UNITED STATES DEPARTMENT OF AGRICULTURE

FOOD SAFETY AND INSPECTION SERVICE

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ADVANCES IN POST-HARVEST INTERVENTIONS
TO REDUCE *SALMONELLA* IN POULTRY

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February 23, 2006
9:00 a.m.

The Loudermilk Center
Atlanta, Georgia

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Daniel Engeljohn

1 P-R-O-C-E-E-D-I-N-G-S

2 (9:00 a.m.)

3 DR. ENGELJOHN: Good morning. This is the
4 second in a series of meetings that we're having on
5 the control of *Salmonella* in raw products, with this
6 particular focus on poultry, and broilers specifically
7 today.
8 Just a few housekeeping issues before we
9 get started. We are net-casting the presentation
10 today so that individuals who are not able to attend
11 can at least hear the presentations and see the
12 PowerPoint presentations. And then our intention is
13 to make available copies by CD and other formats to
14 anyone who would like a copy. We're going to make
15 copies available to all the establishments that we
16 regulate, but, certainly, we'll make the information
17 available to anyone else who requests it.
18 But as we get started today -- we do have
19 28 speakers over the course of the day-and-a-half that
20 you're here, and that's intentional. We have a lot of
21 information we want to ensure that you have access to,
22 and so they're rather short presentations, and we're

1 going to hold the speakers to their time limits.
2 But for other issues related to those of
3 you here in the room, the restrooms are outside the
4 door to the left. There is no refreshment here in
5 this building. So if you go out the building to the
6 right to the United Way building just directly
7 opposite of this building, there are food and beverage
8 facilities there. And then if you hang a left out of
9 this building and go back up Auburn, there are a
10 number of food court choices up the street there.
11 With that, I think we'll get started.
12 Today we will have opportunity for questions and
13 answers from the audience.
14 We are transcribing the meeting so that
15 all the information from this meeting is available to
16 the public. And so I do ask that if you have a
17 question, you queue up in front of this microphone in
18 the center of the room at the appropriate time when we
19 have questions and answers available and that you give
20 your name and the association that you represent so
21 that we can get that into the official record. And
22 then we'll make every attempt to answer your questions

1 today. And if not, we will find another format to be
2 able to answer questions that are raised.
3 (Pause.)
4 DR. ENGELJOHN: They're not able to hear
5 us on the phone lines. So we're hoping that we can
6 get that corrected and you'll let us know if you hear
7 differently. And if you can't hear us, be sure to let
8 us know, as well.
9 I do want to get us started. Dr. Richard
10 Raymond, the Under Secretary of Agriculture for Food
11 Safety, will be our first presenter.
12 Dr. Raymond was appointed as the under
13 secretary on July 18, 2005, and he's responsible for
14 overseeing the policies and programs of the Food
15 Safety and Inspection Service. And he chairs the U.
16 S. Code Codex Steering Committee, which provides
17 guidance to U. S. delegations to the Codex
18 Alimentarius Commission. He has extensive experience
19 in developing and implementing policies and programs
20 designed to improve public health.
21 Prior to joining USDA, Dr. Raymond served
22 as the director of the Nebraska Department of Health

1 and Human Services Regulation and Licensure Division,
2 where he oversaw regulatory programs involving
3 healthcare environmental issues. He also developed
4 several anti-bioterrorism initiatives and a statewide
5 healthcare alert system. Dr. Raymond also played a
6 major role in the development of local health
7 districts that serve Nebraska's 93 counties.
8 Please welcome Dr. Raymond.
9 (Applause.)
10 DR. RAYMOND: Thanks, Dan.
11 Before I start, let me just speak into
12 this microphone and see if the telephones pick it up,
13 just in case that solves our problem.
14 (Pause.)
15 DR. RAYMOND: We're doing a mic check for
16 the telephones.
17 (Pause.)
18 DR. RAYMOND: Maybe they all left.
19 (Pause.)
20 DR. RAYMOND: We can't hear them? Okay.
21 They can't hear us, and we can't hear them.
22 Well, good morning, everybody. And thank

1 you for coming to this very important meeting to
2 discuss advances in post-harvest reduction of
3 *Salmonella* --
4 (Pause.)
5 DR. RAYMOND: They're hearing this now?
6 Okay. Great.
7 -- in poultry. You don't know how good it
8 feels to come to a group which for the most part has
9 had nothing to do with shipping hotel rack veal to
10 Japan.
11 (Laughter.)
12 DR. RAYMOND: It's nice to have a
13 different venue and talk about *Salmonella* for a couple
14 of days. And then we'll go back and talk about veal,
15 I'm sure.
16 (Laughter.)
17 DR. RAYMOND: As most of you know, I hail
18 from Nebraska, as do several other people that have
19 followed the Secretary to the USDA for the second term
20 of the Bush administration. And some of us were back
21 home a few weeks ago and gave a little talk.
22 We were on a panel -- the three of us.

1 And when we opened up questions and answers, the
2 reporter said, What would you like people to say as
3 they walk by your casket about your time in public
4 service? And the first individual said, I would want
5 them to say I worked long, hard hours and was totally
6 committed and dedicated. The second person would say,
7 I would want them to say, "He was an honest man."
8 And it was my turn, and I said, I feel I
9 must apologize a little bit; if I had a more time to
10 think about this than the Secretary and the other
11 gentleman, well, what I would want them to say is, I
12 think he's still breathing.
13 (Laughter.)
14 DR. RAYMOND: So after the last month of
15 dealing with hotel racks, we are still breathing, and
16 it's time to move on to another subject. So today and
17 tomorrow, we're going to dedicate our time to
18 discussing new research and new insights and then
19 learn from practical experience on how we can reduce
20 this prevalence of *Salmonella* post-harvest. And I
21 also hope you do some networking and exchange best
22 practices, which I know the industry is looking at

1 very seriously, in helping each other out to control
2 this problem.
3 Everybody needs to understand that there's
4 going to be changes in how the Office of Food Safety
5 and the Food Safety and Inspection Service approach
6 this important issue of reducing *Salmonella*.
7 You probably heard in 1996, when the rules
8 were published, that it was the goal of FSIS to lower
9 *Salmonella* rates. I don't think that ever happened.
10 You're hearing it again, but it's a new world, we've
11 got a new Administrator, a new Deputy Administrator, a
12 new Under Secretary and a new Secretary, and we
13 believe strongly that this is going to happen this
14 time. And we're going to tell you how, and we're
15 going to tell you why.
16 So just accept that change is coming. And
17 be prepared to work with us, not against us, and we
18 can all make this happen together. Your participation
19 particular today and tomorrow but, also, in the future
20 and in the past is very critical to us at FSIS to
21 develop these best practices to combat *Salmonella*.
22 We, the Office of Food Safety and FSIS,

1 have been at the forefront of the battle to reduce the
2 prevalence of foodborne pathogens in meat, poultry and
3 egg products, and we've done that through
4 communication, cooperation and collaboration with the
5 industry, with consumers and with scientists. We are
6 trying to be open and transparent as we develop these
7 new policies, and *Salmonella* will be no exception to
8 the trend that we have set under Dr. Masters'
9 leadership the last year-and-a-half or so.
10 As you have heard me say before -- those
11 of you who have heard me talk -- this is not new news.
12 My first day on the job, Secretary Johanns told me
13 this should be one of my top priorities: To get our
14 arms around *Salmonella* and lower those rates and
15 protect the public. And believe me, it is one of my
16 top priorities, and we will get this done.
17 We want to explain these slides to you in
18 just a little bit of detail. My job today is to
19 explain to you kind of the groundwork of where we're
20 going.
21 We have been surveying and doing stats in
22 over 100 large plants since 1998, seven years' of

1 experience of doing *Salmonella* stats. These slides --
2 the 20 percent line there is the performance standards
3 that have been established for carcasses. The middle
4 line is one-half of the performance standard, twice as
5 good as you have to be to pass the performance
6 standard.
7 What this slide shows you is in Category
8 1, we have 25 percent of our plants that have always
9 had their *Salmonella* stats come in below one-half of
10 the performance standard, six or fewer positives. We
11 know this can be done. Those plants show us that it
12 can be done.
13 The second category, the majority, 45
14 percent of the plants, have never exceeded the
15 performance standards in those seven years. At times
16 they've been below the 10 percent, at times, they've
17 been above the 10 percent, but they've never exceeded
18 the performance standards.
19 And then we have 30 percent of our plants,
20 who at times are above the performance standard and at
21 times are below the performance standard. What this
22 graph doesn't really truly represent is sometimes

1 they're way below the performance standard; sometimes
2 they're at 5 percent. So the squiggles should be
3 bigger, but that -- I apologize for that.
4 The point is almost two-thirds to three-
5 fourths of our plants performed better than the
6 performance standard in the last seven years. So to
7 the ones that say, "We can't do this; we can't get the
8 *Salmonella* rates down," I ask you to meet with those
9 plants on those bottom two squiggly curves. I think
10 they'll give you perhaps a different viewpoint.
11 I know that if I go to a restaurant
12 tonight and eat a chicken breast, I don't know which
13 plant it came from, and I don't know what the
14 performance sets were. Now, the consumers want to
15 know that. They want to know who's up there in that
16 top 30 percent, and they want to know who's down there
17 in that bottom 25 percent. And we haven't done that.
18 But it's one of those little carrots and sticks that
19 you're going to hear about that we may entertain if we
20 can't get some movement within the industry to get the
21 30 percent coming on down so they more closely mirror
22 the ones with the good performance sets.

1 Now, this is different than the plants.
2 This is the actual sets. I mentioned some of those
3 plants that are in the more variable categories will
4 occasionally have a set that gets the 5 percent or
5 lower.
6 And in actuality, in those same seven
7 years, in all of the sets that we performed, the
8 majority, over 50 percent, were at lower than one-half
9 the performance standard. More than half the time, it
10 was down to below 10 percent. We need to find out
11 what happened the other 49 percent of the time, when
12 they fell into Category 2 or Category 3, and it's not
13 consistent amongst the plants.
14 We had one plant that had a 30 percent
15 rate on the performance set. We did a food safety
16 analysis, and we worked with that plant to point out
17 areas that they should and could improve. In the next
18 set we went in, they had a 2 percent. Now, that is
19 dramatic. What we need to do is move the trends that
20 direction in all of our plants that are in the poultry
21 business. That's our goal. I firmly believe that we
22 can get that done.

1 I know that there are going to be some
2 nay-sayers out there. I've heard them. They say it
3 just can't be done. But you know what? We've got
4 consumers out there that say, You're not doing enough,
5 and you're not doing it fast enough. So this isn't a
6 plan that's going to please everybody. We have
7 listened to the consumers and we have listened to the
8 industry; we've listened to your representatives,
9 we've been in the small plants, and we've listened to
10 the scientists. And what we have tried to do is come
11 up with a plan that has a little bit in it for
12 everybody to get everybody on board.

13 The worst thing that could happen is to
14 have someone lay down on the tracks and say, We're not
15 going to go with this; we're going to stop it on the
16 Hill; we're going to stop it in the Secretary's
17 office, and you know what; we're going to keep doing
18 business like we're doing. And that just isn't good
19 enough.

20 So we have tried to accommodate. We've
21 tried to compromise. We've tried to bring enough
22 people to the table that we can sell this and we can

1 make it work. And again, we'll give you the details
2 later today on how it is going to work.
3 I had one plant owner -- honest to God --
4 who told me in a public meeting that it was the
5 consumers' fault because they didn't cook their
6 chicken and they didn't cook their turkey to the right
7 temperature and it was our fault because we didn't
8 educate the consumers. That's a plant owner.
9 Now, I recognize that is the minority, not
10 the majority, but there's still some outreach that
11 needs to be done, because those people can get very
12 vocal. But I think most or the people we have talked
13 to have the same goal, and that is to reduce the
14 *Salmonella* loads. And I think, together, we can get
15 this done.
16 Back in 1996 to 1998, I'm sure the beef
17 industry said the same thing that some people in the
18 poultry industry may be saying: It can't be done; we
19 can't get *E. Coli* rates down to less than one per one-
20 hundred thousand; it is impossible; it's inherent;
21 it's the nature of the beast; it's a part of the
22 plant. But they had two things happen to them that

1 made them roll up their sleeves and work together and
2 work with us, and they got it done because of the
3 Jack-in-the-Box and because *E. coli* 0157 was declared
4 an adulterant.
5 Now, *Salmonella*, as you know, has not been
6 declared an adulterant. And, thank God, we haven't
7 had a Jack-in-the-Box scenario -- or Chick-Fil-A -- or
8 something like that. We don't want that to happen.
9 But if it does, believe me, the pressure will be
10 intense on the industry. We would rather do it now
11 because it's the right thing to do.
12 We don't have a Jack-in-the-Box scenario
13 for *Salmonella* that I'm aware of, but we do have 14.5
14 people out of every hundred-thousand Americans get
15 sick with culture-proven *Salmonella* every year.
16 That's 42,000 people a year. The CDC estimates it's
17 actually 1.3 million people a year that get sick with
18 *Salmonella;* they just don't sick enough to get a stool
19 culture. And 400 people die with *Salmonella*. It's
20 just that we're used to it because those numbers have
21 been there a long time. We don't have a Jack-in-the-
22 Box.

1 I talked to a reporter yesterday who said,
2 I don't think this will work; I'm not hearing
3 anything -- I'm not hearing bells and whistles, and
4 nothing's happening. And I said, It's because we
5 haven't had a Jack-in-the-Box, but the industry is
6 going to make this happen; stay with us and watch our
7 progress. And I hope she -- hopefully, she'll write a
8 nice article about us.
9 These are positive regulatory samples for
10 *E. coli* over the last five years. The same -- this is
11 human illness in *E. coli* over the last seven or eight
12 years. This last one right here is 0.9. Healthy
13 people in 2010, the goal for the year 2010, is 1.0
14 persons per hundred-thousand. We reached that goal in
15 *E. coli* in 2004, six years ahead of schedule.
16 When it was up here at 2.5 and 2.4, that's
17 when the beef industry was saying, It just can't be
18 done; we can't get down to that; we've set an
19 unrealistic goal for us. But they did it, and I hope
20 we can all take notice of that. And I hope we can all
21 be motivated by what they were able to do.
22 *Listeria*? The same example, without the

1 Jack-in-the-Box. But their sample products have gone
2 down, and the human infection rates have gone down --
3 with *Listeria*. Now, we've got a few risk-based
4 initiatives we took on a couple of years ago when that
5 number popped up there that are going to keep the
6 *Listeria* numbers going down. And that's all good news
7 for the people that consume these ready-to-eat
8 products. We need to work on the chicken and turkey
9 products just a little bit, however.
10 We do believe that those experiences,
11 again, lend proof that this can be done. When the
12 industry works together with the scientists, with the
13 Agency and with consumers and shares best practices,
14 we know we can get this done. Now, we have a long way
15 to go, though.
16 The healthy people in 2010 goal is 6.8
17 infections per 100,000 people. We're at 14.5 people.
18 *E. coli* was at 2; their goal was 1, and they made it.
19 You're at 14.5, and the goal is 6.8. You can make
20 it. You can make it. It's going to take awhile, but
21 we've got six years to get to the healthy people for
22 2010.

1 What you're going to hear speaker after
2 speaker after speaker is the ways we have learned,
3 things that we have learned that are working and the
4 research that has been done. And you're going to hear
5 about our new rollout that we put on the web two days
6 ago that will be posted in the Federal Register, I
7 believe, on the 26th. You're going to see our new
8 stats for 2005.
9 2005 stats coming out in February? That's
10 unheard of. That's how this Agency looks at things
11 now days. We aren't going to wait a year to release
12 data; we're releasing them right now because they'll
13 show if we've got a problem.
14 And you'll hear how we're going to release
15 set data in a different fashion to not wait a year to
16 address issues. We've got the issues to address now.
17 I'll probably be gone in three years or less. I want
18 to see change while I'm here; I don't want to go back
19 home and say, I tried, but we just didn't get anything
20 done. So we want you to work with us.
21 In closing, I do want to say we do have a
22 strong system in place. I'm not saying the sky is

1 falling. We have a great system, but we can always
2 make it better. Any system that doesn't move forward
3 is a system moving backwards. You can't tread water
4 and try to maintain it. It just doesn't happen. The
5 bugs get smarter, and the bugs get more resistant.
6 And we need to work for that.
7 That's something that -- public health is
8 always changing. If we don't change as the bugs
9 change, we lose ground. A hundred years ago, the life
10 expectancy was 45 years when you were born in America.
11 This year, it's 75 years. That's 30 years we've
12 gained in life expectancy, and that's not because of
13 medical science; for the most part, it's public
14 health.
15 In 1906, one out of five coffins was
16 filled with the body of a child that never reached his
17 or her fifth birthday; they died of infectious
18 diseases. In the '40s, we invented penicillin -- I
19 shouldn't say we invented it -- we discovered
20 penicillin. Bacteria could now be attacked.
21 Pneumonia didn't kill little babies, and meningitis
22 didn't kill young children.

1 In 1955, the polio vaccine was licensed.
2 Kids no longer died from polio or were crippled for
3 the rest of their lives. They were not neurologically
4 impaired. Their parents could let them go swimming in
5 the swimming pools without worrying about polio. None
6 of us in this room, I don't believe, had children
7 before penicillin was discovered; there may be one or
8 two that had a child born before the polio vaccine was
9 discovered, and I won't ask you to raise your hand.
10 But, you know, we don't know what it was
11 like for parents a hundred years ago to have children,
12 realizing that children in your community died on a
13 regular basis; it was just an expected event. They
14 died of diarrhea, dysentery, enteritis, diphtheria,
15 smallpox and polio, things that we can now prevent.
16 Nobody would have thought 100 years ago
17 that we would have vaccines that would wipe out
18 childhood diseases. No one would have thought we
19 would have bullets that would kill bacteria. But
20 we've done those things. And so when you say, "I
21 don't think we can do better with *Salmonella*," suck it
22 up. We're going to do better with *Salmonella*.

1 We don't know what that magic bullet is
2 going to be, but one of the reasons kids now live past
3 their fifth birthdays and the life expectancy has gone
4 up is because -- we have those scientific inventions,
5 but we also have safer food, we have safer water, and
6 we have sewage treatment and disposal.
7 But now days, when a child dies of a
8 foodborne illness, it's a disaster. It's a
9 catastrophe. It was expected 100 years ago that it
10 would happen. Right now, it's a problem with product
11 or the handling of the product in the home or the
12 restaurant or some place along the road. And we an
13 and we must do better, because it is truly something
14 that is totally preventable.
15 We all have the same goal. And if we just
16 remember who we're working for here and if we just
17 remember who we're trying to protect, I'm pretty sure
18 we can get there together.
19 So once again, I thank you for attending
20 this conference; I know you're dedicated and committed
21 or you wouldn't be here. We look forward to a healthy
22 exchange over the next two days. Thank you very much.

1 (Applause.)
2 DR. ENGELJOHN: Now, I just want you to
3 know -- just so you don't think that I'm really lax in
4 my job -- I'm giving an exemption to Dr. Raymond and
5 Dr. Masters for how long their speeches take. So I'm
6 not holding them to their time lines, but, all you
7 other speakers, you other 26 speakers, I'm going to
8 hold you to it.
9 DR. RAYMOND: You didn't let me start
10 until ten after, Dan.
11 (Laughter.)
12 DR. ENGELJOHN: Well, we're going to move
13 on. And thank you very much, Dr. Raymond, for those
14 remarks.
15 Our next speaker is Dr. Barbara Masters.
16 Dr. Masters was named the Administrator of the Food
17 Safety and Inspection Service in August 2005. In this
18 position, she is responsible for leading FSIS in its
19 mission of protecting public health through food
20 safety and [inaudible due to failure of in-house PA
21 system].
22 Dr. Masters began her FSIS career as a

1 veterinary medical officer in 1989 near Hot Springs,
2 Arkansas, and has since held a variety of posts
3 throughout the Agency, both in the field and
4 headquarters. Since March of 2004, Dr. Masters served
5 as the Acting Administrator. And during that time,
6 she raised the scientific training investment in the
7 10,000-employee work force to a record $20 million, as
8 well as enhanced communications with both internal and
9 external audiences.
10 Please welcome Dr. Masters.
11 (Applause.)
12 DR. MASTERS: Thank you, Dan.
13 Good morning, all. I'm certainly pleased
14 to be here today to participate in this important
15 meeting.
16 I certainly want to thank our FSIS Office
17 of Policy, Program and Employee Development for
18 hosting this meeting. It's no small challenge to put
19 on a meeting of this magnitude, and it adds to the
20 challenge when they have to put it together in net-
21 cast. So I also want to thank those folks that are
22 working to do the net-cast portion of this meeting.

1 I think it adds to the value when we're
2 able to not only bring those of you that are
3 interested in being here but we're able to reach many
4 more of our stake holders that have an interest in
5 this topic when we're able to do the net-cast. And
6 we're also able to save it.
7 I talked to many of you after the pre-
8 harvest portion that said, There was just a few of
9 those presentations I would have loved to have shared
10 with many of my folks back home in the plant. So
11 we're hoping that we're able to add value to this
12 meeting by having the net-cast portion and also being
13 able to put it on the CDs for you to take back with
14 you so that you can show portions of this to your
15 folks back at home.
16 So we do believe we're reaching many more
17 of our stake holders by having this format. So thanks
18 to those of you that worked very hard to get this
19 meeting put together.
20 As you're aware, as an Agency, we've been
21 working on our farm-to-table approach to food safety.
22 While most of our regulatory authority lies in the

1 plants, we realize that food safety begins long before
2 the product reaches the processing or slaughter
3 establishments.
4 We've had so much information to share on
5 the topic of *Salmonella*. That's why we realized last
6 year we needed to approach the *Salmonella* topic with
7 two meetings. That's why we got together last year in
8 August and had our pre-harvest meeting in Athens.
9 And we had such a long and fruitful
10 discussion on *Salmonella* and the trends and the
11 research relative to *Salmonella* and the discussion
12 that we had on the pre-harvest topic. We discussed
13 controlling *Salmonella* to the maximum extent practical
14 and the impact that that control has at pre-harvest
15 and on the levels of *Salmonella* coming into the plant.
16 We're hopeful that at this meeting, we can
17 talk about the latest data and have discussion at the
18 in-plant level for combating *Salmonella*, because we
19 recognize most of you will use a combination of that
20 information for the solutions for combating
21 *Salmonella*. We want you to know that we have not
22 forgotten all the information that was shared at the

1 pre-harvest meeting and we're very soon going to be
2 issuing some compliance guidelines that will come out
3 of that pre-harvest meeting.
4 As we discussed in Athens, there are
5 things that can be done prior to that product reaching
6 the plant, but we also believe there are many things
7 that can be done at the in-plant level. Again, we
8 recognize most of you will do a combination of things
9 pre-harvest and at the in-plant level, but the bottom
10 line is we need to make sure that we're doing
11 everything practical to control the *Salmonella*
12 organism. It's up to you as an industry holistically
13 to take on that challenge.
14 As Dr. Raymond mentioned, we are aware
15 that individual plants have been and can control
16 *Salmonella* and have been meeting the performance
17 standards. We know this because we have data that
18 represents that plants have been consistently meeting
19 the performance standards.
20 In my opening remarks in Athens, I talked
21 about the fact that we would be using the model that
22 we used for *E. coli* 0157:H7 in the beef industry. Dr.

1 Raymond talked about it at a high level in his
2 presentation, and I want to get a little bit more into
3 detail, when we say we're using that model, what we
4 really mean by that.
5 We're using the *E. coli* model in the sense
6 that what we did for the beef industry is that -- we
7 conducted a risk assessment for *E. coli* 0157:H7.
8 Based on that risk assessment, we had all of these
9 beef establishments re-assess their own HACCP plan.
10 After they had re-assessed their HACCP plans, we went
11 out as an Agency and conducted food safety assessments
12 of those re-assessed HACCP plans. The results that we
13 found have been reductions in the positives in our
14 regulatory samplings, as well as reductions in
15 foodborne illness.
16 I think the significant point we need to
17 understand is that it was -- the industry taking on
18 the challenge to re-assess their food safety programs
19 is what we believe has really made the difference in
20 looking at the reductions in our positive regulatory
21 samples, as well as the reductions in foodborne
22 illness.

1 When we went out and verified for our food
2 safety assessments the changes in the programs, the
3 industry had taken on that challenge of re-assessing
4 their programs, and we saw significant differences in
5 those plants. That was the crux. The industry-wide
6 initiative of re-assessing their programs is when we
7 saw drastic changes industry wide. We believe that
8 the poultry industry can see similar changes if they
9 apply a comparable model.
10 The challenge that I have to you as the
11 poultry industry is to use the data that you're going
12 to gain at this meeting as well as the industry
13 information that we're going to share with you in the
14 form of compliance guidelines from the pre-harvest
15 meeting to significantly start decreasing the
16 prevalence of *Salmonella* in your plants. You do not
17 need to wait on us as an Agency to require you to re-
18 assess your HACCP program; you can re-assess your
19 HACCP program any time you desire.
20 You're going to have general information
21 coming to you at this meeting relative to best
22 practices, relative to lessons learned from ongoing

1 food safety assessments that we've done; you're going
2 to have information coming to you from the literature
3 reviews that we've done. You're going to have
4 specific plant information that we're going to be
5 providing to you.
6 You do not need to wait on this Agency to
7 conduct a food safety assessment. You can take on the
8 challenge to look at your own food safety systems and
9 make the necessary changes in the design of your food
10 safety programs. And we believe we're going to be
11 providing you the type of information to make the
12 necessary changes, based on the information at the
13 pre-harvest meeting and again at this meeting, to make
14 the changes in your program to control *Salmonella* in
15 your establishment.
16 Please don't misunderstand me. We do
17 understand that there are different ways to control
18 *Salmonella*. We understand that plants are using a
19 variety of ways to control *Salmonella* and that there
20 are a lot of different ideas and different approaches
21 to controlling *Salmonella*.
22 Dan mentioned there's well over 20

1 speakers at this meeting; that's because we recognize
2 there's no one-size-fits-all. There's not one magic
3 way to say, Ta-da, we're going to control *Salmonella*
4 in the plant this way. We want you to look at what
5 works best in your plant environment and apply that in
6 your plant environment; the challenge to you is to
7 listen to the speakers, look at your own plant
8 environment, re-assess your program and figure out
9 what works best in your plant environment.
10 Don't wait on us to come into your
11 facility and say, Why didn't you take advantage of the
12 information. You don't need to wait on a food safety
13 assessment for that to occur. Again, you have
14 options, and we encourage you to take advantage of
15 those.
16 We're going to be very transparent with
17 data. You're going to hear from our speakers. Again,
18 we're going to share lessons learned from previous
19 food safety assessments. Learn from those lessons
20 learned.
21 You're going to hear consistently from Dr.
22 Petersen about plants that failed to control their own

1 processes. We're going to share with you, again,
2 compliance guidelines from the pre-harvest meeting.
3 We've done an extensive literature review, and we're
4 going to share that with you.
5 My favorite I -- so many people have heard
6 me talk about an AVMA meeting where we -- there was a
7 great literature review: "Ain't Nothing Good Ever
8 Happens at Picking." Yet, when I walk into many of
9 your poultry operations, I see picking fingers that
10 haven't been changed in months. Take advantage of
11 this information and apply it.
12 We're going to be sharing with you your
13 own *Salmonella* data on an individual -- result by
14 result. You're going to hear that from us. If you're
15 getting those results back and not using them to your
16 own advantage, then you're not going to be making the
17 necessary changes in your own operation. And again,
18 that's the kind of information we're expecting you to
19 use and apply, and you can determine what the best
20 controls are in your own operation.
21 Again, the CDs will be available from this
22 meeting. Transcripts will be available from this

1 meeting. And so we're just imploring you to take the
2 advantage and re-assess your own programs to make the
3 necessary changes. And you need not wait for a food
4 safety assessment for that to occur.
5 Improvements are expected. As Dr. Raymond
6 indicated, changes will occur. We do expect to see
7 significant improvements in *Salmonella*. We do believe
8 the poultry industry is up for it. We've started to
9 see some changes; unfortunately, we're not seeing
10 *Salmonella* numbers go down. You'll see that when the
11 *Salmonella* results are posted. That's not what we
12 want to see. We want to see the prevalence go down.
13 And we do believe you're up for that
14 challenge. You're here, and that's a good sign.
15 You've been meeting with us as an Agency, and we do
16 recognize that you're starting to work together as
17 groups. And so we do believe you're up for the
18 challenge. We want to work with you. We want to make
19 information available to you. So we know that you're
20 up for the challenge, but it is up to you to make that
21 difference.
22 So again, thank you for being here. And

1 we look forward to watching those numbers come down
2 over the next year. Thank you very much.
3 (Applause.)
4 DR. ENGELJOHN: Thank you, Dr. Masters.
5 Our next speaker is Mr. Loren Lange; he's
6 the Deputy Assistant Administrator for the Office of
7 Public Health Science.
8 Loren came with the Agency back in 1979
9 and has held several leadership positions with us, and
10 he had also worked at FDA prior to that. He has his
11 degrees in mathematics from Iowa State University and
12 a master's degree in applied mathematics from Johns
13 Hopkins University.
14 Loren's going to talk to you about the
15 2005 data. On our web page, you have access to all of
16 our data up through 2004, including the serotype
17 information. Loren's going to give us information
18 about broilers and ground products for 2005.
19 Loren?
20 MR. LANGE: Thank you, Dan.
21 Good morning. It's a pleasure to be here
22 to speak to you about -- oh.

1 I've got to clip it on here? Okay.
2 (Pause.)
3 MR. LANGE: Right here? Does that work?
4 THE REPORTER: Yes.
5 MR. LANGE: Our speakers have mentioned
6 that we've got a new Administrator and new Deputy
7 Administrator. And Barbara knows that --
8 Barbara, you were -- a little over a year
9 ago, I got a new boss.
10 And you've got the Under Secretary and the
11 Secretary and the Deputy Secretary.
12 And I was out for dinner one night, and
13 someone said, Well, how are things going at work. And
14 I said, Well, there's two constants: President Bush
15 and me. So at least there's some constant at work.
16 (Laughter.)
17 MR. LANGE: So I'm glad to be here this
18 morning. I'm going to present a brief summary of our
19 2005 results from testing poultry products for the
20 three products -- you know, broilers, raw ground
21 chicken and raw ground turkey -- that are covered by
22 existing performance standards.

1 The results of all seven product
2 categories that we test for are being posted on our
3 web site today; they should be up there by noon, by my
4 understanding. And if you haven't heard or seen it,
5 I'm just going to mention that last Friday, we put up
6 or serotype data for all the seven years, 1998 through
7 2004, and we'll soon be updating that to include our
8 2005 data.
9 Now, before I present the results, I do
10 want to just point out a little bit about the nature
11 of the data that we post on our web site. These are
12 either positive or negative results from our HACCP
13 verification testing; the results are not from
14 statistically designed baseline studies. Thus, the
15 results are not estimates of national product
16 prevalence. However, we do consider that the data do
17 give us an indication of the trends.
18 The data I will present are summaries of
19 individual samples from what FSIS calls A sets. These
20 are the sets that are routinely scheduled to verify
21 compliance in establishments that are either new or
22 past their previous verification step. The data do

1 not include the follow-up steps to verify corrective
2 actions. Thus, I will be presenting the data that are
3 exactly the same that we've been posting on the web
4 site since this program was initiated in 1998.
5 This presentation will cover two topics.
6 I'll first summarize what we found in 2005, and then I
7 have a few slides showing that the results from 2005
8 didn't follow what we have seen as some historical
9 patterns.
10 First, the summary of the 2005 poultry
11 results. This slide shows seven years -- no. I'm
12 sorry. This slide shows the 2005 results for
13 broilers. In the A sets, the percentage was 16.3
14 percent. This is the third year that we have seen the
15 percentage of positive samples go up in broilers.
16 This next slide shows the broilers by year
17 of the percentage of passing sets. And you see -- one
18 sort of observance here is that as the first couple of
19 years that the percentage of positive samples was
20 going up, there really wasn't a huge change in the
21 percentage of the sets that were passing.
22 In fact, we see that actually from 2003 to

1 2004, the percentage of sets that passed actually went
2 up a little bit. But when we got into last year, when
3 the percentage got as high as 16.3 percent over the
4 year, we obviously had a lot more higher percentage of
5 sets that were failing. And it dropped 9 percent,
6 from 90.3 down to 81.3 percent, of sets that were
7 passing.

8 This next slide shows our ground chicken
9 results per year. The percentage was up a little bit
10 in 2005 from 2004 -- well, actually, it was up 32
11 percent. But notice the numbers of samples are very
12 small here. So we really don't consider the ground
13 chicken data to be merely as good an indicator of
14 trends certainly as the carcass data are, but we do
15 put it up on the web, and I am presenting it here for
16 completeness.

17 The next slide shows ground turkey by
18 year. Again, ground turkey went up from 2004 to 2005,
19 up from 19.9 to 23.2 percent. Now, with ground
20 turkey, we do get, you know, approximately a thousand
21 samples every year. And as you can see, generally,
22 we've been averaging around a thousand samples. So we

1 consider this a little better indicator of trends.
2 We sampled ground turkey in, I would say,
3 roughly half of the major turkey slaughter operations.
4 So we're getting -- our information indicates at
5 least half produced this ground product, and we have
6 been sampling in about half the plants.
7 Now, the next slide's where I'm going to
8 point out what I said my second topic is. We have
9 results showing how 2005 did not exactly follow
10 historical patterns by month and by quarter.
11 This slide shows the broiler data over
12 seven years. Just to make sure everybody's clear,
13 what it's showing is that like -- April/May/June, it
14 shows that broilers have averaged 10 percent. Well,
15 that's the April/May/June data from 1998, 1999 and all
16 the way up to 2004 summed up, the percentage of
17 positive samples over seven years.
18 We see this trend that the low has been
19 April/May/June over those seven years, 10.2 percent,
20 and the high has been the fourth quarter,
21 October/November/December, 14.6. Now, relative to the
22 absolute levels, 10 percent and 14 percent, that

1 difference of 4.4 percent, you know, we would say is
2 considerable. So there certainly has been this
3 historical difference between the months.
4 We went back and looked at just the last
5 three years, and we have seen this is, you know,
6 about -- I'm sorry -- the three years, 2002 through
7 2004. And there we saw that, again, April/May/June
8 was 10.4 percent; October/November/December, 15.3
9 percent. So we saw this same pattern: Low in the
10 second quarter; high in the fourth quarter.
11 This is the 2005 data by quarter. It
12 certainly looks different.
13 Here the second quarter was actually 19.7
14 percent, the highest, where it had always been the
15 lowest. And the fourth quarter wasn't the lowest.
16 The third quarter was a little low, but the fourth
17 quarter was down to 14.5 percent, clearly a different
18 pattern. Something changed, and we hope this -- it's
19 a change in certainly, you know, the last two
20 quarters -- is a change in the right direction. And
21 we hope this is the beginning of a new trend.
22 Next I have a couple slides showing the

1 same thing for the broilers by month. And we can see
2 that the lows were always April -- this is the seven-
3 year data. Excuse me. The lows of 9.8 percent were
4 April/May. And we had three months,
5 September/October/November, that over seven years were
6 always above, you know, 15.3 percent. You know,
7 there's tons of data. I mean every time I see this, I
8 think, You don't find real-world data that follows
9 such a nice S curve. I just -- you know, for someone
10 that has spent his whole life looking at data and
11 trying to analyze data, I'm always amazed by -- you
12 don't see curves like this in real-life data. Very
13 nice.
14 But here's 2005. Again what -- that's not
15 a nice curve -- oh.
16 I've got two minutes? Okay.
17 Nice curve, not a nice curve. But guess
18 what. Those low months where the highest -- in fact,
19 May was 21.7 percent. And we were down -- November
20 was the lowest at 13 percent.
21 I've got two minutes.
22 Ground turkey? This goes back to the

1 quarters -- we saw the second quarter. Just in
2 contrast to broilers, ground turkey was highest in the
3 second quarter, where broilers had been lowest. But
4 this is 2002 to 2004, three years of data, and it
5 changed. Something had happened. The last three
6 quarters were all about the same. And this is what
7 happened last year.
8 We saw it increasing by quarter. Now,
9 there's not a lot of samples in each quarter, so we
10 don't know if this is really trend. But we did see
11 the fourth quarter higher than it had been, you know,
12 over the 2002 to 2004. There may be some concern
13 there.
14 Thank you. That's the end of my
15 presentation. I made my time limit.
16 Dr.RAYMOND: Just one more thing.
17 [inaudible due to failure of in-house PA system]?
18 MR. LANGE: No, not here. Dan gave me the
19 two minutes.
20 DR. RAYMOND: For those who don't know
21 Loren well -- I owe him one, because he got me once.
22 But this is the first time I've ever heard Loren walk

1 away without saying, Oh, just one more thing.

2 MR. LANGE: I do have one more thing while

3 I'm walking back --

4 DR. ENGELJOHN: That's enough, Loren.

5 Thank you, though, very much.

6 (Laughter and applause.)

7 DR. ENGELJOHN: And I do want to move on.

8 All of this information is available to you. So just

9 so you know, it will be available.

10 Our next speaker is Dr. Sean Altekruse.

11 Sean is our Deputy Executive Associate in the Office

12 of Policy, and he's responsible primarily for

13 coordinating the statistical and technical support for

14 our policy development. He's also a captain in the U.

15 S. Public Health Service and has his veterinary degree

16 from the University of Georgia, a master's in public

17 health from the University of South Carolina and a PhD

18 from the Virginia/Maryland Regional College of

19 Veterinary Medicine.

20 I do want to preface Capt. Altekruse's

21 presentation with a note that, as Dr. Raymond

22 mentioned earlier, on Tuesday, we did post a Federal

1 Register document that was submitted to the office of
2 the Federal Register for publication. And it will
3 publish in the Federal Register on Monday, the 27th,
4 but it is available to you. It may have some slight
5 formatting changes from what the published is, but it
6 is available to you -- which will clearly articulate
7 what Dr. Altekruse is actually going to present to you
8 now.
9 So, Dr. Altekruse?
10 DR. ALTEKRUSE: Good morning. So what I'd
11 like to talk about today is the *Salmonella* categories
12 that are described in the document that has been
13 posted to the web, which -- our Under Secretary, Dr.
14 Raymond, described the three categories -- and also,
15 the type of data that we're going to be sharing, which
16 our Administrator, Dr. Masters, mentioned,
17 specifically, the serotype information.
18 So specifically, the *Salmonella* categories
19 are -- the current casts the sets into two groups.
20 One is those that are less than 50 percent of the
21 standard, and the other is that they're above 50
22 percent of the standard without failing the standard.

1 And then the third category is the existing category
2 of exceeding the standard. And I'd like to talk about
3 why we think these three categories make a lot of
4 sense.
5 So just briefly, we have looked at data
6 for a variety of product classes. And today, I'm
7 going to be presenting information specific to large
8 broiler establishments, but the same patterns are seen
9 for small broiler establishments and for other product
10 classes, as well. And also, this presentation is
11 specific to data through 2004, although we have looked
12 at data through 2005 and the same patterns continue to
13 occur.
14 So establishments are tested about once a
15 year. One broiler rinse is collected per day, and
16 there are 51 rinses per set. This should be fairly
17 familiar. And then those rinses have results of:
18 Less than 50 percent of the standard, which would be
19 six or fewer positive tests per set; greater than 50
20 percent, which would be seven to 12 positive tests per
21 set, and; exceeding the standard as having 13 or more
22 *Salmonella*-positive tests in a set.

1 So these these categories really make some
2 sense. Category I shows consistent *Salmonella* control
3 is possible. Category II suggests to us that these
4 plants are doing the right things but, with a little
5 bit of perseverance, can do even better. And Category
6 III is what it always has been, which is failing to
7 meet the standard.
8 And why have we selected these categories?
9 The reasons are really pretty straight forward.
10 First of all, Category I is the normal scenario.
11 There are -- and Category II accounts for about 25
12 percent of sets. And Category III is really -- it's
13 an outlier in terms of what we're seeing over the
14 historical time. Less than 10 percent of sets are in
15 Category III.
16 And furthermore, if we look at Categories
17 II and III, that's where the majority of the
18 *Salmonella*-positive tests are. And, even more
19 important, it's also where the serotypes that are most
20 commonly associated with human illness are most likely
21 to occur.
22 So it's really a testimony to the hard

1 work of the great FSIS work force and what they've
2 accomplished. Over the last seven years, they have
3 completed *Salmonella* A tests in about 135 large
4 broiler establishments, so we have -- about once a
5 year. So 762 completed sets. And from that ongoing
6 prospective sampling and verification program, we have
7 serotype information, phage type information and
8 pulsed field data on isolates.
9 So this brings up some questions. What
10 does this historical information tell us? First of
11 all, the vast majority of establishments are in
12 Category -- of sets are in Category I.
13 This schematic shows the distribution. So
14 a huge number of sets are in Category I. And then
15 Category II is about a quarter of all sets. And
16 Category III is way out there. And in a few sets,
17 we've seen as many 30 isolates per set.
18 Now, this slide is intended to provide a
19 little definition about common serotypes. The Centers
20 for Disease Control publishes the list of the 20 most
21 common *Salmonella* serotypes in people each year. And
22 when we look at that list, we see some of the

1 serotypes which also occur in poultry rinses that we
2 collect, particularly Heidelberg, Typhimurium, and
3 Enteritidis, those top three.
4 Now, each one of these serotypes has its
5 own little reservoir. So with Typhimurium, we see it
6 in a lot of product classes; it's not unique to
7 poultry by any means. Heidelberg is -- it does occur
8 in some other classes, but primarily in poultry. And
9 Enteritidis is -- it's really the exception to find it
10 in other product classes, although never say, Never.
11 So -- but really, these serotypes do have their own
12 unique reservoirs.
13 Now, what can we -- what knowledge can we
14 gain from our A sets about these common serotypes?
15 First of all, this first question, "Is the percent of
16 the common serotypes the same in each category," is an
17 important question because if the driver for being in
18 Categories II and III is a serotype that usually
19 doesn't commonly cause human illness, we should know
20 about that.
21 Secondly, are these common human illness
22 serotypes more likely to be found in one of the

1 categories or another? Third, is any category
2 resident for the Lion's share of the total serotypes?
3 And the last question is, How many isolates can you
4 normally expect to see in an A set of these serotypes?
5 So let's go through those questions systematically.
6 Do these serotypes account for the same
7 percent of *Salmonella*? Well, you'd be very surprised
8 if the percentage was absolutely identical across all
9 classes. However, it's very close. It's within 5
10 percent. And when you look at the confidence
11 intervals around the point estimate, they all capture
12 the overall average, which is 48 percent, and they all
13 capture 50 percent, as well.
14 So in other words, just shy of 50 percent
15 of all *Salmonella* that we get from broiler rinses are
16 in these common human serotypes, and there's no
17 statistical difference between the two groups.
18 Now, the second question was, Are any of
19 these categories more likely to be positive, to have
20 sets that are positive, for *Salmonella*? And this
21 would apply -- the table shows, "Was there a human
22 serotype in the set," with the answer yes or no. And

1 you can see for Category I about a third of all sets
2 tested negative for human serotypes. In contrast, in
3 Categories II and III, it was about 5 percent of all
4 sets that tested negative for human serotypes.
5 And so if you look at the odds ratios for
6 those associations, what you see is that there's a
7 very robust statistical association between Categories
8 II and III and the likelihood of testing positive for
9 a human serotype.
10 The third question was, Is there any
11 category that accounts for the lion's share of these
12 serotypes? And the answer to that is also yes.
13 Categories II and III, although -- remember it was 24-
14 and 8 percent respectively of all sets that fell into
15 those two categories. So they -- together they
16 account for 32 percent of sets. They account for 63
17 percent of common human serotypes.
18 And I think an important additional point
19 is that Category II, the middle category, which is
20 sort of the new category that we're describing here,
21 has 737 positives for the common human serotypes. So
22 that category contains the most common human

1 serotypes.

2 And then the last question on that
3 introductory slide was, How many isolates of common
4 human serotypes should you expect within a given A
5 set? And the answer is very few. Twenty-five -- this
6 slide shows the percent of all sets and then the
7 isolates of common human serotypes with -- per set.
8 And you can see that 25 percent of A sets had no human
9 common serotypes in them.

10 And then if you go to, "1," of the common
11 human serotypes, the next increment on the X axis,
12 that's 49 percent of all sets that had one or fewer
13 human serotypes. So I had to say that there were two
14 or fewer -- most sets had two or fewer, because you
15 have to add in two or fewer to get to 64 percent.

16 And then going on out the scale, "3," is
17 75 percent. "8," common human serotypes within an A
18 set is something that you would not expect to see in
19 95 percent of sets. "12," is -- you would not expect
20 to see that in 99 percent of sets. And you have to go
21 to 30 common human serotypes per set to reach the
22 maximum.

1 So in summary, most sets have no or very
2 few common serotypes of human illness in them. And
3 Categories II and III, although they account for only
4 one-third of all A sets, accounted for two-thirds of
5 the common human serotypes. So we really think that
6 those two categories, II and III, are an important
7 place to focus.
8 Now, I've talked about sets, but I'd like
9 to talk about something that Dr. Raymond mentioned, as
10 well, which is the performance of individual
11 establishments over time. We have data on more than
12 100 large plants over seven years, and a quarter of
13 those establishments could demonstrate consistent
14 control of *Salmonella* throughout the entire period of
15 follow-up.
16 Most sets had no or -- wait. So this is
17 looking at the large establishments with five or more
18 sets through 2004. We see that a quarter of the
19 establishments could control *Salmonella,* as defined by
20 consistently being in Category I. And the others were
21 in Category II or III. And most often they were in
22 Category II or III at least twice, but there was a

1 gradient of performance. Some just barely -- some
2 only were in Category II one time out of the entire
3 follow-up period. And some failed as many as three
4 sets.
5 So this graphic sort of depicts that
6 distribution over time, and the biggest category is
7 the middle category. The way I would interpret this
8 is that this is a group of plants that is sort of on
9 the line and, with perseverance, they can move in the
10 direction of the establishments depicted in green,
11 which have lower than 50 percent. But if just allowed
12 to drift, there's also the potential that they could
13 begin to have sets that they fail. And we want to
14 encourage the first and discourage the second.
15 I'd like to also focus specifically on the
16 plants that never exceeded half the standard, because
17 I think this is an important group of plants. These
18 plants, after five, six or seven tests, have never
19 failed a plant. And I think that this isn't an
20 accident. This occurs because of leadership within
21 the establishments: The executives, the quality
22 control management, the plant employees, the

1 investment and testing and the validation of the
2 infrastructure that they're using to control
3 *Salmonella*. And so I would suggest that these plants
4 are the leaders and that they are an important
5 resource for helping the industry to meet this
6 challenge.
7 So in summary, what we see in looking at
8 the data over time is that there is a tendency for
9 patterns to emerge, that plants can demonstrate the
10 very best control or an intermediate level of control
11 or have some variability in their control of
12 *Salmonella*. And I showed this slide earlier, but I'll
13 show it again, because maybe it means a little more
14 after my presentation.
15 Category I indicates consistent *Salmonella*
16 control. Category II suggests that they're moving in
17 the right direction, but some improvement is possible.
18 And category III continues to be failing to meet the
19 standard.
20 Really, this data is the product of seven
21 years of hard work by the FSIS work force, and I'd
22 like to acknowledge just a few of the people who have

1 helped to make this presentation possible. Thank you.
2 (Applause.)
3 DR. ENGELJOHN: Thank you, Dr. Altekruse.
4 Well, I hope that gave you a bit of
5 perspective as to why we have a need to talk today and
6 to continue the dialogue. But to pull all this
7 together, we have Dr. Robert Wills from Mississippi
8 State University, who has done some extraordinary work
9 on pulling together the literature to define, What
10 interventions are available, and how effective are
11 they.
12 We think this information is absolutely
13 critical for you to hear. We're delighted that this
14 work was being done through one of our sister
15 agencies' grant programs; the CSREES within USDA
16 helped fund part of this work, and we think it's just
17 outstanding. And we know that there's other work
18 related to *Campylobacter* that's going to be underway
19 soon.
20 Dr. Wills since 2001 has been an associate
21 professor of veterinary epidemiology in the Department
22 of Pathobiology and Population Medicine at Mississippi

1 State University. He previously was an assistant
2 professor in the department of veterinary and
3 biomedical sciences at the University of Nebraska at
4 Lincoln, in Lincoln, Nebraska.
5 He received his doctor of philosophy from
6 Iowa State University, a college of veterinary
7 medicine degree in Ames, Iowa, and a doctor of
8 veterinary medicine from the University of
9 Missouri/Columbia College of Veterinary Medicine in
10 Columbia, Missouri.
11 Dr. Wills, we're delighted to have you
12 here today.
13 DR. WILLS: Well, thank you.
14 I was a little worried. I thought maybe
15 we'd catch up pretty quick here when we couldn't find
16 my presentation, but we did find it.
17 I want to talk about a method that was new
18 to me; about a year-and-a-half ago, I guess, I've
19 known about it. I think it has great potential to
20 help us to fully utilize the literature that's
21 available -- scientific literature. And I think it's
22 a good tool that we can apply to figuring out what the

1 best intervention strategies are controlling
2 *Salmonella*.
3 I want to take the moment to acknowledge
4 the people that I'm working with on this project:
5 Hart Bailey, who's here; Kris Clements, who's with
6 Mississippi State, as well, and that's helping us with
7 this. I also want to acknowledge Jan Sargeant at
8 McMaster University in Canada, who's really a leader
9 in developing this methodology for use in food safety.
10 I think the premise that most people agree
11 with is that the production of safe food involves
12 intervention strategies to be implemented at all
13 stages of production, from farm to fork. We've heard
14 about that already today. In order to do that,
15 decision makers need information: What strategies,
16 and where to apply them.
17 I think the scientific literature is a
18 good resource for this information, but there are some
19 problems I see with using the literature -- some
20 stumbling blocks. One is sometimes a lack of
21 literature: It's difficult to find information on a
22 particular subject; it may be out thee, but it's not

1 readily accessible.

2 And the other is kind of the flip-side of
3 that, that you have a large body of information and,
4 in fact, so much information that you have an
5 overwhelming quantity of it. And it's difficult to
6 synthesize that and summarize it, and, even when you
7 do, you find conflicting conclusions.

8 So systematic reviews are a way to help us
9 deal with this scientific literature and come up with
10 some answers from it. It's a method of identifying
11 effective treatments or processes based on the
12 available evidence from a variety of sources. It
13 differs from traditional narrative or critical reviews
14 of literature. And I'll go into a little more detail
15 about that in a moment. But it gives us a transparent
16 and replicable scientific methodology to collect,
17 assess and synthesize all available information on a
18 subject.

19 And one of the key components of this is
20 that it could be reproducible in that in the final
21 paper or document you come up with with the systematic
22 review -- within that, the methods should be outlined

1 sufficiently that a person could take that through the
2 same systematic review and, hopefully, come up with
3 the same conclusion.
4 Now, the reason I became interested in
5 systematic reviews was as an outgrowth of the Food
6 Safety Research and Response Network, which is USDA
7 funded, a sub-project of the Food Safety CAP. And we
8 have a sub-project in that looking at *Salmonella* and
9 preventions, and Jan Sargeant, as I mentioned, is
10 leading this effort. Annette O'Conner and Jim McKean
11 at Iowa State University are conducting the systematic
12 review of *Salmonella* interventions in pork or -- in
13 pigs. And then Hart and I are working on one in --
14 looking at *Salmonella* interventions in broilers.
15 I wanted to put up this slide, and you may
16 not be able to read it. But it's "A Guide to
17 Conducting Systematic Reviews in Agri-Food Public
18 Health", and it's kind of a how-to manual on
19 conducting systematic reviews on food safety topics,
20 and it was produced by Jan Sargeant. And I wanted to
21 put it up here because it's a good resource and I took
22 a lot of the information for this presentation from

1 it. I want to acknowledge that.
2 So in looking more at our rationale and
3 significance of systematic reviews, how they've been
4 used and why, they're very commonly used in human
5 medicine fields and used in evidence-based medicine.
6 And they're used primarily to identify effective
7 interventions to reduce disease burden.
8 But they may also be used to identify
9 knowledge gaps that target additional research, and
10 that's one reason I think there's great promise in
11 systematic reviews. Even if you don't come up with
12 the final answer, one of the byproducts of it is that
13 you find out the gaps that you need to know in order
14 to find those answers. So I think that's useful.
15 It can also identify methodological
16 strengths and weaknesses in the available research.
17 We can find out why we don't have the answers.
18 Perhaps it's because the research wasn't conducted
19 appropriately. And that will help us identify, once
20 again, where we need to go with more research. And it
21 can also encourage best study practices development
22 for intervention research. We can find out how to do

1 the research to find the answers.

2 So Jan Sargeant put this slide together,
3 and it shows that there's a lot of systematic reviews
4 used in health science, lots of those. If you narrow
5 it down and look at systematic reviews for public
6 health, there are still quite a few, but it's a
7 reduced number. And then when you get to looking at
8 systematic reviews in microbial food safety, there are
9 really very few of those available.

10 Now, this isn't an exhaustive list here,
11 but I wanted to make a point of it because it's a
12 recently published systematic review for development
13 or -- for control of *Campylobacter* in broilers. It
14 focuses primarily on contributing factors and sources
15 of *Campylobacter* in Great Britain, but I thought it
16 might be of interest. And it demonstrates that these
17 methodologies are being used in the field of food
18 safety.

19 I mentioned I make a comparison of
20 systematic versus traditional reviews. And generally,
21 systematic reviews have a more focused study question;
22 it's defined, and it is a foundation of the review.

1 Systematic reviews also have a more explicit and
2 comprehensive search strategy; the articles are
3 selected according to uniformly applied and specified
4 criteria.
5 The quality of articles is formally,
6 rigorously and consistently assessed. And a
7 quantitative summary is made if possible; it's not a
8 requirement for a systematic review, although the true
9 goal, if you could, would be to do a Met analysis of
10 multiple studies for your final product of the
11 systematic review. And if that's -- if the studies
12 aren't there that allow that, the systematic review
13 can still be done and done successfully in
14 productivity.
15 I wanted to give you a schematic here of
16 the systematic review process, just to get a general
17 idea of it. And then I'll go through it in a little
18 more detail. We start out with the study question,
19 and I've put this in the center of the schematic
20 because I think the whole systematic review really
21 revolves around the question that's being asked. And
22 it has to be a well defined question.

1 Then the next step is to do a literature
2 search and try all the information that can be found,
3 all the primary research that can be found, for that
4 study question. I have a step process of relevance
5 screening so that you find the articles that are
6 relevant to that study question. Once they pass that,
7 they go through a process of quality assessment. If
8 they make it through that, the data is extracted from
9 those articles, that information is synthesized or
10 summarized, and then the final product is a written
11 report.
12 So start off here with the development of
13 a focused study question, and the question needs to be
14 clearly defined *a priori*. And that's, I think, a
15 critical part of this.
16 You don't do the review and then decide
17 what you wanted to find out from it; you start with
18 the question in mind at the very beginning. And that
19 question will include components on the population
20 that's being studied, what intervention is being
21 looked at, what outcome is being assessed, and it
22 specifies the system

1 level or sector of agriculture to be reviewed.
2 And this -- the development of this study
3 question is accomplished by forming a panel of content
4 experts. And they decide how to write this question
5 so that the subject of interest is captured within
6 that question.
7 The next step is a literature search. You
8 want to generate a complete list of all primary
9 research that is relevant to the question. And the
10 search terms, once again, are based on those
11 components that make up the study question:
12 Population, intervention, outcome and agriculture
13 sector.
14 And we're looking at published literature.
15 To do this, we use an exhaustive list of search terms
16 in multiple electronic databases. You'd also need to
17 hand-search journals that -- if you can identify
18 relevant journals that we feel may have the papers on
19 this topic, but they are not listed in any of the
20 electronic journals, we'd still want to go through
21 those and hand-search them.
22 Also -- in addition to the published

1 literature, gray literature is also searched. Now,
2 gray literature is completed-but-unpublished research.
3 This can be found through scanning the internet,
4 electronic and hand searching of conference
5 proceedings and contacting researchers, national and
6 international experts in the field directly.
7 Well, once we have this huge amount of
8 information -- and this -- we may end up with several
9 thousand -- it's quite likely we'll end up with
10 several thousand abstracts after our search -- we then
11 go through a process of screening these for relevance.
12 We want to determine if an article has potential to
13 answer the study question through this screening
14 process.
15 *A priori* criteria for subject relevance
16 and inclusion of material into the review is
17 established, and these are a series of questions that
18 have to be answered based on information in the
19 abstract to determine if the abstract is relevant to
20 answering the study question.
21 And another point here that's unique, I
22 think, with systematic reviews as compared to

1 traditional reviews is that at least two reviewers
2 independently assess each article or abstract to
3 determine whether or not it's relevant. If they have
4 conflicts, if one excludes it and the other one
5 includes it, then they, those two reviewers, have to
6 meet and resolve those conflicts to decide which way
7 the abstract should go.
8 I've put up here a shot of the screen on
9 the software program we're using in our systematic
10 review. I just wanted to point out that we have a
11 series of questions on the right-hand side that we can
12 check off. And the information we have available to
13 make these decisions are the authors, the title, and
14 then an abstract if available. Sometimes abstracts
15 don't come through, and we have to do a little more
16 searching to track those down.
17 In our particular one systematic review,
18 we're categorizing the information, thinking that we
19 will probably come up with several systematic reviews
20 on intervention strategies in broilers, but our first
21 screening process is to divide it into broilers and
22 layers and then different segments of the poultry

1 continuum and then into different intervention
2 strategies.
3 But once we've had an article successfully
4 pass the screening process and at least two reviewers
5 have deemed it relevant, then we find the full article
6 and do a second, stricter level of screening using
7 full copies of the articles. There's a standardized
8 procedure developed for each type of study design, and
9 that's used to evaluate each article. And once again,
10 two reviewers use that standardized protocol to review
11 that particular article.
12 And just a list here of the items that are
13 used in this assessment: A look at the study
14 objectives, the population that's being looked at,
15 intervention strategy -- make sure it's appropriate --
16 a look at the outcome assessment, how withdrawals or
17 loss of data was handled, and, also, how the data was
18 analyzed, and then the conclusions, if they were
19 appropriately made or not.
20 Different types of study designs provide
21 different levels of evidence, and I have a list here.
22 I think I'll skip over it other than that within a

1 systematic review, different types of designs have
2 different values or levels of evidence, and you can
3 restrict it to a systematic review to a particular
4 level if you have sufficient articles; if you don't,
5 you may have to look at everything, and that will make
6 it more difficult to compare the data.

7 So once we've had an abstract to go
8 through the screening process and we get the full
9 article and we've screened it or assessed its quality,
10 the next step is data extraction, where that paper
11 that has made it all the way through is read and we
12 take the different components of it and put those in
13 the data repository so that the can be summarized and
14 analyzed as a whole.

15 And once again, we'll have a form that we
16 use to fill out. It includes information:
17 Descriptive data of the article in context, study
18 characteristics -- what design was used, how many
19 animals, and those sorts of things -- and then study
20 results.

21 Then this information is synthesized or
22 summarized to take the results from multiple primary

1 studies that have met the quality assessments and put
2 them together and analyze them using either
3 qualitative methods or quantitative methods when
4 possible.
5 The data is presented in this step so that
6 similarities and differences between the studies and
7 the level of evidence can be visualized, and the
8 results are presented to show if intervention is
9 consistent and effective. And that's -- the final
10 bottom line that we'd like to achieve is to make a
11 determination of how well that intervention works and,
12 if possible, how well it worked in different
13 situations and different conditions.
14 Now, one of the challenges that we have in
15 doing this is just managing all of the data and the
16 information. You start out with a very large volume
17 of abstracts -- several thousand. We started out with
18 one pass that had over 13,000 and dropped it to around
19 2,500 as our final starting point. But that's a lot
20 of abstracts to keep track of. And if you have to
21 have paper copies of all those, it really becomes a
22 challenge.

1 And then once you move those through the
2 multiple stages of valuation, it becomes even more
3 difficult. And if you have reviewers -- multiple
4 reviewers potentially from different places, then it
5 becomes even more of a challenge.
6 There's some software available -- the one
7 we're using is a web-based program -- that can handle
8 this. Data's loaded into -- the literature's loaded
9 into the program. It contains the standardized
10 protocols, so you can have check-lists and move
11 through it. It keeps track of what reviewers have
12 done, it automatically tracks the movement of
13 articles, it keeps track of what the reviewers have
14 said about those articles and identifies discrepancies
15 between them and keeps track of all the information
16 all the way through data extraction.
17 I have a few things here. I've mentioned
18 the systematic reviews that have been done in pork and
19 the one we're working in broilers. And there's also
20 one being done on *E. coli* 0157 in cattle at McMaster
21 and also one looking at the association between Johnes
22 and Crohn's disease in Canada.

1 Just a few slides here on the strategy
2 or -- on the systematic reviews we're using in
3 intervention strategies in broilers. I mentioned
4 we're taking an approach where we're looking at pre-
5 harvest/post-harvest, looking at broad field -- a lot
6 of different interventions, categorizing those so that
7 we can either look at them at that level or focus in
8 on a fewer number of interventions and do a complete
9 review of those.
10 And these are just the screening process
11 slides that we have here, where we're dividing up into
12 broilers, layers -- we do keep track if a turkey
13 article -- if it refers to turkeys as well as broilers
14 or layers. Then we divide it into the thicker segment
15 of a production continuum. Then we have a list of
16 intervention strategies that we're keeping track of.
17 And then the final question here is, Is
18 this primary research? This is sometimes used as the
19 very first question. If it's a review, you may use
20 that as a source of more articles, but it's not
21 included in the systematic review, only primary
22 research.

1 I'd like to thank Dr. Jay Levine from
2 FSRRN -- he's director of that program -- and Dr. Mary
3 Torrence, who's program manager for the funding for
4 that project. Thanks for your attention.
5 (Applause.)
6 DR. ENGELJOHN: Well, thank you very much
7 for all the information this morning.
8 And as I said, I think we are particularly
9 interested in the work that Dr. Wills is working on
10 with broilers. I think he is nearing completion of
11 what he is doing for broilers, and then there'll be
12 some activities associated with that and then a
13 process for making that information available, because
14 we as the Agency have a special interest in making
15 sure you as the industry and regulated customer have
16 access to that important information.
17 That completes our first segment of today.
18 We have 20 minutes set aside for a break. I'll call
19 you back in when we're ready to go. I know there's a
20 water fountain outside to the left, as are the
21 restrooms. And then there are beverages down the
22 street to the right.

1 (Whereupon, a short recess was taken.)
2 DR. ENGELJOHN: Okay. Some of you have
3 some questions you'd like to ask. And so I thought
4 I'd just take this point while we're still uploading a
5 presentation. We have time for maybe one or two
6 questions -- at least one question.
7 So I've received a couple I'm going to
8 answer. It -- but if somebody has a question that
9 you'd like to ask the panelists, would you please come
10 up to the microphone? And get ready to say who you
11 are and which association you're with, and then we'll
12 get started.
13 There have been questions about whether or
14 not we're making the CD available and how quickly
15 we're making compliance guide information available.
16 Just so everyone knows, as soon as this meeting is
17 over, we will download the information from the
18 internet. The transcripts will not be immediately
19 available, but all the PowerPoint presentations,
20 speakers' notes and so forth will be.
21 And as quickly as we can get that done,
22 we'll make those available. So you should expect that

1 within the next couple of weeks, that information
2 would be available.
3 I think we have somebody ready to ask a
4 question.
5 If you would, give your name and
6 association.
7 (Pause.)
8 DR. ENGELJOHN: And the question is for
9 Dr. Altekruse. So is he here?
10 (Pause.)
11 DR. ENGELJOHN: It looks like he stepped
12 out. So we'll try to answer it.
13 MS. JOHNSON: I'm Patricia Marsh Johnson
14 with V. E. T. Solutions. My question was for Dr.
15 Altekruse actually.
16 So I was confused. In one point in his
17 slides when he was talking about the isolates that are
18 commonly found in human illness, he was stating that
19 about 50 percent of the Set A isolates are serotypes
20 that are common to human illness, but then in a
21 subsequent slide, he said that the vast majority of
22 sets have less than two isolates per set.

1 So I'm not understanding how that jives, I
2 guess, because less than two isolates per set is far
3 less than 50 percent of the isolates being part of
4 those that are commonly found in human illness.
5 DR. ENGELJOHN: I think we'll wait for
6 Sean to get back to answer that one --
7 (Laughter.)
8 DR. ENGELJOHN: -- rather than me give
9 you an answer, which I would probably attempt to do,
10 but I think I will not. So we'll keep that one on the
11 record, and we'll try another one.
12 Does somebody else have a question?
13 MS. NESTOR: I'm Felicia Nestor with Food
14 and Water Watch. And I'm just wondering, after seeing
15 the seasonality in the *Salmonella* data, what will you
16 do -- it sounds like from reading the new Federal
17 Register notice that you're going to be testing on a
18 more consistent basis.
19 Now, how will you deal with the fact that
20 some plants that are going to be tested in certain
21 months are going to be getting a higher percentage
22 just because of the seasonality effect? If I were in

1 the industry, I would definitely be arguing me if you
2 were testing me in the worst months.
3 MR. LANGE: When we -- if you've read the
4 Federal Register notice --
5 DR. ENGELJOHN: Who are you?
6 MR. LANGE: What?
7 DR. ENGELJOHN: Who are you, for the
8 transcript?
9 MR. LANGE: Oh. This is Loren Lange,
10 Office of Public Health Science.
11 As we speak -- Dan could answer this,
12 too -- we have a team of people that we're assembling
13 that beginning at the implementation of the change
14 will review each set result as it's complete. And
15 they'll look at the number of positive sets, and
16 they'll look at the number of serotypes of human
17 health concern in that set.
18 And this team is going to recommend based
19 on certain criteria a pattern for rescheduling. So
20 some establishments could get, you know, re-sampled in
21 a very quick period of time. Others that have either
22 booked low levels or low levels of human isolates will

1 change. We -- certainly, you know, to the
2 seasonality, I mean we expect reduction all across the
3 year. And at the end of the year, we'll sort of be
4 evaluating.
5 Now, it will be -- the one thing I didn't
6 mention is we posted the 2005 results. That is the
7 last year of having any data in that format, because
8 as we're shifting our resources to focus more on
9 establishments where there are higher levels of public
10 health concern due to the serotypes, we are going to
11 change the nature of our data. So we are losing that,
12 but we think it's a good decision to make.
13 DR. RAYMOND: I want to add a couple other
14 things. This is Dr. Raymond, for the record.
15 Felicia, when I go eat chicken, I don't
16 want to worry about which plant it came from or what
17 month the chicken was slaughtered; I want to know that
18 chicken is safe. And so our goal is not to have that
19 S curve that Loren likes so much as a statistician.
20 We want a flat line.
21 And I think you've already seen -- when we
22 were looking at Loren's bar graphs, we looked at the

1 last two quarters. Maybe perhaps we've seen -- it's
2 too early to say this, but, hopefully, we've seen a
3 change. Hopefully, the industry has created some
4 changes that will drop those numbers down and we won't
5 see that seasonality.
6 The second thing. Not to steal thunder
7 from tomorrow, but when we're doing these sets, every
8 time we have a positive culture, we will be letting
9 the plant know. Rather than wait until after the 53
10 culture samples have been taken and then scrub those
11 for a month and then tell them, they're going to know
12 right off the bat that if they have positive, you
13 know, tests the first four out of five, they're
14 probably looking at another set to be done very
15 quickly. So they'll start making changes right then,
16 hopefully, rather than three months down the road.
17 So I think your question is a very good
18 one. I mean I agree with you. I would not want to
19 get tested in October if I had a choice of May or
20 October if I was a plant -- with the current
21 statistics. But we want to change those.
22 DR. ENGELJOHN: We do have our

1 presentations ready. So I'll hold off on more
2 questions. But I --
3 Dr. Altekruse, a question was asked. And
4 if we can, we'll get the person who asked the question
5 to just re-ask the question.
6 (Pause.)
7 MS. JOHNSON: Dr. Altekruse, you had in
8 your slides -- on one slide that 50 percent of
9 isolates were those that were common human serotypes
10 in a set. And then in a subsequent slide, you
11 mentioned that there were usually less than two
12 isolates of those, which is far less than 50 percent.
13 I obviously am not understanding the difference in
14 the way the slides --
15 DR. ALTEKRUSE: Oh. The 50 percent is the
16 ratios of all *Salmonella* isolates that are those human
17 serotypes. Now, a lot of A sets have zero *Salmonella*
18 in them. That's not an uncommon finding. So the
19 average number of human serotypes is two, and that
20 would suggest that, you know, the average is somewhere
21 in the four to six range. There's some variability
22 around it. But there are some plants that have many

1 more -- you know, they're skewed out to the right with
2 30 *Salmonella*-positive tests. And one of those --
3 every single one of those *Salmonella*-positive tests
4 was a human health serotype.
5 But the -- what that slide was trying to
6 portray was, How many human serotypes can you
7 typically expect to see in a set? And the answer to
8 that is very few. Typically, you know, 50 percent of
9 sets have two or fewer in them. So -- and but the
10 averages -- you know, they don't express the ranges.
11 So that average -- the overall average
12 would include all the sets that have zero and the few
13 sets that have 30. And so the total average for all
14 *Salmonella* would be, you know, in the range of about
15 six isolates.
16 MS. JOHNSON: Thank you.
17 DR. ENGELJOHN: Okay. I just heard a cell
18 phone. So I want to make sure that everyone has
19 turned off their cell phones. I haven't heard many
20 yet.
21 And so that we can keep our conference
22 line, we're going to start out now with our second

1 part of this morning's presentation with Dr. Laura
2 Hulsey. She is a veterinary staff officer at our
3 technical service center in Omaha, Nebraska. She has
4 her expertise in poultry slaughter, and she received
5 her degree in veterinary medicine from Oklahoma State
6 University and had a small animal practice in
7 Washington state, as well as working a private
8 practice in Jackson, Wyoming. Please welcome Dr.
9 Hulsey.
10 (Applause.)
11 DR. HULSEY: Thank you. My presentation
12 today is going to be an overview of poultry slaughter,
13 and this is a step-by-step --
14 You can't hear me?
15 (Pause.)
16 DR. HULSEY: Okay. So this, my
17 presentation, will be an overview of poultry
18 slaughter.
19 (Laughter.)
20 DR. HULSEY: Again and again? Okay.
21 (Pause.)
22 DR. HULSEY: And this will take us step by

1 step --

2 (Laughter.)

3 (Pause.)

4 DR. HULSEY: -- okay -- to set us up for

5 the presentations that follow today.

6 The information for the concerns and the

7 controls came from a literature review that we did at

8 the tech center over the last year, and the focus will

9 be on *Salmonella*. Each step in the process will be

10 covered by about three slides, an in-plant picture

11 that's typical of that process and a slide

12 highlighting concerns at that step for *Salmonella* and,

13 also, a slide that lists possible controls for

14 *Salmonella*.

15 We'll try to point out at each step where

16 *Salmonella* can be introduced, amplified, decreased and

17 where it has been found at the highest levels and some

18 of the factors that influence the levels at these

19 steps. And we'll do that briefly, because each of the

20 presentations today delve more deeply into those

21 subject areas.

22 This slide review that we have done is

1 less formal in structure than Dr. Wills has planned,
2 but it has provided a foundation for identifying
3 decision criteria for risk-based inspection systems,
4 and it will pull together information from the papers
5 that describe the interventions and best practices.
6 Those interventions and best practices we are trying
7 to capture in compliance guidelines for use by the
8 industry, and especially for small and very small
9 plants.
10 Okay. So as I said, step by step: Live
11 receiving and hanging, stunning and bleeding,
12 scalding, feather removal, evisceration and chilling.
13 Okay. This is a slide that Dr. Fisher at
14 the technical service center put together. And the
15 purpose of the slide is to show the general trend of
16 *Salmonella* levels through the slaughter process, and
17 it's pretty much what you would expect.
18 He took the averages and found a mean
19 prevalence for *Salmonella* at each process step. This
20 was taken from 23 different research papers. So high
21 at receiving, lowest numbers at scalding, highest at
22 feather removal, moving on down through evisceration

1 and chilling. So we would hope for the lowest levels
2 at chilling. In this particular group of papers, we
3 didn't see that.
4 Okay. So this is a typical picture of the
5 live receiving where we stage the trucks or -- where
6 you stage the trucks and unload. Some of the concerns
7 at live receiving are, of course, the high level of
8 *Salmonella* coming into the plant. The incoming loads
9 can overwhelm in-plant interventions, and they are
10 carried forward to the subsequent steps.
11 In one study that we looked at, there were
12 feather samples recorded at 6.7 logs per gram, and
13 skin samples at 5.9. And as many of you know, the
14 crop has a large concentration of the *Salmonella*,
15 along with the cecum, colon and the cloaca.
16 Controls that can be implemented at live
17 receiving are, of course, feed withdrawal times and
18 coop sanitation and cleaning programs, also unloading
19 and holding area sanitation programs. The holding
20 times, of course, affect the number or the amount of
21 litter ingestion, as do employee traffic patterns in
22 and out of the facility.

1 And I'm sure that you've all had the
2 experience of the live hang employee coming in through
3 the back door of the plant while a white coat comes
4 out of the other door. So traffic patterns can get to
5 be a problem, bringing contamination into the plant.
6 Another factor is the air flow that moves
7 into the plant from live hang. You want a positive
8 flow from inside to outside.
9 The next step is stunning and bleeding, a
10 typical picture of that electrical stunning. Concerns
11 at this step are that the immobilization causes
12 voiding of feces and further contamination of the
13 carcass.
14 We had one study that we looked at that
15 made a comparison of the withdrawal times. And they
16 had a table that showed that the volume of excreta
17 increased as the feed withdrawal time increased. And
18 therefore, shorter withdrawal times may lead to a
19 lower percentage of the broilers producing an
20 excretion. That contamination at stunning, of course,
21 is carried through forward to the scalder and the
22 picker.

1 This is a shot of a scalder without a
2 counter-current flow. And a scalder that doesn't have
3 a good flow to it, of course creates a bath of cross-
4 contamination. The best idea is to have a continuous
5 flow of water that moves the contamination and the
6 feces away from the carcass instead of along with it.
7 Some more of the concerns that we have are
8 that it washes much of the dirt and feces off, more
9 microorganisms are removed during scalding than in any
10 other step, but they affect the quality of the scald
11 water.
12 *Salmonella* and *Campylobacter* are the most
13 common organisms identified. We looked at a study
14 that 75 to 100 percent -- of the samples of the pre-
15 scalding identified 75 to 100 percent *Salmonella*
16 prevalence. And of course, they accumulate over the
17 shift.
18 Okay. Scalding controls. Brush systems
19 are one that we saw throughout the literature review
20 that had a good impact -- also, rinses before the
21 scalder, counter-current flow and multi-tank systems.
22 In one three-stage system in a multi-tank system,

1 there was a report of 3.4, 2.0 and 1.2 logs CFU per
2 mil. coliform reduction.
3 Temperature is important -- and pH. The
4 literature papers reported on both high pH and low pH
5 that decreased the number of microorganisms. In one
6 report, they decreased the pH to 4.3 with 1.1 percent
7 acetic acid and increased the death rate of *Salmonella*
8 Newport and Typhimurium by 91 percent in the scald
9 water.
10 This is a bird scrubber that I just got a
11 picture of before we came here -- at a plant, that
12 they installed. And you can see quite a significant
13 difference from before and after. I don't have any
14 numbers on this, but I hope to have them before we do
15 the compliance guidelines.
16 The next step is typically the highest
17 amount of cross-contamination and the highest level of
18 *Salmonella* -- at feather removal. So we've seen up to
19 100-percent incidence of *Salmonella* at the feather
20 removal step. The picking fingers and the feather
21 follicle are implicated most often at this step and
22 may drive *Salmonella* into the follicle. Also, the

1 cloaca is emptied by the rubbing action of the picking
2 fingers, which increases the contamination.
3 The controls at this step, from the
4 literature, included the National Chicken Council's
5 GMPs that recommend preventing the build-up of
6 feathers, continuous rinses for the equipment and the
7 carcasses and, also, equipment adjustments to minimize
8 cross-contamination.
9 One study talked about if contaminated
10 water from the scald step's driven into the follicles
11 by the picking fingers and then it moves forward into
12 the chiller. It's the chiller cold water, and the
13 follicles contract and hold that contamination in
14 there.
15 Other interventions at this step include
16 post-feather-removal rinses at 160 degrees Fahrenheit,
17 chlorine rinses, acetic acid rinses, hydrogen peroxide
18 rinses and other adjuvants; these produced mixed
19 results.
20 Okay. We're going to move into the
21 evisceration step. The crop removal seemed to be the
22 area of highest concern. The greatest percentage of

1 *Salmonella* was recovered from the crop, also from the
2 ceca, but the highest incidence of carcass
3 contamination occurring at pre- and post-crop, due to
4 rupture and spillage at that step.
5 Okay. One study by Byrd and Hargis in '02
6 recovered marker organisms from broiler crops prior to
7 live hang. They recovered 92 percent pre-crop and 94
8 percent post-crop. So that's a pretty big recovery
9 rate. Also, GI leakage from the equipment and
10 manipulation of that equipment and from the GI tract
11 contaminates the carcasses and the equipment.
12 Controls at this step include, again, the
13 National Chicken Council GMPs, which are -- focus on
14 the most ideal feed and water withdrawal prior to
15 slaughter, the maintenance and the adjustment of your
16 equipment and continuously rinsing and sanitizing
17 evis. equipment. Also, employee hygiene plays a role
18 in this, as do whole-carcass water rinses.
19 Carcass rinses in the studies decreased
20 *Campylobacter*, coliforms and *E. coli*. And you can see
21 a 36-1/2 percent decrease in *Salmonella* when rinses
22 are used, compared to 20.5 percent without; they are

1 not effective against attached pathogens, however.

2 So we have a presentation later today on

3 on-line re-processing in *Salmonella* that will delve a

4 little more deeply into it, but these are some of the

5 on-line reprocessing methods that are reviewed in the

6 papers. At least 23 parts per million Free Available

7 chlorine decreased the incidence from 5- to 2 percent

8 in one study. 10 percent TSP decreased 1.3 logs. And

9 then we went through several others here that we'll

10 touch on later today: The 2 percent lactic acid, and

11 so on.

12 The last step for an intervention to be

13 applied is the immersion chiller. Concerns that were

14 covered in our paper are that lipids are 84 to 98

15 percent of the filterable solids in the chiller; they

16 consume the available chlorine, and they can protect

17 the microorganisms.

18 It's also the major site of cross-

19 contamination between positive and negative flocks,

20 and it can increase the incidence by 20.7 percent on

21 average. *Salmonella*-negative broilers will remain

22 negative if you don't put a flock of *Salmonella*-

1 positives through there first. So that says a lot for
2 tracking pre-harvest before the birds get to the
3 plant.
4 Okay. Other controls. The National
5 Chicken Council, again, recommends proper water
6 replacement, quality and temperature. The chiller
7 health also depends on the pH generally of 6.5 to 7.5,
8 Free Available Chlorine at one to five parts per
9 million, and trying to minimize organic solids with a
10 high flow rate, counter-current direction and
11 cleanliness.
12 So the bottom line over the process
13 through all these steps is that the appropriate
14 interventions applied effectively can decrease
15 *Salmonella* in the slaughter process.
16 I don't know if I mentioned just as a
17 final note at the introduction as things were trying
18 to get going here that we are working on the
19 compliance guidelines that should be out shortly and
20 will capture the information that we have in this lit
21 review. Thank you.
22 (Applause.)

1 DR. ENGELJOHN: Thank you, Dr. Hulsey, for
2 that overview of what we're going to spend the rest of
3 today and tomorrow morning talking about with regard
4 to the production process and slaughter and [inaudible
5 due to failure of in-house PA system] processing.
6 Our next presenter is going to give us
7 some lessons learned, I think, from the perspective of
8 what we've found when we as an Agency conduct our food
9 safety assessments. Dr. Petersen is now the assistant
10 administrator for the Office of Field Operations; he
11 took that role in December of 2005. He recently had
12 been the deputy administrator for the Office of Field
13 Operations.
14 He served here in the headquarters. And
15 then, prior to that, he worked as a senior staff
16 officer in our Office of Public Health Science. He
17 was in private practice for [inaudible due to failure
18 of in-house PA system] time, as well, and has both a
19 doctor's of veterinary medicine and a master's in
20 public health, as well.
21 Please welcome Dr. Petersen.
22 DR. PETERSEN: Okay. Thanks, Dan.

1 Actually, before I get started, I want to
2 circle back -- I can't believe I'm the only one who
3 missed it -- to Dr. Wills' bio. He works at
4 Mississippi State. And I have an administrator who's
5 a big-time Mississippi State grad, and I thought he
6 did some training in Nebraska. And I have an under
7 secretary that -- if anybody thinks he's not a major-
8 league Corn-husker, they need to spend about five
9 seconds with him.
10 So, Dr. Wills, where ever you're sitting,
11 I think you kind of hit all of the numbers. And so
12 very good.
13 VOICE: And Iowa State.
14 DR. PETERSEN: And Iowa State. Boy, he's
15 all over the place.
16 (Laughter.)
17 DR. PETERSEN: Okay. What I want to talk
18 about is things we're finding in our comprehensive
19 food safety assessments that are done by the folks in
20 the Office of Field Operations. And food safety
21 assessments -- of course, we have a directive out on
22 that now. The directives are information that apply

1 to our folks. That's at Directive 5100.1. That
2 outlines the food safety assessment methodology.
3 The food safety assessments are basically
4 a comprehensive look by specially trained people.
5 They look at -- let me hold this.
6 (Pause.)
7 DR. PETERSEN: Okay. The food safety
8 assessments are conducted by specially trained people,
9 as most of you know, and they look at the structure
10 and design of the program and, fundamentally, What is
11 the theoretical basis for your program, what is the
12 rationale for it, and are you delivering that in your
13 food safety system, and are you making necessary --
14 adjustments when necessary when things go awry in your
15 process.
16 I want to start with a few definitions to
17 make sure we're all on the same page. A food safety
18 hazard: Any biological, chemical or physical property
19 that may cause a food to be unsafe for human
20 consumption. Your HACCP analysis, which is conducted
21 to determine the food safety hazard [inaudible due to
22 failure of in-house PA system] occur in the production

1 process and identify the preventive measures that can
2 be applied to control those hazards.
3 And then "reasonably likely to occur" are
4 for those hazards where the establishment would
5 establish controls either because they've historically
6 occurred or they're reasonably likely to occur in the
7 absence of those controls. Okay? Those are all
8 regulatory references not to be confused with
9 statutory definitions, statutory definitions for
10 adulteration.
11 And since we're here to talk about
12 poultry, we're working within the Poultry Products
13 Inspection Act. There are multiple definitions of
14 adulteration, but I really just pulled out two that
15 are the most germane for us here today.
16 What we call a G-1, that's a particular
17 section of the Act, and it basically has two parts to
18 it: "Either bears or contains any poisonous or
19 deleterious substance which may render it injurious to
20 health," or, a little more commonly, in case it's not
21 an added substance, "The article," meaning the poultry
22 product, "shall not be adulterated under this clause

1 if the quantity of such substance in or around the
2 article does not ordinarily render it injurious to
3 health."
4 So we've got a couple things in the second
5 part there. It's not an added substance -- the
6 quantity -- and not ordinarily injurious to health.
7 For your friends on the beef side, *E. coli* 0157 --
8 this is where our authority drives for that. No one
9 would consider an *E. coli* infection anything other
10 than injurious to health.
11 So we don't quite have that with poultry.
12 Some use the reference, "Naturally occurring." But I
13 think, theoretically, at some point -- not today -- we
14 could have a *Salmonella* that has such hospitalization
15 rates, high attack rates, high infectivity rates, high
16 case fatality rates, and then I may wonder if that
17 *Salmonella* is not ordinarily injurious to health.
18 So not today, but that's a possibility if
19 some of the virulence factors continue to evolve.
20 Really more likely what we're dealing with
21 and what we'll talk about as we get into these food
22 safety assessment discussions is the G-4 reference,

1 "Insanitary Conditions": "Where it's prepared, packed
2 or held under insanitary conditions whereby it may
3 become contaminated with filth or where it may have
4 been rendered injurious to health."
5 Insanitary practices such that I find the
6 product adulterated, meaning I can't put the marks of
7 inspection on that product. Adulteration not to be
8 confused with food safety hazards. Two different
9 references.
10 Okay. So what did we find? Food safety
11 assessments -- these are the assessments. We've done
12 about 31 of them or -- 31 that we've looked at that we
13 have initiated since October 1, 2005. Briefly, the
14 results. Ten of which led to a notice of intended
15 enforcement, basically, where I had questions about
16 the adulteration status of that product.
17 Interestingly, having heard Dr. Raymond's
18 first slide earlier, where he -- one of his early
19 slides showed about 30 percent of plants were in that
20 Category III. These aren't the same plants, but,
21 interestingly, about 33 percent of these assessments
22 in plants led to an NOIE.

1 Eleven non-compliance records were issued,
2 meaning there was some miscellaneous non-compliance,
3 but not sufficient for me to pursue an enforcement
4 action. Nine had no action, so were within compliance
5 of the regulations. And one had a 30-day re-
6 assessment letter issued. Yes, we do still do 30-day
7 re-assessment letters. Those are not regulatory
8 issues. They basically mean I have a question about
9 some supporting documentation that you're unable to
10 provide or explain, and we give you a period of time
11 to describe that.
12 What I want to do with these next slides
13 is -- of course, we've got some folks in the room who
14 are quite familiar with our regulatory frame work, and
15 we have others who are less so. And so I've
16 characterized these findings kind of in between those
17 two levels of knowledge.
18 And general observations. And we'll work
19 through these observations, not necessarily synonymous
20 with the regulatory requirements, but I think you'll
21 see some common themes as we go forward.
22 Many establishments do not sufficiently

1 identify *Salmonella* as a food safety hazard reasonably
2 likely to occur, and, yet, when you look at their
3 supporting documentation, all of the supporting
4 documentation says it is likely to occur. But they do
5 identify microbial growth, enterobacteriaceae,
6 pathogenic microorganisms, as the hazards.
7 Inadequate consideration of incoming
8 *Salmonella* loads. I see references to on-farm
9 practices, on-farm vaccination protocols, but more
10 listed just for the fact of listing them rather than
11 using that information or integrating that information
12 into their thought process.
13 Process control steps. Inadequate
14 consideration of incoming *Salmonella* loads and process
15 control steps that affect *Salmonella* levels. We just
16 heard some of that a minute ago on the last slide.
17 Multiple steps in the process can have multiple
18 impacts, but not considered there at all. And
19 intervention's not validated to address *Salmonella*.
20 Inconsistencies between the hazard
21 analysis and the selection of your critical control
22 points and critical limits. Hazards identified in the

1 hazard analysis. And they're simply identified. No
2 indication on whether they're reasonably likely to
3 occur. No supporting documentation for decisions that
4 the hazard is reasonably likely to occur. It's just
5 there.
6 And for those who are using prerequisite
7 programs at some level, insufficient records to
8 describe what it is they do and how they affect your
9 decision making. And of course, we look to --
10 -- supporting documentation to demonstrate the scientific
11 regulatory basis for your program informs what it is
12 you're trying to implement, what it is you're trying
13 to execute.
14 *Salmonella*. Where it is identified and is
15 reasonably likely to occur at some process steps --
16 that's your hazard analysis -- but did not
17 subsequently indicate where in fact it would be
18 prevented, eliminated or reduced. So we have
19 *Salmonella* that's floating out there. At some point,
20 if it's reasonably likely to occur, where are you
21 dealing with it?
22 Some plants put controls in place and take

1 actions consistent with the hazard, but not indicated
2 as reasonably likely to occur. So addressing it in
3 fact, but not addressing it in the thought process and
4 the design of what it is you're trying to do.
5 And interestingly, CCP has established a
6 control of food safety hazard for a hazard that's not
7 even deemed reasonably likely to occur. That's a
8 difficult concept.
9 Key steps without identified hazards. A
10 couple examples: Biological hazards not identified at
11 processing steps -- that doesn't mean they have to be,
12 but some processing steps, we think, may have some
13 biological hazards; Red-water chilling and some of the
14 chilling steps. And if they're not identified,
15 there's no rationale, no subsequent documentation, to
16 justify how those decisions were reached. You can
17 justify the decisions, but you need to have a basis
18 for it.
19 Off-line steps, whether it be wash-out,
20 fecal air sac, IP cut-up or whatever, where we have
21 incoming birds with a hazard reasonably likely to
22 occur, but, somehow, when we get to the more risky

1 parts of the process, the hazard -- there's no hazard
2 identified or none is specifically referenced. And so
3 what is the thinking and what is the theme cutting
4 through your program? And what is the thought process
5 at each step of the way?
6 Water re-use. You are expected to have
7 measures sufficient to prevent or reduce physical,
8 chemical or biological contamination to the extent
9 necessary to prevent contamination or adulteration of
10 product. So have you considered that? Have you
11 considered the impacts of water re-use on your HACCP
12 plans -- subsequent HACCP plans? That, of course, is
13 a regulatory expectation.
14 No supporting decisions for selection of
15 CCPs and critical limits. You've selected them. You
16 have a critical limit. And here largely we're talking
17 about various temperature critical limits, and, of
18 course, there are some common ones: 36 degrees, 50
19 degrees and 55 degrees. Well, why is that? What
20 is -- what are you targeting? What are you trying to
21 accomplish?
22 No supporting documentation for the

1 monitoring and verification frequency selected for the
2 CCPs. Why are you doing your checks at a certain
3 frequency? What is the basis for it, and what do they
4 tell you? How does it tell you the status of your
5 process on an ongoing basis?
6 That's true where the common controls are
7 involved -- whether it be typical fecal contamination,
8 of course, is a common control -- or temperature
9 controls. What's the frequency for your temperature
10 controls, monitoring and verification, and why do you
11 do it at that frequency?
12 CCP validation. Particularly, of course,
13 involving an antimicrobial intervention, is it
14 effective to reduce the identified hazard at the entry
15 point? And your supporting documentation in your
16 hazard analysis of food safety hazard reasonably
17 likely to occur. Where is it dealt with, and how do
18 you know it?
19 Chlorine identified in the hazard analysis
20 at steps as a control measure to prevent the food
21 safety hazard. However, when you go to those steps,
22 chlorine's not even applied. We need some thematic

1 thinking, some systematic thinking, to what we're
2 trying to design and implement. And then, of course,
3 zero tolerance and temperature are not uncommon CCPs.
4 Corrective actions following a critical
5 limit deviation are not implemented, particularly for
6 temperature controls. Say if your temperature is 50
7 degrees and you exceed it, 51 degrees. Well, that's
8 your critical limit. What are you doing? What should
9 you be doing? Are you doing it on an ongoing basis?
10 And if you don't, you need to look at your system.
11 Corrective actions not documented, not
12 implemented, or the preventive measures were
13 ineffective. And here we'll work mainly through some
14 SSOP issues.
15 Repetitive corrective actions: Clean,
16 sanitize, inspect. Clean, sanitize, inspect. Pre-op,
17 for example. On a -- at some level at some point when
18 that happens with some frequency on particularly some
19 product contact zones, what does that tell you about
20 either the effectiveness or the design of your SSOP
21 program?
22 Repetitive documentation of temperature

1 deviations. Well, it's only one degree, so maybe it's
2 not so bad. Well, it is a critical limit. And if
3 they're exceeded, there are some expectations on how
4 we deal with it.
5 Recurrence of a deviation should inform
6 you of something. And what do you do with that
7 information, and how do you improve your program
8 accordingly?
9 And so all of which leads to not
10 conducting appropriate re-evaluation or modification
11 of the design of, say, your SSOP, or what does it tell
12 you about the execution of your SSOP? You can improve
13 the execution if you think the design, of course, is
14 fundamentally satisfactory. But with repetition, one
15 of those should result.
16 So the next couple of slides kind of pull
17 this all together in a *Salmonella* context. Sanitation
18 performance standards. And these are some common
19 themes particularly but not exclusively in the plants
20 that led to enforcement actions.
21 Just fundamental employee hygiene issues.
22 And we heard that with the last slide. Restroom

1 issues, product handling, hand washing and that type
2 of thing. When supplemented with some pest control
3 concerns -- you know, unaddressed pest control
4 concerns -- cockroaches and that type of thing --
5 superimposed with employee hygiene concerns. And
6 ventilation, of course, refers to condensation
7 controlled in a manner sufficient to prevent the
8 creation of insanitary conditions or product
9 adulteration.
10 Control and equipment and facilities. Is
11 the equipment contaminating product? Is it leading to
12 fecal contamination of product at some ongoing
13 frequent basis, and why is that acceptable? That's
14 superimposed with facility concerns: Pipings with
15 the, you know, insulation displaced over product
16 zones, superimposed with the wall sanitary issues.
17 And so these things tend to multiply when we look at
18 what is being executed in that facility.
19 Water re-use considerations. Of course,
20 an SBS issue. We touched on that.
21 So taking that, the findings in those
22 plants, it doesn't -- I'm not suggesting it needs to

1 be all, but when you start to add in facility
2 questions with ventilation questions with employee
3 hygiene questions.
4 And then development and implementation of
5 your SSOPs. What did you want to do, and is it
6 working? And if not, are you making adjustments?
7 That's maintenance and effectiveness, taking
8 appropriate corrective actions. And what do those
9 corrective actions tell you about what's occurring?
10 And then, of course, appropriate documentation.
11 And so those two things, SPS and SSOP,
12 with what we walked through on some fundamental
13 inconsistencies on what it -- what your program says
14 or does. Hazards without controls. Hazards that are
15 reasonably likely to occur with process steps that are
16 not validated for that hazard.
17 The monitoring frequency. How do you know
18 it works for you? And then validation verification
19 and re-assessment. I thought I heard Dr. Masters say
20 something using the words, You don't have to wait for
21 us. And you don't. These are the issues we're
22 finding, and my strong preference is that you find it.

1 You don't need me to find it. And so you have that
2 opportunity.
3 And then the record keeping. That goes
4 with everything else, to document what your system is
5 delivering.
6 So we heard earlier that *Salmonella*-
7 positives have some reflection on the status of
8 process control, Categories I, II and III, and then,
9 of course, exceeding the performance standard. That
10 was the previous bench mark.
11 And a facility with some perhaps serotypes
12 of human health concern now superimposed perhaps in an
13 establishment with some of these repetitive SPS
14 issues, these SSOP issues, these HACCP issues. And so
15 what is your program? How is it constructed? Does it
16 make sense? Is it consistent?
17 And this is kind of the -- I won't call it
18 a hierarchy, but this is kind of the sequence of
19 events that can be an entry point for us to look at
20 your food safety system. And when we've done that --
21 what I outlined is what we typically find.
22 And I just, I think -- I know you can

1 design and implement a HACCP system. And so we need
2 to look at the thought basis behind that and look at
3 the scientific basis and get it done.
4 And that's it.
5 (Applause.)
6 DR. ENGELJOHN: Okay. Can you hear me on
7 this microphone?
8 (Pause.)
9 DR. ENGELJOHN: We're going to have to
10 find a microphone that we can put down there that we
11 are sure that you can ask questions from.
12 But if I can have the members from this
13 morning's presentations come up to the table and --
14 we'll take some questions from the audience. And then
15 I will also ask whether or not there are any questions
16 from our telephone callers. So there were --
17 Dean, I saw you up here.
18 Somebody needs to come up to the
19 microphone.
20 (Pause.)
21 DR. ENGELJOHN: Please state who you are
22 and your association, and ask your question.

1 MR. BENSON: Okay. Mike Benson with
2 Jennie-O Turkey Store. I'm asking this question as a
3 consumer, though, of poultry products. My question is
4 for Dr. Masters.
5 In the presentation Laura had earlier, it
6 was showing the use of a lot of chemicals in the
7 processes. And it would appear from the agenda and
8 from the discussion that the FSIS is recommending or
9 implying the recommendation that we should be applying
10 more and stronger acidifiers, alkalizers, oxidizers,
11 sanitizers, quaternizers and more of these chemicals
12 to our food products.
13 My question is, Dr. Masters, do you think
14 that these are safe? And is that the direction that
15 FSIS is looking for processing companies to go forward
16 with?
17 DR. MASTERS: I think that in my
18 presentation, I indicated that there is --
19 This is Barb Masters. There is a variety
20 of options that you can use to control *Salmonella* in
21 your establishment. I'm aware of some establishments
22 that use no chemicals in controlling *Salmonella*. I

1 think that Dr. Hulsey was presenting to you some of
2 the interventions that are available. I think Dr.
3 Wills presented to you that they are doing a
4 systematic approach to some of the interventions that
5 are available; many of those will be non-chemical
6 interventions, hopefully.
7 And so I'm certainly not suggesting to you
8 that the only approach that you can use is to
9 continuously apply more and more chemical
10 interventions in your process. I think you need to
11 start at pre-harvest and look at what you can do pre-
12 harvest.
13 You need to look at control in your
14 process. And I think that's -- what Dr. Petersen was
15 trying to say to you is you need to look at every part
16 of your process, starting with your employee hygiene,
17 looking at your SSOPs, looking at your sanitation
18 process control and looking at your control of fecal
19 contamination. And you need to look at all of those
20 things in combination.
21 What Dr. Hulsey was providing to you was
22 some of the areas that you might look at some

1 interventions, and that would be your option to look
2 at which of those might be useful in your
3 establishment. But certainly, we're not suggesting
4 that you need to only control *Salmonella* through the
5 use of increased chemical controls in your
6 establishment.
7 DR. ENGELJOHN: Yes. Next question.
8 MR. DANILSON: Thank you, Dan.
9 Probably to Loren, but I don't know. It
10 might be --
11 DR. ENGELJOHN: Who are you?
12 MR. DANILSON: Pardon?
13 DR. ENGELJOHN: Who are you?
14 MR. DANILSON: Oh. Thank you, Dan. Dean
15 Danilson with Tyson.
16 I found it very interesting -- the data
17 that you showed in your red zone plants, the low --
18 the disparity of the number of A sample sets that have
19 been taken on those red zone plants relative to middle
20 zone and green zone. As your efforts in 2006 and 2007
21 increase in sampling activity on those red zone plants
22 relative to the data trend that we're looking at, we

1 are very likely to see a continued increase in
2 *Salmonella* incidents as reported in your trend
3 analysis, because of the bias that will push back up
4 there, which will not look good to whoever is looking
5 at that.
6 And I -- we need to be aware of what that
7 next two years is going to show us on that trend. And
8 I hope that increasing trend doesn't take away from
9 our interest and desires for promoting and making
10 incentives for the green zone plants and take away
11 from those efforts. So, you know, I just ask that
12 that be kept in mind.
13 MR. LANGE: I have a response to that.
14 I -- one thing we will do is have the staff at FSIS
15 looking at all different ways in which we can display
16 the data.
17 And just thinking off the cuff, it may not
18 be something we can do, but we also do have -- through
19 our animal disposition reporting system, we have
20 numbers of carcasses slaughtered. We could generate a
21 new statistic like weighting the last set by
22 production weight for each plant.

1 I'm just throwing out an idea, but we'll
2 consider a lot of different ways of presenting the
3 data because, as I said earlier, this ends that era of
4 1998 to 2005 in terms of at least having some
5 consistency in what we've been posting on the web.
6 DR. RAYMOND: And I'm going to add to that
7 Loren.
8 In that we've talked about this and we
9 recognize very clearly that the trends may get skewed,
10 we also recognize from some of the people who advise
11 us from the outside that we shouldn't even be using
12 these as trends because it's not a statistically
13 significant sample. As Loren said right up front at
14 the very start of his presentation, this is not
15 intended to be a statistically significant sampling of
16 the product; it's to sample perhaps verification and
17 to judge individual plants, but it doesn't say what
18 the *Salmonella* load really truly is from the product.
19 That said, we've also discussed changing
20 how we sample and that it would also skew the
21 statistics even more significantly. Instead of always
22 sampling on the first shift, I strongly believe we

1 need to look at the sampling of other shifts. And
2 that will change our data, also, but we won't know how
3 much it'll change that until we can have a
4 statistically significant number of first shift
5 samples and second shift samples in the same plants to
6 find out what the risk of a carcass is going through
7 the second shift versus the first shift.
8 I mean those are issues that have been
9 raised to me, and I agree that they're issues that
10 need to be addressed. So I don't think we'll be able
11 to say anything from trends in the future, you know,
12 looking backwards. We'll have a new trend data, and
13 we'll use that.
14 But very most importantly is not how many
15 sets are positive or how many carcasses test positive;
16 it's how many people test positive. And so we still
17 have that baseline data going: The number of human
18 beings who have culture-proven *Salmonella* infections
19 per year. That's really the bottom line for me. I
20 mean it's important -- what's going on in the plants,
21 but the bottom line is what's going on when people eat
22 the product.

1 DR. ENGELJOHN: And I'll just point out,
2 too, just because we haven't mentioned it yet but we
3 will later, that we are in fact going to start another
4 national baseline study, which is statistically
5 designed, that's going to start this year, as well.
6 So it will serve as a basis to give us a more accurate
7 picture of true prevalence.
8 Could we have the next question?
9 DR. BYRD: Ken Byrd, with Mionix.
10 Actually, I have a question for Dr. Hulsey.
11 You had mentioned something about a study
12 where in the scald the pH was -- I believe you said --
13 4.1 and you got a certain reduction in *Salmonella*. I
14 didn't get that number. Could you repeat that,
15 please?
16 DR. HULSEY: That's a good question, and
17 I'll have to look. And I'll get back to you on that.
18 DR. BYRD: Okay. Thank you.
19 DR. HULSEY: Okay.
20 DR. ENGELJOHN: I think -- could I ask the
21 operator? Are there any questions being queued by the
22 telephone callers?

1 (Pause.)

2 DR. ENGELJOHN: While we're waiting for
3 that, go ahead and ask your question.

4 DR. O'CONNOR: Bob O'Connor from Foster
5 Farms, and I had a question for Mr. Lange.

6 The seven-year data that you looked at for
7 what we term seasonality -- basically, you broke it
8 into quarters. How many companies within those seven
9 years were tested during the same quarter over those
10 seven years? So if I was Company A and it was Year
11 One and I was tested in Quarter 3, did I get tested in
12 that same quarter the next year? If I was tested five
13 times, how many times did I fall into the same
14 quarter?

15 MR. LANGE: There is a pattern of when --
16 that sort of grows from when we started the program in
17 1998, where it initiated. We scheduled everybody that
18 was a large establishment early in the year. And then
19 by only scheduling one set a year, we did get in the
20 pattern that if you were large, in the early years,
21 you tended to get sampled in that March/April/May/June
22 period.

1 In the last couple of years, we've been
2 trying to sort of, as you can see, diversify that a
3 little bit. And you can see we actually have the
4 largest number of samples last year in the fourth
5 quarter. So it did happen.
6 I can't, you know, tell you -- but, you
7 know, there's a lot of plants that probably found
8 themselves getting sampled at the same time of the
9 year each year. But that will change. Now, so I
10 mean -- but that won't happen in the future, because
11 rescheduling will be based on, you know, public health
12 considerations.
13 I'd like to add one other comment to what
14 Dr. Raymond said. When this issue of a second set
15 came up, I went back and looked at the report of the
16 original 1994-to-1995 baseline that established the 20
17 percent. All those samples were collected on first
18 shift samples. All those samples were Monday through
19 Thursday.
20 So in trying to do studies, I -- we have
21 both microbiologists and statisticians. And they
22 don't always come from the same perspective as to when

1 samples get collected and when samples get shipped,
2 because the microbiologists always want consistency
3 and the statisticians would like it spread across the
4 board. And it's -- you have to find that common
5 ground between the two.
6 DR. O'CONNOR: Okay. I think the reason
7 I'm asking the question is because we kind of deemed
8 that seasonality. And I think from a biological
9 standpoint, if we deem it seasonality, we might try to
10 look for a biological reason for that. But if it's
11 not truly seasonal, if it's more a scheduling incident
12 or coincidence, then I probably wouldn't concentrate
13 on the seasons of the year.
14 MR. LANGE: Yes. There --
15 DR. O'CONNOR: For interventions. That's
16 what I'm saying.
17 MR. LANGE: You would have to do a very
18 sophisticated analysis of trying to account for all
19 the potential variables. Was it the plants that were
20 getting scheduled in that April/May/June period that
21 just always kept that down, or was it a real change?
22 But I -- you know, there's probably both happening,

1 like anything in the real world.
2 DR. ENGELJOHN: Any other questions?
3 MR. POTTER: Bill Potter with George's.
4 My question is about upcoming baseline studies and
5 maybe future *Salmonella* standards and how they're
6 developed.
7 My question is, In the upcoming studies,
8 has there been any consideration of the enumeration of
9 the *Salmonella* colonies on the carcass? Most of us in
10 research are really interested in the enumeration.
11 In other words: Is the carcass containing
12 one DNA strand of *Salmonella*, which would make it
13 positive under the current testing, or does it
14 contain -- I don't know -- a million? To us -- many
15 of us, that is of significance, and it would be good
16 to know. Is that being considered in future studies?
17 MR. LANGE: Yes. As Dan just mentioned,
18 we are in the final process of designing a new broiler
19 baseline. We will quantitate *Salmonella* and
20 *Campylobacter* on all samples. And we have -- over the
21 last year, we have had the National Advisory Committee
22 on Microbiological Criteria for Foods recommend a new

1 *Campylobacter* method that we will use in our baseline
2 studies, and it is currently undergoing validation at
3 the contract lab that will do our baseline study.
4 So we hope to start a shakedown period --
5 we hope -- by the end of March. But if not, we should
6 start in April of this year for young chickens. And
7 probably then the next thing I'll have the staff work
8 on is a turkey carcass baseline.
9 MR. POTTER: Okay. Is -- excuse me. If I
10 may, is the enumeration just for *Campylobacter*?
11 MR. LANGE: And *Salmonella*.
12 MR. POTTER: And they'll -- okay. Thank
13 you.
14 MR. LANGE: They will enumerate for
15 *Salmonella*, too.
16 DR. ENGELJOHN: This is Engeljohn. I'll
17 just add a little more to that.
18 We will also be looking at some [inaudible
19 due to failure of in-house PA system] organisms. And
20 I would also point out that we've changed the
21 structure of this baseline. We traditionally had done
22 baselines in which we looked at the point most

1 immediate to sale or to the consumer, meaning the
2 post-chill sample. You should expect this time we
3 will do a sample at re-hang before the carcasses are
4 chilled and we are going to do it at post-chill in the
5 same facility.
6 And as we get better and have more ongoing
7 baseline studies, which we do intend to do for all the
8 classes of products, we will add more points in the
9 production process by which we will take those samples
10 so that we can see what's happening in the entire
11 production process. But this baseline study is
12 designed to have two points in the production process
13 by which we pull samples.
14 MS. NESTOR: Felicia Nestor, Food and
15 Water Watch. Loren, did I understand you to say that
16 the original baseline was done first shift, that most
17 of the samples were done first shift Monday through
18 Thursday?
19 MR. LANGE: Yes. If you go on our web
20 site and read -- what is it -- June '94 to '95, the
21 actual baseline is posted on there. And there's a
22 sentence that says -- it says, All samples were first

1 shift and Monday through Thursday. So it was -- back
2 then, it all tied in to Fed-Ex delivery schedules.
3 And we were shipping carcasses back then, and the
4 microbiologists all wanted the carcasses shipped the
5 same day they were sampled.
6 MS. NESTOR: And what months did that --
7 MR. LANGE: That would have been across --
8 that would have been designed to so it weighted
9 production by month. So as a baseline study, it is
10 based on production volume to get your national
11 product estimate. So if production was actually
12 changed, you would expect more samples in the month
13 where production was higher. And I don't know if
14 you'll see that fluctuation in production. But it
15 would be evenly across all months, yes.
16 MS. NESTOR: So it's a one-year baseline?
17 MR. LANGE: Yes. It was one year, yes.
18 MS. NESTOR: Okay. And were all sizes of
19 plants tested? Because -- I know in ground beef, the
20 very small plants weren't tested.
21 MR. LANGE: In all except the very
22 smallest. The sampling frame, I think, covered 99.9

1 percent of production. So they were all subject to
2 sampling, but then the number of times -- the way the
3 baselines in the '90s were done, the number of times a
4 plant would be sampled was entirely proportional to
5 its production volume or, at least statistically,
6 that's how the number of samples was derived.
7 MS. NESTOR: Thank you.
8 DR. ENGELJOHN: While we have the
9 gentleman coming up to the phone, could I ask the
10 operator again?
11 Has anyone identified that they want to
12 ask a question?
13 FEMALE VOICE: Yes. [inaudible due to
14 failure of in-house PA system].
15 DR. ENGELJOHN: Loren, were you able to
16 capture that?
17 MR. LANGE: I captured --
18 DR. ENGELJOHN: Could you repeat the
19 question? And we'll just --
20 MR. LANGE: Yes. The second part of it
21 was, Is -- I tried to show both the seven-year data,
22 the 2002 to 2004, I've looked at 2001 to 2003. That

1 pattern of broilers being low in April, May and June
2 and higher in October, November and December appears
3 to have been consistent across all seven years through
4 2004.

5 And the point I was trying to make is it
6 really changed in 2005. And it changed because the
7 third and the fourth quarters actually ended up being
8 the lowest.

9 Now, that answers part of it. I didn't
10 catch the whole thing.

11 DR. ENGELJOHN: The first part of the
12 question that I think I heard was also -- that there
13 appeared to be the 2.8 percent increase from last year
14 to this year, I think, was the question -- and that it
15 appeared to be rather significant. Is that true?

16 MR. LANGE: We don't have the ability to
17 sort of test to say, "Was the increase from 13.5 to
18 16.3 a statistically significant change," because the
19 data isn't set up so that you can run that type of
20 test. But we -- certainly, it was a larger increase
21 than we've seen in previous years.

22 DR. RAYMOND: This is Raymond. And I

1 would say, Caroline, what bothers me the most is that
2 it wasn't a one-year increase on the carcasses on the
3 broilers. It has been four years in a row or three
4 years in a row that it has gone up. Starting four
5 years ago, each year has shown an increase. And I
6 don't think you can deny, if you look at a four-year
7 trend, that there's not a problem.
8 DR. MASTERS: And this is Barb Masters.
9 And I guess that was the real reason we started these
10 meetings last August, and that's why we're here. And
11 that's -- what we're hoping to do with these meetings
12 and the sharing of information and with the policies
13 that we put out that will be in the Federal Register
14 is to turn that trend the other direction. And
15 that's -- what we're hopeful we saw in the last two
16 quarters is the trend going the right direction and
17 that we're, you know, challenging the industry. And
18 we're optimistic that we can start seeing the numbers
19 go the other direction.
20 We do believe that it's a trend in the
21 wrong direction. Whether it's significant or not
22 significant, it is in the wrong direction, and we

1 believe the data needs to go the other direction. And
2 we're optimistic that with -- the policy changes that
3 we have put -- that we're recommending and that we're
4 putting in place are the kinds of things that can make
5 that trend go the other direction.
6 DR. ENGELJOHN: Do I have other questions
7 here in the audience?
8 MR. YANCY: Al Yancy, Gold Kist. This
9 question's for Dr. Petersen.
10 I think I heard you say that there had
11 been 31 FSAs since October 1 of '05. The -- maybe the
12 only question is, Are those -- is that statistic --
13 does that speak only to targeted FSAs for failed A
14 sets, or is that routine FSAs, or is that some mix
15 thereof?
16 DR. PETERSEN: We gave the district
17 managers just some general guidance. They didn't
18 really target for A sets [inaudible due to failure of
19 in-house PA system] performance. [inaudible due to
20 failure of in-house PA system] district managers were
21 aware of some plants where [inaudible due to failure
22 of in-house PA system] they had some lasting

1 *Salmonella* control over time. And at the starting
2 point, that's where we want [inaudible due to failure
3 of in-house PA system].
4 MR. YANCY: Okay.
5 DR. PETERSEN: And so they had [inaudible
6 due to failure of in-house PA system] the ones that
7 [inaudible due to failure of in-house PA system] their
8 minds. And once we better characterize the food
9 safety assessment methods in a, you know, *Salmonella*-
10 [inaudible due to failure of in-house PA system]
11 commodity in all establishments, that's really
12 [inaudible due to failure of in-house PA system].
13 MR. YANCY: The reason I'm asking, as a
14 follow-up is I wondered if there was any direct
15 correlation between the ten NOIEs that were issued and
16 *Salmonella* incidence in those plants. In other words,
17 did the ten plants that got NOIEs -- did they -- if we
18 were to have the Categories I, II and III at present,
19 would they have all fallen into a Category II or III,
20 or do we know?
21 DR. PETERSEN: Well, I can look at that.
22 But that wasn't -- if you look at the basis for any of

1 these actions, it's the exception [inaudible due to
2 failure of in-house PA system] issue. It's that
3 [inaudible due to failure of in-house PA system] of
4 issues that leads you up to failure [inaudible due to
5 failure of in-house PA system] if there was a failure
6 [inaudible due to failure of in-house PA system] these
7 things come together over time.
8 MR. YANCY: Okay. Thank you.
9 DR. ENGELJOHN: If I could -- Dr. Hulsey,
10 if you could, respond to the question that was
11 answered earlier. And repeat the question if you
12 could.
13 DR. HULSEY: I believe your question was,
14 What was the decrease in pH in the scald water for the
15 *Salmonella* killed in water -- if that was correct.
16 That was from a paper by [inaudible due to failure of
17 in-house PA system]. And he determined that
18 [inaudible due to failure of in-house PA system] water
19 with a pH of 4.3 [inaudible due to failure of in-house
20 PA system] 4.1 [inaudible due to failure of in-house
21 PA system].
22 DR. ENGELJOHN: And I would also just

1 reiterate on the point that the information that Dr.
2 Hulsey is sharing with you will all be fully
3 articulated in the compliance guideline that we expect
4 to have ready to go within a matter of days, not
5 weeks, as I typically try to characterize things.
6 Any other questions in the audience here?
7 (Pause.)
8 DR. ENGELJOHN: If I could ask the
9 operator?
10 Is there any other question on the phone
11 line?
12 (Pause.)
13 DR. ENGELJOHN: Okay. We have no further
14 questions. It's about lunch time. We will be back
15 here at one o'clock for the afternoon session with
16 some research information. Thank you very much.
17 (Whereupon, at 12:00 p.m., this conference
18 was recessed, to reconvene at 1:00 p.m. this same day,
19 Thursday, February 23, 2006.)
20
21
22

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

2 (1:05 p.m.)

3 DR. ENGELJOHN: This afternoon, we're
4 going to actually concentrate on actual research and
5 real-life experience that can be shared with you. Our
6 first speaker this afternoon is Dr. Mark Berrang. He
7 graduated from Virginia Tech with a bachelor's in '86
8 and then from the University of Georgia with a
9 master's in '88.

10 He began working with the USDA's
11 agriculture research service in Dr. Nelson Cox's lab
12 in '89. He completed a PhD in food microbiology from
13 the University of Georgia and took a senior scientist
14 position with the Agricultural Research Service. And
15 his specialty has been in the area of poultry food
16 safety microbiology.

17 During the course of his career, he has
18 conducted research at all stages of production and
19 processing, ranging from the hatching egg to further-
20 processed, ready-to-eat poultry meat, and these
21 efforts have resulted in the authorship on 87 peer-
22 reviewed articles published in scientific journals and

1 90 scientific abstracts.
2 Please welcome Dr. Mark Berrang.
3 (Applause.)
4 DR. BERRANG: Thank you, Dan.
5 Can you all hear me okay?
6 VOICES: Yes.
7 DR. BERRANG: As Dan said, I'm going to be
8 talking about *Salmonella* and *Campylobacter* in broiler
9 transport coops. And most of my own work in transport
10 works has actually been done specifically with
11 *Campylobacter*, but I will discuss some information
12 about *Salmonella* today where I can and where it's
13 appropriate.
14 We all know the kind of coops we're
15 talking about: The dump coops that carry broilers
16 from the farm to the plants. And thanks to our
17 interaction with local commercial cooperators, we've
18 been able to get coops to work with so we can use
19 real-life surfaces to sample.
20 We have several different ways that we
21 sample these coops in our experiments and our studies.
22 And the most important surface in my opinion in a

1 transport coop is going to always be the floor,
2 because that's where most of the fecal matter ends up
3 and that's where the birds tend to settle onto and
4 contact during transport.
5 So pathogens in transport coops. It's no
6 big surprise that *Salmonella* and *Campylobacter* have
7 been detected in transport coops. And Stan Bailey
8 published a paper a few years ago, where he found that
9 5 percent of the coops that he sampled were positive
10 for *Salmonella* prior use; 10 percent were positive
11 after use.
12 And in another, Belgian, study, 56 out of
13 128, or close to 44 percent, of their transport crates
14 were positive for *Salmonella* prior to use. And these
15 authors showed that the prevalence of *Salmonella*-
16 positive broilers was increasing during transport, and
17 they suggested the transport crates that they used in
18 Europe were an important source. And of course,
19 similar findings and suggestions have been made
20 relative to *Campylobacter*.
21 So what is the source of *Salmonella* and
22 *Campylobacter* in these coops? Obviously, *Salmonella*

1 and *Campylobacter* are present in the gut and feces of
2 broilers from a positive flock. And feed withdrawal
3 tends to change the microflora, and transport tends to
4 increase excretion. And that's how they get into the
5 coops.
6 Coop flooring and carcass microbiology.
7 Jeff Buhr and some other folks at the Russell Research
8 Center did a study a few years ago where they compared
9 broilers that were transported on solid flooring,
10 traditional fiberglass flooring, to those that were
11 transported on an elevated wire floor. The wire then
12 allowed the fecal matter to drop through, and there
13 was less contact between the bird and the feces.
14 What they found is that feathered
15 carcasses that were transported on the solid flooring
16 had noticeably more fecal contamination right here on
17 the breast than the ones that were on the wire floor
18 and higher numbers of *E. coli*, also. But what they
19 also found is that after those same carcasses had been
20 scalded and defeathered, all the differences in the
21 carcass microbiology had disappeared. So we started
22 to wonder, Well, how really important is this

1 contamination in the transport coop?

2 I did a study with the help of some other

3 folks at Russell Research Center where we looked at

4 cross-contamination in transport coops. And what we

5 wanted to do was examine the possibility that

6 contaminated feces left in these dump coops can cause

7 transfer of *Campylobacter* to birds that were

8 previously free of *Campylobacter*.

9 And what we did is -- we got some

10 transport coops -- new transport coops that had never

11 been used before, loaded them with *Campylobacter*-

12 positive broilers, let them stay in those coops for

13 eight hours, pulled the *Campylobacter*-positive

14 broilers out and replaced them with birds from a

15 *Campylobacter*-negative flock. So we were able to

16 isolate the fecal contamination in the dump coop as

17 the only source of *Campylobacter* to these previously

18 *Campylobacter*-free carcasses.

19 And what we found was that after just two

20 hours in that dump coop, more than half of the

21 defeathered carcasses from the test flock, the

22 *Campylobacter*-negative flock, had now become positive

1 for *Campylobacter* and -- albeit it was at lower
2 numbers. It was still a substantial number on the
3 rinses from these birds that -- carcasses that were
4 from a *Campylobacter*-negative flock.
5 And the important point is that we sampled
6 them after defeathering. So this contamination was
7 transferred in the dump coop and was maintained in the
8 carcass through scalding and picking.
9 So *Campylobacter* can in fact be spread to
10 previously negative broiler by contact with
11 contaminated feces remaining in the dump coop, and
12 this contamination can remain on the carcass through
13 scalding and picking.
14 Now, keep in mind that *Salmonella* is a
15 much more hardy organism than *Campylobacter*. And I
16 feel confident in saying that if *Campylobacter* can be
17 transferred by this route, the *Salmonella* can be, too.
18 In fact there's similar findings that have been
19 recorded in the literature for *Salmonella*.
20 "So should we be washing and sanitizing
21 cages," is the question to pose. And is that -- is
22 washing and sanitizing dump coops the answer?

1 Right now, according to Julie Northcutt
2 and Dena Jones, about 28 percent of U. S. broiler
3 plants have a coop wash or sanitizing procedure in
4 use. There's a much higher percentage of processors
5 in Europe who are washing and sanitizing crates, and
6 they have been for quite awhile.
7 Nevertheless, even though they've been
8 washing and sanitizing crates in Europe, it's not hard
9 to find reports coming out of Europe where they have
10 detected *Salmonella* or *Campylobacter* or both on their
11 transport crates -- even after washing and sanitizing
12 them. And the authors of these studies usually point
13 out human error and the mixing of the chemicals or
14 equipment breakdown or not getting all of the fecal
15 matter off the crates as reasons why they still have
16 to detected these organisms after washing.
17 Having said that, though, there are
18 reports in the literature that show that some
19 experimental washing and sanitizing procedures can
20 work. But when you look at this research, before you
21 run out and adopt a washing and sanitizing procedure
22 based on experimental results, you need to evaluate

1 these results carefully.
2 For instance, were the chemicals
3 inactivated properly before the samples were taken?
4 There's studies that are published where the target
5 bacteria might actually be being inadvertently being
6 killed after sampling during culture, because the
7 chemical has not been properly inactivated.
8 Another thing to consider is, How
9 realistic or commercially viable is the procedure, and
10 how expensive or unwieldy is the equipment? If it
11 involves big immersion tanks or high temperature
12 application of chemicals, it might be more money than
13 is really reasonable.
14 Julie Northcutt and I did a study at a
15 commercial processing plant that does have a coop
16 washing procedure in place. And what we did is -- we
17 measured the numbers of total aerobic bacteria and *E.*
18 *coli* on the floor surface of dump coops, and we also
19 measured the presence of *Salmonella* and *Campylobacter*.
20 And what we found is that by washing these
21 coops with water, just by spraying them out with
22 water, they were able to reduce the numbers of aerobic

1 bacteria and *E. coli*. And then later they applied a
2 sanitizer, which lowered the numbers even more.
3 And the prevalence of *Campylobacter* and
4 the prevalence of *Salmonella* were also lowered, but,
5 even so, after the washing and sanitizing procedure,
6 we were still able to detect *Campylobacter* on two out
7 of 27 coop floor samples, or about 7 percent. I feel
8 like if we had sampled a higher number of coop
9 surfaces after washing and sanitizing, we probably
10 would have found *Salmonella* in some of them, as well.
11 So we decided to look at some novel
12 approaches and try to see if we could find other ways
13 to possibly decontaminate transport coops. And one
14 thing we tested was allowing the coop to dry out and
15 be stored between use for extended periods of time.
16 And our thinking there was that by allowing the fecal
17 matter left in the cage to dry out and be exposed to
18 atmospheric oxygen, we would lower the numbers of
19 *Campylobacter* that we were able to detect in that
20 feces.
21 So what we did is -- we got dump coops,
22 and we loaded them with *Campylobacter*-positive

1 broilers, left those broilers in there for eight
2 hours, removed them and then put the dump coops in a
3 pole shed, where we just left them for 48 hours, and
4 periodically scraped out all of the feces from some of
5 the openings and cultured that for *Campylobacter*.
6 And this graph shows some of the results
7 from that study. The Y axis is marking the number of
8 *Campylobacter* that we recovered per opening. Across
9 the X axis is time.
10 What we found is that we started out with
11 between 10 to the eight and 10 to the nine
12 *Campylobacter* per opening and that stayed steady for
13 the first eight hours of sampling, but, by 24 hours,
14 we saw a two-log decrease, down to about 10 to the six
15 per opening, a two-log being about a 99-percent
16 decrease. By 48 hours, we were down to our limit of
17 detection or even where we were unable to detect any
18 *Campylobacter* from those -- the feces that was left in
19 these dump coops.
20 The *Campylobacter* counts closely mirrored
21 the percent moisture in the feces that was left
22 behind. And this graph shows how the percent moisture

1 in the fecal matter tended to go down as the coop was
2 left out to dry.
3 So by allowing these transport coops to
4 dry out and not be used for 24 to 48 hours, we were
5 able to lower the numbers of *Campylobacter* in that
6 feces, but we were not able to reliably eliminate the
7 *Campylobacter* altogether.
8 And it's important to note that the
9 expense required to maintain enough coops for this
10 would be really impossible to justify. I'm not
11 suggesting that every company needs to go out there
12 and buy three to four times the number of coops and
13 build a huge building and store them. That's not at
14 all what I'm trying to say.
15 Julie and I also did a study looking at
16 washing and sanitizing of coop flooring. And this was
17 designed to test the efficacy of spray washing with
18 tap water followed by an immersion I chemical
19 sanitizers to eliminate *Campylobacter* on the coop
20 flooring material.
21 And this was similar to the field study
22 that we did, but it's a much more controlled

1 environment, because we were doing it in a lab so we
2 had a repeatable way to contaminate the floor surface
3 and a much more repeatable way to wash it and sanitize
4 it.
5 What we were doing in this study is -- we
6 used little pieces of coop flooring that we cut from a
7 large sheet that we got from a commercial cooperator,
8 and we intentionally contaminated these little squares
9 with gut contents from *Campylobacter*-positive broilers
10 with known amounts of *Campylobacter* in them. We
11 allowed the gut contents to dry, and then we'd wash it
12 off with tap water at a known water pressure. And
13 then we dipped the little squares with whatever
14 remaining fecal matter was still on them into a
15 sanitizer to see what would happen.
16 This graph shows what we found when we did
17 the water spray wash followed by immersion in chemical
18 sanitizers for 15 seconds. And the control bar shows
19 the number of *Campylobacter* on a square that was not
20 washed and was not dipped with sanitizer.
21 Where we had about ten to the seven
22 Campylobacter on that little square, just washing it

1 with water lowered those counts by two logs, down to
2 ten to the five. But immersing those little squares
3 of flooring into a quat or a chlorine for 15 seconds
4 did not improve the reduction of the *Campylobacter* at
5 all.
6 And we found the same kind of thing when
7 we immersed them into the sanitizers for 60 seconds.
8 In fact, when we immersed them in the sanitizers for
9 five minutes, the numbers we found from the little
10 squares that were immersed actually went up. And I
11 think they were just getting so wet that the remaining
12 fecal matter was washing off -- coming off of the
13 sample much easier.
14 So spraying the floor surface with tap
15 water lowered the numbers of *Campylobacter*, but adding
16 an immersion of 200 parts per million quad or chlorine
17 did not help.
18 So we know that water spray can lower the
19 counts, and we know that drying can lower the counts.
20 We decided to look at those two together and measure
21 the effectiveness of the water spray followed by an
22 extended dry time to eliminate *Campylobacter*, and this

1 graph shows the results we found in that study.
2 Here in this study, we counted
3 *Campylobacter*, which is shown on the blue bars, and we
4 also counted *E. coli*, shown on the red bars. And,
5 now, *Salmonella* is closely related to *E. coli*. So I
6 would expect *Salmonella* to respond in a similar way to
7 *E. coli* to this kind of treatment.
8 With our control -- again, this was done
9 with little squares of flooring. With the control
10 squares, we found about 10 to the 7 *Campylobacter* and
11 close to 10 to the 6 *E. coli*. Just applying the
12 spray -- low-pressure water spray, we were able to
13 lower those counts significantly, down to 10 to the 4
14 *Campby* and about 10 to the 3 *E. coli*.
15 The 24-hour dry was -- that's just drying
16 and not spraying. That lowered the counts even better
17 than a spray by itself, down to about 10 to the 1
18 *Campylobacter* and half-a-log *E. coli*. But when we
19 used the spray followed by the dry, we had our best
20 reduction; we were not able to detect any
21 *Campylobacter* or *E. coli*.
22 Notice the one red bar there with the

1 little asterisk on it. That's to point out that when
2 the little -- when the fecal matter had dried for 24
3 hours and it was re-wet -- we sprayed it after 24
4 hours to simulate that coop going out on a wet day --
5 the numbers of *E. coli* do rebound up to about Log 2,
6 not the size they were originally -- nowhere near the
7 size they were originally, but it does rebound
8 somewhat. *Campylobacter* did not rebound. But I would
9 expect *Salmonella* to behave like *E. coli* in this kind
10 of situation.
11 So the low-pressure tap water spray
12 lowered the numbers of *E. coli*, but simply allowing
13 the gut contents to dry out was more effective, and
14 using them together in concert was very effective.
15 But the re-moistening -- again, if that dried-out
16 fecal matter becomes re-moistened, *E. coli* and
17 probably *Salmonella* can rebound.
18 So is floor surface drying a reasonable
19 sanitation treatment? It can certainly lower the
20 numbers of *Campylobacter* that we recover from these
21 floor surfaces, but it would really require,
22 obviously, a major change in thinking relative to coop

1 design or management, and we can't do it the way
2 things are right now.
3 We cannot store cages for 48 hours. We
4 would have to either have removable floors that could
5 be stored separate or have a fast way to dry out the
6 surface.
7 So overall, coop washing and sanitizing is
8 an expensive proposition. You have to worry about
9 water costs, your personnel and the time involved.
10 You have to worry about -- what are you going to do
11 with your runoff water? Are you causing cross-
12 contamination in that area by spraying that material
13 around?
14 The efficacy is questionable. Even in
15 Europe, where they've been doing this a long time,
16 they're still finding pathogens on the surfaces after
17 sanitizing. And we're looking at new ways to sanitize
18 these coops now, and we'll see what we come up with.
19 And I think I'm about out of time.
20 (Applause.)
21 DR. ENGELJOHN: Thank you, Mark, very much
22 for that helpful information on getting the birds to

1 the facility.
2 We're going to move now into the facility.
3 And we have Dr. Stan Bailey, a microbiologist also
4 with the Agricultural Research Center, with USDA. Dr.
5 Bailey is the lead scientist and research
6 microbiologist for USDA Agricultural Research Service,
7 where he has directed research toward monitoring,
8 controlling, reducing and ultimately eliminating
9 contamination of live poultry by human enteric
10 pathogens.
11 During his 21-year career, he has authored
12 or co-authored about 500 publications on food
13 microbiology. And today, we have Dr. Stan Bailey to
14 talk about the processing plant and final carcass
15 contamination. So thank you very much.
16 (Applause.)
17 DR. BAILEY: Thank you, Dan. But that's
18 31 years, not 21. Don't take them away; I need them
19 for retirement.
20 (Laughter.)
21 DR. BAILEY: Thank you for the invitation.
22 And the first thing they asked me to talk about today

1 was, What does the effect of what's coming off the
2 farm, the load of bacteria on your chickens, have on
3 what you can do in the processing plant? So I went
4 through some of the work that we've done and some of
5 the literature.
6 But I think if we're going to talk about
7 what's on the chicken, then we need to have just a
8 little basic understanding of what's going on. And
9 the first thing I wanted to talk to you about was,
10 What are the factors that affect *Salmonella*
11 colonization of poultry?
12 And you'll notice I might say,
13 Colonization. And that's because, for the most part,
14 the vast majority of strains of *Salmonella* don't
15 infect the chicken in the classic sense; it's not
16 causing a disease. I mean there are a few strains
17 that will. But for the most part, we're talking about
18 it just setting up shop and growing in the intestinal
19 tract.
20 And there are a number of factors that
21 affect that. Probably the first and foremost is the
22 age of the chick or the chicken. A new-hatched chick

1 is highly susceptible to becoming colonized with
2 *Salmonella*. If you take -- each strain may be
3 slightly different.
4 But if we take the same strain and give it
5 to a day-of-hatch chick, probably 50 to 75 percent of
6 those chicks will become colonized with *Salmonella*
7 with maybe ten to 20 cells. By the time that bird's
8 three or four days old, it'll take maybe 10,000 to
9 100,000. By the time it's a week or more old, it
10 might take a million cells of the same strain of
11 *Salmonella*. Of course, all of that can be affected by
12 stress and bird health, the third factor I show there.
13 Then there's -- how is the *Salmonella*
14 getting there? There's a couple of primary ways that
15 we're always concerned about. I think first and
16 foremost is what's coming out of the breeder stock.
17 If you have your breeder stock
18 contaminated and if they take it into the hatchery and
19 spread it around and, therefore, you're putting birds
20 on the ground that are *Salmonella* colonized, then
21 you're almost beat before you start, because anything
22 you do from that point on -- for the most part, you're

1 going to have a hard time preventing or getting rid of
2 that *Salmonella*.
3 And then, of course, you can get it onto
4 the farm free of *Salmonella*, and then you also have to
5 worry about feed and environmental exposure. And each
6 of those can play a separate role. And I could talk
7 for the 15 minutes I have about all of this, but let
8 me move on.
9 So what are we theoretically seeing
10 happening on farms? For the most part, if you don't
11 have extra stress and other factors affecting you,
12 then this graphic would probably indicate what's
13 happening most of the time. You'll have some low
14 level of *Salmonella* coming out of the breeder's farm
15 or else in the environment or the feed when they first
16 get there, and that will explode up in the first two
17 weeks or so.
18 In around two to three weeks, you'll see
19 your maximum level of *Salmonella*. And if you don't do
20 anything and if they don't have any extra event that
21 causes significant stress or anything, then you'll see
22 that intestinal colonization of *Salmonella* starting to

1 drop off over time to the point they're six to seven
2 weeks old. Again, without any extra stress pressures,
3 it'll drop down pretty low, sometimes 1 or 2 percent,
4 5 percent or 10 percent, just depending on the
5 situation.
6 But it's not just what's in the bird that
7 we're concerned about. Remember, we talked about
8 there at Week 2 or 3, that might be your maximum
9 level. Well, what happens is, unlike *campy*, which
10 Mark was just talking about, *Salmonella* is not very
11 fragile. It sticks around for a long time. So if you
12 ever get it in your flocks, if you get it on the skin
13 or on the feathers, it's likely to stay there and take
14 it into the farm or into the processing plant.
15 I did two studies several years ago where
16 we looked at that a little bit. In the first one, we
17 challenged the chicks the day of hatch with
18 *Salmonella*, and we followed them for five weeks or six
19 weeks, to the time they would go to the processing
20 plant. And at that time, they had been reduced down
21 to that 5 percent level we were talking about in
22 intestinal colonization. But of those same birds, 53

1 percent of them were carrying *Salmonella* on the
2 outside of the bird.
3 Another set of studies we did similarly
4 had 15.5 percent of the birds intestinally colonized
5 compared to 50 percent with *Salmonella* on the
6 feathers. And as we're talking about load coming into
7 the plant, I think that's particularly pertinent
8 because -- most of you are aware -- that know the
9 literature and know what we've seen through time --
10 that probably 90 to 95 percent of the birds in a
11 processing plant that are *Salmonella*-positive are
12 carrying very few cells of *Salmonella* on them,
13 probably less than a hundred and often times less than
14 50 cells of *Salmonella,* on the whole carcass. So it
15 probably is a result of some kind of cross-
16 contamination at transport maybe.
17 So those figures I was just talking about
18 then were what was going on with the birds when they
19 left the farm. There has been several studies done --
20 and I didn't put them all here, for the sake of time.
21 But to summarize, we see in general an increase in
22 the *Salmonella* carriage, either internally or

1 externally, of between 20 and 40 percent for those
2 birds after transport compared to before transport.
3 So being in that coop, being exposed to
4 the fecal material, just the aerosoling, the sitting
5 in the materials, or whatever, will increase the
6 carriage of *Salmonella* from 20 to 40 percent during
7 transport.
8 Another area that we can talk about where
9 I can show you a little bit of the issues that have to
10 do with the carriage had to do with a study we did
11 several years ago, and I thought I'd highlight just a
12 few points of that. And the objective of this study
13 was to characterize on a multi-state basis the
14 prevalence of *Salmonella* from numerous sources in
15 chicken production and processing.
16 We looked at plants in four states each
17 season of the year, and we artificially defined high
18 and low production. We let the companies do that.
19 And those would be production parameters: Feed
20 conversion, just generally good operating plants
21 versus those that weren't perceived as being so good.
22 And actually, in this study we found a very low level

1 of *Salmonella* overall.

2 And I just wanted to show this to you to

3 kind of see the kind of variability we would see. But

4 you can see within plants, there is a big variability

5 by season and by plant. But I threw this slide in for

6 a particular reason. If you want to monitor what's

7 going on in your farm level and you want some really

8 simple way to do it, we've found that used fly strips

9 can catch your flies. They're a natural filter, and

10 they monitor. And we found the best sensitivity of

11 knowing what was going on on our farms just by looking

12 at what was going on with the flies.

13 To the point that Mark was just talking

14 about, transportation coops, look at the difference

15 that we see here. In three of the four times that we

16 looked at this, we had significantly higher levels of

17 *Salmonella* detected in the coop swabs after transport

18 than before. In that particular study, to some of the

19 things that Sean Altekruse was talking bout this

20 morning, we identified 36 different serotypes. Those

21 frequently were Senftenberg, Thompson and Montevideo.

22 And we found one strain that we -- had never been

1 isolated from poultry before.
2 Now I want to spend just a couple of
3 minutes with you talking about a study that we've been
4 doing -- and just completed a couple of months ago --
5 for the last year with FSIS which again is
6 illustrative of the type of load that we're bringing
7 into the plant.
8 With this particular study, we asked some
9 basic questions. One was, Can a reliable measure of
10 process control be determined from one or more post-
11 chill samples, or do samples have to be paired from
12 within a given flock post-pick compared to post-chill?
13 And, if you want to answer that question, how many --
14 if it requires either one at the end or a pair, how
15 many samples would you have to look at to have an
16 indication of what's truly going on to take out
17 statistical variability?
18 And then finally, is there a relationship
19 between the reduction in *E. coli* counts during
20 processing and the reduction in *Salmonella* and
21 *Campylobacter* during processing?
22 We looked at 20 randomly selected plants,

1 again, across all four seasons. FSIS collected the
2 samples and sent them to us in Athens in a blinded
3 manner. I have no idea what plants they were and
4 where they came from. And in this particular study,
5 we looked at ten carcases post-pick and ten carcasses
6 post-chill.
7 In the study, we looked at quantitative *E.*
8 *coli* and coliform and *Campylobacter* levels,
9 qualitatively *Salmonella*. We used Petrifilm for the
10 *E. coli* and coliform and direct plating on Campy Cefex
11 for *the Campy*, and we used the FSIS procedure of a BAX
12 PCR. And if we got a positive screen, then we used
13 cultural procedures to get our *Salmonella* isolate.
14 When we finished the study, we had done a total of
15 6,400 analyses, 1,600 for each of the four organisms.
16 Just to give you a little bit of an idea
17 of the kind of load, as I'm supposed to be talking
18 about incoming load, when we looked at the rehang
19 immediately after the picker, we saw about 72 percent
20 positive for *Salmonella* across all of the samples and
21 all of the seasons, but you can see that, obviously,
22 the processing plants were doing something right,

1 because we had about 19.6 percent of the birds
2 positive by the time they got out of the chill tank.
3 So we did have a pretty significant load
4 coming in, but the processing plant seemed to be doing
5 a reasonably good job. And you can see some
6 variability in the seasons, too, particularly more on
7 what's coming out of the plant than what was going in.
8 I did want to finish with just a couple of
9 slides as we're talking about the importance of what's
10 on the birds as they come in. And I'm going to talk
11 later about some chemical disinfection things, and
12 you'll have other talks this afternoon.
13 But if we go back to the point of, "What's
14 the load coming in," being particularly important -- I
15 did a study with Tonya Roberts a couple years ago
16 where we looked at *Salmonella* control in Scandinavia,
17 particularly Sweden and Denmark. And I'm not going
18 to get into that whole study, but we obviously don't
19 have the same type of industry. We can't do the same
20 control program, but I think we can learn from some of
21 the principles that guide that program and maybe apply
22 some of those to help us here on the farm.

1 And the basic principles of the Swedish
2 program is: If broilers are never exposed to
3 *Salmonella*, then they can't become colonized. In
4 their system, they started off by eradicating all the
5 positive breeder flocks and continue to do so over
6 time, and a number of other things. But we can't do
7 that. That wouldn't be practical. But what we can do
8 is learn from that and know that we have to be able to
9 control what's coming out of our breeder flocks, and
10 there's ways and things we'll look at.
11 Then the other point that comes from that
12 is: Their entire control program -- they have zero
13 *Salmonella* -- or pretty close to zero. Their entire
14 control program is on the farm. They use no chemicals
15 in their processing plant. They use the same basic
16 equipment we do, but they just use water.
17 So what are some potential U. S.
18 intervention strategies that don't involve
19 eradication? Well, on the farm or -- on the breeder
20 farm to start with, we can control *Salmonella*, I
21 believe, to a great extent -- and I know this to be
22 true because I've seen it done -- by controlling

1 what's going into the feed, with biosecurity, with an
2 effective vaccination program, an effective
3 competitive exclusion program, and, particularly, we
4 have to pay attention to moisture control.
5 In a broiler production, again, we can
6 look at the feed, biosecurity, competitive exclusion
7 and moisture control. And we won't get into all the
8 issues we talked about in the fall about getting some
9 of these products approved.
10 So in conclusion, most chicken flocks in
11 the U. S., unfortunately, carry some load of
12 *Salmonella*. It's to our -- what we have to work on is
13 getting that load as low as possible. And again,
14 transportation appears to increase both the internal
15 and external carriage of *Salmonella*. We have seen and
16 will see more today that chemical treatments in the
17 plant can reduce *Salmonella* on processed chickens, but
18 it's important to keep the level of *Salmonella* low in
19 and on the chickens as much as possible, and that will
20 eventually require going back to the farm.
21 So that's all I have. And thank you very
22 much.

1 (Applause.)

2 DR. ENGELJOHN: Thank you, Dr. Bailey.

3 We're now going to move on to a
4 presentation on sanitation and the sanitizers being
5 used. We have Dr. Scott Russell, who is since 1994 a
6 professor of poultry processing microbiology in the
7 department of poultry science at the University of
8 Georgia; he also has had real-life experience as a
9 production manager and microbiologist at a processing
10 facility in Gainesville, Georgia. And with him -- he
11 has done a lot of research that will be helpful to you
12 and I'm sure would be good as research --

13 So Dr. Russell?

14 DR. RUSSELL: Thank you. I'd like to
15 thank the Organizing Committee for having me. It's a
16 real pleasure to be here with you today. And in the
17 [inaudible due to failure of in-house PA system] I
18 have, I'd like to go ahead into the subject.

19 And as [inaudible due to failure of in-
20 house PA system], numerous microbes can adhere to
21 processing equipment surfaces, as most of you know,
22 and they may concentrate and grow in crevices or

1 joints of rubber gaskets that remain moist. And wear
2 on these surfaces can also impact the ability of the
3 organisms to adhere, and any remaining food material
4 that may be left on those surfaces can then provide a
5 growth medium for the bacteria.
6 So part of the whole idea of cleaning and
7 sanitation involves removing a lot of that material to
8 avoid any sort of growth material being there for
9 those organisms.
10 So just by way of a general overview for
11 how to go about cleaning and sanitizing, it's
12 important for the large pieces of trash and things to
13 be picked up and electrical connections to be covered.
14 And then they want to go through a pre-rinse with
15 warm or hot water and then apply usually an alkaline
16 cleanser, as applied through a central system using
17 pretty warm water, and then five to 20 minutes of
18 exposure, and then usually the ceilings and things
19 like that -- floors, walls, equipment -- rinsed with a
20 cleaner.
21 And then it's inspected, and touched up as
22 necessary. And only after all of those things have

1 been conducted, that's when the sanitizer's applied.
2 And I want to go into kind of more in depth as to why
3 it's so essential that we get rid of all of that
4 stuff.
5 Now, most of the commonly used
6 detergents -- and I won't go into this in detail --
7 are alkaline detergents by far. They're especially
8 useful in the poultry industry because of the types of
9 soils that are deposited there. And there are a
10 number of categories of those types of products.
11 Acid detergents are generally used to
12 remove strongly encrusted surface matter, and they're
13 good for cleaning the scale on a fairly regular basis,
14 as well. Some synthetic detergents that are out there
15 are used occasionally -- such as quaternary
16 ammonium -- and then there are soaps that are used --
17 a variety of different things that can be used to do
18 cleaning.
19 Now, with regard to sanitizing, there are
20 a number of different products that have been used in
21 the industry, but I want to point out to you and, as
22 we go through this, I want to make it real clear that

1 I don't -- the sanitizer isn't nearly as important as
2 removing the biofilms in the cleaning process. It is
3 important to use a good sanitizer, and all of these
4 things give you an opportunity to see what's out
5 there, for example quaternary ammonia, a very popular
6 sanitizer used throughout the industry.
7 Some companies use industrial-strength
8 bleach; some will use a chlorine-dioxide mixture.
9 Some use iodine compounds especially in hand-dips and
10 things like that. Some have gone to encapsulated
11 lysozyme, and I'll talk a little bit about how that
12 works. Ozone systems, steam systems, hydrogen
13 peroxide vapor, peroxyacetic and cetylpyridinium
14 chloride are all being used in these contexts
15 throughout the industry at different -- in different
16 areas.
17 This -- I don't want you to read this. I
18 didn't put this here for you to read it or memorize
19 it. There won't be a test afterwards. Okay?
20 The idea here is for you to see what types
21 of characteristics are needed for a sanitizer to be
22 considered effective. Really, you -- the companies

1 that develop these things have to jump over numerous
2 hurdles to make this thing worthwhile to the industry.
3 I mean look at all of these different
4 things. The kill rate, the toxicity, stability,
5 speed, penetration, film-forming, and all of these
6 kinds of characteristics -- proclivity -- have to
7 be -- it has to pass all of those tests in order for
8 it to be effective. And then, of course, we have to
9 look at what area that the product's going to be used
10 in.
11 There are concerns such as, What kind of
12 equipment is in that area? Is it aluminum? Is it
13 stainless steel? Is it wood? Is it plastic? And in
14 all of those areas -- concrete floors, these kinds of
15 things -- it's important to determine, What's the best
16 sanitizer or best cleaning method for those types of
17 equipment? Of course, we don't have a lot of time to
18 go into that.
19 There are other concerns, too. What type
20 of water is coming into the plant? Is it high in
21 iron? Is it hard water? All of these kinds of things
22 are really important. And of course, the cost of the

1 sanitizer is very important, as well, as most of you
2 know -- and then the type of material you're applying
3 it to -- walls, tile walls, rubber belts, and so
4 forth.
5 Now, why is it that the sanitizer -- I
6 mentioned earlier that I don't think sanitizer is
7 nearly as important as removing biofilms removing the
8 bacterial colonies that are already there on the
9 equipment. I want to show you some things as we go
10 along.
11 And this is one of the big problems that
12 we see throughout the food processing industry.
13 Bacteria can be transferred from floor drains, from
14 walls, ceilings and these kinds of places, where they
15 will accumulate and grow -- bacteria like *Salmonella*
16 and *Listeria* -- and they will -- they can easily be
17 transferred by aerosolization.
18 These -- a lot of the employees, as you
19 know, like to use high-pressure hoses. And they'll
20 spray the floor, and these bacteria will aerosolize,
21 and they'll land on a piece of equipment. It could be
22 stainless steel, it could be rubber belting or what

1 have you.

2 These bacteria will then begin to
3 communicate with one another through a processing
4 called quorum sensing, and they send out a signal.
5 Usually, this is an N-acelated-homoserine lactone, AHL
6 for short.

7 And these chemicals basically are
8 signaling to the other bacteria, Hey, I'm not real
9 happy here; there's not many nutrients here; we have
10 got to get together and build a house to protect
11 ourselves. That's in a nutshell what they're doing.
12 Now, I'm not sure if they say it in those words, but
13 that's, you know, my best estimate, anyway.

14 But a number of bacteria do this.
15 *Salmonella*, *E. coli*, *Pseudomonas* -- a lot of them do
16 that. And they form these biofilms sort of -- if you
17 can, envision in your mind -- it's like an ant.
18 Ants -- it's raining outside, and the ants get
19 together, some signal goes out, and they begin to
20 build an ant mound. It's a very similar type of
21 process.

22 Now, as I mentioned to you, they do this

1 to protect themselves, and they do this to prepare an
2 organized communal structure. And it is similar to an
3 ant mound. And I have some pictures here for you to
4 see.
5 In the early stages, the biofilm is
6 composed of a cell layer attached to a surface, and
7 the cells grow and divide, and they form a dense mat
8 numerous layers thick. The bacteria use quorum
9 sensing to signal each other to reorganize, therefore
10 forming a very complex array of pillars and irregular
11 surface structures. And these structures are
12 connected by convoluted channels that deliver food and
13 remove waste, and we're going to see pictures of this.
14 Now, why is this significant? Because if
15 you go along and spray a sanitizer on an already
16 formed biofilm, you may only kill 10 percent to 50
17 percent. And, believe it or not, 50 percent in the
18 bacterial world doesn't mean anything. Okay?
19 We've talked about a significant reduction
20 in bacteria being at least a log; that's 90 percent.
21 If you go and only kill 10 percent or 50 percent,
22 you're not even removing by a log. And that doesn't

1 do you much good. So it's essential that these
2 biofilms be removed, because they will protect
3 bacteria, and they will keep them from being killed by
4 sanitizers.
5 This is a picture of how biofilm forms and
6 how short a time it requires. In the first window up
7 here at the three hour stage, you see single bacteria
8 there. But by as early as eight hours, you start to
9 see the yellow biofilm formation around the colony
10 and, by ten hours, the bacteria almost wholly encased
11 in that biofilm. So you can see, in a very short
12 period of time even during an individual processing
13 day, these bacteria can easily become encased in a
14 biofilm and can become protected from sanitizers.
15 Look at how complex this structure is.
16 There can be everything from yeast, bacteria -- many
17 different strains or species of bacteria can get in
18 this structure. And they're all communicating in
19 there, and they're forming these organized channels.
20 What happens then is -- this is sort of a
21 good diagram. You can see that the aerobic bacteria
22 live on the top of the biofilm, and oxygen can

1 penetrate down to them. But down below them,
2 anaerobic bacteria are living, as well. Food and
3 moisture are able to go through those channels that
4 you could see in the previous slide down to the
5 bacteria and allow them to survive just fine. The
6 waste materials are excreted out the bottom of the
7 biofilm.
8 It's just like an ant mount; it's a very
9 organized communal structure. It makes it very, very
10 difficult to penetrate that with chemical sanitizers,
11 and that's really our challenge. That's one of the
12 problems that we see throughout the industry.
13 This is an example of how the biofilm --
14 the bacteria that form these things can be sprayed and
15 they can aerosolize because of high-pressure hoses and
16 so forth. They fly through the air, they land on a
17 piece of equipment, and they go through these stages.
18 And once they reach like Stage 5 here, as you see on
19 the slide, then they rise to the top.
20 The thing can break off or be rubbed off
21 by incidental surface contact or sprayed off with a
22 hose. Next thing you know, these bacteria are flying

1 to other areas within the cooler or within the
2 processing plant and forming other bacterial biofilms,
3 which, again, makes it very, very difficult to remove
4 these things.
5 So as I mentioned before, you can clean
6 and sanitize. And if these things are in biofilms and
7 you don't remove the biofilms, you can still have only
8 10 to 60 percent elimination, which isn't much, in
9 those cases. And most cleaning programs
10 involve four days with alkaline cleaner and one day
11 with an acid cleaner. And authors Jessen and Lammert
12 found that the effective detergents on biofilms was
13 negligible whereas the most efficient disinfectants
14 were able to eliminate *L. monocytogenes*. Very high
15 strength and long reaction times were required under
16 these conditions. Acid disinfectants composed of
17 hydrogen peroxide and peracetic acid were more
18 efficient than the chlorine-type compounds or
19 oxidants.
20 Similar results are reported by other
21 authors, while others find the efficacy of the two
22 disinfectants or even the opposite efficacy. So

1 there's conflicting studies as to which is better,
2 chlorine or oxidant-type compounds or like peracetic
3 acid-type oxidant compounds combined with acids.
4 Now recommendations for removing
5 biofilms. This has kind of gone through some
6 evolution over time, but Parker and others found that
7 chemicals that attack the polysaccharide matrix of the
8 biofilm -- that is that glycocelic structure -- were
9 particularly effective in killing and removing cells
10 in a biofilm. Treatment of this biofilm on a clean --
11 of either of the biofilms on a clean, stainless steel
12 surface with lysozyme, which is an enzyme produced by
13 a number of different types of cells, killed biofilm
14 cells and prevented the attachment of any bacteria to
15 the surface.
16 Now, lysozyme can easily be isolated from
17 egg shell membranes, in case you're wondering where we
18 might get something like that in large volume. The
19 authors suggested that lysozyme may have potential as
20 an alternative control method for biofilms of
21 bacteria. Gibson and others reported that cleaning
22 produced a .91 log reduction and high spray -- and

1 again, that's not that much. That's not even a whole
2 log reduction, where -- and they were saying that
3 high-pressure sprays and mechanical methods -- actual
4 scrubbing methods -- were the best.
5 But it required quite a high-pressure
6 spray; 17.2 bars of spray were required just to start
7 to remove biofilms of *Pseudomonas* and *Staphylococcus*.
8 Increasing spray times didn't seem to have any effect
9 on the biofilms, and acidic or alkaline or neutral
10 detergents didn't increase the removal of biofilms.
11 However, the acidic and alkaline cleaners or products
12 affected the viability of the organisms and then
13 minimized the spread of contamination later on.
14 So now, we did some studies where we were
15 able to dramatically reduce fully formed *Listeria*
16 biofilms on stainless steel products. And this
17 product was applied by electrostatic spraying. It was
18 created in an all natural way; in fact, the product
19 could be consumed by itself in a fully concentrated
20 form without any problems.
21 Log 10 reductions in our study ranged from
22 3.3 to 7.2 logs when compared to the controls, and

1 caused the *Listeria* biofilm literally to float off of
2 the stainless steel coupon. It was really interesting
3 to see that.
4 Here are the data if you'd like to see
5 them, but we saw reductions anywhere, again, from 3.3
6 logs all the way up to 7.2 logs on fully formed
7 biofilms of *Listeria* on stainless steel coupons. So
8 we had very good results there.
9 Testing for biofilms. Sampling is very
10 difficult because they adhere to equipment so tightly,
11 and scraping or high-pressure swabs are recommended.
12 Traditional methods for microbiological testing are
13 very slow; most of them are 48 hours, and they just
14 provide a retrospective assessment of cleanliness.
15 Rapid real-time methods are much more appropriate.
16 You can see here how difficult it is to do
17 a swab technique, and it takes 48 hours. This is a
18 Petrifilm contact method, to give you an idea, another
19 24 hours. The traditional contact RODAC plate takes
20 another 24 hours to get results. A Hycheck's another
21 way to do that.
22 These are new carbohydrate and protein

1 test strips that are out there. They're real time.
2 And these other carbohydrate test strips are real
3 time, as well. A lot of people have switched to ATP
4 bioluminometers, because you get real-time assessments
5 of these things. And I don't have time to go into
6 here.
7 Conclusion? Proper cleaning and
8 sanitizing is absolutely essential in preventing the
9 cross-contamination of pathogenic bacteria from
10 equipment to food products. Choosing the proper
11 chemical for the food matrix is important in terms of
12 removing that food from the surface.
13 And biofilm formation is a serious
14 problem, because they're difficult to eliminate once
15 they've formed fully formed biofilms. And as I
16 mentioned before, it doesn't take much time to do
17 that. Microbiological monitoring programs should be
18 real time and should not be retrospective. Thank you.
19 (Applause.)
20 DR. ENGELJOHN: Thank you very much.
21 We're now going to hear from Dr. Marty
22 Ewing, who is a graduate from the University of

1 Georgia College of Veterinary Medicine in '87 and
2 practiced in the private clinical medicine before
3 joining USDA FSIS. She then moved to Florida, where
4 she began working for the state department of
5 agriculture and managed the national poultry
6 improvement plan.
7 We're glad to have her here today. She's
8 going to talk to us about a natural disaster and
9 things that she was able to find out from that.
10 So thank you.
11 DR. EWING: Well, I appreciate it,
12 everyone. Thank you to the Organizing Committee again
13 for inviting me to speak here today.
14 I think you might find this presentation a
15 little (different) from some of the others. This is
16 more going to be about the experiences and concerns
17 that we had following what was probably one of the
18 most catastrophic natural disasters we've had.
19 But first, for those of you that aren't
20 familiar with Sanderson Farms, while we now have
21 facilities in Texas and Georgia, we are primarily a
22 Mississippi-based company. In south Mississippi, we

1 have four processing plants. In one just north of New
2 Orleans in Louisiana, there are four hatcheries, three
3 feed mills and over 600 contract poultry houses. And
4 in that region we process approximately 4 million a
5 week.
6 Okay. It's a little ironic, but, almost
7 six months to the day ago, on August 26, we were
8 traveling home from what was then the pre-harvest
9 meeting on *Salmonella* interventions. And at that
10 time, a small Category 1 hurricane was skirting south
11 Florida, and we went home and did the standard
12 hurricane preparations.
13 We filled all the fuel tanks and discussed
14 with our growers, checked generators --
15 (Pause.)
16 DR. EWING: Okay. I'll try to talk
17 louder.
18 We checked all the generators in the
19 hatcheries, plants and poultry houses. We try to
20 deliver as much feed to the growers as we can in
21 advance and, also, pick up any hatching eggs. We
22 communicate with our growers on feed, water, fuel and,

1 again, drainage, to prevent flooding of the houses.
2 And generally, within 24 hours of where
3 the path -- we can distinguish where the storm is
4 going to hit, we'll notify the district office of any
5 plant closures. Unfortunately, on Sunday, we woke up
6 to a Category 5 behemoth, and her name was Katrina.
7 This slide depicts that path of Katrina as
8 she roared across the Mississippi coast line. The
9 star is Laurel, Mississippi. That's our corporate
10 headquarters. And all of our facilities in
11 Mississippi were either affected by hurricane or
12 tropical force winds.
13 After Katrina, there was no power south of
14 I-20. I-20 goes through Jackson, and all of our
15 facilities are south of there. There was no phone
16 service and no communications except for one radio
17 station that we could get intermittently out of
18 Jackson. There was no water in most areas. There was
19 no gas or diesel fuel. And if there were, we couldn't
20 pump it, because we didn't have any electricity.
21 Curfews were ordered in most localities.
22 And the extent of the devastation, although we didn't

1 know it at the time, was over 90,000 miles.

2 Now, the Gulf Coast? It was incredible.

3 If you go west of the Gulfport/Biloxi area, houses

4 that are left standing are uninhabitable. And if

5 they're not uninhabitable, they're just gone. West of

6 Gulfport past Christian, there is nothing but front

7 steps and foundations. And it's still that way.

8 Fortunately, where were -- we were about 90 miles

9 inland -- we were not hit, although we did have

10 tremendous wind damage.

11 This is the road to my son's high school,

12 outside of our house, completely blocked. And blocked

13 roads were everywhere. This is a church down the

14 street from that house. If you'll notice, the windows

15 are very well taped. The joke around our town now is,

16 Gee, they really should have taped the steeple down.

17 Now, the damage in Laurel was particular

18 devastating from the high winds, and many trees were

19 knocked down. This actually belongs to one of our

20 employees in Laurel; this is her home. Fortunately,

21 she and her family are fine, but her home was

22 devastated. We had about five or six others in our

1 offices that had experienced the same thing.
2 And of course, once we got the trees
3 cleared, we had downed power lines everywhere. And
4 something I want to share with everybody because -- I
5 didn't realize this. But even though there's not any
6 power for hundreds of miles, if somebody has a
7 generator hooked up and it's hooked up wrong, these
8 lines can still be hot. So always respect downed
9 power lines.
10 So, anyway, the good news? There was no
11 loss of life to any of our Sanderson employees,
12 contract growers or contractors. And there was also
13 no major facility damages to any of our feed mills,
14 hatcheries or plants, although we did have some live
15 haul sheds that were damaged.
16 The bad news? Seventy-one poultry houses
17 were just totally gone. 90 more houses had
18 substantial damage in that they would take three to
19 six months to repair. And virtually (every) farm in
20 Mississippi sustained some damage. Now, in the houses
21 that were partially damaged but the chickens were
22 still contained, we were able to pick those up and

1 move them either to other houses on that farm or maybe
2 even to another farm. Injured and unconfined birds
3 were euthanized.
4 Here's one of our houses and yet another
5 and another. And you can see the chickens.
6 Bird losses. We lost 3 million birds
7 outright and another 5 million due to reduced
8 placements and egg sets.
9 So housing. In the houses that were left
10 standing, management was a challenge. Most of our
11 houses are tunnel ventilated. We in many places were
12 not able to do that because of curtains, roofs or wall
13 damage. We did experience some heat loss in our older
14 birds because they weren't acclimated to the heat.
15 And unfortunately, September was the hottest September
16 on record in Mississippi.
17 Because of the feed constraints, we had to
18 feed a single ration, and we also couldn't pelletize
19 it. The birds -- instead of just pre-feed, we had to
20 feed them only twice a day. But we were slowly about
21 to get them back on to a full feed by mid- or the end
22 of September.

1 Now, obviously, we had birds that needed
2 to get desperately out of the field. And in many
3 cases, the processing plant was the best way. But
4 there were concerns about re-opening the plants.
5 First we had to make sure that there was no flood or
6 water damage, which, fortunately, we did not
7 experience.
8 There were power outages. So there was
9 potential product abuse. So we had to dispose of 4.7
10 million pounds of product in our coolers and cold
11 storage. Now, a large majority of that was stuff that
12 was stored down in the New Orleans cold storage
13 facilities.
14 Now, prior to reopening the plants, we
15 also had to assure water potability. So it took us --
16 Hammond and McComb were able to come back online that
17 next Saturday, September 3, Collins and Hazlehurst on
18 that Sunday. And at Laurel, because of water
19 potability, even though, ironically, Laurel had power
20 two days after the storm hit, we couldn't start
21 running and processing birds there until the next
22 Wednesday, a week later.

1 Of course, the poor communications and
2 blocked roads, some people were just completely
3 displaced, and employee attendance was low. To help
4 employees when we were able to communicate to them
5 that the plants had reopened, meals were provided. We
6 gave them free ice.
7 Our human resources people were trained to
8 file to help our employees file for FEMA and Red Cross
9 assistance. We also provided fuel to not only our
10 contract growers to run their generators but also to
11 employees just to get to work. And it continues to be
12 a problem in our region. The industries -- not only
13 ours -- continue to be plagued by labor shortages.
14 Obviously, the birds' condition -- this
15 was a challenge. We had -- we were processing birds
16 aging from the range of the low 40s to mid-70s. We
17 normally process birds that are either 50 days of age
18 or 60.
19 Obviously, we had an increase in
20 variability. And this was particularly apparent when
21 you had multiple-age flocks, because we had to move
22 one age of birds into houses with other ages of birds.

1 There was a moderate -- not as bad as I
2 thought it would be -- increase in condemnation and
3 trim. We did see a slight increase in IP,
4 Inflammatory Process, particularly in those flocks
5 that we had to pick up and move. And there was a
6 slight increase in Zero Tolerance deviations, but,
7 fortunately, that was minimal.
8 And of course, as Dr. Bailey was talking
9 about, there was stressed birds. There was concern
10 about maintaining our *Salmonella* performance
11 standards. So USDA started 51-day windows in all
12 plants in the regions. And I'm pleased to say that
13 all of the plants in our regions -- not just ours --
14 met the performance standards. And there was also a
15 special FSIS team sent to survey the plants, and I
16 believe those results are to be published. But
17 basically, what they did is -- they took pre-evis.
18 samples at the rehang table and post-chill samples.
19 The pre-evis samples they tested of *E. coli*. The
20 post-chill samples they tested for *E. coli* and
21 *Salmonella*.
22 The paired samples that we got from --

1 that we shared with the USDA or they shared with us.
2 We had no *Salmonella*-positives post-chill. The
3 average reduction on the *E. coli* amounts in those
4 samples we had -- in each of the plants, you can see
5 fairly, you know -- well, relatively high CFUs for the
6 incoming pre-evis and -- but really low levels, you
7 know, less than 200, for -- well, less than 50 for
8 colony forming units post-chill.
9 *Salmonella* isolations. We routinely
10 monitor pre-evis and post-chill just to see where
11 we're at. This depicts the dates just prior to
12 Katrina hitting, and you can see where she hit.
13 That's where that dip on the pre-chill is, where --
14 the pre-evis numbers.
15 You see that dip and where it goes down to
16 zero. That's because no samples were being submitted.
17 And what you can see is, you know, we have a
18 fairly -- it fluctuates the incoming, what we're
19 seeing, but fairly -- we were able to maintain a
20 fairly low level post-chill.
21 This is another one of our plants. And,
22 again, you can see the dip when Katrina hit. And this

1 is another plant in which -- they did quite well.
2 Now, as a company, we don't like to just
3 depend on the positive/negative aspects of looking
4 just at incidence levels. We also like -- we also
5 monitor MPNs. And we don't do this for every sample,
6 because this is quite arduous.
7 We use an AOAC-approved method. It's a
8 nine-tube method. So each one of these samples
9 represents nine *Salmonella* isolations, which means
10 over a thousand. But we use this to try to assess
11 what kinds of numbers we're having coming out of the
12 chiller.
13 Now, I know there are a lot more people in
14 here that know a lot more about MPNs. So if I say
15 something incorrect, please correct me.
16 But basically, my microbiologist -- she's
17 not going to report anything as sterile without
18 autoclaving or irradiating. So on this first column,
19 the orange one, the less than two basically states
20 that they couldn't find anything. The next column is
21 colony of a most probable number of one to ten, the
22 next one greater than ten to 100. The next one, the

1 3.7, is greater than 100 to -- it's from 100 to 1,000.
2 And we had zero MPNs greater than 1,000.
3 So with that, in conclusion, we feel that
4 we were able to maintain process control and produce a
5 quality product for our customers.
6 (Applause.)
7 DR. ENGELJOHN: Thank you very much,
8 Marty. And that was very helpful information. I know
9 we -- as an Agency, we're particularly interested in
10 what happened during the hurricane. And it's
11 something we all have to be prepared for in the
12 future.
13 Our next presenter is going to talk about
14 processing and sanitation issues unique to very small
15 establishments. We have with us Dr. Patricia Curtis;
16 she's a professor and Director of Poultry Product
17 Safety and Quality at Auburn University. She has
18 worked with the International HACCP Alliance in terms
19 of presenting a course, and she has a special interest
20 in distance learning.
21 With that, she has received her PhD and
22 master's of science degrees from Texas A&M University.

1 We are pleased to have with us Dr. Patricia Curtis.
2 DR. CURTIS: I'm going to talk to you very
3 briefly about the differences in working with small
4 plants or very small plants and the large plants and
5 some of the challenges that they face in trying to
6 meet *Salmonella* standards. If you look at the very
7 small plants, most of those plants -- when we're
8 talking about poultry, there is a very, very small
9 number of plants that actually do the slaughter.
10 Most of those raw poultry plants are
11 purchasing product from the larger plants. But I want
12 to tell you they have no clue as to -- they think, you
13 know, We're getting it from a HACCP-approved plant, so
14 this product is fine. They don't have a clue if
15 you're in the low level, Category I to Category III,
16 of the contamination level, and they don't have a lot
17 of clue in many cases about how to control that when
18 it comes into their operation, because they don't have
19 the scientific background.
20 And what they -- when they got into
21 business, they got into business more as an art than a
22 science; their family may have been in business, and

1 that's the way that they've always done things. So it
2 creates a little bit of a concern when you're trying
3 to teach some of the concepts. They're relying very
4 heavily on those inspectors that were in the plant to
5 tell them what they needed to do and how they needed
6 to do it.
7 Yes, there's exceptions to all of these.
8 And my view may be a little biased, and I'll state
9 this up front. I work primarily with large plants,
10 and I only get called in to the very small plants when
11 there's a problem. Okay? So I'll say that up front.
12 And if any of you are from very small plants and this
13 doesn't apply to you, I apologize, but that's the
14 reason I wanted to say up front that I deal mostly
15 with the plants that are having the problems.
16 So what I see is that they have that real
17 lack of the scientific background, which causes
18 issues, because, as Dr. Russell was saying, when
19 you're trying to do cleaning and sanitizing -- I've
20 been in many of the plants who are going, Well, I want
21 a cleaner and sanitizer combined; why can't I do that
22 and save a step. And it's really hard to make them

1 understand.
2 And if they start cleaning, they will
3 take -- and they'll get -- Oh, well, if I can clean at
4 a certain temperature, I can double that temperature
5 and it'll even be better or I can double the
6 concentration and it'll be better. And obviously, we
7 know that that's not true. If you raise the
8 temperature too high even during pre-cleaning, you'll
9 start denaturing the protein on there protecting the
10 bacteria, and you've got other problems.
11 So they depend a lot on the sales people
12 that come through, because they get very, very
13 frustrated now that the FSIS is not providing them
14 with some of the information on how they need to do
15 things.
16 Often times, they create a wider of
17 product. They will purchase products in, and then
18 they may be simply portion-control sizing it or
19 handling that product in some manner. Or they may be
20 creating ten different products or 15 different
21 products from that raw product that's coming in.
22 Most of their operations are manual, so

1 there's more opportunity for that cross-contamination.
2 And I've seen many, many of the processors,
3 particularly the very small, where it's a mom-and-pop
4 operation or there's just one. They want to know,
5 Well, why can't I go down to Wal-Mart and buy my
6 supplies?
7 You know, they want to go down and get
8 Clorox for their sanitizers; they don't understand the
9 difference between household cleaners and some of the
10 commercial cleaners that are available. They don't
11 know which cleaners work best in certain situations.
12 And other than just seeing the scale forming from the
13 hard water, many times, they don't understand the
14 impact of what that hard water will have on anything
15 from the sanitizers to even how their product may
16 react -- that they're making.
17 They don't have a clue about the pHs and
18 the effectiveness of the pH so that your sanitizers
19 are effective. And they've many times [inaudible due
20 to failure of in-house PA system] the biofilms.
21 Since they have fewer employees, it's very
22 difficult for this group of people to be reached for

1 training programs. And even when they are able to
2 come in and do training programs -- I know that when I
3 was in North Carolina, we spent one summer -- there
4 was a whole group of us extension people that went out
5 and did training for the very small plants.
6 And they couldn't be away from their
7 facility for three days in a row because there was
8 nobody back home to keep the business going. So we
9 would have to go in and do one day a week for three
10 weeks, or something like that, for HACCP training.
11 So the training assistance has to be more
12 creative for the small plants, but the thing that's
13 even more important is being able to follow up with
14 that person after they've attended the training,
15 because they often get back home and they go, Well,
16 that situation really didn't work for me, because I
17 have something a little bit different. And then they
18 have to find someone who can answer those questions
19 for them.
20 And even more recently, the very small
21 plants that I've seen that are trying to start up an
22 operation -- they don't even have a clue of where to

1 turn. They don't have an idea of where they need to
2 find -- Well, you said that I just can't put back that
3 these are germs, you know. And I'm going, No; you
4 have to be more specific. "Well, I don't know what's
5 on there; how do I find out what's on there.
6 So the question of just being able to
7 identify the hazards becomes a very, very big issue.
8 And the smallest of the plants are usually not members
9 of associations. So they don't have that backing of
10 the associations that many of the larger plants to do
11 provide the information that will help them as they're
12 trying to create a HACCP plan. And they certainly
13 don't have the expertise.
14 A lot of them will have a consultant come
15 in and develop their HACCP plan, which is fine until
16 they have the first deviation or the first problem
17 that's associated with their HACCP plan. And then
18 they really don't know how to fix that problem.
19 So -- and as FSIS comes back each year and
20 looks a little bit more critically at their HACCP
21 plan, they're not prepared for that. They don't
22 really have a clue unless their inspector in their

1 plant is really, really helpful to them and provides
2 them some scientific expertise. And we're seeing a
3 movement away from that supplying of information. So
4 those people are having a much more difficult time.
5 And you might say, Well, look at all the
6 information on the internet; there's all kinds of
7 things out there. But let me tell you. I teach HACCP
8 classes, and I have a mixture of large-plant and
9 small-plant people in my classes. And recently, I
10 spent a lot of time in introductory HACCP.
11 All of you that have done the HACCP
12 classes know you have to write a HACCP plan. And you
13 spend a lot of time writing all that stuff down and
14 then putting it on charts to share with the class. So
15 I thought, Okay, this is great; I'll just buy some
16 tablet notebooks, and I'll pass them out, and the
17 groups can use these; and, you know, we can go in and
18 we can look at some of the FSIS web pages and some
19 other resource web pages.
20 Well, I found that the people from the
21 larger plants are very comfortable with that. They're
22 used to using technology. And the people -- and I

1 can't say this is 100-percent true, but I've found
2 that many more of those very small-plant people will
3 just kind of scoot that tablet under the table and get
4 out their notebooks and keep writing, because they're
5 uncomfortable even with that technology.
6 So we can't assume that just because we
7 post it on the internet, that's the way that we're
8 going to reach everybody. I mean that's a good way to
9 share information, but it's not particularly the best
10 way for all the people to obtain the information that
11 they need.
12 The -- also, the people who are from these
13 very small plants -- they're usually making very
14 unique products in many cases. And they may be making
15 chicken jerky or something. I mean, you know, it's
16 amazing, some of the products that they come up with.
17 Well, where do you go even when they call
18 you or find somebody that has some experience in some
19 micro-background or some processing background and
20 their product is so unique that you're going, Well, I
21 don't know anybody that has ever done any research in
22 that area. And they're at a loss, because they still

1 are expected to come up with validation information.
2 Hopefully, there's a safe harbor that you can point
3 them to, but that's not even always the case.
4 So this validation information that's
5 required by small plants is a very, very tough task
6 for many of those. So they had -- and they have no
7 idea how to run validation studies in their own plant
8 and, if they did, they didn't have the equipment to do
9 it.
10 I was amazed a few years ago in North
11 Carolina. As I said, when I was doing this training.
12 We were talking about calibrating different kinds of
13 thermometers. And can you believe there were some
14 very small plants that didn't even own a thermometer
15 at that time, which is kind of scary?
16 But you have to -- they have certain ways,
17 and I think that we're past that and many of the
18 plants are moving in that direction. But just the
19 expertise of how to handle certain equipment, how you
20 plan out validation experiments that would really work
21 for those operations, is just beyond their
22 expectations. So they need lots of help, but I'm not

1 sure how we get that help to them.

2 The other thing that you run into often

3 times in very small plant operations is that you have

4 only one or two people in the plant. So you have the

5 same people monitoring HACCP that are doing the

6 reviews.

7 And if any of you have ever written things

8 and you've gone back to check your own writing, it's

9 very difficult to find your own mistakes. It's a lot

10 harder to go back and proofread something that you did

11 than to proofread something somebody else did, because

12 you keep reading things in there that -- not

13 intentionally that weren't there -- but it's just

14 harder to catch.

15 So it makes it more difficult for small

16 plants when they go back and are trying to do pre-

17 shipment sign-offs and things to make sure that

18 everything gets done.

19 So they definitely are much more dependent

20 on the in-plant inspectors for -- to provide updates

21 on regulatory information and to provide some of that

22 scientific knowledge that they may need in the

1 processing of their products.
2 I don't know exactly how's the best way to
3 go about reaching small plants to try to teach them
4 how to do validation studies, but I think as we look
5 for more and more validation requirements onto the
6 small plants, there's going to have to be some
7 material that is provided to them to help them figure
8 out, What is a validation study.
9 I know that even working with large
10 plants, you wind up with people going, Well, what
11 exactly are the inspectors looking for when they're
12 talking about validation? Well, imagine that even if
13 you're able to tell them, then when you go to the very
14 small plants, they have no clue what you're talking
15 about. And they have no way of getting the scientific
16 magazines to provide some of it or even finding the
17 people with the right expertise to help them design
18 some way in order to do their validation.
19 So basically, I guess the thing that I
20 want to summarize here from this presentation is that
21 there's a lot of very small plants out there and
22 they're producing a lot of very good product. But

1 they have a lack of scientific knowledge to tell you
2 why that product is safe. That's not to say their
3 products are not safe. I don't want to imply that at
4 all, because many of them -- what they're doing is a
5 very good process, but they can't explain to you why
6 that's a good process.
7 And in a lot of the presentations that we
8 heard earlier today, we were seeing that there's being
9 a request for more documentation on why we're doing
10 the things that we're doing. And so I think that that
11 is a key area that we need to pay particular attention
12 to for the very small plants.
13 And from the sanitation area, those
14 sanitation decisions, hopefully, the sales men that
15 are producing and selling the sanitizers and the
16 detergents are providing some good credible
17 information to these small plants and visit them more
18 and help the small plants understand why they need to
19 use more commercial products than trying to rely on
20 some of those household products.
21 And then from the processing expertise, I
22 think it would help a lot if some of the inspectors

1 would emphasize to these small plant processors the
2 importance of continuing education, because I think
3 they truly don't understand what they would gain.
4 They see it as time that is being taken away from
5 their production, from their profits and from their
6 livelihood, but they don't understand that if they
7 don't participate in some of these trainings, it is
8 going to cause them more problems in the future.
9 With that, basically, I'd like to say --
10 conclude with that the small plants are very different
11 than the large plants in the help that they need. I
12 think that they can produce some of the product that
13 is equally as good and safe as large plants', but they
14 need a lot more assistance from the federal government
15 than do the large plants.
16 (Applause.)
17 DR. ENGELJOHN: Thank you very much, Dr.
18 Curtis. And we'll certainly take that as a challenge
19 to the Agency to make sure that we focus on getting
20 the proper type of information to the audience that we
21 have to serve. And we do think that the large plants
22 have a lot of that expertise that they could share, as

1 well. And so we certainly will be looking to see what
2 partnerships we can form there to get the right kind
3 of information out.
4 We're now going to hear something about
5 the turkey industry. We have with us Michael Rybolt,
6 who is the Manager of Scientific and Technical Affairs
7 at the National Turkey Federation. Michael has his
8 background with his work at Mississippi State
9 University.
10 So we're glad to have you here, Michael.
11 MR. RYBOLT: Thank you, Dr. Engeljohn.
12 Yes, you do have another Bulldog in the
13 house.
14 What they asked me to come and talk to you
15 about today are some of the food safety best
16 practices.
17 (Pause.)
18 MR. RYBOLT: Oh, I'm sorry. You can't
19 hear me? I thought everybody could hear me. I'll
20 talk about the food safety best practices that the
21 turkey industry has.
22 A little bit about the National Turkey

1 Federation. Everybody knows we're a national trade
2 association based in D. C. We are the only trade
3 association representing the turkey industry and its
4 allied industries exclusively.
5 Food safety is considered a high priority
6 for the turkey industry and for the National Turkey
7 Federation. To prove that, several years ago, the
8 turkey industry developed the "Food Safety Best
9 Management Practices for the Production of Turkeys."
10 And this is more of your own farm live production
11 BMPs. It's in its second edition now, and it was
12 updated recently, in 2000.
13 The process is actually pretty intensive.
14 It starts out at your foundation and multiplier
15 breeders, and it goes all the way through to the feed
16 manufacturing and delivery. It covers your live haul,
17 and it also covers your meat bird production and grow-
18 out section.
19 Here's a snapshot of the whole program
20 itself, and it has a flow diagram of each one of the
21 processes and has a control point that you should
22 evaluate in each one of those steps. And it also has

1 a monitoring and feedback component.
2 So it follows the HACCP-like program and
3 is fairly intensive, and it covers everything from
4 disease diagnostics, disease prophylaxis, whole
5 quality, [inaudible due to failure of in-house PA
6 system] servicing, biosecurity, which is big, vector
7 control, drinking water and sanitation. So it covers
8 a lot of the different live production components that
9 you would -- that are important for food safety.
10 We also have developed our ground turkey
11 good manufacturing practices; this was developed by
12 the Tech & Reg Committee. The ground turkey GMPs
13 cover everything from receiving, storage, tempering,
14 grinding, packaging, finished product storage and
15 distribution. It doesn't necessarily have the HACCP
16 flow chart that the live production or the production
17 BMPs have, but it does cover all the different
18 components for producing ground turkey.
19 We also have developed -- we contracted
20 with two researchers, Dr. David Caldwell and Audrey
21 McElroy. David is at Texas A&M, and Audrey is a
22 Virginia Tech Hokie now. We contracted with them to

1 do a chiller study, and it's just a snapshot to see
2 what was going on in five different establishments to
3 see what sort of best practices they use within their
4 chiller systems and [inaudible due to failure of in-
5 house PA system] for the *Salmonella* and *Campylobacter*
6 levels to see what works best.
7 And I'm not going to go through the whole
8 study, but I just want to talk about -- the objective
9 was to assess the emergent chilling and best
10 management practices and their effects on the
11 microbial quality of carcasses, whole turkeys, coming
12 out of the chiller, and then to develop the best
13 practices or recommendations for the industry to
14 follow.
15 Some of the conclusions that the group
16 found were that you need to maintain your total
17 chlorine at 15 to 25 ppm. That there is a discrepancy
18 [inaudible due to failure of in-house PA system] some
19 of the other literature that's published, but this is
20 what was going on in these particular plants at that
21 time.
22 There were different types of chlorination

1 being used, and the type of chlorination in this
2 particular study didn't matter; what really mattered
3 was that you maintained your pH appropriately so that
4 you have the effective form of chlorine available.
5 And what -- the main part of the BMPs was
6 that the chiller can serve as an effective part of
7 your overall pathogen control program as long as
8 you're managing it appropriately.
9 The next step for us is to do a process
10 control mapping. I think you'll hear some other talks
11 later on about that. But the NTF Micro group decided
12 that we needed to do this within the turkey industry,
13 and we decided that we would go to different
14 processing points and measure for different organisms
15 to see what was going on at that particular process.
16 I tried to get them to do every single
17 process, but, of course, money comes into play and you
18 can't do that. But we did settle on some of the key
19 points or key processes within the slaughter section
20 of the plants: Before scald, after scald, after the
21 pickers, at rehang, pre-crop, post-/pre-chill rinse or
22 antimicrobial application and then, of course, post-

1 chill.
2 The plants are going to take five wing
3 samples at each one of those locations, and the
4 samples are going to be pooled. And we're going to
5 test for *Salmonella*-positive or -negative at this
6 point in time. We're also going to quantify generic
7 *E. coli* and do APCs, as well. And the pilot is going
8 on right now.
9 We have a handful of plants that are going
10 through this protocol at this point, and we're
11 planning on doing this for about 30 processing days so
12 that we can get enough data and look at the data to
13 determine, Where do we need to concentrate? Do we
14 need to concentrate only at, you know, rehang? Do we
15 need to concentrate only at, you know, pre-chill? And
16 then we'll go back and launch this industry wide to
17 other plants and let them start sending in data.
18 Again, once we're done with the pilots,
19 we'll take the data and analyze it and present it to
20 the entire committee and industry and try to get
21 everybody to participate with shared data to NTF so
22 that we can collect all this data, analyze it and

1 discuss it and talk about what practices the companies
2 are using, what works best and what doesn't work best
3 to try to develop some BMPs there and to build on what
4 we already have in place at this point in time.
5 We have, you know, our live side. And we
6 have our ground BMPs. And now we're going to build
7 onto those with the process mapping.
8 I do have just a snapshot of data. I
9 don't have enough to really show this, but I did -- I
10 thought it was interesting to show what we do have at
11 this point in time. Like I said, we're just in the
12 very beginning of this; we only have a few days of
13 results in.
14 But here you have the APC and generic *E.*
15 *coli* results from some of the plants. And this is --
16 the red is the APC, and the yellow is the *E. coli*.
17 And you can see a reduction in the process from pre-
18 scald all the way down to post-chill.
19 So we are showing process reduction. I
20 don't have any *Salmonella* data. I know this is a
21 *Salmonella* meeting, but I don't have anything to show
22 you at this point, and I do apologize for that. But I

1 guess this is -- Dr. Engeljohn hasn't shown me a sign
2 yet. So we're doing good.
3 The -- again, just to emphasize, this
4 mapping study is in its infancy. We're just getting
5 started, and we're only a few days into this.
6 But the goal of all this is to build onto
7 the already -- the programs that we already have in
8 place within the industry and to further develop our
9 best management practices for the production of
10 turkeys. And, you know, this is going to help in the
11 hurdle approach. You know, there's no one silver
12 bullet, unfortunately. So it's going to build on the
13 programs that we already have in place and, hopefully,
14 help decrease the *Salmonella*.
15 (Applause.)
16 DR. ENGELJOHN: Thank you, Michael, for
17 that presentation on turkeys. And the fact that you
18 don't have information on *Salmonella* -- did you know
19 we are going to start [inaudible due to failure of in-
20 house PA system] turkeys?
21 MR. RYBOLT: I didn't know that.
22 DR. ENGELJOHN: Well, we are.

1 (Laughter.)

2 DR. ENGELJOHN: Well, we now are

3 [inaudible due to failure of in-house PA system] check

4 with the people on the phone. But if you would, as we

5 did this morning, if you have any questions of any of

6 the panelists from this afternoon's session, please

7 come up to the microphone and identify your name and

8 association and then ask your question.

9 MR. WALTHER: Hi. I'm Jeff Walther from

10 Mount Air Farms, and I have a question, I think, for

11 Stan Bailey and maybe a little bit for Mike.

12 And that's, Would you think I was crazy to

13 think that we could -- we're trying to save our birds

14 during the live-haul process by a technique I learned,

15 you know, a million years ago. And we go out there,

16 and we drench the birds coming out of a house on a 90-

17 degree day, and the birds come out of the house white

18 and they arrive at the plant tan.

19 And, you know, our *Salmonella* -- we do a

20 fairly expanded sampling for *Salmonella*. It's just

21 presence or absence. But I expected to see some

22 tremendous numbers in the summer time when we did

1 that, and I haven't. And is it because I just -- I
2 should be looking at colony forming units, or, you
3 know, am I just missing it, or have you got any idea?
4 Is that just a -- is that a bad practice?
5 DR. BAILEY: Can you hear me?
6 (Pause.)
7 DR. BAILEY: Can you hear me now?
8 VOICES: Yes.
9 DR. BAILEY: Maybe I could get a job on
10 the Verizon commercial.
11 (Laughter.)
12 DR. BAILEY: It's a mixed bag. The good
13 thing is you're doing that for a reason, and that
14 keeps the birds from getting stressed. And anything
15 that keeps the birds from getting stressed is going to
16 keep them healthier and keep their intestinal tract
17 more intact. And so that's a good thing.
18 Any time you're adding moisture to
19 anything to do with *Salmonella*, it makes it worse.
20 The fact that you're not seeing elevated levels of
21 *Salmonella* because of the process probably indicates
22 that you've got a very low level coming in. That

1 would be my biggest guess.
2 MR. WALTHER: We do not have a low level
3 coming in. I'm sorry.
4 DR. BAILEY: But -- well, then if you've
5 got a very high level, then it doesn't have that much
6 room to go up. So I don't --
7 (Laughter.)
8 MR. WALTHER: Okay. That's great.
9 (Laughter.)
10 DR. BAILEY: Other questions?
11 (Pause.)
12 DR. ENGELJOHN: If we could ask the
13 operator if there's anyone on the line that may have a
14 question?
15 (Pause.)
16 DR. ENGELJOHN: Does anyone else in the
17 audience have something?
18 MR. SANCHEZ: Marcos Sanchez from Texas
19 A&M. I just want to follow up on the same question he
20 just had on the humidity levels and the increase in
21 *Salmonella*.
22 I know that I mean when I did some work,

1 too -- I mean the drier air we had at -- we had less
2 numbers, too, but we still were able to recover some
3 of it. And I know that there were some publications
4 recently or some findings recently that -- actually,
5 when you have humid environments, you have more
6 competition, too. That competition keeps those
7 numbers down -- of *Salmonella*. And so I was just
8 wanting to know your perspective on that, too.
9 DR. BAILEY: Well, it's true that if you
10 have more moisture, everything's going to grow better.
11 But everything that I've personally worked with and
12 all of the literature that I've seen in the past, from
13 Ed Mallenson's [phonetic] work at Maryland to some of
14 the industry data that has been looked at internally
15 by some of the companies, would suggest that your
16 biggest problem with *Salmonella* is always moisture.
17 Anything you can do to keep an environment
18 dry is probably the greatest thing you can do to
19 reduce the prevalence of *Salmonella* in your chicken
20 houses. Surely, any -- you know, if you increase the
21 good bacteria or your competitive flora, then you'll
22 be making the situation better, but I don't think it's

1 worth the trade-off of worrying about doing that via
2 moisture management.
3 If you want to increase the good bacteria,
4 there are some other ways. You can look at some
5 prebotics or, if you can find it, an approved
6 competitive exclusion-type product. But I don't think
7 I would be attempting to do that with moisture
8 management.
9 DR. ENGELJOHN: While the gentleman is
10 coming up to ask a question, if the panelists have
11 anything you want to add or you -- to anyone's
12 presentation or anything else, also, raise your hand.
13 MR. BENSON: Hi. Mike Benson from Jennie-
14 O.
15 Dr. Bailey, I was intrigued by your
16 measurement of *Salmonella* in the houses with the fly
17 strips. In Minnesota, we don't have many flies in the
18 winter time. I was wondering if you have any
19 suggestions for other measurements on what the load is
20 coming into our processing plants -- other ways of
21 measuring what the live load is.
22 DR. BAILEY: Well, there's a number of

1 ways people go about that. As you -- and you can do
2 fecal or cecal grab samples or litter samples. I
3 think probably the most commonly used sampling methods
4 are either drag swabs or using, like they do in
5 Scandinavia, kind of a foot-sock which is basically an
6 oversized wristband that you put over the bottom of
7 your feet and walk around the house with. That has
8 proved to be a particularly effective measure.
9 There -- each of these methods have some
10 advantages and disadvantages. I would suggest that,
11 whatever method you use, you use it consistently,
12 because it will -- you will be able to get a
13 historical perspective of what you're doing and
14 whatever the positives or negatives of the method
15 you're using will cancel itself out over time and
16 you'll be able to see relatively what you're doing.
17 I personally -- some of the companies I
18 know also do antimicrobial testing on the birds a week
19 or so before they go to the slaughter plant. And they
20 kill, say, three birds in the plant and look at the
21 fat pads for antibiotics. And they take those birds
22 and look at the cecal content of those birds. So

1 that's another potential alternative.
2 As I say, there's a number of different
3 ways you can do it, and I would just be consistent.
4 Decide what you're going to do, and use the same thing
5 all the time.
6 DR. RUSSELL: Yes. I'd like to add to
7 that. I saw a paper I edited one time where they
8 compared the use of drag swabs to just using those
9 surgeons' booties that you put over your feet and
10 walking around the house. And I think they
11 recovered -- 2 to 3 percent of the samples were
12 positive for *Salmonella* using the drag swabs and maybe
13 11 percent with the surgeons' booties. And they
14 concluded that that was a much more effective way to
15 do it. So just sort of to add to that.
16 MR. COUGHLIN: I'm Michael Coughlin from
17 Johnson Diversity. And this question is for Dr.
18 Bailey.
19 Can you speak to the prevalence of
20 *Salmonella* within the feather follicle pre- and post-
21 picking, if in fact that microbiology has been done?
22 DR. BAILEY: I know that in all the 30

1 years -- 32 years I've been working, people say that
2 the *Salmonella* gets driven into the follicles. But as
3 I'm sitting here really thinking about it, I can't
4 honestly say that I personally know of a research
5 paper that has demonstrated that. But maybe somebody
6 else in the audience -- I think it probably is true,
7 but I don't know.

8 Mark?

9 DR. BERRANG: I was involved in some work
10 where we were -- a graduate student developed a method
11 to visualize *Campylobacter* live and dead --
12 *Campylobacter*, now -- live and dead cells at different
13 heights down into a feather follicle. And what she
14 found was that she was able to find some viable
15 *Campylobacter* deep down in the feather follicle, but
16 the numbers were much, much higher on the upper
17 surface of the skin.

18 And most of what I see in the
19 literature -- and Dr. Buhr might have something to say
20 about this, but most of it seems to be anecdotal, you
21 know: "The feather follicles open up, and then they
22 close, and, boy, I'll bet you that's where they are."

1 But what I see with the *Campylobacter* data suggests
2 that the upper surface of the skin is really where we
3 find more numbers of viable *Campylobacter*, anyway.
4 MR. COUGHLIN: Right. And this is what
5 I've noticed, too: That there's very little
6 information in the literature. I was wondering if my
7 search was inadequate.
8 But I'm also wondering if the types of
9 populations in the feather follicle would be dependent
10 upon the oxygen content, like a biofilm, maybe
11 anaerobic at the bottom and aerobic at the top.
12 DR. BERRANG: That was our thinking with
13 *Campylobacter*. That -- you know, since it's a
14 microaerophilic organism, it might really be happier
15 down in there. But, again, it didn't seem to be borne
16 out by the data in that particular study.
17 DR. BAILEY: I would think that would also
18 be very dependent on when you chose to look, because
19 that feather follicle until the feather is pulled out
20 is going to be full.
21 So if you went in, you know, very soon
22 after that, the atmosphere or the lack of oxygen or

1 whatever the micro-environment is there would not have
2 had a whole long time to select for a given
3 population. But if it was, you know, a week or two
4 later after processing, you might see a different
5 situation then than you would immediately after
6 processing.
7 DR. ENGELJOHN: How about on the phone?
8 Did we hear anything?
9 (Pause.)
10 DR. ENGELJOHN: No one from the audience
11 has anything they want to follow up on?
12 (Pause.)
13 DR. ENGELJOHN: Well, with that, then
14 let's take a break. We'll come back in -- at 2:45.
15 (Whereupon, a short recess was taken.)
16 DR. ENGELJOHN: We'll go ahead with the
17 afternoon session. We get to leave early if we get
18 all the presentations done. So --
19 (Pause.)
20 DR. ENGELJOHN: Well, welcome back to the
21 afternoon session. I notice that there are a few
22 empty seats -- not that you're not coming back to the

1 presentation and that people might be standing
2 outside.
3 But I do want to remind everyone that if
4 in fact you're not going to be back tomorrow or you're
5 going to leave early today -- we do have evaluation
6 forms out on the table, and we would like you to fill
7 them out to give us some input as to how we can
8 enhance the future public meetings that we have. So
9 please stop by, pick one up and fill it out.
10 Our next presenter is Dr. John Cason, who
11 is an animal physiologist scientist with the
12 Agricultural Research Service at USDA. He has worked
13 for ARS for 16 years, with most of his research
14 focusing on poultry processing. He has his degrees
15 from the University of Georgia.
16 We're pleased to have John Cason with us.
17 Thank you.
18 (Applause.)
19 DR. CASON: Thank you. Good afternoon to
20 everyone. I'm glad to be here, and I want to thank
21 FSIS for the invitation to make a presentation.
22 Basically, I'm going to be talking about factors that

1 limit the effectiveness of antimicrobial treatments.
2 And in case you can't read this in the
3 back of the room, this is a "Dilbert" cartoon, and
4 he's in his cubicle making a PowerPoint presentation
5 on his computer. And he says to himself, "I no longer
6 feel the need to change the real world as long as I
7 can change these bullet points." And I'm sure
8 everyone here has done a PowerPoint presentation and
9 had that feeling before. To avoid that in my case, I
10 want to start with a photograph of a chicken to keep
11 me connected with reality.
12 (Laughter.)
13 DR. CASON: Okay. So now we're all set.
14 So the question I want to ask is, Why
15 can't we remove or kill all of the bacteria on poultry
16 carcasses? And of course, outside of cooking and
17 irradiation, there really is no way to either wash off
18 the bacteria or kill the ones that are on the carcass
19 or to do that to all of them, anyway.
20 And this is an anecdotal sort of graph
21 here from a paper by Huda Lillard from about 25 years
22 ago -- well, my math might be off a little bit -- 20

1 years. And this is -- the three lines here show
2 carcasses that were hand-picked before being scalded
3 on the top line. And the middle line is carcasses
4 after scalding and picking, and the bottom line is a
5 carcass that has been eviscerated and is about ready
6 to go into the chiller.
7 And these are numbers of aerobic bacteria
8 recovered in consecutive carcass rinses of the same
9 carcass. And you can see that the higher levels --
10 when you have a dirty carcass, there is a bit of a
11 curve to the line, but as you get down through the
12 plant and the carcasses are cleaner, the lines are
13 relatively flat. So that -- each consecutive rinse
14 washes off about the same number of bacteria.
15 And this is the results for the same
16 carcasses, showing the number of enterobacteriaceae
17 removed from carcasses. And of course, this family of
18 bacteria has several pathogens that are of interest to
19 us.
20 And I want to direct your attention to the
21 bottom line here, which is the carcasses just about
22 ready to go into the chiller. And if you look at the

1 level there -- I don't know if we have a pointer up
2 here, and, if we did, I wouldn't know which screen to
3 point at. If you look at the tenth rinse -- there's
4 maybe 4.7 logs of enterobacteriaceae that were removed
5 from that carcass in the rinse.
6 And I did a little calculation down to the
7 40th -- I'll just point at my screen. I did a little
8 calculation down to the 40th rinse. And from ten to
9 40, about a million enterobacteriaceae were removed
10 from that carcass. But a carcass rinse down there at
11 Number 40 was unable to detect that difference. And
12 so whenever we do a sample, we have to keep in mind
13 that our interpretation has to be based on how that
14 sample is taken.
15 Besides that study that I just showed you,
16 there have been five studies that I could find where
17 they did consecutive rinses on enough carcasses to do
18 some statistical analysis. And the table here shows
19 the kind of bacteria that they were checking for and
20 the number of times the carcasses were rinsed, and
21 that's a total of 32 comparisons between two
22 consecutive rinses.

1 And of those 32 comparisons, there was
2 only one report of a significant difference, and that
3 was in aerobic bacteria. Now, I suspect that if you
4 did enough carcasses, there would be many more
5 significant differences found, so I think the problem
6 is not that the number doesn't decline slightly with
7 each successive rinse, but that the level of variation
8 is so great that it's just about impossible to find a
9 significant difference.
10 So why do bacteria persist on carcasses so
11 that we can rinse carcasses 40 times and still get off
12 the same number of enterobacteriaceae? And I'm going
13 to talk about some reasons that have been given in the
14 scientific literature. And I was really pleased to
15 hear some of the earlier discussion.
16 Number One: Bacteria in feather
17 follicles. It was suggested as far as I know first by
18 a British microbiologist in the 1960s.
19 And there was a similar suggestion in a
20 study of turkey carcasses by Avens and Miller where
21 they couldn't understand why they were able to keep on
22 getting bacteria off the carcasses, and they said that

1 maybe some of the bacteria are subcutaneous. The
2 feather follicle idea has been repeated dozens of
3 times; it's in some pretty high-powered publications
4 by national committees and investigatory bodies that
5 should know all about things.
6 And this is an electron micrograph that I
7 found in a paper by Thomas and McMeekin from 1984.
8 And this is chicken breast skin before water
9 immersion. And that little bar down in the lower left
10 is 30 microns. The usual size range for *Salmonella* is
11 about 1-by-3 microns. It's a little rod.
12 And so end to end, you could probably put
13 about ten *Salmonella* bacteria along that black bar.
14 And so if you have the idea now of the size of a
15 *Salmonella*, you can see lots of places down in those
16 little crevices where they could hide.
17 And the next photograph shows the chicken
18 breast skin after it has been soaked in water for 30
19 minutes. And you see the skin takes up water and the
20 crevices become much deeper. And now there are many
21 more places for the bacteria to hide. And I'm not
22 suggesting that they hide consciously, but it's an

1 artifact of -- well, maybe they do, since they can
2 talk to each other, as somebody else said.
3 (Laughter.)
4 DR. CASON: And so I did some
5 calculations. There's a standard formula for
6 calculating the surface area of a carcass. And so I
7 did a 2-kilogram carcass. That's a 4.4-pound
8 eviscerated carcass going in the chiller. It would
9 take about 2-1/2 million of these photographs to show
10 you the entire surface of the skin of that carcass.
11 And on the way over here this morning, I
12 came with some really smart people. So I was asking
13 them how many feathers a chicken has. And the
14 estimates that we have seen in the literature -- this
15 thing is turning on and off up here.
16 The estimates in the literature range from
17 5,000 to 9,000, so I used 10,000 as a convenient
18 number. And that's really a high estimate. I would
19 have to show you on average 250 of these slides for us
20 to see one follicle. Now, granted, some of the
21 follicles are really thick -- the flight feathers and
22 the tail. It would take about four of these high and

1 four wide to show one entire follicle on some of those
2 larger ones.
3 And so even though I could argue that just
4 in terms of numbers follicles should not really be
5 very important, it's more important to do some
6 experiments and ask the chickens.
7 And for the last four or five years at the
8 Russell Center, Jeff Buhr has been maintaining a flock
9 of featherless chickens. Now, this was a spontaneous
10 mutation that arose in a flock somewhere, and Jeff got
11 someone to send him some eggs.
12 And this is a single recessive gene. And
13 if both parents are heterozygous, 25 percent of the
14 chicks are hatched without feathers and -- they're not
15 entirely without feathers. I think you can see there
16 are a few little tufts of feathers here and there.
17 But we've been using this as an experimental model to
18 see what differences it makes if a chicken has
19 feathers or it does not. And of course, if they don't
20 have feathers, they don't have follicles.
21 So we've done at least three experiments,
22 and there are probably several more in the pipeline.

1 And basically, our research shows that follicles don't
2 make any difference.
3 There may be a short in this mic -- or
4 something.
5 We tested de-feathered and chilled and
6 then carcasses that had been stored for a week for
7 lots of different kinds of bacteria. And basically,
8 the chickens without feather follicles are not any
9 different.
10 In agriculture, I don't know if we have
11 urban legends; we probably have rural legends.
12 (Laughter.)
13 DR. CASON: And so I think that feather
14 follicles don't make any difference at all. Of the
15 two studies that I know about where they have
16 published photographs of *Salmonella* and *Campylobacter*
17 bacteria in feather follicles, in both cases, the
18 follicles were soaked for several hours in a
19 concentrated suspension with millions of bacteria.
20 And so I think that neither of those studies with the
21 photographs really reflects the real world.
22 Physical attachment of the bacteria to the

1 carcasses has been suggested. Biofilms have been
2 mentioned, but a traditional biofilm is a mat of
3 bacteria on a physical surface growing out into a
4 liquid. And so I don't think that biofilms really
5 match up with poultry.
6 Physical attachment to the surface to me
7 doesn't seem to be terribly convincing even though I
8 know it happens. And the main reason is that the
9 surface changes so much while the carcass is going
10 through processing. The cuticle is moved in most
11 plants, and part of the epidermis below that, but,
12 yet, the bacteria still persist on the carcass.
13 Some people also suggest there might be a
14 clumping effect and that the clumps are there and
15 every time you rinse, one or two cells break off the
16 clump, and so the numbers don't change. That may be
17 possible, although, I think, in a lot of photographs,
18 there seem to be relatively small numbers of bacteria
19 in groups on the surface, not just big clumps.
20 Surface chemistry has been suggested. The
21 presence of fats and oils can protect the bacteria.
22 The pillae and flagella -- the bacteria -- have

1 sections that recognize chemical receptors on the
2 surface of the skin; however, when rinses have been
3 done with surfactants and detergents and enzymes such
4 as lypasis and proteinasis, the increase of numbers of
5 bacteria that come off the carcasses is really very
6 modest.

7 So I don't think that surface chemistry --
8 even though this may have some effect, I don't think
9 it really explains why we can get so many bacteria off
10 of carcasses.

11 I think surface physics is one of the most
12 convincing explanations. People talk about surface
13 tension in the water, the formation of water layers
14 around the carcass, the lack of ability to get shear
15 forces right up on the skin of the carcass to remove
16 bacteria. And there's a possibility that you might
17 get into an exchange-in-equilibrium situation where as
18 many bacteria are coming back onto the carcass as are
19 coming off as you're doing your rinse.

20 And I have a cartoon here, and, luckily,
21 it's showing up well on the big screens if not on the
22 little screen. I made this up; this is sort of a

1 "Dilbert" sort of thing.
2 And what I have here is -- you can see the
3 yellow chicken skin. And there are some crevices with
4 some bacteria in them, and there's a water layer once
5 the carcass starts through processing. It has been
6 dipped in water and the scalder, and it has been
7 sprayed several times, and there's a water layer that
8 can't be removed; it can be evaporated, but you can
9 take the towel, and you can't get that off.
10 Okay. Now, in this one, this has more
11 blue up above it; you've added the water to do a
12 carcass rinse. And so you start to shake, and the
13 arrows show that there's a good velocity of water out
14 in the rinse, but as you get closer to the surface,
15 there's less and less velocity. And in fact, by the
16 time you get down to those bacteria in those little
17 crevices, they don't even know that somebody's doing a
18 carcass rinse out there.
19 Now, several physical things have been
20 done to carcasses to try to increase their removal or
21 the elimination of the bacteria, including bubbling.
22 Bubbling does increase the removal of bacteria from

1 carcasses; however, it has a side-effect. In a lot of
2 applications, it increases water take-up by the
3 carcasses. So there might be some problems there.
4 Ultrasound in some uses can be expensive.
5 It does kill bacteria. However, ultrasound
6 treatments are used by the people who study biofilms
7 to break up the biofilms so that they can count
8 individual cells, and so ultrasound might be a way
9 where you can kill lots of bacteria and then get a
10 worse result when you do your carcass rinse.
11 Brushing has been tried. I think it
12 recontaminates the carcass about as fast as it removes
13 the bacteria. And I want to show you some preliminary
14 results of some scraping experiments that we've done
15 at Russell just recently.
16 Here's a cartoon again showing that water
17 layer that can't be removed. And we scraped with
18 about a 2-1/2-inch piece of stainless steel. And you
19 can see a lot of material -- perhaps fat and liquid
20 and bits of the cuticle -- can be removed. And there
21 were large numbers of bacteria in this material on the
22 little blades, but when we tested the carcasses by a

1 whole-carcass rinse after chilling, there were no
2 differences.
3 And this is a little cartoon that's
4 supposed to represent the stainless steel scraper
5 blade going over the surface. And the pressure
6 flattens out everything on the surface and closes up
7 the crevices so that bacteria down in the crevices are
8 completely protected. It may remove some from the
9 surface, but, again, it does not reach all of the
10 bacteria on the surface.
11 And so I went over some of the things here
12 that have been suggested as the reasons for why you
13 can't get all the bacteria off of the carcasses. I
14 think a lot of research has been done on looking for a
15 silver bullet, an antimicrobial chemical.
16 But the trouble is not that we haven't
17 found the right silver bullet; it's that we can't get
18 the antimicrobial chemical into contact with the
19 bacteria that are in those crevices on the carcasses.
20 So I think that new approaches are needed to improve
21 the efficacy of antimicrobial treatments.
22 And I just got the two-minute warning. So

1 we're actually running ahead of time, I think. Thank
2 you.
3 (Applause.)
4 DR. ENGELJOHN: Thank you very much for
5 that presentation.
6 Our next speaker is Dr. Jeff Buhr; he's an
7 animal physiologist scientist with the USDA's
8 Agricultural Research Service. He has his degrees in
9 avian sciences and a PhD in veterinary anatomy from
10 the University of California, Davis. His research for
11 the past nine years has focused on broiler processing,
12 specifically feed withdrawal, de-feathering, crop
13 removal and bacterial decontamination, and currently
14 is looking at environmental lighting programs and
15 their effect on colonization by pathogens.
16 DR. BUHR: Well, thank you very much.
17 Thanks for the invitation.
18 I've been asked to talk specifically about
19 scalding, de-feathering and rehang. And my topic will
20 mainly stick with *Salmonella*, but we also have to talk
21 about *Campylobacter*, because I don't have data on
22 everything. And I'm not going to talk about any

1 silver bullets or antimicrobials. And at the end,
2 we'll have a little bit with the featherless chickens.
3 If you look at the relative levels -- and
4 this is something to remember -- when we're talking
5 counts or colony-forming units, how many bacteria,
6 this is easy to do on a whole-carcass rinse on a
7 feathered bird or when it's in a dump coop shoot.
8 However, when we're talking incidence, if we didn't
9 enrich samples post-chill, we're not going to find
10 very much *Salmonella*. So we need to keep -- in
11 "incidence" or "prevalence," we're talking about
12 percentage here.
13 If we look through processing and the
14 presentations we've had this morning, in general,
15 scalding is going to decrease numbers, and it may or
16 may not decrease incidence. And I'll show you some
17 data to support that.
18 Everybody agrees de-feathering is going to
19 increase numbers, increase incidence, specifically
20 related to the decrease we just had with scalding.
21 Rehang is pretty neutral. Most of it's automatic.
22 It's not going to really increase numbers that you

1 can't wash off. It may increase incidence if you
2 don't wash off.
3 Here are some data from Mark Berrang,
4 looking at *Campylobacter*. Now, this one's different
5 from the *Salmonella* we had this morning. This is
6 individual flocks through a processing plant. And if
7 you look at the pink one on the top, you'll see that
8 it tends to be the highest one as we go from pre-scald
9 to post-chill. But the important thing to remember,
10 also, is as we go into post-chill, everybody ends up
11 down there in the same bottom part about one log,
12 whole-carcass rinse, except for the pink one.
13 So it's important when you're sampling a
14 processing plant to make sure you're sampling the same
15 flock as it goes through the plant. Otherwise, you
16 may get increases or decreases that are flock related,
17 not necessarily plant related.
18 This one talks about scalding: Triple-
19 tank, counter-flow, S-shaped. What does scalding do?
20 Scalding, as we've heard, loosens feathers. It
21 removes feces, litter from the process in house, and,
22 also, bacteria. It also enables us to loosen

1 feathers, and it's also going to take off most of the
2 epidermis.
3 What are the positive aspects? The good
4 thing -- when I say, "Positive," I mean good, not
5 positive meaning a positive sample. There's some
6 physical equipment advantages with processing.
7 Multiple tanks. We've seen triple-tank
8 scalders; with three tanks, you can have three
9 different temperatures. We're not convinced that
10 temperature is all that important as opposed to --
11 three tanks appear to do just as good a job.
12 Counter-flow. The water's coming in when
13 the chickens are going out. So clean water is getting
14 on the cleanest chickens.
15 Triple-pass. Each tank, the birds are
16 exiting the tank the opposite from where they entered.
17 The negative aspects of scalding. It's a
18 common bath. Any time you have a common bath or
19 common surface, there's a possibility for cross-
20 contamination. It's immersion. There are very few
21 spray scalders or steam scalders out there that stay
22 in the plants for any length of time. We have the

1 potential for internal contamination.

2 Another disadvantage of making this
3 presentation: Very seldom do people report data or
4 prepare something with scalding without the
5 combination of picking.

6 This is a reference to what Mark said
7 we -- if you'll look at the chicken on the left
8 here -- this is the dirty chicken from the
9 conventional processing fiberglass floor. The one on
10 the right was on the elevated wire, a lot cleaner.
11 And we've seen birds a lot dirtier than this. And
12 yes, these birds have a lot more bacteria -- on the
13 left.

14 So to aid with this, which has also been
15 mentioned, people have added brush machines, either
16 pre-scalding or post-scalding. They have a preventer
17 now that squeezes the bird's empty cloaca. Electrical
18 stimulation is supposed to induce defecation. All
19 claim to have reduced fecal matter going into the
20 scalder so you have a cleaner bird to start with.

21 Here are some samples from John Cason
22 where we're looking at a triple-tank scalder. The

1 birds enter from the left and go to the right. We've
2 shaken these samples up. Visibly cleaner water.
3 Now, if we look at the micro on this, we
4 have the triple-tanks left to right in the columns, we
5 have a single-temperature tank up on the top row,
6 triple-tanks on the bottom. You can see, as you go
7 from left to right, the recovery of *Salmonella* in the
8 water decreases. So we're not recovering very much
9 *Salmonella*.
10 Now, if we look at the carcasses, all the
11 way over to the right, three quarters of the carcasses
12 still positive. Half the carcasses scalding. It
13 isn't doing that much for *Salmonella* cross-
14 contamination. And we're actually reducing it with
15 this triple-tank.
16 What about the respiratory tract? We
17 talked about immersion scalding. It was mentioned
18 this morning about stunning, electrical
19 immobilization, where they're aspirating contents.
20 What happens in a scalder where it's immersion?
21 This is a study we did again with Mark
22 Berrang. We looked at *Campylobacter*, coliforms, *E.*

1 *coli* and total aerobes before and after scalding and
2 picking -- before and after scalding. We didn't pick
3 these birds. I'm sorry.
4 If we look at *Campylobacter*, it's very
5 low. If we did a whole-carcass rinse on these birds,
6 30 out of 30 were positive for *Campylobacter*. In the
7 respiratory tracts, only 11 out of 30 were positive.
8 .7 is our minimum level of detection. That was one
9 colony on two plates -- I mean a one is one colony on
10 one plate.
11 So *Campylobacter* isn't really a problem in
12 scalding. Other bacteria do increase during scalding.
13 Can we prevent this increase? We thought, "Well, can
14 we prevent this increase. What if we put a black
15 cable-tie around the neck during bleed-out? Will that
16 stop it, or is this some other factor breaking up
17 clumps and increasing the number of bacteria in the
18 respiratory tract?"
19 When we looked post-pick -- I'm sorry --
20 post-scald, we saw the blue bars on the right. We
21 completely eliminated that increase. Well, the
22 increase due to immersion scalding is a passive aspect

1 during going to water. The question was raised:
2 Well, what does this have to do with respiration?
3 Could these birds be struggling? What if they were
4 deader? Would it have an effect?
5 And we said, Well, for a bird to
6 ventilate, it has to have neural reflexes from the
7 brain stem; so let's decapitate the birds. We
8 decapitated the birds. No longer ventilation. We'll
9 see if this is purely mechanical or it's a biological
10 effect.
11 If we look at the light blue and the dark
12 blue columns, these are the birds that we decapitated.
13 And we go from pre-scald. The light green are the
14 ones that weren't decapitated -- stunned and bled.
15 Light blue, there's no difference. If we look at
16 coliforms, you know, 4.3 and 4.2, decapitation.
17 So this is purely passive. We're putting
18 the bird under water, increasing pressure and then
19 releasing that pressure. It has physics -- it has
20 nothing to do with the bird being -- ventilation.
21 Let's talk about picking. The positive
22 aspects? It removes the feathers; it also removes the

1 epidermis. But it also removes the bacteria on the
2 feathers and epidermis that were put on there in the
3 house and also in the transportation coops.
4 Negative aspects? As we've heard today,
5 it expresses cloacal contents into the picker and then
6 spreads it around.
7 Here's a study we did with Mark Berrang
8 where we wanted to demonstrate the increase post-scald
9 during the picker of *Salmonella* and *Campylobacter*.
10 The left side is *Salmonella, and the right side is*
11 *Campylobacter*.
12 We put ten-to-the-seventh *Salmonella* in a
13 gel capsule into the cloaca of the bird before
14 scalding. That's the left column. We did a breast
15 swipe post-picking, all negative. The birds that
16 we -- I'm sorry. The left side is post-scald prior to
17 picking. The dark yellow one is after we picked them.
18 We have 84 percent positive for *Salmonella*, 57
19 percent positive for *Campylobacter*, indicating that
20 contents are squeezed out of the cloaca.
21 But this is *Salmonella* and *Campylobacter*
22 in peptone, not in fecal matter. So we did another

1 study. We added ten-to-seven *Salmonella* to one
2 milliliter of cecal contents and spread it on the
3 breasts. We did this every other bird. If you'll
4 notice, the second bird and the third bird have a dark
5 streak on them. We had a leader bird and a tailer
6 bird. So we didn't have an effect of bouncing around
7 the picker.
8 And we also did this with the featherless
9 birds. You see the first featherless bird and then
10 the third featherless bird have a fecal sample on
11 them.
12 We took breast squares off the birds post-
13 pick. You can look at the results.
14 If we look at the two left bars --100
15 percent of the birds that we put *Salmonella* on we
16 recovered it from. That's pretty good. In 100
17 percent of the birds that we didn't put *Salmonella* on
18 we didn't recover any *Salmonella*.
19 After we picked them, the left column, 81
20 percent were the birds we put *Salmonella* on; 88
21 percent of the birds next to that we didn't put
22 *Salmonella* on. It didn't matter what position, didn't

1 matter if they were featherless or feathered; the
2 picker is spreading *Salmonella* around.
3 If we look at the histology section on the
4 left here, we have skin prior to scalding and picking
5 with *Salmonella* on top. You see the epithelial layer,
6 the base membrane right here -- the basal cells.
7 After scalding and picking, that is literally ripped
8 off the chicken. So in addition to redistributing the
9 cuticle, it's redistributing the *Salmonella* on the
10 carcass.
11 We came up with some possible ways for,
12 Well, what can we do to prevent this. And we came up
13 with cloacal plugging. Mike Musgrove did this in '97.
14 And it would decrease *Campylobacter*. We can show you
15 the results here.
16 This is, again, pre-pick and post-pick
17 plugged. In the ones pre-picked, we didn't get any
18 *Campylobacter*. In the third column, we didn't plug
19 them; 100 percent of these birds were positive for
20 *Campylobacter* after picking. In the right column,
21 they were plugged; only 11 percent. So we decreased
22 it, but we didn't get 100 percent. Why didn't we get

1 100 percent? Well, sometimes the plugs leaked. And
2 sometimes we have to go a little bit harder. If we
3 look at some *Campylobacter* data from that
4 transportation study, we had the dirty birds, which
5 would be in the green, the clean-wire birds in the
6 white. We saw *Campylobacter* dramatically decrease
7 after scalding and picking.

8 We plugged these birds and removed the
9 heads and the feet. So scalders are cleaning it up,
10 but we're not getting it down to zero.

11 If we look at featherless birds here --
12 this is where the featherless birds come in -- the
13 left column would be normal birds that weren't
14 plugged. In the right column, they were plugged and
15 sutured closed. We sutured them closed. And you can
16 see we virtually eliminated *Campylobacter* from a post-
17 pick carcass if we sutured the vent closed. And you
18 can see how we can do that.

19 In a normal bird, on the left, sewing the
20 vent closed, you get some feathers in the way. It's
21 not easy to do. On the right, it's easy to sew the
22 vent closed with a dead, featherless bird.

1 This shows them after picking. This slide
2 also on the left side shows how big this feather
3 follicles are. Okay. Everybody can see those. I
4 didn't see anybody say they could see *Salmonella*. So
5 *Salmonella* can go in and out of those feather
6 follicles if it wants to, but it's not really a big
7 factor.
8 What happens with *Salmonella* while in
9 scalding and picking if you eliminate vent leakage?
10 Nothing. The scalder knocks down *Campylobacter*.
11 These are the same birds pre-scald and post-scald. It
12 didn't do anything at all.
13 Well, we thought, What if we pick the
14 birds longer? If we're redistributing the *E. Coli* and
15 the *Salmonella*, what if we did it twice as long? We'd
16 get rid of more of it.
17 The left two bars, we picked them for 30
18 seconds; the right two bars for 60 seconds.
19 *Campylobacter* and *E. coli*, the first bars are
20 feathered and featherless. It didn't make any
21 difference. So picking them longer or more pickers
22 isn't going to make a difference.

1 The third topic I'm supposed to talk about
2 is rehang, where we're going from the kill line to the
3 evisceration line. This is mainly machine transfer
4 now, physically transferring from your de-feathering
5 line to your evisceration line. It's going to
6 minimize external surface cross-contamination if they
7 make a correct transfer.
8 Positive aspects? Physically different
9 lines, plus we're removing the heads and feet from the
10 evisceration line -- sources of contamination.
11 Negative aspects? We can get leakage from
12 the vent, and we can also get leakage from the
13 esophagus and the crop. Time on the rehang table can
14 be a problem especially if you have a slowed-down
15 evisceration line.
16 If we look at the digestive tract of the
17 bird, on the left side, we see the end of the
18 esophagus and the crop, but realize that the
19 [inaudible due to failure of in-house PA system]
20 there. And the bird is hanging upside-down, so it can
21 leak contents.
22 Similarly, on the right side, we have the

1 vent and the ceca contents. I'll show you some data
2 that -- feed withdrawal doesn't do much to their
3 content.
4 Let's start with the ceca and some work we
5 did with Arthur Hinton. We did feed withdrawal from
6 zero hours, on the left, to 24 hours. The green bars
7 show the weight of the ceca. We see feed withdrawal
8 had no effect. We've seen that ten minutes post-
9 mortem the digestive tract is still active.
10 Now, on the other hand, we're looking at
11 *Salmonella,* in the pink bars. *Salmonella*'s still
12 there. So if we're going to leak contents any time
13 during processing, we're going to leak *Salmonella*.
14 Now, we've all looked at crops that are
15 empty and full in birds. Similar data with crops.
16 However, if we look at the left, after about 12 hours
17 of feed withdrawal, we have an empty crop.
18 Unfortunately, during that time period, the
19 concentration of the counts of *Salmonella* are going
20 up. We've removed the lactic acid bacteria feed
21 source, pH has gone up and *Salmonella* is going to
22 bloom.

1 Now, at the end, I was also supposed to
2 talk about the featherless birds. John mentioned
3 them. It is an autosomal recessive that came in a
4 Leghorn background. We had to make three out-crosses
5 to commercial broiler breeders. It's described in
6 Poultry Science if you want some more detail.
7 This is the second generation, a breeder
8 male on the left, a female on the right. They do have
9 a few down feathers; we haven't gotten rid of 100
10 percent of the feathers. We treat them just like
11 normal broilers. You can see they have leg bands.
12 And as John says, when we did these
13 studies, we paired equal body weight of feathered and
14 featherless birds. The only thing they need is a heat
15 lamp in this type of area in this type in the winter
16 time.
17 Let's look at post-chill data, feathered
18 birds on the left, featherless on the right. Pick
19 your bacteria. There's no difference.
20 Let's look at the *Salmonella* and
21 *Campylobacter* we're supposed to talk about. These are
22 counts -- okay -- direct plates, 22 to 28 percent

1 positive. This was one out of -- 8 percent positive
2 for *Salmonella* with direct plating, *Campylobacter* up
3 there at 97 and 94 percent. If we enrich the samples,
4 it jumps up there.
5 So *Salmonella* is now at 83 and 77 percent;
6 it doesn't matter if they're feathered or not
7 feathered. *Campylobacter* was 100 percent. Now, we
8 made these birds positive by challenging them the week
9 before.
10 So the absence of feathers and empty
11 feather follicles does not result in lower carcass
12 bacteria recovery from post-chill carcasses. It's
13 when we're giving the carcass to the consumer.
14 And that's the end of my presentation.
15 Thank you.
16 (Applause.)
17 DR. ENGELJOHN: Thank you, Jeff. That was
18 very interesting.
19 Now we're going to talk about the
20 mechanics of poultry processing, and we have with us
21 David McNeal from Meyn America. David is from here in
22 Georgia. He has been with Meyn since 2004, and he has

1 a food technology background.
2 So welcome.
3 MR. McNEAL: Thank you for the invitation.
4 [inaudible due to failure of in-house PA
5 system] we manufacture poultry processing equipment.
6 And I was asked to speak today about reducing
7 digestive tract contamination on carcasses during
8 processing from an equipment standpoint.
9 I've listed several factors which affect
10 broiler carcass contamination. The microbial load of
11 the live bird which arrives at the processing plant.
12 This includes the bacteria on the skin and inside the
13 digestive tract, which is out of the processor's
14 control.
15 The conditions and content of the GI
16 tract. This includes how much time the birds have
17 been off feed and water and what they have been
18 eating.
19 Processing equipment which is not set up
20 properly. This includes parts and equipment of the
21 wrong size and equipment not adjusted properly.
22 Maintenance of equipment. Parts such

1 as -- that come into contact with the broilers -- with
2 the carcasses that can be bent or scratched up or
3 moving parts that can be worn out.
4 All of these factors are related to
5 minimizing broiler carcass contamination. Processing
6 plants will always be challenged with differing grades
7 of these factors.
8 Here's a picture of the digestive tract of
9 a chicken. I've listed the most critical parts which
10 pertain to contamination.
11 Once feed enters the esophagus, it will
12 first travel to the crop. If there's food already in
13 the gizzard, then the feed will stay in the crop until
14 the gizzard is empty. We know if broilers are without
15 feed for too long, then the crop conditions will
16 change, and the amount of *Salmonella* can greatly
17 multiply.
18 Once food exits the gizzard, it will pass
19 into the duodenum. This portion of the intestine is
20 important to the process because of its location in
21 the carcass. The duodenal loop lies just below the
22 abdominal surface, and this is the portion of the

1 intestines that is most often cut during the opening
2 process.
3 We also know the ceca and colon are
4 locations in which high numbers of *Campylobacter* and
5 *Salmonella* can be found. The colon or bing area is
6 the area most often damaged with the bing cutting
7 machine.
8 There are differences between breeds and
9 strains which affect size and shape of broilers.
10 Size, live average weights and shapes of birds greatly
11 influence equipment setup and adjustment and ultimate
12 performance. For example, a breed raw 708 is bred
13 with a long breast and has longer legs as compared to
14 a Taw, which is thicker with shorter legs.
15 Other factors which affect conformity of a
16 flock are differences in feed conversion between
17 breeds and gender differences. All of these affect
18 the efficiency of the process.
19 Machines are designed specifically to
20 operate within a given weight range, usually within
21 three to four pounds. And generally, a machine setup
22 is only needed if the average live weight changes of

1 the flock more than one pound. If there's too much
2 variation in a flock, then equipment will have to be
3 set to a happy medium; this results in a reduction in
4 equipment performance and an increase in process
5 contamination.
6 Now, these two photos I have here are
7 of -- I've got -- the photo on the left is of vent
8 cutting blades. I've got five different sizes, and it
9 may be hard to tell the difference. But the smaller
10 diameter blades are used for lighter weight broilers,
11 and larger ones are used for heavier birds.
12 The picture on the right is of two
13 different lifting units for an eviscerator. The one
14 on the left has a different angle, and more material
15 is used to position smaller birds. The one on the
16 right is designed to allow for more space when lifting
17 the bird for positioning and machine function. If the
18 processor's live weight changes dramatically, then
19 changes greater than the machine's adjustment may be
20 needed. Parts may need to be changed.
21 The weight variations in this slide I got
22 from our U. S. install list. As you can see, there's

1 a large range of weights which fit into the young
2 broiler class of inspection. We're often asked to
3 deliver systems which can process live weights from 3-
4 1/2 to 8-1/2 pounds. The 3.6 to 5.4 weight range
5 represents markets targeted for cut-up, and weights
6 above 5-1/2 pounds are mainly used for deboned
7 product.
8 Traditional inspection systems, which
9 include Streamline Inspections Systems, SIS, and New
10 Evisceration Line Speed, NELS, were established around
11 the same time the introduction of the 180-degree
12 machines or round machines, as we call them, and were
13 used to improve the performance and quality of the
14 evisceration process.
15 In the last ten years, advances in
16 technology such as Meyn Maestro and Stork Nuova
17 evisceration systems have established a process which
18 completely removes the viscera pack from the bird and
19 presents it separately from the carcass for
20 inspection. By separating the viscera pack from the
21 carcass rather than draping it over the back of the
22 carcass, it was microbiologically proven to reduce

1 internal and external pathogens.

2 Here I have a picture of two different
3 evisceration shackles. The shackle on the left is
4 made of stainless and is what we call a rigid or one-
5 piece shackle. When a carcass is hung in this
6 shackle, the shackle hangs straight, and the carcass,
7 therefore, is tilted out. This can impede the
8 operations of some machines because the machine cannot
9 lift the carcass with complete manipulation.

10 The shackle on the right is plastic. It
11 is hinged from a building. And at the bottom, there's
12 a 90-degree break. This is used to allow the carcass
13 to hang straight for more manipulation and placement
14 by the machine. The more carefully and consistently
15 birds are aligned, the greater the performance of that
16 machine.

17 I want to focus on three pieces of
18 equipment in the evisceration department: The venting
19 machine, the opening machine and the eviscerator.
20 These three all deal with eviscerating the viscera
21 pack and digestive tract in the bird, from which a
22 carcass can be contaminated.

1 The venting machine is the first machine.
2 It's job is to remove the vent and bursa of
3 Fabricius, often called rosebud, and to position it
4 over the back of the carcass. If this is not executed
5 correctly, then the opening cut cannot be achieved and
6 there will have to be manual evisceration; this can
7 cause an increased number of carcasses to be
8 reprocessed.
9 Some possible reasons for a vent not being
10 removed. The bird may not have been fed into the
11 units of the machine. This could be a timing issue
12 with the machine or a misaligned in-feed guide bar.
13 The vent or rosebud could still be
14 attached to the bird. Possible solutions for this are
15 sharpening of vent cutter blades, change of
16 positioning of the bird or lowering the cam to be --
17 the lower cam could be too low.
18 Cutting the intestines and/or back, kidney
19 or hip damage could be causing the machine being
20 adjusted to the wrong height or wrong size parts for
21 average live weight.
22 I've got several pictures here of

1 carcasses just coming out of the vent machine. In the
2 first one, you can see the carcass is hanging straight
3 and it's in a two-piece shackle.
4 The second one is of a carcass with the
5 vent and rosebud hanging over the back of the carcass.
6 And as it has been vented properly, if there's any
7 fecal leakage, it will not drip onto the surface of
8 the carcass. The next picture is just a top view, and
9 this opening created by the vent machine is what the
10 opening machine needs to open the abdominal skin.
11 Opening machine. The function of this
12 machine is to open the abdominal cavity to prepare the
13 carcass for evisceration. It's output demands are no
14 cut guts, no damage to keel or breast meat and to
15 leave sufficient breast meat coverage of the keel
16 area.
17 There are many different kinds of opening
18 machines. There are those which use a blade very
19 similar to a box-cutting blade, which enters at the
20 opening of the vent and springs toward the keel in a
21 swinging motion.
22 There's a scissor-type opener which cuts downward

1 through the skin and membrane in a clamping action.
2 And there's a cross-cut style opener, used mostly in
3 Central and South America.
4 The picture at the bottom is an example of
5 what can happen with the box-cutter-style opener if
6 it's not adjusted properly. As you can see, the skin
7 is exposed, downgrading the product and exposing the
8 breast meat.
9 Here I've listed performance failures for
10 the opening machine. A bird can miss the units and
11 not feed into the machine properly. The same
12 solutions as before: Check the timing of the machine
13 and in-feed guide bars.
14 Cut guts from the opener could be caused
15 from improper timing of the cam with an overhead line
16 or a proper height of machine. A long or short
17 opening or cut keel can be caused from improper
18 positioning of the bird. For example, if the bird is
19 too big to fit into the machine or too small to be
20 positioned, then the cut will not be uniform.
21 All of these pictures here are of a
22 scissor-type opening machine. As you can see, the

1 photo on the left is the unit in an open position.
2 The blade is open. In the middle photo, you can see
3 that -- there's a picture of it closed. And you can
4 also see that the bottom guide bar of the blade
5 prevents the blade from cutting the guts. And the
6 third picture is just of the opening machine in
7 operation.
8 Draw machine. This type of evisceration
9 machine is often called a draw machine because it
10 draws the viscera pack and digestive tract out of the
11 cavity of the bird. It uses a spoon to pull the guts
12 out of the bird and drape them over the back of the
13 carcass. It works off of a central cam.
14 The working principle of this machine is
15 that the spoon enters the abdominal cavity near the
16 keel area, it travels past the liver and positions
17 itself just below the gizzard. As the machine turns,
18 tension is created with the use of the central cam,
19 and the spoon scrapes the viscera pack and digestive
20 tract out of the bird. Each viscera must then be
21 manipulated into a presentation for inspection
22 personnel.

1 The original patented technology for
2 complete viscera removal was developed by Meyn. With
3 the eviscerator, the viscera pack is transferred to a
4 pack take-over device, which is then distributed to
5 different colored pans. The different colored pans
6 are then presented to inspection personnel in a
7 predefined position along the conveyor line with the
8 appropriate carcass.
9 Key actions of the Maestro eviscerator
10 include accurate positioning of the carcass and the
11 spoon, capturing the trachea and esophagus, and
12 complete removal of the viscera pack. Key performance
13 indicators of the Maestro are: Greater than 99
14 percent removed viscera packs, properly presented
15 packs with viscera and carcasses to inspectors for
16 disposition, and a less than 10 percent liver damage.
17 The working principle of the Maestro spoon
18 is that it enters the abdominal cavity near the keel
19 area, travels past the liver and stops between the
20 crop and the gizzard. The viscera pack is then pushed
21 through the spoon with pressure from the central cam.
22 The spoon rotates to close tightly, clamping the

1 esophagus before the spoon lifts out the viscera pack.

2 The photo on the left just shows the

3 Maestro in operation. You can see the spoon is inside

4 the cavity and it's in an open position. The picture

5 on the far right shows the pack take-over device as it

6 deposits the viscera pack onto the trays. And the

7 photo in the middle shows the carcass and viscera pack

8 in line for inspection.

9 I've discussed the operational functions

10 and performance criteria for the machines which deal

11 with gutting the bird, the ones most critical for

12 controlling contamination. But I would like to

13 briefly discuss the methods of prevention, with method

14 of kill, removal of fecal matter and reduction of

15 contamination with an inside/outside bird washing.

16 Here I've pictured a cross-section at the

17 base of a head. Here you can see the location of the

18 jugular veins and carotid arteries in the neck, of

19 which all or some must be severed at the death of the

20 animal. You can also see the locations of the

21 esophagus and trachea.

22 A shallow cut will result in less than an

1 optimal bleed-out and will also leave the crop intact
2 until the head is removed by the head and trachea
3 remover just after picking. Removing the head in this
4 manner will often tear the crop, and its contents will
5 spill upon the carcass. We know from previous
6 research that depending on the length of feed
7 withdrawal, the conditions of the crop could change to
8 favor the growth of *Salmonella*.
9 If the animal's killed by using a deep
10 cut, a faster bleed and death can be realized. The
11 connection of the crop to the head will be severed,
12 and the crop can be removed by the Maestro eviscerator
13 without spilling the crop contents. The cropping
14 machine at the back of the line actually becomes more
15 of a trachea machine.
16 Processors that do whole-carcass deboning
17 already utilize this method of kill. The head must be
18 completely removed because the carcass shell and neck
19 will be mechanically deboned. No skeletal fragments
20 may remain.
21 Fecal removal machine. This machine can
22 be used as a preventive measure. The machine is

1 located in the slaughter line after bleed-out and
2 prior to scalding. This machine expresses fecal
3 matter and rinses it off of the carcass.
4 We know carcasses will deposit fecal
5 contents in the skull and/or in the pickers, thereby
6 spreading and cross-contaminating each carcass which
7 passes through them. The machine is designed to help
8 reduce the organic load at the earliest stage in the
9 process.
10 Ever since the implementation of the Mega-
11 Reg in '96, plants have been required to meet a zero
12 tolerance regulation for physical fecal contamination.
13 Uses of inside/outside bird washers have greatly
14 increased; most plants have two per line. This
15 machine uses many nozzles and various water pressures
16 and consumptions to clean the inside and outside of
17 the carcass.
18 The use of this machine is designed to
19 reduce contamination; it is not designed for
20 prevention. It's only performance criteria is 100-
21 percent cleanly rinsed birds.
22 In conclusion, broiler carcass

1 contamination is influenced by the condition of the
2 bird when it arrives at the slaughter plant.
3 Processors can use preventative methods when possible
4 and maintain equipment properly -- which is used for
5 evisceration to reduce carcass contamination. Thank
6 you.
7 (Applause.)
8 DR. ENGELJOHN: Well, thank you.
9 That leads us to our next presentation,
10 dealing with reprocessing of fecal contaminated
11 carcasses and the use of antimicrobials. We have Dr.
12 Stan Bailey back with us. He is a microbiologist with
13 the Agricultural Research Service at USDA.
14 Stan?
15 DR. BAILEY: Thank you, Dan.
16 (Pause.)
17 DR. BAILEY: Thank you. The second area
18 they asked me to talk about this afternoon was
19 reprocessing. And I guess maybe the reason they asked
20 me to do that is because we did in our laboratory -- I
21 wasn't involved directly in the very first project
22 I'll talk about, but the others after that. We did

1 the very first work in this area back in the '70s.
2 A little bit of background before I get
3 into the actual reprocessing. In the early '90s, '92
4 and '93, after the Jack-in-the-Box *E. coli* 0157:H7
5 issue with beef and with recurring issues with
6 *Salmonella* and other issues with poultry and other
7 animal species, there was a concerted effort made by
8 USDA to begin to try to improve this situation.
9 What came out of that led to a lot of
10 meetings and other things and the Mega-Reg, the HACCP
11 document that we still refer to today. But one thing
12 that was a central tenet of those discussions at that
13 time was fecal contamination, the assumption being
14 that if carcasses were fecally contaminated, then they
15 would have a greater propensity for having *Salmonella*
16 and other pathogens.
17 At about the same time -- well, I'll get
18 into that in a second. With fecal contamination,
19 there's a couple of issues. Obviously, there's the
20 aesthetics of the situation. It's not particularly
21 palatable to think that the meat product that you're
22 buying or want to eat would have feces on it. And

1 then there's microbiological concerns, the assumption
2 being made that if you have fecal contamination, then
3 you have a higher likelihood of *Salmonella*.
4 And one of the central tenets that was put
5 forth by USDA at that time was that all species would
6 be treated equally in terms of allowability of fecal
7 contamination. That is not a bad theory and idea, but
8 it's somewhat problematic when you think that species
9 aren't exactly equal. Many types of products have the
10 skin removed, and that's an entirely different
11 situation than poultry which has skin on. So there's
12 a lot of issues that we could discuss about that.
13 But to the idea of reprocessing, about
14 1972 or maybe even '73, Ken May, who at that time was
15 with Holly Farms, came and talked to Roy Blankenship,
16 who was the research leader in our unit at that time,
17 and asked him if we could take a look at an issue that
18 was really causing a lot of issues for the poultry
19 industry.
20 And at that time, approximately 1 percent
21 of the birds were having to be reprocessed because of
22 visible fecal contamination. And the only thing you

1 could do at that time was to trim the product if it
2 had fecal contamination on the outside. Or if it had
3 internal feces, you had to discard the product.
4 And so we -- that was just slightly before
5 I started working with them. But they initiated a
6 study in 1975 that was the first to show that
7 inspection-passed and offline re-processed broilers
8 were basically microbiologically indistinguishable.
9 And this is one of the data slides from
10 that study. And you can see if you look at the
11 inspection-passed or -condemned product on an external
12 swab -- this is looking at enterobacteriaceae, which
13 is the family that *Salmonella* is in -- there was no
14 difference. And if you washed it with water again,
15 there was no difference.
16 In internal swabs, the condemned were a
17 little higher, and you got somewhat lower, but not
18 microbiologically significant, after washing. And so
19 you see what happened with the initial study.
20 So then a few years later, FSIS had taken
21 that under consideration and rewrote the rules in the
22 Code of Federal Regulation, which allowed that under

1 the supervision of a USDA inspector, reprocessing
2 treatments were allowed, including trimming,
3 vacuuming, washing or a combination of these. If
4 internal contamination's present or treatments other
5 than trimming are used, the entire carcass must be
6 washed with water containing 20 parts per million.
7 So what that means in simple terms is that
8 after this regulation was passed, visually fecally-
9 contaminated birds as identified by an inspector were
10 allowed to be pulled off the line and washed and put
11 back on the line. When that process went into place,
12 there was a lot of concern by consumer groups and
13 others that this was maybe not a good idea.
14 So we revisited that issue and published
15 in 1993 another study showing the microbiological
16 quality of conventionally processed and reprocessed
17 broilers. And you can see here that they're basically
18 microbiologically indistinguishable between the two.
19 And again, I wanted to point out that this study was
20 done with just water alone, no chemical treatments.
21 About this same time, Amy Waldroup and
22 some of her co-workers also looked into this issue as

1 for commercial reprocessing of broiler chickens. And
2 they used 20-parts-per-million chlorine in the carcass
3 wash water and determined the effect also that
4 reprocessing was having on *Campylobacter*.
5 So they were the first ones that looked at
6 the *Campylobacter* issue in reprocessing, and they
7 found that there was some plant variability, but they
8 concluded that current reprocessing procedures were
9 microbiologically justified and that on reprocessed
10 carcasses, there were no significant differences in
11 *Salmonella* prevalence or numbers and that
12 *Campylobacter* were either not affected or were
13 significantly lower than in commercially processed
14 birds.
15 And then in 1997, Dan Fletcher at the
16 University of Georgia published a study where he
17 showed that on-line reprocessing reduced the need for
18 off-line reprocessing by 73 to 84 percent.
19 He found that *Salmonella* and *Campylobacter*
20 incidences were not affected by on-line, versus off-
21 line, treatments, there was no significant differences
22 in treatments between the effects on aerobic plate

1 counts, *Campylobacter* or coliforms, and summarized
2 that on-line processing of visually contaminated
3 carcasses could greatly reduce the number of carcasses
4 being subjected to off-line reprocessing without
5 negative effects on bacteria and pathogen counts.
6 And then in 2003, our next speaker, Julie
7 Northcutt, published a paper where she showed the
8 effect of bird washers on carcass microbiological
9 characteristics. And in this study, she was looking
10 just at the effect of washing and the equipment itself
11 with water, not the chemical effect, and found no
12 differences were found in coliforms or *E. coli* counts
13 due to washing in an inside/outside bird washer, that
14 total aerobic plate counts were lower on carcasses
15 from one plant, but not on carcasses from two others,
16 and washing in water alone did not significantly
17 change carcass bacteriological characteristics.
18 So that brings us to the second half of
19 what they asked me to talk about, and that is the use
20 of chemicals. So we have shown historically that --
21 pretty much almost every study that has ever been down
22 has shown that you can reprocess carcasses and

1 microbiologically it'll be indistinguishable from
2 inspection-passed carcasses.
3 So that has led to the use of a lot of
4 chemicals in the plant. Either it is reprocessing
5 aids or, just in general, to try to get down to meet
6 the *Salmonella* performance standards. And the
7 chemicals -- and I'm sure I left somebody's off. And
8 if you're in this room and I didn't put your chemical
9 on here, I apologize.
10 But the ones that are fairly commonly seen
11 is: Chlorine, probably the most widely used of all of
12 the chemicals -- can be up to 50-parts-per-million
13 product contact; a fairly new chemical on the market
14 place, Cecure, cetylpyridium chloride; Inspexx, which
15 is Peroxyacetic acid; $Safe_2O$, which is an acidified
16 calcium sulfate; Sanova, which is an acidified sodium
17 chlorite, TomCO, which is a CO_2/chlorine system, and
18 then; TSP, or trisodium phosphate.
19 And each of these have been used or tested
20 a great deal, and there's a good bit of publications
21 on them. And I didn't have time to really go into the
22 individual use of each of these, and so I won't try to

1 do that. But I think there are a couple of
2 considerations as we think about all these chemicals
3 that we really have to consider and keep in mind.
4 One, what is the organic load that you're
5 going up against? A lot of these chemicals will kill
6 things in a test tube. The key is: Can you get the
7 active ingredient in the chemical to the bacteria that
8 you're wanting to kill? So the amount of organic load
9 on a bird can be imported. And some people -- it has
10 been variable results with the benefits of using
11 brushes and scrubbers, but there are -- some people
12 have found that using brushes to get that organic load
13 minimized does help the process.
14 Then there's the issue of dipping. Can
15 you -- and you see different permutations that people
16 are using: Pre-chill dips, post-chill dips. The
17 chiller itself, the immersion chiller, is really a big
18 dip tank. I mean usually 30 to 45 minutes in a dip
19 tank. So having that exposure to these chemicals in a
20 dip tank can be effective.
21 And then there are sprays. There's
22 continuous spraying of equipment. I was part of the

1 team and led the studies that did the initial work
2 back in the '70s that led to the regulation requiring
3 continuous spraying of 20-parts-per-million chlorine
4 to all common equipment surfaces that touch birds.
5 And so we know that spraying of equipment can keep a
6 build-up from happening over time.
7 There's external spray cabinets where
8 you're just doing like the old-line final washes,
9 where you're washing the outside of the birds. And
10 then there's inside/outside spray cabinets, as you've
11 just heard some talk about.
12 So all of these can play a role. And you
13 see different companies using different permutations
14 of these.
15 There's a lot of issues and concerns with
16 chemicals, though, as we talk about it. Certainly,
17 export markets are one. Depending on where in the
18 world you might be exporting to, there's different
19 rules and regulations. If you wanted to export to
20 Canada or Europe, you certainly can't use the elevated
21 levels of chlorine. And I don't know all the rules
22 for all the countries, but I know that there are

1 issues you have to deal with.

2 There's organoleptic quality or the

3 potential issues with organoleptic quality for certain

4 of these chemicals. Certain chemicals will do a good

5 job of killing *Salmonella*, but maybe they might

6 discolor or have some off-flavors or something that

7 you'd be concerned about from an organoleptic point of

8 view.

9 I think a really important factor that

10 people have to consider is your water chemistry; all

11 water is not created equal. If it's a municipal

12 system, depending on what they're using -- are they

13 using chlorine or chloramine or some other kind of

14 chemical in the water already -- the hardness of the

15 water, the amount of trace minerals in the water --

16 everything doesn't work the same as everywhere.

17 So if we go in as researchers or as

18 regulatory agencies and we evaluate a plant and they

19 seem to be having real good results, there's a lot of

20 things that could be contributing to that. One, what

21 is the load of pathogens coming in on those birds to

22 start with? That might be a big factor.

1 But another factor may be, Well, this
2 chemical seems to work really well in this plant, but
3 it doesn't work very well over there. Well, that
4 might have to do with water quality or a lot of other
5 issues.
6 There's also worker health issues when we
7 talk about chemicals. Some of these chemicals are
8 pretty toxic. If you go in a plant that's running
9 maximum levels of chlorine, sometimes in those areas,
10 it'll just about knock you over. And some of the
11 other chemicals can have issues that we have to be
12 concerned about for worker health.
13 And certainly, sampling technique is very
14 important. We know from many of the early studies
15 with some of the chemicals that looked particularly
16 effective that what we were doing was not necessarily
17 killing the *Salmonella* on the carcass.
18 What we were doing was -- that carcass
19 carried some of that residual active ingredient of
20 those chemicals with it. And when you did a rinse
21 sample of the carcass to see if it had killed the
22 *Salmonella*, you kept that residual chemical in your

1 rinse water, and it would kill it there. It hadn't
2 killed it on the carcass.
3 So when we're doing these studies, we have
4 to be particularly cognizant of that issue and make
5 sure that we neutralize the chemical during the
6 sampling process.
7 I'm just going to pick a couple of -- I'm
8 not trying to promote this particular chemical in what
9 I'm doing here. I want to make that very clear. But
10 I did want to show you a couple of selected studies
11 where you can see that chemicals when used properly
12 can be effective in helping you reduce your levels of
13 *Salmonella*.
14 In a study that was published by Kemp and
15 co-workers in 2001 -- and it was, I want to point out,
16 a company-funded study, and I think that's always
17 important to know, but -- looking at acidified sodium
18 chlorite spray system, the microbiological quality of
19 fecally contaminated carcasses was found to be
20 significantly better than that of off-line reprocessed
21 carcasses. And all but two of 1,127 carcasses passed
22 the zero fecal tolerance test.

1 So this allowed the -- this was taking the
2 reprocessing step and putting it in a continuous on-
3 line system and using a chemical to help you control
4 the issue. And you were able to do that without
5 having to take things off-line.
6 And this is some of the data from that
7 study, and I won't go over all of it. But if we look
8 at just the *Salmonella* line, after the eviscerator,
9 the carcasses were about 37 percent positive. After
10 the continuous on-line reprocessing, they were 10
11 percent positive as compared to the birds that were
12 taken off-line for reprocessing, which were slightly
13 lower than the post-evis, but still at about 32
14 percent.
15 And even if we take these birds that were
16 continuously on-line reprocessing and going through
17 the chiller, they were still only 12.5 percent
18 positive. So as I say, I'm not trying to selectively
19 pick out and advocate this chemical, but this is an
20 example of some good results that we're seeing.
21 And then at Auburn University, they did
22 also a study looking at the post-chill application of

1 acidified sodium chlorite. And this is something I
2 hadn't talked about earlier, but this is taking a dip
3 after the chill tank.
4 So you've gone through the entire process,
5 and you have a post-chill dip. And using this -- at
6 Auburn University, using this acidified sodium
7 chlorite, you can see the pretty good results that
8 they were getting. The *Campy* levels were down and
9 extremely low, and so were the *E. coli* levels.
10 I'll take about -- the last two minutes I
11 have is almost exactly the amount of time I'll need.
12 This very same study I talked about earlier I won't go
13 over. And it was the 20 plants. We worked with FSIS
14 plants in the post-pick and post-chill. And, again,
15 the same things we looked at.
16 But the reason I wanted to go over this
17 real quickly was to show you that for what -- we have
18 survey data showing us what each of these plants were
19 using, and they're all over the board. People are
20 using different things, but I just wanted to show you
21 what was happening in the plant.
22 If we look at the rehang station and we

1 look at *Campylobacter* levels, we averaged across that
2 study log 2.66. By the time we came out of the chill
3 tank, we were at log .43; in *E. coli*, 3.28 to .92. If
4 we -- you can look at the data by season and see the
5 really pretty straight-line effects that you were
6 getting, very consistent effects, with *E. coli*.
7 And if we look at *Salmonella*, which we're
8 talking about today, you can see that at the rehang
9 station, we were bringing in or after -- by the time
10 they got through the picker, anyhow, about 72 percent
11 of the birds were *Salmonella*-positive. But by the
12 time we came out of the chill tank, over the course of
13 the whole year that we did this study of 1,600 samples
14 each, that -- we had about 19.6 percent positive.
15 So that's not great in terms of where we
16 want to be, but I think it does show that the plants
17 were doing something that was pretty good. I mean
18 they had a very dramatic reduction.
19 The next and last slide. When you pair
20 the samples as we talked about before, you can get an
21 idea of how effective your process control is in your
22 plant.

1 But if you look at the slide here, you
2 can't see all of the individual lines, I don't guess,
3 but each one represents a plant. They represent what
4 was happening at that plant and what the levels of *E.*
5 *coli* were on those carcasses at the rehang station in
6 the first side. And they represent what the rehang
7 station was -- I mean after the chiller on the right-
8 hand side.
9 And if you look at the slope of the line,
10 you can tell how good a job those plants were doing in
11 reducing the bacteria load of an indicator organism
12 that's always there. The problem with doing this with
13 *Salmonella* is it's sporadic; sometimes it's there, and
14 sometimes it's not.
15 By taking -- as Jeff had referred to
16 earlier, in taking paired samples within a flock --
17 when you use within a flock, you eliminate a lot of
18 those other variabilities you're concerned about. You
19 eliminate seasonality. You eliminate weather
20 conditions, whether the carcasses got wet and all
21 that. And you just see what happens in that
22 individual plant.

1 And if you do this over time, you're going
2 to know what you would expect if everything's working
3 properly. So that is a potential to measure process
4 control, we feel.
5 In conclusion, reprocessing and on-line
6 reprocessing can and do result in processed chickens
7 that are equal to or have improved microbiological
8 quality compared to inspection-passed chickens.
9 Chemical interventions in processing are resulting in
10 significant improvements in prevalence of *Salmonella*
11 and in reductions in *Campylobacter* levels in broilers.
12 But I do believe that we do need a
13 caution. And somebody asked the question earlier.
14 Continued use of large quantities of chemicals may
15 lead to increased concerns with export markets and
16 perceived public health issues by some people. So I
17 think that's something we need to keep in mind. And I
18 thank you very much.
19 (Applause.)
20 DR. ENGELJOHN: Thank you, Stan.
21 Realizing it's the afternoon and we've
22 been going for a little over an hour, now we are going

1 to take a ten-minute break. So come back at 4:05, and
2 we'll finish out the afternoon and then have questions
3 and answers.
4 (Whereupon, a short recess was taken.)
5 DR. ENGELJOHN: We have with us Dr. Julie
6 Northcutt, who's going to talk about the impact of
7 chilling on the poultry carcass microbiology. Dr.
8 Northcutt is a research food technologist and lead
9 scientist in the poultry processing research unit at
10 the Russell Research Center, Agricultural Research
11 Service with USDA. And Dr. Northcutt has her degrees
12 in food science biochemistry from North Carolina State
13 University and Clemson University.
14 Welcome.
15 DR. NORTHCUTT: Thank you, Dr. Engeljohn.
16 And I appreciate the opportunity to speak
17 with you this afternoon. And I'd like to thank you
18 all for staying toward the end here. Also, I'd like
19 to thank the rest of the folks at the FSIS who have
20 organized this, and specifically Dr. Patty Bennett and
21 Dr. Bill Shaw.
22 As mentioned, I'm going to talk for the

1 next few, 15, minutes about immersion chilling and air
2 chilling and how they affect poultry microbiology. So
3 as we know, poultry is chilled primarily to reduce
4 microbial growth.
5 And the methods include, as I've already
6 mentioned, traditional immersion chilling, which is
7 the method that is most commonly used in the United
8 States, although air chilling is becoming more
9 popular. Air chilling can either be a dry air chill
10 or what is called an evaporative or spray air chill.
11 And in unique situations, there have been combinations
12 of the two.
13 There have been a number of studies on
14 immersion chilling and very few on dry air chilling or
15 on evaporative air chilling and even fewer projects
16 that have compared the two methods. Many of the
17 methods, on a frustrating level, do not cite the
18 complete information. So it's difficult to dig out
19 through the literature and make a valid comparison
20 because the conditions, the rates and the times are
21 not always complete in some of these studies.
22 There are a few fairly good comprehensive

1 review articles, and I'm sure there are a few others
2 that I have not listed, but this slide shows a few of
3 those. And if anyone is interested in getting a copy
4 of some of these, I would be happy to provide those to
5 you if you would contact me either after this or
6 through e-mail.
7 So when we look at some of the previous
8 studies, what we find is that in terms of *Salmonella*,
9 most of the literature has focused on looking at
10 prevalence of *Salmonella* -- that is: The number of
11 positive carcasses -- and they have not looked at or
12 reported the exact numbers or the counts.
13 Now, overall, *Salmonella* prevalence was
14 reduced by immersion chilling and by air chilling,
15 although for the air chilling, I would like to mention
16 that was just one experiment that I was able to find
17 that looked at *Salmonella*, because there are just so
18 few data out there.
19 *Campylobacter*. We found that we get up to
20 about a two-log reduction with immersion chilling, and
21 that's when we use a whole-carcass rinse for recovery.
22 We get little change in *Campylobacter* with air

1 chilling, but in that method -- that was a neck-skin
2 maceration method from a processing plant that was in
3 Kuwait.
4 And when we look at generic *E. coli* and
5 coliforms, we get about a one-log reduction without
6 chlorine; when we maintain our chlorine between 20 and
7 25 parts per million, we get between a two- and three-
8 log reduction. That's with immersion chilling. We
9 got no significant reduction with air chilling, but,
10 again, that was the neck-skin maceration recovery
11 method, which tends to recover higher levels than just
12 a whole-carcass rinse.
13 I'd like to spend the next few minutes
14 talking specifically about some research that we've
15 done at the Agricultural Research Services and then
16 end with another study, from the University of
17 Bristol.
18 This was a study that I conducted with
19 Mark Berrang, whom you've already heard from, Andrew
20 Dickens, Nelson Fletcher and Nelson Cox in 2003. And
21 what we were doing was looking at the effects of
22 broiler feed withdrawal and transportation on levels

1 of *Campylobacter*, *Salmonella* and *E. coli* on carcasses
2 before and after immersion chilling.
3 And what we did was -- we went out and
4 found a *Campylobacter*-positive commercial flock; at
5 about 28 days of age, we tested the litter. And when
6 we identified the flock, we then went back at 36 days
7 of age and moved them to a university facility. We
8 inoculated them one week before processing with a
9 marker strain of *Salmonella*, and then we processed
10 them at 42, 49 and 56 days of age.
11 We did a whole-carcass rinse after a
12 manual final wash, and that was our pre-chill counts.
13 And then we did another whole-carcass rinse after
14 chilling, and we maintained our chlorine level in the
15 chillers at 20 parts per million.
16 This is just a picture of our little
17 prototype tumble chillers.
18 And this slide shows the data with the log
19 counts on the Y axis -- that's colony forming units
20 per mil -- of the whole-carcass rinse. And the
21 different categories of bacteria are on the X axis.
22 The white bars that you see in the first column of

1 each bacteria represent the pre-chill counts, and the
2 black bars represent the post-chill counts.
3 And what we found is that with the
4 chilling, we got a 1.2 log reduction in coliforms, we
5 got a 1.3 log reduction in *E. coli*, we got a 1.3 log
6 reduction in *Campylobacter*, and we only got a half-a-
7 log reduction in *Salmonella*.
8 If you'll notice, we started off with a
9 very low level of *Salmonella*. And I think the reason
10 is because we inoculated them one week before we
11 processed and we should have waited and maybe
12 inoculated them about two days before we processed.
13 So there were low levels to begin with even though
14 that was a significant reduction and it was only half
15 a log.
16 This next study is from an individual in
17 our group that you've already heard from, John Cason,
18 and co-workers. And in this study, John wanted to
19 look at the effects of pre-chilled fecal contamination
20 on the numbers of bacteria recovered from broiler
21 carcasses before and after immersion chilling.
22 And what John did was -- John came up with

1 this idea of dividing a carcass in half and using one
2 half of the carcass as a control and the other half as
3 a treatment. And this is an excellent way of doing a
4 study because you statistically have the companion
5 comparison, which is a far more superior statistical
6 comparison than one carcass to another.
7 So he divided the carcasses half in two,
8 and then he identified a 3-by-5-centimeter rectangle
9 section on each half. And on one half of each pair,
10 he put .1 grams of fecal material. Then he waited ten
11 minutes, and then he washed it and chilled it in the
12 same prototype chiller that I just showed you, then
13 did a half-carcass rinse and then also recovered the
14 section of skin that was macerated, that3-by-5-
15 centimeter square of skin.
16 And I don't know if you can see this or
17 not, but there are little dots designating the area on
18 the skin here. And this is an example of a paired
19 half, so he has got the 3-by-5 identified on the
20 control carcass and then the fecal material on the
21 other half.
22 So what John found -- and this is just the

1 *E. coli* data, but he found the same thing for
2 enterobacteriaceae and for coliforms. He did not look
3 at *Salmonella*, unfortunately.
4 But for *E. coli* in the rinses on the
5 control half that did not have the fecal material, he
6 found 5.4 log; on the half that had fecal material, he
7 found 5.5 log, not a difference. The skin halves
8 or -- the skin sections that were macerated -- he got
9 comparable counts on those, as well. And as I
10 mentioned, the same results occur for
11 enterobacteriaceae, which is the category of bacteria
12 that includes *Salmonella*. And also, for coliforms --
13 the same data. So no difference.
14 Another study I want to tell you a little
15 bit about is from Doug Smith, John Cason and Mark
16 Berrang, also with ARS. And in this case, they looked
17 at the effects of fecal contamination in immersion
18 chilling on *E. coli*, coliforms, *Campylobacter* and
19 *Salmonella* counts on broiler carcasses.
20 This is a little bit of a complicated
21 design. And I stole this slide from Doug, so I can't
22 take credit for the handy drawing. But what Doug did

1 was -- he got eight carcasses, and he divided them
2 half in two.
3 If you'll look at the left side of the
4 screen -- he took eight carcasses, divided them half
5 in two. One half went into one chiller which he
6 designated as the clean chiller. That companion half
7 went into another chiller that he designated as the
8 contaminated chiller.
9 Then he took another set of eight
10 carcasses, and he divided those half in two. The one
11 half that did not have fecal material on it went into
12 the clean chiller; he put a tenth of a gram of feces
13 on the companion half and then put that into the
14 contaminated chiller.
15 And this is the results that Doug found
16 from this study. The white bars represent the carcass
17 that had the fecal material on it, the black bars
18 represent the carcass halves that were in the
19 contaminated chiller, and then the green bars
20 represent the halves that were in the clean chiller.
21 And he found no difference in coliforms and *E. coli*
22 among any of the treatments. He did find a slight

1 difference with *Campylobacter*.
2 With *Campylobacter*, the control carcasses
3 were about six-tenths lower in log *Campylobacter* than
4 the direct contamination. They were half a log lower
5 than the cross-contamination. So that's a minor
6 difference, statistically significant, but,
7 biologically, we always question whether or not a half
8 a log is of practical significance.
9 In terms of *Salmonella*, he did not find
10 any detectable levels of *Salmonella* in the control
11 carcasses, and less than one log on either the direct
12 or the cross-contamination.
13 I would like to point out that no chlorine
14 was used in the chiller. And when I started trying to
15 figure out how much of a volume of water he used, it
16 worked out to be about .9 gallons per pound. So we
17 decided to do another study where we wanted to look at
18 volumes of water that were used in the chiller.
19 And in this first study, we wanted to use
20 extremes that were not commercial volumes. So we
21 picked a very low volume of water and then something
22 that was eight times that to see if we were going to

1 find a difference in bacterial counts.
2 So what we did was -- we used John Cason's
3 method again of dividing a carcass half in two. And
4 on one half of each pair, we put in .25 gallons per
5 pound, which is typically about half the level that we
6 would see commercially, and then we put the companion
7 half in eight times that, which would be two gallons
8 per pound. This, again, was non-chlorinated water.
9 And what we did was -- we put each half in
10 an autoclave bag with zip-ties. And then this was
11 submersed into a secondary tank of chilled water that
12 had an air agitation in the bottom. And after 45
13 minutes, we then pulled those halves out. We let them
14 drip for five minutes, and then we did a half-carcass
15 rinse and looked at what we could recover.
16 So again, we've got the log counts on the
17 Y axis and the different bacteria on the X axis. The
18 white bars are the pre-chill counts, the low volume of
19 water is in the black bar, and the high volume of
20 water is in the green bar. And from pre-chill, using
21 a low volume of water, we got a 1.5 log reduction. We
22 gained another half-log reduction -- and that's total

1 aerobic bacteria -- when we increased the volume of
2 water.
3 For *E. coli*, we saw a two-log reduction
4 when we used a low volume of water; this was then
5 increased to 2.8 log with a high volume of water. For
6 enterobacteriaceae, we saw a 1.2 log reduction with a
7 low volume of water and a 2.2 log reduction with a
8 high volume of water.
9 *Campylobacter* had our largest reduction.
10 We saw a 2.7 log reduction with a low volume of water
11 and then a 3 log reduction when we used a high volume
12 of water.
13 Interestingly enough, we also collected
14 the water that was in the chill bags, and we looked at
15 how much bacteria was in that. And we did it on a
16 per-mil basis. And oddly enough, we found that on a
17 per-mil basis, each mil of chill water had the same
18 total aerobic bacteria count, it had the same *E. coli*
19 count, it had the same enterobacteriaceae count and
20 the same *Campylobacter* count.
21 So we are actually going to go back and do
22 some additional volumes that are in between these two,

1 but we've found this to be extremely interesting.
2 Maybe there is a possibility that a certain number of
3 bacteria get in each mil and that's going to be the
4 standard.
5 I wanted to end with a study from the
6 University of Bristol by a fellow named Jeff Mead and
7 his colleagues. And this was conducted in 2000. And
8 he looked at the microbial cross-contamination during
9 air chilling.
10 And what he did was -- he used a marker
11 strain of *E. coli* that was a non-pathogenic strain.
12 And he put this on one carcass, and then he ran it
13 through a commercial system. And the commercial
14 system was set up for an evaporative chill with 50
15 parts-per-million chlorine. Then they turned that
16 system off. And they ran it through and did a dry
17 chill.
18 And he evaluated the contamination in a
19 plus-or-minus ten carcass direction, and he also
20 evaluated -- I'm not going to show that today, but he
21 evaluated it on a companion line to see if it would go
22 in all four directions. And in fact, it did. But

1 what he did was -- he put 10 mls of 10^9 colony forming
2 units per ml, and then he sampled the different
3 carcasses.
4 This slide shows the set of data for the
5 evaporative chill. The zero position on the X axis is
6 the carcass that was inoculated, and he went upstream
7 and downstream. And then you will notice that he
8 found at least on log even ten positions away. So we
9 did get cross-contamination there.
10 The same thing for the dry air chill.
11 Although the counts were lower, he was able to recover
12 bacteria as far away as ten positions.
13 So what we know from this is that
14 immersion chilling causes at least a one log reduction
15 in carcass pathogenic bacteria; post-chill, when we
16 have fecally contaminated carcasses, they are
17 microbiologically equivalent to non-contaminated
18 carcasses.
19 And the potential does exist for cross-
20 contamination during immersion and for air chilling,
21 particularly if antimicrobials missing or not used.
22 And I wanted to mention that because I frequently hear

1 that a lot of folks are saying, "Okay, we're not going
2 to get cross-contamination with air chilling," and I
3 think that's a miscommunication. So we will as long
4 as we are chilling chickens.
5 So thank you for that. And I will show
6 you the place where we all work.
7 (Applause.)
8 DR. ENGELJOHN: Thank you.
9 Our next speaker is Dr. Ken Byrd, a
10 veterinarian with Mionix, who will talk to us about
11 experience with managing pH and its effectiveness in
12 processing water. He comes to us with experience from
13 the field; he has worked with industry and is a former
14 FSIS employee, as well.
15 So welcome.
16 DR. BYRD: Thank you very much. I
17 appreciate the opportunity to be here. Let me begin
18 simply by offering a disclaimer. FSIS does not
19 endorse any particular products.
20 Can you all hear me okay in the back? I
21 was -- can you hear me?
22 (Pause.)

1 DR. BYRD: Okay. I didn't know whether
2 there was an issue with the sound or what. I was
3 walking around during the break back there awhile ago
4 and talking, and I heard one of the sound system folks
5 say that they thought there was a loose screw in one
6 of the speakers. So, you know, I don't know for sure
7 how to take that.
8 The topic of my presentation does have to
9 do with chlorine and some of the factors that are
10 issues to make it work. Many if not most of the
11 poultry slaughter operations use some form of chlorine
12 in the plants, whether it's sodium hypochlorite bleach
13 or whether it's calcium hypochlorite, some of the
14 swimming pool-type tablets, or gas or whatever.
15 To make this work, I need to do just a
16 quick review of the chemistry of chlorine. And I --
17 most of you all probably know all this, but there may
18 be someone here that doesn't understand it all. And
19 so I don't mean to insult anybody's intelligence, but
20 I've kind of got to go back to zero and start over on
21 this.
22 To really accomplish the disinfectant and

1 oxidation from chlorine, you'd have to (have a)
2 chemical reaction between the chlorine and the water
3 to form hypochlorous acid. Now, the hypochlorous acid
4 is the particular substance that does destroy the
5 bacteria, and it usually does this by the process of
6 oxidation or simply pulling electrons out of their
7 cell membranes.
8 Now, the kicker to this is: When you
9 produce the hypochlorous acid, it's a relatively weak
10 and unstable substance, and it doesn't stay in that
11 form very well; it will tend to dissociate into the
12 chlorite ion. Now, what -- again, the hypochlorous
13 acid is what is the antimicrobial. Key point: The
14 chlorite ion is not relatively effective.
15 So why is pH control important in this
16 whole scenario? PH is what drives the equilibrium
17 back and forth between whether the hypochlorous acid
18 stays in its chemically active form or whether it
19 dissociates over into the relatively ineffective
20 antimicrobial.
21 The higher the pH, the more the reaction's
22 driven to the right, the less antimicrobial activity

1 you have, because more of it is driven over to the
2 chlorite. The lower the pH, the more it's driven back
3 to the left to stay in the hypochlorous acid form.
4 If I had only one slide that I could
5 present, this would probably be the slide because it
6 pretty well puts into picture what is happening with
7 the hypochlorous acid. For instance, look at if
8 you've got a pH in your water of, let's say, eight.
9 Only about 27 percent of your free available chlorine
10 is actually going to be in the hypochlorous acid form.
11 You're getting the beneficial effect out of about 28
12 percent of your chlorine.
13 Now, typically what do we do when we're
14 using chlorine and we're not getting the effect that
15 we want? Well, you know, call maintenance and tell
16 Boudreaux to crank up the chlorine pump; let's add in
17 some more chlorine, you know. When I first got in
18 around the meat and poultry industry back in the mid-
19 to latter '70s, you know, that was the thing to do:
20 Let's just add more chlorine.
21 But what happens with this scenario so
22 often -- and I've got a bet that there's somebody here

1 in this room that has probably experienced this -- you
2 don't get the results that you want. You add more
3 chlorine. Then you start getting complaints from some
4 of the workers or some of the inspectors: My eyes are
5 burning; my throat's burning; you know, we've got too
6 much chlorine. Okay. Go tell Boudreaux to turn it
7 back down; you know, we're getting some gassing-off.
8 And so it becomes very, very frustrating.
9 What's the issue? You know, we add more chlorine
10 because we need it; now we can't use it because it's
11 gassing off. The key to it is not to add more
12 chlorine, but simply make what you've got work better.
13 And you do this simply by shifting the pH.
14 For instance, again, look at the pH of 8.
15 You have about 27 percent or so of your chlorine
16 that's actually active. If you drop that pH to 6.5,
17 you increase your hypochlorous acid from 27 percent to
18 about 92 percent. What have you done? Have you added
19 more chlorine or more chemical to that? No. You've
20 simply just adjusted the pH and use what you now have
21 already in the water. That's very, very important.
22 And you need to keep a good handle on

1 this, you know. Used to be we used some of the
2 swimming pool kits to measure pH and whatever; you'd
3 pour some of this in and some of that in and you'd get
4 a color reading, and you'd compare it to a chart or
5 whatever. Today, with the new technology, the
6 handheld pH ORP meters are very effective and very
7 inexpensive. I bought one, oh, back in the summer,
8 and I think it was -- well, it was less than $150 for
9 a handheld ORP pH meter that's quite accurate.
10 Again, the key to effective use of
11 chlorine is to keep the chlorine in the hypochlorous
12 acid form. As Dr. Bailey said awhile ago -- and I
13 loved that comment -- not all water is created equal.
14 Well, that's very true.
15 Because how your acidifier reacts depends
16 on your water quality, you need to take this into
17 consideration. Does it come from a well, where it may
18 have a lot of mineral content? Does it come from the
19 surface? Where does it come from? What's the pH and
20 the alkalinity? What's the hardness of it? This can
21 help give you some ideas.
22 When I first got around the meat and

1 poultry industry, our buzz words were, "How much
2 chlorine have we got in there? Well, we've got, you
3 know, 20 parts per million," or whatever. And we
4 thought, "Well, okay, that's good." But this simply
5 measures the total amount of chlorine, which is the
6 bound chlorine, which can't react any more, and your
7 free available chlorine.
8 A little bit later on, we got a little bit
9 more sophisticated, so we started talking about, What
10 is your free available chlorine? And we thought,
11 Okay, now this is the cat's meow. You know, we --
12 this is the buzz word. But there's still an issue
13 with that, because that measures not only the
14 hypochlorous acid, but the hypochlorite ion. So now
15 you know how much free available chlorine you've got,
16 but you don't know whether it's active or not, because
17 if your pH is 8.5 or so, you've probably got, you
18 know, 10 to 15 percent that is actually active.
19 So the best way to actually monitor or to
20 measure the sanitizing effect that's in your water is
21 through oxidation reduction potential. It's not a
22 direct measurement of the hypochlorous acid, but it is

1 rather an indicator of the hypochlorous acid.
2 ORP is a conductive measurement; it's
3 measured in millivolts. To kill free-floating
4 pathogens in water, an ORP of 650 to 700 is usually
5 recommended. You know, we try to hit 700 or a little
6 bit more.
7 It's a range, not a fixed number. And
8 just as a side note, it has been reported that it's
9 not really a practical method for monitoring the
10 antimicrobial potential of water treated with hydrogen
11 peroxide or peroxyacetid acid. And I'm not chemist
12 enough to tell you why.
13 Also, there's -- it has been reported that
14 there's some caution against using citric acid as an
15 acidifier, because it -- some evidence indicates that
16 it may in some way interfere with the lethal action of
17 the HOCl.
18 Okay. I mashed the wrong button.
19 (Pause.)
20 DR. BYRD: Okay. An inorganic food grade
21 acid is what is recommended.
22 This is just an example of some of the

1 pathogens and the kill time with the different ORP
2 readings. As -- for instance, if you'll -- look at
3 *Salmonella*, for instance. At an ORP of -- I'm going
4 to do like Dr. Cason; I'm going to point on my screen
5 here, and you all follow me along.
6 For *Salmonella*, at an ORP of about 485,
7 you can see it takes over 300 seconds for it to be
8 killed. But if you increase your ORP over to 665, you
9 get killing in less than 20 seconds.
10 Now, the acidifiers that are used to
11 acidify the water come in different forms. There's
12 benefits and there's disadvantages to all of them.
13 Dry powders? One of the advantages is
14 it's less freight. Any time that you're shipping a
15 dry powder, well, it's probably more cost efficient,
16 but there are some draw-backs, you know. The dry
17 powders must be mixed. Boudreaux may come in on
18 Monday morning hung over pretty bad, and, you know, he
19 may not be real good at mixing today.
20 There are -- any time you've got to mix
21 something, there's increased labor, and there's higher
22 risk of mixing errors and potentially inconsistent

1 results. There may be some hazards to the workers.
2 And also, if you've got some undissolved solids, these
3 things can plug hoses and nozzles and that type of
4 thing.
5 There's also gas acidifiers such as CO^2.
6 Some of the pros? No mixing is required. They're
7 relatively inexpensive.
8 I am told that some of the things to be
9 aware of in using it is the safety factor. You need
10 to monitor the CO^2 in the air. It's not real easy to
11 automate. And you need some sort of device or system
12 in place so if you lose -- this is a free-flowing
13 system, I'm told. And if you lose electrical power,
14 this system continues to emit the gas. So you've got
15 to have something there to shut it off.
16 Also, there are ready-to-use liquids.
17 With the liquids, no mixing's required. So you don't
18 have the extra labor or the potential of mixing errors
19 and those types of things.
20 Now, any time you start to ship a liquid,
21 you know, it's probably going to cost a little extra
22 freight. One of the ways to address that is just to

1 use bulk shipments. A very, very important point,
2 though: Whatever you're doing, automate your system.
3 Automate your system.
4 When we first got started in acidifying
5 chillers, we were doing it manually. And that was an
6 extremely time-consuming thing, and there were spikes
7 up and down, and whatever. We weren't getting real
8 good, consistent results, and we quickly realized that
9 this had to be automated.
10 And this is just one of our systems
11 sitting on the side of a chiller, as you can see.
12 Again, I'll point on my screen, and you all follow.
13 But if you can make it out on the slide
14 there -- there's a couple of little probes that you
15 see sitting in a little box. One measures pH, and the
16 other monitors ORP. These hook into a PLC which in
17 turn goes to a couple of pumps to increase or decrease
18 the acidifier, as well as the bleach, for chlorine
19 source.
20 Where would you use pH-adjusted
21 chlorinated water? Well, I think some of the speakers
22 that have already been here today have pretty well

1 covered that. Basically, anywhere that you're going
2 to put chlorine in the water, why not use it and use
3 it efficiently? Also, as Dr. Hulsey mentioned, just
4 the scalder -- just the pH in the scalder showed
5 some -- the pH reduction in the scalder showed some
6 very encouraging results.
7 Avoid misuse. As with anything else, use
8 as directed. Isn't that a catchy thing? You know, I
9 have to remind myself of that every once in awhile.
10 Use as directed. Use as written in the food safety
11 documents. You need to educate the user and document
12 the training.
13 Some of the material I've presented today
14 has come from a publication by Dr. Trevor Suslow at
15 the University of California. This is a real good
16 reference on addressing ORP, how it works and how to
17 measure it. And if you don't get this information
18 written down here and you want a copy, well, holler at
19 me, and I'll get this address for you.
20 So in conclusion, let me just simply say
21 this. Chlorine is effective. It's readily available,
22 it's relatively inexpensive, and it's very effective

1 when properly managed. And, "When properly managed,"
2 is the key.
3 That's all I have. Thank you.
4 (Applause.)
5 DR. ENGELJOHN: And here we are at our
6 last presentation for the day. Dr. Scott Russell will
7 come back and give us some input on further
8 processing.
9 Dr. Russell?
10 DR. RUSSELL: Thank you, Dr. Engeljohn.
11 (Pause.)
12 DR. RUSSELL: Thank you. Stan says since
13 we had to give two talks, we get double the pay. So
14 I'm looking forward to that.
15 (Laughter.)
16 DR. RUSSELL: All right. These are some
17 of the topics that I'd like to cover. And again, I'd
18 like to say, like Julie did, thank you for staying so
19 late for the presentation. It's good to have a nice
20 audience at the end of the day.
21 But some of the things we want to cover in
22 this presentation are the regulations involved and how

1 the problem occurs in terms of post-process
2 contamination on fully cooked products and some
3 technologies used to apply sanitizers in these areas.
4 And we're going to look at clean rooms,
5 employee hygiene, drain treatments that have been --
6 that are very novel, biofilm abatement procedures and
7 innovative surface materials and how some companies
8 use in-process sanitation and novel packaging
9 materials, as well. And then we'll talk a little bit
10 about microbial testing.
11 As most of you know, the USDA has stated
12 that official establishments that produce ready-to-eat
13 meat products must prevent adulteration by pathogenic
14 environmental *Listeria monocytogenes*. And the new
15 directive also requires that the plants conduct
16 verification procedures to make sure that the organism
17 is being removed from these foods.
18 And there's a similar regulation with
19 regard to *Salmonella*, as well, on fully cooked
20 products, and it goes sort of like this under
21 '381.150. I'll just read the part on lethality: "A
22 7-log reduction of *Salmonella* or an alternative

1 lethality that achieves an equivalent probability that
2 no viable *Salmonella* organisms remain in the finished
3 product, as well as the reduction of other pathogens
4 and their toxins...is necessary to prevent
5 adulteration."
6 So the same sort of regulation there on
7 both products. Now, how does this occur?
8 And the basic problem, again, very similar
9 as to what I mentioned previously, is that these
10 organisms, either *Listeria* or, in some cases,
11 *Salmonella*, can get on the equipment surfaces --
12 particularly *Listeria*, because it lives well in the
13 drains in the cooling areas. It can get on fans, and
14 it can get on cooling units and on employees. And it
15 can incidentally go into the air as an aerosol that
16 can be blown by these fans in high areas where there's
17 a lot of rapid air movement, and it can get on the
18 equipment surfaces.
19 One of the new technologies that has been
20 developed -- and I say it's new; it has been around
21 for probably 20 or 30 years but not really been
22 applied to the poultry industry this way. And in many

1 of the food industries, it's just now starting to get
2 real notice, and a lot of people are starting to see
3 how this can work.
4 Essentially, this is the electrostatic
5 spraying nozzle. And there's a high-pressure air
6 stream pumped through the middle of the nozzle. The
7 sanitizer is introduced into the air stream, where
8 it's vortexed. And it goes through a round, very
9 small aperture, and it's sheared off into about 30
10 micron particles.
11 After that, it goes through this silver
12 ring that you see here on the diagram, and it's
13 charged. And it works a little bit different than the
14 normal electrostatic sort of painting-type procedures
15 that you see where you charge the metal and charge the
16 spray a different charge and they coat beautifully.
17 This technology works on the basis that you're
18 charging the spray and, as it approaches an object,
19 the object takes on the opposite charge of the spray.
20 It's kind of an unusual thing, but here it
21 is in action. And you can see this is pesticide being
22 sprayed onto a leaf of a plant. And you can see that

1 the spray is defying gravity, coating the underside of
2 the leaf and the back side of the stem.
3 To see this a little bit more clearly, we
4 have a demonstration sort of electrostatic sprayer.
5 Pretend that this is a chicken carcass or an egg or a
6 piece of surface or equipment or food contact surface.
7 We spray the object with a powdery
8 substance to mimic sort of what a sanitizer would look
9 like for, let's say, eight seconds. This is what it
10 looks like. It's sort of like if you sprayed the ball
11 with a can of spray paint; essentially, only half the
12 ball is covered.
13 Now, we can actually spray the same ball
14 with, for only two seconds, much less material using
15 the electrostatic sprayer with the charge on. And you
16 see a much better coverage. Much better coverage.
17 We've seen coatings in these areas in restaurants,
18 food processing plants and further processing plants,
19 where we can use 1/80th the amount of material.
20 1/80th.
21 Now, you might ask, Well, why in the world
22 isn't everybody using this technology? Well, if you

1 were a big company that produced chemicals and I came
2 to you and said, "I can cut the amount of chemical
3 that these companies have to use by 1/80th," what do
4 you think you're going to do? Well, that's the look I
5 get when I go talk to them about these kinds of
6 things.
7 So you can see the difference here. We
8 were in a room, with 400,000 eggs, about the size of
9 this room. And we used one gallon of material to
10 sanitize the eggs and the floors and the walls. It's
11 pretty dramatic, the kind of results that you can get.
12 Now let's talk a little bit about clean
13 rooms. This is a strategy that has been employed very
14 well at a lot of further-processing plants. And what
15 occurs here is that the raw area is truly separated
16 from the cooked area. And when I say, "Truly
17 separated," essentially, where the oven is is a very
18 large wall structure.
19 And I don't have it on the diagram, but
20 just imagine a wall between the oven and the IQF
21 freezer. And the idea is that those chicken pieces or
22 parts prior to cooking will have pathogenic bacteria

1 on them, whether it's *Salmonella*, *Listeria*, or
2 whatever, and as it goes through into that IQF
3 freezer, it's a new world. Okay?
4 That IQF freezer is evaluated on a regular
5 basis for biofilm formation. Extra effort is required
6 to remove the biofilms in that freezer. Special anti-
7 biofilm agents are used in those freezers. So it's
8 tested on a regular basis microbiologically to make
9 sure that that freezer is free of pathogens.
10 So when it goes through that oven, it
11 enters into a whole new space now, and it's in a clean
12 room, essentially. The air that goes into that
13 freezer and into the room after the freezer is handled
14 differently.
15 After it comes out of the IQF, or
16 Individually Quick-Frozen, freezer, it goes into the
17 true clean room. And in this room which is very
18 separate -- it's almost like going into a
19 laboratory -- there's no water on the floor. And as
20 most of you know, in these plants, there's water all
21 over the place in food and poultry processing plants,
22 but not in this room. These floors are kept very dry.

1 The employees from the raw areas are not
2 allowed into those areas. The employees that come in
3 there have to have clean, sanitized boots, smocks,
4 clothes and so forth.
5 And they sanitize these rooms every two
6 hours in some cases. They'll shut the process down,
7 they'll remove all the food from the area, and they'll
8 spray the walls, floors and ceilings with very high
9 concentrations of quaternary ammonium or some other
10 kind of chemical. And then they'll go in. And if
11 it's too high to be legally used, they'll rinse it
12 off. Okay?
13 Let's say they used 400 parts per million.
14 They have to go in there and rinse all that stuff
15 off. Then they begin processing again for two hours.
16 The air from the outside is filtered using
17 hepafilters before it comes in there. It's evaluated
18 on a regular basis for biofilm formation.
19 Again, extra efforts are made to control
20 biofilms, and novel chemical systems are used in these
21 rooms, but there it's almost like a sterile room.
22 It's not sterile, but it is controlled very, very

1 carefully as a means of doing this. And some
2 companies have had very, very good results in terms of
3 *Salmonella* and *Listeria* control using these kinds of
4 rooms.
5 Another area that's of concern is employee
6 hygiene. They want mandatory handwash and sanitizing
7 stations. The hand dips need to be changed on a very
8 frequent basis. I've seen plants were the restrooms
9 have access to the plant, and that's not a good idea.
10 People just walk right out of the restroom right onto
11 the plant floor, not a good idea.
12 A lot of plants you'll see, you go in, and
13 the people that work in the plant also have cows on
14 their farms. And they're out there managing the cows,
15 and then they'll walk right into the plant with the
16 same boots, same jeans and so forth. And they're
17 wearing very short smocks. That's not a good idea.
18 I like to tell people to examine employees
19 daily for illness. I used to walk through the plant
20 when I managed the plant and -- further processing
21 plant and make sure that they're not visibly sick or
22 carriers.

1 It's a good idea to question foreign
2 employees that are coming back from other countries to
3 see whether or not they may be -- may have become
4 sick. I had to serve on a court case one time where
5 86 people went to the hospital and one lady died
6 because of that. So it is important to monitor those
7 kinds of things.

8 And, also, it's important to make sure
9 your employees are familiar with American hygiene
10 customs in the restroom. We have a problem with some
11 folks from some countries that the toilets don't
12 flush. And they'll use the toilet paper and stack it
13 up next to the toilet. And that's, of course, not
14 very hygienic.

15 So let's change subjects a little bit. A
16 new drain treatment has just become -- has just been
17 developed by Dr. Mike Doyle, who's a professor at the
18 University of Georgia down at the Griffin Labs. And
19 what they did was -- they selected two commonly used
20 competitive exclusion bacteria, the two species in
21 particular, *Lactoccus lactis* subspecies *lactis* and
22 *Enteroccus durans*, and they treated floor drains in a

1 poultry processing plant with these bacterial
2 cultures.
3 The results over a five-week period showed
4 a several log reduction in *Listeria* at temperatures of
5 4 to 37 degrees centigrade. And Ecolab now is working
6 with UGA to license the technology. And of course,
7 requests have been made from many major meat and
8 poultry processors to use this to reduce those levels
9 of *Listeria* in the drains, and, thereby, the
10 incidental spraying of the hose into the drains and
11 the incidental aerosolization of *Listeria* and
12 *Salmonella* from those areas won't get on the
13 equipment.
14 So that holds some promise there. It's a
15 new treatment.
16 As I mentioned in the previous talk, the
17 big problem with biofilm is that you have to break up
18 the matrix. And Lysozyme has been effectively used in
19 this area.
20 We had some discussions after the last
21 talk, and some of my colleagues mentioned that it may
22 have difficulty with -- Lysozyme may not work so well

1 on gram-negative bacteria such as *Salmonella*, whereas
2 it may work better on some of the gram-positive
3 bacteria. So that's a concern that I did not notice
4 in the literature, but I appreciate that being brought
5 to my attention.
6 And again, the same sort of thing here:
7 Mechanical methods have been shown to be very good.
8 High-pressure sprays have been shown to be very good.
9 But again, this requires a lot of labor. And it
10 would be great if we could come up with better
11 technologies, better chemistry and better cleaning
12 methodologies for breaking down these biofilms, as
13 opposed to having to use labor and hand-scrub these
14 things off of the equipment.
15 Again -- I showed you this. Most of you
16 were here earlier. And this was a very successful
17 trial in the sense that we formed these *Listeria*
18 biofilms on stainless steel coupons and we were able
19 to get dramatic reductions on those biofilms over
20 many, many reps. So that was very nice.
21 Now, as I mentioned before, this idea to
22 me was fairly relatively new: That a company would

1 shut down every two hours -- literally discontinue
2 their processing, shut that whole plant down, have
3 everybody removed from the processing floor, and go
4 through a process of disinfection of all the
5 equipment.
6 Imagine the labor involved in removing all
7 of their product from the lines because, you know,
8 it's suspect of being, well, contaminated with regard
9 to the chemical.
10 And so they have to remove all of the
11 stuff. The equipment, the wall and the floor is
12 thoroughly rinsed or foamed with 400-parts-per-million
13 quaternary ammonium, for example, and then they have
14 to rinse everything down, bring everybody back and
15 then begin production again. So that is a big bit of
16 labor there, but it has been used to some good effect.
17 Now, there have been some innovative
18 surface materials developed recently. One is called
19 AlphaSan, also called Silveron. It's a product by
20 Milliken and Westlake, a combination of the two
21 companies. And what they do is incorporate this into
22 plastic cutting boards.

1 And we did some studies on this, and we
2 could see -- in a very short period of time, we could
3 see in only one hour a reduction in bacterial numbers.
4 And at 16 hours, we saw a tremendous increase in the
5 efficacy of reducing these bacteria on coupons.
6 Another product that has been studied
7 heavily by Dr. Sheldon, if you're interested in that,
8 is called HabaGUARD. And he did the work on
9 *Salmonella*, *Listeria*, *Campylobacter* and *E. coli* 0157.
10 And they were all inhibited anywhere from 3.6 to 7.7
11 logs.
12 But the idea here is that these products
13 are incorporated into plastic cutting boards, into
14 surfaces that are used for processing. And the idea
15 is if they can control bacterial growth on those
16 surfaces, then they may be able to control some of the
17 biofilm formation, as well.
18 So these are some new products out there
19 that you all should be looking for.
20 Now, another group of scientists have
21 worked with novel edible films. The idea here is that
22 we're using -- on fully cooked or fully processed

1 products -- for example, a chicken patty or a whole
2 chicken carcass -- can we take some material and spray
3 or apply it to that carcass, let's say -- let's just
4 use a whole carcass as an example.
5 Some of these products that can be sprayed
6 on the outside of those carcasses would include lipids
7 and oils, waxes and emulsions, resins like shellac and
8 rosin -- and I don't know about you, but I don't think
9 I'd like to eat shellac, to be honest with you, but --
10 carbohydrates like Celluloses, pectins, chitin,
11 starches, gums, and then proteins. There are
12 proteinaceous ones.
13 And someone once asked me the last time I
14 spoke about this, you know, How about allergies? And
15 of course, some of us are allergic to soy or peanuts
16 or whatever. So we'd have to watch that.
17 But these things are being produced --
18 these films, these edible films -- and you spray these
19 on the outside of a carcass. The idea here is you can
20 add antimicrobial substances to these films, spray
21 them to the surface of a carcass, and it will prevent
22 the growth of pathogenic bacteria. And it will kill

1 anything that might have incidentally gotten on there.
2 Some of the things that antimicrobials
3 would be used for in a coating like that would be
4 organic acids like acetic, benzoic, lactic,
5 proprionic, and so forth, fatty acids, bacteriocins --
6 that's another one. Bacteriocins would be by far the
7 broadest group there.
8 And then there are novel packaging films.
9 And they're really -- these are interesting because
10 these are the films that are used on these packages,
11 and you can incorporate bacteriocins in particular,
12 like nisin, into these things to help control
13 pathogenic bacteria.
14 Now, there are two ways to go about this.
15 You can incorporate the bacteriocins directly into
16 the plastic itself or you can -- and I have lots of
17 data here that shows that we had good reductions, but
18 we're running out of time. You can also coat the
19 bacteriocins on the surface of these bacterial films
20 and get excellent results, as well.
21 Another thing that's so important to
22 remember is microbial testing. When you have these

1 types of further processing environments, it's
2 important to do rapid microbial and accurate microbial
3 testing. Some of the companies here in Georgia do up
4 to 80 rapid tests per day on all of these organisms:
5 *Salmonella*, *Listeria*, Staph, *E. coli*, APC.
6 And it's really important that they are
7 able to test the products and release them based on
8 negative results because in the last few years, we've
9 had anywhere from 100 to $190 million worth of recalls
10 per year because of these bacteria, *Listeria*,
11 *Salmonella* and so forth -- being found on fully
12 processed products. So rapid testing is extremely
13 important. Use of rapid methods is going to help
14 this.
15 So overall, companies that meet the
16 USDA -- they have to meet the USDA FSIS regulations
17 regarding *Salmonella* and *Listeria* on fully cooked
18 foods. Electrostatic spraying is an excellent way or
19 an excellent means of applying sanitizers. Clean
20 rooms generally reduce the risk of the incidental
21 contact with environmental *Listeria* and/or *Salmonella*.
22 And employee hygiene is, of course,

1 essential to preventing cross-contamination. Novel
2 drain treatments are being developed. Novel biofilm
3 abatement treatments are being developed, and
4 innovative surface materials may have some hope in the
5 future for helping us to control the growth of these
6 biofilms.
7 In-process sanitation's being used to good
8 effect. Novel packaging materials now exist, but a
9 lot of companies are slow to uptake these types of
10 things because of the cost involved. Those
11 bacteriocins are expensive. And rapid microbiological
12 testing is essential for data-based release programs
13 to prevent recalls in the future.
14 Thank you all for your attention.
15 (Applause.)
16 DR. ENGELJOHN: Well, thank you very much,
17 Scott, for that. And I think, you know, even though
18 some of that information was related to ready-to-eat
19 products, there's no reason why we can't be looking at
20 what we can do in the raw processing areas to see what
21 can in fact be done more there.
22 With regard to where we're at now, we're

1 five minutes ahead of schedule. And we're ready for
2 some questions and answers from the panelists who
3 talked this afternoon.
4 And I also just want to remind you all or
5 at least -- you don't know this yet, but, tomorrow,
6 we're not meeting in this room. We're meeting
7 upstairs in the amphitheater. So I think it's on the
8 second floor, but, in any case, it's right above here,
9 tomorrow, 8:30.
10 So do we have any questions from the
11 audience for any of the panelists?
12 MR. COUGHLIN: Michael Coughlin from
13 Johnson Diversity. A couple of questions, if I could.
14 One is just from a position of ignorance.
15 I'm not familiar with air chilling, so this is
16 directed to Dr. Northcutt.
17 I'm wondering how it is that you can
18 actually get cross-contamination in an air chill,
19 especially if there's no misting. And of course,
20 there's no mystery how that happens in an immersion
21 chiller, but I don't understand how it happens in air.
22 And the other question is directed to Dr.

1 Byrd.

2 If you're trying to -- you made the point
3 at the beginning of your talk that pH is extremely
4 important in controlling chlorine. That being the
5 case, if you're using two electrical probes, one being
6 an ORP probe and the other -- I imagine it would be
7 ORP and chlorine -- why would you not have just simply
8 a pH probe and a chlorine probe? Basically, why do
9 you need an ORP probe?

10 DR. BYRD: Because the ORP actually gives
11 you a better indication of the killing power of what's
12 in the water, the better indication of the sanitation,
13 because, again, it's a conductance type of
14 measurement.

15 And so you -- what we do -- we measure the
16 pH which or -- monitor the pH, which is fed into a
17 PLC, which then in turn drives an acidifier pump. But
18 also, then by using the ORP probe to monitor the ORP,
19 this goes into the PLC, which in turn drives the
20 chlorine pump.

21 One of the things that we had to figure
22 out on this was the electrical setup, because we were

1 using the 420-millivolt wires. And to make the ORP
2 pump sensitive to what we needed done, we had to
3 reverse that so that it wasn't a 420, but it was a 24,
4 because when your ORP gets lower, then that's when you
5 need more of the ingredient. And so we had some
6 electrical engineering there to do initially.
7 DR. NORTHCUTT: To back up and answer your
8 air chill question, thank you for asking that because,
9 if you need to ask that, then I'm sure there's other
10 folks that needed to hear that, as well.
11 All of the large manufacturers of
12 equipment are now making systems for chilling chickens
13 without immersion, and basically in a cold room. And
14 they may or may not incorporate different phases of
15 that room where they have what they might call a
16 stabilization phase with different temperatures that
17 range in the neighborhood of about 33 to about 37
18 degrees.
19 And in many cases, the air is going to be
20 blown either across the carcass or in the body cavity.
21 And blowing the air in the body cavity is -- gives
22 you a much better removal of the heat. And this is --

1 may or may not be incorporated with a series of
2 misters that will mist the carcass and maintain the
3 humidity at a high level.
4 And so when water is available, then that
5 provides an opportunity for bacteria to survive. And
6 in blowing the air, we have an opportunity to spread
7 bacteria.
8 And so I don't know if that is in some way
9 answering your question, but I will also ask if my
10 colleague down here with Meyn would like to add
11 anything, because I know that they have a system, as
12 well.
13 MR. COUGHLIN: Okay. Are you saying that
14 you're atomizing particulates, basically, the
15 bacteria?
16 DR. NORTHCUTT: Yes. And --
17 MR. COUGHLIN: Just the air currents are
18 enough to drive liquid films of bacteria off the
19 carcass?
20 DR. NORTHCUTT: Yes. Plus you also have
21 the potential for the bacteria to become resident in
22 the room if it is not properly cleaned. And

1 obviously, that could provide some cross-
2 contamination.
3 MR. COUGHLIN: So the movement of air must
4 be actually quite violent to strip off the film of
5 water and aerosolize it and transfer it through the
6 room.
7 DR. NORTHCUTT: And it depends on the
8 system. But in the literature that I've read, it will
9 range anywhere from like a half a meter per second up
10 to three meters per second. And it depends on the
11 system, and it depends on the location in the room.
12 MR. COUGHLIN: Okay. Thank you.
13 DR. ENGELJOHN: Any further comments?
14 (Pause.)
15 DR. ENGELJOHN: All right. Any other
16 questions?
17 MS. NESTOR: I'm Felicia Nestor with Food
18 and Water Watch. And I just wanted to say to all of
19 the panelists that as a consumer representative, I
20 don't know very much about the poultry industry at all
21 or how any of this works. And I just found all the
22 presentations really fascinating, and I really feel

1 like I learned a lot today. So thank you all very
2 much. And now I'm going to show my ignorance by
3 asking these questions.
4 I guess the first one is -- you know, as
5 consumers, we're told to cook the chicken and cook the
6 turkey to 160 degrees inside the meat. So that
7 suggests to me that the contamination is inside the
8 meat and we're not worried about what's on the
9 surface.
10 DR. BAILEY: No.
11 MS. NESTOR: No? Where am I getting it
12 wrong? Why can't we cook it just like a steak, you
13 know, just really fry the outside? And then --
14 (Laughter.)
15 DR. BAILEY: It's a safety factor. It's
16 suggesting that it's not out of the realm of
17 possibility with the processing that it conceivably
18 could on a rare occasion have something get down in
19 the muscle. We -- intact muscle would rarely have any
20 bacteria in it.
21 If you wanted to cook your chicken to
22 where you didn't get an internal temperature of 160

1 but you got a surface temperature of 160, the
2 overwhelming majority of the time you would be okay.
3 But as a safety factor put in, if you know that every
4 point in your chicken is cooked to 160, then you know
5 that you have eliminated the possibility of survival
6 of vegetative cells of many of these pathogens we're
7 dealing with. And besides, chicken tastes pretty good
8 when it's cooked to 160.
9 (Laughter.)
10 MS. NESTOR: Okay. Thank you. I have a
11 couple more.
12 Dr. Northcutt, in -- the studies went by
13 so quickly. In the one where the chicken gets split
14 and one half gets put in the contaminated tank, was
15 that also the single contaminated tank? Is -- it
16 looked like what you were using there was just a small
17 tank. You're not talking about what's commercially
18 used, you know, a big immersion chiller with thousands
19 of --
20 DR. NORTHCUTT: Yes. It was the small
21 prototype. And that was the study that Dr. Smith
22 led -- who is sitting across the room. But yes. It

1 was our -- we have two of those single prototype
2 tumble chillers that allow us to do things that we
3 couldn't do in a commercial setting with inoculated
4 strains and bacteria. And so yes, it was that small.
5 It holds about 40 gallons.
6 MS. NESTOR: And how contaminated was the
7 water?
8 DR. NORTHCUTT: In Doug's study? I'm
9 not -- I don't know.
10 Did you look at the water?
11 DR. SMITH: [inaudible due to failure of
12 in-house PA system].
13 DR. NORTHCUTT: Okay. Do you want to
14 share that?
15 DR. SMITH: [inaudible due to failure of
16 in-house PA system].
17 DR. NORTHCUTT: It was not contaminated --
18 it was contaminated?
19 DR. SMITH: [inaudible due to failure of
20 in-house PA system].
21 DR. NORTHCUTT: I do have a copy of the
22 manuscript that I will actually give you if you would

1 like it.

2 MS. NESTOR: Okay. I'm just wondering how

3 it compares to some of those immersion -- the pictures

4 of immersion chillings that we saw, which -- some of

5 them looked pretty -- you know, like the color of --

6 DR. SMITH: No chlorine.

7 MS. NESTOR: I know there was no chlorine.

8 DR. NORTHCUTT: Yes.

9 MS. NESTOR: Right.

10 (Pause.)

11 MS. NESTOR: Oh. We didn't see any

12 pictures of immersion chillers today? I thought there

13 was at least one.

14 DR. NORTHCUTT: Yes. I showed one at the

15 very beginning of -- a commercial immersion chiller.

16 MS. NESTOR: Yes. Right. Okay. I've got

17 one more question.

18 DR. NORTHCUTT: I'm looking for the

19 numbers.

20 (Pause.)

21 MS. NESTOR: Should I wait, or should I

22 ask my one more question?

1 DR. NORTHCUTT: Sure. Go ahead. And I'll
2 keep looking.
3 MS. NESTOR: Okay.
4 Dr. Bailey, you said, if I've got it
5 correctly, that when you've got these chemical rinses,
6 you have to be careful when you take the sample that
7 you deactivate the chemical that's on it so that it
8 doesn't continue to destroy the pathogen in the
9 sample. Does FSIS do that in its *Salmonella* sampling
10 in any way? Is there any control for that?
11 DR. BAILEY: Yes, they do.
12 MS. NESTOR: And --
13 DR. BAILEY: I was pretty sure they did,
14 but I just checked with Dan. And he said they do.
15 MS. NESTOR: And how do they do that?
16 DR. BAILEY: It depends on the chemical
17 you're using in the plant. If it's chlorine, you'd
18 use something like sodium lauryl sulfate. And
19 different chemicals have different things that
20 inactive them. You just have to know what's being
21 used.
22 MS. NESTOR: Oh. So it's something that's

1 actually in the chemical?
2 DR. BAILEY: In the rinse material that
3 you use.
4 MS. NESTOR: Okay. Thank you.
5 DR. ENGELJOHN: If I could? This is
6 Engeljohn with FSIS.
7 Just to follow up on your question,
8 Felicia, we have over time looked at the issue of
9 whether or not the samples are -- pathogens are
10 actually dying in the rinse samples that are being
11 sent to the lab. And so that is something that we
12 have studied. And so we -- in working with ARS, we
13 actually have the answers to those questions. So --
14 but we do have buffers and so forth that we use that
15 we ensure don't cause a difference in the pathogen
16 load in the samples we send to the labs.
17 DR. NORTHCUTT: Just to finish answering
18 your question -- and I will be happy to give you a
19 copy of this if you would like it. What Doug found in
20 the contaminated chiller is -- for coliforms, he found
21 log 3.2, which is pretty close to what he was
22 recovering from the carcass. And similarly, for

1 *Campylobacter*, he found -- excuse me. For *E. coli*, he
2 found log 2.7; for *Campylobacter*, log 2.9, and; for
3 *Salmonella*, a 1.5 log -- in the contaminated chiller.
4 MS. NESTOR: Thank you.
5 DR. NORTHCUTT: And you're welcome to
6 this.
7 MR. SANCHEZ: Marcos Sanchez from Texas
8 A&M.
9 Actually, we published a couple of studies
10 on air chilling without a mist. And one of the
11 observations there was the importance of the pre-
12 harvest interventions there, because you don't have
13 this coming-out tank where everything gets mixed up
14 and probably at the same level. So if you have a
15 loaded carcass, it may get loaded until the end of the
16 process, because they're individually hanging.
17 So here brings the concern about the
18 enumeration of the pathogens like *Salmonella*. That is
19 rarely done, because the MPN process is very time
20 consuming. So I'm just wondering. With the new
21 baselines that are planned on it, what is the opinion
22 of the panel on becoming -- the importance of these

1 carcasses. If they have the levels -- if they were in
2 the tank, they were probably similar. But if they
3 were individually hanged, I mean we're going to have
4 some variability there.
5 DR. BAILEY: I actually was not aware
6 until Dr. Engeljohn told us this morning that they
7 were planning to do MPNs and enumerate the *Salmonella*
8 levels. I knew they were going to enumerate the
9 *Campy.*
10 I'm not exactly sure of the point you were
11 getting at, except that you probably may see more --
12 there's very -- there's only a couple of air chillers
13 in the country. So that won't be -- there won't be a
14 whole lot of that. There may be a little bit more
15 variability in air chilling because you don't have the
16 washing effect of an immersion chiller.
17 Immersion chillers historically, a long
18 time ago, when we first started working in the area,
19 were known to potentially cause a fair amount of
20 cross-contamination. But a properly managed chill
21 tank where you control the pH and you use your
22 chlorine or other disinfectant properly is actually a

1 significant processing aid to reduce the levels of --
2 whether it's *Salmonella*, *Campylobacter* or any of
3 your -- just total bacteria on the carcass.
4 So it all goes back to proper -- if you're
5 talking about the chill tank, then you will generally
6 have, as you've seen several slides suggest today, up
7 to 2 logs or more lower coming out of the chill tank
8 than you did going in. And -- but that's pretty much
9 dependent on the quality of the job you're doing
10 managing your chiller. And that would specifically be
11 referring to pH control more than anything else, but
12 it's some other issues, too.
13 DR. ENGELJOHN: And I would just add from
14 an FSIS perspective on that that that is also
15 dependent on the quality of the birds and the sanitary
16 dressing that go into that chiller. So from our
17 perspective, we don't want to see situations where the
18 chiller is used to clean up the birds. So the case is
19 that we need to pay attention on that slaughter
20 dressing.
21 MR. SANCHEZ: Okay. Thank you.
22 MR. McNAUGHTON: James McNaughton with

1 Solution-BioSciences. We heard several times today
2 about neutralization of the chemicals in the rinse
3 solutions.
4 Stan, you mentioned it numerous times.
5 We're now working with a lot of acids and
6 some caustic compounds. Are we also in our research
7 neutralizing those products in the rinse solution
8 itself?
9 DR. BAILEY: Anybody who knows anything
10 about what they're doing in research is. And that's
11 as reviewers for -- pretty much all of us sit on
12 review boards for all the journals that we all publish
13 in. I know that's the very first thing that -- any
14 time we get a paper that has a chemical in it, we make
15 sure that proper neutralization takes place.
16 You know, in the early years, when some of
17 the first things started coming out, there were a few
18 people who weren't paying attention, but I think most
19 of the people who do the research now do.
20 MR. McNAUGHTON: Not particularly the
21 chemical itself, but is pH being neutralized?
22 DR. BAILEY: Yes. I mean certainly, in

1 any research study anybody on this panel I think I
2 know I can speak for has ever done. The very first
3 thing we do before we ever start running our real
4 experiments is run prototype experiments. And we
5 monitor the quality and the condition of the rinse
6 fluid, whether -- whatever the pH is. That's
7 certainly one of the very first things we do.
8 You'll always take that rinse fluid that's
9 coming off without any kind of study itself, and then
10 you drop some of your test organisms in it to see if
11 they survive or if they die. I mean it's just a given
12 that that's the first thing you're going to do.
13 MR. McNAUGHTON: Does USDA in their rinse
14 solutions adjust pH in their 51-day window?
15 DR. ENGELJOHN: I don't have an answer for
16 you, but we'll have one before you leave tomorrow.
17 DR. ALTEKRUSE: The rinse is a buffered
18 peptone water rinse. So --
19 (Pause.)
20 DR. ALTEKRUSE: Oh. I thought this would
21 pick it up.
22 Yes. The rinses that we use are buffered.

1 So it does tend to adjust for that, yes.
2 DR. BAILEY: Sean, I think it -- that's
3 what we were just talking about here. But I think
4 it's accurate to say that you are using buffered
5 peptone water rinse. So you're getting buffering to
6 the capacity that -- the buffering capacity of the
7 buffered peptone water is there, but you're not
8 monitoring the pH of those solutions to make sure if
9 you need to do any other adjustments. Is that -- I
10 think that's a correct statement.
11 DR. ALTEKRUSE: That's correct. But they
12 are placed in buffered peptone water on ice and
13 shipped overnight. And then they're put into
14 [inaudible due to failure of in-house PA system]. So
15 that's the extent of the system, but we think it's
16 pretty good.
17 MR. COUGHLIN: If I may, I -- Michael
18 Coughlin again. I have a couple other questions, the
19 first one to Dr. Bailey.
20 Assuming that the bacteria on and in the
21 bird are metabolically more active immediately after
22 slaughter, as opposed to those coming out of the

1 chiller, would it not be more efficacious to have an
2 on-line reprocessing unit pre-chill, as opposed to
3 post-chill?
4 DR. BAILEY: That is where most of them
5 are. It's only recently that there have been some
6 systems put in place where you have a post-chill dip.
7 Almost all reprocessing is done fairly soon after the
8 evisceration step on the processing line prior to
9 chill.
10 MR. COUGHLIN: But can you speak to the
11 efficacy of pre- and post-chill relative to --
12 DR. BAILEY: Are you talking about the
13 efficacy of chemicals pre-chill versus post-chill?
14 MR. COUGHLIN: Uh-huh.
15 DR. BAILEY: A little bit. But don't -- I
16 mean I haven't done an exhaustive evaluation of this.
17 There -- it depends on the chemical you're using and
18 your ability to get the active ingredient of that
19 chemical to the pathogen of concern, in this case,
20 maybe *Salmonella*, whether it's in pre-chill or post-
21 chill.
22 If your -- most of your pre-chill

1 applications are sprays. And so you can get
2 reasonably good contact with a spray if it's a good
3 quality spray inside/outside, but it's likely that
4 you're getting less contact time with the active
5 ingredient to the potential pathogen in a spray pre-
6 chill than if you went into a post-chill dip.
7 Now, if you have a pre-chill dip, you
8 would have for the most part the same likelihood of
9 exposure of your active ingredient to the pathogen.
10 The one thing you do have when you've come out of the
11 chill tank is that you -- with the immersion chillers,
12 you're getting a washing effect.
13 So you have reduced the organic load, the
14 fat content that kind of washes off a little bit,
15 maybe a little blood and other material that's
16 involved with processing. And you have a somewhat
17 lower level of both total bacteria and in your
18 pathogens if it's in a properly managed chill tank.
19 And so you have two issues there. It's
20 not quite a straight-forward answer. It's, Are you
21 talking about a dip versus a dip, or a spray versus a
22 spray, before and after the chiller? And then you

1 probably have a slightly better chance for
2 efficaciousness of the chemical at a post-chill dip
3 because you have reduced the organic load on the
4 chicken and you have reduced the level of bacteria
5 there. So you may get slightly better post-chill.
6 MR. COUGHLIN: Thank you. The other
7 question I have I guess would be for Dr. Buhr or Dr.
8 Cason.
9 I've noticed that carcasses coming out of
10 the chiller -- the skin seems to be easily detached,
11 and sometimes torn. I'm wondering. Is it possible
12 for the bacteria to actually -- you know, during the
13 hour's time that the carcass spends in the chiller,
14 can the bacteria be transferred actually underneath
15 the skin? And might that be a reason why it's
16 difficult to get a complete kill?
17 DR. CASON: Well, I think bacteria can be
18 transferred anywhere that water gets to while they're
19 in the chiller. But there are so many locations where
20 the bacteria can stay that it's hard to say that any
21 one location is more important than another.
22 MR. COUGHLIN: Has that area actually been

1 investigated, though -- the underside of the skin?
2 DR. BUHR: Mark Berrang has done some work
3 with post-chill skin removal and in looking at parts
4 with or without skin. And relatively the same level
5 of bacteria is recovered. So they are contaminated
6 beneath the skin as related to *Campylobacter*.
7 MR. COUGHLIN: Thank you.
8 DR. CASON: Huda Lillard did some
9 experiments a long time ago -- and one other lab, as
10 well -- where they were doing rinses inside carcasses
11 versus outside the carcasses after chilling. And they
12 found that chilling really redistributes the bacteria
13 so that they're pretty equally distributed all over
14 the carcass inside and out after chilling.
15 DR. ENGELJOHN: I'm going to ask if
16 there's anyone on the phone line that has a question
17 now.
18 (Pause.)
19 DR. ENGELJOHN: Any other questions here
20 in the room while we're waiting for the phone?
21 Yes, Felicia?
22 MS. NESTOR: Felicia Nestor, Food and

1 Water Watch. I just wanted to ask this. It's
2 actually more pertinent for tomorrow, but I know that
3 some people in the room will be leaving before the
4 discussion tomorrow.
5 And I'm assuming that the last topic we're
6 going to be talking about is the new Federal Register
7 notice that's out and what the Agency is considering
8 doing with the *Salmonella* results. And one of those
9 things is publishing the results by plant.
10 And I know that in the consumer group
11 meetings, we're going to be discussing this. So if
12 anybody has any ideas about why you think this is
13 unfair or fair or good or bad, I would really be
14 interested in hearing those ideas so that I can take
15 them back to the consumer group and, you know,
16 consider them as much as we can. Thank you.
17 DR. ENGELJOHN: Thank you, Felicia, for
18 bringing that in.
19 We will get into it a bit tomorrow, but
20 the real issue is: We want to hear back from all
21 stake holders on how we can all collectively move to
22 where we want to go, which is a reduction in

1 pathogens. So those are just what -- that's just one
2 of the options that we said we would consider and that
3 we prefer that that be considered, anyway.
4 (Pause.)
5 DR. ENGELJOHN: Okay. Any other questions
6 here in the room?
7 If not, then we'll adjourn for today.
8 Again, we will meet at 8:30 tomorrow upstairs in the
9 amphitheater. Thank you.
10 (Whereupon, at 5:30 p.m., the meeting was
11 recessed, to reconvene at 8:30 a.m., February 24,
12 2006.)
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