In The Matter Of:

Paul Halderson, et al., v.
Star Blends, et al.

Lewis G. Sheffield, Ph.D.
May 30, 2014
Volume 3

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STATE OF WISCONSIN CIRCUIT COURT TREMPEALEAU COUNTY

Paul Halderson and Lyn M. Halderson, N17388 County Road T Galesville, Wisconsin 54630 and Arctic View Farms, LLC N17388 County Road T Galesville, Wisconsin 54630,
Plaintiffs,
vs.
Star Blends LLC 1919 Riley Rd. Sparta, Wisconsin 54656 and ABC Insurance Company, a fictitious company and Northern States Power Company d/b/a Xcel Energy Services Inc. 1414 W. Hamlin Avenue Eau Claire, WI 54702,
Defendants.

Case No. 12-CV-74
Code Nos: 30303 & 30201

STATE OF MINNESOTA
COUNTY OF CASS

IN DISTRICT COURT
NINTH JUDICIAL DISTRICT
11-CV-12-1670

Randall and Peggy Norman,
Plaintiffs,
vs.
Crow Wing Cooperative Power & Light Company,
Defendant.

VOLUME III

Continuing Video Deposition of LEWIS G. SHEFFIELD, PhD, pursuant to Notice of Taking Deposition, and taken before John T. Kirby, a Notary Public in and for the County of Dakota, State of Minnesota, on the 30th day of May, 2014, at 1 South Pinckney Street, Madison, Wisconsin, commencing at approximately 8:59 a.m.

APPEARANCES:

Scott Lawrence, Esquire, of the LAWRENCE LAW OFFICE, S.C., 403 South Fourth Avenue, P.O. Box 117, Saint Nazianz, Wisconsin 54232-0117, 920-773-2811, ATTORNEYS@LDLAWSTN.COM, appeared representing the Plaintiffs, both captions.

Timothy R. Thornton, Esquire, of the firm of BRIGGS & MORGAN, 2400 IDS Center, Minneapolis, Minnesota 55402, 612-977-8400, tthornton@briggs.com, appeared representing Defendant NSP/Xcel Energy.

Charles A. Bird, Esquire, of the firm of BIRD, JACOBSEN & STEVENSON, PA, 305 Ironwood Square, 300 Third Avenue SE, Rochester, Minnesota 55904, charles@-birdjacobsen.com, telephonically representing Plaintiff Norman.

ALSO PRESENT:

Theresa A. Peterson, DVM.

VIDEOGRAPHER:

Todd S. Campbell, CLVS VT, Campbell Legal Video Company, 417 Heather Lane, Fredonia, Wisconsin 53021 262-447-2199, schedule@campbelllegalvideo.com.

EXHIBITS

EXAMINATIONS

MR. CAMPBELL: We are on the record. Today is Friday, May 30, 2014. The time is approximately 8:59 a.m. This is Volume III, Disk Number 1, of the Continuing video deposition of Dr. Lewis G. Sheffield, taken by Defendant Northern States Power Company in the matter of Paul Halderson, et al, versus Star Blends LLC, et al, State of Wisconsin, Circuit Court, Trempealeau County, Case Number 12-CV-74. And also noticed in the matter of Randall and Peggy Norman versus Crow Wing Cooperative Power and Light, State of Minnesota, County of Cass, Case File Number 11-CV-12-1670.

The deposition is being taken at the Law Firm of Boardman and Clark, Madison, Wisconsin.

My name is Todd Campbell. I am the video technician with Haskins Media Services, Apple Valley, Minnesota 55124.

Will counsel please note their appearances after which the court reporter will swear in the witness.

MR. LAWRENCE: For the Plaintiff Halderson, et al, Attorney Scott Lawrence, and for the Plaintiff Norman, Scott Lawrence, and on the telephone listening is Charles Bird. Also appearing working for Haldersons is Dr. Theresa Peterson.

MR. THORNTON: Do you want to tell him about Carlson?

MR. LAWRENCE: Yeah. Thank you. I have an e-mail yesterday from Attorney Paul Carlson representing the Defendant in the Norman case that says, "Scott: Because we have motion hearings tomorrow, I will not be at the Sheffield deposition tomorrow." Just put that on the record.

MR. THORNTON: Tim Thornton representing Northern States Power Company.

LEWIS G. SHEFFIELD,
a witness in the above matter,
after having been first duly sworn,
testified under oath as follows:

CONTINUING RE-DIRECT EXAMINATION

BY MR. THORNTON:

Q Dr. Sheffield, can you please explain the difference between the first Part III study that you did of proteins and the second study that you did by yourself with Messenger RNA?

A There were quite a few differences. The biggest one was what the N point was. The first study we were looking at more functional, if I can describe it that way, functional assays, such as the lymphocyte blastogenesis assay or chemiluminescence assays, and actual protein levels in the blood.

The second study was looking at Messenger RNA, which is a much earlier state of affairs in regulating gene expression.

Q Is it fair to say that the Messenger RNA is what turns on the proteins that become the antibodies?

A In a sense, that's correct. The Messenger RNA encodes the sequence of the proteins. There's actually several ways of increasing the level of protein in the blood.

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It's not just antibodies, it can be any protein that you're detecting.
One of the methods - one method that the cell uses would be to increase the Messenger RNA level. It is not the only method, but it is a method.
Q And there's a relationship, but not a direct relationship, between the Messenger RNA and the protein in the blood?
A Often, yes. There are specific cases where regulation occurs by other pathways, but usually there is some relation.
Q And I asked you this last time, but in re-reading the deposition transcript, I'm not still sure I understand it. What's the difference between an activity change and an expression change?
A I believe what you're referring to is this: Expression is the level of a protein that is present. Activity is its ability to carry out a particular function. For example, some enzymes in the body are regulated not by how much enzyme is there, but by how active the enzyme is. For example, some of the enzymes called protein kinases, these enzymes are present, but often turned off, and some signal then turns them off. At least I believe that's what you're referring to there as an activity, whereas, the expression is the actual amount of it present.
Q So, they might be present in the bloodstream, but not turned on, so to speak, so not in an invading fighting mode?
A That depends very much on the protein. Some proteins are

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regulated as to their activity and some are not.
Q And as I understand the second study, there was no within animal samples taken or samples analyzed?
MR. LAWRENCE: Object to form.
A There was no repeated samples analyzed, if that's what your asking.
Q Well, did you do a before and after on cow X, Y, Z?
A I believe we did collect the samples, but I don't think we ever analyzed them.
Q And that's not the best way to analyze the data, is it?
A It's probably preferable to do a before and after study, I would say, that's correct.
Q And on the first study, the Part III study, Mr. LeMire, he was a statistician, is that correct?
A That's what he did on the study.
Q And you did the second study, the study you did on the Messenger RNA, without a statistician?
A That's correct.
Q Now, take a look at 279, doctor.
A Okay.
Q And the pages aren't - well, the pages are numbered. Page 37.
A Page 37. Okay.
Q And that's comparing a particular Messenger RNA - or, no, that's comparing a protein between the two groups, the control group and the test group?
A Let me orient myself here. Yes, that's correct.
Q And that's IgA serum?
A Correct.
Q And you do have a statistically significant difference

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between the treatment group and the control group of .003?
MR. LAWRENCE: You left off a zero, Tim.
Q I'm sorry?
MR. LAWRENCE: Point triple zero 3.
Q Oh, excuse me. You're right.
A IgA serum. That is what this analysis shows, yes.
Q So that shows that, at least as related to IgA serum, these cows were not random, but quite different?
MR. LAWRENCE: Objection. Form.
A It says they were different. Whether that implies not random or not, I don't know.
Q Well, isn't the point in randomly selecting them is to try to get two as similar groups as possible for comparative purposes?
A Yes.
Q And at least as it relates to IgA serum, these groups are not similar?
A In that respect, they were not.
Q And without having the before and after samples analyzed, you can't make a covariant comparison with a group of animals that you analyzed in the second test, can you?
A I wouldn't know how it could.
Q And one of the major factors that could influence the comparison between the treated group and the control group after is the starting point for those groups?
A That would seem correct.
Q And just so I'm clear, define for me what covariant means.
A It's a statistical term. It means varying together.

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Q And block design is the statistical method that tends to factor out differences between the sampling times, for example?
A I think that's - I think what you're trying to get at is correct.
Q Why don't you get at it so it is correct?
A If we have the - basically, the same thing repeated several times, we have a group of, say, four cows we do an experiment and we do the same experiment on another four cows. A block design would allow removing any difference between the two times.
Q So, if one was done during the summer and one done during the winter, there would be some differences that were associated with ambient temperature?
A Yes.
Q And where they were in the stage of the feed bins?
A Possibly.
Q And the whole point of the block design is to factor out those differences?
A Yes.
Q And did you imply a block design in the Part III study?
A No.
Q Did you imply a block design in the study you did by yourself?
A Oh, excuse me. I got those backwards. The Part III study, meaning the one that these data came from?
Q The one you did with Dr. Reinemann.
A Dr. Reinemann. Okay. The answer to that question is, yes. I'm sorry. And the answer to your second question is no.

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Q Because that was done on one group of animals at one time?
A Correct.
Q So the control group and the test group experienced the same external factors?
A As close as we could manage it.
Q What is it, an array analysis?
A That's the method of detecting the Messenger RNAs. Instead of looking at them one at a time, we looked at them basically all at once, in the one assay.
Q And that was another difference between the original Dr. Reinemann U study and the study you did alone, is that an array analysis was only used on the second study, not the first?
A That's correct.
Q And is that going to create some differences in the two studies?
A Well, you're measuring very different things.
Q And in terms of the administration of electricity in the second test, were you responsible for that?
A I did not design the method to do it, I implemented it, if that's what you mean.
Q So you, physically - -
A I physically put the device on the animals.
Q And that was another difference between the first test and the second test, is that you weren't involved in the administration of the electricity in the first test?
A No, I was not.
Q Can you draw any conclusions to a reasonable degree of scientific certainty from the Part III test, the first

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test that was done with Dr. Reinemann?
MR. LAWRENCE: Object to form.
A To a reasonable degree of scientific certainty, I would be uncomfortable drawing any strong conclusions from that. We saw some possible differences that were fairly, in my opinion at least, modest in terms of what those measures are.
Q Can you draw any conclusions to a reasonable degree of scientific certainty from the second test, the test that you did?
MR. LAWRENCE: Same objection. Form.
A I would make the same comment.
Q And in terms of milk production, were there any differences in the Part III test in terms of comparing the milk production of the control group and treatment group?
A Not that I recall.
Q And in the second test, the test that you did?
A Not that I recall.
Q What about the behavioral responses, was there any difference in behavioral responses between the control group and the treatment group in the first test, Part III?
A I don't recall ever seeing any actual data on that. I don't recall any specific behavioral differences either.
Q What about in the second test, the test that you did?
A I didn't notice any.
Q And the Messenger RNA gene paper, that wasn't even submitted for publication, was it?
A No. Not to my knowledge. I think I would remember

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submitting it.
Q And is there a difference between cow trainers and the duration and the dose of electricity and the duration and the dose of electricity that you and Dr. Reinemann were administering in your two tests?
A Yes.
Q Can you explain that?
A Well, there's a lot of differences. Does everyone here know what a cow trainer is? Do I need to explain that?
Q I'm sure the jury doesn't.
A Should I explain what a cow trainer is? Or will someone else do that?
Q Please.
A Okay. In stanchion barns, a major goal is to keep the cows clean. That's a goal at any dairy operation. The cleaner the cows, the better the quality of milk, the fewer cases of mastitis and so forth.
A cow trainer is designed to train cows to avoid defecating in the stall and defecating in the gutter behind the stall. When cows defecate, they often hunch their back. The cow trainer prevents the cow - or trains the cow to not do this by delivering an electrical shock when she contacts a metal bar. So there's a metal bar above the back of the cow, adjusted for each cow to a certain height. And when the cow hunches to defecate, she contacts the bar that delivers an electrical shock.
This is strong - not strong enough to actually harm the cow, at least it's not thought to harm the cow by most dairy producers, but it is strong enough to cause an avoidance behavior so she will avoid making

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that contact.
Q Train the cow to crap in the alley rather than crap in the stall?
A That's the idea. Now, what this is doing is delivering a very short-term, very intermittent and avoidable exposure. And it's a fairly high voltage; I don't know just what the voltage on those things are. They're often, I think, powered just by electric chargers.
I know - I've never contracted one. People who have, tell me it's to humans fairly painful. On the other hand, the exposures that we used were much, much lower, and they were unavoidable exposures. The cow could not modify her behavior to avoid them, but they were a much lower level of exposure, but prolonged exposure as opposed to intermittent exposure, which is given by the trainers.
Q So, in terms of symptoms, behaviors, milk production of cows that are subject to the trainers, you can't draw a comparison to cows that are subject to low levels of electric current, can you?
MR. LAWRENCE: Objection to form.
A I would be very hesitant to draw a correlation or an inference there.
Q 278. On the first page there, there's typed in numbers and handwritten numbers. Do you know what the difference between the two are?
A I do not know.
Q And, for example, with the interleukin 1, pg/ml, the picograms per microliter, I think. The typed number is .071, which is not statistically significant, and the

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handwritten number is .0175, which is statistically significant. What is the difference between the two, do you know?
A I do not know where - what those numbers, those second numbers, the handwritten numbers, are from, or what they represent.
MR. LAWRENCE: Just for the record, I think the unit is picograms per milliliter, not microliter.
Q You're right. Sorry. 282. Is that a document that you recall ever seeing? On the first page it says, "Lewis Sheffield requested these summary statistics."
A One moment, please. It appears to be something like I might have requested. This was a long time ago, so I don't specifically recall that, but I probably have seen it before.
Q And this was an effort to just generally show that, as to the parameters indicated on the second page, that these cows are random enough?
A That's one of the reasons. Another reason is just - I thought it was important to just have an idea of what the basic characteristics were, such as their days in milk, their milk production and such.
Q Was this at the beginning of the study or after the study had been administered?
A (No response).
Q The note, there's a second - a third - I guess it's the third page that has some different numbers.
A I don't recall.
Q And the one thing that struck me, if you look on the - it's not numbered but it's the third page in the group,

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cow 4212, she has a somatic cell count of zero. That's impossible, isn't it?
A What that would mean is, it is just below detection levels. She would have had a very low somatic cell count. Because that would be unusually low, especially for this herd.
Q What's the difference between 286 and 251?
A Very little. It appears there might have been some editorial wordy changes.
Q It appears to me like 286 is like an iteration of what ultimately became 251.
A That's quite possible.
Q But to the best of your knowledge, 251 was your final draft of your report, "Impact low AC currents on immune function of dairy cattle"?
A I believe that is correct.
MR. LAWRENCE: For the record, sorry for interrupting. Just so it's clear now, I believe 251 is the copy Dr. Sheffield brought for the first deposition, and 286 is one I printed off from the disk, I believe. I think we're agreed on that.
Q I agree.
MR. LAWRENCE: Okay. Very good.
A They're different iterations of the same document, and I can't say for certain which is the older or the newer of the two, but they appear to be iterations of the same.
Q Well, for example, if you look at the second page of 286, in the second paragraph under Animals, there's a wording change, 'was' was changed to 'were.'?
A Yes.

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Q And then it's 'were' in the later document, or in the 251 anyway.
A Yes.
Q All right. Focus on 251. The conclusion of your report is that, "Four possible changes were noted: A slight increase in interleukin Number 1, Messenger RNA 2, a moderate decrease in interleukin 2 and 3, a moderate increase in interleukin 2 and number 3, interleukin 10 Messenger RNA, and a decrease in IgA related to Messenger RNA."
A Where exactly are we at?
Q I'm reading in the middle of the Abstract.
A The middle of the Abstract.
Q The third sentence begins, "However."?
A I think what I heard you say is not quite right.
Q What is quite right?
A I thought I heard you say an increase in interleukin 2. It should be a decrease in interleukin 2.
Q Okay.
A But I could have misheard.
Q But anyway, that "However" sentence, that's the bottom line of your findings?
A "However" sentence?
Q "However, four possible changes were noted."
A Yes. Okay. I see where you're at now. Yes.
Q And when the immune system is turned on by an invader to the body of a cow, there's other things that happen, like swelling, inflammation, temperature changes, things like that?
A Oftentimes, yes.

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Q When a cow is responding to an infection, for example?
A One usually sees that, yes.
Q And did you see any evidence of these other immune responses, symptoms, associated with the electrical treatments that were being administered?
A I did not note any.
Q And when you say in the last sentence of the Abstract, "These results suggest that electrical effects on disease processes are likely to be modest," there's significance to you using the word "suggest" in a scientific paper as opposed "indicate" or "established," isn't it?
A I would say a word like "established," in my opinion, would be a stronger word to use. That's what I would suggest. But language is not always my best suit.
Q Well, based upon the data that you assembled as a result of this test, you couldn't say anything was established, could you?
A I would not use the word "established" in this context.
Q And then, on the second page of 251, - it's not numbered - the last two paragraphs talk about the methodology that was used to deliver the voltage, correct?
A Correct.
Q And you washed the patch of skin, shaved the hair, washed it again and dried it and then attached the electrode?
A Yes.
Q That would be different than any exposure to electricity in a barn that an animal receives, certainly in a free-stall barn?
A Well, there was a reason we did that. But, yes, the method of exposure would be different in the sense, for

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example, there's no layer of hair left. In order to deliver a really accurate current, we shave the hair and put an electrode directly on the skin rather than having it go through the hair, which would introduce a varying amount of resistance.
Q Depending on the hair on the animals?
A And how dirty it was and how wet it was and many other factors.
Q Whereas, in a free-stall barn, if the cow wants to avoid the source of any electricity, it can just walk away?
A Not always.
Q When can't it?
A For example, if it is coming through a water bowl, the cow still has to drink. But in terms of other places, like if it was on a railing or something that the cow would not need to contact to live, like a water bowl, yes, they could generally avoid that.
Q And then, if you look on the third page, under Results and Discussion, you come to the conclusion that, "Milk production was unaffected by current exposure, ...and behavior of the cows was not noticeably affected"?
A Yes.
Q And the next paragraph - excuse me, the last paragraph on that page, you say, "Several major questions are raised by these results. Most measures were not affected, suggesting that those could be Type 1 errors, due to a large number of hypotheses tested." What are you saying there?
A As in any experiment, no matter how big a difference is, you always have to acknowledge the possibility that you

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could be wrong, and the more things you test, the more likely you will be to find what's called a Type 1 error. I think I've explained what this is before.
Q You have.
A That is basically saying there's a difference when there's really not. There's some statistical ways that are sometimes used to account for that, and there's a lot of - at least it's my understanding, as a non-- statistician, there's a lot of debate among statisticians as to how to do that, how to best control for that.
Q Because you sampled almost a hundred different variables, the chances of an error is increased as opposed to if you were sampling one or two variables?
A Yes.
Q Each sample has a 5% chance of being wrong?
A I'm not sure that's exactly the way that it's defined statistically, but that's the way it's often described. That's if you set your significance at 5%, which is typical in scientific literature, by the way.
Q But if you're testing for one variable, the results from the test are going to be a lot more valid than if you're testing for almost a hundred?
MR. LAWRENCE: Object to form.
A You are stretching my knowledge as statistic here. But if you're testing for one variable and you set your P value at .05, there is a 5% chance of a Type 1 error. If you test for 10 variables and each one is at .05, there is something more than a 5% chance that at least one of them will be.
Q Okay. You go on to say, "However, three variables were

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also identified in a previous study, suggesting the possibility that these could be real effects." Given the differences between the study you did by yourself and the study that you did with Dr. Reinemann, both what was looked at, how it was looked at, when it was looked at, how can you even make comparisons between the two?
A That's why I would use a word like "suggest" rather than "show" or "define." They are measuring things that have a relationship, but they are not exactly the same thing. I'm trying to think of a good example.
Q Apples and oranges are both fruits, but they're not the same fruit?
A I'm trying to think of one a little better than that. In the first study we measured the protein, in the second study we're measuring a factor that - one of several factors that induces production in the protein.
Q Well, not only are you looking at different things, you're looking at them in a different way, aren't you? In one, you're using an array analyzer and the other you're using assays?
A You have to look at them in different ways, because one is a protein, one is a Messenger RNA. You can't use the same methods to detect them. They are different measures. And so there is also the possibility that you're not measuring the same thing.
MR. LAWRENCE: Let the record reflect, we just lost the connection with Mr. Bird. He's up in northern Minnesota somewhere. Maybe I'll try him a little later.
MR. THORNTON: Or maybe this deposition

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just put him to sleep.
MR. LAWRENCE: Knowing Mr. Bird, I doubt that.
Q The last sentence, carryover sentence, "However the possibility exists that such small changes in immune function over a long period of times could have important consequences for disease resistance, particularly when considered over long periods." That's speculation, isn't it?
A Yes.
MR. LAWRENCE: Object to form.
Q Is it fair to say that the only conclusion that you really drew from this study is that more research is needed?
A That would be the only one I would feel safe about saying.
Q Take a look at 250.
MR. LAWRENCE: Here you go.
Q Which is the study that you and Dr. Reinemann did together. And this was submitted to the Minnesota Public Utilities Commission?
A I believe so.
Q Was it submitted to anybody else?
A I do not know.
Q And again, if you look at the last sentence of the Abstract, it says, "Collectively, these results suggest that exposure to 1 milliamp of current for two weeks had no significant - -
MR. LAWRENCE: Mr. Bird's returned.
MR. THORNTON: Mute your phone, Charlie,

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please.
Q The last sentence of the Abstract says, "Collectively, these results suggest that exposure to 1 milliamp of current for two weeks had no significant effects on immune function of dairy cattle." Do you have any reason to take issue with that conclusion?
A Based on all I know at the moment, no.
Q And if you look at page 9 of the table, the - I can't pronounce it, con - -
A Concanavalin A.
Q And the next one underneath that?
A Phytohemagglutinin.
Q Those are in bold. Why is that?
A I don't recall. I - -
Q Go ahead. Go ahead.
A As my - I said that, I'm thinking that they're bolded because there was some discussion about maybe there being two of the main measures to look at. But I could be wrong about that. I shouldn't speculate like that, because I really don't remember why those would be bolded.
Q The bolding for the staph. aureas P value number of .038 is because that is statistically significant?
A Probably.
Q And if we look at Page 11, - by the way, what's the difference between the Table 2 on page 9 and Table 3 on page 11? They appear to be the same response variables, but different numbers associated with it.
A Yes. These were different experiments. Table 2 is the voltage exposure. The control group was given no volt-

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age, the treatment group was given 1 milliamp current. Table 3 reports what we refer to as the positive control experiment. I think I've described this already, but I'll do it again in case I haven't.
Q You did describe it, but it may be a function of - I have a little difficulty reading it and understanding it. So give it another shot.
A It's an - the idea is, if we were wanting to measure a change in immune function, could we even do it? If we gave something that we know is a stress to the animal, would that affect our immune function measures? That was the idea.
Q Is that the dexamethasone?
A Yes, the method we chose, I vaguely recall some discussions about this. And the method we chose is a fairly classic method of injecting the animals with a drug called dexamethasone. Dexamethasone is a synthetic glucocorticoid. This is a hormone produced by the adrenal gland in response to a number of signals, but among them, stress. Among its many properties is it strongly suppresses certain aspects of the immune function. It's the type of drug that's often, or used to be used to treat arthritis. Sometimes people with arthritis will have dexamethasone or glucocorticoid injections into joints. So, it's that kind of drug.
So, what we did here was to inject the animals with dexamethasone, make our various measurements, and we see, for example, the concanavalin A, blastogenesis is affected, the IgG is affected, IL1 is affected, and as you'd expect cortisol is decreased.

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Q And that's the result of the injection and not the result of any electricity?
A Yes. There was no electricity used in the results of Table 3.
Q That's all I have.

FURTHER RE-CROSS EXAMINATION

BY MR. LAWRENCE:

Q Okay. That was short. Good. Dr. Sheffield, could you go back to exhibit 251, and I believe it's in front of you, the very last page of the exhibit.
A I've got it, yes.
Q This table (Indicating). And do we have exhibit 254 before you there, too? If not, I'll get one out of my set if you don't have one.
MR. THORNTON: I'll get it.
Q Thank you. Let's go to the second page of 254, that's that long spreadsheet of the measurements taken for the various variables for the treatment and control of cows. Do you recall that?
A Okay. Yes.
Q And in Table 1 on exhibit 251, that is that last page you have in front of you, you have the fold recorded for each gene, correct, or just about every gene anyway?
A Yes.
Q And then, referring to the second page of Table 254, I'd like to go through a few of those with you as they relate to some of those lower P values.

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MR. THORNTON: Just for the record, I'm going to object. This is way beyond the scope of re-direct.
Q Okay. But anyway, on IgJ, for example, in Table 1 on 251, you have loaded a fold of 0.43, correct?
A That's what I see here, yes.
Q And IgJ, the fold noted in those - those bottom four lines, which you weren't sure whether they were your work or not, the fold is also recorded as 0.43, at least to two digits, correct?
A That looks right.
Q With respect to IgA HC, or the heavy chain, the fold in Table 1, exhibit 211, which came out of your file of the first deposition, the fold is .49, correct?
A That looks right.
Q And on exhibit 254, at least to two digits, it's the same number, correct?
A I think so.
Q Interleukin 1 A, or alpha, it's A on the document, the fold on Table 1, exhibit 251, is 1.66, and again at least rounded to two decimal places, the same number appears on exhibit 254, is that correct?
A I think so. Yeah.
Q Next one, the next gene down in Table 1, exhibit 251 notes the fold at 1.78. And again, in exhibit 254 for interleukin 1 B, we have to two decimal places, the same number, correct?
A I think so.
Q Following the next number on Table 1, exhibit 251, is IL1 antagonist, and the fold reported is 1.19. And looking

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at exhibit 254, the fold is the same number to two decimal points, correct?
A I think so.
Q One column over on 254, or one row down on 251, we have the fold for IL2, reported both places as .48, correct?
A It looks right.
Q And then, the following column is IL2 receptor, and that's 1.21, the same number both places to two decimal points, correct, would you agree?
A Yes.
Q So, would it appear that the numbers that you have reported in Table 1, exhibit 251, that came out of your personal files are the same fold numbers, at least for those variables that are reported in exhibit 254?
A It appears to be.
Q So, I think you told us before that, to your knowledge, you were the only person that did any of that statistical analysis on the outcome of the gene expression study, correct?
A That's what I recall.
Q Now, it's fairly easy if one is - especially if one is somewhat facile with the software, to run the P values on these numbers on a two tailed independent T test, correct?
A Yes. Should be.
Q And that can be done, for example, in standard Excel software, as well as many others, correct?
A There's a lot of software, yes, to do that.
Q I'm having some difficulty establishing exactly when the work was performed on the second study, but I would like

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to just discuss that with you a bit.
A Okay.
Q I'll show you what we've marked as exhibit 298, which is a printout of one of the cover sheets from one of the folders in the UW data which has attached to it a whole bunch of milk production data on 20 cows, taken in December of '03 and January of '04.
A Okay.
Q And the - I don't want to waste your time by sitting here comparing them, but the cow numbers appear to be the same as are recorded elsewhere in the data. Does that sound like about the time this work was done on the gene expression study?
A I can't be sure, but it seems reasonable. That appears to be what this would be.
Q Exhibit 299 is just a - well, a shortened and somewhat modified version of a portion of 254 in front of you, where we have printed off the experimental data and the Excel formulas for IgJ, IgA HC, IL1 A, IL1 B, IL2, IL10, IgJ, IgA HC, - excuse me, I'm sorry, I'm on the second sheet. The front sheet shows those variables with the Excel notations for the formulas for the means, the fold and the P value. Do you see that?
A Yes.
Q And this just comes off of any Excel software that you plug the data into, will print you what those formulas are, if you ask it, is that correct?
A I guess so.
Q And then the second sheet of the document, on the second sheet of the document we simply run the analysis and

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printed the numbers in Excel, resulting in the P values that are shown on the document, correct?
A That looks correct, yes.
Q And I believe those P values are identical to those shown on exhibit 254. Or at least they'll do as many digits as they're reported in here. If you can take a look and verify that for us?
MR. THORNTON: I'll stipulate that they are.
Q Okay.
A That appears the same, from what I can see here, yes.
Q And those P values are all - well, the largest of the bunch is 0.0032 something for IgA HC, correct?
A That's right.
Q The rest of them are on the order of - well, a multiple of a number times 10 to the minus 5th or 10 to the minus 6th, correct?
A That's correct, yes.
Q And do you have any information in your possession or do you recall any information that would suggest that these P values derived from that data are wrong?
A I can't think of any.
Q We talked briefly last time about the concept of cytokine induced sickness behavior. I think you told me that you didn't have much familiarity with that concept, correct?
A That's correct.
Q Do you know or know of Robert Dantzer and Keith Kelley, K-e-l-l-y (sic), members of the Department of Animal Sciences, in the case of Dr. Dantzer, the Department of Medical Pathology, in the case of Dr. Kelley at the

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University of Illinois?
A Which Kelley was that now?
Q Keith Kelley.
A Keith Kelley.
Q Would you like me to restate that? I've got a document for you.
A No. No. I'm not particularly familiar with those names. No.
MR. THORNTON: I'm going to object. I'll wait 'til you're done. Go ahead.
Q I'll show you exhibit 300, Dr. Sheffield, and that is a copy of a paper from Elsevieer publication called Brain, Behavior and Immunity by Drs. Dantzer and Kelley, entitled "Twenty years of research on cytokine-induced sickness behavior." Have you ever seen that paper before?
A No, I have not. At least not to my knowledge.
MR. THORNTON: Object as to any questions on that paper based on foundation and hearsay.
Q In the Abstract of that paper, the third sentence, the authors say, for example, quote, near the end of the third line, "It was subsequently shown that physiological concentrations of pro-inflammatory cytokines that occur after infection act in the brain to induce common symptoms of sickness, such as loss of appetite, sleepiness, withdrawal from normal social activities, fever, aching joints and fatigue."?
MR. THORNTON: I'm also going to object that this obviously relates to human beings and not animals. When you're talking about normal social

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activities, I don't know of any normal social activities that cows engage in.
Q Oh, I think the animals I have are full of those, Mr. Thornton. We can save that for another day.
Anyway, this sort of thing is a concept with which you were not familiar with when you performed these studies, is that correct?
A If I understand what you're saying here correctly, I was not familiar with that idea at the time I did these studies, no.
MR. LAWRENCE: Charlie, you need to mute your phone, please.
MR. BIRD: Oh, okay. Hold on a second. I'll call you back.

(At this time a recess was taken - 9:58 to 10:08).

Q Thank you, Dr. Sheffield. Mr. Bird is back with us on the telephone again. I'm showing you (exhibit 301) a copy of a paper published by Cambridge University Press 2008, Animal Health Research Reviews, entitled "Sickness behavior, its mechanisms and significance," by Dr. Ian Tizard, T-i-z-a-r-d. I take it you have never seen that paper before?
A This is the first time I've seen it, yes.
MR. THORNTON: Objection. Standing objection to any questions about it?
Q Sure. And it has a similar discussion of the impact of elevated inflammatory cytokines in its Abstract of similar subject to the Dantzer and Kelley paper we looked

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at a moment ago, correct?
A I haven't had a chance to read it, just - -
MR. THORNTON: The document speaks for itself.
A Skimming the Abstract, that appears correct.
Q Do you know who Dr. Tizard is?
A I seem to have seen that name before, but I don't recall where. I'm assuming, since he is in veterinary medicine at Texas A&M, it was in some connection there. But I don't recall ever meeting him personally.
Q Are you familiar with his text, now in its 9th edition, and I have a copy here - -
A That may be where I have seen the name before then.
Q And that's called Veterinary Immunology, 9th edition, correct? (Indicating).
A I've seen that text before, yes.
Q And that is a standard - one of the standard texts in Veterinary Immunology, correct?
A I don't teach veterinary immunology, but it's a text I have seen before.
Q You have made no attempt, either by yourself or in connection with others, to attempt to evaluate the probability that these P values shown on the second page of exhibit 299, which are also on exhibit 254, may have occurred by random chance, have you?
A That I haven't.
Q Given the small numbers, would that be a useful project to undertake in analyzing the significance of the results of your gene expression study?
A I'm not sure, but I assume so.

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Q The gene expression study as well as the earlier Part III study, were both funded by public entities, correct?
A I believe so, yes.
Q And yet, these very low P values on these several variables that we talked about in exhibit 299 and 254, those have never been made public, to your knowledge, have they?
A I don't know.
Q You're aware that, for several decades now, dairymen have - many dairymen have claimed that exposure to low levels of electricity chronically have affected their herds adversely. You have been aware of that for some time, - -
MR. THORNTON: Objection. Hearsay. - -
Q Have you not?
A I have heard that, yes.
Q And you became aware, during the course of these studies, at some point in time that Dr. Reinemann regularly testifies for electric utilities in that regard, have you not?
A I know that now. I don't know when I first heard that.
Q It may have been sometime after you did the work, is that correct?
A I don't recall when I became aware of that.
Q Don't you think it was important that the public, and in particular the dairy community, know about these very low P values you found in your gene expression study?
A I don't have an opinion on that.
Q We talked a little bit about the equipment with which the gene expression assay work was done?

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A Yes.
Q Can you tell me what department or departments at the university or where that stuff ordinarily resides or resided back at the time that you did this work? Is it one of the Animal Science Departments or some other department or how did that work?
A It was in the Animal Science Building. I don't recall the ownership.
Q And I think you've described to us before that it was typically shared between multiple researchers because it's expensive stuff, correct?
A Yes.
Q And were the technicians who operated the machines, I think you indicated before they were full-time employees of the university, is that correct?
A I think that is correct.
MR. THORNTON: Objection. Move to strike. Speculation.
Q They weren't graduate students, is that right?
A That's correct. They weren't graduate students.
Q So, what's your understanding, if you have one, about the extent of the experience of these folks running that equipment? Can you describe that a little bit?
A They had run the equipment. That's - I don't know what else to say.
Q I think you told us in one of the earlier depositions that basically you don't want the immune system active all of the time because it can do damage if it is, is that correct?
A Well, there are quite a few diseases which override the

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patient of the immune system does damage.
Q And I apologize if this question has been asked, I do not think it has. But are you aware of any studies anywhere as to whether chronic elevation of the cytokines and the one immunoglobulin as shown on exhibit 299 will or will not damage cows? I'm referring to the second page of 299.
A Okay. So let me make sure I understand the question here. You're referring here to the things that are elevated, IL1 and 10. There's a good deal of work showing IL1 can be. I do not know how much of this might be in cows; there's certainly a lot more in rodents. I couldn't point you to any specific papers on that. I'm not saying they don't exist, I'm just saying I can't point you to them now.
Q Are you aware of any studies of cows or of other animals where the depression of the variables as shown on 299 which were lowered, and which is IgJ, IgA HC, IL2 and IL10, would be damaging to cows or other animals, for that matter?
A I would have to go back in the literature and look to find specific studies. But the problem - my overall impression is that they probably would exist for at least some animals.
MR. THORNTON: When you say they, you mean literature would exist for some animals?
A Literature on things what IgA does and what changes in IgA. I recall seeing studies that certain problems were correlated with changes in IgA, but that was a long time ago that I've even looked at those studies, so I'm not

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really up to date on that.
Q And in general, will a decrease in IgA tend to increase susceptibility for disease?
MR. THORNTON: Objection. Foundation. Speculation.
A Well, IgA is involved in what is called mucosal immunity. That is, I think I indicated this earlier, in these studies we're looking at, in the first study we were looking at serum levels of immunoglobulins. This one we're looking at the messenger immunoglobulins. And because IgA is more important in mucosal immunity on surfaces than it is in serum, that does raise the question of what the serum levels may actually be. But mucosal immunity is affected by IgA, so it's very important there.
Q Dr. Sheffield, I assume in your time at the university, you probably had some responsibilities for managing various of the university dairy herds in one fashion or another, or am I wrong about that?
A Not really.
Q Go ahead. I'm sorry.
A I was not involved in any day-to-day management. I occasionally served on committees that would make overall policy decisions on things, such as assigning cows to research projects and such. But I would not be involved in the day-to-day management.
Q But have you ever been responsible for the day-to-day management of dairy herds?
A No.
Q So, you have no - well, what a veterinarian would call

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clinical experience in observing patterns of behavior, production and health in dairy herds over time, is that correct?
A Only in context of research studies. But in terms of fields type work, no, I don't.
Q And you would not be critical, I take it, of a veterinarian or a dairyman taking into account their field observations of their herd in making decisions on how to manage it, would you?
A I'm not entirely sure exactly what you're getting at there. But unless it violated something that I would consider well recognized accepted standard practices, such as properly caring for animals, I can't see where I would object.
Q You have certainly read some of the scientific and popular press literature that exists relating to the impact, or the alleged impact, as Mr. Thornton would say, of stray voltage on dairy herds, have you not?
A Yes, I have read some.
Q And various of that literature describes the course of health and production in herds as improving when steps are taken to reduce the electrical exposure of the herds; you're familiar with that, correct?
MR. THORNTON: Objection. Hearsay.
A I have seen those things they have stated, yes.
Q Conversely, if the electrical exposure is increased, it's been reported that the health of various herds has diminished, correct?
MR. THORNTON: Objection. Hearsay.
A I don't know if I recall seeing that specific claim, but

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it might - it's been a long time since I've looked at some of that, so it's possible.
Q There's certainly nothing inconsistent with your data on cytokines in IgJ, as reflected in the gene expression levels shown on exhibits 299 and 254, that would be inconsistent with the notion that exposure to electricity affects the health of dairy cattle, would it?
A Off the top of my head, I would say I don't see anything that would be inconsistent with that. That, to me, if I'm understanding what you're asking, is a little bit like proving a negative; that, have we proved that it couldn't happen? Of course, not.
Q And, in fact, we do see statistically significant changes in the levels of IgJ and the various - and IgA HC and the various cytokines listed on 299 associated with electrical exposure in your gene expression studies, correct?
A In the messages, yes.
Q Mr. Thornton talked to you about the behavioral changes or behavioral affects.
Charlie, mute your phone, please.
MR. BIRD: Pardon?
Q Mute your phone.
MR. BIRD: Okay. Sorry about that.
Q Sorry about that, Dr. Sheffield, I'll start again.
MR. BIRD: I'm going to have to get off because I can't fiddle with this while I'm driving. I'll just talk to you later.
Q All right.
MR. THORNTON: We'll miss you deeply,

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Charlie.
MR. BIRD: I know. I know. All right.
Q Dr. Sheffield, would it be a reasonable thing to do to analyze the barn notes from the first study, the Part III study that you and Dr. Reinemann and others performed, to see if there were any differences in behavior between the treatment and control animals?
MR. THORNTON: Objection. Foundation. He testified last time he has never seen the barn notes.
A I do not know if enough detail were recorded in there to make an appropriate analysis, so I really can't answer the question.
Q Well, the Part III paper indicates that there were no changes in behavior noted, or words to that effect, correct?
A Yes. Yes.
Q That's got to be based on some kind of data, given that it was published, isn't that correct?
A Yes. I've never seen the actual data, and don't recall discussing how that statement was determined. I wasn't involved in assessing behavior. I'm not a behavior assessor.
Q Who was, with respect to that?
A I don't recall. That might have been - I think the name was Rasmussen. But I could be mistaken on that.
Q Well, if the statement is made in the paper and it is based upon data, that data should be preserved in the UW documentation of the study somewhere, should it not?
A I would think so.
Q Do you have any idea where that - what category of

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documents that might be in, other than the barn notes, if any?
MR. THORNTON: Objection. foundation. Speculation.
A I would not know.
Q And if there is no data to back up that statement in the paper, then that statement would also be simply speculation, would it not?
A I think that would be based on - well, without data, I would say, yes.
Q Going back to exhibit 279 that counsel discussed with you earlier this morning a bit, page 37 in particular. I've got a copy over here to show you what it looks like.
MR. THORNTON: Why don't you look at page 37.
Q Counsel asked you about the data with respect to differences between the treatment and control groups in IgA, I believe?
A Yes.
Q Was that data taken before, after or during the shock treatment?
A I believe that this is data taken both before and during and analyzed to this data. Maybe I'm mistaken about that, but I believe that included - - maybe I can figure this out.
Q Take your time, please.
A I think that includes the before and after data.
Q Was there part of a - in this particular case, demonstrated on or off the top of page 37, thereabouts, part of the covariant study or - covariant analysis or

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not?
A I believe the idea behind this was, this would not have included a covariant in this analysis. I am struggling a bit here with the details of this particular model, but what we have here is a difference in the overall treatment effect. The next line, if we look on page 38, gives us the effect of the difference among the cows. Each cow, of course, is going to be different. So, you get a P value for that. A day effect, and that is whether there was a change over time, and then the final is a treatment by date interaction.
What this shows is whether the two treatments responded differently to time. So, if, for example, you saw a change, and effect of treatment, you would see a treatment by time interaction, because they would start - if they start off at a certain level, and then say, just as an example, the treatment loads, you would see - and the control group did not rise in parallel, you would see a treatment by time interaction. I believe that was the idea behind this analysis.
Q And did there appear to be an effect taking into account treatment and time, or could you tell?
A From what I see here, it looks like, no. The treatments were - the groups were different, but they stayed different over time. That appears to be what's happening here.
Q All right. Going to exhibit 282 that counsel discussed with you, it's that summary of individual cow data for the treatment and control cows in the Part III study.
A I've got it here.

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Q Is one of these looking at data such as this to determine whether the randomization process was done reasonably.
A I'm not quite sure how to answer that, because it gets into the question of what randomization means, and I think a statistician may be able to give a better answer for that.
Q Okay. Fair enough. Let me just ask you the question from your point of view.
A From my point of view, okay. First, if you're wanting random assignment, you would expect most of the things that you measure to not be different at the start of the experiment. On the other hand, if you do measure many things, even with random assignment, you expect a few of them to differ.
So, if I took 20 people, for example, and randomly assigned them to two groups, it wouldn't do anything but just measure 20 different things. Some of those things may be different. Most of them would not be. And the same thing you would see with the cows. It would certainly be possible if you started with these - what is it 24 cows altogether - that as we're making more and more measurements, we would find an occasional one showing up where they look different.
Q You probably answered this, but what is the reason that you randomize, to start with?
A That is to avoid any bias in measuring your treatment effects.
Q Let's just look at some of these various variables that are summarized on the second page of exhibit 282. It appears that the control cows for all three replicates

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are listed, and the numbers at the top of the page, the treatment cows are at the bottom, is that correct?
A That seems to be the case, yes.
Q And if I understand the numbers correctly, the days in milk for the control cows is 213, is that correct?
A Right.
Q Would that be at the beginning of the experiment?
A I think so, but I'm not entirely sure.
Q And for the treatment cows, it was 240, correct?
A Correct.
Q And part of the purpose of this study is to ascertain the effects, if any, of the electricity on stress on the cows, is that correct?
A That's correct.
Q Are cows later in lactation generally less subject to the effects of stressors than cows earlier in lactation?
A I don't know.
Q The next column is daily milk average amount, correct?
A Correct.
Q And looks like control cows were just over 90 pounds, correct?
A They're averages, yes.
Q And the treatment cows were just over 81 pounds, correct?
A Correct.
Q Generally speaking, are cows - are higher producing cows more subject to the effects of stress than lower producing cows?
A More subject to or more stressed?
Q Well, thank you. I'll ask a different question. Are higher producing cows generally more stressed than lower

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producing cows?
A That's the general consensus, although I'm not sure how strongly the hard data are to support that. It depends a little on what you define as stress.
Q And I think the definition of stress in the veterinary dictionary goes something like, anything that adversely affects the homeostasis of the organism. Does that sound about right?
A That would sound like the kind of definition you would see in scientific literature.
Q It's sort of like saying stress is stress, in a way, is that correct?
A It's a little more precise than that.
Q Okay. And, therefore, are higher producing cows generally more susceptible to additional incremental stress than lower producing cows?
A I'm not sure how well that's supported, but I think many people would believe it.
Q Then, at the means somatic cell count, the next column, for the control cows, it is 46.75, correct?
A Correct.
Q And that's in thousands, of course, correct?
A That's in thousands per milliliter, yes.
Q And for the control cows, it was 58.5, correct?
A Right.
Q Both very low numbers in the world of somatic cell counts?
A Those are very low numbers, yes.
Q You then have the days carrying calf, correct?
A Yes.

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Q Just under 80 for the control cows and a little over 86 for the treatment cows, correct?
A Correct.
Q The lactation number mean for the control cows was 2.75 and for the treatment cows it was 2.83, correct?
A Correct.
Q The next column is just a trial number, which, of course, averages - -
A Just two.
Q And then we have the mean for the age of the cows in years, and for the control cows it is 4.65 and for the treatment cows, 4.94, correct?
A Correct.
Q Overall, at least based upon the parameters listed here, does it appear that the researchers' efforts to randomize the control and treatment cows was successful?
A Looking at the standard deviations, those are very similar numbers. So, it appears that they were random.
Q Counsel talked to you, or you talked to counsel about how cows can't always just walk away from electricity in their environment, for example, the example you used was a water bowl, correct?
A Yes. It depends on where the source is as to whether they can, yes.
Q And have you ever become familiar with the concept of measuring current in barn floors or step potentials to be different points on barn floors?
A I've heard the terms, but I'm not familiar with the measurements and how those things are done.
Q And if the cows are getting shocked from hoof to hoof in

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their environment at various points in their environment, that's something that they may not be able to get away from, depending on the specifics, would that be fair?
MR. LAWRENCE: Objection. Foundation.
A I wouldn't know for certain either way on that.
Q Exhibit 250, the Part III paper, for just a moment. Pages 9 and 11, those two tables that Mr. Thornton talked with you about earlier. In Table 3, the positive control, the dexamethasone study that didn't involve electricity.
A Yes.
Q And again, this was one group, not a block design, correct?
A I believe that's correct.
Q And of the 13 variables studied, four showed a statistically significant response at .05, indicating significance, correct?
A That's correct.
Q And what was your conclusion based upon that?
A Well, to begin with, there weren't many cows in this, as you notice, so it was not a very powerful study to start with. But we concluded that we were, at least for some of the measures, able to detect differences induced by a known stress.
Q And with respect to immune response, a particular stress may effect some responses and not others, isn't that correct?
A That's true.
Q And you have never taken the opportunity, I take it, or reviewed the results of the Part III study involving

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shock, which is summarized in Table 2, where the authors summarized it in Table 2, based upon a statistical analysis that accounted for the block design of the study, is that right?
A I don't recall seeing that.
Q I believe you told me in past sessions that the use of block statistics, or what some people call replication statistics, would be an inappropriate means of analyzing the data from the shock study in the Part III paper, correct?
A From my understanding of the statistics, yes.
Q And if the results of that analysis using the replication or block statistics showed that three of the 13 variables showed a statistically significant response and a fourth was closing to equal .06, the conclusions of the paper might have been different, might they not?
MR. THORNTON: Objection. Speculation. Objection. No foundation. Objection. Hearsay. Objection. Relying upon an expert that hasn't been identified or made any disclosures in this case.
A Without knowing more, I would say it would be possible. Not definitively yes or no though at this point.
Q In your Messenger RNA study, the second study, you utilized a current level of 1 milliamperes, right?
A I think that's right. Yes.
Q And as I understand it, a wave form was used that was not a pure 60 cycle wave form, is that correct?
A That's correct.
Q That was based on studies that were, I believe, done up at the - I think it was an experimental study by Mr.

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Allen Bahr (ph) and others?
A I don't remember where it was done.
Q It was based on some - -
A It was based on some measurements, and I don't know much about how those things were measured or any more than that.
Q Have you made any review of the scientific literature about whether or not exposure to 1 milliampere will elicit behavioral responses in some portion of the cow population?
A I don't recall anything.
Q Thank you. Off the record for a moment.

(Discussion held off the record - 10:49 to 10:54).

FURTHER RE-DIRECT EXAMINATION

BY MR. THORNTON:

Q Dr. Sheffield, can you say, to a reasonable degree of scientific certainty, that there's any biological significance to the P values on the second page of exhibit 299?
MR. LAWRENCE: Object to form.
A At this point, I would say I would be uncertain.
Q And you were asked questions about the people that were collecting the samples and doing the analysis. Your report and your notes indicate that there were problems running the array analyzer, at the beginning of testing?
A We did have some problems with that, yes.

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Q And that was the first time that that sort of testing device was ever used at the University of Wisconsin?
A I'm not sure about that, but that could be.
Q Certainly was the first time it was ever used - -
A First time we had used it, yes.
Q By we, you mean your research people?
A Yes.
Q And you said the significance of some of these proteins or Messenger RNAs were most significant for mucosal immunity rather than in the blood stream?
A For the IgA, that's correct.
Q And you didn't do any mucosal immunity testing?
A No, we did not.
Q So you can't say one way or the other whether the mucosal immunity was affected?
MR. LAWRENCE: Object to form.
A I would not say that I could make a conclusion about that. Let me rephrase that. I think that came out very strange.
I would not make a conclusion about mucosal immunity in these studies.
Q And then you were asked whether the numbers reflected in exhibit 299 were not inconsistent with adverse health effects in animals. And you said something to the effect that that's like proving a negative. My question is, can you conclude from the data in exhibit 299, to a reasonable degree of scientific certainties, that the gene levels reflected on 299 adversely affect animal health?
MR. LAWRENCE: Object to form.
A I would not conclude that.

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Q That's it.

FURTHER RE-CROSS EXAMINATION

BY MR. LAWRENCE:

Q One follow-up for the record. The gene expression study was conducted over a three week period of shocking activity, I think, is that correct?
A It was two or three weeks. I would have to look, but it's in there as to how long it was.
MR. THORNTON: First time, Scott, I've ever seen a lawyer say I've got one more question and only asked one more question.
Q Well, I was known to violate that. So there is nothing in these studies that would give us any specific results that would relate to how a cow would be affected if she lived with electrical exposure all or most of her life, correct?
A I don't think this would have any relevance to that, it's tissue exposure.
Q Thank you. That's all I have.
MR. THORNTON: Dr. Sheffield, neither one of us represent you, but you have a right to read and sign your deposition transcripts, in your case, plural transcripts. You have already indicated you think that's a good idea.
A Yes.
MR. THORNTON: I would recommend, given the scientific nature and the terms that you talked about

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that you, if you don't mind, take the time to read it and send a copy to Mr. Kirby?
A Yes, I intend to do that. I will do that when I get the final copy.
MR. THORNTON: I think we both agreed that he can have 30 days from the time that he gets the last transcript to do that?
MR. LAWRENCE: For all three transcripts, that correct.
MR. THORNTON: All three transcripts.
MR. LAWRENCE: That's correct. Any statutory provisions to the contrary notwithstanding.
MR. THORNTON: Because we've got Minnesota rules and Wisconsin rules, and if we just come to an agreement, I think that solves it if you have a problem.
MR. LAWRENCE: Yes, that's the intent.
MR. THORNTON: And if you have a problem, get in touch with Mr. Kirby and we'll get you more time.
MR. LAWRENCE: And, Mr. Kirby, do you want to send a copy direct to Dr. Sheffield?
THE REPORTER: Right. Do you have a copy of the first two?
A I have a copy of the first two.
MR. LAWRENCE: Yes, I sent them before, and this one may just as well come from you.

(10:59 o'clock a.m.)

* * * *

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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 5-30-14, 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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STATE OF MINNESOTA)
COUNTY OF DAKOTA) ss.

Be it known that I took the video deposition of LEWIS G. SHEFFIELD, PhD, Volume III, on the 30th day of May, 2014, at Madison, Wisconsin;
That I was then and there a notary public in and for the County of Dakota, State of Minnesota, and that by virtue thereof, I was duly authorized to administer an oath;
That the witness before testifying was by me first duly sworn to testify to the truth and nothing but the truth relative to said cause;
That the testimony of said witness was recorded in computerized Stenotype and thereafter transcribed by myself, and that the testimony is a true record of the testimony given by the witness to the best of my ability;
That I am not related to any of the parties hereto nor interested in the outcome of the matter;
That the reading and the signing has been executed as evidenced by the preceding page.

WITNESS MY HAND AND SEAL THIS 2ND DAY OF JUNE, 2014.

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