The disease

The hosts

Johne's disease, also known as paratuberculosis, is a wasting condition of cattle and other ruminants that progressively damages the intestines of affected animals and in cattle results in profuse and persistent diarrhoea. While cattle remain susceptible to infection throughout their life, they are at their most vulnerable in the first few months of life, but the advanced signs of the disease are seen most commonly in animals at three to five years of age. In the United Kingdom the disease occurs as a herd problem in both the dairy and the beef suckler sectors. Significant problems are also found in sheep, goats and deer. Surveillance of wildlife has confirmed that the infection can be found in many species other than wild ruminants. In particular, in Scotland, rabbits have been found to become infected and to pass the organism in their faeces.

The organism

*Mycobacterium avium* subspecies *paratuberculosis* (*Map*) is the bacterium that causes the disease and it is in the same family of bacteria as those that cause tuberculosis and leprosy. *Map* is slow growing and takes several months to grow to detectable levels in the laboratory. Outwith the confines of the laboratory it can only multiply within an animal, but nevertheless survives well in the environment and can be isolated from pasture, slurry and water after periods of up to one year.

The Spread of Infection

The bacterium is passed in large amounts in the faeces of infected animals, but can also be found in milk and particularly colostrum. Calves may be infected in the womb, but are more commonly infected through drinking colostrum and whilst sucking teats that are soiled with faeces. Purchasing colostrum from infected dairy herds is also a potential source of infection. In the dairy herd practices such as pooling colostrum and feeding waste milk to calves that are destined to be herd replacements are considered to be important in the rapid within herd spread that is sometimes observed. Despite the concerns over the disease in other species it is accepted that most herds become infected by the purchase of apparently healthy cattle that have come from infected herds and are already in the early stages of the disease.

Herd dynamics of the disease

As a consequence of the long period of time before the disease becomes apparent in a herd, many other animals within the herd will have been exposed to infection during this time. Therefore the disease will be developing in several other animals. For every diseased animal that is seen in a herd there will be a group where the disease is already affecting their milk output or fertility; there will be a group where the disease is taking hold and the effects will be seen in later years.
Johne’s disease in cattle

Crohn’s disease

There are similarities in the pathology observed in Johne’s disease and the condition known as Crohn’s disease in humans. It has been suggested that the same organism may be involved in both conditions and indeed some researchers claim to be successful in isolating Map from the intestinal lesions in the majority of Crohn’s disease patients they examine. There appear to be differences between the few isolates of Map from human disease that have so far been characterised successfully and those strains of Map found in cattle. No epidemiological study has been carried out to determine the risk factors of this disease with a view to examining a possible link with the disease in domestic ruminants. Therefore it is unclear at present how closely related Crohn’s disease is to Johne’s disease. However as Map has been found in small numbers in pasteurised milk at retail outlets, the Food Standards Agency have advised that the precautionary principle should be observed. This issue is currently causing concern for many of the countries of the world where dairy production is important. To date the beef sectors have been largely unconcerned about this aspect of Johne’s disease. However there is at least potential for the organism to enter the food chain from beef animals, as the infection is not confined to the intestines and spreads through the body in infected white blood cells. There is also the potential for faecal contamination of carcasses at slaughter. The numbers of organisms that may enter the food chain in this way will certainly be small and less likely to survive the processing of meat in contrast to milk. The risk from meat is considered to be insignificant.

DEFRA have produced a document on the control of the disease in the dairy herd that is at the consultation stage (DEFRA 2002).

Diagnosis and diagnostic tests

Once an animal has severe diarrhoea and is losing weight (clinical disease) the disease can be readily confirmed by a blood antibody test (ELISA) and the Map organism can be cultured from the faeces in the laboratory. However diagnosis of the presence of the infection in animals in the silent period of the disease is difficult. They seldom pass detectable numbers of the Map organism in their faeces until they are beyond two years of age. Similarly they tend to produce the antibody that is detected by the blood test relatively late in the disease. In some individuals it may be difficult to confirm the presence of infection in the live animal. This means that animals that have been infected early in life may give negative results for the disease in several annual tests before they eventually test positive. Therefore testing animals at the point of sale or on arrival in their new herd will not prevent the introduction of infected animals to the herd. When considering control this is the single most difficult feature of the disease.

Using the blood antibody test 50% of all infected animals can be detected. This rises to 90% of animals with clinical disease and falls to less than 50% for animals in the early stages of infection. Culture of faeces detects a similar proportion of infected individuals as the blood test but not the same individuals. Therefore, testing blood for antibody and faeces by culture in the same animal gives the highest detection rate but is expensive and is unlikely to be cost effective for whole herd screening on most farms but may be justified for purchased individuals such as bulls.

Prevalence in the UK herd

There is no reliable prevalence data for the UK. The trend in numbers of diagnoses as indicated by Veterinary Investigation Diagnosis Analysis (VIDA) has been upwards. It is of some value to compare the data from other countries (Table 1). The USA national survey indicated a dairy herd prevalence of 21.6%. A point of slaughter survey in the USA found an individual cow prevalence of 2.9%. In the only similar abattoir survey carried out in the UK, workers at Bristol Veterinary School found an individual prevalence of 3.5% in cull cows in the south west of Britain. Extrapolating from this it may be suggested that the herd prevalence of Johne’s disease in the UK dairy herd will be of a similar order to that in the USA. Several surveys at the state level have been carried out in the USA beef herd and herd prevalence figures have been from 30 to 40%.

Table 1: Johne’s disease prevalence (%)

<table>
<thead>
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<th>Herd</th>
<th>Animal</th>
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<th>Beef</th>
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</tr>
<tr>
<td>Canada</td>
<td></td>
<td>5.5 (1991)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Britain</td>
<td></td>
<td>3.5 (1996)</td>
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Significance for the beef herd

**Commercial herds**
Despite the lack of prevalence data it appears to those working with beef cattle that in the past ten years Johne’s disease has become increasingly important. In severely affected herds annual incidences of 5% and even up to 10% of cows affected are not uncommon. The trend towards rearing replacements from within the herd rather than purchasing heifers may serve to reduce the risk of introducing the disease, but once the disease is introduced it will favour the build up of disease. While no attempt to study the exact financial losses in affected herds has been made, modelling of the disease has estimated that the average annual loss due to the disease in a commercial suckler herd to be £1600 per year in a 100-cow herd. Clearly in severely affected herds the losses are far higher.

**Pedigree herds**
The losses are even more difficult to estimate in herds where the animals being culled early may represent particularly valuable bloodlines. In addition there is the concern for the owners of infected herds that they will sell stock that may develop clinical disease a short while after arriving on the purchaser’s farm.

**Breeding replacements**
As added animals from infected herds are the principal way in which the disease is spread commercial herds are at risk through the purchase of dairy bred replacement females and terminal sires from infected herds. Replacement females from other beef herds such as three quarter bred beef animals and traditional beef crosses such as the Galloway/Whitebred shorthorn may also introduce the disease to a herd although impressions are that the risks associated with this are lower.

Significance for the dairy herd

**Commercial Herds**
As for the beef herd the disease appears to be increasing in prevalence. A number of factors may be contributing to this including the importation of high genetic merit heifers from countries with a high prevalence of infection. However the increase in average herd size, compromised cleanliness of cows at calving and feeding pooled colostrum and waste milk ensure that once the disease enters a herd it can quickly become established. The financial loss is due to reduced milk output. In the lactation before the disease becomes apparent cows produce 10% below their potential and in the lactation in which the disease becomes apparent milk production will be reduced to 25% of their potential. Modelling exercises have translated this in to average herd losses of £2600 per 100 cow herd when the milk price is 18 pence per litre. It is important to recognise that losses in severely affected herds will be significantly higher than the estimate provided by the average.

**Pedigree / high genetic merit herds**
Concerns are of a similar nature to those of the pedigree beef herd.

**Breeding replacements**
Where herds are not infected it is important that the few replacement heifers that they may purchase are free from infection. As most herds rear the majority of their own replacement heifers the introduction of infection is likely to lead to a build up of infection in the herd.

Options for control

**Vaccination**
A live vaccine is licensed for use in the UK, but can only be given to animals in the first month of life. This may result in a reduction of clinical disease in infected herds, but will not lead to eradication of infection. Vaccinated animals frequently break down when sold onto other herds, negating the value of this control measure for herds selling breeding replacements. There is also potential for interference with the skin test for tuberculosis. As a result of this, and because of vaccination with a live organism that may be capable of causing disease in humans, live vaccines are not favoured by several countries. Killed vaccines are however used elsewhere and while a positive cost benefit has been reported when they are used, nevertheless the disease remains active in the herd. Research into improved vaccines is being undertaken in many countries, but as with vaccines for tuberculosis significant progress is unlikely in the near future.

**Test and cull**
The main problem with this approach is the long time interval between infection and the time when the animal will test positive. It is therefore not possible to carry out a couple of years of herd testing and remove all positives. In reality a period of three to five years can be expected to pass before
herd owners will see progress. And perhaps a further period of three to five years before the disease disappears from the herd. In addition to removing animals that test positive from the breeding herd it is recommended that offspring from these animals should not be retained for breeding either. This is because calves born to infected mothers are at high risk of being infected. This risk is at its highest for the youngest of the positive cow’s calves. To date no controlled study to assess the success of the test and cull programme to control Johne’s disease has been completed.

Accreditation of freedom from disease
The aim is to provide a pool of tested free herds that will allow commercial producers to buy breeding replacements that are free from disease (or carry a very low risk of introducing the disease). Despite the limitations the biology of the disease imposes on the ability to detect infection in the individual animal, whole herd testing is a reliable way in which to demonstrate freedom from the disease. The power of this is related directly to herd size and the number of years the herd has tested clear. Hence a large herd that has tested free of infection for four years is more likely to be free from the disease than is one of 12 cows that has tested clear of the disease for two years. Under the Cattle Health Certification Standards a certified programme is in place and delivered at the present time by SAC’s Premium Cattle Health Scheme, Hi-Health and Herdcare.

Herd hygiene and biosecurity
Whether the plan is to control the disease or to keep infection out the same management rules apply. Purchased animals should come from sources known to be free of disease (not achievable at present as insufficient accredited stock exists). Failing this added animals may be subjected to an annual screening test. Efforts must be made to reduce the exposure of young stock to faeces from adults. This can be difficult to achieve in both the beef and even in the dairy herd where hygienic management of dry cows and calving cows can be problematic in some herds. Wherever possible water should be piped and ponds and streams should be fenced off. When cattle are housed measures should be taken to minimise faecal contamination of drinking troughs. Control of rabbits is a sensible precaution in the light of current research findings. Limiting contact with sheep is also advisable, but rarely practised.

Control measures specific to the dairy herd
Dry cows should be kept in as clean an environment as possible to reduce to a minimum the faecal contamination on the coats of the cows. There should be calving pens that are cleaned out between each calving with the aim of removing all faecal contamination and birth products. Once the calf has received colostrum from its dam it should be removed from the cow and placed in a rearing area that is not subject to faecal contamination from adult stock. Colostrum should not be pooled as this is an effective way in which to spread infection. Similarly waste milk should not be fed to calves that are to be reared as breeding replacements.

Conclusions
Johne’s disease is of increasing international significance because of the putative relationship to Crohn’s disease in man. At present attention has been focused on the dairy industry. Johne’s disease itself is a significant source of financial loss in beef and dairy herds and appears to be increasing in prevalence. The long time period between infection and the development of the disease and the inability of diagnostic tests to identify infected animals during this “quiet” phase of the disease makes this one of the most difficult cattle diseases to control. The disease poses particular problems for infected herds selling breeding stock. An accreditation programme exists but for progress to be made membership needs to increase significantly in order to provide sufficient replacement stock free of disease.

Acknowledgements
Much of the above is taken from the DEFRA funded project “Assessment of surveillance and control of Johne’s disease in farm animals in GB” carried out by SAC. The full report can be found on DEFRA’s web site (www.defra.gov.uk/animalh/diseases/sac2.PDF). The rest is from Premium Cattle Health Scheme (www.cattlehealth.co.uk) experience with control of the disease.

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