UNITED STATES
DEPARTMENT OF AGRICULTURE

UNITED STATES DEPARTMENT OF

AGRICULTURE/FOOD SAFETY AND

INSPECTION SERVICE

AGENDA

Pages: 1 through 271

Place: Arlington, VA

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Date: February 29, 2000

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Contaminated With Escherichia Coli O157:H7

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Recent Developments Regarding Beef Products
Contaminated With Escherichia Coli O157:H7

Holiday Inn

Rosslyn Westpark Hotel

1900 N. Fort Myer Drive

Rosslyn Ballroom

Arlington, VA 22209

Tuesday,

February 29, 2000

MEETING PARTICIPANTS:

DR. CATHERINE WOTEKI, Under Secretary For Food Safety
MR. THOMAS BILLY, Administrator, Food Safety and Inspection Service
DR. WILLIAM C. CRAY, JR., USDA
DR. DANIEL ENGELJOHN, USDA
DR. MARK POWELL, USDA
DR. SONJA J. OLSEN, Centers for Disease Control and Prevention
DR. CAIRD REXROAD, Agriculture Research Service
DR. DELL ALLEN, Excel Corporation
MR. JAMES HODGES, American Meat Institute
DR. KEITH BELK, Colorado State University
DR. ANN HOLLINGSWORTH, Keystone Foods
DR. WAYNE BIDLACK, California State Polytechnic University
DR. NARAIN NAIDU, California State Polytechnic University
DR. JAMES REAGAN, National Cattlemen's Beef Association
DR. KEITH BELK, Colorado State University
DR. ANDREW BENSON, University of Nebraska at Lincoln
DR. COLIN GILL, Agriculture and Agri-Food Canada
DR. RANDALL PHEBUS, Kansas State University
MS. NANCY DONLEY, Safe Tables Our Priority
MS. CAROLINE SMITH DEWAAL, Center for Science in the Public Interest on behalf of the Safe Food Coalition

P R O C E E D I N G S

(9:10 a.m.)

MR. BILLY: At this time, it's my pleasure to introduce Dr. Catherine Woteki, who is the Undersecretary for Food Safety in the U.S. Department of Agriculture. Cathy?

DR. WOTEKI: Thank you very much, Mr. Billy. I'd like to welcome everyone here today to what I view as being a very important public meeting on E. coli 0157:H7. For the past five years, the Food Safety and Inspection Service has pursued a strategy to make the food supply even safer. And I believe that the agency has made tremendous progress, along with the meat and poultry industry.

Just last month, we reached a major milestone with the third and final phase of implementation of the Pathogen Reduction and Hazard Analysis and Critical Control Points Rule. And evidence from many sources, including the salmonella performance standard data collected so far, show that this new system really is working to significantly reduce levels of contamination.

Now the organism, E. coli 0157:H7, has played a prominent role in the agency's strategy for change for a

number of different reasons. First of all, it's a pathogen of great concern because of its virulence. And as a result, FSIS has declared it to be an adulterant in ground beef in 1994, one of the first steps taken by the agency when it began an aggressive strategy to reduce foodborne illnesses.

Second, because of the seriousness of this pathogen, it served as a catalyst for change enabling FSIS as to make major improvements in all aspects of its food safety programs. Before E. coli 0157:H7 emerged as a pathogen of concern, that progress occurred, but it occurred very slowly.

Third, E. coli 0157:H7 has played a prominent role in our strategy for change, because it's a prime example of a food safety issue where FSIS had to take action to protect the public health, even though the scientific data were incomplete. The process we're going through now to reevaluate our policies, as new information becomes available, is a process that you will see repeated again in the future for various hazards as science moves forward.

And fourth, E. coli 0157:H7 is a good example of how government, academia, industry, and consumers have

come together to address a single, very important food safety issue. Today's agenda reflects the tremendous efforts that have been put forth to make sure that our decisions will be informed decisions.

Now our goal has been, and will continue to be, to ensure that our policies for E. coli 0157:H7 protect consumers to the fullest extent possible and that it's based on the best scientific information available. This meeting will help us to achieve that goal. And I look forward to hearing the various presentations that are on today's agenda.

Now, I very much appreciate all of your interest in participating in this meeting today, and I'd like to now turn over the meeting to Mr. Tom Billy, Administrator of the Food Safety and Inspection Service, who will be moderating the meeting today. Tom?

MR. BILLY: Okay. Thank you very much. And I, too, would like to welcome all of you to this public meeting. As you know, E. coli 0157:H7 is a pathogen of great concern to FSIS. In 1994, as Cathy indicated, the agency declared the pathogen to be an adulterant in ground beef. And the agency instituted an end-product sampling program, first, to stimulate action by industry,

and second, to help keep adulterated products out of the marketplace.

In January 1999, FSIS issued a notice to clarify that E. coli 0157:H7 adulterate not only ground beef, but any nonintact product or intact product that is to be further processed into a nonintact product. Now, as Dr. Woteki said, our goal has been, and will continue to be, to ensure that our policy on E. coli 0157:H7 protects consumers to the extent possible and is based on the best scientific data available.

Thus, FSIS is very interested in new information that would enhance our understanding of the pathogen and the appropriateness of our strategies. Such new information is, in fact, available from a number of sources. And that is why we're here today. We want to hear this new information and share it with you to keep all of you informed.

We also want to allow both the agency and the public to ask questions and to receive any comments you may have. We believe that the information presented here today may well have a bearing on the agency's policy on E. coli 0157:H7 or on the implementation of that policy. In particular, FSIS wants to move forward on the January

1999 Federal Register notice regarding intact versus nonintact products.

Verification of this policy by field employees has been on hold, although industry must adhere to the policy. In moving forward on the 1999 policy, FSIS has posed a number of questions regarding implementation of this policy on which it would like to receive input. These questions were listed in the Federal Register notice announcing this public meeting.

For example, we are asking whether it would, whether we should, redesign our sampling program that is, our testing program. And for example, should we establish alternatives to that testing program, and whether a plant's testing and verification programs should influence the degree of FSIS testing.

Based on all of the new information presented and comments we have received from a variety of sources, we will present our current thinking on E. coli 0157:H7 to the National Advisory Committee on Meat and Poultry Inspection which will meet in Washington, D.C., on May 16th and 17th. FSIS will, then, take whatever actions are necessary to implement its policy and verify industry compliance.

I'd like to take a moment to review the agenda before we start. We have a number of presentations scheduled today, and time will be allowed for questions after each one. To facilitate this, we have established a panel from the agency that is, from FSIS.

And the panel consists of Dr. William C. Cray, Jr., who a microbiologist with the Microbiology Division in FSIS; Dr. Daniel Engeljohn, who is the Director of the Regulations Development and Analysis Division in the agency; and Dr. Mark Paul, a risk analyst for the Epidemiology and Risk Assessment Division in FSIS.

The panel members will have an opportunity to ask questions first. Then, we will open it to the audience to ask questions, as well. If you'll quickly look at the agenda, I'd like to go over the agenda very quickly. As you can see, we intend to lead off with some agency presentations to share new information and data that we have, to bring you up to date with regard to the work within the agency.

Then, we will open it up to other federal agencies and the work that they are doing, as well as work, important new work that's being done in the private sector, as well. We plan to break for lunch at 12:30.

We also plan to take a couple of breaks in the morning and the afternoon.

There are a couple of changes in the agenda. First, at the presentation just after lunch at 1:30, my understanding is that Dr. Gary Weber will be making that presentation. Is that right, Gary? And then, the presentation at 3:15 this afternoon, regarding work done at Kansas State University, that will be a joint presentation involving both Dr. Randy Phebus and Dr. Jim Marsden.

Finally, if you wish to speak during the comment period between 4:45 and 5:45 this afternoon, we request that you sign up at the desk out front here outside the room. Several people have already done that and we welcome that. We request that you sign up to speak if you want to make a presentation.

Okay. With that, what I'd like to do, then, is move on to the presentations. And the first presentation will be done by Dr. William Cray, Jr. And he will focus on the new methodology, the new testing procedure that we're using for E. coli 0157:H7.

DR. CRAY: I want to begin by giving an overview of the new method. The new method was developed

through a collaboration with Dr. Jerry Crawford of the USDA Agricultural Research Service. His laboratory is at the Eastern Regional Research Center in Winmore (phonetic), Pennsylvania.

All agents and supplies used in the new method are commercially available. The new method is posted on the FSIS Website. The mention of specific brand and trade names for a product, medium, chemical, or agent does not constitute endorsement or selectivity by USDA over similar products that might also be suitable.

Analysis for E. coli 0157:H7 can be divided into four steps -- enrichment screening tests, isolation, identification, and confirmation. The new and old method use the same enrichment screening tests and identification confirmation. The difference in the method is in the isolation steps.

MR. BILLY: Dr. Cray?

DR. CRAY: Yes?

MR. BILLY: May I interrupt just a second? For those that may not be able to see what's on the screen, it's also in this handout that looks like this. And if you'll look in there, you'll find the same slide is in it. Sorry.

DR. CRAY: Enrichment step, the meat sample is mixed with nutrients and chemicals in a broth to encourage the growth of E. coli and discourages the growth of other bacteria. In the screening test, the sample enrichment broth is analyzed by performing an immunochromatography based E. coli 0157 dipstick screening test, if negative analysis stops. If positive, the sample is considered a potential positive.

In the old method, samples of the enrichment broth were diluted and spread onto MSABCIG aggre (phonetic.) E. coli 0157:H7 appeared colorless on MSABCIG aggre. Other bacteria could also appear colorless, making E. coli 0157:H7 difficult to detect. On the new method, E. coli 0157:H7 cells are concentrated by using immunomagnetic separation and spread onto rainbow agar.

E. coli 0157:H7 typically appears as dark colonies on rainbow agar. For identification and confirmation, biochemical tests identify the isolate as an E. coli and serological tests confirm the presence of the 0157:MH7 antigen. This is a slide showing a representation of immunomagnetic beads in E. coli 0157.

These green structures represent E. coli 0157, and these represent the immunomagnetic beads. The immunomagnetic beads have an iron core, and they are coded with antibody. Now, the antibody acts like a molecular Velcro. And when the immunomagnetic beads make physical contact with an E. coli 0157 cell, they will adhere. The cells and beads are very small, 300 cells placed end to end, which would equal about 1 millimeter.

This is a photograph of immunomagnetic beads, which have been mixed with a pure culture of E. coli 0157. These rodlike figures are the E. coli 0157 cells. And these are the immunomagnetic beads. The beads in the attached E. coli 0157 cells are concentrated using a column and a magnet. The enrichment broth containing the beads is poured through the column.

The beads have an iron core and are held in the column by the magnet. The colored dots represent bacteria that are not E. coli 0157. The black dots represent E. coli 0157. Buffer is poured through the column to wash away most of the bacteria that are not E. coli 0157. However, there are always some bacteria that stick and cannot be washed away.

This shows the column after it has been removed from the magnet and buffer is added, and the beads can be flushed out of the column along with the E. coli 0157. These beads are then plated onto an agar plate. And the plates are placed in a warm incubator to allow the bacteria to multiply. After 24 hours incubation, a single cell that initially we could not see will multiply and form a colony that we can see, which will contain hundreds of millions of cells.

This line illustrates the differences between the old and the new agar. On the old agar, the E. coli 0157:H7 cells and some other bacteria will appear colorless. On the new agar, E. coli 0157:H7 appears as dark colonies. Now, as you can see, there are a lot of other bacteria on here. And these were the bacteria that were sticking to the beads. We weren't able to wash those off. And we refer to those bacteria as background.

And you can see when there is a low number of E. coli 0157 on a plate that it's much easier to pick these dark colonies, out on the new agar than to pick which of these colonies -- of these colorless colonies are E. coli 0157. Now, on these plates, we put an equal amount of beads in E. coli 0157 cells, so there are approximately 20 E. coli 0157 cells on the new agar and 20 on the old agar. And it would be very difficult to pick the 0157 out of these background colonies that are also colorless.

Now, I'd like to show you how the method is performed in our laboratory. We have the magnet on a stand and a pan to collect anything that is washed out of the column. And we also have a small centrifuge. From a dipstick positive enrichment broth, we will pipe that about 5 mls and place that in a tube. Then we add a screen with a mesh onto a second tube.

And we then pour the enrichment broth through the mesh. And this withholds large particles of meat which could clog the column. We take 1 ml of the enrichment broth, and we add it to a small tube that has the immunomagnetic beads. We place the tube on the mixer, and this agitates the tube so that the beads will thoroughly mix throughout the enrichment broth.

While the beads are mixing, we place our columns on the magnet. When the mixing step is finished we then add the enrichment broth with the beads to the column. And we allow it to go through. The magnet will hold the beads in this area. There is a matrix in this area, which allows the liquid to flow through, but will impede the beads from flowing through.

We then add a buffer to rinse away as many of the background bacteria that we can. When the rinsing steps are finished, we remove the column and the beads are still in this area. And surface tension holds the beads in the matrix. We then place the column on the tube, and a buffer is added to the column.

And a plunger is used to force the beads down through to the bottom of the tube. Now we have about 1 ml of our solution of beads. And we add a 10th of an ml to an agar plate. And we then spread that on the agar plate. We return to our tube, and we take a 10th of an ml from this and make a 1-10 delusion. And we'll plate that.

Then, we still have about .8 ml of the bead solution. And we take that and put it into a small tube. And then, we put that into our centrifuge. And this will spin around and force all of the beads to the bottom of the tube. We then collect these beads and plate those.

So for every sample, we'll have a plate with undiluted beads, a 1-10 solution, and then our concentrated beads for a total of three plates. And we are doing this, because if the E. coli are present in very high numbers, then it will be too hard to get isolated colonies if we use undiluted beads.

And on the other hand, if they are sparsely populated, then we need to use the concentrated beads to ensure that we will be able to isolate the E. coli 0157. We, then, place the plates in an incubator, and we incubate them for 24 hours.

And at the end of the incubation period, we examine the plates for dark colonies which are typical of E. coli 0157. At this point, we perform a serological latex bead test for the 0157 antigen. Colonies that are

positive for the 0157 antigen are then stripped onto blood agar.

After incubation, colonies on those plates are analyzed serologically, and biochemical tests are performed to identify them as E. coli 0157. The agar that we're using is not perfect, in that not every dark colony is an E. coli 0157. But it is an improvement over the old agar.

The new method is, at least, four times more sensitive than the old method and reduces our analysis time by one day. And now, I'd like to show you the results of raw beef products analyzed for E. coli 0157:H7. The number of samples analyzed are on the y-axis, and this shows fiscal years '95, when the projects first began, '96, '97, '98,'99, and 2000 up to February 13th.

And this shows the number of positive isolates in '95, '96, '97. And I don't know if you can read this for three in '95, four in '96, two in '97. In 1998, there is an increase. There are 14 positives. In 1995, '96, and '97, the sample size analyzed was 25 grams. And in 1998, the sample size was increased to 325 grams, so this increase in positives in fiscal year '98 is attributed to the increase in sample size.

The increase in 1999, where we had 29 positives, is attributed to the introduction of the new method late in the fiscal year. And the data for 2000 is

incomplete, but we would anticipate that the numbers for the fiscal year will be higher than 1999. Questions please?

MR. BILLY: Okay. Are there any questions from the panel? No. I have one question just to be clear. I think I heard you say that this new method is four times more sensitive than the old method that we were using, is that correct?

DR. CRAY: Yes.

MR. BILLY: And I know that some of the studies will be reported on later today were also using the similar kind of new method, so it would be important when presenters talk about their study to be clear what methodology was used, so that we understand the sensitivity of the method that's associated with that data. Are there any other questions anybody has? Yes, Kim?

MS. RICE: Kim Rice, AMI. Dr. Cray, can you clarify on the 325-gram sample that you now pull, you actually run 565-gram tests, correct?

DR. CRAY: Correct.

MS. RICE: So does that number indicate the samples taken or the tests run?

DR. CRAY: This number indicates the samples, and so the subsamples would be five times that number.

MS. RICE: So the number of tests run since the change to 325 is actually five times that number, correct?

DR. CRAY: Yes, yes.

MS. RICE: Okay.

MR. BILLY: State your name and your affiliation.

MR. WOOD: Richard Wood with Fact Food Elements Concerned Trust. You mentioned that this test takes one day less than the earlier test. What is the total test time, then?

DR. CRAY: Samples that are analyzed on a Monday, we would have their result on a Friday. So about four days. And that would be if there were no extenuating circumstances. For example, if the E. coli 0157 colony was in a crowded area on a plate, it would have to be restreaked. And that would add an additional day.

MR. WOOD: And the sample is a meat sample that is taken. Can this test -- and perhaps I'll learn this as we hear the presenters -- but can this test be used for fecal samples or any other kind of sampling?

DR. CRAY: We haven't evaluated that. But the principles of using the beads, CDC uses those now for that.

MR. WOOD: Thank you.

MR. BILLY: Yes?

MS. HOLLINGSWORTH: Ann Hollingsworth with Keystone Foods. You stated that this test is four times more accurate than the other test. On what basis are you making the assumption? And has that data been peer-reviewed and acknowledged?

DR. CRAY: The data was obtained by running samples in parallel with the old method and the new method. The data is in a manuscript which will be submitted next month for publication. It has not been peer-reviewed.

MR. BILLY: Yes, Caroline?

MS. DeWAAL: Can the new agar --

MR. BILLY: Caroline, state your name.

MS. DeWAAL: Caroline Smith DeWaal, Center for Science in the Public Interest. Can the new agar be used without the magnetic beads?

DR. CRAY: We find that the -- that it's a combination of using the magnetic without the -- the advantage of the new agar is that it's easier to see the colony's typical E. coli 0157. However, the immunomagnetic beads are necessary to concentrate the cells, which might be present in low numbers.

MS. DeWAAL: But is there any reason for people, whether or not they are using the new magnetic beads, is there any reason for people to be using the old agar? The new agar seems so much more superior.

DR. CRAY: We no longer use it.

MR. BILLY: Sonja?

MS. OLSEN: This is Sonja Olsen from CDC. I'd just like to -- I think what you've already said is that CDC currently uses immunomagnetic beads. And there are published accounts of its use in humans. I don't know about, in terms of beef samples, but it's used very frequently in human samples. And it's found to be much more sensitive.

MR. BILLY: Yes. Mark?

Mr. POWELL: Mark Powell, FSIS. Our analysis of the scientific literature on the IMS method also suggests that it's approximately four times more sensitive. The sensitivity is also a function of the concentration of the samples. At very low levels of spiked samples, it appears to be about four times more sensitive as the concentration in the samples elevates.

There's not such a stark contrast. But at the very low levels that you would expect to find, it's approximately four times more sensitive.

MR. BILLY: Okay. Randy, did you have anything?

MR. PHEBUS: In terms of being able to use --

MR. BILLY: Your name?

MR. PHEBUS: Oh, excuse me. Randy Phebus, Kansas State University. In terms of being about to use this as a technique that you would use in in-house laboratories, do you think that there's potential worker-

safety risk with all the pipe heading and transferring and aspirating and centrifuging that is being done? Or should that be a consideration?

DR. CRAY: Well, E. coli 0157 is serious, so we adhere to all the safety regulations in our facility USDA regulations. And as I showed on the slides, all of these operations can be done in a safety cabinet.

MR. BILLY: Thank you.

MR. DANIELSON: Dean Danielson with IDP. I would like to point out that there are several industry companies that have adopted or adapted this new procedure in the last two or three years. In fact in 1997-1998, these systems, more sensitive systems, became known. And some of us have been using those for a period of time.

One question to you or the agency is, I am aware of some labs still using the old methods today. I'm also aware through a secondhand source that when queried, the agency will say either method can be used.

Now, in terms of defining policy, it seems to me if there's two methods out there, one being less sensitive and more sensitive, we need to get a little more consistent in that, I would -- would be my opinion. Is there a plan with the agency to specify this new method?

DR. CRAY: I can respond in the sense that this is precisely the kind of input and thoughts that we'd like people to share with us as part of this meeting. We

recognize that it was a relatively new method, and we're hoping that there would be people switching over to the new method and gaining experience with it.

And as that process occurred, then we would reconsider our policy. I mean, there are tradeoffs. If the question is, you know, are they -- do we either do the old method or no testing, we'll take the old method. If they are going to do testing and make a choice between the old and new methods, then we would prefer the new method. But we can make that clear in terms of what we -- what comes out of this meeting.

MR. BILLY: Kim, you have a --

MS. RICE: Kim Rice, American Meat Institute. One point of clarification, my understanding is that the new method, it's the beads that make it more sensitive and that the use of the rainbow agar just simplifies during the isolation. It's the use of the beads and then the agar simply makes it easier for the technicians in the lab to pick off colonies.

DR. CRAY: The beads make it more sensitive. If there are a lot of background colonies that mimic E. coli 0157, then in our understanding, the rainbow agar helps us select the E. coli 0157. If there are not a lot of background colonies with it of similar coloration, in that situation, there isn't an advantage.

For example, on one sample, 128 silver-tone negative colonies were picked from a plate. And only one

of those was E. coli 0157. On a plate inoculated with the same material, rainwater plate, we were able to pick five out of five. And so the E. coli, if they are there in low numbers, the rainbow benefits, and also if they are in -- if there are a lot of confining organisms, background organisms.

MR. BILLY: Okay. Dr. Naidu and then --

DR. NAIDU: I'm Dr. Naidu. I'm from Center for Antimicrobial Research. On the same question, the sensitivity and specificity of your method depends on the antibody that is sitting out that has been kept on your magnetic plate. How much polyclonal is your antibody and whether it will recognize all types of E. coli 0157 and how the life of the antibody during your testing would influence the result?

DR. CRAY: We use commercially prepared Dynel immunomagnetic beads. These are used in many laboratories in the U.S. and Canada and Norway. And my understanding is it's proprietary information exactly what their antibodies are. But my understanding is that it's a polyclonal antibody to E. coli 0157.

That's why we use a serological test. When we're picking colonies typical of E. coli 0157 off of our media, we have to confirm chemically if they are E. coli 0157, that they are E. coli and we serologically confirm that they are 0157 in itself H7. We also perform toxin tests on all of the isolates.

So if an isolate is E. coli 0157-positive serologically, and if the H7 test is inconclusive, then we will -- and if it is toxin-positive, we will report that out as E. coli 0157:H7.

MR. BILLY: Okay. Last question.

MR. WOOD: Rich Wood with FACT. Are the costs of the new tests different than the costs of the former tests? Is there an incentive one way or another?

DR. CRAY: The materials are more expensive for the new tests. But the labor costs are markedly reduced.

MR. WOOD: So it's an even trade?

DR. CRAY: We think it's -- there's more benefit to the new test. It costs less.

MR. BILLY: Okay. I'd like to move on. Thank you very much, Dr. Cray. The next presentation will focus on the area of irradiation of meat products. It will be made by Dr. Dan Engeljohn.

DR. ENGELJOHN: Good morning. I, too, have a handout in the back of the room that follows through each of these slides. I'd like to point out the FSIS website that's on this first page, as well, where we have most of the information I'm presenting today is already available on our website. And we will be having, or adding, more information to that website shortly.

I'll talk briefly about the final regulation that just issued. It issued in the Federal Register, Volume 64 on December 23, 1999. It became effective on

February the 22nd of this year. It involves, for specific the topic today, it involves the refrigerated or frozen beef in the uncooked state. It could be packaged or unpackaged, seasoned or unseasoned.

And the sources of irradiation can be from gamma, which would include the Cobalt 60 or CZM 137 sources or the machine sources which would include x-rays and high-energy electron beams. One other piece of information issued this last week was our FSIS Directive No. 7700.1, which includes the instructions to our employees of how they would do verification activities within the irradiation facilities. That too is available on our website.

This week, I would hope this week we would be issuing a question and answer that contains many of the questions that we've received since the regulation published. And it addresses issues related to labeling, process control, and so forth. And we'll update that as we get in more questions and post that to the website.

With regard to the controls that we have in place, it's our expectation that radiation facilities will identify critical points within their HACCP system. And this would be for radiation on-site or radiation at a contracted facility that may be off-site. Within that control program, we would expect that there would be both symmetry addressed, as well as documentation.

And most of that documentation relates to licensing or registration, training of the employees that operate the irradiation program, as well as operating the food perishability aspects of their program. In addition, there would be issues related to worker safety.

And we've added a criteria for citations that might be received from other federal agencies or other regulatory authorities related to the operation of an irradiation facility. And then, we have issues related to packaging. On the packaging issue, I'll talk about next, those packaging criteria are listed in 21 C.F.R. 179.45.

I do want to point out that yesterday we did receive a letter from the Food and Drug Administration that will allow, upon the request of FSIS, recently, it will allow for a one-year trial, the use of all radiation materials that are approved for gamma sources to also be used for electron sources or x-ray sources. And so we will be getting instructions out on that that we've received a number of questions on that. I was glad to get that letter from FDA yesterday.

With regard to labeling, this is the area where we get most of the comments. But I'd like to point out that product that's irradiated in its entirety -- and that could be either in the package form or in an unpackaged form -- the requirements would be that labeling would include the logo which is pictured here on

this slide in any color and a statement. And that statement could be treated with radiation or treated by irradiation.

Or if irradiated is in the product name that, too, would suffice. But in any case if it's irradiated, if the product is irradiated in its entirety, it would have both the logo and some identification of the radiation treatment.

If irradiated beef is used as an ingredient in a multiingredient product, such as beef used to make fermented sausage, then the irradiated beef would be listed in the ingredients statement in the order of predominance as it's used in the formulation.

This last slide talks about the radiation sensitivity of E. coli 0157:H7. 0157:H7 is particularly radiation-sensitive when compared to salmonella or to listeria. The d values for E. coli 0157 in the refrigerated state is .25 kilocurie (phonetic.) In the frozen state, it's .45 kilocurie. To point out for salmonella, in the refrigerated state, the d value is .4.

And for listeria, it's .48, so it is quite a bit more sensitive to radiation than the two other pathogens of primary concern in beef. To give you an idea of what it would take to irradiate beef to eliminate it to the levels that would be representative of cooked beef, we have in place regulation 9 C.F.R. §318.23, which

is our cooked beef patty regulation for fully cooked beef patties.

That is associated with a 5-log reduction for salmonella and E. coli 0157:H7. It would require 1.25 kilocurie to irradiate beef in the refrigerated state to achieve a log reduction for 0157:H7. In the frozen state, it would take a higher dose. It would take 2.25 kilocurie minimum to achieve a 5-log reduction. That would make it equivalent to a cooked meat patty.

In terms of the recently issued performance standard rule for cooked roast beef, where the log reduction for salmonella was 6.5 kilocurie -- I mean, I'm sorry -- 6.5 logs, the equivalent in terms of what that would take with the irradiation would be 1.63 kilocurie in the refrigerated state and a minimum of 2.93 kilocurie in the frozen.

And the reason the difference between the refrigerated and frozen state is that the water particles are tied up in the frozen state, and it takes a higher dose to accomplish the same effect. With regard to the organoleptic (phonetic) properties of irradiated meat, our expectation is that there would not be any noticeable or discernible differences in the taste, the color, the odor, or other attributes associated with raw ground beef.

And for those reasons, we believe that irradiation is extremely effective in reducing or

eliminating 0157 from raw ground beef. And it is our expectation that we will have irradiated beef available fairly soon. We do have a training program for our inspectors in the field.

We have roughly four plants that we know are up and getting ready to irradiate beef, mostly in the midwest in the Chicago area and the Sioux City, Iowa, area, and then in the Florida area. Other than that, we're waiting to see what kind of response we do get from the irradiation and meat industry for this technology. Any questions?

MR. BILLY: Any questions from the panel? Okay. Other questions? Perhaps, I can kick one off, Dan. There's a follow-on petition, I believe, from industry to FDA regarding irradiation of meat products. Could you speak briefly about that and what it's about?

DR. ENGELJOHN: Certainly. There are a number of petitions that have been submitted to the Food and Drug Administration for irradiation. Actually, there are seven that have been submitted. And they are being handled by FDA in an expedited manner, in the sense that they have antimicrobial properties. And they will be reviewed on a first-in, first-out-type of basis.

And so I would point out that two petitions submitted by FSIS for hot bone meat and for poultry to change poultry requirements to be the same or consistent with red meat were number 5 and 6 on that list of

petitions. And then, the industry petition for ready-to-eat product was number 7. So it's the last one that's received.

And so our expectation would be that FDA would resolve the petitions that have come in prior in the order they were received. The regulation that we just issued deals with raw meat only and can have non-food seasonings added to it. But that's the extent of additions that could be to the raw meat.

The industry's petition related to ready-to-eat meat products. And it was very broad in the sense that it covers all ready-to-eat meat and poultry products. And that would include all the additives and binders and treatments that occur with ready-to-eat meat.

FDA did begin the process of redoing some of the additives and binders that would be in meat products when they originally began the review for the raw meat petition that was submitted back in 1994. So they've begun the process. But there's an enormous amount of work that also has to be reviewed, particularly from a nutritional and from a toxicological safety standpoint.

It is our hope that we would be able to help with the review of that petition by providing them some expertise. But I think that it would probably be awhile before they are able to address that particular petition that would deal with ready-to-eat products and primarily for the effect of Listeria monocytogones control.

MR. BILLY: Thank you. Question?

MR. MARSDEN: Yes. Jim Marsden, Kansas State University. Dr. Engeljohn, one approach that industry's looking at is to utilize an integrated HACCP plan to reduce the bioburden in the raw meat product prior to irradiation as much as possible, and then to irradiate or pasteurize that product using irradiation at low doses.

But what they documented bioburden control using microbiological testing to document control. How does the agency look at that? You were talking about a 5-log reduction and higher log reductions that would be consistent with other regulations that are in place.

If bioburden were controlled and held at a very low level, we could effectively pasteurize the product without having negative sensory effects, and so on. Is that something that you would look favorably on?

DR. ENGELJOHN: I would certainly agree with the statements that you made about the effect of a total process control where you're integrating a variety of barriers to reduce the bioburden on the product. That is exactly what we would hope industry would move towards in terms of all their processing in meat and poultry products that irradiation would be one of those hurdles that could be added to it.

And you certainly could reduce the level of irradiation that you might want to apply to that product. The log reductions that I've provided in the slide, which

relate to the 5-log reduction and the 6.5 log reduction, were meant as an indicator as to what industry may need to consider if, in fact, 0157:H7 was contaminating raw beef.

Right now, product that is contaminated would need to either be fully cooked or treated so that the pathogen would be eliminated or reduced to a safe level. Those would be the requirements if you were to cook that product.

And so I provided those 5-log reductions as a relationship to ground beef and 6.5 as a relationship to roast beef as examples of the type of reduction that we would, at this point, would view as being clear evidence that you've addressed that particular pathogen, that pathogen only in the product. Radiation is selective for the pathogens.

Again, their radiation sensitivity is considerably different. And so the issue of taking care of 0157 if it's contaminating the product is one thing. If the issue is trying to go for a labeling claim, as an example calling your product pasteurized, we have put discussion in the preamble to the final rule that we believe pasteurization is possible.

It may not be feasible today, but through the controls that you've mentioned of controlling the bioburden, certainly, a manufacturer may be able to remove the vegetative cells of the pathogens that are

there that are of concern to a level equivalent to a ready-to-eat product.

MR. BILLY: Okay. Caroline?

MS. DeWAAL: Thank you. Caroline Smith Dewaal, Center for Science in the Public Interest. Dan, we asked because of concerns that companies might use irradiation as a substitute for good process control and good sanitation in their plants, we actually asked the agency to mandate microtesting prior to meat being irradiated so that, in fact, you could evaluate the amount of bacteria in the product. Why didn't the agency choose to do that in the final regulation?

DR. ENGELJOHN: I'll hedge and say that we addressed it to some extent in the preamble to the final rule. But a general response would be that the agency believes the system we set up with the sanitation standard operating procedures and the written programs associated with that, and then the associated HACCP regulations that we have in place in combination don't provide the opportunity for there to be lax sanitation in combination with irradiation.

Again, our expectation is that irradiation would, in fact, be identified as a critical control point in any processing plant. And we don't see that there would be the opportunity to make more lax the sanitation procedures in place. We also have in place the pathogen reduction requirements for salmonella at this time.

We did put in our directive that if a manufacturer was operating an irradiation process in a HACCP system and included irradiation as part of that, that the checks for the pathogen reduction, the salmonella testing, would occur after the radiation process, as opposed to before.

MR. BILLY: Nancy?

MS. DONLEY: Nancy Donley from STOP, Safe Tables Our Priority. Caroline actually asked my first question. And I'm just going to add, kind of, a comment to it is the necessity that to know the bioload prior to going in that you can have -- if you have a 6-load coming in and you're using a 5-log reduction of a 6-log load and a 5-log reduction, it's not going to be effective. That's a major concern of ours.

Second is maybe you can explain, Dan, why do the agencies choose to, what I'm going to call, roll back the poultry regulation, which had required that the poultry be irradiated in the final packaging? And so they actually rolled that back to remove that requirement to make it consistent with the new rule for red meat.

DR. ENGELJOHN: I would say it's the agency's opinion that, by allowing the flexibility with the poultry, which we hoped we'd be able to do partly through the regulation we issued, and then raising the maximum doses that FDA had previously approved for poultry is that we provide the opportunity for more raw product,

more poultry to be irradiated, and then used as a secondary ingredient in other products.

Back in the early '90s when we issued the poultry regulation FSIS did, in fact, submit a petition to FDA for that particular approval. And we very specifically identified that we believed it needed to be for retail-ready-only product, because at the time there wasn't a great deal of accommodated poultry available in the marketplace or other processed poultry products.

And there certainly were not a great deal of the low-fat poultry products and the sausage products that are available today. So we believe that the irradiation process for the raw materials used as secondary ingredients in products which today cannot be irradiated in their entirety would enhance the public safety and the system that we would have in place for protecting the public health. So I would view the poultry regulations as not being a rollback, but one which would further enhance public safety.

MR. BILLY: Dr. Naidu?

DR. NAIDU: Narain Naidu, Center for Antimicrobial Research. I would like to expand on Caroline's question. In medical devices, when you radiate it, after irradiation you test for pathogens to seem how much bacteria load was initially there.

Is the agency looking at anything on looking for what is the microbial quality? Is bacteria live or

dead? It is still an implementary pathogen. Are you planning to do any testing? After irradiation, what is a dead mass of bacteria in like, for example, pathogen content of the meats?

DR. ENGELJOHN: I would say that the agency has in place a number of microbiological monitoring-type programs. Again, we would view that the irradiation of raw meat, in conjunction with the HACCP system, would be one where we would follow through with our pathogen reduction testing for salmonella.

If a plant were to make a health claim or a labeling claim, such as a very specific reduction for a pathogen or a specific statement about the effect of the irradiation process, that would be something that we would, in fact, verify as part of the HACCP plan documentation that the plant would have as to how they were able to achieve what they are claiming on that label.

MR. BILLY: Okay. Thank you very much. I think we're going to move on now for the next presentation, which focuses on the area of our risk assessment. The presenter will be Dr. Mark Powell from the Office of Public Health and Science in FSIS. Mark?

DR. POWELL: Thank you. I'm going to apologize that hard copies were not available. However, if you'd like to request a hard copy be sent to you, you can do so at the registration desk. This presentation file will be

made available electronically at the OPHS Website. And I'll put up the website address for that in the final slide.

On behalf of the FSIS E. coli 0157:H7 Risk Assessment team, I'm pleased to have this opportunity to summarize the draft findings of the agency's assessment of E. coli 0157:H7 in ground beef.

My presentation today will cover four areas: first, a brief background on the process by which the risk assessment was developed; second, our best estimate of the magnitude of the public health problem; third, the process risk model's predictions regarding the occurrence of E. coli 0157:H7 in ground beef production; and finally, the modeled results of some alternative mitigation scenarios.

In the interest of time, during my presentation, I'll refer to E. coli 0157:H7 simply as 0157. The 0157 risk assessment has been a large team effort. And I'd like to take this opportunity to recognize the many contributions of team members, consultants, and scientific peers.

The 0157 Risk Assessment Project began taking form in March 1998 when I formed a resource group during the formulation stage of the assessment. In October 1998, a meeting was held to solicit public input at an early stage of the process and to release a preliminary

document describing the modeling approach and summarizing the data acquired by the team to date.

We have emphasized peer input during the development phase of the assessment through presentations at SRA and IAMFES and by convening a week-long, interagency workshop on microbial pathogens in food and water that involved microbial risk assessment practitioners from USDA, FDA, EPA, United Kingdom, and New Zealand.

The peer-review process began in December 1999 with presentations to SRA and the National Advisory Committee on microbiological criteria for food. The draft results that I will present today reflect changes that have been made to the process risk model in light of comments received through the peer-review process.

The 0157 process risk model covers all aspects of ground beef production and consumption from farm to table. The exposure assessment consists of three sequential model segments. The production segment outputs the prevalence of 0157 in live cattle. The slaughter segment outputs the prevalence and levels of 0157 in beef trimmings destined for grinding.

The preparation segment outputs the prevalence and levels of 0157 in consumed ground beef servings. And this final output of the exposure assessment feeds directly into the dose response assessment. The final

output of the model is the annual number of 0157 cases due to ground beef in the U.S.

The scope of the assessment is limited to ground beef as a vehicle of infection, and therefore, does not include cross contamination to, or from, ground beef or person-to-person secondary transmission. The scope of the present assessment is also limited to 0157, and therefore, does not include all interohemorrhagic E. coli.

However, the paucity a replosity of reported outbreaks due to non-0157, combined with the higher isolation rates of seratype 0157:H7 in prospective studies indicates that other EHEC's do not attain the public health importance of 0157 in the United States. The scope of the assessment is also annual and national, although data are available at some points to model at seasonal or regional scales.

Insufficient data are available to model slaughter, processing, preparation, and other processes at seasonal or regional sales. The scope of the draft assessment includes cooked ground beef products. The present draft assessment does not include products containing ground beef that are prepared by means other than cooking, for example, fermented sausages.

We also have not included raw ground beef consumption which is a very uncommon practice in the U.S. But the ingested doses would be analogous to very

undercooked ground beef. And this is considered. Intact steaks and roasts are excluded, because potential surface contamination would very likely be eliminated during cooking.

The present draft assessment does not yet cover other nonintact cuts of beef, such as steaks or roasts that have been blade-tenderized, or injected with needles that may introduce surface contamination into the interior muscle tissue. However, FSIS does plan to address the other nonintact products in the subsequent iteration of the risk assessment.

This table presents our best estimate based on epidemiologic data independent of the risk assessment model of the magnitude of the 0157 problem attributable to ground beef and places it in the context of the magnitude of the problem from all sources. We estimate that somewhere between 16 and 40 percent, with a most likely value of 18 percent of all cases, are due to ground beef.

The estimated distribution of the total number of cases of 0157 due to ground beef has a median of approximately 16,000 and a 95-percent interval of approximately 9,500 to 29,000. Approximately 10 percent of the cases are characterized as severe; that is, bloody diarrhea for which the patient seeks medical care.

The estimated annual number of deaths due to 0157 in ground beef ranges from 5 to 20. This figure

compares the epidemiological estimate that the number of cases of 0157 due to ground beef with the results predicted by the risk assessment model under the baseline, or as-is scenario.

The broader red curve, which peaks at about 15,000 cases per year, this characterizes the full range of uncertainty about the epidemiologic data, while the narrower blue curve with a peak around 20,000 0157 cases per year only represents our uncertainty about the central tendency or the most likely value, if you will, of the draft risk assessment model.

The full range of uncertainty in the risk assessment model would be much greater, but the degree of overlap between these two curves suggests that we may draw inferences from the model with some degree of confidence. This figure presents the model's estimated prevalence of 0157 at various points in the ground beef production process, including the complete upper and lower bounds of uncertainty in the risk assessment model.

Here, CB connotes cow bull, and SH means steer/heifer. Our best estimate of the prevalence of 0157 in live cattle destined for ground beef production is 11 percent. The bounds of uncertainty depend upon the class of animal considered, fed or culled, but range from less than 5 percent to greater than 15 percent.

The estimated prevalence of 0157 on carcasses in the chiller ranges from a fraction of a percent to

approximately 3 percent. For the cow bull plants, the estimated combo bin prevalence is 15 percent with a range of uncertainty of 6 to 28 percent. For steers and heifers, the estimated combo bin prevalence is 41 percent with a range of 22 to 59 percent.

Our best estimate of the prevalence of 0157 in grinder loads is 89 percent with a range of uncertainty from 71 to 96 percent. Now, I'll proceed to the modeled results under a series of alternative mitigation scenarios. In each of the scenarios considered, we do not specify how the mitigation would be achieved, but simply pose what-if questions to the risk assessment model.

In each case, we estimate the effect, leaving everything else in the model the same of the mitigation on the estimated annual number of 0157 exposures. This figure, then, presents our current best estimate of the annual number of 0157 exposures in ground beef servings after cooking.

The range is large, about 240 to 340,000. But as in the model comparison in slide 9, this figure does not capture the full extent of uncertainty in the draft risk assessment model. This figure presents the estimated reduction in 0157 exposures, leaves everything else in the model unchanged due to a 25-percent reduction in the prevalence of 0157 fecal-shedding, live cattle prior to slaughter.

This scenario estimates the effect of a 25-percent reduction in the prevalence of 0157 on carcasses at the chiller after decontamination measures. This figure shows the estimated effect of reducing by 25 percent the frequency of internal ground product temperatures during storage that are in excess of 41 degrees Fahrenheit.

This figure, then, compares the three mitigation scenarios just considered. Each appears to have a significant effect in reducing the number of exposures. We have not yet modeled the cumulative effects of multiple mitigations.

And while each of the hypothetical mitigations presented appears to have a significant effect in reducing exposures, these results need to be interpreted cautiously, and further analysis of the process risk model is needed before we can quantify the public health effects of these mitigations. For example, we should not expect to find a direct proportional correspondence between the frequency of exposures and the number of cases.

In other words, the 25-percent reduction in exposures may translate into a reduction in the 0157 illnesses of greater than or less than 25 percent. Nevertheless, these what-if examples demonstrate the real utility of the risk assessment model as a tool to support risk management decision-making.

This final figure shows the estimated effect of testing 25 to 100 percent of grinder loads produced at plants using the current FSIS method and rendering pathogen-free any loads that are detected positive. Again, the effect in this range of testing appears significant.

A 100-percent testing scenario could be considered as an upward-bound estimate of the direct impact of such a program at this point, although a testing program would also have indirect impacts that may be difficult to predict or to quantify. The next step for the 0157 Risk Assessment team is to draft a report documenting the baseline risk assessment model.

We anticipate releasing a draft for public comment and peer-review in the spring. Before concluding, I'll draw your attention to the project's website where the draft report and other project-related information, including this presentation file, will be made electronically accessible.

MR. BILLY: Any questions? Questions from the panel? Dan?

MR. ENGELJOHN: This is Dan Engeljohn with USDA. Mark, could you clarify one of the statements you made about the ground beef that was used for your modeling the information that you put in here? Was it for ground beef that's used specifically for ground beef,

for raw ground beef? Or was it used for ground beef that may be used for other ground beef products?

DR. POWELL: We considered ground beef meals being 100-percent ground beef and another category of servings in which ground beef was an ingredient in the serving. So the full range of servings that include ground beef.

DR. ENGELJOHN: This is Dan Engeljohn again. Could that include product that would be used for cooked meatballs?

DR. POWELL: Yes.

DR. ENGELJOHN: Okay.

MR. BILLY: Yes, Caroline?

MS. DeWAAL: Thanks. Caroline Smith DeWaal, Center for Science in the Public Interest. I have two questions. First is where did you get your prevalence number for the incidence of E. coli 0157:H7 in the live animal?

DR. POWELL: This was based on a number of studies that have looked at the gastrointestinal prevalence of 0157. We did not consider the hide prevalence, the GI-positive prevalence of live animals that we're estimating.

MS. DeWAAL: So this does not include some of the most recent data on the prevalence of the GI tract? Is that what you're saying?

DR. POWELL: It does include the most recent evidence on the gut prevalence. It does -- we do not meddle the hide prevalence. There has been some reports recently on hide prevalence.

MS. DeWAAL: And what's the high-end prevalence estimate?

DR. POWELL: For GI-positive?

MS. DeWAAL: Yes?

DR. POWELL: Our best estimate is 11 percent, and the bounds is 5 percent -- less than 5 percent to greater than 15 percent for GI-positive live animals destined for ground beef production.

MS. DeWAAL: And secondly, of all the scenarios you did test, you found that testing 100 percent of the grinder loads really rendered the greatest public health benefit?

DR. POWELL: That was intended to be an upper-bound estimate on the effect that, not only testing, but also rendering pathogen-free any grinder loads that were detected positive could have.

MS. DeWAAL: Did you model any testing at the carcass level?

DR. POWELL: We have not done that yet.

MS. DeWAAL: And just to clarify for myself, the prevalence fact that you have identified, the prevalence in the combo bins was 41 percent, and the prevalence in the grinders was 89 percent.

DR. POWELL: Let me go back to that.

MS. DeWAAL: So 89 percent of grinders, you're estimating, are contaminated or may render contaminated product?

DR. POWELL: For cow bull plants, the estimated combo bin prevalence is 15 percent with a range of uncertainty of 6 to 28 percent. For steers and heifers, the estimated combo bin prevalence is 41 percent with a range of 22 to 59 percent. And then, our best estimate of prevalence of 0157 grinder loads is 89 percent with a range of uncertainty from 71 to 96 percent.

I should note that most of the levels predicted by the model are at very low levels that would be very unlikely to be detected by available testing methods.

MR. BILLY: Dr. Gill?

DR. GILL: Colin Gill, Agriculture Canada. The models, I believe, are constructed on the basis that all contamination with E. coli 0157:H7 occurs as a result of contamination of carcasses with feces from shedding animals.

Seeing as how the mouth of the animal, persisting populations of bacteria, and improperly cleaned equipment, and bacteria which grows in equipment which warms during processing, are potentially major sources of E. coli, and therefore of E. coli 0157:H7, what effect do you think taking these sources of

contamination into account would have on the predicted value of your models?

DR. ENGELJOHN: The model currently does include the potential for contamination during fabrication. We model, the correlation between the GI-positive animal and the likelihood that a carcass is contaminated. It may be contaminated by itself. It may be contaminated by the environment. It may be contaminated by an adjacent carcass.

We don't specify the mechanism by which a carcass is contaminated. We rely simply on the empirical evidence that establishes our best estimate of the fraction of carcasses that become contaminated, given an incoming prevalence of GI-positive animals. So we cannot specify the mechanism by which the carcasses become contaminated.

However, the model as it is currently drafted and composed does seem to comport reasonably well with our estimate from an independent source of data, the epidemiologic data. Therefore, we feel that looking at these alternative scenarios and the effects that they might have, it's reasonable to draw inferences from the current drafted model.

MR. BILLY: Kim?

MS. RICE: Kim Rice, American Meat Institute. The rest of the data on combos and product, where did you

get that? Where's that data coming from? You said you got the carcass data from studies done on GI levels.

DR. ENGELJOHN: Right.

MS. RICE: The other data, I missed that.

DR. ENGELJOHN: The model has been constructed to predict at various points. And we have "ground-truthed" the data at the point of ground beef production with the FSIS testing data that's been done.

We've also "ground-truthed" the data on the carcass prevalence from the FSIS testing that's been done, taking into account the sample size, the sensitivity of the tests. We presented this information at the National Advisory Committee meeting in December. And so you can get a more complete description of the underpinnings of the model from that presentation.

MS. RICE: So the numbers that you have on, not this chart, but the one where it says -- yes, that -- those for the combo bin and grinder levels, those are estimates? They are not actual data that you have on incidence rates?

DR. ENGELJOHN: Well, they are modeled estimates that, at the grinder load, are -- there's an overlap with the prevalence that would be estimated directly from the FSIS testing data, taking into consideration the sample size and the sensitivity of the test.

MR. BILLY: Nancy?

MS. DONLEY: Nancy Donley from STOP. I actually have two questions. Number one is how much of this research was new research that you commissioned to have? And were there other parts of this research that were studies that were given to you?

DR. ENGELJOHN: We have relied on the available, publicly available, information for the most part. There has not been a lot of data that's been submitted to the docket in response to our request in October of '98 for data submissions. So we have used, for the most part, the publicly available data, the data that's produced by FSIS.

We have had a couple of submissions from a couple of plants regarding their testing data in addition to that. And as I said, we were able to incorporate some of the new information on the live animal prevalence that has been coming out. But a lot of the reported findings have not yet made their way into the published literature.

MS. DONLEY: And then, my second question is I find it interesting that you kind of did a what-if scenario on this end of the chart, if you will, at the grinder of your testing 25 percent at the grinder and 100 percent at the grinder level. Did you take it the other direction and look at it in the live animal and say what if we reduced it 25 percent or 100 percent in the live animal and what would the results be?

DR. ENGELJOHN: The scenario, the live animal scenario that we considered, again without specifying how it might be done was what if the live animal prevalence were reduced by 25-percent? And that was estimated to have a significant effect. Again, we aren't at the point yet where we can quantify the effect, but we're confident, based on this estimate, that there is a significant effect.

MR. BILLY: Could you, just for everyone here, explain what the red dot means in the context of this graph?

DR. ENGELJOHN: Right. The blue curve shows the -- our estimate, our most likely estimate, of the number of exposures after cooking under the baseline scenario. We have some estimate of our uncertainty regarding that most likely estimate.

We have done one run under this 25-percent reduction of live animal prevalence scenario and achieved about a 210, 205,000 annual exposures. Because it lies well outside the lower tale of this distribution, we feel confident in saying there's a significant effect, a significant reduction achieved by reducing live animal prevalence by 25 percent.

MR. BILLY: Okay. Thank you. You can speak.

MR. BOLTON: Lance Bolton, Dupont Quality Con. My question is whether you have taken into account the methods used to determine baselines like the number of

organisms in live animals and GI tracts and the relative sensitivity of those methods and whether or not the model has been adjusted for those?

DR. ENGELJOHN: We have modeled sensitivity as a function, both of the sample size and the concentration in the sample.

MR. BOLTON: But the actual methods used were the same methods used in the tests that you put together?

DR. ENGELJOHN: We have adjusted all prevalence estimates to actual prevalence that would be inferred, given the test sensitivity.

MR. BILLY: Okay. The next person I have is Rosemary.

MS. MUCKLOW: I'm playing catch-up. I'm sorry I was a little late this morning.

MR. BILLY: Would you say your name and affiliation?

MS. MUCKLOW: Excuse me. Rosemary Mucklow with the National Meat Association. The data that you base this on, could you tell me again, I think you mentioned it, the time frame that it was collected on, the data that this is based on? Is this new data, recent data, old data? What sort of time frame was it collected over?

DR. ENGELJOHN: There's a wide variety, voluminous data. We have used the most recent data available. In some cases, obviously, the surveillance

data, it will be more time-sensitive. The more recent data will be more reflective.

However, other data that may be on, say, the efficacy of a process, there's no need to think that that would become outdated as long as the report is well-documented. I'd refer you to the preliminary pathways and data book that lays out a lot of the information that's been used.

MS. MUCKLOW: And if I go to your website, as listed up there, will I find the backup for what you have explained to us this morning and the various charts, and so on?

DR. ENGELJOHN: The draft report will be made available on this website.

MS. MUCKLOW: When?

DR. ENGELJOHN: We anticipate releasing the draft report for public comment and peer-review this spring.

MS. MUCKLOW: I just recently became aware of a paper that was published and peer-reviewed called Topics in Microbial Risk Assessment Dynamic Flow-tree Process which was, I understand, funded by FSIS. And Harry Marks was the lead investigator. This is above my grade level in statistics. Were the findings of this paper included in what you discussed this morning?

DR. ENGELJOHN: We have utilized the data that is the base, the clinical trial data that is the basis

for that paper that you're referring to. We have modeled those responses for 0157:H7 somewhat differently. The Shigella species that Marks and colleagues reported are considered to be an upper-bound on the infectious dose for E. coli 0157:H7 with the enteropathogenic E. coli.

The EPEC's are considered to be a lower-bound, and so we have modeled the dose response as bounded by an envelope, essentially, between those two dose response curves with the most likely value for 0157 that is derived from outbreak data.

MS. MUCKLOW: Are there any other published papers that were funded by FSIS? This is the first I've heard of this one that's a year and a-half old. Are there any others that were included as resource material? Or will we wait until you issue the White Paper before we know that?

DR. ENGELJOHN: Again, I would refer you to the Preliminary Pathways and Data Book that's been available since October 1998. That documents the available data to that point. We have since acquired and been made aware of other data. And that will be fully documented in the draft report that is to be released soon.

MS. MUCKLOW: And the Preliminary Pathways Data Book is up at that website?

DR. ENGELJOHN: That's correct.

MS. MUCKLOW: Thank you.

MR. BILLY: Okay. Next, Ann?

MS. HOLLINGSWORTH: Ann Hollingsworth, Keystone Foods. I have two questions, the first probably is just from ignorance. You used a term called "ground-truth". Could you explain that in a little more detail so I can understand what you meant?

DR. ENGELJOHN: That is our term for evaluating the consistency between the model's prediction and the empirical data that's available. Another term would be validate, but that's perhaps a little bit too strong. There's a lot of discussion within the statistical circles about just what validation of a model is.

Just because it agrees with the observed data doesn't necessarily mean that, you know, a stopwatch is right twice a day. It doesn't mean the model is necessarily working. But that is our comparison of the available empirical data with the model's predictions.

MS. HOLLINGSWORTH: Okay. The second question when you were discussing the grinders portion where you said that 89 percent was your best estimate of contaminated product. You said that you compared that to the USDA data for E. coli 0157:H7 and testing procedures that were outlined in 101 (phonetic).

That only includes those portions of the grinders that choose not to test within the limits of that directive and does not include a vast number of grinders to test that product. Does this include the

data from a representative portion of the grinders, I don't believe?

DR. ENGELJOHN: That is the best empirical data that we have. We would welcome more representative data if it were to become available.

MR. BILLY: Okay. I'm going to take two more, then we're going to have a brief break. So the next person I have on the list is Dean.

MR. DANIELSON: Thanks Tom. Dean Danielson, IDP. Mark, I didn't -- don't believe that you took into account or at least spoke to potential seasonal and regional differences or activities of 0157:H7 that we know occur out there. I've really got three points to make. That's one.

Number two, that 89 percent level, that gets to be a pretty significant number. I'm curious as to how you got that. I would be interested in reviewing that in more detail, but in particular with a 15 percent cow bull rate of 41 percent fed beef, and then all of a sudden we leaped 89 percent on grinder loads. It's a pretty interesting leap that kind of has baffled me for the moment.

DR. ENGELJOHN: Okay.

MR. DANIELSON: Then I'll finish that, what is a grinder load to start with? I don't understand what that term means. That would be a point. And the second question and the third question is you define a current

FSIS method for testing grinder loads. I'm not aware of a defined FSIS method for testing grinder loads, so some clarification.

DR. ENGELJOHN: I'll try and remember your questions in turn. We're modeling the average over a year throughout the model, because although we're aware that at certain points there's data available to, that suggests seasonal variation, at other points in the model there, we don't have that sort of seasonal data.

And therefore, we're not looking at geographical or seasonal variation throughout the model. We have to go to the lowest common denominator of the available data and model at the annual national level. One of the reasons, again, that the prevalence is higher than previously reported is that the vast majority of the positive grinder loads, positive carcass bins, combo bins that the model predicts are on the order of one log per combo bin, okay, or one -- zero logs per grinder load.

So these are large quantities of product contaminated at very low levels. Nevertheless, the model predicts that they contain, you know, if a grinder load that is composed of, you know, three or four combo bins, 2,000-pound combo bins is predicted to contain one organism, it is a positive combo bin, I'm sorry, grinder load.

We model grinder loads containing anywhere from one to several 2,000-pound combo bins that would be

ground together. That's what we refer to as grinder loads. So it's a variable quantity.

MR. DANIELSON: Okay.

DR. ENGELJOHN: Did that address all of your questions?

MR. DANIELSON: Yes.

MR. BILLY: One last question, and then we're going to take a break. Jim? Just speak up.

MR. HODGES: Jim Hodges, American Meat Institute. I have two questions. When you presented this data to the Micro Advisory Committee, they had several questions, suggestions, even criticisms of the way of projecting through the system about comparable data to support some of the conclusions. Do you plan to return to the micro committee for advice and guidance after you've provided your model? And if not, why?

DR. ENGELJOHN: We plan on making the draft risk assessment available for public comment and peer-review. And the National Advisory Committee has been part of that peer-review process.

MR. BILLY: So the answer is yes.

MR. HODGES: Will you go back at the committee to have a discussion about that at the micro committee to have questions?

DR. ENGELJOHN: Yes.

MR. BILLY: Yes.

MR. HODGES: The second question, in your projections at the combo and grinder levels, I'm predicating on incidence levels that are occurring in carcasses, I believe that you are projecting that those are standing to the system on a theoretical basis. You're saying that 89 percent of a grinder load has a potential to contain at least one organism.

How do you reconcile that with the data, the ground beef data, that FSIS selects in their 0157 sampling program and shows somewhere in the neighborhood of .4 percent? And secondly, what can you infer from that about testing and its effects?

DR. ENGELJOHN: One of the analyses that we presented at the micro committee showed the overlap between the model's predictions and that which would be inferred from the FSIS Ground Beef Testing Program. Again, the levels that are predicted by the model are such that it would be very unlikely, given a 325-gram sample from a very large quantity of product contaminated at low levels, to be detected.

The vast majority of servings, for example, that would come out of a large grinder load that would contain one log total of contamination would obviously be negative. There would only be a small number of servings from such a grinder load that would contain any 0157:H7.

MR. BILLY: Okay. We're a little bit behind schedule, obviously, so what I'd like to do is try to

compress this break and ask that you return by 5 after 11.

(Whereupon a recess was taken.)

MR. BILLY: If people would take their seats, please, I'd like to get started. Okay. I'd like to, before I introduce the next speaker, just make a brief comment which is that to reiterate what was said about this preliminary risk assessment. Again, the results that have been presented are preliminary results that will be contained in a draft report.

That report will be subject to further peer-review, as well as public input. We recognize that there are new data that will be presented today from various sources. It would be our intent to use that data to the extent that we can, in terms of further refinements of the model, and obviously, the results that it would predict. So this is an iterative process.

We feel we've come a long way, in terms of the development of the model. Obviously, it's critically important that we have the best data that we can find to use in the model to make these kinds of predictive results most useful. So we look forward to the additional presentations today, as well as further comment and input that we'll receive when the draft report comes out this spring.

Next, I'd like to introduce Dr. Sonja Olsen. She is with the Foodborne and Diarrheal Diseases Branch

in the Division of Bacterial and Mycotic Diseases at the Centers for Disease Control and Prevention. She'll make a presentation on E. coli 0157:H7, a continuing threat to our food supply. Dr. Olsen?

DR. OLSEN: Thank you. Good morning. Today, I'm going to talk to you about E. coli 0157:H7, a deadly pathogen which continues to threaten our food supply. For simplicity and brevity during the rest of the talk I will refer to this organism as 0157.

First, I'll give you a little background on the organism. Then, I will tell you about CDC's estimates of the burden of illness from 0157 in the United States. After that, I will discuss recent trends over time, and then end with a discussion of the sources of infection.

0157 was first identified as a cause of human illness in 1982, following two outbreaks of bloody diarrhea that were linked to hamburger patties served at fast-food chain restaurants. Since these first outbreaks, we have learned a great deal about 0157 and the illness it causes. The organism has a very low infectious dose. Less than 10 organisms can cause infection.

It is shed in the feces of healthy cattle. 0157 is one of a number of sero-types of Shiga toxin-producing E. coli that can cause disease. After ingestion, illness begins with nonbloody diarrhea and

abdominal cramps. In many, but not all persons, the illness then progresses to bloody diarrhea.

In most persons, the illness results within a week, but in approximately 6 percent, it progresses to hemolytic-uremic (phonetic) syndrome. Hemolytic-uremic syndrome is a life-threatening condition characterized by anemia, low platelet count, and kidney failure. It affects persons of all ages, but the highest rate is in children less than five years old.

In U.S. children it is the major cause of acute kidney failure; 3 to 5 percent die, and 10 percent have stroke or chronic kidney failure. There's no specific treatment for 0157 or HUS. Antibiotics do not cure the illness. Therefore, prevention is critical.

So how big is the problem of 0157 in the United States? To estimate the burden of illness for specific diseases, we rely on surveillance data. This slide shows a surveillance pyramid which represents the burden of illness for any given disease. As you can see, there are limitations with surveillance data.

In order for a case of any disease to be captured by routine surveillance, the following events must occur: The ill patient must decide to visit a health care provider. Provider must decide to obtain a stool culture. The stool culture must be tested for the organism. And the test must be positive. And finally, the results must be reported to public health officials.

At each of these steps, some proportion of cases are lost, or at least could be lost. Recognized outbreaks account for only a very small proportion of reported cases, essentially, the tip of the iceberg. To quantify the degree of attrition at each step, CDC, FSIS, FDA, and state health departments created the Foodborne Diseases Active Surveillance Network, or FoodNet.

The network includes nine sites, the most recent addition being Colorado, with a combined population of 28 million residents under active surveillance. FoodNet sites connect active surveillance for seven bacterial pathogens, including 0157.

In addition, FoodNet employs a series of surveys that help us better understand the degree of underreporting at each stage of the pyramid. First, the population survey in which people are contacted at home and asked whether they've had acute gastroenteritis recently, and if so, did they seek medical care?

Second is a physician survey in which health care providers are asked how often they obtain stool cultures from patients presenting with acute gastroenteritis. Third is a laboratory survey to determine how often stool cultures are tested for specific pathogens and how often the results are reported to health officials.

Through the system of surveys, it is possible to work backwards from the number of cases detected

through active surveillance to determine the number of cases that likely occurred in the population. Using this approach, it can be shown that for every culture-confirmed case of 0157 reported to CDC, there are a total of 20 cases that occurred in the community.

Thus, we use a multiplier of 20 to estimate the true number of infections in the country. Using FoodNet data, CDC recently derived national estimates for foodborne illness. This slide shows the results for 0157. We estimate that 0157 causes over 73,000 illnesses, 2,100 deaths, no hospitalizations, and 61 deaths overall. Of these, foodborne transmission is estimated to cause over 62,000 illnesses, over 1,800 hospitalizations, and 52 deaths.

Now, I want to discuss some of the recent trends. Several sources of data have led some to believe that the number of 0157 infections per year is increasing. The next two slides are meant to explain why this is not necessarily true and may be an artifact of reporting. This slide shows the number of outbreaks of 0157 infection in the United States between 1982 and '98.

There were a total of 206 outbreaks reported to CDC. As you can see, it appears that the number of outbreaks is increasing dramatically. However, 0157 was not discovered until 1982. And since this time, our diagnostic and reporting capabilities have been improved dramatically. Further, after the Western states outbreak

in 1992 and '93, 0157 became a nationally notifiable disease.

In addition, PulseNet, a network designed for states to compare molecular patterns of isolates from foodborne pathogens such as 0157, was added in 1998. As a result, we are now able to detect smallersized outbreaks that were probably occurring all along, but not detected through routine surveillance.

Another way of showing that our reporting has improved is to look at the number of states reporting by year. Shown here are national surveillance data for 0157 infections for the period 1993 when 0157 became reportable through '98. The yellow line indicates the number of states reporting, and the red bars indicate the number of cases reported.

As you can see, the number of reported cases has increased in recent years. But most, if not all, of this increase can be attributed to the growing number of states reporting. Perhaps, the best data we have to assess recent trends are FoodNet data, which is based on diagnosed cases of 0157.

Because FoodNet has a defined area, it is possible to calculate rates. Therefore, these data most accurately reflect current trends in the United States. This slide shows the most recent trends in 0157 infections using FoodNet data. Shown here are the number

of cases per 100,000 persons of 0157 infection and pediatric HUS.

As you can see, the rate of 0157 infection was 2.7 in '96, 2.3 in 1997, and 2.8 in 1999. Although there are only three years of data, the rate of infection seems to be fairly stable. Similarly, the rate of pediatric HUS was relatively stable at 0.58 in '97 and .7 in 1998.

Now, I'm going to discuss what we know about the sources of the infection of 0157. Much of what we know about the epidemiology has been learned from outbreak investigations. This slide shows the various modes of transmission in 206 outbreaks reported to CDC since 1982. Foodborne transmission accounts for the majority of recognized outbreaks.

Person-to-person transmission accounts for 20 percent. However, it's important to note that 0157 does not naturally live in the human intestine. Therefore, most outbreaks, due to person-to-person spread, often begin with a person who ate a contaminated food. Drinking water or swimming was a mode of transmission in 10 percent of recognized outbreaks.

Now, I'm going to focus just on those outbreaks that were foodborne. This slide lists the major food categories implicated in foodborne outbreaks with a known source of transmission. As you can see, ground beef accounts for the greatest proportion of outbreaks, 55 percent.

Other types of meat, such as beef or game meat, account for about 10 percent of outbreaks. And produce accounts for 20 percent. Although there's some speculation that in recent years the number of outbreaks due to ground beef is decreasing and the number due to produce is increasing, our data do not support this trend.

Now, we have discussed the sources of transmission ascertain from outbreak data, which actually represent a very small number of cases, so what do we know about the source of infection from sporadic infection? There have been several case control studies to look at risk factors for sporadic illness.

Sporadic infections are single cases that don't have any obvious connection with any other case. The first sporadic case control study was conducted in 1990 to 1992 in 10 medical centers throughout the United States. In uniformed and varied analysis, illness was significant associated with eating hamburger, eating uncooked hamburger, and eating in a fast-food restaurant.

These findings confirmed and expanded on our knowledge of 0157 transmission from outbreaks. A second case control study of sporadic infections was conducted from 1996 to 1997, using cases in participating FoodNet sites. The results were intriguing. Again, illness was significantly associated with eating pink hamburger or ground beef at home or at a restaurant.

However, in marked contrast to the previous study, illness was associated with eating hamburger at a restaurant that was not part of a fast-food chain. Why should this be? What we'll call an important event that occurred between these two studies, namely the massive Western states outbreak of 0157 in fast-food chain restaurants that occurred in 1992 and 1993, as a result of this outbreak, we suspect that the fast-food industry implemented several changes, including improved quality control of meat and cooking methods with higher temperature and longer times.

These process control measures mean that ground beef served in fast-food restaurants is safer than it was before. And as a consequence, people may now be less likely to become infected with 0157 by eating ground beef served at a fast-food restaurant.

In 1996, USDA introduced the pathogen reduction and Hazard Analysis and Critical Control Points, or HACCP Rule. The objective was to reduce pathogens in our food supply with process control and microbiologic testing at the slaughterhouse and grinding level. Just as surveillance is critical to monitoring disease trends in humans, it is equally important to monitor trends of contamination in the food supply.

CDC feels that HACCP is a rational, scientifically sound program that will ultimately help reduce the incidence of illness due to foodborne

pathogens. As early as 2000, HACCP was still in the process of implementation for ground beef. Therefore, it's too early to expect to see significant declines in the incidence of 0157.

However, we know that the incidence of other diseases has decreased following targeted HACCP-like programs. A good example is the decrease in salmonella enteriditis (phonetic) following a flock-based quality control program. I want to briefly mention that in addition to 0157, there are other serotypes of Shiga toxin-producing E. coli.

Other current surveillance for these organisms in humans is limited. Efforts are underway to improve them. Like 0157, these pathogens, including 0111 and 026, have been found in cattle and ground beef and are known to cause severe illness and even death in humans.

As our diagnostic and surveillance tools improve, non-0157 E. coli are likely to play a larger role in human disease. For this reason, it's important to be thinking of these organisms as potential food contaminants. Fortunately, because of their similarity to 0157, it is likely that current efforts, such as HACCP, will effectively reduce contamination from these pathogens, as well.

In summary, 0157 infection remains a serious problem in the United States. Infection with the organism can cause severe illness and even death. CDC

estimates that there are approximately 73,000 illnesses and 61 deaths each year in the United States due to 0157. Foodborne transmission accounts for the majority of infections, both outbreak-related and sporadic.

And ground beef continues to be identified as a major risk factor. Our case control studies have shown how HACCP and other interventions by USDA and the meat industry have helped to reduce specific problems, namely in the fast-food industry.

Foodborne transmission of 0157 is preventable. And changes in the meat industry are an important part of this prevention. Comprehensive prevention strategies from farm to table are needed. Thank you.

MR. BILLY: Okay. Thank you very much. Any questions from the panel? Dan?

DR. ENGELJOHN: Dan Engeljohn with USDA. On the slide that you had about CDC supporting HACCP with micromonitoring, could you explain or go into that in a little more detail what you meant by that?

DR. OLSEN: Yes. I think we think it's an important part of the HACCP and control part point, control process that just as we are, you know, monitoring for pathogens in humans, that it's important to monitor for these pathogens in the food supply to know what the level of contamination is and, as you know, a way of monitoring the different control points.

DR. ENGELJOHN: If I could follow up, so you would envision that as an FSIS-directed, monitoring-type program?

DR. OLSEN: Correct.

DR. ENGELJOHN: Okay.

MR. BILLY: Bill?

DR. CRAY: Bill Cray, FSIS. Are you aware of any differences in the actual isolates that are associated with produce foodborne illness, and say, beef isolates?

DR. OLSEN: You mean in terms of virulence or --

DR. CRAY: Yes, any distinguishing features.

DR. OLSEN: No, I'm not aware of any further characteristics of isolates at that level. As far as we know, it's the same, you know, in terms of virulence and in terms of --

MR. BILLY: Okay. Other questions? Yes.

DR. NAIDU: Narain Naidu, Center for Antimicrobial Research. One thing is puzzling to me. If E. coli 0157 in so exclusively associated with cows and cattle, why there is such a low or no incidence of disease in farm workers and their families? Why should it only be with hamburgers?

DR. OLSEN: Well, that's a very good question. There's some speculation that actually people who are exposed over long periods of time, perhaps, on the farm

or through, say, chronic water contamination might develop some immunity to the pathogen. In fact, I investigated a fascinating outbreak where we saw just that.

We were looking for serologic evidence of immunity, and there were two groups of people. There were the town residents, who had a much lower attack rate, and there were a bunch of out-of-town visitors who had come to this town for the weekend. And it was a waterborne outbreak. But their attack rate was much higher, suggesting that perhaps, you know, if you're chronically exposed, you may develop some immunity. So that may play a part in --

MR. BILLY: Okay. Dr. Gill?

DR. GILL: Colin Gill, Agriculture Canada. There's been two international discussion groups on the purposes of microbiological testing in relation to the safety of meat in the last year. And both those groups came to the conclusion that end-product testing is a total waste -- for pathogens is a total waste of time. Could you please elaborate on why CDC apparently thinks it's a valuable tool for assuring the safety of the meat?

DR. OLSEN: Right. End-point testing, you mean the packaged ground beef?

DR. GILL: Yes.

DR. OLSEN: I don't -- that's not what I meant to imply. I apologize if I did. I think we're saying

microbiologic testing at different points are important, you know. And the farther back you can push that testing is, you know --

DR. GILL: Well, we're getting very little prevalence of organism. And it's a very dubious value for -- in relationship to implementing a HACCP, why do you think the testing of the pathogens is a useful tool?

DR. OLSEN: Well, I think as we're developing these new diagnostic tools, like the immunomagnetic beans, we're not really sure what the prevalence of this pathogen is in beans. And you know, similarly, we didn't know what it was in humans. And you know, if you look you might find it.

And I think it's just, you know, you can implement the control process at different points, but, you know to make changes that should have an effect on the pathogen load in meat. But I think you're not going to know until you test it.

DR. GILL: I would take it from that, your suggestion from CDC focuses on continuing to do testing to better inform all of us about the impact of various preventive control measures in the farm-to-table continuum that you're not necessarily saying that end-product testing is a procedure for controlling pathogens, such as 0157 in the food supply.

DR. OLSEN: Correct.

DR. GILL: Is that correct?

DR. OLSEN: Yes.

MR. BILLY: Other -- yes, Dr. Naidu?

DR. NAIDU: Your answer for the previous question that probably certain populations could develop antibodies against E. coli 0157, now does it mean because this opens up a different Pandora's box, does it mean that you have healthy carriage of E. coli 0157:H7 in normal populations which can contribute very good handling for the transmission of E. coli 0157:H7?

DR. OLSEN: I think it's a very good question that we don't currently know the answer to.

DR. NAIDU: And number two is has CDC or anybody has done any serological surveillance of what is the antibody levels against E. coli 0157 in healthy populations with age groups?

DR. OLSEN: Yes. I mean, there hasn't been systematic, you know, sampling of the U.S. population, but from various healthy populations, it seems to be fairly low. And I think there's some evidence to suggest that it may vary by urban or rural location.

And I think in the next few years, we're going to see a lot more published on that. There currently isn't a lot of data, but I think that's one thing, you know, at least CDC is interested in looking at.

MR. BILLY: Okay. Thank you very much. I'd like to call to your attention a slight change in the agenda. We'll next have the presentation from Dr.

Rexroad as scheduled. And then, the following presentation on the results of the carcass survey previously scheduled for 11:30 will be moved back to right after lunch.

And the presentation on the antimicrobial blocking agents will occur about noon as scheduled. So the presentation of the carcass survey will be dropped back to right after lunch. Okay.

Now, it's my pleasure to introduce Dr. Caird Rexroad, who is the Associate Deputy Administrator of the Agricultural Research Service. And he's going to be making a presentation on research they've conducted on preharvest food safety.

MR. REXROAD: I thank you for the opportunity to be here today. And indeed, I do want to present to you our research program. In preharvesting safety, I want to describe its extent with the glossy behind it, some of the approaches that we use. And to do that, a number of the slides that I present will not refer to 0157.

However, towards the end of the presentation, I will provide a summary of some of the data that we've recently collected, particularly at the Meat Animal Research Center that relates to 0157. Probably, as you can see, it does say associate deputy administrator. That means I won't be able to answer many of your

scientific questions. But I'll do my best and see that we get that information for you. Okay.

Our total program in terms of funding is for food safety research. It's about $82 million; 228 scientists apply to that effort. We look at preharvest pathogen control, $27 million. Much of that has come over the last few years as a result of the Presidential Food Safety Initiative. So it's been a big increase, and we're just now beginning to see some of the benefits of that investment into this preharvest food safety research. Okay.

I'll use some of the locations that we have as a way of pointing out some of the kinds of things that we're doing, whether they relate to 0157 or not and some of the philosophy. In this particular location in Beltsville, here in Maryland or across the river in Maryland, we're looking at dairy management for pathogen reduction. So we're looking throughout the production system.

We're trying to build teams of people that know about management research and the likely interventions that we need to be doing, teaming those folks with microbiologists together to look at the production system and where the burden from pathogens can come in that production system.

The Meat Animal Research Center certainly is one of the places where we focus largely on production of

livestock from meat consumption. We do look at the meat species -- cattle, swine, and sheep -- doing a number of things related to epidemiology, ecology of the organisms, and trying to develop interventions to reduce pathogen burdens. We'll speak more about the data.

For instance, you've heard something about feeding regimes. We'll talk a little bit about that. Manure management is quite an important issue here, because it's not only what's in the animal, but what's in the environment that we think is important, particularly as it relates to the safety of the water supply.

And of course, some of the organisms that we're looking at at Clay Center are the salmonella and 0157. In College Station, Texas, we have a little bit of a different approach where we're looking, primarily, at diagnostics and interventions. And they are looking for new methods to reduce pathogenic bacteria, and particularly looking at competitive exclusion.

You've probably heard a lot about replacing the endogenous flora (phonetic) with flora that is likely to be non-pathogenic and using that as a method of keeping away the pathogens. We're now extending that from poultry to other species and look forward to learning more about the efficacy of that approach.

Well, I'll skip this one. It mostly relates to poultry this morning. The National Animal Disease Center, again, we think one of the largest parts or kinds

of investments that we need to be making over the next few years are microbial genomics.

We see that investment taking place everywhere, and we think in terms of understanding how these pathogens, bacteria trade pieces of antibiotic resistance, how they adapt to their environment, how they become pathogens is very important. And we think a fundamental understanding of the genome of pathogens is extremely important.

We also work here on manure management, and as I'll point out in a little bit, vaccine research and the development of models for 0157. As you know, most livestock species are not impacted with illness, as a result the presence of the organism. So it's sometimes difficult to have an optimal kind of a model on which to study the organism.

Just a few slides to summarize some of our research and some of this, the kinds of things we're trying to do, again, are detect for specific types in the live animal. And we'll say more about that in a minute.

Manure and meat and the environment even becomes an important issue, occurrence in the production environment; where does the pathogen burden come relative to the kinds of production practices that we have, the effects of things like feeding and transport, particularly as we near the market, develop interventions

that are changes in the practices, changes in feeding, feed additives that could reduce the pathogen burden.

We need to learn more about the organism. As indicated, 1982 maybe is a long time ago in some ways, but there's still many things that we simply don't understand about this organism and other organisms that are likely to emerge as problems.

So we need to invest, again, in understanding them, and then some little additional work on specific kinds of treatments, some of which you'll hear about here today. I'll tell you a little bit about some of our recent findings, some of which will be published in the near future. And you can get better reference to that.

We do find a relatively high prevalence of 0157. A lot of this has to do with increasing sensitivity of the assays that are used to detect the presence of the organism, not necessarily in any sense an increase in the organism. For instance, we found that in 13 or 15 herds, that at least one animal had feces that were positive for the organism.

But many of the animals have been exposed based on their serology; that is, the evidence of having reacted to the presence of the organism. We did do a study that suggested that hay-feeding could reduce the incidence of the organism in feces when presented later. However, we also found, at the same time, that transport

with water available and no food reduced the frequency and the presence of the organism.

We think that we still don't know enough about the ecology of the organism to really explain these particular findings. And that's especially true for the feeding, because there's a lot of disparity in the kinds of scientific reports that we've had in the United States about the report of feeding.

So they do these studies as we're now beginning to be able to do them with, these more sensitive diagnostics do indicate that there's some interesting things about the ecology of the organism. But I don't think we can say that we understand it on that basis.

We looked at the incidents in nine states. And we found that using the sensitive essay that there's a wide variation in the incidence of the organism, being nearly absent in the wintertime in the December to February time, having higher peak incidents in July to October. And we've done some preliminary work on hygiene related to the incidence of the organism and found that it had no effect.

So one of the things to remember is that this thing does have, at least in our studies, highly seasonal incidence. A study which is to be published soon, which we were asked to talk a little bit about, and that's just what I'll do, is to look at the presence, again, using

our new sensitive assay methodology as animals are presented for slaughter.

And we find that in this study where there were 30 lots of animals, a total of 357 carcasses to be studied, of 29 lots studied, at least 72 percent of those lots had a positive fecal sample, and 38 had positive hide samples. As we looked at the kinds of interventions that are being used in industry today, we saw a tremendous decrease in the presence of the organism down to less than 2 percent.

And I should remind you that this study was completed during that time when we find a very high prevalence of the organism; that is, the closer to 50 percent in the feedlot. So it was done during a high-incidence time of the year.

With some suggestion, and I think this needs additional study, but there's some correlation between -- it seems to make common sense, but you can't also be sure -- some correlation between what's on the hide and the feces and what may be there postintervention. If so, then that suggests, again, that working in the preharvest arena to reduce the pathogen burden would have some benefits.

Again, I think that really needs to be evaluated more closely. These data will be presented soon for publication, are already in the review process. And I think they've probably been mentioned here today.

We do other research. This particular research is at Ames, Iowa, at the National Animal Disease Center where we're developing some technology to distinguish among 0157 and others Shiga toxin-producing E. coli. I think that is very important.

As I mentioned, it's very difficult to have a model animal to repeatedly to be able to find the presence of 0157 in an animal. So we need an animal model to study how it colonizes in the animal and what methods or interventions can be used to clear the animal. And there, we're looking at pig models to do that, particularly trying to answer questions about the surface proteins that have a role in the adhesion and the capacity, then, to colonize.

And one of the other things that we just happen to be doing is testing an active agent as a method to reduce the incidence of E. coli. Again, I want to mention that we -- part of this research, not only in the Food Safety Program, but in our environmental programs is to address the incidence and the transport of manure, its nutrients, and any associated pathogens in the environment.

And certainly, that's part of what we will do that will relate to our ability to answer questions about 0157. And again, our goal is in this preharvest program is to develop management practices that will reduce

exposure to -- of the animal to these pathogens and to reduce the pathogen burden.

Okay. That's everything I have to say. If I can answer questions, I will. And I will be glad mostly, probably, to talk about the program. But I probably can answer a few questions about the data, which are not my personal research data.

MR. BILLY: Okay. Thanks very much. First, to the panel. Mark?

DR. POWELL: Thank you, Doctor. Mark Powell, FSIS. Just to clarify, thank you for your presentation on this study that will be coming out soon. The 72-percent fecal positives, the 38-percent high positives, that was a cluster prevalence, a herd prevalence or a lot?

DR. REXROAD: That was a lot prevalence.

DR. POWELL: A lot prevalence.

DR. REXROAD: We studied 30 lots, and that meant that just at least one animal in that lot could be measured with one sample taken out of that lot.

DR. POWELL: So that would not, then, be directly comparable to our animal estimates. Are you aware of whether that study will be also reporting the within-herd prevalence rates

DR. REXROAD: I don't know.

DR. POWELL: Okay. Thank you very much.

MR. BILLY: Okay. Bill?

DR. CRAY: Bill Cray, FSIS. Are you able to comment on some preharvest innovation strategy which may reduce the incidents of E. coli 0157?

DR. REXROAD: Well, a number of the interventions have been developed. The rinsers, the steam processing over the last years have, obviously, in this study were very effective methods in reducing the incidence on those carcasses. Is that what you're referring to? I'm not sure I can go a whole lot further. I can --

DR. CRAY: Yes, yes. On the farm --

DR. REXROAD: At this point --

DR. CRAY: -- feedlot.

DR. REXROAD: -- I think we're still in the position of really working to develop the kinds of interventions and management practices. We're still, as a lot of these funds are very new funds to the agency, still trying to sort out the ecology of the organisms and the epidemiology, just looking to see where they are entering into the production system. So we really haven't developed a lot yet.

MR. BILLY: I have a question related to the same study that Mark referred to. Did I hear you say that 3 percent of the carcasses after slaughter were positive?

DR. REXROAD: If I remember right, it was less than 2 percent. I don't know the exact number.

MR. BILLY: Okay.

DR. REXROAD: Of course, that's based on our research data.

MR. BILLY: Great. Thank you. Dr. Gill?

DR. GILL: Yes. That study interested me, as well. Was this work carried out in a commercial plant? Or was this under an experimental situation?

DR. REXROAD: This was carried out under commercial conditions in several plants.

DR. GILL: Just a comment on that, there's considerable amount of data in the literature relating to the effects of high condition and microbiological contamination of meat. And all of it says, basically, there's no relationship whatsoever.

That it all depends on your dressing process. So your results may be appropriate for one dressing process and totally irrelevant to another dressing process.

DR. REXROAD: Yes. I think your comment that there needs to be additional data is appropriate.

MR. BILLY: Okay. Other questions? Caroline?

MS. DeWAAL: Caroline Smith DeWaal, Center for Science in the Public Interest. First, just a comment on the last comment; and that is, it brings me back to hearing from a gentleman representing the government of New Zealand who felt that actually the conditions of the animals, as they come into slaughter, is actually very

indicative of the safety of the products coming out. When you said that 72 percent of 29 lots were positive, how large are those lots?

DR. REXROAD: I can't say exactly. I think there are at least 30 animals on those lots. But I'd have to look again. There were 300-and-some animals total in that study, 30 lots. So there were 10 or more animals in the lots.

MS. DeWAAL: And you do have data on the peranimal positive?

DR. REXROAD: We have. In some of the feedlot studies we have on per animal, the 50-percent incidence was on a per animal in the feed lot. And that was in the summertime and also the comparable data for the wintertime where it was 1 percent or less. But because of their cost contamination things, I presume, is why we have the data in lots going into the slaughterhouse studies.

MS. DeWAAL: And it was 50-percent positive on animals in the summertime coming into the lots?

DR. REXROAD: In the feedlots for the fecal samples.

MS. DeWAAL: In the feedlots. Thank you.

MR. BILLY: Rosemary?

MS. MUCKLOW: Rosemary Mucklow, National Meat Association. You mentioned seasonal differences in the

findings. Did you find any kind of regional differences? Or was it strictly only seasonal?

DR. REXROAD: We haven't really evaluated this for regional differences. And they tend to be the packing plants, if I remember correctly, tend to be in the same region. I think that's an important question.

MS. MUCKLOW: So you may not have the data to be able to evaluate regions.

MR. WOOD: Rich Wood back with Fast-Food Elements Concerned Trust. With the seasonal differences that you're looking at and the high figures that you found, apparently, during the summer months, are your intervention strategies being developed, in any way, to account for those seasonal differences? Or are you finding that the intervention strategies must be constant throughout the production?

DR. REXROAD: I can't really answer that right now. I haven't communicated recently with the scientists that are doing intervention strategies. But, certainly, we'll pay attention to our own data, I hope.

MR. BILLY: Okay. Thank you very much.

DR. REXROAD: Thank you.

MR. BILLY: Again, with the shift in the agenda, the next presenter will be Dr. Narain Naidu. He is the director for the Center For Antimicrobial Research at California State Polytechnic University in Pomona.

And his presentation will be on antimicrobial blocking agents in food safety. Dr. Naidu?

DR. NAIDU: First, I would like to thank Tom Billy and USDA and FSIS for giving me an opportunity to present our findings. My today's talk will be like reinventing the wheel. There is in nature, for example, a cow can protect itself. It doesn't need Ph.D.s. It doesn't need M.D.s. It doesn't need anybody.

A cow in a pasture can happily protect itself, and it can shed the E. coli 0157:H7 through its feces. It goes away. But once when you slaughter the animal, dehide the animal, eviscerate the animal, you make it into food. The food for us is also a food for the bacteria.

So now, I would like to walk you through a technology that is present in the first place in the cow itself which protected it. And we are depleting it during the process and how we can replete it back so the meat can protect itself. My talk for today is activated lactoferrin a new way to protect meat from harmful bacteria.

Today, it is E. coli 0157. Tomorrow, it may be enterotropic specium (phonetic.) Day after tomorrow, it could be another thing. On this planet, we share our lives and bacteria, so there can be any bacteria that can emerge as a pathogen. So the concept I would like to present you today is how nature protects a life form like

cow and how we can give back to production, back to cow after slaughter.

I will walk you through in this talk. What this activated lactoferrin technology means, how this technology works in the laboratory, in the pilot scale, and exactly on the surface of the beef tissue, and what are the research results we have so far in terms of optimization of this technology, plus efficacy data and our future directions of where we would like to take this technology.

I come from California State Polytechnic University, Pomona. And two and a-half years ago, we have started a Center for Antimicrobial Research. It was our intention that food safety is not a medical problem. It is no more a food microbiologist's problem.

We would like to integrate medical technology the way we know how we can handle pathogens in medicine; how we can transfer the technology to the beef industry, so that we can go for prophylactic measures to prevent pathogens in tissues. We established the Center 1997. We conduct both basic and applied research on various antimicrobials. And we have been focusing mostly on natural antimicrobials.

And we also explore the application of these natural antimicrobials in clinical medicine, oral health, animal sciences, for food safety, water quality, et cetera. And let me take you to lactoferrin.

Lactoferrin, as the name sounds, it is an iron-binding protein present in milk. And when we say that breast milk is the best and it protects the infant, probably, the first primary food any infant gets from its mammalian mother, is milk.

There are so many protective factors that protect an infant. And one of the important primary line of defense is lactoferrin. And lactoferrin has been discovered some 50 years ago, and for the past 30 years, medical researchers have skinned this molecule in and out.

If you go to Medline and put a key word on lactoferrin, you would explore something like some 7,000 to 8,000 publications. And lactoferrin is currently being investigated in AIDS research. It is investigated in cancer research, in as an immunomodulator in vaccine delivery mechanisms.

And some 12 years ago, when I got into this research, we started investigating how this molecule acts as an antimicrobial agent and what are the exact mechanisms of this molecule on various microorganisms that include bacteria, viruses, fungi, and parasites. And one of the things that intrigued me was when mother gives the first cholesterin, the cholesterin is full, rich with lactoferrin.

And our entire gastrointestinal tract is like a beautiful ion exchange column for a biochemist. This

thing goes and flushes everything. And this molecule -- and we wanted to study how bacteria colonizes in the intestinal tract and how bacteria flushed away from the intestinal tract.

That was of great medical interest for us to understand infantile diarrhea, which is the worst number one killer there is. About 20 million children get sick, and about 3 to 4 million children die annually.

So we wanted to understand in these immunocompromised populations how the milk and the milk components, particularly lactoferrin, would establish bacteria in the gut and how it would detach bacteria, how it would detect lines (phonetic) to certain bacteria, and how it would allow good bacteria to grow, and it would allow pathogenic bacteria to get out.

And this molecule is also a multifunctional molecule. It has plenty of -- you name it. It should probably go into Medline and make a -- what this molecule could do. And as I already told you, it is a broad spectrum antimicrobial.

Now, when I work as a scientist, I always tell my students nature is always perfect. As scientists, we are imperfect. Our technology's imperfect. That's the reason we improve upon Pentium 1, Pentium 2, Pentium 3. We go on adding our inefficiencies and trying to improve upon. And when we try to purify a natural molecule from its natural niche like, say, milk, that molecule's no

more in that same confirmation to do the function what we expect the molecule to do.

And it needs an activation process to bring back that structure of that molecule to that confirmation that it will do its biological job. So it took us almost 10 years. That small little molecule, it has been x-rayed, photographed. It has been studied so extensively. And we've had to spend more than 12 years to understand how this molecule could be brought to a confirmation that it would exactly behave the way it would act in the intestinal tract.

So lactoferrin, when it's isolated from milk, it is structurally compromised to deliver the right kind of antimicrobialness. So it needs an activation process, as you could see, that molecule binds to iron if these two lobes on your left-hand side. And the jaws of lactoferrin, as we call it -- some three years ago, there was a beautiful article on the cover page of Nature, "Jaws of Lactoferrin."

For the lactoferrin molecule to work as an antimicrobial, the jaws have to be opened. So that is where, actually, we require an activation process is. And we have found a particular component that is very similar to what you find in the mucus of the gastrointestinal tract that would go to the internal and stretch (phonetic) this one low and immobilizes the

lactoferrin molecule and opens the jaw. So it is now ready for business.

And before I go a little further, I would like to tell you that lactoferrin is not only present in milk Lactoferrin is present in tears. It is present in saliva. It is present in every mucous secretion that bathes the mucal surface. Every form of secretion has lactoferrin, including the neutrophils in a response. They spew lactoferrin. So lactoferrin is present in tissues.

It is present in the cylinder (phonetic) pool. It is present on the mucous surface. So now, we wanted to find a way to put this lactoferrin on a beef surface. We know how it works in medicine. We wanted to transfer this technology and try to see how this molecule -- we could put it on a beef surface for the beef surface and protect the beef surface from E. coli and other pathogens.

Now, I would like to take you to how bacteria become pathogens. Number one, if a bacteria does not have an ability to colonize, it is no longer a pathogen to be flushed off. Bacteria needs to have specific mechanisms to stick to a surface and be there.

Like intestinal tract, it's like California is like -- an earthquake flushing everything off. If a bacteria has to cause a disease, it has to have specific mechanisms to stick. This is an enterotoxigenic E. coli

you could see here. Those haylike projections you could see are fimbria. In ETAC, we call them as colonization factor antigens.

And in the presence of activated lactoferrin, within minutes the bacteria turns off its fimbrial expression. And it loses its ability to colonize the intestinal tract. And activated lactoferrin, what all we know in medicine, when we wanted to bring this technology to beef research, the first thing is we would like to optimize this activated lactoferrin to function in a beef safety issue, in a beef safety situation.

So we wanted to optimize this lactoferrin molecule against E. coli 0157:H7. That includes both human strains, bovine strains. There are some differences in how human strains and bovine strains and species specificity about human-to-human, bovine to bovine. That probably is a different talk altogether, and also different kinds of other enteric bacteria.

To activate the lactoferrin to take and inhibit this bacteria growth multiplication. At six logs -- concentration, we optimize the lactoferrin also with -- at 10 CFU per milliliter. And also, I want to remind you there's a plethora of radiation-resistant bacteria. At least we have tested some eight different radiation-resistant bacteria. And we could also contain them, control them with this activated lactoferrin repression.

Once we have optimized this lactoferrin in the laboratory, we took it further. Now, we wanted to use this activated lactoferrin in beef processing. As you could say down in the beef processing and that multiple-hurdle mechanism, it is so much like the gastrointestinal tract. Gastrointestinal tract is the perfect multiple-hurdle mechanism. I want to remind you, nature is always prolife, never intends to kill anything.

It always wants to put everything on equilibrium. And when you look in here in an intestinal tract, you have saliva that takes care of certain bacteria. Then, it goes into the stomach. You have acid wash there. You have hydrochloric acid much stronger than your lactic or gastric acids, enzymes. If the bacteria could pass, which E. coli 0157:H7 could, it comes into the intestinal tract.

There, again, you have a microbial blocking it, which would take care of this bacteria and flush it out. That's the reason when you find so much of bacteria in the feedlots, they are not happy campers. They wanted to just get out. And you have those musocal barriers in the intestinal tracts of healthy cows. And you don't see a cow coming and complaining of hempolytic-uremic syndrome. It's a turnoff.

So now, the activated lactoferrin we want to do in this, as this thing says out of the hurdle (phonetic), the last hurdle you see in the intestinal tract that

would dispose of all the bacteria. We wanted to see that thing pass one more additional hurdle. The meat processing research has done tremendous progress with all those interventions.

And those interventions should be in place, plus this would come as one more additional hurdle for the pathogens to jump over. And the electrostatic application we started working because it's not like medicine. You have about 20,000 cattle that's been processed in one day. It's not like a doctor to patient.

We don't have more than some two seconds to, actually, to handle a carcass. So we doubled up and we started working with an electrostatic spray system. In less than a second, we can put, cover the entire and pet carcass, like, it is coated and coated in a uniform on a mucosal surface. We can daily work activated lactoferrin in a biologically functional manner in less than a second, onto the surface, uniformly.

And we would create a protective barrier like a shield on the carcass until the carcass goes to the consumption level, because in some of the processes, you may have a postharvest contamination, not a post-processing contamination, so that thing would go through various different steps.

And finally, you would get a carcass that has not only displaced the bacteria, but the lactoferrin would stick to the surface and remain there and retain

its biological activity till it goes to the consumer. And from there, we took this to the pilot scale system. In the pilot scale system, we have built a digitally simulated spray system which exactly mimics a beef processing plant.

Since we started working with E. coli 0157:H7, I was not interested in stomaching the things and getting the bacteria in the liquid. The problem with E. coli are pathogens, as I told you earlier, is the bacteria that are loose and stick. You can easily detach them -- are not the ones that cause the problem.

The ones that would stick strongly to the surface and resist any detachment, these are the bacteria that would cause the problem. So we have to study directly the bacteria attachment on the tissues. So we have used a labeling technique to get into the DNA and label the DNA of the live E. coli 0157:H7.

We need a -- time of labeling, so we can track down E. coli 0157:H7 wherever it is going. In this digital system, the spray system, we can exactly program how many seconds of wash we can keep, how much temperature we can keep, and how fast the belt would move. And we can inoculate the E. coli 0157:H7. We can purposefully contaminate, in fact, a tissue.

And we could study and compare different sanitizing systems, all without lactoferrin at the end. And as you could see here, E. coli 0157:H7, 0157 has an

adhesive called integrin, plus it has other adhesion mechanisms, as well. It has a specific affinity, especially to collagen type 1 and collagen type 2.

As you could see here, those little sausage-like creatures there, they are E. coli 0157 so beautifully embedded in the collagens fimbrials in a tissue matrix. And our pilot studies have shown that if you have one million cells per gram of beef infested with E. coli 0157, all the treatments combined -- it means I'm talking about a sanitizing assembly where you have a water wash, then you have an organic acid wash, then you have a hot-water wash, then you will go water wash and then again an acid rinse, and so on -- it could remove only from 7 percent of E. coli 0157:H7 on the tissue.

So you still will have at the end approximately some 25 percent of the bacteria still left. And those are the bacteria that will cause all the problems. When you stomach this tissue, these bacteria don't come out. Whatever the sampling you have, it is representative of those loose 7 percent that come into your liquid phase. These bacteria are still there on the tissues.

So these are the bacteria that could be removed if you add one more step of lactoferrin, activated lactoferrin at the end. And we tested lactoferrin against a variety of gram-negative and gram-positive bacteria, including the currently most feared microorganisms in meat industry, of course E. coli 0157,

and we looked into different kinds of salmonella, both the pork, cattle and the poultry pathogens, including DT-104.

We have gone with the campylobacters. We went with shigella. Shigella is considered to be the mother of fecal E. coli 0157. And we went with clostridium and various other bacteria. And about the safety and tolerance of lactoferrin, lactoferrin has been consumed by mankind, or by mammalians, since the evolution. That's probably the first coating, actually, we got into our mouths ever.

And the anticipated level of -- we want to apply is one thousand times less than what is actually found in a single serving of milk, a glass of milk. But it is already a less amount of lactoferrin that we can activate and effectively dispose of those -- that amount on the entire beef carcass. And there are various ingredients in this formulation that would keep this molecule active.

And all those ingredients in this formulation are GRAS. And lactoferrin is now going to a GRAS petition, as well. And there is no reason to suspect any adverse impact of lactoferrin on nutritional quality. And probably when you took your cup of tea or coffee with milk in it, or you have your yogurt or your cheese, you have taken your dose of lactoferrin.

But it is not an active form, however. It doesn't affect the nutritional quality, sensor characteristics, or product safety. For example, the taste you feel in your saliva in your mouth, lactoferrin is there. And there is an abundant source of lactoferrin. Currently lactoferrin has been produced or isolated as one of the many bioactive ingredients from cheese whey.

And these large quantities of whey are available and lactoferrin is being commercially produced by many, many major dairy companies around the world. And there is enough of lactoferrin to protect the entire meat supply. And, as I told you, lactoferrin is a commercial commodity.

And the next step is right now we're looking for some regulatory approvals of our in-plant testing on beef carcass and ground beef applications. We want to expand our research to pork, poultry, and other processed meats. And we are still awaiting such regulatory approvals from FDA and USDA. And applications are beyond me.

I, basically, belong to the medical sciences, and there is a tremendous amount of data over there that we wanted to get into medical applications, which we are currently looking at. And in summary, this is a natural protective mechanism that primarily existed in a cow.

For the past 12 years when I started working with cow milk lactoferrin in human diseases, I never even had the faintest of idea that we're talking about a homologous situation, a cow milk protein getting back to cow. Nature has designed this molecule for cows. And it is one of the natural food safety solutions that Mother Nature has provided.

We just borrowed a page from Mother Nature to bring it back to the beef industry. This is a normal application of an extensively studied natural protein. And it is consumer and producer friendly. And thanks for your attention.

MR. BILLY: Okay. Thank you, Dr. Naidu. Questions from the panel? Okay. Go ahead.

MR. GOYAL: Raghugir Goyal, FSIS today. I'd just like to ask have you made any study or made an attempt to study long-term bioassays or how it could chronically affect the safety of the body?

DR. NAIDU: Of what, of lactoferrin?

MR. GOYAL: Yes.

DR. NAIDU: There are various studies. Lactoferrin is currently being used as an ingredient of infant food formulas all over Europe and in Southeast Asia. So it is being consumed by the most sensitive population on the planet, that is the infants.

MR. GOYAL: No. I'm just asking if any of the experimental studies like chronic studies, like two-year bioassays done in the annual studies --

DR. NAIDU: Yes.

MR. GOYAL: -- have you done to prove there's any chronic effect on the -- any type of live animals or -- the surrogate animals or sometimes for --

DR. NAIDU: Yes.

MR. GOYAL: -- those studies done in --

DR. NAIDU: Yes. Yes, sir. Those studies were done. There are 29 different animal studies have been done in the last 15 years. And there was a big review article where recently we published -- probably, you could get the article, and see it in the safety and tolerance section, you can read that. There was 10 pages of section there. We have listed all the trials, animal trials.

MR. BILLY: Yes, Mark or Dan, and then Kim.

DR. ENGELJOHN: Dan Engeljohn with USDA. What level of residue would you expect to be coated onto the product?

DR. NAIDU: Could you please repeat your question?

DR. ENGELJOHN: Would there be a residue left of the lactoferrin on the processed meat products?

DR. NAIDU: We wanted lactoferrin to remain on the surface of the meat, so that it would give a lasting protection.

DR. ENGELJOHN: Okay. And so in the petition, I'm assuming that you're putting together, or have put together, on this visit, would it identify the level that would need to be there?

DR. NAIDU: Yes. I think we have assays, and we are keeping those in place.

DR. ENGELJOHN: And it's specific for meat and poultry?

DR. NAIDU: Right now, we are focusing mostly on beef and beef products, yes, sir.

DR. ENGELJOHN: And does it create a sufficient amount of protection in the sense that it allows for competition by other organisms, anaemicrobic organisms, or other organisms that may survive all tied up?

DR. NAIDU: Lactoferrin is also a prebiotic. The only organism that it would allow to grow is -- Lactobacilleae and other organisms don't grow. And we have done these shelf-life studies for 45 days, seeing that how lactoferrin could put down the bacteria to multiply.

MR. BILLY: Okay. Kim?

MS. RICE: Kim Rice, the American Meat Institute. Can you explain how you went about doing your control study, the level you inoculated at and how long

you let it grow, and then how you came by measuring the effect of the interventions, and then the lactoferrin? Could you briefly explain that?

DR. NAIDU: What we do is we try to -- we take E. coli 0157; we grow them in the presence of treated thiamin. The thiamin gets incorporated with the DNA. We did those bacteria, and we tried to make a standard curve as to three different variations with variables with total platlet counts and how much DPM it would come to. And also, we do an OD (phonetic) determination and also correlate how much in the disintegrations per minute.

And then, we take a measured volume of measured DPM of bacteria, so we exactly know how many bacteria we are putting there. And when our E. coli 0157 sticks to a surface, it needs a minimum of 30 minutes interaction for that lock-and-key mechanism to establish an equilibrium.

If you put 100 cells, only 7 cells will bind of the equilibrium. If you put a million cells, only 7 percent, so for that 7-percent equilibrium, you have to keep it for 30 minutes. And after that, we subject it to different kinds of treatments. And at different steps, we take those meat pieces, and we disintegrate the entire meat piece in a tissue modernizer, put it into a meat account and measure, and then correlate it with how many bacteria are left. And we wanted to see zero with lactoferrin.

MS. RICE: And can I ask a followup question? The number of cells you were measuring, that was just the ones that were marked in the DNA? It was not live or viable? It was just --

DR. NAIDU: They are live. They are live bacteria.

MS. RICE: They were all live?

DR. NAIDU: Yes. All of them are live. This is a technology we use in cancer research to study live cancer cells.

MS. RICE: And you allowed it to bind for 30 minutes only, or was it longer than 30 minutes?

DR. NAIDU: We put even sometimes for two hours, a minimum of 30 minutes -- 30 minutes to 2 hours. After two hours, no matter how much you keep it, it's equilibrium.

MS. RICE: Okay. Thank you.

MR. BILLY: Nancy?

MS. DONLEY: Nancy Donley from STOP. I think I heard you mention at the very end of your presentation that you're looking at this for other uses. Perhaps, have you looked into this at all with -- as a medical treatment for people who are infected with E. coli 0157?

DR. NAIDU: Oh, yes. We have been working on that. That is actually my main focus for the past 20 years. We have been working on that, but for the past two years, our entire focus went onto beef. My plate is

full. I could not even go look into any other products. Our total 100 percent focus is on beef safety.

MS. DONLEY: But you looked at it as a medical treatment for humans for 20 years?

DR. NAIDU: For 12 years, yes.

MS. DONLEY: Twelve years?

DR. NAIDU: Yes, ma'am.

MS. DONLEY: Really, nothing ever came out of it?

DR. NAIDU: There are; there are treatments going on. And I think in oral hygiene, it is going on; in -- intestinal, it is going down. But the point is, activated lactoferrin is a pretty new discovery. This would allow a different kind of ballgame. If you wanted to take lactoferrin as a prophylactic or a therapeutic that we're exploring now how to get there, but after we finish the beef story.

MR. BILLY: Okay. Andrew?

DR. BENSON: Andrew Benson, University of Nebraska. A couple of questions here. Any idea what the incidence of allergy is to lactoferrin?

DR. NAIDU: To our knowledge, no. And if I had to go, whenever you take a protein, if a protein is denatured by any process, then any protein can become an antigen. And lactoferrin, as a native protein, it is nonallergenic.

DR. BENSON: The question was, in terms of those persons that are allergic to milk products, are any of those persons allergic to lactoferrin, in particular? Or do you know?

DR. NAIDU: No. So far, there are no reports. The only complaints you have is lacto-intolerance, which is a carbohydrate-associated intolerance coming from the milk products. In certain cases, it can be casing. And actually, if I had to put a little spin on lactoferrin, lactoferrin decreases the inflammatory responses and it decreases any allergic responses. Lactoferrin is now currently being tried to reduce rheumatoid as a treatment.

DR. BENSON: The second question was a little bit about the mode of antibacterial action that you have here. You suggest in one of your experiments that it's inhibiting growth and multiplications. So it's obviously not killing the organisms. Would you clarify that for me, in terms of what its mode of action was?

DR. NAIDU: Okay. In medicine in the past five or six years, the way we build up, killing a bacteria meant a bacteria was alive or dead, it still has the ability to cause immunostimulation in immunomodulation. It's still a proimplementary breather that can lead to other events, cellular events. So nature never kills anything, unless it takes --

DR. BENSON: But you referred to a specific experiment, though, where you were looking at growth inhibition.

DR. NAIDU: This particular lactoferrin, the way we activated it, it acts as a bacterial static agent. It stunts the bacteria and does not allow bacteria to multiply.

DR. BENSON: And what is the mechanism of that?

DR. NAIDU: Iron deprivation.

DR. BENSON: Iron deprivation?

DR. NAIDU: Yes, sir.

DR. BENSON: So iron deprivation also would explain the loss of pilae in the experiment with the endotoxins.

DR. NAIDU: No. That's a different mechanism. For that, lactoferrin has to bind to FNOC, and then it has to put a small little fragment inside and inhibit the plasmid. That's an altogether different mechanism.

MR. BILLY: Marty?

MR. HOLMES: Marty Holmes, North American Meat Processors. The graph you showed that showed the reduction, pathogen reduction, using the lactoferrin, did that include any other interventions? Or was that strictly lactoferrin?

DR. NAIDU: No. As I wanted to recall that slide back to you, it is plus-lactoferrin. You held all

interventions in place. And the last step is a lactoferrin formulation for 10 seconds.

MR. HOLMES: Okay. And that's what I'm talking about. Did you do any tests, lactoferrin only, to see what result it was?

DR. NAIDU: Yes.

MR. HOLMES: And what was that?

DR. NAIDU: It comes around 95.

MR. HOLMES: Ninety-five percent reduction?

DR. NAIDU: Yes.

MR. HOLMES: Okay. Thank you.

MR. BILLY: Okay. Caroline?

MS. DeWAAL: Thank you. Caroline Smith DeWaal with the Center for Science in the Public Interest. I have a couple of questions. One is how large is this study that you're reporting on in terms of how many samples have you run? I mean, what level of confidence do we have in the result here?

DR. NAIDU: We have been testing for the past one and a-half years almost on a regular basis. And three of my students are running this plant almost three times a day and hundreds and hundreds of samples. We have piles of data, and three students are going to finish the pieces on this.

So we have quite a good amount of data. What we did, Caroline, is we already know in medicine how it works. We just wanted to translate this thing to a

different ballgame. Here, we're talking about a big surface, and we have a very short period of time to move things up. So we understand. I think we need to do a lot of -- lot of work which we have done on a pilot scale. But we still have to go for the in-plant testing for approving us.

MS. DeWAAL: I note that, although it was hard to read the list of pathogens on one particular slide, you didn't have -- I was struck by the fact you didn't have salmonella typhimurium, which is a type of salmonella which is frequently associated with beef.

DR. NAIDU: There were listed -- a list of a lot of -- we could not put them. DT 104 was there, typhimurium three, four different serotypes we tested.

MS. DeWAAL: Okay. Fine. I just noticed that in the salmonella list, it didn't include typhimurium. And finally, and I think this is really a followup or, maybe, the same questions Marty just asked. Why so late?

I remember with the TSP work, the trisodium phosphate, they tried to put it way at the end of the process and found that if they put it earlier in the process, it was far more effective. Have you tested out the lactoferrin earlier in the process before the other hurdles that you have tested? Why are you putting it so late in the process?

DR. NAIDU: It was two and a-half years ago, I was approached by a few people. And my work back in

Europe and back in so many other countries -- this is my 20th country -- I have met a gentleman by the name of John R. Miller. He is the CEO of Farmland National Beef. And then we went on some discussions. And I started talking about how medical technology can take care of E. coli 0157 in a clinical situation.

From there, the whole thing has been spun. And today, here we are. And medical people usually don't want to step down to food microbiology, because all your colleagues would say that, probably, your period is going -- doldrums. So we never go downward. But this is a children problem.

All my life I have worked with infantile illnesses as a medical person. And you see a child dying, I think without even explaining, you cannot explain the symptoms. It is so pathetic. And all my life, I worked with infantile infections, and this is a disease with an immunocompromised host, especially children who are immunocompromised. And we wanted to transfer this technology. And that's how it is so late to bring this technology to the beef industry.

MS. DeWAAL: And perhaps, I didn't state my question very clearly. Have you tested it on warm carcasses, as well as carcasses right before they go into the chiller? Why are you using lactoferrin so late in the slaughter process? And have you tested it earlier

before you utilized all the other -- you know, you had washing? You had the antimicrobial --

DR. NAIDU: Oh, yes. I'm sorry. We have tested lactoferrin before going through all those processes and after those processes. As one of our friends has asked, if we try to take lactoferrin alone, it is effective. However, you need to have a mechanical way of removing those bacteria. Lactoferrin could inhibit radiation. You need to have a mechanical way of flushing the bacteria out.

And all those different steps of washing and the flowing through the carcass would help that mechanical flushing out. I'm sorry I didn't understand your earlier question.

MR. BILLY: Rosemary?

MS. MUCKLOW: Rosemary Mucklow, National Meat Association. I think what you're really telling us, Narain, is that lactoferrin is not a substitute for good cleaning practices, that we need to do all of the other things that we've always done. And this is just one extra safeguard that has a very impressive result, certainly, in the research and, hopefully, in the real world. Is that a fair statement?

DR. NAIDU: Absolutely. I would actually give a take-home message. Nature has never devised a silver bullet. There's never one thing that could take care of anything. And Rosemary is precisely correct. This is

one of those various multiple hurdles. And all those hurdles that are out there right now, they have to be there in place.

MR. BILLY: Absolutely. Marty?

MR. HOLMES: I had one further question. That is --

COURT REPORTER: I'm sorry. Your name, please?

MR. HOLMES: Marty Homes, North American Meat Processors. I know we're not that far yet, but are we talking about if this is a wash or a rinse similar to organic or lactic acid rinses, is there some labeling requirements that would be -- need to be looked at?

Are we talking, you know -- Dan, you brought up questions about residues. You know, this is all fine and good, but if we start talking about a lactic acid or putting this on the label, that just raises some concern. I just want to make that comment.

DR. NAIDU: Well, again, we have a team of regulatory guys working with us. Maybe, I think they are the right people to answer it. I'm a scientist in my little lab, so thank you for your comment.

MS. WALLS: Isabelle Walls, International Sciences Institute. Have you got any data showing after you treat the meat with the lactoferrin, what percentage do not attach to the meat? Do you have any data to show that?

DR. NAIDU: It was actually 99.9 percent.

MS. WALLS: That you had --

DR. NAIDU: Yes, because it very effectively attaches to most of the bacteria.

MS. WALLS: So after treatment, then, if you challenge it -- have you tried it at different periods of time after you challenge it?

DR. NAIDU: Oh, yes. Yes. It protects the meat from the bacteria. The bacteria would not attach, because the surface charges and the collagen matrices, which are the receptors for E. coli, have been blocked. This thing will competently go there, occupy those sides, and does not allow an in-coming bacteria after the lactoferrin treatment to get to the surface and colonize.

MS. WALLS: I'd be interested in that data.

MR. BILLY: Bill?

MR. BROWN: Bill Brown, ABC Research. Two quick questions. One, have you tried it on listeria monocytogenes? And two, is it heat-stable?

DR. NAIDU: Number one, Listeria monocytogenes is a different kind of pathogen. Lactoferrin, when it exists in different sites in our body, it is meant to take care of different kinds of bacteria. The way we have tuned this molecule and activated it is specifically against the bacteria I was talking about.

Yes, we could tune and activate this lactoferrin molecule against listeria. And we know how to do that. And your second question about the heat in

this thing. At pasteurization temperatures, lactoferrin, we have done a lot of differential econom tric studies on folding and unfolding of lactoferrin. To pasteurize these temperatures, it can unfold and recoil back, but when you go to extremely high temperatures, like any other protein, it would be nature.

MR. BILLY: Okay. Thank you very much.

DR. NAIDU: Thank you. Now, we'll break for lunch.

(Whereupon, a luncheon recess was taken.)

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A F T E R N O O N S E S S I O N

MR. BILLY: Everyone take their seats, please. I'd like to do a few housekeeping things to get started. Again, we're running about a half hour behind, so I'm going to press forward. Before I do, I'd like to remind everyone that we do have a time schedule at the end.

We may eat into some of that time, depending on our adhering to the agenda scheduled for this afternoon. But nonetheless, we welcome comments. We encourage people that wish to make a statement or comments to do so by going out to the registration desk and letting us know. And we'll make that time available, first, to those that sign up and provide us that information to the extent that time's available.

Getting back to the agenda, the next presentation was one that was delayed or carried over from this morning. And it is a group presentation. It reflects a piece of work that was carried out by coalition and industry, looking at the incidence of E. coli 0157:H7 on carcasses, as well as other places in the slaughter operation.

The presentations will be made by Dr. Dell Allen, who is Vice President of Quality and Training for Excel Corporation; Mr. Jim Hodges, who is the President of the American Meat Institute Foundation; Dr. Keith Belk, who is the Assistant Professor at Colorado State

University, the Department of Animal Sciences; and Dr. Ann Hollingsworth, who's Vice President for Food Safety for Keystone Foods. So, folks, whoever is first.

DR. ALLEN: On behalf of the Beef Industry Coalition that worked on this project, I want to thank USDA for the opportunity to present our results. I think, first of all, you all are at risk as an audience. I have been turned loose with a computer up here to do this presentation. And I've never done that in my life. So we'll learn together and enjoy it together.

A couple of points I'd like to make, first of all, as I told somebody when I went to lunch today, the first thing I've learned -- I've learned a couple of things today -- number 1, E. coli 0157, evidently, is all around us, but thank God for drinking milk. So that's the good news out of the morning that I'd say.

I think another thing that I would like to stress, we've talked a lot about 0157. We've talked a lot about testing. And as I sat through, particularly the early morning sessions, you know, it reminded me, as I told Kim who was sitting next to me -- Kim Rice -- we need Ann Murray here for a little more good news. You know the song she had, "A Little Good News Today," because it was pretty gloomy for awhile.

But I would remind everybody, and I think it needs to be publicly stated, that my company alone will perform about 18,000 tests for E. coli 0157:H7 this year.

And I think my peer companies -- I think I can speak for them -- although I don't know exactly what they do, I would dare say that they would be somewhere in that same type of vicinity anymore.

So despite what may be the perception, there is a lot of work that has been done and is being done to try to get at this organism and to try to take it and remove it from the food supply on the part of an industry that has taken this as a very, very serious challenge over the years. With that in mind, we'll go ahead and get into the formal presentation if I can remember to hit the down arrow.

First of all, last year on January 19th, USDA came out with a policy clarification on their adulteration policy on 0157:H 7. And basically, what it was doing is clarifying that it was adulterated in ground beef, but potentially also should be considered such in trim, as well as muscle meat, where they were stenciled for an intact surface penetration.

As a result of that, this group will be presenting the information to you as form. And out of that early genesis of a group came the recommendations that we made in March of last year to go ahead, and before we got into this too much further, really try to start looking at what was the incidence level of 0157 in our plant.

We realized that we needed to work hard, even harder than we were, maybe at getting information out into the public. We felt like, definitely, all segments needed to be involved. And by all segments, everybody in the segments represented in that meeting represented everybody from cattle producers through retailers in that group.

It's still our feeling, and I think a lot of people's feeling, that the end-product testing that is being done and is done is equivalent to closing the barn door after the horse is out. And the logical alternative to that is to go back upstream somewhere and try to identify it earlier in the production stream, such that it can be prevented from being, even entering food streams.

With that in mind, we looked internally at our industry and said the logical choke point may be in the carcass form, at least initially, and that if we can identify it on a carcass, we could at least take that carcass and isolate it and get it out of the food system, and that the carcass so contaminated would either be sent to condemnation or processed in a way of cooking which would kill the organism.

Basically, strongly stated in that group, presumptive positives had to either be treated as a confirmed positive, or you take them out to confirmation and find out where it is. We felt like that message

needed to go out loud and clear to everybody in the industry. We also wanted to come up with some way, if we could, and encourage USDA to come up with a system that basically encourages testing and does not discourage testing.

In many cases, the current policy actually discourages or causes people not to test. Sometimes, it's the attitude, "it's better not to know whether it's there or not." And that's really the ostrich-head-in-the-sand approach. And we need to get that ordered if we can, and we want to work with the agency to try to get that done.

Looking at that, we asked the USDA to consider the revision of their directive 10010.10, which had come out previous to that. I don't know; it's been a year, two years ago. Well, actually, it was about two years ago they came out.

And in that directive, which allows for a reduced -- for a plant to enter into a reduced testing period by USDA, one of the criteria in there is that you have to have six months of negative data up front before you qualify for that, where you test in your system for six months and you get six months, negative information. And then, you can qualify to operate under that directive.

And that six-months testing period is actually a deterrent to some people to even enter into. And so we

recommend taking a look at that and doing away with that six-month requirement. Basically, we felt like option three, which is in that directive, and allows plants that have microbial intervention systems in their system to qualify under that directive.

Basically, should be revised to formalize, if you will, a process verification testing as a part of that directive, and then basically to also to allow the eligibility for FSIS's reduced testing, if I qualify for it in my plant, for that to be passed onto my customers. Out of that, then, came a recommendation from this group that we do a pilot survey, a pilot test, if you call it.

Basically, we were coming up with a carcass-testing process. We had a written program. Basically, it stated in that program any positive, confirmed positive, that we found to be removed from the system and any presumptive positive that was only taken at that stage would be treated as a confirmed positive, identified the swabbing sites to be used as the same flank, brisket and rump sites that the USDA uses in their generic E. coli sampling.

And we recommended a minimum level of sampling of one carcass per 300 slaughtered and/or, if a plant killed less than that, then the slaughter, at minimum, to sample a minimum of one per day. And with that, I'm going to turn it over to our next speaker in line, Jim Hodges.

DR. HODGES: In the Carcass Testing Pilot Project, we started with the objective to evaluate the feasibility of a carcass testing program to routinely verify slaughter plant controls for E. coli 0157:H7. This was not a designed research project, but simply on a plant-by-plant basis to look at whether the carcass testing program that had been proposed was feasible and workable.

The survey design included 12 plants. Those 12 plants were geographically disbursed across the United States. They included both steers and heifers and cows and bull slaughters in various plants. And in each plant, there was at least one microbial intervention.

But those intervention systems would vary between the various plants. One in 300 carcasses were tested and were tested at three points during the slaughter process -- on the hide before hide removal, prior to carcass wash, and after final microbial intervention.

The carcasses, the hides and carcasses, the same ones were tested throughout the system, and matched sides and alternating matched sides were tested prior to carcass wash and after final microbial intervention. Trend data, if it was normally done by the plant or the plant could obtain that trimmings information from their customers, we did collect that during the course of their

normal business activities. But that was not a part of the routine design of the program.

The test ran for a one-month period immediately after Labor Day into October. For the carcass handling, each carcass was identified as an individual lot and held until confirmed negative for 0157. Carcasses confirmed positive for E. coli 0157:H7 were rendered or cooked, those carcasses that tested positive after the final microbial intervention.

And positive tests required a reassessment of the slaughter procedures and carcass intervention systems. The hides were sampled with a 24-square-inch area along the brisket midline in one area. They were analyzed using a modified USDA ARS hide-sampling method that was mentioned this morning.

The difference between the ARS method and what we did is because this was a commercial survey. Those hide sponge samples were transported and not immediately gone into incubated enrichment as the ARS protocol called for. And all presumptive positives were confirmed for 0157.

The hide samples were sent to a central laboratory, the Penn State E. coli Reference Center. The carcass sampling procedures was done by the individual plants or a laboratory, private laboratory of the plant's choosing. This was to simulate what would happen in commercial practice.

We did not use a centralized laboratory for the carcass sampling programs. We sponged 40 square inches on the 3-side areas, brisket, flank and rump. We analyzed using the, what I've classified as, the modified FSIS Microbiologic Laboratory Guide Book Procedures.

The difference between the current procedures that is used by FSIS and what we used is we used the MSA VCIG agar, instead of the rainbow agar. We did, however, consult with FSIS about that choice that we had started with. And with their concurrence, we elected to stay with our original plants.

And again, all presumptive positive samples were confirmed. We transferred our -- the data was brought to AMI. We coded that data and gave it to Colorado State for analysis.

DR. BELK: Thank you, Jim. This slide reflects data from the first six plants included in the study that was received by Colorado State University. Very quickly, I'd like to outline a couple of interesting notes here. Firstly, this column is the number of observations collected at each one of the plants involved in the study at each of the three processing sites within the harvesting system in those plants.

One of the unique aspects of the study was the fact that the hide samples, with the exception of the first plant out of the first six plants, resulted in positive incidents of E. coli 0157:H7. The incidents

ranged from down here at 0 percent up to a high of almost 19 percent on the surface of the hides.

For these first six plants, prior to washing, but after hide removal, only one of the six plants actually exhibited positive incidence of E. coli 0157:H7. And following application of the intervention systems within the plant, none of the six plants exhibited positive incidence of E. coli 0157:H7.

Similarly, for the second set of six plants, once again, only one plant out of the second set of six did not have any positives detected on the surface of hides. And one again, the range and the incidence was 0.00 up to a little over 18 percent incidence on surface of the hides.

Three of the plants did have positive samples obtained from the surfaces of carcasses after removal of the hide, while the other three plants all had zero frequency of E. coli 0157:H7. And once again, all six of the plants reported no positive incidence of E. coli 0157:H7 after application of the intervention systems.

If you look at the total of all plants combined, we collected approximately 2,248 samples at each of the processing sites included in the study. Hide on, the average mean incidence of E. coli 0157:H7 was 3.56 percent. After hide removal, but prior to intervention application, the incidents dropped to .44

percent. And this was a statistically different number from the hide-on incidents.

And in following intervention or application of the intervention systems, the incidence dropped to 0 percent. And that also was a statistically significant reduction in the frequency of E. coli 0157:H7. In addition to this, as Jim Hodges mentioned, samples were collected of trimmings, which were sent to laboratory for evaluation of E. coli 0157:H7.

And in this study, none of the beef trimmings samples were found to test positive for 0157:H7. To summarize these numbers very quickly, at the hide-on stage of processing, succumbing into the packing plant, only 2 of 12 plants did not run into some incidence of E. coli 0157:H7 on the exterior of the hide.

Prior to washing or prior to the application of interventions, 8 of the 12 plants did not experience a positive incidence of E. coli 0157:H7, and samples collected from the carcasses following application of the intervention systems resulted in 12 of the 12 plants not experiencing a positive incidence of 0157:H7.

Some of the conclusions that we devised from analysis of these data, firstly, the current protocols that the monitoring system for E. coli 0157:H7 are resulting in about an average of 6,373 samples collected per year at the retail ground beef level.

If a carcass testing program were capable of being applied in such a manner that we were testing one of every 300 carcasses slaughtered, both fed beef and in market bull plants, that would result in the cumulation of over 120,000 samples per year, about a 19-fold increase.

In addition to that, the surface sponging protocol that was used in this study would result in about a two-and-a-half-fold increase in the amount of surface area currently been sponged via the generic E. coli verification programs of the house regulation.

Testing for pathogens to ensure food safety cannot be successful. Pathogen contamination is an infrequent, unpredictable event. And there's no such thing as zero risk. And that was, once again, clearly shown this morning with the presentation of the model risk assessment systems.

This results in somewhat of a disparity in current FSIS policy. If you look at the definition for a critical control point in the HACCP regulations that recognizes the fact that there's no such thing as zero risk, while on the other end of the spectrum downstream in the production process, you have adulteration policy that, by definition, implies that there is such a thing as zero risk.

We would encourage anybody that would like to implement a testing program to consult the American Meat Science Association's publication from last year dealing with the scientific perspectives of sampling within production.

Logically, pathogen testing upstream would at least increase the probability of effectiveness, particularly from the verification or a process control perspective. The reasons we think this would be the case is because positive carcasses can be removed from commerce prior to fabrication and grinding if they are detected within the packing plant.

And secondly, if a positive were to be detected post`intervention, appropriate corrective actions would be allowed to occur which would allow and enhance the continuing improvement theory of the preventive food safety programs currently implemented in the packing industry. I'll stop and turn it over to Dr. Ann Hollingsworth.

DR. HOLLINGSWORTH: Thank you, Keith. In summary, we would like to recap what it is that we have, as a beef industry coalition, put together in our attempt to continue to aggressively pursue the death of E. coli 0157:H7, in other words, to eliminate it from a pathogen of concern in the food industry.

We believe that taking a process-control approach is the way to go about this. And we believe

that because it would encourage industry testing, it is to our advantage as industry to eliminate this organism from the food supply. And we're very serious about doing so. However, some of the methodologies that we are -- some of the procedures that we're currently being subjected to do discourage testing in some facilities.

The proposal that we've put together would lead to an aggressive approach to the control and eventual elimination of E. coli 0157:H7. And it also involves all segments of the industry from the slaughter facilities all the way through to the retail establishments.

Briefly to remind you what our proposal is, we would like to revise our request, the revision of FSIS Directive 10,010.1 to remove the six-month negative requirement which states that you must have six months of negative results before you are allowed to be a part of this program.

Secondly, we would like that option three be formalized, revised to formalize a process verification testing procedure. And thirdly, we would like to ask that the eligibility for reduced testing by FSIS be passed through the chain for hides that have been tested upstream. We believe that this all should be part of a carcass testing program that includes a written program.

It would be a formal program that would include a protocol that stated the sampling frequency in the sites, the methodologies that would be utilized, and it

would provide for process reassessment activities which would include that all presumptives, presumptive positives for E. coli 0157:H7 be treated as positives, unless they are confirmed to be negative at further stages down the confirmation process.

In addition, a process evaluation should and could include an investigation of the process operation, a trace back to the supplier, a review of any other data that might be, might shed some light on what's going on in the plant, the generic E. coli that is required by FSIS in the processing facilities today. And we believe it should, probably, include an ability to do increased sampling.

And finally, based on the results of the survey that you've just viewed from my colleagues, we believe the Beef Industry Coalition that FSIS should have gotten the recommendations of our coalition. And we believe that this is supported by those survey results. Thank you. And I and my colleagues would be willing to answer any questions.

MR. BILLY: Thank you very much. Are there any questions from the panel? Yes, Dan?

DR. ENGELJOHN: Dan Engeljohn with USDA. Could you identify the interventions that were noted in the 12 plants?

MR. HODGES: As I mentioned -- Jim Hodges, American Meat Institute -- as I mentioned from the

various interventions that were applied in the past, it was not selected to data from a specific plant, because that was not the purpose of the survey. The survey purpose was to look at the carcass testing to be applied and used to verify the process control in the individual plants, so you did not collect the data on a plant-by-plant basis.

MR. BILLY: Could I ask a followup on that? Is it possible to provide examples of the types of interventions, not whether they were all in each plant, but to --

DR. ENGELJOHN: Provide the types of interventions that all plants were to have at least one intervention, which is pretty much -- the case. The interventions range from pasteurization to hot-water thermal pasteurization, organic acid rinses, and all of the other types of slaughter procedures to prevent contamination up to the steam bath.

MR. BILLY: Thanks. Bill?

DR. CRAY: Bill Cray, FSIS. Do you think that a more sensitive assay for E. coli 0157 would help you assess the value of interventions?

MR. HODGES: Our attempt was to use the most sensitive setting we had available to us at that time. Clearly, on the hide samples, there was a great deal of differences of scientific opinion about how that ought to

be done. The method we chose was sensitive. The same thing occurred with carcass sampling.

When we initiated this study, that was before the new methods were used, immunomagnetic separation and the rainbow agar was announced. That was announced on September the 10th, if I recall right. And we had started to do the project immediately after Labor Day on the 6th. But we did have some concerns about that.

But it was decided at that time that we should move forward, because we had already -- it was to our advantage to incorporate the new pathogen with the beads. It was the only difference in the agar that we chose to stay with, because it was technician-specific in a variety of other supplies in place, and we would have to restart the project all over again.

DR. CRAY: That's okay. I have a followup. I didn't ask that as a criticism of your study. I was thinking more about the future. As the testing becomes more sensitive, will that be of value for you?

MR. HODGES: Absolutely.

MR. BILLY: Okay. Dr. Gill?

DR. GILL: Thank you. Colin Gill, Agriculture Canada. Can you offer any explanation for the failure of some plants to find any E. coli 0157:H7 at the height of the shedding season, while other plants were finding some 20 percent of their animals were --?

DR. ALLEN: Dell Allen. Colin, I was relying on you to do that. You're the microbiologist. In seriousness, first of all, I don't know that this was the height of the shedding season. We didn't get started until September.

And basically, based on what I've talked to the RS people, I think it happens a little earlier than that in the year. And the other one, again, I don't know where these plants were, but I would suspect there's some regional differences potentially in there, as well. I don't know. I can't answer that.

MR. BILLY: Okay. Caroline?

MS. DeWAAL: Thank you. Caroline Smith DeWaal, Center for Science in the Public Interest. Were the carcasses tested at the three different points, the same carcasses? And maybe you said that and I missed it.

MR. HODGES: Yes.

MS. DeWAAL: Okay. So you were testing the same exact carcass at each point?

MR. HODGES: We tested the hide of the animal. The animal had been slaughtered, eviscerated, and then we would take one side and test the prior carcass portion and the matching side would be tested after farm interventions.

MS. DeWAAL: Okay. My second question is, really, has to do with your conclusion on pathogen testing. And maybe, I'm misunderstanding it, but the

slide that says testing for pathogens to ensure food safety cannot be successful. Perhaps, you mean as -- I guess, my takeaway message of viewing your slides is that pathogen testing is quite important as a verification that your process is working.

I mean, you're showing us that, you know, up to 20 percent of the carcasses can be contaminated coming in the door, and yet, at three different points you're finding that the final number on the carcass postintervention is zero. So, in fact, the pathogen testing is documenting, is process-control verification. So I guess I'm just a little unclear what this one slide said when it seems like the actual takeaway message is to the contrary.

DR. BELK: Keith Belk. You're exactly right. Testing can be used effectively for verification of process control. And what the slide has written is, it says that if you're going to test to ensure safety procedures somewhere down the stream at that point, then it won't be successful. It's a scientific factor, as long as scientists have been around. Another big difference between ensuring the safety of a product using food safety or using testing versus --

MS. DeWAAL: So could it be said a different way that testing isn't a substitute for interventions or for process control? I mean, I just -- I mean, clearly, it's used here very effectively to document process

control and as a verification technique. It's not necessarily a substitute.

DR. BELK: I think there'll be some speakers dealing with that. I think there'll be some speakers later in the program who will discuss the NSA guidelines and recommendations for using the testing procedures.

And I think it was basically the feeling that experts had put together that document that pathogen testing probably wouldn't be the best selection of the methodology for ensuring process control. However, since it is apparent that we will probably be testing for pathogens, then we went on to make the following recommendations.

MS. DeWAAL: Okay. Thank you.

MR. BILLY: Mark?

DR. POWELL: Thank you for your presentation. I have just a couple of questions for clarification. You said that there was a statistically significant difference between the prevalence following the intervention that was with respect to the high prevalence or to the -- prior to treatment prevalence? Which one was that difference?

The zero prevalence following intervention was -- you reported it to be statistically significant. It was statistically significant from what, from the high prevalence, from the part of the wash?

MR. HODGES: From all three processing steps, there was a significant reduction statistically in the incidence of the organism. When you're superstrict across that road, you get a, b, c.

DR. POWELL: And so, again, the difference is some -- is due to some mixture of treatments which varies across plants?

MR. HODGES: I think it would relate to prerequisite programs through manufacturing practices, to hygiene and standard operating procedures employed by the plants, in conjunction with the use of intervention systems as part of the HACCP.

DR. POWELL: I guess I'm getting to the point of it being hard to distinguish what the definition of the treatment is. You have, you know, a pretreatment, posttreatment when that treatment is variously defined across plants. And so it's hard to interpret that numerical difference in terms of a statistically different, statistically significant difference based on a treatment when the treatment is loosely defined.

MR. HODGES: I guess I'm unclear as to where you're finding the word treatment.

DR. POWELL: You talk about --

MR. HODGES: The application of intervention systems and before application of intervention systems.

DR. POWELL: Substitute intervention systems for treatment, it's -- you've got a pre and post intervention. And the interventions are --

MR. HODGES: Essentially --

DR. POWELL: -- they are not the same across all plants.

MR. HODGES: Correct.

DR. POWELL: And so it's difficult to interpret, then, you know what the effect of that intervention is, because it varies across plants.

MR. HODGES: The intention was to look at -- define the system and the effects of that, including intervention throughout the program. And I think we've provided it with a handout that has a table in the back that's more specific relative to the statistical tests that were conducted. But all three of those sampling sites, the frequencies, when compared to the sample -- statistics and those are all statistically different than the .025 level.

DR. POWELL: Right. But the statistical -- the application of that statistic implies that you have a consistent treatment that's being applied.

MR. HODGES: Why?

DR. POWELL: You're comparing one prevalence at one point to another prevalence at another point. You would need to have a consistent set of interventions to --

MS. DeWAAL: The purpose of the study was not to compare interventions. It was to see if -- go ahead, Ann.

DR. HOLLINGSWORTH: Very simply put, what the survey shows is that you do take the systems and that each plant was effective in reducing, or in this case, eliminating E. coli 0157:H7 from the carcasses that went out. It's this whole system that we're concerned about, and not individual treatments.

DR. POWELL: If you were to -- well this is, perhaps, getting a little technical, but a more appropriate statistical treatment would be to group all the plants that had similar treatments and evaluate their effect, rather than pooling all the data across all the various treatments.

MR. HODGES: I disagree, because if that were a possibility, then you wouldn't have to have HACCP plants developed for each individual plant and another plant, even within the same company. I mean, the whole basis for this is the fact that there was a whole different set of environmental conditions and other conditions that are going to influence the safety of product in any given plant. And to try and standardize those conditions over all plants is just unrealistically impossible.

DR. HOLLINGSWORTH: Additionally, if you're combining a set of treatments into one treatment, as we did here, the ability to show a difference is to find

differences, significant differences. It says our systems are very effective.

The other way around, if we had to combine six or seven different ones and one had had a program and one had not, the one that had not would have remained much more unlikely that we would have shown any differences.

MR. BILLY: Okay. Other questions? Go ahead.

DR. PHEBUS: Randy Phebus, Kansas State University. Would you explain one more time quickly how the hide samples were taken? Was that also a sponge?

MR. HODGES: The hide samples was a sponge along the midline on basically a 2 x 12 inch area and it was designed to show if there was testing control or not.

DR. PHEBUS: Is there a particular reason you chose the midline as the sampling site for the hide?

DR. ALLEN: Because in the plant, Randy, if you go try to do it somewhere else, you're in dire danger. That was the simplest, easiest, and most effective way to get out a hide sample in the commercial setting of multiple plants, multiple locations.

MR. BILLY: Yes, go ahead.

MS. SOSA: Meryl Sosa for Food Animal Concerns Trust. I have a -- I'm sorry -- Meryl Sosa for Food Animal Concerns Trust. I have a question about the coalition. At the beginning in the background, you mentioned that the logical preventive point is carcass/live animals.

And you mentioned that producers are part of your coalition and what I wanted to find out was whether you've considered or are funding any kind of research to determine any kind of intervention or mitigation strategies that you would think might be helpful as far as you could have cleaner animals coming into the slaughter plant?

DR. BELK: There was a presentation earlier in the discussion about some of the harvest, as it were, clearly sustaining quarterly stated in our slides that we raise the bar -- and we're looking at all kinds of matters that we can use the incidence of live animals coming into our facility. You will know about it.

MS. SOSA: Is that coming from the coalition? Or is that just generally --

DR. BELK: It would be members of the coalition, and it's also the industry atlarge, too.

MS. SOSA: Okay.

SPEAKER: I have a question.

MR. BILLY: Yes, go ahead.

SPEAKER: In terms of the specific testing methodology, I understand why you didn't use the rainbow agar. You started before that was all announced. In terms of the immunomagnetic separation, though, did you use the FSIS method, but just without the rainbow agar?

DR. BELK: Yes.

SPEAKER: So it's exactly the same?

DR. BELK: We followed the main -- I should qualify that. We followed the manufacturer's -- the instructions to the plants, followed the manufacturer's recommendations on how the test should be used. I have looked at that protocol versus FSIS's, and it appears to be similar.

MR. BILLY: Caroline?

MS. DeWAAL: Thank you. Caroline Smith DeWaal, Center for Science in the Public Interest. Can you just talk a little bit about the range of laboratories that were used by the plants? Were some in-house laboratories versus external?

DR. BELK: I have no specific knowledge if the laboratory test was used. I do know the laboratory test for the labs used by the individual plants. It's my understanding some of those tests were conducted in-house by laboratories company. A couple of others were done by private laboratories that were chosen by the individual plants.

MR. BILLY: Mark?

DR. POWELL: Thanks. Just one question for clarification. I wanted to be sure that I understood correctly the enrichment step that was used, that was used in the same manner both for the high prevalence, as well as the carcass prevalence. Was that consistent across the hide in the carcass?

DR. BELK: The enrichment of the carcasses was not clear until it got to the laboratory of the plant's choosing. The enrichment on the hides during incubation did not occur until it got to the Penn State Laboratory. I will provide FSIS the directions in each one of the plants certainly, specific analysis that I also have at Penn State and medical procedures that they use on evaluating -- general practices.

DR. POWELL: Thanks. And just as a final follow-up, I wanted to thank you for supplying the state of what will be an important, I think again, a reality check on our risk assessment model.

Like, when we just at first glance taking into account the sensitivity of the method that was used, it seems that the carcass prevalence is pretty consistent with -- that we're predicting is pretty consistent with what you have found in terms of the reported prevalence and the hide prevalence will be very valuable input. We hadn't had access to this sort of data up until this point, so thank you.

MR. BILLY: Okay. All right. Thank you very much. I'd like to move on now to the next presentation, which will be by Dr. Gary Weber. Gary?

DR. WEBER: Thanks, Tom. I'm fully willing to talk over the noise if you want me to.

MR. BILLY: You want to talk over it?

DR. WEBER: Yes. Why not?

MR. BILLY: Let's go ahead.

DR. WEBER: Thank you very much. Thank you. As Tom said, I'm Gary Weber. I'm the executive director of Regulatory Affairs for the National Cattlemen's Beef Association. Principally, I work in the regulatory area around animal health issues in the Washington office in meat inspection, food safety-related issues.

Dr. Reagan was going to be here today. And a personal matter came up, and he was unable to attend. But I thought it would be appropriate to share with you a little about where we've been on this issue of food safety. In 1989, we had policy on the books regarding 0157:H7 before the 1993 incident.

In '91, we began tests on organic acid rinses and started investing research dollars at that particular point in time. In 1994, an interesting thing happened that really galvanized our emphasis on the direction that we've taken over the last several years. And that was the Pathogen Reduction Act of 1994. During that time period -- I think he's going to shut it off. All right. Thank you.

(Applause.)

DR. WEBER: And now we know it's not a way out. Anyway, as I said, in 1994 some things started to happen here in Washington, D.C., that really galvanized our focus on this issue. And that event was the Pathogen

Reduction Act which would have quarantined farms and ranches for E. coli 0157:H7.

But more importantly, there were a number of individuals from the research community and others who were on the Hill talking about the need for this legislation and that it was, indeed, warranted because soon there would be vaccines and probiotics (phonetic) available on the market that take care of this.

Now, if that wasn't bad enough, because here we are six years later and where are these developments? And as Dr. Rexroad mentioned, the investments in the preharvest side have been immensely problematic and it's very, very complicated, long-term work.

But to make matters worse, the companies who were beginning long-term investments in things that would become steam-vacuum and, I would assume, steam-cabinet pasteurization or organic acid-rinsing systems, a number of the people responsible for the R&D in this area said to us if this is true, if this is true, we don't want to be investing in something that companies and the industry aren't going to need.

And it became very clear to us, we better get busy making sure that act didn't occur, that this misrepresentation of research data that there was on-farm solutions ready that could jeopardize everything we have today that you've seen.

And Mike Taylor, and us, and many other people in the industry came together to expedite the approval of interventions that are now contributing real savings in terms of food safety. One of the other things that we were looking at in here was this choke point, if you want to call it that, that we've got about a million people that raise cattle and calves. And of course we've got 250 million consumers.

And so trying to change behaviors at both ends of that spectrum in order to benefit food safety, yes, we need to do that. But in the near term with limited dollars, we wanted to hit this bug, this issue where we could really make a difference. And so we began an investment program led by the Blue Ribbon Task Force Committee in a prioritized way to target where we could make a difference.

In the back of the room, there's a report that documents the time line and the commitments. And I want to take this opportunity to have Dr. Belk come up and talk a little bit about some of those interventions and some of the things that have come. And then, I want to tell you a little bit about the research we have planned for the year and the years ahead that fit into this whole continuum.

DR. BELK: Thank you, Dr. Weber. I'm probably the only guy you get to listen to twice today. And I think it's just because I was already here. Several of

these studies, researchers at Colorado State University have been involved with, basically, since their inception back in the very early 1990s.

And so it's kind of a privilege from our perspective to have the opportunity to very generally go through with you some of the research that we've conducted on behalf of cattlemen to address this growing food safety issue, E. coli 0157:H7.

The first studies -- and I'm going to be very brief and general here, as I mentioned -- the first studies that were instituted back in the early part of the 1990s will be referred to as microbial mapping studies. The first microbial mapping study we call microbial mapping 1. It was initiated in 1994 and completed in 1996.

Now, basically, the objective of the study was to identify critical entry points for pathogens in the slaughtering and harvesting process and use that information to help extend and improve the process for preventative maintenance of food safety. We felt at the time that these data would be crucial in development of HACCP regulations and HACCP plans within plants.

And we thought that this would help determine how much contamination is introduced from outside of the actual production chain. And so it was a series of evaluations to determine exactly where in the process flow we would have an opportunity to intervene or to

introduce process management techniques to improve the safety of beef.

Microbial mapping two came along about a year and a-half later. This was a similar sort of study. And it was designed to provide an assessment of where pathogens could enter the beef chain, following the slaughtering and chilling processes all the way through to retail.

In the case of this study and the previous study combined, the opportunity then became available to use -- as one example, to determine the prevalence of pathogens in the different seasons, at different points in the processing system where they might be introduced or reoccur. We're currently in the process of conducting the third in the series of studies called microbial mapping three.

This particular study actually has been designed to develop additional intervention systems that could be used during the fabrication and grinding processes to improve the safety of beef from the chilling cooler on. And we think they will identify additional methods to allow us to reduce the risk of pathogens being transmitted to consumers and will ultimately address all of our food safety objectives.

So as kind of an overview of all three of these studies, microbial mapping one was designed to map critical entry points for pathogens at the harvesting

step and within the cooler. Microbial mapping 2, then, addressed whether you could control or exert process control during fabrication and then distribution to food service and retail. And then, microbial mapping 3 is an ongoing project we're currently working on.

And when we finish with that, we would hope to have some intervention systems that could then additionally be applied during the fabrication and grinding processes. Relative to the development of intervention systems themselves, the first of these studies, as Dr. Weber mentioned, was actually started in 1991 before the Pacific Northwest outbreak. It was a four-year study.

It was determined -- it was designed to determine whether the use of natural food acids could be used to help decontaminate or remove pathogens from the carcasses. And the beef industry worked very closely as it conducted the study with USDA to develop and test specific rinses that would be effective towards this objective.

Second, a series of studies that was conducted addressed washing versus trimming issues. Zero tolerance had been implemented at that point in time. It was extremely labor-intensive and costly to trim away visible contamination on carcasses. It led to USDA approval. These studies led to USDA approval and implementation of the steam-vacuuming technologies.

And it also addressed the use of hot-water in organic rinse interventions which are now recommended in the FSIS regulations. Hot-water and steam-vacuuming studies were conducted next. Actually, five different universities were involved in the research that led to the development of some of these systems.

Steam vacuuming has one of the offshoots of that -- of those series of studies is now used in virtually every major packing plant in the country and is probably applied to more than 90 percent of the fed cattle carcasses. Hot-water pasteurization came along next. This kind of evolved at about the same time that steam-pasteurization technologies evolved.

In this case, hot-water pasteurization was developed to wash carcasses with water temperatures that actually made contact with the carcass in excess of 160 degrees Fahrenheit. And that helped to serve as a kill step in the elimination of pathogens on the surface of carcasses.

This pasteurization system is often followed by a rinsing system called final wash, and then subsequent to that, usually some application of organic acid. And it was imperative that these sorts of technologies be researched, both for their effectiveness from the food safety perspective, but also from the impact that they would have on quality and color of the product that was being generated.

Preevisceration washing came along during the latter part of the '90s. And studies that we completed in 1998 and 1999 preevisceration washing of carcasses is an additional hurdle that pathogens have to jump over to make it to the consumer. And so it made sense that you would implement another hurdle in the process.

You could reduce the risk of a pathogen reaching a consumer. The system helps eliminate pathogens and particles that may remain on the carcass immediately after hide removal and application of steam vacuuming and also helps prevent attachment of bacteria and formation of biofilm as the carcasses are processed.

And that goes to what was discussed this morning relative to some other studies that are currently being conducted. From this research, kind of a new terminology developed or evolved that we commonly call today multiple hurdles. Multiple hurdles is essentially the linkage sequentially of a whole bunch of interventions within the processing system on harvesting floor.

And starting in 1999, we decided that we needed to go out and, essentially, conduct a study to see how these interventions worked in aggregate when they are implemented sequentially within the plant to determine the value of that industry-funded process relative to the safety of beef.

Very briefly, multiple-hurdle strategies create barriers for pathogens and are highly effective in reducing the risks that a pathogen would be transmitted to consumers. In this particular study it's conducted in eight commercial plants that were geographically disbursed and included both fed beef and market cow and bull plants.

They had standardized their multiple hurdle system across the entirety of their harvesting operation and included steam-vacuuming. It included application of an evisceration wash unit, along with application of organic acid at the preevisceration level. And then, following evisceration, later down the stream, there was hot-water pasteurization.

And, in this case, the hot water actually made contact with the carcass surface at about 180 degrees, followed by organic rinsing. The results of this study across -- in total, the eight plants that were studied, it resulted in a 99.75 percent reduction in total plate counts or total aerobic plate counts, 99.79 percent reduction in total coliform counts, and a 99.55 percent reduction in generic E. coli counts.

NCBA has also funded several studies since, to begin moving the process downstream towards the consumer at the various points where we can now begin to identify process control opportunities. One example of such studies was a study conducted a couple of years ago

relating to the use of raw materials in ground beef manufacturing systems.

In this study, basic general conclusions that resulted from the experiment, raw material trimmings that were greater than 30-percent fat content tended to have a higher microbial count than other types of raw material trimmings. Fed beef trimmings had higher plate counts than trimmings from market cows and bulls, dairy cows or imported frozen product that was boxed and in a different state of refrigeration.

The same study of purged bacterial counts tended to be higher than counts that were obtained using poor sampling techniques and has led to some further investigations that I think Dr. Weber will talk about that are ongoing at the moment. The detectable bacteria counts increased as product moved through the grinding process, which wasn't a complete surprise to anybody.

The last study that we've just completed this past fall -- and this is the only study that, out of the series, that I've been through with you that is not currently in peer-review press or in the acceptance process -- has to do with raw materials that are used for production of ground beef to be marketed at retail.

In this study, samples were collected from both packing plants, processing plants, or further processing plants, and retail stores that were pretty geographically disbursed. A total of 1,158 samples in aggregate were

collected. Not one of those samples was found to have positive E. coli 0157:H7. And this would have been using the electromagnetic beads.

One combo bin sample did contain 0157 nonspecific H group that was considered to be nonpathogenic and one ground beef patty sample that contained an 0105:H8, which is a rare H-type group that has not been linked with human beings as being a pathogen. So I would turn it back over to Dr. Weber. Thank you.

DR. WEBER: Thanks, Steve. I wish that Dr. Reagan could be here, because really I'm sharing with you the results of their current process of developing a set of strategies for the next phase of investments in this area. And these are the results of counsel from a number of experts, scientists, government, industry leaders sitting down and deciding where's the best way to focus our limited resources.

Basically, there's a couple of key areas here. One, there's a lot of investment going into engineering and evaluating the dehairing process which is a chemical dehairing that looks at basically cleaning the outside of cattle to minimize that contamination, since that appears that highest probability for carcass contamination is hide-related, as opposed to intestinal contents, which is a result of rupturing the gut during evisceration.

We are developing and continually monitoring the preharvest side, watching for opportunities there. As Dr. Rexroad mentioned, there's about $27 million in ARS alone this year. We're having a real serious problem trying to monitor what goes on within the land grant institutions which contains both federal dollars, state dollars, and private industry dollars to find out what's going on there.

And until we really have a sense of that and we're really pursuing that, it's hard to really find out where should you invest as a partner in trying to make some of these things happen. But we are serious about monitoring that and where there are opportunities, we will invest there.

But there's a lot of activity there that's really coalescing and hopefully will result in some things that we can go into a validation mode on. We'll continue to look at the post-cooler interventions to see what we can do there.

The sampling systems for combos, I was mentioning to Mark Mina at lunch that we appreciated as a result of one of our meetings with Tom Billy and others that we find one of these combos that's positive, we want to be able to remove it from the plant and just take that entire combo apart.

And I think, Mark, this relates to some of your data on what this really looks like in terms of a

positive in a grinding lot in the context that it may just be in one little part and maybe one piece. And how does that really affect the risk or how we view how that may contaminate the system? So we've got at least one of those, and it's completely being disassembled into integral pieces and tested.

With any work on the nonintact raw materials, the blade-tenderized issues and others to look for E. coli and salmonella and ways of looking at kill steps there, after the listeria issues raise the potential for aerosols and air purification needs, we're looking at investments there, as well as equipment cleaning and sanitation issues.

We continue to look at ways of helping to ensure consumers can make an informed choice on irradiated products. And there is some question about dose levels, and we're continually monitoring that to make sure that there is, sort of, this low-dose relationship to help people out.

We've had a long-term relationship with the American Digestive Health Foundation, looking at the human side of it and seeing if there's ways we can partner and encourage or help us access research dollars on that side, too. This is an ongoing process. And as in the past, I hope that we can focus in as these things develop.

We'll be handling briefings with FSIS and with consumer groups and others as we have developments here. And again, I wish that Dr. Reagan was here. He could go into more detail on this. And as Dr. Rexroad said, I'm just the deliverer of that, not necessarily the architect.

So if there's technical questions, Doctor, well we'll just forgo that. I'll let you handle them. So with that, thank you much and look forward to some bold initiatives and new solutions as we further invest in this area to improve food safety and improve consumer confidence in our products and the regulatory agencies that ensure that. So thanks a lot.

MR. BILLY: Okay. Thank you very much. Are there questions, first, from the panel? No. Okay. Okay, Dr. Gill?

DR. GILL: Colin Gill, Agriculture Canada. Two questions. The multihurdle data for carcasses, does that relate to carcasses that are being inoculated? Or was this describing their natural flora, because there is often the great difference between the effects you get with the two situations and the other thing on the work on dehairing carcasses, the published data show there's no microbiological effect of dehairing carcasses before dressing. Have you any further data that would contradict that?

DR. BELK: I can answer the first question. Relative to the first question, multihurdle studies have been conducted in eight plants and others have been conducted in plants so there wouldn't have been any inoculation. That was basically to monitor indicator organisms as parts moved into the process. And that publication is accepted and should be out there in the -- on a table.

DR. GILL: Dehairing?

DR. BELK: Dehairing, I'm not the expert for dehairing.

MR. BILLY: Anyone else that has any information on that? Okay. We'll have to let that pass. Other questions? Yes, Nancy?

MS. DONLEY: Nancy Donley from STOP, Safe Tables Our Priority. And I think Gary Weber left the room, because I really was going to direct -- well, maybe, someone else here knows. I wanted to ask about -- I think multiple hurdles is a really important idea. And it's a valuable one.

But I still didn't hear, he made some comments about, you know, monitoring what was going on in ARS research in the animals. But I didn't hear anything that was specifically mentioned coming out of the National Cattlemen's Beef Association on on-farm or preharvest studies. And also, if maybe somebody knows, too, when

that White Paper is -- you mentioned a White Paper, but when it might be released.

MS. KOSTY: I can try and answer that for you. This is Lynn Kosty with the National Cattlemen's Beef Association. The White Paper that we are working on probably won't be released until sometime this fall. That is an ongoing process.

As far as on-farm research that we are doing, currently we don't have it in our agenda to do on-farm research. We are lobbying very hard for those dollars for agencies that are more capable to conduct those long-term studies, like ARS, to conduct those.

But as far as what we have heard from researchers and scientific experts, their feeling is that our dollars are better spent elsewhere. And that is where we can make the most impact on public health in the near future.

MS. DONLEY: Elsewhere meaning postharvest?

MS. KOSTY: Exactly.

MS. DONLEY: And so is the White Paper also on postharvest interventions or --

MS. KOSTY: I believe that it will target both. I think that the greater problem that we have right now is the fact that, as Dr. Rexroad said this morning, we are seeing numerous studies that come out about on-farm practices, such as the hay-feeding study. But then, a

few weeks later, we see something else that comes out that points to the opposite conclusion.

And I'm going to repeat his words in saying, basically, that we just don't know enough about the ecology of the organism to get very far right now on on-farm practices.

MS. DONLEY: Well, do you mind if I follow up one more time? I guess if it's not the cattlemen who are doing -- looking for it or figuring out how this is happening, who else is there to look? Who else is there to do the research?

MS. KOSTY: Well, I think we're counting on the government to help us out in that area. I think, quite honestly, if you look at the area of animal disease, which is very similar, and the eradication of tuberculosis which has also taken years and years -- it's taken us 50 years to eradicate that disease.

And I think that you know you can't look at that and expect us to solve this problem overnight. It's not that we don't want to help. It's not that we're unwilling to have government researchers on our farms. I think if you speak to most of our members, they are more than happy to help. And they are very interested, but we just simply don't have the money to conduct a 50-year survey.

MS. DONLEY: How much money are you spending now on postharvest research.

MS. KOSTY: That, I couldn't tell you. You'd have to speak with Bo Reagan.

MS. DONLEY: Okay. Okay.

MS. KOSTY: But I can tell you it's significantly less than $27 million.

MS. DONLEY: But it's more than zero, which is what's being spent on preharvest?

MS. KOSTY: That's correct.

MS. DONLEY: Okay.

MS. GLAVIN: One of the things that I heard Gary talk about was that detectable bacterial counts increased as the material moved through the process. And I wondered if, you know, when we look at the information on interventions, they appear to be quite good, you know, approaching maybe 100 percent good.

But obviously we continue to find 0157. Is there any thought that after intervention, the incidence is so low that we're not finding it, and then as the product moves through commerce, it grows out and then it's findable? Is that possible? Any comment on that?

DR. BELK: I think it's a matter of what percentage of potentially contaminated surface area that is being tested. All of the samples are being enriched, so there it ought to be down.

MS. GLAVIN: Okay.

DR. PHEBUS: Randy Phebus, Kansas State University. In relation to growing out there in

commerce, that's not going to happen with E. coli 0157:H7 based on its growth temperature characteristics. I think one thing that we're forgetting relative to finding this organism and, particularly in ground beef, is that a lot of technologies have been directed at the carcass level.

And there's still a significant amount of product that goes into ground beef that there's no intervention at this point to take care of that.

DR. ALLEN: I'd speculate a little bit --

MR. BILLY: You need to say your name.

DR. ALLEN: Dell Allen. And it's a good speculation, probably, that's appropriate here. I think it's an opposite, Maggie. From what I've talked to the RS researchers at Clay Center, 0157:H7, fortunately for us, is not a real competitive organism and easily gets overshadowed.

And I think, in fact, it's fairly fragile, in particular a cold environment, at least it's my impression in talking to the researchers there. So I think when we have a problem, it's probably one where there's been a fairly heavy contamination or cross-contamination level is when it occurs, not in the typical, probably, is not going to make it through, nor is it going to grow afterwards.

And it also goes back to when James Jay, who's -- I was in a meeting with him one time -- reminded me that where our microbial counts were very, very low in

the ground beef, he says you're running in danger, then, of any organism like 0157 if there is that contaminant level where you get it. Then it doesn't have the competitive exclusion thing to help you out.

And I think our counts industry wide are considerably lower now than they were 10 years ago. So we may be running on that fine line. And don't ask me how you tell people not to be so clean. But I think we are actually approaching some of those levels in some cases.

MR. BILLY: Good. Dean?

MR. DANIELSON: Thank you, Tom. Dean Danielson. I need to understand your question just a little bit more. Could you repeat that?

MS. GLAVIN: Well, my question really was based -- it was more of an observation that the data on interventions looks so good, but we're still finding 0157. And I was looking for some speculation on, you know, is that from -- you know, is this speculation that it's from plants that aren't using interventions? You know, what is the speculation?

DR. ALLEN: Okay. I guess I would offer another thought on that. If you look at the carcass data shown in the carcass study, .44 percent FSIS data, which is done at various points throughout including retail, you're looking at rates of .2, .3, .4 percent. The data that we have on trimmings over the years puts us in that

.1 to .3 percent range, depending upon the year and depending upon when various interventions come in place.

To me, exactly what you're saying, but then the arrested growth, once we get temperature control on these carcasses and arrest that growth, we show very -- you know, quite similar levels at the carcass stage, at the trimmings stage, and at the ground beef stage in the FSIS testing.

So there is huge reductions occurring on the slaughter floor, you know, very significant reductions. And we're not, I don't believe, we're seeing whole-scale temperature abuse. We're not seeing whole-scale growth of this pathogen or this organism in the meat supply once we get past the carcass stage. That would be my observation to that question.

MS. GLAVIN: Okay.

MR. BILLY: Yes, Dr. Gill?

DR. GILL: Colin Gill, Agriculture Canada. Just a comment on the temperature control. I've just been involved in a rather large-scale study of the temperature during distribution of beef in the Canadian system. And it turns out that the degree of temperature control is extremely good, particularly for ground beef, for manufacturing beef, and ground meat products.

Throughout the system, it's generally cooled very rapidly and maintained at temperatures below 6

degrees centigrade right to the retail level. Things do tend to go wrong in the retail case, though.

We also have data that suggests that there is no change in E. coli numbers throughout this distribution system until you get to the retail case where you can get temperatures up to 15 degrees centigrade for prolonged periods which does allow bacterial growth.

MR. BROWN: Bill Brown. Maggie, a couple of changes have been made, one, increasing sample size by 13-fold from 25 graphs to 325. That has a difference. And then, the new method is four times as sensitive.

MS. GLAVIN: Oh, no, I understand. We're not comparing. We're comparing two different things. Thank you.

MR. BILLY: Okay. I think we'll move on. Thank you very much. The next presentation is by Andrew Benson, who is the Assistant Professor of Food Microbiology at the University of Nebraska at Lincoln. He's going to be speaking on research on 0157:H7 in feed yards. Dr. Benson?

DR. BENSON: Thank you, Mr. Billy. I'm not real sure where the title "0157 in Feed Yards" came from, because that's not exactly what I want to tell you about. But I do have a message today that I do want to communicate that I think is important. So hopefully you'll bear with me on that.

And if everybody will follow this, I'm a geneticist trained as a geneticist, and so I look at this problem from a slightly different standpoint than many of you do in this room. And what I want to tell you about today is a comparative genomic analysis that we've done sort of to get at the question of the E. coli genetics and the E. coli ecology of E. coli 0157:H7.

And hopefully, you'll understand as you walk away from this that this is an approach that can be used, in general, for other E. coli and other pathogens, as well. Before we get started, though, we all have to have a little bit of a course here in bacterial genetics and population genetics, so that we all speak the same terms.

I always have to do this, and everybody laughs at me. But it helps that we're all on the same page. And first of all, the thing I need to get across is that most bacterial populations -- I guess this isn't a pointer. Here it is. Most bacterial populations are clonal. And what that means is that they are comprised of a founding cell and all of the daughters of that particular cell.

Now, that's not to say that all of the daughters will be genetically identical, because over time subclones happen. Any number of types of alterations can occur in that chromosome. And that will mark that subclone from its peers here. And over time, as a clone spreads geographically, it accumulates

alterations in the chromosome and also undergoes certain types of selections in different niches, and therefore, becomes adapted to particular niches.

Therefore, over time if one looks in different niches, you'll find that although the bacteria are very, very similar, there are distinct differences that you can find amongst them to distinguish them one from another. Okay. So that's enough of the little lesson here in population genetics.

The reason I told you that is because there was a bit of puzzling data with regard to 0157:H7. Back in 1993, Tom Widham (phonetic) at the Penn State University had demonstrated by looking at E. coli 0157 isolates from cattle and from humans all across the planet that, in fact, 0157:H7 is a clone, that is, it arose from a single founding cell that subsequently spread geographically.

However, wholesale genomelectrophoresis, which is a very standard typing scheme used by epidemiologists demonstrated that, in fact, there's significant genome diversity amongst the 0157:H7 strain.

So on the one hand, you have one method saying that they are all very, very similar. On the other hand, you have a different method saying that, in fact, there's a tremendous amount of genetic diversity. And the problem was that looking at the diversity with the pulse-field data (phonetic) it was very difficult to

understand the relationships in the strains one to another.

In fact, the only instance that we understood the genetic relationships of the strains one to another were those few instances in which we had isolates from human cases of disease that had been linked epidemiologically to contaminated food sources. Short of that, we couldn't say much about the relationship of the strains one to another.

Now, based on the fact that we have this genetic diversity, this suggests that, in fact, there are lots of subclones out there and also suggests that, perhaps, some of those subclones could have unique virulence or physiological properties.

This is what we needed at the time, is a very high resolution method to identify subclones and to map the role of genome alterations. That is, so to speak, let Mother Nature do the genetic experiments for us. We'll go find the alterations and walk backwards, finding or determining what genes those alterations are in and subsequently trying to understand the impact on the physiology or virulence of the sub.

Well just very, very briefly, I don't want to go into detail about our methodology, other than to say it's called OBGS. It stands for Optimum-Based Genome Scanning. And it relies on a phenomenon of skewed iligmers (phonetic.) These are very short words. You

can think of them from the short words that occur in a chromosome over and over and over again.

And not only are they overrepresented, they also occur much more frequently on one strand than the other. And we simply mix and match these different sequences and use them as mileposts and use the former H-chain reaction to look at the distances between it. And so here's just a little short section of the chromosome I've shown here. There's watts and strands (phonetic) on top and a thick strand on the bottom.

And here, these little lines are just the occurrence of these little specific segments we use. And these little pieces here are the little pieces between them that we can look at. So using this method, we can look at thousands and thousands of pieces of the chromosome from each different isolate and get a very, very high-resolution fingerprint.

In fact, we can use whatever resolution we want here to get a very high-resolution fingerprint of the different isolates that we're looking at. So that's the methodology. We, then, take those PCR products. We run these in an automated DNA sequencer, so this is a very automated process.

As you can see here, each of these tracts, there's a different isolate, and here's an alteration I've shown up here in only in some of the isolates, not in the others. And here's some down here that occur only

on a couple of red guys occurring here. So we can identify these different alterations.

Now, a little bit more on methodology. How do we make that useful? Well, what we do with that is to convert that image I just showed you over into a binary file of 1s and 0s. One presents the presence of a band or a segment of a chromosome. Zero represents absence.

And once you convert something over into a binary file, you can essentially do anything you want with it, computationally, which is really nice. So we do cluster analysis on those. And the way the cluster analyses are rendered is that they are rendered by dendrograms (phonetic.) And, essentially, the closer each sample is on the leaves on the dendrograms, the more highly related they are genetically. Okay. So that's the approach.

Now, here's what we do. We started off with a set of isolates from Wisconsin, from a three-county region in Wisconsin and were collected by Charlie Casper and John Luchanski (phonetic) for a period of about three or four years. Part of the isolates came from a longitudinal cattle study of four different cattle herds that occurred in that region.

The other isolates came from humans in that same three-county region during that same time period. We looked at 1,251 different markers from each of the isolates. That's about 20-percent genome for a single

nucleotide resolution. So if there was a single-base difference within these regions, we'd pick them out.

In contrast to what we expected, what we found was that the bulk of the animal isolates clustered together. And the bulk of the human isolates clustered together with one single animal isolate up there. Now, that kind of puzzled us, because we weren't expecting that, because conventional wisdom said, at that point, that ground beef was the primary source of transmission and was the most effective source of transmission to humans.

So we scratched our heads a bit and asked ourselves what this might mean. There was two explanations for the result that we had here. One explanation was that what we were looking at was regional bias. Those cattle herds were confined to that three or four-county region during that three or four-year period that Charlie and John were sampling then.

The humans, however, were not confined to those regions, nor were their food sources. So what we could be looking at here is a regional phenomenon. And this is a region subclone of 0157, and the humans obtained their clones -- I hate to put it that way -- but the humans were infected with clones from outside this region.

The other explanation is that, rather than regional formal phenomenon, we're looking at an animal-specific clone here. And in fact this is a clone

that you very seldom see in humans. It's a subclone that colonizes animals that is either less arivulent, arivulent, or is ineffectively transmitted to humans.

So the way to test that hypothesis to discriminate between those two is to collect samples from all over the place. And we went out and did that. We collected samples across the nation from 16 different states' worth of cattle and, it seems to me, almost 20 different states' worth of humans. I forget the exact numbers now.

The bottom line is when you do this experiment, you get the same results. Again, right here is this little cluster of isolates from Wisconsin, and you see that the bulk of the animal isolates that we had, or that we looked at, clustered with those animal isolates from Wisconsin. In fact, there are no human isolates in this clustering, till you get down to here.

Of course, there were some animal isolates did cluster up with the humans, and we fully expected that. In fact, I would have been shocked if we didn't see that. And I really would have questioned whether a method was working right or not. But the way we interpret this is that, in fact, there are two very different subclones, or at least two very different subclones, of this organism out there.

One of them -- both of them can be isolated from cattle. One of them you see in cattle, but you very

rarely see in humans. In fact, you may never even see it in humans. The other clone you do see transmitted to humans and the way we interpret that is that, in fact, one of these subclones is less virulent or virulent where it's just not efficiently transmitted from cattle to human.

One example, it's easy to see if it was missing virulence factors, if it had lost a virulence gene, why it might be arivulent. On the physiological side, on the transmission side, you might think of this subclone being temperature-sensitive, let's say. So it doesn't survive cooking as well as these guys do, and therefore, it's not transmitted as efficiently. That's just an example.

That's -- I don't know that that's the case. So anyway, that's two genetically distinct lineages of 0157 out there, at least two. We want to know what the genetic differences are, and then we want to convert that back to what the genetic differences are. That is to say, how did the genetic differences correlate back to the differences in the traits of this organism, the character traits in virulence or in physiology? And how do we go about doing that?

I don't have time to describe how we landed on this, but just -- you'll have to trust me. Most of this is published, by the way. So you can look at the details in the publications. One of the things we do know that's contributing to the diversions or the differences between

these subclones are little bacterial viruses called bacteriophage.

Bacteria, just like we do, have viruses that infect them. And sometimes those viruses choose, rather than to blow the bacterium apart, to sit down in its chromosome and become one with the bacterium for awhile. We've pulled one of these viruses out of 0157:H7, and in fact, it encodes one of the Shiga toxin genes. This virus is called HB 4 down here.

Now, to make a long story short, what we've noticed is that we've sequenced almost all of the genome of this bacteriophage now, and what we've noticed is that these green regions right here are regions that are at least 95 percent identical to these other bacteriophages. And the main takeaway message I want you to get here is the fact that bacteriophage diversity contributes to a lot of the genome diversity that you see in 0157:H7 isolates.

And bacteriophage, since they evolve very rapidly by swapping segments of their chromosome, can contribute to very rapid evolution in 0157:H7 and, perhaps, in other bacteria. All right. So there's one genetic difference that we know of and that we're pursuing.

The other thing we're interested in doing now is comparing the entire genome. The studies that we did that I've just showed you were done at 20 percent genome

coverage. Now that we understand the genetic relationship amongst those isolates, we want to cover the entire genome. And the reason we want to do that is we want to identify all of the lineage-specific polymorphisms. That's a $2-dollar word. What that stands for, call it OSP.

A lineage-specific polymorphism is an alteration that occurs in one subclone that's not in the other and vice versa. Okay. We call those OSPs. We want to identify all of them. And we want to do that for a couple of reasons.

First of all, we want to design LSP tests, so that we can test for the different subclones very, very rapidly. Right now, it's sort of a difficulty for us to distinguish between these subclones. It takes us a week or so to do it. All right. So we want to develop a test that'll greatly facilitate the epidemiological studies, because we want to know something about the distribution of these subclones.

What is the real prevalence of these subclones in different populations? The other thing that it'll allow us to do is if we cover the entire genome is to learn something about the genetics and the physiology of these sub-clones. So just to show you that we thought about the problem and calculated how we're going to do this, each primer combination that we do provides about 200 KB of coverage, on average.

It'll take us about 30 different OBGS combinations to get 1-x coverage of the genome; 120 different combinations will give us 4-x coverage. That'll allow us to account for any regions where these priming sites are cold stops in the genome.

There's on an average, what we've seen so far, that three of these lineage-specific polymorphisms for any primer combinations -- so we're probably going to be looking at anywhere around 360 different lineage-specific polymorphisms. And at 4-x coverage, then that number 360 you could essentially divide it by four. That will be the actual number, because many of them should be overlapping.

This just sort of gives you an example of how you can picture these lineage-specific polymorphisms. The white here, the white lines, represent just different isolates of E. coli 0157:H7. And that's the identical part. That's the part that would be identical between any isolate, any pair of isolates that you looked at out there. Okay.

The 0157:H7-specific markers are shown in yellow. Those would be the markers that discriminate 0157:H7 from other types of E. coli, other flavors of E. coli you find out there. And then, these red and pink markers would be the OSP's, the subclone-specific markers, or lineage-specific polymorphisms that occur only in subgroups of 0157:H7.

And then, we have even smaller groups that we call Clade-specic (phonetic) markers. So those are the different types of markers that you would come across. Just to show you real quick how, in fact, we can find these quite readily, here we've cheated a little bit.

And this is what we have to do to find these things and that we've lined the organisms up, or the samples up, and file a genetic order -- that is, in the order of the genetic relationships on the automated sequencer here. And here, you can see bands that are present only in one lineage.

There's another one present only in another lineage. Same thing here. Same thing here. Same thing here. So you can find these examples of these things. It's not terribly difficult for us to find. If we've covered the genome, if we've done our job, each lineage-specific polymorphism will be picked up on different segments, different primer combinations by our methods, so we'll have enough overlap here to be sure that we've covered the entire genome.

And once we identify these things, then we have specific specialized equipment that we can use to cut those bands out. Here's an example of two of the bands. Here's one band here. Here's another one here. We've actually cut it out. We've used PCR to reamplify it. It's purified now. We can go through some PCR chemistry and some magic here and get the DNA sequence of this

particular product and know exactly where it is in the chromosome and pinpoint it.

And this is just one example that I have where we've done that. This happens to be a nine-base duplication that occurs in the gene encoding methenyltetrahydrofolate cyclohydrolase. It's a gene that's -- that's really a $2-dollar word. But that's a gene involved in being a synthesis.

And in what we've referred to as the bovine lineage, there was an eight or nine-base duplication that occurred right in the lighter region of that gene and actually looks like the footprint of a transposer jumped out of there at some point in the evolution of this bug.

So anyway, what have I told you about? Well, the bottom line is that there's at least two genetically distinct subclones of 0157 that can be isolated from cattle in the U.S. One of these subclones is rarely isolated from patients with hemorrhagic colitis, at least in the U.S. The subclone is, perhaps, less virulent or not as readily transmittable.

That's our conclusion and reason -- genomics to identify the specific alterations that distinguish these subclones to begin to go back here and understand whether this is a virulence of a transmission phenomenon. And lastly, these are just the people who contributed to my work and my collaborators in the funding. Thanks.

MR. BILLY: Okay. Thank you very much. Are there any questions from the panel? Yes, Bill?

DR. CRAY: Bill Cray, FSIS. Have you looked at isolates from produce at all?

DR. BENSON: I'd love to. I would like to look at them. We haven't yet. And if anybody in this room would send them to me, we would be glad to look at them.

DR. CRAY: Also have you looked at isolates from deer or sheep?

DR. BENSON: I do have isolates, a couple from deer, one from a raccoon that I've looked at. And in fact, they group in there with the cattle isolates so far. But it's not a large enough sample to say that's how they are all going to shake out.

DR. CRAY: You mentioned in the U.S. that the human isolates are in the first group. Have you looked at European isolates? And I think that you mentioned --

DR. BENSON: Yes, yes. So what we've done is to get at this from an epidemiologic -- it's very difficult to actually demonstrate whether a test -- whether or not one of these lineages are virulent or not, because you can't do the real test. And I can't get volunteers. That's not a real laughing matter in this room, but it's the truth.

So what we've done is there's an epidemiological approach we've taken. And there's a phenomenon in Central Europe and also in Australia. It

turns out in those countries they have E. coli in their cows, but not in their people. So they have 0157:H7 in their cows at about the same prevalence that we have here in the United States. However, it's very rarely isolated from patients.

In fact, it's other serotypes of hemorrgagic E. coli that cause problems over there. So this is very preliminary evidence now, and I'm not finished with the analysis, which is why I don't have the slide. But I'm comfortable enough to tell you, we've looked at a set of isolates from cattle and from humans, the few human 0157:H7 isolates that are from Australia.

And in fact, they all look like that lineage, too. They look like the, what I've referred to, the bovine lineage, a lineage, too, here in the United States. So my interpretation of that is that, in fact, that lineage is virulent, perhaps, less virulent.

And the other interpretation you would make you would say it's less virulent, because it's in their cattle. But very seldom does it ever cause disease in their humans. If it's the primary clone in the cattle, then you would expect it to be the one you'd isolate from the humans, as well, or what few patients there were.

MR. BILLY: From your work, have you been able to determine if there's -- I know you haven't done the virulence study, but the known virulence markers attaching and facing hemolysin and Shiga toxins --

DR. BENSON: They are all there.

MR. BILLY: They are all there.

DR. BENSON: They are all there.

MR. BILLY: Are they all there, maybe, two or three in humans versus one in the cow?

DR. BENSON: Yes, that we haven't done. We haven't quantified that. But all I know is, of the isolates we've looked at, most of the isolates that all of those tests have been run on, all of the known virulence factors are present. I don't know anything about whether the genes are expressed or not. It could be that they are turned off in one lineage and not in the other. But I know that they are at least present.

MR. BILLY: All right. Stan?

MR. EMERLING: Stan Emerling representing NET (phonetic). Just a question. Are any of these differences in the genetic makeup that you were talking about, E. coli 0157:H7, are they peculiar to any specific breed or breeds of cattle?

DR. BENSON: I don't have any data regarding that right now. There could be. I think you'd have to design a study to ask that very question.

MR. EMERLING: But it could be possible?

DR. BENSON: Oh, it's possible. I can tell you this going back to the Australian isolates that we've look at, I said they did look like the lineage, two isolates. But nonetheless, they were distinct. I could

tell you that they were from another country. It was clear that there is some geographic isolation to them. So there could be in animals, as well.

MR. EMERLING: And that carries over into human beings could be, perhaps, some trait also or not? I mean, the fact it showed in some and not in others?

DR. BENSON: I don't know.

MR. EMERLING: I thought you showed a difference in the genetic makeup.

DR. BENSON: Right. So the idea is that there's several populations of 0157 out there, several of these subclones out there. Okay. And you can isolate these different subclones from animals, but not all of those subclones the way you isolate from humans that humans that have disease.

And so what that suggests is that, just because you isolate 0157:H7 from an animal, it makes you question, well, are all of them capable of causing disease? Or can all of them do it with equal efficiency? That's what I'm getting into.

MR. BILLY: Dean, then Sonja.

MR. DANIELSON: Thank you, Dr. Benson. That was very interesting. It's a fairly new piece of information that's come about. So I have two questions. Has this theory or this discovery of yours been -- it's very complex and it's way over my head. But has it been

duplicated or verified by a second independent source? Or is this just emanating through your laboratory?

DR. BENSON: It's in the process of being verified independently by another source. It has not been reported in peer-review literature yet, that other source. What I can tell you, though, is while it sounds really striking that we find this phenomenon, if you take a step back and think about it, if you're really looking at the same sort of genetic drift and genetic shift that you see with influenza or any infectious disease, cholera, you name it, it's the same type of phenomenon that you're seeing there.

MR. DANIELSON: Okay. It'll be very interesting if and when that second source becomes available. The second question, if you have an 0157:H7 test and a result, let's say, with a new standard method or the new method and it says it's positive, do you know if that's a virulent or a nonvirulent 0157, based upon that result?

DR. BENSON: On the test that's currently being done, no.

MR. DANIELSON: How about with PCR?

DR. BENSON: Not necessarily.

MR. DANIELSON: Thank you.

MR. BILLY: Kay?

MS. WACHSMUTH: Yes. I noticed Tom Widham is one of your collaborators. Has Tom run this clone in the

multi -- because that would eliminate any of the potential problems you see with this diversity in bacteriophage and other, maybe, non -- I mean, since he looks at core enzymes?

DR. BENSON: Right, right.

MS. WACHSMUTH: Has he done these?

DR. BENSON: In a lot of these -- I wouldn't say all of them, but a lot of them he's run in the past. And, again, the reason we developed our method was to get around the limitations of his method. And the limitations of his method is it's not sensitive enough to discriminate subclones of 0157:H7.

MS. WACHSMUTH: But it does have a basis in genetics, since he's looking at how the sequence of those genes and code enzymes --

DR. BENSON: Yes. Now he's doing -- exactly. Now he's doing it by sequence.

MS. WACHSMUTH: To me, it might be more meaningful to have that link to potentially look at repeat sequences that you don't really know what they might code for. Or is something like that --

DR. BENSON: No, no, no. That's -- these aren't repeat sequences like you normally --

MS. WACHSMUTH: Those are just to generate the --

DR. BENSON: These are frequent words, but they are very short. They are eight bases.

MS. WACHSMUTH: But you don't know the gene products right?

DR. BENSON: Pardon?

MS. WACHSMUTH: You don't know the gene products right?

DR. BENSON: No. They are too short to encode gene products. There are frequent words, eight bases and links that occur in a very nonrandom distribution on a chromosome. And this phenomenon has been documented now in almost every bacterial chromosome that's been sequenced so far.

MS. WACHSMUTH: No. I wasn't taking any issue with that.

DR. BENSON: Yes.

MS. WACHSMUTH: I just am saying in terms of genetic relationships of strains, it seems that it might be more meaningful to look at those enzymes that are housekeeping --

DR. BENSON: But you can -- Tom and I argue about this all the time. You can look at four low --

MS. WACHSMUTH: I worked with Tom. That's why --

DR. BENSON: -- you can look at 451. To me, I'd go with 1,250 data points over 4.

MS. WACHSMUTH: You know what the four really are when you sequence the genes.

DR. BENSON: Right. But we can find out what the alterations are. That's what I'm trying to get across is we can go in and fish out the alterations that are relevant and find out exactly what they are, which is what we're doing right now.

MR. BILLY: Sonja?

DR. OLSEN: Sonja Olsen, CDC. I was just curious about your human isolates you used, if you knew if they were from sporadic infections or outbreak-associated --

DR. BENSON: Funny you should ask. I have a very difficult time getting isolates, particularly from humans and particularly from CDC. (Laughter).

I'm sorry to bring that up, but it's a problem I've had. The isolates that we did have from humans were from both sporadic cases and from outbreaks.

DR. OLSEN: Okay. And did you see any specific clustering --

DR. BENSON: We did tend to see clustering of outbreak isolates. But I'd have to look at a whole bunch more before I would really want to make that statement.

MR. BILLY: Go ahead.

MS. KOSTY: Dr. Benson, this is Lynn Kosty with NCBA. Just one question for you. In light of your data, what does this mean for things like Dr. Powell's risk assessment where there is some concern now that maybe not all E. coli strains are equal while looking in the feed

lot or on the incoming hides? What is your opinion there?

DR. BENSON: My opinion there is that if you really want to do a risk assessment, then one needs to include a factor in there if you could come up with a wobble factor for virulence. And again, I don't know how to predict virulence.

I'm not necessarily sure that anybody knows how to do that fairly well, particularly with 0157:H7. But my suggestion would be to come up with some wobble factor where you can account for the different subclones or quit counting just E. coli 0157:H7 and start classifying them as subclone A, subclone B, subclone C. That's my suggestion. You're talking about an awful lot of work to do that.

MR. BILLY: I concur. The person at that microphone.

MR. BOLTON: Lance Bolton, Dupont Polycon. Just a quick question. I think you've about answered this, but before I get to that, I'd like to say very impressive work.

DR. BENSON: Thank you.

MR. BOLTON: I really find it very fascinating. But what I was wondering is if it would be possible to develop a set of primers for PCR that would capture most, if not all, of the human virulent subclones.

DR. BENSON: That's what we're doing, yes. Yes.

MR. BOLTON: Would that ever be actually a practical test, so that you could get the number down, the number of --

DR. BENSON: It would be very practical, because then you'd -- that's one of the reasons we're covering the genomes, because we want to pull out all of them. We'll take 20 of them -- 20 is a nice number -- to make a very robust test for. That's the 20 most relevant markers that can discriminate the two subclones one from another. It's a multiplex test, so you can do it in one shot.

MR. BOLTON: So about 20 would actually get --

DR. BENSON: Or we can do 50. We could do -- the problem is not everybody can afford automated DNA sequencers like we have. So we have to try and design it, so it'll fit different types of electrophoresis formats.

MR. BOLTON: Thank you.

DR. BENSON: Yes.

MR. BILLY: Okay. Bill?

DR. CRAY: Bill Cray, FSIS. All of the cattle isolates from the 1991 genomes survey were examined for virulence attributes. And all of those were toxin-positive, EAG-positive, and also EHEC-plasma positive. Based on that, would you say that we should

still, at this point, consider all E. coli 0157 isolates to be potential human pathogens?

DR. BENSON: If it were me, because I have young children, I would say yes. At this point, until we learn more about these subclones, we need to learn more about their distribution. I think we need to map that better, because what we did wasn't really a prevalence study.

What we did was simply a genetic-relatedness study. Somebody needs to go out and do a real prevalence study. And, you know, that's something that I'd be interested in hooking up with people to do once we've generated these specific markers that make our lives a lot easier in terms of data analysis.

I should say that those 1,251 markers should -- meat samples are scored manually, because there was no software on the market that could deal with that. So that's another issue that we have which, again, is another driving force for us to generate this specific test.

MR. BILLY: Mark?

DR. POWELL: Mark Powell, FSIS. That answered the question that I was going to raise. And all kidding aside, not only would it make a lot more work for the risk assessment team, but also that doesn't give us a handle yet on the relative prevalence occurring in the wild population.

And, as well, because at least some of the bovine isolates are found in human isolates match, there may be some differential infectivity, but knowing that and quantifying that are two very different things.

DR. BENSON: And I agree with that. But the point I want to make is that we do need to change our thinking in that not all 0157:H7 are identical. There are some very, I think, quantifiable differences, both genetically and probably physiologically, as well. And we need to hammer away at that and identify what those are to determine whether we need to worry at all on 0157:H7.

MR. BILLY: Okay. All right. It's now 3:30, and I'd like to take a break for 15 minutes.

(Whereupon, a 15-minute break was taken.)

MR. BILLY: Okay. We're sort of on the home stretch. And we don't want to short any of the remaining speakers' time in terms of their contributions to what I think is a very important meeting. So if you all take your seats, the next speaker is Dr. Colin Gill.

He is a meat preservation and hygiene microbiologist with Agriculture and Agrifood Canada. His presentation will be on interventions for assuring the microbiological safety of raw red meat. Dr. Gill?

DR. GILL: Thank you. Right. Thank you. Sorry for the size of my paper, but I thought there was a few bumps that had to be made. The first thing is that

intervention is to give surety of safe microbiological conditions will only be effective if they are implemented as part of our effective HACCP system.

To be effective, the HACCP system for raw meat must be implemented on the basis of appropriate microbiological data. That's not just any old microbiological data, but appropriate data. Consequently, current systems at meat plants are not HACCP systems at all. They are quality management systems for assuring the quality of compliance with regulatory requirements. Whether or not those regulatory requirements are effective can be questioned.

They are, after all, much the same sort of requirements that have been enforced for 30 or 40 years. And they haven't worked before, and changing the name to HACCP isn't going to make them work now. Effective HACCP systems can be based on the enumeration of appropriate indicator organisms. There is very little point in chasing after specific pathogens, because there are just too few of them on which to base a system for process control.

When using indicators to evaluate the microbiological performance of a process, it is necessary to look at their numbers on the product passing through the process, not on the numbers as are hugely done, because during a process, the variation in the

distributions can change. And if you don't take account of that, you can get the wrong answer.

You can also look at individual operations with a process, including decontaminating treatments. And it's in decontaminating treatments often influence the variance greatly if they don't take the change in distribution into account. You will quite often get the wrong impression as to what your intervention is doing.

Sequel indicator organisms are total -- coliforms, generic E. coli -- and we hope sometime in the not-too-distant-future generic listeria. The more indicators you look at, the better. The understanding of your microbiological effects of your processor are going to be -- because none of these indicators are interchangeable.

Your reduced total aerobic count it doesn't necessarily mean you've reduced numbers of E. coli and vice versa. Interventions are four types. You have those for preventing microbiological contamination, and that's as much an intervention as any of the other ones; those for decontaminating selected areas of product surfaces, maybe on carcasses; those for decontaminating all surfaces or the whole mass of the product, the holistic approach; and those for dealing effectively with misprocessed product.

In some processes, most of the microbiological contamination will occur during only a few of many

operations. This is the classic situation where those operations are your critical control points. If you misidentify them, then you won't be in control of your process.

I will give an example which is the skimming of beef process carcass hindquarters which can cause -- does cause more contamination in the meat at some plants and not at others. Here's a set of results from three plants. As you will see, the microbiological performance of those operations on the carcasses at plant A are far more deleterious to its microbiological condition than the operations at the other two plants.

When some of the procedures used at plant B and C were implemented at plant A, contamination on the carcasses at plant A were reduced. Of course, that reduction will only be effective in the final products. And in this case, we were looking at the dressing process, so we considered the carcasses leaving the dressing operation or leaving the dressing floor.

And as you can see, the intervention effectively reduced contamination with -- and E. coli by something over one log unit, which is a nice handy little number to have since it didn't cost anything, just a little bit of effort. You will also notice that the coliform numbers haven't changed at all, and that is because the coliforms on these carcasses were being

deposited on the carcass mainly from improperly cleaned equipment later on in the process.

So there's no shift in that, but it tells you something that something else is going on further down the process that is well worth looking at. In other instances, there may be fundamental problems with the procedures or the equipment used in processes. And these would have to be addressed by radical changes before the contamination of a product could be brought under control.

For example, procedures for cleaning personal and fixed equipment are inadequate at most meat packing plants. Here's an example of the bacteria recovered from equipment immediately before the start of work on a number of days. As you will see, we recovered large numbers of bacteria from all but one sample from gloves, mesh gloves, and items of fixed equipment. And those included, in some cases, substantial populations of generic E. coli.

And you're going to run your meat through this. Consequently, most of the bacteria on the meat dispatched from many packing plants are deposited on the product during the carcass-breaking process, not during the carcass-dressing process where all -- on which all eyes are fixed.

Here is the result from four plants before and after the carcass-dressing process -- the carcass-

breaking process. This is carcasses entering and cuts leaving. And as you will see, the numbers of E. coli out of these products go up dramatically at plant A.

We have a 5-log increase in the numbers of E. coli on the product. And we had 20 logs at plant B, one log at plant C, and plant B looks as though it's got everything under control. There is, however, one point that we're looking at the moment. We know we recover by swabbing or by incision similar numbers of bacteria from carcasses. It doesn't matter which procedure you use, you'll get much the same numbers.

Looking at manufacturing beef recently, we find that swabbing will recover only about 1 percent of the bacteria that are present on it. We're just looking at cuts at the moment. But those increases in those numbers could, in fact, be a hundredfold greater than those earlier indicated. Not a pretty picture.

Such situations will be remedied if procedures for assured effective cleaning of personal equipments are adopted. And that is mainly a matter of management, designs for cleanable meat plant equipment are developed, and existing equipment is modified to be cleaned or it is replaced.

The big problem here is that the equipment used at meat plants has usually been designed without any thought to its cleanability, whatsoever. It is just assumed that it will be cleanable in due course and is

required. The consequence is that some of it is not cleanable at all and, in fact, isn't cleaned. And finally, with that, effective cleaning procedures for meat plant equipment are implemented and are implemented on a regular basis.

I don't believe -- I don't mean to imply that everyone isn't trying very hard to clean this equipment, but the fact of the matter is it is not being effectively cleaned. It is essential that this aspect of hygiene control is looked at urgently, because it could be the major factor in compromising the safety of meat.

Treatments for decontamination in selected areas, the old tradition of trimming, vacuum cleaning which has been used on some parts of the carcass for many years, and the now presently trendy vacuum-cleaning while treating the surface with hot water or steam, all are effective for using visible contamination which, after all, is the prime purpose.

Cleaning for selected areas, therefore, are usually guided by the presence of visible contamination. You treat the carcass and you treat the carcass to get rid of this more contamination. Used in this manner, these treatments are largely ineffective. There's some data, basically, as microbody and microbiological treatments, they don't work.

But as for removing visible contamination, which is not a bad idea after all, you can go with 31 of

them, and they will give you a similar result. Trimming can be somewhat effective if it is supplied to an area likely to be contaminated, irrespective of the appearance. Here's a case where they get a live drop in E. coli numbers as a result of trimming in an area that's usually heavy contaminated.

They are not trimming it to reduce microbiological contamination. They are trimming it to remove fat. So for that reason, I wouldn't say any of these interventions were necessarily ineffective, but I want to indicate that they may or may not be ineffective in your system, unless you find out from direct microbiological data in each particular process you do not know what it's doing.

And you cannot assume that it's doing something. Vacuuming, hot-water vacuuming treatments are likely to be ineffective however they are, for they are applied vacuuming because all this is going to do is remove visible contamination.

And hot-water vacuuming, because you're not applying heating the carcass surface for long enough for it to have any microbial effect, to be effective, you've got to heat to a greater degree centigrade for about 10 seconds. Since you're applying these treatments to a surface area of several hundred square centimeters through a head that's only 50 square centimeters and serve in an area, you simply cannot apply it for the

requisite time to heat the carcass surface to give you antimicrobial effects.

Washing of carcasses is usually ineffective. That's the usual result you get and really no effect at all. You will notice there is an apparent increase in the number of coliforms in E. coli. It's half a log, not important. It isn't a real increase. The water isn't heavily contaminated.

What's happening is you're getting redistribution, and because you can undertake a limited number of samples, you get the illusion of these -- that the numbers have increased. However, washing of carcasses can be performed to reduce bacterial contamination and washing of at least some offals may usually reduce bacterial contamination.

Here are some examples: There is a carcass washing process where you're getting a good log reduction in E. coli numbers by washing of tails and tongues. Particularly, you get very large reductions as a result of washing these things. I may, however, add that in none of these cases did any of the people involved in these washing processes know what the microbiological effects of the washing processes were.

These things are not being washed to reduce bacterial numbers. They are being washed to remove visible contamination. The thing is if you know what microbiological effects they are having, it should be

possible to adjust various operations of these types to achieve consistent large reductions or useful reduction in bacterial numbers.

If you don't know what the bacterial -- the microbiological effects are, they are not -- they will be useless. They probably will have no such effects at all. Decontamination with antimicrobial solutions, they are highly effective in laboratory circumstances. They are probably largely ineffective in actual practice.

The reasons are that complete coverage of a product, particularly carcasses, is difficult without using uneconomically large amounts of solution. Bacteria in the natural flora vary widely in their susceptibilities to antimicrobials. It's quite usual to see large differences in the numbers of bacteria destroyed in the experimental circumstances with inocula and with a natural population.

Many of the antimicrobials tend to act on the meat, rather than the microbe. You've got very little microbe and a great deal of meat. And they'll tend to react with the meat itself.

And solutions and concentrations in temperatures that are effective for destroying bacteria tend to be damaging to the product, so there is a natural tendency if you don't know what the microbiological effects are to reduce the concentration to the

temperatures down to levels where they are ineffective for bacterial decontamination anyway.

There are few very reports of the effects of in-plant antimicrobial treatments. I presume that there is possibly some more information out there in the industry, but very little of it has been reported up till now. What data there is does tend to confirm that, in practice, these are ineffective.

This was a study of decontamination using acetic acid at four plants. And there was found to be no difference in the microbiological condition of the meat from the four plants. The only thing I find puzzling about that is the uniformity of the four plants. I've never come across four plants that were all similar. That is the reported data.

Pasteurizing toxins with steam or hot-water can be effective. There's some data for each of them, a good two- to three-log reduction in E. coli numbers, considerably less with total aerobes. Pasteurizing the carcasses with steam is rather more complicated. You need clean, dry carcass surfaces to do this, or otherwise, you're just heating the dirt on the surface or the film or water on the surface, rather than dealing with the bacteria.

You need a single treatment chamber to get the uniform condensation of steam onto the object to be pasteurized. And the effective treatment is a carcass

surface temperature of 103 centigrade for 6.5 seconds. There isn't a report in the literature which describes the treatment of, I think, 80 degrees centigrade for about 5 seconds. And it is quite obvious from the microbiological data that it's ineffective.

Pasteurizing carcasses with water is rather easier and cruder which is nice for meat plants, carcass surface temperatures of greater than 80 degrees centigrade for 10 seconds, and carcasses don't need to be clean or dry or anything because you have the mechanical effect of dumping large quantities of water onto them. Manufacturing beef, too, can be pasteurized with hot-water.

The treatment times, however, are considerably longer, over 30 seconds. In this case, we got effective decontamination at 45 seconds, or at least we couldn't find anything much after 45 seconds. But in other cases there is so much variability between manufacturing beef that even 45 seconds has little -- therefore we have to go to a 60-second treatment which is considered to be longer than you have to go with carcasses.

Carcass-cleaning processes, most probably, have little effect on the microbiological quality of the product. There's a couple of typical cases, air cooling and spray chilling. Both of them, essentially, they maintain the bacterial load just where it was when they went into the chiller. But all carcass-cooling processes

can give large reduction in the number of E. coli and other gram-negative bacteria.

And there's a couple of cases in air cooling processes which is giving you a nice log reduction in E. coli numbers and a spray chilling process, which is giving a two-log reduction in E. coli numbers. I'm not quite sure how that spray chilling process is doing it. But I think it involves freezing of a film of, essentially, pure water onto the carcass surface when they drop the temperature to minus 5 degrees centigrade at the end of the spraying process.

It's something that would be nice to have a look at. Various other slow freezing processes may also reduce bacterial numbers. However, few plants would know the microbiological effects of their cooling processes. And none at all likely operate their cooling processes to assure reductions in the numbers of E. coli on product.

But as you see, you can get reductions that are at good or better than some after interventions that are being used with carcasses. Radiation treatments, you've had a talk about that. I'm not very enthusiastic about it. It does seem to be technological overkill, and it doesn't seem to be -- it's not really necessary, there are alternatives.

One shortcoming with meat plants is they do not generally include specific procedures for reacting immediately to misprocessing events as they occur online,

nor do they include procedures for treating misprocessed product to assuredly return it to the microbiological condition of properly processed product or to reject the product from usual processing if its conditions cannot be assured.

What usually happens is that misprocessed is identified on the basis of visible contamination. It is pulled off the line. The visible contamination is removed, and it is returned to the line. There is no surety whatsoever that its microbiological condition has not been grossly compromised by the misprocessing. We don't know what its microbiological condition is at all, usually.

And varying procedures for dealing online and effectively with misprocessed product are essential if heavily contaminated product is not to sporadically enter the process to compromise all the rest of the production. So my conclusions, proper implementation of HACCP systems at meat plants can give meat an assured microbiological safety.

A proper HACCP system must include procedures for minimizing microbiological contamination, procedures for decontamination product, and procedures for dealing with misprocessed product in timely and effective manners. A system that lacks those elements is not an effective HACCP system. Procedures currently advocated

in and employed for HACCP implementation do not give effective HACCP systems.

And finally, an effective HACCP system should give meat with total aerobes at less than 2 logs CFU per square centimeter and E. coli at less than 0 logs CFU per thousand square centimeters. These are levels approaching the microbiological condition of potable water. And if you can do that, there really is very little point of going pathogen hunting. Thanks.

MR. BILLY: Okay. Thank you. Are there questions from the panel? Dan?

DR. ENGELJOHN: Dan Engeljohn with USDA. I have two questions. On the carcass-cooling information you presented, is that related to cooling the carcass once it's eviscerated down to a certain surface temperature? Or do you know what those temperatures were related to?

DR. GILL: All carcasses are cooled to a nominal deep temperature. In theory, carcasses are not moved from the chiller before they fall to 10 degrees centigrade at their warmest point which is usually the deep-pit temperature.

In practice, however, you'll find that a lot -- there's always a fraction of carcasses that are substantially warmer than that when they are moved out of the chiller, because in the backs of chillers, the air distribution is uncertain. And some carcasses are always

shielded from effective cooling. What is done about this, I'm not entirely sure. I think it varies considerably between plant to plant.

But, no, there is no defined end point. You will always get a range of end points in temperatures. Surface temperature is not a consideration. Some of the deep temperatures is a consideration. But, in fact, in running these operations, the major consideration at most plants is the avoidance of weight loss, shrinkage. And that's the main operating parameter for the chilling system.

DR. ENGELJOHN: I did have a follow-up. On your very last slide there, an effective HACCP system should give meat what the total aerobes in the numbers you have there. Is that generally for carcasses, processing meat?

DR. GILL: You can produce carcasses of that level. In fact, I know of one or two plants who are doing something very near to that at the moment. I mean, just because I say, because they haven't got an effective HACCP system -- it's simply that you haven't got a HACCP system you can check up on. Some plants are doing an extremely good job, and some plants are getting very near to that.

There's no darn reason why you can't maintain that condition right all the way through the rest of the

process. In fact, you should. The fact that many plants don't is a real problem.

MR. BILLY: Okay. Randy?

DR. PHEBUS: Randy Phebus, Kansas State University. Can I ask you how you put these data tables together? You don't provide any references as to where these figures come from. The second question is how common or, as far as the numbers that are presented here, were they obtained using the same type of sampling method and analytical method?

DR. GILL: Well, they were all obtained using the same method. Yes, what we do is --

DR. PHEBUS: All of them use incision sampling; is that right?

DR. GILL: No, no, swab sampling.

DR. PHEBUS: Well, okay. All right. Go ahead.

DR. GILL: I have data that shows the swab -- not only I, but others, have data that shows on carcasses swab sampling is as effective as incision sampling for recovering bacteria. As I say, that's all obtained the same way.

What we do is we take 25 random samples from randomly selected carcasses moving through the process and estimate from there the log mean numbers of bacteria on the product. This is a process control system we're trying to estimate what the process is doing.

DR. PHEBUS: So all these data figures are from your personal research?

DR. GILL: Yes. I can send you all the papers if you like. Most of it's been -- well, all of that's been published.

MR. BILLY: Okay. Kim, and then Nancy.

MS. RICE: Dr. Gill, Kim Rice, the American Meat Institute. I missed your first few slides and I apologize. But did you say -- is all of this information based on experience in Canada or the U.S.?

DR. GILL: It's from North American plants.

MS. RICE: How many plants are U.S.-federally inspected plants involved in your findings?

DR. GILL: I'm sorry. I'm not prepared to discuss my sources at all, except to tell you that these are all commercial plants. I worked in New Zealand for a long time, as well.

MS. RICE: So let me ask you this: Is this your -- is this a culmination of just experience or --

DR. GILL: No. This is all published data.

MS. RICE: Okay.

MR. BILLY: Nancy?

MS. DONLEY: Nancy Donley from STOP. Dr. Gill, you mentioned a couple of times today that the low prevalence of E. coli 0157:H7 and that you said it's not necessary to look specifically for it, because it is in such very low prevalence.

But we've hearing today that it's quite the controversy with these more sensitive testing methods that, in particular, there appears to be a lot more of it. Do you use the testing method that we heard about this morning, the magnetic beads and the --

DR. GILL: I think, perhaps, you misheard me. What I said was it was pointless to look for E. coli 0157:H7 in relationship to controlling the process. What you're trying to do is control the process. So you need to have numbers, appropriate microbiological data to control that process. You simply cannot get useful numbers of E. coli 0157:H7.

What you can do with this sort of process is reduce your numbers of indicator organisms to such low levels that your risk is contained. This is exactly what's done with milk. It's exactly what's done with water. And there's no reason why you can't do it with meat. But chasing around after specific pathogens simply does not solve your problem for you, because if you don't find a pathogen, it doesn't tell you it's not there. It just says you didn't find it that time.

MR. BILLY: Okay. Caroline?

MS. DeWAAL: Thank you. Caroline Smith DeWaal, Center for Science in the Public Interest. And this question follows up on Nancy's question. Are you aware of data from some fast-food plants in the U.S. showing that systems which combine both indicator organisms and

pathogen testing actually give you the best of both worlds, because you get a good sense of what's going on with the indicator organisms, but you also know what your pathogen load is on products? And do you have any objection to that kind of a broad-spectrum testing approach?

DR. GILL: Well, the only thing is that testing for pathogens distracts attention from controlling a process in the first place.

MS. DeWAAL: Excuse me, though, if I could. The process is supposed to control the pathogens. Isn't the best measure of process control then control of the pathogens? What else is the process control about?

DR. GILL: Well, unfortunately, no, it's not, because all you get from -- if you're trying to control your process, you've got to have some information to work on. And all you get if you go chasing pathogens is a string of zeros. You can't do anything with that, because those zeros don't tell you that pathogen isn't present. It only tells you you've got a string of zeros.

MR. BILLY: Let me follow up on that. As I understand what you're saying, you're recommending to use a certain species of bacteria to monitor process control. And those are bacteria that are present in numbers that you can detect differences from the various process control procedures and antimicrobial treatments, or whatever that you happen to be using.

How do you feel, then, about the periodic use of tests for pathogens to verify that, in fact, the results you're getting from indicator organisms are, in fact, equating to effective controls? I understand your point about zeros don't tell you anything. Is there some way where you believe that some adequate number of tests would be a good indication of the effectiveness of the controls?

DR. GILL: Just as I stated, you don't use microbiological data to control your process. You use it for validation of your control procedures and for verification of your maintaining control over your procedures. You do not use it for online testing. You can't do enough of it.

You can't do it frequently enough to use it for online testing. That has to be done by your standard operating procedures which you've set up for your process. Having said that, the only reason I could see for using -- testing for pathogens would be for surveillance purposes to see what this actually means, but then you'd have to set up your surveillance properly.

But, yes, I could see it would be useful for regulatory agencies to sort of carry out surveillances of that sort. But really, it has no part at all in relation to a HACCP system if you're operating a HACCP system properly. And if you're operating a HACCP system properly, the whole point of it is you get progressive

improvement of your process to a level where you have a very high degree of assured safety.

Testing for pathogens won't do anything for you. You cannot test safety into a product. It's one of those long-established things that seems to be forgotten at the moment.

MR. BILLY: Okay. Rosemary?

MS. MUCKLOW: Rosemary Mucklow, National Meat Association. I'm beginning to feel like I hang out with Colin Gill, because we spent three days with each other last week and a day this week. The three days we spent together last week, Colin chaired a very distinguished panel of microbiologists from both our country and international companies.

And I wonder if you could, Colin -- better than me anyway -- put into just a few -- couple of sentences the conclusions that those microbiologists came together under your leadership last week. The basic principles, which are not different than you've enunciated here, but maybe you could restate it as the views of, probably, some of the best world microbiologists who look at this system.

DR. GILL: Well, simply put I was gratified and very relieved to find that all of the people present ultimately agreed on almost every one of the -- they did agree on all the major areas. Basically, the conclusions were that the point of microbiological testing in

relationship to food safety should be for the implementation of HACCP systems. There is no other reason for doing it.

If you're going to use microbiological testing in relation to HACCP systems, then you have to go for indicator organisms, because pathogen counts -- counting pathogens won't -- isn't any help in this respect. You are using them in relation to HACCP systems for validation and verification of your -- validation of your control processes and verification that your processes are under control.

That end-product testing is of no value and, in fact, is completely contrary to the whole concept of HACCP implementation. And finally, that if you're going to use these procedures for -- you're going to use microbiological data in relationship to HACCP systems, then you have to go to variables, sampling plans rather than to attribute sampling plans, because if you go to attribute sampling plans, you use much of the information you need for process improvement.

MR. BILLY: Yes, Caroline?

MS. DeWAAL: Caroline Smith DeWaal, Center for Science in the Public Interest. I've heard these arguments for so long. And they are so troubling. How do you deal with a prevalence of, you know -- on some of the plants that we saw today, we saw to 20 percent of the cattle coming in with E. coli 0157:H7. Or in the poultry

industry, we've seen salmonella rates of 20 percent and 25 percent, actually, when the original rule was published.

So the idea that you can't find pathogens because, you know, they are just not going to show up assumes very low levels of pathogens. How do you deal with situations and how do you address the data that was presented today showing that we have higher levels coming into the processing plants and, in fact, testing of carcasses for 0157:H7 documents process control?

I just -- you know, the language is stuff I've heard a lot before, but it assumes a very low incidence of pathogens which, in fact, has not been the case in either our beef industry or our poultry industry.

DR. GILL: Well, I think we should -- you can't equate poultry in this. I mean, as far as the poultry industry is concerned, I quite agree you can use your pathogens as indicator organisms. But these are red meats. I mean, hey, that's all right. But the thing is you looked at that data. All they were talking about was prevalence, found it or didn't find it. The amount of information you get out of that is very small.

They found it on animals. They found it on animals wandering up to the place. Once it was into the plant, it wasn't there. Where's all these organisms? They are not there. You don't know what your processes do. All you can say is we couldn't see any. But you

don't really know how hard you were looking, or it gives you no information, except that you didn't find them.

You need information to be able to control the process. That's why, I'm sorry; I didn't make this up. Other people agree with me. I'll go over it with you, if you'd like, bit by bit. But that is the situation. Would I lie?

MR. BILLY: Okay. Thank you very much. I think we'll move on. The next presentation is going to be a joint presentation by Dr. Randy Phebus, who is the Professor of Food Microbiology at Kansas State University; and his colleague, Dr. Jim Marsden, who is a Regents Distinguished Professor from Kansas State University. Their presentation will be regarding ongoing studies at KSU to characterize pathogen risk in non-intact beef and pork products.

DR. PHEBUS: Okay. I appreciate the opportunity to be here today to present work that we have been doing over the last year-and-a-half, I would say, on risk assessment of non-intact meat products.

Specifically, what we're going to talk about today, a little bit different than what the program actually says, we're going to be looking at blade tenderization of beef products. We're not going to present work that we've done with pork and salmonella. But we have completed a lot of that type of work. And we're in the process of analyzing the data. But this is

specifically directed toward E. coli in blade-tenderized products.

Back in 1997, the National Advisory Committee made the statement, "Due to the low probability of pathogenic organisms present in, or migrating from, the external surface to the interior of beefsteaks, cuts of intact muscle," which means steaks, should be safe if the external surfaces are exposed to temperatures sufficient to affect a cooked color change.

But they also said that there's a lack of scientific data to address the hazards associated with those processes that may cause translocation of the pathogens to the interior of the meat products. So this led to the initiation of our studies to try to generate data to characterize what these processes do.

In case you're not familiar with blade tenderization, it's a process that's used extensively in the beef industry. This is a unit -- this is actually a raw unit that is one of the most popular ways that beef sub-primals are blade-tenderized.

And that particular unit has two of these heads that have these long, slender blades that penetrate the product from the top and go all the way through the sub-primal as the sub-primal works its way down a conveyor belt. So there will be two heads, kind of, stamping this product and cutting the muscle fibers in order to tenderize the product.

Just to kind of put this in a little context, we have looked through the literature, and there's really no foodborne illnesses that have been traced to blade-tenderized product to date. There were two salmonella outbreaks linked to beef roast that had been needle-injected. And this is a different technology than needle injection, but that was really related to undercooking.

Federal law now requires these roasts to be cooked to 145 degrees internally to assure their safety. But since these outbreaks, E. coli 0157:H7 has emerged as our problem in beef. The objectives of our study were, first, we wanted to microscopically visualize these organisms and how they are carried and to what extent they are carried into this processed muscle. So I'll show you how we did that very shortly.

But then, we wanted to determine the effectiveness of cooking processes that are generally used in the commercial scale from well -- from rare to well-done temperatures, own controlling the amount of contamination that might be carried into the center of that sub-primal. Here, we looked at -- we took some green fluorescent E. coli, and this would be the top surface of the sub-primal that we inoculated at approximately six to seven logs, depending on the rep.

And then, it was passed through the equipment, and this would be a penetration point as you're looking

down on the top of that sub-primal. As you can see there along that needle channel, there are probably 20 to 25 green fluorescent E. coli. That is right at the very surface. This is at the very bottom of that sub-primal, at the bottom of the needle channel.

As you can see, there's a lot more here than here, but there's still three to four cells that were carried down in that one channel to the very bottom of the surface. We went a little further to actually quantify how much E. coli is carried from the surface to various levels in the sub-primal to see how it diluted itself out.

Here, we inoculated E. coli by misting approximately seven logs per square centimeter -- excuse me -- six logs per square centimeter on the top surface. And we also looked at a lower inoculum level of 10 to the 3 CFU's per square centimeter and passed those one time through the blade tenderization unit.

And I might say that we have done a couple of surveys since this work, and there's quite a bit of variation in how blade tenderization is actually used in the industry. Actually, there's probably a lot of the people average about two passes through a unit, so it's different, depending on the processor and how they are using the technology.

But anyway, one pass leads to 32 blade penetrations per square inch. We, then, took that sub-

primal that had been processed and we took cores, aseptically took cores -- these dotted arrows represent the path of the needle going in, the blades going in. This would be the inoculated surface.

This is the non-inoculated surface, so we actually cored from this direction from the lowest to the highest concentration, and then pulled the core back out the back end, so that we didn't artificially carry the organisms further down into the core. We, then, looked at various subsections of that core to enumerate the amount of E. coli 0157:H7 there per gram.

And this is what we found, and this data has been very consistent over several replications that we've performed, both at high and low concentrations. As you can see at the surface, which would be right there, we had approximately six logs. And at the very bottom of the core, we had approximately 2.8 logs when we started with the high inoculum.

At three logs, initially, we had about a half a log at the bottom. But the geometric center of that sub-primal, which would be the geometric center of steaks cut from that sub-primal, which theoretically would be the slowest to heat during cook processes, would be approximately at this point. And that corresponds to about 3 to 4 percent of whatever was on the surface was carried to the geometric center. Okay.

So a summary of this part of our work was that E. coli 0157:H7 on the surface of meat were translated throughout the entire volume of the sub-primals by the penetrated blades and that the geometric center contained about 3 or 4 percent of that surface contamination.

Now, if we look at a hypothetical example, say, we're cutting steaks out of this particular sub-primal, and we did have that three logs on the surface -- let's say 3,000 CFU's per gram hypothetically, which that would be an extremely contaminated piece of meat at the center -- we would expect to get about 100 CFU's per gram.

So our conclusions are if pathogens are present on the surface of the sub-primal, adequate cooking is the key to providing safe blade-tenderized products. But then what is an adequate cooking process? So this leads us into the second part of the study, which we looked at oven boiling in the data that I'm going to present to you here, and how that affects control of E. coli 0157:H7 in the product.

In this study, we looked at three sub-primals that were tenderized and three that were left intact as non-tenderized products. And we repeated this study four times. We, then, hand-sliced aseptically into different steaks' thicknesses, a half inch, three-quarters of an inch, and 1.25 inch. And this really mimics what's typically done in food service.

We trimmed those trimmings on the non-inoculated side to 5, 8, and 12 ounces, respectively. We, then, cooked these products, and we tried our best to have an accurate cooking-temperature recording method. And if you've ever done these types of studies, you know it's very difficult to accurately measure internal temperature, because it's a lot of that goes into that. But we feel that we did the best job that we could with this.

We did this in oven boiling. We looked at six target internal temperatures, 120 to 170 degrees Fahrenheit. I just checked with Marty, and the 140 would be considered rare by NAP guidelines for cooking. So this would be very rare. This would be very undercooked, very rare, rare, medium; 145 would be medium rare. And then, we also compared these to non-cooked inoculated controls. Okay.

The steaks were cooked, again, at an ambient air temperature of 300 degrees Fahrenheit. Okay. And this is the data, and this is where I'm going to turn it over to Dr. Marsden, not that I couldn't explain the data.

DR. MARSDEN: Thank you, Randy. What we found in terms of the results were that, in order to be assured of a 5-log reduction across all the different treatments, we had to cook to an internal temperature of 140 degrees. At 130 degrees, which is really below any temperature

that would be likely to be seen in commercial practice, we were -- the data was variable.

For the thicker steaks, the three-fourths inch and inch-and-a-quarter, we did get a five-log reduction. For the thinner steak which is cooked, of course, for a less period of time in order to achieve that temperature, there was quite of bit of variation. And it was generally below five logs. But at 140 degrees, we were able to get a consistent 5-log reduction. Again, 140 degrees is a rare- cooked steak.

One of the phenomenon that we noted was that there is a continued rise in temperature after the steaks are removed from the heat source. We tried to control that by placing the steaks in an ice bath immediately after coming off the heat source. And we still, you can see from this slide, had a temperature rise in each case. From 140, for example, crept up to 145 to 151 degrees; 130 crept up to 137 to 142.

And this is with a deliberate intent to restrain that rise in temperature. In actual practice, that rise in temperature occurs naturally. So there is, if anything, what we're doing here is underestimating the lethality of the process as it would exist in a commercial practice. We concluded that blade tenderization does not significantly affect the safety of beefsteaks when they are cooked to temperatures of 140 degrees or above using this oven broiling method.

It was variation around that five-log reduction. But, in general, we feel confident that we're at that five-log reduction, similar to what we'd do with ground beef if it were cooked to 160 degrees. Significantly, though, there was no difference in risk between the steaks that were tenderized and the steaks that were not tenderized. Okay.

Some regulatory issues, if the objective is, in fact, to achieve a five-log reduction, as we have with ground beef, then internal temperatures slightly higher than 140 degrees may be required, depending on the cooking method and also the thickness of the steak.

If the objective, however, is to ensure the safety of the blade-tenderized steaks, assure that it's equal to non-tenderized product, then 140 was sufficient for all cooking methods. We are looking at, as we've been discussing all day, an integrated HACCP plan that includes a lot of different elements.

One is a validated antimicrobial treatment as a critical control point in the slaughter process, thermal pasteurization or some validated critical control point. Segregation of the slaughter process from the post-slaughter process to prevent cross contamination is important. Control of refrigeration temperatures and the chilling of carcasses is important.

And then, also prerequisite programs to address plant sanitation and personal hygiene issues all come

together to greatly reduce the likelihood of contamination on sub-primals to start with. Now, we looked at high inoculation levels, six logs and three logs, in order to demonstrate this desired five-log reduction.

In the context of this integrated HACCP system, the risk of E. coli 0157:H7 contamination of sub-primals that are destined to be blade-tenderized, obviously, is significantly reduced. With these HACCP systems in place and appropriately applied, the probability of having high levels of contamination on the surface of a sub-primal prior to being blade-tenderized is remote.

Now, in order to make a recommendation regarding the level of reduction that's required to assure the safety of non-intact steaks, we believe that a scientific risk assessment should be conducted which takes into account these upstream reductions, reductions that are associated with each of the critical control points in the overall HACCP system and production process.

Another thing that the agency needs to take into account -- and this is extremely important -- is that non-intact is not a generic description that's appropriate to all the meat products that are not intact muscle. We looked here at blade-tenderization which results in a fairly low level of translocation of

bacteria from the surface into the interior of the muscle.

There are other technologies. For example, a sectioned and formed product may actually resemble ground beef in terms of the potential for translocating bacteria. So just saying non-intact is not enough. The actual process that's involved needs to be considered. The risks are going to be different, depending on the amount of translocation that occurs and translocation that's associated with the various technologies.

So needle-injected or needle-tenderized may be completely different in terms of its risk from sectioned and formed. And finally, by combining an effective HACCP plan with customary cooking practices -- and I noticed that's a term that's used in the USDA regulation -- in this case defined as cooking to a rare degree of doneness, or 140 degrees, assures the safety of blade-tenderized steaks.

Now, there is additional research that's ongoing in this area. One is we're identifying methods of reducing the variability associated with commonly used cooking methods. That's similar to what happened in the ground beef industry for cooking hamburger patties in restaurants after the 1993 outbreak. We've made strides in terms of improving the consistency that goes with the cooking process.

We're also evaluating other technologies. We're evaluating the fiber-mix technology, which involves section and forming of the product. And we're conducting inoculation studies to determine the risks associated with those other types of technologies. Prime rib and rare roast beef prepared from blade-tenderized sub-primals are being evaluated.

There are reported cooking temperatures for prime rib and rare roast beef that are well below the 140 degrees that we validated in the steak study. And we're looking at those temperatures to determine whether or not they are safe. And finally, we're doing a salmonella risk assessment for -- we've already done one for non-intact pork products. We are in the process right now of doing one for non-intact beef products, as well. So with that, I guess we can have questions.

MR. BILLY: Thank you very much. Questions from the panel? Yes, Dan?

DR. ENGELJOHN: Jim, I had two questions. On these non-intact steaks, did you look at the same quality grade? Was the fat content, intramuscular fat, the same in those steaks? Since we know that in hamburger patties, fat content greatly affects the D-value, so I was curious about the quality grade of the steaks.

DR. MARSDEN: Randy, did we look at all Choice?

DR. PHEBUS: Yes, everything was Choice. And the controls and the tests of sub-primals came from the same lot, the same box.

DR. ENGELJOHN: So you would expect them all to be the same degree of intramuscular fat within the Choice grade? They wouldn't be high, low, medium? You don't know?

DR. PHEBUS: These were all exactly the same sub-primals out of the same box. So they would have been identical. We didn't specifically look at, say, high Choice or Prime or anything like that, in selecting our raw materials. But the ones that we did select were the same between control and the test treatments.

DR. ENGELJOHN: Okay. And then the second question, if I could, on those products with a double-pass, do you expect the translocated organism to be there in a double quantity, then?

DR. MARSDEN: I don't know if it'll be exactly double. I think what happens is that the translocation is proportional to the disruption of the surface and carrying that surface into the center of the product. So two passes would certainly result in the greater translocation than one pass.

DR. PHEBUS: I might add on that that it would still be probably 6 percent versus 3 percent. And you're still on the same log cycle range.

DR. MARSDEN: Exactly.

DR. PHEBUS: So I would expect it to be fairly minimal.

MR. BILLY: Bill?

DR. CRAY: Bill Cray, FSIS. How did you enumerate the cells on your cook studies? And also, did you have a resuscitation step in your --

DR. MARSDEN: I'll have Dr. Phebus answer that.

DR. PHEBUS: Okay. On the cook studies, we had to directly plate so that we could enumerate to figure our reductions. We actually played it on PRSA agar which is much less selective and much more attuned to recovering injured cells. So we've got several studies that verify that as being the case. When we had truly negative product, we did enrich and do a qualitative assay to determine whether we could completely eliminate it or not.

MR. BILLY: Other questions? Okay. Marty?

MR. HOLMES: I just wanted to make a statement that, although a large number of our members do send product through two times, two passes, the machine you saw had two heads. Many of our members' machines only have one head. So it may go through two times, but it's going through a one-head machine, not a two-head machine. So that's just something you might consider in the future.

DR. MARSDEN: As Dr. Phebus pointed out, the log level is going to be similar, whether it goes through

one pass or two passes. I wouldn't be too concerned about that.

MR. BILLY: Okay. Nancy?

MS. DONLEY: Nancy Donley from STOP. Jim, did you say that the risk assessment should be done based on that list that you had of a good HACCP system which included, for instance, a CCP in the slaughter phase?

DR. MARSDEN: Uh-huh, I did. That's exactly what I mean is that I think that the risk assessment should be conducted with the precondition that an effective HACCP plan exists which, in my view, would include a critical control point being a validated antimicrobial step that would occur during the slaughter process and the other conditions that I laid out, as well, separation of the slaughter portion of the plant from the post-slaughter portion of the plant, adequate refrigeration, and so on.

MS. DONLEY: But right now, that's not the real world.

DR. MARSDEN: Well, it's actually very often the real world. You have the largest beef plants in the United States all have a validated antimicrobial step in their process. And increasingly, that's the case in smaller plants, as well. So I don't know what the percentage is, but the percentage is probably greater than 90 percent of the beef slaughter plants in the United States have validated intervention technologies in

place, and then whatever extent, to some extent, the other things, as well.

That may be variable in terms of how they separate the slaughter process from the post-slaughter process and refrigeration capabilities and that type of thing. But it's becoming, in my view, certainly in larger plants the norm that these validated interventions are, in fact, in place.

MS. DONLEY: But isn't it in some of these, actually, in some of the smaller plants that a lot of this processing is done? It's not done in --

DR. MARSDEN: Well, the processing is usually done as further processing, so they are not -- it's not like you have slaughter plants, generally, that are doing this. They are buying their sub-primals or raw materials from other plants.

So it's not 100 percent of validated intervention is a pre-condition of bringing this into the plant, and then maybe it should be, but it's generally meat purveyors that are preparing steaks, and so on, for restaurants that do this tenderization step, not slaughter plants.

MR. BILLY: Okay. Marty, and then Mark.

MR. HOLMES: I was going to ask you a question, Jim. You inoculated the surface with six logs. That is a surface of a primal that, typically, would have been trimmed more than once. I mean, you would have had a

carcass that would have been trimmed, a sub-primal trimmed before it ever got to a blade-tenderizer.

DR. MARSDEN: That's right. That's another thing that needs to factored in the risk assessment is that there's a lot of trimming that occurs from the carcass level down to the sub-primal before it enters this machine. And that needs to be factored in, as well.

MR. HOLMES: Can you give me some feel for what would be a typical surface contamination of 0157 on a carcass?

DR. MARSDEN: On a carcass?

MR. HOLMES: On a post-intervention carcass.

DR. MARSDEN: It would be approaching zero.

MR. HOLMES: And then, we're talking about possibly trimming those even further before it goes through a blade tenderizer, so I just want to make that point.

DR. MARSDEN: With all of these provisions in place, the probability of having any E. coli 0157:H7 would be remote, let alone three logs, six logs.

MR. BILLY: Yes, Mark?

DR. POWELL: In the current ground beef risk assessment, rather than taking a worst-case scenario, like you describe for the experimental study that provides very useful information about the performance of cooking, rather than taking worst-case scenario or the alternative that you seem to be proposing which is a

best-case scenario, the agency's approach has been to try and model the as-is scenario, the range of practices, the range of concentrations that are the full range that are existing, given the mix of practices that we see.

DR. MARSDEN: That's right. If you were to do that and, and say for the sake of argument, that you estimate a one-log contamination level, then you're going to translate or translocate 3 percent of that into the center. That's the kind of thing I'm talking about with the risk assessment. And then you could do, like you say, a moderate estimate risk assessment on what cooking requirements would be necessary to assure the safety of the product.

DR. POWELL: And to that end, it would be very helpful to know about the range of practices in the industry, as well as the relative frequency of those practices in the industry.

DR. MARSDEN: Uh-huh, I agree.

MR. BILLY: Okay. Thank you very much. We'll move on now. The next presenter is Ms. Nancy Donley. She is the president of Safe Tables Our Priority. And she's going to provide a consumer's perspective in terms of their expectations regarding this organism.

MS. DONLEY: I'd like to thank you for inviting me here today to present STOP's comments on what we consider a very critically important topic. We come from an radically different perspective than, I dare say, just

about anybody else in this room. STOP's very origins are a direct result from the problem, E. coli 0157:H7, contaminated ground beef that we are discussing today.

Most of you are familiar with STOP, but for those of you who may not be, let me give you a very brief introduction of who we are. Safe Tables Our Priority was founded in 1993 in the wake of the Jack-in-the-Box E. coli 0157:H7 epidemic that sickened over 700 and killed a documented four children in California and the Pacific Northwest.

Grieving parents and concerned friends got together to mourn, vent, and then to discuss ways on how to prevent the horror that they experienced from happening again. The channeling of personal grief and the progressive efforts to effect reform makes STOP unique for many activist organizations. We consider ourself actionists. We are not willing to simply point blame.

We want to work together with all food safety stakeholders to produce the safest possible food to feed our families. We want to see good-industry citizens excel and the bad ones put out of business. We are pleased that FSIS is moving forward in implementing the adulteration policy announced over a year ago.

STOP has supported closing this food safety loophole since 1998, and we appreciate the agency's efforts to strengthen the random sampling program to

incorporate non-intact meat. The public strongly supports testing for E. coli 0157. And I've got in this bag several hundred petitions from consumers who just want to weigh in with the agency their commitment to 0157. I'd be happier going back tonight; my suitcase will be lighter.

The public really strongly supports, as I said, testing for E. coli 0157. STOP knows from thousands of phone calls that we've received on our hotline that the public mistakenly believes that the government is conducting now routine testing for E. coli 0157 in meat plants.

They equate E. coli, generic E. coli, that they read or hear about as the government's new scientific inspection system, with the pathogenic strain. They think they are one in the same. We are also pleased that the agency wants to ensure that this policy is implemented based on the best available information and in a manner that will best protect public health.

We also wish to commend FSIS for expanding on the questions listed in the White Paper to lead and lend more balance to the discussion that we're having today. Some of the points that have been clearly articulated here and in the White Paper is that, number one, E. coli 0157:H7 is not as rare as previously thought in live animals, up to 50 percent, and in ground beef, 40 percent positive under the new sampling technique; that is,

Centers of Disease Control and Prevention has nearly quadrupled the estimate of illnesses from E. coli 0157; that the infectious doses for this organism is extremely low, fewer than 10 organisms; and that E. coli can produce severe and fatal consequences, particularly in children and the elderly.

I'd like to share with you a few examples of these consequences. This is Damian Piercing, and he contracted E. coli 0157:H7 at a Boy Scout camping trip in 1992. He had over seven surgeries, four of them heart surgeries. He no longer -- the lining of his heart had to be removed. His kidneys failed, and his intestines were punctured. He had to learn to stand, sit, and eat again.

Seven years after his illness, his mother says this disease is never over. Damian was hospitalized last year three times with small bowel obstructions due to abdominal scarring. At one point in Damian's illness, they did have to remove his entire colon.

And I don't know if you can see. It's kind of hard to see, but this red line here is where he was cut open. And they had to remove -- and had to go over his intestines inch-by-inch looking for punctures. And the doctors cannot pack them back -- the intestines back in the way God originally put them in our bodies. And he suffers problems today.

Now, this is Brianne Kyner (phonetic). She was one of the children hospitalized in the Jack-in-the-Box outbreak in 1993. She spent two months in intensive care and nearly six weeks in a coma. Her hospitalization lasted nearly six months. She suffered from thousands of seizures and three strokes. Every organ of her body failed. She had swelled so much that they could not close -- that's her abdomen -- they could not close her up. She had to be left open while the swelling went down, so that they could finally stitch her.

This is little Amy Ermo. This picture was taken just two weeks before she became sick, just a beautiful little girl, as you can see. Amy lives near Sacramento, California. She got sick two weeks later and was put in the hospital and in intensive care. She had to receive two surgeries, and she was put on dialysis.

These pictures were taken when she was not at the full height of her illness. Her parents were just too concerned at that point that they couldn't even bear to -- they didn't want to remember Amy this way if she did die. Amy has survived, but they are very anxious about, particularly AIDS test, coming back.

These are the lucky ones. What I just showed you are the lucky ones. Their parents feel very lucky and extremely blessed, because they still have their children. Little Scotty Hinkley, he died, three years old. Kevin Scott died in Seattle, Washington, a year

after Jack-in-the-Box when his parents thought it was safe again to eat a hamburger.

All of these children, by the way, are hamburger E. coli 0157:H7 accidents. This is Mrs. Metts. She was 88 years old, very active, very fit until she ate a hamburger with her daughter at home. This is Lauren Rudolph. Lauren was the first to die in the Jack-in-the-Box outbreak. And last of all, that's my son. My son, Alex, didn't make it home either. So what I have left are pictures like this and a death certificate telling me he died of hemolytic uremic syndrome.

At a time when we're seeing a higher prevalence of 0157 and higher numbers of E. coli illnesses should be recognized as a time for implementing more, not less, aggressive plans in combatting the problem. It is a time for government and the beef industry to acknowledge that 0157 is, in fact, a hazard reasonably likely to occur and that HACCP plans from slaughter through processing must address this hazard.

And I want to emphasize that each operation must include 0157 as a hazard likely to occur. A single accounting for it, either in the front or a back end of the beef production process, won't suffice. It's simply not enough to address E. coli 0157:H7 as a hazard reasonably likely to occur only at a single point during the slaughtering and processing of beef.

There are simply too many steps along the continuum where cross-contamination can occur. It is time for FSIS to broaden its pathogen reduction program and include a comprehensive government testing program for 0157 within its inspection program. The testing program should be scientifically and statistically designed to detect and prevent E. coli 0157-contaminated beef from entering the marketplace.

Once a comprehensive testing program were implemented at production, the need for the random sampling program could be reviewed or modified, for instance, to only test at the retail level. The current random sampling program by FSIS was developed to encourage voluntary testing by industry.

It had an added benefit of being able to detect and prevent a limited number of adulterated product from entering the marketplace, and it also had, again, a limited effect on identifying contaminated product at the retail level and effecting a recall. I use the term limited, because the size of the sampling program is minute when you compare it to the tonnage of ground beef produced each year.

STOP has always encouraged voluntary testing by industry, but the public needs to have government-conducted inspection programs for pathogens. Now, the industry's proposal of testing one out of every 300 carcasses to replace or reduce the need for FSIS's

random sampling program of non-intact beef does not provide adequate protection to the public.

Testing results would not be available to the public. And we certainly could not agree to a modified Directive 10,010.1 to pass over or exempt production lots all the way from slaughter through retail from FSIS's Random Sampling Program simply because one out of 300 carcasses had been swiped by a plant employee. We will not find that acceptable.

STOP has currently urged industry to develop and implement additional testing programs of their own. Supplemental approaches should always be considered as an alternative, but they very rarely are. STOP has been alarmed at the way industry wants government to horse trade on food safety issues. This "I'll give you this if you give me that" is not in the public's interest.

The objective should be not to maintain the status quo, but rather to raise it. Let's see what the outcome is with both programs. Industry, do your carcass testing and, government, do the current random sampling. And let's see what the results will be.

In the Federal Register notice, there were a number of questions posed. And I'm just going to briefly answer a couple of them. Question two asked a series of questions regarding a redesign of the current program. And as we stated earlier, we support 0157 testing as part

of government's HACCP-based inspection program within each plant.

Until that time, FSIS should increase the number of samples taken to allow for testing of non-intact product, in addition to the current testing of ground beef. What we're saying here is that the current size of the sampling program need to be expanded to account and adjust for non-intact, this new definition. FSIS's current policy requiring 15 consecutive negative samples, following a positive one, is a -- positive result is a sound one.

So far, follow-up tests have yielded 11 positives. In Fiscal Year 1998, one plant tested positive two consecutive times and another tested positive three consecutive times. And in Fiscal Year 1999, one plant tested positive three times within two months. Clearly, these plants were not operating as they should. Consecutive 0157 testing identified that there were systemic flaws which posed a serious threat to public health.

There is no question that consecutive testing provided an incentive for these plants to address their food safety problems. STOP strongly encourages the continuation of the consecutive testing policy. We are interested in FSIS's plans to conduct some sampling to assess the feasibility of identifying E. coli 0157:H7 on carcasses and establishing a routine, and that an

agency-directed sampling program -- and then you had the words "To supplement or replace FSIS's ongoing ground beef testing."

We're pleased to see the term "supplement." But we're distressed to see the term "replaced" at this premature time. For the same reasons that we stated earlier about the industry's carcass-testing proposal, we urge FSIS to do comparative studies including either/or and both scenarios before modifying or replacing its ground beef testing.

Really, again, I commend the agency for looking at both sides of the issue, looking at what if we do less and what if we do more? And that's the way I think you can make a really good decision on what type of policy and how to implement it. Testing at all levels of the production and distribution would have the most powerful effect in encouraging the regulated industry to institute pathogen reduction interventions, specifically for 0157.

In question three regarding the salmonella and E. coli 0157 outcomes within plants, we are not aware of any studies showing the correlation between the presence of salmonella and E. coli 0157:H7. So we feel research is needed before one can make any sort of definitive comment on whether FSIS should target its sampling program to plants with poor generic E. coli and salmonella results.

STOP does support improved efforts to detect 0157 in food, and common sense suggests that these plants would be a good place to target sampling efforts. We still argue that the best strategy would be for FSIS to test for 0157 as part of a HACCP-based inspection system. And then, at question five is that STOP urges FSIS to treat blade-tenderized beefsteaks and roasts the same as other non-intact product.

Research presented in March and today by KSU does show that 3 to 4 percent of surface contamination was transferred into the interior of the muscle during a blade-tenderization process. Because of the infectious dosage of 0157 and the possibility of life-threatening illness, we see no reason that these products should be treated any differently than any other non-intact products.

After determining in fall of 1999 that there is insufficient information regarding the hygienic processing of muscle systems to narrow the scope of products affected by the E. coli 0157:H7 policy, FSIS urged the industry to label their intact and non-intact primal and sub-primal cuts with appropriate cooking statements from the 1999 Food Code.

We, at STOP, would be interested in knowing to what extent industry followed FSIS's advice. We are, lastly, we are aware of the budgetary concerns on the part of FSIS in implementing additional or supplemental

programs. You're having trouble meeting the current program. But the public cannot and will not accept this as an excuse for not doing everything possible to protect them from harmful pathogens in their beef.

If this project cannot move forward because of budgetary problems and/or inspection shortages, we want FSIS to go back to the well. We also call on industry here and those absent to lobby the administration, Congress, and Secretary Glickman for additional funding to fill inspector shortages. Consumers want government-inspected meat and poultry. And we will aggressively challenge any movement toward company self-inspection.

Alternative inspection plans, even interim inspection plans, are not acceptable. And we would challenge the mark of inspection. STOP plans to submit more extensive comments, written comments, by the April deadline. But I would like to express, again, our appreciate at being invited to present our views here today. You saw just a few examples of the horrible toll that E. coli 0157:H7 can take on its victims.

I don't have a little boy waiting for me anymore at the end of my trip. For many people in this room, it's about protecting a job, a company, or even a whole industry. For us, at STOP, it's about protecting lives. And we're here today on our own time and our own dime to thank you at FSIS for working to strengthen

policies that will help prevent the tragedies you saw today. We beg you to move swiftly. Thank you.

MR. BILLY: Thank you very much. Are there any questions from the panel? Anyone else have any questions? No? Okay. Thank you very much, Nancy.

MS. DONLEY: You're welcome.

MR. BILLY: The last presenter will be Ms. Caroline Smith DeWaal. She's the director of Food Safety for the Center for Science in the Public Interest. And she's speaking this afternoon on behalf of the Safe Food Coalition.

MS. DeWAAL: Can you hear me? Okay. Thank you.

MR. BILLY: I can hear you fine.

MS. DeWAAL: Great. I just want to say thank you. I think Nancy and I both appreciate that at the end of this very long day where we've heard about five government presentations, five industry and academic presentations, one including numerous industry representatives, and even an international presentation that you saved a little room on your agenda to hear from consumers. We do appreciate that.

Good evening. I guess we're entering evening already. I'm speaking today on behalf of the Safe Food Coalition. And we represent numerous consumer organizations. Today, the Consumer Federation of America, National Consumers League, and Government

Accountability Project, as well as CSPI and others are endorsing this statement.

CSPI represents nearly 1 million consumers in the U.S. and Canada on food safety and nutrition issues. My remarks probably won't come as a surprise to most people here. Pathogen testing is an essential weapon in the government's arsenal against foodborne illness. Testing at many levels is needed to maximize consumer and public health protections.

Microbial testing at multiple levels was built into the pathogen reduction and HACCP regulation which utilizes both industry and government testing. USDA requires all beef, pork, and poultry slaughter plants to test their own products for generic E. coli. Government tests these slaughter operations and some beef, pork, and poultry processors for salmonella.

As this data shows, this program has had a marked improvement on salmonella contamination levels across the meat and poultry industry. And I'm giving you just some example here given out recently by Secretary Glickman at a meeting in Washington. But we see reductions from one quarter to almost 50 percent of salmonella in some meat and poultry -- portions of the meat and poultry industry.

Unfortunately, the lessons of the last few years have also shown us that control of this one hazard does not result in control of all hazards. In fact,

recalls for other hazards in meat continued at a high level last year. And this is just data for 1999.

We had Listeria-contaminated meat and poultry products recalled 33 times and E. coli-contaminated meat and poultry products were recalled 10 times. So, though now we're seeing improvements in some areas, clearly more work can be done.

The lesson of HACCP implementation -- in addition, I just want to make this final note. I'm sure we'll all remember the fact that 1998 and 1999 also gave us one of the most deadly outbreaks of foodborne illness when Listeria monocytogenes in a processed meat product sickened 100 people and killed 21.

The lessons of HACCP implementation show clearly that systemic pathogen testing of meat and poultry products is an essential adjunct to HACCP systems. Microbial testing provides both the food industry and the government an objective measure for evaluating HACCP's effectiveness. Recent improvements in salmonella-contamination rates show that testing and performance standards also provide incentive for meat and poultry processors to improve.

It is time to expand this testing to cover more products and more pathogens, especially those that trigger adulteration standards. There is another model for pathogen testing that differs from the model used in the HACCP regulation. It is being used for two

pathogens, Listeria monocytogenes and E. coli 0157:H7. And this model is marked by the fact that it utilizes limited random government testing for the pathogen of concern.

And when the pathogen is found in the food, according to this government test, the products are subject to a voluntary recall. This model has been used by the government to address serious hazards in certain food products which the industry hasn't been able to adequately control.

The E. coli 0157:H7 Adulteration Policy and Ground Beef Testing Program was announced in 1994, over one year after a major outbreak -- actually, I'll go back up -- after a major outbreak sickened over 700 consumers and was linked to the deaths of four children. In situations like this, the government must act promptly to address serious safety concerns and to restore consumer confidence.

However, many years later -- six years later, actually, there are many criticisms that can be leveled at this limited approach to testing. It is reactive, instead of prevention-oriented. The number of tests conducted each year is highly inadequate to address the problem. It's like throwing darts at -- the program is not systemic and provides inadequate coverage.

It's like throwing darts at a dart board. Although the government hits the target occasionally,

it's clearly missing a lot of the problem. The same is true for the government's Listeria testing program currently in place for ready-to-eat meat and poultry products. And CSPI recently petitioned USDA to change this program.

And I believe that our analysis here is applicable to both hazards. We support changing the E. coli testing system to one that is more systematic, that is more prevention-oriented, and that gives consumers greater assurance that it is actually catching the hazards in the food supply.

In 1994, Michael Taylor, the Administrator of the Food Safety and the Inspection Service, announced the E. coli policy that we have today. In his speech to the American Meat Institute annual conference, Taylor said, "In the case of the 0157:H7 in raw ground beef, the only satisfactory public health goal is to eliminate contamination.

"We must look for ways to reduce the likelihood that contaminated animals will enter the stream of commerce, the risk that any pathogenic bacteria present in the intestinal tract will contaminate meat during the slaughter process, and the potential for subsequent growth of any organism that may be present.

In short, technological innovation and production, slaughter, and processing must be harnessed

and applied aggressively if we're to move effectively towards our public health goal."

Taylor's words provide an important reminder of the challenges that continue to face the beef industry. We strongly support FSIS in expanding the adulteration policy to non-intact meat. This step is essential if all segments of the beef industry are to share responsibility for meeting these challenges.

Some things have changed, however, since the original testing program for E. coli 0157:H7 was announced. We now know that this hazard is much more prevalent in live animal than was assumed in 1994. Also, CDC's estimate of the annual burden of human illness has greatly increased. Obviously, previous efforts to control the pathogen have not worked.

It is time to bring this problem into the HACCP area. First, E. coli 0157:H7 should be considered a hazard reasonably likely to occur for beef slaughter and processing operations. And the hazard should be included in their HACCP plans. And they should institute technological controls to address it.

If a company has evidence indicating that E. coli is not a hazard for a particular type of cattle that it slaughters, the burden should be on the company to seek an exemption from the general policy and make supporting data available to FSIS. Second, government

testing for E. coli 0157:H7 should be continued and industry testing should be added.

When Mike Taylor announced the original policy on E. coli, consumers believed that government testing would provide greater incentives for the industry to test their own products. We were wrong. Unfortunately, as with Listeria, food lawyers advised the meat industry that it is better not to know if their product contains a deadly hazard.

The government must counter this hide-your-head-in-the-sand approach to product testing, product safety with the testing mandate. Microbial testing for E. coli 0157:H7 should achieve the following objectives: First and foremost, it should verify that the plants' HACCP systems are effective in controlling E. coli 0157:H7 and also identify problems so that corrective actions can be taken.

Second, the testing should improve the likelihood that contaminated products are detected and either further processed to eliminate pathogens, recalled if the product is already in commerce, or destroyed. It can also provide incentives for companies to implement effective interventions against E. coli both on the farm and during the slaughter process and to conduct their own thorough testing.

Finally, testing can fulfill the -- facilitate the acquisition on data on such questions as the seasonal

and geographic prevalence of the pathogen, the effectiveness of various intervention measures implemented by the industry, and the relative utility of carcass sampling versus bin sampling versus finished, raw product sampling.

Neither government sampling nor industry sampling alone would achieve these objectives. Instead, FSIS should develop a comprehensive E. coli 0157:H7 strategy that includes both systematic microbial testing by both the government and the industry.

USDA should consider the following element for its sample program:

Mandatory industry testing of carcasses and trimmings in slaughter houses and grinding operations.

Slaughter houses and grinding operations should be required to test both carcasses and trimmings for the presence of 0157:H7 at least until sufficient data exists to demonstrate that thorough carcass sampling obviates the need to test the trimmings. It may be appropriate to allow plants that conduct more frequent carcass testing to reduce the testing of trimmings.

As part of its rule-making, FSIS should consult an independent expert body regarding how lot size for carcass testing should be determined. The number one in 300 is a number that the industry came up with for carcass sampling. But it really appears to have come out of the air. And we need a better estimate of what the

appropriate sampling frequency should be and how the lot size should be determined, given that sampling frequency.

Consumer protection must be maximized in determining both lot size and the sampling frequency. And actual carcasses that are sampled should be held pending test results.

Any positive tests should trigger appropriate corrective actions, including step-upped sampling in the plant, and repeated positives should trigger revalidation of interventions and possibly changes in the slaughter processes.

All positives should be reported to FSIS immediately, and the agency should take appropriate action, including asking for product recalls.

Second, random FSIS testing based on risk:

FSIS should significantly increase the number of E. coli 0157:H7 tests conducted annually and should establish a protocol for conducting the tests in order to evaluate a plant's process controls.

FSIS should target establishments that do not conduct their own testing and/or do not employ validated interventions initially. But once the entire industry is required to perform its own testing, FSIS sampling should be focused on those plants and raw meat products that historically have posed the greatest risk.

Until sufficient 0157:H7 data are obtained from the plants, the results from the salmonella and generic

E. coli testing can be useful in determining where to sample. Once industry testing is fully implemented, all plants should be subject to random government testing. In a pooled system, similar to the one in use today, for the salmonella testing program.

The trade-offs currently reflected in the FSIS Directive 10,010.1 should be eliminated. As all beef slaughter and most processing plants would be required to conduct systemic testing. FSIS's program should be dynamic, and not static. And the agency should alter its testing program based on the data derived from both the government and the industry testing.

The focus should be on identifying the riskiest plants and products and taking the appropriate measures to assure their safety. Now, in my talk, I also addressed a bunch of other issues posed by USDA, but I'm going to spare you all. And you can read it if you want in my written text.

So in conclusion, I just want to say that USDA should take responsibility to devise a new testing policy for E. coli 0157:H7 that is more systemic, more prevention-oriented, and one that gives the consumers greater assurance that it is actually catching the hazards in the food supply. In addition, the new policy should utilize the lessons learned from HACCP implementation, including the importance of testing at several levels to maximize public health protections.

Combining both industry and government testing for E. coli 0157:H7 would significantly improve consumer protection from this deadly bacteria. It's important that FSIS bring the E. coli policy into the HACCP era. First, E. coli has to be considered a hazard reasonably likely to occur. With the new data, especially on prevalence in the live cattle, this is very important.

And it's also the trigger that will mandate -- that the industry's actually implement the interventions that appear to be available to control it. And industry testing should be mandated to verify that the controls are working to eliminate the hazards. The data we saw from the industry coalition today was quite striking in providing evidence for how testing can be used to verify process control.

Finally, as FSIS modernizes this policy, it also should mandate pathogen testing in other areas such as listeria in ready-to-eat meat and poultry products and -- capturvacture (sp) in poultry products. These are the next logical steps to incorporate the science of microbiology in order to modernize FSIS's regulatory program and to improve food safety.

These were the promises made to us in 1994. And it is time to move forward on them. These steps are essential if the government and the industry want to continue the work that was begun in 1994 to improve

consumer confidence in meat and poultry products. Thank you.

MR. BILLY: Okay. Thank you. Any questions from the panel? Other questions. Yes, Kim?

MS. RICE: Kim Rice, American Meat Institute. It's not a question. I just want to clarify for the record that the one in 300 frequency that the industry used -- the beef industry coalition used was based not only on the USDA sampling frequency for generic E. coli and salmonella or -- excuse me -- for generic E. coli, it was also based on a outside recommendation from a testing laboratory based on using already used combo sampling programs through the fast-food customers that the sampling at the rate of one in 300 was as -- the same, basically, as the combo sampling that's used by the fast-food customers. So it wasn't just pulled out of the air.

MR. BILLY: Other questions? Okay. Thank you very much. Oh, yes, Jim?

DR. MARSDEN: Yes.

MR. BILLY: State your name, Jim.

DR. MARSDEN: Jim Marsden, American Meat Institute. You're advocating many tames more testing for pathogen and it does still occur in the low incidence rate in natural product. So obviously, your desire is to improve public health, not just simply to test and reduce the numbers in the product. But I think the ultimate measure here is the public health hazard.

If you take an analogy and you look at the numbers that you've gotten from Sec. Glickman, a 25 percent reduction on salmonella and 33 percent in ground beef and a big percentage in -- and you compare that to the CDC illness data, it leads me to at least question whether more testing leads to a better health out lay. I'd like to hear your opinion.

MS. DeWAAL: I'm a little confused. What exact comparison are you making with the CDC figures?

DR. MARSDEN: Well, their estimates on illness, in particularly.

MS. DeWAAL: Well, the estimates on illness were revised recently. Are you interpreting that as an increase? Are you using the FoodNet data? What data set are we working from for CDC? The CDC figures for illness are over 70,000 illnesses a year. I believe it's 73,000 from E. coli 0157:H7, the vast majority of which are from beef products and the highest, clearly 55 percent, as I saw, were from ground beef.

So this is a major contributor to a public health problem that -- and testing of ground beef and testing of beef carcasses would clearly help to maximize consumer protections in this area. So I guess I'm concerned -- I'm confused about which CDC data you were trying to rely on in your question.

DR. MARSDEN: Maybe, I should ask for clarification of some of the data. I can't even -- find

it -- when you look at the most recent estimates, those estimates have --

MS. WACHSMUTH: No, they've gone down.

MR. BILLY: Identify yourself.

MS. WACHSMUTH: Kay Wachsmuth, FSIS. The last report from FoodNet indicated a downward trend. That was the '98 report; '99 hasn't come out yet. But I'm told if you look at the original five sites, it might still be that way for campobacteriosis (sp) and salmonellosis.

It's not dramatic, but I think Mark brought attention to the fact in his presentation that, in effect, some place in the chain may not have an exact causal effect on the outcome. The 25 percent reduction in the products may mean an increase in risk, but it might not be a 25 percent risk. If Mark wants to --

DR. POWELL: That's right. And I just wanted to try and clarify, I think, some of the confusion. Since Dr. Olsen's not here, I'll pretend to speak for CDC. Our numbers were very consistent with CDC's estimates. And the increase in the reported estimate of the number of illnesses nationwide, a large majority of that increase is due to CDC capturing the 90 percent, roughly, of cases that result in less severe health outcomes, non-bloody diarrhea for which the patient does not seek medical care.

And so if you look at their case control studies, there's only been two, but the idealogic

fraction estimated from those two case control studies, the proportion of illnesses attributable to ground beef appears to be in decline from the initial case control study to the second case control study, which has not yet been firmly reported, but has been presented at various scientific conferences.

So our best estimate, based on both the case control study and the more recent outbreak data is that currently somewhere between 16 and 40 percent of the total can be attributed to ground beef, but our most likely estimate was in that range which is consistent with the most recent case control study is that it's on the order of 20 percent.

MS. WACHSMUTH: The only data that you can actually use to say something's gone up or down is data that have been collected in the same way over a period of time. The FoodNet data are the only data that can address whether it's gone up or down. And they are showing a slight downward trend. It think it's very confusing, the new burden of disease document.

It makes it look like something's changed. But what's changed is just the way that CDC's been able to gather more data and different data and look at it in that document, that study. But the FoodNet data, over time, are the only trend data we have. And I think Sonja's slide for 0157 specifically didn't show much of a change one way or the other over the three years.

And HUS seemed to be about the same the last two years. But the salmonella and camperia (sp) they seem to be coming down.

MS. DeWAAL: I believe she did have a slide, though, and it may have been the outbreak data where it showed that the outbreak -- this 16 percent doesn't jive with one of the slides she had, which showed that about 55 percent of the -- probably the illnesses linked to outbreaks are from ground beef and another ten percent from other beef products.

SPEAKER: And that's the percent of reported outbreaks the likely vehicle identified for reported outbreaks dating back to '83 or '82, I believe.

MS. DeWAAL: And I'll be certainly interested to see your rationale for having that number at 16 percent in the risk assessment. But I'm sure that will be interesting reading.

MR. BILLY: Are there other questions? Okay. Thank you, Caroline.

MS. DeWAAL: Thank you, Tom.

MR. BILLY: We have the room till six o'clock, and there are five people that have identified their desire to make some comments. I want to ask all of them to keep their comments as brief as possible. The first person is Steven Grover, who is with the National Restaurant Association. Hi, Steve.

MR. GROVER: Hi, Tom. I'll make my comments brief. Can everybody hear? I'm pretty loud. I only have a couple of points to make. The National Restaurant Association represents about 40,000 independent corporations which represent about 200,000 restaurants. There are about one million restaurants in the country today. We're a major consumer of ground beef and ground beef products.

We encourage FSIS to work closely with the beef industry and consumers such as ourselves to focus on a science-based prevention of this pathogen. You need to keep in mind bacteria are a normal part of most raw foods, including ground beef.

No matter how much we wish for, simple silver-bullet solutions, they are just not going to work if they are not scientifically based or statistically valid. No matter how many samples we take for low-level pathogens, I don't think we're ever going to take enough to give us the assurance of food safety that we would like to see.

Quite frankly, I think we need to shift focus to a more proactive approach where we implement pathogen control programs and we use non-pathogenic organisms to assure the statistical validity of the program. I think the science is clear on that that low-level pathogens and, quite frankly, I think you confuse the subject when

you start talking about salmonella, listeria, and E. coli.

These are all unique pathogens. They have unique problems. There's unique problems associated with it. And when you talk about it together, you confuse the entire topic. We're talking about E. coli here today. We're talking about a low-level pathogen. It doesn't happen. It's hard to find. No matter what you say, no matter what the prevalence is, it's hard to find.

And I like the comment if you get zero, it doesn't tell you anything. If you get consistent zeroes and you get consistent zeroes on sampling, it's not telling you anything. It's not telling you anything about your process. It's not telling you anything about your controls. And it's not telling you how good a job you're doing, whether you're controlling it or not.

We need to find a better way to do it. It does the restaurant industry no good. It does consumers no good to simply find out that the ground beef we ate was contaminated with E. coli after the fact. Sampling, it's just not going to work. You can sample till the cows come home at all levels.

While the question of scientific basis and statistical justification for the current E. coli 0157:H7 policy is in debate, we strongly feel that FSIS should set science-based standards for the production of ground beef. But they must be scientifically-based and

statistically valid. I don't want there to be the same chance of finding E. coli as there is for hitting the lottery.

I mean, the bottom line is we need to make sure that what we're doing is right and making a marked improvement in the production of beef and the final end product. To solve this problem, we need to consider available science, quick fixes, and overly simplistic schemes must be avoided, no matter how attractive they may seem to be.

Again, pathogen testing of any low-level pathogen in food is no assurance of food safety to the restaurant industry. Finally, we encourage FSIS to continue working with the industry, continue working with consumers. And we dedicate our resources to helping solve this problem. We would like to see an improvement in the scientific understanding of this pathogen.

And we would like to work with FSIS and the beef industry to implement meaningful, proactive controls that prevent illnesses from happening, not just simply sampling. Thank you.

MR. BILLY: Okay. Thank you very much. The next commenter is Richard Wood from FACT.

MR. WOOD: Thank you for this opportunity to provide comments regarding FSIS policy on E. coli 0157:H7. I am Richard Wood, the Executive Director for Food Animal Concerns Trust, or FACT. We're a non-profit,

not-for-profit organization that advocates for the use of better farming practices to improve the safety of meat, milk, and eggs.

Our response is focused on issues related to the first and sixth questions that are before us. Question one, should E. coli 0157 be addressed in animal production HACCP plans? FACT supports the use of on-farm HACCP pathogen controls for all producers raising cattle for food consumption.

With the new data that the prevalence of 0157 is existing increasingly so in cattle, or at least it's being identified there in increasing numbers, the FSIS food safety system should be one that truly moves from farm to table. FACT believes that the stakes are too high to allow contaminated cattle to enter the slaughter house door and then to trust that everyone else, in all their technology, is vigilant from that point on to the dinner table.

FACT wants the farm and the feed lot to be an integral part of any FSIS pathogen control response. We need to move forward with the science that we have in hand now. A growing body of evidence exists in the literature regarding on-farm mitigation steps and this pathogen. These studies should form the basis of our on-farm response with other steps being added or modified as new studies warrant.

For example -- and I'll just mention a couple here -- research has shown that a transmission point for E. coli 0157 among cattle is the water troth where this pathogen can survive for at least four months in its sediments. Research has found that keeping water troths clean and regularly changing the water for cattle appears to be a most effective barrier to the disease.

Regarding feed, the NAMS (sp) cattle and feed evaluation found that that cattle receiving barley were two point seven five times more likely to have a positive sample than cattle receiving barley. Another study found significantly higher prevalence of 0157 in herds where corn sodage was fed. The use of propionic and acidic acids appear to inhibit growth of fecal E. coli. Adding such acids to feed stored outside should be evaluated as a mitigation step to protect cattle from 0157.

Regarding stress, research has demonstrated that stress may cause calves and full-grown cattle to become more susceptible to infection by 0157:H7 and that management measures to prevent stress may reduce the spread of infection. Continued research is needed, focusing on such issues as stress-related decrowding, transportation, and changes in diet. It's time for on-farm controls.

Question six, how effective are voluntary producer actions? FACT believes that while quality assurance programs are good producer education tools,

they are no substitute for a nationwide HACCP need required by the current situation with E. coli 0157:H7. Reliance on voluntary programs will not provide consumers with the confidence that the food is safe.

First, the voluntary programs are not accountable to the public through the regulatory agencies. There is no publicly available data as to the actual number of producers participating in these programs. And you meet with producer groups and talk and you find they speak in the terms of high numbers, but the NAMS (phonetic) cattle and feed evaluation said there was only 18 percent of cattle producers that participated in training programs.

And the numbers are probably higher now. But there's no public accountability in terms of participation. There's no public accountability as to what program requires regarding pathogens and whether or not the requirement are verified mitigating steps and other commodity groups where individual state programs exist has led to a patch work of diverse programs and requirements.

This situation gives consumers little confidence when faced with a production system where feed lots in several states ship to processing plants in other states. We can ill afford a patch work response to E. coli 0157:H7. FACT calls for a federally regulated, on-farm HACCP pathogen program.

This program would assure consumers that the federal response to E. coli 0157 involves all producers, that these producers are meeting the same standards of pathogen controls and that these standards are the same throughout the industry in the U.S. Thank you.

MR. BILLY: Okay. Thank you very much. Is Joe Maas here? Okay. The next speaker is Marty Holmes with the National Food Processors Association.

MR. HOLMES: Dave Bernard would be glad to know that they've added me to his staff.

MR. BILLY: Oh, sorry, Marty.

MR. HOLMES: That's all right. No problem. Dave and I need to go fishing anyway.

MR. BILLY: I just read off the sheet.

MR. HOLMES: That's all right. North American Meat Processors Association. I appreciate the time, and I will make this very brief. A couple of things that, as it relates to mechanical tenderizer -- blade-tenderized products, a couple of facts I wanted to reiterate. They were made previously, though.

The National Advisory Committee for microbiological criteria for foods suggested that the agency do a full risk assessment on these products before making any regulatory action on those products. No cases of 0157:H7 have been documented by CDC or anyone else regarding mechanically tenderized products that we're aware of. Caucuses are treated with a pathogen

intervention method they are further trimmed as primals and sub-primals before entering a blade tenderizer.

And so if there was a gross contamination on the surface, the odds of that ever reaching the blade tenderizer is very very small. And the only study that's been done so far is KSU's data which used a inoculated surface that's far beyond anything that we see in nature.

And because of these points and in the wake of that data, we respectfully request that the agency remove the policy clarification on mechanically tenderized and blade-tenderized products until they've done their own full risk assessment based on these facts that have been presented. Thank you.

MR. BILLY: Okay. Thank you, Marty. And that's Marty from the North American Meat Processors Association. The final speaker is Jimmie Keaton.

SPEAKER: Collis Powell is going to speak for him.

MR. BILLY: Is he? Okay.

SPEAKER: He had to leave to get his plane.

MR. BILLY: Okay. Collis Powell with the American Meat Science Association.

MR. POWELL: Yes, I'm executive director of American Meat Science Association. Jimmie Keaton of Texas A&M is our current past president. He had to catch that last flight back to Dallas so that he could get back to school to teach in the morning. What we wanted to

bring to you was, again, very similar to the testimony we presented a year ago.

Back in January of 1999, the American Meat Science Association, which consists of over 1,000 meat researchers from around the world, pulled together 35 of the best minds in statistics, microbiology, food microbiology, and brought them together to answer the questions of what can you do with microbial sampling in a beef food safety program? We wanted them to evaluate the concept of microbial sampling, say what you can, cannot do.

And they also looked at evaluating approaches to existing sample plans. And when you get 35 scientists in one room to get them to agree on anything is sometimes like pulling teeth. We managed to reach consensus on eight points. The points are highlighted in the complete report that we have, the scientific perspective on the role of microbiological testing in beef food safety programs.

I think it's appropriate that we end with this today, a reminder of what the scientists say is possible and is not possible with microbiological testing. Very quickly, the eights points. Number one, the main purpose of microbiological testing of foods is to validate and verify process control measures in the context of a properly implemented HACCP program.

Number two, effective microbiological testing programs are based on sound food safety objectives with defiable microbiological performance criteria. Number three, pathogen testing at any stage will not assure food safety. Number four, foodborne pathogens will not be detected consistently when they are not randomly distributed and/or they occur at a low incidence.

Number five, pathogens or other micro organisms at a low incidence cannot be used to assess process control. Number six, testing for appropriate non-pathogenic organisms will allow validation and verification of process control systems designed to improve food safety. Number seven, declaration of a foodborne pathogen as an adulterant in raw products discourages testing for that pathogen.

It leads to a false sense of security among consumers. It discourages evaluation of potential control measures, and it encourages the inappropriate use of microbiological testing. And finally, number eight, microbiological testing of foods in production is important, but is only a part of the overall strategy for controlling food safety. And they suggested education concerning proper handling and cooking is essential. Thank you.

MR. BILLY: Okay. Thank you very much. I'd like to thank all of you for your participation. I know this has been a long and somewhat arguous process. On

the other hand, it's a very critical subject of importance to everyone that's here. We very much appreciate all the material that's been presented by the various speakers.

We also appreciate the participation of those of you in the audience. We will very carefully consider all of this input and weigh it as we move forward to develop a revised White Paper, which as I indicated in my opening remarks, we plan to present to the National Advisory Committee for Meat and Poultry Inspection -- I believe, the meeting is in May.

So we look forward to that, and hopefully many of you will participate as part of that process, as well. So, again, thank you all very much.

(Whereupon, the meeting was adjourned at 6:00 p.m.)

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