Northern Ireland Assembly

Committee for Agriculture and Rural Development

OFFICIAL REPORT (Hansard)

Bovine TB Review: Queen's University Belfast

1 May 2012

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Members present for all or part of the proceedings:
Mr Paul Frew (Chairperson)
Mrs Jo-Anne Dobson
Mr William Irwin
Mr Kieran McCarthy
Mr Oliver McMullan
Mr Robin Swann

Witnesses:

Professor Christopher Elliott	Institute of Agri-Food and Land Use
Dr Irene Grant	Queen's University Belfast
Dr Neil Reid	Queen's University Belfast
Professor Ian Montgomery	University of Ulster

The Chairperson: I welcome Professor Chris Elliott, the director of the Institute of Agri-food and Land Use; Professor Ian Montgomery, the director of Quercus; Dr Irene Grant, a lecturer in microbiology and food safety; and Dr Neil Reid, the manager of the Natural Heritage Research Partnership. I hope that I got that all right. I will give you time to get settled.

Thank you for your attendance here today. This is a very important inquiry to the Committee, as has been demonstrated by the time that it has taken to get to you. I apologise for the fact that you have had to wait for so long, but I hope that you have found it as interesting as the members have. I am sure you have a presentation. Please keep it as brief as possible so that we can go directly to questions. I would appreciate that.

Professor Christopher Elliott (Institute of Agri-Food and Land Use): You got our names, ranks and serial numbers correct, so I will not go through those again. We are very pleased that as a result of the research that has been conducted at Queen's over quite a long period, we can have an input into your important inquiry. As we have sat and listened with a great deal of interest, two key themes have emerged. One relates to the testing and the actual diagnostics of the disease, and the other is the role of the badger in the spread of the disease. Those are the two key areas that we have been conducting research on.

Mr McMullan was absolutely correct: bovine TB is an unbelievably complex disease. The organism is very intelligent. It can hide away in the system of an animal for months before the immune system

recognises that it is there and can take action. During that period of a couple of months, the disease has the ability to spread within farms, because the disease can be spread as animals move around.

What happens during the two months when the bacteria is hiding in the animal? The present testing regime, which is based on the skin test, cannot detect the disease until it has been there for about two months. There is a second test called the gamma-interferon test; you have heard information on that test. It is a faster test, so it gives an indication of disease presence earlier than the skin test does. However, I have heard a lot of accurate information to say that it is not a very reliable test, because it is not measuring TB; it measures an acute phase protein, which can be elevated by many other different types of disease. So, it is not TB-specific.

My research group set out to look at that early period of infection — the first couple of months, when animals are subjected to the infection. We set out to look at what changes occur in the molecular fingerprint of the animals: what genes are switched on, what genes are switched off; what proteins the animals produce, and what proteins the animals have a decreased production in. To cut a very long story short, we were able to find a number of those very specific gene and protein markers between two and four weeks after infection. We were able to produce a fingerprint of the disease. That fingerprint tells us some of the underlying mechanisms of what that bacterium is doing for the two-month period when it appears to be dormant, but is far from it. We searched many scientific databases to try to find out if there was any evidence that the markers we detected were linked with other diseases. In most cases, the answer was yes. However, the particular fingerprint that we have produced is very specific to bovine tuberculosis. In our laboratories at Queen's, we have come up with a number of very interesting targets that could be used to advance the diagnostics of bovine TB. That research was completed recently, and I presented it to the Department of Agriculture and Rural Development (DARD) before the end of last year.

Dr Irene Grant (Queen's University Belfast): I want to make the Committee aware of a couple of mycobacterium bovis (M.bovis) projects funded by the Department for Environment, Food and Rural Affairs (DEFRA) for which I have been principal investigator. The projects are concerned with improving the diagnostic or detection methods for M.bovis. The current means of detecting M.bovis in lymph nodes of cattle taken at slaughter is culture, which takes eight weeks, so it is not rapid. It is very slow and may or may not indicate M.bovis presence when, in fact, the organism is there in low numbers.

I have almost 20 years' experience working with mycobacterium paratuberculosis, which is the relative of M.bovis. We used some of the things that we have learned about paratuberculosis and mycobacteria generally to approach M.bovis from a different angle. We have been looking to develop a method that will pull the organism out of the tissue sample to make it more detectable by a subsequent test method. This is called immuno-magnetic separation (IMS). In simple terms, you use microscopic beads with an iron core, coat the beads with an antibody or peptide to your organism of interest, and mix the beads with the sample. If M.bovis is there, it sticks to the beads. You can then apply a magnet, pull the beads out of the sample with any M.bovis attached to them, get rid of the rest of the sample, wash the beads and then do what you like with the bovis that you pulled out of the sample. So, you can use a polymerase chain reaction (PCR) DNA test or try to culture it. There are various other things you can do.

The first project that DEFRA funded, starting just over two years ago, was for two years and is just completed. We submitted our final report to DEFRA at the end of March. It was a collaboration between Queen's and the Agri-Food and Biosciences Institute (AFBI). It sought to develop or generate antibodies or peptides that could be used on those beads; prove that the bead system worked in pulling the M.bovis out of the tissue samples; and carry out a fairly substantial survey of bovine lymph nodes taken at slaughter, and compare the statutory results with our new test results.

We managed to generate several M.bovis-specific monoclonal antibodies and peptides. We successfully applied those to the beads. We then employed the beads in conjunction with PCR DNA tests and the culture test. We carried out a large-scale survey. The results showed that the IMS bead-based methods detected around 27% more M.bovis-infected lymph nodes than the current statutory culture method. The vast majority of those extras were from non-visibly-lesioned lymph nodes.

The positive IMS PCR results show an initial impression of M.bovis positivity was obtained by the PCR route within 48 hours. It was still eight weeks — the current statutory culture period — before the culture results became available. Generally speaking, however, the culture results mirrored the initial PCR results. So, you got an early indication of M.bovis positivity, which was backed up by culture results later on. The IMS method provides a potential means of taking a different angle to testing for M.bovis. On the basis of a survey of 280 lymph nodes, we are getting better results, ie more M.bovis positives from lymph nodes from reactor cattle.

The second project started in January, also funded by DEFRA. It is using the antibodies and peptides generated in the first project but putting them into a different test format, a lateral flow device test, that may be used in the field to test badger faeces for M.bovis. My collaborators on that project are Professor Montgomery and Dr Neil Reid at Queen's, a diagnostic company in York and folks at the Food and Environment Research Agency, Woodchester Park, Gloucestershire.

The project will run for 18 months. We are in the test development stage. Once we have developed the test, we will evaluate it in the lab. We plan then to go out to badger setts in the Province in TB-affected areas and non-TB-affected areas. We will not interfere with the badgers at all, but we will collect faeces, do an in-field test on the spot and then take the sample back to the lab and test it there also. Then we will compare the results of the field test with the laboratory test and see how well the field test performs. However, the idea is that you can detect M.bovis-infected badgers, use GPS to say exactly where they are and link them in with TB breakdowns around the Province to provide that kind of information.

The Chairperson: Those are three very distinct projects, but they all tie in together.

Professor C Elliott: Exactly.

The Chairperson: Sorry, Dr Grant, were you finished?

Dr Grant: Yes.

The Chairperson: Is there someone else due to speak?

Professor Ian Montgomery (University of Ulster): Yes, I will give a brief review of work on two areas of badger ecology and epidemiology. Since the early 1990s, we have been funded largely by DARD studentships, which have been extremely valuable and appreciated. Without that, we would have very little knowledge of what is happening with respect to the badger population. Very briefly, the results indicate similarities with GB but also some differences, which might be significant when it comes to implementing the control measures. Those differences might be related to landscape or to farming practice. It is not entirely clear, but there are some significant differences there.

In short, our badger groups tend to be smaller. They also tend to make use of habitats that are not used heavily by badgers elsewhere, such as field boundaries, for their setts, in the absence of significant woodland. We also see a huge difference in the density of badgers from one area to another. Landscape is very influential, and there can be as much as a 30-fold difference in population density. The GB experience always refers to particular studies which are in high-density areas. Very often, we deal with relatively low densities of badgers by comparison to GB. There are aspects that are very similar. They live in territorial groups, and males in particular can wander across the countryside. Significantly, one of the reasons why it is a difficult disease to control is that badger groups do not really line themselves up with farms. A single badger group could cover up to nine or 10 different farms, and it is likely that most farms have only one badger group, but it spans all the neighbours.

Some preliminary data on particular areas for the level of badgers that are exposed to the pathogen comes out at around 40%, but it is a limited study. We have something like 14% excretion of the pathogen in some of our data, and, in a later study, 6% came out, with 2% being super-excreters, that is, they excrete on more than one occasion of being captured. Therefore, we have some idea what the disease is like in a living group of badgers in at least one area of the Province. We also have some preliminary data on biosecurity. For example, we have been able to show that 60% of farms graze

cattle next to their neighbours, and there is no barrier to contact between cattle. That is clearly a major problem. There are a variety of other areas of data which are important, but I will not go into that because it is in the written submission.

The second area is to do with population change in badgers and persecution. That work was funded primarily by a competitively won tender with DARD. That work shows in a nutshell that population size and the number of social groups have not changed between the 1990s and 2007-08 when the last survey was done. There is probably every reason to believe that we underestimate badgers by that method. We have some genetic evidence which suggests that we can catch four out of five badgers. Therefore, when we make the correction, it comes out at roughly 41,000 badgers in Northern Ireland. There are wide margins of error, but that is the nature of the beast. It shows a huge range of densities, and, consequently, when we sample the population, we get quite wide confidence limits for the population size. Therefore, the population has not changed. It was fairly stable, as far as we can tell, between the 1990s and roughly five years ago.

The persecution aspect is also interesting. Of course, it is very topical. There was a lot of publicity recently, and there is anecdotal evidence that there has been an upsurge in persecution. When we did the work in 2007-08, however, we showed that there was a decrease in persecution from the 1990s. There was a major decrease in disturbance at setts in general but also a big decline in digging activity at setts indicative of people going into the setts with dogs.

Disturbance of that nature is important because it has a direct effect on the disease. Research done through the Central Science Laboratory (CSL) in England shows clearly that small groups have a higher level of disease, that badgers that are disturbed become mobile across the landscape and that more of the disease is put into the environment when you get disturbance at setts. In fact, when that sort of thing happens, it has a detrimental effect on the whole farming activity in certain areas.

Broadly speaking, those are some of our results. There are details there, so I will now shut up and take any questions that you may have.

The Chairperson: OK. Thank you very much. Again, I ask members to keep to the two-question rule, and I, too, will try to abide by that.

The three distinct yet linked projects that you talked about should be part of the research. However, how close are we to getting data into a practical solution that DEFRA or DARD can implement, and is there acceptance of the findings of those bodies?

Professor C Elliott: The answer to the first part of your question is this: for however long it takes. What we did at the university is really the fundamental research to say that we have the markers. Those results now have to be taken into a field study to validate them. It is one thing to do something in a nice academic environment but different when you get into the blood and guts of what is going on in real life.

My estimate is that it will take probably a minimum of a year to 18 months to do that study. It will not be a trivial undertaking. I costed it for DARD, and the ballpark figures that we came up with were between £500,000 and £1 million.

Will the data be accepted outside Northern Ireland? The answer is yes if you publish your research findings, which we do in international journals, so that would not be a fear.

The Chairperson: On the work and research that you have on the badger, its nature and its movements, have you compiled that evidence and impressed it on the recent plans in England and, before this month, Wales for the proposed culls? Did you look at the details of the proposed culls and form an opinion on whether that would work? Having the intelligence that you have on the badger, is it there, is it right, is it nearly there or is it completely wrong? Can you give us some indication or steer as to what your view is?

Professor Montgomery: We have to look to a very substantial area of scientific literature, which is freely available to all concerned and has come out of the randomised badger culling trial (RBCT). That is the single biggest mammal epidemiology wildlife disease study undertaken ever, anywhere in the world. It is a huge study that has gone on from 1975, culminating in the Krebs trial — the actual experimental investigation — which confirmed the involvement of badgers in the disease. However, it also pointed out absolutely clearly that there is nothing to be gained, as far as we can ascertain, from conducting culling as a means of control. That comes out in paper after paper, and the arguments are very well thought out.

The contrary view has been taken in the Republic of Ireland, where they did a very different study, which was not designed to elucidate the perturbation that came out of the Krebs trial. It was done under very different circumstances, not in randomised fashion but in selected areas, which were selective because of their physical characteristics. As a result, that information does not travel, because it is very specific.

The motivation for us to do the research on badgers at Queen's was simply that there was a complete lack of information on what was going on with badgers in Ireland. Nobody else was doing any work on it at that time. I asked to get involved and have been involved in a marginal fashion over the years. It is not my prime area of interest. During that time, however, we built up a certain amount of expertise on how to interpret the scientific data coming out of the CSL, which has developed in that very large-scale study. We have been able to apply that information to what we know about badgers in our own backyard, so to speak.

That is one of the motivations on distribution abundance. Dr Reid has been able to model the distribution of badgers across the landscape, so we can predict — I hope fairly accurately — the population density. That will help us, whether we use a vaccine in future or simply try to ensure that badgers and cattle do not come into conflict in particular areas.

That sounds like a long-winded response, but the answer is that we can apply the information that we have now to any discussions. Most of our information is specific to Northern Ireland, but a lot of the information that was gathered in the past at the Central Science Laboratory, as it is now — it used to be the Ministry of Agriculture, Fisheries and Food (MAFF) — is directly applicable to what we experience as well.

Mr McMullan: You strongly advise against the culling of badgers.

Professor Montgomery: Yes.

Mr McMullan: That is your main thing. You are totally against, or strongly advise against, culling.

Professor Montgomery: There is no scientific argument in support of it.

Mr McMullan: Your — these big words get me — experiments on this new thing that means that you can get the diagnosis within 48 hours. What are the benefits of that?

Dr Grant: It means that you would have an indication much earlier of positivity that bovine tuberculosis (bTB) is potentially present. It is not a confirmation, but you could tag a herd as a stronger suspect earlier.

Mr McMullan: Do we not already know in theory that there is bTB in those hot spots and different areas?

Dr Grant: Oh, yes, TB is there but the way in which it works currently is that if skin tests are carried out and you get reactors, the animals are slaughtered. The lymph nodes are taken, and they are either visibly lesioned or non-visibly lesioned, but they are all tested. Currently, the vast majority of the visibly lesioned ones will turn up M.bovis positive in culture and a very limited number of the non-visibly lesioned lymph nodes test M.bovis positive, so they are reported back as M.bovis negative.

Our test is picking up 25% more culture positives from the non-visibly lesioned lymph nodes, which means that an extra 25% of animals, and whatever number of herds that that represents, are positive, and farmers are being told that there is no evidence of M.bovis on the basis of current statutory culture.

Mr McMullan: Is there a possibility of the breeding of a super-badger? When we talk about strain types in badgers parallel to the strain types in cattle, what happens if badgers from different areas with different strains breed? Can that lead to immunisation or — I am being very flippant with my words — to what I call a super-badger? Is there a possibility of different strains in the breed confounding your analysis or anybody's reports on the strains of TB in badgers?

Professor Montgomery: The nature of the disease is that it can be present in an individual for a long period, so there is nothing to stop that animal from breeding. Consequently, there is not so much selective pressure on it. It is not likely that you will have a development of a super-badger spontaneously without very strong selective pressure. Consequently, I see that as being something that will not happen.

The disease is a conundrum. It can be with a badger throughout its life and never have an obvious detrimental effect. We very rarely see badgers that show external signs of any disease, even though they test positive.

Mr McMullan: What is the outcome of different strains of TB in badgers when they breed?

Dr Grant: It has nothing to do with it. They can breed and produce offspring, but the TB infection will not cross over at the same time.

Mr McMullan: The TB remains the same?

Dr Grant: Yes.

Mr McMullan: Are we sure about that?

Dr Grant: I think so, because it would have to be exchanged between the two bugs, not between the animals.

Mr McMullan: That has never been tested, then? We talked about different areas. We heard earlier that we have the County Tyrone strain, something else and something else again. If a badger from, say, County Down went into County Tyrone —

Professor Montgomery: You see a very similar pattern of distribution of those strains in the badger population to what you see in cattle. That has been demonstrated not just here but elsewhere. There is information to that effect in evidence from AFBI, based, I think, on the roadkill survey. Therefore, that is there already and has been done elsewhere.

We have evidence from a recent study that would suggest that very rare strains can show up in a badger, and there is some link between that and the local cattle population where it has shown up as a very rare strain. That is strong evidence that something is going on between the two populations of hosts of the disease.

The Chairperson: OK, I am going to move on. If you have any further questions, Oliver, get them to the Committee Clerk, and we will pass them on.

Mrs Dobson: Thank you for your presentation. Under project 1 in your submission, you say that a reservoir of undetected infected cattle exists because both TB tests do not identify all infected animals at the early stage. How big is that reservoir in percentage terms?

Professor C Elliott: Stats have already been quoted about the current tests. I think that the skin test has around a 50% to 60% accuracy rate. The gamma-interferon test is quoted at around 70% to 80%.

That means that, in roughly 20% of cases, animals are diagnosed as being positive that are not but in 20% of cases animals are positive and are not being diagnosed. That is the kind of error that we are working with.

Mrs Dobson: You touched on this, but what practical changes would you recommend or have you recommended to the way in which we test cattle for TB as a result of your research?

Professor C Elliott: The skin test is the standard method, and that will not change for a long time. That is being supplemented by the gamma-interferon test. That is a very expensive test, because the person who invented it was very clever and patented on it. However, that patent has now expired. Therefore, my recommendation to DARD was to stop buying commercial test kits and do the test itself, because it would come to a fraction of the cost. That would reduce the cost of the current testing but not improve its performance. We believe that if DARD introduces some of our fingerprinting techniques, that would greatly improve the chances of detecting accurately more than 90% of infected animals.

Mrs Dobson: Could a more accurate and cheaper alternative skin test be introduced? Is that a possibility?

Professor Montgomery: It is very unlikely. A lot of work has gone into that for a long time, and it has not improved substantially. Data was produced, and different people say that it is 50%, 80% or 90% reliable. Our feeling in Northern Ireland is that it is closer to the lower end, at 50% to 60% reliability.

Mr Irwin: I am sorry that I had to leave for a few minutes during your presentation. It probably proves how complex this disease is when even the possibility of a dual test still gets only 90% detection. That does prove how complex it is and how difficult it will be to detect 100%. Is that right?

Professor C Elliott: It is absolutely right. I would not claim now to be anywhere close to 100% accuracy for diagnostics.

Mr Irwin: That is what I thought.

The Chairperson: Thank you very much for your presentation and answers, and for attending this very important inquiry.

Professor Montgomery: Thank you.