Salmonella Interventions for Swine
Jim McKean

[1] Well let me introduce Dr. James McKean of Iowa State University, College of Veterinary Medicine, Ames, Iowa, to discuss the salmonella interventions for swine. Dr. McKean is an extension veterinarian and Associate Director for the Iowa Poultry Industry Center. Dr. McKean's research interest is focused on integration of research and public policy to develop education and demonstration programs in swine disease control and eradication and for pork safety and quality initiatives. He has received many awards for his outstanding service to the swine industry. Please help me in welcoming Dr. Jim McKean. Thank you Randy. My mother couldn't have written that any better. I appreciate the opportunity to be here and I want to say that this presentation is an amalgamation of work that Drs. Rostagno, Hurd, and Ed O'Connor and others at Iowa State have assisted in. And so most of the opinions are mine. Most of the data is jointly owned and we'll try to separate the two as we go through this afternoon. We're going to talk about salmonella interventions in swine. And I want to talk first of all about on-farm interventions and then try to integrate that into a more fully active program.

[2] We see the problem as sub-clinical infections and carriers in pigs that are identified as a food safety problem and then the risk for pork contamination coming from those carcasses. That sort of defines the problem as we've looked at it over the years. And we'll talk a little bit about that. Proposed solution for many people and I think a first principle for some is that reducing the meat contamination by reducing or eliminating contaminants at the pre-harvest level and that is the on-farm. And my question is that an appropriate strategy? Is that an appropriate way to attack this problem in this species? It may be appropriate in other places.

[3] I come to that looking at intervention considerations. The epidemiology and ecology of the hazard, we recognize and part of the way that I got started in this business is I went through the sulphamethazine residue in pork issue in late 70's and 80's. It was also an FSIS issue. We were handed a residue rate in the neighborhood of ten to thirteen percent and a fairly high testing program. And we were told to assist producers in getting their animals back on to market. And what we very quickly found out is that we didn't know very much about sulphamethazine even though we'd used it for twenty some years. So we needed to do some work. But it becomes clear that antibiotic residues, pesticides are in fact residue or are in fact contaminants that must be dealt with at the farm source. They are not added at the plant. They do not multiply under handling conditions. And so they become things that must be handled at the farm level. I would argue that microbes may or may not be the same as our level of knowledge increases, we may be able to look at those. But microbes have the ability to multiply under handling conditions. So what starts out to be a safe product, if in fact it's mal-handled anywhere along the chain, you have the risk of it becoming an infectious
product. Correlated with that is the potential for downstream contamination. Again with the antibiotic residues is our marker on one end of the spectrum. Once that product is in the stream there’d be very little opportunity to add to that contamination level. I’d also remark that there’s very little opportunity to remove it. And therefore, it must be dealt with at the farm. There must be and Dr. Angulo talked about attribution and I think we must get into the issue of attribution of contamination and its significance. Makes a lot of difference in an industry. Dr. Angulo showed that pork was attributed in Denmark to ten to fifteen percent of the residues, excuse me of the salmonella in human cases. That means that you could eliminate all of the pork-related salmonella infections, you would not change that number by a great number. And I think what one of the things that we need to look at is we need to look at the different parts of the production system. And our group is focused primarily on the on-farm to the cooler and not the post harvest. But if you segment that group, you can segment into about three groups, the on-farm, the transportation and abattoir, and then the processing sides. And we need to spend a little more time in terms of attribution of contamination in those areas. For on-farm we need to look at feasibility and costs. Feasibility not only is can this, can this intervention be applied, but does it consistently across all levels and types of production, does it, does it produce a reasonable and expected result? And then the last issue is one of control measures in terms of implementation. By that I mean can you measure the success or the failure of that intervention? Can you put an incentive or a penalty on that will encourage producers to in fact implement that intervention in the appropriate way, in a proper way so that in fact you have a predictable result. And lastly can you monitor for that activity so that you know that in fact it was completed or the efficiency by which it was completed, so that you then can put the incentives or the penalties for it.

[A couple of other things, salmonella serovars are everywhere in large numbers. They are in a wide range of hosts. They are an environment, Dr. Angulo mentioned Javiana as an environmental issue. We believe there are a number of others. And we’ll talk a little bit about some of that as we go on. Exposure levels in pigs for different serotypes. We’ll give you carriage and/or infection but they appear to be different for serotypes. And so that becomes an issue when we treat all salmonellas alike, what might be successful for one may not be for others. We believe that the environment, salmonella can survive for extended periods of time, plus or minus six years. That means it’s going to be with us all the time. It’s going to be available to both pigs and other species in the slaughter environment to amplify and then bring forward. We found very little ability to distinguish between carriage and infection. And I guess at the slaughter point, it really doesn’t make any difference. But from an intervention standpoint, it may very well make a difference in whether these animals are merely reflecting what’s in their environment or whether they in fact are infected and may be intermittent shedders or contaminators of the environment. Randy mentioned the fact that I]
work in animal disease control is my other half brain, my other half time. And we deal in those conditions with detection levels that are sensitive in the ninety-five plus percent range. With salmonella in pigs, cultures been reported depending on which paper you read or which process you follow or the sample size that you take, to be some place between thirty and seventy percent sensitive. Antibodies are used as a method for identifying carrier animals or animals that have been exposed to salmonella species prior. It does not help us with shedding necessarily. In fact many of the animals that have antibody positives may not be shedding. It appears to be effective at herd but not individual levels, so it’s hard to find those animals at least with our current technology that we want to remove from the herd as shedders and contaminators of the environment. And that creates some problems. And then we’ve got PCRs of a variety of ranges. But their test costs at least in large term large basis projects their test costs make them difficult to see how they’re going to be active.

[5] As I said earlier, the first principle for many people is to stop the contamination at the farm. We need to look at the practicalities, the attribution of risks, and the downside or downstream recontamination. Because I think that that first principle while it sounds very good, is going to be difficult at our current level of experiences. And I would almost say ignorance in many cases. On-farm salmonella controls, most of the experiences in Europe they’ve been working at it for a large number of years, I think Sweden that Dr. Angulo mentioned early on, Sweden started their program in the mid-fifties and have been at it for a long time. The Danes spent ten years developing a salmonella program. What I hear as I listen to these groups talk is that there is still some level of dispute as to when you have success. And I think that that’s an issue that we in the United States are going to need to deal with as we move forward because we don’t deal with the kinds of marketing systems that they do in Europe. We tend to shoot the outliers economically. And that may sound funny, but it really isn’t for people that have to work with those folks and try to bring them back in ergo my experience with sulphas. Current focus in the European theater is on feeds and feeding practices and we’ll talk a little bit about that as we go through.

[6] Now one of the things that people say is improve your hygiene and your biosecurity. And I would agree with that. This list is a list of papers that you can find that will all say that they will, they are capable of reducing the prevalence of salmonella either by antibody testing or by culture at the farm level. Hand washing prior to entry to swine facilities, toilet facilities, this is Julie Funk’s work down in North Carolina where she found that those facilities that had toilets on the finishing facilities, had lower salmonella prevalence. The idea I think being that if the owner cared enough to put toilets in these facilities that are not located close to a house, that they had a higher level of sanitation in their system. Boots and outer clothing changes prior to entry is a standard biosecurity issue and it has been shown to be effective. Reduced human entry to the site.
This is one that causes one to scratch his head. But there is evidence that those sites that have higher human traffic also have higher salmonella prevalence's. Cleaning and disinfection has been equivocal. It’s been tried in a lot of different environments. And sometimes it works and sometimes it doesn’t. And I'll give you a little idea later about why some of those things might occur. All-in and all-out have been set as a way to clean and disinfect and move animals in and leave them there for their finishing lifetime. Then move them out to harvest. Clean and disinfect, bring the next group in. And not add animals to that group has been a method that on its face sounds very logical. And from a disease standpoint would be very logical. But it has had equivocal results. The sad part is that if you take all-in and all-out, boots clothing changes at entry and hand washing prior to entry of the facility there are studies that consistently say that those three things together may be, gives you an additive effect and a positive and a more reproducible effect. And so now we get a little clue. And then you come along with a counterintuitive story that says that pen sanitation may not influence culture results. That in fact dirty pens may be less likely to have positive cultures than others. We need to look at that one and be sure that in fact that’s consistently the case.

[7] The Europeans have looked at on-farm interventions primarily as feed based now that they've, they've gone through the all-in and all-out. They've gone through a variety of other things. They would agree with the sanitation, the hand washing and the changes of boots and clothing as a method. They would not agree with the all-in and all-out. They, at least the Danes wouldn't. Their success with that it was equivocal at best. Feed as a source of contamination. We hear this fairly regularly. If you look at the literature, if you look at the experience, feeds are rarely contaminated with the serotype that you find in the animals. And in some cases, the animals being positive are merely a reflection of the fact that they're being fed the contaminated feed. And in some cases I think and I think we've done this in some of our on-farm stuff, if you're not very careful with your sampling protocols as to where you collect those feed samples from you may in fact have the feed samples reflecting what's in the environment and what's in the animals. And therefore, which comes first the chicken or the egg? Common environmental contaminants are found in feeds and that should not surprise. The feeds are coming out of the environment and you would expect that. One of the questions is do they reach an infective dose? Do they reach a level that would keep that animal infected if he were exposed to that feed? I think that's part of the reason for the disconnect. Then one of the things that the Danes and the Swedes and the like have given us in terms of information is the fact that there are variable effects on processing. Pellets versus mash, clearly again applying first principles, one would say that pellets should be safer than mash. And in fact the studies and the experience in the field that the Danes and the Swedes have had is that pelleting does reduce the microbiological load in the pellets at the time of pelleting but that does not consistently, is not consistently maintained. And the Danes have
shown that if you use the ELISA test and serology as your marker that animals on pelleted feeds are more likely to have high prevalence than animals on home raised, home mixed and much coarser feeds under similar conditions. So particle size appears to be protective. And if the larger the particle size, it appears to be more protective or the larger particle size appear to be more protective. That has implications because what we'd like to do for feed efficiency is we'd like to grind those, in pigs we'd like to grind those particle sizes down. It may be that that is not a useful strategy if our goal is salmonella prevalence. Wet versus dry feeders, clearly wet fermented feeds are more protective than wet unfermented feeds. And dry feeds are less than the fermented feeds. So you got the wet/dry issue but you've also got superimposed in that in the feeding systems in Europe, you've got the issue that at least partial permutation occurs with many of those wet feeds. Fermentation appears to the issue that gives rise to benefit.

[8] And what, that's where the Danes have come with acidification. Acidification of feed or water, the natural form if you need natural in your vocabulary in this regard is a fermentational liquid feeds is where they first identified it. It is in fact where it is clearly an issue at hand. The addition of whey as a major feed ingredient will also provide some assistance as will the introduction of organic and inorganic acids. And the acid de jure at the moment is benzoic acid at somewhere around the one percent level. That creates, in feed that creates some real problems in terms of the environment. And you can apply it in either feed or water as you so desire. That is an area that we, I think we need to look at farther. Source of miscellaneous other interventions. Stocking density has given equivocal results again. There are some studies that say if you have high stocking densities, you'll see increased prevalence. And others it has not been the case. Seasonality or location in this country appears to have some impact. Southeast U.S. seems to have more Typhimurium then we do in the Midwest. And it appears that winter and spring both here and in the Danish experiences seem to be higher at times. Although I will tell you that at least in the ante mortem pens that our highest rates of recovery are during the summer months and I attribute that to the ability to maintain environment in those ante mortem pens. So seasonality may very well have to do with different stages in production. And then the other one is intergenerational transfer from infected moms to kids. And you can break that with early weaning.

[9] So I'll take all of that together and after ten years of intensive on-farm in-field experience, the Danes would say since their problem is primarily salmonella Typhimurium that an infected sow herd can be expected as those animals move to the finisher to throw higher prevalence herds, higher prevalence groups of pigs than does the non-salmonella Typhimurium infected animals. The question is, is that salmonella, does that Typhimurium specific Typhimurium is a organism that is at least somewhat specific to pigs or at least has a predilection for pigs. And is that because the animals truly become infected as opposed to merely exposed? Acidification of feed and
water, expense is a problem as is the effects on equipment. It really
creates some rather difficult situations on flooring and various types of
equipment if it's not properly prepared for. The issue of coarse
grains versus fines, fine grained. I've already mentioned the feed
efficiency issues. What you find in the Danish experience is that
people will do these things for short terms to get themselves out of a
Level 3 situation. But they prefer not to do it for very long because
several of these things add substantially to their costs. They've
looked at farm level classification, reducing abattoir and transport
contamination. And I think that is an issue that we probably ought to
look at but their experience in terms of total contamination of the
facility, the facility being the abattoir, those classifications may
not have been as helpful. It does limit, there's a limit to continued
reductions in terms of interventions with classification. They
dropped from two and half to three percent down to about a percent and
a half fairly rapidly in their ten-year experience. And if you look
at their numbers over the last few years, they've really plateaued
out. So there may be a limit at how much the on-farm interventions
can provide in terms of total activity.

[10] So now as Paul Harvey would say, now for the rest of the story
and I'll try very quickly to look at this in terms of why those
things, those experiences that I've just laid out to you and the lack
of clear cut measures might be the case.

[11] The traditional is the on-farm infection either going directly to
the cecum and gut-associated lymph nodes, which we commonly find or
going through transport and lairage. And the idea if you read the
early papers, the idea of stress in transport causing these animals to
shed and more animals to become infected. Then going through the ante
mortem pens, going through the dressing with a high percentage of the
animals positive. And then the issues related to HACCP in the plant.
The other area that I think bears looking at is the fact that there
may be an on-farm infection that causes the non-gut associated lymph
nodes to become infected. But that then comes down into carcass
contamination. There has to be a reason why, as the animal, as the
carcass moves through post processing we oft times see increases in
salmonella levels. It's got to be, something's got to affect that and
contaminate that equipment as we go through. And it appears to be
larger than one could explain simply by carcass, external carcass
contamination.

[12] So we get good manufacturing practices and the issues related
to processing controls associated with HACCP and the abattoirs. We have
the issues of post mortem or ante mortem pens with routine
recontamination, I'd say in the seven or eight years that we've been
doing this. In a wide range of packing plants for both culled sows
and boars and for market hogs and then even the barbequed hogs. We've
very rarely find a pen that is not salmonella positive prior to the
introduction of animals. There's a rapid infection from the
environment. And in order to cut this out, it may require complete
isolation of those animals.
This is a rapid infection that Dr. Scott Hurd and others did. The important point is that at two hours, eight of ten tissues that were sampled were, eight of ten animals were sampled at least in one tissue. That means that those animals picked it up. And I will tell you from our experience in plants, it’s twenty or thirty minutes is enough to do it. So just having the animals on load and get in line may not be the answer. I give you that as, a couple of these now are just food for thought cause I don’t know the answers to them.

We did one study where we had ten, we went ten weeks. We broke out the serotypes. They’re listed here. Derby was found on that farm at a very low rate. You’ll notice that Derby was consistently found all the way across the ten weeks. I will tell you that we PFGE’d a number of those Derby’s and they didn’t all come from the same place. They are not the same as came off that central farm. And we took these animals the same day each week. Took them through the same routine. Took them to the same plant. And this is the kind of run we got in terms of ileocecal cecal contents on these animals and the serotypes that came out of them.

The idea of stress has been raised. This was a sequence to the study I just showed you. Three hundred pigs in ten weeks, so fifteen stayed at home. Fifteen went for a slumber party and at the National Animal Disease Lab. And then were taken to the plant. The question with stress. There’s no difference between on-farm in the colon contents at slaughter. The idea of stress and we’ve done other studies since looking at on-farm and when the animals arrive at the plant. And the stress of transport I think is overrated. We’ll have to go on.

Just so you don’t think that it’s only in market hogs. This is a sow study. We took the animals and cultured them on-farm and that’s the top row. Then we went to the collection point. The collection point thought we, they were going to do our kids a favor. They put the animals in individual in small pens instead of throwing them out in the group with the rest of them. Put bedding on the floor. And when we went to culture those, we couldn’t find culture-positive animals the next morning before they were loaded to send to the plant.

But not to worry, by the time they got to the abattoir and spent the night in the abattoir, the listing of serotypes that showed up on any given week are listed there. And you’ll see a wide range. And they certainly didn’t all come from that production facility. So the ante mortem pen is an issue.

So what is the farm level problem?

Dr. Angulo and Dr. Engeljohn both mentioned the fact that there are salmonellas of concern in humans. Whether this list is not necessarily this year’s list but it’s just for me it represents what we normally get in pigs and what the humans get. And Derby is our heavy hitter. And we very rarely do you see a Derby in associated...
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with a human infection. And that has implications I think.

[19] So we’ve got all these good manufacturing practices. The question is what, how are we going to measure their success?

[20] Clean up on farms and the system will be safer. That’s the first principle that we’ve been dealing with thus far to date. I think there’s a limitation in terms of measurement either with culture or ELISA. And we’ll talk about that in just a minute. Reduction of contamination, is it a reduction in contamination and prevalence? Or do we have to go to zero? Is zero even possible? Where to measure the farm level effect? That issue has not been clearly delineated. The research, there are many people that do research. They do it at the time the animals are going to leave the farm in terms of the farm effect. But is that the appropriate place to measure it? Or is it in the carcass? And that’s an issue that needs to be rationalized I think as we go forward. The other thing is serotypes at least, our experience thus far a serotype is considered to be important.

[21] My question is, based on the fact that Derby is a predominant in humans or in animals but rarely shows up in humans, is Derby equal to Typhimurium? This is the other high one in both humans and pigs. Is Agona equal to those? Is Infantis equal to, where are we in terms of the serotypes? Should we be separating them out? And can we in fact do that? We know that Infantis can cause human illness. There was a reason the Danes put up their program and spent the millions that they did. So we don’t want to minimize these. But the question is, are they all created equal? And should we treat them as equals? Is there a link between the farm and the pork product, a direct link? And that’s a question that I think needs to be examined. Here’s one, a dilemma for the research folks. These are six herds, six portions of a herd. They have common genetics, common feed, common production SOPs. They are handled in the same ostensibly in the same way. We took and sampled sequentially both fecal and serologically.

[22] And you can see the variation in fecal results. And if you’re really good and I don’t have a pointer, so I can’t point them out. But if you’re really good, follow the yellow and the purple because they seem to be some correlation.

[23] But the point of this slide is the variability within those finishing facilities over a period of about twelve weeks. So these were sampled every two or three weeks for six different sample sets. And you’ll notice a tremendous amount of variation in terms of the prevalence with either fecals or with serologic evaluation. Now as someone that’s going to do an intervention, which week and which plant do you want to take it at? I leave that for your consideration. Because I think that is one of the problems with interventions. If you use these measures, they vary substantially in terms of how you’re going to, how you’re going to evaluate it.

[24] All right. Clusters, intermittent shedding and then resolution
of infection epidemics I think are all possible explanations for that.

[25] So where should GMPs be applied? On-farm? In transport and concentration at the abattoir? And I suspect we need more information. Probably all three at some point, but there’s need for more information I think from the top down in terms of how we’re going to be successful. And I would argue that transport may be not as important as some people would believe or want to believe, at least in pigs. But that concentration point at the end is.

[26] There’s for those of you that have never seen it. That’s the all American pig.