The Paratuberculosis Newsletter

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All contributions should be sent to ssn@life.ku.dk

Søren Saxmose Nielsen
Editor

1. IAP Business

A New Information Partnership

Kenneth E Olson Ph.D., PAS

The IAP and its members are dedicated to sharing knowledge about MAP research as widely as possible. To further this objective we are pleased to announce that the IAP has partnered with the American Dairy Science Association® (ADSA®) to share information from the International Colloquium on Paratuberculosis through the Searchable Proceeding of Animal Conferences (S-PAC®). The collaboration also provides a new benefit for IAP members, access to this unique and powerful information portal at "member rates."

S-PAC is an on-line, user searchable, database of proceedings from many of the top animal conferences in the world. Each year many state, regional, national and international conferences are held for scientists, producers and ag professionals, where "cutting edge" research and management information is provided. Often the first, and sometimes the only, place where this information appears is in the conference proceedings. S-PAC was established as a convenient way to provide access to this wealth of valuable information. It allows a much broader audience than just the conference attendees, or even those who happen to find the conference proceedings, to use the information provided at the conferences. It should also be noted that several of the proceedings in the database are only available electronically through S-PAC.

So, what is available through S-PAC? In addition to ICP proceedings, others that may be of special interest include:

- The American Association of Bovine Practitioners (AABP)
- The U.S. Animal Health Association (USAHA)
- The Johne's Disease Integrated Program (JDIP)
- The Western Dairy Management Conference
- ADSA Annual Meeting Abstracts

A total of 412 proceedings from 44 different conferences are currently available to S-PAC subscribers. New conferences and additional proceedings are added frequently, making S-PAC an information accessing tool of ever increasing value.

Each article is stored individually so that it may be searched in any way you wish. This means that rather than going to 10 sets of proceedings that are sitting on your bookshelf and paging through them for information that you need, or checking 15 separate websites for information that you think may have been presented at a conference, a visit to the S-PAC site allows you to rapidly search all 400+ proceedings in the database for the information that you are seeking.

Visit http://spac.adsa.org/ to check out S-PAC and all the proceedings currently available. In addition to the primary search function for the database, you will find a calendar of upcoming conferences and links to websites for many of the conferences. You can "test drive" the system at the special rate of "$5 for 5 days". This allows you full access to the system as many times as you would like during those five days, and you can sign up for this offer repeatedly or, if you are ready to add S-PAC to your information tool kit, IAP members can subscribe for the year at the "Member Professional rate" of only US$75---a modest business expense, here are the steps to sign up:

1. Go to the S-PAC® site http://spac.adsa.org/ and click on "Subscribe to S-PAC; then, click on the line "Click here if you are a member of a Partner Organization"

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If you have additional questions about S-PAC®, please contact Ken Olson
Ph: +1-630-237-4961 or e-mail: keolson@prodigy.net

IAP Book Purchases

The association has a number of past International Colloquium proceedings available for distribution. We currently have the following in stock:

8ICP Proceedings – Book
8ICP Proceedings – CD-ROM
7ICP Proceedings – Book
6ICP Proceedings – Book
5ICP Proceedings – Book
4ICP Proceedings – Book

Proceedings are available FREE to members, but shipping charges of $15 (USA) or $35 (outside of USA) will apply. Non-members may purchase the Proceedings for $25 plus shipping costs.

Furthermore,
The History of Paratuberculosis compiled by Rod Chiodini
is available for 50 USD + shipping for members, and $125 +shipping for non-members.

To order please send an e-mail to Secretary-Treasurer Ray Sweeney at:
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The number of proceedings is limited so we operate by first-come-first-served principle. Please place your order no later than 1 April 2012.

Also note that the 7th, 8th, 9th, 10th, and 11th Proceedings are available on-line at www.paratuberculosis.info.
Starting with the 9th ICP, a print version of the Proceedings are no longer produced by IAP. However, print versions of 9th, 10th, and 11th ICP can be purchased at http://www.proceedings.com/6219.html

12th International Colloquium on Paratuberculosis

The 12th International Colloquium on Paratuberculosis will take place in Parma 22-26 June 2014. Visit the official website at: http://www.icp2014.eu/

2. Short scientific communications

Mycobacterium avium subspecies *paratuberculosis*—An environmental trigger of type 1 diabetes mellitus

Coad Thomas Dow

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Type 1 diabetes mellitus (T1DM) is an autoimmune disease. The etiology of T1DM is incompletely understood but environmental agent(s) are thought to trigger T1DM in the genetically at-risk. Humans are widely exposed to *Mycobacterium avium* subspecies *paratuberculosis* (MAP), a proven multi-host chronic enteric pathogen that is mostly studied in ruminant animals and causes the inflammatory disease paratuberculosis or Johne's disease. In humans, MAP is the putative cause of Crohn's disease and has been linked to sarcoidosis, autoimmune thyroiditis, multiple sclerosis and autoimmune diabetes. The role of MAP as a trigger for T1DM was first postulated in 2005; subsequent studies suggest a link. This article discusses MAP, human exposure to MAP, genetic susceptibility to MAP and MAP in human disease including T1DM.

The paper has been published in Journal of Diabetes Mellitus, 2, 88-95. Link to full access

A case report of Johne's suspected cattle calf by ante-mortem and post-mortem examinations

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Johne's disease (JD) or bovine paratuberculosis is a chronic, progressive, and incurable intestinal disease caused by *Mycobacterium avium* subsp. *paratuberculosis* (MAP) in domestic and wild ruminants (Roupie et al., 2011). The disease results in severe economic losses due to reduced milk production (Stable, 2006). Infected cattle with clinical signs shed the organism in feces (Clarke, 1997; Stehman, 1996) and milk (Taylor et al., 1981) resulting in greater risk for animals as well as human exposure. Serological test and intradermal skin test were performed to screen cows in an organized farm for the incidence of JD. One of the young cross-bred male calf of 8 to 10 months old was turned to be serologically positive and was suspected for JD but no clinical signs were observed.

The serological assay was conducted with *Mycobacterium paratuberculosis* antibody ELISA kit (Pourquier® ELISA paratuberculosis-paratub.serum-S, Institut Pourquier, France) according to manufacturer's instructions. The presence of MAP specific antibodies was determined based on the ratio of absorbance of experiment sera samples to absorbance of positive control sera sample (S/P percentage). The S/P % value of the suspected calf was 139.7% and 136.5% for the paired sera samples collected with a month interval indicates a high level of MAP specific antibodies in the serum. Fecal samples for bacterial culture and whole blood samples for Interferon gamma release assays (IGRAs) were collected before performing the Johnin intradermal skin testing. The animal showed positive reaction in single intradermal test with johnin (I.V.R.I., Izatnagar, India) with a skin thickness difference of 6 mm.

The animal was euthanized and post-mortem was conducted. Mesenteric lymph node was slightly enlarged and congested. Gastrointestinal tract did not exhibit any characteristic changes. Various organs such as intestine/rectum, mesenteric lymph nodes, and spleen, liver, sub-mandibular and pre-scapular lymph nodes were collected in 10% formalin for histopathological observation. Prescapular lymph nodes, submandibular lymph nodes, liver, spleen and mesenteric lymphnodes were collected for tissue specific IGRAs. Mesenteric lymph node, rectum and fecal samples were collected for MAP isolation.

Lack of clinical symptoms and specific pathological changes in organs pose difficulties in arriving at a definitive diagnosis. The blood collected from this animal was stimulated with JD specific recombinant antigens showed 3 fold increase in stimulation compared to media controls as measured by BoIFN-gamma IC-ELISA. The ELISPOT assay was performed with the PBMCs using JD specific antigens as per Maroudam et al., 2011 showed 5-6 fold increase JD specific spot forming units compared to media stimulated well has encouraged to move further into other in-vitro JD diagnostic investigations.

Tissues of prescapular lymph nodes, submandibular lymph nodes, liver, spleen, and mesenteric lymph nodes were processed as per the Freshney (2004). The cells were isolated from the tissues and analyzed for the presence of CD4 T cells and CD8 T-cells by immunophenotyping. The isolated cells were stimulated with JD specific antigens and showed significantly high JD specific IFN-gamma release in spleen and prescapular lymph nodes. The tissues such as rectum and mesenteric lymph nodes turned MAP culture positive when inoculated into BACTEC MGIT culture tubes and cultured in BACTEC 960 as per the manufacturer's instructions (BD Biosciences).

Transmission of disease to calves during the first year of life is by swallowing contaminated feed and through milk/colostrum through from infected dam and rarely by in-utero infection. Clinical diagnosis of JD is difficult because the disease can present in any one of the four forms viz latent, sub-clinical, clinical and clinical, advanced stage (Whitlock et al., 1996). The major problem in control and possible eradication of paratuberculosis is the difficulty in identifying the MAP infected animals in early stages of the infection (Jungersen et al., 2011). The prolonged course of infection, the predominantly subclinical nature of the disease and lack of tests for accurate detection of subclinically infected animals (OIE manual 2008 In http://www.oie.int/) complicates the JD diagnosis. Additionally all serological test and bacterial isolation attempts fail to detect the latent form of JD (Whitlock et al., 1996). In many animals, shedding of bacteria occurs long before a clinical sign of the disease starts (Cocito et al., 1994) and these shedders contribute to the infection of other animals by contaminating the environment. Thus diagnosis of the disease by fecal culture is considered as the most reliable and gold standard method to diagnose the MAP infections (Merkal et al., 1968). In the present study the organisms were not isolated/cultured from fecal samples due to other bacterial contaminations which resulted in test failure (Collins et al., 1996; Whipple et al., 1991).

IGRAs which detects the cell mediated immunity (CMI) against MAP by measuring IFN-gamma release from sensitized lymphocytes has been used as an early diagnostic test for bovine paratuberculosis infection (Wood et al, 1989 and Kalis et al., 2003). However the available IGRAs tests for MAP diagnosis uses crude PPD and hence to augment the specificity of the IGRAs well defined and MAP specific antigens were included in this study. The induction of MAP specific IFN-gamma release in various in-vitro assays proved that the disease can be diagnosed before the appearance of clinical signs using these assays. Although IGRAs are sensitive test for early diagnostics, there is a need to identify and evaluate the novel JD specific IFN-gamma inducing candidates in the field /farm conditions in order to improve the present assays that use crude PPD (Nagata et al., 2005).

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3. Histories, Comments and Opinions

My Personal MAP-Quest

Judith Eve Lipton, M.D.

May 3, 2012

Single case reports are no longer the fashion in modern medicine. We all know that an n of 1 plus 1 plus 1 plus 1 ad infinitum equals nothing in terms of proving causality. However, once in a while, it might be well to consider a case report that is startling and suggestive, even if it is statistically meaningless. Here I present the history of my battle with Crohn's Disease, knowing that it doesn't prove anything by itself, but believing that it matters nonetheless insofar as it suggests the desirability of pursuing a more detailed scientific inquiry. In brief, I think I have been "cured" of Crohn's Disease with antibiotics directed at the eradication of *Mycobacterium avium* subspecies *paratuberculosis*.

I was a generally healthy physician, wife and mother of three until the summer of 1986, when I suffered my first bout of what was called ulcerative colitis. It remitted with steroids and time. I had several subsequent minor episodes of colitis that remitted quickly with oral or rectal steroids. In 2003, I underwent a routine colonoscopy, which showed penetrating abscesses in my colon, but no action was taken. In February 2004, I became increasingly fatigued and anorexic, with fever and abdominal pain; the presumptive diagnosis was appendicitis. However, colonoscopy revealed Crohn's Disease with diffuse abscesses of my entire colon. I was treated with IV prednisone, plus oral mesalamine and azathioprine – the standard medical treatment for Crohn's - with little improvement. In July, 2004, I received an infusion of infliximab, which helped dramatically. After several more infusions, I relapsed and was readmitted to the hospital in October, 2004, whereupon I again received infliximab as well as steroids.

While lying in bed, soon after discharge, I read Dr. Salah Naser's article in The Lancet, "Culture of *Mycobacterium avium* subspecies *paratuberculosis* from the blood of patients with Crohn's Disease." This article was immediately salient to me, since I knew about Johne's Disease because I was keeping angora goats at the time. In fact, in December, 2003, one of my goats died in my arms of a mysterious wasting disease. Here are my survivors:

Maybe because I was still a little agitated on prednisone, or maybe it was just good instincts, but I immediately connected the sudden onset of my severe Crohn's with the death of my goat, and I decided to contact experts in the field of MAP to look for appropriate antibiotic treatment. (Retrospectively, I think I was wrong, I don't believe that I caught Crohn's Disease from a sick goat, but at the time, it seemed compelling.) I read most of the references from Dr. Naser's article and emailed many of the scientists. Dr. Robert Greenstein, author of many articles about MAP including several in The Lancet, was kind enough to refer me to Dr. Thomas Borody in Australia. Dr. Borody had extensive experience with treating Crohn's Disease as an infection caused by MAP.

I printed a ream of papers for my own gastroenterologist and my primary care physician, internist Dr. Anita Shaffer, of The Polyclinic, Seattle. The gastroenterologist did not believe a word of it, and wanted to continue with infliximab. Dr. Shaffer, however, was intrigued. I paid for a phone card so she could call Dr. Borody (in Australia) directly, which she did. After reviewing the literature and discussing the situation, we decided to give Dr. Borody's protocol a trial. Before this, blood was drawn and sent to Dr. Naser's office for culture and PCP. The results were negative.

On December 10, 2004 Dr. Shaffer started me on the Borody protocol: clarithromycin, rifabutin, clofazimine and ethambutol. I also had one more infusion of infliximab in January, 2005. I continued to take mesalamine, and tapered off prednisone, but developed secondary adrenal insufficiency requiring supplementation with hydrocortisone at physiological replacement doses (5 mg daily). I quit the ethambutol fairly quickly because of my fear of side effects. Within a month, it became impossible to obtain clofazimine in the US because the drug company discontinued selling it in the US, and donated their stock to agencies that treat leprosy, largely in the Third World. However, Dr. Shaffer and I were able to obtain clofazimine via the US government under a "compassionate use" protocol, with extensive help from Swedish Medical Center, where we were both on the medical staff.

I took the three medications – rifabutin, clofazimine, and clarithromycin - from December, 2004 until May of 2010. I don't recall when I stopped the mesalamine, probably around 2007. I had no side effects. By the summer of 2005, I also had no trace of GI disease. Repeat colonoscopy in 2007 was entirely normal except for one polyp that was removed. I retained a bit of adrenal insufficiency and took small doses of hydrocortisone (5 mg or less). I never took prednisone again. In the fall of 2009 I closed my psychiatric practice in order to retire to a small, rural village in Costa Rica. I am writing this article from Playa Potrero, Costa Rica, in view of the Pacific Ocean, and can honestly say that despite eating Third World food, for three years, including local milk, seafood and meats, as well as diverse foods sold in fiestas and tiny pulperias (corner grocery stores) I have not had a single day of abdominal cramps or diarrhea. I also no longer need or take hydrocortisone, unless I have a severe stressor.

Dr. Borody and I have discussed the "c word": Am I cured? I have no evidence of GI or inflammatory process, either by symptoms or regular blood tests looking for inflammatory parameters. Is it possible that receiving the antibiotics just after and even overlapping one infusion of infliximab eliminated MAP from my gut? I suppose we will never know.

Although in itself my story proves nothing, it should at least provoke serious thought among MAP researchers and especially among skeptical gastroenterologists, who overwhelmingly adhere to the traditional view that Crohn's is primarily if not exclusively an autoimmune disease. I thank Drs. Borody, Naser, and Greenstein, and especially Dr. Anita Shaffer for saving my life. It is highly unlikely that my recovery was unrelated to antibiotic anti-MAP therapy. In fact, I believe that the opposite is true: I strongly suspect that I had a serious MAP infection that responded beautifully to appropriate treatment. I am also aware that the idea of a Crohn's-MAP connection is controversial, not only at the purely scientific level, but because it goes counter to current "best practices" in gastroenterology. Hospitals, drug companies, gastroenterologists and the beef and dairy industries profit greatly from the status quo, based as it is on the regnant medical "wisdom" that essentially discounts MAP as a human pathogen.

In 2007, thanks to Drs. Marcel Behr and Carol Nacy, I was invited to participate in a Colloquium sponsored by the American Society for Microbiology to explore whether or not MAP is pathogenic in human beings. Our conclusions were equivocal, but we ended by pointing to the precautionary principal as a mandate to continue research, but also to take steps to eliminate MAP from the human food chain. This principle states that when a problem is very large, such as nuclear war or global warming, it is expedient and appropriate to take steps to prevent it, even if 100% proof of the phenomenon is lacking. It is only common sense, in keeping with the precautionary principle, to eliminate sick animals from the human food chain.

I strongly believe that if nothing else, my personal experience should encourage researchers as well as clinical physicians to question the traditional paradigm and to explore the possibility that in some cases at least, there may be an intimate connection between MAP and Crohn's Disease.

UK experiences with a national Johne's Engagement program

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How do you engage a farmer to tackle a disease he doesn't think he has got? How do you persuade practising cattle vets to commit to further training and drive Johne's control when there is little financial gain for them? If we start an engagement program how do we track and monitor progress? Indeed what should we monitor? Disease status? Within herd prevalence? Or simply evidence of robust controls? How could all of this be co-ordinated in such a way that we could demonstrate the "Johne's Journey to control".

These were some of the problems that we had to deal with in 2008.

Prior to this the UK only approach was to offer a Cattle Health Certification Standards (CHeCS) approved program which would seek to demonstrate the absence of MAP based on annual blood screens. Dairy vets and farmers had dabbled with this and the experiences had not been very satisfactory. Adopting the rigorous test and cull program based on a single blood test was expensive in terms of testing and the losses associated with culling test positive cows. Although cows were generally removed early in the disease process, but not infrequently test positive cows would be retained, tested again whilst in remission and test negative, causing some confusion amongst vets and farmers who did not fully understand the disease and the test. Vets and farmers lost faith in the program. The most successful and popular control program to evolve inadvertently let the disease build up within a herd only for the farmer to then sell the cows to somebody else! The "one size fits all" test centric approach was not widely adopted by the industry.

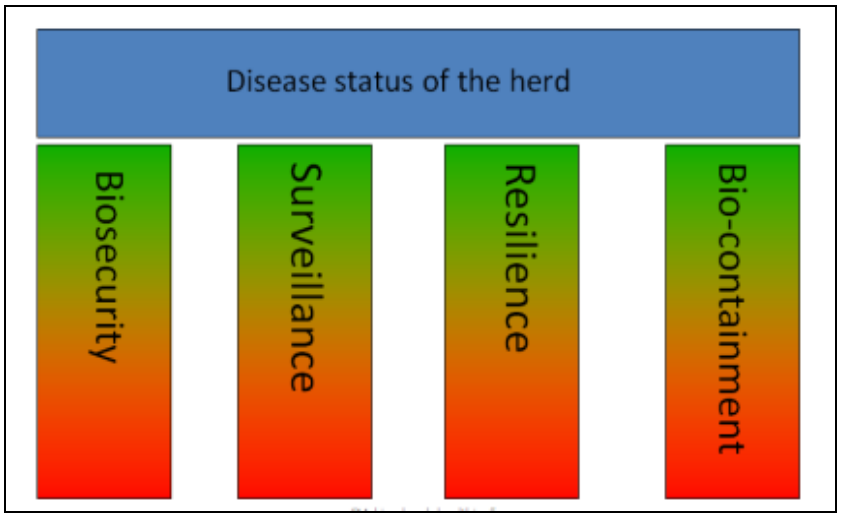
The incidence of Johne's disease was driven upwards by the increasing risks of introduction and spread within herds. The Foot and Mouth Epidemic of 2001 prompted widespread movement of cattle between herds during re-stocking. The incidence of bovine tuberculosis subsequently rose and in the TB hotspots dairy farmers had little choice but to purchase replacements of unknown MAP status to replace animals removed as a consequence of rigorous TB controls. Over the same time period milk prices hit an all-time low and the response by many farmers was to expand their milking cow herd to help defray the fixed costs of the farm. Maternity areas were not increased in proportion. Waste milk feeding became more widespread not least due to the euthanasia at birth of 400.000 male dairy calves prompted by the ill-advised ban on calf exports (due to consumer pressure relating to transport of calves overseas for the veal trade). The scene was set for a major increase in Johne's disease in the national dairy herd.

There had to be a better way to deal with this disease.

The first major breakthroughs were prompted by Soren Nielsen's presentation at the British Cattle Veterinary Association conference in Glasgow 2007. He neatly illustrated how a quarterly testing program combined with simple risk management could be used to help control MAP in Denmark. This risk based program would at least provide a realistic option for control.

National Milk Laboratories created a non-interpretative testing program called "Herdwise" based on the Danish program.
A parallel development was the creation of Myhealthyherd- a web based relational database which would allow for the more effective management of infectious disease. The program was developed so that farmers, vets, monitoring organisations and labs could all access the

program with permissions set according to their service levels. The simple traffic light interface was well suited for the illustration of risks and disease status.
The infectious disease module was broken down into 4 areas. Risk of disease introduction (biosecurity), risks of disease spread and control(bio-containment), immunity and resilience (including vaccination) and surveillance. The disease status is supported by the strength of each of the elements.



With the majority of our dairy herds the four pillars were far from robust with most being absent!

The work by Rossiter et al in USA voluntary control program clearly indicated the approach that could be adopted for the biocontainment module. A hybrid version of the Rossiter/Nielsen model was adopted within the program. However a completely new biosecurity module had to be created to identify the likely presence of any disease within a herd. These two risk assessments could then be traffic light scored and would help shape the likely surveillance and control plan for the herd. Herds could be classified into risk combinations. Red: red herds were typical. These had a high risk of disease being present and also high risk of disease spread. Once this status is established this is the first step on the Johne's engagement process.

The risk model was an inspirational development as the ability to predict disease presence proved to be more useful than relying on the typical “test and treat” approach adopted by the majority of vets. Surveillance, by definition, is retrospective and “waiting for disease” to build seemed to be a unintelligent approach. By the time the disease is seen and recognised as a clinical problem in a herd the subclinical disease levels can be significantly high.

Control plans and Surveillance strategies had to be developed which reflected current practice. A decision was made at an early stage to ensure a program was developed which would provide a solution for all farmers. We did not want to fall into the trap where only a “high level or high aspiration” control programs were offered as this would disengage herds where MAP control may be of a lower priority.

So how were going to get the farmers engaged? We followed Deng Xiaoping advice; “ it doesn’t matter if the cat is white or black as long as you catch the mouse!”.

Several routes were adopted. Vets could engage farmers directly using the Myhealthyherd program. Typically they held an evening meeting and educated their farmers about the disease. This was then followed by vets under taking risks assessments with their farmers and then following this up with a structured surveillance program based on these risks. The vets charged for their time and worked with their farmers to create the appropriate plans.

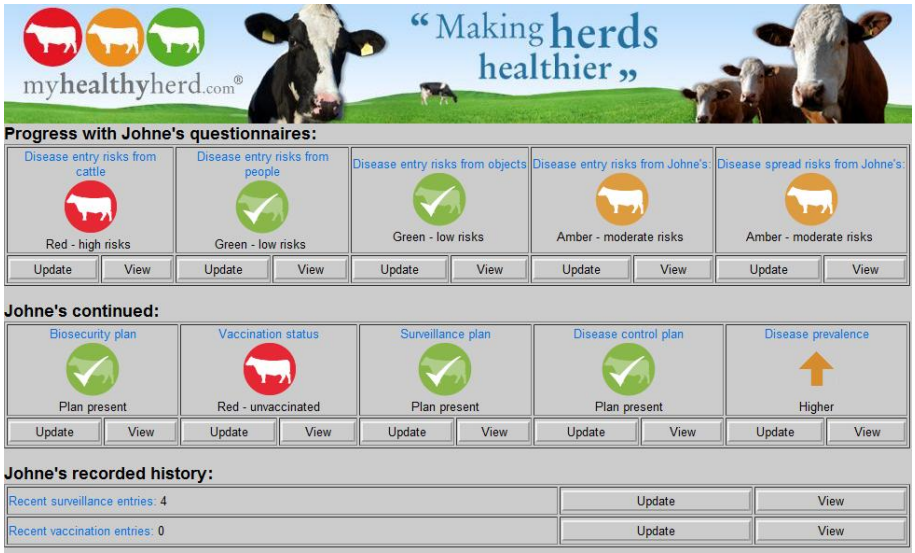
This particular route really appealed to the proactive vets who already had business models that would allow for the effective charging of time.

An alternative route which proved to be more effective in engaging farmers was the structured training of farmers using the milk processors as the hosts for the meetings. They would encourage their producers to attend meetings. The risk assessments were conducted in the break during the meeting and then at a later stage entered into Myhealthyherd by National Milk Laboratories staff. Each farmer was provided, free of charge, an option to undertake a targeted 30 cow screen of high risk animals (cows 3-7 years of age with any symptoms of low milk yield, scouring, high cell count etc) as a crude but relatively sensitive indicator of Johnes status of the herd. A simple Myhealthyherd Prevalence report was then produced and sent to both farmer and vet which highlighted their risks and more importantly highlighted what the true herd prevalence may be and how this may change if their risks remained unchecked. This provided the building blocks for decisions to be taken by the farmer and vet for effective control and protection.

The final route adopted was funded veterinary involvement. Regional Johne's programs were developed using a structured approach which involved “one to one” and group training in MAP control. Participating vets had to attend training to become an approved deliverer. The vets were funded up to £500 to install effective surveillance, control and protection plans on farms. The regional program only funded the initial 30 cow screen and they often dovetailed in with the processor driven education programs as well. This avoided repetition and ensured the same presentation and messages went out to both vets and farmers.

The typical approach of simply subsidising testing that was used in may other countries to encourage engagement in Johne’s control was observed and considered. However, testing never cured anything! Johne's is a disease which has to be managed by risk management. All testing achieves is a facilitation or improved reliability of the control programs. By channelling resources into the risk management, farmer training and veterinary engagement this provided a much wider base to work from. For the engagement program to be a success we must reach as many farmers as possible whilst also be able to demonstrate progress.

The myhealthyherd.com relational database allowed tracking of progress at a national, regional and vet practice level.



An illustration of the Myhealthyherd progress page. In this herd disease risks are amber/ amber for introduction and spread. A robust surveillance, biosecurity and control plan is in place.

The key to the success of the program is about getting the "ducks in a row".

Ensuring collaboration rather than competition developed between the various labs and organisations was a key challenge. Every lab could engage in the program without prejudice. The choice of strategy and control was effectively decentralised to the vet and the farmer rather than defined centrally.

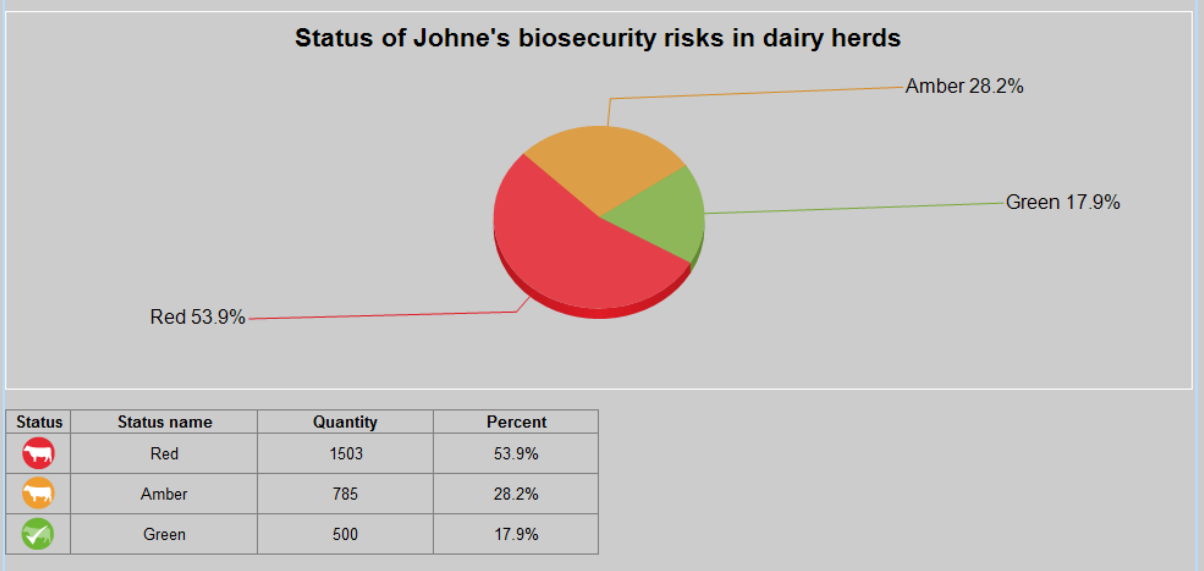
Without the generous support and guidance from Soren Nielsen, National Milk Laboratories and the tireless work from the Myhealthyherd team it is unlikely that the program would have been a success.

Dairy UK (representative body for milk processors) set up a Johne's Action Group chaired by Ed Komorowski ensuring that a consistent message was delivered and created an opportunity for the wider farming industry to contribute to the program.

The control program has to be owned by the grass roots farmers and vets who should ultimately see Johne's control as an opportunity rather than another centralised disease control program for them to follow. The didactic centralised model will not work for Johne's in our view with UK farmers.

So how far have we got? Has it worked?

Engagement has been good. In the South West over 30% of the dairy farmers have engaged in Phase 1 (training). Not all have progressed to Phase 2 (regional funded control and protection). Survey results have shown that some farmers at this stage adopt a control program directly via their vet (they know enough).



Status	Status name	Quantity	Percent
	Red	1503	53.9%
	Amber	785	28.2%
	Green	500	17.9%

By May 2012 2788 dairy herds had completed the risk of Johne's disease introduction module. This is equates to nearly a third of all dairy producers in the UK.

The current challenge is how to progress to the next stage? Is there a need to progress to more demonstrable evidence of control based on surveillance results?

Again, the view taken at this stage is ensuring that as many farmers as possible have an **effective robust control program designed to meet their aspirations and resources** which can be regularly reviewed. If we can ensure this is in place then the "R" value for the disease will be less than 1 and the Johne's epidemic brought under effective control. Within the Myhealthyherd program a scoring tool has been added to traffic light score the

robustness of each of the seven control strategies and also to allow review of the risks. This will allow a completion of the circle and the ability to graph the predicted prevalence of disease based on risk and test data. This then allows a much greater flexibility for monitoring progress as we envisage that as at least a third of producers may opt for Improve Farm Management alone as a control program (i.e. testing will be undertaken as part of surveillance at lower level rather than an integral part of control)

If we track the disease prevalence there is a real risk that this may seek to disengage producers who believe that compulsory declaration of status may impair their trading status.

So what do you want to achieve? Farmer engagement or National Control? Should a compulsory or voluntary approach be adopted? Big questions.

All avenues are open at this stage to the UK. The plan will be to maintain engagement and if the industry or global market defines that demonstrable control programs are required then the educated Johne's producer can simply migrate into the appropriate surveillance program to demonstrate test prevalence. At least this is a possible route that others may wish to learn from and adapt for their own regions. If you require further information please read the full paper at the Lessons Learned 3rd ParaTB conference in Sydney or email us on enquiries@myhealthyherd.com

3. List of Recent Publications

Ansari-Lari M, Haghkhah M, Mahmoodi F. Association of *Mycobacterium avium* subspecies *paratuberculosis* infection with milk production and calving interval in Iranian Holsteins. Trop Anim Health Prod. 44:1111-6.

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Gurung RB, Purdie AC, Begg DJ, Whittington RJ. In silico identification of epitopes in *Mycobacterium avium* subsp *paratuberculosis* proteins that were upregulated under stress conditions. Clin Vaccine Immunol. [Epub ahead of print]

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Kaittanis C, Boukhriss H, Santra S, Naser SA, Perez JM. Rapid and sensitive detection of an intracellular pathogen in human peripheral leukocytes with hybridizing magnetic relaxation nanosensors. PLoS One. 7:e35326.

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