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Livestock Health & Greenhouse Gas Emissions

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Executive Summary

Background

Methane emissions from ruminants are responsible for approximately 50% of the greenhouse gas (GHG) emissions associated with agriculture in Scotland. Reducing the emissions intensity (i.e. the amount of GHG emitted per unit of meat or milk produced) of ruminants is, therefore, key to reducing agricultural emissions in Scotland.

Scottish Government commissioned ClimateXChange to carry out a rapid evidence assessment to explore the potential for eradicating or controlling major livestock diseases in Scotland, as a contribution to reducing the intensity of GHG emissions.

Key findings

- GHG emissions savings were identified for all twelve diseases evaluated, but some diseases proved more tractable than others
- Overall, the evidence suggests that emissions intensity could be reduced through control measures relating to
    - milk yield and cow fertility rates (dairy systems),
    - cow/ewe fertility and abortion rates, calf/lamb mortality and growth rates (beef and sheep systems), and
    - feed conversion ratios, FCR (all systems).
- Three diseases, one from each the major livestock sectors, were considered more cost-effective and feasible to control: neosporosis (beef cattle), infectious bovine rhinotracheitis, IBR (dairy cattle) and parasitic gastroenteritis (PGE; sheep).

Discussion

Endemic, production-limiting diseases are a significant constraint on efficient and sustainable livestock production in Scotland and around the world. Dealing effectively with endemic livestock diseases represents an opportunity to reduce emissions from the livestock sector, often without compromising productivity or farm economics. This report delivers a rapid evidence assessment of the potential contribution that can be made towards reducing the intensity of GHG emissions from Scottish animal agriculture by eradicating or controlling livestock diseases, focusing on the main livestock species, cattle and sheep.

This assessment provided a comparative analysis of the available evidence for the control or eradication of twelve of the major livestock diseases in Scotland in terms of GHG abatement potential, cost-effectiveness and feasibility. This was based on qualitative analysis of the published and grey literature and expert opinion on disease prevalence, impacts on productivity and current control options. This assessment was further underpinned by quantifying the impacts of selected diseases on emissions and production using established GHG modelling methods.

Overall, the evidence suggests that reductions in GHG emissions intensity (EI) could be achieved through the implementation of cost-effective control measures that impact on the parameters EI is particularly sensitive to, i.e. (a) milk yield and cow fertility rates (dairy systems), (b) cow/ewe fertility and abortion rates, calf/lamb mortality and growth rates (beef and sheep systems), and feed conversion ratios, FCR (all systems).

GHG emissions savings were identified for all twelve diseases evaluated, but some diseases proved more tractable than others in terms of the availability of practical diagnostic and control options. Based on comparative disease analysis, we identified a 'Top 3' diseases, one each from the major livestock sectors, to consider for potential eradication and/or government policy intervention. These were neosporosis (beef cattle), infectious bovine rhinotracheitis, IBR (dairy cattle) and parasitic gastroenteritis (PGE; sheep), respectively. Abatement was possible, economically viable and practically feasible for IBR and PGE. Although neosporosis is the major cause of abortion in beef cattle and therefore expected to impact significantly on EI, insufficient data were available to substantiate abatement potential and feasibility of its control.

Approach

Focusing on cattle and sheep, which are the major livestock species in Scotland, we assessed the available evidence for the control or eradication of the major diseases in terms of (i) **abatement potential**, and (ii) **cost-effectiveness** (in both financial terms, and in units of carbon dioxide equivalents (CO_2e) saved compared with business as usual)**.** *Major* is defined as those diseases that are (i) most economically costly, and/or (ii) the most wasteful in GHG emissions intensity (EI; where it is known or can be inferred). The assessment should address those diseases listed in Table 1, below, as a minimum.

Table 1. Coverage of endemic livestock diseases by host species*

Cattle	Sheep	Both
Johne's Disease	Sheep scab	Liver fluke
Leptospirosis	Foot rot	**Gastrointestinal nematodes**
IBR	Jaagsiekte	**Lungworm**
Mastitis	**Chlamydia**	
Lameness	**Toxoplasmosis**	
Neosporosis		

*Additions to original SG/CXC project specification highlighted **in BOLD**.

Contents

List of Abbreviations

AP - Abatement potential, i.e. the reduction in total emissions (in kg or t)

AR - Abatement rate, i.e. the reduction in emissions per ha or per farm etc.

CE - cost-effectiveness

CHeCS - cattle health certification standards

CO_2e - CO_2 equivalent

CW - carcass weight

DIVA – differentiation of infected and vaccinated animals

DM - dry matter

ELISA – enzyme linked immunosorbent assay

FCR - feed conversion ratio, e.g. kg feed in/live weight gain

GHG – greenhouse gas

IBR – infectious bovine rhinotracheitis

LFA – less favoured area

LW - live weight

MACC - marginal abatement cost curve

OPA – ovine pulmonary adenocarcinomatosis

PCR – polymerase chain reaction

PGE – parasitic gastroenteritis

QMS – Quality Meat Scotland

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1. Introduction and Background

Efficient, sustainable and profitable livestock production is important in Scotland. Approximately 80% of Scotland's agricultural land is classed as Less Favoured Area (LFA), ideally suited to livestock grazing, which in turn is responsible for the appearance of much of the country's landscape, a key asset to the Scottish Tourism industry. The Scottish red meat sector is also a key contributor to the rural and national economy and the world-renowned Scottish Food and Drink industry. However, ruminant production also contributes significantly to the carbon footprint of livestock farming and The Climate Change (Scotland) Act 2009 sets ambitious emission reduction targets for Scotland, requiring all sectors, including agriculture, to reduce GHG emissions to mitigate anthropogenic climate change.

1.1 Livestock and Greenhouse Gases (GHGs)

According to the UK National Atmospheric Emissions Inventory, Scottish farming produced 9163 kilotonnes (kt) CO_2e in 2013 (not including land use change), or 17% of Scotland's territorial emissions (Figure 1). Cattle and sheep produced 99% of the enteric and manure GHG emissions within Scotland and a significant percentage of the soil emissions (these are not quantified for Scotland in the UK inventory submission, but in Ireland's 2013 submission urine and dung deposited by grazing animals produced just under half of the direct nitrous oxide (N_2O) from managed soils). Emissions of enteric and manure methane from sheep and cattle accounted for just over half (52%) of Scotland's total agricultural GHG emissions in 2013 (Salisbury et al., 2015). There is also still some debate about the relative contribution of grassland as a carbon sink and whether this is adequately captured in the CO_2 calculations. While these figures do not include important off-farm sources of emissions (e.g. the production of feed and other inputs), they illustrate the importance of ruminants in this context (Figure 2). Reducing the GHG emissions intensity (EI; the amount of GHG emitted per unit of meat or milk produced) of ruminants is, therefore, key to reducing agricultural emissions in Scotland.

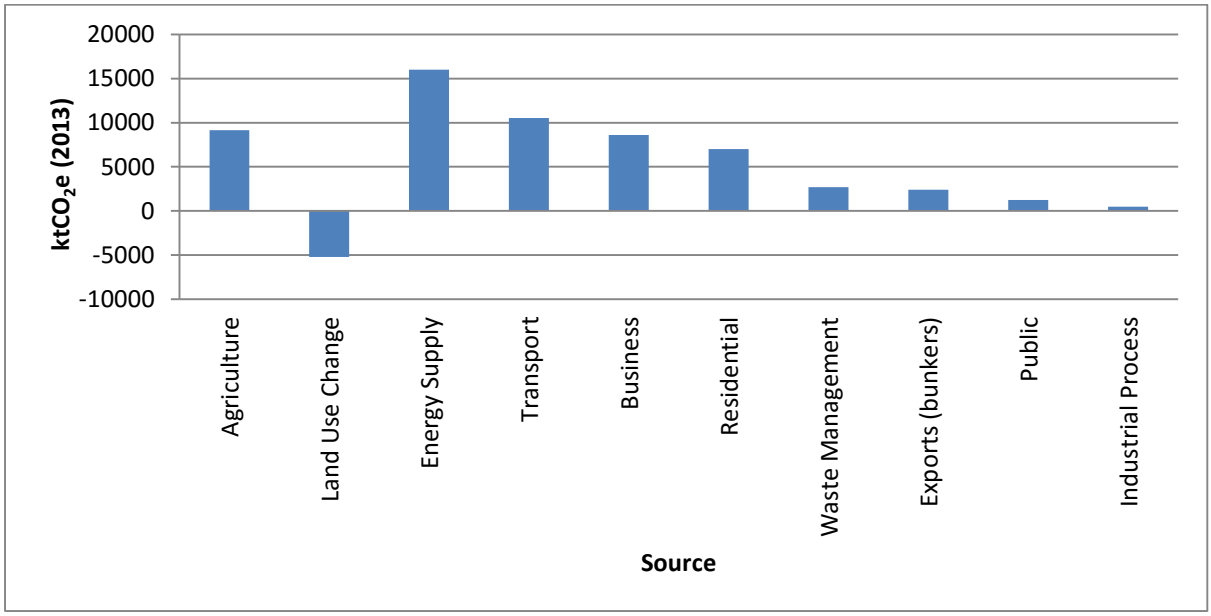


Figure 1. GHG emissions by source: Scotland, 2013 (based on Salisbury et al. 2015)

The level of GHGs emitted by an animal depends on a number of factors, including level of feed intake, quality of feed and efficiency of feed conversion. Monogastric livestock (e.g. pigs and poultry) are more efficient in terms of GHG emissions per unit product than ruminants (e.g. sheep and cattle) but Scotland's LFA are not suitable for monogastric livestock production. GHG emissions represent a loss of energy, hence improving animal performance can often lead to improvements (i.e. reductions) in EI. The biological efficiency of livestock production can equally be optimised through improvements in animal health as diseases impact negatively on EI. The role of improving animal health in reducing GHG emissions from agriculture has recently been

recognised by the Scottish Government (SG). In her update to SG on Scotland's progress in tackling climate change, Dr Aileen McLeod, Minister for Environment, Climate Change and Land Reform announced a number of measures to tackle climate change including 'new action to reduce wastage by improving livestock health'. http://news.scotland.gov.uk/News/Climate-change-action-heats-up-19c8.aspx (last accessed 24 FEB 2016).

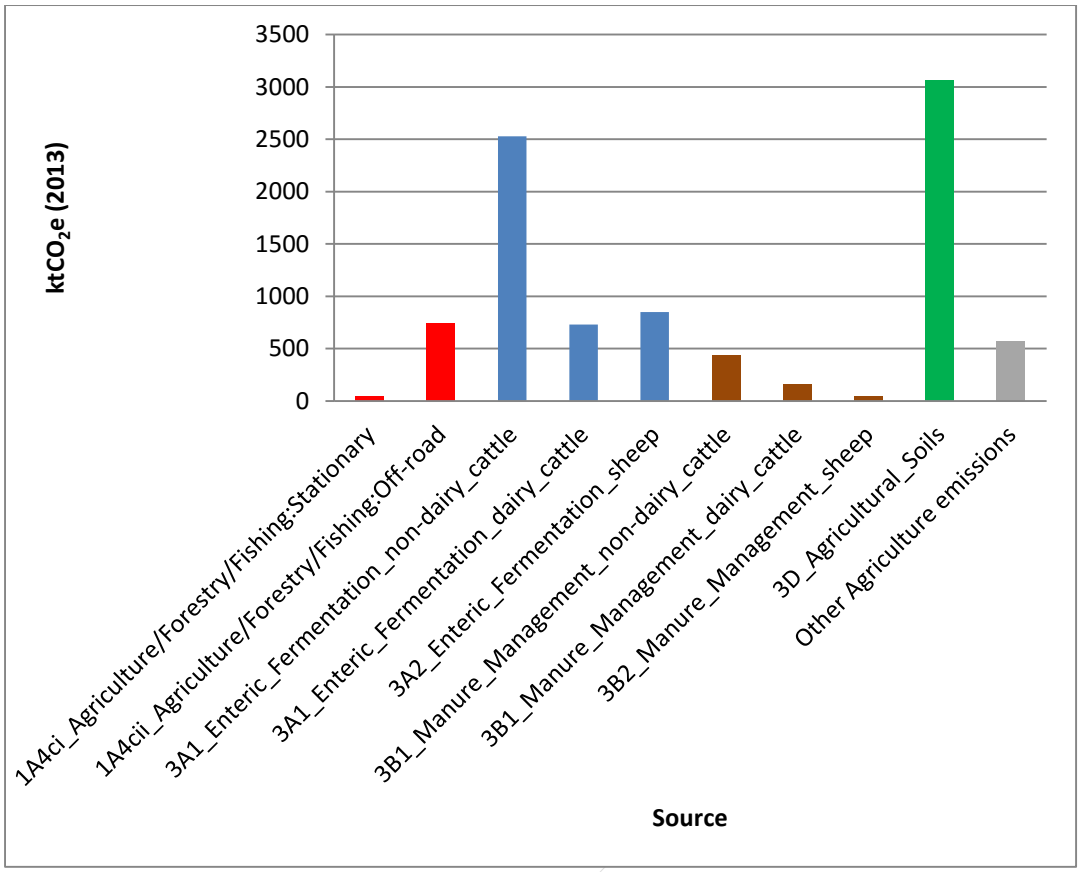


Figure 2. Agricultural GHG emissions by source: Scotland, 2013 (based on Salisbury et al. 2015)

While the connections between animal health, mortality/morbidity, nutrition, production and GHG emissions appear obvious and intuitive, only a few studies have directly addressed this topic (Hospido and Sonesson, 2005; Stott et al., 2010; Guelbenzu and Graham, 2013; ADAS, 2014; Skuce et al., 2014; Eory et al., 2015b). As a consequence, there are relatively few data available to support the link between animal health, productivity and GHG EI. Such studies are logistically challenging to perform and complicated by the fact that grazing livestock are naturally infected with multiple pathogens at the same time, so attributing production and/or GHG emissions effects to individual health conditions is difficult. However, animals emitting GHGs while not growing or producing efficiently would be expected to add significantly to a livestock farm's environmental footprint. GHG emissions produced while the animal is growing become a net loss to the system if the animal dies before its productive value is realized or if the value of that product, whether that is a litre of milk, a kg of meat or a healthy lamb or calf, is reduced due to poor animal health status.

One of the most comprehensive studies in this area to date has been carried out recently as part of a large DEFRA/AHVLA project, FFG1016 (ADAS, 2014). In that study, the authors addressed the impact of ten cattle health 'conditions' on GHG emissions per unit product (milk) in UK dairy cattle. The study utilized the Cranfield Life-Cycle Assessment (LCA) model to calculate GHG emissions associated with healthy versus affected cattle in terms of mortality, morbidity, lost production etc. The study also modelled the GHG emissions associated with veterinary, physical or management intervention(s) designed to control infection e.g. farm visits, fencing, production of a vaccine/drug etc. All conditions had a negative impact on GHG emissions per unit milk output. The individual conditions produced a range of associated GHG emissions compared to healthy cattle, with the

lowest being calf scours at <0.5% and the highest, Johne's disease, at 24%. The impacts for BVD and infertility were also high, in the region of 16-20%, as was liver fluke at 10%. Mastitis proved to be the most intractable condition and BVD the most tractable. In this report, we have concentrated on diseases of beef and dairy cattle as well as sheep within a Scottish context, hence our selection of diseases differs from that covered in ADAS report (Annex 1). Because an official eradication programme is already in place in Scotland, BVD is not considered in our report.

The impacts of endemic disease on production efficiency

There have been recent concerns about incursions of 'exotic' diseases, such as Foot and Mouth Disease, Bluetongue and Schmallenberg. However, the most significant constraint on efficient and sustainable livestock production in the UK, now and in the short-to-medium term, comes from endemic diseases of livestock, i.e. diseases that are routinely present in many herds or flocks. These diseases can be caused by viral, bacterial and parasitic pathogens, and some syndromes, such as lameness, infertility and calf scours, may have non-infectious causes. Some if not all have life-stages in the environment, so their prevalence, seasonality and geographic spread can be affected by climatic conditions, farm management practices and land use strategies. These diseases can affect single or multiple host species, usually as multi-pathogen infections and sometimes have wildlife or environmental reservoirs making them difficult, if not impossible, to eradicate. They also vary in prevalence in space and time, from farm to farm as well as within and between years.

Endemic diseases are production-limiting and can impact on the biological efficiency and productivity of livestock in a number of ways. They can cause a spectrum of outcomes ranging from sub-clinical disease, where the impact on productivity is insidious and may be difficult to diagnose, to clinical disease, where disease is visible and mortalities may occur. Some diseases have a short but significant impact during their acute phase, others become chronic with long-term impacts on production, fertility, feed-conversion or culling. Examples of losses include (i) fewer units of product e.g. milk, meat or wool; (ii) animals taking longer to reach their target market weight; (iii) delayed onset and reduced quality of production e.g., for milk; (iv) lost production i.e. lambs or calves aborted due to infection; (v) premature culling; (vi) waste of animal products condemned at abattoir; (vii) reduced reproductive performance; or (viii) premature death of animals.

This report aims to deliver a rapid evidence assessment of the potential contribution that can be made towards reducing the GHG EI from Scottish animal agriculture by eradicating or controlling the major endemic livestock diseases, focusing on the main livestock species, cattle and sheep. The assessment provides a comparative analysis based on the available evidence in the published and grey literature, expert opinion and well-established GHG modelling methods.

2. Methodologies

The authors consulted the published and grey literature on livestock disease and impacts on GHG emissions. We then sought opinion and specific disease information from acknowledged experts, on areas such as health and welfare implications, prevalence, production effects, climate change impact/implications, mitigation and adaptation options, disease control options and likely costs. This information was collated and is presented as disease-specific proformas (Annex 2).

Quantifying the impacts of disease on emissions and production

For the 'Top 3' case studies, a Microsoft Excel version of GLEAM (the Global Livestock Environmental Assessment Model, see MacLeod et al. 2016; see Annex 3 for details) was used to compare emissions and production between a healthy herd/flock and one with infection or disease.

3. Results

The expert opinion and specific disease information was used to generate the comparative table below (Table 3), synthesizing available data on disease impacts, control options and feasibility of eradication.

Some diseases will have more GHG abatement potential than others based on their prevalence, impact on infected animals etc. However, decisions on which diseases to prioritise for control and/or eradication must also take into account the cost-effectiveness and feasibility of GHG mitigation measures in practice. Such decisions are also compounded by the multi-factorial nature of some of these conditions, most notably mastitis and lameness, where infectious agents are only one possible cause, among many, that may contribute. For this reason, mastitis and lameness have not received as much attention as other diseases in this report and do not feature in the proforma section (Annex 2). Examples of diseases that are difficult to control include Johne's disease and liver fluke, both of which have diagnostic tests with severe limitations and environmental reservoirs. Based on the semi-quantitative analysis of information from Table 3 (details in Annex 2) and the sensitivity analysis (Annex 4), the 'Top 3' consisted of neosporosis in beef cattle (major cause of abortion), IBR in dairy cattle (significant impact on milk production; eradication feasible) and parasitic gastroenteritis, PGE (impact on growth and FCR) in sheep.

Disease name	Pathogen	Host affected				Impact				Control options					Wildlife reservoirs	Eradication feasibility
		Cattle	sheep	goats	Other	Reproduction	growth	production	waste	Treatment	Diagnostics	Vaccine	Management	Other		
Neosporosis	Parasitic	✓	×	×	✓	C	C	C	C	×	C	×	C	C	×	✓?
IBR	Viral	✓	×	×	×	C	ND	C	C	×	C	C	C	C	×	✓
Parasitic gastroenteritis (PGE)	Parasitic	✓	✓	✓	✓	C,S	C,S	C,S	C,S	C,S	C,S	×	C,S	C,S	✓	×
Liver fluke, Fasciolosis	Parasitic	✓	✓	✓	✓	C,S	C,S	C,S	C,S	C,S	C,S	×	C,S	×	✓	×
Parasitic bronchitis, lungworm	Parasitic	✓	✓	✓	✓	C,S	C,S	C,S	C,S	C,S	C,S	C	C,S	×	×	✓?
Leptospirosis	Bacterial	✓	×	×	✓	C	C	C	C	C	C	C	C	×	✓	✓?
Sheep scab	Parasitic	×	✓	×	×	S	S	S	S	S	S	×	S	×	✓	✓?
Johne's	Bacterial	✓	✓	✓	✓	C,S	C,S	C,S	C,S	×	C,S	S	C,S	×	✓	×
Toxoplasmosis	Parasitic	×	✓	✓	✓	S	ND	ND	ND	×	S	×	S	×	✓	×
Lameness	Multifactorial	✓	✓	✓	×	C	ND	V	C	C, S	C, S	×	C, S	C, S	×	×
Footrot	Bacterial	×	✓	×	×	S	S	S	ND	S	S	S	S	S	×	×
Mastitis	Bacterial/Multifactorial	✓	✓	✓	×	C	S	C, S	C, S	C, S	C	C	C, S	C	×	×
Chlamydiosis	Bacterial	✓	✓	✓	✓	C,S	×	ND	C,S	C,S	C,S	S	C,S	×	✓	×
Jaagsiekte, OPA	Viral	×	✓	✓	×	ND	ND	S	S	×	S	×	S	S	×	✓?

Table 2. Comparative table of endemic livestock diseases, production impacts and control options (details in Annex 2)

Key - C, impact in cattle; S, impact in sheep; ND, no data; V, variable (positive and negative associations reported); ✓? feasibility of eradication uncertain (eradication is technically feasible but difficult in practice)

Top 3 – Neosporosis

Disease summary - Neosporosis, caused by the protozoan parasite *Neospora caninum*, is the primary cause of abortion in beef and dairy herds in the UK. It has high economic impact with loss of the calf, less milk produced and higher reproductive costs.

Aborted calf foetus as a result of neosporosis, showing loss at an advanced stage of pregnancy (Image: Moredun)

Justification - This is a disease of primarily one livestock host species (cattle), is a single pathogen disease and has a number of control options e.g. good diagnostics and management options with no known wildlife reservoirs. Dogs play a key role in its transmission but dogs are generally under the control of humans and can be included in control strategies.

Impact of disease on Emissions Intensity – The main impact in beef cattle is through reduced birth weight, reduced liveweight gain and reduced feed conversion ratios. The economic effect of *Neospora* infection is also associated with the cost of abortion, either directly as the loss of a calf but, just as importantly in the dairy industry, failing to get a cow back into milk, which may result in her experiencing prolonged dry periods.

GHG abatement potential – Emissions associated with non-productive pregnancy. AP is estimated at 2.2 to 4.5%, depending on model assumptions and within-herd prevalence (Annex 3). Considering that non-dairy cattle are the major contributors to GHG from animal agriculture (Figure 2), a modest reduction in EI here could have a bigger impact than potentially larger proportional reductions elsewhere.

Control strategies and effects - Preventing cow-to-calf transmission by excluding infected animals from breeding, preventing cattle-to-dog transmission by keeping afterbirths and foetal material away from dogs, and preventing dog-to-cattle transmission by keeping dog faeces away from cattle, cattle feed and grazing areas. Introduction into a herd may occur through purchase of cattle. Current diagnostic tests cannot detect all infected animals reliably, but purchase from accredited sources reduces the risk of introduction.

Feasibility/cost effectiveness – Neosporosis was added to Cattle Health Certification Standards (CHeCS) in March 2015, following evidence of rising levels of infection in the UK and Ireland. It is the first new disease in 15 years to be added to CHeCS, the regulatory body for cattle health schemes in the UK and Ireland, and joins Johne's disease, BVD, IBR and Leptospirosis.

Policy options - Public awareness campaigns to alert dog walkers to the risk their dog poses to grazing cattle and to ask them to pick up after their dogs in the countryside. Given uncertainties in prevalence estimates, a sero-survey of *Neospora* in Scotland would be merited, to more accurately gauge the GHG savings that could be made through improved control/eradication. Mathematical models suggest that eradication from herds is possible, but often not profitable. Hence, financial incentives may be needed to encourage disease control for the sake of curbing GHG emissions.

Top 3 – Infectious Bovine Rhinotracheitis (IBR)

Disease summary – Infectious Bovine Rhinotracheitis (IBR) is a disease of the upper respiratory tract and the lungs that is caused by bovine herpes virus 1, leading to pneumonia and, sometimes, death. The virus may also cause poor fertility and a severe drop in milk yield. IBR has been eradicated from parts of Europe and forms a barrier to export.

Justification - This is a disease of primarily one livestock host species (cattle), is a single pathogen disease and has a number of control options e.g. good diagnostics, vaccine and management options with no known wildlife reservoirs. Examples of successful eradication from other countries exist.

Impact of disease on Emissions Intensity - The main impact in dairy cattle is through the cost of abortion, either directly as the loss of a calf or failing to get a cow back into milk, which may result in her experiencing prolonged dry periods. Significant reductions or complete cessation in milk yield. Mortalities in adult cattle.

Naso-pharyngeal lesions associated with IBR in cattle. (Image: http://homepage.usask.ca/

~vim458/virology/studpages2009/VirusWebsite/ibr_nose.jpg

GHG abatement potential – Emissions associated with lost production i.e. milk yield, fertility, abortion and mortality. Several control measures are available, and each of these measures would be likely to lead to a reduction in EI of milk of approximately 1.5 to 3% (Appendix 3).

Control strategies and effects – Diagnosis (PCR, ELISA) to detect infection (individual & herd); DIVA (differentiation of infected and vaccinated animals) vaccination to prevent infection and transmission; management options e.g. fencing, avoid co-grazing.

Feasibility/cost effectiveness – IBR has been eradicated previously in several countries through test & cull strategies, often in combination with the use of a marker vaccine; semen screening, movement restrictions.

Policy options - Several European countries or regions have eradicated IBR, or have compulsory or voluntary eradication schemes in place. In the UK, individual herds can become accredited through CHeCS. However, voluntary control or vaccination programmes are unlikely to lead to eradication at national level. Coordinated and compulsory control would be needed to achieve national IBR-free status.

Top 3 – Parasitic gastroenteritis (PGE)

Diarrhoea due to PGE (image: Moredun)

Disease summary - Parasitic gastroenteritis (PGE) is a dose-dependent condition (i.e. influenced by worm burden) caused by a range of parasitic roundworms that impact significantly on the performance and productivity of livestock, especially small ruminants.

Justification - This is a disease complex but is highly prevalent on Scottish/UK farms. Has major impact on productivity and farm economics. There are practical diagnostic, treatment and management options.

Assumptions about disease impact - Although parasite populations tend to be over-dispersed i.e. most of the infection is harboured by relatively few host animals (the '80:20 rule'), in reality, it is more likely that 100% of stock carry infection to some extent. The actual impact of infection is complicated by the fact that some host animals will continue to be relatively productive in the face of a parasite challenge, whereas others become less productive and/or display clinical disease. This host effect is the basis of breeding for host resistance/resilience and also for targeted (selective) treatment strategies.

GHG abatement potential – Emissions are associated with lost production i.e. reduced liveweight gain, longer finishing times, premature culling of infected stock, mortality. Reduction of PGE in sheep would seem to have a significant technical abatement potential, provided the parasite burden can be reduced in a cost-effective way. If the proportion of sheep affected by PGE were to be reduced from 20% to 0%, this would result in a reduction in EI of sheepmeat of ca. 9% in all production systems (Appendix 3).

Control strategies and effects – Practical cost-effective diagnostics, effective treatments (in absence of resistance); grazing management strategies; alternative strategies e.g. breeding, nutrition

Feasibility/cost effectiveness – Optimising anthelmintic treatments can be readily achieved through increased monitoring, evidence-based decision making and improved treatment administration e.g. accurate weighing of animals and targeted (selective) treatment strategies.

Policy options - Farmer awareness campaigns on anthelmintic efficacy, optimising treatments and sustainable drug usage. Promotion of targeted (selective) treatment strategies to encourage best practice for sustainable parasite control. Financial incentives to purchase electronic identification devices and portable weighing equipment to facilitate this.

4. Discussion

This rapid evidence assessment indicates that improving livestock health represents an opportunity to reduce GHG emissions from animal agriculture in Scotland. Overall, the available evidence suggests that reductions in EI could be achieved through the implementation of cost-effective control measures that impact on the parameters EI is particularly sensitive to, i.e. (a) milk yield and cow fertility rates (dairy systems), (b) cow/ewe fertility and abortion rates, calf/lamb mortality and growth rates (beef and sheep systems, and FCR (all systems).

There are a number of important limitations and assumptions implicit in the disease-specific inputs into the GHG model calculations. For most endemic diseases, there is a complete lack of active surveillance, with limited passive surveillance and inconsistent reporting. Without knowing the prevalence and incidence of individual diseases, the likely impact of control on GHG emissions cannot be predicted accurately. Furthermore, where data on prevalence

or incidence are available from peer-reviewed or grey literature, they do not always reflect recent changes in disease epidemiology and occurrence, e.g. as a result of vaccine use or climate change (Kenyon et al., 2009). Where possible, data were used from Scotland, the UK, the British Isles, or Europe (in that order), rather than from other countries, but in many cases, we were reliant on estimates of prevalence, incidence and production impacts derived from other nations and countries, where climatic conditions, land use, and farm management practices may be considerably different from those in Scotland. Even within the UK, major differences exist, e.g. between dairy and beef farming, between lowland and hill sheep farming conditions and between different geographic regions. As a result, quantifying the abatement potential and cost-effectiveness of health improvement measures presents a variety of challenges. Performing the analysis using (national) averages can furthermore obscure cost-effective measures. Also, some potentially important disease impacts are poorly understood. For example, feed conversion rates, which are a key determinant of EI, are not routinely measured for ruminants. It would be useful to undertake a literature review, challenge or field studies of the effect of key diseases on the feed conversion rates of dairy cattle, beef cattle and sheep. Some data on this may be generated within Workpackage 2.2.9 of the 2016-20 RESAS programme.

Another consideration is the interaction between multiple pathogens e.g. gastrointestinal nematodes and Johne's or liver fluke and bovine TB (Claridge et al., 2012), and the implications of controlling one disease on the potential outcome and impact of another. We have focused our report on those diseases that are, at least clinically, attributable to a single pathogen (virus, bacterium, parasite) or multiple, closely-related pathogens, as in the case of PGE, where the causative agents are known and their epidemiology reasonably well understood. In real life, production is affected by many parameters, including breed, nutrition, co-infections, etc. Hence, estimated production impacts may not always be exclusively attributable to the organism or disease of interest. One of the challenges of estimating the abatement potential of multiple diseases is in understanding the net effect of implementing multiple measures for different diseases. The improvements in performance will not always be additive, which raises the possibility of double counting of abatement. Double accounting may also occur within the calculations for individual diseases, e.g. when early embryonic death due to *Neospora* infection is included as a reduction in fertility as well as an extension of the average calving interval. Interactions between measures are a common challenge in GHG mitigation analysis (see MacLeod et al. 2015, p17). In order to address this, ADAS (2014) assessed the total abatement from improving cattle health using a scenario-based approach to quantify the effects of a 20% and 50% movement from a reference to a healthy cattle population. A similar approach was used by Eory et al. (2015, p107) to assess the abatement potential of improving UK sheep health. Top-down estimates of the abatement potential of improving animal health (total abatement potential) provide a benchmark against which to compare bottom-up estimates of individual diseases. If the top-down estimate is lower than the sum of bottom-up estimates, this suggests the possibility of double accounting in the latter. However, there is also the possibility of positive synergies between measures, e.g. a reduction in GIN may lead to a reduction in PGE and in flystrike.

In addition to uncertainties about occurrence and production impacts, there is still a lack of information relating to the effects of animal health status on GHG emissions from livestock. There are numerous studies in the scientific literature describing direct production losses and economic impacts of disease in food-producing animals, but these were never designed to provide input into GHG models. However, it is now recognised that animal health status has direct and indirect effects on GHG emissions per unit of livestock product and that dealing effectively with disease in particular, whether it be exotic or endemic, can help reduce the carbon footprint of livestock farming (Gill et al., 2010). To make the outcome of GHG models more accurate, data should be gathered on the distribution of prevalence and impact, so that we can calculate the distribution of AP and CE rather than talking in terms of national averages. Disease is rarely evenly distributed across farms, and use of averages may mask abatement potential on a subset of farms where high prevalence or "abortion storms" may result in significant impacts. The uptake of control measures across farms, e.g. most farms taking up some control measures vs. some farms taking up most control

measures, may also impact on AP. This will help to inform the need for policy as an instrument to influence uptake of control measures.

Sustainable control of endemic, production-limiting disease represents a potential win-win situation, with wins from an economic and environmental standpoint. Farm management measures that are likely to improve the biological efficiency of livestock production are also most likely to improve the economics of livestock farming and reduce its environmental impact. However, a number of caveats apply, as described in the policy section below. If GHG mitigation options are to be widely taken up by livestock farmers, as they must be to ensure sufficient impact, then they must be relatively straightforward, practical and cost-effective to implement. This latter aspect can be estimated through economic modelling, however, animal health status rarely features in calculations that underpin Marginal Abatement Cost Curve (MACC) analysis for the agricultural sector. Where animal health has been included in MACC analyses, this has tended to be an overall improvement in the health status of the flock or herd as a whole, rather than being specifically attributed to any given disease (e.g. Eory et al. 2015b). Inclusion of disease-specific information would allow policy makers to make informed decisions about the mitigation options available for reducing GHG emissions from animal agriculture, in the relatively short-term, balanced against the contribution of livestock products to local and global food security.

5. Policy implications

When considering prioritisation of diseases for control or eradication, a number of factors should be taken into account, such as economic impact, impact on biological efficiency, and availability of control tools. For example, Johne's disease has a major production impact and a relatively large GHG abatement potential (ADAS, 2014), but there is no treatment, no vaccine, diagnostic tests have poor sensitivity, and there are wildlife and environmental reservoirs of the causative agent, limiting our ability to control the disease. At the other end of the spectrum, eradication strategies have been implemented successfully in several countries for IBR, and many tools for control or eradication of this disease are available.

Another major consideration for control or eradication is the extent of farmer uptake. For example, a national IBR eradication campaign in The Netherlands was abandoned when the use of contaminated vaccines resulted in disease outbreaks. This undermined farmers' willingness to participate in the campaign and IBR control was made voluntary rather than compulsory. However, model calculations have clearly demonstrated that a voluntary vaccination campaign is highly unlikely to result in eradication. In a situation like this, there is a clear place for national disease control policy.

National policy may also have an important role to play when the cost of disease control is similar to or even outweighs the economic impact of a disease, as for example in the case of neosporosis. At an individual farm level, benefits of neosporosis control may not outweigh costs, but at a national level, the balance may be different because of the GHG emissions abatement potential of a disease control strategy.

Because cooperation of farmers and farmers' organisations is essential to the success of any control programme, stakeholder initiatives may need to be taken into account when setting priorities for disease control or policy intervention. When selecting a sheep disease for the 'Top 3' control targets, the economic and environmental impact of PGE was deemed to outweigh that of footrot or enzootic abortion of ewes (chlamydiosis). The difference, however, was small, and may fall within the range of uncertainty surrounding EI estimates. In such a situation, it may be advantageous to build on existing industry initiatives. For example, Quality Meat Scotland conducted a footrot and lameness control campaign in autumn 2015 (Anon; 2015). In a series of farmer meetings, the costs of lameness and the industry-accepted five-point sheep lameness reduction plan were discussed. Such initiatives could be a springboard for policy-supported action to encourage disease control and reduce GHG emissions.

A final but important policy consideration is the risk posed by disease eradication. Although endemic diseases affect production, part of the reason they do not receive as much attention as incursions of exotic diseases, e.g. Foot and Mouth Disease, Schmallenberg or Bluetongue, which may cause major outbreaks, is that such outbreaks are unusual for endemic diseases. This is in part because our herds and flocks have frequent exposure to the pathogens causing those diseases, providing a level of immunity and protection to many of them. By eradicating a disease, we essentially turn an endemic disease into an exotic disease. This does have production benefits and reduces GHG emissions from livestock production, but upon reintroduction of a disease into a herd, flock or country, major outbreaks of disease may occur once the population is immunologically naive to it. Such outbreaks are low risk, high impact events and there may be a role for Government in underwriting the risk of such events for individual farmers.

6. Recommendations

- Encourage improvement in livestock health to contribute to reduction in GHG emissions from animal agriculture in Scotland.
- Discuss prioritisation of target diseases with industry stakeholders, e.g. NFUS, QMS.
- Improve evidence base underpinning GHG model calculations for priority diseases, e.g. neosporosis in beef cattle, IBR in dairy cattle, and PGE or footrot in sheep, including estimation of impact on feed conversation ratios.
- Consider relative improvements in EI per industry sector as well as contribution of each industry sector to overall GHG emission from animal agriculture in Scotland.
- Account for heterogeneity in disease prevalence and incidence and uptake of control measures
- Consider intended and unintended consequences of disease control and eradication, including vulnerability to reintroduction of disease through animal movements.
- Consider feasibility of control and eradication, including examples of similar efforts in other nations or countries.

7. References

ADAS (2014) Study to model the impact of controlling endemic cattle diseases and conditions on national cattle productivity, agricultural performance and greenhouse gas emissions, Report No Defra AC0120. http://randd.defra.gov.uk/Default.aspx?Menu=Menu&Module=More&Location=None&ProjectID=17791&FromSearch=Y&Status=3&Publisher=1&SearchText=ghg&SortString=StartMth&SortOrder=Desc&Paging=10#Description (last accessed 22 FEB 2016)

Anon (2015) Stamping out footrot. http://www.thescottishfarmer.co.uk/livestock/sheep/stamp-out-footrot.27754352 (last accessed 26 FEB 2016)

Claridge, J., Diggle, P., McCann, C.M., Mulcahy, G., Flynn, R., McNair, J., Strain, S., Welsh, M., Baylis, M. and Williams, D.J., (2012). *Fasciola hepatica* is associated with the failure to detect bovine tuberculosis in dairy cattle. *Nature communications*, *3*, 853.

Eory, V., MacLeod, M., Rees, B. (2015a) Short policy briefing on the abatement and cost-effectiveness of selected greenhouse gas mitigation actions Edinburgh: ClimateXChange

Eory, V., MacLeod, M., Topp, C.F.E., Rees, R.M., Webb, J., McVittie, A., Wall, E., Borthwick, F., Watson, C., Waterhouse, A., Wiltshire, J., Bell, H., Moran, D., Dewhurst, R. (2015b) Review and update the UK agriculture MACC to assess the abatement potential for the 5th carbon budget period and to 2050: Draft final report London: The CCC https://www.theccc.org.uk/publication/the-fifth-carbon-budget-the-next-step-towards-a-low-carbon-economy/

Gill, M., Smith, P., & Wilkinson, J. M. (2010). Mitigating climate change: the role of domestic livestock. *Animal*, *4*(3), 323-333.

Guelbenzu, M. and Graham, D. (2013) Prevalence of BVD in Northern Ireland dairy and suckler herds, Report No Booklet 28, Agri-Food and Biosciences Institute.

Hospido, A. and Sonesson, U. (2005) The environmental impact of mastitis: a case study of dairy herds. Science of the Total Environment 343, 71-82.

Kenyon, F., Sargison, N. D., Skuce, P. J., & Jackson, F. (2009). Sheep helminth parasitic disease in south eastern Scotland arising as a possible consequence of climate change. *Veterinary Parasitology*, *163*(4), 293-297.

Leip**,** A., Weiss, F., Wassenaar, T., Perez, I., Fellmann, T., Loudjani, P., Tubiello, F., Grandgirard, D., Monni, S., Biala, K. (2010): Evaluation of the livestock sector's contribution to the EU greenhouse gas emissions (GGELS) – final report. European Commission, Joint Research Centre. http://ec.europa.eu/agriculture/analysis/external/livestock-gas/full_text_en.pdf (last accessed 22 FEB 2016)

MacLeod, M., Vera Eory, Guillaume Gruère and Jussi Lankoski (2015), "Cost-Effectiveness of Greenhouse Gas Mitigation Measures for Agriculture: A Literature Review" OECD Food, Agriculture and Fisheries Papers, No. 89, OECD Publishing, Paris. http://dx.doi.org/10.1787/5jrvvkq900vj-en

Salisbury, E., Thistlethwaite, G., Goodwin, J., MacCarthy, J. (2015) Emissions of the basket of 7 Kyoto GHGs according to Devolved Administration 1990-2013 (xl file available at: http://naei.defra.gov.uk/reports/reports?report_id=810) (last accessed 22 FEB 2016)

Skuce, P., Houdijk, J., Hutchings, M., Waterhouse, T., MacLeod, M. (2014) The Impact of Animal Health Status on Greenhouse Gas Emissions from Livestock http://www.knowledgescotland.org/briefings.php?id=378

Stott, A., MacLeod, M. and Moran, D. (2010) Reducing greenhouse gas emissions through better animal health pp. 1-8. SAC.

Annex 1: Comparison of the scope of this project and the DEFRA/AVHLA study of cattle diseases (ADAS 2014)

	ADAS (2014)	This project	Notes
Cattle diseases			
Lameness	Yes	No	Multifactorial, including infectious and non-infectious causes; ADAS (2014) indicated high cost of abatement
BVD	Yes	No	Eradication policy already in place in Scotland
Calf pneumonia	Yes	No	ADAS (2014) indicated small abatement potential
Calf scour	Yes	No	Sign rather than a disease, with many potential underlying causes, including infectious and non-infectious etiologies
Liver fluke, fasciolosis	Yes	Yes	Increasing prevalence reported across Scotland, major health problem in both cattle and sheep, good abatement potential (ADAS, 2104)
IBR	Yes	Yes	Highly prevalent in both beef and dairy herds, significant production losses. Moderate abatement potential (ADAS, 2104)
Infertility	Yes	No	Effect rather than a disease; multifactorial, including infectious and non-infectious causes. Individual infectious underlying causes addressed in current report (e.g. IBR, fasciolosis, neosporosis)
Johne's	Yes	Yes	Ranked highest in ADAS report 2014, large abatement potential
Mastitis	Yes	No	Multifactorial, ADAS (2014) indicated moderate abatement potential
Salmonella	Yes	No	ADAS (2014, p142) indicated moderate abatement potential and likely lower impact in Scotland
Neosporosis	No	Yes	Major cause of infertility in cattle in Scotland
Parasitic gastroenteritis (PGE)	No	Yes	Major cause of production loss in cattle in Scotland, increasing prevalence
Parasitic bronchitis, lungworm	No	Yes	Increasing prevalence reported in Scotland
Leptospirosis		Yes	Zoonotic disease (human health risk); requested by SG
Sheep diseases			
Sheep scab	No	Yes	Eradication policy already in place in Scotland
Footrot	No	Yes	"Stamp out Footrot" campaign launched by Quality Meat Scotland (SEP 2015). Considered major industry concern
Jaagsiekte, OPA	No	Yes	Farmer/industry concern in Scotland
Chlamydiosis	No	Yes	Major cause of infertility and abortion in sheep in Scotland
Liver fluke, fasciolosis	No	Yes	Increasing prevalence reported across Scotland, major health problem in both cattle and sheep
Parasitic gastroenteritis (PGE)	No	Yes	Major cause of production loss in sheep in Scotland, increasing prevalence linked to climate change.
Parasitic bronchitis, lungworm	No	Yes	Increasing prevalence reported in Scotland
Toxoplasmosis	No	Yes	Major cause of infertility and abortion in sheep in Scotland

Annex 2: Disease-specific information

For each of the diseases listed in Annex 1, disease experts were asked to compile an overview of disease prevalence, impact and control options. This allowed for qualitative assessment of abatement potential, based on both impact and feasibility of control or eradication. For example, Johne's disease has a major impact on EI, but the abatement potential is limited. Conversely, IBR has moderate impact on EI but tools for its eradication exist and have been implemented successfully in numerous countries.

For each disease, information was compiled on:

- Disease cause and manifestation: Brief description of disease
- Hosts affected: indication of eradication potential, e.g. harder for diseases with wildlife hosts
- Health & welfare implications: impact on individual animal level
- Prevalence in Scotland & UK: impact at national level
- Economic impact – indicative a how much disease costs and of how much control could cost
- Climate change impact/implications: Impact of climate on disease occurrence.
- Climate change mitigation strategies: Impact of disease occurrence on climate
- Climate change adaptation strategies: Response to climate impacts
- Eradication: Feasibility of eradication based on available control options and host range

For production impacts and control options, results are presented in tabular format so as to create an inventory of inputs for modelling of EI and abatement potential. As this was a rapid evidence assessment, information was based on review of existing evidence. A list of references used as evidence is provided for each disease.

2.1 Neosporosis

Disease: Neosporosis, caused by the protozoan parasite *Neospora caninum*, is the primary cause of abortion in beef and dairy herds in the UK. This has high economic impact with loss of the calf, less milk produced and higher reproductive costs.

Host(s) affected: Cattle are the intermediate host of the parasite, *Neospora caninum*. The definite host is canine, hence the name "*caninum*". In canids, sexual reproduction of the parasite takes place, resulting in release of eggs in faeces. Contamination of feed or water with eggs from dogs' faeces results in infection of cattle. In cattle, transmission is vertical (dam-to-calf) but not horizontal (cow-to-cow). In the UK, dogs are the canid host. Foxes do not play a role in transmission.[1-3] Rodents may also act as intermediate hosts, resulting in infection of dogs with the risk of subsequent infection of cattle via eggs from dogs' faeces.[2-4]

Health & welfare implications: Infection with *N. caninum* does not cause clinical disease in adult cattle but the unborn calf may be affected. When this happens early in gestation, this can result in poor fertility (late return to oestrus) or, at 3-4 months of gestation, in mummification of the foetus, which may go unnoticed for months until the animal does not give birth at the expected due date. When infection of the foetus occurs after 4 months in gestation, the calf is aborted within 48 hrs. Occasionally, calves are born alive but brain damaged. Most calves are born as healthy carriers, resulting in maintenance of the parasite in the herd.[1]

Prevalence in Scotland/UK: In south-west England, herd and animal level seroprevalence of Neosporosis were estimated at 94% and 12.9% respectively, with 90% of herds consistently seropositive over 4 years. Within-herd seroprevalence ranged from 0.4% to 58%, with a median of 10%[4]. In the Netherlands, within-herd seroprevalence <15% is considered acceptable whilst a higher seroprevalence is a trigger for control actions[1.]

In the UK, *Neospora* has been the most frequently detected and attributed cause of infectious bovine abortion for several years. Estimates for Scotland range from 18.8 % in 2007-2010 to 25.6% of diagnosed abortion submissions in 2014[2.]

Table1. Production effect(s):

Effects	Impact	Estimate of losses
Growth	Reduced weight gain	Significant reductions in birth weight (4.2 kg lighter), weight gain (7.5kg slaughter weight) and feed efficiency (2.2 kg extra feed for 1 kg weight gain) were associated with the presence of antibodies against *N. caninum* in post-weaning beef steers[5,12]
Production	Reduced milk production	In herds with abortion problems, seropositive cattle produced less milk, whereas in herds without abortion problems, *N. caninum*-seropositive cattle produced the same amount of milk as seronegative cattle[6]; Dairy Herd Improvement Association data showed that milk production of seropositive cows was less for milk (1.4 kg/cow/day), fat-corrected milk (1.6 kg/cow/day), and fat (0.06 kg/cow/day) than production of seronegative cows[7].
Waste	Abortion	Cattle with *Neospora* antibodies are 5 to 7 times as likely to abort as cattle that are seronegative[2].
	Culling	Risk of a seropositive cow dying was not different from that of a seronegative cow. Seropositive cows were culled 6.3 months earlier than seronegative cows, and had a 1.6 times greater risk of being culled, compared with seronegative cows, after adjusting for culling risk associated with abortion. For cows culled for low milk production, culling risk for a seropositive cow was twice that for a seronegative cow.[8]

Economic impact: The economic impact of *N. caninum* infection in dairy cows can include reduced revenues from decreased milk production, which may warrant culling of young, seropositive replacement stock[7]. Economically, "do nothing" may be better than test-and-cull strategies in herds with <20% seroprevalence[9]. Other analyses suggested that whole herd testing and excluding daughters from seropositive dams as potential replacements provided the best economic return compared to e.g. culling of animals that fail to give birth, replacement of seropositive animals by seronegative replacement cattle or mass treatment.[10,11]

Climate change impact/implications: Reduced productivity contributes to increased carbon footprint of dairy and beef production. Prolonged grazing season may increase risk of exposure to dog faeces.

Climate change mitigation strategies: Control of neosporosis at herd level to reduce risk of abortion.

Climate change adaptation strategies: Not applicable.

Disease control options and costs: Infection occurs via two routes: (a) from cow to calf, and (b) from dog to calf/cow. Cow-to-calf transmission is the main source of infection, and results in maintenance of the parasite in the herd over many cattle generations. Dogs become infected by eating aborted materials, birth fluids or placentas. Infection in the dog results in excretion of eggs with their faeces, leading to contamination of cattle feed and drinking water. Control is aimed at preventing cow-to-calf transmission by excluding infected animals from breeding, and at preventing dog-to-cattle transmission by keeping dogs away from cattle. This includes public awareness campaigns to alert dog walkers to the risk their dog poses to grazing cattle and to ask them to pick up after their dogs in the countryside.[1] Introduction into a herd may also occur through purchase of cattle.[4]

Table 2. Disease control options and costs: (a) applicability (i.e. what % of the affected animals could the option be applied to (b) effect of treatment.

	Control option	Applicability	Effect	Cost
Diagnostics	Antibody ELISA	Serum, milk (individual, bulk tank), foetal fluids	Diagnosis of infection	£5.50 (serum)
	PCR	Aborted foetus	Diagnosis of infection	
	Immunohistochemistry	Aborted foetus	Diagnosis of infection	
Treatment	None[2]	n/a	n/a	n/a
Vaccine	None[2]	n/a	n/a	n/a
Grazing/pasture management	Prevent dogs from having access to cattle feed, pastures, fields for the production of cattle forage and water sources[1,2]			
	Inform dog walkers of the potential risk dog fouling in rural areas may represent to cattle[1,2]			
Alternatives	Dispose of afterbirths (even those from normal calvings) aborted foetuses and other animal tissue leftovers promptly and in a safe manner[2]			
	Control rodents on farm			

	Selective breeding: Breeding heifers born from seronegative dams only[2]		Decrease the within-herd prevalence over time and reduces the risk of abortion	unknown
	"Breed to beef" in dairy herds: Inseminating seropositive cows using beef bull semen[1,2]	Dairy herds	Reduced risk of abortion; Decrease the within-herd prevalence over time	unknown
	Test and cull: The removal of *Neospor*a seropositive animals and their offspring[1,2]	Economically sustainable only in herds with low seroprevalence where only a small proportion of animals would need to be removed	Reduce the within-herd prevalence over time	
	Embryo transfer: Implanting embryos from a seropositive dam to a seronegative recipient[1,2]	Cattle of high genetic merit only due to high cost of ET	Prevents vertical transmission to calf	
	Control concomitant infections and risk (BVDV, IBR, mycotoxins in feed)[2]		Reduced risk of abortion if infected	unknown
	Biosecurity: only purchase seronegative animals[2,4]			

Eradication: There are no reports of eradication of Neosporosis from countries or regions, but control programs at herd level do exist[1.] In Scotland, accreditation of *Neospora*-free status is possible via CHeCS herd health programmes.

References:

1. www.gddiergezondheid.nl (Dutch Animal Health Service).
2. Guido, S. et al. , Katzer F, Innes E. The Moredun Foundation News Sheet 2016 6(9).
3. Dubey, J. P and JP, Schares, G. Neosporosis in animals--the last five years. *Vet Parasitol*. 2011 Aug 4;180, (1-2):90-108. 4. doi: 10.1016/j.vetpar.2011.05.031.
4. Woodbine, K. KA, Medley GF, Moore SJ, Ramirez-Villaescusa A. et al., Mason S, Green LE. A four year longitudinal sero-epidemiology study of *Neospora caninum* in adult cattle from 114 cattle herds in south west England: associations with age, herd and dam-offspring pairs. *BMC Vet Res*. 2008 Sep 15;4, :35. 5. doi:10.1186/1746-6148-4-35.
5. Barling, K. S. et al. KS, Lunt DK, Snowden KF, Thompson JA. Association of serologic status for *Neospora caninum* and postweaning feed efficiency in beef steers. *J Am Vet Med Assoc*. 2001 Nov 1;219, (9):1259-62.
6. Hobson, J. C. et al. JC, Duffield TF, Kelton D, Lissemore K, Hietala SK, Leslie KE, McEwen B, Cramer G, Peregrine AS. *Neospora caninum* serostatus and milk production of Holstein cattle. *J Am Vet Med Assoc*. 2002 Oct 15;221, (8):1160-4.
7. Thurmond, M. C. and Hietala, S. K. MC, Hietala SK. Effect of Neospora caninum infection on milk production in first-lactation dairy cows. *J Am Vet Med Assoc*. 1997 Mar 1;210, 72(5):672-4.
8. Thurmond, M. C. and Hietala, S. K. MC, Hietala SK. Culling associated with *Neospora caninum* infection in dairy cows. *Am J Vet Res*. 1996 Nov;57, (11):1559-62.
9. Reichel, M. P. and MP, Ellis, J. T. JT. If control of *Neospora caninum* infection is technically feasible does it make economic sense? *Vet Parasitol.* 2006 Nov 30;142, (1-2):23-24.
10. Larson, R. L. et al. *J Am Vet med Assoc*. RL, Hardin DK, Pierce VL. Economic considerations for diagnostic and control options for *Neospora caninu*m-induced abortions in endemically infected herds of beef cattle. J Am Vet Med Assoc. 2004 May 15;224, (10):1597-604.
11. Häsler B, Regula G, Stärk KD, Sager H, Gottstein B, Reist M. Financial analysis of various strategies for the control of *Neospora caninum* in dairy cattle in Switzerland. *Prev Vet Med*. 2006 Dec 18;77, (3-4):230-53.

12. Haddad, J. P. A., I. R. Dohoo, and J. A. VanLeewen. A review of *Neospora caninum* in dairy and beef cattle-a Canadian perspective. *Can. Vet. J*. 2005 46:230-243.

2.2 Infectious Bovine Rhinotracheitis (IBR)

Disease: Infectious Bovine Rhinotracheitis (IBR) is a disease of the upper respiratory tract and the lungs that is caused by bovine herpes virus 1 (BoHV-1). The virus may also cause poor fertility and a severe drop in milk yield. Once infected, animals are carriers for life, with reactivation of virus shedding and disease at times of stress. IBR has been eradicated from parts of Europe and forms a barrier to export[1-4].

Host(s) affected: IBR primarily affects cattle. BoHV-1 may also infect sheep, goats and deer. Interspecies transmission is of limited epidemiological relevance[2,3]. IBR is an important cause of financial loss on dairy and beef farms[1,4]. The virus affects young stock and adult animals and may cause disease of the genital tract in male and female cattle. The virus can be spread via bulls, semen used for artificial insemination and embryo transfer.

Health & welfare implications: BoHV-1 may cause a range of clinical conditions in cattle, including respiratory disease and severe or fatal pneumoniae; genital tract infections, infertility and abortion; conjunctivitis, encephalitis and neurological disease; enteritis and dermatitis, all of which affect animal health and welfare.

Prevalence in Scotland/UK: Based on bulk milk surveys in 1998 and 2008-2010, the herd level prevalence of BoHV-1 in the UK is estimated at ca. 70%[5], with similar estimates for dairy and beef herds in Ireland[1].

Production effects: There is conflicting evidence on the production effects of IBR, in part because of differences between strains of BoHV-1. The major production effects include failure to conceive, abortion, and milk production losses as well as morbidity, mortality and growth retardation due to respiratory disease[1].

Table1. Production effect(s):

Effects	Impact	Estimate of losses
Growth	Respiratory disease in calves	
Production	Milk yield	BoHV-1 seropositive cows produce 2.6kg/d less than seronegative cows[6]; Acute disease: severe drop in milk yield or complete cessation with recovery in 5-7 days, with up to 75% of animals/herd affected[1]; Subclinical outbreak: 0.92 kg of milk per cow per day during a period of 9 wk[7].
	Fertility	Conflicting evidence with regards to association between BoHV-1 serostatus and fertility (conception rate), with negative impact reported in some studies and no impact in others[1]
	Abortion	Within-herd abortion rate from 5 to 75% in outbreaks[1,8]; Ca. 3% of abortions in cattle in the British isles attributed to IBR[1]
Waste	Mortality	Outbreaks in BoHV-1 free suckler herds may cause 5% mortality of cows[8]; Outbreaks in BoHV-1 free dairy herds occurred in 2% of herd-years at risk[9].
Other	Ban on export of live cattle to IBR free countries/regions	Trade losses
	Embryo transfer and artificial insemination stations must be free from BoHV-1[3]	Trade losses

Economic impact: IBR is associated with significant losses due to disease and restrictions on trade, although detailed estimates of production impacts are difficult to obtain or highly variable[1,2,4].

Climate change impact/implications: BoHV-1 causes latent infections that can be re-activated by stress, e.g. transportation, movement and mingling. It has been suggested that extreme weather events, e.g. heat waves, may also act as stressors and result in virus reactivation[3,10].

Climate change mitigation strategies: BoHV-1 control reduces the biological efficiency of production and reproduction.

Climate change adaptation strategies: Virus transmission is primarily directly from animal to animal or via semen. There is no clear evidence for an impact of climate change on IBR prevalence or incidence.

Disease control options and costs: Biosecurity measures and vaccination can be used to control IBR. Marker vaccines exist, allowing for differentiation of infected and vaccinated animals (DIVA). There is no treatment for IBR and animals remain infected for life. Screening and eradication programmes are offered at herd level. CHeCHS licences the IBR Accreditation program, which can certify herds as IBR free using UKAS ISO17025 accredited tests[4].

Table 2. Disease control options and costs: (a) applicability (i.e. what % of the affected animals could the option be applied to (b) effect of treatment.

	Control option	Applicability	Effect	Cost
Diagnostics	Virus isolation	Tissue, nasal swab		£50 (AFBI)
	PCR	Tissue, nasal swab		£20 (AFBI)
	Antibody detection in blood	Beef cattle, non-lactating cattle	Individual status, herd level surveillance	£3.67 (non-marker); £7.50 (marker; SRUC)
	Antibody detection in milk	Dairy cattle	Individual status, herd level surveillance	£3.60 to £6.00 (BioBest, SRUC)
Treatment	Intranasal vaccination	Start of outbreak only	Induce interferon production, limit damage caused by outbreak[1]	Ca. 2.50
Vaccine	Inactivated		Prevention of clinical signs, reduced risk of infection and transmission[11]	Ca. £2.50
	Live vaccine	Rapid protection during outbreaks	Prevention of clinical signs, reduced risk of infection and transmission[11]	Ca. £2.50
	Marker vaccine (live or inactivated)	Control and eradication programmes; IgE-specific ELISA has low sensitivity at individual animal level (70%) but is adequate at herd level.	Differentiation of vaccinated from infected animals (DIVA) based on serological detection of IgE-specific antibodies, critical for trade restrictions[2]	Ca. £2.50
Grazing/pasture management	Double fencing		Prevent transmission via boundary fence[4]	
	Avoid co-grazing		Avoid transmission via direct contact or aerosol[3,4]	
Alternatives	Biosecurity: avoid introduction of cattle, incl. bulls		Prevent introduction via infected animals (nasal discharge, coughing, semen)[5,9]	
	Avoid cattle markets, shows		Prevent introduction[3,9]	
	Protective clothing for professional visitors	Traders, veterinarians, AI staff, feed consultants, etc.	Prevent introduction[3,9]	

Eradication: IBR has been eradicated from several European countries (Norway, Sweden, Finland, Denmark, Austria and Switzerland) and from some regions in other countries[3]. Measures to achieve eradication have included test-and-cull programmes, movement restrictions, a ban on use of semen from BoHV-1 positive bulls, vaccination, and monitoring programmes based on bulk milk or blood testing[11]. Voluntary vaccination schemes are unlikely to achieve eradication[12] and some experts have argued that economic benefits may not always outweigh the risk[11]. Addressing infection with bovine herpes virus-1 (BoHV-1) in the Irish cattle population has been identified as a priority for Animal Health Ireland and Animal Health Northern Ireland[1].

References

1. Graham DA. Bovine herpes virus-1 (BoHV-1) in cattle-a review with emphasis on reproductive impacts and the emergence of infection in Ireland and the United Kingdom. Ir Vet J. 2013 Aug 1;66(1):15. doi: 10.1186/2046-0481-66-15.
2. Muylkens B, Thiry J, Kirten P, Schynts F, Thiry E. Bovine herpesvirus 1 infection and infectious bovine rhinotracheitis. Vet Res. 2007 Mar-Apr;38(2):181-209.
3. Raaperi K, Orro T, Viltrop A. Epidemiology and control of bovine herpesvirus 1 infection in Europe. Vet J. 2014 Sep;201(3):249-56. doi: 10.1016/j.tvjl.2014.05.040.
4. http://www.sruc.ac.uk/info/120112/premium_cattle_health_scheme/658/.
5. Williams D, Winden SV. Risk factors associated with high bulk milk antibody levels to common pathogens in UK dairies. Vet Rec. 2014 Jun 7;174(23):580. doi: 10.1136/vr.102049
6. Statham JM, Randall LV, Archer SC. Reduction in daily milk yield associated with subclinical bovine herpesvirus 1 infection. Vet Rec. 2015 Oct 3;177(13):339. doi: 10.1136/vr.103105.
7. van Schaik G, Shoukri M, Martin SW, Schukken YH, Nielen M, Hage JJ, Dijkhuizen AA. Modeling the effect of an outbreak of bovine herpesvirus type 1 on herd-level milk production of Dutch dairy farms. J Dairy Sci. 1999 May;82(5):944-52.
8. Holzhauer M, Dijk R, Mars J. [Infectious bovine rhinotracheitis outbreak on a mostly BHV-1 free farm can result in great damage]. Tijdschr Diergeneeskd. 2003 Oct 1;128(19):593-5.
9. van Schaik G, Schukken YH, Nielen M, Dijkhuizen AA, Barkema HW, Benedictus G. Probability of and risk factors for introduction of infectious diseases into Dutch SPF dairy farms: a cohort study. Prev Vet Med. 2002 Jul 25;54(3):279-89.
10. Nardelli S, Farina G, Lucchini R, Valorz C, Moresco A, Dal Zotto R, Costanzi C. Dynamics of infection and immunity in a dairy cattle population undergoing an eradication programme for Infectious Bovine Rhinotracheitis (IBR). Prev Vet Med. 2008 Jun 15;85(1-2):68-80. doi: 10.1016/j.prevetmed.2008.01.001.
11. Ackermann M, Engels M. Pro and contra IBR-eradication. Vet Microbiol. 2006 Mar 31;113(3-4):293-302.
12. Vonk Noordegraaf A, Buijtels JA, Dijkhuizen AA, Franken P, Stegeman JA, Verhoeff J. An epidemiological and economic simulation model to evaluate the spread and control of infectious bovine rhinotracheitis in The Netherlands. Prev Vet Med. 1998 Sep 1;36(3):219-38.

2.3 Parasitic gastroenteritis (PGE)

Disease: Parasitic gastroenteritis (PGE) is a dose-dependent condition (i.e. influenced by infection level) caused by a range of parasitic roundworms that impact on the performance and productivity of livestock. PGE is a disease complex characterised by diarrhoea, dehydration, ill-thrift, inappetance, weight loss and/or anaemia and, in very acute cases, death.

Host(s) affected: Endemic within livestock viz. sheep, cattle, goats but some species also found in deer, horses and wildlife e.g. rabbits, hares. The predominant sheep roundworms in the UK include *Haemonchus contortus* (Barber's Pole worm), *Nematodirus battus, Teladorsagia circumcincta* (brown stomach worm) and *Trichostrongylus* species (black scour worm). In cattle, the predominant species are *Cooperia oncophora, Ostertagia ostertagi*, *Nematodirus* and *Trichostrongylus* species. Rarely found in humans (reported cases in immuno-compromised individuals).

Health & welfare implications: Infections range from acute, where mortalities may occur, chronic, where morbidity and premature culling may occur, to sub-clinical, where the impact on productivity is insidious and difficult to diagnose. The prevalence of anthelmintic resistant nematodes is increasing in livestock across the UK, this has an overall impact on the ability of producers to effectively control many roundworm populations.

Prevalence in Scotland/UK: No active surveillance in the UK, scientific studies in sheep suggest that roundworm species like *Teladorsagia* and *Trichostrongylus* are ubiquitous, with high prevalence of species like *Haemonchus, Nematodirus* and *Cooperia* [1]. The situation in cattle is less well documented but small scale studies suggest that *Ostertagi* and *Cooperia* species are ubiquitous, with *Nematodirus*, *Trichostrongylus* and *Haemonchus* species being important locally [2].

Production effect(s): The magnitude and scale of losses can differ considerably and can be influenced by a range of factors e.g. breed, sex, nutritional status, previous exposure etc. Losses attributed to growth, production and wastage can be observed.

Table1. Production effect(s):

Effects	Impact	Estimate of losses
Growth	Liveweight gain	28-62% difference in liveweight gain in first season grazing calves (sub-clinical-acute infections versus treated animals)[3]. 24-45% difference in liveweight gain in uninfected lambs compared to infected lambs [4-7] or 9kg over grazing season [7]
Production	Carcass quality	34-52% reduction in protein deposition in moderate *Teladorsagia* infections in lambs [6]
	Meat quantity	2.8-4.7 kg decrease in carcass weight with a 10-14% decrease in carcass value in sheep and cattle [7, 8]
	Milk quantity	~1kg per day greater milk production in "optimally" managed cows compare to less well managed herds [9] Economic impact and impact of lamb/calf development
	Lower wool production	~18-45% lower wool growth in *Trichostrongylus* infected lambs compared to uninfected lambs [7, 10, 11],
	Reduced feed conversation rate (FCR)	~20% in *T. circumcincta*-infected sheep. May require supplementary feeding
Waste	Culling unproductive stock	Current figures not available
	Purchase of replacement stock	Current price ~£1-2 per Kg live weight for commercial lamb and cattle stock
Other	Reduced body condition scores	Captured by reduced growth and carcass quality
	faecal breech soiling	Lambs more susceptible to fly strike –with losses associated with disease and subsequent treatment

Economic impact: A conservative estimate of the cost of PGE in lambs to the British sheep industry in 2005 was £84 million [12]. Reduced growth rate and lost performance in lambs is estimated to cost £63.7 million and treatment and control is estimated to cost £20.3 million (£11.7 million in labour costs and £8.6 million on medicines)[12] .

Climate change impact/implications: Seasonality, prevalence, geographic spread, disease outbreaks driven largely by prevailing climatic conditions, especially temperature and rainfall. Roundworm risk forecast to increase over coming decades, based on UK climate projections. Roundworm infection contributes to carbon footprint of livestock production through reduced biological efficiency and increased waste.

Climate change mitigation strategies: i) Appropriate choice/timing of treatments, control through better use of diagnostic or electronic weighcrates, to ensure accurate dosing, and drafting systems leading to increased biological efficiency & reduced waste, efficient treatment of livestock leads to animals reaching target market weight earlier thus reducing GHG emissions intensity per unit livestock product[13], ii) altering timing of turn-out and/or housing shown to be beneficial in First Season Grazing (FSG) cattle and lambs by reducing exposure of naive animal to high infection rates, iii) Effective quarantine treatment reduces the risk of importing anthelmintic resistant parasites onto farms. Resistant parasites can lead to sub-optimal growth and productivity and waste time and resources in control, iv) reducing stocking densities leads to lower contamination rates on pasture and subsequently lower infection rates in susceptible stock v) nutrition - concentrates and or bioactive forages; improved nutritional status of animals facilitates better development of immunity.

Climate change adaptation strategies: i) Changing breed and/or selective breeding for resistance/resilience to infection has been undertaken, but results are ambiguous, difficult to effect in the field, timescale is long, often comes at the expense of more desirable traits ii) changing pasture management and/or grazing strategies to minimise the exposure of susceptible lambs to high infection levels, iii) changing timing/delaying of mating/artificial insemination, delaying turnout has been shown to be beneficial in FSG cattle and lambs by reducing exposure of naive animal to high infection rates iv) intensive creep feeding of young stock at grass to improve nutritional status of animals to facilitate better development of immunity.

Disease control options and costs: Some of the impact of control strategies will depend on the factors such as stocking densities, topography of the enterprise etc.

Table 2. Disease control options and costs: (a) applicability (i.e. what % of the affected animals could the option be applied to (b) effect of treatment.

	Control option	Applicability	Effect	Cost
Diagnostics	Faecal egg counts	All stock can be examined	More efficient /sustainable use of treatments (up to 50% ↓ usage in anthelmintics)	£10-20 per count, various suppliers
	Test for examining anthelmintic sensitivity	All drug classes and stock can be examined at treatment	Efficient drug treatment can improve quantity and quality of livestock products (see table 1, above)	£30 per treatment; SACVIS
Treatment	Therapeutic (curative) or prophylactic (preventative) administrations	All stock can be administered anthelmintic treatment (> 6 week old)	Efficacy make be compromised by anthelmintic resistance	£0.1-1 per animal

	Quarantine treatment of new/retuning livestock to avoid dissemination of resistant roundworms.	All stock can be administered anthelmintic treatment (> 6 week old)		£0.1-1 per animal
Vaccine	None currently commercially available in the UK.	N/A	N/A	N/A
Grazing/pasture management	Rotational and co grazing with other stock	Requires access to livestock and sufficient pasture	Reduce pasture contamination and thereby reduce exposure of naive animal to high infection rates	Unknown/variable
	Rotational grazing through various paddocks	Requires access to sufficient pasture		Unknown/variable
	Clean grazing	Requires access to sufficient pasture		Unknown/variable
	Bioactive forages e.g. chicory and sainfoin	Influenced by topography, soil biology	Bioactive forages improve nutritional status of animals and act as natural anthelmintics	Reseed cost (~ £140 for 30Kg grass mix)
Alternatives	Selective breeding for resistance or resilience to worm infection	Applicable to all age classes of livestock	Less pasture contamination/less anthelmintic usage	EBV estimation required
	Improved nutrition	Ewes, cows	Better body condition score, more immune to parasite infection	£220/t for general mix sheep/cattle feed.
	Improved nutrition	Lambs, calves	Reach marketable weight sooner, less pasture contamination	£200/t for protein sheep nuts, £435 for lamb creep feed.

Eradication: Not perceived as possible, due to wildlife reservoirs and set stocking rates

References

1. Burgess C. et al.,2012, A survey of the trichostrongylid nematode species present on UK sheep farms and associated anthelmintic control practices. Vet Parasitol 189, 299-307
2. McArthur C. et al., 2011, Assessment of ivermectin efficacy against gastrointestinal nematodes in cattle on four Scottish farms. Vet Rec 169
3. Shaw D. et al., 1998, Gastrointestinal nematode infections of first-grazing season calves in Western Europe: general patterns and the effect of chemoprophylaxis. Vet Parasitol 75, 115-131
4. Coop R.L. et al., 1982, The effect of three levels of intake of *Ostertagia circumcincta* larvae on growth rate, food intake and body composition of growing lambs. J Agric Sci 98, 247-255
5. Coop R.L et al., 1983, Parasitol 87, R5; 6. Coop et al. 1985, Effect of experimental *Ostertagia circumcincta* infection on the performance of grazing lambs. Res. Vet. Sci. 38, 282-287
6. Miller H. et al., 2012, The production cost of anthelmintic resistance in lambs. Vet Parasitol 186, 376-381
7. Sutherland I. et al. , 2010, The production costs of anthelmintic resistance in sheep managed within a monthly preventive drench program. Vet Parasitol 171, 300-304
8. Charlier J. et al. 2005, A survey to determine relationships between bulk tank milk antibodies against *Ostertagia ostertagi* and milk production parameters. Veterinary Parasitology 129, 67-75
9. Kimambo A.E. et al. 1985, The effect of prolonged subclinical infestation with *Trichostrongylus colubriformis* on the performance of growing lambs with emphasis on the fate of nitrogen. Anim Prod 40, 534

10. Coop R.L. et al. 1984, Effect of anthelmintic treatment on the productivity of lambs infected with the intestinal nematode, *Trichostrongylus colubriformis*. Res Vet Sci 36, 71-75
11. Nieuwhof G. & Bishop, 2005, Costs of the major endemic diseases of sheep in Great Britain and the potential benefits of reduction in disease impact. Animal Sci 81, 23-29
12. Kenyon, F. et al., 2013 Reduction in greenhouse gas emissions associated with worm control in lambs. Agriculture, 3(2), 271-284.

2.4 Sheep scab

Disease: Sheep scab is a highly contagious disease of sheep skin, caused by infestation with the sheep scab mite, *Psoroptes ovis*. The feeding activities of the mite, and the deposition of mite faeces containing allergens, cause a rapid inflammation of the skin following infestation and severe pruritis, leading to exudation of serum onto the skin and scab formation at the skin surface.

Host(s) affected: Endemic in sheep in the UK; also infests goats, llamas and alpaca. *Psoroptes ovis* has been successfully experimentally transferred from sheep to goats, rabbits and calves, but not in the opposite direction[1]. *P. ovis* also infests cattle but is infrequent in the UK, usually seen on imported beef cattle; recent incursions in Scotland[2] were controlled. Some concern persists about whether the recent cases in England and Wales[3] have led to the establishment of the parasite in cattle in these areas.

Health & welfare implications: Loss of serum by exudation, coupled with the potential for secondary bacterial infection of wounds and stereotypical behaviours related to the irritation associated with the disease (nibbling, lip smacking, tongue protrusion, convulsive movements, scratching on fence posts and with hind hooves, neck craning, epileptiform fits in some cases) make this a serious production- and welfare-limiting disease. Mortalities may occur but usually only in infested young lambs or in lambs feeding from infested ewes in poor condition as result of the disease[4].

Prevalence in Scotland/UK: Scottish Government survey in 2006 indicated 14.7% of respondents had experienced sheep scab in their flocks the previous 5 years[5]. Within herds, up to 90% of the herd can be infested at the point of veterinary intervention[4].

Production effect(s): Ewe and lamb mortality, loss in body condition, secondary infections, hypothermia, low birth weights, reduced milk yield and lamb growth rates, reduced wool, pelt and leather values

Table1. Production effect(s):

Effects	Impact	Estimate of losses (based on a lowland flock)
Growth	Lamb finishing time	Increased by 2 weeks due to poorer milk yield and growth rates, reducing price obtained by 10p per kg carcase weight per week (based on 2007prices). Additional creep feed intake of 1kg per day for these additional 2 weeks[6]
Production	Lamb losses	Increase lamb losses by 25%, reducing output to 1.575 lambs sold per ewe[6]
	Ewe condition	Additional 10kg of concentrate per ewe as a result of lower body condition at time of lambing[6]
	Fleece	Reduction in fleece value by 50%[6] Economic impact and impact of lamb/calf development
Waste	Purchase of replacement stock	Current price ~£1 – 2 per kg live weight for commercial stock
Other	Notifiable disease in Scotland, leaving producer with decision to send diagnosed lambs for slaughter (maybe earlier than planned) or compulsory treat (with implications of drug withdrawal periods before lambs can be sent for slaughter to enter food chain)	Dependent on timing of diagnosis/treatment of lambs

Economic impact: A 2005 estimate of the cost of sheep scab to the British sheep industry was £8.3 million[7]. Using an alternative model, Stubbings (2007)[6] calculated a reduction in profit of £18.84/ewe in a lowland farm after a

winter outbreak of scab while a recent ADAS report[8], commissioned by EBLEX, estimated £12.30 to be the cost per ewe of a sheep scab outbreak.

Climate change impact/implications: Sheep scab has traditionally been seen as a winter disease with most outbreaks occurring between September and March, although it can occur year-round[9]. Research from the University of Bristol identified elevation, temperature and rainfall all as accurate predictors of risk. [10]

Climate change mitigation strategies: i) Appropriate choice/timing of treatments, control through better use of diagnostics to reduce waste, efficient treatment of livestock leads to animals reaching target market weight earlier thus reducing GHG emissions intensity per unit livestock product, ii) Effective quarantine treatment reduces the risk of importing sheep scab onto farms. Infestation can lead to sub-optimal growth and productivity and waste time and resources in control, iii) Increased biosecurity (double fencing etc., limiting use of common grazing leads to lower infestation rates

Climate change adaptation strategies: i) Changing breed to animals with finer, lighter wool may make them less susceptible to infestations taking hold[11,12] and/or selective breeding for resistance/resilience to infection may have some effect. ii) changing pasture management and/or grazing strategies to minimise the use of common grazing, which is the highest risk factor associated with sheep scab transmission.[13]

Disease control options and costs: Some of the impact of control strategies will depend on the factors such as stocking densities, topography of the enterprise etc.

Table 2. Disease control options and costs: (a) applicability (i.e. what % of the affected animals could the option be applied to (b) effect of treatment.

	Control option	Applicability	Effect	Cost
Diagnostics	Skin scrape	Only to animal with obvious lesion	Select correct treatment	Currently free to producers through SAC if scab is suspected
	Blood test	Can detect recent/subclinical infestation as well as clinical	Select correct treatment	~£5 per test
Treatment	Therapeutic (curative) or prophylactic (preventative) administrations of OP dip or ML injection	Dependent on animal's age, administration of certain foot-rot vaccines and planned withdrawal period	OP dips and ML injectables still effective, Some reports of emerging resistance to both	£0.1-1 per animal
	Quarantine treatment of new/retuning livestock to avoid introduction of parasites	Dependent on animal's age, administration of certain foot-rot vaccines and planned withdrawal period	OP dips and ML injectables still effective, Some reports of emerging resistance to both	£0.1-1 per animal
Vaccine	None currently commercially available in the UK.	N/A	N/A	N/A
Grazing	Avoiding common grazing	Requires access to sufficient alternative pasture	Prevents exposure to parasite	Not known
Alternatives	Selective breeding for resistance or resilience to infestation	Only anecdotal evidence to support	Less infestation/less acaricide usage	Not known

Eradication: Previously achieved in the UK in 1952 following sustained, enforced, compulsory dipping scheme. Successfully eradicated from Norway, New Zealand and Australia. Current eradication campaign in South Africa. In the UK several local eradication campaigns have had success but need sustained effort.

References:

1. Kirkwood, History, biology and control of sheep scab.*Parasitol. Today* 1986 2, 302-307.
2. Jones et al., Psoroptic mange in a Scottish beef herd. *Vet Rec*. 2014 174, 509-10.
3. Wall, Psoroptic mange in cattle and the Ghost of Christmas Yet to Come. *Vet Rec*. 2012 170, 357-358.
4. Sargison et al., Effect of an outbreak of sheep scab (Psoroptes ovis infestation) during mid-pregnancy on ewe body condition and lamb birthweight. *Vet Rec.* 1995 136, 287-289.
5. Blissitt et al., Ongoing battle against sheep scab: History and Progress 2012. *Vet. Times* 42, 5-7
6. Stubbings, L. The prevalence and cost of sheep scab 2007 *Proc. Sheep Vet. Soc.* 31, 113-115.
7. Nieuwhof & Bishop Costs of the major endemic diseases sheep in Great Britain and the potential benefits of reduction in disease impact *Animal Science* 81, 23-29 (2005).
8. Wright, Economic Impact of Health and Welfare Issues in Beef Cattle and Sheep in England (2013). ADAS report
9. Baird, G. Increase in sheep scab cases decreases treatment options *Vet Times* (2011) 41, 32.
10. Rose et al., Mapping risk foci for endemic sheep scab (2009) *Vet Parasitol* 165, 112-118.
11. Fourie et al., The growth of sheep scab lesions in relation to sheep breed and time of the year 2002. *Exp Appl Acarol* 27, 277-281.
12. Meintjes et al., Host preference for the sheep scab mite, *Psoroptes ovis* 2002. J S Afr Vet Assoc. 73, 135-136.
13. Rose and Wall Endemic sheep scab: risk factors and the behaviour of upland sheep flocks (2012) *Prev Vet Med*. 104:101-6

2.5 Johne's disease (JD)

Disease: *Mycobacterium avium* subspecies *paratuberculosis* (MAP) is the infectious agent that causes Johne's disease (JD); a chronic wasting disease of the intestine that infects ruminants worldwide and is endemic in the UK/Scotland. The majority of individuals will be infected soon after birth. However there can be a long incubation period of many years before symptoms are observed (most commonly 3-5 years, but up to 15 years in some cases). Once clinically identified, the effects of diarrhoea and dehydration result in a chronic wasting disease which leads to the death of the animal. Therefore, this disease has a significant effect on performance and productivity, specifically in reproduction, milk yield and meat production, in all economically relevant ruminants.

Host(s) affected: Endemic within ruminant livestock. The most economically significant host species are cows, sheep, goats, and deer. However, there are also a number of wildlife reservoirs for this disease including squirrels, rabbits and hares. Found in humans and has been associated with Crohn's Disease [1,2]. JD is transmitted by both faeces and milk but also through semen and in-utero

Health & welfare implications: Due to the potentially prolonged incubation time of MAP, when there is an absence of clinical symptoms, many infected animals will be silent carriers of disease, but still have the ability to shed the bacteria via faeces and milk. Early signs of the onset of clinical disease are a reduction in milk yield, poor body condition and longer calving intervals. If the infected animal is not culled, they will become emaciated, due to mal-absorption of nutrients in the gut, with the continual shedding of infected diarrhoea. Clearly this is a welfare issue for these individual animals and they should be culled. Removal of these individuals also prevents further spread of the disease to other animals in the flock or herd as well as between species where co-grazing occurs. There is no economically viable treatment unless animals are of high value [3].

Prevalence in Scotland/UK: In several countries, studies have assessed the prevalence and economic impact of this disease. In the UK, a prevalence study in the cattle dairy industry was conducted in 2009 [4], but no prevalence studies have been done for beef cattle, sheep, deer or goats. This is partially due to difficulties in diagnosis and that it is not a notifiable disease. This has led to a gross underestimation of the prevalence and financial impact of this disease in the UK [5]. The estimated prevalence is dependent on the diagnostic test used, as the specificity and sensitivity of each test is different and is dependent on the type of sample taken [6]. As a result, within herd prevalence using a serum ELISA may vary between 0 and 4.9%, however, when using the liquid faecal culture method prevalence varied between 0-13.6% [7,8]. Therefore comparisons between prevalence studies can be challenging due to differences in diagnostic tests, diagnostic strategies, and sampling design [9,10]. However, some assessments have been made. It has been suggested that for every clinically identified dairy cow, 25 will be subclinical within the herd [8]. George Caldow *et al*., [11] have suggested that up to 20% of beef and dairy herds are infected in the UK, based on related studies in other countries, with those farms of high prevalence also, having higher replacement frequencies.

Results from the UK study of dairy herds published in 2009 showed that from 13,688 cows within 136 dairy herds 65% of herds had at least one animal testing positive based on the ELISA test for JD and 2.5% individual animals tested were positive (4). A survey of cull cows from a single slaughter house in the south west of England [12] identified MAP in 3.5% of cull cows.

Table1. Production effect(s):
The effect of JD on production varies significantly from one herd/flock to another. The explanation for this is multifactorial, but mainly due to the inability to diagnose the disease early enough to cull out those affected before they transmit disease to other animals and, whether offspring of infected animals are used for breeding. Eradication of the disease, once identified on a farm is difficult. However, farmers have managed this disease effectively by using a range of control measures; both to prevent the introduction of the disease onto uninfected farms and also to control the disease once a farm becomes positive for JD.

Effects	Impact	Estimate of losses
Growth	Live weight gain	Two studies carried out in 1999 and 2009 assessing the production effect on beef cattle herds considered that calves from MAP positive cows (either by faecal culture or serum ELISA) had a reduced birth weight of 9.7% to 14.9% respectively, implicating this disease does have a significant production effect 13.
Production	Meat and milk quantity: Reduction in beef and milk yield, in all ruminants infected with MAP.	A Michigan based study reported that a 10% increase in MAP apparent prevalence in a herd, was associated with a decrease in mean weight (culled cows) of 33.4 kg 14. Financial losses due to reduced weight of culled cows were estimated to be $1,150 annually for each 10% increase in herd prevalence of JD. In a separate study, the effects of JD on the slaughter weight and slaughter value of dairy cows and estimated weight losses were up to 31% and slaughter value losses up to 48% compared with MAP-negative cows (with at least 2 ELISA-negative tests) [15]. In the UK, it was estimated that there were 1,000 cases of Johne's disease, in the year 2000, increasing to 2,400 in 2004, affecting between 20 to 50 per cent of UK herds. Estimated culling/mortality rates, in infected herds may be 1 to 5%. However, losses due to subclinical disease (weight loss, reduced milk yield; poor fertility) are predicted to be substantial. The financial losses are estimated to be £2600 in a 100 cow dairy herd with clinical cases but this is likely to be a gross underestimate [5].
Reproduction	Reduction in lambing and calving frequency.	The estimated reproduction cost of JD in a herd of 100 suckler cows where 10% of the cows are JD infected, predicts that 5% less calves would be produced per annum and 5% of the calves produced each year weigh 36 kg less at weaning [16].
Waste	Culling due to reduced production and welfare. Restocking from other herds/flocks. Significant environmental faecal contamination on pasture, in barns and birthing pens. The spread of contaminated slurry on fields.	Replacement frequency increases, which is an economic loss, but also a potential threat to introducing the disease from other herds/flocks of unknown disease status. JD is transmitted predominately in faeces with super-shedders being the most prolific transmitters containing high concentrations of MAP [17].
Other	Susceptibility to other diseases.	Villarino and Jordan [18] estimated that dairy cows testing positive for MAP were more likely to be lame and to develop digestive disease, mastitis and/or respiratory disease than MAP-negative cows. On the other hand, stress, parturition, inadequate nutrition, concurrent infections such as parasitism and immunosuppression associated with some infections (e.g., bovine viral diarrhea virus, BVDV) may influence the onset of clinical JD 19,20.

Economic impact:

The actual cost to the cattle industry in 2009, in Scotland, has been estimated by Scotland's Rural College (SRUC) to be £13 million per year [21].

Annual cost of JD in 100 suckler cow herd would be £4532.00; based on spring calving with an average calf weaning weight of 270 kg, and the following assumptions: that 10% of the cows are infected resulting in 5% less calves being produced per annum; 5% of the calves produced each year weighing 36 kg less at weaning; 2 cows with clinical JD being slaughtered and unable to enter the food chain due to emaciation; and in an additional 3 cows being culled each year [16].

In the UK, the average cost of paratuberculosis, per animal, per year has been calculated as £26.00 for dairy cattle (approximately $47 based on the average exchange rate in 2004) and £17.00 for beef cattle (approximately $31 based on the average exchange rate in 2004) [22].

The economic impact is affected by a number of factors including: milk production, weight loss and beef production, premature culling, increased mortality, and replacement-associated costs, infertility and predisposition to other diseases, diagnostic testing, veterinary costs, animal welfare impact, marketing, and public health-related issues [8].

Climate change impact/implications: Based on UK climate predictions, changes in temperature and rain fall will potentially result in an increase in the sustainability of MAP in contaminated pastures. MAP bacteria can potentially survive in water environments for up to 48 weeks, providing a potentially important reservoir for infections [23,24]. Increased resilience of MAP in the environment would increase infection frequencies and result in a higher carbon foot print by the reduced productivity of animals, and the increase in diarrhoea. The Life Cycle Analysis (LCA) analysis done by Cranfield University, indicates GHG emissions for a functional unit of milk (1,000 litres) of 0.89 t CO_2e (net GHGE abatement) for a 'healthy' animal. The current national herd performance is 6% higher at 0.95 t CO_2e. However, GHG emissions increase per unit of milk up to 25%, for JD [16,25]. The GHG emissions associated with 1,000 kg of beef carcass weight from a healthy herd is estimated to be 17.1 t CO_2e. The current national herd performance is 6.6% higher at 18.2 t CO_2e. However, GHG emissions increase by 40% when JD is detected. For dairy beef JD increases GHG emissions per unit output by 4% [16,25].

Climate change mitigation strategies:

Mitigation strategies could include: adaptation of grazing strategies, in field and paddocks; biosecurity during and after calving or lambing; slurry composting; regular test and cull strategies, maintaining a closed herd/flock policy. Test replacements and obtain them from tested herds/flocks. Avoid breeding from cows/ewes with a positive test result and remove from herd/flock as well as any off spring from these animals. Improve nutritional levels of sheep and reduce parasite load if known to be high. Use vaccination as part of a management control strategy for sheep. Do not used pooled colostrum in herds/flocks with a known history of JD. In cases where cows infected with MAP are calving, place in separate birthing pens away from JD free animals and remove calf as soon after birth as possible and provide pasteurized colostrum or colostrum from a JD free source. Maintain barn areas, specifically birthing areas clear of soiled bedding. Provide good hygiene levels around the teats and udders of cows with calves.

Climate change adaptation strategies: Developing JD control measures will depend on the farm type (intensive or croft) and species farmed. With JD free farms, the management control strategies would focus on maintaining this status. For farms with a history of JD, reducing the number of new infections would be the focus of a management strategy. Keep water troughs regularly cleaned and remove faecal contamination from areas where cows are most frequently gather, such as feeding pens/bins, milking parlours and entrances to barns. Introduce composting into

manure management to kill MAP. Avoid grazing young animals where fresh manure has been spread on fields. Control rabbits if farm is heavily populated, as rabbits are known to carry MAP.

Table 2. Disease control options and costs: (a) applicability (i.e. what % of the affected animals could the option be applied to (b) effect of treatment.

	Control option	Applicability	Effect	Cost
Diagnostics	Serum ELISA Milk ELISA Faecal culture Sheep: MAP culture +PCR (pool of 10 faeces samples) Faecal PCR		Correctly identifying the infection status of cattle depends on the diagnostic tests but also on the stage of the infection process; in general, cattle with clinical infection are more likely to be detected [26]. This fact together with the presence of a high number of animals in subclinical stages may explain in part why the overall sensitivity of diagnostic tests for paratuberculosis is low.	£4.80 per test £5.00 per test £38 per test £72.50 per test £27.50 per test
Treatment	There is no economically viable treatment available.	A course of antimicrobials over an extended time course can be used, but is only feasible for very high value animals.	Not known	Not known
Vaccine	Gudair® Dead attenuated strain of MAP commercially available in the UK – distributed by Virbac	Single vaccination between 1 month and 4 months after birth.	Can cause some tissue damage at sight of inoculation. Does not prevent infection or transmission. However, vaccination does prevent clinical symptoms developing. Vaccinated animals cannot be differentiated from those infected. The vaccine can also cross-react with the bovine TB test.	£2.75 per head
Grazing/pasture management	No co-grazing of animals on farms with known history of JD. Pastures can remain contaminated with MAP for up to 47 months [11].	The separation of grazing areas of cattle and sheep. Ideally, cull JD test positive cattle/sheep as soon as possible. Keep new replacements in quarantine until test result and clinical examination completed.	Effect of control measures may take several years (>3) in combination with other control measures, but reductions in clinical cases are observed.	
Alternatives				

Eradication: Not economically possible due to a number of factors including: poor detection at early stage of infection, environmental contamination, wild life reservoirs and a non-protective vaccine. However, disease management strategies have reduced clinically affected animals on farm.

References:

1. Groenendaal, H., & Zagmutt F.J., 2008. Scenario analysis of changes in consumption of dairy products caused by a hypothetical causal link between *Mycobacterium avium* subspecies *paratuberculosis* and Crohn's disease. *J Dairy Sci* 91(8):3245-58
2. FSAI, 2009. *Mycobacterium avium* subsp. *paratuberculosis* and the possible links to Crohn's disease. Report of the Scientific Committee of the Food Safety Authority of Ireland. https://www.fsai.ie/workarea/downloadasset.aspx?id=8552
3. Manning E.J., & Collins M.T., 2001. *Mycobacterium avium* subsp. *paratuberculosis*: pathogen, pathogenesis and diagnosis. *Rev Sci Tech* 20(1):133-50
4. Anon, 2009. DEFRA SB4022: An Integrated Strategy to Determine the Herd Level Prevalence of Johne's Disease in the UK Dairy Herd. http://archive.defra.gov.uk/foodfarm/farmanimal/diseases/atoz/documents/johnes-report0911.pdf
5. NADIS (National Animal Disease Information Service). 2014. Johne's disease (Paratuberculosis). http://www.nadis.org.uk/bulletins/johne%E2%80%99s-disease-(paratuberculosis).aspx
6. Nielsen, S.S., & Toft, N., 2008. Ante mortem diagnosis of paratuberculosis: a review of accuracies of ELISA, interferon-γ assay and faecal culture techniques. *Vet Microbiol* 129:217–235
7. Smith, R.L. *et al*., 2009. A longitudinal study on the impact of Johne's disease status on milk production in individual cows. *J Dairy Sci* 92:2653–2661
8. Garcia, A.B. & Shalloo, L., 2015. Invited review: The economic impact and control of paratuberculosis in cattle. *J Dairy Sci* 98:5019–5039
9. Muskens, J., *et al*., 2000. Prevalence and regional distribution of paratuberculosis in dairy herds in the Netherlands. *Vet Microbiol* 77:253–261
10. National Research Council. 2003. Economic implications of Johne's disease. In Diagnosis and Control of Johne's Disease, National Academy Press, Washington, DC., p9–103
11. Caldow, G., *et al*., 2001. Assessment of surveillance and control of Johne's disease in farm animals in GB. Veterinary Science Division, Scottish Agricultural College. www.johnes.org/handouts/files/Scottish_Report_JD.pdf
12. Cetinkaya, B., *et al*., 1996. An abbatoir-based study of the prevalence of subclinical Johne's disease in adult cattle in South West England. *Epidemiol & Infect* 16:373-379
13. Bhattarai, B.G., *et al*., 2012. Comparison of calf weaning weight and associated economic variables between beef cows with and without serum antibodies against or isolation from feces of *Mycobacterium avium* subsp. *paratuberculosis*. *J Am Vet Med Assoc* 243:1609–1615
14. Johnson-Ifearulundu, Y., *et al*., 1999. Herd level economic analysis of the impact of paratuberculosis on dairy herds. *J Am Vet Med Assoc* 214:822–825
15. Kudahl, A.B. & Nielsen, S.S., 2009. Effect of paratuberculosis on slaughter weight and slaughter value of dairy cows. *J Dairy Sci* 92:4340–4346
16. ADAS report, 2015. Study to Model the Impact of Controlling Endemic Cattle Diseases and Conditions on National Cattle Productivity, Agricultural Performance and Greenhouse Gas Emissions. *randd.defra.gov.uk/Document.aspx?Document=13320*_AC0120Finalreport.pdf
17. Whitlock, R.H., *et al*., 2005. Another factor in the control of Johne's disease. Proc. 8th Int. Colloq. Paratuberculosis, Copenhagen, Denmark. International Association for Paratuberculosis, Kennett Square, PA
18. Villarino, M.A., & Jordan E.R., 2005. Production impact of subclinical manifestations of bovine paratuberculosis in dairy cattle. In Proc. 8th Int. Colloq. Paratuberculosis, Copenhagen, Denmark. International Association for Paratuberculosis, Kennett Square, PA
19. Allen, W.M., *et al*., 1986. *Mycobacterium johnei* infection of cattle: The effect of corticotrophin and anabolic steroids. *Vet Rec* 82:562–567

20. Hasonova, L., & Pavlik, I., 2006. Economic impact of paratuberculosis in dairy cattle herds: A review. *Vet Med Czech* 51:193–211
21. Moredun (2010). Moredun - 90 Years of Excellence in Animal Health Research. Moredun magazine. Issue 1, Winter 2010
22. Gunn, G.J., *et al*., 2004. Comparison of the modelled effects and consequential losses due to Johne's disease outbreaks for beef and dairy herds in Great Britain. Cattle Pract 12:1
23. Whittington, R. J., *et al*., 2004. Survival and dormancy of *Mycobacterium avium* ssp. *paratuberculosis* in the environment. *Appl. Environ Microbiol* 70:2989–3004
24. Whittington, R.J., *et al*., 2005. Survival of *Mycobacterium avium* ssp. *paratuberculosis* in dam water and sediment. *Appl Environ Microbiol* 71:5304–530
25. ADAS (2010) Feasibility of Green House Gas (GHG) mitigation methods. Defra project AC0222. http://randd.defra.gov.uk/Default.aspx?Module=More&Location=None&ProjectID=17110
26. Weber, M.F., 2006. Risk management of paratuberculosis in dairy herds. *Ir. Vet. J.* 59:555–561.

2.6 Ovine pulmonary adenocarcinoma – OPA, Jaagsiekte

Disease: **Ovine pulmonary adenocarcinoma** (OPA; also known as sheep pulmonary adenomatosis and Jaagsiekte) is a fatal lung disease of sheep that is caused by a virus named Jaagsiekte sheep retrovirus (JSRV)[1]. JSRV targets the lung in infected sheep and triggers the growth of lung adenocarcinoma. Sheep affected by OPA typically appear thin and have difficulty breathing and may cough and puff, particularly after exercise. In many cases, fluid accumulates in the lungs, and may be discharged from the nose when the animal lowers its head [1, 2]. The disease is characterised by a long subclinical period prior to the onset of clinical signs.

Host(s) affected: OPA occurs in sheep and, rarely, in goats[3]. Other species, including humans, are not affected. OPA is endemic in UK sheep. There are anecdotal reports of increased susceptibility in specific breeds but there have been no systemic experimental studies to address this question.

Health & welfare implications: Infections may be subclinical for several months or perhaps years, with no apparent effect on the overall health of the animal. However, advanced clinical OPA produces significant respiratory difficulty and wasting, ultimately resulting in death of the animal. This may become progressively more severe over several weeks and, therefore, represents a significant welfare problem. Bacterial lung infections are also common in OPA-affected sheep. The subclinical nature of early OPA increases the likelihood that the disease is introduced to unaffected flocks e.g., through the purchase of apparently healthy sheep. Even for flocks with good biocontainment practices, which are 'closed' with respect to ewe replacements, the disease may be brought in with rams[4].

Prevalence in Scotland/UK: (a) within flock: Available figures suggest within flock prevalence of JSRV infection may be as high as 50% in some flocks[5]. Clinical cases of OPA in affected flocks may be as high as 20% of breeding ewes in the first few years following introduction of the disease to a flock. This typically reduces to 1-5% losses per year in flocks were the disease is established[6]. Clinical disease is observed predominantly in adult sheep but may also be seen in lambs 6-12 months old. **(b) between flock prevalence:** Precise figures about the prevalence of OPA are not available due the lack of reliable diagnostic tests and because the disease is widely thought to be under-reported. A Moredun/BioSS/SRUC survey in 2005-8 estimated that 11% of flocks in Scotland carry the disease[7]. A 2013 study of fallen stock in Yorkshire found OPA in 6 of 106 sheep (5.2%) analysed[8]. By comparison, the same study found a similar prevalence for Johne's disease and pneumonic pasteurellosis. A slaughterhouse study earlier this year found the disease in almost 1% of apparently healthy cull adults[9], showing that it is also common in the apparently healthy sheep population.

While it is difficult to estimate the overall impact on UK sheep farming, it is clear that OPA can be severely damaging for individual producers. In some cases, losses due to the disease may be as high as 10% per annum, with costs running into several thousands of pounds per year for some farmers.

Table1. Production effect(s):

Effects	Impact	Estimate of losses
Growth		
Production	Loss of productive ewes and resulting lambs Loss of affected rams	No data
Reproduction	Effect of subclinical OPA on lambing efficiency is unknown	No data
Waste e.g. increased culling, mortality etc.	Culling (or death of) affected stock Purchase of replacement stock. Carcass removal.	No data
Other	Body condition of pre-clinical cases?	

Economic impact: Cost to UK sheep industry unknown. No (reliable?) estimates. Cost to individual producers is high (running into thousands of pounds per year for large flocks or if high value rams are affected).

Climate change impact/implications: No available data, but infectious JSRV may survive in the environment for longer periods during wet and cold conditions. Climate change resulting in more sheep being housed indoors is likely to promote transmission and increase the incidence of the disease.

Climate change mitigation strategies: Reduction of losses to OPA would reduce carbon resource wasted in production and management, thereby reducing impact of the disease.

Climate change adaptation strategies: Uncertain.

Table 2. Disease control options and costs: (a) applicability (i.e. what % of the affected animals could the option be applied to (b) effect of treatment.

	Control option	Applicability	Effect	Cost
Diagnostics	Reliable laboratory tests not currently available Ultrasonography, blood PCR test and novel laboratory tests under study for early detection	Individual animals Subset of flock tested as flock test	Test and cull approach Accreditation	Current PCR test >£40 per animal. Costs could be considerably reduced if part of a multiplex with other tests. Ultrasonography £1-2 per sheep
Treatment	None available	N/A	N/A	N/A
Vaccine	None available. Absence of natural adaptive immunity in infected sheep perceived as barrier to vaccine development.	If available, individual sheep and flocks.	N/A	N/A
Grazing/pasture management	In theory, extensive grazing should reduce transmission. Focal feeding points (licks etc.) may facilitate transmission.	Whole flocks	May reduce but not eliminate transmission. Vet advice should be sought to weigh up reducing transmission vs providing appropriate nutrients.	Unknown
Alternatives	'Snatching' of lambs/motherless rearing. Embryo transfer Culling out and replacing whole flocks	Whole flocks or individual ewes. Whole flocks	How do you make sure to keep the flock OPA free? (Need to buy rams) How do you ensure replacement flock doesn't also have OPA	Expensive. Very labour intensive. Selling flock at cull price and replacing at costs for breeding stock could be as much as £60 per ewe

Eradication: Difficult due to subclinical nature of infection. Stringent culling policy led to eradication from Iceland in 1950s, but also required no new importation of sheep to that country. Robust screening/culling scheme could

reduce impact and this would be possible if a preclinical diagnostic test becomes available. There is a parallel here with Maedi Visna in Sheep and Goat Health Scheme.

References

1. Griffiths, D.J., H.M. Martineau, and C. Cousens, Pathology and pathogenesis of ovine pulmonary adenocarcinoma. J Comp Pathol, 2010. 142(4): p. 260-83.
2. Cousens, C., et al., Jaagsiekte sheep retrovirus is present at high concentration in lung fluid produced by ovine pulmonary adenocarcinoma-affected sheep and can survive for several weeks at ambient temperatures. Res Vet Sci, 2009. 87: p. 154-156.
3. De las Heras, M., L. González, and J.M. Sharp, Pathology of ovine pulmonary adenocarcinoma. Curr Top Microbiol Immunol, 2003. 275: p. 25-54.
4. Scott, P., D. Griffiths, and C. Cousens, Diagnosis and control of ovine pulmonary adenocarcinoma (Jaagsiekte). In Practice, 2013. 35(7).
5. Salvatori, D., Studies of the pathogenesis and epidemiology of ovine pulmonary adenocarcinoma. 2004, University of Edinburgh.
6. Sharp, J.M. and J.C. DeMartini, Natural history of JSRV in sheep. Current Topics in Microbiology and Immunology, 2003. 275: p. 55-79.
7. Lewis, F.I., et al., Diagnostic accuracy of PCR for Jaagsiekte sheep retrovirus using field data from 125 Scottish sheep flocks. Vet J, 2011. 187(1): p. 104-8.
8. Lovatt, F.M. and B.W. Strugnell, An observational study involving ewe postmortem examination at a fallen stock collection centre to inform flock health interventions. Vet Rec, 2013. 172(19): p. 504.
9. Cousens, C., et al., Prevalence of ovine pulmonary adenocarcinoma (Jaagsiekte) in a UK slaughterhouse sheep study. Vet Rec, 2015. 176(16): p. 413.

2.7 Toxoplasmosis

Disease: Toxoplasmosis, this disease is endemic within the UK

Host(s) affected: Sheep, goats and humans (affected by abortions and congenitally infected offspring), cats (the only definitive host that produces parasite oocysts, the environmentally stable and infective stage of the parasite), all warm-blooded animals can act as intermediate host but usually no clinical signs of disease are attributed to *Toxoplasma gondii* infection in these hosts if they are immunocompetent. Once a host is infected, it will stay infected for life. Marine mammals and marsupials are particularly vulnerable to infection and will often develop acute disease. It is thought that this is mainly because these species have evolved away from the cat and, therefore, have not built up resilience against the parasite.

Health & welfare implications: The main health and welfare implications associated with *T. gondii* infection in livestock are abortions and congenitally infected offspring. Abortions and congenitally infected offspring in sheep are only seen following a first infection of the host during pregnancy. Re-infections or re-activation of the parasite in subsequent pregnancies does not result in abortions or congenital transmission of the parasite. Initial infection of sheep and lambs will result in a rise in temperature/fever but generally symptoms are so mild that they go unnoticed. Other food animals e.g. cattle may become infected with *T. gondii* and will rarely show clinical signs or disease, however, these infected food animals are a source of transmission of *T. gondii* to people through the consumption of infected, undercooked meat. Infection in humans may result in serious disease in the developing foetus (deaf, blind, brain damaged) and in immuno-compromised individuals. Ocular toxoplasmosis is also a consequence of human infection with new evidence that particular strains of *T. gondii* are more virulent than others. Measuring disease impact I humans using Disability Adjusted Life Years (DALYs) has shown that *T. gondii* is one of the most significant food-borne pathogens worldwide.

Prevalence in Scotland/UK: (a) within-herd and (b) between-herd prevalence. A seroprevalence study, based on 125 representative sheep flocks within Scotland, has provided information about within- and between- flock prevalence for Scotland (Katzer et al., 2011). This study tested an average of 27 breeding ewes from these flocks for antibodies against *T. gondii* and revealed a seroprevalence (presence of specific antibody) of 56.6% within the 3333 breeding ewes tested. The study has shown that all 125 flocks tested had at least one ewe that was infected and, for 4 flocks, every ewe tested had antibodies against the parasite. The study also revealed an increase in seroprevalence as animals become older, with 73.8% of over 6 year-old ewes testing positive. This increase of seroprevalence with age is a clear indication of the widespread environmental contamination with *T. gondii* oocysts. The median within-flock seroprevalence varied significantly across Scotland, with the lowest seroprevalence of 42.3% in the South and the highest seroprevalence of 69.2% in the far North of Scotland and the Scottish Islands, while the central part of Scotland had a seroprevalence of 57.7%.

Veterinary Investigation Diagnostic Analysis (VIDA) studies into infectious causes of abortions in sheep during 1996-2003 have shown that 28.9% of abortions were due to *T. gondii* infection, which was the second most common cause of infectious abortions following *Chlamydia* infection. This trend continued at least until 2010.

Table1. Production effect(s):

Effects	Impact	Estimate of losses
Growth	No data	No data
Production e.g. reduction in quantity/quality of meat, milk or fibre produced	No data	No data
Reproduction	Foetal reabsorptions Mummified foetuses Abortions Stillbirths	The overall cost of abortion for the UK has been estimated to be £32 million to UK sheep industry. If we assume that 28.9% of abortions were due to *T. gondii* infection, the cost will be £9.25 million. A different calculation could be based on the number of breeding ewes within the UK, which in 2010 was 6.5 million. Annual incidence rates of clinical toxoplasmosis have been predicted to occur in 1% to 2% of breeding ewes[1], which means that an estimated 65,000 to 130,000 pregnancies are lost annually due to *T. gondii* infection. At an average cull ewe cost of £70 this would result in a loss of £4.55 to £9.1 million to the UK industry.
Waste e.g. increased culling, mortality etc.	Neonatal loss due to weak lambs. Culling of barren sheep. Foetal loss due to abortion	No data
Other	*T. gondii* infection is zoonotic and is a significant cause of human disease. It may be transmitted through the consumption of oocysts contaminating the environment ; eating undercooked meat from food animals infected with *T. gondii* cysts or vertically from mother to foetus.	Congenital toxoplasmosis is a serious disease in humans and the incidence varies across the world e.g. in Brazil it has been reported to affect 1: 1000 births[2], whereas in Europe it is 3: 100 000[3] Calculations using Disability Adjusted Life Years (DALYS) has shown *T. gondii* to be one of the most significant food-borne pathogens globally [4]. Recent data from South America has shown that some strain of *T. gondii* may be highly virulent for people resulting in acute clinical disease and blindness in affected immune-competent individuals[5].

Economic impact: Bennett and Ijpelaar (2003)[6] estimated that the cost of *Toxoplasma* infection for the sheep industry is £12 million annually.

Climate change impact/implications: If the climate gets milder and wetter, then oocyst sporulation and survival within the environment will increase because the oocysts do not survive freezing or drying out. Another important implication of climate change will be an increase in heavy rainfall events. This will mean that oocysts will be washed away from cat defaecation sites by rain water and spread within the environment. Recent research by Moredun in collaboration with Scottish Water showed that detection of *T. gondii* DNA in water was highest at times of high rainfall[7]. Even low oocyst densities are sufficient to cause infection as single viable oocysts are capable of causing an infection in a naive host. Another implication may also be that the cat breeding cycle is extended so that female cats can give rise to more kittens each year, which will increase the number of definitive hosts available to produce parasite oocysts. Increases in oocysts numbers, larger numbers of viable oocysts and a bigger geographic spread of the oocysts will increase the potential exposure of naive sheep to the parasite during pregnancies and, therefore, result in an increase in abortions. Higher losses to the sheep industry will also mean that potentially more GHGs are produced in order to produce the same amount of lambs in order to compensate for losses.

Climate change mitigation strategies: More efficient use of the available vaccine against *T. gondii*: The seroprevalence study by Katzer et al (2011)[8] has shown that less than a quarter of sheep farmers used the vaccine. Better uptake of the vaccine would result in reduced losses due to toxoplasmosis and thereby reduce the impact of the disease on GHGs.

Climate change adaptation strategies: Neutering of female cats within farms will help to reduce kitten numbers, which in turn will reduce the numbers of oocysts produced on a farm and thereby limit the exposure of sheep flocks to the parasite. Euthanasing old or potentially immune compromised cats on the farm will also help as these cats may start to shed parasite oocysts again following challenge. Vaccination of sheep to help prevent congenital toxoplasmosis. Further recent work by Moredun has shown that vaccination of lambs (Katzer et al 2014)[9], and pigs (Burrells et al 2015)[10] will significantly reduce the *T. gondii* tissue cyst burden in food animals.

Table 2. Disease control options and costs: (a) applicability (i.e. what % of the affected animals could the option be applied to (b) effect of treatment.

	Control option	Applicability	Effect	Cost
Diagnostics	Serology	Live animals	Identify naive and infected animals	
	Molecular testing	Abortion cases and placentas, usually for research purposes only	Identify if aborted foetuses were infected with *T. gondii*	
	Pathological examination of abortion cases	Abortion cases and placentas	Identify if abortions were due to *T. gondii* infection	
Treatment	Monensin	Not practical because it will need to be given on a daily basis during pregnancy	Suppresses parasite growth but does not clear the infection	N/A (banned in UK?)
Vaccine	ToxoVax	Vaccination of replacement stock	Effective reduction of abortions due to *T. gondii* infection.	£3 per dose
Grazing/pasture management	N/A	-	-	-
Alternatives	Keep only healthy, adult, neutered cats	Farms that keep cats	A reduction in oocyst contamination of pastures as oocysts shedding is usually seen in young cats	
	Rodent control not based on cats		This will reduce the number of intermediate hosts that are potentially infected with *T. gondii*	

Eradication: Eradication is unlikely to be achievable because there are so many intermediate hosts that are infected and infection will last for the duration of their lives. A vaccine that could be given to cats and that would prevent oocyst shedding could result in a reduced prevalence of the parasite within the sheep population but a further challenge will be that the vaccine has to be administered to kittens before their first exposure to the parasite.

References

1. Blewett DA, Trees AJ: The epidemiology of ovine toxoplasmosis with especial respect to control. Br Vet J 1987, 143:128-135.
2. Bennett, R. and Ijpelaar, J. (2003). Economic assessment of livestock diseases in Great Britain. Final report to Defra; ZZ0102 University of Reading)
3. Burrells, A et al (2015) Vaccination of pigs with the S48 strain of *Toxoplasma gondii* – safer meat for human consumption. Vet Research 46: 47
4. Carellos E.V et al (2013) Congenital toxoplasmosis in the state of Minas Gerais Brazil: a neglected infectious disease? Epidemiol Infect, 1-12
5. Ferreira, I. M et al (2011) Toxoplasma gondii isolates: multilocus RFLP-PCR genotyping from human patients in Sao Paulo State, Brazil identified distinct genotypes. Exp Parasitol, 129: 190
6. Gilbert, R et al (2006) Symptomatic toxoplasma infection due to congenital and post natally acquired infection. Arch Dis Child. 91; 495-498
7. Havelaar et al (2012) Disease burden of food borne pathogens in the Netherlands. International Journal of Food Microbiology, 156: 231
8. Katzer F, Brülisauer F, Collantes-Fernández E, Bartley PM, Burrells A, Gunn G, Maley SW, Cousens C, Innes EA. *Toxoplasma* Seroprevalence in 125 Sheep Flocks in Scotland; increased positivity with animal age provides evidence of widespread environmental contamination by *T. gondii* oocysts. Veterinary Research (2011) 42:121.
9. Katzer, F et al (2014) Immunization of lambs with the S48 strain of Toxoplasma gondii reduces tissue cyst burden following oral challenge with a complete strain of the parasite. Vet Parasitol. 205:46
10. Wells, E et al (2015) Molecular detection of *Toxoplasma gondii* in water samples from Scotland and a comparison between the 529bp real-time PCR and ITS1 nested PCR. Water Research 87: 175

2.8 Ovine enzootic abortion (EAE)

Disease: Ovine enzootic abortion (also known as enzootic abortion of ewes or ovine chlamydiosis) is caused by the bacterial pathogen *Chlamydia abortus* (formerly known as *Chlamydia psittaci* serotype 1 or *Chlamydophila abortus*). It is the most commonly diagnosed infectious cause of lamb loss in many countries worldwide, particularly in lowland flocks that are intensively managed at lambing time. Disease manifests with the discovery of dead lambs usually 2-3 weeks before expected lambing. As well as stillbirths and abortions, lambs can be born weak and fail to thrive dying within 24-48 hrs of delivery.

Host(s) affected: Infections are endemic in sheep in the UK and most European countries. Disease in goats is similar to that occurring in sheep. The disease can also affect cattle, deer, horses, pigs and yaks, amongst other animal species, although infection is more sporadic and epizootic. The organism is zoonotic and infections can occur in humans, principally immunocompromised individuals and pregnant women, where infections can cause influenza-like symptoms, spontaneous abortion, stillbirths and in rare cases death of the pregnant mother.

Health & welfare implications: Following introduction into a naive flock, infection remains inapparent until abortion starts occurring and during this persistent or latent stage it is currently not possible to diagnose infection. Thus, the infection can rapidly spread following introduction with a few abortions in year 1 and up to around 30% of ewes losing their lambs. Infection and disease is propagated through exposure of naive ewes to products of abortion. Thus, the disease is a serious production and welfare issue. Secondary bacterial infections and metritis can develop as a result of retained placentas, which occurs most commonly in goats and cattle, adding to disease severity.

Prevalence in Scotland/UK: Annual figures provided by the APHA for diagnostic testing of submitted abortion material (Veterinary Investigation Diagnosis Analysis (VIDA) reports) show that the disease is responsible for around 45% of all diagnosed cases of ovine fetopathy [1], making it the most common diagnosed cause of infectious abortion in the UK. Other estimates suggest that 8.6% of flocks [2] equating to around 1.7 million sheep are affected annually. Within herds, prevalence during an abortion storm can be as high as 50-60%, with 30% aborting, with annual incidence thereafter being 5-10%. Epidemiological modelling rates show the transmission rate (i.e. contact) and the number of infected replacements introduced at the start of an outbreak to be the main important factors for the development of disease in a flock [3].

Production effect(s): Mainly affects lamb mortality (aborted and stillborn), with low birth weights in weak born lambs that fail to survive. Some ewe mortality possible due to secondary infections and complications. May lead to increase in number of barren ewes. May see a reduced performance in surviving lambs.

Table1. Production effect(s):

Effects	Impact	Estimate of losses
Growth	No data	No data
Production	Meat quantity due to lamb loss	During abortion storm up to 30% of ewes may abort, affecting up to 30% of lambs (a ewe with twins or triplets can have 1 or 2 surviving lambs)
Reproduction	Lamb loss	Estimated to result in losses of £20M per annum [4]
	Barren ewes	Market cost and disposal costs for ewes
Waste	Culling of infected animals	Market cost and disposal costs for ewes
	Purchase of replacement stock	Current price of £1 per kg live weight for commercial stock
Other		

Economic impact: A conservative estimate of costs associated with lamb loss to the UK sheep industry has been estimated at £20M per annum [4]. Average cost of losses per affected ewe has been estimated at £121 for a 500 head flock with a 10% disease incidence, and assuming affected ewes are kept rather than culled; culling would result in higher costs [5].

Climate change impact/implications: This is principally a disease of intensively managed animals that are brought in for lambing with spread of infection resulting from close proximity of animals. Changing temperatures and wet conditions will impact on the survivability and environmental spread of the bacterium and thus it's potential for transmission to livestock and to humans.

Climate change mitigation strategies: (i) Appropriate choice and timing of treatment, control through better use of prophylactics, diagnostics and therapeutics, leading to increased biological efficiency, reduced waste, and reduced spread of infection and disease burden, thus reducing GHG emissions intensity per unit livestock product; (ii) effective screening and vaccination of all replacement animals brought in to reduce risk of importing the disease; (iii) increased farm biosecurity (double fencing, carrion, wildlife etc) to limit risks of introducing disease.

Climate change adaptation strategies: Reduce risks of transmission from environmental contamination and from other infected ewes by changing lambing management strategies to ensure (i) affected animals are isolated, (ii) any products of abortion are disposed of carefully, (iii) affected areas are cleaned with appropriate disinfectants, (iv) and adhering to strict hygiene procedures through careful washing of hands, use of gloves, safe washing of contaminated clothing, etc.

Disease control options and costs: The impact of control strategies will depend on factors such as stocking densities, whether animals are housed for lambing, whether lowland/hill/upland flocks, whether a pedigree flock. Following a survey of UK farmers conducted in 2007 the preferred options for controlling EAE are either through vaccination and/or keeping flocks closed [6]. However, further analysis of data indicates that implementation of these strategies does not provide a guarantee of exclusion of disease from flocks and thus further work is required to improve on current intervention strategies.

Table 2. Disease control options and costs: (a) applicability (i.e. what % of the affected animals could the option be applied to (b) effect of treatment.

	Control option	Applicability	Effect	Cost
Diagnostics	Macroscopic examination of placenta [7,8]	Only when placenta is found and experienced in recognising disease characteristics	Gives a rapid presumptive diagnosis that requires confirmation	Free
	Impression smear to detect organisms [7,8]	All affected animals	Rapid indicator of presence of bacteria that requires confirmation	<£1 per test
	Blood test to detect antibodies to organisms [7,8]	All animals – most show rise in titre at abortion	More specific diagnosis allowing appropriate treatment	Varies (£4-8 per test)
	Antigen detection tests [7,8]	All affected animals (swabs) or those with placental lesions	More specific diagnosis allowing appropriate treatment	Varies (£10-20 per test)
Treatment	Long-acting antibiotics (eg oxytetracycline) [9]	All animals in a flock once abortions start occurring	Reduces bacterial burden, limiting further pathological damage to placenta	<£1 per animal

			and thus reducing lamb losses	
Vaccine	In the UK, 3 commercial vaccines available (2 live, 1 inactivated) with different advantages and disadvantages [9]	All animals should be vaccinated in an affected flock as well as all incoming replacements	Although animals may still abort eventually abortion numbers will be reduced	Varies (£2.50-£4 per animal)
Grazing/pasture management	Rotational grazing following contamination of pasture	Requires access to sufficient pasture	Reduces exposure to contaminated pasture and thus potential risk of infecting naive animals	Unknown
Alternatives	Obtain replacement ewes from flocks in accreditation scheme (PSGHS) [6]	For obtaining disease free replacements	For reducing risks of introducing infection into flock	Unknown
	Closed flocks [6]	Ensure good biosecurity to keep disease out from neighbours and wildlife	To keep disease out	Unknown

Eradication: Not realistic. The two live vaccines have been demonstrated to cause disease in some animals. None of the vaccines completely eradicate shedding of infectious organisms at the time of lambing or abortion, which is an important factor in the propagation of infection. Vaccinating previously infected animals does not necessarily protect them from disease. Animals can appear to be completely normal but still infective, while it is currently impossible to detect whether they are infected or not. There is also no current information on wildlife reservoirs, which are also likely to play an important role in the spread of infection.

References

1. https://www.gov.uk/government/statistics/veterinary-investigation-diagnosis-analysis-vida-report-201
2. Leonard et al., 1993. An estimate of the prevalence of enzootic abortion of ewes in Scotland. Vet Rec 133:180-3
3. Milne et al., 2009. Epidemiological modelling of chlamydial abortion in sheep flocks. Vet Microbiol 135:128-33
4. Wood et al., 1992. Enzootic abortion costs home industry £20m pa. Farmers Weekly 117:60;
5. Wright, 2013, Economic Impact of Health and Welfare Issues in Beef Cattle and Sheep in England;
6. Longbottom, et al., 2013. Evaluation of the impact and control of enzootic abortion of ewes. Vet J 195:257-9
7. Longbottom, D. and Sachse, K. (2012). Enzootic abortion of ewes (ovine chlamydiosis). In: Manual of Diagnostic Tests and Vaccines for Terrestrial Animals. 7th Edition. World Organisation for Animal Health (OIE) Chapter 2.7.7, pp. 1008-16
8. Sachse et al., 2009. Recent developments in the laboratory diagnosis of chlamydial infections. Vet Microbiol 135:2-21
9. Essig, A and Longbottom, D, 2015, *Chlamydia abortus*: New Aspects of Infectious Abortion in Sheep and Potential Risk for Pregnant Women. Curr Clin Micro Rep 2:22–34

2.9 Lungworm - Parasitic bronchitis

Disease: Parasitic bronchitis is commonly caused by a range of roundworm (specifically lungworm) infections in a wide array of hosts. In cattle, the syndrome is commonly referred to as hoose or husk. Acute infections are characterised by persistent coughing, increased respiratory rate, ill-thrift, weight-loss and in serious cases mortalities are common. In addition to the damage caused by the primary infection secondary bacterial infections of the lungs may also occur. The disease is unpredictable, can affect both young and adult stock, and although it can be caused by a number of different worms, is predominantly caused by *Dictyocaulus* species.

Host(s) affected: Endemic within livestock viz. young and adult sheep, cattle, goats, deer, equids, llamas, alpacas and pigs. The predominant species are; *Dictyocaulus viviparus* in cattle, llamas and alpacas, *Dictyocaulus filaria, Protostrongylus rufescens and Muellerius capillaries* in sheep and goat, *Dictyocaulus eckerti* in deer *Dictyocaulus arnfieldi* in donkeys and horses and *Metastrongylus apri* in pigs.

Health & welfare implications: Infections range from acute, where mortalities may occur, chronic, where morbidity and premature culling may occur, to sub-clinical, where the impact on productivity is insidious and difficult to diagnose. Reported morbidity rates in infected herds range from 6-100%, with morbidity generally >50% of herd [1, 2)]. If exposure to larval challenge in young stock is low (i.e through overuse of anthelmintics or movement of animals from clean areas to contaminated land) animals are unable to build a strong immunity to infection. If these animals are subsequently exposed to a high challenge, they can develop what is referred to as 're-infection syndrome'. The prevalence of anthelmintic resistant lungworm is currently unknown but anecdotal reports suggest that it is present across the UK; this will have an overall impact on the ability of producers to effectively control these infections.

Prevalence in Scotland/UK: No active surveillance in conducted in the UK. Regional Veterinary laboratory reports suggest that parasitic pneumonia was the highest cause of non-bacterial respiratory disease in cattle in Great Britain (VIDA) and Ireland (AHI).

Production effect(s): The magnitude and scale of losses can differ considerably and can be influenced by a range of factors e.g. breed, sex, nutritional status, previous exposure etc. Losses attributed to reproduction, growth, production and wastage can be observed.

Table1. Production effect(s):

Effects	Impact	Estimate of losses
Growth	Liveweight gain	Reduced DLWG as result of ↓ milk yield in heifers, ~22 weeks delay in reaching slaughter weight [3]
Reproduction	Reduced fertility performance	Delays in calving interval (~10-52 days) Delays in first service to conception[4] Increased inseminations per conception ~↑ 0.2-1.3 inseminations
	Abortion	Respiratory distress leading to loss of calf ~3% abortions following moderate infection[4]
Production	Carcass quality	
	Meat quantity	
	Milk quantity	15-30% (3-6kg/cow/day (5); COWS) loss in output in affected animals and as a result of milk being discarded if zero day withdrawal anthelmintic product not used
Waste	Lung condemnation	Information from processor unavailable
	Culling unproductive stock	
	Mortalities followed by purchase of replacement stock	~5-14% of herd (2-5), Current price ~1-2 per Kg live weight for commercial stock

Other	Reduced body condition scores	Difficult to quantify, but impacts on overall productiveness of animal.
	Susceptible to secondary infection	Anthelmintic cost (~£6 per head), antibiotics cost in treatment (~£ per head) and veterinary time

Economic impact: A conservative estimate of the cost of moderate to severe lungworm outbreaks in dairy cattle range from €160-300 per cow [4, 5]. The estimates include production and reproductive losses as well as additional expenditures incurred as a result of infection (e.g. diagnostics and anthelmintics).

Climate change impact/implications: Seasonality, prevalence, geographic spread, disease outbreaks driven largely by prevailing climatic conditions, especially temperature, humidity and rainfall. Lungworm risk forecast to increase over coming decades, based on UK climate projections. Lungworm infection contributes to carbon footprint of livestock production through reduced biological efficiency and increased waste.

Climate change mitigation strategies: i) Appropriate choice/timing of treatments, control through better use of diagnostic leading to increased biological efficiency & reduced waste, efficient treatment of livestock leads to animals reaching target market weight earlier, thus reducing GHG emissions intensity per unit livestock product, ii) minimise co-grazing of paddock with susceptible stock for example horses and donkeys or sheep and goats thereby reducing exposure of naive animal to high infection rates, iii) Effective quarantine treatment reduces the risk of importing anthelmintic resistant parasites onto farms. Resistant parasites can lead to sub-optimal growth and productivity and waste time and resources in control, iv) reducing stocking densities leads to lower contamination rates on pasture and subsequently lower infection rates in susceptible stock v) strategic use of vaccine prior to the start of grazing or exposure to probable infection.

Climate change adaptation strategies: i) changing pasture management and/or grazing strategies to minimise the exposure of susceptible animals to high infection levels e.g. housing pig.
Disease control options and costs: Some of the impact of control strategies will depend on the factors such as stocking densities, topography of the enterprise etc.

Table 2. Disease control options and costs: (a) applicability (i.e. what % of the affected animals could the option be applied to (b) effect of treatment.

	Control option	Applicability	Effect	Cost
Diagnostics	Baemannisation/larval counts	All stock can be examined	More efficient use of treatments	£10-20 per count, various suppliers
	Bulk milk ELISA	Lactating heifers, relates to incidence of lungworm-related morbidity than to prevalence of lungworm infection Sekiya et al 2013 IVJ	More efficient use of treatments	
	Test for examining anthelmintic sensitivity	All drug classes and stock can be examined at treatment	Efficient drug treatment can improve quantity and quality of livestock products (see Table 1, above)	£30 per treatment; SAC VIS

Treatment	Therapeutic (curative) or prophylactic (preventative) administrations	All stock can be administered anthelmintic treatment (> 6 week old)	Efficacy may be compromised by anthelmintic resistance	£0.1-1 per animal
	Quarantine treatment of new/retuning livestock to avoid dissemination of resistant roundworms.	All stock can be administered anthelmintic treatment (> 6 week old)		£0.1-1 per animal
Vaccine	Bovilis® Huskvac	All stock > 8 week old, two doses ~ 4 weeks apart.		~£6 per dose

Eradication: Not perceived as possible, due to wildlife reservoirs and set stocking rates

References

1. David, 1993, Increased prevalence of husk. Vet Rec. 1993;133(25/26):627;
2. David, 1997, Survey on lungworm in adult cattle. Vet Rec. ;141(13):343-4;
3. Borsberry, 2012, Impact of lungworm on cattle health and future production. Vet Times [Internet]. 2012 accessed 26JAN16. Available from: http://www.vettimes.co.uk/article/impact-of-lungworm-on-cattle-health-and-future-production/;
4. Wooley, 1997, The economic impact of “husk”, Cattle Practice 5(4):4;
5. Holzhauer et al. 2011, Lungworm outbreaks in adult dairy cows: estimating economic losses and lessons to be learned. Vet Rec.;169(19):494.

2.10 Liver Fluke, Fasciolosis

Pathogen/Disease - Fasciolosis; Common name: Liver fluke disease; Causative agent, parasitic flatworm, *Fasciola hepatica*. Mammalian hosts become infected by ingesting fluke cysts (metacercariae) shed by infected mud snail intermediate host, typically *Galba truncatula* in UK.

Host(s) affected - Mainly grazing livestock viz. sheep, cattle, goats, but also horses and wildlife (deer, rabbits, hares). Liver fluke is zoonotic and occasionally found in humans in UK/Europe, considered a Neglected Tropical Disease in some parts of the world e.g. S. America.

Animal Health & welfare implications - Acute fluke disease can cause sudden death of previously healthy animals, especially sheep. Chronic disease causes abdominal pain, inappetance, reduced weight gain, ill-thrift.

Prevalence in Scotland/UK - No active surveillance, prevalence indicated by diagnosable submissions to regional VI Centres (e.g. SAC, APHA, AFBI), liver condemnation rates at UK abattoirs and occasional scientific studies/surveys. Recent estimates are ~10% in sheep and ~25% in cattle.

Production effect(s) – the extent of production effects observed is dictated by the host species, fluke burden and whether the infection is acute, sub-acute or chronic. Typically, reduced growth rates; extended time to slaughter, reduced feed conversion ratios; reduced reproductive performance; reduced quality/quantity of meat & milk; increased waste through liver condemnations at slaughter, see Table 1, below.

Table1. Production effect(s):

Effects	Impact	Estimate of losses
Growth	Reduced liveweight gain	10% in ewes, 30% in lambs[1], 10-15% in cattle[2]
Reproduction	Reduced reproductive performance	Increased barren ewes & cows, extended lambing/calving interval (13 days)[2]
Production	Extended time to slaughter	Study of 450,000 prime beef cattle – 'fluky' cattle 2.5kg lighter and 27 days older (Harbro Ltd., 2014)
	Reduced feed conversion ratios (FCR)	Sheep FCR uninfected 4:1, infected 20:1 i.e. £1 to put on 1kg liveweight becomes £5; (W. Thomson, Harbro Ltd., pers comm)
	Reduced quality/quantity of meat & milk	Reduced fat content & milk yield (8-15%)[2]
Waste	liver condemnations at slaughter	100s of kgs/day (Scotbeef, pers comm)
Other	Reduced body condition score (BCS)	Captured by reduced growth & carcase quality

Economic impact - Liver fluke estimated to cost GB sheep and cattle sector £13-15 million in 2011, recent work suggests it could be significantly higher[3]. £200/head for beef/dairy cattle in Switzerland[2]; £10-£25 per infected sheep in UK[1]; uncontrolled outbreak in Scottish sheep cost ~£20K = £8/ewe[4]. Liver condemnations in cattle alone cost over £1.7 million annually[5], similar losses have been estimated for the sheep sector[6].

Climate change impact/implications - Seasonality, prevalence, geographic spread, disease outbreaks driven largely by prevailing climatic conditions, especially temperature and rainfall. Fluke risk forecast to increase over coming decades, based on UK climate projections[7]. Fluke infection contributes to carbon footprint of livestock production through reduced biological efficiency and increased waste.

Climate change adaptation/mitigation options – Adaptation: (i) change breeds and/or select for resistance/resilience to fluke? No evidence of any heritable capacity in sheep or cattle to suggest this is feasible. (ii) House animals to reduce risk of fluke infection? Expensive in terms of feed requirements, also brings added health & welfare issues, possible fluke risk in silage/haylage. Mitigation: improved/sustainable disease control leading to increased biological efficiency & reduced waste, fluke-free animals reach target market weight earlier thus reducing GHG emissions intensity per unit livestock product.

Disease control options - Diagnostics: faecal egg counting, coproantigen ELISA, serum ELISA, liver/bile duct enzymes; Vaccination – highly desirable, focus of international research effort but none commercially available, compounded by lack of natural protective immunity in sheep or cattle; Anthelmintic drugs – not many to choose from and have emerging resistance problems, especially with triclabendazole, the drug of choice for acute fluke control; Quarantine treatment of incoming stock rarely practiced properly, if at all; Grazing/pasture management: on-farm risk analysis, temporary/permanent fencing to prevent stock gaining access to high risk pasture at high risk times; Pasture improvement to reduce snail habitat e.g. rolling poached areas, removing rushes; Improved drainage – can run counter to agri-environment/wetland schemes

Diseases control costs - Diagnosis £7-20 per test (individual and/or composite, e.g. SAC VIS); Flukicidal drugs @ ~50p (sheep)-£3 (cattle) per treatment, labour costs, facility cost; Pasture improvement ££: Fencing ££; Drainage £££?

Eradication – Not feasible because of ubiquitous snail intermediate host and presence of wildlife reservoir hosts**.**

Table 2. Disease control options and costs: (a) applicability (i.e. what % of the affected animals could the option be applied to (b) effect of treatment.

	Control option	Applicability	Effect	Cost
Diagnostics	Faecal egg count (FEC)	All stock can be examined	More strategic use of chemical treatments i.e. better product choice, timing	£10-20 per sample, depends if individual animal or group pooled/composite; various providers e.g. vet practices, SAC VIS
	Testing for anthelmintic efficacy, faecal egg count reduction test (FECRT)	All stock can be examined	Informed decision-making regarding which treatments are effective and which are not; confirmation of resistance status	£20 for a post-drench efficacy check; ~£70-100 for a complete FECRT
	Coproantigen ELISA (cELISA)	All stock can be examined	More strategic use of chemical treatments i.e. better product choice, timing	£16 per individual animal, £30 composite, various providers e.g. SAC VIS, BioBest
	Serum antibody test (AbELISA)	All stock can be examined	Indicates if an animal has been exposed to fluke, not necessarily currently infected	£7-10 per animal, various providers e.g. SAC VIS, BioBest
	Blood biochemistry i.e. liver and/or bile duct enzymes	All stock can be examined	Indicates bile duct and/or liver damage, not specific for liver fluke	£10 per animal, various providers, SAC VIS
Treatment	Chemical flukicides (anthelmintics) targeting different fluke stages, available as injectables, drenches & pour-ons	All stock >6 weeks of age	Efficacy can be compromised by over-use, misuse or resistance. Withdrawal periods apply for meat & milk	£0.1-1 per animal, depending on host, drug & route of administration
	Quarantine treatment of incoming/returning stock	All stock >6 weeks of age	To avoid bringing fluke (possibly resistant) onto a farm	£0.1-1 per animal, depending on host, drug & route of administration
Vaccine	Subject of international research effort; none currently available	n/a	To protect animals from infection and/or clinical signs of disease	n/a
Other	Improved drainage	Yes, but runs counter to current agri-environment schemes & flood risk management policy	To reduce intermediate host snail habitat	~Large-scale drainage would be expensive, but small gains could be made by repairing broken drains, fixing leaking water troughs, rolling heavily poached areas etc.
	Fencing	Yes, doesn't have to be permanent	To reduce contact between livestock & marginal/snail habitat at high risk times	Large-scale fencing likely to be expensive, but temporary (electric?) fencing feasible; may be win-wins with other agri-environmental objectives e.g. catchment management, riparian strips etc.

	Breeding for host resistance to fluke	No	No evidence of any genetic component to host resistance in any breed or host species	n/a

References

1. Sykes A. et al., 1980. Chronic subclinical fascioliasis in sheep: effects on food intake, food utilisation and blood constituents. *Res Vet Sci* 28(1): 63-70
2. Schweizer G. et al. 2005. Estimating the financial losses due to bovine fasciolosis in Switzerland. *Vet Rec*. Aug 13;157(7):188-93
3. EBLEX stock briefing 2012, http://www.eblex.org.uk/documents/content/publications/stock-briefing_12-03autumn_liver_fluke050412.pdf
4. Sargison N.D. & Scott, P. 2011. Diagnosis and economic consequences of triclabendazole resistance in *Fasciola hepatica* in a sheep flock in south-east Scotland. *Vet Rec*. Feb 12;168(6):159
5. EBLEX stock briefing 2012, http://www.eblex.org.uk/documents/content/publications/stock-briefing_1205autumn_liver_fluke011012.pdf
6. ADAS Report for EBLEX, Economic Impact of Health and Welfare Issues in Beef Cattle and Sheep in England. 2012, pp 19, 36
7. Fox N. et al., 2011. Predicting Impacts of Climate Change on *Fasciola hepatica* Risk. *PLoS One* Jan 10;6(1):e16126

2.11 Leptospirosis

Disease: Leptospirosis is a disease caused by bacteria of the genus *Leptospira*, which can affect multiple animal species. In beef and dairy cattle, leptospirosis is associated with infertility, abortion and stillborn or very weak calves. It may also cause fever and milk drop syndrome. Leptospirosis is a zoonosis, i.e. the organism is shared between animals and people and causes disease in people.

Host(s) affected: Leptospirosis occurs in a wide range of animals, including (in the UK) cattle, pigs, horses, dogs and rodents [1-5]. Within the genus *Leptospira*, multiple pathogenic species and serovars are recognized. Cattle in the UK are primarily affected by *Leptospira* Hardjo serovars. The nomenclature is complicated, but *Leptospira borgpetersenii* serovar Hardjo (Hardjobovis, HB) and *Leptospira interrogans* serovar Hardjo (Hardjoprajitno, HP) are the most frequently documented serovars in cattle in the UK. Other ruminant hosts such as sheep and deer may also be infected with those serovars but infection is typical subclinical.

Health & welfare implications: In people, leptospirosis ranges in severity from a mild or subclinical infection to a severe, life-threatening illness. Most cases of leptospirosis in the UK are travel-associated. In the epidemiology of UK-acquired leptospirosis, two main patterns are important, whereby leptospirosis may be transmitted to people via urine from wild rodents (e.g. rats) or from infected domestic cattle. Dairy farmers are the main group at risk for cattle leptospirosis, with additional cases in vets, meat inspectors, butchers and abattoir workers. Sewage workers and people in contact with canal and river water are also at risk[4]. In cattle, acute infection is often subclinical, but milk drop syndrome (sudden drop in milk production in all four quarters with or without fever, soft flabby udder with colostrum-like appearance of milk) is seen in dairy cattle, and acute systemic disease is also reported. Chronic infection may cause infertility, abortion, stillbirth, premature birth and reduced calf viability [2].

Prevalence in Scotland/UK: Prevalence in the UK has been estimated based on detection of antibodies, which can result from infection or from vaccination. From 1997 to 2001, ca. 75% of 12,504 bulk milk samples from England and Wales were positive for *Leptospira* serovar Hardjo antibodies[6]. Between 2008 and 2010, 72% of ca. 1,000 unvaccinated herds throughout the UK tested were positive based on bulk milk testing[7]. The prevalence in Scotland may be lower, with 30-40% of dairy and beef herds positive for leptospirosis antibodies according to a 2015 report in Scottish Farmer.[8] Similarly, the prevalence in 109 breeding bulls in south west Scotland was estimated at 27% in 1992/1993[9]. A recent review of global leptospirosis estimated the total average UK burden of morbidity of human leptospirosis as 0.72 – 4.27 cases per 100,000 per annum[10]. In Scotland, based on data from 2010-2015, there are ca. 3 cases per year, of which roughly half are travel associated (Lynda Browning, NHS; personal communication).

Production effects: Leptospirosis affects milk production of dairy cattle, fertility of bulls and female beef and dairy cattle, and foetal and calf survival. Reduced birth weight and retained placenta may also occur.

Table1. Production effect(s):

Effects	Impact	Estimate of losses
Growth	Reduced birth rate	
Production	Milk drop	Estimates are highly variable. According to some sources, most animals return to almost full milk production in 10–14 days[2]. Other reports range from no yield loss to loss of 10% of annual yield in affected cows, with the proportion of affected cows dependent on whether infection is new or chronic[11,12].
	Reduced fertility	"Abortion, stillbirth, premature birth, the birth of weakly calves and reduced birth weight are the most important economic aspects of chronic leptospirosis in cattle"[2]

		"In endemically infected herds, where young stock are exposed to infection before breeding, levels of associated reproductive wastage are very low"[2]
Waste	Abortion	
Other	Occupational zoonosis in humans	Herd owners have responsibilities towards dairymen working in milking parlours under COSHH regulations.

Economic impact: "Abortion, stillbirth, premature birth, the birth of weakly calves and reduced birth weight are the most important economic aspects of chronic leptospirosis in cattle"[2], but specific estimates of incidence of infection, abortion, infertility etc. or of costs associated with those are difficult to find. "Control decisions are not always based on the obvious ones of reducing/preventing human infections and preventing clinical disease and economic loss in animals. Other considerations include achieving economic or strategic advantage for either the national or individual herds"[2]

Climate change impact/implications: Leptospirosis affects the biological efficiency of food production, e.g. by reducing milk yield and calf crops, resulting in a higher carbon footprint per unit of milk or meat. Many Leptospira serovars are environmentally transmitted and heavy rainfall and flooding events may contribute to the survival and spread of the organism. . After widespread flooding in the UK in December 2015, veterinarians alerted dog owners to the increased risk of the Weil's disease variant. However, the cattle derived Hardjo-serovars are mostly transmitted through direct contact and have poor environmental survival.

Climate change mitigation strategies: Clinical leptospirosis in cattle is rare due to widespread use of vaccines. There is limited data on cost and benefits of vaccine based vs. vaccine free control strategies.

Climate change adaptation strategies: Risks are reduced in housed herds compared to extensively managed herds, and in closed herds compared to herds that purchase animals or bring in bulls[2,7].

Disease control options and costs: Current control in the UK is largely based on vaccination, although alternative control options, including eradication from individual herds, exist.

Table 2. Disease control options and costs: (a) applicability (i.e. what % of the affected animals could the option be applied to (b) effect of treatment.

	Control option	Applicability	Effect	Cost
Diagnostics	Culture	Outbreak investigation, blood, milk, foetus	Detect bacteria. High specificity but low sensitivity and labour intensive.	Not known
	PCR, real time PCR	Currently post-mortem, could be used in vivo	Detect bacteria, e.g. in kidneys, urine	£28.15 (APHA)
	ELISA for antibodies to *Leptospira* Hardjo serovars in blood (individual)	Beef and dairy	Serovar specific: Detect antibodies to Hardjo serovars only. No distinction between infection and vaccination. False positives and false negatives may occur.	£5.70 (APHA, screening); £5.40 (SRUC)
	ELISA for antibodies to *Leptospira* Hardjo serovars in milk (individual or bulk tank)	Dairy cattle only. Good to demonstrate freedom of disease and comply with COSHH; false positive results may occur due to cross-reactivity		£5.30 (APHA), discount for packages covering multiple infectious agents; £5.50 (SRUC).
	MAT for antibodies to *Leptospira* serovars in blood (individual)	Demonstrate exposure and confirm infection in clinical cases.	Can be used in any species (cf. ELISA). Detect antibodies and can confirm acute infection with paired serology (rising titre). No distinction between infection and vaccination. MAT results can be used to gain a broad picture of circulating serogroups at the population level but cannot be used to identify the infecting *Leptospira* species.	£7.50-£9.50 per serovar (APHA). Typical panels range from 1-12 serovars per test.
Treatment	Antimicrobial (whole herd treatment with Dihydrostreptomycin)	Control of active chain of infection.	Bacterial infection cleared	
Vaccine	Vaccine	Widely used in UK	No DIVA vaccine. Most diagnostic test cannot differentiate between infection and vaccination. Reduces shedding, improves fertility	£2.30
Grazing/pasture management	Piped drinking water		Prevents transmission of bacteria from urine via streams and contaminated pasture	Not known
	Removal to clean pasture after treatment			Not known
	Avoid shared grazing with other herds			Not known
	No co-grazing with sheep		Prevents transmission from sheep	Not known
Alternatives	Biosecurity: purchase of replacements	Screen animals prior to purchase	Prevention of introduction via urine, maintenance of free status	Not known
	Biosecurity: use of clean bulls or artificial insemination	Screen animals prior to use as breeding bull	Prevention of introduction via semen or urine	Not known

Eradication: Eradication is theoretically possible in closed cattle herds due to the existence of a vaccination for Leptospira Hardjo and potential to treat infected animals with antibiotics to clear chronic infection. Care should be taken on mixed units as small ruminants and deer are also susceptible to Hardjo infection and may act as reservoirs for cattle. The feasibility of control and eradication may differ between extensively farmed and housed herds[11]. In Scotland, accreditation of freedom of disease is possible through herd health schemes such as those offered by SRUC, Biobest Herdcare or HerdSure. In some countries (e.g. The Netherlands) milk for human consumption must come from certified *Leptospira* free animals. Eradication is also an important consideration for farmers who wish to sell bulls into AI stations or be involved in embryo transfer[2].

References:

1. Ball C, Williams N, Dawson S. Prevalence of *Leptospira* cases in the vet-visiting dog population in the UK. Vet Rec. 2011 Jul 30;169(5):132. doi: 10.1136/vr.d4810.
2. Ellis WA. Animal leptospirosis. Curr Top Microbiol Immunol. 2015;387:99-137. doi: 10.1007/978-3-662-45059-8_6.
3. Gelling M, Zochowski W, Macdonald DW, Johnson A, Palmer M, Mathews F. Leptospirosis acquisition following the reintroduction of wildlife. Vet Rec. 2015 Oct 31;177(17):440. doi: 10.1136/vr.103160.
4. http://www.hse.gov.uk/pubns/indg84.pdf-289.
5. Whitwell KE, Blunden AS, Miller J, Errington J. Two cases of equine pregnancy loss associated with *Leptospira* infection in England. Vet Rec. 2009 Sep 26;165(13):377-8.
6. Pritchard G. Milk antibody testing in cattle. In Practice 2001 Oct;542-549.
7. Williams D, Winden SV. Risk factors associated with high bulk milk antibody levels to common pathogens in UK dairies. Vet Rec. 2014 Jun 7;174(23):580. doi: 10.1136/vr.102049.
8. http://www.thescottishfarmer.co.uk/livestock/dairy/data-confirms-lepto-threat-in-scotland.26804526.
9. McGowan AC, Murray RD. Health status of bulls used for natural breeding on farms in south west Scotland. Zentralbl Veterinarmed B. 1999 Jun;46(5):311-21.
10. Costa F, Hagan JE, Calcagno J, Kane M, Torgerson P, Martinez-Silveira MS, Stein C, Abela-Ridder B, Ko AI. Global morbidity and mortality of Leptospirosis: A systematic review. PLoS Negl Trop Dis. 2015 Sep 17;9(9):e0003898. doi: 10.1371/journal.pntd.0003898.
11. Little TW, Hathaway SC, Boughton ES, Seawright D. Development of a control strategy for *Leptospira hardjo* infection in a closed beef herd. Vet Rec. 1992 Oct 24;131.

2.12 Footrot

Disease: Footrot is a contagious, debilitating disease of sheep, causing major economic losses and welfare problems in most sheep-producing countries. The causative agent is the bacterial pathogen *Dichelobacter nodosus*. Depending on the virulence of the bacterial strain, clinical signs vary from a mild interdigital dermatitis (benign footrot) to severe underrunning of the horn of the hoof (virulent footrot; VFR)[1].

Host(s) affected: Footrot, as caused by *Dichelobacter nodosus*, is primarily a disease of sheep, although transmission of the organism with subsequent disease is possible in cattle[2]. In other host species, e.g. cattle and goats, the term "footrot" is also used, but it generally refers to a clinical hoof condition that is dominated by the presence of pathogens other than *D. nodosus*, notably *Fusobacterium necrophorum*[3].

Health & welfare implications: Footrot is one of the most important health and welfare issues for sheep farming in the UK. Footrot is an extremely painful disease and affected animals can lose weight rapidly. Sheep with footrot can be very lame, remain recumbent for long periods and may not bear weight on the affected leg. When both forelimbs are affected, sheep walk on their knees. Footrot reduces feed intake and performance, and sheep with footrot raise fewer lambs than healthy sheep[4,5].

Prevalence in Scotland/UK: Prevalence of lameness at any one time is > 10% of the national flock in the UK, equivalent to ca. 3 million sheep. In well-managed flocks, the prevalence of lameness can be as low as 2 per cent and this figure represents an achievable target in most situations[4]. Up to 90% of cases is attributed to footrot[4,5].

Production effect(s): Footrot affects feed intake, ewe condition, fleece weight, lambs born per ewe, and time to slaughter for lambs.

Table1. Production effect(s):

Effects	Impact	Estimate of losses
Growth	Poor feed intake, reduced growth[3,6]	Growing animals with average footrot severity suffered weight losses of 0.5 to 2.5 kg live weight, but most animals regained lost live weight as footrot healed following vaccination[8]; It is assumed that growth in lambs will have a reduction of proportionately 0·18[7]; £1.5M/year[7]
Production	Ewe condition	Mean body weight of infected animals was 7.3 kg (11.6%) below that of the control group[9]
	Fleece	Fleece weight of the infected group being 0.4 kg (8%) lighter than that of the controls[9]
	Reproduction losses[5,6]	It is assumed that ewes infected with footrot will have a reduction in lamb output of proportionately 0·18; £5.3M/year[7]
Waste	Culling of chronically affected animals	No data
	Mortality[3]	No data
Other	Welfare impact: painful condition. There is a large amount of legislation detailing the necessity for daily inspection of intensively-managed sheep (lowground and upland farms) and their prompt and correct care and treatment. Farmers must be familiar with the Code of Recommendations for the Welfare of Livestock - Sheep (2002). Other regulations include The Welfare of Farmed Animals Regulations (2007).	

Economic impact: The cost of footrot is estimated at £24 million/yr in the UK (£14M is for preventive measures, £7M for lost production, ca. £3M for culling and treatment[7]). A reduction of proportionately 0·10 in incidence across flocks would have national benefits of £1·0 million annually. If this reduction was achieved by eradication of the disease, considerable additional benefits could be had by reduction of preventive treatments, although eradication is unlike in the UK due to its climate[7]. Animal and flock level cost estimates range from £8.38 per incidence of lameness to £15,000 when having 8% lame sheep in a flock of 1,000 animals[5].

Climate change impact/implications: Rainfall and moisture contribute to the occurrence of footrot and may explain why footrot is easier to eradicate in dry as compared to wet climates[10]. Increased rainfall and flooding events may result in a higher risk of footrot in the UK.

Climate change mitigation strategies: Control of VFR would contribute to improved biological efficiency of production and should contribute to a reduced carbon footprint per unit product.

Climate change adaptation strategies: *D. nodosus* can survive for more than 30 days in soil at low temperatures (5°C)[11]. Improved control of VFR in sheep flocks may reduce the environmental load of *D. nodosus* in soil, hence reducing the risk of re-emergence after periods of rainfall or flooding. The average estimate for heritability of footrot in ewes was 0.2, which means it has a low to moderate heritability and conventional breeding approaches can be used to improve resistance and assist in footrot control[12].

Disease control options and costs: Hoof trimming was long seen as a major tool in control of footrot but based on scientific evidence and education campaigns for farmers and veterinarians, it is increasingly replaced with antimicrobial treatment and vaccination[Winter]. Additional control options include selective breeding, culling of non-responders, and footbathing with the effect of footbathing dependent on country (successful in Australia, much less so in the UK) and quality of footbaths. Foot trimming should be avoided[6,10,13,14].

Table 2. Disease control options and costs: (a) applicability (i.e. what % of the affected animals could the option be applied to (b) effect of treatment.

	Control option	Applicability	Effect	Cost
Diagnostics	Inspection			Not specified
	qPCR [15]			Not specified
	Serotyping [15]			Not specified
Treatment	Topical treatment (oxytetraycline)	Clinically affected animals	Improved recovery	£0.05/dose
	Parenteral treatment	Clinically affected animals	Improved recovery	Ca. £2/dose
Vaccine	Footvax	As part of a 5-point control programme (vaccination, avoiding spread, quarantine, culling, treatment) ; whole flock approach	Reduction susceptibility, improved recovery	£1.10/dose
	Footvax	As emergency treatment	Reduction in clinical lameness	£1.10/dose
Grazing/pasture management	Separate lame animals at pasture		Reduced risk of transmission[16]	Not specified
	Reduce stocking density		Reduced risk of transmission[16]	Not specified
	Avoid spread at gathering at handling	Improving cleanliness and drainage of handling area; use mobile handling unit	Reduced risk of transmission[5]	Not specified

	Avoid spread around water troughs	Put lime around water troughs	Reduced risk of transmission[5]	Not specified
Alternatives	Culling	If a ewe has footrot more than once in a season she should be given a cull tag to help prevent the cycle of infection; may be high in first year but will decrease[5, 17]	Reduction in spread to other animals	Dependent on cull price.
	Quarantine	Separate bought-in stock for 4 weeks after purchase, inspect, treat as needed[5]	Reduction in spread to other animals	Not specified
	Footbathing	Prevention, treatment	Contradictory evidence of positive effect[10, 16]	Not specified
	Hoof trimming	Discouraged	Slower recovery[6,10,13,14]	Not specified

Eradication: In parts of Australia, eradication of VFR has been achieved through quarantine, total destocking, inspection and culling of affected animals, or treatment with or without use of foot-bathing[18]. Vaccination with flock specific vaccines has been used for eradication of VFR in Australia and Nepal[19]. Vaccination, inspection and culling has also been used for eradication of intermediate footrot from individual flocks in Australia[20]. The success of footrot control in Australia is partly attributable to climatic conditions (transmission does not occur at certain times of the year), and partly to higher awareness of available control methods[21]. VFR was introduced into Norway in 2008 and eradicated by 2011 through a concerted eradication campaign, with decreased contact between flocks, footbathing and inspection and culling as major control tools[22]. In recent years, considerable progress has been made in farmer education and lameness reduction in England[13], and awareness campaigns have been conducted in Scotland[23].

References:

1. Kennan RM, Gilhuus M, Frosth S, Seemann T, Dhungyel OP, Whittington RJ, Boyce JD, Powell DR, Aspán A, Jørgensen HJ, Bulach DM, Rood JI. Genomic evidence for a globally distributed, bimodal population in the ovine footrot pathogen *Dichelobacter nodosus*. MBio. 2014 Sep 30;5(5):e01821-14. doi:10.1128/mBio.01821-14.
2. Knappe-Poindecker M, Gilhuus M, Jensen TK, Vatn S, Jørgensen HJ, Fjeldaas T. Cross-infection of virulent Dichelobacter nodosus between sheep and co-grazing cattle. Vet Microbiol. 2014 Jun 4;170(3-4):375-82. doi:10.1016/j.vetmic.2014.02.044.
3. Bennett GN, Hickford JG. Ovine footrot: new approaches to an old disease. Vet Microbiol. 2011;148(1):1-7. doi: 10.1016/j.vetmic.2010.09.003.
4. http://www.nadis.org.uk/bulletins/foot-trimming-of-sheep.aspx
5. http://www.msd-animal-health.co.uk/binaries/Footvax_Lameness_Guide_6pp_tcm80-151756.pdf
6. Wassink GJ, King EM, Grogono-Thomas R, Brown JC, Moore LJ, Green LE. A within farm clinical trial to compare two treatments (parenteral antibacterials and hoof trimming) for sheep lame with footrot. Prev Vet Med. 253.2010 Aug 1;96(1-2):93-103. doi: 10.1016/j.prevetmed.2010.05.006.
7. Nieuwhof GJ, Bishop SC. Costs of the major endemic diseases of sheep in Great Britain and the potential benefits of reduction in disease impact. Animal Science 2005;81:23-29.
8. Nieuwhof GJ, Bishop SC, Hill WG, Raadsma HW. The effect of footrot on weight gain in sheep. Animal. 2008 Oct;2(10):1427-36. doi: 10.1017/S1751731108002619.
9. Marshall DJ, Walker RI, Cullis BR, Luff MF. The effect of footrot on body weight and wool growth of sheep. Aust Vet J. 1991 Feb;68(2):45-9.
10. Smith EM, Green OD, Calvo-Bado LA, Witcomb LA, Grogono-Thomas R, Russell CL, Brown JC, Medley GF, KilBride AL, Wellington EM, Green LE. Dynamics and impact of footrot and climate on hoof horn length in 50 ewes from one farm over a period of 10 months. Vet J. 2014 Sep;201(3):295-301. doi: 10.1016/j.tvjl.2014.05.021.
11. Muzafar M, Green LE, Calvo-Bado LA, Tichauer E, King H, James P, Wellington EM. Survival of the ovine footrot pathogen *Dichelobacter nodosus* in different soils. Anaerobe. 2015 Dec 30;38:81-87. doi: 10.1016/j.anaerobe.2015.12.010
12. Connington et al. http://hccmpw.org.uk/medialibrary/publications/Footrot%20article.pdf

13. Winter JR, Kaler J, Ferguson E, KilBride AL, Green LE. Changes in prevalence of, and risk factors for, lameness in random samples of English sheep flocks: 2004-2013. Prev Vet Med. 2015 Nov 1;122(1-2):121-8. doi:10.1016/j.prevetmed.2015.09.014.
14. Kaler J, Daniels SL, Wright JL, Green LE. Randomized clinical trial of long-acting oxytetracycline, foot trimming, and flunixine meglumine on time to recovery in sheep with footrot. J Vet Intern Med. 2010 Mar-Apr;24(2):420-5. doi: 10.1111/j.1939-1676.2009.0450.x.
15. http://www.langfordvets.co.uk/diagnostic-laboratories/services/molecular-microbiology-diagnostics
16. Kaler J, Green LE. Farmers' practices and factors associated with the prevalence of all lameness and lameness attributed to interdigital dermatitis and footrot in sheep flocks in England in 2004. Prev Vet Med. 2009 Nov 1;92(1-2):52-9. doi: 10.1016/j.prevetmed.2009.08.001.
17. http://www.thescottishfarmer.co.uk/livestock/sheep/fighting-the-footrot-battle.27754347
18. Mills K, McClenaughan P, Morton A, Alley D, Lievaart J, Windsor PA, Egerton JR. Effect on time in quarantine of the choice of program for eradication of footrot from 196 sheep flocks in southern New South Wales. Aust Vet J. 2012Jan-Feb;90(1-2):14-9. doi: 10.1111/j.1751-0813.2011.00872.x.
19. Dhungyel OP, Lehmann DR, Whittington RJ. Pilot trials in Australia on eradication of footrot by flock specific vaccination. Vet Microbiol. 2008 Dec 10;132(3-4):364-71. doi: 10.1016/j.vetmic.2008.05.027.
20. Abbott KA, Egerton JR. Eradication of footrot of lesser clinical severity (intermediate footrot). Aust Vet J. 2003 Nov;81(11):688-93.
21. Abbott KA, Lewis CJ. Current approaches to the management of ovine footrot. Vet J. 2005 Jan;169(1):28-41.
22. Grøneng GM, Vatn S, Kristoffersen AB, Nafstad O, Hopp P. The potential spread of severe footrot in Norway if no elimination programme had been initiated: a simulation model. Vet Res. 2015 Feb 20;46:10. doi: 10.1186/s13567-015-0150-y.
23. http://www.thescottishfarmer.co.uk/livestock/sheep/stamp-out-footrot.27754352

Annex 3: Calculation of Abatement Potential

The parameters that diseases most commonly impact on, and the extent to which the impact can be quantified in GLEAM, are summarised in Table 2., below. The case studies provide details of the actual changes in parameter values that were used to quantify the disease impacts. In most cases, the limiting factor in quantifying the impacts of disease will be the quality of data on disease (e.g. prevalence, impact and vaccination or treatment efficacy) rather than the functionality of GLEAM.

The sensitivity of the EI to a given parameter will depend on the specific details of the system, e.g. in dairy systems the relative importance of different drivers will depend on whether or not sexed semen is used, baseline milk yield etc. In general, the EI of milk will be most sensitive to milk yield and cow fertility rates. In suckler beef and sheep systems, the EI will tend to be sensitive to cow/ewe fertility and abortion rates, calf/lamb mortality and growth rates. The EI of all systems are likely to be sensitive to rates of feed conversion as it affects both the emissions arising from feed production and the emissions arising from excretion (of volatile solids and nitrogen).

Disease impacts on:	Can impact be quantified in GLEAM at present?
Age at first parturition	Y
Age at slaughter	Y
Weight at first parturition	Y
Weight at slaughter	Y
Milk yield	Y
Growth rates	Y
Fertility rates	Y
Fecundity	Y
Calving/lambing interval	Y
Abortion rate	Y
Mortality rate during week 1	Y
Mortality rates of growing animals	Y
Mortality rates of mature animals	Y
Cow culling rate	Y
Replacement rate	Y
Quantity of output	Reduced yield (of milk, meat or wool) captured; condemnation (of lungs, livers etc.) could be captured if rates are known.
Quality of output	Partially, e.g. fat and protein content of milk and meat can vary.
Additional ME (metabolic energy) for immune response	Y, if the effect on ME is known.
Effect on feed conversion rate (FCR)	Y, if the effect on FCR is known.

Table 3. Parameters that diseases impact upon, and the extent to which these impacts can be captured in GLEAM.

To gauge the impact of different parameters from Table 2 on estimated changes in EI, sensitivity analysis was conducted for the three animal categories (beef, dairy, sheep; Annex 4). Together with the qualitative comparative analysis, the sensitivity analysis was used to prioritise diseases for inclusion in the 'Top 3'.

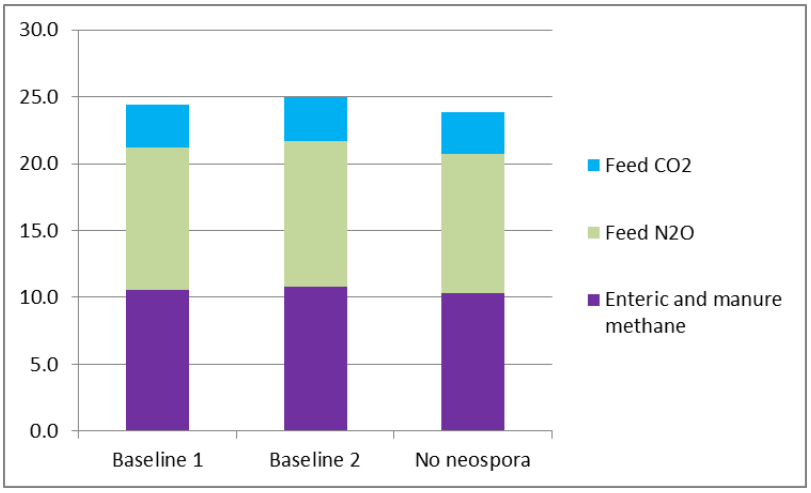
3.1 Neosporosis in Beef Cattle

The emissions and production from an upland suckler beef herd were calculated for three scenarios: no *Neospora*, 10% prevalence and 20% prevalence.

Parameter	No *Neospora*	10% of cattle positive (baseline 1)	20% of cattle positive (baseline 2)
Between herd prevalence	0%	90%	90%
Within herd prevalence (% of cows infected)	0%	10%	20%
Age at first conception (years)	2.49	2.5	2.51
Cow replacement rate	0.136	0.14	0.145
Calving interval (years)	0.995	1	1.005
Abortion rate	3.0%	4.0%	5%
FCR adjustment	98%	100%	102%

Table 4. Input assumptions for modelling of abatement potential for neosporosis

It was assumed that half of aborting cows were culled, and half were retained and mated a year later, leading to a small increase in the herd average age at first conception, calving interval and cow replacement rates. However at the herd level these changes are small as only 1% of cows abort due to *Neospora* (in the baseline 1 scenario), based on the assumption that the average abortion rate is 4%, with 25% of abortions attributed to *Neospora*. The FCR of infected cattle is assumed to be 20% higher (based on Haddad et al. 2005), so the herd average FCR will be change by 20%*10% = 2% for a 10% change in prevalence. GHG by emission category for the three prevalence scenarios are shown below.



The main sources of emissions are methane (mainly from enteric fermentation) and "feed N_2O", which is primarily N_2O arising from the excretion of N by cattle onto pasture.

The EI in the no *Neospora* scenario is 2.2% lower than the baseline 1 scenario, and 4.5% lower than the baseline 2 scenario.

Drivers of EI are shown in Table X.

Figure 3a. Emissions intensity (kgCO2e/kgCW) for the three levels of *Neospor*a prevalence.

Change in	Abortion rate	Age at first calving	Cow rep. rate	Calving interval	FCR
Total GHG emissions	0.5%	<0.1%	-0.5%	-0.2%	-2.0%
Meat output (LW)	1.0%	<0.1%	-0.6%	-0.4%	0.0%
Emissions intensity	-0.5%	<0.1%	0.1%	0.2%	-2.0%

Table 5. Change in total GHG, meat output and emissions intensity arising when the values of the input assumptions are changed individually from the Baseline 1 value to the No Neospora value.

Most of the parameters have little effect on the EI, apart from decreasing the abortion rate (which leads to increased LW output) and decreasing the FCR, which reduces the emissions from both feed production and consumption. Importantly, *Neospora* is the most common cause of abortion in beef cattle, which is why neosporosis was selected for the Top 3.

Caveats/recommendations - The fertility rate of cows infected with *Neospora* (but not aborting) may be lower than those not infected, however we were not able to find evidence to support quantification of this effect. A change in the fertility rate of infected cows (and heifers) is likely to lead to a significant increase in EI, and merits further investigation. The effect of a change in FCR also merits further investigation as it potentially affects all *Neospora* +ve cattle, including young stock and steers/bulls.

Summary - We have reasonable confidence that a reduction in *Neospora* prevalence could lead to a modest reduction in EI via reduction of the abortion rate. There is the possibility of further abatement via improved feed conversion efficiency and cow/heifer fertility. However, evidence on these effects is currently mixed, and largely derived from other production systems, e.g. dairy cattle in the UK or beef cattle in North America. Further work is required to estimate both the prevalence and the reproductive and productive impact of *Neospora* under Scottish conditions and to refine the calculation of abatement potential.

3.2 IBR in Dairy cattle

ADAS (2014, pvi) identified IBR reduction (along with control of liver fluke and Johne's) as one of the key opportunities for GHG abatement in UK cattle because of "its extensive prevalence combined with low estimates of current uptake (below 50%) for the MMs (mitigation measures)" ADAS (2014, piii). Three measures were estimated to have significant abatement potentials at a negative cost (i.e. while providing a net financial benefit to the farmer), see Table 6. Implementation of one of these measures would be likely to lead to a reduction in emissions intensity of milk of approximately 1.5 to 3%.

	AP (ktCO2e)	CE (£/tCO2e)	Ease of implementation	Reduction in disease
Vaccination	277	-95	Med/High	75%
Screening test and cull	231	-4	Med	65%
Double Fencing and Buying Policy	226	-7	Med	50%

Table 6. Abatement potential and cost-effectiveness of three mitigation measures for IBR (adapted from ADAS 2014)

Caveats/recommendations – This table is based on the ADAS report and not specifically adapted to the Scottish situation. Prevalence estimates for Scotland may be available through CHeCS laboratories and/ SRUC. In depth exploration of the feasibility, risks and benefits of IBR control (see Annex 2) could be conducted and should involve

industry stakeholders, GHG modellers and disease modellers, e.g. from EPIC (Centre of Expertise for Animal Disease Outbreaks).

Summary – Control of IBR is technically feasible and would result in reduction of EI for milk production.

3.3 Parasitic gastroenteritis (PGE) in sheep

The main sources of emissions are enteric methane and "feed N_2O", which is primarily N_2O arising from the excretion of N by sheep onto pasture (see Figure 3b). There is considerable variation between systems, reflecting primarily differences in mortality, fertility and fecundity. Three systems are considered here, i.e. lowland, upland and hill sheep.

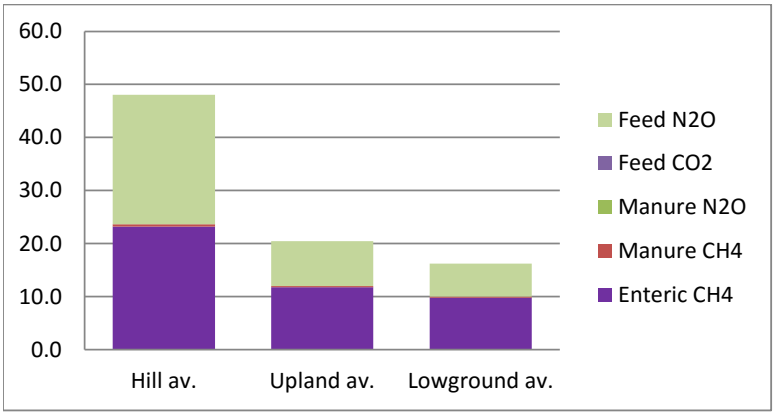


Figure 3b. Emissions intensity (kgCO2e/kgCW) for three sheep production systems with no impact from worms

The input assumptions in Table 7 were used to model the effects of different levels of GI worms. Note that 20% GI means 20% of sheep have performance reduced by GI (not 20% infected by GI).

System	Parameter	No worms	20% GI	40% GI
Hill	Male lamb weight at slaughter (kgLW)	39.3	37.4	35.4
	Female lamb weight at slaughter (kgLW)	36.7	34.8	33.0
	Age at first lambing (years)	2.0	2.1	2.3
Upland	Male lamb weight at slaughter (kgLW)	41.4	39.3	37.3
	Female lamb weight at slaughter (kgLW)	38.6	36.7	34.7
	Age at first lambing (years)	2.0	2.1	2.3
Lowground	Male lamb weight at slaughter (kgLW)	43.5	41.3	39.1
	Female lamb weight at slaughter (kgLW)	40.5	38.5	36.5
	Age at first lambing (years)	2.0	2.1	2.3
	GE intake adjustment to reflect increased feed conversion rate	1.0	1.046	1.092
Hill	Wool (kg sold /ewe/year)	2.0	1.88	1.76
Upland	Wool (kg sold /ewe/year)	2.0	1.88	1.76
Lowground	Wool (kg sold /ewe/year)	2.5	2.35	2.2

Table 7. Input assumptions for modelling of abatement potential for control of PGE in sheep

The results for the three sheep systems with no GI worms ("av."), and with 20% and 40% of sheep affected (not just infected) by GI worms are given in Figure 3c. PGE has a significant effect on EI, with a move from no sheep affected to 20% affected increasing the EI by 9.6%, 8.9% and 9.1% in the hill, upland and lowland systems, respectively.

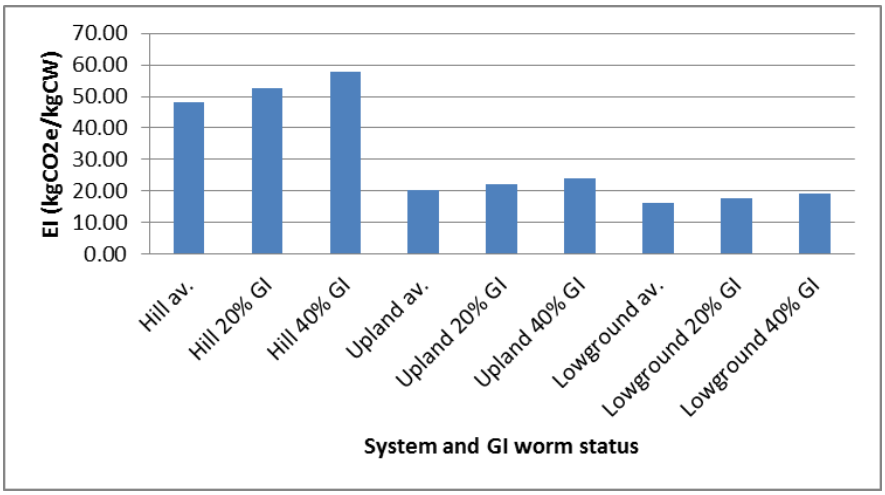


Figure 3c. Sheep emissions intensity (EI) by system and proportion of sheep affected by GI worms.

The decrease in slaughter weight (and growth rate), increase in age at first lambing and the increased feed conversion rate all had a significant impact in the EI (see Figure 3d).

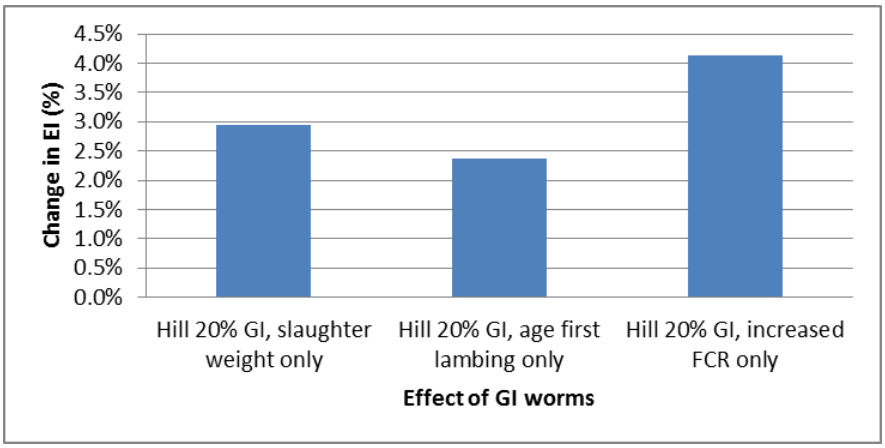


Figure 3d. Change in EI arising from separate impacts of GI worms

Caveats/recommendations - Reduction of PGE in sheep would seem to have a significant technical abatement potential, provided the parasite burden can be reduced in a cost-effective way.

Summary – We, arguably, know enough about sustainable worm control in sheep to perform better than we do. We require improved uptake of best practice advice and a move towards more targeted control of PGE in sheep.

Annex 4 – Sensitivity testing

Estimates of the impact of diseases on EI, and hence on AP, are sensitive to model assumptions. Often, exact quantification of the impact of a disease on production, reproduction, morbidity or mortality is not possible, as studies conducted at different times, in different countries and different production systems with different study designs yield different and sometimes conflicting estimates. To gain a better understanding of which input parameters are most important for estimation of EI and hence need to be quantified most accurately to get an accurate measure of AP, a sensitivity analysis was conducted

Dairy cattle – When calculated with GLEAM, the EI of milk is highly sensitive to feed conversion ratio, cow/heifer fertility rate and milk yield (Figure 4a). Fertility rate and milk yield/day have a direct effect on the total amount of milk produced, while FCR affects the amount of feed that needs to be produced (and therefore the amount of feed related emissions) per unit of output. An increase in FCR also increases the enteric emissions and the amount of volatile solids and N excreted (which leads to higher rates of CH_4 and N_2O emissions from manure management and grazing).

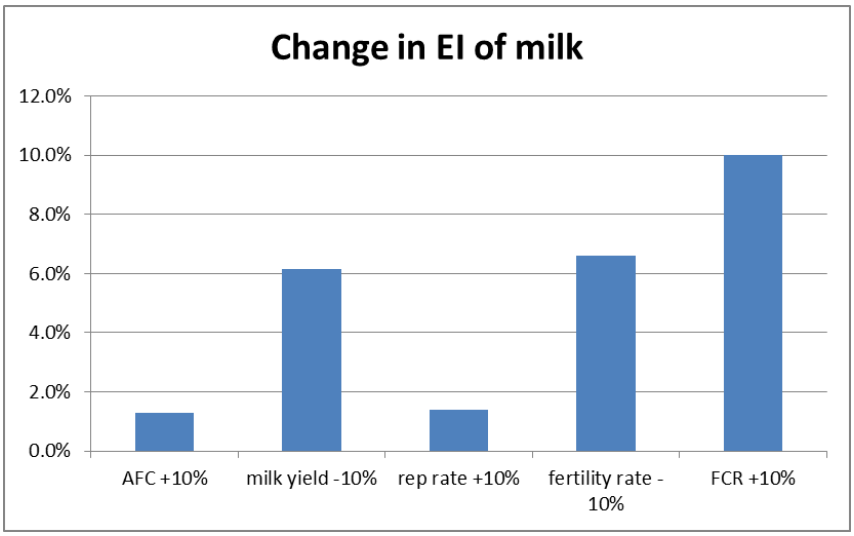


Figure 4a. Change in the emissions intensity of milk with a 10% change in: age at first calving (AFC), milk yield, cow replacement rate, cow/heifer fertility rate and feed conversion ratio (FCR).

The sensitivity of EI to increased cow mortality on EI depends on the assumptions made about how it is managed. Increasing cow mortality by 10% may lead to an increase in cow replacement rate (and an increase in EI). If mortality increases but the replacement rate remains constant then it implies a lower rate of elective culling of poorly performing animals, which would lead to a decrease in herd performance over time.

For calf mortality (pre- and post-natal), the effect on EI depends on the timing of death and how it impacts on milk yield. If calf mortality leads to a significant reduction in milk yield then the cow may be culled (and the replacement rate increased) or kept in which case the herd average fertility rate is reduced. Post-natal calf mortality is likely to have little direct impact on EI as it simply reduces the amount of surplus dairy calves reared for beef, although if sexed semen is used, most calves will be raised for replacement rather than beef. Calf mortality could have indirect effects on EI through (a) reducing the pool from which replacement animals can be taken may lead to a decline in genetic merit and performance over time, and (b) reducing the amount of surplus dairy calves will reduce dairy production, necessitating an increase in the amount of beef produced in (higher EI) suckler systems.

Beef cattle - When calculated with GLEAM, the EI of meat from an upland suckler system is highly sensitive to feed conversion ratio, cow/heifer fertility rate and calf growth rate (Figure 4b). Cow mortality, calf mortality and cow replacement rate have little effect on EI, partly because the baseline values for these parameters are low (2%, 4% and 14% per annum respectively). Therefore, a 10% increase only increases cow mortality from 2% to 2.2%. However, in herds with significant disease challenges, mortality may be much higher and EI may therefore be more sensitive to reductions in it.

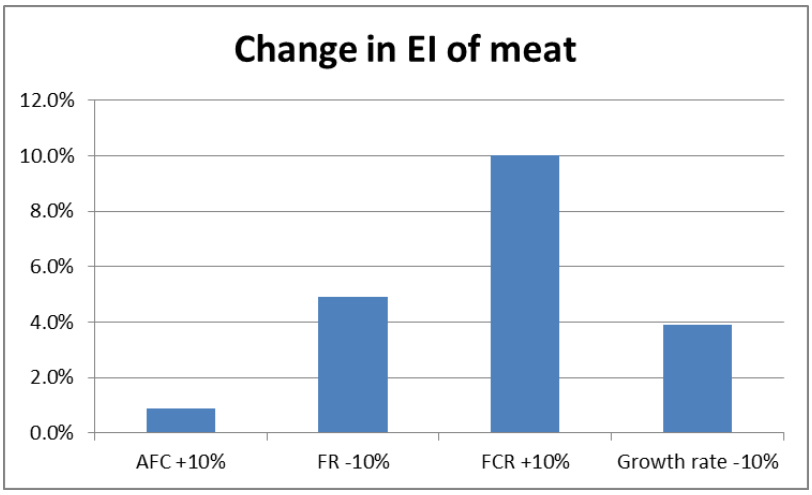


Figure 4b. Change in the emissions intensity of meat from an upland suckler herd with a 10% change in: age at first calving (AFC), cow/heifer fertility rate, calf growth rate and feed conversion ratio (FCR).

Sheep - Analysis undertaken in Eory et al (2015, p110) indicates that the EI in all three sheep systems is sensitive to ewe fertility, lamb mortality and growth rate (reflected in the time taken to reach target weight). Although not quantified in Eory et al (2015), the EI would also be sensitive to the feed conversion ratio.

	Hill	Upland	Lowground
Ewe fertility +5%	-4.8%	-3.9%	-4.0%
Lambs scanned per ewe mated +5%	-4.8%	-4.0%	-4.0%
Lamb mortality from scanning to birth -5%	-0.2%	-0.2%	-0.3%
Mortality aged 0-1 year -5%	-0.5%	-0.3%	-0.2%
Mortality >1 year -5%	-2.3%	-0.4%	-0.6%
Time to target weight -5%	-0.3%	-0.5%	-0.5%

Table 8. % change in EI arising from changing the values of single parameters by + or – 5%

Conclusion - The sensitivity of the EI to change in a given parameter will vary a great deal depending on the starting performance of the herd or flock, and those with below average health status are likely to provide scope for larger and more cost-effective reductions in GHG. It is therefore important that the baseline situation is specified more precisely, in terms of both the physical and economic performance.