UNITED STATES OF AMERICA
DEPARTMENT OF AGRICULTURE
FOOD SAFETY AND INSPECTION SERVICE

ADVANCES IN PRE-HARVEST REDUCTION OF SALMONELLA IN POULTRY

Auditorium
Russell Research Center
950 College Station Road
Athens, Georgia

Thursday, August 25, 2005
12:30 p.m.

MODERATOR:

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C O N T E N T S

P R O C E E D I N G S

OPENING REMARKS:

DR. GOLDMAN: Good afternoon. I think we will begin, we're only a couple of minutes late.

My name is David Goldman and I am the -- in FSIS the Assistant Administrator for the Office of Public Health Science. And I have the first opportunity to officially welcome those of you who have come from across the country, in various professions and walks of life, but all of you have an interest in poultry production and *Salmonella*, to this conference.

Frankly, we were a little bit overwhelmed with the number of people who registered -- pre-registered for this conference. We are very pleased to see our colleagues from the industry and from the research community, both within the government and within academia, helping us to understand more about this particular problem.

It is also part of my duties as the first person at the podium to introduce to you the official party, those from both FSIS and the Office of Food Safety as well as the Office of Research Economics and Education, who will officially welcome you to this conference and will help us set the tone for this very important technical meeting.

So first, it is my pleasure to introduce to you our new Undersecretary for Food Safety, Dr. Richard Raymond,

who was named Undersecretary for Food Safety on July 1, 2005. He played an important role in the efforts in Nebraska to protect the food supply from terrorist threats and he brings that valuable insight to the USDA.

In addition to his role in protecting the food supply, Dr. Raymond also served as the head of the Nebraska Department of Health and Human Services Regulations and Licensure. He was also appointed Chief Medical Officer in January 1999, by then Governor Mike Johanns. He also served in Nebraska as an interim director of the Department of Health and Human Services Finance and Support Committee in 2000 and as the interim director of HHS in Nebraska in 2004.

Dr. Raymond was instrumental in his role in Nebraska in the development of the local health districts that serve Nebraska's 93 counties.

Dr. Raymond graduated with high distinction from the University of Nebraska College of Medicine in Omaha, Nebraska. Please welcome Dr. Raymond.

(Applause.)

DR. RAYMOND: Thank you, David and good afternoon everyone. It is an honor to be here and it is a pleasure to be here both at the same time. For the next couple of days you all are going to be listening to and discussing and hearing about new research and getting insights from other persons' experiences that hopefully apply to preventing

Salmonella from getting into the processing plants.

That really truly is the essence of prevention. And I would like to tell you a little analogy that people that have been in public health know and maybe some of you have heard it. And maybe some of you will hear for the first time. But I think it might help guide you for the next two days.

The story goes something like this, there was a fisherman on a river, his usual spot that he went almost every day. He was retired. A body went floating by, a person struggling trying to swim to shore and didn't make it and he had no way to help this person because the current was too strong for him to wade out. He thought that was really too bad.

The next day he was back at his fishing spot and two people went struggling by. And he was able to get out to one just in time to pull them to shore, but he could not resuscitate that one and the other one drowned.

He thought, maybe I could help these people if I brought a boat with me tomorrow. So, he brought a boat. And this time there was three or four people going down the river struggling and he got in this boat and he rowed out there and he was able to pull two to shore and resuscitated one, the other one died. And the other two that he could not get to died.

He thought if I had somebody that really knew CPR I could probably do a better job resuscitating these people. So, he got two more people down there with him. One to help row the boat and one to pull the people into the boat and one to do CPR and, you know what, they saved a couple of lives that day and only lost six. Because that day there was eight people that went down the river.

They realized for those that they were saving there lives some of them needed intensive care, so they had an ambulance to haul them to the hospital so they could continue to recover. But the hospital got overwhelmed so they had to add on to the critical care unit. And of course they needed more boats because there were more people coming down the river every day drowning.

And eventually they built a brand new hospital right by the river so people could get medical care immediately.

And you know what's wrong with the story, is nobody ever went up river or upstream to find out why everyday more people were falling into the river. And that is what we are going to do today.

We are going to go way upstream, instead of figuring out how much money we could put into our laboratory systems to detect *Salmonella* more quickly and to get the sub-species identified more quickly and to put more money

into antibiotic research since the *Salmonella* is becoming multi-drug resistant, so the doctors will have an antibiotic to treat people who are sick. Or putting more money into our efforts to create safer plants, maybe we could go upstream far enough and keep the *Salmonella* or reduce -- I shouldn't say keep, I do not think we will ever keep it from coming in the processing -- but if we could reduce the load up top, then think what we can do. Now, I have heard people say that we can not do that, you will never be able to reduce the *Salmonella* load on the farms. And I do not think probably you people who are here -- since this is pre-slaughter and that is why we are here today -- I do not think you people believe that. So, I am hoping you all can get together, and give us guidance and ideas to the industry on how we can get upstream far enough to reduce that load.

I want to tell you that *Salmonella* is not something we are here today because I think it might be important -- or that we might need to see what we can do about *Salmonella*. This is a priority of the Secretary. When I first met with Secretary Johanns and Deputy Secretary Connor, between the confirmation and the actual taking of the job, they gave me a list of things that they expected me to solve. And one of them was, "*Salmonella* problem". So we will have his attention and we will certainly have his support as we go through this process. And this is not a

two-day meeting, this is a process that I intend to have as a priority for the next three years.

By the introduction, maybe you could tell that Governor Johanns -- and he quite often asked me to take over interim this and interim that, wherever he thought he saw a problem I became a interim something or other until we solved it. And then I went back to being Chief Medical Officer. And he has tasked me with this problem to solve and he brought me here for a reason. And I do not intend to let him down. The one thing that we do have with Secretary Johanns, that I admire is a man of high ethics, morality, but also a man of conviction to promote public health.

I wouldn't have left Nebraska the only state I've lived in all my life, to come to D.C. into this environment if it wasn't for the man that I'm working for right now. Because I know he will support us in this effort, so trust me, we've got leadership at the top that will make sure we can get this done.

We've done a lot -- we meaning the industry, FSIS and others -- have done a lot in the last five or six years. If you just look at the numbers that the CDC confirms with human illness load, but we also confirm with our product sampling load, the decrease, the marked decrease in *E.coli* in humans of 42 percent over that time period. *Listeria* by 40 percent, *Campylobacter* fell 31 percent, *Yersenia*

decreased by 45 percent.

Then if you just want to just be the positive spinmeister on this you'd say yeah, *Salmonella* Typhimurium decreased by 38 percent, we did great with *Salmonella*. But in my short six weeks I certainly have come to learn that *Salmonella* Typhimurium does not reflect *Salmonella* totally. And we have certain *Salmonella* that have increased in fact in those same time periods.

I saw some graphs yesterday that were rather dismaying about the increase in some of the *Salmonella* species. Now how dangerous are those sub-species to the human life? I don't know that yet, those are some things we need to find out. Why are some increasing and some are decreasing? What are the characteristics of the different *Salmonella* sub-species that don't allow what we are doing already to have across the board effect in reducing the disease itself in the product sampling.

And I heard someone say the other day, well, it's different, you can't compare us to red meats. We have skins intact and the skins -- the *Salmonella* sticks to the skins, so there's no way we can be as good as we did with *E.coli*. And I would say, just look at the graphs for the last six years and see the product sampling and see how it's gone up. And I don't think anything's changed with the chicken skins. It might be that the bug has changed or it might be

the process has changed. But that's what we need to find out. It might be the load coming into the plants has changed. And that's why we are here today, to see if that's part of it and if that's part of the area we can contribute to the improvement.

We do have a strong system in place. I didn't come from Nebraska and leave my support network and my safety nets back there to come here and just be a caretaker of a good system. And it is a good system. We have the safest food product in the world. But if you could do better, then good is not good enough. At least it's not good enough for me. And we must do better and we will do better. I'm not a caretaker.

With the public health background that I've gained in the last six and a half years, particularly since Governor, now Secretary Johanns has asked me to leave private practice and come and come and be his Chief Medical Officer. And my dealings with all of the major public health associations in the country and media, national media and the D.C., life coming in out of D.C. for many meetings as a president of my parent organization of ASTO, those experiences have taught me more then I knew as a practitioner, when I was down at the bottom of the river, I was taking care of the sick person, treating them with antibiotics and IVs and sending them back home and not

worrying about what happened up river.

But in the six and a half years of trying to build systems in Nebraska that you heard mentioned -- building of the multi district county health departments to cover Nebraska with health districts that have never been covered before. And doing all of our bioterrorist and public health preparedness, I found the way to get this done isn't by thinking you know the answers. It's by getting all the people in the room that have different insights and different outlooks and different visions, and different thoughts, and communicating, and cooperating, and collaborating. And there's a difference in those things. Communicating -- I'm communicating today with you, I'm talking to you. Cooperating means we may take turns listening to other people talk, we may listen. Collaborating means we put something on the table and we take some risk. We collaborate, you know, the industries are going to say we are willing to go down this road with you if you are willing to do this for us. And we're going to put our collective reputations at risk on this project. And we are going to win. We will involve industry. We will involve other branches of government including CDC who are here today also, and the industries, and the consumers and we'll all be at the table together doing the collaboration that we need to get this done.

And so, I really am happy to see that FSIS already had this on their agenda when I came to town. Since I met with Barb Masters and said we need to do something about *Salmonella*, she said, you need to get to Athens, Georgia, because that's where we are going to start this project.

There's three things that I think we should have as goals. And the first, we've got to determine what interventions are already currently available to the producers that can form the basis for best management practices that will reduce the load of *Salmonella* in poultry before slaughter.

Second, we need to look at the research, I failed to mention when I said in the room we have the producers and the consumers and CDC and FSIS, but we also have the researchers. And we will listen to them also. But we need to look at the research for promising new hazard interventions. Identify what needs to be done, what can be done and what will work to make sure we use them at the production level to lower those *Salmonella* loads.

And finally we need to make sure we take in a full accounting of where we stand in regard to research. So we can identify gaps in our current thinking and there are gaps there. These gaps then can be filled with action from government academia and industry to reduce that *Salmonella* load.

We believe at USDA that everyone has an important role to play in the farm-to-table chain in food safety. And that's one of the reasons why I'm very pleased to have Dr. Merle Pierson and the other leading scientists from the Office of Research Education and Economics here today with us and tomorrow to hear and to listen and to discuss with us.

And of course, Merle comes from Food Safety before he went into research. So, he can look at it from many different angles, more than I can at least. But this is a chance for the health professionals, and the science professionals, industry, trade, farm groups, consumer interest groups, all to share their ideas and find common ground to tackling this problem.

And I leave you with one last thought that a successful public meeting cannot be measured simply by the research that's being presented or by the quality of the research being presented. Because some of the research being presented will need discussion and debate after it is presented. But we have to take into account the numerous opportunities that we have today and tomorrow to build new contacts with others in the field and to share new and innovative ideas openly that we can take back to our offices, to our agencies and to our universities. And I know for one that I will value this meeting as an

opportunity to get to know some of you better. And to get to meet some of you for the first time to open up my avenues of communications. I'm encouraged that you're all here. I know you're dedicated to the issue. We don't have the naysayers here, as Beth might say, we have people who want to think outside of the box here today. So welcome again and thank you for taking time to come to this conference.

(Applause.)

DR. GOLDMAN: Thank you, Dr. Raymond, and thank you for letting us all know exactly why we're here today and tomorrow.

Next it gives me pleasure to introduce someone who is probably familiar to many of you already, and as Dr. Raymond just said, Dr. Merle Pierson was recently a part of the Office of Food Safety. But was just recently appointed the new Deputy Undersecretary for Research, Education and Economics last month. Dr. Pierson was appointed as Deputy Undersecretary of Food Safety in February of 2002 and more recently served as the acting Undersecretary of Food Safety since 2004 until July of this year.

Prior to coming to USDA, Dr. Pierson served as a professor of food microbiology and safety at the Virginia Polytechnic Institute and State University, or Virginia Tech. During his tenure at Virginia Tech, he served as the head of the Department of Food Science and Technology from

1985 to 1994 and as acting Superintendent for the Center for Seafood Extension and Research from 1992 to 1994.

Dr. Pierson is internationally recognized for his work with HACCP, the Hazard Analysis and Critical Control Point Systems and research on the production and control of foodborne pathogens. Dr. Pierson is a native of South Dakota and received his BS in bio-chemistry from Iowa State University and then his Masters of Science and PhD in food science from the University of Illinois.

Please welcome Dr. Pierson.

(Applause.)

DR. PIERSON: Thank you, David, and good afternoon, everyone. Thank you, Dick, for the excellent overview and insight to introduce this meeting, the purpose of it. And I would also like to publicly congratulate Barb Masters who is now not just acting Administrator of the FSIS, but for real Administrator of FSIS. I'm very, very pleased to see Barb in that role.

It's really great to be here, to see many colleagues and folks that I've known for quite a few years. Some of you know, especially those of you in the poultry industry, we've had some discussions in recent months that I'm very, very, keen on addressing the *Salmonella* in poultry issue and *Salmonella* in eggs issue and being able to effectively address this whole area.

You know, I'm kind of the new guy at Research Education and Extension and I really appreciate the excellent working relationship the REE has and its agencies have with FSIS and other groups, especially the ARS group here in the Russell Research Center who have done just some absolutely superb work on interventions both pre- and post-harvest relative to poultry.

My experience as a food safety scientist and with food safety regulation and policy within USDA only reaffirms my belief that research is essential to a strong food safety program. I think, Dick, you very wisely pointed out the importance of research. It's not just doing research to publish another paper, but doing research that can be effectively communicated and effectively implemented in tying in with the policy folks such as FSIS, and obviously it takes a lot of cooperative effect too. It's not just regulations that makes things happen. But it's the industry that implements and really effects those controls.

I can only encourage, you know, further interaction between our government agencies as well as with other universities and other researchers and the like. I'm very pleased to see people here from other government agencies, you know, our land grant institutions, industry, and the like. This is just an absolutely excellent turn out for this meeting. It's -- I really appreciate your interest

and it shows your dedication to, quite frankly attacking this issue of *Salmonella*.

As you all well know, reducing *Salmonella* contamination of poultry has been one of the most intractable challenges in food safety research. Collaboration is vital to finding effective pre-harvest and post-harvest interventions. And as you all know from your own experiences, the best way to control *Salmonella* and other pathogens can be starting at the primary production system. It's one point to start and clearly you don't ignore the rest of the system, but you have to look at a fully integrated approach to addressing such issues.

USDA place as much value in working with our stakeholders to find solutions and from my perspective Secretary Johanns is very much committed to improving food safety, as Dick pointed out. And I might say that he's very, very committed to applying the best available science to policy and to articulating policy. There's a very serious dedication there and research is a foundation behind that.

USDA is extremely fortunate to have some of the best scientists anywhere, who have devoted their careers to working on developing pre-harvest intervention strategies. One of those researchers is Nelson Cox -- now why would I mention Nelson by name? Not just to embarrass Nelson. As

many of you know, you can't embarrass Nelson, okay. However, let me say that Nelson will be joining a very elite group of agricultural scientists when he's inducted into ARS Hall of Fame later this year. Congratulations to Dr. Cox and this is a very, very, very distinct honor to be inducted into the ARS Hall of Fame.

I know you have a great line up of experts including Dr. Cox in the program during the next two days and on behalf of Undersecretary Jen and all the REE agencies I hope you have an enjoyable, productive conference and the very best to you, thank you.

(Applause.)

DR. GOLDMAN: Thank you, Dr. Pierson, I appreciate that introduction and we'll appreciate our continued collaboration with you in your new role.

Dr. Barb Masters, as what was just pointed out, was recently appointed the Administrator of Food Safety and Inspection Service on August 1, 2005, after having served as the acting Administrator since March of 2004. She began her FSIS career in 1989, as a veterinary medical officer and has held a variety of posts since that time throughout the agency, both in the field and at headquarters.

Previous positions in the agency include director of a slaughter operations staff, branch chief in processing operations, and she supervised the HACCP hotline for

employees and industries at the Technical Service Center. Most recently prior to her serving as the Acting Administrator, she was the Deputy Assistant Administrator for Field Operations.

Dr. Masters graduated from Mississippi State University with a doctor of veterinary medicine degree and served in a food animal internship at Kansas State University. And she has continued to further her education by taking advance course work in biotechnology at Texas A&M.

Please welcome our new Administrator Dr. Barb Masters.

(Applause.)

DR. MASTERS: Thank you, Dr. Goldman, and I certainly want to thank Dr. Raymond and Dr. Pierson for their remarks. And I have the pleasure of welcoming all of you on behalf of the Food Safety and Inspection Service to this public meeting to talk about the advances that we can make in the pre-harvest reduction of *Salmonella*.

When I went to Dr. Goldman and asked him if he would be willing to put together this meeting on behalf of the agency, he stepped up to the plate and I certainly want to acknowledge the work that the Office of Public Health and Science has done in putting this meeting together. And I also want to thank the Eastern Laboratory for hosting this

meeting. It's no small challenge to put together a meeting of this magnitude and it's no small challenge to get you all through security gates and into buildings like this, so again, thank you, Dr. Goldman and your staff for putting this together.

Certainly, our agency has always looked at food safety from the farm-to-table approach. As Dr. Raymond, explained you have to look upstream, up river if you're going to make the kind of changes that we want to make. As we know, food safety doesn't start at the processing establishment or the slaughter establishment. In fact, if you look at our HACCP regulations, we ask the industry to consider hazards before, during and after entry into the establishment.

What happens before the animal gets to the establishment certainly has a great impact on the establishment's ability to address hazards at the processing establishment. And it certainly has an impact on our agency's ability to verify what the establishment is doing to address those hazards. While we recognize our regulatory authority is at the regulated establishment, we realize it's critical and what critical impact we can have by looking at the pre-harvest level. Some of the things that we do in that regard is to work with producers to educate them about pre-harvest food safety. Dr. Goldman has a staff and their

whole role is to work with producers and to educate them in that area.

We work with Dr. Pierson and his missionary and also with private researchers to look at pre-harvest food safety. We conduct farm-to-table risk assessments, with the goal of looking at hazard reduction along the farm-to-table chain. And we hold scientific meetings such as this, so that we can help further best management practices that will hopefully reduce the load of *Salmonella* in poultry before slaughter.

I also think it's important for us to recognize that because we are talking about pre-harvest food safety does not mean we as an agency are not going to continue to pay attention to what's happening at the regulated establishments with regards to *Salmonella*. That is where our regulatory authority lies and we are continuing to be concerned about what we are seeing in the establishments. And we recognize by working with the industry, we need to continue to have an impact there. But as Dr. Raymond said, we recognize that you've got to think about where that problem started. The people that were drowning in the river started somewhere.

And so, that's why we're here today to think about where is this problem starting. Those chickens didn't get the *Salmonella* at the processing establishment. So, that's

what we're here to talk about and hopefully further make some impact.

But I do want to share a model that we believe has been extremely successful. We as an agency worked with another segment of the industry. We worked with the beef industry. When we did a risk assessment based on some outbreaks, based on some recalls of E. coli O157:H7 in the beef industry. We asked all beef slaughter and processing establishments to reassess their HACCP plans. And in doing so we've seen a reduction in our regulatory sampling for *E. coli* O157:H7 and we've also seen reductions based on CDC data and foodborne illness relative to *E. coli* O157:H7. And we truly believe that those reductions have occurred in a large part due to significant industry changes in practices and the design of their food safety systems based on those reassessments.

And we believe that you can see similar changes if there's a similar model applied in the poultry industry. When you look at the prevalence of *Salmonella* in our pathogen reduction testing in the poultry industry, if you look at it aggregately, if you put all of our data together we're seeing a downward tend. But if you single out and take the poultry data, we are not seeing that same downward trend, particularly when you look at broiler chickens and at ground chicken. And that is certainly something that causes

us to be concerned. Again, that's why we're here today.

We've begun to do food safety assessments in the poultry industry. And whether it's based on our doing the food safety assessment of whether it's based on the industry doing their own reassessment of their food safety systems, we believe there can be changes if the industry starts looking at the design of their own food safety systems, that can lead to improvements in the processing of those chickens.

In fact, I believe that can lead to our next public meeting -- getting a little ahead of ourselves here. But as Dr. Raymond said, we need to continue moving down. So there's the upstream, but also the midstream. When I say this next meeting, I believe if we could start looking at the improvements made by the industry based on the changes in their food safety systems and if we also look at the scientific literature, we are starting to look at that literature and see what's happening at picking, what's happening at in the scalders, what's happening based on the health of your chilling system?

Then let's take the aggregate *Salmonella* data, and what's happening when you start making those processing improvements, and let's talk about all of that in aggregate at our next public forum. I think that will be a good public discussion for us to have in the near future.

Because if we take what we learn at this meeting and move that forward, hand in hand, this farm-to-table approach is what ought to get us where we need to be going when we look at *Salmonella*.

To borrow again from Dr. Raymond, I also want to recognize there's the downstream. Because we don't want to forget that when we look at *Salmonella*, we also have to remember farm-to-table. Because food handlers also have a critical role in reducing foodborne illness. And I don't want to not recognize the good work being done by some of our outreach programs for the food handlers. FSIS also have programs that look at food handler education with our Fight Bac Campaign. And our Food Safety mobile, and our USDA meat and poultry hotline. So, I do want folks to recognize that we have the pre-harvest. We need to be looking what is happening at the processing plant and we also need to recognize that there's things that happen downstream. Because it's going to take all of this to make a difference

Because in closing I think it's important that we all recognize public health is in our best interest. If you didn't believe that, you wouldn't be here. I think we all recognize and all believe that we want to make a difference in reducing *Salmonella* in poultry. And we all want to get at reducing foodborne illness related to *Salmonella*. If we had the magic answer we wouldn't be sitting here, we would

just be out there implementing the magic answer.

I've been in five poultry plants in the last two days. Everybody is using different interventions. Everybody has a different idea how to approach this problem, there isn't a magic answer and I don't think there ever will be one simple thing that gets us where we want to go. But I think we're heading the right direction by getting together to talk about this. And I think that we're all here together to say, let's come up with some ideas. And I think that the pre-harvest is the place to start.

If we start upstream and start thinking about some of the research and some of the ideas, I think it is going to get us where we need to go. So, I certainly applaud your commitment and I challenge you to keep that commitment up. I think that we're headed in the right direction and I think if we work together, and that's why I want to share some ideas of where we might be going, so you recognize our commitment to this.

I think if we work together, we can make the difference, so again, I applaud you and I want to let you know that we're in this together. And collaboratively I believe is a word that I heard Dr. Raymond use -- collaboratively we can make the difference and we can overcome the challenges that we face. So, again, good luck with the conference and I look forward to learning something

the next two days as well.

So, thank you very much.

(Applause.)

DR. GOLDMAN: Thank you, Dr. Masters. We're going to move immediately into the first official section. So I want to offer my thanks to the -- to our leaders here, Doctors Raymond, Pierson, and Masters, for giving us all a very clear charge. And what you've heard from them is not only a clear charge but rather a high level overview of why we are here. This first session we're going to have will help to get into a few more of the details, both from the FSIS perspective as well as from the human health perspective and our colleagues at CDC.

So, next on the agenda we have a session titled Why We Are Here. So again, we'll go a little bit more into detail.

And first we'll hear from Dr. Alice Thaler from FSIS. She joined FSIS over 20 years ago as a supervisory medical officer, after owning and managing a private veterinary practice for four years. She supervised inspection activities in meat and poultry slaughter in processing plants for six years. She was branch chief of the inspection systems development system in the Office of Policy for eight years where she integrated technical advances into policies and programs and implemented

strategies for strategic plans for reducing regulatory burden, increasing accountability and measuring results.

In 1999, she joined the Animal Production Food Safety staff as the national program leader for poultry.

Since 2003, she's served as the Director of our Zoonotic Disease and Residue Surveillance Revision in OPHS where she leads chemists, toxicologists and veterinary epidemiologists, who help to develop and implement the national residue program and lead scientific evaluation of new and emerging zoonotic diseases as they relate to meat, poultry and egg safety.

Please welcome Dr. Thaler, and I'll also ask Dr. Angulo, if you'd join us on the stage as well, as we move into this next session.

(Applause.)

WHY WE ARE HERE:

FSIS PUBLIC HEALTH PERSPECTIVE

DR. THALER: Just before we start we just have a few housekeeping things. Of course, the logistics are we are on a very, very tight schedule, so we have one of our veterinarians down in the front row who's going to hold up a little two minute warning sign that's green with a big two on it for the speakers so they kind of know that they're getting to the end and then a nice big zero with a red background so they'll know when we need to cut them off.

And the moderators will try to hold the speakers to the schedules, because we do have a tight schedule.

We're going to have question and answer sessions after the various sessions of speakers. So if you can hold your questions until that time, we will be carrying microphones through the audience, because there will be a transcript. So we will be reminding you to identify yourself and use a microphone so we can capture all these good ideas.

For restrooms, they're certainly on this level, the floor you came in and the second floor. Actually, they said the second floor has a little bit larger facilities than if you tried this floor and the first floor. So that will get us started.

All right, when FSIS looks at the FoodNet surveillance data we appear to be on track for our 2010 national health target objectives which are in the far right column. With regards to *Campylobacter*, *E. coli* O157:H7 and *Listeria monocytogenes*, this doesn't appear to be the case with *Salmonella*. Of the most common *Salmonella* serotype in people, Typhimurium is actually the only one that has had a sustained decline in incidence over time.

And here we see the serotype prevalence of the top broiler and ground turkey isolates from the samples we've taken under a HACCP program. The ground chicken is

basically the same isolates as the broilers, which you would expect. You will notice three of them are starred, the Senftenberg, the Redding and the Agona and those are the three that we find in turkeys that we don't find in the broiler top ten.

Of the serotype common to poultry products and people you'll see Typhimurium is the predominant human serotype, but it's only the fifth most common in broilers and ground chicken. And it's the seventh most common serotype found in ground turkey.

Then when you look at Heidelberg well, it's number two in broilers, number one in ground chicken and ground turkey and it is a fairly frequent serotype causing human illness. So, there's lots of questions about serotypes and hopefully a lot of discussion today regarding that.

Here we have the *Salmonella* prevalences of the PR/HACCP verification samples divided by the baseline prevalence to give you an idea of how far up to the baseline standard we are bumping when we get our HACCP results. And you'll see for the most part, poultry products are meeting the regulatory requirements that were established, our baseline levels. And that's a good thing.

But when we look at the data and we look at all sizes of establishments and looked at the combined *Salmonella* prevalence, we see this increase for broilers,

ground chicken and ground turkey, when you look between 2002 and 2003.

So, what does this tell us, we're looking at all size establishments together? So if we break it down by size of establishments to give you an idea where the increase is coming from, you can see for broilers it's pretty much across the board for large, small, and very small plants. For ground chicken it's basically the small and the very small plants that seem to be contributing to the increase. But then in ground turkey it's the large plants, we're not sure actually what that means.

If you look at the A set samples, and this is a string of samples we take to evaluate whether or not the HACCP sample results indicates that the plant have their food safety program in control, we're doing pretty well as far as meeting and passing the A set, which is the first set of samples. But between 2002 and 2003 we've seen a decrease in the percentage of sets that pass for broilers, ground chicken; and ground turkey, fortunately is not in that category. They have continued to pass 100 percent.

Looking at the broilers and the ground chicken by establishment size again, you can see that for broilers that the large and small plants, I guess we don't have the samples for the very small plants, are contributing, and for the ground chicken it's only the small plants that seem to

be having fewer A sets pass.

So as scientists, we're concerned in general about what does this increase mean and what could we do to reverse what we see as a potential trend when looking at all size establishment and for these three product categories. So, the agency will be examining *Salmonella* data from 1998 to the present to try to identify which specific plants appear to be displaying these negative performance trends. And we have our standard procedures of having enforcement investigation and analysis officers conduct in depth HACCP and sanitation verification reviews at the facilities to try to see if we can make sure this trend doesn't continue.

Achieving reductions in pathogens again, we hope that will reduce illness and again a reminder that it's important for all segments of the food production chain and consumers to properly handle poultry products to guard against foodborne disease. But this is where we play a role at the slaughter plants.

A little bit of an overview of this meeting now, to have an idea of what we're going to cover, what the speakers will cover. You've looked at the agenda, but the public meeting's going to consist of presentations on research and on practical experiences at reducing *Salmonella* in poultry, at the production level, and how that hopefully integrates into poultry that comes to the plant. How that

improves quality of poultry being presented for slaughter.

The meeting's an opportunity to provide input into the process and raise concerns about areas that are not currently under investigation. Are we looking at the right things? We're also very interested in the economic impact of implementing new practices. There may be something that works but it's not going to be economically practical and it needs to be feasible. And then the impact of food safety hazards on the market stability of poultry products. For example, there are foreign countries that have set a standard of zero *Salmonella* and how does that relate to our ability to reach those global markets.

We have three main goals for the meeting. The first is to determine whether the interventions available to producers now can form the basis for best management practices to reduce the load of *Salmonella* in poultry before they enter slaughter.

The second goal is to identify promising interventions and determine what steps are needed to be taken to make these interventions available at the poultry production level.

And the third is to identify which research gaps with respect to *Salmonella* control at the production level should be the focus of the research community, and that would include government, academia and industry.

Our intent and we stated it in the Federal Register notice is to try to pull all the information together that we get from this meeting and from any other sources we can get our hands on, and develop some level of compliance guideline material that would be available for producers. Basically, our version of a best management practice. So, that is the intended outcome.

So, we're going to try to do what we can to put up the barriers to *Salmonella* and get on with food safety improvement.

Guys, do we have a blank slide.

(Applause.)

Our next speaker is Frederick J. Angulo. Dr. Angulo is the Chief of the Foodborne Diseases Active Surveillance Network and the National Antimicrobial Resistance Monitoring System Unit for the Center of Disease Control. He's been an medical epidemiologist and epidemiologist intelligence service officer for CDC since 1995.

Before joining CDC, Dr. Angulo worked for NIH UCLA School of Public Health and served in the United States Army Veterinary Corp. He's received the CDC Neperno Citation for outstanding scientific paper, and the CDC James Steele award for outstanding contributions in veterinary public health.

Dr. Angulo received his MS in microbiology from

the University of San Francisco and his doctorate of veterinary medicine and masters of preventive veterinary medicine, specializing in epidemiology and a doctorate in philosophy in epidemiology from the University of California. And his presentation will be human health burden of *Salmonella* infections in the United States and the contribution of poultry.

(Applause.)

HUMAN HEALTH BURDEN OF *SALMONELLA* INFECTIONS IN THE UNITED SATES AND THE CONTRIBUTION OF POULTRY

DR. ANGULO: Thank you very much for the invitation to be here. I'd like -- much of the information that I have about the -- that I'll cover is coming from FoodNet data, so just to remind us of the history of FoodNet. FoodNet was established in 1996 and it is a collaboration between two different departments, the Department of Health and Human Services and the U.S. Department of Agriculture and ten state health departments.

FoodNet has the objective to determine the burden of foodborne diseases, to monitor the trend of foodborne disease over time and to attribute that burden to specific sources. FoodNet currently has, as I mentioned, ten states that are participating. This is 15 percent of the U.S. population in the FoodNet sites. Citing 2004 data, *Salmonella* is the most common -- was the most commonly

isolated bacterial pathogen in surveillance.

As has been described, there is exciting things happening with the surveillance today, there are important declines that have occurred with several pathogens. But there's been little change of *Salmonella*. These show some of those declines. You read the scale, anything below the one is a decline and you see across these pathogens listed, you see the declines that have occurred.

In particular, I'd like to highlight as others have mentioned, this remarkable decline of *E. coli* O157 infections, especially in the last two years. In fact, the decline of *E. coli* O157 has been so remarkable and rapid that we are below the healthy people 2010 objective, the national health objective that was established. And we sought to reach that goal by the year 2010. So we're there five years ahead of time because of this remarkable decline. It's noteworthy that this decline occurred in the face of the decline that are also seen with the FSIS data. Many of you who are familiar with these data recognize that prior to the year 2000 there was a less sensitive test method that was used.

After 2000, then the data become comparable. And you see in the years 2000, 2001, and 2002, there was little change in *E. coli* O157 prevalence in ground beef and that is also the same as the human illness incidence. And then a

remarkable decline in '03 and sustained decline in '04 captured in the FoodNet surveillance data of ill persons.

We're in the process of doing a collaborative study with the national cattle, excuse me, with the American Meat Institute in which they are surveying all the meat processing plants to find out what were the interventions that did take place that led to this remarkable decline. I highlight it because as others have mentioned that it really is a wonderful success story that we want to capture this case study of a successful intervention. It's also quite exciting could have this collaboration with the American Meat Institute, with some good news. To try to capture what exactly contributed to this decline.

Which brings us to *Salmonella*. And what is happening with *Salmonella*? Well, there's been little changes I've mentioned in *Salmonella*, there's some noises that have gone up and down. But it has not declined. In fact, it's not declined to such an extent that we are in danger of not achieving our national health objective. In fact, we're at the same place that we started when we set this goal. And therefore the total burden that we estimate caused in terms of human illness caused from *Salmonella* is that there are over a million people infected each year with *Salmonella*. Resulting as you see on the slide in tens of thousands of hospitalizations each year and hundreds of

deaths.

The USDA Economic Research Service estimates that *Salmonella* costs over $3 billion a year. So that begs the question, why isn't *Salmonella* declining in the face of remarkable interventions being taken by many food processing groups. Well, it's useful to look at *Salmonella* across the multitude of serotypes and in particular highlight the serotypes that cause the most human illness. And this is Typhimurium which causes 20 percent of human illness, Enteritidis 15 percent, Newport 10 percent, Javiana 5 percent and Heidelberg 5 percent. And there's a newcomer to the list number 8 in the top serotype to humans, which I will highlight at the end. But together these lists of serotypes result in 60 percent of human illness.

And it's worthwhile to note that amongst all those top serotypes, some of them are declining, and this is *Salmonella* Newport, the third most common serotype of humans and notice the similarity between the decline in *Salmonella* Newport and the decline of *E. coli* O157 in the last two years. It matches our understanding of the reservoir of Newport, that being cattle, and we believe matches the -- is a reflection of intervention made -- that beef processing that resulted in this remarkable decline in Newport.

So, my question is, is it possible that *Salmonella* is declining in beef but is in fact increasing in other

meats? This is a slide, to answer this -- to begin to look at this complex question we can use other sources of data. In particular we have a collaboration with the Food and Drug Administration in a FoodNet/NARMS retail meat study. In this study we -- in each of the ten FoodNet sites personnel go to grocery stores and purchase 10 packages of four different types of meat each month and test it in the laboratory for presence of *Salmonella* and *Campylobacter*.

What's noteworthy on this and granted this is not a random sample of meats in grocery stores, it's a convenience-based sample. But even with that non-random sampling, it is notable that the prevalence of *Salmonella* found on chicken breasts in grocery stores has remarkable increase in the year 2004 compared to the prevalence that was seen in 2002 and 2003. In many ways, this seems to reflect what has happened in the HACCP samples that we just saw demonstrated.

So, our question, the impression is that *Salmonella* is increasing in chicken -- the question is, is there a consequential increase in chicken associated human *Salmonella* infection? To look at this more closely we find it might be helpful to focus on four of the serotypes that you see listed. These four serotypes together make up 43 percent of all human illness of *Salmonella*. When you look at the three most common of those serotypes, this picture

emerges. The bottom line is Typhimurium. Typhimurium had an interesting decline in the first couple of years since baseline. And has been relatively constant since 2000. Enteritidis and Heidelberg shown hovering around the central middle line have had no decline since baseline.

What do we know about *Salmonella* Typhimurium? While from a wide variety of data sources we know that it is found in all food animal reservoirs and that we are currently working on efforts to attribute the burden of Typhimurium to the specific reservoirs. Important to that process is we know recognize that 5 percent of human Typhimurium infections in the United States are acquired for travel. We do recognize poultry as an important source of Typhimurium. We cannot say with extreme precision what proportion of illness, of Typhimurium illness, is attributed to poultry.

What about Enteritidis and Heidelberg? Well, with Enteritidis and Heidelberg, we recognize that with Enteritidis in particular that eggs are an important source and also the broiler meat is an important source. We've done several sporadic case control studies, two of them recently, that demonstrate that a way to acquire *Salmonella* Enteritidis infection is by eating or by contact with -- contact with broiler meat.

Also, note that 22 percent of *Salmonella*

Enteritidis infections are associated with international travel. It's amongst the greatest percentage of travelers of all the serotypes. So the total burden of *Salmonella* Enteritidis, when you look at domestically acquired burden should -- would be lower.

What about *Salmonella* Heidelberg? We also recognize a new development in the last several years that Heidelberg has been increasingly associated with eggs. Now, we recognize Heidelberg as a broiler meat issue predominantly, but the finding of Heidelberg in outbreaks that involve eggs is noteworthy. And in fact, there's been very interesting work done by Dr. Gast, here at the Agricultural Research Service demonstrating the ability of Heidelberg to be passed in an intact egg.

Well, this doesn't project very well and I apologize, but this is the other serotype that I wanted to highlight. And the scale is different here because the increase is so remarkable it doesn't fit on the other scale. And shown in a color that you cannot see but maybe you see the black dots that's Typhimurium which you see the subtle decline of Typhimurium since baseline, it's the lower collection of dots. The line that's at the top is a new serotype of *Salmonella* that we are calling and others are calling monophasic Typhimurium, it's actually more actually known by it antigenic formula name of 1 4,[5],12:i --

monophasic Typhimurium will also describe it and it has had a remarkable increase in the last couple of years.

In fact, the increase has been over a 1000 percent since baseline. So where is 1 4,[5],12:i, this is a group B *Salmonella* and where is it coming from? Well again looking at the FoodNet/NARMS retail meat study, it's only isolated 15 times off of meats. And all 15 were from chicken. Our impression is that chicken may be an important source and in fact one of the key scientific questions is to discern what the contribution of this new serotype is in the last -- it really has emerged in human illness in the last two years -- to really understand its contribution to this last two years of events in the HACCP data. Only one percent of human infections of the monophasic Typhimurium are associated with travel.

So, in general it appears that *Salmonella* is increasing in chicken HACCP data and a limited amount of data from retail meat sampling. And it also appears that chicken-associated human illness may be increasing, which leads us to the last objective of FoodNet.

That is, FoodNet sees as a fundamental objective to attribute the burden to specific sources. This is our attribution exercises which are in their infancy. These CDC attribution exercises include trying, can be viewed as a qualitative risk assessment taking to human illness and in a

top down approach partitioning it to the specific sources through a variety of different techniques. One technique is point of consumption attribution. A second technique is point of processing attribution.

With point of consumption attribution we will use information from the outbreaks with are the actual foods that the people ate that made them sick. And we'll also use information from sporadic case control studies. And we'll blend that information together to get a single best measure of the sources of the food that cause the illness in people that became ill.

A second approach is a point of processing attribution it's a molecular library approach by comparing fingerprinted isolates from HACCP samples and human samples. We can find indistinguishable strains in both collections and attribute the illness to those collect -- to the sources of where those indistinguishable strains have been identified. This approach, the point of processing attribution approach, has been very successful in Denmark.

Each year Denmark and their annual zoonoses report publishes a *Salmonella* count. And this is the *Salmonella* count from Denmark from 1998, more recent counts are available. You see a pie chart and this is human illness partitioned to the sources. And so they -- of all the human *Salmonella* infections that occurred in 1998, they judged

that 12 to 17 percent of them were due to travel and that 45 to 50 percent of them were due to eggs. We are well progressed to try to develop a similar model in the United States. Why is this exciting? Because, this is the trend data, and when you look at trend data in essence each of these years is a pie chart and each, therefore, when you put all those pie charts together year to year, you can look at trends of commodity associated illness.

And notice in yellow, the trend of broiler associated *Salmonella* infections, this is human infections that are broiler associated through this model and you notice the -- the remarkably high count in 1988, and then a decline, and a sustained low amount. I highlight that because they have well documented successes in controlling *Salmonella* in the broiler meat industry that results in a consequential decline in human illness.

The other information on this graph shows a surge in pork associated *Salmonella*, which came under control and a surge of table egg associated *Salmonella* human infections that also came under control based upon different interventions. I think there's clear evidence from Denmark that it is possible to reduce *Salmonella* in chicken and thereby reduce human illness.

So, in summary I would conclude that the human health burden of *Salmonella* is high, additional efforts are

needed to meet the national health objectives. Human illness data is consistent with the retail food data and also with the HACCP data. Suggesting an increased prevalence of *Salmonella* contamination of chicken in the food supply and a possible increase or at least not a decline in chicken associated human illness.

And further attempts are needed to specifically attribute the serotypes to their sources. And additional sub-typing efforts will be helpful in this regard. Poultry are an important source of several of these serotypes including stable serotypes like Enteritidis and Heidelberg and emerging serotypes like monophasic Typhimurium. And we have particular concern that this monophasic Typhimurium may be a major contributor to what we're seeing in the HACCP data. Efforts have been successful in other countries to reduce the prevalence in chicken and to reduce human illness.

Next steps, attribution needs to continue, in particular we have an exciting collaboration with ARS and other partners under -- trying to understand *Salmonella* Kentucky. We recognize *Salmonella* Kentucky to be common in -- in chicken, but uncommon source of human illness, however, it's clearer that they share the same strains. So some Kentucky do cause human illness. But we need to learn about pathogen load and infectious dose. We recognize all

serotypes of *Salmonella* are capable of causing human illness, that in fact it's an issue of infectious dose.

And then, finally, I think it's critical that we understand the contribution of monophasic Typhimurium. With that I'll be glad to participate in discussion.

(Applause.)

DR. THALER: Can we have the lights on?

So we have just a short question and answer session, but we want to get it off to a good start, so we can get questions answered up front about the broad picture. And we have microphones that will be going through, if you would please raise your hand and speak into the mic and identify yourself for the record. Any questions.

DR. GONDER: This is Eric Gonder. How has the incidence of human salmonellosis in Denmark changed over the years please?

DR. ANGULO: Well, it's a complex -- the question was how has incidence of human *Salmonella* in Denmark changed over the years. And that graph that I showed in fact, was human data that has been partitioned to the source of the human infections through this model. And so, overall there has been a decline, but in some commodity associated illness there's been increases and in others there's been declines; so it's a mixture of many sources. That's the beauty of the model. So, you see I tried to highlight on the graph, the

yellow bars were the broiler -- were the chicken associated *Salmonella* incidence which declined over time. But you notice there was a surge of swine associated human illness and also surge of egg associated human illness.

So, your question is trying to look across all that and there's so much noise in that, that we want to get down to the specific source associated human illness.

DR. KELLEY: Lynda Kelley. What methods were they using in Denmark for attribution, if they were just trying to .... human illness?

DR. ANGULO: Right.

DR. KELLEY: Were they using epidemiologic data as well or strictly some type of that?

DR. ANGULO: Thank you, for the question. It's a molecular library approach. It's a complex approach that actually has -- it's quite mature from a statistical point of view now. It's a Bayesian Monte Carlo simulation approach but in essence it boils down to ultimately being a molecular library approach where you compare the human strains to the animal strains and then partition human illness.

DR. KELLEY: What method are they using for molecular typing, is it Steele, is it Smith, what are they using?

DR. ANGULO: It varies on the degree of

specificity they need. In many incidences serotype is sufficient because in some farm animal reservoir -- it's only found in one, some serotype is only found in one reservoir. In other incidences like Typhimurium they have to use phagetyping and sometimes that's not sufficient they use MLST or PFGE, there is not a consistent approach because you only do what is necessary to fingerprint a strain.

DR. KELLEY: Thank you.

DR. STERN: It's always interesting to hear the relating of Nordic countries to the United States. There are some differences between the Nordic countries and the United States. One is in scale of industry and up until very recently I don't know where the EU is really going, but how -- there is a certain amount of size consideration. We probably will have a much more difficult time of reproducing the sort of work that you're looking to do as to what was done in Denmark and I was wondering if you could comment on that?

DR. THALER: For the record just identify yourself.

DR. ANGULO: That was Norm Stern.

DR. THALER: I know.

DR. ANGULO: And he was asking about the similarities and differences between the U.S. poultry industry and Denmark poultry industry. And of course

they're remarkably different in terms of scale. Although, 20,000-bird houses in Denmark are the norm just like they are the norm for many parts in the United States. So, although the scale is much larger, the actual production units might be very similar. But at production there are many differences. There are EU oversight on the use of chlorine. There is no use of chlorine, they use -- they don't use water chillers, they use -- they will use air chillers. So, there are differences in processing, that's what's great, thank goodness there are differences because it let's explore what's successful and what is less -- what doesn't work. And then, let's take advantage and capitalize on those that appear to be working.

DR. THALER: We probably have time for one more question. Do you see any. All right, thank you very much, Dr. Angulo.

(Applause.)

DR. THALER: I'd like to introduce the next speaker, which is Dr. Bhabani Dey, he has a degree in veterinary medicine and an MS in microbiology, an MPH and PhD in Food Science from the University of Missouri, Columbia, Missouri. He works for the USDA, FSIS, where he coordinates and manages activities in food safety and animal and egg production projects. His areas of research interest are food microbiology, veterinary public health and chemical

residues in meat, poultry and egg products. He's authored many scientific articles and presented numerous papers nationally and internationally.

Dr. Dey was the lead editor of the FSIS microbiology lab book in 1998. He's a member of the American Society for Microbiology, Sigma Xi, Gamma Sigma Delta, and the National Registry for Microbiologists.

And his topic is Industry Efforts to Control *Salmonella* Enteritidis, *Salmonella gallinarum* and *Salmonella pullorum*.

INDUSTRY EFFORTS TO CONTROL *SALMONELLA* ENTERITIDIS, *SALMONELLA GALLINARUM* and *SALMONELLA PULLORUM*

DR. DEY: Thank you, Dr. Thaler, for those kind words. I'm going to do the moderator and not the speaker. And the first session, Industry Efforts to Control *Salmonella* Enteritidis *Salmonella gallinarum* and *Salmonella pullorum* has three papers.

The second session is Entitled Current Broiler and Turkey Pre-harvest Production Practices, has two papers. So, the first session will have three papers and the first paper will be presented by Ms. Kennedy.

Ms. Kimberly Kennedy, is the Pennsylvania Egg Quality Assurance Program Coordinator for the Pennsylvania Department of Agriculture. She currently coordinates and monitors environmental manual test program and flock

inspection on 311 PEQAP flocks. She formerly was research -- senior research technologist at the Penn State University with *Salmonella* Enteritidis PEQAP program. Ms. Kennedy's work at Pennsylvania State included the isolation and the identification of *Salmonella* species from other bacterium and differentiation of species of *Salmonella*.

Ms. Kennedy has a bachelor of science degree in animal bioscience technology and management from Penn State University. Ms. Kennedy.

REVIEW & UPDATE OF PENNSYLVANIA EGG QUALITY ASSURANCE PROGRAM

MS. KENNEDY: Okay, hello. PEQAP is the Pennsylvania Egg Quality Assurance Program. Basically it's the program that we work with the industry to help reduce *Salmonella* Enteritidis, SE, in eggs. It is a HACCP type program. The hazard is the SE in eggs, the critical control points are SE-free chicks, SE-clean environment and egg refrigeration and processing. This program began in 1994 and currently as of July of this year we have 313 flocks that are on the program that I monitor. And that's about 85 percent of Pennsylvania's shell egg production.

This chart shows how the numbers have been decreasing from positives since 1992.

Environmental testing is what this program is based on. We require test of chick papers and manure drag

swabs throughout different stages of the flock's life cycle. These are some other swabs that we collect if we cannot collect manure drag swabs. Our swab testing is done at two of our laboratories. New Bolten Center is one and the other is Penn State University.

Now, if an environmental manure drag swab or chick paper goes positive we require egg testing and it's a 1000 eggs that are required, four times at two week intervals. And if any eggs are positive, immediate diversion is required. And testing will continue. And egg testing is done once again in the two laboratories and it's very similar to the environmental testing.

Okay, we require our flocks to be in compliance, and it's where every flock is inspected twice a year by a Pennsylvania Department of Agriculture inspector. We use the same inspection form and we do have -- I think it's Maryland and Ohio, we have some out of state flocks that are on our PEQAP program. They're required to send their inspection forms in. And also, those two states pay for the testings. And the states within Pennsylvania that are on PEQAP, PDA picks up the charge for the testing.

C&D inspections, cleaning and disinfection, that's also required for a house that's gone positive. And something the we newly started this year was, paying -- to try to get these flocks back into compliance if they fail

inspection to go and test environmental positive, they're going to be paying for that testing for the flock unless they can get back into compliance by requesting a reinspection.

Now, I'm going to go over our inspection form a little bit here. I have a lot of slides, but I'm trying to get through this quick. We have 10 criteria that we inspect the house on and if you pass eight out of ten you get an 80 percent and you will pass inspection. Now, they get a reinspection, that's what I just touched upon a little bit, briefly. You can have two reinspections within a six month period for your flock to get back into compliance and it's highly recommended because then we won't charge you for the testing. This is what our inspection form looks like, it's a standardized inspection form.

Okay, I'm just going to touch briefly, I have some pictures here on what our inspectors actually look for. This is along the outside of the building. We want vegetation and debris maintained. This is acceptable. We have unacceptable, we have overgrowth of vegetation is unacceptable. You can use livestock to control your vegetation. Now, any type of debris leads to rodents and through a lot of research they have come to the conclusion that rodents are definitely carriers of SE. There's a rodent on the shelf there, I don't know if you can see it.

Okay, another criteria is holes, which is structural architectural rodent exclusions. Poultry holes, holes within the poultry building, you really want to try to minimize them. And that's what our inspectors look for and talk with our flock owners and producers about. That's just an open pit door. If you can prove that you can seal the door you will be acceptable, that's something that we just newly passed. Before if the pit door was open you got failed, but now, you just have to prove you can close it.

If you can put anything up against the door, and a new regulation with organic flocks, there's organic outdoor access and PEQAP just recently approved that you can have outdoor door open and you will pass an inspection as long as you prove you can close it.

Bait stations and tin cats are a big part of our PEQAP program. Anywhere there's any opening by the pit door you should have bait stations or the tin cat. The tin cat is a way that a mouse can go in, like a mouse trap, and they stay in there live and then it's up to the flock owner to dispose of them. That's a maintained bait station.

Rodent control log book, a lot of our poultry producers require this of each owner, and also we require it on the PEQAP program, just so that we know that you're actually keeping bait inside, you're changing your bait. You're actually record the number of mice. This is what a

log looks like.

Rodent indexing, this is not counted in our pass/fail. We used to count number of rodents, but that's changed. Industry wanted us to change it, but we do keep track of it, but it is not counted against anyone.

Sanitation, keep the place clean. We don't want dirty equipment, dirty egg cooler. We don't want any debris when it can actually be cleaned down. This is unacceptable of course. That's very clean, probably brand new equipment. Same thing, no garbage piled in the coolers. And believe me, we have seen it and they do get failed for it. That of course is acceptable.

Temperature is 55 degree or less. It's very, very important, if it's 56, 57 -- we are pretty stringent on this. It has to be 55 or less.

And then, tin cats, this is what you use to calculate the rodent index, we have to have a number -- a minimum of 12. If you have any less you're going to fail. If they are not functioning properly you're going to fail. So, our inspectors are pretty stringent on this too. And they're very easy to pick up and clean out.

Now, this is just -- we recommend more bait stations and tin cats inside the house. That just shows a bait station, that's actually, shows some baits, even though the lid's off. You can put bait on pit ledges. Bait types,

we think that bait should be rotated.

Tracking powder can be used. Just fill any type of holes, if you have holds inside we want them filled, just like holes outside. Insulation, that's a thing to look for. Cinder blocks you can patch those holes up with a bunch of materials. This is open, rodents are everywhere in poultry houses, you've just got to try to keep them out. Different materials we use, suggest to use for holes.

And basically, as long as the poultry building inside and out is cleaned you're going to pass for a tier 11, which is poultry sanitation. We don't want no unnecessary amounts of feeds or birds in the pit. We fail that part of the inspection would probably be failed.

That just shows that the feed spill is not acceptable at the end of the feed bin. Egg and feed spills at the front of the pit are unacceptable for our program. Eggs in the aisleway, we do not approve of that. Manure on beams, it has to be below eight inches. Rodents, will actually go and live underneath that manure and get into those beams, I've seen it. That's a good pit basement, we look for clean. Outside the building, that's not acceptable. That's just bad sanitation.

Packing supplies we work with our producers, we don't grade this against them. If there's dirty packing supplies that come in, we will just make a note of it and

myself or we work with Penn Ag Industries will contact the producer -- the supplier of the packing supplies for that producer. And there's just a picture of the condition of packing supplies.

Our program works with, it's a team effort with Pennsylvania Department of Agriculture, industry. We do trace back investigations. I haven't partaken in one, but I know one recently occurred last year. So, we do keep very accurate records. There's many people that did help me with this presentation.

But basically, what I want to say, it does start at the level where the producers are, that's from what I'm learning. If you can work with the producers, work with your industry, you can try to reduce *Salmonella* in poultry.

Thank you.

(Applause.)

DR. DEY: Thank you. The next paper on this session will be Prevention of *Salmonella* Enteritidis in Shell Eggs During Production. It will be presented by Howard Magwire, Director of Government Relations, United Egg Producers.

Mr. Magwire, after retiring as the Deputy Administrator of Poultry Programs for the Agricultural Marketing Service in 2001, joined the United Egg Producers and the United Egg Association in Washington, D.C. as the

Director of Governmental Relations in 2004.

UEP, the United Egg Producers, represents the nation's shell egg producers while United Egg Producers -- United Egg Association, represent the further processors of eggs into liquid, frozen and dried egg products.

He's a graduate from Wayne State College, Wayne, Nebraska.

Mr. Magwire.

(Applause.)

PREVENTION OF *SALMONELLA* ENTERITIDIS IN SHELL EGGS DURING PRODUCTION

MR. MAGWIRE: First, like Dr. Raymond and Dr. Dey said, I moved from Nebraska to Washington. But it was a long time ago and I don't remember why anymore.

(Laughter.)

MR. MAGWIRE: Thank you for the opportunity to be here and talk about what egg farmers are doing. And particularly Dr. Raymond and Dr. Pierson and Dr. Masters for giving us the opportunity to explain some of the experience that U.S. egg producers have had in reducing *Salmonella*, specifically *Salmonella* Enteritidis in shell eggs. And I might mention that this is also a learning experience for me, because I plan to take away a lot of good information from here.

When I got the invitation to speak, it said, use

your research knowledge or practical experience to talk about reducing *Salmonella* at the poultry production level. Well, I'm going to talk about practical experience in reducing *Salmonella* in eggs at the production level.

As noted up here, the majority of our products does not move through an FSIS inspected plant right now. About two thirds of the shell eggs that are produced go to processing plants where they're washed, graded, sized and packed into cases or cartons for consumers. USDA does come to those plants FSIS or AMIS goes into them each quarter to look for diversion of certain low quality eggs. And also, FSIS has a refrigeration requirement.

UEP represents about 95 percent of the shell eggs that are marketed in the United States. United Egg Association represents a little over 90 percent of the liquid, frozen and dried egg products that are marketed in the United States. They take up about a third of all the shell eggs produced here. And of course all of those plants are under continuous FSIS inspection.

Going back a little bit in history, the contents of eggs, that is the egg meat, were long recognized as practically free from bacteria. We thought that all we had to do was properly wash and sanitize them, refrigerate them and store them, and we would not have problems. But in fact, I think back in the 1980s the American Egg Board,

which promotes eggs, even had several egg-containing recipes for food that recommended the use of raw shell eggs in uncooked food products. Of course they no longer do that.

In the late '80s a medical doctor working at CDC, Dr. Mike St.Clair, recognized that there was an increase in the number of *Salmonella* Enteritidis outbreaks in the United States. And before that we haven't heard about *Salmonella* Enteritidis. But most importantly to the egg industry, Dr. St. Louis (sic) observed that many of those outbreaks were associated with the consumption of shell eggs or food containing eggs.

For about a two year period back in the late '80s and early '90s, I followed every outbreak of *Salmonella* Enteritidis, that we heard about from industry, from CDC, from USDA or FDA. There were a lot of outbreaks to follow. And each one of those at that time, we found there was sometimes temperature or other abuse of the eggs or the food that had the eggs incorporated into it.

Nevertheless, when USDA, at that time began trace backs to find the cause of the outbreak, they sometimes could identify a farm where the shell eggs originated from and in some instances they found small numbers of SE in eggs at those farms. It is hard to confirm, but they did find them. Obviously we had to do something about it.

At that time the things that the egg people

thought about were washing and sanitizing and things like that. And then the researchers told us about a new phenomenon to egg producers and that was transovarian transfer of the organism. So we had to learn what that was about and regroup and take action accordingly.

To digress for just a little bit here from the egg farms to the egg processors, since they are regulated by FSIS, and why would egg further processors be concerned with SE? Well, of course in '88 that became an immediate problem for all farmers and we'll talk about that in a little bit. But FSIS requires manufacturers of liquid, frozen, and dried egg products to pasteurize all their products and test finished products for the presence of *Salmonella*.

So, why should we be concerned? It began over 30 years ago, processors recognized that if they wanted to consistently produce high quality *Salmonella*-negative product they needed to improve the quality of shell eggs broken. That is, they need to keep bacteria level as low as possible in raw materials coming into the plants, including, levels of SE. This seems pretty elementary today, but 40 years ago pasteurization was sometimes thought of as a silver bullet that would take care of any major microbiological problem. I guess as Dr. Raymond, also mentioned we weren't thinking very far up river at that time.

While not yet a USDA requirement, many processors have implemented HACCP programs in their facilities. And FSIS has indicated that they will propose a rule for HACCP in all egg products plants. We expect that they will have performance standards for assuring a safe product along with those pending regulations. And to meet performance standards for finished products processors are going to need to establish the efficacy of their processing methods and their pasteurization processes. It follows that raw product with high micro counts are going to require a more severe pasteurization process to assure safety of the final product. A more severe process is not necessarily a desirable thing when you're dealing with a delicate protein product.

I note that we're not aware of any outbreaks of *salmonellosis* in humans attributed to egg products since USDA implemented the Egg Product Inspection Act in 1971, but we certainly look at things differently now with HACCP and performance standards.

Also, today, many further processors where they once took surplus eggs from the table egg industry, they now have their own dedicated flocks. Sometimes they want us to divert the surplus eggs from those flocks into table use, particularly when the market's right. So, they know that they need to have the *Salmonella* Enteritidis out. In fact,

we'll talk about it in a bit, FDA is going to make sure they have it out.

As the last 34 years following the Egg Products Inspection Act demonstrated, pasteurization will kill most of these organisms, but yet some of our customers have -- many of the customers have fairly stringent standards on the raw material that's going into the product and that needs to be addressed.

Just a couple or three slides on *Salmonella* Enteritidis cases and before we talk about a few more the actions that egg producers have initiated. On this first one here, you can see that we think these procedures have been effective. If you look at the graph, it tracks data from 1970 through 2003, you'll see the trend upward in SE cases that happened there in about the late '80s through the mid '90s. You recall Mike St.Clair brought this trend to the industry and others' attention back in '88. It was in the late '80s and early '90s that the industry and folks like the State Department of Agriculture in Pennsylvania started to look at ways that they could reverse this trend and controls that they might have. They started doing that by '97 total outbreaks of SE in the United States from all sources was headed downward.

This slide if you can see it, is basically the same information but it's by region. And the two lines

after the blue and the pink lines are for the northeast and mid-Atlantic states. And you'll see that they were some of the first areas to have a problem with SE, as the lines went up pretty high. They were also the first areas to address the problem. Pennsylvania, working with FDA and USDA, established the Pennsylvania Egg Quality Assurance Program that Kim has talked about. Maine, up in New England states initiated something about this same time. And you can see that as we go out and get past '95, '96 the trend in those regions of the country is down.

The white line represents the Pacific -- the West Coast. And up until about '93, California really didn't have any SE problems. They were kind of proud of that. All of a sudden they started popping up out there. And you'll see the white line went up that graph went up. They immediately got on it. They had people like Pennsylvania and Maine and some of the other states to look at, they worked through their state department of ag out there and got another very tough SE control program, egg quality assurance program. And you can see now how that line has gone back down.

This chart here shows something that we like is egg producers, overall by this data SE incidence went down. But the percent of SE attributed to shell eggs in foods went down even further as this chart shows here with one

anomaly and that's in 2001, when out of 46 outbreaks in the United States, three of them were associated with eggs. But those three outbreaks accounted for over 60 percent of the individual cases. In the end I think one of those three cases was actually linked to SE in eggs on a farm. As you can see that number was not something that we want to have happen.

So, what happened? The voluntary egg quality assurance program that Kim talked about and actually 15 -- producers in 15 different states worked with their state departments of agriculture, with USDA, with FDA and developed egg quality assurance programs. They tended to be in the states that first saw the SE problems.

UEP developed a five-star program for assuring egg safety. Many of these companies developed their own programs working with the company veterinarians and state veterinarians. And then U.S. egg producers made a commitment to fix the problem.

What are the producers doing in these programs? Briefly they're securing chicks from NP, National Poultry Improvement Program, *Salmonella* Enteritidis-monitored flocks. And then, they are either testing the chick paper or requiring that the hatchery submit tests when they deliver the chicks showing that they're *Salmonella* negative. They don't want to invest a lot of money in those chicks

obviously before they know they're starting with a clean product.

After researching best practices producers have implemented and improved and rigorous clean and disinfection methods for houses. Some houses are even fumigated if they have an SE problem. Producers have implemented biosecurity measures that control movement of employees and cleaning and disinfection equipment if it's used between houses.

As Kim showed, they have buffer barriers around the houses, strong rodent pest control programs. They do routine testing of environments and eggs when necessary. If eggs are found SE positive, the flock production is diverted to breaking and pasteurization. Some of the producers don't test eggs if they get a positive environment they just divert them to breaking and pasteurization. Producers are following these kinds of practices from placement of chicks all the way through the time that the hen ends its life. And they continue testing and continue these practices.

Many, many producers, particularly in regions of the country that have experienced problems with SE now vaccinate their flocks. Most of them are administering two or three doses of live vaccines and some are going ahead and doing a dose of dead vaccine also.

Coincidentally, three or four years back, the United Egg Producers implemented some science based animal

care guidelines that's kind of a thing that all us in the animal community do now. But we're seeing an unexpected pay back from those. As we've given more cage space and improved animal husbandry practices, we've seen the health of the birds improve. We've seen production on individual birds go up. And that has also helped reduce the number of *Salmonella* Enteritidis that we're seeing.

After 17 years of working on this problem, we have not identified a silver bullet and I think somebody else made a reference to that. But we have an arsenal of tools when used together and used completely have had a dramatic effect of reducing the incidence of SE.

A couple of other impacts on us that have had positive effect if you can say that. Agri-terrorism, that's made producers become much more concerned about security in their operations, the control of people coming in, the control of employees moving from house to house. Similarly the concerns over avian influenza in the United States along with the traditional poultry diseases has heightened biosecurity. And now, where producers once looked at their farm from a biosecurity program, they have biosecurity programs in some cases for each house on that farm.

Pending government actions, we talked about FSIS's anticipated HACCP rule. Back in 1999, FSIS and FDA announced the egg safety action plan and part of that plan,

FSIS said that they might at some point regulate egg processing in those grading plants out there. In September of last year, FDA did propose an egg safety rule for the on-farm. It was a 78-page rule, so I'm not going to try to read it here. But some of things in that rule or many of the things in that rule are what we're doing now. We obviously -- since that's what we do we send in comments, but we went on public record supporting FDA's efforts in that regard. Looking at the components of the program proposed by them, we think we see some similarities in the practices that we're now doing. Excuse me, I'm going too fast here.

Look at this short list of chick procurement, biosecurity, pest and rodent control, cleaning and disinfection of houses, refrigeration and environment and egg testing. Indeed we laud FDA for doing a lot of homework in looking at the state programs out there before they actually came out with the rule.

When Dr. Raymond started -- I'm sorry to pick on him so much here, but when he started he stated that there are three goals for this meeting. The first was to determine if interventions available at the processors can form best management practices to reduce the load of *Salmonella* in poultry, eggs before slaughter, processing. And egg producers say, yes, to that, we think we've shown

that. And in fact, I think that's reflected by FDA adopting in their proposed rule many of the interventions that we're doing.

A second goal that Dr. Raymond mentioned was to determine steps to make these interventions available at the production level. FDA's doing it. FSIS has some additional pasteurization information that there's been discussion about incorporating in the current regulations. They're working on the HACCP rule. Don't misunderstand me here, I'm not standing up here saying a producers are begging for additional regulation. But we think that some of this stuff is fitting.

The third goal mentioned was to identify research gaps. One that we're seeing particularly as FDA further works on their rule is vaccination in commercial poultry. There's research on what happens in the lab and we know it's effective, but we can't quantify it. And we need research to quantify what that does as well as the other things that we've implemented.

For example, we need additional research to show what are the best ways to clean a poultry house. You deal with manure out there, you're dealing with live birds. What's the best way to clean and disinfect.

And we would also like to see some research on the actual -- and I've heard someone else mention that -- but

the actual incidence of SE in eggs today. At one time several years ago, it was estimated about one and every 20,000 eggs might contain the organism. And we probably need to update that.

Thank you.

(Applause.)

DR. DEY: Thank you, Mr. Magwire. The last paper in this session is Historical Achievement of the National Poultry Improvement Plan, it will be presented by Dr. Gast. But he will be presenting for Andrew Rhorer the Director of National Poultry Improvement Plan.

Dr. Gast is a research leader and microbiologist with the USDA Agriculture Research Service, Egg Safety and Quality Research Unit in Athens, Georgia.

He obtained his MS and PhD in poultry science from the Ohio State University. Dr. Gast's research focuses on the detection and control of *Salmonella* infections in poultry, *Salmonella* contamination of eggs.

Dr. Gast has received a number of awards and recognitions including a cooperative research award from ARS and FSIS, the American Egg Bowl Research Award and Poultry Science Association award.

Dr. Gast.

HISTORICAL ACHIEVEMENT OF THE NATIONAL POULTRY IMPROVEMENT PLAN

DR. GAST: Thank you, good afternoon. I guess since I'm the first ARS speaker of the today, I should take the opportunity to welcome you all to the Russell Research Center, and encourage those of you that are visitors, please sometime while you're here find an opportunity to take a minute or two and catch one of the ARS scientists from this facility and take them aside and tell him or her some of what your agency or your industry needs in terms of research that we might be able to accomplish. That's very valuable to us to have that opportunity.

About six weeks ago in Minneapolis, I had the privilege of hearing a talk -- trying to get this to advance. Thank you -- sorry -- had the privilege of hearing a talk by my colleague Andy Rhorer, who is a USDA APHIS employee and is administrator and director of the National Poultry Improvement Plan. And Andy gave a very nice outline of the history of this program and its 70 years of successful track record in addressing a wide variety of significant poultry disease problems. And when Andy couldn't make it here today, because he has a prior commitment, I thought this would be a good opportunity to take Andy's talk and scale it down and focus on the *Salmonella* portions of what the program has done. And so, what I would like to do for the next few minutes, is go through some of Andy's talk and tell you what NPIP is and

how it came to be and what it's accomplished. And then at the end I'd like to add a little bit of personal spin to this and try to put it in perspective and tell you why I think NPIP has worked. And maybe that will be a little bit of a lesson I think for us in looking at some other *Salmonella* control issues in poultry.

The story of NPIP is really about the convergence and cooperation of the efforts and interest of the government and the industry and the scientific community. And the story really begins in the late 19th century when developments were stirring in all three of these communities at the same time that eventually would culminate in the NPIP.

From the government side of it, beginning in the latter part of the 19th century, the government began to recognize it had a responsibility and interest in disease control. In the 1880s the Bureau of Animal Industry was established and the picture you're looking at there, by the way, is Daniel Elmer Salmon, who was a USDA veterinarian, who for a time headed the veterinarian division of the Bureau of Animal Industry. And of course after whom the genus *Salmonella* is named.

There were also at that time significant developments going on in the scientific community. This is the period in which we were beginning to understand the

microbial cause of many infectious diseases. One that was of particular consequence to poultrymen at the time was a disease called Bacillary White Dysentery. And during the later part of the 19th century, through some work that Leo Rettger did at Yale and elsewhere, an organism that was called *Bacterium pullorum* was identified as the cause of this disease.

And then in the early part of the 20th century, it was determined that in fact this disease was transmitted in eggs from parents to progeny, from hen to chick. And during the same period of time, there was a considerable evolution from back yard poultry operations into the evolution of a truly large scale commercial industry. In 1895, a farm in Pennsylvania instituted the use of a huge -- huge by that standard, the standards of that day -- 20,000 hatching egg capacity hatchery system, hot water heated. And it's this period that we're making the transition from folks that had chickens in their yard to people that are raising chickens for profit.

In the early part of the 20th century, we also began to develop some diagnostic tests for Bacillary White Dysentery, the first of which was the tube agglutination test. You can see there on the right side of it positive samples when blood samples from infected birds were mixed with an antigen preparation and incubated, you get a nice

looking sort of snowflake pattern of agglutination. This test by the way despite the fact that it's extraordinarily simple and ancient by today's standards is still in use and still effective. And it was the basis of the first organized state control effort of Bacillary White Dysentery in Connecticut in 1914.

About this period as well, one of the developments that began to influence the spread of *pullorum* disease was the institution of the shipping of chicks on a national basis by the Postal Service, which meant that not only were chicks shipped nationally, but diseases included Bacillary White Dysentery went with them and were distributed from point sources of origination all across the country.

And then in the 1920s we began to modernize a lot of our thinking about what this disease Bacillary White Dysentery is. In 1925, the organism was renamed in recognition of the fact that it's actually a member of the genus *Salmonella*, and was called *Salmonella pullorum*. In 1928, the name Bacillary White Dysentery or Bacillary White Diarrhea was abandoned all together in favor of the more modern term *pullorum* disease.

And by the 1930s this disease had become extremely significant in U.S. poultry commercial operations. *Pullorum* disease can in some instances cause 80 percent mortality. It was an extremely significant concern at the time.

There's another *Salmonella* disease of poultry that I'm ignoring all together in this discussion, although NPIP is concerned with it as well, which is the -- the disease caused by *Salmonella gallinarum*, which is called fowl typhoid. Which is a similar disease, but although that disease has been extremely significant historically throughout much of the world, it's never been highly consequential in the U.S. So, I'm glossing over it a little bit. But it's part of the subtext of the discussion here as well.

By the 1930s, we were also beginning to understand -- I mentioned earlier we knew the disease was egg transmitted. So there was this concept evolving and this illustration from the Storrs Agricultural Experiment Station Bulletin in 1931 shows. It almost -- if any of you have seen more recent illustrations of cycles of infection between poultry and food and humans, and so on. But we were beginning to understand at this point the cycle between hens and eggs and chicks and back to hens and so on.

Better diagnostic tests became available in the 1920s. In 1927 a rapid serum test was introduced in which you could simply take serum and mix it on a plate with an antigen instead of having to incubate it overnight as you did with the tube test. Even better tests showed up in 1931. A rapid whole blood plate test, where you can simply

take a loop full of blood, mix it on a plate with an antigen, you get an instant answer as to whether that bird has antibodies against *Salmonella pullorum*.

So, by the early 1930s, three big pieces of the puzzle from different directions had all fallen in place in regard to what we want to do about *pullorum* disease.

First, a growing nationally interconnected industry needed help desperately with an economically significant problem.

Secondly, we had developed an understanding that this disease was transmitted vertically from breeding flocks to progeny.

And third, we had dependable, efficient, inexpensive tests available to us. And in one of those reassuringly, I shouldn't say rare, but in a reassuring moment, government responded to this and acted and Congress passed an act that created the National Poultry Improvement Plan in 1935. The provisions that Congress acted on came from recommendations from the scientific community, from industry organizations, such as the International Baby Chick Association, which was very influential at the time; from states; from other government agencies and so on. Compiled all of these and created the program.

The slide Andy has here is a bit more technical, in essence I think you could distill the objective of NPIP

as it's worked over the years is to take the best available scientific information and turn it into action to protect the nation's poultry from infectious diseases.

How NPIP works is a bit of a unique exercise in comparison to what we see with a lot of other control and regulatory programs. Because the provisions of NPIP are voted upon by representatives of the industry, the government and the scientific community together in periodic conferences. And these plans are instituted and shaped by the people that for the most part will in fact be affected by the decisions that are made.

NPIP is divided into what are referred to as subparts that apply to different types of poultry including egg-type chickens, meat-type chickens, turkeys, water fowl, exhibition, game, back yard flocks and ratites such as ostriches.

The core testing provisions of NPIP include most prominently the rapid whole blood test, which is the principal qualifying test for status in regard to *pullorum* typhoid and then other types of samples that are instituted and performed as necessary when we get positive whole blood tests, including additional blood testing, collection of hatchery debris and in some cases organ sampling from positive reactor birds.

I kept this slide in just only as a curiosity for

a very small number of you that might be interested. This is a 1947 slide of some folks doing a *pullorum* rapid whole blood plate test. The most significant part for any of you that have been around the poultry disease community is the gentleman doing it there is Dr. Hiram Latcher. Who has, for over 60 years been involved in poultry veterinary community.

The principal classification for *pullorum* typhoid disease from the National Poultry Improvement Plan is the *pullorum* typhoid clean status. This status is achieved primarily by testing. Blood is tested at age four months, from 300 birds in a flock. If the samples are all negative the flock is given status as *pullorum* typhoid clean. If those samples are not all negative, a series of further tests have to be done, both to clarify what's going on and eventually a flock will not get that status until it manages to pass a qualifying test.

And if we look at what this program's been able to do. If you go from the mid-1930s when *pullorum* positivity in the blood samples that were collected was relatively high and project forward to the present, *pullorum* disease has virtually has gone away in the U.S.

Sort of an interesting element and Andy included this I think to illustrate this particular point, if you look at where *pullorum* disease in this country largely come from, one of the striking things is the influence of

backyard flocks, the red part of the bar accounts for a very, very significant portion of our continuing isolates, far more so then do commercial flocks.

In the late 1980s, an additional responsibility was given to NPIP and it struck out in an entirely different direction, with the inclusion of some provisions to test for a food safety organism that was not inherently a pathogen for poultry, *Salmonella* Enteritidis, the egg transmitted pathogen that's become of increasing concern in the 15 years or so to the egg industry.

In 1989, a provision for testing for SE in laying flocks was instituted and then, in the mid 1990s, *Salmonella* provisions that applied to meat type birds were also added to NPIP.

I'm going to show you three quick slides regarding some of the methodology for some of these programs for the food safety *Salmonella* and the NPIP, not because the provisions are so inherently so important, but just to give you an idea briefly of where the emphasis is. In the case of the SE clean program for egg-type chickens, the program includes both the blood testing type of component that is found in the *pullorum* program. It also includes some environmental sampling for the organism and it includes some more proactive efforts as well. The requirement that rendered feed be used and the requirement that bacterins be

used in multiplier flocks.

In regard to meat-type chickens, provisions are relatively similar, a little bit more emphasis on where the stock comes from, a little bit different twist to the environmental sampling. But again the same general type of program with the core element of it being the blood testing component.

A somewhat different program came along subsequently to those others is the *Salmonella* monitored program for breeding chickens, because the focus here is not serotype specific. The goal is to certify the overall *Salmonella* status of a flock. And in this case there's more frequent environmental monitoring and there's also institution of a paratyphoid *Salmonella* vaccine in the program.

And if we look at what NPIP has achieved in regard to controlling the food safety pathogens, the record is equally as impressive in a shorter time frame than what the program achieved with *pullorum* typhoid disease.

I promised the personal spin of why I think NPIP has been successful and this is partly why I glossed over how NPIP works. There are a lot more subparts and a lot more specific programs. And I didn't really want to emphasize in any great detail the provisions and what producers have to do. I wanted to emphasize why I think the

program has managed in both food safety and disease control venues to be successful. And I think it's really these two things.

The first is that the industry represented themselves shaped the NPIP provisions. Therefore, there's extremely widespread support in the industry for a plan that is known to be practical.

And secondly, the biennial conferences of NPIP where the provisions are reflected upon, voted upon and often modified provide an opportunity for incorporating the best, most current, new scientific ideas into a program that therefore is able to continuously evolve to stay at least at pace with an equally rapidly evolving problem from the disease itself.

And I've appreciated your time.

(Applause.)

CURRENT BROILER AND TURKEY PRE-HARVEST PRODUCTION PRACTICES:

DR. DEY: Thank you. The next session would be right now, which is Current Broiler and Turkey Pre-Harvest Production Practices. And the first paper will be presented by Dr. Bruce Stewart-Brown, and his paper topic is Growout Farm Influence on *Salmonella*.

Since 2003, Dr. Stewart-Brown has been working as a Vice President of Food Safety and Quality for Perdue Farms. In his role, he's responsible for food safety

quality and health for all Perdue Farms. Earlier Dr. Stewart-Brown, worked as a director of health services for Perdue Farms, Incorporated and coordinated health programs in all operations within the Perdue Farms, focusing specifically on the farmer raised poultry and as a director of poultry vaccine production for Salisbury Laboratories.

Dr. Stewart-Brown.

GROWOUT FARMS INFLUENCE ON *Salmonella*

DR. BROWN: Thanks very much. I will make a number of comments through the course of this talk. Most of them are a little bit specific to us and what we do. Having said that, I spent a lot of time talking with my colleagues throughout the chicken and turkey industry. And we spent a lot of time exchanging -- I guess I want to assure everybody that on food safety kinds of things, we -- although we're competitive companies, we're awful wide open as it relates to exchanging information and ideas and issues as it relates to food safety. Same can be said for health and some other things. So, I'm pretty proud of the fact that we try to make that go forward in the best way that we can.

I've been working a little bit in the last few years on growout farms in particular. And I -- I'm not undermining this philosophy that you control *Salmonella* from the top down. In other words from the breeders, pedigrees through GPs, through parents, and on down, because I do

believe that's absolutely necessary. Having said that, I don't think that will get us there. And I -- I think that other people would share this feeling and this kind of thought, especially as you -- the more and more you understand of the U.S. industry.

I think it's a much cleaner philosophy in different parts of Europe and we had some mention of different ways that poultry is raised throughout the world. It's really, really, critical that as we look at food safety and as we work on *Salmonella* and other food safety organisms that are in the pre-harvest type positions, that we understand the U.S. industry's make up, some of the specific challenges and incorporate our best minds into solving those solutions as it relates to the industry that we have.

We started a BMP program, and you've heard comments about one of the outputs of this meeting might be BMPs. Well, BMPs are a great way to work in the poultry industry. They are hugely important to us at Perdue Farms. They're really the basis in which we work. We have BMPs and have for years on production parameters. They usually have to do with simple things like feed and water and air and temperature. And we get more specific than that, but that's in essence a BMP. Now, we might not have called them that years previous. But certainly they are BMPs and then

we audit them on a routine basis. We audit them every week. That's essentially what the flock supervisor does, is he or she goes to a farm and looks at this farm that BMPs would be in place as it relates to production BMPs. Well we added Food Safety BMPs and we've added also welfare BMPs in the last few years. So our flock supervisors -- our relationships with growers tend to be communication of those BMPs. And you have to make them, you know, something that everyone can understand. And they have to be enough -- a small enough number that people can get their arms around them. So there's usually five or six maybe even three BMPs that you go and educate based upon that.

And once you've educated and got the program in place, that's essentially what you monitor on. And you end up with a percent compliance to a BMP and that allows you to assign a key initiative for next year kind of thing. It really works well in the management scheme of things to say, you know what that number three BMP in the hatchery, we're just not as compliant to that as we need to be. We're 70 percent compliant last year. We're going to be 90 percent next or 95 percent next year. Let's go educate on the BMP, make sure everybody understands what it is. And then make sure we get somewhere on the compliance to it. You can be successful with BMP programs.

Having said that, food safety BMP programs are a

huge, huge challenge. I put up the basic component to our food safety BMP program and it's not unlike other companies. It might have different terms or different discussion. But for instance, the breeders have seven basic BMPs, hatcheries got six, growouts got four, feed mills got three.

Let's just look at one of them real quick. Say the -- let's say the feed mill. Pelleting temperatures, pest control, housekeeping and sanitation. Pretty simplistic; however, you have to look and check paperwork based on pelleting temperatures. Assure that that process is in place and you do an audit on a routine basis to make sure. Now, that doesn't solve, of course, feed associated *Salmonella*, because coolers are a big challenge. It does get you going on it. Pest control within the feed mills important and housekeeping and sanitation, all the aspects of keeping a feed mill right.

Now, I would say this, as it relates to these BMP programs, we had mixed reception to the whole program. Breeders really took the BMP program and worked hard on it. It was really well accepted. Our growers actually really loved it. They loved to -- they wanted to have a great understanding of what we expected of them. They wanted to and felt good about all the aspects of the BMP program and they loved to get their grade essentially. They wanted to know how they did. And they were excited about it. And it

was successful and the audit scores over the years have reflected a general continuous improvement. The feed mill BMPs are really well accepted and successful and scores reflected continuous improvement. Hatchery took some work. However, after we worked on it and educated more and more and more and helped and figured out why in this hatchery that BMP might need to be run a little bit differently, I would call it successful and the audits are improving.

Growout stalled and it stalled pretty hard for us a couple years ago. It stalled from an acceptance, it stalled from a general perception within our own company that we had in fact got the right BMPs at all. And in fact if those BMPs were in place, did that make any difference. Not that they weren't good ideas. It's just that there was a lack of confidence among them. And one of the biggest components to the BMPs and the growout BMPs in particular, I guess everybody understands this, but I'll say it anyhow. The hatcheries are ours, the feed mills are ours, the breeder farms are generally contract. However, highly motivated different kinds of people. They have these birds for half a year or a year. They work in a different way.

Growout farms are contract farms, and they are a challenge as you run any kind of program to make people believe it. They've been doing it for 40 years, never had to do this before. Don't know why you're bothering me with

this kind of thing. I've got other stuff that I've got to do besides your BMP program. So, it takes a lot of education, a lot of work, but you can have and we do have successful programs at the growout level.

Well, this made us look at, and I'm going to spend a little bit of time on this. If you said, what -- let's look at these Food Safety BMPs and decide if we really think that we believe them. We need to convince ourselves and -- and aside from this kitchen sink approach which was through everything you ever heard of anybody doing and all that, and put that all in place and I want 100 percent compliance with that. And Europeans have this and this and this, and the primary breeders did this, how about we do all that. Aside from that approach, how do you determine what to put into your programs so that you can make some -- make some steps forward.

Well, we understood that although there's good research on it. Research I would say, I'll say this later I think, but of all the research we ever did and we do a lot of research. The food safety is the least reproducible research we've ever done. And the least productive research. You get a study in -- in a small pen trial. It looks really good, this intervention seemed to do a lot and then you put it in the field and it doesn't do anything like that. And a matter of fact, you run another pen study it

doesn't come out the same way. And I'm not, and I'm sure tons of -- I talk with a lot of you and I know this same experience exists. I just want for those of you that don't know, it's really a struggle to get good research, reproducible research.

We started to pull ceca from fat pad birds. And fat pad birds are those that you pull the fat pad for pesticide residue, do it every house and every farm. And it's usually a -- well it's at least six birds off a farm, three out of every house. If you have a single house farm you'd pull six pads. If you had two houses, it's three and three. If you have four houses, it's three, three, three, and three. So, really small sample size. Having said that if you do it long enough in a thing like food safety or even on a thing like infectious disease, you start to see trends over time.

So in this particular complex and we did this every house every farm, have for three years. Continue to do it. It's about 150 ceca a week from 40 houses on 17 farms, that's what kind of it averages out to be. Started in June '02 and analyzed the flocks that were processed through March of '05. I think I actually got some July numbers in here.

This is what if you wonder the correlation of ceca to processing plant data, this is a chart from that. And

essentially -- I guess one of these is a pointer, but you can tell. So this top line is the ceca percent from each month, from all those birds on that complex rolled up into one. Okay, so it's all that ceca. Get a weekly number, I look forward to seeing it every week. Roll it up into a month number and you can see, I think there's a little trouble getting started here, but you'll see it climb here in '02 through the winter. I've got a couple comments about that. It comes down in the summer, it will come down in the summer. Hit a huge number for ceca, I don't know how many of you run that kind type of assay, but 80 percent positive ceca is a tough number. And have worked on down then as it comes down to the current time period. We're running about 30 percent, 30-40 percent positive ceca, that's a great number. And there is a corresponding, albeit got some ups and downs in it, as you bring the ceca positives down, the plant seems to work more efficiently. That's not great, that's not huge, I think everybody understands that and probably believes it.

And it's not necessary, I think one of things when you talk about ceca people go well, what are you doing ceca for. We're not processing ceca, are you hitting a lot of ceca at the plant? And the answer's no, not doing that. But is there a correlation between what's in the ceca and what's on the bird, that's a -- and it's a clean number.

It's a farm number, so that's a farm number at 35 days. Doesn't it have a plan in it, doesn't have transportation in it. All those are good questions, need to be worked on, a part of the program. But if you said just the farm it's a decent number to look at. And it correlates to a certain degree with what you're taking to the plant.

Couple of comments here, one is we lost control of gut health hard in the winter of '02. We had coccidiosis and clostridial disease in the intestines. And if you lose gut health, you will lose your *Salmonella* control. If there is a single BMP or one that's very consistent, it's don't lose control of your gut health. Now, that -- that puts you in a quandary, if you're really working hard at your antibiotic use. Because you'd like to not have antibiotics in birds at any particular time that -- without a reason perhaps. Having said that, if you lose control don't get on it, don't get it treated, get the coccidiosis treated as well as the bacterial treatment. I can't -- I don't think you're doing the right thing. At least I don't believe that's a good balance of the risks.

In addition to that, at about this time and you'll hear some talks here through the course of this -- about this time birds processing were coming from 100 percent vaccinated hens. And I believe that once you get hens vaccinated, you see some response to the overall cecal

carriage in their progeny, to a certain degree. What we were trying to is try to get to this winter which is the winter of '04-'05 and cut this number so that it stayed through the summer time number and you can see that we did that. That's a really good result to take the summer number and put it into the winter and then run a new -- and this is probably a simplistic way to do it. But take the summer number, move it to winter and get a new summer number is what we were trying to do.

I want to show you a little bit of the whole results of this, what we call high-low, study, which is I didn't -- I don't want to put it, you know, a grower might come and go with Perdue, it might go to another company. It might be somebody that built new houses. So in essence, I didn't want to put into this data of a farm or houses that have not had at least seven submissions. Because one submission means nothing. I don't care if this time it was 0 percent and the next time it was 100 percent doesn't tell me anything. But after you get seven, eight, nine flocks you start to see you got some numbers. Now you got 50, 75, ceca to look at. And you've got something that may be suggestive of a farm issue or a farm -- representative of a farm.

These are all those different farms that had over seven submissions and I'll -- I'll try do this quickly, but

there are 42 farms that had between 51 and 60 percent positive ceca on all their submissions. So, there's the averages running right in there, if you look at it over time. Well, there's two farms down here that over three years -- over three years they've had hardly 5 percent positive ceca, that's over all seasons, all breeder flocks, everything, bad chicks, good chicks, all that kind of stuff and they got 2 percent or 3 percent or 5 percent positive ceca, well that's interesting. And then, you've got these farms over here, all though that three year time period there's two farms that give you nothing but positive. So, through the 14 flocks that we've had from those two farms you can just about assure yourself that the bird that you pick up to test has got *Salmonella* in the ceca.

So, okay, now I think we have something to study and the idea is back to trying to help you what the BMPs are. Well, if you can't figure out what the BMPs are, maybe you can figure out what the high guys are doing and compare it to what the low folks are doing. And take the low folks practices and make them your BMPs. And that -- that might work.

Well, to show you a little bit over time what a high farm might do, so this has 13 flocks in it. Here's a high farm that bounces around 60, 70, got a couple of flocks in a row where a 100 percent of the ceca were positive.

That's a 100 percent of let's say 12 ceca. So, 12 out of 12, and came down, whatever. But this farm is in that 80 percent kind of thing, what we would call a high farm. Here's a low farm, it's not always negative, but it had like four in a row, five, four in a row, five in a row that were absolutely every bird in there was negative.

Now, this is not doing any big intervention type thing. I'll just show you real quickly some farm shots. This is a low farm, low farm, low farm, low farm, junk everywhere, got weeds. Low farms, room, you got kind of kick stuff out of the way to get in there. So, it's not -- I'm trying to say it's not -- it's not the great things that we have talked about. High farm, looks nice. Nicely done, beautiful, really got it well groomed, do these people care they do well, they finish in the top for performance. They've got a lot of *Salmonella* though. If we got into these farms one of the things we found is subsurface moisture was pretty high in the -- the high *Salmonella* farms. The surface moisture was not necessarily going to tell you that. In other words, the litter on top might be quite dry. But if we dug down in this, actually that's brand new litter down there. And these had five flocks, four or five flocks on it. Brand new litter was put in actually put in wet. Never did dry out.

This flock, or this farm is a high farm and the --

it's in a very low area and doesn't drain well and actually is losing its floor. So, you get some seepage from the -- we're trying to keep the water from coming in under the foundation in that particular house.

So, anyhow, one of the things is, I wanted to say here's the things -- I don't want this whole talk to be about this exercise, but basically what it came out of was, these are the things that don't appear to be related. Water, livestock, other birds, wild birds, proximity to the road, proximity to other farms, outside appearance in general, as I showed you. PLT use, flock supervisor, treatment, black bugs, rodents, all that didn't relate. Gut health does. Litter conditions, subsurface moisture does. Maintaining the floors in older houses, having a floor in new houses. People are in a real hurry to put up new houses these days and sometimes we don't even put in a clay pad or don't build a floor like we used to build pads.

Farm size, generally probably associated with labor, but the smaller farms probably a little lower. Not that farm size has anything to do with it. But it has probably to do with maybe some of the maintenance and the other care issues. House preparation before a flock comes in is important.

Another benefit to this study and I want to get to this is that controlled food safety studies have been the

most unreproducible studies that we've ever run. We became focused on food safety research on the high farms. So, in other words if -- if you took an intervention to the high farm, one of the high farms, and it dropped to 20 percent I'm confident that did something. Because we've been looking at it for three years, it's never been 20 percent. So, if you can take you intervention to this high farm and show it to do something, you made a difference. That's a good place to test. And a good place to study.

Over -- our goal -- our 2005 goals have some BMPs or interventions that change the high farms. One of the things about high farms that we did was a temporal study, which said from one week to seven weeks how -- what percent *Salmonella* do you get out of those birds at each week? Well, the high farms and the low farms start out about the same, as you'd expect, they came from the same hatchery, same kind of breeder flock mix over time. And essentially something about the farm really kicked it up. It started to go down. I would have expected this kind of thing. Having said that, there's a second surge here even in the low farms. And the low farms, going to the plant or at the -- as you say the low farms are in this range in here generally. And in this particular case they were 30 percent or so when -- 40 percent when we tested them. That would have been a little bit on the high side for low farms. It's

been one of the highest times they'd have. But having said that a low farm has a different curve than a high farm.

There's probably and at least what we're working towards it trying to identify some issues that -- that are in this time frame. It gives you, here's the farm, and here's the time that I need you to focus on. Tell me what we're doing here at this time versus these other farms at the same time.

I want to tell you we've had three high farms. I think we've got some reason to be optimistic about. These three high farms, here's how they've been running for 12 flocks in a row. And we got several, at least one of the high farms, high farm three, that I think is three flocks are relatively successful. Here's two flocks that are relatively successful. This is really -- I'm not sure we changed these guys yet. But anyhow, most of it is associated with what I would call a newer set of BMPs or an evolution of BMPs. And we're still trying to best define them.

My -- my conclusion to all that is to say that BMPs are not necessarily what you think they are. Nor do we really, really know what they are. We've got a lot of work to do. It scares me, honestly scares me a little bit, to sit down and write BMPs for food safety and *Salmonella* and do a kitchen sink thing. I really want to do that, I really

am interested in doing that. We as an industry are working on that. I think that you can see some of that. We are dedicated to finding these BMPs and we want them to be real. And we want them to make a difference and I think we're getting somewhere.

If you do a kitchen sink, you won't ever maintain it. You can't keep it in a system long enough. It's too expensive, too -- you don't have believers, you've got too many people bailing out about a kitchen sink thing, because they can't believe all that is necessary or important. You need to get the right BMPs, then you need to make sure that they're in place.

And we need that, we can focus, people once we've got them, I don't have any -- I don't have any doubt that they would be implemented and really of all the groups, of all the food animal groups that I understand and know much about, if you get the poultry industry on a BMP program that they believe in it will be run and run successfully.

Food safety research, I was so proud -- I was really proud of myself for -- for putting collaborations in here, because I heard it a number of times, and food safety is such a big deal on collaboration, because you have to get all that you know and all that you can think of as it relates to research and then get with somebody that's got chicken houses and interest and focus and then let's figure

out if they really come out all that, that they're shaped up to be.

Thanks.

(Applause.)

DR. DEY: The last paper will be Reduction of Risk in a Turkey Production System Including Breeder and Hatchery Operations, will be presented by Dr. Eric Gonder, who is representing National Turkey Federation.

Dr. Gonder is the Senior Staff Veterinarian for Goldsboro Milling Company in North Carolina. He has a DVM and a Master's in Poultry Microbiology from the University of Minnesota and a PhD from North Carolina State University in veterinary pathology.

He has previously worked as a technical veterinarian, research biologist. He's a Diplomate of the American College of Poultry Veterinarians and he is currently licensed in North Carolina.

Dr. Gonder.

REDUCTION OF RISK IN A TURKEY PRODUCTION SYSTEM INCLUDING BREEDER AND HATCHERY OPERATIONS

DR. GONDER: I'll try and behave myself and stand behind the microphone. Those of you who know me understand that may be somewhat difficult. If I start to wander off, if someone will pull me short before I fall off the stage, I would appreciate it.

I would like to thank my staff, there were a number of other people that were involved beyond this with the company. My associate Becky Tilley, our laboratory supervisor Sharon Jackson, our QA supervisor Amanda Howell.

The historical background for our *Salmonella* reduction program at Goldsboro Milling Company began as a reduction program focused at a clinical problem with *Salmonella* Arizonosis in turkey poults. We had range breeders at that time -- when I say range breeders I mean that with about 50 percent of them the only thing that was under housing was the nest. The other 50 percent had access to exercise yards on the outside. We were experiencing about a 40 to 50 percent of those flocks being Arizona positive and we were experiencing clinical Arizonosis in the poults generally, between about 7 to 10 days of age. That situation became unacceptable, we started the reduction program actively in 1999, directed specifically at that time at the Arizona problem.

Now we did have some structural advantages within our company that are a little bit unique to us. Substantially different than other turkey companies and with a goodly number of the broiler companies, we're geographically compact. Everything that we have is within a 70 mile radius of the mill, one single complex including the processing plant. The breeders are company owned, everybody

that works on a breeder farm is a company employee. Same thing with the hatchery, the hatchery sanitation and quality control I have a number of difficulties with our hatchery manager. His attention to detail on cleanliness is not one of them. He is a maniac. That worked out very well for us.

We do have a large in house laboratory staff that we were able to divert to do additional testing. We had very strict feed mill quality control. The company has been obsessed for years with pellet quality. We have high temperature pellet lines, expanders, our conditioners recirculate, any dust is recirculated back in to the mash going back into the pellet mill. We tested a large amount of finished feed for period of about eight or ten years, finally discontinued it as not being particularly productive since we weren't really finding anything. And despite the fact that we were not using ATPI approved incoming ingredients. So you can work with the feed, but I'm probably going to reproduce that within this year, since it's been about five years since I've done that. And we did have very good enthusiastic management support for this effort throughout.

The history lessons that we learned through this entire effort are that cleaning failures were our number one problem, especially at the breeder farm level. These farms were quite old and a number of them required structural

improvements, especially to the floors, as Bruce mentioned, to make them easy to clean, maintain the houses in a much drier condition. That helped out considerably.

One of the things that we had to emphasize over and over and over and over again was that you cannot disinfect manure or wet surfaces. We messed around a little bit changing disinfectants, it was unproductive. When we started concentrating on physical cleanliness and dryness things moved forward much more rapidly.

Due to the geographic compaction and the large staff, we were able to engage in uniform inspections on the breeder and hatchery facilities. Those were concentrated, two people did them all. The general guidance on cleanliness was anything larger than my thumb nail or thicker than a nickel had to go. And we were able to do quite a bit of training over two to three to four year periods. That worked out quite well for us. Part of the reason that it worked out was we have very low staff turn over.

In the breeder organization the managers have all been there from between 12 and 15 years. The on farm labor in some cases has been there as long as 20 years. It's a dedicated group. We didn't have to spend a lot of time training new people. We didn't have to spend a lot of time on re-education. Management supported this with financial

incentives, especially, as far as cleaning, passing test was concerned, incentives that kind of thing.

The plan of action that we developed initially on this began to work primarily with the Arizona problem was to confine the breeders. This was an immediate cost of about one and a half eggs per breeder over the life of the breeder. But from what we had determined through a couple of experimental flocks that we had run on a confined basis, it was essential to the progress of the program.

We started vaccinating breeders for Arizona and *Salmonella* Typhimurium. We were not experiencing a problem with Typhimurium in the breeders, but our associated hog organization is lousy, I repeat lousy, with Typhimurium Copenhagen. The farms are collocated. They're feed out of the same feed mills. I did not want to risk a crossover introduction into the breeders if we were moving ahead with this program.

Again, we went to breeder housing inspections and environmental swabbing after cleaning, but prior to disinfection. We did drag swabs on each flock for -- each breeder flock -- quite a period of time, about every three weeks. We expanded the egg quality control program to 500 eggs per flock per week, the emphasis on dirty eggs. This is the physical inspection just making sure the eggs are clean on each flock as they're coming in.

Little later on about two or three years into the program, we added *Salmonella* Heidelberg to the autogenous vaccine preparation. There were two reasons we did that. We were experiencing a relatively low level of Heidelberg in the breeders, and it was present in the human serotypes that I was concerned about. We increased the site sampling on breeder farms as the positives on the environmental samples began to decline over the years. We started doing more testing.

We're currently focusing on meat bird brooder house cleaning and disinfection. Essentially trying to push into the meat bird organization what we learned on the breeder farms.

Now, the results of this over time I'll have to explain some of the nomenclature here. L-1 for us is first cycle breeders. We molt some turkey breeders, these results are all from first flight breeders. And it's a combination of information from cull poults at the hatchery and hatch residue. We were going back and forth between the two, we need to change as the incidence began to drop. The red line is from 2000 about the first 12 months after we first began the program, began to just start in with it. The blue line is 2004, which is the last year that I had complete data. The week of the year 1 through 52, is down at the bottom. You see the program overall made a very great difference in

vertically transmitted *Salmonella* from the breeders.

The serotypes that we isolated from these eggs or cull poults are presented here in the 2000 data is mostly on the left in blue. 2004 is on the right, cross hatch. You notice that we got a lot less in 2004 overall than we did in 2000, probably can't appreciate it, but the numerators and denominators are over the top of each bar. We had a big shift from *Salmonella* Arizona. Okay here's Arizona here and in 2000, you notice that there was none in 2004. That was 121 out of 142 positives that year were Arizona, Senftenberg we had 14 out of 124 and virtually disappeared in 2004. Javiana, there was the Heidelberg, that I was worried about, I didn't want that to spread, is the reason we started putting the Heidelberg in the vaccine. Muenster, Berta, this was an interesting one. As the incidence of these others dropped the incidence of Hadar on a percentage basis at least increased and we only got three samples out of that -- out of this cull egg stuff in 2004. But two out of those three were Hadar. The other is one that I cannot pronounce at least probably not correctly, represented one third of them, or one out of three positive samples. Again, we had quite a pronounced reduction, but the vast majority of it was in Arizona.

The clinical cases that we saw followed along with it. The stuff that was presented to our laboratory, usually

poults with problems. In 1999, we had 52 positive cases which represented about 1.5 percent of our total case load. 2003, we were down to about 1/10 percent. 2004 we had none. This would been again stuff presented for clinical Arizonosis, or diagnosis of clinical Arizonosis.

There was an associated change in management that went along with this period. It was not done for this problem. But we were doing it in any event, and this was some movement from single age -- sorry, multiple age production where we had brooders and finishers on the same farm, to single age farms, where we had a farm that would brood and at four and a half to six weeks of age those birds would be removed to a separate finisher farm. The brooder farm would be sanitized and the next flock brought in on to the next finishing farm, like that. Between 2000 and 2004 we had a very large increase in farms of that management type. Again, moving more to single age.

Now, as we did that, we started sampling the birds coming from the brooder house. When we transferred the birds from the brooder house to the finishers on the single age farms, we would have six birds brought into the laboratory. We'd perform cecal cultures on those, delayed secondary enrichment, the whole nine yards.

Okay, here's the 2002 data in the blue line on the percent negative on these birds that transfer. Red line is

2004, you can see it looks like we were starting to make more progress late in 2004. But there isn't a real clear path through here. So despite the fact that we're seeing a fairly significant reduction in the vertically transmitted stuff from the breeders we didn't seem to be making a lot of progress in the brooder house. Even though we'd gone to these single age farms.

If you look at the serotype distributions on these pre-move *Salmonella*s the time period here is a little different than the first slide, 2002, off to the right again is 2004 in the cross hatches. So you've got somewhat of a change in Senftenberg, I apologize we did not get the numbers up on top of these like I'd hoped. We did have an increase in Muenster. There was Heidelberg again, at least again it disappeared in 2004, whether that was related to the use of the vaccine or not is speculative. But it at least happened. Hadar stayed about the same as did Anatum, Mbandacka, another non-modal Agona. And then we had a couple of odd ones.

Again, what we are focusing on currently is trying to move forward with this is brooder farm inspections, and we're pushing that program on out into the brooders, it will be interesting to see if we can do a better job there. We're concentrating more on dry cleaning. That's going to be a little bit of an exercise. There's been a lot of

emphasis on washing within our company over the years. The use of high volumes of water. At least for *Salmonella* control, that does not appear to be a good idea for us. We're concentrating more on using blowers, compressed air that kind of thing. We're going to go back starting to use additional drag swabs on the meat birds. And if we get an opportunity in the meat birds, we're going to reevaluate competitive exclusion products. We've looked at several of those in the past. They haven't been particularly successful, but with the lower overall incidence that we have now, we think that we may see something. At least it's worth another try.

We did have a kind of unique opportunity in 2004 to do a market bird study. I'll try and explain this so that it makes some sense. We have two growout companies, ourselves and another company, that both feed into the same processing plant. We took ten individual cloacal swabs per flock from those two companies over a period of about two or three months on arrival at the plant. If any of those samples were positive -- in other words, if one sample out of ten was positive, we considered the flock to be positive for *Salmonella*. By that standard, more than 50 percent of the meat bird flocks coming into the plant were positive. Unfortunately, from both companies. So once again despite the fact that we had a four year program underway, there

didn't appear to be any discernable effect at the plant. And we'll get into that a little bit more later.

Salmonella Hadar, was the most common serotype that we found through that. That was 15th I believe, 337 cases in the CDC data I believe from 2002 or 2003. Out of the serotyping data there were no other serotypes besides Hadar, that were in the human top 20, which leaves me wondering some what about the public health significance.

The next slide is the serotype breakdown. Company one is us, company two is the other company. GT is ground turkey, which is current performance standard for turkeys. We were sampling ground turkey at the time. See here we've got percentage of samples off the side here. We didn't have a particularly good match in most cases between the Agona and the cloacal swabs. At least this one, the Hadar, looked like it matched up. We did have the highest numbers there. The only point where we had a relationship with human 2002 data was primarily on the Hadar. We did not pick up any Heidelberg in this series. And the vast majority was not in the top 20 for humans for that year. I don't know what that means, I'll leave that to y'all to figure it out.

The problems that we encountered with this as we tried to implement this program and these problems still exist today, is the requirement that Bruce mentioned for intensive uniform management to make these programs work.

You cannot do it with poor training. You cannot do it with high turnover in the help. You cannot do it without administrative support and good teamwork. If you don't have good relationships with the people involved, the program will fail, because they will not believe in it. Because there is no direct economic pay day for them. So, they have no particular financial incentive unless you create one.

We had problems with the autogenous vaccine regulations. I mentioned that we had -- we started out with Arizona and the autogenous vaccine. Went to Hadar -- or Heidelberg -- I'm trying to use Hadar now. The problem with the current autogenous vaccine regulations -- some of you in the audience may find this hard to believe -- if the vaccine works too well, in other words if you can no longer isolate the organism from your birds, you must discontinue use of the vaccine within 15 to 24 months.

The National Turkey Federation, the National Chicken Council and the Triple AP protested the situation to the Center for Veterinary Biologics since 2002. So far the situation has not changed. It's an impediment.

Okay, I've got more time than I thought, but I will try to move along. This is something that I hope we can move forward on because it does stand in the way of trying to maintain a consistent program if you have environmental exposure. In other words, you can isolate it

from the environment but you can't get it from the bird. Because to make the autogenous vaccine you have to get it from the birds.

The other thing that I hope has been changed since it's my understanding that vaccines for food safety claims are being transferred back to USDA from FDA is that we can make progress on competitive exclusion products, especially on undefined products.

Nurmi found out about this I believe in the '60s or '70s. As yet, we still cannot use this approach in the United States. That's a long time to wait, folks -- a long, long, long time. We should be able to move past that.

Okay, I've got some problems with turkey performance standards and I think you saw it in that data there. I cannot influence the current turkey performance standard by anything that I do in the field. Part of the reason is the way the performance standard is set up is you take a 25 gram sample once per shift for 53 shifts. Okay, that's 25 grams of ground turkey per day. We put 1.2 million pounds live weight into that plant daily. Okay, bad sampling. Serotyping has no effect on the results nor is there any quantification. One *Salmonella* is as bad as six logs of *Salmonella* typhimurium DT104. You fail the test in either case, or at least the bird is positive. That is not helpful when you are trying to reduce things by a percentage

in the field. We need to find another way.

Live interventions generally can only reduce *Salmonella*, they may stay positive. I may have a program that's 99 percent successful but that still means there's one percent positives there, the test will be positive, should be positive Non-human serotypes or at least those that don't appear to occur frequently in humans carry the same weight as those that cause problems.

Okay, I virtually eradicated or we virtually eradicated *Salmonella* Arizona within our system. What does that mean? It didn't appear to change the human incidence of Arizonosis at all. But it does mean that if we found *Salmonella* in the ground turkey samples, we failed the test. Is that helpful? Little hard to say.

Does the standard improve public health as it is now or should we replace it with a HACCP based standard. Now to me, a HACCP based standard means that you go in there with a plan, a plan to reduce the problem. It may take time. It took me four to five years to get around this Arizona deal in a company that was relatively tightly organized, with some regulatory impediments. You can't pull the trigger and get a problem that's breeder related to go away all at once. Unless you kill the breeders. Which is one solution I guess.

(Laughter.)

DR. GONDER: Some difference between turkeys and broilers that need to be kept somewhat in perspective. We're in the field about three times as long as broilers. There is more opportunity for exposure, likewise hopefully the intestinal milieu will become somewhat more stable as the birds age. We don't know that for a fact yet. The industry itself isn't completely vertically integrated. The company that I work for is a little unusual in that regard. There are a lot of other people that get poults and eggs from the outside. Don't really have good control over the feed mills, that kind of thing. Our current standard is based on ground product -- I've been over that. We can clean most of the live haul equipment fairly easily. We're still using fixed coops on trailers. Little easier for us to handle that and our primary breeders are virtually *Salmonella* free.

And I imagine that I'm out of time. I'll quit there.

(Applause.)

DR. DEY: Now we'll have a question and answer session. So may I request all the presenters to come and sit on the podium, please.

VOICE: We'll hand you a live mic. Please don't press the buttons.

MS. RICE: My question is just for the person that

didn't give handouts, are we going to have those available to us?

DR. DEY: Some of the handouts already are.

MS. RICE: Right some of them are.

DR. DEY: We didn't the other handouts.

MS. RICE: Will we be able to or have access to get those?

DR. DEY: No.

(Laughter.)

DR. THALER: Could you identify yourself to when you have a question.

MR. WARD: Could you address the importance of sanitation between flocks and then the procedures that you go into there?

DR. DEY: Please identify yourself and ask who should answer your question.

MR. WARD: Casey Ward, EcoLab.

DR. STEWART-BROWN: Was that addressed to anybody in particular. I think it was addressed to you.

DR. GONDER: Okay, essentially we remove all litter from the house, and when I say all litter again, it goes back to stuff that's basically the size of my thumb nail and no thicker than a nickel. The house is allowed to dry. We do go ahead and blow it down, or wash down -- let me start over.

We've taken the birds out of the house, the first thing we is do is kill the insects. The second thing we do is to go in there with soap and water essentially and a high pressure sprayer. Wash everything that's overhead down into the litter. We then remove the litter as I said, down to the size I specified, let the house dry. At that point, we'll go back in and disinfect. Is that what you needed?

DR. STEWART-BROWN: You know, in some of those high and low farms, there were litter change-outs and clean outs in between there. For instance, let's just say some of the high farms, you had some litter, complete litter removal during those three years, sometimes probably a couple of times. And yet those high farms stayed high and the low farms stayed low. One of the things that we believe, if you put in -- if you clean out, put in new litter and that new litter is wet you did the wrong thing. It's not -- that's not a good -- that's not a good move. If you -- and getting good dry, plentiful litter is a real, real big challenge. However, having said that, that's what it needs to be. It needs to be good litter and dry litter.

In our primary breeder operation, we've done a lot sampling of litter as some of the others have, I'm sure. Even good dry litter has had SE in it. At least a number of times. So, there's a lot of the components of cleaning out and I know I didn't address the disinfection piece and all

that. But I wanted to touch on the litter and litter removal and litter control. Sometimes that sounds like a really good idea, and sometimes it's a really bad idea.

DR. GONDER: There's one thing that I -- again, it's kind of a regional difference. Where we are, we're very lucky we've got a relatively inexpensive source of kiln dried shavings. So when we replace we can go back in with dry stuff. The other thing again that I just harp on you can't really disinfect a wet building. So, we try and concentrate on getting those buildings dry before we disinfect.

QUESTIONER: This question is for Kim, with the Pennsylvania Egg Quality Assurance Program. How much government -- what is the government invested in the program in terms of the state government pays for the program or any other governmental sources? Have you compared the price of eggs in Pennsylvania versus neighboring states that don't have quite as intensive program?

MS. KENNEDY: I have not compared the price of eggs. But you mean who's paying for the testing for the program?

QUESTIONER: The whole program.

MS. KENNEDY: The Department of Agriculture pays for the testing. Each sample I think an environmental sample --

QUESTIONER: No, annually.

MS. KENNEDY: I do not have those figures.

QUESTIONER: Tens of millions of dollars?

MS. KENNEDY: I think it can run, if they test -- let's say they run a sample through, I would say it probably cost us for each flock of samples, if they're negative it would probably be about, oh if I had to guess maybe a few thousand dollars. But if they go positive, it can get really high up to five to 10 because you're testing the eggs and if they decide not to divert to divert to break plus continue testing until the flocks out. But I've never compared numbers.

DR. THALER: Would you be able to say that it's been kind of a constant cost because the number of positives have come down as more plants are added, more production facilities?

MS. KENNEDY: It's a constant cost to the Department of Agriculture of Pennsylvania?

DR. THALER: No, the budget, do you know if the budget has kind of been constant or has it been an increasing cost?

MS. KENNEDY: I think it's been going -- well, we're getting more flocks in the program. So, I'd say it's probably constant, but the number of positives have been going down.

MR. TREAT: Gary Treat, with Pilgrim Pride. Eric, this is for you. I noticed you had an interesting statements on antibiotic use. Would you mind sharing just a couple of minutes with that part of your program?

DR. GONDER: Do you want me to go over the standard statements, is that what you're saying? Okay, if you'll all turn to the back page of my presentation. I don't have one along with me, you'll find what has become properly known I guess as my standard statements. I include them in virtually every presentation that I give. And if I can remember them correctly no hormones are used in U.S. poultry. That's one that I get asked fairly frequently from the outside and from our own plant brokers.

There's no documented cases of which I'm aware of human treatment failure due to antibiotic resistant bacteria acquired from USDA inspected meat. I tried to check that as closely as I can. If I'm missing sources and someone can furnish me a correction, I'll be happy to amend the statement.

No one treats whole flocks for single bird infections, with fluoroquinolones, or anything else for that matter.

No fluoroquinolones are used in U.S. feed. And cooked meat cannot transmit antibiotic resistance.

The most recent one I added is one that -- it's at

the far end, that I'm really kind of fond of. Because to me this epitomizes the needs for a terminal step in food preparation. And I apologize for appropriating a non-sequitur obvious man's emblem, which is I refer to as the no duh sign, "cook your meat." And I deliver that message at every opportunity, because while we can talk here about reducing *Salmonella* and reducing food borne pathogens, the likelihood that we will be able to reduce them to zero is remote. Which means as several people have mentioned before there will be a continuing need for emphasis on proper food handling. That relates somewhat again, to the farm-to-fork deal. Yes, we might be able to reduce *Salmonella* somewhat coming into the plant and the field. But at least in our case we were able to accomplish more with plant interventions within six months than I was able to accomplish in the field in five years. If our plant was running entirely a cooked operation we'd be completely successful. Is that what you were hitting at?

MR. HENRY: Craig Henry, Food Products Association. Bruce, could you and Eric comment on your serotyping data about the differences if any on the serotypes that you found on the farm among the houses.

Most of the data usually presented is a summary of the farm and/or what you find from a flock overall. What types of differences did you find especially over time on

the serotypes within the farm, between houses?

DR. GONDER: I haven't looked at our data that way, I don't know if Bruce has or not.

DR. STEWART-BROWN: If you're saying here's a -- for let's say a six house farm and you look at those houses, would there be multiple sero groups or serotypes on that given farm or would it be -- tend to be the same. I never really look at -- at a single sample on our data, for the most part unless we're doing a real research study and we're going to take 30 samples out of a house and then I might do that. So, even in any given single sample type thing I wouldn't have really gone to serotype. I can say that there's trends in serotypes for us. And they -- they appear to be seasonal, which is a little bit surprising to me. And I also think that if you watch the ebb and flow of serotypes -- and people have talked about this before -- but you will tend to knock one down and another one comes in pretty hard. And so, you're -- to Eric's point about autogenous and your approach to *Salmonella*, to go after one knock it down and get some low levels and then to stop your approach to it, one thing is probably and frequently does another serogroup is now your target, and needs to be. To let up on that one that you've beat down doesn't seem like the right thing to do at all. And of course there's some limit to perhaps the number you can go after at one time and make real

appreciable progress.

Back to serogrouping or serotyping, we tend to see Kentucky, I don't really understand this, but Kentucky would be a higher incidence for us through the summer time and Typhimurium higher incidence through the winter time. And that's gone on for a couple of years now, I'm not sure what the logic is and/or that it would continue to do that kind of thing. But we are doing a lot of serotyping, it's a necessary thing, it's important to understand, you know, the whole picture of things. But it's not, it's something that you have to spend a lot of time with over time.

DR. COX: I'd like to make a comment. Having looked at a lot of flocks a particular serotype might predominate, but here at Russell Research Center, looking at commercial poultry, we found four and five serotypes on one chicken carcass, so you can't get too hung up on one particular serotype, you find an abundance of them in a house and you may find as I said four or five or six on one food product.

DR. GONDER: That is one limitation with most of the stuff that we were doing. In virtually all cases we're picking one colony off the plate to send off to serotype, that's right.

Now, kind of going back to the environmental thing, that's jogged my memory a bit. With Arizona, we were

fortunate in that we can identify Arizona with malonate. We were able to do a little bit more work with Arizona without having to resort to serotype. It was quite easy to find Arizona in the environment outside the house. And it apparently is not uncommon in other environmental locations. I did have one rather unfortunate episode where we reclayed the floor in a breeder house, could immediately pick up Arizona from the environmental samples. We had run out of time, we had to place the flock. The flock subsequently went positive, I got back in that clay pit and we could isolate Arizona from the clay in the pit that the new floor had come from. So, it's not just a turkey bug. At least there weren't any turkeys in the clay pit.

MS. COOK: Leisa Cook, FSIS, Center for Learning. And this question is for the Pennsylvania program. What kind of education or training outreach do you do?

MS. KENNEDY: I work close with the Pennsylvania Department of Agriculture inspectors right now. Any type of ideas, we work together and then we go back to industry and also, Penn Ag Industry works with us. We get together with the industry people. We try to do, I think it's -- we certify everyone every two years, and then, we do recertification yearly.

DR. O'CONNOR: Bob O'Connor, Foster Farms, along the same lines, you've seen a decrease in the incidence of

positive farms, correct with your program, Pennsylvania?

MS. KENNEDY: Yes there is decrease.

DR. O'CONNOR: Have you studied that relative to the incidence of Enteritidis in human illness in Pennsylvania?

MS. KENNEDY: I don't get involved in that, so I don't have an answer to that.

MR. MAGWIRE: I could add, do you remember the one chart or table that had showed CDC numbers by region. You can see after they implemented their program and they started getting reductions on the farms that they -- reductions in the numbers -- there were reductions in the numbers also in human outbreaks then, in that region of the country.

DR. DEY: Any more questions? Apparently not. This concludes our session here. Please give our lecturers here a big hand.

(Applause.)

DR. DEY: We'll be having a break and come back here at 10 minutes to 4.

(A short recess was taken.)

BREEDERS, LAYERS, AND HATCHERY

DR. BAUER: We want to get on with the next session which is titled Breeders, Layers and Hatchery. And my name is Nate Bauer. I'm a scientific liaison with the

Office of Public Health Science and I network on a continuing basis with research scientists in academia and government that do -- conduct research on pre-harvest food safety issues, like *Salmonella*, *Campylobacter*, *E. coli* O157:H7 and other pathogens in livestock and poultry.

Our first speaker is Dr. Peter Holt, he's going to present Update and Review of *Salmonella* Enteritidis Vaccinations.

He's spent the past 17 years studying various aspects of poultry immunology, focusing primarily on the effect of stress on immunity in chickens. But also, devoting significant time and effort studying the elicitation of immunity in mucosal surfaces and developing vaccinations regimens to increase resistance to infection by *Salmonella* Enteritidis. He's authored or co-authored more than 85 scientific papers, as well as a number of book chapters and he works with the Egg Safety and Quality unit at Russell Research Center and here at Richard Russell Research Center.

UPDATE AND REVIEW OF *SALMONELLA* ENTERITIDIS VACCINATIONS

DR. HOLT: Thanks, Nate. Good afternoon, everyone. By the way, Copper Creek there's A-number one beer there. So, I highly recommend it if anybody has a chance to go.

One of the nice things about coming in and speaking later on in the afternoon, is a lot of the previous speakers have already given a lot of your preliminary data. So, I can kind of zoom through a lot of the early stuff. Nate asked me to talk about vaccination for *Salmonella* Enteritidis, what's currently known, and so that's where I'm going to go.

This is the bad guy, this is *Salmonella* Enteritidis. Everybody knows what kind of a problem it's created for the egg industry, and that it's been fighting it out with *Salmonella* Typhimurium for the top spot for quite a number of years. The number of outbreaks kind of peaked in the early '90s and then decreased, but it still maintained quite a high number, in the 40s, every year since the turn of the century.

Primarily where the outbreaks occur is in some kind of a institutional type of location, and so a lot of the intervention has been focused on trying to reduce those problems. Because what's happening is generally through some kind of abuse of the egg or egg product. So, the problem, the crux of the problem really is people are getting sick from *Salmonella*, and *Salmonella* many times is coming from poultry or poultry products. And as a result people are eating these contaminated products and getting sick.

Now, there are a number of solutions, the cheapest solution is to cook the food. That was brought up earlier and I heartily agree, but the whole premise is that you need to deliver as safe of a product to the consumer beforehand. So, you have to move on to less cheap alternative. And so you can institute irradiation and pasteurization. And there is a certain percentage of the market that does this. And it is growing. And finally, you can go even less cheap and that's where you start doing you interventions either on farm or during processing.

Now there has been a lot of industry work on preventing it, getting clean birds, chicks, et cetera, cleaning up the farm. And as was mentioned earlier, the work in Pennsylvania, they have been pretty successful in reducing the amount of SE that's causing problems in the food chain.

I think that another thing that is very important is and I would love to see more money being spent is education to the consumer. I think that's been way under funded and let's get people more involved in cooking the food.

I think what's been implemented quite successful also, is serving pasteurized eggs at institutional situations so that pulled eggs enter in.

And finally, there's vaccination. Now, what is

the function of vaccination? Vaccination is essentially supposed to mimic an infection. And when an organism such as SE infects a naive individual -- I know you're working. Oh, well. I'm challenged with the buttons, too. When it infects -- infects an individual it invades the tissues and replicates and disseminates out to the organs after a certain amount of time, the bird develops an immune response and will hopefully clear the organism. So, when this immunized individual then gets infected a second time it enters the host and hopefully the immune system will kick in and will block the ability of the organism to invade tissue and replicate and abrogate the infection. And this is where vaccination comes in, it's trying to mimic the infection.

Now, there are two broad categories of vaccines, your live and your inactivated. But there's also a number of permutations and combinations which I won't go into between the live and inactivated. Live are attenuated to reduce infectiveness both for the host and for humans. It is administered in the water, feed, and also, as an aerosol. Inactivated are generally killed organisms that are resuspended into generally a water and oral emulsion and injected into the bird.

The live vaccines, just as it is for anything, there's pros and cons for it. For pros, it causes infection so the infection is more closely resembling am organism, it

develops -- the bird develops both the cellular and humoral immune response and also with the humoral immune response it develops an immunity in the gut as well. It spreads within the group, so-called herd immunity.

Now, as far as time goes it also, this spread within the group can possibly be a negative. Since, the longer the organism stays within a group of birds the more chance it has of increasing its virulence. And because it's a live organism, storage and viability comes into play as being a problem.

There are currently three live vaccines that are being -- that are marketed in the United States. Megan Vac from Megan Health in St. Louis. Salmune from Biomune and Poulvac ST from Fort Dodge Animal Health.

Now, we did some work about four or five years ago. Looking -- using Megan Vac as the live vaccine and using our molt model as the way to study it. Now, for those of you that aren't familiar, birds that are molted via feed withdrawl are extremely susceptible to an SE infection. And what we developed is a transmission model where we have rows of 11 birds per row that are molted. And then if they form molt we infect just the center bird and then follow the transmission of the organism down the line. So, we set up two groups of birds, ones that we vaccinated and ones that weren't.

Now, this is three day post challenge. The center bird was infected, you can see that with the non-vaccinated birds, we already had five birds that were positive as opposed to just one with the vaccinated. By day ten post-challenge, there were 15 out of 20 of the non-vaccinated birds as opposed to just 9 out of 20 of the vaccinated. But what you pay attention to is the amount of SE that's being shed, 10 to the 3, 10 to the 5th, as opposed to 9. And nine means that the birds on direct plating we could not find SE. This was after enrichment. So the amount of SE that's being shed is much, much reduced.

I think even more telling though is what did you find inside the bird as far as internal organs. The ovary percent positive was completely negative for the vaccinated bird, whereas about 40 percent positive with the non-vaccinated bird.

What about the bacterins? Again, you have the pretty much the same players as with the live vaccine. You have Biomune has their larimume SE. Lowman Animal Health, which is formerly Maine Biological Laboratory has Inactivat SE4 and Fort Dodge Animal Health has their Poulvac SE.

The pros and cons of using a bacterin -- pros, they're very inexpensive and they protect reasonably well. Cons, you do have some danger via the injector. You know, you inject yourself with some of these vaccines you can get

very, very serious inflammation. It's fairly labor intensive, you have to handle the birds and inject them. And generally you have to give multiple injections. And finally, the protection is not complete, it is partial.

However, there's been, you know, a multitude of studies that have shown that using a bacterins provides a lot of protection -- reduced clinical signs, shedding, organ positivity, but I think the most telling thing is, you get the number of positive eggs that the bird is producing is significantly decreased. Now, I have down here, note that the key word is reduced. You can't use that as kind of your magic bullet. You have to use vaccination as an overall management tool along with maintaining the clean integrity of the house as well.

This was a study that was done by Dr. Richard Gast, who's our research leader for the Egg Safety and Quality Research Unit. And I want you to ignore this molted hens up here. The individual that typed up this figure, I'd fire him if I didn't want to keep my job for a little while longer. I'm so into molting for some reason, I had to put that in there. But as you can see with the studies, vaccination had a very significant effect on extra intestinal dissemination to organs, both the spleen and the ovaries and oviducts, significant reduction in the positivity.

I think what's even more telling though is the work that was done over in England. In 1997, they instituted the Lyon Code of Practice where they set up standards for the egg industry over there that dealt with eggs, egg freshness, sell by date, the hygiene standards were improved. And they also had mandatory *Salmonella* vaccination. And in those four or five years the human salmonellosis was cut in half and they tout that vaccination was the primary doer for that. So vaccination can be very, very important.

Now, go back to that original -- that slide a couple slides back. The key word is reduced and not clear. Our work has been involved in looking at trying to improve the vaccines, the killed vaccines, a little bit more. One of the things that we noticed is that if you vaccinate with oil emulsions and change the emulsions with the second vaccination, you get a much, much improved boosting effect, than if the birds receive the same emulsion both times. The emulsions we're working with, just as the commercial ones, they're water and oil. And all the ingredients are food grade or cosmetic grade, except for the SE that we put into it.

We also evaluated the vaccination regimen by taking the serum from the bird and separating the IgG subpopulations in the serum into -- into subpopulations

using an iron chelate column. And then, running ELISA titers on the subpopulation and also avidity indices. And this chromatograph just shows how the serum is broken down into your different subpopulations of IGG. Now, the ELISA titer is the last solution of the serum to give a reading of 1.5 times the negative control sera.

The avidity is the tightness of fit of the antibody to the antigen. And it's been shown in a number of studies that the higher the avidity antibodies the more protection you get. And actually, this graph -- slide kind of gives you an indication of just what we're talking about. If you have your little Shih-Tzu dog here, if a burglar comes in the house, they will offer just a little bit of protection. They're going to yap at the burglar and create an alarm, maybe puddle on the floor, create a slippery situation, but not much more. However, you get you higher avidity antibodies like the German Shepherd, it's going to clamp down, it's going to hang onto the burglar, and that's what you want, you want the big dog type of antibodies to be developed.

Now, when we compared our emulsions with the commercial and these guys got the same commercial emulsion, both vaccinations. They gave a good response, 12, 8, that would 12,800 titers, 25-6. But when you use an emulsion and change it with the boosting emulsion, you can see that you

get quite a substantially higher ELISA titer in those guys, in all the different emulsions we worked with. With the relative avidity index this is using 6M urea, this is going to be your low avidity, your Shih-Tzu type of antibodies that are in there. And you see that overall there's not a lot of difference. Between 1.2-1.5 times greater with the -- using the different vaccines.

However, when you look at the -- the big dog type of antibodies, you see a substantially increased amount of high avidity antibodies. Generally, it's anywhere from 1.5 to 2 higher.

One of the things that I want to show is, and the reason I have emulsion C highlighted is that one of the controls that you use in vaccination is you'll have a control where you vaccinate with just the emulsion without antigen, just to see what effect the emulsion has. And something that we discovered early on is the emulsions can have a very substantial effect on the ability of the birds to be primed. And what we did was is we set up an experiment where the birds were primed with the emulsions without antigens and then we come back with our -- our standard southeast poultry bacterin and looked at the response.

And what we found was -- and these were all birds that received emulsions without the SE antigen in them --

the birds that received the same emulsion twice, which are SEPRL 1 emulsion, it gave good ELISA titer, 25,600. But take a look at emulsion C, these guys really -- these guys were primed with -- with -- to give a very, very strong response. So, why this is happening? We really don't know, we're in the process of trying to figure out just what the mechanisms are of this. And that is the direction that we're going in right now.

And with that, that's it. Thank you.

(Applause.)

DR. BAUER: Thanks, Pete. Our next speaker is Dr. John Glisson. He's going to be making a presentation for Dr. Charles Hofacre, Chuck Hofacre is in Istanbul, Turkey at the World Veterinary Poultry Congress.

Dr. John Glisson is involved in various aspects of the teaching, research and service functions of the Poultry Diagnostic and Research Center at the University of Georgia.

His research focus has been on bacterial diseases of commercial poultry. Dr. Glisson teaches in the master of avian medicine program and served the poultry industry through field diagnostics and disease prevention for several years as a poultry clinician. He currently serves as the Director of the Poultry Diagnostic and Research Center and is head of the Department of Population Health, College of Veterinarian Medicine, University of Georgia, Athens,

Georgia.

Dr. Glisson is presenting this paper, as I stated previously, for Chuck Hofacre.

SALMONELLA REDUCTION IN POULTRY PRODUCTION

DR. GLISSON: Thank you, very much. It's not -- I'm not accustomed to giving papers for other people, particularly people that are on vacation in Turkey.

Chuck would argue that he is not on vacation, he is representing Triple A-P [AAAP] at the World Poultry Congress. But I'll see how this goes.

One of the first things that I want to try to do is help the folks in here that are not poultry people try understand what we poultry people deal with every day. And I thought it would be good just to go through this. For you poultry people, you can nod off.

Integrated poultry industry is very difficult for people who have not been involved in it. There's many layers in the industry. We talk about generations of birds. And the companies that are out there producing the chicken that you eat or the turkeys that you eat or the eggs you eat generally don't have more then a couple of generations of birds.

The broilers as we call them, there's -- this country is producing about eight billion of those. Those come from 75 million broiler breeders. So, all the poultry

countries in the U.S. take all their breeders together, is about 75 million.

The turkeys that you eat, there's about 270 million of those. They're coming from about 3-1/3 million turkey breeders. There's a huge multiplication effect here. The commercial layers that are laying the eggs that we all eat come from about 2 million commercial layer parents.

So, you can see when you start thinking about *Salmonella* control at different levels within the industry, that controlling it at this level is very different than trying to control it at this level, just simply because of the number of animals involved, the number of facilities and sites involved.

Now, if you just concentrate on the broiler industry, which is the largest segment of our industry, it becomes I think worth the time to just think about the money that's involved in some of these animals. Breeder pullets are -- are the females that we use that are going to become the breeders to produce the eggs that we hatch to produce the broilers. That take about 20 weeks. And then those birds are physically moved from there to a breeder layer farm. So, the number in the U.S. is about 75 million breeder pullets, about 73 million breeders. And this is the approximate value of each of those birds, about $3.50 for a breeder pullet, about $9.00 for a breeder female. That's

including the males that go along with them. About 10 percent of the flock is males, by the way.

Now, they're going to produce eggs. Those eggs, we'll use a generous figure and say they're going to produce about 160 eggs per hen and the value of each of those eggs is about 10 cents. Those eggs will be hatched in 21 days and we produce about 8.8 billion chicks that are going to be worth about 18 cents at the time they hatch. Then those are taken to a broiler farm from the hatchery. We'll use 49 days, there's a lot of variation, depending on what size bird you're going to raise. Then those are going to go to the processing plant and have more or less a value of 60 cents a pound.

What does all this mean? If you think about investing money to reduce *Salmonella*, it's very difficult to invest a dollar in that when it's only worth a dime. Very difficult to invest a nickel in an 18 cent chick. So -- so there's some economics involved here that have to guide us a little bit as we think about where the poultry industry is going to invest its money. Because this *Salmonella* reduction program at the end of the day is an investment for the company in the quality of its product. So it's very -- I think very important for us to understand where we can afford to spend money, where the investment is highest.

Chuck has put this talk together to focus on that

concept. And he has here several critical factors to reduce *Salmonella*. And we're going to go through several of these some more thoroughly than others. First one we're going to talk about is the breeder supplier. This is -- I showed you the integrated poultry industry, the way it's structured. Above that are the genetics companies, where all the genetic supply comes from that these integrated companies then use to multiply to make the broilers or the turkeys or the layers.

There are a number of primary breeding companies in this country and they're all basically built the same way. Some differences, but basically this is the structure. At the very top there are elite animals those are single sire matings, highly selected and those produce what we call great grandparents and then grandparents and then parent stock. Parent stock is what the poultry companies buy. They buy day old parent stock. They raise those out to produce the animals that we eat. Now this is shaped like a pyramid for a good reason. Actually the pyramid, the shape of it would be even more exaggerated than that in reality. One elite male -- Bruce can probably help me -- how many broilers that's going -- broiler progeny he's going to have once it gets down through the system. But it's probably maybe several hundred thousand maybe, I don't know. But the multiplying effect is very, very large.

Again, this is important to understand, if you have an organism that is being transmitted vertically, it's being transmitted vertically all the way through this system. All the way through. We all know that a lot of what we're dealing with with *Salmonella* is various forms of vertical transmission. So, this picture carry it in your mind as we go along.

Biosecurity practices would be the next topic. We could spend hours talking about this and I'm not going to do that. But the important thing here to realize is *Salmonella* can enter our system in a number of different ways, vertical transmission is simply one of those. But anywhere that you have contact with anything else that's alive, that's very, very important. Humans, do you believe that humans can transmit *Salmonella* to chickens. We don't talk about that much, but if you go as a veterinarian, you're going to help one of the primary breeders and you're going to go into their elite birds or their great grandparents, they're going to do a *Salmonella* swabs on you before you come because they know that humans transmit *Salmonella* to chickens.

So, we have to think about those things -- humans on the farm. Other animals are very, very important. Because chickens are certainly not unique in that they carry *Salmonella*.

Feed, there's been a lot of discussion about that.

And again, this is a very, very big topic and we're not going to spend too much time on that. There are companies that have invested quite a bit of money in reducing *Salmonella* through their feeding program. Dr. Gonder talked about what they do at Goldsboro Milling. We know that there are ingredients that have a lot higher risk than others. That's all well known. There are a number of different ways to decontaminate feed from using additives in the feed that kill microbial organisms to the extreme of actually cooking the feed and various variations on that theme, like pelleting. One of the real problems is recontamination of the feed once it's manufactured. Feed delivery is -- can be a real problem in that regard. And then of course monitoring the process.

This is a very expensive process, is having clean feed. Remember the poultry industry is buying the same corn and soybeans as everybody else, and the same raw materials as everybody else. And it's not *Salmonella* free.

This is an interesting thing, just again, as a point of perspective, what's in the chicken intestinal tract. And this John Maurer and Margie Lee do this type of work where they look at the microbial composition in the intestinal tract, making 16S clone libraries. And this is a composite of what's in a broiler's intestinal tract over the 49 day period. The interesting thing is, you can see that a

lot of *Lactobacillus* in the ileum. A lot of clostridia in the cecum -- where's the *Salmonella*? *Salmonella* is in this tiny little fraction right here, which is one of these lines right here. There's a lot of stuff going on in there, inside the intestinal tract of the chicken. And *Salmonella* is certainly a very, very minor component of what's in there.

Also, it's been mentioned competitive exclusion. We're very, very frustrated in this country by our inability to use competitive exclusion. The poultry industry wants very badly to use competitive exclusion and there's all sorts of legalities that keep that from happening. I hope that can be solved.

I think Eric said it's been over 30 years since Nurmi showed this work. And a lot of people spend a lot of time working on this. We know how susceptible newly hatched chicks are, extremely susceptible to colonization. And we know that we can reduce that susceptibility tremendously, very significantly with competitive exclusion.

Vaccination is another very important tool that we have. I'm going to back up one slide and point out a couple words on this slide I didn't point out, that I thought Chuck chose very well. Two important words -- achievable and reduction. Achievable strategies are something I think Bruce spent a lot of time talking about. What are the

things that we know we can do, what of the things we know will have an impact to reduce the *Salmonella*, and CE is one of those.

Another is vaccination. I've spent a lot of my career working on bacterial diseases other than *Salmonella*. And there's a real general thing you can say about birds, is that they don't respond well to bacterial vaccines. You can almost say that about any animal including humans. The history of successful bacterial vaccines is pretty poor. But we do know how some of these things work. Live vaccines stimulate mostly secretory, humoral and cell mediated response. And because of that, they have been used primarily as what we call priming vaccines, a vaccine that gets the birds immune system prepared for an inactivated vaccine. And then, the inactivated vaccines give a good humoral response.

Basically the way it works in our industry is the live vaccines will be given very early in the pullet stage, followed by two doses of inactivated vaccines separated by at least four weeks. That protects the pullets and also provides maternal antibodies to the next generation, which is a very important concept.

Now, Chuck makes a comment there at the bottom that vaccination will take at least a year before you seen an effect. Now, one of the things that I want to try to

explain about that, say today a company decides to change its vaccination program in its breeder pullets. These breeder pullets are -- they're starting new flocks every week and have flocks that are being killed at the end of their lay cycle every week. It takes a year to replace the breeding stock in the poultry company. So, if you change the vaccination program today, it takes you a year to get all your chickens vaccinated.

Now that's not what Chuck's talking about. What Chuck is talking about it takes an additional year for you to really begin to see the effect because we're talking about a reduction program and a reduction program is a reduction all through the system. And I'll talk about that just a little more as I go along.

So, when you get into trouble at the processing plant with your *Salmonella* counts, people all of sudden get excited about vaccinating breeders. That's not a short -- short term solution, that's very long term.

This is a study that was done in the UK in layers. And what they wanted to do is look at the comp using CE and vaccination together. And you can see basically how they set this up. They had controls and then they had a group that got competitive exclusion at day old and 14 weeks. They had a group that got *Salmonella* Enteritidis bacterin at 10 and 14 weeks. And then you had a group that got CE plus

bacterin. And then these birds were challenged. And what you see is how many -- you see the number of *Salmonella* -- the number of birds shedding *Salmonella* either 7 days or 14 days post-challenge here. And you can see that it -- you don't see much on the seven day, but on the 14 day post-challenge you can see a big effect. And the interesting thing and many companies have found this out on their own, private companies that do their own work, that there is either a synergistic or an additive effect, probably just additive effect to using CE and vaccination. So we got two tools right there that can be used in an economic way to achieve *Salmonella* reduction.

Here's one we're going to talk about that a lot of people probably don't want to talk about. Can you use antibiotics for *Salmonella* reduction? The answer is yes. It's been done. And I'm not going to go into a lot of details, but companies have done this on their own primarily. But primary breeders have used antibiotics to clean up *Salmonella* out of breeder flocks.

Strategic use of antimicrobial followed by replacing the intestinal flora with CE. Now this is done -- can be done at several different times. But usually it's done before the breeder pullets are moved to the layer facilities. Try to get them clean before they go to the layer facilities. What many of these companies have found,

that failure to replace the flora after the antimicrobial therapy actually can make the birds more susceptible to *Salmonella*. So those these two things need to go together.

This is data from a company that did that and they -- they did just that. They used an antibiotic and CE on the pullets at the end of their growing life, instituted really good rodent control at the breeder house. They moved those to the farm and this is data from 5 million broilers off of breeders that they did that to and 5 million that they did not do that to. This fluff is fluff in the hatchery. They didn't see a lot of difference there. But what they did see is a fairly significant reduction at processing, which is what we're trying to do. So, again this is another example of things that can be done to reduce *Salmonella* as it spreads vertically through the food chain.

So what are we talking about here? We're talking about that breeder vaccination reduces the number of birds positive and reduces the number of *Salmonella* in the intestine. And we think that in breeders that's probably a very good thing for the overall situation of *Salmonella* in the company. We know that live vaccine stimulates CMI and inactivated vaccines give a good humoral response.

What we don't know real clearly is will live vaccination followed by inactivated vaccination protect both the breeders and the broilers? So Chuck has done some work

over at PDRC to try to figure this out. This is a pretty big study that he's worked on for quite sometime in cooperation with a company in this area. So these breeders here had -- this breeder company had these *Salmonella*s in their system -- Hadar, Kentucky, Heidelberg, Enteritidis -- and essentially in this experiment what happened, they took Hadar, Kentucky and Heidelberg and made an autogenous bacterin. And used this protocol on these -- in this company. Gave live *Salmonella* Typhimurium vaccine, like Pete was talking about, one day, two weeks and six weeks, followed by killed autogenous at ten weeks and 18 weeks containing these three serotypes.

Those birds were brought into production -- again this is a commercial flock. Other flocks did not get this vaccination and were used as controls. The eggs were hatched at PDRC across the street and -- and then the groups were challenged and they were challenged, different challenge groups with all four of these organisms of course separately. And then we looked at the results. And the challenge was done by a fairly real life situation using seeder chicks, put them in at one day of age and let them mix with the other birds. And then the ceca were cultured at 21 days of age plus or minus and also enumerated. And this is what you see, there's no *Salmonella* Enteritidis in the bacterin and you see no reduction there.

But you can see a reduction with all the others. And the same with the enumeration. What you'll find out, none of these difference here are statistically different. And that's typically what you see in a controlled situation like this. This was done four times. What does that mean? That's -- I think that's what we see in the field also. The reductions are small but it's cumulative. And that's why Chuck says after you get everything vaccinated, it takes about a year to really begin to see the effect. Because you're reducing the *Salmonella* load all the way through the system -- the hatchery, the broiler farms, the processing plant. But after about a year of this, you begin to see significant reduction.

So, let me just finish up and summarize here. We know that live and killed vaccination of commercial broiler breeders with *Salmonella* contributes some protection by maternal antibodies. Decreases the number of positive birds, which decreases the *Salmonella* in the ceca. It may reduce *Salmonella* incidence in the processing plant over a period of time. Vaccination of broiler breeders may be an ideal strategy when *Salmonella* levels are high in the processing plant. However, this program may take a year to see the beneficial effects.

It's been shown that the *Salmonella* isolated from a chick at one day of age will most likely be the

predominate strain throughout its life. Number of people have shown that. We need to look at all inputs onto a farm and minimize the chance that they will bring in *Salmonella*, and make the bird more resistant to *Salmonella* colonization by CE and/or live vaccine and/or inactivated vaccines and/or others.

No silver bullet, no magic potion, no single remedy. Thank you very much.

(Applause.)

DR. BAUER: Our next speaker is Dr. J. Stan Bailey. He's lead scientist and research microbiologist for USDA ARS right here in Athens. He's responsible for research directed toward monitoring, controlling, reducing and ultimately eliminating contamination of live poultry by human enteric pathogens. During his scientific career, Dr. Bailey's authored or co-authored over five times ten to the two scientific publications in the area of food microbiology concentrating on controlling *Salmonella* in poultry production and processing, *Salmonella* methodology, Listeria methodology and rapid methods of identification.

Dr. Bailey's recognized nationally and internationally and has received numerous awards including the 2002 USDA ARS outstanding senior research scientist of the year award. Dr. Bailey was elected secretary of the International Association of Food Protection in 2005 and

Stan will become president in 2009, thanks a lot.

VACCINATION OF BROILER BREEDERS AGAINST PREDOMINATE *SALMONELLA* SEROTYPES

DR. BAILEY: Thank you, Nate. And I too want to welcome you to the Russell Center. This is one of the bigger groups we've had here in a long time. It's very good to see you here.

Several of my colleagues and I have been working in this area for a long time. This is my 32nd year working here at the Russell Center. And the whole time has been trying to work on *Salmonella* in poultry. And several of us made a conscious decision in 1985, that -- prior to that we worked in the processing plants a great deal. We'd worked with feed and other areas and we made a conscious decision that if we were ultimately going to ever significantly impact *Salmonella* in poultry that we needed to move back to the farm level. So we started directing our research at the farm level at that time. And so, I'm going to be giving a couple of talks in the next couple of days over some various aspects. But today I wanted to share with you some of the work we've done with broilers and broiler breeders and vaccination.

First I wanted to acknowledge several people. I am not an immunologist. But I work with several people on this project. First was a graduate student of mine Ariel

Rolon, who was a brilliant man -- young man from Bolivia who did the primary bulk of this work. We worked very closely with Chuck Hofacre, whose work you just heard talked about. Peter Holt, who gave a talk before that and Jeanna Wilson at the University of Georgia.

As a bit of a background, the history of vaccination of poultry flocks against different *Salmonella* serovars can be traced to the layer flocks, where oil-emulsion vaccines against *Salmonella* Enteritidis have been used for many years with some success in diminishing transmission of this serovar to the eggs.

Vaccination of flocks undergoing molt has been found to be useful.

And breeders have also been vaccinated in an effort to prevent vertical transmission and increase resistance to early exposure.

More so in other countries than here early on, we saw a lot of use of vaccination to control *Salmonella gallinarum* and *pullorum*, because it's a bigger issue in developing countries, in Latin America particularly. We've also seen a great deal of vaccination work as a reported earlier with *Salmonella* Enteritidis in the UK particularly.

These reports however, are based on statistical comparisons of vaccinated and non-vaccinated flocks. No studies correlating vaccination protocols and resistance to

challenge models had been reported at the time we did this work. There is also few reports on the interaction of passive immunity in live vaccines during the first weeks of life.

In addition given the use of the live vaccine at day of age on chicks when maternal antibodies, the potential of passive immunity interference on early live vaccine effects need to be further assessed.

So what I want to do in the next series of slides is -- is cover a lot of work that Ariel did in his dissertation and those three papers are all at the Journal and will be out soon. And I obviously can't cover all that. But I think it's some very interesting and important work in this area and will contribute a great deal. So what we want to do is give a brief summary of the findings and if you want more complete information on these just feel free to contact me and I'll be happy to provide you with that.

So the first objective was to evaluate humoral and mucosal immune response in broiler breeders under the different vaccination protocols that I'll share with you in a minute. So I want to here this -- this first objective was looking at the humoral and IgA response in these birds.

And then secondly, we wanted to assess the effectiveness of the different vaccination protocols, using both live and killed autogenous vaccines, on the protection

of broiler breeders during rearing, under simulated industry conditions.

So what were the four treatments that we used? They were applied to breeders during grow out, using a commercially available *Salmonella* Typhimurium live vaccine. So the live vaccine the we'll talk about here is a *Salmonella* Typhimurium vaccine. And an autogenous killed vaccine containing three *Salmonella* serovars, a group D1 a B and a C2 prepared by a commercial vaccine company.

Treatments were as follows:

Our controls received no vaccinations, the next treatment we gave two killed vaccine patient one at 77 days or 11 weeks and one at 18 weeks or 126 days. So treatment two received two killed vaccines. Treatment three got two live and two killed. The two lives at one day and three weeks. And the two kills again at 11 weeks and 18 weeks. And then the fourth treatment or third actual treatment was three lives and one killed as you see there. I'm not -- don't want you to even try to interpret all this because it's just too much and there's about 100 of these slides that we generated out of all the data.

But we looked at gut immune response and we did it against *Salmonella* Enteritidis LPS as a captured antigen and also against *Salmonella* Typhimurium.

And again, we looked at the gut, we looked at the

serum immune response and we also looked at the chicken serum. So, we have all of that data that I'll be happy to share with you but it was just too much for the time frame here.

So, what did we find in this study where we were looking at immunological response? Not looking at the *Salmonella* itself. We found using optical densities to measure the immunological response were consistently stronger on the *Salmonella* Typhimurium LPS than they were on the *Salmonella* Enteritidis LPS. We found IgA serum from the crop and the gut lavages as well as the IgG of the crop lavage gave short-lived peaks after the first killed vaccine only and that didn't last a particularly long time.

We got a strong gut lavage IgG after the first live and both killed vaccine events were observed, the killed response lasted much longer than the live. The serum IgG responses were observed after killed vaccine events and lasted throughout the 40 weeks.

Chick serum and egg yolk IgA were negligible and IgG comparable among all treatments throughout time. These results showed that killed antigen is vital in eliciting an adequate IgG response in serum and in gut. Live vaccination with the Aro-A mutant ST vaccine enhanced the gut IgG and possibly aids in conferring adequate immunity during the breeder's first weeks of life.

So then, we moved on in the same model system and went beyond what the immunological response was. And we measured what was the response in terms of *Salmonella* in the hen and in the offspring. We evaluated the resistance of *Salmonella* challenge of vaccinated breeders and their day old progeny using a multiple *Salmonella* strain model. We evaluated the effectiveness of competitive exclusion treatment alone or in combination with vaccines of breeders to reduce the *Salmonella* in the progeny.

So, what did we do? Broiler breeders were challenged using a three marker strain -- three marker strains of *Salmonella* and at days 21, 42, 77, 119, and 154 a sub-sample of breeders were challenged with those three *Salmonella* listed there, Enteritidis, Typhimurium and Thompson.

At weeks 29, 34 and 40 of the breeders' age chicks from each of the breeder treatment groups were hatched. Half of the progeny of each treatment was administered competitive exclusion, which as the version of competitive exclusion that we had developed here at this laboratory. That I'll talk a little bit more about tomorrow. Chicks were challenged with the three strains of *Salmonella* and assessed for colonization after one week. Additionally, counts two weeks after challenge were determined at weeks 34 and 40 of the breeders age.

Just briefly, you can see that the very little response in the level of *Salmonella* with just the killed, the purple. It was almost identical to the controls. But where there was a combination of kill and live vaccine, we did see a reduction -- a significant reduction at three and six weeks of age, where we used a combination of the live and the killed vaccine. This is in the breeders now, this is not in broilers -- this is in the breeders. This had diminished by 10 weeks of age at which time they were getting some additional treatments and we see a variation. But overall even out at 22 weeks of age all three of the treatments, those that just got the two killed and the combinations in the breeder hens themselves, we did see a diminished level of *Salmonella*, significantly diminished level.

If we look at the progeny though, we can see very little measurable effect. At 40 weeks of age we do see some reduction in comparison to the control. But as was just talked about in the previous talk, it's not dramatic, it's small reductions and they add up over time.

If we look at the progeny post-challenge with and without the competitive exclusion product, we see a far more dramatic effect. Which is very consistent with what we've seen for many years with competitive exclusion. You see the light purple bar is the competitive exclusion. And from 34

weeks on, you see a very dramatic difference in the level of *Salmonella* in the breeder -- in the progeny from the breeders in comparison to those that did not receive the competitive exclusion.

And from this section of the work we concluded that breeders *Salmonella* count showed significant difference between live vaccinates and non-vaccinates at three and six weeks of challenge showing the commercially available Aro-A *Salmonella* Typhimurium vaccine confers some early protection.

By ten weeks there were not discernible difference in *Salmonella* level in challenged and control chicks, indicating protection by one day and three weeks. The live vaccines had diminished at this time. All programs reduced *Salmonella* counts compared to controls at 22 weeks.

Chick *Salmonella* counts showed little consistency between breeder vaccine treatments.

No clear differences were observed in susceptibility of chicks from vaccinated and control breeders. Passive immunity did not show consistent reduction of challenged chick *Salmonella* counts. So, the immunity that they brought over as they hatched out from the dams or from the mother hens, did not give a significant reduction.

Treatment with MSC reduced *Salmonella* counts

consistently regardless of breeder vaccination treatment or breeder age. These results show that live vaccination with ARO-A *Salmonella* Typhimurium vaccination decreases *Salmonella* counts during the first six weeks of age of the breeders as do all programs by 22 weeks of age. And that competitive exclusion is the most effective treatment in reducing progeny *Salmonella* counts.

Our third objective was to evaluate the gut humoral immune response of hatchlings from hyper-immunized breeders and challenged with *Salmonella* at day 3, 13, and 34 of age. And to assess the effectiveness of early vaccination with an ARO-A mutant live *Salmonella* Typhimurium vaccine in protecting hatchlings from hyper-immunized breeders against *Salmonella* challenge at days 3, 13, and 34 of age.

Basically what we did in this study -- and this study was actually carried out in Bolivia in commercial situation -- was to take a commercially prepared autogenous bacterin and a Poulvac *Salmonella* Enteritidis serovar Enteritidis bacterin and non-vaccinated controls. And vaccinate at 40 and 43 weeks of age and collect the eggs for incubation at weeks 46 of age.

Again, just briefly some of the data you see that you see gets very difficult to -- you see non-statistical difference in reduction in counts. But you see numerically

somewhat lower. We did with the live *Salmonella* Typhimurium see a statistical difference in the first two treatment days. If we looked at the heart, liver, spleen counts for what was internalized, we see the same type of situation. The live gave a pretty dramatic reduction at day 3 and a statistical difference at 13, pretty dramatic but not statistical at day 34.

So, the conclusions from this -- this part of the study were maternal IgG is important up to 13 days. Higher optical densities are obtained on the LPS than on the SE LPS, which was consistent is what we saw in the first study. The live ARO-A vaccine enhanced IgG up to 34 days with titers starting to decrease at this time. Effect better measured when it's assayed on ST LPS. The diminished protective effect of ARO-A vaccine after 34 days probably indicates the needs for another vaccination to sustain protection after this age, that may important in breeder management.

There were no maternal IgG as expected. The short IgA peak measured at 13 but not 34 days indicates that the gut IgA might peak as a response to primary exposure to antigen, other isotypes being more prevalent thereafter. No interference -- and this is a fairly significant finding, we think -- no interference of maternal antibody on the live vaccinations. Vaccine's ability to stimulate IgA was

demonstrated. And that had not really been reported on the literature before. And for those that were worried about the carry-over effect of passive immunity on the live vaccine we felt like that was a very important observation.

Live vaccines and maternal antibodies decrease overall *Salmonella* counts about 1 log and .3 log for cecal and internal organ samples respectively. Total counts diminish with age. This has been previously reported. Live vaccine and maternal antibodies decreases overall *Salmonella* counts about 1 log-- and then finally although vaccine decreases overall bacterial load, *Salmonella* still present at considerable numbers highlights the importance of vaccine as a complementary tool in controlling *Salmonella* in poultry and not as a substitute for integral biosecurity programs.

So where are we with all this? What does all this mean? I reported in a very fast manner trying to go through a lot of data. Currently there's three live *Salmonella* Typhimurium mutants on the market. Most people who vaccinate breeders -- and it's an increasing number of you in this room that I know for sure are doing it -- most people who vaccinate breeders are using either two of three live and one or two killed. Vaccination with live and autogenous killed has been shown to give incremental reductions in *Salmonella* which over time will carry through the processing plant.

And I wanted to take just a minute to talk about this last bullet point. APHIS needs to revise its autogenous vaccine rules. The rules that were written for autogenous vaccine have been in effect for 20 or 30 years or more. And they don't really apply to the situation we're dealing with with autogenous killed vaccines for *Salmonella* in chickens. Because if you have something that's effective and you reduce those particular strains of *Salmonella* your autogenous vaccine was against, within a year you have to quit using that vaccine. That doesn't make any sense. You can't combine an autogenous vaccine with a commercial live vaccine, that's against the rules. There's just a lot of issues with autogenous vaccines that need to be readdressed. And I would encourage those of you who have any influence to speak with and continue to carry forth this idea that APHIS needs to readdress those rules. We're not talking about doing anything that would increase any -- from my perspective -- any discernible increase in public health risk or anything. But I think it's an important issue that if we want to be able to use this tool that's one of many that we need to have available to us, then we need to bring the rules up into the current date.

And my final thoughts. *Salmonella* is often pervasive in the poultry environment, including breeder farms and the growout farms. And it is likely that no one

intervention will completely control all *Salmonella*. I think it's more than likely. I think that we can safely say that's a fact. And multiple intervention approach including vaccination, competitive exclusion, biosecurity, insect and rodent management and moisture management will be needed to achieve significant on-farm control of *Salmonella*.

And that's all I have and thank you very much.

(Applause.)

DR. BAUER: Okay, our last talk today is by a scientist who has seen it all and done it all. He is a research microbiologist right here in Athens at the Poultry Microbiological Safety Research Unit. And I just want to say a few things that kind of sum up his career.

Dr. Cox has seven issued patents which have been licensed by commercial companies. He has since 1971 worked for ARS and authored or co-authored over 700 publications, 450 of those publications have been in the last 15 years. He was the ARS distinguished senior scientist of the year 2003, Dr. Richard Gast also made a comment about Dr. Cox saying that he always had about 15 collaborations going on with about 20 people at the same time.

And last Dr. Cox, has been -- is going to be inducted in the ARS Hall of Fame. Please join me in welcoming Dr. Nelson A. Cox.

(Applause.)

ROLE OF BREEDERS, EGGS, AND HATCHERY IN TRANSMISSION OF *SALMONELLA* IN THE BROILER INDUSTRY

DR. COX: Thank you very much. And welcome to my house. I got here June 13, 1971. And since that time I've had the same phone number and the same address. And those of you that don't know that haven't contacted me enough in the 30 -- 34 years. And I got to tell you, when we came here, there was only 60 people working in the building from the area director down to the janitor. And at that time they told us they were going to put moon rocks on the seventh floor. And they made some adjustments to the various rooms so we would have moon rocks. Well, after 34 years later we don't have moon rocks in this building, I guess we got the next best thing on the seventh floor, that's FSIS, right.

Anyway, I got to tell you all if I wasn't giving this talk, if I wasn't giving this talk I wouldn't be here, because this is interfering with my happy hour. And I grew up in south Louisiana somewhere between New Orleans and Baton Rouge in a little town call Napoleanville, named after Bonaparte, and I told people for many years that the bars I drank in in New Orleans, you didn't dare throw your cigarette on the floor. And they'd say why, Nelson, is it a nice place. I'd say no, you don't want to burn somebody's face.

(Laughter.)

DR. COX: I appreciate Nate inviting me to give this talk. I didn't want to talk about this. I worked on *Campylobacter* now and I wanted to talk about *Campylobacter*, but he insisted I talk about this because it's a *Salmonella* meeting. So, I said, you know, Nate, the stuff I'm going to be talking about is so old that the reprints are yellow. And Beth Krushinskie always says that I never talk about anything new. But Beth this stuff is so old it might be new to some of you younger people. And I guarantee you, Eric Gonder has all of these yellow reprints, I've been to his office. At the break I went up to Eric Gonder, and I said, Eric why don't you tell what you really think. I love him, I can't believe he's not eligible for retirement. You usually have to get eligible for retirement before you give that kind of talk.

So I'm going to talk about chicken science and I got this from a pretty good source. They tell me that when the scientists at NASA are sitting around working on a difficult problem and they can't come up with a solution, they're scratching their head, and they can't come up with a solution, one of them will look at the others and say, come on guys let's solve this problem, after all this isn't chicken science, right. So, we -- so some of us who have PhDs in poultry science and have been doing this for 34

years, we're sort of proud of what we do. Some of the folks in the audience, I went to graduate school with and hadn't seen some in many, many years. And be honest with you I haven't seen this many people in one pile here at Russell Research Center since we had a recent fire drill.

We no longer have a cafeteria so, you know, we don't have a gathering like this. So, it's kind of nice to see this many people. So, when my presentation is over -- I don't have any handouts. And if any of you are interested in this stuff that's old, just give me your business card and I'd be more then happy to send you reprints. And if you're interested in some other area whether it's *Campylobacter* or competitive exclusion or feed or you name it just write that on the back of your business card and give it to me. With 700 publications, trust me I've touched on a little bit of everything. In fact we worked with Eric Gonder and the turkey people for awhile. And the competitive exclusion I think dropped the contamination I think from 47 percent down to 3 percent in some of the flocks. So we've done an assortment of things and with -- with breeders, with broilers. My dissertation was on table eggs. So, I sort have gone the whole gamut.

So let me kind of get started here, to begin with there's nothing new. The people knew 80 years ago in the poultry industry or more that *Salmonella* was in the

hatchery. When the *pullorum* and the *gallinarum* was killing the chickens before all of us in this room were born, the poultry industry knew that they could find these organisms in the hatchery. So there was nothing really new about going in the hatchery and finding *Salmonella*.

In 1985, we started working with live birds, so, you know, I got into the hatchery because I knew we were going to find *Salmonella* there and maybe we can give industry some advice and some suggestions on how they might reduce that contamination. And first of all to assess what the contamination actually was.

But only by identifying these critical control points and not leaving any out are you ever going to get close to elimination. I don't think we're ever going to idiot proof the food supply. I don't really believe that we're going to get *Salmonella* zero tolerance on these kind of food products. But if we're ever going to approach zero or dramatically reduce it, we can't leave any of the critical control sources out.

And all of you have seen this diagram, a speaker or two already today have used this particular one. And usually there are double lines coming down from the breeder and the hatchery to the growout. So it's no doubt that, you know, no one's going to stand up here and tell you that is the only source, but it has been a primary source of all of

the bacteria, not just *Salmonella*.

We go into the commercial broiler hatcheries and one percent of all our samples are *Listeria monocytogenes* and I realize one percent is small, but it's there. And it's trickling into the processing plant through the breeders, through the growout. And when it gets to the processing plant, it flourishes because it's cool and damp. But it starts in places like the breeder flocks and the hatcheries. *Clostridium perfringens* coming through the egg, you can find it in the hatchery without even trying. *Campylobacter* too, and I'll talk about that at the very end.

Oh, excuse me, let me back up here. Okay, we -- working for the government you cannot -- you have to have an open door policy. If somebody walks through the front door, which they can no longer do, because of our security -- but when they used to be able to do this, if they were coming with a little bucket of gamish and they wanted us to test it to kill *Salmonella* on the -- on the fertile hatching eggs or whatever, we have to have an open mind, and say, okay we'll test this chemical A-Z. And we basically did that. We looked at everything that we could lay our hands on. And through the years what we basically found was regardless of the chemical, if you dip it -- if you dip your egg into a solution you're going to have a better chance of killing the *Salmonella* than if you spray it. And if you spray it, it's

better than foam.

And we also, tried fogging. The problem with fogging is some of these chemicals can't safely be fogged and breathed in. So we didn't do a whole lot of fogging. But the dipping is not practical. Maybe with turkey eggs, that are worth a great deal, you might have a chance to argue that. But with broiler eggs, it's going to have to be a spray or something that can be automated.

Now, the bottom line here, talking about the time, if you inoculate *Salmonella* onto a chicken egg, and you apply a chemical one minute after you inoculate it, I don't hardly care what chemical it is, it's going to kill the *Salmonella*. If you wait five minutes you'd probably kill 90-95 percent. Four hours, you're not going to get many. By 24, it doesn't hardly matter what you apply you can't get the chemical in there deep enough to kill the *Salmonella*. All of these chemicals are direct contact chemicals. They have to touch the cell wall of the *Salmonella* in order to kill it. So, basically a lot of these chemicals to ever reach their peak effectiveness have to be applied at the farm. And that's just not done in our industries. Some farm application, but for the most part, the eggs are moved from the breeder house, breeder farm into a cooler at a hatchery, and then -- if they are going to receive any chemical treatment, it's usually at the hatchery. And it's

more then 24 hours after the *Salmonella* has been -- has gotten on the egg shell and has penetrated.

The hen laying the eggs through the same opening that she defecates and her body being significantly warmer than just about any place except Phoenix in July, the temperature deferential causes the *Salmonella* to get pulled through the shell and into the membranes and so forth where they have an excellent chance of surviving. So the application is not always what chemical you apply but how rapidly it's applied.

We look at all sorts of chemical -- the quaternary ammonias works. The one we probably had the most success with was hydrogen peroxide, about a 1.5 percent. And I think industry was using this to some degree. Particularly some of the breeder companies. But it's my understanding that they've noticed some corrosion of some of their equipment with this. And Phenol works okay. So we're still looking. Even through 90 percent of my work is with *Campylobacter* right now, I'm still working with trying to find an ideal chemical for killing *Salmonella* on these fertile eggs.

In fact next week we're spending the whole week with a group from Madison, Wisconsin that has developed a new process to break a liquid chemical down to a half a micron size particle, which we believe has a better chance

to get very deep inside the egg. And so we're going to look at some novel chemicals and a novel way of applying them. So, we're still looking.

The chemical at the bottom down here PHMB Polyhexamethlyene biguanide hydrochloride was an extremely effective chemical that would eliminate *Salmonella* instantly and it was not a problem and we could -- we applied it on chicken carcasses, on the eggs. But every time you find a chemical that works, there's always a down side. This particular chemical you couldn't fog it or make real small particles, because if you breathe it, it has an affinity for our lung tissue. So, it could not be breathed in small particles or in a fog. So, that kind of didn't allow it to be used in the hatching cabinets. We applied it on the chicken carcasses and the chemical had been fed to every animal that was on Noah's Ark. And they know it was a safe chemical, you can drink it, you bathe in it and all this other stuff, but the FDA said, well, Nelson, no matter how small amount you put in the chill tank, even if it's 10 or 15 parts per million, we put chicken in baby food. And you've got to have a rapid test so we can determine what's the residual amount of this chemical coming out of the processing plant. And there was the catch 22 that tripped us up on that particular chemical. Because it's a biguanide, and when you mix it with chicken skin you can't

have a rapid test to determine the residual amount. It takes two or three days in the laboratory to separate this out.

So, you know, for one reason or another a lot of chemicals sort of got tripped up right at the finish line. So, in our minds we found the ideal chemical many times, but there's always one or two things for each chemical that will cause it not to be ideal. So, we continue to search.

The ozone -- the ozone we found worked wonderful in the hatching cabinet to prevent cross contamination from -- when the little chicks start to hatch out and you have this Mount St. Helen effect in there. So, rather than allowing, if you only have a hand full of eggs that have the *Salmonella*, this prevents it from being spread to an awful lot of the little 15,000 chicks in the hatching cabinet. And the hatchability was not adversely affected.

Now with all of these chemicals, let me tell you how we approached it. We were not into trying to fool our selves. You can take any chemical, you can inoculate *Salmonella* on your fertile egg, you can dip it in your chemical and then you can immediately analyze that egg for your *Salmonella*. And chances are if the chemical's effective at all, you're not going to find the *Salmonella*, and so your going to say ah-ha, we've eliminated the *Salmonella*. But if you really want to know if the chemical

works, you inoculate the egg, you dip it in your chemical. You then put it in the incubator and incubate it and hatch it, 21 days later you get a little chick. You take that chick and grow it up in an isolator for seven days. And then you cut out the ceca, and look for you marker organism. And we have never, ever found a chemical that you didn't have at least five percent of those birds positive after that kind of treatment. We call that the acid test. And even with hydrogen peroxide which we consider to be an excellent chemical, if you did that test properly you still had some positive chicks after that seven day growout in the isolator. So, we haven't really eliminated them. We still had some small percentage of positive.

Now, we went into the hatcheries in 1990 with the cooperation of the poultry industry because we went to five or six commercial broiler hatcheries and they let us come in and we pulled our samples. And the samples that we pulled were egg shells, chick pads, fluff, anything, horizontal swabs or what have you. And 75 percent or slightly more than that of every sample we drew in 1990 -- and this was from all different companies, five or six different large companies in this country -- over 75 percent of all the samples were positive for *Salmonella*. And more alarming 95 percent of these positive samples had greater than 10 to the three *Salmonella* per sample.

The breeder hatcheries were 11 percent positive. This is where you got to shower in and shower out -- shower in. We actually went to a pedigree hatch, 56 percent positive. So even at the tip of the pyramid.

So basically we just kind of published a paper showing hatchery one through six. We didn't name any companies, but they were significant companies in this country, and it showed that the hatcheries were just throbbing with *Salmonella* in 1990.

Then what we did was, we wanted to see five years later after we have given a lot of these talks and, you know, talked to them about how to try to treat with the chemical as early as possible. And paying attention to sanitation in the hatchery of horizontal surfaces. We went back to the same six hatcheries -- five or six hatcheries with the same personnel using the same methodology to see if things had been reduced. And incidentally all of this hatchery work that I said was done so many years ago, the other people involved or the people on this paper -- Stan Bailey, Mark Berrang, Joe Maulden, Jeff Brewer, also, John Casin was involved and Mike Musgrove, Jeanne Wilson, so a lot of people from the University of Georgia and other scientists here at Russell Research Center was involved in this hatchery work.

So, we went five years later and we saw a

significant reduction. Just looking at three hatcheries right off the bat were 90 percent and went down to 52, 75 down to 22 and the hatchery 66 down to 12. Now, the overall reduction was 77.7 down to 29. Now, that's still not great, but now ten years later we haven't gone back to these hatcheries. I would feel the percentage positives have even decreased even further. But it did show that with enough attention paid to the sanitation and -- and just trying to get rid of *Salmonella* any possible way you can, that it had an effect.

Some of the reasons we think that this happened was use of more effective chemicals, whatever they might have been using. Maybe they tried something based on something some of us did or showed was more effective than what they were using. More diligent cleaning, changing of nesting material on the farm. Improved ventilation in the hatcheries. Overall improvement in the hatchery sanitation, not just at the broiler hatchery but the breeder hatcheries. And so, all of that probably played a significant role.

Okay, newly hatched chicks can become colonized with very low levels of *Salmonella*, they just don't have a gut micro flora to resist colonization. And these become seeder birds and they spread this contamination very rapidly through the flock through an assortment of body openings which I'll show on another slide here in a minute. And then

these seeder birds can diminish the effectiveness of a treatment like competitive exclusion or vaccination or whatever.

You have a reservoir of *Salmonella* in these commercial hatcheries. If you go into a hatchery in those days at least and you couldn't isolate *Salmonella*, you need to check your methodology in your lab, because it was there, you just wasn't picking it up. The bird is extremely susceptible at the day of hatch and all of the possible routes of entry into the animal. If you look at -- talking about the age of a chick. If I take a little chick on the first day of its life and I gave it ten to the nine *Salmonella*, which I wouldn't do, I'm going to probably kill it. But if I wait till maybe 14 days of life that ten to the nine might not even be enough to get it to stick to the gut of that -- of that 14 day old chick because the gut has matured. And here you can see a two day chick, day of hatch ten to the one, ten cells. By the time that bird is just four days old it takes between a 1000 and 100,000 *Salmonella* to stick to its gut and so forth. The number just keeps going up. Because the intestinal tract is maturing. That's why competitive exclusion works, it's an instant maturation of the gut micro flora.

Now, you look at the routes of entry, all the different openings into that little bird's body. I can take

two *Salmonella* and put it in the eye of a chick and between 24 and 48 hours later that chick is spitting out 10 million *Salmonella* for every gram of droppings that it has. So, it's become a little *Salmonella* factory and it's hard to imagine in a hatching cabinet that you're not going to have *Salmonella* getting in to something like the eye of a chick, if it's bouncing around in its hatching cabinet. And the navel, you know, you got this unabsorbed yolk material that the bird sort of encloses its body around this yolk and uses that for nutrition until it learns to metabolize the feed.

We find *Salmonella* and *Campylobacter* and some of these other pathogens in this yolk even as late as 65 week breeder birds still has some of this unabsorbed yolk material floating around in its body that's still carrying some of these food borne pathogens in this particular material. And the hatchery contamination, as I said before, limits the effectiveness of all these other treatments. For instance, if you're vaccinating birds and, you know, the immune system of a bird is not fully competent until ten or 12 days of age, and you got *Salmonella* coming from the hatchery. And these birds are in a house, 20,000 chicks in a house are spreading its *Salmonella* around and getting it all on their feathers and skin, it doesn't really matter what kicks in two weeks later to clear their gut. The damage may have already been done. And the same thing with

competitive exclusion.

If they come from the hatchery loaded with *Salmonella* and shedding it in that house, it's going to get spread around on the outside and even those that become heavily contaminated inside, the competitive exclusion may not be able to go in and drag that contamination out. If you get it there before the *Salmonella*, it causes the gut to be mature and can repel the *Salmonella*. If the *Salmonella* beats the competitive exclusion or vaccination to the punch you've got a mess in the house.

So, you know, it has an effect on other effective tools. The importance of this contamination has been clearly demonstrated the world over. I think there was fellow named Goren G-o-r-e-n in the Netherlands who, him and his co-workers did a study with God knows how many birds, 8 million I think, and they were looking at 4 million with competitive exclusion and 4 million without competitive exclusion. They wind up isolating something like 29,000 *Salmonella*, from everything that you can think of. And they serotyped all these *Salmonella*. And so they did it from all of the possible sources and all the way to the final bird coming out of the processing plant. And they were not able to show any connection, for instance, between feed and the final carcass. But they showed a tremendous correlation between the serotypes they isolated from the hatchery and

what they found on the final bird going to the consumer. And other studies have showed the same thing, including some of them that -- that we've done in this facility.

I think that's my last slide. I just want to say that the *Campylobacter* that I'm working on now, we also, it's transmitting to the egg. Now, there's an awful lot of people that don't believe that. Mainly because the organism is difficult to culture from dry material, like in the hatchery. So, if a microbiologist doesn't find something on a sample, he automatically assumes it's not there. And once people have kind of closed their mind to the fact that the fertile egg is not transmitting *Campy* they don't want somebody like me to come along and open that can of worms. But the direct evidence is -- is at our feet now. There's a dissertation just finished in Puerto Rico where a woman did 960 fertile hatching eggs and found about 20 of them *Campylobacter* inside the eggs. And found three percent outside the eggs.

And also, we have a fellow who's going to be on the program tomorrow, Allen Byrd at Texas A&M. He routinely cultures *Campylobacter* from the chick pads. No PCR, none of this. He routinely cultures *Campylobacter* from the chick pads.

So, why would you think one organism didn't pass through this egg when all of the rest are? What would --

what would prevent that? So, for FSIS, who I know this is a *Salmonella* meeting, but *Campylobacter* is also an important organism. And so, I'm telling you *Listeria*, *Clostridium perfringens*, all of them are found in these hatcheries and they are all involved in some way with the fertile egg.

So, I think that's the last of my slides. And it is interfering with my happy hour, but I'm sure all of us are going to be able to take questions, and I'll hang around a little while in case anyone wants to get me one of their business cards. I'll be happy to send you all this pack of yellow reprints, thank you.

(Applause.)

DR. BAUER: Thanks. Could we have all the speakers from the last section up here -- Stan Bailey, Peter Holt, John Glisson, we have time for a few questions and we absolutely have to be heading for the door about no later than 5:45 for sure, they close the building at 6:00. So, if you want to leave now, go ahead. We have time for just few questions for Dr. Cox, Dr. Bailey, Dr. Holt and Dr. Glisson.

Yes, could somebody get a microphone here for this gentleman. Could you state your name and your affiliation.

MR. BAHL: I'm Aren Bahl, I work for a company called Immudyne.

The question I have is, first of all, all the papers were very excellently presented keeping the industry

problems in mind. And I need to congratulate every one of you. Most of the speakers this afternoon touched or talked about the mucosal immune system, but there was no elaboration or no further quantification as to how the mucosal immune system can be matured or how does it mature, or what are the cells involved? So we are talking about the mucosal immunity but we are not getting deeper into the mucosal immunity, we're still talking about humoral immunity alone. Could anyone please comment on that area.

DR. HOLT: As far as mucosal immunity goes, the easiest way of measuring it is the humoral immune response. And so that's what we have focused on an awful lot. So, as far as the cells are involved, and that is primarily the B cells. There has been, you know, a fair amount of work over last four or five years looking at cell mediated immunity primarily done by our sister lab up at Beltsville looking at the T-cell immunity. And it's very much involved and both the CD4/CD8 are involved in that.

We are very heavily involved in the humoral aspect trying to look at the hierarchy as far as the immune response goes in the mucosal system and just where in the mucosal system immune response against a gut organism occurs. And we do find it in the gut, we find it in the crop and actually we just finished up a study looking in the lung and lung secretions and it's found there as well.

So, the immunity, as far as mucosal immunity goes, you know, occurs everywhere.

DR. BAUER: Are there further questions?

DR. O'CONNOR: Bob O'Connor, Foster Farms.

Just a quick question about broiler vaccination with *Salmonella* vaccines. The theme seems to be that the live vaccine acts as a good primer. But if you're not going to follow those live vaccines with a killed vaccine, what's your opinion on the effect of live vaccines exclusively on broilers?

DR. BAUER: Who did you want to answer that?

DR. O'CONNOR: Open question.

DR. BAILEY: Bob, as you know I'm not an immunologist. I'd say that what we saw in our studies and what I've talked to Chuck about in the past, is that the live vaccines alone work pretty well for the homologous strains of *Salmonella*. As long as it's either the same serotype or a very similar serogrouping. But they have some trouble against heterogeneous *Salmonella* which are not closely related antigenically. So -- but the combination of using a live vaccine to prime the system with autogenous that are against the primary serotypes that you see in your area in doing the multiple treatments that I talked about, seemed to be giving far better effect then just the live alone.

DR. O'CONNOR: I think that's a very good point that people need to take away from this meeting, that there might be vaccines available, homologous vaccines for vaccination of broilers might work for that serotype. But heterologous serotypes it may not be effective against.

It sounds like a simple solution but I don't think it's as simple as it sounds.

DR. BAILEY: No, it's not and that's a good point that you made and I should have -- I should have pointed that out in my talk. But that's clearly been demonstrated both in research labs and in people who looked at it in the field.

DR. HOLT: Actually, the live vaccines, to my knowledge, the only live vaccines that are going to be allowed are the Typhimuriums that are licensed right now. And you get an awfully good cross protection there.

DR. BAILEY: But you don't get particularly good against [group] Cs.

DR. HOLT: [Group] Cs do become pretty difficult, yeah. And there is a certain amount -- and I'm going more into mouse data know, there hasn't been a lot of data on live vaccines in chickens. But you know, in mice, they've shown with live vaccines, that you can generate a specific immune response, you know a cellular immune response, and it will also provide a certain amount of non-specific cellular

immunity with natural killer cells, that type of thing. So, if you keep on boosting with that live vaccine, you very well may provide a certain low grade protection, you know, even against something like a group C. But to actually kick the guys over, you know, going with a killed is going to be your best bet, you know, in the end.

I think what you're getting at probably is that, going with a killed you're going to have to go ahead and take the birds and inject them, which can be fairly labor intensive. It's much, much easier to give a live organism, just put it in the water or in the feed or whatever.

DR. BAUER: We've got a question right back here.

DR. STAYER: This Phil Stayer with Sanderson Farms.

I had a question concerning these vaccines. If they are better on homologous strains, should we not be focusing on human pathogens versus this generic *Salmonella*? What we see in chickens is rarely found in humans. Are we chasing the wrong goal?

DR. BAILEY: That's a different, I mean that -- that question has a lot of levels to it. Not the least of which is that at current, for regulatory purposes, all *Salmonella* are created equal. And for you meeting your specifications as laid out by FSIS, then one's as bad as another. We -- it is certainly a debatable question if that

is the way it should be ultimately. Certainly if we get into attribution and do a better job than we've done in the past and have a better understanding of the predominant serotypes from humans and those from chickens and we see a big disconnect, then that is a debate that may be addressed at a different time. But for the purposes of where you are today, then you have to consider for your regulatory purposes that all are created equal. Even though that -- even though we know they're not.

DR. BAUER: We've got time for a couple of more questions.

QUESTIONER: I have to ask a question to Dr. Cox, to get him going. Dr. Cox, John Glisson did an excellent job presenting the data that 8 billion chickens lead to how many billion dollars worth of live chickens. And live chickens lead to so many billion dollars worth of further processed chicken or chicken products, either cooked or uncooked. The question I have is, the pig industry and the cattle industry has partially looked at activated lactoferrin as a spray mechanism on the carcass to reduce the *Campylobacter*, *Salmonella* and *Listeria*. Is there any work in broilers on turkeys to look at activated lactoferrin spray?

DR. COX: I don't really know the answer to that. Does anybody know? Not to my knowledge but that doesn't

mean it's not happening. We're not looking at that here.

DR. BAUER: Any more questions?

(No response.)

DR. BAUER: Housekeeping, please remember to wear your badge tomorrow. We start 8:30.

Also, Copper Creek Brewing Company tonight between 7:00 and 8:00 p.m., hopefully some of the speakers will be there. Let's give our speakers another round of applause.

(Applause.)

DR. BAUER: And we do have to be out of the building, they close down at 6:00 p.m. Thanks. Thank you for you attention.

(Whereupon, the meeting was adjourned at 5:45 p.m., they reconvene at 8:30 a.m. on August 26, 2005.)

1