next item - the next variable is  
 4 CuZnSOD.  
 5 A That's right. I will agree with his assessment of that.  
 6 Q And can you tell us what the next variable is,  
 7 Desmogelin?  
 8 A Yes.  
 9 Q I think Dr. Chase couldn't make it out, so he doesn't  
 10 have any restriction.  
 11 A As near as I can tell, that is a rather bad typographical  
 12 error on there. I believe that that is a protein called  
 13 Desmogelin.  
 14 Q Can you spell that for John?  
 15 A Yes. I expected to spell that. And I am a terrible  
 16 speller, so I'm going to get it close. If I misspell it,  
 17 please forgive me on that.  
 18 Q We will.  
 19 A It is spelled, D-e-s-m-o-g-e-l-i-n. I think that is  
 20 correct.  
 21 Q Okay. What is that protein and what's its function?  
 22 A This is one that, at first glance, you would not expect  
 23 to see at all, if you look at the classic definition of  
 24 what it is and where it's found. Desmosomes,  
 25 D-e-s-m-o-s-o-m-e-s, are what we call junctions. They  
 26 are very common in epithelial cells and sometimes found in  
 27 other cells as well.  
 28 They basically hold cells together. If  
 29 you've got a sheet of cells, something has to hold them  
 30 together. There are several things that do this, and

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1 desmosomes are among them. So we see this very commonly  
 2 in epithelial cells.  
 3 I was surprised to find very much of it in  
 4 these cells at all. It turns out, however, that there  
 5 are some lymphocytes that do seem to express this, and it  
 6 seems to be involved in their ability to invade tissues.  
 7 Lymphocytes sometimes need to attach to tissues and  
 8 epithelial tissues, the lining of the vascular system,  
 9 and lead to vascular system. It's been studied in a few  
 10 disease processes where this occurs.  
 11 Q Well, at the time - -  
 12 A At the time I was kind of surprised to even see it there.  
 13 Q So, I take it this perhaps falls into one that you didn't  
 14 expect to see an effect or - -  
 15 A I would not have expected to see any effect of that. I  
 16 actually would have expected to see very low levels of  
 17 it.  
 18 Q I take it from that, that if there were - if it were  
 19 detected and if there were a change, you wouldn't  
 20 consider that likely to be immune specific, is that  
 21 correct?  
 22 A That would have been my initial reaction to it.  
 23 Q As you sit here today, would that have changed?  
 24 A I would be maybe a little more qualified. There is not a  
 25 lot known about it in the lymphocytes. It is not well  
 26 studied, because it, as I said, it has been traditionally  
 27 thought to be in epithelial tissues. That's where almost  
 28 all the work on it has been done.  
 29 Q And the knowledge of the expression of lymphocytes is  
 30 something that has come about since this study was

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1 performed, I take it?  
 2 A I think there were a couple studies before this study was  
 3 performed. I may be mistaken on that, but I believe  
 4 there were a couple that showed that it might be present  
 5 in lymphocytes.  
 6 Q Well, I guess, obviously, in general, the human knowledge  
 7 of gene expression in human or cattle cells is a subject  
 8 that is just exploding over the last decade or two, is  
 9 that correct?  
 10 A In the last ten years, it has exploded, yes. There's a  
 11 lot we know now that was not known when this was done.  
 12 Q Would it be fair to say the same is true with respect to  
 13 immunological function of humans and animals, such as  
 14 cows?  
 15 A I would say that's true, yes.  
 16 Q The next item on the spreadsheet, and I'm going from left  
 17 to right on exhibit 254, but I think you can do the same  
 18 thing on 275. The next one is FAS. Same question.  
 19 A I will agree with his assessment here of what is FAS, as  
 20 well as FASLigand. This, possibly you could see some  
 21 changes in FAS. I think changes in FASLigand, would be  
 22 much more likely to see if you're seeing immune system  
 23 effects.  
 24 Q And, Dr. Chase in the immune specific - excuse me, immune  
 25 system specific effect column, had a no for FAS and a yes  
 26 for FASLigand. Go ahead. I'm sorry. Sounds like you  
 27 are sort of in the same ballpark?  
 28 A I am. I am. FAS is a receptor. It's present on any  
 29 cells. It induces cell death. So, you're going to see  
 30 effects, very wide spread effects in distribution of FAS.

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1 FASLigand is the Ligand for that receptor.  
 2 Q GAP is one you didn't expect to see change. So let's  
 3 proceed to GlutPerox.  
 4 A Yes. Yes. I will agree with what he says about that.  
 5 Q Again, a change in that item would be likely immune  
 6 specific, correct?  
 7 A Yes. I'll agree to that.  
 8 Q And then, going to the right, the next is GluTrans - it  
 9 should be Roman numeral IV?  
 10 A It should be Roman numeral IV, not V.  
 11 MR. THORNTON: It's Roman IV on 254, but  
 12 it's Roman V on 275?  
 13 A Yes.  
 14 Q It's actually Roman I on 254, but --  
 15 A 254 is sort of IV, quantity 4.  
 16 Q That's right.  
 17 MR. THORNTON: No, it's 4.  
 18 A IV, four.  
 19 MR. CARLSON: I think you might have said 4  
 20 before - you might have said 1 before, but it's actually  
 21 4.  
 22 A Whoever typed this just left the I out. It should be  
 23 Glucose Transporter 4.  
 24 MR. THORNTON: Roman IV.  
 25 A Roman IV, yes.  
 26 Q Assuming that we don't know that Dr. Chase had that in  
 27 mind, do you agree with his comments or is --  
 28 A What he actually says, Glucose Transporter IV in the  
 29 typed section. It is just in the column, for the  
 30 abbreviation he has the V.

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1 MR. THORNTON: But it's Arabic 4 in the --  
 2 A He has Arabic 4. You will see it written both ways. And  
 3 I will agree with what he says about that.  
 4 Q The next item on 254, is the first one on the lefthand  
 5 side of exhibit 254, is GMCSF. Same question.  
 6 A I will agree with what he says.  
 7 Q Next is HexoKinase1. Excuse me. I'm sorry. That's one  
 8 you did not agree with.  
 9 A That's one I wouldn't expect to see a change.  
 10 Q And the next one then is HSP70. Same question.  
 11 A I will agree with his assessment of what it does.  
 12 Because this is a very common protein to see expressed  
 13 during stress situations, you might see it expressed with  
 14 inducing some kind of stress here. It would not be  
 15 specific to the immune system, that we can say almost  
 16 universal protein.  
 17 Q The next one is IGF1Rb. You were, I'm sure whether you  
 18 would have expected to see an effect on that one. So  
 19 same question with respect to what Dr. Chase has  
 20 summarized.  
 21 A His description I will agree with. This is the receptor  
 22 for IGF1, insulin-like growth factor 1. It's very  
 23 widely distributed. I was not sure if I would have seen  
 24 a response or not. If I had to choose a side, I would  
 25 have said less likely than likely.  
 26 Q Would the same be true of the next item, IGF1R?  
 27 A Yes.  
 28 Q You agree with Dr. Chase's characterization?  
 29 A His characterization, yes, I'll agree with that.  
 30 Q And then we have IgG1HC. Same question.

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1 A And I agree with what he says.  
 2 Q His response would likely be immune specific, correct?  
 3 A Yes. This is an immunoglobulin. Immunoglobulins are  
 4 antibodies. So, yes, that would be a very important  
 5 immune system response.  
 6 Q I sometimes have been asking you follow-up questions  
 7 about the righthand column on Dr. Chase's spreadsheet.  
 8 I'll just assume the general question includes that also,  
 9 okay?  
 10 A Okay.  
 11 Q If you would also. Thank you. The next one is IgG2HC.  
 12 Same question.  
 13 A Same answer. I agree with what he says.  
 14 Q Then, IgJ.  
 15 A And I agree with what he says about that.  
 16 Q Why did you choose that one in particular?  
 17 A The IgGJ?  
 18 Q IgJ, yes.  
 19 A We wanted to include as many of the immunoglobulins as we  
 20 could. Circulating in blood, there are four major  
 21 immunoglobulins. There's IgG1, IgG2, which are very  
 22 similar, but slightly different. IgG immunoglobulin A,  
 23 and immunoglobulin M, IgM, we didn't get a good probe for  
 24 IgM, unfortunately.  
 25 Q I'm sorry, You say you did not get a good what?  
 26 A Probe. Assay for IgM. Immunoglobulin AG, or not,  
 27 Immunoglobulin G is the dominant immunoglobulin  
 28 circulating in blood. The most common antibody in the  
 29 body, however you total up, is probably not IgG, it's  
 30 actually IgA. This antibody is involved in what we call



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1 mucosal immunity. It's actually secreted in mucosal  
 2 tissue, such as the GI tract, the lining of the lungs and  
 3 the mammary duct. So, it is a very important antibody.  
 4 And this is a piece of that antibody, it's  
 5 sometimes called secretory piece, that is necessary for  
 6 it to be secreted into those foods.  
 7 Q So, what consequences would a decrease in IgJ following  
 8 treatment have for the immune function of a cow?  
 9 A There's a couple of things that could be going on here.  
 10 One is, if the animal is producing less immunoglobulin G,  
 11 then that could result in a lowered mucosal immune  
 12 response. As you imagine, the tissues I mentioned, the  
 13 lining of your lungs is actually outside your body. It  
 14 is exposed, potentially exposed to all kinds of  
 15 pathogens, as is the lining of the GI tract, for  
 16 instance. And this mucosal immunity plays a key role in  
 17 that protection.  
 18 The limitation to this study is, we looked  
 19 at what circulates in the blood, not what's in the  
 20 mucosal secretions. It's also possible that, what's  
 21 happening is, the cells producing the IgA are leaving the  
 22 circulation, even though they are still there producing  
 23 it at a different cycle. Does that make sense?  
 24 Q Well, I think it does. If a change in IgJ is found,  
 25 what, if any, conclusions can be drawn about changes in  
 26 the immune function of the cow?  
 27 A (No response).  
 28 Q I think you kind of answered that, but I am just asking  
 29 you to expound a little more.  
 30 A Yes. I think that I probably answered it in a rather

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1 round about way. In this particular study, we are  
 2 looking at expression of IgJ protein in cells that are  
 3 circulating in the blood. That's where our cells came  
 4 from. So you can make - there's two possibilities here,  
 5 two major possibilities, anyway. One is that the IgJ  
 6 production has actually gone down, which would impair  
 7 immune function. The other is that the cells producing  
 8 the IgJ gene - or IgJ unit, are not circulating. That  
 9 would suggest they have been recruited to somewhere else,  
 10 which would mean a change, but not necessarily an  
 11 inhibition of the mucosal immunity.  
 12 Q So, to summarize, I think what you just told me is that  
 13 decrease in IgJ in the blood can indicate either an  
 14 impairment of immune function if its overall production  
 15 has gone down, or it could simply be indicating that  
 16 there's an immune response going on in the animal  
 17 somewhere?  
 18 A Yes.  
 19 Q Somewhere, taking it out - -  
 20 A Yes, I think I'll agree with that.  
 21 Q Now, in that particular item, and according to the  
 22 specifics or the run that summarizes the spreadsheet, the  
 23 P value arising from the t-test was 8.21, if I understand  
 24 the notation correctly, times ten to the minus fifth, is  
 25 that correct?  
 26 A That's what this says. And I do recall that that was  
 27 different. I didn't - I don't recall - didn't recall the  
 28 exact number. But I do recall that IgJ and IgA were both  
 29 lower in the treatment groups.  
 30 Q And to be a little more specific, 8.21 times 10 to the

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1 minus 5th would be .0000821, is that correct? Four zeros  
 2 and 8121?  
 3 A Four zeros and 821, that's correct.  
 4 Q Four zeros to the right of the decimal point. And that  
 5 is about - well, it's almost a hundred fold less than the  
 6 .05 usually considered as statistically significant,  
 7 correct?  
 8 A I'm not sure you can do that kind of calculation with P  
 9 values, but it is much lower than your typical .05.  
 10 Q And that's just mathematically.  
 11 A Yes. It is - it is - that would be considered a  
 12 significant response. You get no dispute on the  
 13 significance of a P value that small.  
 14 Q It's fairly extraordinary to see a P value that low in  
 15 any experiment, is it not?  
 16 A I don't know. Oftentimes in experiments, we're looking  
 17 for things that haven't been discovered before, so we're  
 18 often looking for things where the response is fairly  
 19 subtle.  
 20 Q But it's certainly one of the larger responses that you  
 21 would see?  
 22 A It's a highly significant statistical.  
 23 Q If you recall, did you or anyone also, to your knowledge,  
 24 do any further statistical analysis related in particular  
 25 to IgJ beyond what is summarized on the spreadsheet?  
 26 A Not to my knowledge.  
 27 Q Would that be of any of these variables on the  
 28 spreadsheet, was any further statistical analysis done  
 29 beyond what's on this spreadsheet, to your knowledge, by  
 30 you or anyone else?

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1 A Not to my knowledge.  
 2 Q With respect to the results of this experiment, the one  
 3 involved in Messenger RNA expressions we've been  
 4 discussing, generally speaking, whose job was it to do  
 5 the statistical work? Was that yours or somebody else's?  
 6 A I did the statistical analysis on this particular study.  
 7 Q And I take it, nobody else did any further analysis of  
 8 any kind that you're aware of?  
 9 A Not that I'm aware of.  
 10 Q Proceeding to the next variable is IgAHC, is that  
 11 correct?  
 12 A That's correct.  
 13 Q And my rudimentary understanding is, HC means heavy  
 14 chain?  
 15 A Heavy chain, correct.  
 16 Q Same question that we were going at some time ago about  
 17 Dr. Chase's summary on exhibit 276?  
 18 A His summary I will agree with.  
 19 Q And again, there we have a P value based on the t-test of  
 20 0.003211, correct?  
 21 A That looks correct.  
 22 Q And with respect to the immunological function of the  
 23 cow, what does that tell us, if anything?  
 24 A The same thing the IgJ would. One actually would expect  
 25 those to change more or less in parallel, although IgJ is  
 26 involved in another antibody immunoglobulin M that it's a  
 27 major part of the IgA molecule. So you might expect  
 28 those to change in about the same way.  
 29 Q And again, the P value for IgAHC is less than .05,  
 30 correct?

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1 A Correct.

2 Q Dr. Sheffield, Mr. Thornton discussed with you last time

3 Bonferroni adjustments or computations and we talked

4 about that generally a little bit, about the controversy

5 and statistics surrounding that sort of adjustment.

6 Did you, in the course of your analysis of

7 the statistics related to this study summarized on 254,

8 consider making adjustments of that general nature, given

9 that you studied many variables?

10 A I didn't do that. I do think it probably would be a

11 reasonable thing to do. But I did not do that.

12 Q And this, I'm going to ask you this question and maybe

13 one that you can't answer on the fly very well, and if

14 so, then so be it. But given that there were quite a few

15 variables here that you did not expect to change when the

16 experiment was designed, how would you apply that sort of

17 adjustments to these circumstances? Could you, either

18 generally or specifically, to the extent you can address

19 that?

20 A That is one of the problems with the Bonferroni. If you

21 have maybe, let's just use as an example, ten

22 measurements that you would expect to change and then ten

23 that you don't expect to change, but maybe they will

24 change, do you do the Bonferroni correction based on the

25 ten or the 20? I can't answer how I would do that. I

26 would probably call a statistician I know and ask him how

27 to do that.

28 Q If I understood the answer or series of answers you gave

29 Mr. Thornton the last time we were together, I take it

30 your experience is that if you call ten statisticians,

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1 you might get ten different answers, is that about right?

2 A I don't know if I would go that far, but you will see

3 discrepancies from time to time about things like, what

4 is the best way to do adjustments for something like

5 this.

6 Q And given that there were, I think it's a little less,

7 but approximately a hundred variables studied in this

8 experiment, and let's assume it's a hundred, that's not

9 quite right, but it's close, okay? If one were to do a

10 very simple Bonferroni adjustment based on 100 variables,

11 the procedure as I understand it is to reduce your P

12 value for significance by dividing the number of

13 variables into .05, is that correct?

14 A It's something like that. I don't know if that's exactly

15 right. I would have to look up the Bonferroni

16 correction. But it's along those lines anyway.

17 Q And with respect to a number like the one for IgJ, which

18 is reported on the spreadsheet as 8.21 times 10 to the

19 minus 5th, if you make that simplistic adjustment, based

20 on 100, you would compare that to a P value for signifi-

21 cance under these assumptions of .05 divided by 100,

22 correct?

23 A If that is correct, that's what you would - let me - say

24 that again.

25 Q Sure. Could you read it back, John? I think I said it

26 right, and I don't think I can say it better. So, I'll

27 have him read it back.

28

29 (The last question was read aloud by

30 the court reporter).

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1

2 A I believe so.

3 Q And .05 divided by 100 would be, I believe, 5 times 10 to

4 the minus 4, is that correct?

5 A If I can do multiplication in my head, that's right.

6 Q Well, .05 is 5 times 10 to the minus 2, correct?

7 A Yes. 10 to the minus 2. That's correct.

8 Q So, even doing what - well, what one could call a full

9 Bonferroni adjustment for all approximately 100

10 variables, the change in IgJ still appears as statistic-

11 ally significant after a fine Bonferroni in that manner,

12 if one were to do that, is that correct?

13 A That seems right at the moment, yes.

14 Q I tell you what, we've been going for an hour and a half.

15 I told Mr. Bird I'd call him and see if we can include

16 him at about this time. Shall we take a short morning

17 break? Perhaps you could use a break, doctor?

18 A Sure.

19

20 (At this time a recess was taken - 10:37 to 10:54.

21

22 Q Dr. Sheffield, we'll get back to the spreadsheet in just

23 a moment, but Mr. Bird on the phone over the break

24 reminded me to ask you, I believe that my office sent you

25 a copy of the transcript of your first deposition in this

26 matter after it was taken. Did you receive that?

27 A I don't recall. I would have to look to see if I did. I

28 might well have.

29 Q I take it you haven't read the transcript then?

30 A I think I would remember if I had actually read it.

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1 Q Sure. Well, even without having read it, can you recall

2 anything today that you said the first time around that,

3 upon reflection, you would like to correct or change?

4 MR. THORNTON: I object. That's way too

5 broad.

6 Q It is broad.

7 A I would want to read it before giving a definitive answer

8 to that. But off the top of my head, I'm not - you're

9 asking me to recall something that was over a month ago,

10 and it might be something - I'd have to read the

11 transcript.

12 Q You are correct on all of those accounts, but if there's

13 anything that came to mind, I just wanted to know about

14 it. Thank you.

15 Going back to the spreadsheet, exhibit 254,

16 and Dr. Chase's summary, exhibit 275, the next item on

17 the list is IL1a, where I think it's frequently referred

18 to as IL1Alpha, is that correct?

19 A Alpha is what it's usually called.

20 Q And it's probably true of all the small subscript A's

21 throughout the spreadsheet, is that correct?

22 A I think that's - at least most of them, yes.

23 Q And do you agree with Dr. Chase's summary in exhibit 276

24 (sic) about the function of IL1a, or alpha?

25 MR. THORNTON: 275?

26 Q 275. Excuse me. You're right.

27 A Yes.

28 Q And the next item is IL1b, or beta, correct?

29 A Correct.

30 Q I think you discussed with counsel the distinction

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1 between them, which is minimal, is that correct?  
 2 A They are different, but there's - they're very, very  
 3 similar.  
 4 Q And in the case of IL1alpha and beta, the P values, as  
 5 calculated and indicated on the spreadsheet, are, in the  
 6 case of alpha, 8.74 times 10 to the minus 6; in the case  
 7 of beta, 2.55 times 10 to the minus 6, is that correct?  
 8 A That is what I'm reading here, yes.  
 9 Q And which are even smaller than the IgJ P value of 8.21  
 10 times 10 to the minus 5th, correct?  
 11 A That is correct, yes.  
 12 Q So, if one were to perform a very simplistic Bonferroni  
 13 adjustment to the P value, as we discussed before, these  
 14 would still be statistically significant if P equals 5  
 15 times 10 to the minus 4, correct?  
 16 A If that correction is the way it's done, that would be  
 17 correct, yes.  
 18 Q I'm not implying that one way or the other. But if one  
 19 were to do it that way, that would still be true?  
 20 A Yes. Correct.  
 21 Q All right. Then, the next item is IL1. Could you tell  
 22 me how that word is pronounced?  
 23 A It's an abbreviation for antagonist, or inhibitor.  
 24 Q But pronounced - -  
 25 A Pronounced antagonist.  
 26 Q Do you agree with Dr. Chase's summary relating to it?  
 27 A Yes.  
 28 Q The next item is IL2, is that correct?  
 29 A Correct.  
 30 Q And do you agree with Dr. Chase's summary with respect to

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1 it?  
 2 A Yes.  
 3 Q The P value there is also - well, it's in the same  
 4 general ballpark as IL1alpha and IL1beta, correct?  
 5 A Looks like these are a little out of register, so I have  
 6 to make sure I'm looking at the right column. That's  
 7 correct.  
 8 Q Specifically, it's 4.98 times 10 to the minus 6, correct?  
 9 A That's correct. Yes.  
 10 Q The next item, could you tell us what it is and whether  
 11 you - just how you say the full name, in other words,  
 12 whether you agree with Dr. Chase?  
 13 A Yes, this is the IL2 receptor. And that is what he  
 14 thought it was, and what he says about it is correct.  
 15 Q Moving on to the right, next is IL3. Same question.  
 16 A And I agree.  
 17 Q Next item is IL4. Same question.  
 18 A And I agree with what he says of that.  
 19 Q Next is IL6. Same question.  
 20 A And I agree.  
 21 Q Next item is IL8. Same question.  
 22 A Looks like IL8, yes.  
 23 Q Oh, I'm sorry, did I say IL6? I'm sorry.  
 24 A No. No. I just said, I was meaning to imply that that  
 25 looks like an accurate description of IL8.  
 26 Q Okay. Thank you.  
 27 MR. THORNTON: You didn't say IL8.  
 28 A I'm sorry.  
 29 Q The next item is IL10. Same question.  
 30 A And I agree with that.

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1 Q If I understand correctly, IL1 is a pro-inflammatory  
 2 cytokine, and IL10 is an anti-inflammatory cytokine. Is  
 3 that correct?  
 4 A That is the sort of the general one word description of  
 5 them. Like a lot of these, they have many functions, but  
 6 that's a fair summary.  
 7 Q And by the measurements you made in the experiment, the  
 8 fold for IL1 alpha and beta were around 1.65 for one, and  
 9 1.78 for the other, correct?  
 10 A Correct.  
 11 Q In other words, the serum levels of those cytokines went  
 12 up in the treatment counts, correct?  
 13 A That's correct. Yes.  
 14 Q And in the case of IL10, the serum levels halved,  
 15 essentially, is that correct?  
 16 A That's about right. Yes. IL10 was lower.  
 17 Q And I think you discussed with Mr. Thornton the last time  
 18 that the immune responses seen in this study were smaller  
 19 than you would typically see in an acute disease  
 20 outbreak of some form in a cow?  
 21 A Yes, you would see if an animal has an acute infection,  
 22 you would see much bigger changes in IL1, for instance.  
 23 Q And perhaps in some of the others?  
 24 A And some of the others as well, yes.  
 25 Q By the way, I don't think that TNFalpha was on your list  
 26 here, is that correct?  
 27 A For some reason, I thought it was.  
 28 Q Maybe I missed it.  
 29 A Yes, it is.  
 30 Q Okay. Where is it?

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1 A It's on the spreadsheet on page 4.  
 2 MR. THORNTON: Chase's deal, it's - -  
 3 A About a third of the way in. And - -  
 4 MR. THORNTON: Page 11 on 275.  
 5 Q Okay. Very good.  
 6 A Yes, it is - it is on this.  
 7 Q Then we will get to that. I'd forgotten. I'm sorry. Is  
 8 there a concept in biology called cytokine induced  
 9 symptoms behavior?  
 10 A Probably. I'm not all that familiar with that specific  
 11 term.  
 12 MR. THORNTON: Objection. Foundation.  
 13 Q And I take it you either haven't reviewed, or if you  
 14 have, you don't recall much of the specifics of any  
 15 literature about that subject?  
 16 A I don't recall reviewing any literature on that -  
 17 anything that was called that.  
 18 MR. CARLSON: Let me just clarify. Are you  
 19 referring to the cytokine storm as well? Is that the  
 20 same thing, called it a cytokine storm?  
 21 Q I guess I wouldn't choose to say they mean the same thing  
 22 or not. I don't know.  
 23 A I am familiar with the cytokine storm. But I haven't  
 24 kept up with it very much, but I do know something of  
 25 that. And I was assuming you meant something different  
 26 with this.  
 27 Q What is a cytokine storm, in your understanding?  
 28 A During inflammation, you get a - frequently get a massive  
 29 release of a variety of pro-inflammatory cytokines. The  
 30 cytokines are actually important in inducing responses to

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1 an infection, protective responses, but they can also  
 2 damage normal tissue. And in many cases, it's the  
 3 production of massive amounts of these inflammatory  
 4 cytokines, they cause some of the deleterious effects  
 5 during infection and inflammatory responses.  
 6 Q All right. And if things are going well with the body,  
 7 and with this massive cytokine release, one would hope  
 8 that the inflammation serves its purpose and passes  
 9 relatively quickly, is that correct?  
 10 A That's what one would hope.  
 11 Q Doesn't always happen that way though?  
 12 A No, it doesn't.  
 13 Q Are you familiar with any studies in any type of organism  
 14 of the impact, if any, of a long-term elevation of  
 15 cytokines at the levels of approximately two-fold or a  
 16 little less than we're seeing in this study? In other  
 17 words, I'm asking if there's been anything studied about  
 18 --  
 19 A There has been.  
 20 Q -- the consequences of that is a chronic process rather  
 21 than an acute process?  
 22 A There have been.  
 23 Q Can you describe what you know about that, in general,  
 24 please?  
 25 A This is not an area I have reviewed recently.  
 26 MR. THORNTON: Objection. Speculation. No  
 27 foundation.  
 28 Q Please continue.  
 29 A So, I was familiar with this some years ago. I haven't  
 30 looked at the most recent literature, except in

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1 preparation for teaching, which is a little bit lower  
 2 level than what one would do if I were going to be doing  
 3 research on this area.  
 4 Q Do you recall who --I'm sorry. Go ahead.  
 5 A What I was going to refer to is, two points. Certain  
 6 diseases, cardiovascular diseases, are felt to be caused  
 7 by, or related to - maybe caused is not the right word -  
 8 related to low level chronic inflammations. Some of  
 9 these, not the interleukins, the TMFAlpha that you  
 10 alluded to earlier, for example, is produced in adipose  
 11 tissue. And this is actually where my familiarity years  
 12 ago with this came from, that it's thought that some of  
 13 the adverse effects of obesity on things like cardio-  
 14 vascular health might be mediated by this long-term  
 15 sub-acute inflammation, in other words, TMFI is usually  
 16 implicated in that, in my understanding of that, rather  
 17 than the interleukins, I don't know about the IL1 and its  
 18 implication in that.  
 19 Q All right. Do you recall who any of the folks who you  
 20 would consider to be the leading researchers are or were  
 21 in that area?  
 22 A Not off the top of my head. If I were going to look at  
 23 that again, it would be fairly easy to find it on  
 24 health.ed. But I don't recall the names, off the top of  
 25 my head.  
 26 Q What would you search for on health-ed? What kind of key  
 27 words?  
 28 A Probably --  
 29 MR. THORNTON: Objection. Speculation.  
 30 A The word that's often used is the adipokine.

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1 Q Can you spell that, please.  
 2 A A-d-i-p-o-k-i-n-e. Or adipokine hypothesis, which is  
 3 this idea that adipose tissue produces hormones, and that  
 4 is considered a subset of that hypothesis. So, you could  
 5 look for words like TNFAlpha and adipose tissue.  
 6 Q Now back to IL10 for a moment. I think you've answered  
 7 my basic question about that. The P value that is  
 8 indicated in your summary is 2.93 times 10 to the minus  
 9 5th, correct?  
 10 A Correct.  
 11 Q And again, just grossly, if you were to make that very  
 12 simplistic Bonferroni adjustment that we went through  
 13 earlier, not saying it's right or wrong, that number is  
 14 still less than 5 times 10 to the minus 4, and then this  
 15 item would be - would still be considered statistically  
 16 significant, even if one made such a Bonferroni adjust-  
 17 ment, correct?  
 18 A That looks right, yes.  
 19 Q Next item on the spreadsheet is IL12Alpha, I believe, is  
 20 that correct?  
 21 A Correct.  
 22 Q Put the same basic question relating to 275, Dr. Chase's  
 23 summary.  
 24 A I'll agree with that.  
 25 Q Next item is IL13. Same question.  
 26 A I will agree with that.  
 27 Q Next one is IL15. Same question.  
 28 A Yes, I'll agree with that.  
 29 Q Then, IL16. Same question.  
 30 A And I'll agree with that.

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1 Q Next item is labeled INTb1. What's the full name of that  
 2 item, and then do you agree with Dr. Chase or not?  
 3 A The full name is interferon, i-n-t-e-r-f-e-r-o-n, beta 1.  
 4 And I'll agree with what is said there.  
 5 Q The next item is Int2? Could you give us the full name  
 6 and whether you agree with the summary on 275?  
 7 A That is interferon 2, and I will agree with that.  
 8 Q Next item, which is the lefthand column on page 3 of  
 9 exhibit 254, could you pronounce the full name for us and  
 10 answer the same basic question.  
 11 A The full name is lactose peroxidase. Do you want me to  
 12 spell that?  
 13 THE REPORTER: I can look it up.  
 14 Q It's in the exhibit, so I think we're fine.  
 15 A And let me find it again.  
 16 MR. THORNTON: On Page 6.  
 17 A Yes. I'm just looking to make sure. He's asking me if I  
 18 agree, and I wanted to make sure I know what I'm agreeing  
 19 to.  
 20 Q Please.  
 21 A Yes, I'll agree with that.  
 22 Q Next item is Leptin. Same question.  
 23 A Yes, I'll agree with that.  
 24 Q Next item is mmp1, and is spelled out in full in exhibit  
 25 275, so the same basic question.  
 26 A Yes, I'll agree with that.  
 27 Q Next item is mmp3. Same question.  
 28 A I'll agree with that one.  
 29 Q And we have mmp9. Same question.  
 30 A And I'll agree with that.

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1 Q Next item is PLC. Same question.  
 2 A I'll agree with that.  
 3 Q The next item is PLCa, and in Dr. Chase's summary, he  
 4 says, "Not sure what this is." Can you expound on that  
 5 one in some length, please?  
 6 A PLCa stands for phospholipase C alpha. And it's one of  
 7 many forms of phospholipase C, which is an enzyme that is  
 8 involved in a wide variety of cell signaling pathways, in  
 9 many cells in the body, not specific to the immune  
 10 system.  
 11 Q And so in terms of whether a change would probably relate  
 12 to immunological function, that would be a no - it could,  
 13 but not likely?  
 14 A It could, but it's the sort of enzyme that you might  
 15 expect activity to change more than expression.  
 16 Q Next item is PGDSynth, S-y-n-t-h, or Prostaglandin D  
 17 synthase, is that correct?  
 18 A That's correct.  
 19 Q Do you agree with Dr. Chase's summary there?  
 20 A Yes.  
 21 Q Next item is labeled PGSH2, and Dr. Chase asks us to  
 22 double check with you as to just what this is. Could you  
 23 tell us, please?  
 24 A Okay. It stands for Prostaglandin synthase H2. It's an  
 25 enzyme involved in Prostaglandin synthase, much like the  
 26 PGD synthase is. These enzymes go by a wide variety of  
 27 names. An alternative name for this one is  
 28 Cyclooxygenase, or COX-2.  
 29 Q Is that the item we had - is that one of those items that  
 30 one of the drugs for humans that was inhibiting that was

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1 pulled off the market a while back?  
 2 A Exactly.  
 3 Q All right. Thank you. At least I got that right. And  
 4 is this an item, a change in which you would expect  
 5 ordinarily to relate to immunological function?  
 6 A Yes. It's involved in inflammation.  
 7 Q And it's pretty specific to inflammation?  
 8 A There are other places and times when it does occur, but  
 9 it is not exclusively, but it is very often associated  
 10 with inflammation.  
 11 Q Next item is pim1, and the same basic question about Dr.  
 12 Chase's comments on 275.  
 13 A I would agree with those assessments.  
 14 MR. THORNTON: Excuse me. You didn't ask  
 15 him specifically whether he agreed or disagreed with  
 16 Chase's assumptions on PGSH2.  
 17 A He didn't make any assumptions on PGSH2 because he wasn't  
 18 sure what it was.  
 19 MR. THORNTON: Well, but there's a  
 20 description here.  
 21 A That is actually referring to the PGD, which is also  
 22 called COX-1, is the way I interpreted that. I may be  
 23 misinterpreting it.  
 24 MR. THORNTON: I don't think so. Well.  
 25 It's unclear.  
 26 Q Well, Dr. Chase makes no summary about COX-2, which is  
 27 what the PGSH2 is, or another name for it.  
 28 A I think I want to make sure this is clarified here.  
 29 Q Please.  
 30 A Or should. COX-1 is the same thing as PGDSynthase. So,

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1 what is written beside the PGSH2, referring to COX-1, I  
 2 will agree with, if we assume that that's referring to  
 3 the PGDSynthase, or COX-1. The PGSH2 is also called  
 4 COX-2. Its roles are fairly similar to COX-1, it's  
 5 involved in synthases across the glandins, and it has an  
 6 immune specific effect in inflammation. It does have  
 7 some other roles, but its main role is mediating  
 8 inflammation.  
 9 Q That's why I didn't ask, because I don't think it's  
 10 clear. There is a comment there.  
 11 MR. THORNTON: The comment is sort of  
 12 screwed up. Usually, when you've got the wikipedia  
 13 reference, that's the end of the comment. But the  
 14 comment for PCGDSynth appears to go down all the way to  
 15 pim1 1, and there's two wikipedia references.  
 16 A Without having the Web site here, I don't know what that  
 17 wikipedia reference says.  
 18 Q Sure. And that's why I didn't ask the question. It is  
 19 not clear what that is referred to, and Dr. Chase,  
 20 obviously, was having trouble identifying what PGSH2 was,  
 21 which is why he asked to double-check it with Dr.  
 22 Sheffield. Okay. Yeah, I think you answered the pim1.  
 23 The next item is PKAbCAT.  
 24 A Okay. These next few that start with PKA are various  
 25 sub-units and forms of an enzyme called protein kinase A,  
 26 and his description I will agree with on this.  
 27 Q I think on these, on all of these except maybe PKAlpha,  
 28 you indicated you really didn't expect to see a change in  
 29 any of those, is that right?  
 30 A I wouldn't have expected changes.

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1 Q Would that include PKAlpha?  
 2 A That is a rather different enzyme. And that is another  
 3 one that one doesn't always see changes in expression  
 4 level, but often sees changes in activity level.  
 5 Q And by expression level, you mean the expression that  
 6 would show up in MRNA?  
 7 A Correct. Correct.  
 8 Q Okay. After the PKs, the various PKs, the next item is  
 9 PRASG. Again, that's one you didn't expect a change,  
 10 correct?  
 11 A Correct.  
 12 Q The same with the next one, RhoGDI, correct?  
 13 A Yes.  
 14 Q Then we have a series of five or six STATS, is that  
 15 correct?  
 16 A That's correct.  
 17 Q And, generally speaking, maybe it's fair to ask these  
 18 together, maybe we need to take them one at a time, but I  
 19 suspect you're going to agree with Dr. Chase that changes  
 20 in all of those would tend to be associated with immune  
 21 function, is that correct?  
 22 A Let me just look through the list to make absolutely  
 23 sure.  
 24 Q Sure.  
 25 A Yes.  
 26 Q Proceeding on to page 4 of exhibit 254, and which is near  
 27 top of page 10 on exhibit 275, the next item is TEK, and  
 28 I think you described that one as interesting before.  
 29 A Yes.  
 30 Q And I guess I'll ask you to respond to the standard

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1 question, do you agree with Dr. Chase's summary?  
 2 A What he says is correct. I would add that the statement  
 3 is expressed almost exclusively, and that is correct.  
 4 But he almost doesn't indicate that there are a few other  
 5 places it can get expressed.  
 6 Q Such as?  
 7 A There are some, as I discussed earlier, some, a few  
 8 studies that suggest it might be expressed in leukocytes  
 9 that have roles in them.  
 10 Q Any particular type of leukocytes?  
 11 A I'm not recalling that off the top of my head for that  
 12 one. I would have to look that up.  
 13 Q And do you agree with Dr. Chase, that changes in PK would  
 14 not be particularly immune specific?  
 15 A No, they wouldn't.  
 16 Q Next item is TGFb1. Same question.  
 17 A I agree with what he says about that.  
 18 Q Next is TGFb1 as it's shown on my spreadsheet, which is  
 19 different on 275.  
 20 MR. CARLSON: Looks like EB on the  
 21 spreadsheet.  
 22 Q I guess, let me ask you more broadly then, Dr. Sheffield.  
 23 Can you tell us what that is?  
 24 A I believe that refers to a transforming growth factor  
 25 binding protein, not P2. Now, what he says, it is the  
 26 same, TGFB2, that's the same function as B1, that is  
 27 correct. But there is a trans - for many factors like  
 28 this, there are proteins that are called binding proteins  
 29 that are secreted, bind to the growth factor, and  
 30 modified its function. Sometimes they stimulate its

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1 function, sometimes they inhibit it.  
 2 These are best known for insulin-like  
 3 growth factors, but they exist for other things as well.  
 4 And I am almost positive that this is referring to trans-  
 5 forming growth factor binding protein, not the B2  
 6 protein.  
 7 Q Would a change in that item tend to be indicative of - -  
 8 A That's a possibility.  
 9 Q Let me finish the question. Would such a change tend to  
 10 reflect an immune system specific effect?  
 11 A It's not specific. These things would be present in a  
 12 lot of places in the body, just like TGFb1 would be. But  
 13 it is important in the immune system. It's one of these  
 14 things that covers a lot of ground. TGFTransforming  
 15 growth factor proteins are involved in a huge variety of  
 16 processes. They're involved in the immune system, but  
 17 they're involved in things like limb pattern formation in  
 18 embryonic development, to give you an example of how  
 19 involved their actions are.  
 20 Q The next item is - well, let me ask you, tell me what the  
 21 spreadsheet says. It appears to be Tie1 or maybe Roman 1  
 22 or I, I'm not sure what it says, frankly.  
 23 A Yes. This is Tie 1 and 2.  
 24 Q All right. Dr. Chase does comment on those. Standard  
 25 question.  
 26 A Let me look at his comment a little more carefully.  
 27 MR. THORNTON: He's got a different - -  
 28 A He has it listed as TiaI. And I want to make sure if he  
 29 is referring to the same thing that I am here. I'm not  
 30 sure he is referring to the same thing that I am

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1 thinking.  
 2 Q Okay. Could you please tell me what your item is on  
 3 exhibit 254, please?  
 4 A What I believe those are, are two very closely related  
 5 proteins that are involved in cell death processes. I'm  
 6 not sure if they are the same as Tia that he is stating  
 7 here, but they are involved in what is called program  
 8 cell death.  
 9 Q And would a change in either of those tend to be some-  
 10 thing that would be immune system specific effect?  
 11 A It would not be specific to the immune system. These are  
 12 fairly - they're more often associated with the immune  
 13 system's, if I recall, distribution, but they are found  
 14 in other places as well. But you would expect to - a  
 15 change in them, in the immune system cells, might be seen  
 16 in alterations of the immune function. They could be  
 17 seen in other things as well.  
 18 Q The next item is TIMP3. Same question. Standard  
 19 question, I should say. Bottom of page 10 on 275.  
 20 A Yes, I'll agree with that.  
 21 Q Then we have TNFalpha or tumor necrosis factor alpha.  
 22 Standard question.  
 23 A Yes, and I'll agree with his assessment there.  
 24 Q Next item is TNFRec, tumor necrosis factor receptor, is  
 25 that correct?  
 26 A That's correct.  
 27 Q Same question.  
 28 A Yes, I'll agree with that.  
 29 Q Next item is TPA. Same question.  
 30 A I'll agree with that.

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1 Q Next item is Urokinase. Same question.  
 2 A I'll agree with that.  
 3 Q Then we have IL1Rec or interleukin 1 receptor. Same  
 4 question.  
 5 A I'll agree with that.  
 6 Q Then we have a series of variables that start with PK, I  
 7 guess, I believe it's the next six. I'll ask you the  
 8 same question for each. If you prefer to deal with them  
 9 collectively, that's fine.  
 10 A I would make one correction, and that is, the one that  
 11 says PKARI, Roman numeral I, alpha, that one is not an  
 12 isomer of PKC, it's an isomer to PKA.  
 13 Q I see. With respect to those six variables, do you agree  
 14 that those that Dr. Chase has in the column on immune  
 15 system specific effect?  
 16 A Yes. These are the sorts of enzymes you find in most  
 17 cells of the body in varying amounts.  
 18 Q Then we have INFalpha. What is the full name of that  
 19 item?  
 20 A Interferon alpha.  
 21 Q And same question, the standard question.  
 22 A I'll agree with his statement.  
 23 Q Then we have those last four items, which I think we have  
 24 already covered in detail when we got started?  
 25 A Correct.  
 26 Q I see, for example, with Casein, Dr. Chase says, "Good  
 27 control," like that of some of those others. Okay.  
 28 MR. THORNTON: Before you leave that, could  
 29 you explain what Empty is?  
 30 A Yes. We didn't put anything there. There's no DNA probe

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1 at all. So you'd expect - that's not background. You'd  
 2 expect it to be close to zero. That's sort of a 'no  
 3 signal at all' control.  
 4 Q We still get a number, but they're much, much smaller?  
 5 A Yeah. You will always - you will always get a - like if  
 6 you're trying to detect for radiation, there's always a  
 7 background radiation that you have to correct for, and  
 8 that's what that means.  
 9 Q All it means is, there's something in that well  
 10 generating the light that the lab machine is detecting?  
 11 A Right. Right. It's another of those controls for how  
 12 specific things are.  
 13 Q Oh, before I forget this one, you didn't review the  
 14 transcript, but there's, I think two - approximately two  
 15 occasions in the transcript of your first deposition, you  
 16 were describing that machine detecting the light signals,  
 17 and the area of the intensity and so forth, where Mr.  
 18 Kirby got the word protons, and I think you meant to say  
 19 or said - I think you said photons?  
 20 A Photon is what it should have been.  
 21 Q Resisting the urge to ask you whether light is a wave or  
 22 a particle.  
 23 A Yes.  
 24 Q We will pass on that one. Oh, by the way, Dr. Chase got  
 25 his PhD in veterinary science, veterinary medicine here  
 26 at Madison in 1990. Do you recall knowing him at all?  
 27 A I do not recall knowing him. I may know the person he  
 28 studied with. But I don't recall that name.  
 29 Q You never had any professional interaction with him, I  
 30 take it?

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1 A I don't recall any. I may have met him at a seminar or  
 2 something at some point, but I don't remember.  
 3 Q Sure. For example, I happened to attend a seminar given  
 4 by him most of the day in Wisconsin Veterinary Medical  
 5 Association in the fall of 2009 over - it was over  
 6 farming methods and vaccinating dairy cows and that sort  
 7 of thing. He did that commonly. Have you ever heard of  
 8 one of those?  
 9 A I know that they're there, but I have never been to one.  
 10 Q In with the materials that were provided by the  
 11 University back in 2008, and following in late 2007,  
 12 there are a number of memos, either from you or to you,  
 13 usually between yourself and Steven LeMire, the gentleman  
 14 who is doing the statistics on that first study, about  
 15 various alternative information and alternative  
 16 statistical tests, and I'll get some of them out and talk  
 17 about them here in a moment. But do you recall anything  
 18 about your asking for alternative statistical analyses of  
 19 the data from the first study, the one that was submitted  
 20 to the Minnesota State Government, after the study was  
 21 published in the year 1999?  
 22 A That was a long time ago. I don't recall anything  
 23 specific. I think we did have some discussions about how  
 24 to analyze the data. And I don't recall the time-frame  
 25 of any of those, whether that was before or after various  
 26 events occurred.  
 27 Q Well, I would like to show you some of those documents  
 28 and talk to you about them a bit. I understand it was a  
 29 long time ago, and they're not - fortunately, they're not  
 30 lengthy documents, though some of them have tables and

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1 numbers and that kind of thing which are in a lot of  
 2 information. So, in doing so, I will certainly try to  
 3 give you plenty of time to look at them. In fact, if you  
 4 would like, I could stack them up and we can take a lunch  
 5 break and you can take a look at them while we're on  
 6 break and take them up afterwards, and I'll provide those  
 7 to counsel, too. Does that sound reasonable?  
 8 A (No response).  
 9 Q It might go a little quicker than if I simply hand them  
 10 to you and give you the opportunity to read them and  
 11 spawn through them. But if you've got - -  
 12 A It's up to you. It depends how long they are and how  
 13 many there are. Might be able to respond quickly or  
 14 might need to look at them.  
 15 Q Well, let's give the first one a try.  
 16 A Why don't we try that.  
 17 Q Sure. About 8 or 9 of them, I think, total. Actually, I  
 18 have one here and the others are back here. I think I'll  
 19 try to do them in chronological order, that might be  
 20 easier. The first one doesn't involve you, I don't  
 21 think, but start with that.  
 22 Exhibit 278 is a series of documents that  
 23 were produced by Professor Reinemann in response to the  
 24 subpoena, and appears to be a memo from Steven LeMire to  
 25 Doug Reinemann, dated June 28, '99, just a couple days  
 26 before the first paper was sent to the Minnesota State  
 27 Government.  
 28 Do you recall ever seeing this document  
 29 before, particularly the cover sheet?  
 30 A I don't recall seeing it. I recall seeing the informa-

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1 tion that's in it, but I don't recall seeing the  
 2 particular document.  
 3 Q On it are - there is a - obviously a portion of it is  
 4 typewritten and then there's a whole bunch of hand  
 5 written notations. Do you know whose handwriting is on  
 6 that document?  
 7 A No, I do not.  
 8 Q And - -  
 9 A It does not appear to be mine, I can tell you that, but I  
 10 have no idea whose it is.  
 11 Q As an example, Table 1 on the cover sheet, for the  
 12 variable interleukin 1, micrograms per milliliter. We  
 13 have a P value shown for serum of 0.071, the typewritten  
 14 number, that is, over in the righthand column. Does that  
 15 appear to be correct?  
 16 A That's what I see here, yes.  
 17 Q And up at the top of Table 1, it says, "Table 1. Blood  
 18 file names and different responses in natural logs.  
 19 Sample size is 12 per group." Did I read that correctly?  
 20 A Yes.  
 21 Q And we were talking about a blocked experimental design  
 22 with multiple replicas the last time we got together?  
 23 A Yes.  
 24 Q Does it appear from this table that Mr. LeMire is  
 25 analyzing the treatments and controls as the treatment  
 26 group of 12 and control group of 12?  
 27 A I can't tell for certain from - just from this table. I  
 28 don't see any indication on here of any blocked  
 29 correction.  
 30 Q I guess we have to run the numbers to really know that,

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1 is that correct?  
 2 A Or get a printout of where those numbers came from, the  
 3 program that was used to do it.  
 4 Q It appears that the following pages, which were all  
 5 pretty much sequential in Professor Reinemann's materials  
 6 relate to the SAS, S-A-S, program that was used for  
 7 analysis. Is it possible that by examining those pages  
 8 we could tell?  
 9 A Assuming those relate to this table, it appears that you  
 10 might be able to.  
 11 Q Could you take a look and tell us whether you can tell  
 12 whether a block design is accounted for in this analysis  
 13 or not?  
 14 A I do not see anything that would suggest it is. It looks  
 15 like it was not.  
 16 Q And, for example, I'll refer to Bates number pages down  
 17 in the lower righthand corner where it says Reinemann  
 18 with a number, numbers in the 2600 plus range. For  
 19 example, on page 2656, the last variable on that page is  
 20 iL1 serum, apparently, is that correct?  
 21 A That's what that would mean.  
 22 Q And it appears that the input to the SAS program is 12  
 23 controls and 12 treatments, correct?  
 24 A Correct.  
 25 Q And if this were a blocker, a replica analysis we would  
 26 see something a little different, is that correct?  
 27 A Not necessarily. Let me put where this is, what this is.  
 28 This is simply a calculation - I believe this is simply a  
 29 calculation of the means, not the actual analysis, not an  
 30 actual analysis of variance. If you've done an analysis

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1 of variance, it would look different than that.  
 2 So, let me look at the means. I believe  
 3 that is the printout. Yes, that is a simple P test.  
 4 That does not have blocks in it and you would see some-  
 5 thing different if it did.  
 6 Q So, I take it, there's no indication in these documents  
 7 that a blocked analysis was performed?  
 8 A That's correct.  
 9 Q And you told us the last time, that you were not involved  
 10 at all in the statistical analysis of the data from this  
 11 first study, correct?  
 12 A Other than reviewing what was done and sometimes  
 13 discussing what might be done next or in addition with  
 14 Steve, I didn't do any of the actual computations.  
 15 Q The number, the typewritten number for iL1 serum on  
 16 exhibit 278 of .071, is, I believe, the same number that  
 17 appears for the P value for iL1 serum in the final  
 18 publication, which we marked as exhibit 250 the last  
 19 time. We can check that. Exhibit 250, page 9, Table 2,  
 20 shows a P value for iL1 serum of 0.071, the same number  
 21 that is on exhibit 278, correct?  
 22 A Yes.  
 23 Q Do you have any understanding of why an analysis of the  
 24 experiment accounting for the three different blocks that  
 25 was performed and was not done in this data?  
 26 A If you're referring to this table only (278), I don't. I  
 27 don't know why Steve decided to run the initial analysis  
 28 like this.  
 29 Q I think you told us the last time that it would be  
 30 appropriate to do the block analysis where the study was

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1 done in block, correct?  
 2 A There Actually - it could be. There's another caveat in  
 3 there, but I'm sure we will get to that in a bit.  
 4 Q All right. Well, this is as good a time as any. What is  
 5 that caveat?  
 6 A Well, if I recall correctly, one of the analyses that  
 7 were done was to use the initial values before treatment  
 8 was applied as a covariant, and do an analysis of  
 9 co-variants. That controls for a lot of the block to  
 10 block variability, although possibly not all of it. And  
 11 so it still might be appropriate to run a block, include  
 12 a block of that in that analysis.  
 13 Q Do you know if Steve LeMire did, in fact, do a covariant  
 14 analysis on this data?  
 15 A I believe so.  
 16 Q Again, not something you've ever run the numbers on?  
 17 A I haven't.  
 18 Q Do you have any understanding one way or the other as to  
 19 whether the numbers published in the report that was  
 20 sent to Minnesota, which is exhibit 250 here, utilized a  
 21 covariant analysis? Feel free to look at this document,  
 22 if it helps.  
 23 A That may help.  
 24 Q Sure.  
 25 A It appears it was not.  
 26 Q And you are referring in particular to the paragraph  
 27 labeled Immune Function Responses at the bottom of page  
 28 8, just before Table 2?  
 29 A Yes. What it appears was done here in generating this  
 30 table was to take the difference from base line level -

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1 yes, this is a - as I understand this analysis, and since  
 2 I didn't do it, I could be mistaken, is that what Steve  
 3 did was take the control group and look at it before and  
 4 after change, and then do the same thing with the  
 5 treatment group; and then look at the difference between  
 6 those changes and do a t-test on the difference between  
 7 those changes.  
 8 Q And apparently the t-test was run on all 12 control cows  
 9 and all 12 treatment cows as one control group and one  
 10 treatment group, correct?  
 11 A Correct.  
 12 Q In these circumstances, would that be the most appro-  
 13 priate statistical test or would the block analysis be  
 14 more appropriate?  
 15 A I am not a statistician. In my opinion, as a  
 16 non-statistician, this is not the best way to analyze  
 17 these data, and some kind of analysis or variance which  
 18 might include block effects would be more appropriate.  
 19 Q And we discussed that a little bit the last time around,  
 20 in general. And I take it, that's a subject you've never  
 21 had occasion to discuss with Steve LeMire or Douglas  
 22 Reinemann, is that correct, in relation to this study?  
 23 A I don't recall those in any discussion like that.  
 24 Q It's about the lunch hour. I'm going to mark another  
 25 series of 8 or 9 memos, and you're welcome to have copies  
 26 of them before lunch, including you, Professor Sheffield.  
 27 I'll ask you about those the first thing after lunch,  
 28 okay?  
 29 A Okay.  
 30 Q Why don't we go off the record and we'll mark those



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1 items.  
2  
3 (At this time the noon recess was taken - 11:59 - 1:03).  
4  
5 Q Just briefly, Dr. Sheffield, before I get back to those  
6 memos. In the first study, the one, the output of which  
7 went to Minnesota State Government, did the workers in  
8 the barn, who were with the cows daily, keep notes in  
9 that study, do you recall?  
10 A I don't recall any.  
11 Q There are what appear to be some barn notes in the  
12 records.  
13 A That could be. I just don't recall what they were or  
14 seeing them.  
15 Q We will get back to that, perhaps. The memos in front of  
16 us, we have covered, I believe, 278, which is the  
17 analysis with the handwriting. Exhibit 279, and I  
18 believe - excuse me. Exhibit 279 has a cover sheet upon  
19 which it says Analysis of Part III requested by Dr. Lewis  
20 Sheffield, 10/6 of '99. Did you have some opportunity to  
21 look at this over the lunch hour?  
22 A Yes, I have.  
23 Q First of all, do you think that's accurate that you were  
24 requesting some further analysis?  
25 A I probably did. I don't recall how detailed I would have  
26 requested it.  
27 Q Do you recall why you were requesting it?  
28 A Yes. Basically, I had some questions about whether the  
29 way it would have been analyzed in this initial 278,  
30 whether that was the best way of analyzing the data.

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1 Q And there are a number of memos relating to further  
2 analysis of the data that followed that, which we have  
3 marked as exhibits. Have you had a chance to look at all  
4 of those exhibits?  
5 A I glanced at them, and we can go through those.  
6 Q Okay. On the cover sheet of 279, in someone's  
7 handwriting, it appears to be written, "See iga serum."  
8 Is that your handwriting or somebody else's?  
9 A I don't think it's mine. It doesn't look like mine. But  
10 someone did write that on there.  
11 Q Okay. All right. And tell me what it was you were  
12 requesting upon this occasion as reflected in 279, and  
13 what the conclusions are from the re-analysis, if any?  
14 A Well, this analysis, these two methods of analyzing the  
15 data, one is called general linear models, which is a  
16 fairly standard analysis. The problem with the general  
17 linear models analysis is, it assumes that your  
18 observations are all independent of each other. Or how  
19 one should analyze data that, where that isn't true, for  
20 example, when you take one animal and measure it  
21 sequentially as a very long history in statistics, it's  
22 not all that simple to figure out how to correctly  
23 analyze that, because the methods that ignore the  
24 potential for correlated error terms, if the errors are  
25 correlated can give erroneous results.  
26 I don't recall the history of this, but the  
27 SAS, Statistical Analysis System, released sometime in  
28 the 1990s, and I don't recall when, a method called Proc,  
29 P-r-o-c, mixed. I, at the time, was not very familiar  
30 with that, I'm still not very familiar with the ins and

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1 outs of how Proc Mix works.  
2 MR. THORNTON: Objection. Foundation.  
3 Q Go ahead, doctor.  
4 A But the procedure, as I understand it, accounts for the  
5 correlation that could exist from one animal to another,  
6 - or not from one animal to another, but one sample to  
7 another within the same animal. How it does that, I  
8 don't know.  
9 So this was analyzed using Proc Mixed, and  
10 Proc GLM, taking into account the effects that individual  
11 cows have on the results.  
12 Q Are the results of that analysis tabulated anywhere  
13 within the document?  
14 A Yes. In fact, most of the document is the results of  
15 that. For example, page 15 begins with The Models, is  
16 the title on it. The first variable analyzed here is  
17 called CHEM, which stands for chemiluminescence. The  
18 first analysis here is the mixed procedure, mixed model  
19 for chemiluminescence. The results of this of the SAS  
20 output is shown here.  
21 Q Now on page 16?  
22 A Page 15, and continuing to page 16. At the top of page  
23 16 you'll see a table that says, Tests of Fixed Effects.  
24 The mixed, in Proc Mixed, refers to what we call a mixed  
25 model. In statistics, we think of - often think of  
26 things as either being fixed effects or random effects.  
27 Fixed effects are things you decided on, is the easiest  
28 way to describe it, like which treatment you apply. We  
29 decided the treatments.  
30 Random are things that are selected at

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1 random, like the cows were selected at random, they were  
2 randomly assigned to the treatment, so the cows would be  
3 a random effect.  
4 We usually, in these types of experiments,  
5 are interested in the fixed effects. The random effects  
6 are things you want to control for. And controlling for  
7 them can reduce error variance, but we're not really  
8 interested in comparing this cow with this cow, we are  
9 interested in comparing the two treatments.  
10 In this table you will see - -  
11 MR. THORNTON: You're at the top of page  
12 16?  
13 A The top of page 16. You'll see source, degrees of  
14 freedom, type 2, which stands for type 2 sums of squares,  
15 and then the statistical test, which is Pr greater than  
16 F, that is the P value for treatment effect, day effect,  
17 and treatment by date interaction.  
18 Q So, in this case the treatment effect by this computation  
19 came in with a P value of 0.07?  
20 A That's correct. Well, 0.08 - .7958.  
21 Q Oh.  
22 MR. THORNTON: It's zero point, not zero  
23 point zero.  
24 A Correct. Zero point seven-nine-five-eight.  
25 Q Okay. I'm sorry. I misunderstood. I was getting some  
26 post-it notes when you were giving part of that  
27 explanation. I apologize. Let's just run through the  
28 columns again to make sure I've got it straight. I  
29 apologize.  
30 The F means what?

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1 A I'm not sure.  
 2 Q DDF, degrees of freedom?  
 3 A NDF would be degrees of freedom. Yes, because there's  
 4 one treatment, 5 day effects, 5 treatment by day. DDF,  
 5 I'm not sure what that stands for.  
 6 Q Okay. The third column is what?  
 7 A That is the sums of squares, from an analysis of errors.  
 8 SAS uses something called a type 3 sums of squares. And  
 9 the final column is the P value.  
 10 Q Do you see a low P value in the day row?  
 11 A Day row.  
 12 Q What's the significance, if any, of that?  
 13 A It fluctuates from day-to-day, and I don't know what the  
 14 significance of that would be.  
 15 Q Then I take it there are similar analyses for the other  
 16 variables?  
 17 A Correct. It follows with the glm model for chemilumi-  
 18 nescence, which is a different analysis. And as we  
 19 follow through this, on 15, we have the beginning of the  
 20 analysis of variance table on 16. And then on 17, that  
 21 continues.  
 22 About the middle of page 17, it appears to  
 23 be expecting mean squares. Least square means for the  
 24 two treatment variables. I'm looking through here to  
 25 find a least squares means by date for these. Here's  
 26 what I was looking for. At the top of page 17, we have  
 27 the analysis, the variance table giving treatment, cow  
 28 within treatment, effect, the effect of the individual  
 29 cows, the day effect, and a treatment by day interaction.  
 30 And again, in the final column you have the P values for

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1 these.  
 2 Q And again, the P value for treatment in this case is  
 3 0.7958, which I think is exactly the same as was noted in  
 4 the prior table we spoke of a moment ago?  
 5 A Correct. If you notice, the treatment effect here, --  
 6 MR. THORNTON: Where are you. Top of page  
 7 17?  
 8 A I'm on the top of page 17. We have a treatment effect,  
 9 and there's a table here in which it shows that P value  
 10 as .5066. Now this is something about statistics  
 11 programs. When you do an analysis of variance, every-  
 12 thing you include in the model is a model effects.  
 13 Everything that's left over is assumed to be your error.  
 14 That's not always correct. That's where  
 15 this expected means squares table comes in. What we  
 16 really want to use is the error term, not the residuals  
 17 what's left over, but the cow within treatment effect.  
 18 Q And how is that reflected in these tables?  
 19 A That is what this last line, which is a test of hypo-  
 20 theses using the type 3 means square in this, means  
 21 square, for cow (treatment) as an error term.  
 22 Q So that is the row that has in the last column P value  
 23 you're looking for?  
 24 A Correct. That was included as a line within the commands  
 25 given to the SAS Program to use that as the error term  
 26 for testing treatment.  
 27 Q Which happens to be exactly the same P number --  
 28 A It should be similar. It should have been similar.  
 29 Q As the four digits that appears, it was the same as under  
 30 the mix model that you discussed on 16?

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1 A Yes.  
 2 Q Then, --  
 3 A And then it repeats for all the other variance.  
 4 Q Is there any one of these models, either the mixed model  
 5 or glm model, that you consider most appropriate for  
 6 these circumstances as set forth in these calculations?  
 7 A Most people today, I believe, would use the Proc Mixed  
 8 procedure.  
 9 Q And many of these results as expressed in the P value are  
 10 different than what was reported in the original  
 11 publication provided to Minnesota?  
 12 A Well, it is a different way of computing the P values,  
 13 and that's not too surprising, but there were a couple  
 14 that were --  
 15 Q Different by quite a bit?  
 16 A -- were different by quite a bit. And the IgA in  
 17 particular, is one.  
 18 Q And it looks as if interleukin 1 is serum?  
 19 A Interleukin 1, I think 2, also. Let's see.  
 20 Q Let's go through those results --  
 21 A We will have to go through those.  
 22 Q -- a little bit?  
 23 A I think IgA was the first one of those where it looked  
 24 very different.  
 25 Q And that's, I believe those are the results that are  
 26 found on about pages 36 and 37?  
 27 A 36, yes, page 36 and 37.  
 28 Q Under the mixed model, for IgA, what's the P value?  
 29 A Under the mixed value, it says .0003.  
 30 Q That's at the top of page 37?

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1 A Top of page 37, yes.  
 2 Q As originally reported in exhibit 250, the P value under  
 3 the two tailed independent test, 12 treatments and 12  
 4 controls that we spoke about earlier, was 0.796, is that  
 5 correct?  
 6 A Yes.  
 7 Q And that is an established --  
 8 A That is very different. And when you look at the raw  
 9 data, you begin to see why that is. I'm going to go -  
 10 let me find it here. Beginning on page 11, we have a  
 11 table of needs for control and treatment groups for each  
 12 day. And if we come down on page 11, where it says,  
 13 Observation, which is row 37 in this table, IgA serum,  
 14 the 3 in the day means this is taken on day 3. 7 means  
 15 day 7. Control and treatment. We see here the control  
 16 and the treatment start off very different. And the same  
 17 on day 7. Now, if I recall correctly, day 3 and 7 no  
 18 treatment had been applied yet. So, for reasons that I  
 19 can't even guess, for whatever reason the serum IgA was  
 20 different at the very start of the treatment. So, when  
 21 we did a before and after study or analysis, as was done  
 22 on this table in exhibit 250, it didn't show much effect.  
 23 This particular calculation could have  
 24 all of the days in there, whether they're just looking at  
 25 after the treatment had been done. And so it's including  
 26 a difference that was at the very start before any  
 27 treatment was applied. And that's why, I think, it is so  
 28 radically different.  
 29 Q Then, with respect to interleukin serum, I think the  
 30 results of that under the mixed model, the next few

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1 pages, I think I'm seeing them on 39 and 40?

2 A Yes. That's correct. And again, I think this is because

3 of the differences in this. When you look - this

4 particular analysis, basically, didn't recognize - the

5 first two points in there were not actually points of

6 where the treatment had been applied. And when you take

7 that into account, you get that the effect is bigger than

8 what it appeared in this analysis.

9 Q So, would it be fair to say, in summary form with respect

10 to exhibit 279, although the method of analysis you've

11 tested it, that was attempted to be applied, might be

12 more appropriate than the original paper, it wasn't

13 applied correctly to this data set?

14 A I would say that's correct.

15 Q So, then there are some more follow-up analyses that you

16 requested, starting with exhibit 280 a couple

17 weeks later, is that correct?

18 A I don't remember requesting 280, but that is a follow-up

19 analysis, and in looking at that, I'm not entirely sure

20 what that analysis was. It appears to be a multi-

21 variable analysis of some sort. But there's not enough

22 of it there for me to see what - it appears that it's

23 being applied to IgA serum, but I don't see the actual

24 SAS code for it, so I am not a hundred percent sure what

25 was actually done with that.

26 Q Fair enough. Then 281. This goes several months later,

27 into February of 2000, according to the date on the

28 document. Do you recall requesting this re-analysis?

29 A I recall discussing with Steve using those days when no

30 treatment was applied as a co-variant barrier to the

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1 analysis.

2 Q For some reason, it looks like we don't actually have the

3 numbers - -

4 A All I see is what looks like a cover page for a report

5 that Steven has sent me.

6 Q Then, 282 is a memo directed from Steve LeMire to Doug

7 Reinemann. It indicates that you had requested the

8 summary statistics that are on the second and third page.

9 Do you recall requesting this information?

10 A I am pretty sure I would have requested that information.

11 I don't recall the specific request, but it's certainly

12 something I would have requested.

13 Q And why is that?

14 A Well, these are just the basic characteristics of the

15 cows that we were using in the study to just - to

16 document what the animals were like, what their milk

17 production was, how old they were, which lactation they

18 were in, those kinds of things.

19 Q And then, for each of those types of statistics or

20 variables, there's a mean, a standard variation given in

21 the table, correct?

22 A Correct.

23 Q From that data, or will that data reflect at all upon

24 how, shall we say, successful the randomization process

25 was?

26 A I don't know if it would reflect on how successful

27 randomization was.

28 Q Or how random it was, I guess is what I was looking for.

29 A It would basically reflect on whether the two groups were

30 really similar. As I recall, these were randomized. I

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1 believe Steve actually did the randomization, probably

2 using a computer program that is essentially a random

3 number generated to assign the cows to treatment. I

4 believe that's - I could be mistaken on that.

5 Q Well, does it appear that the two groups of cows were

6 reasonably similar?

7 A From the types of gross variables, like age of the cows,

8 milk yield, lactation number, that they looked fairly

9 similar. That's fairly typical of what you see with a

10 well-randomized experiment.

11 Q And then, exhibit 283 appears to be the result of another

12 request for a different type of statistical analysis from

13 yourself, is that correct?

14 A Actually, just a different variable. One of the things

15 that we had recorded, and had not included in the initial

16 analysis, was body temperature, morning temperature of

17 the cow's body temperature. And that appears to be

18 what's being analyzed here.

19 Q On the third page of that document, there is a table 2,

20 it has both typewritten and handwritten numbers. Any

21 idea what the handwriting is all about?

22 A Let's see. The very first here, over where it says

23 treatment and treatment by time, it's just to indicate

24 that those are the P values in those columns. I do not

25 know what that split plot refers to. The check marks are

26 just apparently checking with the computer printouts to

27 make sure that the numbers were typed correctly in the

28 table. The Concanavalin A blastogenesis that has a square

29 around it indicates that before the .413 should actually

30 be point .4013.

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1 Q Do you know whose handwriting it is on this page?

2 A I am not entirely sure. It looks a little like mine, at

3 least in places. So, I may have written that. The split

4 plot does not look like my handwriting. My handwriting

5 is not that neat. But the scribble at the bottom does

6 look like my handwriting.

7 Q Up at the top it talks about - -

8 A Yes, the P, I'm pretty sure I wrote that P. That looks

9 like the way I would write Ps, at least the first one.

10 Q And on table 2 at the end of the typewritten introduction

11 there, the third line, it says, "n equals 12 treated and

12 12 control cows," correct?

13 A Where?

14 Q Top of the third page, third line.

15 A Top of the third page, third line. Yes, that's correct.

16 Q Would that again indicate that this was run as a simple

17 two tailed t-test and not with a block design or

18 analysis?

19 A No. This was run, as I recall, as an analysis of co-

20 variance design, where the initial values were included.

21 I do not believe that a block was included in it. With

22 that much of a block effect, it's included in the

23 co-variant effect. Not always, but it is possible that

24 much of it would be.

25 Q So it's a different way of getting at some of the

26 concerns about multiple groups, is that correct?

27 A It's - no, it's a way of getting at the concern I

28 mentioned earlier about the IgA, that the cow started off

29 with a different IgA. That's what it's really trying to

30 get at.

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1 Q And as I think you indicated earlier, the original  
2 analysis in the published report, published in the sense  
3 that it went to the Minnesota government, the variable  
4 that was analyzed was the difference in cows from the  
5 first week test when no treatment was being applied to  
6 the later test, is that correct, with later assays, I  
7 should say?  
8 A Yes.  
9 Q And if one did that and one also took into account the  
10 block design of the experiment, that would yield an  
11 appropriate statistical analysis, would it not? That is,  
12 using the differences, the variable of interest in  
13 analyzing pursuant to replication of blocked statistics.  
14 A It might. I would run that by a statistician.  
15 Q Fair enough. 284. Another memo from Steve LeMire to  
16 Doug Reinemann, copy to yourself, indicating it was  
17 another analysis that you had requested, dated August 8,  
18 2000, correct?  
19 A That's what it says.  
20 Q Do you recall requesting this one?  
21 A No, I don't. I do know what it's about, so it's quite  
22 possible that I requested it, but I don't recall making  
23 the specific request for it.  
24 Q Okay. And what is it about?  
25 A A question came up. If this is the one I'm thinking it  
26 is, a question came up of a positive control. If we were  
27 to take cows and do something to them that we knew  
28 affected immune function, would we be able to detect it  
29 with the assays we were using? I do recall some  
30 discussions about that. What are we going to do? And

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1 the decision was made. I don't remember who actually  
2 came up with this suggestion, but it was suggested that a  
3 classic immune suppressant treatment is high dose  
4 glucocorticoids.  
5 Q Doctor, the first two words were high dose?  
6 A High dose, yes.  
7 Q And that was, such a control test was run in the original  
8 Part III study using dexamethasone, is that correct?  
9 A That's correct.  
10 Q Go ahead.  
11 A Yes. It was actually after we had finished the results  
12 we had been discussing up to this point. We did a short  
13 study with a fairly small number of animals where we  
14 injected dexamethasone. I think it was a three or four  
15 days of treatment, probably says in here - yes, four days  
16 treatment, and made our various measurements on them.  
17 And this was just an analysis of those results to see if,  
18 in fact, we did effect immune function.  
19 Q Sure. And they were also analyzed in a different  
20 statistical manner in the original paper, correct?  
21 A Were they recorded in the original paper here or not?  
22 Q Page 11 on exhibit 250.  
23 A We're looking at 250. I didn't recall those even being  
24 in this paper.  
25 Q Isn't that page 11 reports in exhibit 250?  
26 THE REPORTER: I'm sorry, I'm not hearing  
27 you.  
28 A It says positive control. Let me read this. It makes  
29 the statement that they were suppressed, but I'm trying  
30 to find the table that actually shows the data. So I do

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1 not know how it was analyzed at this particular study.  
2 Q I don't know that we have the data, the table with the  
3 data, but Table III on page 11 gives the results of the  
4 statistical analysis, I believe. Looks very similar to  
5 table 2, which has the electrical treatment.  
6 A Yes, I believe that is correct. The analysis here is a  
7 different analysis, a different way of analyzing it. But  
8 you're correct on that.  
9 Q Would this again be a t-test, basically?  
10 A This is a t-test, this Table III on page 11 is a t-test.  
11 Q And there's no issue with respect to the blocked or  
12 blocking issue because there's only one replicate?  
13 A Correct.  
14 Q And with a known immune depressant and dexamethasone for  
15 the 13 values reported were less than .05, correct?  
16 A That's correct.  
17 Q Okay. Then exhibit 285 appears to be another memo from  
18 Steve LeMire to Doug Reinemann, copy to you, about some  
19 more analysis you had requested, according to the  
20 document. Do you recall requesting this analysis?  
21 A This appears to be the analysis co-variants I was  
22 referring to.  
23 Q With respect to the positive control study?  
24 A Let me look at that. I don't think so. Must have been.  
25 There's not enough cows there.  
26 Q Under Introduction, it says, "It covers the positive  
27 control blood data"?  
28 A Okay. Yes. That's what that says.  
29 Q All right. In the course of all this analysis and  
30 re-analysis, did Steve LeMire, or anybody else associated

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1 with this experiment, that is, the Minnesota funded one  
2 resulting in the Part III paper, exhibit 250, ever do an  
3 analysis of the statistics of the difference between the  
4 after treatment and before treatment levels using a block  
5 design?  
6 A Not that I'm aware.  
7 Q Was that ever discussed among yourself and Mr. LeMire and  
8 Professor Reinemann or others associated with the  
9 experiment?  
10 A Not that I recall.  
11 Q So nobody ever expressed reluctance to do it that way, I  
12 take it?  
13 A Not that I recall.  
14 Q And it was Mr. LeMire who was primarily in charge of that  
15 part of the work, correct?  
16 A Correct.  
17 Q And the person overseeing - who is the person overall in  
18 charge of the whole experiment? Was that Professor  
19 Reinemann?  
20 A That would have been Professor Reinemann.  
21 Q Then, with respect to the second experiment involving the  
22 Messenger RNA, the gene expression, who would you  
23 describe as the person in overall charge of that  
24 experiment, if there was one?  
25 A That would have been me.  
26 Q In the materials from the University, there were - the  
27 one graph of that I found in those materials came out of  
28 Professor Reinemann's file. Maybe I can find that.  
29 (Exhibit 286). I printed off the sheets from Professor  
30 Reinemann's disk right after that.

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1 How was it that this document came to be  
2 drafted? Did you write it? Did somebody else write it?  
3 Did you have assistants or somebody else? How did that  
4 work?  
5 A This appears to be - this appears to be something that I  
6 wrote, and would have shared with Dr. Reinemann, who was  
7 involved in this, particularly in figuring out how to  
8 deliver the current to the animals.  
9 Q This particular item, again, which is off a disk supplied  
10 by Professor Reinemann, has some typed in red, appears  
11 that editing was going on by someone. Do you know what  
12 that was all about?  
13 A I do not know who would have made those edits. They may  
14 have been changes that I made in the document, they may  
15 have been things that someone else suggested. I wouldn't  
16 know.  
17 Q But your last - at the first portion of your deposition  
18 in March, another version of this document was marked as,  
19 I believe it's exhibit 251. I believe the text is the  
20 same. We can check that, but.  
21 A It would look very similar.  
22 Q It didn't have the red type and so forth, but I think it  
23 was the same text in the first four pages plus the  
24 references anyway.  
25 Would you turn to the third page of that  
26 document, and I think the text will be the same on the  
27 other one. Under Results and Discussion, in the second  
28 paragraph, the third sentence, it says, "There was a  
29 tendency for IL1a, or alpha, and IL1b beta mRNA, to  
30 increase slightly, P less than 0.10, but it did not reach

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1 significance at P less than 0.05." Did I read that  
2 correctly?  
3 A That's what that says, yes.  
4 Q And yet, in the tables of data and analysis that we went  
5 through this morning, exhibit 254, those P values were on  
6 the order of approximately 10 to the minus 5th, or 10 to  
7 the minus 6th, something like that. Do you recall that?  
8 A Yes. And I do not recall - that's why that maybe in  
9 looking at these columns, I don't know that I generated  
10 those numbers in that table. I don't know where those  
11 numbers came from.  
12 MR. THORNTON: The last four lines?  
13 A The last four lines of this (254). I'm not sure.  
14 Because this does not look like a version of this that I  
15 actually had. It's got a lot of the numbers in it. So,  
16 I don't know where those numbers came from. I don't  
17 recall putting in those particular numbers, so I can't  
18 vouch for the veracity of those last four lines of this  
19 table.  
20 Q Okay. You supplied documents to Kathleen Erwin, the  
21 University's counsel, in response to the subpoena that  
22 was served on the University back in late 2007 and  
23 through early 2008, correct?  
24 A That's correct.  
25 Q That those documents were subsequently copied when in  
26 document form and provided to myself and others who were  
27 interested in obtaining those documents.  
28 Did you ever send that documentation back  
29 or did it stay with the University?  
30 A As far as I know, it is still with the University. I

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1 don't have it.  
2 Q Well, I will represent to you that it was provided to us  
3 on a disk whose cover was, as marked on exhibit 276, and  
4 on that disk - mark some of these if we need to later,  
5 but I would just like to work through it before we do.  
6 There are files named "503 cow data sheets." And then,  
7 "Array 503 gene. Array 503 layout. Array 503  
8 statistics. And collated Array 503," as it appears on  
9 the disk produced by the University.  
10 Do you have any idea what the 503 number  
11 refers to?  
12 A Just a number we gave the file.  
13 Q And that particular spreadsheet, including the bottom  
14 four lines, appears in the file labeled "Array 503  
15 statistics." I'll also represent to you that Professor  
16 Frank Martin, who is a retired statistics professor at  
17 the University of Minnesota, with a long time appointment  
18 at the veterinary school, has reread those numbers and  
19 verified them.  
20 A Okay.  
21 Q And again, it would just be a matter of - -  
22 MR. THORNTON: I'm going to object to that.  
23 You are testifying, number one. Number 2, Frank Martin  
24 hasn't been identified as an expert in this case and  
25 hasn't made any expert disclosures in this case.  
26 Q In any event, coming up with that analysis is just a  
27 matter of running the math, either by computer or  
28 however, correct?  
29 A Correct.  
30 Q Where did the .05 to .10 P values come from?

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1 A I don't recall where those would have come from. They  
2 could just be things that I entered incorrectly in this  
3 tape. I may have inadvertently got my decimal point off  
4 and meant .01. I honestly don't recall.  
5 Q And by table, you are referring to the table 1, which is  
6 part of the exhibit - -  
7 A Table 1, yes.  
8 Q It appears that there are no P values in that table, is  
9 that correct?  
10 A That's correct.  
11 Q However, the fold number, or a fold number anyway, is in  
12 that table, correct?  
13 A Yes.  
14 Q Did you ever have occasion to discuss the P values from  
15 the gene expression study, the second study that is, with  
16 Professor Reinemann?  
17 A I don't recall any such discussion.  
18 Q Dr. Sheffield, I would like to ask you to assume,  
19 hypothetically, that the P values from this second study  
20 are, as set forth in exhibit 254, pursuant to a t-test as  
21 we discussed this morning, and that the P values for IgJ  
22 of 8.21, times 10 to the minus 5th; interleukin 1 alpha  
23 of 8.74 times 10 to the minus 6th; IL1 beta of 2.55 times  
24 10 to the minus 6th; IL2 of 4.98 times 10 to the minus  
25 6th; and IL10 of 2.93 times 10 to the minus 5th, do  
26 reflect an accurate two tailed P analysis of the  
27 experimental data.  
28 Under that hypothetical assumption, would  
29 the Results and Discussion section of the paper need to  
30 be redrafted, in your estimate, if it were to be

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1 submitted for publication?  
 2 A Well, it would certainly, when it refers to IL1 alpha and  
 3 beta, would need to be modified from slightly to  
 4 significantly, with a PS to .01, which is how very low P  
 5 values are usually reported, and he did not reach  
 6 significance would be struck. The significance of the  
 7 IL2 and IL10 is not changed, just the P value changes.  
 8 And the same thing with .04, the IgA, heavy chain, and  
 9 secretory piece.  
 10 Does that answer it?  
 11 Q Sure. And again, as we have already discussed, an  
 12 increase of interleukin 1, either alpha or beta, that is  
 13 somewhat under two-fold, is certainly not indicative of a  
 14 change associated with an acute infection, correct?  
 15 A Correct.  
 16 Q The main objective of a commercial dairy herd, certainly  
 17 one of them, is to produce high levels of milk production  
 18 and good components, correct?  
 19 A Yes. That is a major objective.  
 20 Q Do you have any opinion one way or the other as to what  
 21 the likely consequence of chronically elevated  
 22 interleukin 1 alpha and beta between 1.5 and two-fold,  
 23 what would be there without electrical exposure, would  
 24 have to the productivity of a commercial dairy herd?  
 25 MR. THORNTON: Objection. Foundation.  
 26 A I am not familiar with anything that would let me assess  
 27 that reliably.  
 28 Q Is it true, as I've heard some people express, that  
 29 elevated interleukin 1 is one of the principal factors  
 30 determining how we feel lousy when we have a cold or

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1 other infection?  
 2 MR. THORNTON: Objection. Relevancy.  
 3 A I've heard that.  
 4 MR. THORNTON: Objection. Hearsay.  
 5 Q Do you have any professional opinion as to whether or not  
 6 its true or do you know?  
 7 A I don't know.  
 8 Q Do you know of any studies or publications indicating  
 9 that one of the symptoms of chronically elevated  
 10 inflammatory cytokines, such as interleukin 1, use some  
 11 degree of inappetence?  
 12 A Excuse me?  
 13 Q Some degree of inappetence, not getting hungry?  
 14 A I'm not aware of that. But I haven't read the literature  
 15 on this in a while.  
 16 Q I think you would probably agree that when dairy cows get  
 17 some degree of inappetence, that's a big deal to a  
 18 commercial dairy, fair statement?  
 19 A Yes, feed intake is a big issue in dairy production.  
 20 Q I have way too much paper here. What, if any, are the  
 21 consequences of a decrease in IgA, either in serum or in  
 22 tissue, to local immunity of the utter of a cow?  
 23 A In tissue, it's fairly important. Circulating, I'm less  
 24 certain about how important that would be.  
 25 Q And why are IgA levels in the utter important to local  
 26 immunity of the utter?  
 27 A Well, IgA in many mucosal tissues is the major anti-body  
 28 that is secreted into a secretion. It turns out in cows,  
 29 they are a little bit different in their mammary glands  
 30 than most other species, in that cows' milk actually has

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1 more IgG in it than most other species do. Humans, for  
 2 example, the major anti-body in milk is IgA. Cows have  
 3 significant amounts of IgA in their milk, but they also  
 4 have large amounts of IgG. But IgG is still an important  
 5 part of immunity in these tissues that are exposed to the  
 6 environment, such as the utter. If you think about it,  
 7 the inside of the utter is outside the body.  
 8 Q Explain that a little bit more, please. That last  
 9 comment, I mean.  
 10 A Well, the mammary gland is lined with an epithelium.  
 11 Milk is produced in small structures called alveoli and  
 12 is transported through a system of ducts to the outside.  
 13 So, it is just like the lining of your GI tract, it's  
 14 actually outside the body. That means it is very easy  
 15 for bacteria to enter through the teat, what we call the  
 16 streak canal, and infects the utter. And the utter  
 17 has, as you'd expect, a lot of defense systems, among  
 18 them, various anti-bodies in milk.  
 19 Q Unfortunately, the next document I want to refer to is  
 20 sitting down in the trunk of my car. I can get it if I  
 21 need to.  
 22 MR. THORNTON: Do you want to take a short  
 23 break?  
 24 Q Yeah, we could do that. Maybe I can ask you if you can  
 25 recall anything about Professor Reinemann making a  
 26 presentation at a seminar relating to - or entitled Stray  
 27 Voltage, something like that, in Campbelltown, Penn-  
 28 sylvania, in the spring of 2003, in which his references  
 29 included a reference to a paper that was reported in the  
 30 document in press, being submitted in The Journal of

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1 Dairy Science, in which you were a co-author at that  
 2 time.  
 3 Does this make any sense to you?  
 4 A Possibly. I think there was a paper that we were  
 5 drafting. I don't recall it ever being actually accepted  
 6 for publication. I assume that is what he is referring  
 7 to.  
 8 Q I believe in 2003. Would that be approximately the  
 9 likely - -  
 10 A That would sound like about the right time-frame. I'm  
 11 assuming that's the paper he's referring to. It was  
 12 never published.  
 13 Q Is that the same paper that was marked in your earlier  
 14 deposition as exhibit 249?  
 15 A Let me see exhibit 249.  
 16 Q Here we go.  
 17 A Yes. That's the one I'm assuming he's referring to  
 18 there.  
 19 Q To your knowledge, was that paper put out for peer  
 20 review?  
 21 A I don't recall.  
 22 Q You never saw any feedback from any peer review - -  
 23 A I don't recall anything like that. It could have been.  
 24 That was a long time ago. I don't recall that.  
 25 Q Were the results of the second study with the MRNA gene  
 26 expression ever put into a form that was intended to be  
 27 submitted for publication?  
 28 A No, they weren't.  
 29 Q And you personally have no results of statistical  
 30 analysis of that data from the gene expression study at

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1 all, I take it, unless they are in what you brought to  
 2 the first deposition? I don't believe there's any --  
 3 A I don't believe I do.  
 4 Q Back in the late '90s and the very early 2000s as this  
 5 work was going on, did you know that Dr. Reinemann had  
 6 been testifying for utilities in stray voltage litigation  
 7 since approximately the early 1990s?  
 8 A I know now that he had been. I honestly can't remember  
 9 if I knew at that time if he had been or not.  
 10 Q Not a subject that ever came up at the time?  
 11 A I don't recall any discussions with him about it. I may  
 12 have known it, but I may not. I just don't remember what  
 13 I knew when.  
 14 Q Other than responding to the subpoena here today and the  
 15 last time we got together, you have never been involved  
 16 in litigation --  
 17 A I have never been involved in any such litigation.  
 18 Q Sure. You've never been involved in any litigation as an  
 19 expert witness, is that correct?  
 20 A No, I have not.  
 21 Q At least until whatever we've done here. I don't think  
 22 I'll bother going down to the car. I think we have  
 23 covered it.  
 24 I've got copies of these, but may not be  
 25 very significant. Let me just ask you some questions  
 26 regarding them.  
 27 Exhibit 287 is what I understand to be some  
 28 notes taken by folks working in the first research  
 29 project, the Part III paper project, taken off the  
 30 materials provided by Dr. Reinemann in response to the

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1 University subpoena.  
 2 Does that refresh your recollection at all  
 3 as to whether there were any barn notes taken?  
 4 A Well, that is what this appears to be. I don't recall  
 5 seeing these specific notes before, but I knew that  
 6 things like water consumption and temperatures were being  
 7 recorded. I didn't see the raw records, but that's what  
 8 this appears to be.  
 9 MR. THORNTON: Object to that document.  
 10 Foundation.  
 11 Q The document, the Bates number from the University  
 12 materials, the documents start with Reinemann 1595, for  
 13 the record, and runs through -- well, there's a number of  
 14 pages there, The last one of which is Reinemann 1611.  
 15 And at the bottom of the sheet where the  
 16 water and temperature and so forth are recorded, there's  
 17 a Comments section, is that correct?  
 18 A That's what it seems to be.  
 19 Q And what was the purpose, if you know, of having that  
 20 Comments section on the form?  
 21 MR. THORNTON: Objection. Foundation.  
 22 A I would only be able to speculate, since I did not --  
 23 MR. THORNTON: Objection. Speculation.  
 24 Q Since you did not, what?  
 25 A Since I did not design the form or have any input into  
 26 it.  
 27 Q Well, were you not the person in charge of experimental  
 28 design as it related to things having to do with the cows  
 29 themselves?  
 30 A No.

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1 Q Who was in charge of that?  
 2 A Dr. Reinemann did most of the setup, designing the stalls  
 3 and working with the barn crew. I was responsible for  
 4 handling the samples after -- the blood samples that were  
 5 coming into the lab.  
 6 Q There's a person named Misty, I believe the last name is  
 7 Davis, that appears in these forms. Are you familiar  
 8 with her?  
 9 A She was someone that worked with Dr. Reinemann. I don't  
 10 know if she was a graduate student or post doctorate or  
 11 her exact status, but she was an employee with Dr.  
 12 Reinemann in Ag. Engineer.  
 13 Q Well, I tell you what, why don't we take a short break.  
 14 I'm close to done. And these gentlemen will want to ask  
 15 you some follow-up questions, I'm sure, especially Mr.  
 16 Carlson, who wasn't here before. Why don't we take ten  
 17 minutes, shall we say, or your pleasure. It's your  
 18 flight, so I'll make it shorter, if you want.  
 19 MR. THORNTON: That's fine.  
 20  
 21 (At this time a recess was taken - 2:15 - 2:26).  
 22  
 23 Q Dr. Sheffield, we've marked as exhibit 288 another packet  
 24 of barn notes, if you will, the water meter, et cetera,  
 25 measurements on them and comments, and I believe these  
 26 are from Replica 2, and were taken in January, 1999.  
 27 Would you turn to the third page of those  
 28 barn notes, Bates number 1663. Look through some of  
 29 these quickly. I'm sorry. Actually, turn to the fourth  
 30 page, if you would, 1669. There's a fairly long hand-

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1 written description about cow number 3861 having a bad  
 2 day there. Do you see that?  
 3 A Yes.  
 4 Q The initials at the bottom I think, appears to be RK. Do  
 5 you know who that is?  
 6 MR. THORNTON: Objection. Foundation.  
 7 A A watcher, and I think the last name was something like  
 8 Kasper, if I recall. There was a Roger Kasper that was  
 9 involved in this, but I can't say for sure if that's who  
 10 that's referring to.  
 11 Q Roger was a one-time State Agriculture Department  
 12 employee, I believe. Did you know that?  
 13 A I seem to recall that, yes.  
 14 Q And toward the middle of the page, it's indicated by the  
 15 author of this note, -- I'll point to where I am on the  
 16 sheet. "Decided to share concern with Doug and then  
 17 Lewis." Do you see that?  
 18 MR. THORNTON: About half way. The line  
 19 that begins with "Jerry and --"  
 20 A Yes. Okay. I see that.  
 21 Q Then, toward the bottom of the page, it's indicated,  
 22 "Lewis, Doug and Josie felt okay to stay in trial." Do  
 23 you see that?  
 24 A Yes.  
 25 Q And up on top, it describes her having trouble getting  
 26 up, struggling to get up and things of that nature. Do  
 27 you see that?  
 28 A Yes.  
 29 Q Do you recall anything about that cow as you sit here  
 30 today? Long time ago, I realize.

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1 A There was at least one instance. This may be this  
 2 particular instance, where someone, and I don't remember  
 3 who it was, possibly - probably Josie, since she worked  
 4 with me, came and said, "We've got a cow that seems very  
 5 bad. Can we come look at her?" By the time I got there,  
 6 she did not seem to be having those problems. But that's  
 7 really all I recall about that.  
 8 I do recall at least one instance where  
 9 there was a cow they were concerned about in the morning,  
 10 but by afternoon seemed to be doing okay. But I don't  
 11 recall any more details about it.  
 12 Q Seems to be several pages into the document, about seven  
 13 pages in is a typewritten memo.  
 14 MR. THORNTON: What's the Bates number?  
 15 Q The date on it is January 25, 1999.  
 16 MR. THORNTON: I'll just note the page in  
 17 front of it is 1674, then we go to 1394.  
 18 Q It is. Taken out of a different section of Dr.  
 19 Reinemann's materials.  
 20 In there it talks of cow 3861 having  
 21 fallen, so forth. Do you see that?  
 22 A I see that.  
 23 Q And further down is a duplicated, an e-mail from Roger  
 24 Kasper, to apparently Dr. Reinemann and others.  
 25 Are you among the recipients of that  
 26 e-mail?  
 27 A I don't recall seeing this. I will look at the Cs on it  
 28 to see if I'm on there. I don't seem to be on the CCs to  
 29 that. My e-mail address doesn't appear on this, and I  
 30 don't recall seeing this.

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1 Q Sure. In the second paragraph below the addresses,  
 2 there's a line, indicates, "Doug, Lewis and Jerry  
 3 discussed the situation, decided giving her a tube of  
 4 Calcium Oral Gel would help boost her energy. Also some  
 5 surface ointment was applied to the spot on rear hip  
 6 where the fur has rubbed off from rubbing against the  
 7 stall support." Do you see that?  
 8 A I see that.  
 9 Q And if we go through these notes, we will find notes  
 10 relating to cow 3861 on January 22, 23, 27, 28 and 29. I  
 11 don't necessarily want to go through all of them with  
 12 you, - -  
 13 A I appreciate.  
 14 Q - - but just take a quick look and verify whether or not  
 15 there are multiple days with notations about that cow?  
 16 A Okay.  
 17 Q Is there multiple entries about that cow?  
 18 A It does appear that way, yes.  
 19 Q On multiple occasions either down or having trouble  
 20 getting up, that sort of thing?  
 21 A There seems to have been some concern about her mobility.  
 22 Q And from the treatment prescribed, it appears that the  
 23 consensus was that she was hypocalcemic?  
 24 A I don't recall there ever being - I don't recall a  
 25 discussion about that particular treatment. I mean, it's  
 26 possible she was, but - let me. I would have to spend  
 27 some time looking through where she was in lactation and  
 28 all of that. But I don't remember a discussion about  
 29 treating her with calcium.  
 30 Q But it is indicated in Roger Kasper's e-mail though,

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1 correct?  
 2 A It is indicated that she was treated with that.  
 3 Q And if that were done, the only reason for that would be,  
 4 the thought process was, she was hypocalcemic?  
 5 A That would be what you would use that for.  
 6 Q Now, according to the table in exhibit 282 that we went  
 7 through earlier, I'll just show you my copy here, it  
 8 indicates that that cow was, I believe 175 days in milk  
 9 at the start of her participation in the study?  
 10 A That's what that says, yes.  
 11 Q Is it normal for a cow in mid-lactation, such as that, to  
 12 be hypocalcemic?  
 13 A Not normally. That's usually something that occurs early  
 14 in lactation.  
 15 Q I believe the Part III paper indicates that there were no  
 16 noted differences between treatment and control cows in  
 17 behavior, or makes that indication at some length, is  
 18 that correct?  
 19 A I think it does.  
 20 Q Would it have been a reasonable thing to evaluate  
 21 statistically the number of cows, treatment and control,  
 22 through these three replicas that exhibited some unusual  
 23 behavior or some health problems, such as hypocalcemia?  
 24 A Because the incidents of it is so low, I don't know how  
 25 you could make any conclusion from that.  
 26 Q Well, that would certainly be true for any one condition?  
 27 A Or any one condition, yes.  
 28 Q How about, would it be reasonable to evaluate the  
 29 incidents of any unusual behavior for health problems in  
 30 the population of cows?

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1 A I'm assuming you mean by this, simply having a health  
 2 problem, yes or no. I don't know, just because that  
 3 seemed like a fairly vague thing to do. Most researchers  
 4 would want to break it out into exactly which health  
 5 problem you're talking about.  
 6 Q Understood. But the more you break it out, the more you  
 7 run into the difficulty of small numbers of cows in - -  
 8 A Yes.  
 9 Q - - experiments that are practical, right?  
 10 A Yes.  
 11 Q In exhibit 286, the draft paper that was not published,  
 12 there's a reference in perhaps more than one place, but  
 13 one of them is at the bottom of page 1, the very last  
 14 line, and on the top of page 2, about epidemiological  
 15 study of over 15,000 Swedish cows. Do you see that?  
 16 A Yes. I remember reading this study.  
 17 Q And that had to do with herds that use or did not use  
 18 electric cow trainers?  
 19 A That's correct.  
 20 Q I'm going to show you what we have marked as exhibit 289,  
 21 which is out of an LC of your journal, titled Preventive  
 22 Veterinary Medicine, and it appears to be a copy of an  
 23 article entitled, "Associations between use of electric  
 24 cow-trainers and clinical diseases, reproductive  
 25 performance and culling Swedish dairy cattle," and the  
 26 lead author is Pascal A - not even attempt to pronounce  
 27 this, O-l-t-e-n-a-c-u. Is that correct?  
 28 A That's correct.  
 29 Q Does that appear to be the article that you were  
 30 referring to in your draft?



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1 A That does appear to be the article. Let me double-check  
2 this. Yes, that appears to be the same article.  
3 Q Then, on the fourth page of your paper, the page where  
4 the references start at the bottom. The draft paper  
5 says, starting on the fourth line, "An epidemiological  
6 study by Oltenacu - for want of a better pronunciation -  
7 et al, 1998, that found mastitis and reproductive  
8 problems were associated with the use of cow-trainers.  
9 This could suggest impacts of electrical exposure. But  
10 other explanations are also possible, including the herds  
11 with such problems may be more likely to use cow-trainers  
12 to solve them." Correct?  
13 A That's what it says there, yes.  
14 Q And do you recall that at least one of the herds studied,  
15 a larger herd utilized one group of its cows and in the  
16 before and after use of cow-trainers - -  
17 A Yes, that was in some of the data in there, yes.  
18 Q And even in that herd using the same cows as their own  
19 control, if you will, there was found to be an effect, is  
20 that correct?  
21 A It has been a while since I've read this paper.  
22 Q Well, and I don't want to make you sit here and read it.  
23 A I think I do recall that.  
24 Q Okay. Fair enough. We can all read the paper and find  
25 out.  
26 Have you had any occasion to read exhibit  
27 252, which is marked from the last time, which is a group  
28 of materials prepared in connection with other litigation,  
29 not this case, by Dr. Frank Martin, retired physics  
30 professor I have referred to?

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1 A I have read it. I haven't read it in incredible detail.  
2 By I have read through that, yes.  
3 Q With respect to Dr. Martin's statistical methods applied  
4 to the data from the Part III study and/or the MRNA gene  
5 expression study, do you have any critiques or issues  
6 with the statistical methods he approached those with?  
7 MR. THORNTON: Objection. Foundation.  
8 Objection. Identifying the work of a witness who has  
9 neither been designated as an expert or provided a report  
10 in this litigation.  
11 A Okay. I was not sure exactly what he did in his  
12 re-analysis, but the idea of using the general type that  
13 he referred to would seem reasonable.  
14 Q Counsel talked to you about the issue of blood samples  
15 collected from the cows in the second study, the gene  
16 expression study, which is referenced, I believe, in the  
17 middle of the second page of exhibit 286 or exhibit 251,  
18 whichever you prefer, and which indicates blood samples  
19 were collected via the tail vein immediately prior to  
20 applying current and at the end of a three week exposure  
21 period." And I think you told Mr. Thornton when we got  
22 together before that you didn't know where the data was  
23 from the initial blood draw?  
24 A I don't think we ever analyzed those samples.  
25 Q I see. Is there a particular reason why or why not?  
26 A Running out of time and money, and the analysis is quite  
27 difficult, tedious and expensive to perform.  
28 Q And then these gene expression assays are expensive and  
29 the use of sophisticated equipment, correct?  
30 A Yes.

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1 Q I think you mentioned earlier that people had to share  
2 the University's equipment with other departments, it's  
3 not like it was available to you for as long as you  
4 wanted, I take it, is that correct?  
5 A For some of it, that's true.  
6 Q By the way, did you ever talk to Mr. Thornton before the  
7 first leg of the deposition in this matter?  
8 A Only to reschedule the deposition.  
9 Q Have you ever had occasion to discuss with anyone what  
10 you were going to be asked about in this deposition  
11 before it occurred, and I don't mean just today, but the  
12 first leg of it?  
13 A No, other than to state your opinions on this work.  
14 Q But have you had occasion to discuss something with Mr.  
15 Thornton just as we took a break awhile ago? Did it have  
16 anything to do with this case or just pleasantries?  
17 A Just the contents, you know, about, I think the comment  
18 that I made had to do with I can be kind of frustrating  
19 at times because I will answer a question by saying this,  
20 but on the other hand.  
21 Q In other words, lawyers like more defined answers than  
22 scientists often have?  
23 A Yes, I think sometimes I could be frustrating because  
24 everyone seems to want me to give a yes or no answer, and  
25 I'm seeing nuances in things.  
26 Q I have enjoyed listening to your nuances, Professor  
27 Sheffield, but that's all I have at least for now.  
28 Counsel may have some follow-ups.  
29 MR. CARLSON: Do you have anything, Tim?  
30 MR. THORNTON: Go ahead. You haven't had a

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1 chance to talk to him at all. I kind of get the feeling  
2 we're going to be coming back here.  
3  
4 RE-DIRECT EXAMINATION  
5  
6 BY MR. CARLSON:  
7  
8 Q Dr. Sheffield, correct?  
9 A Correct.  
10 Q I just want to kind of go over a few things. I apologize  
11 in advance if I cover any ground that's already been  
12 covered, and I'll try not to. And I have read the  
13 transcript of the first portion of your deposition.  
14 Have you had any contact with attorneys  
15 Will Mahler or Charlie Bird or Jeremy Stevens, or anyone  
16 working on behalf of Randy and Peggy Norman?  
17 A No.  
18 Q Is it your understanding that your testimony or March 14  
19 and today will be to render opinions regarding your work?  
20 A Yes.  
21 Q Now, the work of Frank Martin has been described to you a  
22 few times, and I'll represent to you that he has done  
23 some work in the case of mine, including the Randy Norman  
24 versus Crow Wing Power case, which is why I'm here. Dr.  
25 Martin testified that you've lost data, and he's  
26 referring to that initial blood test data.  
27 I take it that you were referring to the  
28 exhibit 286 abstract research that was done.  
29 MR. LAWRENCE: Object. Foundation. Go  
30 head.

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1 Q My question is, did you lose any data?  
 2 A Not that I know of.  
 3 Q And when you testified just a few moments ago that time  
 4 and money were running out and you had to spend money, I  
 5 guess more wisely, you didn't analyze the initial blood  
 6 tests?  
 7 A The initial blood tests were not analyzed. It wasn't  
 8 that the data weren't collected and lost. I don't  
 9 believe we ever analyzed those.  
 10 Q And do you agree that it would be helpful - it would have  
 11 been helpful to have analyzed that initial blood test  
 12 data so that you could compare it to what happened later?  
 13 A I think more data is also better served. I would say it  
 14 would have been better to have done it.  
 15 Q And when I say what happened later, what I should have  
 16 more artfully said, compare it to the end of research  
 17 blood test results?  
 18 A Yes.  
 19 Q Are you aware of any authoritative referenced table that  
 20 establishes what are good or acceptable levels or ranges  
 21 of levels for various substances found in the immune  
 22 system, such as IL1, IL2, IL3, IL10, IgA and I think  
 23 you've been calling it IgG, I have heard it called Ig3  
 24 sometimes. Is there any such authoritative reference  
 25 table that you're aware of?  
 26 A There are accepted values for what is normal for some of  
 27 those. I couldn't point you to a specific reference, off  
 28 the top of my head. But for others, I don't think there  
 29 are.  
 30 Q I take it, off the top of your head, you can't tell me

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1 which ones that there are basically no - -  
 2 A Well, - -  
 3 Q If I could finish. I take it that you're not aware, off  
 4 the top of your head, of which one of these or any of the  
 5 other - I'm calling them substances, I don't know if  
 6 that's the best term, but better analyzed in exhibit 254  
 7 and described by Dr. Chase in exhibit 275. You couldn't,  
 8 off of the top of your head, say that there's known  
 9 values for these that are acceptable?  
 10 A Well, those are different than protein values. Those  
 11 values are Messenger RNA values, and I think it would be  
 12 very difficult to find standard values of what those  
 13 would be.  
 14 Q What about on the various cytokines, the pro-inflammatory  
 15 and anti-inflammatory, and I'm going to say compounds, if  
 16 that's okay. I'm not in your field. But is there any  
 17 table of known, accepted, you know, reasonable values for  
 18 those?  
 19 A Not that I'm aware of for cattle.  
 20 Q And what I'm getting at is, I go to the doctor and he  
 21 does a - runs a blood test on me. Gives me back a report  
 22 and says, "You're good and bad cholesterol levels are  
 23 this, these are the acceptable ranges. Your enzymes are  
 24 in this range, and this is the acceptable or good range."  
 25 So there's nothing like that for dairy cattle regarding  
 26 their immune system?  
 27 A For immunoglobulin levels, there probably is. There are  
 28 some numbers that you might think are high or low. But  
 29 for the cytokines, I'm not aware of anything like that.  
 30 Q And IL1, 2, 3, 10, IgA, IgG, those are all cytokines?

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1 A No. The IL interleukins are cytokines.  
 2 Q Okay.  
 3 A The ones that start with Ig are immunoglobulins. Those  
 4 you might find standard values for. I couldn't point you  
 5 to a specific table, but there might be such.  
 6 Q Are you familiar with a method called, Repeated Measures  
 7 Design?  
 8 THE REPORTER: Called what?  
 9 Q Repeated Measures Design.  
 10 A Yes.  
 11 Q And was that used at all in your work?  
 12 A That's what the Proc Mix was, is a type of repeated  
 13 measures design.  
 14 Q And you use Proc Mix for some of them?  
 15 A For some of the analysis, yes.  
 16 Q And those are at least denoted in the exhibits, correct,  
 17 which ones have been used for?  
 18 A I think so.  
 19 Q That's all I have for now.  
 20 A Okay.  
 21 Q Oh, wait. I'm sorry. I missed something here. I just  
 22 want to read to you something.  
 23 Dr. Frank Martin has opined in the Randy  
 24 Norman case that he will testify that in the 1999 Science  
 25 Advisors, Part III experiment, 1 milliamp current had an  
 26 effect on behavior and health of the treated cows at a  
 27 highly statistically significant level. Do you agree  
 28 with that statement?  
 29 A I'm not familiar with the behavior data that was  
 30 collected on that, so I can't give an opinion whether I

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1 agree or disagree with that.  
 2 Q What about the effect of those levels on the health of  
 3 the treated cows, did the 1 milliamp current have a  
 4 statistically significant effect on their health?  
 5 A Not that I know of. But again, I don't have the specific  
 6 data about health measures for that.  
 7 Q So, were your conclusions in the 1999 Science Advisors,  
 8 Part III, he calls it an experiment, I believe it was  
 9 research, so you didn't come to these conclusions that  
 10 Dr. Martin comes to here, is that correct?  
 11 A We are referring here to - -  
 12 MR. THORNTON: 250.  
 13 A 250.  
 14 Q Yes.  
 15 A Okay. As I recall, that was a statement that was made  
 16 based on the - I don't know what that statement was based  
 17 on about behavior.  
 18 Q Are you talking about Frank Martin's statement?  
 19 A Yes. I don't recall - I don't know what that statement  
 20 was based on. I am not aware of anything in that study  
 21 that showed behavioral differences, but I'm not aware of  
 22 data - specific data on behavior.  
 23 Q So did you, in exhibit 250, in that report, was a change  
 24 in cow behavior one of the things that was being  
 25 measured?  
 26 A That was not the major purpose of this. The major  
 27 purpose was to measure the immune function.  
 28 Q Was there any data obtained from that research which  
 29 would allow someone to make any type of - or draw any  
 30 type of conclusion about changes in cow behavior during

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1 your 1 milliamp exposure testing?  
 2 A There were some things that one might call behavior.  
 3 Q What were those?  
 4 A Things like feed consumption and water in-take were  
 5 measured. I don't know if you'd call those behaviors or  
 6 not, but those were measured. They were analyzed. I  
 7 don't recall the results of those. As I - well, I better  
 8 not say, because I really don't recall the results of  
 9 those particular analyses. I don't recall anyone ever  
 10 discussing any usual changes in feed or water  
 11 consumption.  
 12 I'm not aware of any other measures of  
 13 behavior other than general comments that were made in  
 14 the barn notes, which I had never looked at before today.  
 15 Q Such as the cow - -  
 16 A Such as - -  
 17 Q If I can finish. And that's what I was wondering. Was  
 18 there any effort made during your research for exhibit  
 19 250 that would have recorded or in some way measured  
 20 animal behavior, such as lapping at water, stomping,  
 21 kicking, things along that - twitching, things along  
 22 those lines?  
 23 A Not that I'm aware of.  
 24 Q And you were involved in that research from start to  
 25 finish, correct?  
 26 A I did not do very much in the barn. Other people were  
 27 involved in that. I was mostly involved in analyzing the  
 28 laboratory bench-type analysis of the blood samples that  
 29 were collected.  
 30 Q Did you see a copy of exhibit 250 before it was submitted

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1 to the State of Minnesota?  
 2 A I believe so.  
 3 Q And in you review of that, did you see any indications  
 4 that cow behavior, other than like feed in-take and water  
 5 in-take, the things I was talking about, the lapping at  
 6 water, fidgeting, and that sort of thing, did you see any  
 7 indication that any of those behaviors were ever measured  
 8 or recorded?  
 9 A I don't recall them being recorded, and I don't recall  
 10 any discussion of recording specifically like that for  
 11 this study.  
 12 Q And if I'm correct, and I apologize if I'm rehashing.  
 13 Did that report, the Part III report, conclude that 1  
 14 milliamp current had an effect on the health of treated  
 15 cows at a highly statistically significant level?  
 16 A I don't recall that conclusion.  
 17 Q Have you seen Frank Martin's complete re-analysis of your  
 18 work where he comes to that conclusion?  
 19 A No, I haven't.  
 20 Q So, I take it you're not able to opine as to whether  
 21 Frank Martin's methods and conclusions are valid?  
 22 A Without seeing it, no, I can't.  
 23 Q That's all I have.  
 24  
 25 RE-DIRECT EXAMINATION  
 26  
 27 BY MR. THORNTON:  
 28  
 29 Q Dr. Sheffield, you did all the statistical work for the  
 30 second study that you did?

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1 A I think so, yes.  
 2 Q And you're not a trained statistician?  
 3 A No, I am not.  
 4 Q And you said it would be best to adjust for the multiple  
 5 variables that you were looking for, but you didn't do  
 6 that?  
 7 A I did that initial analysis. No, I did not.  
 8 Q And you're not an electrical engineer or have no  
 9 particular expertise in electricity, do you?  
 10 A No, I have no expertise in that area.  
 11 Q Who handled the administration of the electricity for the  
 12 tests that ended up in table 254?  
 13 A Dr. Reinemann suggested an individual, whose name escapes  
 14 me now, to design the device to administer the current.  
 15 Q And do you know - -  
 16 A So he designed the equipment to administer the current  
 17 to the cows. Do you have another question?  
 18 Q I want to make sure you're done with your answer.  
 19 A I was the one that actually attached it to the cows.  
 20 Q Do you know anything about the credentials, skills or  
 21 competence of the individual that designed the device?  
 22 A At the time I had looked at his credentials, but I don't  
 23 recall what they were.  
 24 Q And you lack credentials to make sure that the device was  
 25 properly attached to the cow, didn't you?  
 26 A Well, I can tell if it was properly attached. It had  
 27 indicator lights on it to indicate that it was working.  
 28 What I would lack is expertise in determining whether it  
 29 was properly designed.  
 30 Q And why did nobody else participate in the second study

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1 that you did?  
 2 A Well, Dr. Reinemann did participate in assisting in  
 3 designing the equipment and figuring out how to  
 4 administer the electricity to the cows, the current to  
 5 the cows. This was very heavily involved in looking at  
 6 the immune function. We weren't measuring a lot of other  
 7 things in it. And so, I was - for this part of that  
 8 study, and the time, we had no need to have other people  
 9 involved in there.  
 10 Q Well, in my experience, there is usually multiple authors  
 11 of these types of reports. You're the only one who's  
 12 indicated as an author on this study. Why is that?  
 13 A That's because I had prepared the report. And that's the  
 14 reason I put my name on there. There are a few other  
 15 people I probably could have put on there.  
 16 Q Had you had any kind of falling out with Dr. Reinemann?  
 17 A Not that I know. He may have felt so, but I don't recall  
 18 anything that was a falling out with him.  
 19 Q We talked to some extent when we talked the last time  
 20 that there's a difference between biological significance  
 21 and statistical significance?  
 22 A Yes.  
 23 Q What would you describe the term biological significance  
 24 to mean?  
 25 A Biological significance refers to affecting the overall  
 26 function of the organism in some way.  
 27 Q And did you see any evidence of biological significance  
 28 in either the first study or the second study?  
 29 A The second study, you really could not assess biological  
 30 significance in, because we were simply measuring

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1 Messenger RNA levels, not any higher level functions.  
 2 The first study we - the study was not big enough to  
 3 measure things like disease incidents. So, if you're  
 4 looking for that as a biological response, it would have  
 5 been an inadequate study for that.  
 6 In general, I would say little effect. You  
 7 might consider something like IgA to be a biological  
 8 significance. I wouldn't. I would consider biological  
 9 significance to mean something, such as in this case,  
 10 mastitis incidents, milk production or other such things  
 11 that would be of potential interest to a dairy farmer as  
 12 opposed to serum antibody levels, that's of more interest  
 13 to a researcher.  
 14 Q And you saw none of those symptoms?  
 15 A Well, we didn't report it, just because we knew the study  
 16 would be too small to report them.  
 17 Q And is there any conclusions that you can draw to a  
 18 reasonable degree of scientific certainty from either  
 19 study other than more study should be done?  
 20 A Regarding possible biological significance or?  
 21 Q Anything that you can draw - any conclusions, any  
 22 opinions that you can come to to a reasonable degree of  
 23 scientific certainty based on either study, other than  
 24 more study needs to be done?  
 25 MR. LAWRENCE: Object to form.  
 26 A All of the conclusions that I could come to reasonably  
 27 would be about things like Messenger RNA levels, or  
 28 levels of protein in the blood, and would be suggestive  
 29 of overall health effects at best. Rather than saying  
 30 health effects to a scientific certainty, if that's kind

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1 of what you are getting at, I guess I don't know quite  
 2 what you're getting at in the question.  
 3 Q Well, I think you indicated and your paper indicated that  
 4 there was some issues with assay collections and assay  
 5 analyzation, is that correct?  
 6 A Issues?  
 7 Q Problems.  
 8 A Well, early on, yes, some of the - some of the assays  
 9 were not as feasible as we thought they would be.  
 10 Q And problems with the assay collection and assay  
 11 analyzation is going to effect the data integrity?  
 12 A If not corrected, they could, yes.  
 13 Q And explain this to me. You say in some cases the  
 14 activity changed, but the expression level didn't, when  
 15 you're talking about genes. What do you mean by that?  
 16 A In the second study we are measuring Messenger RNA  
 17 levels. In the first study, we're measuring the levels  
 18 of protein in the blood. There's several steps in  
 19 between those. The first step in producing the protein  
 20 is called transcription.  
 21 This is the production of the Messenger RNA  
 22 using the DNA and the cells nucleus as a template. That  
 23 Messenger RNA then under-goes a process called  
 24 translation in which it's used as a template to produce a  
 25 protein sequence. In the case of proteins that are in  
 26 serum, that protein is then processed and excreted from  
 27 the cells. And what you measured in the serum is a  
 28 mixture of the rate at which that protein is secreted,  
 29 the volume that it gets distributed in the body, and the  
 30 rate at which it is destroyed by the body or excreted.

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1 So there are many steps between the  
 2 Messenger RNA and the actual protein being detected. For  
 3 some genes, it's possible to see a bigger change in one  
 4 than the other. They often parallel each other, but they  
 5 don't have to.  
 6 For example, for certain cytokines, there  
 7 are reports where the level of Messenger RNA changes more  
 8 than the level of protein in the blood does. And I  
 9 believe that's what I was referring to there, was the  
 10 fact that they're measuring very different things and  
 11 they don't always - one does not always reflect the other  
 12 one. This is because there are things affecting the  
 13 protein in addition to the amount of Messenger RNA that  
 14 is there.  
 15 Q So, Messenger RNA is associated with the electricity  
 16 that's being administered, or can be?  
 17 A The Messenger RNA could be affected by that. The  
 18 Messenger RNA - I'm trying to think how to explain this.  
 19 It's the first step in making a biological response, or  
 20 is often the first step in making the biological a  
 21 response.  
 22 Q And the biological response is the creation of proteins  
 23 to fight the antigens?  
 24 A That could be a biological response, yes.  
 25 Q But if I'm understanding you correct, some of the  
 26 proteins that were being observed were not necessarily  
 27 associated with the Messenger RNA?  
 28 A I think that what we're getting at here is two different  
 29 ideas. The Messenger RNA is not perfectly correlated  
 30 with the protein in terms of how much of it is there.

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1 That's the point I was trying to make. The Messenger RNA  
 2 corresponds to, in a certain sense, the protein in terms  
 3 of the sequence of the Messenger RNA determines the  
 4 sequence of the protein. But the level of the Messenger  
 5 RNA is one of several factors that determines how much of  
 6 the protein is being made.  
 7 Q But there is a relationship between the production of  
 8 antigen fighting protein and the Messenger RNA?  
 9 A Yes. There is a relationship. It is not a perfect  
 10 relationship. That was my point.  
 11 Q Okay. Why did you decide that your second paper wasn't  
 12 worth publishing?  
 13 A I never published it. It was a fairly - it's - although  
 14 there's a lot of Messengers there, it was a fairly small  
 15 study in terms of the sorts of things that were measured.  
 16 I looked at it and I thought as soon as I tried to  
 17 publish it, it's very likely that someone in the review  
 18 process would say, "But have you actually seen the change  
 19 in the protein levels," or other things that could have  
 20 been measured. And I - I just did not think it would  
 21 withstand peer review because of that possible criticism  
 22 of it.  
 23 Q So, not only was there a singular author, there was never  
 24 a peer review?  
 25 A No. That's correct.  
 26 Q Why was the study done the way it was?  
 27 A In terms of measuring the Messenger RNA?  
 28 Q Right.  
 29 A Well, that is the first step in producing these proteins.  
 30 And it's allowed us to assess a fairly large number of

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1 possible responses; whereas, measuring proteins is much  
 2 more tedious.  
 3 Q You talk at some length with Mr. Lawrence about block  
 4 analysis. Could you please explain that in a way that  
 5 somebody as dumb as me can understand it?  
 6 MR. LAWRENCE: Object to foundation.  
 7 A Yes. In this Part III, there were 12 cows total in each  
 8 treatment group. But they weren't all used at the same  
 9 time. We did the experiment once with a total of 8 cows  
 10 for treatments in 4 controls. Collected all of that data  
 11 at the same time from those 8 cows.  
 12 Sometime later, we did the same thing with  
 13 8 different cows. At some time later, the same thing  
 14 with a final 8 cows, for a total of 24 or 12 per  
 15 treatment.  
 16 A question comes up, are the conditions the  
 17 same in the first group and the second group? For  
 18 example, in dairy cattle research, this might be  
 19 reflected in the temperature of the barn.  
 20 Q We know the environmental conditions - -  
 21 A The environmental, it might change. Could that change  
 22 the results? And that is what is meant by the blocking  
 23 effect. And it's the statistical technique to correct  
 24 for the fact that the cows weren't - the three groups  
 25 weren't all at the same time, there was a sequential  
 26 factor to it.  
 27 Q So how do you correct for that?  
 28 A There's a fairly well known statistical technique that  
 29 does correct for it, called, in this case, it would be a  
 30 randomized block design. Off the top of my head, I could

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1 not describe the mathematics of it. But there is - it is  
 2 a statistical technique that, in doing an analysis of  
 3 variant, you have what is called a model, and then  
 4 residual effects. You include the three replicates as  
 5 part of the model.  
 6 Q But cow performance, stress on a cow, is going to be  
 7 different in January and May and August, isn't it?  
 8 A Yes. Yes.  
 9 Q Now, in the first test, 250, you talked about  
 10 co-variants. You did a comparison of the animals, what  
 11 their condition was when they started, and what their  
 12 condition was when you stopped the test in terms of  
 13 protein production, correct?  
 14 A That's right. Yes.  
 15 Q And you didn't do that in the second test?  
 16 A That's correct.  
 17 Q And what do you mean by covariant?  
 18 A A covariant is a variable that might influence the  
 19 results that you were trying to control for.  
 20 Q So, for example, when Mr. Lawrence pointed out to you  
 21 that the P value on exhibit 278 for IgA serum was .7932,  
 22 that was the beginning and end - that was based on data  
 23 at the beginning and end of the treatment, correct?  
 24 A Let me get to that exhibit. You're referring to 278?  
 25 Q 278, it's about the fifth box from the bottom. Do you  
 26 see there's handwritten in there, IgA serum?  
 27 A IgA serum.  
 28 Q And it shows a P value of .7932?  
 29 A Okay. Yes.  
 30 Q And that's not statistically significant because it's

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1 greater than .05, correct?  
 2 A That's correct.  
 3 Q And then, when you did the same analysis, as I understand  
 4 it, and looking then to page exhibit 279, page 37, you  
 5 got a P value of .0003, and that's based upon comparing  
 6 the same protein between two different groups of animals?  
 7 A That's correct.  
 8 Q So, if we compared the control group with the treatment  
 9 group, even before any treatment was administered, the  
 10 difference in the IgA serum levels was statistically  
 11 significant?  
 12 A Correct.  
 13 Q But when we compared the treatment group at the beginning  
 14 and at the end, the difference between the IgA serum  
 15 levels was not statistically significant?  
 16 A That's what this result indicates.  
 17 Q And the difference between the two is dramatic,  
 18 statistically speaking, right?  
 19 A Yes.  
 20 Q So, when you only made the comparison between the control  
 21 group and treatment group in the second study, not  
 22 knowing what the difference was between the two groups  
 23 when they started, that really calls into question any of  
 24 your data, doesn't it?  
 25 A The fact that we randomized the assignment of cows to  
 26 treatment should prevent that. I have no idea of why it  
 27 didn't in this case. But it does - it is something I  
 28 would concede as a possibility, yes.  
 29 Q Well, you thought the cows in the initial study were  
 30 randomly selected.

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1 A Well, they were randomly selected or randomly assigned to  
 2 treatments, and I do not know why there was a difference  
 3 initially at the start in IgA.  
 4 Q But there definitely was?  
 5 A There was, yes.  
 6 Q And that would indicate, as to this criteria, these cows  
 7 were not random?  
 8 A It just indicates that they were different. I don't know  
 9 if that gets into a question of what random means. But  
 10 we had - when we assigned them to the treatment, we had  
 11 no idea what the IgA levels were. So. But it is some-  
 12 thing that was different in the two groups.  
 13 Q I gotta hit the airport. Let's go off the record.  
 14  
 15 (3:24 o'clock p.m.)  
 16  
 17 \* \* \* \*  
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READING AND SIGNING CERTIFICATE

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I, LEWIS G. SHEFFIELD, do hereby certify that I have read the foregoing transcript of my deposition, recorded by John T. Kirby, of 5-9-14, and believe the same to be true and correct, (or except as follows, noting the page and line number of the change or addition and the reason why):

WRITING IN TRANSCRIPT WILL NOT BE ACCEPTED

\_\_\_\_\_  
DATE

\_\_\_\_\_  
SIGNATURE

1 STATE OF MINNESOTA )  
  ) ss.  
2 COUNTY OF DAKOTA )

3  
4 Be it known that I took the deposition of  
5 LEWIS G. SHEFFIELD, on the 9th day of May, 2014, at  
6 Madison, Wisconsin;

7 That I was then and there a notary public  
8 in and for the County of Dakota, State of Minnesota, and  
9 that by virtue thereof, I was duly authorized to  
10 administer an oath;

11 That the witness before testifying was by  
12 me first duly sworn to testify to the truth and nothing  
13 but the truth relative to said cause;

14 That the testimony of said witness was  
15 recorded in computerized Stenotype and thereafter  
16 transcribed by myself, and that the testimony is a true  
17 record of the testimony given by the witness to the best  
18 of my ability;

19 That I am not related to any of the parties  
20 hereto nor interested in the outcome of the matter;

21 That the reading and the signing has been  
22 executed as evidenced by the preceding page.

23  
24 WITNESS MY HAND AND SEAL THIS 12TH DAY OF MAY, 2014.  
25  
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