

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

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*Lewis G. Sheffield, Ph.D.*  
*March 14, 2014*  
*Volume 1*

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*Metropolitan Court Reporters, Inc.*  
*13306 Huntington Circle*  
*Apple Valley, Minnesota 55124*

1 STATE OF WISCONSIN CIRCUIT COURT TREMPEALEAU COUNTY  
 2 - - - - -  
 3 Paul Halderson and Case No. 12-CV-74  
 Lyn M. Halderson, Code Nos: 30303 & 30201  
 4 N17388 County Road  
 Galesville, Wisconsin 54630  
 5  
 6 and  
 7 Arctic View Farms, LLC  
 1919 Riley Rd.  
 8 Sparta, Wisconsin 54656,  
 Plaintiffs,  
 9  
 vs.  
 10 Star Blends LLC  
 11 1919 Riley Rd.  
 12 Sparta, Wisconsin 54656  
 and  
 13 ABC Insurance Company,  
 14 a fictitious company  
 15 and  
 16 Northern States Power Company  
 d/b/a Xcel Energy Services Inc.  
 17 1414 W. Hamlin Avenue  
 Eau Claire, WI 54702,  
 18 Defendants.  
 19 - - - - -  
 20 VOLUME I  
 21 Deposition of LEWIS G. SHEFFIELD, PhD, taken  
 22 pursuant to Notice of Taking Deposition, and taken  
 23 before John T. Kirby, a Notary Public in and for the  
 24 County of Dakota, State of Minnesota, on the 14th day  
 25 of March, 2014, at 1 South Pinckney Street, Madison,  
 26 Wisconsin, commencing at approximately 12:35, p.m.  
 27  
 28  
 29  
 30

1 EXHIBIT INDEX  
 2 249 9-21 251 28-20 253 56-14  
 3 250 18-28 252 (No ref) 254 67-8  
 4 EXAMINATIONS  
 5  
 6 By Mr. Thornton: 3.  
 7  
 8 By Mr. Lawrence: 41.  
 9  
 10 \* \* \* \*  
 11  
 12 WHEREUPON, the following proceedings were duly had:  
 13  
 14 \* \* \* \*  
 15  
 16 LEWIS G. SHEFFIELD, PhD,  
 17 an expert witness in the above matter,  
 18 after having been first duly sworn,  
 19 testified under oath as follows:  
 20  
 21 CROSS EXAMINATION  
 22  
 23 BY MR. THORNTON:  
 24  
 25 Q It's Dr. Sheffield, right?  
 26 A Whatever. It is. It is.  
 27 Q You have a PhD?  
 28 A I do have a PhD, yes.  
 29 Q As you just heard, doctor, I represent Northern States  
 30 Power Company in this lawsuit. Do you know anything

1 APPEARANCES:  
 2 Natalia Blaskovich, Esquire, of the firm  
 3 of REYNOLDS & KENLINE, LLP, 110 East Ninth Street, P.O.  
 4 Box 239, Dubuque, Iowa 52004-0239, 563-556-8000,  
 5 blaskovich@rkenline.com,  
 6 -and-  
 7 Scott Lawrence, Esquire, of the LAWRENCE  
 8 LAW OFFICE, S.C., 403 South Fourth Avenue, P.O. Box 117,  
 9 Saint Nazianz, Wisconsin 54232-0117, 920-773-2811,  
 10 ATTORNEYS@LDLAWSTN.COM, appeared jointly representing  
 11 the Plaintiffs.  
 12  
 13 Timothy R. Thornton, Esquire, of the firm  
 14 of BRIGGS & MORGAN, 2400 IDS Center, Minneapolis,  
 15 Minnesota 55402, 612-977-8400, tthornton@briggs.com,  
 16 appeared representing Defendant NSP/Xcel Energy.  
 17  
 18 Catherine M. Rottier, Esquire, of the firm  
 19 of BOARDMAN & CLARK, LLP, 1 South Pinckney Street, Suite  
 20 410, P.O. Box 927, Madison, Wisconsin 53701-0927,  
 21 608-257-9521, crottier@boardmanclark.com, appeared  
 22 representing Defendant Star Blends.  
 23  
 24 ALSO PRESENT:  
 25 Theresa A. Peterson, DVM.  
 26  
 27 VIDEOGRAPHER:  
 28 Mark C. Haskins, HASKINS MEDIA SERVICES,  
 29 1071 Whitney Drive, Apple Valley, Minnesota 55124,  
 30 952-997-6455, mark@haskinsmediaservices.com.

1 about this lawsuit?  
 2 A Very little. I knew it existed and that's about it.  
 3 Q All right. Have you talked to any of the lawyers except  
 4 for the brief conversation that you and I had on the  
 5 telephone?  
 6 A And introducing ourselves in the lobby. That was all.  
 7 Q What do you do for a living, doctor?  
 8 A I teach biology courses and occasionally chemistry at  
 9 MATC, Madison Area Technical College, in Portage.  
 10 Q How far is Portage from here?  
 11 A I don't know. Takes me about 45 minutes or so to drive  
 12 there. Mileage, I'm not sure.  
 13 Q And can you give me a brief overview of your educational  
 14 background?  
 15 A Yes. I received a bachelor of science degree from  
 16 Clemson University in animal science, stayed there for a  
 17 master's degree, received a masters in 1980.  
 18 Q Also in animal science?  
 19 A Well, it was called animal and food industries, but  
 20 essentially animal science, yes. 1983, I received a PhD  
 21 in dairy science at the University of Missouri, studying  
 22 mammary gland development. From '83 to '86, I was a  
 23 post --doctoral researcher at Michigan State University  
 24 with Dr. Shuford Welch, who is a breast cancer  
 25 researcher.  
 26 Q Is there an overlap between mammary development and  
 27 breast cancer?  
 28 A Yes.  
 29 Q And what did you do after 1986?  
 30 A I joined the faculty at the University of Wisconsin in

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1 the Department of Dairy Science.  
 2 Q Go ahead. I don't mean to interrupt you. Go ahead.  
 3 A I was just going to say, I was initially assistant  
 4 professor, left there in 2010 as full professor.  
 5 Q And was there any area of specialty that you had at the  
 6 University of Wisconsin?  
 7 A I worked predominantly on mammary gland development,  
 8 which is what my training was in.  
 9 Q Did you have any special focus on immunology or genetics?  
 10 A Immunology. I did work a little bit in immunology,  
 11 mostly on the stray voltage related work. I would not  
 12 have considered myself an immunologist, per se, but I did  
 13 do some work on that. Genetics, in terms of gene  
 14 expression, I did some work on that, if that has some  
 15 relationship to mammary gland development. So, I guess  
 16 that depends a little bit on how you define genetics.  
 17 Q What did you do - can you give me a brief overview of  
 18 what you did in the area of stray voltage?  
 19 A Yes. I don't recall the year, but sometime in the '90s,  
 20 a researcher that was affiliated was in a different  
 21 department, Ag. Engineering. Dr. Douglas Reinemann was  
 22 doing some research on stray voltage, and the question  
 23 arose: "Are there ways to measure physiological  
 24 responses that might be relevant to immunology, or  
 25 stress, in general?" And that began some work that we  
 26 did in collaboration with him, measuring various aspects  
 27 of immune function in dairy cattle that had been exposed  
 28 to voltages.  
 29 Initially, we were measuring - I'm trying  
 30 to think of the best way of wording it - levels of

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1 various proteins associated with immune function in the  
 2 blood, such as immunoglobulins and interleukins, which  
 3 are chemicals that regulate function of the immune  
 4 system. And we measured activity of neutrophil cells,  
 5 which are major phagocytic cells, and - -  
 6 Q Doctor, when you say some of the words like phagocytic,  
 7 it would help Mr. Kirby if you spelled them, because  
 8 you're probably the only one who can.  
 9 A Okay. Phagocytic. P-h-a-g-o-c-y-t-i-c.  
 10 THE REPORTER: Thank you.  
 11 Q Sorry I interrupted you.  
 12 A I apologize. If I use a term that you're not familiar  
 13 with, please let me know and I can define it. Phagocytic  
 14 simply means cell eat. And a group of cells called  
 15 lymphocytes. Lymphocytes are important in a variety of  
 16 aspects of immunology, and in the initial studies we  
 17 measured the ability of the lymphocytes to respond to  
 18 various stimulants. These are chemicals that are  
 19 recognized by the lymphocytes as something they should  
 20 respond to.  
 21 Q So, antigens, for example?  
 22 A Well, yes. Antigens, that's a good way of putting it.  
 23 That's accurate.  
 24 Q So, when a bacteria or a virus or some invader that  
 25 enters - -  
 26 A Yes. In fact, one of the things we measured in response  
 27 to was a particular bacteria, staphylococcus aureus.  
 28 Others were lectins from Pokeweed.  
 29 Q What is lectins?  
 30 A Lectin. It's a compound protein that binds to cell

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1 surface with receptors on the surface of lymphocytes, and  
 2 it often stimulates it to undergo cell division.  
 3 Later, we did another study in which we  
 4 measured the level of messenger RNA of certain genes,  
 5 some of which are associated with immune function. This  
 6 is - perhaps I should explain what messenger RNA is, is  
 7 each of the cells in the immune system has a set of  
 8 genes. Coding for - a wide variety of things that they  
 9 need to carry out their cell functions. The way they  
 10 express these proteins, the interleukins, for instance,  
 11 the anti-bodies are proteins, is, the, DNA is copied into  
 12 the intermediate molecule called Messenger RNA, a  
 13 processed called transformation. That is then used as a  
 14 template to direct synthesis of the specific protein, a  
 15 process called translation.  
 16 Early on, we had studied the production of  
 17 the actual proteins. We then studied the production of  
 18 specific messenger RNA's coding for those proteins.  
 19 Q And this work was done in cooperation in connection with  
 20 Dr. Reinemann?  
 21 A Yes.  
 22 Q He did the engineering side of it and you did the  
 23 biological side of it?  
 24 A Yes.  
 25 Q And did that culminate in a paper, a published paper?  
 26 A No, it did not.  
 27 Q What happened to that work?  
 28 A That, we never published that. We did not find a lot of  
 29 great statistically significant findings. Some of the  
 30 things were, I want to say inconsistent, erratic, and we

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1 did not pursue publication of that, or at least I did  
 2 not. I do not know if Doug might have published any of  
 3 that work or not.  
 4 Q What is CALS at the University of Wisconsin?  
 5 A College of Agriculture and Lab Science, that is the  
 6 College of Agriculture.  
 7 Q Is there a unit called the ICCUC that meets with experi-  
 8 menters to make sure that their work is statistically  
 9 correct and significant?  
 10 A That's not the purpose of that committee. That stands  
 11 for the Institutional Animal Care and Use Committee.  
 12 They monitor the use of experimental animals to make sure  
 13 appropriate animal welfare regulations are followed.  
 14 Q Is there any entity associated with the University of  
 15 Wisconsin that examines the statistical work of  
 16 researchers?  
 17 A None that I'm aware of.  
 18 Q Was there any review of the statistical work that you and  
 19 Dr. Reinemann did early on?  
 20 A The statistical analysis on this early work was done by  
 21 Steven LeMire.  
 22 Q Who is Steven LeMire?  
 23 A He was associated with Dr. Lyman. I do not recall the  
 24 details of that association, as to whether he was a  
 25 graduate student or research associate or what the  
 26 details were.  
 27 Q But was he a statistician or did he have expertise in  
 28 statistics?  
 29 A He had expertise in statistics. I do not know exactly  
 30 what his background was.

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1 Q And what's your basis for concluding that your early work  
2 didn't come up with anything that was statistically  
3 significant, and, in fact, was contradictory?  
4 A Well, I didn't say - I didn't mean to imply contra-  
5 dictory.  
6 Q Okay.  
7 A If you look at the work, we have a table here that is  
8 Table 2.  
9 Q What are you looking at now?  
10 A I'm looking at this file here (indicating).  
11 Q Okay. Can we mark that as an exhibit?  
12 A Yes. You have a copy of that. I sent you a copy of  
13 this.  
14 Q My people didn't give it to me, unfortunately.  
15 A Oh, okay.  
16 MR. LAWRENCE: Perhaps I can help. If  
17 that's the same data as the paper that was eventually  
18 published, I've got it along.  
19 Q I've got the published paper. This is the early work.  
20 Why don't we mark it as 249.  
21 A Okay. This is what I'm referring to.  
22 Q Okay. Can the court reporter mark your copy as 249?  
23 A Yes.  
24 Q We'll get it back to you. That's what you missed, Scott,  
25 first 248 exhibits.  
26 MR. LAWRENCE: Thank God.  
27 A Okay. I'm referring here to Table 2.  
28 Q Table 2. What page?  
29 A 22.  
30 Q Page 22. Okay.

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1 A Okay. Statistically, what these numbers mean, treatment  
2 is the effect of treatment, and treatment here is  
3 exposure to voltage. So, the smaller the number, the  
4 greater the degree of significance. Biologists generally  
5 want to see a number less than .05 to consider it  
6 statistically significant.  
7 Q You're talking about P values?  
8 A P values, that is what these numbers are.  
9 Q Maybe for the record, why don't you just explain what P  
10 value is?  
11 A P value is a measure of statistical significance. It  
12 ranges from zero to 1. And, although I'm not a  
13 statistician, so my interpretation here might not be  
14 exactly what a statistician would give, it is generally  
15 considered the probability of being wrong if you say  
16 there's a difference between two treatments. So, we want  
17 that number to be small.  
18 Q .05.  
19 A .05 is often used as the criteria.  
20 Q And that's 95 percent certain?  
21 A 95 percent certain that it's not due to random chance, or  
22 a 5 percent chance that it is due to random chance.  
23 Q And - -  
24 A And if we look down, most of these numbers are fairly  
25 large, with one major exception, and that is,  
26 immunoglobulin A. Serum IgA, and my lines are not  
27 numbered here, but it's very easy to find them.  
28 Q And that's the .360 number? No.  
29 A No, .015. So, Serum IgA, and the Treatment column,  
30 .015.

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1 Q Oh, got it.  
2 A The other thing that you see, if you look at the figures  
3 here, on page 25, you see things like, Figure 2, we have  
4 an elevation, or we have a level, it drops and then it  
5 comes back up. We don't see flat lines or lines that are  
6 diverging. I'm not sure what that means. It's what we  
7 found. But the only thing in here that we found that was  
8 statistically significant was the IgA levels.  
9 Q And that's under the treatment column, the .015?  
10 A Yes. Yes. Correct.  
11 Q And so you came to a conclusion that this research, to a  
12 reasonable degree of scientific certainty, didn't  
13 necessarily mean anything?  
14 MR. LAWRENCE: I'm going to object to the  
15 form as leading. All right. Go ahead.  
16 A I don't know if I would say it doesn't mean anything.  
17 Q Could you draw any conclusions from these data to a  
18 reasonable degree of scientific certainty?  
19 A To a reasonable degree of scientific certainty, this  
20 study, based on 12 treated and 12 controlled cows, showed  
21 a probability that IgA was lower in terms of statistics.  
22 That is not the same as biologic significance.  
23 Biological and statistical significance are different  
24 ideas.  
25 Q Two different animals, right?  
26 A Correct. Correct. So, statistically we saw a difference  
27 in Serum IgA.  
28 Q Could you say to a reasonable degree of scientific  
29 certainty that there was biological significance in  
30 anything you - -

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1 A I've always - - I'll let you finish the question.  
2 MR. LAWRENCE: Object to form. Go ahead.  
3 THE REPORTER: Wait until he finishes his  
4 question.  
5 Q I don't mind if you interrupt me and Mr. Lawrence, but it  
6 makes it hard on Mr. Kirby, and he's an old man, and we  
7 try and go easy on him.  
8 A I'm sorry. I've never done this before, so if I do  
9 something wrong, let me know.  
10 Q Just do your best not to interrupt Mr. Lawrence or I, and  
11 we'll do our best not to interrupt you, okay?  
12 A Okay. Now, biological significance. There were two  
13 observations, IL1 and IL2, that were close to significant  
14 statistically. The one that was significant was IgA.  
15 Now, an important part in interpreting these data is to  
16 know what IgA actually is. Ig stands for immunoglobulin.  
17 That's effectively is an antibody. The major immuno-  
18 globulin that circulates in blood is the immunoglobulin  
19 G4. There are different forms of these immunoglobulins.  
20 Immunoglobulin A makes a very minor contribution to  
21 immunoglobulins in circulation. It's importance is in  
22 what is called mucosal immunity. The mucosal tissue is  
23 what lines many of the cavities of the body and surfaces.  
24 For example, the lining much of the intestine is a  
25 mucosal tissue.  
26 Most of the IgA in the body is not found  
27 circulating, it's found associated with mucosal tissues.  
28 Q In the digestive tract?  
29 A And other surfaces. Digestive tract I used as an  
30 example, but there are many others, the lining of the

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1 lungs, the lining of the mammary gland, the lining of the  
2 genital urinary tract, and so forth.  
3 Q The areas of the body that are most likely to come in  
4 contact with antigens?  
5 A Yes. Those surfaces. So, where you would expect to find  
6 large amounts of IgA would be in lymphoid tissue, that is  
7 immune system tissue associated with surfaces, and  
8 secreted into the - sometimes into the secretions from  
9 these surfaces. So, a major question is whether the  
10 Serum IgA reflects the change in mucosal immunity or not.  
11 And I don't know the answer to that. It doesn't  
12 necessarily reflect a change in mucosal immunity. One  
13 could certainly imagine seeing no change in IgA or a  
14 change in Serum IgA that isn't reflective of mucosal  
15 immunity. It suggests a possibility. It doesn't  
16 establish it to a biological certainty.  
17 Q So, if you were going to attempt to draw any conclusions  
18 to a reasonable degree of biological certainty, these  
19 data don't enable you to do that?  
20 MR. LAWRENCE: Object to form. Leading.  
21 Go head.  
22 A Very rarely do you see a single study in which you can  
23 say something to certainty. I'll start with that. I  
24 would suggest that - it suggests the possibility that  
25 further work might be worth doing, but it doesn't  
26 establish a change in mucosal immunity.  
27 Q And ultimately you decided that these data were not  
28 significant enough or not certain enough to warrant  
29 publication, you and Dr. Reinemann?  
30 MR. LAWRENCE: Objection to form. Leading.

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1 Go ahead.  
2 A I do not know Dr. Reinemann's opinions. I only know  
3 mine. So I can't speak to Dr. Reinemann's opinions on  
4 this. I was not very excited about publishing it. I  
5 wouldn't object to publishing it, but I did not think it  
6 was a particularly exciting study from that standpoint.  
7 Q In the scientific community, what does it mean to have a  
8 paper peer reviewed?  
9 A The most scientific journals have an editorial board, and  
10 when a paper is submitted to those journals for possible  
11 publication, it is sent to reviewers who are, in the  
12 judgment of the editor at least, sufficiently expert in  
13 the subject matter of the paper to pass judgment on  
14 whether it meets that journal's criteria for publication.  
15 It varies considerably with a journal as to  
16 what that might mean. Some of the things that are  
17 typically evaluated are novelty of work. Is it reporting  
18 something that hasn't been reported before? Appropriate  
19 methodology, whether the right measures were made,  
20 appropriate controls, whether, statistically, whether the  
21 experiment was big enough, for example, did you use  
22 enough animals?  
23 And something that is a little harder, at  
24 least for me to get a grasp on, and that is, the signifi-  
25 cance of the finding; does it actually change the way we  
26 look at a particular field.  
27 The reviewers evaluate these. They send a  
28 report as, in my experience, always anonymously, back to  
29 the editor who then communicates this to the author as to  
30 whether the paper is acceptable for publications, needs

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1 some modification, a very common thing, maybe generally  
2 acceptable, but they have some questions, or is not  
3 acceptable for publication in that it doesn't meet some  
4 of the criteria.  
5 Q Does the validity or accuracy of the conclusions or  
6 findings have anything to do with the function of the  
7 reviewers?  
8 A I'm not sure what you mean by validity or accuracy.  
9 Q How about, let me restate it. Is there ever a situation  
10 where the reviewers say the experimentation, the data,  
11 simply doesn't support the conclusion?  
12 A Yes. If, as an example, if you do an experiment, you  
13 observe a certain observation, and you make inferences  
14 far beyond what your data will actually support, yes,  
15 that comment can be made.  
16 Q In any event, you decided 249 was not worthy of publica-  
17 tion?  
18 A That was my opinion.  
19 Q By the way, what's a Type 1 error?  
20 A I know the answer, I'm trying to think of how to explain  
21 it to you.  
22 Q You're a teacher?  
23 A Yes. Yes, but sometimes when I haven't explained  
24 something in a very long time, I have to think of it  
25 before I get into this.  
26 Q If you can explain it so that I can understand it.  
27 A I will use this experiment as an example. We had, for  
28 each of these measurements, we had two groups, control  
29 and treated. If I - I can make two decisions. The  
30 control and the treated are the same, they're equal, or I

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1 can decide that they're different. There is a truth,  
2 they either really are the same or they really are  
3 different. Now, if I say that they're the same, then  
4 they really are the same, there's no treatment effect.  
5 Then I haven't made a mistake. If I say they're  
6 different, and they're really different, then I haven't  
7 made a mistake. If I say they're the same, but they're  
8 really different, that is an error. And if I say that  
9 they're different, but they're really the same, that's  
10 also an error. But those two errors aren't the same. If  
11 I say they are different when, in fact, they're really  
12 the same, that's called a Type 1 error. And if I say  
13 they're the same when I really should have said they're  
14 different, that's called a Type 2 error.  
15 Q So you're focusing on the pre-treatment conditions of the  
16 animals to be able to make a valid comparison between the  
17 control and the treatment group?  
18 MR. LAWRENCE: Object to form. Leading.  
19 A No. We're focusing on the differences after applying  
20 treatment.  
21 Q But there can be pre-treatment differences between two  
22 groups of animals that are going to affect the end  
23 results after the treatment, is that correct?  
24 A That's possible, yes.  
25 Q And what did you do to ensure that, in your initial  
26 study, that the animals' pre-treatment really were the  
27 same?  
28 A The initial study, the first thing that's done in any  
29 study is randomization. You randomly assign animals to a  
30 treatment group, so that you don't, for example, take the

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1 12 highest milk production cows and call them a control  
2 in 12 lowest ones, treatment. Where they're housed in  
3 the barn is random, for example. We don't house all of  
4 the treatment group together and all of the control group  
5 together, in case there's some local environmental  
6 effect.  
7 So that's the first thing, and probably the  
8 most important in any experiment, is the randomization  
9 part.  
10 The second, in this initial study, is a  
11 technique called analysis of co-variance. Analysis of  
12 co-variance makes a measurement at the start of the  
13 experiment. It doesn't have to be the same as what  
14 you're measuring later, but it can be; and statistically  
15 correct for any difference there between the two, the  
16 groups.  
17 It's mostly the measure of actually  
18 reducing variability. And I'm not a statistician, so I'm  
19 hoping that makes sense. But that's the best I can do.  
20 Q In your initial study, you were looking for a number of  
21 outcomes?  
22 A Correct.  
23 Q And in the study, the Part 3 of the Minnesota Science  
24 Advisory, that also looked for a number of outcomes?  
25 A Correct.  
26 Q And in the unpublished abstract that you did, that looked  
27 for scores of outcomes?  
28 A That's correct.  
29 Q Have you ever heard of statistically the Bonferioni  
30 Adjustment?

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1 MR. LAWRENCE: Do you mean Bonferioni?  
2 A Bonferioni?  
3 Q Bonferioni.  
4 A Yes.  
5 Q You didn't use those in these studies?  
6 A I do not know that I used them for these studies or not.  
7 Q Okay.  
8 A I have heard of it though.  
9 Q So you didn't do the statistics, is that correct, doctor?  
10 A That's correct.  
11 Q What was the next project that you did relating to bovine  
12 immune systems after 249?  
13 A Okay. That was the study looking specifically at  
14 messenger RNA reference. When we started this, we  
15 thought it would be technically feasible to make very  
16 large numbers of measurements. We were able to get  
17 measurements on a reasonable number around the hundred or  
18 so.  
19 Q Of different immune responses?  
20 A Different messenger RNAs. Not all of them were related  
21 to immune responses. We included some that we were  
22 pretty sure wouldn't see an effect as a control for that.  
23 We also included some things that we shouldn't have even  
24 seen in the cells to make sure that we weren't detecting  
25 spurious signals. Does that answer - is that an answer  
26 to your question?  
27 Q Yes. Well, let me just ask you this. Exhibit number  
28 250. Do you have a copy?  
29 A Yes, I have a copy.  
30 Q Is that the next research that you did?

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1 A I believe he was referring to this (indicating).  
2 Q No, no. Part 3.  
3 A Oh. Okay. This is the same - that is this (indicating).  
4 This is just a different summary of this.  
5 Q So, 250 is just a summary of 249?  
6 A This is what was submitted to the Minnesota Public  
7 Utilities. This wasn't. (All indicating).  
8 Q But 250 is just a different compilation - -  
9 A 250 is a different compilation.  
10 Q - - of 249?  
11 A Of the same work. At least - -  
12 Q Go ahead.  
13 A To the best of my knowledge, that is true.  
14 Q And at the time 250 was submitted to the Minnesota  
15 Science Advisory, you're indicated to be a professor of  
16 dairy science?  
17 A That is correct.  
18 Q And Dr. Reinemann was just an associate professor?  
19 A That's what this says, and I don't recall, but that's  
20 what it says.  
21 Q And Steve LeMire, he was the guy who was in charge of the  
22 statistics?  
23 A He was in charge of the statistics. I don't know if he  
24 did other things as well, but that's correct.  
25 Q What did Morten Dam Rasmussen, PhD, do?  
26 A He was an associate of Dr. Reinemann's, and I'm not quite  
27 sure. Dr. Reinemann felt his name should be associated  
28 with it, I do not know why.  
29 Q What about Milo Wiltbank?  
30 A Milo Wiltbank did some of the assays. I believe the

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1 assays he did were the assays for the hormone cortisol.  
2 C-o-r-t-i-s-o-l.  
3 Q Why don't you explain for the record what assays are?  
4 A-s-s-a-y-s?  
5 A Correct. Measurements.  
6 Q And there is a certain number of - at least in exhibit  
7 250, there were a certain number of indicators or markers  
8 of the immune's function response that were identified  
9 for observation and testing. Why did you identify and  
10 test those?  
11 A They are often used in immunology to assess immune  
12 function. They are accessible, meaning they were things  
13 we had the ability to actually assay. Those were the  
14 major reasons.  
15 Q When we talk about immune response, are there two types  
16 of proteins and cells, those that stimulate an immune  
17 response and those that control an immune response?  
18 A There are many proteins in the body that affect the  
19 immune response. Affect spelled with an A. Some of them  
20 stimulate certain activities, some of them inhibit  
21 certain activities. For example, there are pathways that  
22 stimulate antibody production, there are pathways that  
23 inhibit antibody production.  
24 Not being an immunologist, I am not quite  
25 sure how to answer that, but I'm not sure I would say  
26 that it's quite as simple as a protein is always either  
27 one or always the other.  
28 Q And if you look at the abstract of 250, the last sentence  
29 says, "Correctively, these results suggest that exposure  
30 to 1 milliamp of current for two weeks has no significant

Page 21

1 effect on the immune function of dairy cattle." Was that  
 2 the conclusion of this report?  
 3 A I did not write that, but that was the conclusion of this  
 4 report.  
 5 Q Was this report peer reviewed?  
 6 A Not to my knowledge.  
 7 Q Is peer review the gold standard in your business?  
 8 A For publication awards, it certainly is. For - yes, I  
 9 guess I could say that that's correct.  
 10 Q Now, the study reflected by 249 and 250, which is the  
 11 same data, I guess you're telling me, was that in a  
 12 stanchion barn or a free-stall barn?  
 13 A This was in a stanchion barn.  
 14 Q And how the current was delivered to the animals?  
 15 A It was a long time ago, but if I recall correctly, there  
 16 were special stalls constructed with conductive mats on  
 17 the floor, and I believe it was an AC. I'm not the  
 18 engineer, so I may be remembering this wrong. But it was  
 19 delivered through the floor of the stanchion, if I recall  
 20 right. I didn't design the stalls, so I am relying on an  
 21 old memory here to answer that.  
 22 Q And the animal was tethered in the stall so she couldn't  
 23 escape the introduction of the electricity?  
 24 A I believe that is correct.  
 25 Q And going on the second page of exhibit number 250, the  
 26 first full sentence says, "The consensus of the science  
 27 advisors was that current in the earth can only interact  
 28 with dairy cows through their associated electrical  
 29 fields, magnetic fields and voltages, and that these  
 30 parameters should be the focus of the analysis." Do you

Page 22

1 know what the author is attempting to say there?  
 2 A I am not entirely sure. Or - -  
 3 Q Let me - well, go ahead.  
 4 A I was just looking at it and seeing if I could think of a  
 5 different way of saying the same thing. If we have a  
 6 current in the earth, you need to create an electric  
 7 field that the cow is exposed to, current flow and  
 8 electric field and the magnetic field. So, either the  
 9 electric field or the magnetic field interact with the  
 10 cow or the current flows through the cow. I believe  
 11 that's what you're trying to say, but I'm actually not  
 12 sure that would be.  
 13 Q Would you agree with me that the early research on stray  
 14 voltage primarily focused on behavioral responses?  
 15 A What I am familiar with, that's correct. There may be  
 16 some things that I'm not familiar with, but that's what I  
 17 am familiar with.  
 18 Q And the point of this research that you and Dr. Reinemann  
 19 were involved in was to see if there were other  
 20 responses?  
 21 A Yes.  
 22 Q And specifically, if there were immune responses?  
 23 A Correct.  
 24 Q Now, exhibit number 250 and 249, both of which tested the  
 25 control group and the test group before the treatment,  
 26 correct?  
 27 A I believe that's correct, yes.  
 28 Q So you had not only a comparison between the test group  
 29 and the control group, but you had a comparison before  
 30 and after the test was conducted?

Page 23

1 A That's correct.  
 2 Q And that's the best way to do it, isn't it?  
 3 A That is a powerful way of doing it. I don't know if I  
 4 would say it's the best. There are many possible ways,  
 5 but that certainly is a way of reducing the variability.  
 6 Q It is a more powerful way of reducing variability than to  
 7 just compare the test group to the control group after  
 8 the test is completed with no baseline comparison?  
 9 A Under most situations, that's correct. It would be - it  
 10 is technically possible that that's not correct, but  
 11 those situations would be pretty rare.  
 12 Q The second last sentence in the last full paragraph on  
 13 page 2 says, "The absence of significant changes in these  
 14 laboratory data in treatment cattle over time (each cow  
 15 serving as her own control), as well as a lack of  
 16 difference between treatment and control cows, indicate  
 17 there was no alteration in circulating volume or  
 18 acid-base balance, nor was there significant stress (as  
 19 measured by glucose concentration) or muscle injury  
 20 inflicted by the treatment." Are you talking there - or  
 21 is the author there talking about the testing you did or  
 22 testing that had been done?  
 23 A I believe - let me read the whole paragraph for a moment  
 24 to put it in context.  
 25 Q All right.  
 26 A I believe that is referring to this study cited  
 27 Reinemann, et al, 1996, which is in the references of  
 28 this.  
 29 Q Okay.  
 30 A That is not referring to this particular study.

Page 24

1 Q If you look on page 3 under Objectives, that was the  
 2 objective of the study that's reflected by this paper?  
 3 A Excuse me?  
 4 Q Page 3, where it says Objectives, that was what this  
 5 study hoped to accomplish, by this study that's reflected  
 6 by exhibit 250?  
 7 A That, referring to the immune function?  
 8 Q Yes.  
 9 A Correct.  
 10 Q And do you agree with me that the stress that an animal  
 11 is subjected to, is, in part, related to herd management?  
 12 A Yes.  
 13 Q And the way one group of cows might be treated, if it was  
 14 different than the way another group of cows is treated,  
 15 you might expect to see different stress responses?  
 16 A Could you repeat that?  
 17 Q Yes. If you had two herds, and their daily protocol,  
 18 their daily management, was different, one would expect  
 19 to see different stress responses in those two different  
 20 herds?  
 21 A That's certainly possible.  
 22 Q And on the bottom of page 3, the last paragraph on the  
 23 bottom of page 3 talks about how this herd was managed.  
 24 A Okay. Yes.  
 25 Q And it wouldn't be appropriate to draw necessarily a  
 26 comparison between this management style and a completely  
 27 different management style?  
 28 A (No response).  
 29 Q It looks like you're struggling with the question.  
 30 A I'm struggling a bit for several reasons here. If I

Page 25

1 understand the question correctly, you're asking, can we  
 2 extend the results from this study, which was done in a  
 3 stanchion barn, UW Madison herd, their particular  
 4 genetics and so forth.  
 5 Q Milk two times a day?  
 6 A Milked twice a day, fed a certain type of ration.  
 7 Q May or may not have been administered BST, we don't know  
 8 that.  
 9 A I do not recall when UW started using BST.  
 10 MR. LAWRENCE: I'll straighten that out. I  
 11 don't mean to interrupt, but it's discussed in a lot of  
 12 the page 3 also, if that helps.  
 13 A The extent to which these results could be extended to  
 14 other herds. Basic biology is still constant. I mean,  
 15 there are certain principles of biology that can be  
 16 extended. Certainly it limits some types of responses.  
 17 For example, if you are - and I believe there is research  
 18 to support this, although it has been a long time since I  
 19 looked at it, an animal that is housed in such a way that  
 20 it can avoid a stress shows less of a stress response  
 21 than one that's housed in such a way that it can't avoid  
 22 the stress.  
 23 Q So, extrapolating that, an animal in a free stall barn  
 24 that can avoid a stress is going to have less stress than  
 25 an animal in a stanchion barn where the stress is  
 26 administered?  
 27 MR. LAWRENCE: I'll object to the form as  
 28 leading. Go ahead.  
 29 A There are some studies that would suggest that, if I  
 30 remember the literature correctly. To state that that's

Page 26

1 always true I think is a bit of an overstatement. So.  
 2 Q It could be true?  
 3 A It could be true, but I wouldn't say it is true.  
 4 Q You wouldn't say it's always true?  
 5 A I wouldn't say it's always true.  
 6 Q All right. Then, on Page 4, the bottom of the last  
 7 paragraph says, "The differences of the treatment cows  
 8 were compared to the differences of the control cows  
 9 using and independent t-test." What's a t-test?  
 10 A It is a statistical method of determining significance.  
 11 Q On Page 6, how is it determined when the blood samples  
 12 were collected? The samples were collected - -  
 13 A Are you referring here to the duration of - I'm not sure  
 14 what exactly you're referring to.  
 15 Q It says, "Samples were collected for one week before  
 16 exposure and for the two weeks of exposure."  
 17 A Yes.  
 18 Q Who decided how and when to collect the blood samples and  
 19 what to make the comparisons to? I mean, that sounds  
 20 like more biology than it does engineering.  
 21 A It does. One of the factors - are you referring here to  
 22 the duration of the collection, that it was for, say two  
 23 weeks of treatment, or to the exact date it was collected  
 24 on?  
 25 Q The latter.  
 26 A The latter. Okay. One of the things - let me refresh  
 27 myself on how often we actually didn't collect these.  
 28 One aspect of this is, these - some of these assays are  
 29 quite difficult to conduct. It can't be conducted on  
 30 stored samples. Some of the things like serum cortisol

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1 levels, if you put the sample in the freezer and do the  
 2 analysis at anytime. But some of these tests require  
 3 living tissue collected from the cow, and they take  
 4 several days to actually conduct.  
 5 That timing allowed us the opportunity to  
 6 collect the sample, process it and then go back and  
 7 collect and process the next sample. We simply didn't  
 8 have the personnel to, for example, take twice daily  
 9 samples and process all of that for some of the assays  
 10 that we were doing, like chemiluminescence in the  
 11 lymphocyte blastogenesis assay, in particular, are vary  
 12 laborious assays.  
 13 Q On page 9, are those data the same data that were in 249?  
 14 Because I noticed it in IgA serum, the mean difference is  
 15 .017 rather than .015.  
 16 A I believe that it's based on the same row data set, but  
 17 we did some slight differences in, I think, - I think  
 18 that is reflected in a statistical difference that was  
 19 made in how the details of how the statistics were  
 20 analyzed that makes a slight difference in the exact  
 21 number. But I - it does appear that these should have  
 22 been the same data. There was only one data set with all  
 23 of this.  
 24 What's in this first report may have been  
 25 analyzed by a slightly different technique, and so the  
 26 numbers may show very small differences, like the  
 27 difference between .017 and .015.  
 28 Q But just from looking at, with my rudimentary  
 29 understanding of P-values and statistics, there isn't  
 30 anything in the far righthand column that's less than

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1 .05, is there? Well, I guess there is, staph. aureas.  
 2 A Staphylococcus aureas. I did not do those statistics.  
 3 So that detailed independent t-test. Okay. Let me see  
 4 what the difference is. Staphylococcus aureas. I am not  
 5 sure what that refers to.  
 6 Q Okay.  
 7 A That is something that a statistician did that I do not  
 8 know what that even refers to.  
 9 Q Take a look at page 13, Conclusion. The conclusion says,  
 10 "Collectively, these results suggest that exposure to 1  
 11 milliamp of 60 hertz electrical current for two weeks had  
 12 no significant effect on immune function of dairy  
 13 cattle." Was that the conclusion of this study?  
 14 A That would have been the collected conclusion of the  
 15 authors in the study.  
 16 Q And you were one of the authors?  
 17 A I was one of the authors. There is, like I said, the one  
 18 observation that was significant.  
 19 Q Now, 251 is your abstract?  
 20 A That's correct.  
 21 Q Is that the next research that was done on this subject  
 22 matter?  
 23 A That I was involved in, that's correct.  
 24 Q And why was this abstract never published, Dr. Sheffield?  
 25 A I had - for basically the same reasons as the first. The  
 26 study had not shown a lot of significant effects, and I  
 27 doubted it would stand peer review.  
 28 Q Why did you doubt that it would stand peer review?  
 29 A By the time - when we started this study, the technology  
 30 we were trying to use was in its infancy.



Page 29

1 Q A ray analyzer?  
 2 A Yes.  
 3 Q Did you have a lot of trouble with that?  
 4 A Yes. As we progressed through this, some of the things  
 5 that we would liked to have done were technically not  
 6 feasible, at least not at the time with the technology  
 7 that we had available.  
 8 Q The ray analyzer was a new - -  
 9 A It was a very new technology and it really had not been  
 10 applied to cattle at the time.  
 11 Q And the notes, underlying notes, seems to suggest that  
 12 your lab assistant had some difficulty using that  
 13 technology?  
 14 A That's correct.  
 15 Q And you heard of the expression, garbage in, garbage out?  
 16 A Yes.  
 17 Q And there's some aspect of garbage in, garbage out in the  
 18 data that were generated, is that correct?  
 19 MR. LAWRENCE: Object to form. Leading.  
 20 A I don't know if I would say in the data that were  
 21 generated. The - well, I'll just say that.  
 22 Q Well, is there some reason to believe that the underlying  
 23 data that were suspect because of the new technology and  
 24 the unfamiliarity of the people who were applying that  
 25 new technology?  
 26 A I think the data included here are as reliable as we  
 27 could have made them.  
 28 Q I understand that. I know that - -  
 29 A You're asking me about technical abilities. Certainly,  
 30 today there are much better ways of doing it than what we

Page 30

1 did.  
 2 Q I'm not suggesting - -  
 3 A By today's standards, the results would be very noisy.  
 4 Q By the way, was there any observation in the work that  
 5 you and Dr. Reinemann did before exhibit 251 of a drop-  
 6 off in milk production or an adverse effects on animal  
 7 health associated with the administration of electrical  
 8 currents to the animals?  
 9 A We did not notice any change in milk production.  
 10 Q And is that true - - go ahead.  
 11 A And, well, you asked also about animal health. The  
 12 numbers would have been pretty small to have detected any  
 13 health effects at all.  
 14 Q And is that true with the animals in the experiment of  
 15 251, no drop-off in milk production?  
 16 A I do not recall any.  
 17 Q Now, in the abstract, the last sentence says, "These  
 18 results suggest that electrical effects on disease  
 19 processes are likely to be modest, probably more long-  
 20 term and likely to be very difficult to detect in small  
 21 samples." Was that the conclusion of this study?  
 22 A That's what I would have concluded, yes. Perhaps I  
 23 should define modest, meaning, we basically found, out of  
 24 a hundred genes, only a couple of things were actually  
 25 different that we could detect at all, and when you're  
 26 doing the hundred statistical test, you expect a certain  
 27 number of false resuts.  
 28 Q False positives?  
 29 A Yes.  
 30 Q So, some of the results that you see could just as likely

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1 have been attributed to false positives as they could be  
 2 to the effects of the administration of - -  
 3 A I don't know if I - -  
 4 MR. LAWRENCE: Object to form. Go ahead.  
 5 A I don't know if I would say just as likely, but it could  
 6 be.  
 7 Q And you also said there could be a Type 1 error in the  
 8 data that you generated?  
 9 A I probably said that in the caveats here somewhere. That  
 10 sounds like something that would be in here.  
 11 Q And again, this was in a stanchion barn?  
 12 A Correct.  
 13 Q And unlike exposure in a real life situation, you  
 14 essentially attached electrodes to the legs of the  
 15 animals so they were constantly administered electric  
 16 current?  
 17 A Correct.  
 18 Q They couldn't avoid it?  
 19 A Not without physically detaching the electrodes by  
 20 rubbing against the stanchion.  
 21 Q Did that happen?  
 22 A Well, we did check those. Each time the cows were  
 23 milked, that got checked. You will occasionally - we  
 24 would on occasion see the electrodes detached. They were  
 25 immediately repaired. But, in general, they did stay in  
 26 place.  
 27 Q And in the last paragraph on the second page, first  
 28 sentence, it says, "In a previous study." Is that  
 29 referring to the study that you and Dr. Reinemann did?  
 30 It says, "In a previous study, we observed that

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1 electrical exposure of dairy cattle had minimal effect on  
 2 most immune function measures, including  
 3 chemiluminescence, lympho - how do you pronounce it?  
 4 A Lymphocyte blastogenesis, is how it's pronounced.  
 5 Q So that was - -  
 6 A That refers to the previous study that we've just  
 7 discussed, yes.  
 8 Q All right. And you're talking about interleukin 1  
 9 approached significance of less than .01, but I thought  
 10 statistical significance was less than .05.  
 11 A Where do you see this? Next sentence. "Increase in  
 12 serum interleukin 1 approached significance at P of less  
 13 than .01."  
 14 A No. .10.  
 15 Q Excuse me. .10.  
 16 A That is greater than .05. That's why we say "approached"  
 17 rather than "reached."  
 18 Q So that could be attributable to chance?  
 19 A Anything can be attributable to chance. It's more likely  
 20 to be chance than if it were a smaller number. That's  
 21 what that means.  
 22 Q In your business, .05 is what's regarded - -  
 23 A Most - - excuse me. You're correct. Most biologists  
 24 consider .05 to be, for lack of a better word, the gold  
 25 standard.  
 26 Q And it says underneath the animals, so the CALSIACUC,  
 27 that makes sure you're not abusing the animals?  
 28 A That's correct. I served on that committee, so, if you  
 29 want, I could discuss for you what they do. But that's a  
 30 short version and accurate enough.

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1 Q Bottom line is, you're not electrocuting the animals?  
 2 A That's correct. If we had tried to give them a voltage  
 3 that the committee felt was truly dangerous, for example,  
 4 we're going to use 110 volt 20 amps, which is quite  
 5 serious stress.  
 6 Q Probably wouldn't get the assignment.  
 7 A You probably wouldn't get the approval to do that. But  
 8 that is what they assess, yes.  
 9 Q Do they look at what they do with the rhesus monkeys and  
 10 the mice and the rats?  
 11 A Well, the college of agriculture's committee isn't the  
 12 one that does that. But there is a committee that does.  
 13 Any vertebrae animal research goes through such a  
 14 committee at the University of Wisconsin.  
 15 Q Hopes of keeping the PETA people happen, huh?  
 16 A I wouldn't comment on that.  
 17 MR. LAWRENCE: If you ask if that's true in  
 18 Harry Harlow's days, it was.  
 19 A These laws are more recent than Dr. Harlow's work. His  
 20 work would not have been subjected to that.  
 21 MR. LAWRENCE: Thank you.  
 22 Q Then you say underneath Animals, "Blood samples was  
 23 collected - probably should be were collected - via the  
 24 tail vein immediately prior to applying the current and  
 25 at a end of a three week exposure period." So you  
 26 took two blood samples, one at the beginning and one at  
 27 the end?  
 28 A That's correct.  
 29 Q How come your data doesn't reveal anywhere what the blood  
 30 samples showed at the beginning of the test?

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1 A I do not know.  
 2 Q Your comparison is between the control group and the test  
 3 group?  
 4 A That is the comparison we did.  
 5 Q No cow to cow - within cow comparison?  
 6 A No, as I recall, we did not do that.  
 7 Q And then, if you look at, it's not numbered, but the next  
 8 page, under Results and Discussion. Are you with me?  
 9 A I'm with you, yes.  
 10 Q You said, "Most measures were not affected, suggesting  
 11 that those that were could be Type 1 errors, due to a  
 12 large number of hypotheses tested." What do you mean by  
 13 that, doctor?  
 14 A You mentioned earlier - well, I guess you explained it.  
 15 If you measure one thing and you have a Type 1 error of 5  
 16 percent, there's a 5 percent chance that if you measured  
 17 the conclusion, that there's a difference, there's a 5  
 18 percent chance of being wrong. Follow me so far?  
 19 Q I think so.  
 20 A If I'm measuring one thing, let's say milk production,  
 21 and I should conclude milk production was changed at a P  
 22 value or Type 1 error P value of .05, there's a 5 percent  
 23 chance to be wrong. If I measure two things, there's a 5  
 24 percent change of each one, of either one. So, the  
 25 chance that at least one of them is a Type 1 error goes  
 26 up. The more things you measure, the greater the chance  
 27 that at least one of them will show a difference even  
 28 though it wasn't really there.  
 29 Q Kind of like, if you flip a coin a hundred times, every  
 30 time you flip it, there's 50-50 chance it will be tails.

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1 But the chances that if you'll be able to flip it a  
 2 hundred times, it'll come up tails becomes less and less?  
 3 A I don't know if that's a good analogy or not. But.  
 4 Q But the point is, one of the reasons the Bonferioni, if  
 5 I'm pronouncing that correctly, the adjustment is, is  
 6 taking into consideration that possibility?  
 7 A Yes. Now, that is a major issue in statistics anytime  
 8 you're making mini comparisons. And the Bonferioni  
 9 approach - again, I'm not a statistician, but I think I'm  
 10 getting this close to right. The critical question is,  
 11 what should the Type 1 error rate be based on? Each  
 12 individual comparison for the whole experiment. And  
 13 there's great debate, at least in my understanding, among  
 14 statisticians about how to correctly do those  
 15 corrections. The Bonferioni is one approach. Some  
 16 statisticians criticize it by saying it over-corrects.  
 17 But that is the idea of the Bonferioni approach, is to  
 18 correct that.  
 19 Q But what you're saying in your reference to Type 1  
 20 errors, when you were studying dozens of outcomes and  
 21 only three or four showed it's a statistically  
 22 significant difference, that could be due to chance?  
 23 A It could always be due to chance. It's more likely to be  
 24 due to chance when you've got a large number of  
 25 comparisons and a small number of significant results.  
 26 Q That's why many scientific studies just look for one  
 27 outcome?  
 28 A I don't know. Many scientific studies, at least in large  
 29 animals, look for multiple outcomes. So, I guess I don't  
 30 know if I would have an opinion on that one way or the

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1 other.  
 2 Q Now, on the last page of your report, you say, "In  
 3 conclusion, these studies suggest that electrical impacts  
 4 on immune function are of relatively small impact  
 5 compared with infection and inflammation." What are you  
 6 saying there, doctor?  
 7 A All right. Good question.  
 8 Q All my questions are good.  
 9 A This is important. Let's suppose we took a cow and,  
 10 whether intentionally or unintentionally, gave her  
 11 something that the immune system recognizes as poor, this  
 12 happens with vaccination, for example, if you give her a  
 13 vaccine or intentionally give an infection.  
 14 Q That's what a vaccine is, is introducing a foreign --  
 15 A Yes.  
 16 Q -- entity into the animal.  
 17 A The immune system responds very strongly to these. When  
 18 that happens, you see major changes in the immune  
 19 functions, much larger than what we saw here. And that's  
 20 what I was referring to there. The magnitude of the  
 21 changes that we did see are generally small compared to  
 22 what you would expect to see if the cow were truly ill.  
 23 For example, if you gave the cow a strong vaccine, you  
 24 would expect to see bigger changes than this in at least  
 25 some of the immune function measures. That's what that's  
 26 referring to.  
 27 Q So, if what you were observing was biologically  
 28 significant as opposed to statistically significant, you  
 29 would expect to see a much greater reaction.  
 30 MR. LAWRENCE: Object to form. Leading.

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1 Go head.

2 A Not necessarily. And here is the reason - well, a reason  
3 for this, and a weakness of this study, by the way. So  
4 I'm kind of being critical of myself, but I think you  
5 should do that. This study measures base line responses,  
6 what the base line is. These cows were, as far as we  
7 knew, healthy. They weren't being exposed to any known  
8 pathogens other than the things that's normally in their  
9 environment.

10 An important thing to remember about the  
11 immune system, you don't really want the immune system to  
12 be active all the time, because it's very damaging.  
13 Inflammation is very damaging, but it's also very  
14 beneficial because it gets rid of infections.

15 What we didn't look at in this study was  
16 how strongly and rapidly the immune system responds to a  
17 challenge. So, what we looked at was, you got a base  
18 line here, and that base line didn't change.

19 A second important question that we didn't  
20 assess was, if you give a challenge, a vaccine or a  
21 disease, would the immune system respond strongly or  
22 would in one group the response be less than the other  
23 group? So, what's not assessed here is that ability of  
24 the immune system to respond to a challenge. But in  
25 terms of base line, we didn't see, except for, I believe  
26 it was IgA, we did see a drop in the IgA message. But  
27 other than that, the base lines were the same.

28 Q So, is there sufficient or insufficient data here to be  
29 able to draw any conclusions to a reasonable degree of  
30 scientific certainty about the animal's immune systems

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1 ability to respond to an insult?

2 A This says very little, if anything, about ability to  
3 respond to an insult. It's just a base line study.

4 Q And then you go on to say, "Any effects observed appear  
5 to affect only a small set of immune response regulators,  
6 compared to most disease processes, which affect a wider  
7 spectrum of regulators." Are you saying there that, when  
8 you introduce a real disease, there's a much more robust  
9 response in the animals than the response you saw to the  
10 administration of current?

11 A I think that's what I was trying to say, yes.

12 Q And that you conclude by saying, "As a result, the  
13 impacts of electrical exposure on animal health and  
14 disease is likely to be difficult to detect reliably,  
15 particularly without examining a large population." We  
16 need to study a whole lot more animals before we can come  
17 to any conclusions. Is that what that means?

18 A Okay. That sentence is referring to disease processes.  
19 So. One of the questions that comes up is, even if you  
20 see something such as, say a change in IgA levels, if you  
21 see that, will that indicate that this animal is more  
22 susceptible or less susceptible, depending on what you  
23 see, to a disease. To actually study a disease itself  
24 generally takes a very large number of animals, much  
25 larger than what was involved here.

26 For example, if you look at mastitis, small  
27 studies don't have a lot of what is called statistical  
28 power, and that's what I was trying to get at here,  
29 although it probably didn't do a very good job of  
30 explaining. Remember this Type 1 error, if you say

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1 there's a difference, you might be wrong. That's the  
2 Type 1 error. The Type 2 error is also important, that  
3 is, if you say there is no difference, you could still be  
4 wrong about that also. That's called a Type 2 error.  
5 That's the biggest reason why we use the largest number  
6 that's practical in experiments.

7 To have a low Type 1 error depends on  
8 several factors. One is, it depends on how big an effect  
9 you're looking for. If I want to see something with a 10  
10 percent change, that's going to be the larger Type 1  
11 error than if I'm looking for 10 volt change.

12 Another thing that influences Type 1 error  
13 is what you said calls significant, what your P value is.  
14 Most biologists use .05.

15 Q Even .05, there's a 5 percent chance of being wrong?  
16 A If you say there's a difference. It's a little more  
17 complicated than that. There's a 5 percent chance that  
18 you would see that big a difference by random chance.  
19 It's not quite the same as you'd be wrong.

20 But a big factor that influences Type 1  
21 error, that you have a lot of control over, is not just  
22 how big an effect you're looking for, but also how many  
23 animals you use. The bigger your experiment, the more  
24 reliably you can say that there is no difference when you  
25 make that conclusion. And that's what takes large  
26 numbers of animals - that's what was trying to get at  
27 here.

28 If you wanted to study disease, if I take a  
29 dozen animals and look at an instance of a particular  
30 disease, I'm probably not going to find any difference,

Page 40

1 simply because 12 animals for most diseases is not merely  
2 enough. So, for a study of actual disease instance, and  
3 whether something affects that, does take very large  
4 numbers of animals.

5 Q So, is it fair to say, doctor, that, based upon Dr.  
6 Reinemann and your joint studies, and the abstract that  
7 you did, the data simply isn't sufficient to draw any  
8 conclusions to a reasonable degree of scientific  
9 certainty about disease effects on animals associated  
10 with electricity?

11 MR. LAWRENCE: Object to form. Leading.

12 A We did not measure a disease itself. That's important to  
13 know. We measured some things that may be correlated to  
14 sensitivity to disease. We found most of those measures  
15 were unchanged, and a few were changed, few enough that I  
16 cannot reliably conclude that it's not due to random  
17 change.

18 Q Now, last topic. You were a full professor at the  
19 University of Wisconsin, tenured, specialized in the area  
20 of mammary gland development?

21 A Correct.

22 Q You are now teaching at a junior college in a suburban or  
23 a small town in Wisconsin. How come?

24 A I reached a point in my life where I simply disliked the  
25 various stresses associated with doing research and  
26 wanted to do something that was more pure teaching.

27 Q Got tired of publish or perish?

28 A And getting grants and various other stresses. My life,  
29 I don't know that the job is any easier, but I find it  
30 more enjoyable.

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1 Q So now you're teaching basic biology rather than highly  
2 specialized mammary gland development?  
3 A That's correct. Yes. I teach - this semester I'm  
4 teaching not only Physiology I, mostly to nursing  
5 students, Anatomy Physiology II, the microbiology.  
6 Q Your choice?  
7 A Yes.  
8 Q That's all I have.  
9 MR. LAWRENCE: I have a bunch, doctor.  
10 We've been going about an hour and a half. If you'd like  
11 a break, we can take one, if not, we can go for a while  
12 longer.  
13 MR. THORNTON: I have to leave at 3:30 to  
14 catch an airplane.  
15 A Actually, I'm fine.  
16  
17 (At this time a recess was taken - 2:00 to 2:09).  
18  
19 RE-DIRECT EXAMINATION  
20  
21 BY MR. LAWRENCE:  
22  
23 Q Mr. Sheffield, let's go back to exhibit 249 and 250 for a  
24 moment, please, and I would like to look at Table 2 on  
25 those two documents with you for a moment. It's on page  
26 9 in 250, and I'm not sure what page it's on in 249.  
27 A Table 2, you said?  
28 Q Yes. Page 9.  
29 MR. THORNTON: I think Table 2 is on page  
30 22 of 249.

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1 Q Okay. Then get the same table out from exhibit 249. I  
2 think that was the - it is page 9, originally paginated  
3 on the published paper. Let's talk about the - well,  
4 let's talk about the various columns. I'd like to cover  
5 this with you in some detail.  
6 First of all, and just as background, could  
7 you please describe what your major functions were in  
8 both designing and carrying out these studies that  
9 resulted in the data that's set forth in Table 2 on  
10 exhibit 249 and 250?  
11 A I was responsible for coming up with the list of things  
12 we would measure, so I made the assessment as to what to  
13 measure, what I reasonably felt we could measure, and my  
14 laboratory did the actual measurements.  
15 Q So those would be your two principle functions with  
16 respect to this work?  
17 A Yes.  
18 Q And that would probably apply to the second studies where  
19 messenger RNA assays were used?  
20 A Yes.  
21 Q Generally speaking, do you think these measurements were  
22 done in an appropriate and accurate manner?  
23 A I think so.  
24 Q From your discussion with Mr. Thornton this morning  
25 (sic), I take it you had no responsibility for the  
26 statistical analysis that resulted in Table 2, is that  
27 correct?  
28 A No. You are correct.  
29 Q Thank you.  
30 A I was not responsible for statistics.

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1 Q Thank you.  
2 A Sorry.  
3 Q No, the problem was my question, not your answer. I  
4 apologize. We get double negatives in this business too  
5 often.  
6 And could you briefly describe for me your  
7 academic training in immunology in terms of course work  
8 you've taken and the research you've done as it relates  
9 to that subject? And I'm looking for the short version  
10 of that, a short version.  
11 A Well, like any graduate student whose specialty was  
12 physiology, I had a reasonable amount of immunology in  
13 courses. I was included in a lot of other courses I took  
14 in microbiology and physiology. I have used immuno-  
15 logical techniques as research tools for some time. Some  
16 of these assays were new to me, but in terms of actually  
17 doing them I was familiar with what the assays were, but  
18 I had not actually performed them before doing this  
19 study.  
20 Q The assays involved on Table 2, page 9 of exhibit 250, in  
21 particular, or other ones in the other study?  
22 A Well, we'll just talk about these for now. But, yes,  
23 these I - some of these would have been new assays to me.  
24 Q And who actually did the physical work of making the  
25 assays? A number of people? Can you describe who they  
26 were or what - -  
27 A The end is, there's technicians. I believe all of these  
28 were done - this was a very long time period. By  
29 full-time technicians as opposed to graduate students,  
30 although it might be possible that a graduate student

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1 carried out some of the assays. I do not know about the  
2 cortisol assay. That was not one that I was responsible  
3 for. I believe Dr. Wiltbank had done that. But most of  
4 them would have been done by full-time technicians of  
5 working under my supervision.  
6 Q For example, were the assays involving the cytokines,  
7 c-y-t-o-k-i-n-e-s, with interleukin 1 and interleukin 2,  
8 those would have been done by full-time technicians, is  
9 that correct?  
10 A That's correct.  
11 Q That was the machinery or the equipment and the apparatus  
12 and whatever else was needed to do those assays, is that  
13 something that all had been in the lab for some time?  
14 A We had had access to. Some of the equipment is very  
15 expensive and so shared by several labs, so some of it  
16 may have been physically located somewhere else. These  
17 things they would have used before.  
18 Q Okay. That was my next question. Thank you. By "they"  
19 you meant the technicians?  
20 A The technicians, yes.  
21 Q Thank you. The units for each of the various variables  
22 are indicated in parentheses, and it looks like you made,  
23 you or the statistician, made a logarithmic transfer on  
24 each of the numbers, is that correct, or transformation -  
25 -  
26 A That's what the other one would refer to, ye.  
27 Q That's a referral to natural logarithm?  
28 A That's a logarithm, yes.  
29 Q So you take the absolute number and before the  
30 statistical analysis is performed, the natural logarithm

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1 of that number is the number that's actually used to  
 2 analyze, correct?  
 3 A That's my understanding of what was done, yes.  
 4 Q All right. Was that done, the use of the natural  
 5 logarithm done at your direction?  
 6 A I do not recall how that procedure was arrived at. I  
 7 believe, if my memory serves me correctly, that Steve,  
 8 the individual who was doing that work, was concerned  
 9 about the statistical problem called heterostevasticity  
 10 (ph).  
 11 THE REPORTER: Called what?  
 12 A A-l - - Let me use a different word. The heterogeneous,  
 13 h-e-t-e-r-o-g-e-n-e-o-u-s. Unequal. Let's use this  
 14 word. Unequal variances. V-a-r-i-a-n-c-e-s.  
 15 Q And making a natural log transformation, is a standard,  
 16 unique in those circumstances?  
 17 A Is one of several commonly used techniques. I believe  
 18 Steve - -  
 19 MR. THORNTON: Try not to interrupt.  
 20 A Sorry.  
 21 MR. THORNTON: Or we're just going to have  
 22 a terrible transcript. Because this is hard enough as it  
 23 is.  
 24 Q Why don't we do that one more time, just for the record,  
 25 doctor, to make sure John got it correctly.  
 26 Is it true that making a natural  
 27 logarithmic transformation in the circumstances you  
 28 described is standard statistical technique?  
 29 A That's true.  
 30 Q And in your opinion, was it appropriate?

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1 A I did not - I did not go through the data in extreme  
 2 detail to check that, but it seemed reasonable.  
 3 Q With respect to the first two main response variables,  
 4 concanavalin A and phytochemagglutinin, the units appear  
 5 to be DPM, is that correct?  
 6 A That is correct.  
 7 Q What does that mean?  
 8 A Disintegrations per minute. These - should I explain the  
 9 assays?  
 10 Q Please.  
 11 A These assays are based on taking lymphocytes from blood,  
 12 culture them in the presence of a stimulant, and  
 13 measuring their DNA symphysis. The DNA symphysis is  
 14 measured by adding a radioactive isotope of the phymidine  
 15 p-h-y-m-i-d-i-n-e, and measuring how much of the  
 16 phymidine is incorporated into the cells. And for this,  
 17 we measured the amount of radioactivity in the cells that  
 18 the units for that were disintegrations, how many radio-  
 19 active phase per minute occurred.  
 20 Q And then, I'm sorry, this is all done out of the body?  
 21 A Correct, yes.  
 22 Q The term of that is in vitro or in vivo, one or the  
 23 other.  
 24 MR. THORNTON: In vivo.  
 25 Q In vivo.  
 26 A In vitro is literally in glass. So that's in test tube.  
 27 In vivo is in the whole body.  
 28 Q Thank you. I always get them mixed up. So these were  
 29 done in vitro, is that correct?  
 30 A That's correct.

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1 Q In fact, I think everything here was - that would apply  
 2 to, is that true?  
 3 A Not quite. Many things it does.  
 4 Q Okay. The third main response variable,  
 5 chemiluminescence, PMA. First of all, what does PMA  
 6 mean?  
 7 A Phorbol miristate acetate. I know I'm going to have to  
 8 spell this. P-h-o-r-b-o-l, m-i-r-i-s-t-a-t-e, I believe.  
 9 Acetate, a-c-e-t-a-t-e.  
 10 Q And then the number apparently has the acronym, RLU, is  
 11 that correct?  
 12 A That's correct. That stands for relative luminescence,  
 13 l-u-m-i-n-e-s-c-e-n-c-e, units.  
 14 Q Describe the assay in some detail, if you would,  
 15 including what relative luminescence units means.  
 16 A Yes. Here we take lymphocytes from the blood, and we add  
 17 to them a stimulant. There's several that we could have  
 18 used. Phorbol miristate acetate, or PMA, is the one that  
 19 we used here. This stimulates certain cells, mostly from  
 20 blood, a cell type called a neutrophil, which is a  
 21 component in the immune system that engulfs some digest  
 22 type bacteria.  
 23 We also add a detector, I believe, luminol,  
 24 l-u-m-i-n-o-l, was added. And the active neutrophils  
 25 produced oxygen radicals, this is part of the pathway  
 26 that they use to kill bacteria. This interacts with the  
 27 luminol and gives off light, hence the name,  
 28 chemiluminescence.  
 29 The instrument that we use to detect the  
 30 light is called a luminometer, it's effectively a light

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1 detector. And the relative luminescence units is simply  
 2 how many protons of light we detected for the output of  
 3 the instrument. It's called relative luminescence units  
 4 because it really has no specific number, like  
 5 disintegrations per minute does with radioactivity. It's  
 6 used in association with it.  
 7 Q So, every unit would be a whole bunch of protons, is that  
 8 correct?  
 9 A Probably. I don't know the details of that.  
 10 Q All right. Is there a particular reason or reasons that  
 11 you chose these three at the top of Table 2 as the main  
 12 response variables? And please describe that system.  
 13 A I don't recall that discussion at all about how that was  
 14 going to be presented in the table.  
 15 Q Well, picking out these various variables was your  
 16 primary responsibility, is that correct?  
 17 A Picking out the whole list was my primary responsibility.  
 18 But I don't recall discussing calling any of them primary  
 19 and secondary. I don't know why that distinction is made  
 20 there.  
 21 Q Well, who was the lead author of the Part 3 table, if it  
 22 wasn't - -  
 23 A The composition of it, as I recall, was by Dr. Reinemann.  
 24 Q So he would have done the drafting?  
 25 A He would have done the drafting of this paper, I believe.  
 26 Q And I would assume - -  
 27 A I did not. So, I am assuming that Dr. Reinemann did.  
 28 Q Was it then circulated for comments to all the  
 29 co-authors?  
 30 A I believe so.

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1 Q Going then down the list, the next variable, the next  
2 response variable, which is the lead one, top one, under  
3 secondary response variables, is *S. aureus*, or  
4 *staphylacoccus aureus*, is that correct?  
5 A That's correct.  
6 Q But again, if they were measuring DPM, which would be the  
7 same procedures as before, is that correct?  
8 A That's correct.  
9 MR. THORNTON: Mr. Lawrence, you should  
10 probably make clear that you're dealing with the table on  
11 249, excuse me, 250 or 249, you started talking about  
12 both and now a different one.  
13 Q You're absolutely correct. We are looking at the table  
14 on 250 at the moment, correct?  
15 A That is the one I'm looking at.  
16 Q Thank you. Thank you, Mr. Thornton.  
17 Going down the list in 250, the next  
18 response variable is pokeweed. You may have explained  
19 this to Mr. Thornton a bit. Can you tell us what that's  
20 all about, briefly?  
21 A That is an agent causing in pokeweed that stimulates  
22 certain lymphocytes to proliferate.  
23 Q So again, lymphocyte proliferation that's being  
24 determined here?  
25 A That is correct.  
26 Q And that would be true of the *staph. aureus*?  
27 A That's correct.  
28 Q And then the next one is IgG in the serum, correct?  
29 That's correct.  
30 Q And the units are milligrams per milliliter, correct?

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1 A That is correct, yes.  
2 Q And again, the logarithmic transformation made on the  
3 absolute number, correct?  
4 A Correct.  
5 Q And then the statistics are round?  
6 A Correct.  
7 MR. THORNTON: Doctor, if you could - -  
8 A I'm sorry, I'm just - I'm just wondering that if it  
9 should be micrograms per milliliter rather than  
10 milligrams.  
11 MR. THORNTON: Doctor, take your hand down.  
12 Q If you would.  
13 A Excuse me.  
14 MR. THORNTON: It was okay when you were  
15 looking at me, because the court reporter is between us.  
16 But now you're facing the other way.  
17 Q Okay. With that understanding, we will continue.  
18 What's the difference between IgG in the  
19 serum and the next variable IgG in vitro?  
20 A In serum, we collect the blood sample from the cow and  
21 measure the IgG in serum of that cow at that time. The  
22 in vitro, we collected cells, placed them in culture and  
23 measured their ability to produce IgG in culture.  
24 Q Then the next variable is IgA in serum, correct?  
25 A Correct.  
26 Q And the reported units are the same again, milligrams per  
27 milliliter, correct?  
28 A Correct.  
29 Q And then you explained to Mr. Thornton what IgA is, so I  
30 won't ask you that again.

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1 We then have IL1 of first in serum and then  
2 in vitro, with the units being picograms per milliliter,  
3 correct?  
4 A Correct.  
5 Q And hypo - that prefix indicates 10 to the minus 12, is  
6 that right?  
7 A Correct.  
8 Q So, we're a couple order - well, as compared to a  
9 milligram per milliliter, we're a couple orders of  
10 magnitude down, is that correct?  
11 A Much more than that.  
12 Q Okay. Well, comes to the minus 6 down, correct?  
13 A Yes.  
14 Q All right.  
15 A There is considerably more IgG than there is interleukin  
16 1.  
17 Q And what is interleukin 1?  
18 A Interleukin 1 is - interleukin means between leukocytes.  
19 So, it is the factor, protein factor, produced by certain  
20 leukocytes in the body that regulate other leukocytes.  
21 Q Would the chemical messenger be another way of expressing  
22 it?  
23 A That would be another way of expressing it, yes.  
24 Q And what is the significance of serum interleukin 1  
25 levels to the status of immune function in a cow at a  
26 particular time?  
27 A Elevated interleukin 1 levels is often associated with  
28 inflammatory processes and disease processes.  
29 Q Are there things other than inflammation that cause  
30 elevated interleukin 1?

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1 A Possibly. I am not familiar enough with the work on that  
2 to know for certain.  
3 Q All right. If there are no inflammatory processes going  
4 on in a cow, would you expect to find any interleukin 1  
5 in the blood?  
6 A You would expect to find small amounts.  
7 Q Is there a particular reference that you would - that  
8 refers to a discussion of these various subjects related  
9 to interleukin 1?  
10 A Not off the top of my head, but they do exist.  
11 Q Well, for example, there's a standard text, perhaps even  
12 a couple of them on veterinary immunology, one of them is  
13 by Tizard, T-i-z-a-r-d, is that correct?  
14 A I am not familiar with that particular work, but it could  
15 be. There are standard texts available. That could be  
16 one of them.  
17 Q Okay. You can't think of the name of one as you sit  
18 here?  
19 A Not off the top of my head.  
20 Q Are there any standard immunology texts, not necessarily  
21 directed just at animals, but at humans, that you rely on  
22 in your - that you have relied on in your immunological  
23 studies in the past?  
24 A There are. I would have to go back to my records and  
25 look them up to give you an exact reference, but there  
26 are such references available.  
27 Q Have you ever done work in the nature of, for example, of  
28 doing vaccine trials for drug companies and that sort of  
29 thing?  
30 A No, I have not.

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1 Q Would that type of work typically be done by veterinary  
2 immunologists?  
3 A I would think so.  
4 Q That's not something you have ever been involved in?  
5 A I have not.  
6 Q All right. Why did you look at both interleukin 1 in  
7 serum and in vitro in this particular study?  
8 A The interleukin 1 in serum gives us a base line of where  
9 the animals are at. In vitro, as I recall how these  
10 studies were done, we're measuring a stimulation, so  
11 we're measuring the ability of the lymphocytes in the  
12 blood to elevate interleukin 1 in response to a  
13 challenge.  
14 Q And the challenge in this case was, hopefully, the  
15 electric shock that was going on at levels or something  
16 else?  
17 A No, no. The challenge in this was - I hope I am  
18 remembering this correctly. Method section for this.  
19 The challenges that were used for this was propylene  
20 nitrogen. What was done was, the cows were treated  
21 either as control or voltage. We took the lymphocytes  
22 from the blood of both control and treated cows, and we  
23 stimulated them with propylene nitrogen. This will  
24 elevate their production of the interleukin. And we  
25 measured how much elevation we saw. So we're going from  
26 very, very little, essentially none, if I recall  
27 correctly, without the stimulation, to detectable levels.  
28 So we're measuring whether the voltage changed, whether  
29 or not they could produce interleukin 1 and 2 in response  
30 to the propylene nitrogen.

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1 Q Okay. Is that described in the text of the paper  
2 somewhere?  
3 A It is described in - -  
4 MR. THORNTON: 249.  
5 A 249. I don't know if all of it is described in detail in  
6 this one or not, but it is described.  
7 Q We can look at 250. Well, let's do the math here a  
8 little bit as to the interleukin 1 in serum. The next  
9 column over has two numbers, one on top and one below,  
10 correct?  
11 A That's correct.  
12 Q And those numbers represent the mean change of controls  
13 on top and the mean change of treatment on the bottom,  
14 correct?  
15 A That's what it looks like, yes.  
16 Q And the treatments would be those cows getting the shock  
17 from what's described in the paper, exhibit 250, correct?  
18 A That's correct.  
19 Q So, if we are looking at the concentration in micrograms  
20 per milliliter of the controls, the mean change when  
21 exposed to the pokeweed was - the natural logarithm of  
22 that number is minus 0.085, correct?  
23 A That's correct. Yes.  
24 Q And that indicates a very small change, correct?  
25 A I don't have a variance associated with that, so I can't  
26 really say that. But it looks to me to be a small  
27 change.  
28 Q All right. Well, if we're looking at the absolute value  
29 of the change, we'd have to invert the natural logarithm,  
30 in other words, raise E - the number E to that power to

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1 see what the factor is, correct?  
2 A That's correct.  
3 Q And E to the zero power is 1, indicating no change,  
4 correct?  
5 A Correct.  
6 Q And the number we have associated with the interleukin 1  
7 mean change controls is very close to - not equal to  
8 zero, but very close, correct?  
9 A That's what it looks to me like, yes.  
10 Q Then the mean change of treatments is 0.450, correct?  
11 A That's what this shows, yes.  
12 Q Okay. If you wanted to get the absolute number, you  
13 would raise the number E to that power, correct?  
14 A That would give you the actual levels, or it would give  
15 you the geometric means of that number, yes.  
16 Q Well, when you say - well, let's go through this a bit  
17 more. I want to make sure I've got this right. When you  
18 say the mean change of the treatment, how is that number  
19 0.450 calculated from the data? Could you describe the  
20 math?  
21 A I did not do that calculation, and I am not entirely sure  
22 exactly how this table was calculated. I would interpret  
23 that, just based on what is here, as before treatment and  
24 after treatment. What I don't know is which time point  
25 after treatment would have been used for this table.  
26 Q Well, assuming one before treatment measured and one  
27 after, whenever they were taken before and after, what's  
28 the math by which you arrive at the 0.450?  
29 A You would take the mean after treatment, the way I would  
30 do it. I would interpret this as taking the mean after,

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1 and subtract from that the mean before.  
2 Q And then taking the natural logarithm of the resulting  
3 number?  
4 A No. The natural logarithm would be taken before the -  
5 before the means were taken.  
6 Q Okay. So you determined the mean, you take the natural  
7 logarithm of that number after and subtract - -  
8 A You take the natural logarithm would be raw data, and  
9 take the mean of that natural logarithm.  
10 Q And then take the difference of those numbers?  
11 A That's how I interpret what was done here.  
12 Q Okay. Very good. Let me then show you exhibit - I think  
13 I handed you exhibit 253, which I will represent to you  
14 is that same Part III paper, but it was printed off the  
15 electronic data that was produced by the University in  
16 response to subpoena back in late 2007, from the data  
17 that was compiled that were labeled as yours, as the copy  
18 service hired by the University indicated, and it appears  
19 there's a whole bunch of data attached to that copy of  
20 the Part III paper.  
21 A Okay.  
22 Q Are the documents attached, do they look familiar to you?  
23 A They don't really look familiar, but that's because I  
24 haven't looked at this in a very long time.  
25 MR. THORNTON: You're talking about, Mr.  
26 Lawrence, Appendix 3?  
27 Q Yes. I'm talking about - well, actually Appendix - yeah,  
28 it would start with - -  
29 MR. THORNTON: Sheffield 304.  
30 Q 304, maybe even 303.

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1 MR. THORNTON: Thank you.  
 2 Q And on through the end of that document.  
 3 Assuming that data came from the disk  
 4 produced by the University in response to subpoena back  
 5 in late 2007, do you have any argument with the  
 6 conclusion of, that that's data from this study, Part  
 7 III?  
 8 A I see no reason, from what I'm seeing here, to say it  
 9 otherwise.  
 10 Q Okay. Then back to Table II on page 9 of 250, you have a  
 11 pair of variables for IL 2, or interleukin 2 in serum,  
 12 and then the following one in vitro, correct?  
 13 A That's correct.  
 14 Q By the way, where did you draw the cells from the cows to  
 15 do the in vitro measurements? What part of the cow did  
 16 it come from?  
 17 A They came from the - I believe they came from the tail,  
 18 that's where we usually collect blood samples from.  
 19 Q But the cells would come from there also?  
 20 A Yes.  
 21 Q All right. And why did you choose to study interleukin  
 22 2?  
 23 A Interleukin 2 is a - one of the interleukins that is  
 24 often changed in response to inflammation and infection.  
 25 It's also, at the time we did this, if I recall  
 26 correctly, interleukins were not very easy to study in  
 27 cattle, as opposed to humans, of immunological assays.  
 28 To measure them very easily wasn't available, so we were  
 29 having to rely on rather tedious bio-assays for doing  
 30 these. So we did not have the ability to measure a lot

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1 of different things. These were two that we felt we  
 2 could measure.  
 3 Q Well, were the assays and the measurement techniques  
 4 utilized in this study any different than would be done  
 5 on human blood or human cells to determine interleukin 1  
 6 or interleukin 2 levels?  
 7 A They were assays that could be done on human blood, and  
 8 in the past were done on human blood. But today they  
 9 have been supplanted by other methods.  
 10 Q Was that true back in '99 or 2000 when this work was  
 11 done?  
 12 A I don't recall for certain, but I believe that the  
 13 immunological assays would have been available at that  
 14 time for humans, but not for cattle.  
 15 Q And then, the last response variable is cortisol,  
 16 correct?  
 17 A That's correct.  
 18 Q And what is cortisol?  
 19 A Cortisol is a glucocorticoid produced by the adrenal  
 20 gland. It's often seen elevated in stress situations.  
 21 Q Will all of these variables necessarily show change for  
 22 any challenge of any type to the immune system?  
 23 A Not necessarily.  
 24 Q Are there many, many other response variables associated  
 25 with immune function of cattle that could be studied as  
 26 part of - of studies such as this?  
 27 A Yes.  
 28 Q And I take it you've studied quite a few more and did the  
 29 follow-up study later, is that correct?  
 30 A We studied some more. Fewer than I would have liked to

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1 have studied, but some.  
 2 Q You indicated about a hundred total, but not all  
 3 associated with immune function?  
 4 A Right.  
 5 Q Then we get, in the third column on Table 2, exhibit 250,  
 6 the column with the mean difference or treatment minus  
 7 control, is that correct?  
 8 A That's what it says.  
 9 Q And the arithmetic there is simply to subtract one number  
 10 from the other that's contained in the column to the  
 11 left, is that correct?  
 12 A That's what it appears to have been done, yes.  
 13 Q And in that column, under IgG serum, we see the number  
 14 0.017, correct?  
 15 A That's shown here, yes.  
 16 Q And you spoke to Mr. Thornton about that earlier this  
 17 afternoon, correct?  
 18 A I recall discussing IgGs. I don't recall if I recall  
 19 talking about that specific number, but, yes.  
 20 Q And I think you indicated that the comparable number in  
 21 exhibit 249 was 0.015, is that correct?  
 22 A What I said there was based on a misunderstanding that I  
 23 had at the time. I recall this discussion now. I was  
 24 comparing apples to oranges there.  
 25 Q Okay.  
 26 A Let me go back and correct.  
 27 Q Please. That's what I was getting to.  
 28 A Let's go back, because I was getting a little confused  
 29 here. In exhibit 250, the number here is a difference in  
 30 means, it's not a P factor. Table 2 in exhibit 249 is

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1 just the P value. So they're completely unrelated -  
 2 well, they're not completely unrelated, but they are not  
 3 comparable numbers. You would not expect them to be the  
 4 same. I apologize, I - I was looking at the two tables  
 5 and I was thinking this was a P value and it's not.  
 6 Q Can I take a look at 249, because I don't have a copy of  
 7 that one. I'll get a copy of that one after today's  
 8 deposition.  
 9 A I have an extra copy for you.  
 10 Q If you could. I appreciate that. While she is copying  
 11 249, let's talk a little bit more about 250.  
 12 In 250, the P-value is, as calculated by  
 13 Mr. LeMire on behalf of the researchers, is in the far  
 14 right column directly across from the label IgA serum,  
 15 correct?  
 16 A That seems to be correct, yes.  
 17 Q And that P value is 0.796 as reflected in Table 2,  
 18 exhibit 250, correct?  
 19 A Oh, yes, IgA. IgA, yes.  
 20 Q So, the response of IgA was nowhere even near statistical  
 21 significance, correct?  
 22 A That's based on this test. That's what that would say to  
 23 you.  
 24 Q Okay. Go ahead. I'm sorry.  
 25 A However, the statistic done in exhibit 250 is more  
 26 extensive than what's done here and it did show the  
 27 difference.  
 28 Q Exhibit 250 is the one in front of you - -  
 29 A Oh, okay. I'm getting my exhibits mixed up.  
 30 MR. THORNTON: When you say here, - -



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1 A This, exhibit 250, as I recall, this is just a simple  
2 t-test, and there are other statistical ways of assessing  
3 this.  
4 Q The simple t-test as reflected on Table 2 of page 9 of  
5 exhibit 250 is the one that the researchers collectively  
6 decided to include in the published paper again, is that  
7 correct?  
8 A That is what was in the - this part. I do not recall the  
9 time course of when the various ways of analyzing this  
10 was done. This may have been done before the statistics  
11 of the other paper were done. Probably was. But I don't  
12 know that for certain.  
13 Q Well, exhibit 250, which is, the front sheet entitled,  
14 "Dairy Cow Response to Electrical Environment, Final  
15 Report, Part III, Immune Function Response to Low-Level  
16 Electrical Current Exposure, submitted to the Minnesota  
17 Public Utilities Commission. That was the final paper  
18 that came out of that initial study, correct?  
19 A That was the final submission to the Minnesota Public  
20 Utilities, that's correct.  
21 Q By the way, did you have anything to do with the  
22 activities leading up to the University or Professor  
23 Reinemann's obtaining the contract, if you will, from the  
24 Minnesota Public Utilities Commission of terms - the  
25 request for proposal or anything like that?  
26 A No, I was not involved in any of that work. My involve-  
27 ment came after the involvement with that.  
28 Q And, generally speaking, what was your understanding of  
29 what the study was supposed to have done, in general,  
30 broad terms?

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1 A My understanding was that the initial study was to  
2 initially look at animal behavior responses and stress  
3 responses in response to voltage. I was not entirely  
4 clear from the very beginning as to the exact nature of  
5 the very initial proposal.  
6 MR. THORNTON: Can I just ask one question.  
7 Q Oh, sure. Go ahead.  
8 MR. THORNTON: When you say initial  
9 proposal, are you talking about the entire Minnesota  
10 Science Advisors' study or are you talking about Part  
11 III?  
12 A I'm talking about the entire one.  
13 Q And there were papers labeled Part 1 and Part 2 in this  
14 series also, correct?  
15 A That's my understanding, yes.  
16 Q But they did not address items that were specifically  
17 aimed at assessing immunological function, is that a fair  
18 --  
19 A That's correct.  
20 Q Okay. Let's look at exhibit 249, the comparable table,  
21 if you will. I realize it's not exactly the same format.  
22 A Page 22.  
23 Q Thank you. And I think you told Mr. Thornton this  
24 morning (sic) that exhibit 249 was something on the order  
25 of a preliminary draft of exhibit 250. Do I have that  
26 straight or not?  
27 A I don't think so.  
28 Q Okay.  
29 A This is written as if it were to be submitted to a  
30 journal for publication. I do not recall the order that

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1 these were prepared in. It may be that 249 was prepared  
2 subsequent to 250. I don't know.  
3 Q Okay. And in 249, page 22, under - well, the columns to  
4 the right have a P value over the top of both of them it  
5 appears, is that correct?  
6 A That's correct.  
7 Q So, what do those numbers mean? Can you tell us starting  
8 with the chemiluminescence as an example, the top one?  
9 A Yes. An alternative way of looking at the statistics  
10 here, that I would, with my non-professional understand-  
11 ing of statistics, say, is better than what was done in  
12 this table. But that is perhaps debatable.  
13 MR. THORNTON: This table you pointed to  
14 was exhibit 250?  
15 A This table is 250. So, in the table in 249, there were  
16 two things that were going on here. If you look at the  
17 figures that follow, you will see that there are two  
18 lines shown here, say on page 24 for chemiluminescence.  
19 One line, which has solid filled in circles, is the  
20 control group, the other line that has an open circle is  
21 the group exposed to current. So, there are two things  
22 that you can look at. You can look at whether this had  
23 changed over time and whether there's a treatment  
24 difference. So the treatment, in effect, is averaging  
25 all of these together and say, is the overall effect of  
26 treatment different?  
27 The other thing is, an important question  
28 is, perhaps the overall effect isn't different, but  
29 you've got two lines that are not parallel, the two lines  
30 - the control group isn't changing and the treatment

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1 group is going down. And that's what that treatment by  
2 time interaction, the third column, is measuring. And  
3 for chemiluminescence, for example, we see a P value of  
4 .679, suggesting that there's no difference in the  
5 average chemiluminescence. But the treatment by time is  
6 whether those two lines are parallel to each other or  
7 converging with coming together. That's what that column  
8 will represent.  
9 Q Would these P value calculations also be done following a  
10 natural logarithmic transformation?  
11 A I believe that is correct.  
12 Q Of the two columns on page 22 of exhibit 249, does the  
13 treatment column, those numbers, should they correspond  
14 to any of the columns in exhibit 250?  
15 A Not directly, no.  
16 Q Why not?  
17 A The methodology was different in how they were assessed.  
18 This (indicating) was done using the technique known as  
19 analysis of co-variance.  
20 MR. THORNTON: You've got to say which  
21 number you're talking about when you say "this."  
22 A 249 was done using a technique called analysis of  
23 co-variance.  
24 Q And 250 was not?  
25 A 250 was done using a t-test, which is not directly  
26 comparable.  
27 Q All right. Let's go back to 250 for a moment, Table 2.  
28 The paper itself describes three different groups of 8  
29 cows of 4 treatments and 4 controls for each group,  
30 correct?

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1 A That's correct.  
 2 Q And they were done with different cows, different times,  
 3 is that right?  
 4 A That's correct.  
 5 Q In your judgment, if you have a judgment on this subject,  
 6 what would be appropriate statistical analysis of data  
 7 collected in that manner be?  
 8 A Given that we were measuring things at multiple times, at  
 9 the time this was done the method used in 249 would  
 10 probably be considered the most appropriate.  
 11 Q Why?  
 12 A It takes into account the trends over time, which the  
 13 method in 250 I don't believe does as complete the job.  
 14 Q Okay. Fair enough. And again, you don't claim to be a  
 15 professional statistician, but you've had much contact  
 16 with the subject?  
 17 A I have had contact with the subject. It is not my  
 18 profession.  
 19 Q All right. Is there a concept in statistics that  
 20 attempts to analyze multiple replications of the same  
 21 experiment on different subjects?  
 22 A I'm not quite sure what the question is.  
 23 Q All right.  
 24 A So let's try to clarify that.  
 25 Q Fine. Is there such a thing in statistics as a two-way  
 26 cross analysis?  
 27 A The terminology that you're using there, that I think you  
 28 are referring to, is a crossover design, where - you  
 29 could be referring to a couple of different things.  
 30 Q Okay.

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1 A This type of design, where you have the same experiment  
 2 repeated three separate times, is called a blocked  
 3 design, and there are statistical methods of dealing with  
 4 that. I do not recall, it may be mentioned in here, if  
 5 that was accounted for in the analysis that was done or  
 6 not, but there are statistical ways of doing that.  
 7 The other thing that I wondered if you were  
 8 referring to was taking one cow as a treatment, but then  
 9 later making their control and switching the group  
 10 around.  
 11 Q I wasn't referring to that.  
 12 A You were not. Okay. So, yes, I do not know if the  
 13 blocking effect, the fact that it was done on three  
 14 separate times was accounted for or not, but there are  
 15 fairly standard ways of dealing with effect.  
 16 Q You would generally not do the statistical mathematics  
 17 the same with a blocked design as you described, 8 cows  
 18 per block for treatment or control, as you would in all  
 19 12 cows control and 12 cows treatment had the work done  
 20 at the same time, is that a fair statement?  
 21 A The second situation that you mentioned is a little bit  
 22 simpler analysis. The analysis was actually very  
 23 similar, but you would normally account for the fact that  
 24 it was done on three separate occasions by including a  
 25 time called block in the statistical model.  
 26 Q The details of that would be more appropriate - -  
 27 A That would be done by a statistician, yes.  
 28 Q Do you know whether, in exhibit 250, Table 2, the P value  
 29 for the detailed independent test, was that done simply  
 30 aggregating all of the data together and treating it as

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1 12 controls and 12 treatments without accounting for the  
 2 block design?  
 3 A I do not know.  
 4 Q Okay. Going on then to the second set of experiments  
 5 involved in messenger RNA, I have a set of data that I  
 6 would like to discuss with you. That will become 253.  
 7 THE REPORTER: 254.  
 8 Q 254. Excuse me. And I'll represent to you, Dr.  
 9 Sheffield, that that data came out of the materials again  
 10 that were provided by the University of Wisconsin  
 11 subpoena seven years ago, and they were among your  
 12 materials, I've got a photocopy of the disk they came off  
 13 of, if it will help.  
 14 MR. THORNTON: I don't think that's  
 15 correct. I think the last four lines were calculated by  
 16 somebody else.  
 17 Q I don't believe so, but we can find out.  
 18 MR. THORNTON: At least the data he  
 19 produced in response to a subpoena had the upper data out  
 20 of the last four lines.  
 21 A Not everything that I provided to you is, not everything  
 22 that was in the original subpoena, I have kept copies of.  
 23 What I sent to you was a - what I currently had. There  
 24 are things that are still - I do not know where they are,  
 25 I assume they are still at the University of Wisconsin,  
 26 Legal Services, but I don't physically possess them now.  
 27 So, it is possible that this table was part of what I  
 28 had, but it no longer is part of what I have in this  
 29 form. Does that make sense?  
 30 MR. THORNTON: The only thing I'll tell you

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1 is that the top two blocks of numbers were in the  
 2 material that you gave me, the last four lines, the  
 3 control mean, the test mean, the fold T over C and the P  
 4 value line were not.  
 5 Q You may be right about that. In any event, do you recall  
 6 - and I'll stick to the block, for the moment, maybe  
 7 forever.  
 8 MR. THORNTON: You can get to whatever you  
 9 want.  
 10 Q I will.  
 11 MR. THORNTON: I think there's an issue  
 12 about who did the last four lines.  
 13 Q You may be right. Regardless. Do you recall the second  
 14 experiment where the messenger RNA techniques were used,  
 15 which you described earlier, resulting in a set of data  
 16 that looks like these top two blocks across the page - -  
 17 A Yes.  
 18 Q - - for the variants?  
 19 A Yes.  
 20 Q And this goes on and on for four pages of - -  
 21 A Right.  
 22 Q And those would be almost a hundred variables that you  
 23 talked about?  
 24 A I think there's actually a little more than a hundred,  
 25 but in that vicinity.  
 26 Q What is the - can you describe - well, let's talk about  
 27 the interleukins in particular for a moment, find them  
 28 here. I think they are on page 2.  
 29 A These are mostly in alphabetical order.  
 30 Q Right. And about halfway across, from left to right,

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1 page 2, we have IL1a and IL1b, is that correct?  
 2 A That's correct.  
 3 Q I think those are usually referred to as interleukin 1  
 4 alpha and interleukin 1 beta, is that correct?  
 5 A That's correct.  
 6 Q And this set of experiments distinguish between the two  
 7 sub types with interleukin 1, correct?  
 8 A Correct.  
 9 Q What's the difference between the two? Can you describe  
 10 what it is and the significance biologically, briefly?  
 11 A It's been a long time since I have looked into  
 12 interleukins, but they are very similar. They are what  
 13 we refer to, if I recall correctly, as molegus genes,  
 14 that is, they originated as a gene duplication. So they  
 15 are slightly different protein sequences. In terms of  
 16 the biological activity, I believe they are very similar.  
 17 That's why, in the bio-assay that we did, previously we  
 18 could not detect the difference between - we were  
 19 detecting total interleukin 1 and you can't detect the  
 20 difference between alpha and beta forms.  
 21 Q Then, for example, in the interleukin 1a or alpha column,  
 22 as an example, what do the numbers mean?  
 23 A These are best known as - how to explain this? Relative  
 24 means, they are intensity of light multiplied by the area  
 25 that that light covers. That's the best way of thinking  
 26 it. They really don't have any standard mix of measures  
 27 associated with it, like disintegrations per minute, or  
 28 micrograms per milliliter.  
 29 Q Does that intensity of the light correspond to something  
 30 about interleukin 1 alpha or beta?

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1 A Not directly.  
 2 Q How about indirectly?  
 3 A There are a number of factors that affect it. Obviously,  
 4 the amount of interleukin 1 alpha, for example, messenger  
 5 RNA affects it. So, within interleukin 1 alpha, you can  
 6 make comparisons. So, if you see a larger number, you  
 7 would interpret that as having more interleukin 1 alpha  
 8 messenger RNA. What you can't do is go across genes and  
 9 say that it means a bigger number for interleukin 1 alpha  
 10 than interleukin 2 means that there's more interleukin 1  
 11 alpha than interleukin 2. You can't make that  
 12 comparison.  
 13 Q So we can't get to, for example, picograms per  
 14 milliliters?  
 15 A No.  
 16 Q But you can compare the quantity of interleukin 1 alpha  
 17 to itself in two different times or two different groups  
 18 of cows, fair to say?  
 19 A Fair enough.  
 20 Q And were these numbers then the basis for the analysis of  
 21 changes in interleukin 1 alpha and interleukin 1 beta and  
 22 the various other paramaters here?  
 23 A Yes.  
 24 Q And they were the basis upon which the statistical  
 25 analysis was performed which are reflected in the draft  
 26 paper, draft abstract that counsel discussed with you  
 27 this morning (sic)?  
 28 A Yes.  
 29 Q I believe that was in 251.  
 30 I would like to then go through with you

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1 what these various variables are. I know we've got about  
 2 a hundred of them, close to it. And maybe with respect  
 3 to each one, if you can tell us why you chose to study  
 4 it, if you can recall?  
 5  
 6 (Discussion held off the record).  
 7  
 8 Q Dr. Sheffield, I would like to take the variables on  
 9 exhibit 254 and have you explain to us what each one is a  
 10 little bit, and whether serum or vitro or something else,  
 11 and a little bit about why you chose to study each one,  
 12 if you can recall. I realize it's a long time ago, and  
 13 there's a lot of variables here. You may not recall.  
 14 A I will do the best I can - -  
 15 Q Thank you.  
 16 A - - on this. First of all, some of these - many of  
 17 these, our initial hope was to actually study far more  
 18 than these. Technically, we were not able to do that.  
 19 These were chosen, in part, because they are the ones  
 20 that we had reliable ways of studying in cattle. That  
 21 was an important thing, because the technology to do  
 22 things in cattle often lagged behind what it is in  
 23 medicine for many reasons. But, okay. Almost need a  
 24 magnifying glass.  
 25 Q I actually brought one along, believe it or not,  
 26 somewhere. I'll get it for you.  
 27 A ACK2 is a fairly general gene for acetate kinase. It has  
 28 important roles in a lot of different cell metabolisms.  
 29 So it's not something that would be restricted to the  
 30 immune system. But adenylate cyclase is an enzyme. By

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1 the way, all of these are messenger RNA.  
 2 MR. THORNTON: You're going to have to  
 3 spell some of these, doctor.  
 4 A Let me finish my thought here. All of these are  
 5 messenger RNAs. So the messenger RNA is a cell. I've  
 6 tried to indicate whether the protein is something that  
 7 is going to be in the cell or outside of the cell. But  
 8 adenylate cyclase, a-d-e-n-y-l-a-t-e, c-y-c-l-a-s-e.  
 9 This is a cell protein that's very important in hormone  
 10 C. It is found in many cells, probably most cells in the  
 11 body, there may be some that don't have it. So you would  
 12 find it in the immune system, but a lot of other places.  
 13 It's one of these signal pathways that some  
 14 hormones used to give their signals across the membrane  
 15 into a cell.  
 16 ATP synthase is a very general gene. It  
 17 does exactly what the name suggests, it is responsible  
 18 for producing ATP, so it's something all cells would  
 19 have, and expressed at a fairly - maybe not completely  
 20 constant level, but it's responsible for using ATP, which  
 21 is the main energy source of cells.  
 22 CFos, F-o-s, is what is called a  
 23 transcription factor. This is a protein that binds to  
 24 the DNA in the nucleus of the cell to promote messenger  
 25 RNA production of certain genes. It's widely  
 26 distributed, and it is often seen elevated during stress  
 27 events in the cell. But there are quite a few things  
 28 that will elevate CFos.  
 29 Those same comments hold for the next  
 30 column, cJun. J-u-n. CaATPase, the Ca refers to

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1 calcium. This is an enzyme that grades ATP and  
 2 transports calcium across cell membranes. Cells often  
 3 use calcium as a message. Normally, calcium in the  
 4 cytosol cell is very, very low. And the calcium ATPase  
 5 is involved in pumping calcium out of the cell to keep  
 6 its concentration in the cytosol very low.  
 7 The next column stands for casein,  
 8 c-a-s-e-i-n, kinase, k-i-n-a-s-e. There are two of  
 9 those, and the same comments will apply to both of these  
 10 columns.  
 11 At first glance, this is a bit of a  
 12 misnomer. Casein kinase you might think of as the enzyme  
 13 that transfers phosphate to casein in the mammary gland.  
 14 And we do call that that enzyme casein kinase, but this  
 15 is a different casein kinase. It's an old terminology.  
 16 Kinase, by the way, is an enzyme that transfers a  
 17 phosphate from ATP to a protein or to something. Doesn't  
 18 have to be a protein, but in this case it is a protein.  
 19 Many years ago, these were named sometimes  
 20 based on what they transferred phosphate to. This one,  
 21 it was found that casein would receive the phosphate very  
 22 easily, so it was called that, even though  
 23 physiologically, it does far more than that. These are  
 24 often involved in hormone signaling mechanisms inside the  
 25 cell.  
 26 The next are a series of proteins. CD 14,  
 27 23, 8, 3. These are cell surface antigens found in  
 28 different types of lymphocytes. They are involved in  
 29 self cell recognition and interactions of the cell with  
 30 their environment. These are frequently found in

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1 lymphocytes. Different lymphocytes would express  
 2 different proteins.  
 3 Cdk1 is cyclin, c-y-c-l-i-n, dependent  
 4 kinase. The cyclins are a group of proteins that  
 5 regulate proliferation of cells. And the cyclin  
 6 dependent kinase is a part of this family.  
 7 The next column is cleavage poly adenosine.  
 8 A-d-e-n-o-s-i-n-e. Or cleavage poly A. Messenger RNAs  
 9 in most messenger RNAs, carryoffs. After they're  
 10 initially transcribed, that is, the gene is used to  
 11 synthesize the messenger RNA. The messenger RNA has to  
 12 undergo several processing steps. One of those  
 13 processing steps is called poly adenolation.  
 14 A large number of about 200 adenine  
 15 residues, a-v-e-n-i-n-e, is added in somatically at the  
 16 end of the messenger RNA. It's true in most adenine  
 17 messenger RNAs, but not quite all.  
 18 Q But maybe?  
 19 A But maybe, yes.  
 20 MR. THORNTON: You got a duce.  
 21 A The lower is not absolutely required to make a protein,  
 22 but it does seem to have a role in stabilizing that RNA.  
 23 Messenger RNAs not only need to go up, you need to be  
 24 able to downgrade them to get rid of that is no longer  
 25 needed. One of the first things that frequently happens  
 26 is degrading the unneeded messenger RNA that poly adenine  
 27 is bleeded off. That's what poly A does. This again is  
 28 going to be a very widely distributed gene.  
 29 CREB1 and 2, we often pronounce those CREB.  
 30 That stands for cyclic A&P response element. And in B1

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1 and B2, which is different forms of this. Earlier I  
 2 mentioned adenylate cyclase. This produces a compound  
 3 called cyclic A&P. Cyclic A&P is what's called a second  
 4 messenger. One of its effects is to activate a series of  
 5 enzymes culminating in various responses. One of those  
 6 responses is to increase the expression of certain genes.  
 7 Those genes are those that have what's called a  
 8 cyclically impede response element in the DNA sequence.  
 9 The CREB is something that, it is a protein that actually  
 10 binds to the DNA to promote expression of those genes.  
 11 I hope that makes sense.  
 12 Q That's fine. Is there any particular reason you chose  
 13 CREB 1 and 2 to start with?  
 14 A They are cyclically a major regulator of cell functions.  
 15 The next column is copper, zinc, super  
 16 oxide. Dismutase. D-i-s-m-u-t-a-s-e. This one is  
 17 extremely important in nonspecific types of immunity and  
 18 in antioxidant responses. During metabolism, the body  
 19 produces large numbers of oxygen radicals. These are  
 20 very damaging, and we have a whole series of enzymes that  
 21 detoxify these oxygen radicals, using one of the enzymes  
 22 involved in this.  
 23 The next one, I do not recall exactly what  
 24 that is.  
 25 Q Fair enough. Looks like the initials are F-A-S, I think.  
 26 A No, there's another one before that.  
 27 Q Oh, I'm sorry.  
 28 A And I'm not quite sure about that. FAS is another  
 29 regulator of self-signaling.  
 30 Q What do the - -

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1 A I do not recall off the top of my head what it stands  
 2 for. I know it stands for something, but I just don't  
 3 recall what.  
 4 Q Fair enough.  
 5 A The next column is FASLigand. The FAS, you can think of  
 6 like a hormone receptor, the ligand is what binds it.  
 7 There are several things that could refer to in the next  
 8 column.  
 9 Q And you're not sure of the - -  
 10 A I'm not entirely sure which that refers to.  
 11 Q Okay. Fair enough. The last two on the page?  
 12 A Glutathione, g-l-u-t-a-t-h-i-o-m-e, peroxidase,  
 13 p-e-r-o-x-i-d-a-s-e. This is another of the enzymes  
 14 involved in oxygen radical metabolism.  
 15 The final column stands for glucose  
 16 transport IV. Glucose does not cross cell membranes very  
 17 well. So, we have to have specific cell membrane  
 18 proteins to carry it across. This is one of the more  
 19 common of the glucose transporters that would be present  
 20 in cells.  
 21 Q Let's stay on Page 1 for just a moment, Dr. Sheffield,  
 22 because it is approaching 3:30. Let me ask you a couple  
 23 more questions about this data, and we'll be done because  
 24 of Mr. Thornton's schedule being concluded for today.  
 25 MR. THORNTON: Sorry about that.  
 26 Q Okay. But for each of these variables, there are a  
 27 series of a block of 10 numbers below, and then a space,  
 28 and then a block of another 10 numbers below, is that  
 29 correct?  
 30 A That's correct.

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1 Q And my understanding is, the top block of 10 represents  
2 the control, the 10 control animals in this study, and  
3 the second group represents the treatment animals?  
4 A That's what it looks to me like, yes.  
5 Q And the application of the electricity to the treatment  
6 animals is as described in your draft abstract, correct?  
7 A That's correct.  
8 Q And that would hold - that characterization of the data  
9 would hold true throughout all four?  
10 A As far as I know, it would, yes.  
11 Q And in this case, this study was not done in separate  
12 blocks, but all 10 and 10 were studied at the same time?  
13 A That is correct.  
14 Q So, it's the same set of 10 animals or same sets of 20  
15 animals, if you will, throughout?  
16 A Correct.  
17 Q And in those circumstances, a simple two tail t-test  
18 would be one appropriate - -  
19 A That would be a reasonable thing to do.  
20 Q It would be a reasonable statistical methodology, you  
21 wouldn't have to worry about the blocking effect, is that  
22 correct?  
23 A There is no blocking in that, so you wouldn't worry about  
24 it.  
25 Q Fair enough. Unfortunately, why don't we go off the  
26 record. Let's go off the record for a moment.  
27  
28 (Discussion held off the record - 3:28 to 3:30).  
29  
30 MR. LAWRENCE: Doctor, while we were off

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1 the record, it was agreed that Friday is good for your  
2 schedule, and we've agreed to continue this May 9 at 9:00  
3 a.m.  
4 MR. THORNTON: Sure.  
5 MR. LAWRENCE: And I know counsel mentioned  
6 a subpoena earlier by mail. Is that sufficient for you,  
7 if I write you, send somebody else to serve you with a  
8 subpoena.  
9 MR. THORNTON: I can give you one right  
10 now, if you like.  
11 A I will be here May 9, 9:00 a.m.  
12 MR. THORNTON: Okay.  
13 MR. LAWRENCE: Very good. Thank you.  
14  
15 (3:31 o'clock a.m.)  
16 \* \* \* \*  
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READING AND SIGNING CERTIFICATE

I, LEWIS G. SHEFFIELD, PhD, do hereby certify  
that I have read the foregoing transcript of my  
deposition, recorded by John T. Kirby, of 3-14-14, 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

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1 STATE OF MINNESOTA }  
2 COUNTY OF DAKOTA } ss.  
3  
4 Be it known that I took the deposition of  
5 LEWIS G. SHEFFIELD, PhD, Volume I, on the 14th day of  
6 March, 2014, at 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 16TH DAY OF MARCH, 2014.  
25  
26  
27  
28  
29   
30

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