

Zoonoses Report

UK 2009



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This document was last updated on 24 January 2011.

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Preface

This Annual Report on Zoonoses in the United Kingdom (UK) presents a summary of reported cases of zoonotic infection in humans in 2009 and their trends and sources. The data have been compiled from statutory notifiable or reportable disease reports, national scanning surveillance systems, control programmes, research programmes and from data submitted to the European Community via the Trends and Sources Report, under the Zoonoses Directive 2003/99 by agencies contributing to the Report.

This report is a collaborative publication produced by:

- Department for Environment, Food and Rural Affairs (Defra)
- Department of Agriculture and Rural Development Northern Ireland (DARDNI)
- Scottish Government
- Welsh Assembly Government (WAG)
- Food Standards Agency (FSA)
- Health Protection Agency (HPA)
- Veterinary Laboratories Agency (VLA)
- Public Health Agency, Northern Ireland
- Health Protection Scotland
- Public Health Wales

Occasional corrections and amendments to the data, many of which are derived from dynamic databases, may occur following publication; these will result in minor changes to subsequent annual reports. Where this is the case for 2008 figures, they have been marked with an ^a symbol.

The format of the report has been changed significantly for 2009. The report now includes feature articles exploring key outbreaks or developments in zoonotic disease in the UK during the year. This is followed by an A-Z section detailing zoonotic diseases which may occur or may be found in the UK. Diseases are no longer split into 'Foodborne and Waterborne', 'Notifiable diseases' and 'Other' as in previous years. In addition, the 2009 report also includes an appendix showing laboratory-confirmed cases of zoonotic disease in humans in the UK, 2000-2009 (Appendix 2) and laboratory-confirmed cases of zoonotic disease in animals in Great Britain (GB), 2000-2009 (Appendix 4).

Executive Summary (2009)

A number of noteworthy incidents of anthrax, VTEC and pandemic influenza H1N1 occurred during 2009, some of which are highlighted in the feature articles. There were also significant trends in a number of human and animal infections which are summarised below, and which emphasise the need for both continued vigilance and surveillance, and collaboration between veterinary and human health practitioners.

Anthrax

Human infection with anthrax is rarely reported in the UK, however 2009 saw the start of a large outbreak of anthrax amongst heroin users in Scotland. Eleven cases were reported during the year with five deaths, and the outbreak continued into 2010. The likely source was heroin contaminated with anthrax spores, either directly or via a cutting agent. All heroin samples tested have been negative.

Pandemic influenza H1N1 (2009) in humans

Initial research indicated that the human strain of the pandemic influenza H1N1 (2009) virus was most closely related to pig strains of the virus in all of its gene segments. However, a perfect match in all of the virus genes has not yet been identified in a single virus in pigs or from any other host. Once the strain emerged there was widespread person-to-person transmission. In humans in the UK, there were two waves of activity in early summer and autumn 2009 with a total of 28,164 laboratory-confirmed cases of H1N1 influenza. This would only have represented a proportion of cases, after the strategy was changed from laboratory reporting to clinical case definitions as fewer cases were referred for laboratory confirmation once the policy was changed. There were also outbreaks of pandemic H1N1 infection in pigs whereby the virus spread from humans to pigs.

Vero cytotoxin-producing *Escherichia coli* O157 (VTEC)

In August and September 2009, a large outbreak of *E. coli* O157 occurred at an open farm in Surrey. This has more than 200,000 visitors per year, including between 1,500 and 2,000 per day during the summer school holidays. There were 93 people known to be affected, of whom 65 were primary cases i.e. infected on the farm, 13 were secondary cases and 15 were asymptomatic carriers. Most of the cases (81.7%) were under 10 years of age. This led to an independent review by Professor George Griffin and a number of recommendations aimed at reducing the risk of infection to visitors to open or petting farms.

In England and Wales about 15% of general VTEC outbreaks have been linked to direct or indirect animal contact, but prior to this large outbreak these have generally each comprised less than 10 cases. During 2009, the VLA assisted with the investigation of 14 outbreaks, including the open farm mentioned above.

In 2009, there were 1,306 laboratory-confirmed cases of VTEC O157 infection reported in humans in the UK, a 6% increase on cases reported in 2008, with Scotland continuing to report higher rates of infection compared to the rest of the UK.

Campylobacter

Campylobacter continues to be the most frequently reported bacterial pathogen in humans. In 2009, the number of laboratory-confirmed human cases of campylobacter infection increased significantly, with 65,000 human cases reported from the UK. The increase was observed throughout the UK, and poultry was considered a main source of infection. The FSA made tackling campylobacter in chicken a priority in reducing foodborne disease using a targeted approach in its 2010-15 strategy released in December 2009.

The national survey for Campylobacter in poultry at slaughter from 2007-2009 found that the estimated prevalence was over 75%. This is consistent with the EU-wide baseline survey carried out in 2008, which estimated that the UK prevalence for campylobacter in broilers at slaughter (caecal contents) was 75.3% with 86.3% broiler carcasses contaminated.

Cryptosporidiosis

In 2009, there were 5,577 human cases of cryptosporidiosis reported in the UK which was 13.6% higher than in 2008. The increase in reported cases was mainly from England and Wales. Typing of isolates has shown that around half the cases reported are *Cryptosporidium parvum* and half are *C. hominis*. *C. parvum* is mainly zoonotic and represents the spring peak, whereas *C. hominis* is mainly transmitted from person-to-person and is responsible for the autumn peak.

There were 1,347 diagnoses of animal infection with cryptosporidium recorded in GB, and 90 in Northern Ireland in 2009. Of these, 1,373 were in cattle, 48 were in sheep, five were in goats and 11 were in other animals. Recorded incidents in cattle and sheep showed a distinct seasonal distribution, with a peak in the spring.

Listeria

Listeriosis remains a relatively uncommon infection in humans, but has a high mortality rate and is a targeted disease under the FSA strategy. The level of reported listeriosis infections in the UK has remained relatively stable over the last 5 years. There were 235 cases in the UK in 2009 (of which 35 were pregnancy-associated cases), representing an increase of 13% compared to 2008, with the greatest rise in England and Wales.

Lyme borreliosis

The number of reports of Lyme borreliosis has seen a steady rise in recent years. In 2009, there were 1,395 serologically confirmed cases of *B. burgdorferi* infection in humans in the UK, including 420 laboratory confirmations in Scotland. The cases in Scotland represent a 47% increase on the 285 cases reported in 2008 and are consistent with the upward trend which began in 2005. Investigations are ongoing to establish the reasons behind this apparent increase in incidence.

Salmonella

The number of laboratory-confirmed human cases of salmonellosis continued to fall in 2009, with almost 10% fewer cases than in 2008. As in previous years, *Salmonella* Enteritidis was the most commonly reported serovar, despite the number of *S. Enteritidis* cases decreasing significantly. By comparison, overall in the UK *S. Typhimurium* infections only showed a slight decrease over 2008.

The efforts of industry and government, including the Poultry National Control Programmes for Salmonella, means that the UK chicken breeding, layer and broiler sectors are now largely free of the *Salmonella* serovars of public health importance. In 2009, Official Control Sampling gave an estimated prevalence of salmonella of 0.12%; 0.36% and 0.043% in breeding, layer and broiler flocks respectively. Results for 2009 are therefore well below the Community target of 1%.

Toxoplasma

Under-reporting was known to occur within the national routine laboratory reporting system. An enhanced surveillance system was therefore introduced in England and Wales in 2008 by the HPA, in collaboration with the National Toxoplasma Reference Laboratory in Swansea. In 2009, 422 cases of toxoplasmosis were reported through this scheme, of which 286 had acute infection.

In 2009, exposure to toxoplasma was confirmed in 48.6% of diagnostic sheep sera sampled in the UK.

Introduction

Zoonoses are defined by the World Health Organisation as “diseases and infections which are transmitted naturally between vertebrate animals and man”. Transmission may occur by a number of routes, ranging from indirect contact through food or drink to direct contact through occupational exposure on farms, and from pets or through leisure pursuits. National surveillance schemes for outbreaks of infectious disease and laboratory-confirmed infections, enhanced surveillance schemes for specific zoonoses and notification of infectious diseases all help to build a picture of the burden of zoonotic infection in the human and animal populations.

Notification and Reporting of Zoonotic Diseases

Some (but not all) zoonotic infections are statutorily notifiable or reportable under veterinary and/or human health legislation. A list of these can be seen in Appendices 1 and 3. Relevant animal legislation includes: the Animal Health Act 1981 and its subsequent amendments; the Zoonoses Order 1989; the Specified Animal Pathogens (Amendment) (England) Order 2008; the European Communities Act 1972 and the Transmissible Spongiform Encephalopathies (England) Regulations 2008, Devolved Administrations have equivalent legislation. The relevant human legislation includes the Public Health (Control of Disease) Act 1984 and the Public Health (Infectious Diseases) Regulations 1988. Employers and the self-employed are required to report work-related incidents and diseases (including specified infections) to the Health and Safety Executive (HSE) under the Reporting of Injuries, Diseases, and Dangerous Occurrences Regulations (RIDDOR), 1995 (www.hse.gov.uk/riddor/). Further relevant legislation is listed in Appendix 5.

The significance of notification differs in human and veterinary contexts. In animals, there is an obligation for any person having in their possession, or under their charge, an animal affected or suspected of having a notifiable disease (as listed in the Specified Diseases (Notification and Slaughter) Order 1992, the Specified Diseases (Notification) Order 1996 or the Transmissible Spongiform Encephalopathies (England) Regulations 2008) to immediately notify the local Animal Health Office in England, Wales and Scotland (www.defra.gov.uk/animalhealth/) or the local Divisional Veterinary Office in Northern Ireland. Procedures for notification and control of specified diseases are outlined in the legislation detailed above.

For human cases, medical practitioners in England and Wales have a statutory duty to notify the proper officer of the local authority (usually the Consultant in Communicable Disease Control (CCDC) of the Health Protection Agency (HPA) in England or Public Health in Wales) immediately on suspected clinical diagnosis of a notifiable disease, under the Public Health (Control of Disease) Act 1984 and Public Health (Infectious Diseases) Regulations 1988. In Northern Ireland the equivalent legislation is the Public Health Notifiable Disease Order (NI) 1989 and in Scotland the Public Health (Notification of Infectious Diseases) (Scotland) Regulations 1988 requires similar notification but also includes Lyme disease and toxoplasmosis. A revised list of notifiable diseases, and for the first time a list of notifiable organisms,

were introduced in Scotland, England, Wales and Northern Ireland during 2010 and will be reported in more detail in next year's report.

Surveillance and Recording of Zoonotic Diseases

Humans

In addition to notification of specified infectious diseases, voluntary laboratory reporting and outbreak surveillance are conducted for each of the constituent countries of the UK (Appendix 2 and Appendix 8). Most zoonotic disease surveillance in the UK is based on voluntary laboratory reporting. The UK has to collect and submit data annually under the Directive 2003/99/EC of the European Parliament (The Zoonoses Monitoring Directive), and for publication in the EFSA Community Summary Report on trends and sources of zoonoses, zoonotic agents and foodborne outbreaks in the European Union each year (2008 report published at www.efsa.europa.eu/en/scdocs/scdoc/1496.htm).

A series of steps must be undertaken for data to be recorded in a national surveillance database. An infected individual, if they have symptoms, may consult a clinician, who may then arrange for a specimen to be taken and referred to a microbiology laboratory, which may isolate or positively identify a pathogen, and either the laboratory or clinician then submits a report to their communicable disease surveillance centre. Thus, as not all ill patients will visit a clinician, and not all clinicians will take and submit samples to a laboratory; sampling tends to be biased towards more clinically severe cases in high-risk groups or outbreak related cases. New legislation outlined above in the UK will place a statutory obligation on clinical microbiological laboratories to report the isolation of specified organisms.

The national surveillance centres also receive and collate reports of general outbreaks of foodborne gastrointestinal disease from laboratories, health protection units and local authority environmental health (Public Protection) departments. The minimum dataset on each outbreak is then collected from the appropriate health authority/board through a standardised questionnaire. Surveillance provides information on specific risk factors associated with different pathogens and on trends in the importance of these factors. Enhanced surveillance schemes are also established, either nationally or locally, to provide information on specific aspects of a zoonosis.

Data from the surveillance schemes are reported on national surveillance centre websites and for England and Wales quarterly in the Health Protection Report available at www.hpa.org.uk/hpr/archives/Infections/2009/zoonoses_09.htm. This information for the UK is published annually in the Zoonoses Report.

Animals

In GB, zoonotic diseases in livestock are monitored for the appearance of notifiable or novel diseases or changing trends in endemic diseases, including actual and potential zoonoses. This is done by the following: the Veterinary Laboratories Agency (VLA); the Scottish Agricultural College (Veterinary Sciences Division) (SAC); Meat Hygiene Service (MHS) (from April 2010 this became Food Standards

Agency Operations); and Animal Health (AH). A similar function is performed by the Agri-Food and Biosciences Institute (AFBI) and the Department of Agriculture and Rural Development (DARD) in Northern Ireland. In addition, information may be available from universities, veterinary research organisations and other private veterinary laboratories (Appendix 7).

The VLA and SAC undertake scanning surveillance on behalf of Department for Environment, Food and Rural Affairs (Defra), Welsh Assembly Government (WAG) and the Scottish Government, through the collection, collation and analysis of disease data. This is performed by analysis of test results from clinical diagnostic samples submitted by veterinary surgeons to the VLA Regional Laboratories and to the SAC Disease Surveillance Centres. The results are entered onto the Veterinary Investigation Diagnostic Analysis (VIDA) database, and collated into reports covering GB which are published monthly, quarterly and annually.

These reports, including those specifically relating to non-statutory zoonoses and infections shared between man and animals, are available on the internet: www.defra.gov.uk/vla/reports/rep_surv.htm.

SAC reports can also be found at:

www.sac.ac.uk/consulting/services/s-z/veterinary/publications/gbdiseasereports/

The number and type of samples submitted to veterinary laboratories is influenced by a number of factors including: the within herd prevalence; severity of clinical signs; the level of awareness of a suspected disease; its perceived importance; the value of the animals and the general economic climate.

During 2001, the large outbreak of Foot and Mouth disease disrupted routine animal disease surveillance, and caution is therefore required when comparing information on zoonotic infections in animals in 2001 with information collected in other years.

Risk assessment and control advice for zoonoses

Control policies have been introduced to reduce the prevalence of pathogens in the food chain and other areas. These include the implementation of legislation relating to the production of drinking water and food. The UK Food Standards Agency (FSA), the Health Protection Agency (HPA) and the Local Authorities Co-ordinators of Regulatory Services (LACORS) operate national microbiological food sampling programmes and carry out studies focusing on particular foods, food processes and the production environment. This work enables potential food safety issues to be identified, as well as establishing current levels of microbial contamination in a range of foods. Local authorities also carry out food sampling activities, and data from this activity is systematically collated through the implementation of the FSA's UK Food Surveillance System.

Under the auspices of the FSA, the Epidemiology of Foodborne Infections Group (EFIG) and the Advisory Committee on the Microbiological Safety of Food (ACMSF) bring together UK surveillance data on humans, animals, and food relating to foodborne risks.

More widely, the UK Zoonoses, Animal Disease and Infections (UKZADI) group, which replaced both the UK Zoonoses Group (UKZG) and the Surveillance Group on Diseases and Infections in Animals (SGDIA), also provides a high-level strategic overview and a means of ensuring overall coordination of public health action on zoonoses across in the UK. The multi-agency Human, Animal Infections and Risk Surveillance (HAIRS) group meets monthly to act as a forum to identify and discuss infections with potential for interspecies transfer (particularly zoonoses) and undertake or commission risk assessments where appropriate.

Feature articles

Feature Article 1: Outbreak of Anthrax in Scotland in 2009

Lynda Browning and Susan Brownlie, Health Protection Scotland

In December 2009, two injecting drug users were hospitalised in Glasgow with blood cultures positive for *Bacillus* species. Further testing confirmed these cultures as anthrax. Over the next few weeks more cases were reported and investigated across Scotland. Diagnosis of anthrax was confirmed by the Special Pathogens Reference Unit, HPA Porton by isolation of *Bacillus anthracis* from blood culture or wound tissue, by polymerase chain reaction (PCR) detection or by positive serology. A national Outbreak Control Team (OCT) was established with representatives from public health, microbiologists, Strathclyde Police and other agencies including the Scottish Drugs Forum. All cases reported taking heroin either by injection and/or by other routes including snorting or smoking.

Generally, the cases presented with relatively localised inflammatory lesions, or abscesses related to sites of heroin injection. However some cases presented with symptoms initially suggestive of a sub-arachnoid haemorrhage or haemorrhagic meningitis with rapid deterioration before treatment was available. The range of presentations was wide and inconsistent and included necrotising fasciitis as a co-infection.

Given the nature of the outbreak and the severity of illness in some cases, there were difficulties during this investigation in obtaining reliable and accurate histories of recent drug use. Some cases died before complete histories could be obtained.

The outbreak has continued well into 2010 and the total number of confirmed cases is still being finalised. However, in Scotland alone provisional figures of 46 cases with 13 fatalities have been reported up to the end of June 2010; this represents a case fatality rate of 28%.

Despite extensive alerts and coverage throughout Europe and the rest of the world, only a handful of similar cases were reported from England and Germany.

Only one case of anthrax in an injecting drug user had been reported previously and that was in Norway in 2000¹.

Information was released via the press advising the drug injecting community of the additional risk associated with taking heroin and that they should seek urgent medical advice if they developed an infection. By mid-January 2010, specific information leaflets and posters were developed in collaboration with the Scottish Drugs Forum to inform and advise drug users.

¹ Ringhertz, S. H., et al. (2000). Injectional anthrax in a heroin skin-popper. *The Lancet*, 356(November 4), 1574 - 1575

Possible reasons for the outbreak

The OCT formulated three working hypotheses. Firstly, that there were anthrax spores in the heroin and these may have entered the supply chain at any point from its original source to the final point of acquisition. Secondly, that either the dissolving agent or cutting agent was contaminated with anthrax spores. Thirdly, that there was an as yet undiscovered link between the cases.

It is estimated that the majority of heroin reaching Europe is produced in Afghanistan². Anthrax is more prevalent in agricultural countries such as Afghanistan, Pakistan and Iran which may lack veterinary health infrastructure, and it is possible that heroin was contaminated with anthrax spores in the country of origin. If this were the case, it may never be known how this particular batch came to be distributed primarily in Scotland and why so few cases were reported from other countries.

There is anecdotal evidence that animal hides are used in the transportation of heroin along the main drug routes. It is possible that anthrax spores were passed from contaminated hides to the drugs during transportation. However, if this were the case, the question remains as to why the outbreak was not more widespread.

Another possibility is that a cutting agent used at some stage in the dealing and distribution process contained anthrax spores which contaminated the heroin. Many of the confirmed cases interviewed reported using a variety of dilution products when preparing their drugs, suggesting that the contaminant was introduced further up the distribution chain. Bonemeal fertilizer was identified as a source of anthrax infection in England in 1974³ and bonemeal may have been mixed with the heroin at some stage as a cutting agent, although no evidence of this has so far been found.

Possible links between the cases have been investigated but no plausible associations which could explain the outbreak, other than heroin use, have been established.

An extensive police investigation, which was driven by the imperative to protect public health and not to criminalise drug users, led to the testing of numerous heroin supplies. To date, no anthrax spores have been detected in any of the samples tested.

² European Monitoring Centre for Drugs and Drug Addiction. (2009). Opioid use and drug injection. www.emcdda.europa.eu/situation/opioids/2

³ Health Protection Agency. Epidemiological Data: Human Anthrax in England and Wales. www.hpa.org.uk/Topics/InfectiousDiseases/InfectionsAZ/Anthrax/EpidemiologicalData/

Feature Article 2: Swine Influenza - A disease of renewed global interest

Ian H Brown, VLA Weybridge

Swine influenza (SI) is a highly contagious acute viral disease of the respiratory tract in pigs which is distributed worldwide. The disease is economically damaging primarily due to reduced food intake which causes weight loss and reduced weight gain, but on occasions infection can be severe, particularly if exacerbated by the presence of other pathogens or factors. On rare occasions, individuals frequently exposed to pigs may contract infection with influenza viruses known to be circulating in the pig population.

The epidemiology of influenza viruses in pigs is unique and can clearly be differentiated from related strains in other hosts including humans. Analysis of all the stable lineages of influenza viruses in pigs has revealed that they originated from an avian or human source and then acquired the capability to infect and transmit between individual pigs, so as to persist within swine populations.

The GB pig population has been closely monitored through Defra funded surveillance activity since the early 1990s. This programme has provided detailed information on virus strains and changing epidemiology of SI within pigs and this provides a good platform from which to study the emergence of new strains. In the UK and Europe, the avian-like swine H1N1 viruses have become the dominant strains since the mid 1990s. These viruses, originally transmitted as 'whole' avian viruses, have received genes from human viruses during reassortment and spread within pigs. In addition in the UK, H1N2 virus has become a widespread strain in pigs following reassortment events with endemic swine strains. Both H1N1 and H1N2 are frequently associated with significant disease problems in pigs. In the UK, they have not been described as causing zoonotic infection and have only rarely been described as such in Europe. However, new UK studies initiated following the emergence of the pandemic 2009 H1N1 (pdmH1N1) strain have revealed that transmission to humans, especially those occupationally exposed, may occur more frequently than was previously thought, although these events do not appear to have been associated with disease in humans.

The pig as a mixing vessel for influenza viruses

For some time, pigs have been considered as one of several potential hosts for the mixing of influenza viruses through genetic reassortment. This would lead to the production of progeny virus that may be capable of infecting and spreading within other host populations, especially humans, and which may ultimately result in the production and emergence of a pandemic strain. There is good evidence in nature that genetic reassortment can occur in pigs, suggesting that avian and human viruses may freely co-infect pigs, and progeny viruses may share genes from both avian and human sources. It has been postulated that the basis for this apparent equivalent susceptibility in pigs to both avian and human viruses is that swine carry receptors for both human and avian viruses in their respiratory tract. However, the

dynamics of infection are complex, and recent work suggests that it is not the receptors alone that put the pig in a unique position in influenza virus ecology.

It should also be noted that at no time has a pandemic strain been identified in pig populations prior to the emergence of the strain in the human population. This equally applies to the pdmH1N1, that whilst ostensibly showing highest similarity to strains in pigs, the precise genotype that transferred to the human population and caused a pandemic has not been definitively detected in pigs prior to its emergence in the human population. Therefore, the pig as a host from which pandemic strains may emerge is as yet unproven.

Pandemic H1N1 (2009) virus and pigs

In March 2009, retrospective analysis revealed that the first cases of human infection with a novel H1N1 virus showing close similarity to swine strains detected in North America was reported in people in Central America. Whether this is where the pandemic truly began or whether the virus was moved to Mexico through infected humans or animals remains uncertain. Nevertheless, detailed studies on the genetic characteristics of the virus revealed that it contained a unique gene constellation not previously described or reported in viruses in human or pig populations. Given the high similarity in all of the gene segments to potential progenitor strains circulating in pigs, the virus was likely to have a high capability to spread from humans to pigs following subsequent global dispersion of the virus through the human population. In April 2009, soon after the declaration of the pandemic, the first cases of infection of pigs with pdmH1N1 were detected in a herd in Canada. This was the forerunner to subsequent cases in South America, Australasia, Europe and Asia with currently in excess of 20 countries worldwide reporting infection in pigs to The World Organisation for Animal Health (OIE) under the new and emerging diseases category. Furthermore, it was rapidly demonstrated that pigs are highly susceptible to infection, can present with typical clinical signs as seen with other strains of influenza and will readily transmit the virus to in-contact susceptible animals. This indicates that the virus could, in the right circumstances, become established within pigs independent of the human population. This hypothesis is supported by natural events that have been reported in several countries, although structured surveillance has been lacking to demonstrate long term persistence of the virus. In the UK to date, 26 pdmH1N1 virus infected swine herds have been confirmed. Detailed epidemiological investigations have revealed that the virus has been transmitted between pig herds and therefore does not rely on contact with infected humans for the source of virus, although this may also occur.

The future dynamics of swine influenza infection is likely to be complex, especially given the immune status and characteristics of viruses circulating in pig populations in different regions of the world. The impact of prior immunity to other H1N1 viruses upon infection with pdmH1N1 is not yet understood but, based on previous data with swine H1 viruses, some protection may be afforded against incursion of pdmH1N1. The long term epidemiology of the virus strains circulating in pigs may be significantly affected by the presence of the pandemic virus. Further reassortment of the pandemic virus with strains known to be infecting pigs has already been reported in Hong Kong and demonstrates that a likely increase in genetic diversity and emergence of further genotypes containing genes from the pandemic strain appear

probable within global pig populations. It is important to note that these emerging influenza viruses have the potential to produce a significant economic impact on the global swine industry. Monitoring of such reassortment events in both pigs and humans is therefore necessary to allow early identification of an increased threat to veterinary or public health.

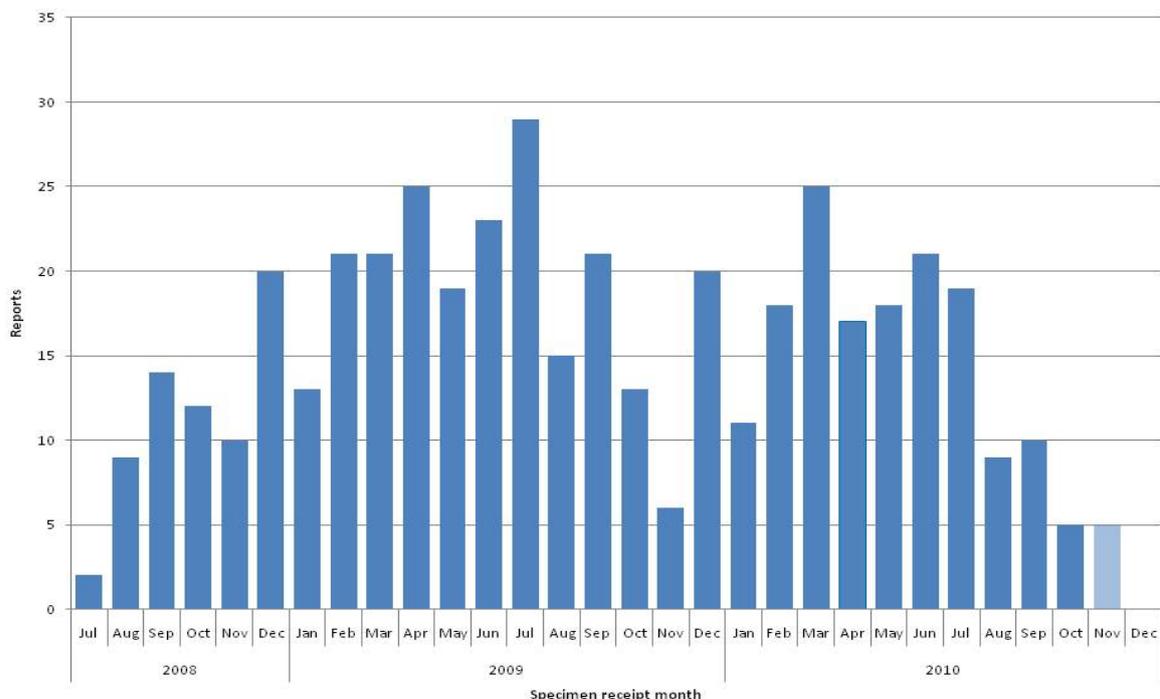
Feature Article 3: *Salmonella* Typhimurium DT 191a infections from frozen feeder mice in the UK, 2008 – 2010

Christopher Lane, HPA

This article is related to Feature Article 4 – Monophasic *Salmonella* Typhimurium-like strains.

In December 2008, the Health Protection Agency Centre for Infections (HPA CfI) detected a gradual but sustained increase in a new phage type of *Salmonella* Typhimurium, designated definitive type 191a (DT 191a). This specific strain is a monophasic *Salmonella* Typhimurium variant (see feature article four) designated with a specific antigenic formula S. 4, 5, 12:i:-. The increase was predominantly in children, and most isolates received at the Laboratory of Gastrointestinal Pathogens, HPA Colindale were resistant only to the antimicrobial tetracycline. A similar increase was also found in Scotland, affecting the same age group. A case-control study in England found an association between contact with or handling pet reptiles and infection. Most cases reported contact with snakes, predominantly colubrids (corn snakes), and results from an analytical study indicated that cases who had exposure to pet reptiles were more likely to have been ill with *S. Typhimurium* DT 191a than those who had no reptile contact (OR 16.82; P = 0.001; 95% CI 2.78- ∞). Most cases who reported contact with snakes also reported that the pets were fed with thawed frozen mice.

Figure 1: Reports of tetracycline resistant *Salmonella* Typhimurium DT 191a to the HPA Centre for Infections, UK, 2008-2010.



An environmental investigation to determine the source of the outbreak followed-up major importers and wholesalers of frozen feeder mice for the pet reptile trade. Frozen mice specimens were collected from six major import and distribution companies in England (representing approximately 80% of market supply lines, and 90% of import lines to the UK). These were analysed at the VLA, Weybridge. Positive isolates were found in products from a single UK importer ('importer A'), who sourced the stock from a single producer in the United States (US). Isolates were phage typed as *S. Typhimurium* DT 191a, and further found to have a Pulse Field Gel Electrophoresis (PFGE) pattern that was indistinguishable from human isolates. Fifty seven percent of samples from importer A were positive, with nine out of 10 product lines tested having at least one positive sample. The importer voluntarily ceased trading the implicated stock and informed his supplier in the US.

A multinational, multiagency Incident Control Team (ICT) involving the HPA, the VLA, the Department of Health, Defra and the Scottish Government was convened in June 2009 to determine how to address the issues relating to both the withheld imported stock and any future imports. The ICT decided to allow the withheld feed to be marketed on the condition it was labelled indicating the potential for infection and the need for hand washing after handling the product. To strengthen controls, new import authorisations were issued, requiring: clear labelling of imported stock "Intended for feeding to reptiles only"; commercial documents to accompany consignments; and the introduction of random testing at Border Inspection Posts (BIPs), with positive results passed to the relevant local authority in England and Wales. This allowed for random testing for such contaminants as *Salmonella*, but would only apply if imported from outside the European Union. The HPA communicated directly with the producer in the US and were told that interventions had been applied at source to reduce further contamination in the product being supplied. Random testing at BIPs began in September 2009, and in November, following three successive positive samples of mice; the implicated importer ceased bringing in products from the contaminated supply in the US. Revised guidance for reptile owners and handlers was also produced and is available on the HPA website at: www.hpa.org.uk/web/HPAwebFile/HPAweb_C/1259152367287.

In January 2010, the Centres for Disease Control and Prevention (CDC) in the US began to detect cases of salmonellosis in children and owners of pet reptiles. The molecular profile of cases in the US was indistinguishable from human and frozen mouse isolates tested in the UK. By July 2010, over 35 cases had been reported from 17 states in the US, prompting the Food and Drug Administration (FDA) to visit the previously implicated supplier. Environmental and mouse specimens were found to be indistinguishable from isolates in humans both in the US and in the UK, and mice tested in the UK. In July 2010, the implicated supplier initiated a voluntary global recall of produce shipped since May 2009, and introduced compulsory irradiation of all produce leaving the facility.

Between August 2008 and November 2010 (Figure 1), 422 cases of tetracycline resistant *S. Typhimurium* DT 191a were reported to HPA CfI with an average of 17 cases per month. After the introduction of controls at the US production plant, the number of cases fell to an average of 7 cases per month, with 5 cases reported in November. The situation will continue to be monitored.

Feature article 4: Monophasic *Salmonella* Typhimurium-like strains

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This article is related to Feature Article 3 – *Salmonella* Typhimurium DT 191a infections from frozen feeder mice in the UK, 2008 – 2010.

Introduction

Salmonellosis is one of the most common foodborne bacterial diseases of humans worldwide, usually characterised by acute gastroenteritis. Severity of the disease varies depending on the age and immune status of the host, the number of bacteria ingested and the pathogenicity (ability to cause disease) of the implicated *Salmonella* serovar.

Of the approximately 2,600 designated types (serovars) of *Salmonella*, certain types are considered of greater public health significance, and the most important of these are *Salmonella* Enteritidis and *Salmonella* Typhimurium. These two serovars account for the majority of human salmonellosis cases both in the UK and in the EU as a whole.

Recently, there have been an increased number of reports of variants of *Salmonella* Typhimurium, known as “monophasic” *Salmonella* Typhimurium. These strains, which cannot be fully typed by conventional serotyping, have shown the ability to spread rapidly in food animals and humans in many countries. The rapid emergence of one particular monophasic *Salmonella* strain (‘DT193’) in pigs and cattle, and occasionally in other species, has led to fears that this strain may become a successor to the previous epidemic strain, *S.* Typhimurium DT104, if immediate action is not taken. Following discussions with Member States, the European Commission (EC) requested a scientific opinion from the European Food Safety Authority (EFSA) on how authorities should identify and control monophasic *S.* Typhimurium-like strains. The opinion is available at the following link: www.efsa.europa.eu/en/scdocs/scdoc/1826.htm

The Organism

Salmonella bacteria can be characterised into different groups by laboratory testing, such as serotyping and phagetyping. Serotyping of *Salmonella* is based on detecting variations in structural components of the bacteria’s cell wall (antigens). Most *Salmonella* strains have specific physical characteristics, such as the ability to be mobile, related to the presence of the flagella (‘tails’ with which the organism propels itself towards nutrients). The majority of *Salmonella* serovars (notable exceptions are *S.* Enteritidis, *S.* Dublin, *S.* Gallinarum/Pullorum) have two different sets of flagella, known as “phase one” and “phase two”. The presence of specific antigens is denoted by letters and numbers. For example *Salmonella* Typhimurium has the antigenic formula 1,4,[5],12:i:1,2. This serovar has two flagellar phases (‘biphasic’); the first phase flagellar antigen being denoted by ‘i’ and the second phase by ‘1,2’.

Most monophasic *S.* Typhimurium-like strains express only the first phase flagellar antigen, as genes governing expression of the second phase (H1,2) antigens have

either been deleted or partially deleted as a result of natural genetic mutation. The most common type of monophasic *S. Typhimurium* is S.1,4,5,12:i:-. There is only one other serovar that has this combination of antigens and that is *Salmonella* Lagos (S.1,4,5,12:i:1,5), which is extremely rare in the UK, but does occur in poultry in the Netherlands and could therefore be imported with hatching eggs, day-old chicks or poultry meat. Monophasic *S. Typhimurium* strains lacking the O5 antigen (i.e. S.1,4,12:i:-) also occur, but less commonly. All of these stable clones of monophasic strains are quite different from the occasional *S. Typhimurium* strain that does not fully express flagellar antigens. In the latter, the relevant genes are still present but there is a deficiency in production of the flagellar proteins.

Salmonella variants lacking all flagellar antigens (S.1,4,5,12:-:- or S.1,4,12:-:-), known as “aphasic” variants, have also been described, particularly in France. These strains may be non-motile and are therefore less easy to detect in Modified Semi-solid Rappaport-Vassiliadis medium (the EU designated enrichment method for culturing *Salmonella*). Currently such strains are very rarely identified in the UK but will be kept under review.

Monophasic *S. Typhimurium* strains can be confirmed as variants of *S. Typhimurium* by various means. Most isolates will produce a recognised *S. Typhimurium* phage type with the usual panels of typing phages. The currently emerging monophasic strains in the UK are predominantly DT193, with DT120 also occurring to a lesser extent. These two phage types are very closely related genetically amongst current monophasic *S. Typhimurium* strains. A further method for differentiating strains is via antimicrobial resistance profiling – DT193 and DT120 are commonly resistant to four antibiotics (tetra-resistant), while the DT191a strain (see feature article 3) is only resistant to tetracycline (as has also been reported from monophasic *S. Typhimurium* strains from chickens and humans in the US).

Definitive confirmation as variants of *S. Typhimurium* can be made by various Deoxyribonucleic acid (DNA) analysis methods such as Polymerase Chain Reaction (PCR), micro-array or ‘genetic fingerprinting’ PFGE. These identify common genetic factors in *S. Typhimurium* and monophasic strains. There is a specific predominant genetic fingerprint type associated with currently circulating monophasic *S. Typhimurium* DT193 strains.

Importance of these strains

A marked increase in these strains during the last 20 years has been reported from many countries, particularly Spain (U302 variant), US, South America, Germany, France, Luxembourg and Italy, as well as from several countries in the Far East. In most cases there has been a concurrent rise in such strains in pigs, but in the US poultry are also involved. In the UK there are a significant number of isolations from pigs and cattle, as well as occasional cases in sheep, horses, dogs and zoo animals. It is likely that pigs are the main species involved in evolution of these strains, as they have been with various other *S. Typhimurium* phage types, e.g. U288.

There is also an unrelated monophasic strain (known as DT191a) which has been prominent in humans in UK and US since 2008/9 and has been associated with

frozen feeder mice fed to exotic reptilian pets and zoo animals (see Feature Article 3).

The rapid emergence of various monophasic *S. Typhimurium* strains to become one of the most common serovars in pigs and humans in multiple countries shows that they have the potential to spread rapidly. This rapid emergence is reflected in the close genetic relationship of strains of related phage types, e.g. the most recent DT193 strain. The mechanism for this rapid spread is unclear, but trade in pigs and pig meat is a possibility. It is also possible that such strains have the ability to be shed in large numbers in faeces compared with other strains, thus promoting extensive environmental contamination and onward spread (via contamination of food animal or pet feed ingredients or human food products), as occurred with *S. Typhimurium* DT104.

In order to reduce the number of human cases of salmonellosis, legislation was implemented in 2003 to harmonise the monitoring and control of *Salmonella* within the EU (Directive 2003/99/EC and Regulation (EC) No. 2160/2003). This has led to the implementation of National Control Programmes (NCPs) in all European Union (EU) Member States which aim at reducing the *Salmonella* burden at the farm level towards an agreed target. Currently, targets are in place for chicken breeding flocks for *S. Enteritidis*, *S. Typhimurium*, *S. Hadar*, *S. Virchow* and *S. Infantis*; and for laying flocks, broiler flocks and turkey flocks for *S. Enteritidis* and *S. Typhimurium* only. Official controls are put in place if any of the above-mentioned serovars are found, depending on the type of flock and the serovar. These range from enhanced abattoir hygiene measures and mandatory heat treatment of eggs to culling of breeding flocks, with the overall aim of controlling the spread of *Salmonella* and limiting the risk to public health.

Serovars other than the named target serovars are currently not included in the NCPs, as they were not considered to be of major zoonotic importance at the time the legislation was put in place. This means that although the monophasic *Salmonella* Typhimurium strains are now recognised variants of *Salmonella* Typhimurium, and have shown the ability to spread rapidly in food animals and humans in many countries, until now they could be considered to be legally outside the statutory controls currently in place. This has particular implications in breeding flocks, where infection with these monophasic strains could potentially result in widespread dissemination of infection.

The EFSA Opinion mentioned above deals with the classification, pathogenicity and legal basis for control of monophasic *S. Typhimurium*. Since EFSA consider the public health risk posed by these emerging monophasic *S. Typhimurium* strains with the formula 1,4,[5],12:i:- to be comparable to that of other epidemic *S. Typhimurium* strains, the Opinion recommends increased formal regulatory control of monophasic *S. Typhimurium* in food animal production in the EU. These strains will therefore be included within the statutory requirements of the *Salmonella* NCPs in poultry and pigs. Therefore, the relevant official controls will now be applied to prevent further spread and control the risk to public health should a monophasic *S. Typhimurium* 1,4,[5],12:i:- strain be detected in chicken or turkey flocks (and in pigs herds, as these become subject to EC regulatory control in the coming years).

Zoonoses A-Z

Anthrax (Bacillus anthracis)

Anthrax is caused by the bacterium *Bacillus anthracis* (*B. anthracis*). Under certain environmental conditions *B. anthracis* can convert into a spore, which may survive in the environment for many decades in an inert state. In this form the organism shows great resistance to the effects of heat, drying, UV light and many disinfectants.

Recent human cases of anthrax in the UK have been reportedly associated with drum making using imported animal hides, or with contaminated heroin.

Anthrax can occur in all mammalian species, and has also been reported in some birds. The clinical presentation varies between species with three forms of anthrax recognized in animals; peracute/apoplectic, acute and chronic. Anthrax infection in humans classically causes one of three types of disease which affect either the lungs (inhalation/ pulmonary), the digestive tract (intestinal) or the skin (cutaneous). In 95% of naturally-acquired cases, the infection is cutaneous.

Anthrax (animal and human) has a worldwide distribution and cases are much more common in tropical countries, with disease being endemic in many areas. In recent years sporadic anthrax cases have occurred in cattle in the UK, presumably from exposure to anthrax spores present in soil and originating from cases that occurred decades earlier.

Anthrax is a notifiable disease in both animals and humans. Any suspicion of an anthrax infection in animals should be reported immediately to Animal Health or to the local Divisional Veterinary Office in Northern Ireland.

Infection in humans

Symptoms: In cutaneous anthrax a lesion is commonly seen on the hands, forearms, head or neck. A raised, itchy, inflamed pimple appears one to seven days after exposure, followed by a papule that turns into a blister. Extensive oedema or swelling accompanies the lesion, which is usually painless. The blister then ulcerates and two to six days later the classical black eschar develops. If left untreated the infection can spread to cause blood poisoning. Cutaneous anthrax can be treated with antibiotics and will rarely lead to death.

Inhalation anthrax symptoms begin with a flu-like illness followed by severe respiratory difficulties and shock two to six days later. Untreated disease is usually fatal, and treatment must be given as soon as possible to reduce mortality.

Intestinal anthrax is a severe disease which can be fatal. It is found in parts of the world where the nutritional value of an animal that has died unexpectedly outweighs any fears of contracting the disease.

'Injection anthrax' has recently been described in heroin users (see Feature article 1: Outbreak of Anthrax in Scotland in 2009). These have been severe soft-tissue

infections at injection sites, often with necrotising fasciitis, cellulitis or abscesses. Many cases have been fatal.

Transmission: The most common route of transmission is direct contact with tissues or hides from infected animals. In the UK this mostly occurs among people working with contaminated animal products (e.g. hides from abroad). Direct contact generally produces the cutaneous form of the disease.

Infection may also arise following exposure to aerosols of anthrax spores, usually in industrial processes such as the tanning of animal skins, and processing of wool or bones from abroad. Recent inhalation cases in the UK and elsewhere have been related to drum making using imported animal hides. Intestinal anthrax follows ingestion of meat from an animal which has died of anthrax.

Cases of cutaneous and inhalation anthrax occurred in the US in 2001 following distribution of mail which had been deliberately contaminated with spores⁴.

It is extremely rare for anthrax to be transmitted from one person to another.

Cases: Between 2001 and 2008, four human cases were confirmed; two cutaneous cases and two cases associated with drum making.

In December 2009, an outbreak of anthrax amongst heroin users started in Scotland, believed to be due to the circulation of a batch of heroin contaminated with anthrax spores. In 2009, there were 11 confirmed cases in drug users with five deaths, all in Scotland.

Infection in animals

Clinical signs: Susceptibility to infection differs between animal species with herbivores showing least resistance, pigs and horses intermediate resistance and carnivores the greatest resistance. In the case of cattle, an animal previously in good health may be found dead a few hours later. Those seen alive may show signs of fever, dullness, depression, tremor, loss of milk production, congestion of mucous membranes and collapse followed by death. Chronic infection (presenting as either pharyngeal or intestinal forms) occurs in species such as pigs, cats and dogs, which show greater resistance.

Frequency: Anthrax has been reported in all continents except Antarctica. The application of effective surveillance and control measures has a considerable effect on the occurrence of disease. However, sporadic cases in the UK occur as a result of animals becoming exposed to the spores of *B. anthracis* present in the soil at certain sites, usually following some form of soil disturbance. The last case of anthrax in animals in GB was in cattle in 2006, affecting six cows (two confirmed) on one farm in Wales. In Northern Ireland the last case of anthrax was in 1990, affecting one cow on a farm in County Antrim.

⁴ Centre for Disease Control and Prevention (2001) Update: Investigation of Anthrax Associated with Intentional Exposure and Interim Public Health Guidelines, October 2001. *Morbidity and Mortality Weekly Update*. 50(41):889-893. www.cdc.gov/mmwr/preview/mmwrhtml/mm5041a1.htm

There were no cases of anthrax detected in animals in the UK in 2009.

Control measures

A number of control methods are in place in the UK to limit the risk of anthrax infection. Import controls on animal feedstuffs require that material does not carry *B. anthracis* spores. All suspected cases in animals must be notified to Animal Health; automatic movement restrictions are applied. Should a case be confirmed, appropriate carcass disposal and disinfection of any sites that may have been contaminated is required. The controls available are set out in the Anthrax Order 1991.

Annual costs of anthrax government veterinary expenditure are low. They include the cost of primary testing carried out on cases of sudden animal deaths where disease is suspected, maintaining laboratory facilities for confirmatory testing and the costs of maintaining capability in Animal Health to deal with cases.

Summary of risk

Animal control measures are considered to be highly effective in the UK. The incidence could increase if *B. anthracis* were to be imported in animal feedstuffs, but this is unlikely as animal feedstuffs are now strictly controlled. The incidence of anthrax in animals is likely to remain rare. If an anthrax case occurred, it is most likely to be a small outbreak with minimal spread.

Further information

Further information on anthrax infection in humans is available from HPA:
www.hpa.org.uk/Topics/InfectiousDiseases/InfectionsAZ/Anthrax/

Further information on anthrax infection in animals is available from Defra at:
www.defra.gov.uk/foodfarm/farmanimal/diseases/atoz/anthrax/index.htm

Bovine tuberculosis (*Mycobacterium bovis*)

Bovine tuberculosis (bTB) is caused by the bacterium *Mycobacterium bovis* (*M. bovis*) and should not be confused with *Mycobacterium tuberculosis* (*M. tuberculosis*), the main cause of tuberculosis (TB) in humans.

The *M. tuberculosis* complex includes *M. tuberculosis*, *M. bovis* and *M. africanum*. Human tuberculosis is usually caused by *M. tuberculosis* (generally acquired by person-to-person transmission). Rarely, *M. africanum* or *M. bovis* can infect humans and give rise to clinical TB which is indistinguishable from the disease caused by *M. tuberculosis*.

M. bovis is an obligate intracellular pathogen, primarily causing respiratory disease. Infection most often occurs when airborne droplets of moisture (aerosols) containing the organism are inhaled, but can also occur by eating or drinking contaminated material and, more rarely, through contamination of skin wounds.

M. bovis is an important cause of disease in cattle and has also been found in other farmed livestock such as sheep, pigs and goats, wildlife such as foxes, rats and deer, camelids (e.g. alpacas and llamas), domestic cats, dogs and humans. Some species appear more susceptible than others. In large areas of the UK, there is a significant reservoir of disease in badgers (*Meles meles*), and the infection can spread in either direction between cattle and badgers.

Tuberculosis used to be widespread in cattle in the UK until a compulsory eradication campaign began in 1950. This programme, underpinned by routine screening of herds using the comparative tuberculin skin test and slaughter of test reactors, gradually reduced infection to a very low level by the early 1980s. However, since then, the number and geographical distribution of new cases in cattle herds ('breakdowns'⁵) have steadily increased. This trend accelerated after the foot and mouth disease outbreak in 2001, during which the routine TB testing and slaughter programme was suspended for several months. Disease in GB is predominantly found in the South West of England, the West Midlands and South and Mid-Wales, where it is considered endemic. Other parts of the country are largely free from disease, with only sporadic incidents often attributed to movements of infected cattle from the endemic areas. Scotland was declared an officially TB free region of the UK by the European Commission in 2009 (Decision 2009/761/EC) and as such implements strict controls regarding the movement of cattle from the rest of the UK. Disease in Northern Ireland is widely distributed geographically and whilst there are areas of lower incidence, no area is considered to be disease free.

M. bovis is notifiable in humans and animals across the UK.

⁵ TB incidents are also known as 'breakdowns', i.e. herds in which at least one animal was a reactor to the tuberculin skin test or culture-positive tuberculous lesions were detected during commercial slaughter of a non-reactor animal.

Infection in humans

Symptoms: The symptoms associated with infection with *M. bovis* depend on the site of infection. For infections with *M. tuberculosis* and *M. bovis*, fatigue, fever, night sweats and weight loss may occur initially, while localised symptoms of cough, chest pain, haemoptysis (coughing up blood) and hoarseness become prominent in the advanced stages. In addition, infection with *M. bovis* in humans can cause swellings of the lymph nodes of the neck. Treatment with a long course of three or four different anti-tuberculosis drugs usually results in a cure. Not everyone infected with the bacterium develops disease. The bacteria may remain inactive in the body for many years and, in a small percentage of these latently infected individuals, reactivation occurs later in life.

As with human infection with *M. tuberculosis*, susceptibility to *M. bovis* infection is increased by human immunodeficiency virus (HIV) infection and other forms of immune-suppression and debilitating diseases such as chronic renal failure, cancer and diabetes. Once disease develops, about half of patients will die within five years if untreated, the majority within the first 18 months.

Transmission: Transmission of *M. bovis* can occur between animals, from animals to humans and, more rarely, from humans to animals and between humans. The consumption of unpasteurised milk or dairy products from infected cows was an important cause of childhood tuberculosis in the UK until pasteurisation became widespread in the mid-20th century. Occasional cases may still occur in the UK through this route of infection. Infection through consumption of contaminated meat is thought to be very rare or non-existent in countries like the UK, where all cattle carcasses undergo veterinary meat inspection. Inhalation of the organism is also an established, but low risk, route of transmission for people working with infected animals. Other potential but less likely sources of infection for people are infected deer, badgers and companion animals. Rare cases of cutaneous human *M. bovis* infection have also been documented in people handling infected animals or their carcasses.

Person-to-person transmission of *M. bovis* is usually the result of prolonged close contact with an infectious case. Exposure to airborne aerosols containing the bacteria created by coughing and sneezing is one route of infection.

Cases: *M. bovis* accounts for approximately 0.5% of all culture-confirmed *M. tuberculosis* complex diagnoses in humans in England, Wales and Northern Ireland annually.

In 2009, there were 17 culture-confirmed cases of human TB caused by *M. bovis* in England and Wales, seven cases in Scotland and one case in Northern Ireland.

Table 1: Patients with culture-confirmed tuberculosis in the UK, 2009

	England & Wales	Scotland	NI	UK Total
<i>M. bovis</i>	17	7	1	25
<i>M. tuberculosis</i>	4,660	310	48	5,018
<i>M. africanum</i>	32	4	0	36
Total	4,709	321	49	5,079

Sources: Enhanced Tuberculosis Surveillance, Enhanced Surveillance of Mycobacterial Infections (Scotland), UK Mycobacterial Surveillance (laboratory data).

Note: numbers are based on live data and may be subject to change (e.g. de-notifications and late reports).

The distribution of human cases of *M. bovis* in the UK has remained similar over the last 15 years. There were 25 cases of *M. bovis* diagnosed in 2009. A country of birth was given for 18 cases, of which 83% were of white ethnicity and UK-born (15/18). Thirteen of these UK-born cases were over 50 years old (13/15) and likely to be due to reactivation. The three non-UK-born cases were from Morocco, Nigeria and the Russian Federation and all had been in the UK for more than five years at diagnosis. This suggests that a majority of the cases seen in the UK are attributable to reactivation of latent infection, probably acquired prior to widespread implementation of disease controls.

Infection in animals

Clinical signs: The disease in animals is chronic, and characteristic signs may not be evident in cattle even when many organs are affected. Signs are non-specific for respiratory disease and include coughing, difficulty in breathing, and acute respiratory distress as well as enlarged lymph nodes of the head and neck, intermittent diarrhoea, extreme emaciation and weakness. In addition, a drop in milk production may be observed in high-yielding dairy cows. In countries where the disease in cattle is of limited duration because of a test and slaughter policy (as in the endemically infected areas of the British Isles), animals are rarely seen in an advanced stage of the disease.

Frequency: *M. bovis* is currently endemic in cattle and badgers in most of Ireland and large tracts of the West of England and South and Mid-Wales, although Scotland is TB-free.

There were 84,515 cattle herds registered in GB at the end of 2009. A total of 4,574 new bTB incidents were recorded in GB in 2009, an 8.3% decrease on the number of new bTB incidents recorded in 2008. Ninety-eight percent of these incidents occurred in England and Wales. Post-mortem evidence of lesions characteristic of bTB and/or culture evidence of *M. bovis* infection was detected in 2,468 (46%) of these new bTB incidents. In Scotland, the number of new total (n=49) and post-mortem and culture-confirmed (n=10) bTB incidents was similar to 2008.

In GB, a total of 34,765 cattle were slaughtered as tuberculin skin or interferon-gamma (blood) test reactors in 2009, a drop of 6.2% on 2008.

Sporadic cases of *M. bovis* infection were reported in four deer species, farmed alpacas, cats, dogs, domestic pigs and sheep, virtually all of them located in areas of

high endemic bTB incidence. The prevalence of infection in badgers is highly variable in GB, but can be as high as 50%.

In Northern Ireland there were 26,287 cattle herds with 1.6 million cattle registered during 2009. There were 1,293 new reactor herds, both confirmed and unconfirmed, and 8,198 reactor animals, and at the end of the year 1,053 herds (4%) were still under bTB restriction.

Control measures

BTB surveillance and control policy is a devolved matter; Defra, WAG, Scottish Government and DARD have responsibility for defining the control programmes in their own jurisdictions. Implementation of control policies is by DARD in Northern Ireland and by AH in GB.

Ongoing routine national screening by the comparative tuberculin skin test using bovine and avian tuberculins means that cattle with suspected bTB are usually identified before they become clinically affected. In England, cattle herds are tested every one to four years, the interval depending upon the local history of incidence of infected herds. All herds in Wales have been tested annually since October 2008. Scottish herds are bTB tested every four years. In Northern Ireland all cattle herds are skin tested annually as a minimum. The tuberculin skin test is supplemented with the interferon-gamma blood assay to enhance the sensitivity of the bTB testing regime in some bTB breakdowns with post-mortem evidence of *M. bovis* infection. Movement restrictions are placed on herds where infection is suspected, and all reactor cattle and other cattle likely to have been infected by contact are slaughtered.

All cattle compulsorily slaughtered in abattoirs for bTB control purposes (i.e. test reactors and contacts) undergo ante- and post-mortem inspection to establish the extent of any visible lesions. Tissue samples are taken for culture and genotyping (where appropriate) and to assess their suitability for human consumption by meat inspectors of the FSA and their veterinary contractors. Under EU regulations, where pathological lesions typical of bTB are found in more than one organ or anatomical region of the carcass, the whole carcass is condemned and declared unfit for human consumption. Those carcasses with lesions of bTB in a single organ (or part of the carcass) and associated lymph nodes can be considered fit for human consumption once the affected part has been removed. If no bTB lesions are detected, the whole carcass is fit for human consumption (with regard to bTB).

Defra funds a wide programme of research into bTB, currently focusing on the development of cattle and badger vaccines and supporting diagnostic tests that could help to reliably differentiate vaccinated from infected cattle. Government expenditure on bTB control (including testing, compensation and research) in GB during the 2008/09 financial year was £108.4 million. In Northern Ireland, government expenditure during 2009 was approximately £23 million.

Summary of risk

The number of cattle herds under restriction as a result of *M. bovis* tuberculosis in cattle has increased significantly since the mid-1980s in the South of England and in South and Mid-Wales, and there is considerable evidence for infection in badgers in areas where TB is endemic in cattle. However, large parts of GB remain free of disease and Scotland has gained "Officially Tuberculosis Free" status. Northern Ireland experienced a significant increase in prevalence and incidence of bTB between 1995 and 2002, but the prevalence and incidence then fell, and they now remain relatively constant.

The overall public health risks posed by cattle infected with *M. bovis* are currently considered very low because of the implementation of meat inspection procedures, wide scale milk pasteurisation and the cattle screening programmes, which are effective controls. There are potential additional risks arising from *M. bovis* infections in domestic animals other than cattle, including pets and wild mammals.

Further information

Further information about tuberculosis in humans is available from HPA at:
www.hpa.org.uk/Topics/InfectiousDiseases/InfectionsAZ/Tuberculosis/

Further information about tuberculosis in animals is available from Defra at:
www.defra.gov.uk/food-farm/animals/diseases/tb/

More detailed statistics on bTB in GB are published monthly in Defra's bTB website at:
www.defra.gov.uk/foodfarm/farmanimal/diseases/atoz/tb/stats/index.htm

For historical annual bTB incidence and charts (1998-2008):
www.defra.gov.uk/foodfarm/farmanimal/diseases/atoz/tb/stats/county.htm

For annual policy updates, see the Annual CVO Reports at:
www.defra.gov.uk/corporate/about/who/cvo/report.htm

For additional information for Wales go to:
<http://wales.gov.uk/topics/environmentcountryside/ahw/disease/bovinetuberculosis/?lang=en>

For Scotland:
www.scotland.gov.uk/Topics/farmingrural/Agriculture/animal-welfare/Diseases/disease/tuberculosis

For Northern Ireland go to:
www.dardni.gov.uk/index/animal-health/animal-diseases/tb.htm.

More detailed statistics on bTB for Northern Ireland can be found at:
www.dardni.gov.uk/index/dard-statistics/animal-disease-statistics.htm

Brucellosis (*Brucella* spp.)

The livestock (cattle) population of GB has been Officially Brucellosis Free (OBF) since 1985; Northern Ireland has not yet achieved this status. As a result, brucellosis in humans is generally acquired abroad (usually *B. melitensis*) although cases of *B. abortus* are rarely acquired in Northern Ireland where infection remains in cattle. Infections with *B. ovis*, *B. melitensis*, *B. suis* and *B. microti* have never been detected in the animal population in the UK.

Two new brucella species have been isolated in recent years from a number of marine mammals washed up on the coast around the UK. These were identified as *Brucella ceti* (with cetaceans as their preferred host) and *Brucella pinipedalis* (with pinipeds as the preferred host). Recently, a further novel species of brucellosis, named *B. inopinata* was isolated from a breast implant in Scotland. The source and natural reservoir of this isolate remains unclear. This is the 10th species of *Brucella* to be recognised⁶.

Infection with *Brucella* spp. is notifiable and reportable in animals.

Infection in humans

Symptoms: Cases of human brucellosis are associated with symptoms such as intermittent fever, arthralgia, profuse sweating and headache. Onset can be acute or insidious. Most cases have a self-limiting disease, but some develop long-lasting or chronic disease.

Transmission: Most reports are due to *B. melitensis* infection, acquired overseas, mainly in Middle Eastern and Mediterranean countries where the consumption of unpasteurised milk and dairy products is common. Other brucella exposure routes are usually occupational, occurring in veterinarians, farm and laboratory workers and those in the meat industry in endemic areas.

Cases: Between 2000 and 2008 an average of 22 cases of brucellosis were identified in humans each year. This level of infection has remained relatively stable, with a slight decline in recent years.

In 2009, there were a total of 17 cases of brucellosis in humans identified in the UK (Table 2), eight of whom had infection with *B. melitensis* (all in England and Wales). Four cases were identified as having had a previous infection, one of whom had been previously treated in Iran. Whilst the sources or countries of infection were not generally reported, it is believed that all the infections identified during the year had been acquired overseas. There was one case of brucellosis in Scotland and four cases of *B. abortus* identified in Northern Ireland, three of which were associated with farming.

⁶ Scholz, H.C., et al. (2010) *Brucella inopinata* sp. nov., isolated from a breast implant infection. *Int J Syst Evol Microbiol.* 2010 Apr; **60**(Pt 4): 801-8.

Table 2: Reports of brucellosis in humans in the UK, 2009

	England & Wales	Scotland	NI	UK Total
<i>B. abortus</i>	0	0	4	4
<i>B. melitensis</i>	8	0	0	8
Other brucella spp.	4	1	0	5
Total	12	1	4	17

Infection in animals

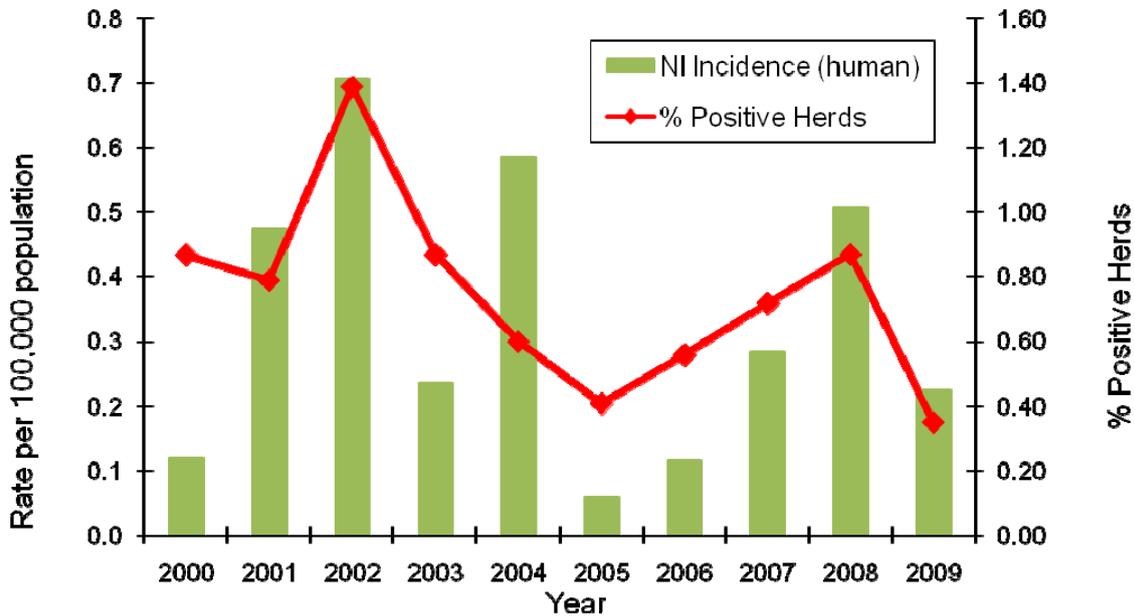
Clinical signs: *B. abortus* infection in cattle is characterised clinically by abortions in the last third of the gestation period, and endometritis (retained afterbirth). It is a disease which can spread rapidly through the herd and cause serious economic losses through abortions, reduced milk yield and reduced fertility.

Frequency: Bovine brucellosis was largely eradicated from Northern Ireland during the 1980s and only sporadic outbreaks occurred during 1990 to 1996. In 1997, three primary outbreaks resulted in secondary and tertiary spread to more than 60 farms. The presence of *B. abortus* in cattle in Northern Ireland continues to constitute a risk to public health but its prevalence has fallen since the peak of infection in 2002, which coincided with the peak of reported human cases of *B. abortus*. The incidence of brucellosis in humans closely corresponds to trends in the percentage of positive herds in Northern Ireland during the period 2000-2009 (see Figure 2) and has shown an overall decrease since 2000.

Typically, cattle herds in Northern Ireland are small in size, fragmented, and subject to a high rate of between-herd and within-herd movement⁷. One recent epidemiological report indicated that contiguous spread (spread between herds within the same locality) was responsible for approximately 44% of outbreaks, while post-abortion outbreaks accounted for approximately 18%. To address the former, pre-movement testing, other than to abattoirs, is compulsory, and at risk herds (e.g. neighbour infected cattle) are subjected to repeated, short-interval testing. DARD has also undertaken a number of initiatives to encourage reporting of abortions.

⁷ Abernethy D.A., et al. (2006). The NI programme for the control and eradication of *Mycobacterium bovis*. *Veterinary Microbiology*, **112**(2-4):231-237.

Figure 2: Incidence of *Brucella abortus* in humans and percentage of positive herds in NI, 2000-2009



In Northern Ireland in 2009, 20,181 herds with eligible cattle were tested for *B. abortus*. Seventy-six herds (0.38%) were positive, a decline from 0.94% in 2008. Of these, 71 new herds were positive during the period. One hundred and sixteen of the 888,898 animals that were tested were positive (0.013%), 13 confirmed by bacteriological culture. This is a decrease from the 0.042% positive in 2008.

GB has remained an OBF region since 1991 and a programme of surveillance is still carried out to ensure that this status is not compromised, in order to underpin trade. Cattle surveillance includes monthly testing of bulk milk samples from all dairy herds, risk-based investigations of cattle abortions, and premature calvings and post-import testing of breeding cattle.

An annual survey is also conducted in the UK to demonstrate the absence of *B. melitensis* in sheep and goats as a requirement of EU regulations. Evidence of absence of *B. melitensis* is supported by submissions of abortion samples from sheep and goats.

No cases of brucellosis were detected in animals in GB during 2009. Tests were carried out on 150,941 bulk milk samples, 6,485 cattle abortions and premature calvings, and 4,577 and 2,104 imported breeding cows, 60-days after importation and post-calving respectively.

The sheep and goat survey found no evidence of *B. melitensis*.

Control measures

Control measures focus on maintaining the absence of disease in GB. This includes the surveillance programme as described above, and the removal of suspected positive cattle where identified.

In Northern Ireland, a programme is underway to eradicate brucella from cattle through the notification and removal of suspected cases. Advice is provided to cattle farmers regarding the safe handling of abortion and post-calving tissues.

Pasteurisation of milk and milk products prevent transmission to people through a foodborne route.

Summary of risk

For people in GB, the principal risk of infection is from the consumption of unpasteurised milk or milk products from infected animals when abroad in endemically infected countries. In Northern Ireland, additional risk comes from direct exposure to aborting and naturally calving cattle, and their birth products. The low level of risk in Northern Ireland has remained relatively constant over the last few years, but recent enhancements to surveillance and control actions are designed to eradicate the disease so as to reduce the level of risk to that of the rest of GB.

Further information

Further information about brucellosis in humans is available from HPA at:

www.hpa.org.uk/Topics/InfectiousDiseases/InfectionsAZ/Brucellosis/GeneralInformation/bruc001GeneralInformation/

Further information about brucellosis in animals is available from Defra at:

www.defra.gov.uk/foodfarm/farmanimal/diseases/atoz/brucellosis/

Further information about brucellosis in animals in NI is available at:

www.dardni.gov.uk/index/animal-health/animal-diseases/br.htm

Campylobacteriosis (*Campylobacter* spp.)

Campylobacter spp. are the most commonly reported bacterial pathogen in humans in the UK.

The species of greatest public health importance are *C. jejuni* and *C. coli*. The Study of Infectious Intestinal Disease in England demonstrated that approximately 90% of campylobacteriosis is caused by *C. jejuni*⁸. However, most laboratories do not routinely speciate strains isolated from clinical specimens.

C. jejuni and *C. coli* (thermophilic campylobacters) can be found in a wide range of livestock and wildlife species. *C. fetus* is a common cause of abortion in sheep and may occasionally cause serious systemic disease in humans. Other species, such as *C. sputorum*, *C. hyointestinalis* and *C. lari*, are present in mammals and birds in the UK but are not generally considered of public health importance.

The role of *C. jejuni* in human enteric illness was first clearly demonstrated in 1972. By 1986, campylobacter had replaced non-typhoidal salmonella as the most commonly reported gastrointestinal pathogen in the UK.

Infection with *Campylobacter* spp. is not notifiable or reportable in animals.

Infection in humans

Symptoms: The principal symptoms of campylobacteriosis are diarrhoea and abdominal pains. Cases may also have nausea, vomiting, headaches, fever, malaise and joint pains. Symptoms usually last for 5-7 days but can persist for weeks. In most cases the disease is self-limiting. Approximately 4% of all cases are admitted to hospital.

The severity of symptoms is dependent upon the age of patients. The very young and the elderly are most at risk of developing severe disease. In extremely rare cases, complications such as bacteraemia, joint disease or more severe neurological conditions such as Guillain-Barré syndrome may arise, although these are poorly ascertained through laboratory-based surveillance. A long term association with Inflammatory Bowel Syndrome has also been reported.

Transmission: Transmission to humans is through the "faecal-oral" route, usually by the consumption of contaminated foods or water. Infection from meat can be prevented by thorough cooking to kill the organism. However, cross-contamination of uncooked foods during processing or food preparation by contaminated juices is a well-recognised route of transmission⁹.

⁸ A report of the Study of infectious intestinal disease in England. 2000. FSA.

⁹ Food Standards Agency, (2008). *Report on UK publicly-funded research relating to Campylobacter*. www.food.gov.uk/science/research/foodborneillness/microfundders/msffg/campylobacterupdate/

Transmission can also sometimes occur through contact with pets with diarrhoea. Person-to-person spread tends to only occur when incontinent patients or infants are involved, and outbreaks have been reported in nurseries.

Cases: The incidence of infection has been recorded since 1982. The number of laboratory confirmed cases in humans in the UK peaked at 65,720 cases in 2000. Numbers then declined and, from 2002, remained relatively stable across GB until 2006, after which the numbers increased again. In Northern Ireland, annual numbers have had an upward trend since 2004.

In 2009, 65,000 cases were reported across the UK, a 17% increase from the 55,609 cases reported in 2008 (Table 3). In England and Wales, 57,608 cases were reported in 2009, an increase of 15% from 2008. In Scotland, 6,415 cases were reported in 2009, an increase of 32% from 2008. Nine hundred and seventy-seven cases were reported in Northern Ireland in 2009, representing an increase of 15%.

Table 3: Number of campylobacter reports in humans, 2000-2009

Year	England & Wales	Scotland	Northern Ireland	UK
2000	58,236	6,482	1002	65,720
2001	55,081	5,435	888	61,404
2002	48,133	5,121	821	54,075
2003	46,285	4,445	743	51,473
2004	44,544	4,365	841	49,750
2005	46,724	4,581	891	52,196
2006	46,868	4,857	937	52,662
2007	51,975	5,194	885	58,054
2008	49,883	4,878	848	55,609
2009*	57,608	6,415	977	65,000

*These figures are provisional and may be subject to change due to delayed reporting.

Within the UK, epidemiological studies have indicated that the ratio of unreported human infection in the community to reports to national surveillance for campylobacteriosis is approximately 8 to 1¹⁰. This suggests that, in 2009, there could have been as many as 585,000 *Campylobacter* cases in the UK.

The rates of reported infection are higher in infants than adults, but there has been a significant increase in the incidence rate in older age groups since 1990.

Campylobacter in food

Campylobacter spp. are present in a significant proportion of animals and poultry entering slaughterhouses, resulting in potential for widespread contamination of meat (especially poultry meat) during the slaughter process and at retail¹¹.

A Scientific Opinion Paper, published by the EFSA in 2009, indicated that the handling, preparation and consumption of broiler meat may account for 20% to 30% of human cases of campylobacteriosis, while 50% to 80% may be attributed to the

¹⁰ A report of the Study of infectious intestinal disease in England. 2000. FSA.

¹¹ www.food.gov.uk/science/surveillance/fsisbranch2009/fsis0409

chicken reservoir as a whole. This report is available on the EFSA website: www.efsa.europa.eu/en/scdocs/scdoc/1437.htm

Infection in animals

Clinical signs: Infection in poultry is generally subclinical as the organism is thought to be a normal gut commensal. *Campylobacter* spp. (*C. jejuni* and *C. coli*) may cause diarrhoea in companion and young animals, and abortion (mainly *C. fetus* but also *C. jejuni* and *C. coli*) in cattle, sheep and goats.

Frequency: Analysis of the EU baseline survey in 2008 on the prevalence of campylobacter in broiler batches estimated that the UK prevalence for campylobacter in broilers at slaughter (caecal contents) was 75.3%, and that 86.3% of broiler carcasses (skin samples) were contaminated¹². The UK prevalence was similar to the prevalence in other EU Member States with large broiler industries.

A national survey undertaken from 2007 – 2009 to examine the prevalence of *C. jejuni* and *C. coli* in poultry at slaughter gave an estimated prevalence of over 75%, consistent with previous cross-sectional surveys carried out in the UK. This survey incorporated the EU baseline survey completed in 2008.

Prevalence of campylobacter is seasonal with a summer peak during which prevalence levels can reach 100%.

There were a total of 99 recorded cases of thermophilic *Campylobacter* spp. detected in the UK in 2009: 29 in cattle, 67 in sheep and three in pigs. Most isolations of campylobacter in animals other than poultry are due to abortion investigations. In 2009, there were 217 *Campylobacter* spp. isolates from cattle and sheep abortions, a decrease of 11% from 2008. Of these, only 26% were the thermophilic species *C. jejuni* or *C. coli*, which are of the greatest public health importance. Seventeen percent of the isolates were other thermophilic species and 57% were non-thermophilic species. The source of *C. fetus* in humans is likely zoonotic but the routes of transmission are not well understood.

Control measures

Animals are infected via the faecal-oral route. Personnel and equipment can be responsible for transmission of infection between flocks, especially during the planned partial depopulation of the flock.

The FSA launched a campaign in January 2004 to help improve hygiene measures on broiler farms and ensure that best practices are followed at all times. An FSA strategy aimed to reduce the prevalence of campylobacter in UK-produced chicken on retail sale by 50% by 2010. A survey of the prevalence of *Campylobacter* spp. in chicken at retail published in October 2009 suggests that the target of reduction has not been met. The FSA strategy for 2010-2015 was published in 2009. This included a new draft campylobacter risk management programme.

¹² www.efsa.europa.eu/en/scdocs/scdoc/1503.htm

This will be described more fully in the 2010 Zoonoses Report and further information can be found at:

www.food.gov.uk/news/newsarchive/2009/dec/strategy

Good practice guidelines are published at:

<http://food.gov.uk/safereating/microbiology/flocks/>

In 2008, a Defra and FSA funded critical review of interventions and strategies to reduce campylobacter on the poultry farm was published at:

www.foodbase.org.uk/results.php?f_category_id=&f_report_id=384

The main recommendation of the review was the optimisation of biosecurity measures and the use of farm based interventions such as water or feed additives that may potentially complement an optimised biosecurity approach.

Summary of risk

Current risk mitigation is based on abattoir hygiene controls and advice to the consumer on food preparation and avoidance of cross contamination. Research is being undertaken by Defra to understand the epidemiology of campylobacter and assess the practicality and cost-effectiveness of medium and long term control options in livestock such as vaccination, competitive exclusion, bacteriophage and bacteriocins¹³. Abattoir based interventions such as physical or chemical carcass decontamination have been proposed and are implemented in some countries (e.g. USA). Chemical decontamination is not permitted in the EU. Physical treatments of fresh meat are permitted in the EU and are being investigated in the UK.

Further information

Further information about campylobacter in human is available from HPA at:

www.hpa.org.uk/Topics/InfectiousDiseases/InfectionsAZ/Campylobacter/

Summary profile for campylobacter in animals is available from Defra at:

www.defra.gov.uk/foodfarm/farmanimal/diseases/vetsurveillance/profiles/documents/sp-campylobacter.pdf

¹³ Bacteriocins are *proteinaceous toxins* produced by *bacteria* to inhibit the growth of similar or closely related bacterial strains.

Chlamydiosis and Psittacosis - including *Chlamydophila abortus* and *Chlamydophila psittaci*

Ovine chlamydiosis (*Chlamydophila abortus*)

Ovine chlamydiosis caused by infection with *Chlamydophila abortus* causes enzootic abortion in ewes (EAE), goats and occasionally cattle. The main route of transmission is through the inhalation of aerosols and contaminated dusts. This infection can cause serious zoonotic disease in pregnant women, resulting in stillbirth or abortion. However, infections appear to be rare, with very few reports of *C. abortus* in England and Wales each year.

Infection with *C. abortus* is not notifiable or reportable in animals.

Infection in humans

Symptoms: Infection may be asymptomatic. Where symptoms occur, they can resemble influenza: headache; chills; fever; joint pains and non-productive cough. Photophobia, vomiting, sore throat and myocarditis may also occur. The disease may be more severe in pregnancy, characterised by systemic illness with disseminated intravascular coagulation and renal and hepatic complications. This may be life threatening in the mother, and can lead to stillbirth or abortion.

Transmission: Human infections are believed to follow inhalation of aerosols and dust contaminated with *C. abortus* from livestock (such as organisms from sheep abortions).

Cases: The number of human cases of *C. abortus* occurring annually is uncertain as routine serological testing does not distinguish between *C. abortus* and other *Chlamydophila* species. Diagnosis of *C. abortus* is dependent primarily on clinical suspicion in a person with relevant exposure to sheep/lambing.

There are usually only one or two cases of *C. abortus* each year in pregnant women in England and Wales that result in abortion or stillbirth (numbers peaked at five cases in 1986). There have been no reports in Scotland. It is very rarely reported in Northern Ireland, with no reports in 2009.

Infection in animals

Clinical signs: Following infection, the organism remains latent until late pregnancy when it invades the placenta leading to abortions in the last two to three weeks of gestation, stillbirth and birth of live but weak lambs and kids. Aborting ewes occasionally appear dull, sometimes with a vaginal discharge immediately prior to abortion. Abortion storms affecting up to 25% of ewes may arise the year after introduction of the organism into naive flocks. It can also cause abortion in cattle and deer, although this is uncommon.

Frequency: In 2009, *C. abortus* was identified as the cause of abortion in 372 (41%) of 899 sheep and goat submissions to VLA and SAC laboratories in GB where a diagnosis was reached (Table 4). This is a 6.6% increase compared to 2008. A diagnosis is reached in 60-70% of all sheep and goat abortion submissions each year. *C. abortus* continues to be the most commonly diagnosed infectious cause of abortion in sheep. *C. abortus* was also confirmed in abortion material from two cattle. In Northern Ireland, 39 cases of *C. abortus* were identified in 2009, compared to 37 in 2008.

Table 4: Laboratory confirmed reports of *C. abortus* in animals in the UK, 2009

	GB	NI	UK Total
Sheep and goat abortions submissions to VLA and SAC in GB, and AFBI in NI, where a diagnosis is reached	899	203	1,102
<i>C. abortus</i> confirmed as the cause of abortion in sheep and goat submissions	372	38	410
Isolation of <i>C. abortus</i> in goat abortion material	5	0	5
Isolation of <i>C. abortus</i> in sheep abortion material	367	39*	406*

* In one isolation of *C. abortus* in sheep material in NI, *C. abortus* was not confirmed as the cause of abortion

Control measures

A live vaccine has been available for over 15 years and is widely used. Control of disease is achieved by maintaining a closed flock, purchase of EAE-free accredited stock from members of an accreditation scheme, or by vaccination. When dealing with an abortion storm, the use of antibiotics may be justified.

Summary of risk

Although the disease is widespread in animals in the UK, the number of cases occurring in pregnant women and other at-risk groups appears to be low. Intensive flocks and those housed for lambing tend to experience more significant disease problems due to a greater chance of transmission from contamination of the lambing environment by birthing fluids and abortion material containing the organism. Where antibiotics are used in late pregnancy in infected flocks to reduce the number of abortions, ewes may still shed the organism and the zoonotic risk remains for people for a period of up to a few weeks after birth or abortion.

Further information

Further information on chlamydiosis (EAE) risks during the lambing season is available from HPA at:

www.hpa.org.uk/Topics/InfectiousDiseases/InfectionsAZ/ChlamydiaAbortus/GeneralInformation/

Further information on chlamydiosis in animals is available from Defra at

www.defra.gov.uk/foodfarm/farmanimal/diseases/atoz/zoonoses/lambing.htm#chlamydia

Psittacosis; Ornithosis (*Chlamydophila psittaci*)

Psittacosis is an infection caused by the bacterium *Chlamydophila psittaci*. It has been described in over 130 species of birds but is most common in psittacines (parrots and parakeets). Other birds commonly affected include pigeons and doves, whilst turkeys, ducks and geese can also be infected. Transmission of *C. psittaci* from birds to humans results in an infection known as psittacosis, ornithosis or 'parrot fever'.

Infection occurs most often via infectious aerosols, and the presence of strong air currents may be a factor in its spread. Farm workers, processing plant operatives, bird keepers including pigeon fanciers, and veterinarians are particularly at risk. Precautions should be taken when handling infected birds or contaminated materials.

Infection with *C. psittaci* is not notifiable or reportable in animals, except in Cambridgeshire where a local anomaly exists (see Appendix 3).

Infection in humans

Symptoms: Psittacosis typically presents with influenza-like symptoms with fever, headache, muscle aches and respiratory symptoms, and may include atypical pneumonia with pleuritic chest pain. Although human disease is usually mild or moderate, it can be severe especially in untreated elderly or immuno-compromised individuals. Encephalitis, myocarditis and thrombophlebitis are rare complications. Relapses can occur.

Transmission: Apparently healthy birds may shed *C. psittaci* in respiratory secretions or faeces. Transmission of disease occurs mainly through inhalation of aerosols, respiratory secretions, or dried faecal or feather dust. Alternatively, people may be infected from handling infected birds' plumage and tissues. Rarely, person-to-person spread may occur during acute illness with paroxysmal coughing, although a lack of specific serological testing means that reported cases could have been caused by *Chlamydophila* species other than *C. psittaci*¹⁴. It is likely that most, if not all, cases of psittacosis are attributable to exposure to birds or bird products.

Cases: In 2009, there were 60 laboratory reports of human infection with *C. psittaci* in the UK; 58 cases in England and Wales and two in Scotland. This is similar to the 66^a cases reported in 2008 in the UK.

Infection in animals

Clinical signs: Clinical signs in birds are highly variable and may be absent, mild or severe. Common features tend to include conjunctivitis, ruffled feathers, greenish diarrhoea, depression, anorexia and loss of body condition, nasal exudate and

¹⁴ Control of Communicable Disease Manual, 19th Edition. Heymann DL, Eds. Publisher: American Public Health Association

^a There has been an amendment to the 2008 figure printed in last year's report as the data is derived from a dynamic database.

respiratory rates. The most specific sign in birds is often yellow urates, which indicates liver involvement, and a uni- or bilateral conjunctivitis and rhinorrhea.

Frequency: In recent years the popularity of keeping birds, in particular psittacines, and the sale of birds and other pet animals by large store chains has increased. Tests used on animal samples do not differentiate between different strains of *Chlamydophila*, so it is not possible to report the number of *C. psittaci* infections in animals in 2009.

Control measures

C. psittaci is susceptible to several antimicrobials. Treatment of infected and in-contact birds with broad spectrum antibiotics for periods of up to 45 days is effective in reducing infection, and in some cases may eliminate it entirely. No vaccines are commercially available in Europe for the protection of poultry against ornithosis.

Hygienic precautions are essential to minimise spread of infection to other birds and to human attendants. If a confirmed case of *C. psittaci* involving domestic poultry is reported, restrictions may be placed on movement of stock (including eggs, droppings, feed and litter) under the Psittacosis or Ornithosis Order 1953, followed by the recommendation of cleaning and disinfection of the premises.

Commission Decision 2000/666/EC, requiring specific action to be taken on detection of infection in psittacines, now applies to imported birds entering quarantine in the European Community. On suspected or confirmed infection of psittacines with *C. psittaci* during quarantine, all birds in the consignment must be treated by a method approved by the Regional Veterinary Officer in England and Wales or the Divisional Veterinary Manager in Scotland or the Divisional Veterinary Officer in Northern Ireland. The quarantine period must then be prolonged by two months after the last recorded case.

Summary of risk

C. psittaci can be carried by asymptomatic birds. This means that handlers of susceptible bird species are at risk of being exposed.

Domestic birds may be infected by wild birds. Where contact with wild birds can occur, such as free range farming systems and where birds are temporarily released into the wild (e.g. racing pigeons), there is an increased risk of birds acquiring infection. Owners of such birds are therefore at slightly increased risk of exposure and infection. Routine biosecurity measures, focussed on preventing mixing of wild and domestic birds, will reduce the likelihood of transmission to domestic poultry.

Further information

Further information on psittacosis in humans is available from HPA at: www.hpa.org.uk/Topics/InfectiousDiseases/InfectionsAZ/Psittacosis/

Cryptosporidiosis (*Cryptosporidium* spp.)

Cryptosporidiosis is a disease caused by protozoan parasites of the genus *Cryptosporidium*. Of those species with the greatest public health impact, *C. hominis* (and the related subtypes) is normally only recovered from humans and *C. parvum* (and its related genotypes and subtypes) is found in both animals and humans. *C. hominis* and *C. parvum* each represents almost half of all human cases in the UK and together are responsible for up to 97% of cases¹⁵.

Preweaned calves (aged 1-3 weeks) are the major animal reservoir for *C. parvum*, but infection can also be readily acquired from other species, particularly lambs and goat kids.

C. parvum is considered to be endemic on the majority of cattle holdings in the UK, and also common in sheep flocks and deer. Infected animals, especially lambs, may often shed significant numbers of oocysts without exhibiting any clinical signs and they can be a source of infection to man. *C. hominis* occurs in humans and spreads mainly by person-to-person transmission.

Infection with *Cryptosporidium* is not notifiable in animals in the UK.

Infection in humans

Symptoms: Cryptosporidiosis causes a range of symptoms including severe watery diarrhoea and abdominal cramping, preceded by anorexia and vomiting in children, although asymptomatic carriage can also occur. Immuno-compromised patients can suffer from prolonged watery diarrhoea that causes protein loss, malabsorption and a failure to clear the parasite causing fulminant disease.

Transmission: There is a bimodal seasonal pattern of confirmed reports of cryptosporidiosis in humans, with higher incidence occurring in spring and early autumn (Figure 3). In GB, the spring peak has been identified as predominantly *C. parvum* cases and most likely acquired from an animal source. This peak has declined since 2001, presumably as a result of improved drinking water quality. In contrast, the early autumn peak has a rise in *C. hominis* cases many of which are associated with travel outside the UK.

Cryptosporidium oocysts can be widely detected in aquatic and agricultural environments. Waterborne infections and outbreaks usually involve the consumption of contaminated drinking water. These can derive from human and animal sources. The majority of cases are sporadic and not part of recognised outbreaks.

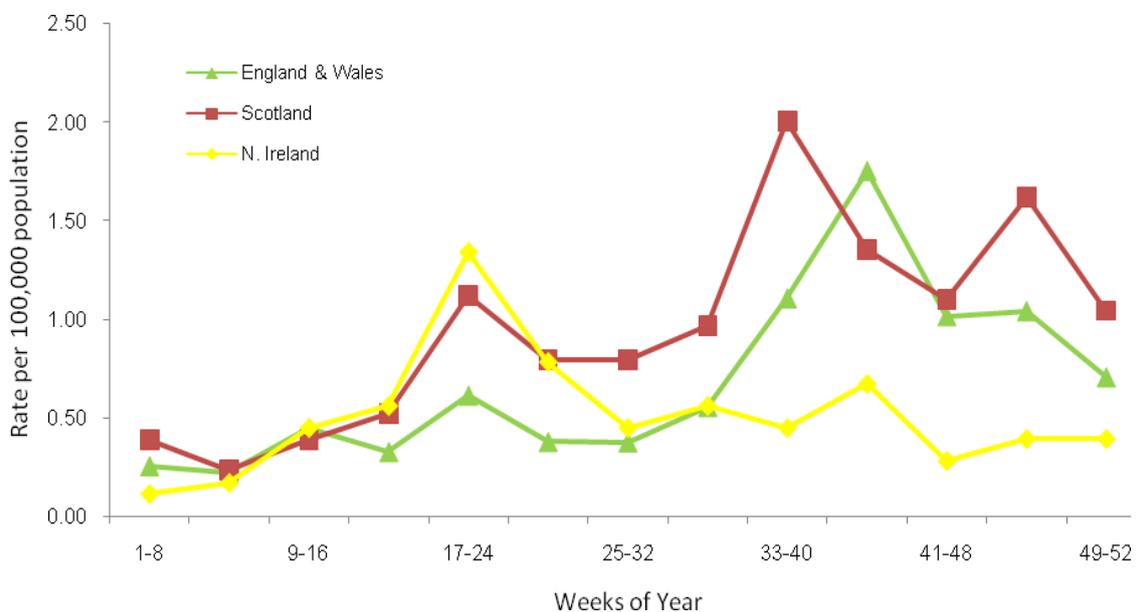
Foodborne infections can result from faecal contamination of foodstuffs by the use of contaminated surface water for irrigation. Direct contact with animals, by farm

¹⁵ Chalmers R. M, et al. (2009) Long-term *Cryptosporidium* typing reveals the aetiology and species-specific epidemiology of human cryptosporidiosis in England and Wales, 2000 to 2003. *Eurosurveillance* 2009 **14**(2) 15

workers/ veterinarians or the general public, and particularly by young children at open farms can also result in infection.

Cases: In 2009, there were 5,577 cases reported in the UK, 13.6% higher than in 2008 (n=4,909^a). In England and Wales, 4,821 cases of cryptosporidiosis were reported, which was 16.1% higher than the 4,151^a cases reported in 2008. Person-to-person spread, swimming pool-related outbreaks, and clusters contributed to the overall increase, as well as one major zoonotic outbreak linked to an open day on a commercial farm attended by 8 primary schools. The likely cause of this outbreak was direct contact with calves and goats. Contamination from the farm environment may also have been a factor. Hand washing facilities were limited and there was over reliance on the use of alcohol hand gel. In Scotland in 2009, there were 638 positive laboratory confirmations of cryptosporidiosis and 118 in Northern Ireland, which was about the same as 2008.

Figure 3: Laboratory confirmed reports of cryptosporidiosis in humans in the UK, 2009



Infection in animals

Clinical signs: Animal infection is often subclinical, but signs of diarrhoea, dehydration, dullness and inappetence can occur in young animals, particularly calves.

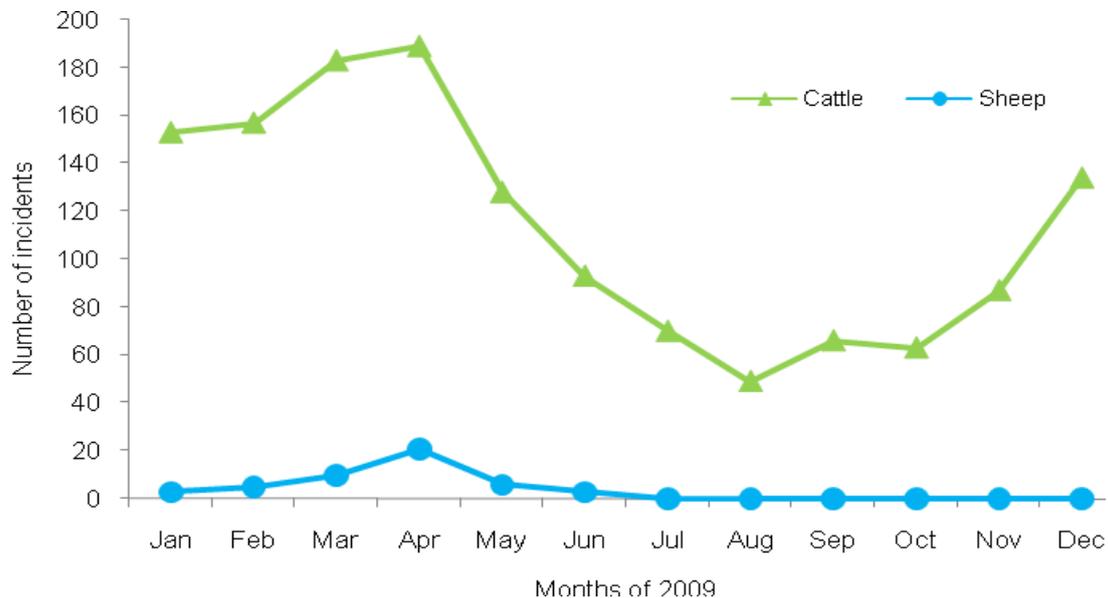
Frequency: Cryptosporidiosis is regarded as a common infection of livestock worldwide. Infection with *C. parvum* is frequently associated with young cattle or lambs, in which it may cause clinical disease¹⁶. Data from the VLA VIDA indicates that infection with *Cryptosporidium* accounts for 13.5% and 2.4% of clinical

¹⁶ Santin, M and Trout, J.M. (2008) in: *Cryptosporidium* and Cryptosporidiosis. (ed Ronald Fayer, Lihua Xaio. 2nd edition. Chapter 18 Livestock. 451-484

^a There has been an amendment to the 2008 figure printed in last year's report as the data is derived from a dynamic database.

submissions to government diagnostic laboratories in GB for cattle and sheep respectively.

Figure 4: Recorded diagnosis of cryptosporidiosis in cattle and sheep in GB, 2009



There were 1,346 diagnoses of animal infection with cryptosporidium recorded in GB, and 90 in Northern Ireland in 2009. Of these, 1,373 were in cattle, 48 were in sheep, five were in goats and 11 were in other animals. Recorded incidents in cattle and sheep show a distinct seasonal distribution, with a peak in the spring (Figure 4).

Control measures

The principal control measures for cryptosporidium in animals include improved farm biosecurity and stockmanship so as to reduce stress in, and transmission by, calves and lambs. Prophylactic treatment can be used to reduce levels of shedding of oocysts in livestock where cryptosporidiosis persists as a problem.

Measures to reduce the risk of waterborne transmission of cryptosporidium by protection of water catchment areas and improved water treatment have been instituted over recent years. A statutory requirement for water companies to undertake risk assessments and monitor drinking water from sources that are most vulnerable to contamination with the parasite was introduced in England and Wales in 1999; similar requirements were introduced to Northern Ireland. As a result, infection relating to animal contamination of drinking water supplies has declined since 2000.

Currently, since farm level measures do not prevent the shedding of oocysts into the environment by livestock, the avoidance of infection with *Cryptosporidium* relies principally upon personal hygiene when exposed to the farming environment, and mains water treatment. Effective hand hygiene after handling animals, especially children on farm visits, is important in preventing infection.

Summary of risk

The principal zoonotic risk of human infection is from young ruminants. There is a regional and seasonal pattern associated with the animal related cases of cryptosporidiosis in the UK. This is principally related to seasonal agricultural practices and the distribution of livestock as well as quality and protection of drinking water supplies. Thus, changes to the level of zoonotic risk for humans depend upon minimising the risk of infection from contact with young ruminants on farms open to the public, and controlling oocyst shedding and environmental contamination¹⁷. Reducing the risk of contamination presents challenges in some circumstances.

Further information

Further information on cryptosporidiosis in humans is available from HPA at:
www.hpa.org.uk/Topics/InfectiousDiseases/InfectionsAZ/Cryptosporidium/

Further information on cryptosporidiosis in animals is available from VLA at:
www.defra.gov.uk/foodfarm/farmanimal/diseases/atoz/index.htm#cryptosporidiosis

¹⁷ Chalmers RM and Giles M. (2010) Zoonotic cryptosporidiosis. *Journal of Applied Microbiology*. **109**: 1487-1497

Hydatid disease – including *Echinococcus granulosus* and *Echinococcus multilocularis*

Cystic hydatidosis (*Echinococcus granulosus*)

Echinococcus granulosus is a tapeworm which inhabits the small intestine of canines. The larval form of the worm is found as hydatid cysts in organs, commonly the liver or lungs of herbivorous intermediate hosts. The infection is potentially zoonotic, causing cystic hydatid disease (echinococcosis) in man. Human infection occurs when man acts as an accidental intermediate host. The main cycle of infection is between farm dogs and sheep. Nine *E. granulosus* strain genotypes are now recognised worldwide, of which two are present in GB: a sheep adapted strain involving a dog to sheep life-cycle, and a horse adapted strain involving a dog to horse life-cycle. Only the horse strain is present in Northern Ireland and there is no evidence that it is infective to man.

Sheep are the main intermediate host in the UK. They acquire hydatidosis by grazing on pastures contaminated with dog faeces containing cestode eggs, or by ingesting other feed contaminated by the eggs. Cattle can also be infected, but cysts are usually sterile. Dogs in turn are infected by ingesting viscera containing viable cysts.

The sheep strain is of worldwide importance as a zoonotic infection. It is common in countries where there are large sheep populations and dogs are allowed free access to carcasses. Cysts are slow growing and take several months to develop.

Surveillance of infection rates in dogs provides a more accurate indicator of the current regional prevalence and risk to the human population than abattoir surveillance, but is only carried out as time-limited surveys.

Infection with *E. granulosus* is not notifiable or reportable in animals in the UK.

Infection in humans

Symptoms: Cysts may grow for 20 or more years before becoming large enough to cause a range of symptoms depending on the affected organ and the location of the cyst. In locations such as the abdomen, where growth of the cyst is not restricted by anatomical structures, it can grow very large and contain several litres of fluid.

Diagnosed cases usually involve major organs and require complex invasive treatments and chemotherapy. Rupture of the cyst or spillage during surgery may release material that can infect other organs. Some cases may be inoperable and some are fatal. The fatality rate for individuals is highly variable and is dependent on the location of the cyst and the duration of infection when first diagnosed¹⁸.

¹⁸ Gemmell, M.A. & Roberts, M.G. Cystic echinococcosis (*Echinococcus granulosus*) (1998) In "Zoonoses". Eds. Palmer, S.R., Lord Soulsby & Simpson D.I.H. Oxford University Press, 665-688

Transmission: The main route of infection is through direct contact with infected dogs or their faeces. Transmission between humans does not occur. Young children in close contact with free roaming dogs living in the countryside are considered to be at higher risk of infection.

Cases: During 2009, nine confirmed cases of hydatid disease in humans were reported in the UK. All cases were reported in England and Wales and were thought to have contracted disease outside the UK. The number of indigenously acquired human cases in the UK is currently very low, with one new indigenously contracted case identified approximately every five years.

Infection in animals

Clinical signs: Neither sheep nor dogs show signs of clinical infection. The impact of the disease on the health of the individual animal is negligible.

Frequency: Meat inspection is carried out in all abattoirs in the UK under the control of an Official Veterinarian on behalf of the FSA. All carcasses and offal, as required by Regulation, are routinely inspected visually and, depending on species, by palpation and incision. Hydatid cysts may be found in almost any part of the body but most often in the liver and lungs. Other affected organs may include brain, muscles, kidneys, bones, heart and pancreas.

The following figures are reported findings of hydatid disease at post mortem inspection of sheep and cattle for human consumption at licensed abattoirs in GB¹⁹. During 2009, there was a throughput of 14,982,297 sheep, of which 74,491 (0.50%) were affected with hydatid cysts; there was a throughput of 1,710,984 cattle, of which 1,422 (0.08%) were affected with hydatid cysts. Figures are slightly lower than in 2008, when 0.53%^a of sheep and 0.14%^a of cattle in GB licensed abattoirs were recorded as affected with hydatid cysts.

In Northern Ireland there was a throughput of 453,726 sheep and 582,299 cows during 2009. There were no post mortem detections of hydatidosis in any species slaughtered throughout 2009. The last recorded detection in Northern Ireland was in June 2006.

There is evidence to suggest a rising trend in dog infestation in South Powys, Wales, following a free dog worming campaign in the eighties²⁰. However there is no current indication that transmission to the human population has increased.

Data from the South Wales pilot hydatid control programme indicates a prevalence of 9% of farm dogs sampled²¹. However, one or more dogs on 20% of farms tested positive, and this represents a potential human health risk at one in five farms in

¹⁹ Data from FSA Operations, York

²⁰ Buishi I, et al. (2005) Re-emergence of canine Echinococcus granulosus infection, Wales. *Emerg Infect Dis.* 11(4):568-71

²¹ <http://wales.gov.uk/topics/environmentcountryside/ahw/disease/hydatiddisease/hydatiddiseasecampaign/?sessionid=JWtnMQ2CT70WCXMnXI2mWLLKPVHgpLM95Z0nDnqTC4bbG9J3fY1b!-25131489?lang=en>

^a There has been an amendment to the 2008 figure printed in last year's report as the data is derived from a dynamic database.

South Powys. There have been no recent studies in any other part of GB and no firm conclusions can be made about risk to humans in the rest of GB. Abattoir post mortem inspection suggests that hydatid is found in animals from various parts of GB. Infection is spread by movements of infected sheep or dogs.

Control Measures

Control or eradication requires long term cross government support for education, dog worming, control measures and enhanced surveillance to monitor progress. UK animal by-product regulations require appropriate disposal of livestock carcasses and offal, which minimises the scope for dogs and other scavengers to consume potentially infected material. Livestock infection causes only marginal economic losses to farmers from condemnation of affected organs at slaughter.

Education programmes in areas with historically high human prevalence have been carried out to raise awareness of the risks and to highlight to stock owners their legal and social obligations regarding the prompt and appropriate disposal of fallen stock, to discourage the feeding of uncooked offal and carcass meat to dogs, to encourage good personal hygiene and the regular worming of dogs.

Although there appears to be a very low incidence of UK acquired human infection, concerns were raised by the suggestion of a rising prevalence in dogs on farms in South Powys. In 2008, the Welsh Assembly Government launched a Wales-wide hydatid disease awareness campaign and a South Powys pilot eradication scheme with follow up testing throughout 2009. Analysis of the results is ongoing and will be published when this is completed²⁰.

Summary of risk

The distribution of hydatid disease in humans has historically not been uniform across the UK and this is believed due to factors beyond just the risk from livestock and dog contact. Although there have been very few cases in humans over the last two decades, there is an ongoing known low risk for people in the South Wales sheep farming areas and an unknown, but anticipated, very low risk in the rest of GB.

Alveolar echinococcosis (*Echinococcus multilocularis*)

Echinococcus multilocularis causes alveolar hydatid disease, which has a wide geographical distribution throughout North America, Asia and Europe. Alveolar hydatid disease is a much more invasive disease in man than cystic hydatidosis. The life-cycle normally involves foxes and small rodents, particularly voles. Dogs, cats and wolves may also act as definitive hosts. *E. multilocularis* is absent from the UK, however there is evidence that its distribution is spreading in northern Europe^{22,23,24} due to increasing fox populations. Particular concern has been expressed in relation

²² Takumi, K., et al (2008). Evidence for an increasing presence of *Echinococcus multilocularis* in foxes in The Netherlands. *Intl J for Parasitology*, **38**(5):571-578.

²³ Berke, O. et al. (2008). Emergence of *Echinococcus multilocularis* among red foxes in northern Germany 1991-2005., *Veterinary Parasitology*, **155**(3-4):319-322.

²⁴ Vervaeke, M. et al. (2006). Spatial spreading of *Echinococcus multilocularis* in red foxes across nation borders in Western Europe. *Preventive Veterinary Med.*, **76**(3-4):137-150.

to the increase in the number of urban foxes. Dogs and cats entering the UK from mainland Europe are currently required to receive treatment for *E. multilocularis* under the 'Pets Travel Scheme', detailed in the Rabies section.

Further information

Further information about hydatid disease in humans is available from HPA at:
www.hpa.org.uk/Topics/InfectiousDiseases/InfectionsAZ/HydatidDisease/

Further information on hydatid disease in animals is available from Defra at:
www.defra.gov.uk/foodfarm/farmanimal/diseases/atoz/zoonoses/hydatid.htm

Influenza – including pandemic H1N1 (2009), avian and swine influenzas

Influenza is a respiratory infection caused by viruses of the orthomyxoviridae family. The virus is usually considered to be of avian origin that, over time, has evolved so that certain subtypes are now capable of causing human infection. There are three influenza genera (influenza A, B and C). Of these, influenza A viruses can affect many species of animals, including humans and this text is therefore limited to this genera.

The segmented nature of the virus genome allows the creation of new subtypes through genetic re-assortment when different subtypes co-infect the same host. There are several potential hosts where genetic reassortment can occur, leading to the production of a virus that may be capable of infecting and spreading within other host populations (Feature Article 2: Swine Influenza: A disease of renewed global interest).

Avian influenza, also referred to as 'Fowl Plague' or 'Bird Flu', is a disease of birds caused by type A influenza viruses. It is highly infectious and can affect many species of birds. Swine influenza is caused by influenza A virus of multiple serotypes in animal hosts. Avian and swine influenza are examples of influenza from a host species (birds and pigs) which are potentially capable of infecting humans.

Animal adapted influenza viruses do not readily infect people. However, spontaneous mutation or reassortment of influenza genes between human and animal strains can occur. Some of these strains can potentially be readily transmitted between people and can lead to pandemic spread in humans, as in the case of the 1918 Spanish flu pandemic.

Infection in humans

Symptoms: A typical influenza illness consists of fever, cough, myalgia and, in children, gastrointestinal symptoms may occur. Occasionally influenza infections can lead to the development of pneumonia and death. The symptoms in humans associated with infection of animal strains vary depending on the subtype and can include conjunctivitis and diarrhoea. Infection with most animal strains is likely to be mild or sub-clinical. However, the H5N1 avian influenza strain in Asia and Africa has produced severe or fatal illness in a high proportion of people who become ill.

Infection of humans with swine-adapted strains is infrequent, generally mild and rarely fatal. However, acute cases of respiratory disease with additional complications have been reported in immuno-compromised patients.

Transmission: Transmission of animal strains to people usually occurs only after a high level of exposure to an infected animal (such as a bird or pig) or their excretions, and so is an occupational risk. It is not usually possible to confirm the route of infection, but it is assumed that this takes place when droplets of moisture containing virus are inhaled or possibly when they enter through the conjunctiva.

Neither highly pathogenic avian influenza (HPAI) nor swine influenza are known to be transmitted to people via cooked food. The FSA does not consider avian or swine influenza to pose a food safety risk²⁵. However, HPAI viruses may be present in the meat and eggs of infected poultry or food products as a result of contamination by HPAI from infected bird faeces. Nevertheless, it is unlikely that H5N1 could be passed on to people, even by raw meat or eggs. Cooking food properly would inactivate the virus and eliminate this potential risk²⁶.

Cases: Human cases of avian influenza in the UK are very rare. In 2006, there was one confirmed case of H7N3 in a farm worker. In 2007, there were four cases in owners who kept birds associated with a H7N2 poultry outbreak. All viruses were of low pathogenicity for poultry. No cases were reported in 2009.

There have been no deaths reported as a result of avian influenza in the UK, however, a number of people worldwide have died after infection with certain strains of avian influenza, notably H5N1. Between 2003 and the end of 2009, 468 human cases of H5N1 had been reported worldwide, resulting in 282 deaths²⁷. Almost all of these people had close contact with birds infected with H5N1.

Sporadic human cases infected with various strains of swine influenza have been reported around the world²⁸. There were around 50 human cases worldwide between 1958 and 2005, with no cases reported in the UK. Subtypes of these swine influenza infections were mainly influenza AH1N1 with some AH3N2. The overall case-fatality rate was reported as 14%.

Further information: Travel advice and advice for travellers returning from countries experiencing avian influenza outbreaks can be obtained from the Department of Health or from the National Travel Health Network and Centre (NaTHNaC):

www.nathnac.org/travel/factsheets/flu_advice.htm

Pandemic H1N1 (2009) influenza in humans

Initial research indicated that the human strain of pandemic (H1N1) 2009 influenza was most closely related to pig strains of the virus in all of its gene segments. Reassortment of two viruses in pigs is a plausible but yet unproven explanation.

Symptoms: The symptoms of pandemic (H1N1) 2009 influenza are similar to those of human seasonal flu, and are mild in most people. However, in a small minority of cases, particularly young adults with certain risk factors, such as respiratory, heart, liver disease or pregnancy, infection can be severe.

Transmission: Transmission occurs between people in the same way as seasonal flu; via droplets from coughing or sneezing by an infected person, direct contact with an infected person, or by touching contaminated surfaces or objects (e.g. door handles) and then touching mouth, eyes or nose without first washing hands.

²⁵ www.food.gov.uk/multimedia/webpage/birdflu/birdflufaq/

²⁶ EFSA (2005). European Food Safety Authority, press release no 1193, 26 October 2005.

²⁷ www.who.int/csr/disease/avian_influenza/country/cases_table_2010_12_09/en/index.html

²⁸ Myers, K.P. et al (2007). Cases of swine influenza in humans: a review of the literature. *Clinical Infectious Diseases*, **44**:1084–8.

During the 2009 H1N1 pandemic there was widespread inter-human transmission across the UK with two waves of activity in early summer and autumn 2009. There were 28,164 laboratory-confirmed cases in humans, although only a proportion of cases would have been confirmed after the laboratory testing strategy changed to utilising a clinical case definition.

Control Measures: Guidance on how to limit the spread of infection was distributed to households across the UK in May 2009, and a vaccine for (H1N1) 2009 was made available in October 2009. Advice was principally based upon the application of good hygiene measures. Details of all of these measures can be viewed at: www.direct.gov.uk/prod_consum_dg/groups/dg_digitalassets/@dg/@en/documents/digitalasset/dg_177903.pdf.

Further information: on H1N1 influenza in humans can be found at:

- www.hpa.org.uk/Topics/InfectiousDiseases/InfectionsAZ/SwineInfluenza/
- www.nhs.uk/conditions/pandemic-flu/Pages/Introduction.aspx
- www.who.int/csr/disease/swineflu/en/

Avian Influenza in animals

Avian influenza is one of the most important poultry diseases, potentially causing high mortality and having a significant impact on production of poultry meat and eggs. It is not normally present in the major poultry species kept in the UK. Infections with influenza A viruses of H5 and H7 serotypes are statutory notifiable diseases in poultry and must be reported to Animal Health.

HPAI can cause severe disease in poultry, with a high flock death rate (up to 100%) in affected flocks. This disease can develop so rapidly that birds may die without showing any previous signs of disease. Other strains known as "low pathogenicity avian influenza" (LPAI) viruses can result in milder, less significant disease. However, LPAI viruses can mutate into highly pathogenic strains. Both HPAI and LPAI describe the virus pathogenicity in 'Galliformes' species of poultry (chickens and turkeys) only.

The highly publicised H5N1 HPAI strain has been responsible for considerable poultry losses across Asia and in recent years in Europe and other parts of the world as well. The UK has maintained a high vigilance for avian influenza in recent years due to westward spread of H5N1 from Asia and occasional incursion of other influenza viruses to European poultry.

Clinical signs: A wide spectrum of clinical signs can be presented with avian influenza. HPAI is highly visible in gallinaceous poultry where there is rapid onset of signs, decrease in food or water intake, respiratory signs, oedema of the head and face, haemorrhages on the legs and skin, occasional neurological signs and high mortality. In domestic waterfowl (ducks and geese) the clinical signs can be variable dependent on the virus strain but are less likely to be apparent, presenting as a mild respiratory disease with diarrhoea and will frequently be asymptomatic. Infection in chickens and turkeys may induce a decline in egg production with an increase in the number of abnormal eggs.

Frequency: During 2009, there were no notified cases of disease in birds as a result of infection with HPAI.

Active surveillance of UK poultry stocks for viruses of H5 and H7 subtypes has been undertaken annually since 2003. Infrequently, antibodies to H5 or H7 infection subtypes have been detected in a small number of sampled birds, which is most likely indicative of prior exposure to H5 or H7 LPAI virus strains, and in ducks these were most likely to indicate non-specific reactions which are recognised to occur.

During 2009, two of 453 holdings sampled in the UK had birds with antibodies to avian influenza viruses of subtypes H5 or H7; one detection was from a duck flock, and another involved a flock where antibodies were detected in both ducks and geese. This compared with eight detections from 374 holdings sampled in 2008.

During 2009, a total of 2,144 wild birds were sampled in the UK. The majority of birds (80%) were sampled by live trapping, while the remaining 20% were birds that were found dead by the public or warden patrols of wetlands and reserves. Most of the birds sampled (60%) were of the order *Anseriformes* (ducks and geese). H5N1 HPAI (notifiable in wild birds since 2003) was not detected but other influenza A viruses of several subtypes (consistent with continual maintenance in these reservoir populations) were found in 19 positive birds.

Control measures: Statutory import controls on live birds, eggs and poultry products prevent the introduction of exotic strains of Avian Influenza to the UK. Vaccines are not currently authorized for general use in the UK although English zoos are able to apply for permission to vaccinate their birds, subject to meeting certain eligibility criteria.

Identification of Avian Influenza would result in a full disease outbreak response including antiviral prophylaxis and the use of personal protective equipment by those involved with the birds. If disease is confirmed, all susceptible birds on affected premises are culled, together with high risk contacts on other premises. Movement restrictions in the surrounding area and action to re-establish disease free status are specified by EU legislation. Compensation is payable at market value for healthy birds compulsorily culled. Statutory controls are also implemented in laboratories working with the virus.

Summary of risk: Stringent import controls exist, however HPAI could be introduced into the UK through live poultry or poultry product imports, or by infected migratory wild birds. As the UK is a large importer of these products and cannot control the movement of migratory birds, the low risk of HPAI being introduced will always be present.

New strains could arise either by mutation of an existing virus strain, or by a process known as "genetic reassortment" where co-infection with different subtypes in a host at the same time can cause the mixing of genetic material of the two viruses to produce new strains, as outlined previously.

Further information: is available at:
www.defra.gov.uk/vla/science/sci_ai.htm

www.defra.gov.uk/foodfarm/farmanimal/diseases/atoz/ai/keptbirds/#natsurvey

http://ec.europa.eu/food/animal/diseases/controlmeasures/avian/res_surv_ai_annual_09_en.pdf

www.defra.gov.uk/vla/reports/docs/rep_survrep_qtlyw0409.pdf

Swine influenza in animals

Swine influenza is a contagious respiratory disease of pigs that occurs worldwide and is caused by infection with influenza A viruses. Short distance transmission between pigs can occur by droplets generated by infected pigs coughing or sneezing, by direct or indirect contact, or by the movement and introduction of infected pigs not showing clinical signs. Factors, such as the presence of other infections, can make disease appear more severe or seem to last longer.

Infections of influenza A viruses in pigs are notifiable in Northern Ireland but not in the rest of the UK.

Clinical signs: The main symptoms are a catarrhal inflammation of the respiratory tract, laboured jerky breathing and a hard paroxysmal cough. Recovery is rapid and mortality is usually low.

Frequency: Influenza A viruses of H1N1, H1N2 or H3N2 subtypes are thought to be endemic worldwide, except in Australasia. This situation presents a continual interface with humans and thereby increases the risk for potential zoonotic spread.

Following the human pandemic H1N1(2009) event in 2009, there was subsequent spread to pig populations on all continents except Africa.

Data collected from submissions to VLA and SAC laboratories using the VIDA system show that there is a relatively low, stable level of swine influenza diagnoses recorded in pigs in GB. In 2009, there were 18 diagnoses of swine influenza in pigs in the UK (Table 5) including cases of infection with pandemic H1N1. It should be noted that these were detected subsequent to global spread in humans including the UK.

Table 5: Laboratory confirmed cases of swine influenza in pigs in the UK, 2005-2009

Diagnosis	2005	2006	2007	2008	2009
GB	23	13	9	17	14
NI	0	0	0	0	4
UK Total	23	13	9	17	18

Despite the low number of recorded incidents, herd infection levels are high, based on serological studies²⁹. At herd level up to 60% may have encountered infection with one or more influenza viruses during their production life.

Control measures: Good hygiene and biosecurity practice within the swine industry is implemented to minimise the impact of infectious disease, including swine influenza. A code of practice was developed by Defra and key industry stakeholders to provide advice and guidance on how to minimise spread and the impact of infection.

Details can be found at:

www.defra.gov.uk/foodfarm/farmanimal/diseases/atoz/swine-flu/documents/cop090803.pdf.

In GB, routine active surveillance programmes and strategic research activity examining the epidemiology of infection of swine influenza in animals and addressing zoonotic risk cost around £250k per year.

Summary of risk: There is continual circulation of endemic swine influenza viruses in pigs, and the appearance of new strains of influenza virus in swine is mediated through changes in the epidemiology of infection in people and poultry. The situation is dynamic and changes may occur in the virus through mixing of co-circulating strains. There is no data to show that these strains necessarily provide any increased risk for either animal or human infection.

Further information: on Swine Influenza in animals can be found at:

www.defra.gov.uk/foodfarm/farmanimal/diseases/atoz/swine-flu/index.htm

www.defra.gov.uk/vla/science/sci_si.htm

²⁹ Brown I. H. (2008). The Role of Pigs in Interspecies Transmission, In: Avian Influenza. Monogr Virol.; Klenk H-D, Matrosovich MN, Stech J (eds): Basel Karger, 2008, vol27, pp 88-100.

Leptospirosis (Leptospira interrogans serovars)

Leptospirosis is a zoonotic disease caused by the bacterium *Leptospira interrogans*; of which only some strains are pathogenic. *L. Icterohaemorrhagiae* is the main serovar causing human disease. Leptospire are widespread amongst feral and domesticated mammals, which are maintenance hosts for over 250 known serovars³⁰. Only a small number of serovars are endemic in any particular country or region. Those encountered most frequently in farm livestock in the UK are *L. Hardjo* (cattle), *L. Bratislava* (pigs) and *L. Icterohaemorrhagiae* (which affects a wide range of wild and domestic species). Leptospirosis is a major cause of economic loss to the intensive cattle and pig industries of the developed world³¹, and is still an important occupational disease risk for people working in agriculture.

Clinical disease in animals in GB is less common than in the past although it is still a significant problem in Northern Ireland.

Leptospirosis is not notifiable or reportable in animals in the UK.

Infection in humans

Symptoms: Infection with leptospira can be asymptomatic, or can cause a mild flu-like illness, or a more severe illness called Weil's disease, with jaundice and kidney failure. The lack of a characteristic clinical presentation makes laboratory confirmation essential for an accurate diagnosis. Generally, cases will recover fully within two to six weeks but some may take up to three months. In most cases a full recovery is made, however two to three people in England and Wales die every year from leptospirosis.

After infection, immunity develops against the infecting strain, but may not fully protect against infection with unrelated strains.

Transmission: Humans mainly acquire infection by direct contact with infected urine through mucous membranes, eyes, cuts and abrasions. Infection can also occur indirectly through contact with water, soil or foods contaminated with urine from infected animals³². Leptospire may be present in cows' milk in acute cattle cases at the peak of fever, although the bacteria do not survive for long in undiluted cows' milk (about 30 minutes) and are destroyed by pasteurisation³³. Recent human cases of *L. Icterohaemorrhagiae* infection have been linked to pet rats. Person-to-person spread is very rare.

³⁰ Higgins, R (2004) Emerging or re-emerging bacterial zoonotic diseases: bartonellosis, leptospirosis, Lyme borreliosis, plague. In: Emerging zoonoses and pathogens of public health concern. *Rev. sci. Off. int. Epiz.* **23**(2), 569-581

³¹ Ellis, W.A (1998). Leptospirosis. In: Zoonoses Eds S R Palmer, Lord Soulsby and D.I.H. Simpson Oxford, Oxford University Press, p115-126.

³² Acha, P.N & Szyfres, B (2001) Leptospirosis in: Zoonoses and communicable diseases common to man and animals, Vol 1, Bacterioses and mycoses, Pan American Health organisation, Washington pp 157-168.

³³ Radostits, O. et al (2007) Leptospirosis in: Veterinary Medicine, 10th Edition, Saunders Elsevier, Edinburgh 1094-1123.

Cases: During 2009, 54 cases of leptospirosis were reported in the UK: 38 were acquired indigenously, and 16 were acquired through travel. Serovars were determined for 14 of the 38 indigenous cases by the Leptospira Reference Unit. These were *L. icterohaemorrhagiae* (n=10); *L. Saxkoebing* (n=2); *L. Australis* (n=1); *L. Autumnalis* (n=1); and for 24 the infecting serovar was not determined. Two patients died as a result of their infection. Thirty-three infections occurred in men and five in women, with ages ranging from 14 to 78 years.

Of the 24 people with indigenously acquired infections for whom risk data was available, nine infections were likely to have been acquired through occupational activities, and 15 through recreational exposures.

Sixteen people acquired leptospirosis whilst overseas in 2009, mostly from South-East Asia and Central America where the serotypes encountered may differ from those found in the UK. Infections in this group of people are most likely to occur in travellers who have participated in recreational water sports.

Table 6: Laboratory confirmed reports of leptospirosis in UK residents, 2000-2009

Year	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009
England & Wales	54	48	54	28	29	41	44	74	62	52
Scotland	0	0	3	0	2	4	3	6	13	2
NI	0	0	1	0	1	1	4	1	1	0
UK	54	48	58	28	32	46	51	81	76	54

Infection in animals

Clinical signs: In the UK, leptospirosis (*L. Hardjo*) is primarily a reproductive disease of cattle, although it also causes reproductive failures in sheep in Northern Ireland. In classical acute infection with *L. Hardjo* there can be a short systemic illness which usually affects several cows in a herd, causing fever, malaise and anorexia with variable sudden milk drop, sometimes with a change in the consistency of the milk produced and 'flabby bag syndrome'. The latter is now very uncommon. Cattle can be infected for months or even years without showing signs, and the first sign will usually be abortion or infertility. Uterine infections, including those acquired as young calves, can cause chronic infertility, with increased numbers of services per conception and greater calving to conception intervals.

In sheep, late abortions, full-term stillbirths, weak lambs and agalactia have been reported in Northern Ireland, although such clinical features appear to be rare in GB³⁴.

Frequency: The constituent countries within the UK use different diagnostic methods, and the diagnostic criteria required for disease confirmation have also changed in recent years, so it is difficult to make comparisons between countries and time periods.

³⁴ McKeown, J.D & Ellis, W.A. (1986), *Leptospira Hardjo* agalactia in sheep. *Veterinary Record*, **118**:482.

During 2009, only one of 394 diagnostic specimens from submissions to VLA regional laboratories in England and Wales examined by real time- polymerase chain reaction (RT-PCR) for pathogenic leptospires was positive. There were five confirmed cases of leptospirosis in animals in GB.

However, in Northern Ireland in 2009, of 764 suitable samples (including cattle samples) examined by the fluorescent antibody test or serology (microscopic agglutination test), there were 84 positives (11.0%).

There were also 9,853 serum samples from a range of species from England and Wales examined by the VLA for various reasons, including export (mainly dogs). A summary of the positive samples is given in Table 7. The data are further biased because only a few samples were examined for the full range of serovars. Note that these data only indicate serological evidence of exposure and/or vaccination, which is widely practiced, and not clinical disease.

Table 7: Isolations of *leptospira* spp. from serum samples in animals in England and Wales, 2009*

	Dogs	Cattle	Pigs	Horses
Total samples	3,803	4,926	546	270
Positive <i>L. Canicola</i>	1,571	-	-	3
Positive <i>L. Icterohaemorrhagiae</i>	681	-	-	2
Positive <i>L. Hardjo</i>	2	1,210	-	-
Positive <i>L. Bratislava</i>	3	8	223	3
Positive <i>L. Zanoni</i>	7	-	-	-
Positive <i>L. Copenhageni</i>	9	-	-	7

*It should be noted that results only reflect the serological tests requested for each submission, and therefore significant titres could be missed.

Serological testing of dairy herds in England and Wales in 2009 to monitor *L. Hardjo* status continued to show evidence of potentially active infection and/or extensive vaccination in about 50-60% of herds.

Control measures

Rodent control is important for reducing the risks from *L. Icterohaemorrhagiae* infection in all species. Rats are not involved in the epidemiology of *L. Hardjo* in cattle because infected cattle (and possibly sheep) are the reservoirs of infection. The main routes of infection for *L. Hardjo* in cattle in GB are infected animals added to a group (including hire bulls), and shared watercourses. These risk factors should be considered when reviewing biosecurity precautions. The main control measure for cattle is vaccination; strategic antibiotic treatment can also be used in infected herds. Routine monitoring for leptospirosis using bulk milk antibody testing is a cornerstone of health control programmes in dairy herds. Awareness amongst farm staff is important to prevent human disease, especially as leptospires can survive in damp soil for more than 30 days.

Summary of risk

Leptospirosis in cattle (*L. Hardjo*) mainly poses a risk to farm and abattoir workers. The general population is mostly exposed to leptospira (including *L. Icterohaemorrhagiae*) through recreational activities involving watercourses contaminated with urine from infected animals.

Further information

Further information on leptospirosis in humans is available from HPA at:
www.hpa.org.uk/Topics/InfectiousDiseases/InfectionsAZ/Leptospirosis/

Further information on leptospirosis in animals is available from VLA at:
www.defra.gov.uk/vla/science/sci_lepto.htm

Listeriosis (*Listeria monocytogenes*)

Listeria monocytogenes is widely distributed in the environment, including soil, decaying vegetation and fodder in silos in which the bacteria can multiply.

Listeriosis is most commonly associated with pregnant women, neonates and people over the age of 60 years with a range of underlying medical conditions including cancer and diabetes.

Listeriosis in animals is chiefly a disease of farmed ruminants, with cattle and sheep considered the most important species. Infection occurs due to direct ingestion of soil, or through soil-contaminated feed, notably spoilt silage. Animal health controls focus on the provision of good quality silage and reducing soil intake in feed.

Infection in humans

Symptoms: A person with listeriosis may show signs of fever, muscle aches, and sometimes gastrointestinal symptoms such as nausea or diarrhoea. If infection spreads to the nervous system, this may present with headaches, stiff neck, confusion, loss of balance, or convulsions. Infected pregnant women may experience only a mild, flu-like illness, although infections during pregnancy can lead to miscarriage or stillbirth, premature delivery, or infection of the newborn.

Listeriosis is a rare but severe disease with high mortality. An estimated 35% of people with invasive listeriosis die due to complications that arise from the infection.

Transmission: Consumption of foods contaminated with *L. monocytogenes* is the main route of transmission for human infection. Foods particularly associated with infection include unpasteurised cheeses, cold cooked meat, pâtés and smoked fish. Although listeriosis is rare in the UK in comparison to other foodborne illnesses, a rise in the number of cases has occurred since 2001, particularly in people over the age of 60 years. The HPA and FSA have been working closely to try to identify foods and other factors which may have contributed to this rise in cases.

Human infection acquired directly from animals is possible although cases reporting an animal contact are rare. Direct animal contact can cause minor skin infections and is chiefly a risk for farmers or veterinary surgeons attending calving or aborting ruminants. Faecal contact is also a possible route of infection. Transmissibility between humans is thought to be extremely rare.

Cases: The level of reported listeriosis in people in the UK has remained relatively stable over the last 5 years. There were 235 cases in the UK in 2009, of which 35 were pregnancy-associated cases (Table 8). This is an increase of 13% compared to the 208^a cases reported in 2008, with the greatest rise (18%) in England and Wales. In Northern Ireland the number of cases returned to more usual levels following a single outbreak of seven cases in 2008.

Table 8: Laboratory reports of listeriosis in humans in the UK, 2000-2009

	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009
England and Wales										
Total cases	101	145	139	233	211	189	185	227	182	214
Pregnancy associated cases	13	18	10	34	21	25	25	28	20	34
Others	88	127	129	199	190	164	160	199	162	180
Scotland										
Total cases	11	12	19	11	15	28	17	23	15	17
Pregnancy associated cases	0	0	0	1	1	2	1	2	0	1
Others	11	12	19	10	14	26	16	21	15	16
NI										
Total cases	4	5	2	3	4	3	6	5	11	4
Total	116	162	160	247	230	220	208	255	208^a	235

Listeria in food

Two surveys in 2009 investigated food as a source of *Listeria* spp. One hundred and thirty-three samples were taken as part of a curtailed EU harmonised survey of *Listeria* spp. in ready to eat foods. Forty-seven samples of meat product, 45 samples of soft cheese and 41 samples of smoked fish were tested for the presence of *Listeria monocytogenes*. *L. monocytogenes* was found in two samples of smoked fish (both at less than 20 cfu/g, as the legal limit under the microcriteria regulations for these types of product is 100 cfu/g, no action was taken on the positive results). There were no positive results for the meat products or for the cheeses, although one cheese sample was positive for *Listeria seeligeri* and one for *Listeria welshimeri*. A full survey is due to be repeated in 2010/11.

In 2009, the FSA also conducted an imported foods survey which sampled 115 fresh chickens and 32 cooked shellfish products. One chicken sample and one shellfish sample were found to be positive for *L. monocytogenes*.

Infection in animals

Clinical signs: Disease is confirmed most frequently in farmed ruminants. The clinical manifestations most commonly recognised are encephalitis and abortion, although an enteric form has also been described (rarely) in sheep. In cattle, abortion due to listeria infection is diagnosed approximately twice as often as encephalitis, but in sheep and goats encephalitis is diagnosed two to three times as often as listeria abortion. Septicaemia, gastrointestinal and eye infections can also occur in some animals. Disease occurs most frequently in association with the feeding of stored ensiled forage, where soil contamination has occurred and conditions result in poor anaerobic preservation, the bacteria being able to multiply where the acidity has not fallen to less than pH 5.5. Disease outbreaks are usually sporadic and affect only a few animals in each herd.

Frequency: In the UK the majority of cases occur between January and April when animals are housed.

During 2009, 196 incidences of listeriosis were diagnosed in animals in the UK (Table 9). Of these, 177 occurred in GB compared to 191 in 2008, a decrease of 7%; two-thirds of the diagnoses were from sheep and goats.

Table 9: Confirmed listeria cases in animals in the UK, 2009

Year	<i>L. monocytogenes</i>	<i>L. spp. unspecified</i>	<i>L. ivanovii</i>	Total positives
Alpacas	1	0	0	1
Birds (at farm)	0	3	0	3
Cattle	18	45	0	63
Sheep and goats	8	116	4	128
Voles	1	0	0	1
Total	28	164	4	196

Control measures

Prevention of listeriosis depends on good quality silage making and storage, and on reducing the potential soil intake in animals that are grazing or fed root crops.

Summary of risk

Direct transmission of listeria from animals to humans is very rare. Most human cases are associated with foodborne infection, as a result of environmental contamination and inappropriate storage of food.

Further information

Further information on listeriosis in humans is available from HPA at:
www.hpa.org.uk/Topics/InfectiousDiseases/InfectionsAZ/Listeria/

Further information on listeriosis in animals is available from Defra at:
www.defra.gov.uk/foodfarm/farmanimal/diseases/atoz/index.htm#listeriosis

Lyme Borreliosis (*Borrelia burgdorferi*)

Lyme borreliosis (also known as Lyme disease) is a spirochaetal infection caused by the bacterium *Borrelia burgdorferi* and is transmitted to humans and animals through the bite of an infected tick (*Ixodes* species). It is the most common tick-borne infection in the temperate northern hemisphere and has shown a steady increase within the UK since 2003. The majority of cases are indigenously acquired in the UK, often through recreational activities including walking, trekking and mountain-biking³⁵.

Diagnosis in humans is primarily clinical, especially in early infection. Well known regional foci of Lyme borreliosis include the New Forest, Salisbury Plain, Exmoor, the South Downs, parts of Wiltshire and Berkshire and Thetford Forest. Similar foci are known on the West coast and islands of Scotland. There are no notable foci of human infection in Wales or Northern Ireland.

In non-endemic areas, there may be lower awareness of Lyme borreliosis and the diagnosis may be overlooked, particularly when the patient experienced only transient or unrecognised exposure to ticks (e.g. on a holiday or day trip). Laboratory evidence should be sought to support the clinical diagnosis.

Lyme disease is notifiable in humans under public health legislation in Scotland.

Infection in humans

Symptoms: *B. burgdorferi* can spread via the bloodstream and lymphatic system to many tissues, and may migrate through the blood-brain barrier, evading the host's immune response. The earliest and most common manifestation of Lyme borreliosis is a rash spreading from the site of a tick bite (erythema migrans), but other more serious problems can occur. These include viral-like meningitis, non-specific flu-like symptoms with tiredness, headaches, arthralgia and myalgia. More serious complications may develop weeks or months later if an infected bite is left untreated, including temporary facial paralysis, pain, weakness or loss of sensation in the arms, legs or trunk and arthritis.

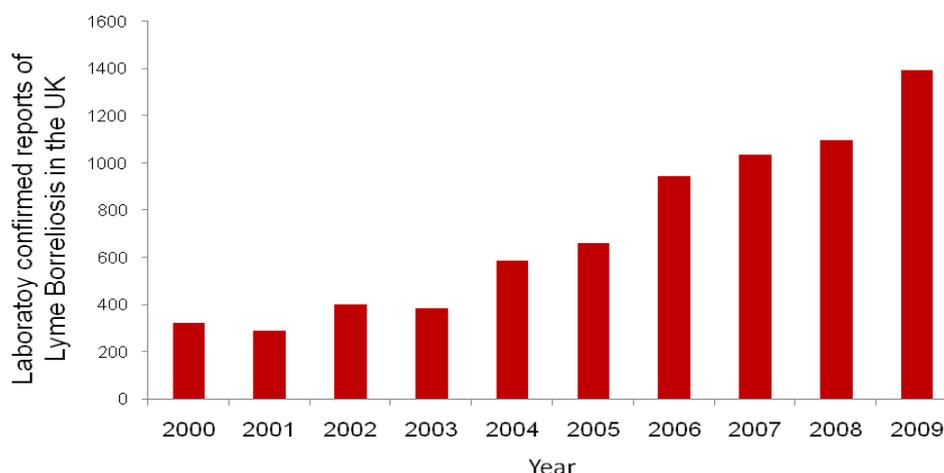
Symptoms resolve quickly with antibiotic treatment. Early recognition and treatment is important and helps to prevent more serious complications from developing.

Transmission: Lyme borreliosis in humans is caused by bites from infected ticks. Humans are incidental hosts and infections occur mainly in late spring, early summer and autumn; the peak periods for tick feeds. The annual incidence of Lyme borreliosis can vary, depending on climatic factors affecting tick population density and activity. There is strong evidence to show that both the prevalence of *B. burgdorferi* infected ticks and the incidence of Lyme borreliosis in Europe is highest in eastern countries and decreases westward across the continent, including the UK. Throughout Europe, neuroborreliosis is the most common complication of Lyme borreliosis.

³⁵ www.hpa.org.uk/NewsCentre/NationalPressReleases/2010PressReleases/100505Betickaware/

Cases: The number of reported cases of Lyme disease has risen in recent years. There were 1,395 serologically confirmed cases of *B. burgdorferi* infection in humans in the UK in 2009. This is a 27.0% increase on 2008 (Figure 5).

Figure 5: Number of laboratory confirmed human cases of Lyme borreliosis in the UK, 2000-2009



There were two laboratory-confirmed cases of Lyme borreliosis in Northern Ireland in 2009, compared to none in 2008³⁶. In Scotland there were 420 laboratory confirmations in 2009, this is a 47% increase on the 285 cases reported in 2008, consistent with the upward trend which began in 2005. Investigations are ongoing to establish the reasons behind this increase in reported cases. Of the 420 reports in Scotland, information on two cases suggests that their infection was acquired abroad.

In England and Wales, there were 973 cases of Lyme borreliosis in 2009. Of these, 173 (18%) cases are believed to have been acquired overseas, giving a UK acquired total population incidence of 1.79/100,000 population. The seasonal pattern in 2009 was consistent with previous years. Approximately 60% of patients were tested in July, August, September and October, representing a likely peak of onset of symptoms in the early summer. This is consistent with the major tick feeding period which occurs in the late spring and early summer months.

Table 10: Source of infection with Lyme borreliosis of cases resident in England and Wales, 2000-2009

Year	UK acquired	Overseas acquired	% overseas	Total
2000	246	76	23.6	322
2001	215	53	19.8	268
2002	269	71	20.9	340
2003	234	31	11.7	265
2004	425	75	15.0	500
2005	488	107	18.0	595
2006	677	91	11.8	768
2007	705	92	11.5	797
2008	722	91	11.2	813
2009	800	173	17.7	973

³⁶ The number reported in the 2008 report for NI was an error and has since been corrected.

Cases of Lyme borreliosis in residents of England and Wales continue to be diagnosed following overseas travel; these cases are mainly in holidaymakers visiting continental Europe (84%) and the US (16%).

Approximately two-thirds of indigenously-acquired infections were reported in residents of the southern counties of England (the South-West and South-East Regions, with population-specific rates of 3.95/100,000 and 4.85/100,000 respectively). The regional distribution of human cases reflects a similar regional pattern to that of the infected tick population.

Control measures

The prevention of Lyme disease in people relies upon avoiding tick bites in endemic areas. The use of protective clothing and the avoidance of heavily infested areas at key periods in the life cycle of ticks (spring to autumn) reduces the likelihood of being bitten. However, there are no effective controls for wildlife reservoirs.

Summary of risk

The number of indigenous cases of Lyme disease in people has risen over the last few years. No structured control programme is in place to reduce the level of either ticks or borreliosis in the UK. Tick control is not usually seen as a high priority for livestock keepers and therefore there is a risk that the distribution of disease from known foci could spread.

Further information

Further information on Lyme disease in humans is available from HPA at:
www.hpa.org.uk/Topics/InfectiousDiseases/InfectionsAZ/LymeDisease/

HPA Guidelines for health professionals is available at:
www.hpa.org.uk/Topics/InfectiousDiseases/InfectionsAZ/LymeDisease/Guidelines/lymGuidelines/

Lyme borreliosis; recent UK and European trends:
www.eurosurveillance.org/ViewArticle.aspx?PublicationType=W&Volume=11&Issue=25&OrderNumber=1

Pasteurellosis (*Pasteurella* spp.)

Pasteurellosis is a zoonotic bacterial disease that occurs sporadically worldwide. In humans *Pasteurella multocida* is the most commonly reported organism of the *Pasteurella* genus. The most common mode of transmission in humans is by cutaneous infection following dog or cat bites, scratches or licks. The organism is also found in the upper respiratory tract of many animal species including chickens, turkeys, cattle, rabbits and rodents.

Cases of infection with pasteurella are not notifiable in animals in the UK.

Infection in humans

Symptoms: The most common symptom of pasteurellosis in humans is a local wound infection, usually following an animal bite or scratch. Complications include abscesses, cellulitis and joint infections. The organism can also infect the respiratory tract and cause sinusitis and ear infections, and rarely more severe symptoms including pneumonia or lung abscesses in those with underlying pulmonary disease. Other uncommon presentations of *P. multocida* infection include cardiovascular infection, septicaemia, eye infections, meningitis, gastrointestinal problems and urinary tract infections. Very rarely, the infection is fatal. Five patients with severe pasteurellosis have died in the UK since 1993. The infection usually responds well to antibiotic treatment.

Transmission: Pasteurella infection is transmitted to humans through a bite from an infected animal. Cat bites are more likely to become infected with *P. multocida* than dog bites. Infections have been associated with a range of other animals including rabbits and cows. Person-to-person transmission of *P. multocida* has not been reported.

Cases: There were 560 laboratory-confirmed reports (from 559 cases) of human pasteurellosis in the UK in 2009, a 14.5% increase on the 489 cases reported in 2008.

Table 11: Laboratory confirmed reports of pasteurellosis in humans in the UK, 2009

Serovar	England and Wales	Scotland	NI	UK total
<i>P. multocida</i>	333*	62	5	400
<i>P. pneumotropica</i>	11*	6	0	17
<i>P. haemolytica</i>	5	0	0	5
<i>P. other named</i>	5	4	0	9
<i>Pasteurella</i> spp	102	25	2	129
Total	456	97	7	560

* Includes one person with dual infection

In 2009, 456 cases were reported in England and Wales (333 of which were *P. multocida*) compared to 436 (including 328 *P. multocida*) in 2008. One case had a dual infection (with *P. multocida* and *P. pneumotropica*). There were 97 cases reported in Scotland in 2009 (62 of which were *P. multocida*) compared to 57 (including 42 *P. multocida*) in 2008, an increase of 70%. Seven cases were reported

in Northern Ireland in 2009 (five of which were *P. multocida*) compared to only two cases (both *P. multocida*) in 2008.

Infection in animals

Clinical signs: In animals, pasteurella is both a common commensal organism and a pathogen. However, *P. multocida* may produce a wide range of clinical signs depending on the species; in rabbits *P. multocida* causes a flu-like illness and is sometimes referred to as ‘rabbit flu’, in birds and poultry it causes ‘fowl cholera’, in pigs atrophic rhinitis and respiratory disease, and in cattle, sheep, pigs, mice and rabbits it causes septicaemia. *P. pneumotropica* is a major cause of pasteurellosis in laboratory rodents including rats, mice, hamsters and guinea pigs. Infection, particularly in cats and dogs, can result in local sepsis or abscess, as in humans.

Frequency: There were 532 cases of pasteurellosis (*P. multocida*) diagnosed in animals in the UK during 2009. Of these, 311 occurred in GB, a small increase on the 281^a cases reported in 2008. Table 12 shows the number of reports per species in 2009 and 2008. The change in laboratory diagnoses is unlikely to reflect any change in the prevalence of animal infection, but is more likely a consequence of differing submission levels in the two years.

Table 12: Laboratory confirmed reports of *P. multocida* in animals in the UK, 2008-2009

Year	2008 GB	2008 NI	2008 UK	2009 GB	2009 NI	2009 UK
Cattle	142	81	223	171	145	316
Sheep	60	5	65	91	6	97
Goats	1	0	1	2	46	48
Pigs	63	21	84	38	22	60
Birds	9	2	11	6	0	6
Miscellaneous/ wildlife	6	4	8	3	2	5
Total	281^a	113	394	311	221	532

^a There has been an amendment to the 2008 figure printed in last year’s report as the data is derived from a dynamic database.

Control measures

Prevention of infection in people largely depends upon avoiding being wounded by cats and dogs. Advice on immediate post bite or cat scratch wound cleaning and treatment may help reduce infection.

There are a number of vaccines widely used against pasteurella in livestock, and occasionally antibiotics are used in the treatment of infection. These have little impact on the level of disease in humans.

Summary of risk

Infection in humans is generally unrelated to pasteurella in livestock. Zoonotic infection principally occurs through bites and scratches from cats and, to a lesser extent, dogs and is therefore dependent upon the level of cat and dog ownership and contact. This is relatively stable and so there is little indication that an increase in human infection is likely.

Further information

Further information on pasteurellosis in humans is available from HPA at:
www.hpa.org.uk/Topics/InfectiousDiseases/InfectionsAZ/Pasteurellosis/

Q Fever (*Coxiella burnetii*)

Q fever is caused by the bacterium *Coxiella burnetii*. For many years its aetiology was unknown, hence the name 'Query' (Q) fever. In its spore-like form the organism is very robust and resistant to desiccation and common disinfectants. It can survive for long periods in the environment and be transmitted in aerosols or by fomites, including dust particles.

Clinically, *C. burnetii* infection is seen mainly in domesticated ruminants (cattle, sheep and goats), which present the greatest zoonotic risk. However, infection is also present in a wide range of wildlife species and other animals including arthropod vectors (mainly ticks).

During 2007-2009 there was a very large human outbreak in the Netherlands linked to airborne spread from nearby, intensively farmed dairy goat herds^{37,38}. This significantly raised the profile of Q fever within the EU and stimulated interest in improving standards of diagnosis and surveillance in both animals and humans.

Currently in GB there is no statutory legislation for control of Q fever. It is not notifiable in animals, and specific screening for infection is not routinely undertaken. Abortion outbreak investigations are undertaken by microscopic examination of stained smears as part of routine abortion investigations in ruminants where a placenta is received. This has proven to be a reliable method of detecting the organism in recent cases. In Northern Ireland, Q fever is a designated organism under the Zoonoses Order (NI) 1991. If found during post mortem, the Agri-Food and Biosciences Institute (AFBI) will notify DARD, and an advisory letter which includes public health advice will be issued to the animals' owner.

Infection in humans

Symptoms: Most human infections are asymptomatic, but acute cases may present with a flu-like illness including fever, severe sweats, headache and malaise. An atypical pneumonia or abnormal liver function tests may also be found. Acute disease is rarely fatal, but chronic infections may be life threatening, and relapses may occur. Chronic Q fever, particularly endocarditis in patients with pre-existing cardiac valve defects, can develop following an acute infection which may or may not have been symptomatic. Immuno-suppression and pregnancy are associated with more severe disease and progression to chronic infection. Delays in diagnosis have an important effect on the prognosis of chronic Q fever.

Since 1999 an average of 16% of cases diagnosed annually are chronic³⁹ and the proportion has increased in recent years. Chronic infections require prolonged courses of treatment and possible surgical intervention. Death may result in 1-2% of

³⁷ Schimmer B, et al. (2009). Sustained intensive transmission of Q fever in the south of the Netherlands. *Euro Surveill*, **14**(19):pii=19210

³⁸ van der Hoek W, et al (2010). Q fever in the Netherlands: an update on the epidemiology and control measures. *Euro Surveill*, **15**(12):pii=19520

³⁹ HPA data

cases but is usually confined to immuno-compromised individuals or following chronic infection and sequelae⁴⁰.

Transmission: The greatest risk of exposure is at parturition when aerosols containing large numbers of *C. burnetii* are shed in the products of conception. Transmission to humans can also occur by exposure to aerosols arising from reproductive discharges, contaminated dust particles or bedding. Windborne infection by these routes can occasionally cause large outbreaks. Other reported but rarer routes of transmission include: drinking unpasteurised milk from infected animals; tick bites; and through cuts or abrasions. Farm workers, veterinarians, and abattoir workers have historically been at high risk of infection, however the source and route of transmission for most sporadic cases is not determined.

Direct person-to-person spread is very rare. Reported methods of transmission include respiratory, blood transfusion, and handling the products of conception from an infected pregnant woman.

Cases: In 2009, routine laboratory surveillance identified 15 cases in England and Wales, while two cases were reported in Scotland and two in Northern Ireland (Table 13). Enhanced surveillance data based on diagnoses from the two HPA reference laboratories in Bristol and Porton identified 32 cases, compared to 60 in 2008.

Table 13: Laboratory confirmed reports of Q fever in humans in the UK, 2008-2009

Year	Scotland	NI	England & Wales (Enhanced Surveillance ^{**})	UK total*	England & Wales (Routine Surveillance)
2008	1	11	60	72	37
2009	2	2	32	36	15

*The UK total includes the enhanced surveillance data but not routine surveillance data for England and Wales, as cases reported routinely may also be reported to enhanced surveillance

**Acute and chronic cases from the Enhanced surveillance database in England and Wales.

Infection in animals

Clinical signs: In cattle, sheep and goats, most infection is subclinical, however Q fever can cause sporadic abortions or outbreaks ("abortion storms") as well as other reproductive failures.

Frequency: A study in England and Wales in the 1990s using Enzyme-Linked Immunosorbent Assay (ELISA) tests for bulk tank milk samples suggested that about 21% of dairy herds were infected with *C. burnetii*⁴¹.

In 2009, the VLA undertook a structured serological survey of samples collected from sheep and goats in GB in 2008. Approximately 9.7% of sheep flocks and 2.8% of goat flocks were positive for *C. burnetii* but the within flock prevalence was much higher in goat herds (41.7%) compared with sheep flocks (2.2%), which may reflect the size of flocks and the intensive husbandry practices associated with goat farming in GB.

⁴⁰ Parker, N.R. et al (2006). Q Fever. *The Lancet*, **367**: 679-688.

⁴¹ Paiba, G. et al. (1999). Prevalence of antibodies to *Coxiella burnetii* (Q fever) in bulk tank milk in England and Wales. *The Veterinary Record*.

In 2009, two cattle and one goat were clinically diagnosed with Q fever from abortion specimens submitted to VLA and SAC laboratories in GB.

Control measures

The prevention and control of Q fever in animals is problematic and principally relies on hygiene and husbandry methods on farms. Careful disposal of placenta and material contaminated with birth products reduces the risk of spore formation and subsequent infection of animals and people.

Pasteurisation of milk and milk products prevents foodborne transmission of Q fever to humans.

Summary of risk

C.burnetii is considered to be endemic and widespread in the UK. The organism is highly resistant to disinfectants and can persist in a spore-like form for several months or years in the environment. It is therefore difficult to eliminate *C.burnetii* and the risk of infection from a farm or area. It is therefore mainly a risk to occupationally exposed groups such as livestock farmers and vets, but large outbreaks can occasionally occur amongst the general population from windborne aerosol spread from infected ruminant farms.

Further information

Further information on Q fever in humans is available from HPA at:

www.hpa.org.uk/Topics/InfectiousDiseases/InfectionsAZ/QFever/

Information on Q fever infection risks during the lambing season are available at:

www.hpa.org.uk/Topics/InfectiousDiseases/InfectionsAZ/QFever/GeneralInformation/qfevQFeverRisksLambingSeason/

Q fever information for farmers is available at:

www.hpa.org.uk/web/HPAwebFile/HPAweb_C/1210834106356

Rabies (*Rhabdoviridae*) – including Bat rabies (European Bat Lyssavirus (EBLV))

Rabies is an acute viral infection of the central nervous system, caused by a lyssavirus in the family *Rhabdoviridae*. It affects all mammals, including humans, cats, dogs, wildlife and farm animals. The disease is absent from land mammals in the UK. The last case of rabies in an animal outside of quarantine in GB was a dog in Camberley in 1970. In Northern Ireland the last case was reported in 1923. The last case of rabies in quarantine was reported in 2008 in England.

The last case of human terrestrial rabies acquired in the UK was in 1902, however occasional travel-related cases do occur. Worldwide it is estimated that there are around 50-70,000 rabies cases in humans each year, predominantly occurring in less developed countries.

In 2002, it was recognised that UK bats carry European Bat Lyssavirus 2 (EBLV-2), a genetically-similar strain of rabies virus. Further information can be found in the European Bat Lyssavirus (bat rabies) section.

The suspicion of rabies in humans or animals on the basis of clinical appearance and/or behaviour must be notified to the relevant government departments so that further investigations can be undertaken.

Infection in humans

Symptoms: The incubation period of the disease is generally between 3 - 12 weeks but in rare cases can be several years. Early symptoms may include numbness or tingling around the bite site, fever, headache, loss of appetite, tiredness and weakness. Later, stimulation may provoke violent and painful muscle spasms and can lead to extreme fear of water due to involuntary contraction of the muscles involved in swallowing and respiration. Hallucinations or manic behaviour may alternate with periods of full lucidity. The disease progresses to paralysis and coma. Death is almost inevitable, with very few documented survivors⁴².

Transmission: The virus is present in the saliva of affected animals, and the most frequent method of transmission is by bites, scratches or licks to broken skin or mucous membranes. Dogs are the most common source of infection worldwide. Bats carrying classical rabies have also been reported as a source of human infection in the Americas. Person-to-person transmission via saliva has not been substantiated. Other rarer routes of transmission include the transplantation of organs or tissues from infected patients, and consumption or handling of raw infected material.

Cases: There were no human cases of rabies infection in the UK in 2009.

In the last 10 years there have been four cases of imported human rabies in the UK (from Nigeria, Philippines, India and South Africa). The most recent case of human

⁴² Jackson AC. (2010). Why does the prognosis remain so poor in human rabies? *Expert Rev. Anti Infect. Ther.* **8**(6), 623-625

rabies in the UK was in December 2008, and resulted from a dog bite in South Africa two years earlier⁴³.

Individuals potentially exposed to rabid animals require post-exposure prophylaxis, which places a significant demand upon health services in countries where the disease is endemic. Over 800 people each year require post-exposure prophylaxis in England and Wales following potential exposure in endemic countries⁴⁴.

Infection in animals

Clinical signs: The clinical course of rabies in animals is similar to that in humans. Three forms are classically described; prodromal, excitement (furious) and paralytic (dumb). The first signs are usually the non-specific signs typical of viral encephalitis (e.g. behaviour changes), which may or may not be accompanied by signs of neuropathic pain at the site of infection. Subsequently, either or both the excitement and/or paralytic forms of the disease may be observed depending upon the species affected, although the disease most commonly progresses to the paralytic form. Clinical signs of rabies are rarely definitive so diagnosis requires the examination of brain tissue post-mortem.

Frequency: In 2009, nine cats, 14 dogs and two foxes were submitted to the VLA for laboratory testing. None of the samples were positive.

Control measures

Rabies is not found in animals in the UK. Protection of public and animal health depends upon the continued exclusion of rabies through risk-based border controls, vaccination and advice to travellers. Control and eradication plans are in place in the event of rabies returning in animals to the UK⁴⁵.

The Pet Travel Scheme (PETS) was launched in 2000 to allow people to bring in or travel with their pets (dogs, cats and ferrets), while ensuring the UK remains free from rabies and certain other exotic diseases. During 2009, 7,128 cats, 89,376 dogs and 55 ferrets entered the UK under the PETS. In total, 662,499 pet animals have entered the UK under PETS since 2000 (ferrets have only been able to enter under the scheme since July 2004), however there have been no cases of rabies in the animals.

The annual government veterinary expenditure on rabies and EBLV is estimated to be over £5 million. This includes surveillance, research and field delivery services in wildlife, and monitoring the operation of quarantine facilities and disease control actions in the face of a suspect case. There are also costs associated with policy development occurring at the EU level and with Border Inspection Post inspections and import controls undertaken by Animal Health and HM Customs.

⁴³ Hunter, M. et al (2010). Immunovirological correlates in human rabies treated with therapeutic coma. *Journal of Medical Virology* 82; 1255-1265.

⁴⁴ Unpublished HPA data

⁴⁵ www.defra.gov.uk/foodfarm/farmanimal/diseases/atoz/rabies/index.htm

Vaccination is available for travellers to rabies endemic areas and is advised where contact with animals in these areas is likely.

Summary of risk

The risk of human infection principally arises when travelling to endemic areas and is mitigated by the use and availability of vaccination and advice on prevention.

Rabies is unlikely to enter the UK through any legal route due to the implementation of controls including the PETS and quarantine of dogs and other species that enter from countries considered to have a higher risk. Rabies has the potential to spread between dogs and to become established and endemic in the fox population if introduced to the UK. However, the availability of effective vaccines, continued surveillance, and limited routes of transmission mitigate this risk⁴⁶.

Further information

Further information on rabies in humans is available from HPA at:

www.hpa.org.uk/Topics/InfectiousDiseases/InfectionsAZ/Rabies/GeneralInformation/

Further information on rabies in animals is available from Defra at:

www.defra.gov.uk/foodfarm/farmanimal/diseases/atoz/rabies/index.htm

Further information on PETS and the conditions of the scheme can be found at:

www.defra.gov.uk/wildlife-pets/pets/travel/pets/index.htm

⁴⁶ Banyard, A.C. et al (2010). Reassessing the risk from rabies: A Continuing Threat to the UK? *Virus Research*. **152**(1-2); 79-84.

Bat rabies (European Bat Lyssavirus (EBLV))

European Bat Lyssaviruses (EBLVs) 1 and 2, commonly referred to as 'bat rabies', are found in bats in Europe. EBLVs have been known to infect other animals and humans, presumably through a bite or scratch from an infected bat. Since 1977, there have been five human deaths in Europe (three confirmed, two possible) from EBLVs. In all cases the person had been bitten or scratched by bats and had not received rabies vaccination either before or after the incident⁴⁷.

Confirmed cases of EBLV in animals are notifiable to Animal Health.

Infection in humans

Symptoms: Symptoms of EBLV in humans are similar to those caused by the rabies virus (see Rabies section).

Cases: The only case of EBLV-2 in a human in the UK was in 2002 when a bat handler was infected following a bite from a Daubenton's bat (*Myotis daubentonii*) in Scotland.

Infection in animals

Frequency: A three year targeted surveillance programme for testing bats for EBLV in England took place between 2003-2006⁴⁸. Surveillance focused on the two species of bat most likely to host these viruses: Daubenton's (*Myotis daubentonii*) and Serotine (*Eptesicus serotinus*) bats. Results indicated a low seroprevalence estimate of EBLV-2 of about 2.2% in the 363 Daubenton's bats tested, and antibodies against EBLV-1 were found in only one of 273 Serotine bats surveyed.

In 2009, the targeted seroprevalence of EBLV in Scottish bats has continued an annual decline to 2.5% from a high of 15% in 2005.

Over 1000 dead bats from GB were submitted to the VLA as part of an ongoing passive lyssavirus surveillance scheme. One tested positive for EBLV-2⁴⁹, bringing the total to nine bats, all Daubenton's (Table 14).

⁴⁷ Note that bats are a protected species and should only be handled by licensed persons. It is advised that any member of the public finding a bat behaving abnormally, found in an unusual place or under unusual circumstances, should not attempt to handle or move the animal, but should contact their local bat conservation group or the Bat Conservation Trust.

⁴⁸ Harris, S.L. et al. (2009). Targeted surveillance for European bat lyssaviruses in English bats (2003-06). *J Wildlife Disease*, **45**(4):1030-41.

⁴⁹ Brookes, S.M. et al. (2005). European bat lyssavirus in Scottish bats. *Emerging Infectious Diseases* **11**(4): 572- 578.

Table 14: Isolations of EBLV-2 in bats in the UK (all were *M. daubentonii*)

Date	No. isolations	Location	Sex
1996	1	Newhaven, East Sussex	Adult female (Pregnant)
2002	1	Carnforth, Lancashire	Juvenile, Female
2004	2	Staines, Surrey Blackburn Lancashire	Juvenile, Female Adult male
2006	1	Abingdon, Oxon	Juvenile Female
2007	1	Stokesay Castle, Shropshire	Adult Female
2008	2	Teddington, Surrey Stokesay Castle, Shropshire	Adult Female Juvenile, Male
2009	1 ⁵⁰	Linlithgow, West Lothian	Adult Female

Control Measures

There are no control measures available for the prevention of EBLV in bats. Pre-exposure immunisation is advisable and free for all licensed bat handlers.

If a person is bitten, scratched, or there is direct contact with a bat, the area should be cleaned thoroughly with water and soap and they should seek medical advice regarding vaccine and human rabies immunoglobulin.

Summary of risk

The risk principally applies to those who handle bats. Given that there are no control measures available, pre-exposure immunisation and post-exposure prophylaxis are the mainstays of public health protection.

Further information

General information including guidance on post exposure prophylaxis is available from the HPA:

www.hpa.org.uk/Topics/InfectiousDiseases/InfectionsAZ/Rabies/

Advice for bat workers and their GPs can be found at:

www.hpa.org.uk/web/HPAwebFile/HPAweb_C/1194947347180, and
www.snh.org.uk/pdfs/species/BatsBuildings.pdf

General information on bats is available from Scottish Natural Heritage:
www.snh.gov.uk/about-scotlands-nature/species/mammals/land-mammals/bats/

Information on identifying bat species and their distribution in Britain is available from Scottish Natural Heritage:

www.snh.org.uk/publications/on-line/wildlife/batsandpeople/identify.asp

Information on bats is available online from the Bat Conservation Trust at:
www.bats.org.uk

⁵⁰ Horton, D.L., et al. (2009). European bat lyssavirus in a Daubenton's bat from Scotland. *The Veterinary Record* **165(13)**; 383-4.

Further information on rabies and EBLV is available from Defra at:
www.defra.gov.uk/foodfarm/farmanimal/diseases/atoz/rabies/#bats;

Results of the Scottish Natural Heritage bat lyssavirus monitoring programme:
www.snh.org.uk/press/detail.asp?id=2104

Salmonellosis (*Salmonella* spp)

Salmonellosis is a worldwide disease of animals and humans caused by *Salmonella enterica*. All animals can carry salmonella and the species is subdivided into over 2,600 serovars.

S. Typhi and *S. Paratyphi* are human host adapted serovars and are not associated with zoonotic transmission. The disease caused by *S. Typhi* and *S. Paratyphi* is known as “enteric fever”. The illness associated with the other serovars of *S. enterica* is known as non-typhoidal salmonellosis. The two non-typhoidal serovars, *S. Enteritidis* and *S. Typhimurium* account for 60 – 80% of all human salmonellosis. The sections below summarise the salient features of these non-typhoidal infections.

In domestic animal species, clinical cases of salmonellosis are most common in cattle. Subclinical carriage is most common in pigs, poultry and reptiles.

Infection in humans

Symptoms: Non-typhoidal salmonellosis is characterised by diarrhoea, abdominal pains, vomiting and fever. Illness usually lasts for three to five days although symptoms may persist for longer in some cases. Infants, the elderly and immunocompromised are particularly at risk and rarely, cases may develop longer term sequelae such as reactive arthritis. Septicaemia and death may occur in these at-risk groups.

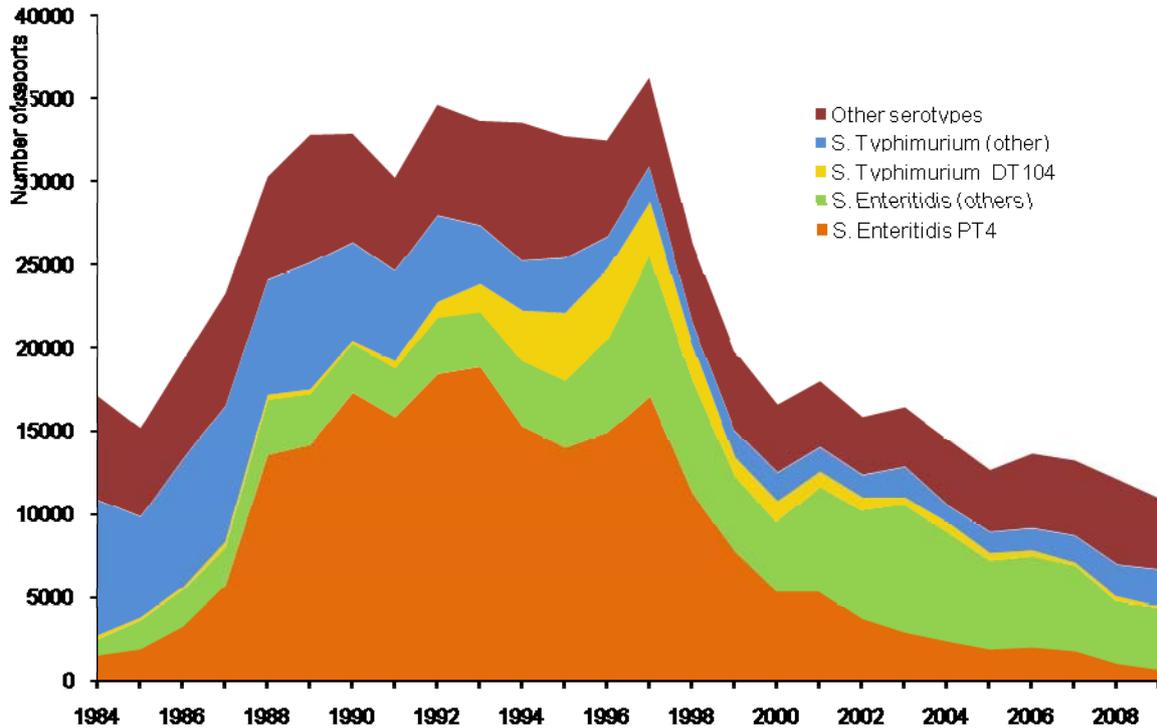
Transmission: Most salmonellosis is acquired via the foodborne route. The most common vehicles of infection are eggs, chicken, other meats and dairy products. Outbreaks have also been associated with a wide range of sources including salad vegetables and herbs which can become contaminated as a result of irrigation with untreated or poorly treated water, the activities of wildlife or faults in the production or distribution chain.

Cases: In 2009, 10,071 cases of laboratory confirmed salmonellosis were reported in the UK. This continues the ongoing decline in salmonellosis in humans dating back to the late 1990’s (Figure 6). The 2009 UK figure represents a fall of 9.1% from 2008^a and for England and Wales, Scotland and Northern Ireland falls of 7.9%, 19.1% and 14.5% respectively. However, for every laboratory-confirmed report of disease caused by salmonella made to the national surveillance scheme, there are estimated to be approximately three unreported cases in the community⁵¹. This means the total number of cases in the UK in 2009 could be as high as 40,000.

⁵¹ Wheeler, J.G. et al (1999). Study of infectious intestinal disease in England: rates in the community, presenting to general practice, and reported to national surveillance. *The Infectious Intestinal Disease Study Executive, BMJ*. **318**:1046-50.

^a There has been an amendment to the 2008 figure printed in last year’s report as the data is derived from a dynamic database.

Figure 6: Laboratory reports of human salmonella cases in the UK, 1984-2009



S. Enteritidis remained the most commonly reported serovar in 2009, accounting for 39% of cases, although S. Enteritidis PT4 reports fell by over a third between 2008 and 2009, to 640 cases (Table 15). S. Typhimurium was the second most commonly reported serovar and only decreased by 0.8% from 2008. Reporting shows a consistent seasonal pattern with a distinct peak of infection observed in the third quarter of the year.

Table 15: The number, rates per 100,000 population and change since 2008 of the most common laboratory confirmed *salmonella* serotypes isolated from people in UK in 2009

Serotype	England & Wales		Scotland		NI		UK	
	Cases	Rate	Cases	Rate	Cases	Rate	Total	Change from 2008
S. Enteritidis	3,834	7.00	265	5.10	44	2.46	4,143	-10.8%
S. Enteritidis PT4	581	1.06	54	1.06	4	0.22	640	-36.2%
S. Typhimurium	1,855	3.37	137	2.64	54	3.02	2,046	-0.8%
All	9,100		846		153		10,099	-8.8%

Salmonella in food

In GB in 2009, there were 41 food vehicles implicated in 28 outbreaks of salmonellosis. There were no such outbreaks in Northern Ireland. The most frequently implicated foodstuffs were composed of mixed ingredients, in which the actual source of infection could not be differentiated, followed by poultry meat and eggs (Table 16). However, more than one food vehicle can be implicated for any one outbreak.

Table 16: Food vehicles associated with outbreaks of salmonella, GB 2009

Food Vehicle Category	England & Wales	Scotland
Poultry meat	9	1
Red Meat	2	1
Vegetables & fruits	3	0
Condiments & sauces	2	0
Desserts, cakes & confectionary	3	0
Rice	2	0
Eggs & egg dishes	6	0
Composite/ Mixed foods	10	0
Unknown	2	0
Total	39	2

A one year long, UK-wide LACORS and HPA survey, completed in March 2009, confirmed the presence of salmonella in 3 (0.13%) of 2,359 samples of ready-to-eat speciality meats from market stalls and specialist retailers. A second 6-month long survey also completed in March 2009, confirmed 3 (0.10%) of 2,886 ready-to-eat nut kernel samples of different varieties were contaminated with salmonella.

Infection in animals

Of all the incidents of salmonella in farm livestock in UK, only a proportion is responsible for outbreaks of clinical disease. The majority of incidents reported from animals during the year are detected as a result of statutory surveillance under legislative programmes to control salmonella in flocks of domestic fowl, or voluntary disease surveillance activity, mostly within the poultry industry sector.

Salmonella organisms are ubiquitous. Prevalence in farmed livestock is dependent on the animal species, production type and stage of production. Transmission of most salmonellae occurs readily between livestock kept in groups. In many cases, carriage of salmonella in groups of farm animals resolves spontaneously, but cycling of infection between different groups of animals may prolong the persistence of infection. This effect can be reduced by the use of systems that enable periods of being stock-free, known as “all in-all out”.

European legislation (Directive 2003/99/EC and Regulation (EC) No 2160/2003) requires the implementation of Salmonella National Control Programmes (NCPs) to reduce the prevalence of salmonellas of public health importance in livestock. The chicken broiler NCP was implemented at the beginning of 2009 and sets out the monitoring and controls that producers must follow to reduce and/or control the prevalence of *S. Enteritidis* and *S. Typhimurium* to 1% or less by 31 December 2011. Thus far, NCPs have been implemented for breeding chickens (2007), laying chickens (2008) and fattening (broiler) chickens (2009). The results of the active scanning surveillance undertaken in these NCPs cannot be directly compared with the less structured surveillance undertaken in species for which there is no NCP. More information on the NCPs is available in the 2008 Zoonoses Report.

Currently, approximately 90% of pigs are produced under an assurance scheme that includes a programme to reduce the level of salmonella in pigs - the Zoonosis National Control Programme for salmonella in pigs (ZNCP).

Feedstuff contaminated with salmonella may be a source of infection for animals. In order to reduce this risk, salmonella is monitored and controlled, according to guidelines described in Codes of Practice, at a number of points in the feed production process.

Clinical signs: In animals, clinical signs of salmonellosis range from symptomless carriage to enteritis, septicaemia, abortion, pneumonia (in calves) and death. Fowl Typhoid and Pullorum disease also cause severe illness and reduced egg hatching ability in breeding poultry and game birds, although these two *Salmonella* serovars (*S. pullorum* and *S. gallinarum*) are not zoonotic pathogens.

Frequency: Animal data for salmonella is usually reported as “incidents” rather than the total number of isolates, since multiple isolates may be obtained from a number of samples taken simultaneously from a premises, group or animal environment. An incident is defined as the first isolation and all subsequent isolations of the same serovar (or serovar and phage type combination) of a particular salmonella from an animal or epidemiologically distinct group of animals occurring on a single premises, usually within a 30 day period. Changes in the number of incidents also need to be treated with caution in view of the inherent biases associated with data collection.

Cattle, sheep and pigs

In GB in 2009, there was a 25% increase in the number of reported incidents in cattle to 764 compared to 2008 (Figure 7). The main factor was an increase in *Salmonella* Dublin following increases in liver fluke populations after the wet summers of recent years and an increase in cases of *Salmonella* Mbandaka and Montevideo; thought to be linked with contamination of soya bean residue meal during processing to extract vegetable oil. In contrast, there was a 13% decline in the number of reported incidents in sheep (125) and no change in the number of incidents in pigs (182) over the same period.

In Northern Ireland there were 122 confirmed isolates in cattle, 18 in pigs and 10 in sheep in 2009.

Figure 7: Number of laboratory-confirmed incidents of salmonella in animals in GB, 1998- 2009

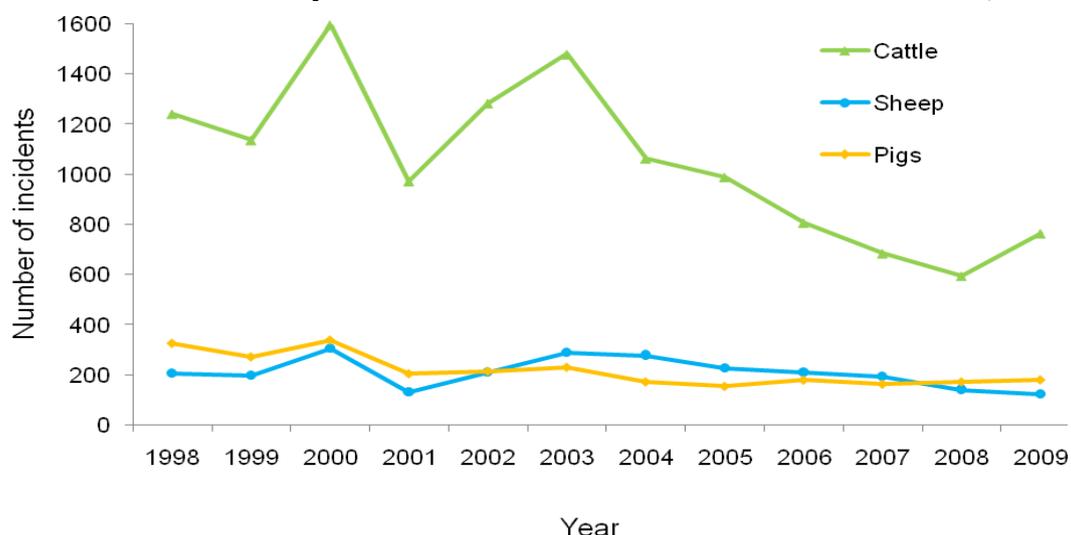


Table 17: Proportion of cattle, sheep and pig incidents by *Salmonella* serovar in the UK, 2009

Species	Cattle	Sheep	Pigs
Serotypes	% of total	% of total	% of total
S. Dublin	69.9	10.4	-
S. Typhimurium	7.7	3.0	70.0
S. Mbandaka	7.0	-	-
S. Montevideo	3.2	11.1	-
S. Anatum	2.4	-	-
S. <i>enterica</i> subsp <i>enterica</i> 4,5,12:i:-	1.6	-	6.0
S. <i>enterica</i> subsp <i>diarizonae</i> 61:k:1,5,7	-	44.4	-
S. <i>enterica</i> subsp <i>diarizonae</i> 61:-:1,5,7	-	16.3	-
S. Newport	0.9	-	-
S. Ohio	0.6	-	-
S. Kottbus	0.3	-	-
S. Derby	-	-	4.0
S. Rissen	-	-	3.5
S. Kedougou	-	-	3.0
S. Reading	-	-	2.0
Other	6.5	14.8	11.5
Total	886	135	200

S. Dublin, which is seldom associated with human foodborne infection, continues to account for 2 out of 3 incidents in cattle, whilst nearly 2% of incidents were caused by monophasic S. Typhimurium; S. 4,5,12:i:- (Table 17). S. *enterica* subspecies *diarizonae* 61:k:1,5,(7), which is also not common in humans, was the most frequently reported serovar in sheep. In pigs, S. Typhimurium was the most commonly recorded serovar, accounting for 70% of incidents, whilst S. 4,5,12:i:- represented 6% of incidents.

Poultry

Chickens

Management of the disease in chickens is largely carried out by specialist poultry practitioners. Consequently, most clinical and laboratory diagnoses are not undertaken by government laboratories, and such figures are therefore not representative of the wider chicken industry. However, the results of the NCPs testing do provide a representative view of the salmonella status of the poultry flocks within them. The NCP results are provided below where available.

Breeding sector: The UK chicken breeding sector is now largely free of serovars of public health importance. During 2009, a total of 1,637 adult breeding flocks were subject to at least one Official Control Sampling. Two samples were positive for *S. Typhimurium* giving an estimated prevalence of 0.12%. This is well below the European Community target of 1%.

Laying flocks: In 2009, a total of 4,466 adult laying flocks were subject to official testing in the UK. Sixteen flocks were found to be positive for *S. Enteritidis* and/or *S. Typhimurium* giving an estimated prevalence of 0.36%. This was an approximate two-thirds reduction compared with 2008, and indicates that substantial progress continues to be made in controlling salmonella in the layer sector.

Broiler flocks: During 2009, the first year of the broiler chicken NCP, approximately 27,780 flocks were tested. Of these, 10 flocks were positive for *S. Enteritidis* and two were positive for *S. Typhimurium*. This gave an estimated UK prevalence of 0.043% for the designated *Salmonella* serovars in broiler flocks, well below the European Community target of 1%.

Turkeys

There were 71 reported disease incidents in turkeys in GB in 2009, a 25% increase from 2008, the majority of which were detected as a result of voluntary monitoring. There was one incident of *S. Typhimurium*, a reduction from 12 in 2008 which may have resulted from the effective application of control measures prior to the start of the NCP for turkeys in 2010. There were no cases of *S. Enteritidis* in turkeys in 2009. No incidents were reported in Northern Ireland in 2009.

Ducks/geese

In GB, there was a 9% increase in reports of salmonella from ducks compared to 2008, to 303, although *S. Enteritidis* (n=1) and *S. Typhimurium* (n=9) were not commonly reported. No incidents were reported in Northern Ireland in 2009.

Animal feed surveillance for salmonella

The isolation rate of salmonella from animal feedstuff and feedstuff ingredients in GB has continued to remain stable. In GB in 2009, 0.7% of samples were positive (310 salmonella isolates from 39,647 samples). Approximately 4.8% of these isolates were of salmonella serovars considered to be of greatest public health significance (i.e. *S. Typhimurium*, *S. Enteritidis*, *S. Hadar*, *S. Virchow*, *S. Infantis*). Salmonella was isolated from 0.8% of processed animal protein for feedingstuffs use, and from

2.0% of premises that process this material. The isolation rates in oilseed meals and mineral (and other) ingredients were 0.9% and 0% respectively.

Control measures

Control measures to prevent human infection are principally related to the clean preparation and minimisation of contamination of food and the application of personal hygiene, especially washing of hands prior to eating, when in an environment potentially contaminated with animal faeces.

Controls in animals depend on the species and production sector in question. Vaccination is widely used in broiler breeders and laying hens, and occasionally in response to infection in cattle. The majority of the table egg industry either operates to a Code of Practice which requires commercial flocks of laying hens to be vaccinated against salmonella, or uses vaccination voluntarily. Farm biosecurity and management, including pest control, particularly for rodents, is very important.

Where an NCP is in place, control measures are specified. Particular measures of note include the mandatory culling of breeding flocks of chickens and turkeys and movement restrictions on hatching eggs if regulated serovars are detected. Eggs from such flocks are required to be pasteurised prior to human consumption. These measures aim to prevent the transmission of salmonella down the production pyramid to commercial flocks.

Contaminated feed is a major risk for the introduction of salmonella to livestock and so effective selection of ingredient sources, mill hygiene control and effective heat/chemical treatment of feedstuffs are all important control measures that are widely practiced.

Total UK government expenditure on monitoring of salmonella and antimicrobial resistance in salmonella, implementation of the NCPs and research projects is more than £5 million per year.

Summary of risk

Salmonella is present in some animals and the environment. Indeed, under optimum conditions it can persist in the environment for long periods of time with a high potential for silent spread, especially in pigs and poultry. Therefore, infection through contaminated food and the environment remains an ongoing risk to humans. New serovars of salmonella periodically result in waves of infection that run through animal and, sometimes, human populations.

The rate of spread on and between infected premises can be high. Wild birds, rodents and other wildlife species may act as reservoirs or vectors for infection. Effective monitoring, including the identification of serovars, phage types and resistance patterns of strains is therefore essential for determining routes of infection and confirming the effectiveness of control measures.

Further information

The 2009 LACORS/HPA survey reports can be found at:

www.hpa.org.uk/Topics/InfectiousDiseases/InfectionsAZ/FoodSampling/HPALacorsReports/

A description of salmonella data collection and reporting in animals is included in the Salmonella in Livestock Report:

www.defra.gov.uk/vla/reports/rep_salm_rep09.htm

The results of mandatory baseline prevalence surveys in laying hen flocks (2004-2005), turkey flocks (2006-2007), slaughter pigs (2006-2007), breeding pigs (2008) and broiler carcasses (2008-2009) have been reported in previous editions of the UK Zoonoses Report:

www.defra.gov.uk/foodfarm/farmanimal/diseases/atoz/zoonoses/reports.htm

Details of the UK NCPs can be found at:

www.defra.gov.uk/foodfarm/farmanimal/diseases/atoz/zoonoses/ncp.htm

Further background to the pig ZNCP initiative is available at the British Pig Executive's website: www.bpex-zap.org.uk

Industry Codes of Practice for the monitoring and control of salmonella in animal feedstuffs can be found at:

www.defra.gov.uk/foodfarm/farmanimal/diseases/atoz/zoonoses/salmonella-cop.htm

Toxoplasmosis (*Toxoplasma gondii*)

Toxoplasmosis is caused by the protozoan parasite *Toxoplasma gondii*. The organism is carried by cats and can infect virtually all warm-blooded animals. The resistant oocysts are excreted by cats in their faeces, and can survive in the environment for many months.

Toxoplasmosis infection is notifiable in humans in Scotland.

Infection in humans

Symptoms: Most healthy people who acquire toxoplasma infection do not experience any symptoms, however about 10% of people develop a mild to moderate self-limiting flu-like illness. Lymphadenopathy is the most common manifestation, lasting up to 4-6 weeks, but fatigue, sore throat, myalgia, rashes, arthralgia or hepatosplenomegaly may occur. In rare cases these symptoms may persist due to chronic active infection. In immunocompromised individuals (such as HIV-infected, those with leukaemia, or recipients of organ transplants), primary or reactivated infection may cause a maculopapular rash, muscle aches, chorioretinitis, or cerebritis.

After an initial infection, people develop immunity. However, the parasite commonly establishes a long-lived "dormant" infection in the form of tissue cysts and, if the immune system subsequently becomes compromised, the infection may re-activate.

If a pregnant woman becomes infected with *T. gondii* for the first time during the first trimester, the foetus has approximately a 25% chance of becoming infected⁵². The foetus may then suffer serious damage to the brain or other organs, abortion or stillbirth. The brain damage may result in cognitive and learning impairments or physical abnormalities such as hydrocephalus ("water on the brain"). Some infected children develop eye damage from the parasite, which may not be immediately apparent.

Transmission: Humans are infected with *T. gondii* by four major routes:

- Ingesting oocytes from water, food or soil contaminated with the faeces of infected cats
- Ingesting or handling undercooked or raw meat (mainly pork or lamb) that contains tissue cysts
- Transmission from a newly infected mother to the foetus
- Receiving organ transplants or blood products from donors with toxoplasmosis, although this is rare

Apart from mother to foetus, direct person-to-person transmission has not been reported.

⁵² Cook, G. C. & Zumla, A. (2003). *Manson's Tropical Diseases*, 21st Edition.

Higher rates of infection have been found in occupational groups where increased exposure might be expected⁵³ and studies have shown that up to a third of the UK adult population has evidence of past infection⁵⁴.

Toxoplasma cysts are believed to be widely disseminated in the environment. To minimise the risk of infection at a critical time, pregnant women are advised to wear gloves when gardening, to avoid handling cat litter trays, and to wash their hands afterwards. They should also avoid assisting with lambing or handling aborted lambs.

Cases: A total of 158 laboratory-confirmed cases of toxoplasmosis were reported in the UK during 2009, 69 of which were reported in Scotland (Table 18).

Under-reporting is known to occur within the laboratory reporting system and an enhanced surveillance system was introduced by the HPA in England and Wales in 2008 in collaboration with the national Toxoplasma Reference Laboratory in Swansea. In 2009, 422 cases of toxoplasmosis were reported through this scheme; 191 (45.3%) of these cases were male and 215 (50.9%) were female (16 were reported with unknown gender), and 286 cases had acute infection (67.8%).

Table 18: UK confirmed human cases of toxoplasmosis, 2000-2009

Year	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009
England & Wales Laboratory reporting	103	106	94	75	79	102	94	106	75	86
Scotland	20	16	32	17	20	11	33	44	56	69
NI	5	7	12	7	1	2	0	2	0	3
Total	128	129	138	99	100	115	127	152	131	158
England & Wales Enhanced surveillance*	NR	NR	NR	NR	NR	NR	NR	NR	407	422

* Figures for enhanced surveillance are not included in the total

Infection in animals

Clinical signs: Clinical signs in sheep and goats depend upon the timing of infection. In non-pregnant animals, the infection is almost always inapparent, whilst in pregnant animals toxoplasma infection usually causes abortion. Infection in the final month of pregnancy may result in weak or infected, but clinically normal, lambs.

Frequency: In 2009, exposure to toxoplasma was confirmed in approximately 49% of diagnostic sheep sera sampled in the UK (Table 19).

⁵³ Thomas, D.R. et al. (1999). Occupational exposure to animals and risk of zoonotic illness in a cohort of farmers, farmworkers and their families in England. *J Agricultural Safety and Health*, **5**(4):373-382.

⁵⁴ Walker, J. et al (1992). Longitudinal study of toxoplasma seroprevalence in South Yorkshire. *Epidemiol Infect.*, **108**:99-106.

Table 19: Sera testing of Toxoplasmosis in animals in the UK, 2009

Sera testing	GB	NI	UK
No. sheep samples sera tested	732	552	1284
No. separate submissions*	174	234	408
No. positives <i>T. gondii</i>	321	303	624
No. goat samples sera tested	9	10	19
No. separate submissions	4	5	9
No positives <i>T. gondii</i>	2	5	7
No. alpaca samples sera tested	10	0	10
No. separate submissions	5	0	5
No. positives <i>T. gondii</i>	1	0	1
No. pig samples sera tested	10	6	16
No. separate submissions	3	6	9
No. positives <i>T. gondii</i>	1	2	3

*Each submission may contain a number of samples

In 2009, toxoplasmosis remained the second most commonly diagnosed cause of abortion in sheep in GB, accounting for 23.1% of all incidents of fetopathy investigated by government veterinary laboratories. Toxoplasmosis was also confirmed as the cause of abortion in one goat in 2009.

Seroprevalence in cats increases with age although, due to acquired immunity after initial infection, it is mainly young kittens that excrete oocysts. Infected cats usually excrete the organism for about ten days, during which time they may shed several million oocysts⁵⁵. Local bird and rodent populations are reservoirs of infection and prey for cats.

Control Measures

Prevention of human infection depends upon avoiding the ingestion of oocysts and is particularly important for pregnant women and those with underlying disease. This is achieved by applying hygiene precautions when handling sheep and their bedding, particularly around lambing time and if sheep are aborting. Precautions should also be taken when handling cat faeces, gardening, and handling and eating raw meat.

There are no statutory control measures for toxoplasmosis and control is therefore the responsibility of the animal's owners. Control in animals is principally achieved by interrupting the parasite's lifecycle through the application of on-farm biosecurity measures such as avoiding contamination of sheep and goat feed by making feed stores cat-proof and the hygienic disposal of abortions and birth products. Licensed vaccines against *Toxoplasma*, and the feeding of anti-protozoal agents to pregnant animals, are widely used in the UK for the prevention of abortions in sheep.

⁵⁵ Tenter, A.M. (2000) *Toxoplasma gondii*: from animals to humans. *International Journal for Parasitology*. **30**:1217-1258.

Summary of risk

Infective oocysts are widely disseminated in the environment, particularly in sheep farming areas. Thus, the risk of human infection in these areas is principally from direct contact with a contaminated environment, pregnant sheep (particularly if aborting), and cats.

Further information

Further information on toxoplasmosis in humans is available from HPA at:
www.hpa.org.uk/Topics/InfectiousDiseases/InfectionsAZ/Toxoplasmosis/

Further information on toxoplasmosis in animals is available from Defra at:
www.defra.gov.uk/foodfarm/farmanimal/diseases/atoz/index.htm#toxoplasmosis

Trichinellosis (*Trichinella* spp.)

Trichinellosis is caused by a small parasitic worm (*Trichinella* spp.) 'the muscle worm', which can infect many species of mammals and some birds. It is a foodborne parasitic disease that is spread primarily by the consumption of raw or undercooked meat containing trichinae, the infective, immature (larval) stage of the worm.

Whilst GB is free from the parasite in animals, *Trichinella spiralis* has recently been found in two foxes in Northern Ireland. There have been no human cases of trichinellosis acquired from meat produced in GB for over 30 years.

There are eight zoonotic species of trichinella, of which *T. spiralis* is the most common species in Europe⁵⁶.

Infection with trichinellosis is not notifiable in humans or animals in the UK.

Infection in humans

Symptoms: The symptoms of trichinellosis in humans are highly variable depending on the severity of the disease (which is related to the number of larvae ingested) and the time after infection. A few days after infection there may be diarrhoea and other enteric symptoms associated with the presence of the adult worms in the intestine. Once new larvae have been produced and have invaded the muscles, then symptoms are fever, muscle pain, joint pains, and facial or periorbital oedema ("puffy eyes"). In severe cases, individuals may experience difficulties with coordinating movements as well as heart and breathing problems, and death may result due to myocardial failure.

Transmission: The main source of human infection is undercooked or raw meat products from horses and pigs (including wild boar) from countries where the disease is present. The trichinae are present in the striated muscle and can be easily killed by thorough cooking. The disease cannot be transmitted from person-to-person.

Cases: No human cases of trichinellosis were reported in the UK in 2009. Trichinellosis is present in Europe where outbreaks are regularly reported.

Nine cases of trichinellosis were diagnosed in England and Wales between 2000 and 2008, which included an outbreak of eight cases in 2000 associated with the consumption of imported meat products. The remaining case was travel-related.

UK livestock is thought to be free from trichinella and all imported foodstuff is tested or made safe by freezing. There has been no significant increase in the number of trichinellosis infections in the EU since 2001⁵⁷. However, numbers of infections

⁵⁶ Pozio (2007) *Vet Parasitology*, **149**(1-2) p3-21

⁵⁷ The Community Summary Report on trends and sources of zoonoses, zoonotic agents and foodborne outbreaks in the European Union in 2008 available at: www.efsa.europa.eu/en/scdocs/scdoc/1496.htm

reported in EU animals and humans have increased significantly since Romania and Bulgaria joined in 2007.

Infection in animals

Clinical signs: Animal infection is most likely to occur in carnivores and omnivores such as foxes, wild boar, bears, pigs, raccoons and rats, but infection may also occur in horses that have been fed on waste food containing infected meat. Infected animals usually show no obvious signs of disease unless the infestation is very severe. When they do occur, the clinical signs are variable, depending on the number of trichinae ingested and the immune status of the host. There may be diarrhoea in the early stages of disease, followed after 1 - 2 weeks by fever, stiffness and pain in the affected muscles, and dyspnoea. Blood results show a marked eosinophilia. In rare cases severe infection can cause death.

Frequency: Historically, cases have occurred in GB in swill-fed pigs, with infection attributed to the inclusion of infected imported meat into swill that was not adequately cooked.

All evidence indicates the absence of trichinella in the UK. Pigs and horses are routinely monitored for the presence of trichinella. In 2009, 208,357 breeding sows and boars and 1,727,877 fattening pigs (including an estimated 311,139 outdoor reared pigs) in the UK, and 5,136 horses, 1,011 farmed wild boar and 159 feral wild boar in GB were tested. All samples examined were negative.

An ongoing survey of trichinella in foxes is being carried out by the FSA and Defra in the UK, and from 2006 other wildlife has also been tested. In 2009, 454 foxes in GB were tested and none were positive; 170 were tested in Northern Ireland and one was positive for trichinella. Three seals in GB and 67 badgers in Northern Ireland were also tested, and all samples were negative.

Control Measures

EU trade rules prevent the importation of infected meat and meat products to all EU Member States as the main control measure to prevent infection in the UK. There is also a prohibition on feeding waste food to pigs. Routine testing for trichinella is undertaken on carcasses of all sows, boars, wild boar and horses for human consumption, and on a proportion of finishing pigs.

Summary of risk

Infection has been absent from UK domestic livestock since 1979 and no infection has been found in GB wildlife in the last 50 years. However, infected foxes were found in Northern Ireland in 2007 and 2009.

The risk of human infection in the UK comes from the consumption of infected meat or meat products produced in endemically infected countries. Trade controls prevent the importation of infective meat.

Further information

European outbreaks are reported at:

www.eurosurveillance.org/ViewArticle.aspx?ArticleId=590

Further information on trichinosis in animals at available from Defra at:

www.defra.gov.uk/foodfarm/farmanimal/diseases/atoz/index.htm#trichinosis

Variant Creutzfeldt-Jakob disease (vCJD) in humans and Bovine Spongiform Encephalopathy (BSE) in animals

Variant Creutzfeldt-Jakob disease (vCJD) in humans

Creutzfeldt-Jakob disease (CJD) is a rare and fatal transmissible spongiform encephalopathy (TSE) of humans. Sporadic CJD is the most common form and was initially described in 1921. In 1996, a new variant, vCJD was recognised and was strongly linked to Bovine Spongiform Encephalopathy (BSE) in cattle.

Symptoms: vCJD initially causes behavioural changes, anxiety and depression followed by incoordination and twitching. These symptoms are followed by memory disturbances and severe cognitive impairment, finally resulting in an inability to move or talk. The disease is progressive and fatal.

Transmission: vCJD is believed to have originated from human exposure to the BSE infectious agent in bovine meat and meat products. Transmissibility between humans is rare. There have been four probable secondary infections associated with blood transfusion in the UK. Other forms of transmission have been reported such as treatment with contaminated human-derived hormones, contaminated surgical instruments, transplants and implants such as dura mater grafts.

Cases: There were three deaths from definite or probable vCJD in the UK in 2009 (Table 20) bringing the total number recorded since 1996 to 167. The number of deaths peaked in 2000.

Table 20: Number of deaths from definite or probable vCJD in the UK, 2000-2009⁵⁸

Year	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009
Deaths	28	20	17	18	9	5	5	5	1	3

There have been no vCJD cases in those born after the 1980s. The government introduced leukodepletion of blood in 1999, and in 2004 implemented a policy that people who had received a blood transfusion in the UK since 1980 would no longer be able to give blood.

The 17th Annual Report (2008) on CJD Surveillance in the UK provides an analysis of the UK vCJD epidemic (www.cjd.ed.ac.uk/report18.pdf).

Further information

The Department of Health:

www.dh.gov.uk/en/Aboutus/MinistersandDepartmentLeaders/ChiefMedicalOfficer/CMOtopics/FeaturesBrowsableDocument/DH_4102718

The National Creutzfeldt-Jakob Disease Surveillance Unit:

www.cjd.ed.ac.uk/

⁵⁸ University of Edinburgh. (2009). The National Creutzfeldt-Jakob Disease Surveillance Unit: CJD Figures. www.cjd.ed.ac.uk/figures.htm

The Health Protection Agency:

www.hpa.org.uk/webw/HPAweb&Page&HPAwebAutoListName/Page/1191942152861?p=1191942152861.

The CJD Incidents Panel:

www.hpa.org.uk/webw/HPAweb&Page&HPAwebAutoListName/Page/120403151121?p=120403151121.

The Spongiform Encephalopathy Advisory Committee:

www.seac.gov.uk.

The 17th Annual Report (2008) on CJD Surveillance in the UK:

www.cjd.ed.ac.uk/report18.pdf

Bovine Spongiform Encephalopathy (BSE) in animals

BSE is a disease of domestic cattle. BSE has caused a major epizootic in cattle and smaller epizootics in exotic ruminants and domestic and exotic felines. Worldwide there have been two naturally occurring cases of BSE in goats: one in France and one in the UK. The transmissible agent is suspected to be an abnormal form of a host-encoded protein called the 'prion protein', although recent findings suggest infection may be transmitted independent of the presence of detectable prions.

BSE is a notifiable disease in animals.

Clinical signs: BSE is a progressive, fatal, neurological disease of adult cattle. Most clinical cases show at least one of the following: apprehension, hyperaesthesia or ataxia, and can include a change in behaviour, apprehension/nervousness, repeated exaggerated reactions to touch or sound, ataxia or hypermetria, reluctance to cross drains/enter doorways, aggression, excessive kicking when milked, head shyness, recumbency, tremors, loss of condition, excessive nose licking.

Frequency: The UK BSE epidemic peaked in 1992 with over 37,000 cases and has since declined steadily, with just 12 cases in 2009 (Table 21). There have been a small number of cases in North America, the Middle East, and Asia.

Table: 21 Number of confirmed cases of BSE cattle in the UK, 1999-2009⁵⁹

Year	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009
Cases	1,430	1,187	1,137	611	343	225	114	67	37	12

Control Measures

Control measures that prevent the feeding of ruminant protein to ruminants are the key to the control of BSE in animal populations. National and European legislation was first introduced with the 'ruminant to ruminant' feed ban (UK) in 1988. It has since developed, and further detail can be found on the Defra website.

⁵⁹ Department for Environment, Food and Rural Affairs. (2009). Bovine Spongiform Encephalitis (BSE) Statistics: www.defra.gov.uk/foodfarm/farmanimal/diseases/atoz/bse/statistics/index.htm

Other control measures include active and passive surveillance for BSE in cattle and TSEs in other species, animal identification requirements to allow cohorts (and certain offspring of affected female cattle) of confirmed BSE cases to be excluded from the human food chain by culling, and the regulated disposal of all animal by-products.

The total cost of BSE (including BSE research) to Defra alone in 2009/10 was approximately £21.5M.

Summary of risk

Effective, widely applied, and enforceable control measures have led to a progressive, significant reduction in the annual incidence of BSE cases in UK cattle. The incidence of BSE-related Transmissible Spongiform Encephalopathy (TSE) in other species (exotic ruminants and exotic and domestic felines) has also fallen significantly. The current controls are expected to continue to reduce the incidence of BSE in animals in the UK, and the present import and feed controls should ensure that imports from other countries with BSE do not compromise this progress.

Further information

Further information on BSE in animals is available from Defra at:
www.defra.gov.uk/foodfarm/farmanimal/diseases/atoz/bse/index.htm

Vero cytotoxin-producing *Escherichia coli* (VTEC)

Escherichia coli is a bacterial species which normally inhabits the guts of animals, including humans. Most strains are considered to be harmless; however there are a number of subgroups that are associated with human disease. In clinical terms the most important of these are the Verocytotoxin-producing *E. coli* (VTEC), and in particular VTEC O157.

Although VTEC O157 is the most common zoonotic serogroup affecting people in the UK, other serogroups such as O26 and O111 may be important in some countries.

Cattle are the main reservoir of VTEC O157 in the UK, but the organism is also commonly found in other ruminants, especially sheep, and has been isolated from a wide range of other livestock and wildlife species. While VTEC causes illness in humans, it does not normally cause disease in other animal species.

VTEC O157 has emerged in the last 25 years as a pathogen of worldwide importance. It was first reported in England and Wales in 1982 and in Scotland in 1984. On average, 1,200 cases in humans have been reported annually over the last five years.

In August and September 2009, there was a large outbreak of VTEC O157 associated with an open farm in Surrey. This has over 200,000 visitors per year, including between 1,500 and 2,000 per day during the summer school holidays. There were 93 cases (of whom 76 were under 10 years of age). This led to an independent review by Professor George Griffin and a number of recommendations aimed at reducing the risk to people visiting such establishments⁶⁰.

Infection with VTEC is not notifiable in animals in the UK.

Infection in humans

Symptoms: Symptoms range from mild diarrhoea through haemorrhagic colitis (bloody diarrhoea) to haemolytic uraemic syndrome (HUS). Patients generally recover within two weeks. The use of antibiotics is not recommended and these may boost toxin release by the bacteria and worsen symptoms. The most severe symptoms, including HUS, are usually seen in children below the age of five years and those over 65 years. VTEC infections may be asymptomatic, particularly in adults.

Transmission: VTEC O157 has a low infectious dose and can be transmitted to people in several ways. These include:

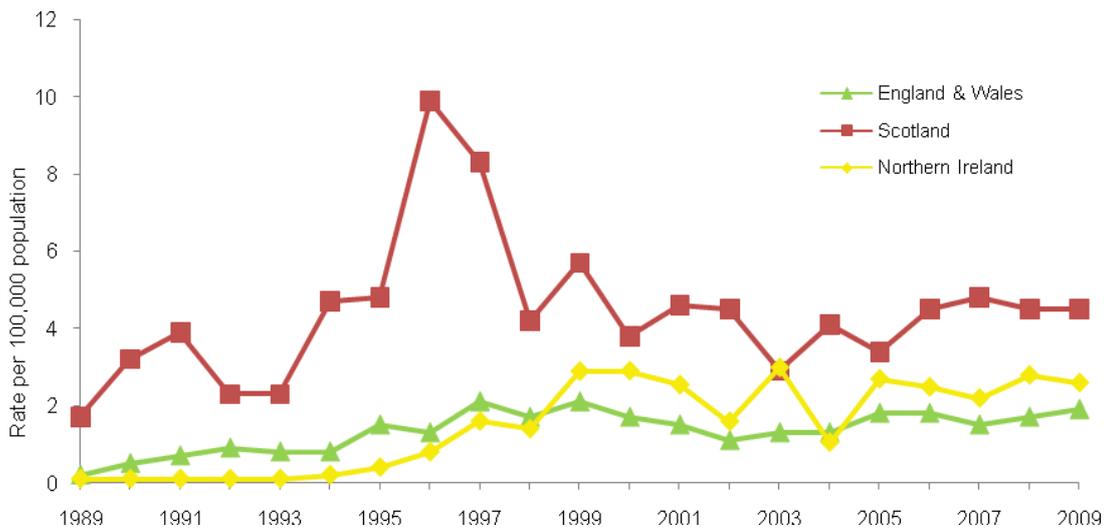
- Consumption of contaminated food or water
- Direct or indirect contact with animals, their faeces or contaminated environments
- Person-to-person spread

⁶⁰ www.griffininvestigation.org.uk/

Cases: In 2009, there were 1,306 laboratory-confirmed cases (1,034 in England and Wales, 228 in Scotland and 44 in NI) of VTEC O157 reported in humans in the UK, a 5.6% increase on the 1,237 cases reported in 2008.

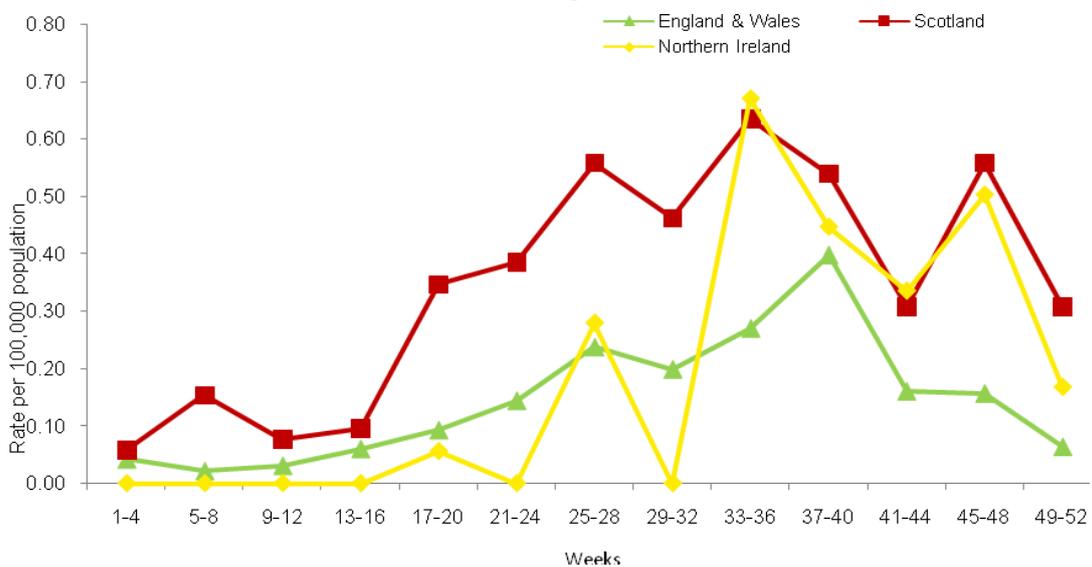
There are clear differences in the geographical distribution of laboratory-confirmed cases within the UK, and Scotland has consistently recorded the highest rates of infection per 100,000 head of population since the late 1980's (Figure 8). The 1996 and 1997 Scottish figures are inflated due to the central Scotland outbreak.

Figure 8: Annual rates of laboratory confirmed reports of human VTEC O157 infections in the UK, 1989–2009



Infections with VTEC O157 show a seasonal distribution with most cases occurring between July and September, although this may be affected by the occurrence of large outbreaks at other times (Figure 9).

Figure 9: Seasonal distribution of laboratory confirmed reports of VTEC O157 in humans in the UK during 2009



In England and Wales about 15% of general VTEC outbreaks have been linked to direct or indirect animal contact. Prior to the large outbreak at an open farm in 2009, these have generally each comprised fewer than ten cases. Most large outbreaks have been related to food rather than direct contact with animals. In contrast, about 80% of human cases appear to be sporadic and unattributed to an identifiable source, although case-control studies suggest that contact with farm animals and the rural environment may be a major contributing factor.

VTEC in food

Consumption of contaminated raw meats is an important route of foodborne infection. VTEC O157 has a low infectious dose and cross-contamination from contaminated raw meats is an important route of transmission. Cold cooked meats, dairy products, minced beef product and salad vegetables have all been implicated in foodborne outbreaks.

At abattoirs, Food Business Operators are required to check the hide or skins of livestock presented for slaughter for faecal contamination, and take the necessary steps to avoid contamination of the meat during slaughter.

In 2009, the HPA undertook a butcher's shop survey where 1,944 samples were taken and tested for VTEC O157; there were no positive results.

Infection in animals

Clinical signs: Animals do not present with clinical signs when infected with VTEC O157. However, enteric disease in animals, particularly calves, can be associated with non-O157 VTEC strains such as O26 and O111.

Frequency: VTEC O157 infection is widespread in cattle in the UK. However, because shedding of the organism is intermittent and it does not cause disease in cattle, prevalence figures are of limited help in assessing the degree of risk to humans.

During 2009, the VLA assisted the HPA with the investigation of 14 outbreaks, including the largest recorded human outbreak in Britain linked to animals at an open farm in Surrey, as mentioned above. The main findings are summarised in Table 22.

Table 22: Summary of VLA investigations of potential animal sources of 14 VTEC O157 investigations in England and Wales, 2009

Premises/ Month	No. people with illness linked to outbreak	Species Tested	VTEC O157 positive	Phage Types
Open farm (Devon, May)	3 primary cases+4 secondary contact	Lambs, pigs, rabbits, guinea pigs, chickens, ducks	Lambs	21/28*
Open farm (Lancs, June)	9 cases	Multiple species (≥10)	Cattle, sheep, goats, pigs, deer, llama, equines	21/28*
School field/ brook (Leics, July)	16 cases	Dog, goose	None	
Dairy/ Dance festival (Pembs, Aug)	No details	Cattle, badger	Cattle	2*
Open farm† (Surrey, Sept)	65 primary cases, 13 secondary and 15 asymptomatic	Multiple species (≥10)	Cattle, sheep, goats, pigs, equines	21/28*
Open farm † (Surrey, Sept)	No cases	Sheep, goats, pigs, deer, rabbits, ducks, chickens	None	
Open farm † (Devon, Sept)	2 primary, 3 secondary cases	Cattle, sheep, goats, pigs, rabbits, chickens	Pigs, mixed samples from sheep, goats, chickens, rabbits	34, 54*
Open farm † (Notts, Sept)	6 cases	Cattle, sheep, goats, pigs, alpaca, llama, equines	Cattle, sheep, goats	2, 8*
Commercial farm (Norfolk, Sept)	3 cases	Cattle, wild rabbits	Cattle	2*
Open farm † (N. Yorks, Sept)	8 cases	Multiple species (≥10)	Cattle, sheep, goats, pigs, hens (in cattle area)	21/28*
Domestic setting (Cornwall, Nov)	2 cases	Goats, pigs	Goats, pigs	21/28*
Commercial farm (Cheshire, Nov)	4 cases	Cattle, sheep, goats, pigs, hens	Cattle, sheep, goats	1, 8*
Open farm (Devon, Nov)	3 cases	Cattle	Cattle	32
Domestic Setting (N. Wales, Nov)	4 cases	Pigs, donkeys, chickens	Pigs	21/28*

† Details of VLA involvement in these outbreaks was reported and regularly updated as Q&As on the VLA website: www.defra.gov.uk/vla/news/new_ecoli.htm

*Molecular profiling indicated matches between human isolates and some or all of the isolates from animal species in this investigation.

Details of all VTEC O157 investigations on open farms and other animal amenity premises open to the public carried out by VLA over a 10 year period were published in 2009⁶¹.

⁶¹ Pritchard, G.C. et al (2009). Verocytotoxigenic *Escherichia coli* O157 in animals on public amenity premises in England and Wales, 1997-2007. *Veterinary record*. **164**:545-549

Control Measures

Available controls for VTEC, including VTEC O157 in animals, rely on the application of good husbandry and hygiene measures particularly at the point of provision of food production. These principally require the hygienic production and pasteurisation of milk, the provision of clean animals to slaughter, and the application of hygiene practices in the processing of these animals and the meat produced from them. In addition, controls to minimise the risk of zoonotic spread on farms require the application of appropriate risk management procedures based upon those suggested for open farms⁶².

Research has shown that the level of VTEC O157 may be reduced by keeping young cattle on very dry bedding, rearing young stock in closed groups and not bringing bought cattle on to the farm, and by reducing contact between herds⁶³. By following these control measures, within-group prevalence was reduced by 30% over a four and a half month period, yielding a risk reduction of approximately five times compared to the control group. The application of potentially expensive on-farm control measures for VTEC need to be balanced against the risk of human infection.

Visitors to livestock farms, including those open to the general public, ramblers and workers on commercial livestock farms are all at risk of exposure, and should ensure good hand hygiene is observed. Risk of foodborne human illness can be reduced by thoroughly cooking meat and meat products, and by avoiding cross-contamination of work surfaces and ready-to-eat foods.

Summary of risk

Most cases of VTEC O157 are sporadic and these have a strong association with exposure to animals. However, although the risk of infection from open farms is low, the consequences of infection, especially in younger children, may be high. VTEC has been the cause of large foodborne outbreaks, and although VTEC infection in animals is not of any direct economic significance to livestock farmers, control measures are in place to reduce the risk of VTEC entering the food chain.

Further Information

Advice leaflets on minimising the risk of infection with VTEC can be found at:

- www.defra.gov.uk/foodfarm/farmanimal/diseases/vetsurveillance/documents/vtec-leaflet.pdf
- www.hse.gov.uk/pubns/ais23.pdf
- www.scotland.gov.uk/Publications/2005/03/20839/54388

Further information on VTEC in animals is available from Defra at:

www.defra.gov.uk/foodfarm/farmanimal/diseases/vetsurveillance/profiles/documents/sp-vtec.pdf

⁶² Advice sheet to farmers available at: www.hse.gov.uk/pubns/ais23.pdf

⁶³ Ellis-Iversen, J et al. (2007). Farm practices to control *E. coli* O157 in young cattle – a randomised controlled trial, *Veterinary Research*, **39**(3).

Further information on VTEC in humans is available from HPA at:
www.hpa.org.uk/Topics/InfectiousDiseases/InfectionsAZ/EscherichiaColiO157/

Yersiniosis (*Yersinia* spp.)

The genus *Yersinia* includes the zoonotic species *Y. enterocolitica*, *Y. pseudotuberculosis* and *Y. pestis* (plague). Plague does not exist in the UK. Yersiniosis in humans is mostly caused by *Yersinia enterocolitica*, and humans usually acquire infection through food contaminated with the faeces of infected animals.

Y. enterocolitica has been isolated from many domestic and wild mammals, birds and some cold-blooded animals. More than 50 serotypes have been identified, not all of which cause disease in animals and man.

Y. pseudotuberculosis has been isolated from various species of wild and domestic mammals, birds and reptiles.

Infection with yersiniosis is not notifiable in humans or animals in the UK.

Infection in humans

Symptoms: Clinical presentations of non-plague yersiniosis include fever, watery diarrhoea and abdominal pain that may mimic appendicitis, and chronic arthritis. The likelihood of serious morbidity is low.

Transmission: The mode of transmission from animals to humans is presumed to be ingestion of faecally-contaminated food (with a particular association with raw pork and pork products) or water, and direct contact with infected animals. Pigs, dogs and cats are the most common carriers of the serotypes associated with disease in man. Person-to-person spread occurs, but is uncommon.

Cases: In 2009 there were 61 cases of yersiniosis in people reported in the UK, compared to 62 in 2008 (Table 23). The majority of cases were reported in people over 45 years of age. No cases of yersiniosis were reported in Northern Ireland during 2008 or 2009.

Table 23: Confirmed human cases of yersiniosis in the UK, 2009

	England & Wales	Scotland	NI	UK total
<i>Y. enterocolitica</i>	40	13	0	53
<i>Y. other species</i>	7	1	0	8
Total	47	14	0	61

Infection in animals

Clinical signs: *Y. enterocolitica* infection in animals is a mainly subclinical infection, although it can cause enteric disease in young sheep. Pigs are the most important livestock carriers of *Y. enterocolitica* serotypes associated with human disease.

Y. pseudotuberculosis infection is also usually asymptomatic in animals but has been associated with clinical disease in poultry and has been occasionally reported

to cause fever, abortion and diarrhoea in cattle, sheep and pigs. Signs of *Y. pseudotuberculosis* infection in poultry are non-specific and include diarrhoea, weakness, ruffled feathers, lameness and progressive emaciation. It is also an uncommon cause of abortion in sheep. *Y. pseudotuberculosis* and *Y. enterocolitica* occasionally cause chronic ill-thrift and/or diarrhoea in young sheep.

Clinical disease (mainly enteric) due to *Y. pseudotuberculosis* has been recorded in zoological collections, caged birds, domestic poultry and small mammals such as guinea pigs. Clinical disease is sporadic in domestic animals although outbreaks associated with significant mortality have been reported in guinea pigs, hares and rabbits.

Transmission between animals often involves direct or indirect contact with wild rodents. Any changes which alter the number and distribution of wildlife carriers of *Yersinia* species could potentially affect the incidence of disease in domestic animals and man.

Frequency: During 2009, 37 cases (33 in GB) of yersiniosis (including fetopathy) were diagnosed in animals in the UK (Table 24). This is a slight increase from the 32 cases reported in 2008.

Table 24: Laboratory confirmed cases of yersiniosis in animals in the UK, 2009

Species	Sheep	Goats	Birds	Wildlife & Miscellaneous	Total
Cases	12	4	7	14	37

Control Measures

The risks to people can be mitigated by the careful application of good personal hygiene during and after handling pets, and good food hygiene practices.

There are currently no specific measures available or implemented to control or monitor yersiniosis in animals in the UK, apart from adopting good on-farm standards of husbandry, hygiene and vermin control. This is due to the ubiquitous distribution of the organism and the difficulties associated with detecting carriers in the domesticated and wildlife animal population.

Summary of risk

The risk to humans is associated with the consumption of food contaminated with animal faeces and has remained stable over recent years.

Further information

Further information on yersiniosis in humans is available from HPA at: www.hpa.org.uk/Topics/InfectiousDiseases/InfectionsAZ/Yersinia/

Reports on yersinia in animals are produced by the VLA in the Non-Statutory Zoonoses Reports that can be found at: www.defra.gov.uk/vla/reports/rep_surv_zoonoses.htm

Appendices

Appendix 1: Notifiable zoonotic diseases and organisms in humans in 2009

Disease	Notifiable*** in humans under public health legislation in			Reportable under RIDDOR* to HSE
	England & Wales	Scotland	NI	
Anthrax	✓	✓	✓	✓
Brucellosis				✓
Psittacosis	See footnote**			✓
Chlamydiosis (ovine)				✓
Food poisoning	✓	✓	✓	
Leptospirosis	✓	✓	✓	✓
Lyme disease		✓		✓
Q-fever				✓
Rabies	✓	✓	✓	✓
<i>Streptococcus suis</i>				✓
Toxoplasmosis		✓		
Tuberculosis	✓	✓	✓	✓

* RIDDOR: Reporting of Injuries, Diseases and Dangerous Occurrences Regulations

** A local anomaly exists in Cambridgeshire where psittacosis is reportable to the local CCDC under a local bylaw.

*** A revised list of notifiable diseases, and for the first time a list of notifiable organisms, were introduced in Scotland, England, Wales and Northern Ireland during 2010 and will be reported in more detail in next year's report.

Appendix 2: Laboratory confirmed cases of zoonotic disease in humans in the UK, 2000-2009⁶⁴

	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009
Anthrax	1	2	0	0	0	0	1	0	1	11
Avian Influenza	0	0	0	0	0	0	1 ⁶⁵	4 ⁶⁶	0	0
<i>Mycobacterium bovis</i>	28	31	20	16	20	39	34	22	21	25
<i>Brucella abortus</i>	1	8	13	4	9	1	4	8	9	4
<i>Brucella melitensis</i>	4	6	6	5	9	9	9	7	5	8
<i>Brucella sp</i>	14	13	17	15	13	2	3	0	1	5
<i>Campylobacter</i>	65,720	61,404	54,075	51,473	49,750	52,196	52,662	58,054	55,609	65,000
<i>Cryptosporidium</i>	7,083	4,482	3,663	6,626	4,197	5,288	4,360	3,671	4,909 ^a	5,577
Hantavirus	0	0	0	0	0	0	0	0	0	0
Hydatid	17	11	10	5	8	11	14	10	18	9
Leptospirosis	54	48	58	28	32	46	51	81	76	54
Listeriosis	116	162	160	247	230	220	208	255	208 ^a	235
Lyme disease	348	296	384	347	586	691	945	1,036	1,098 ^a	1,395
Orf	7	9	4	6	3	2	2	2	3	1
<i>Pasteurella</i>	253	412	302	375	395	434	486	466	489	559
Psittacosis	209	142	94	106	75	72	40	54	66 ^a	60
Q fever (acute and chronic infections)	112	88	163	51	39	25	148 ⁶⁷	62	72 ^a	36
Rabies 'classical'	0	2	0	0	0	1	0	0	1	0
Rabies EBLV	0	0	1	0	0	0	0	0	0	0
<i>Salmonella</i>	16,983	18,420	16,319	16,343	1,729	12,833	13,060	13,218	11,075 ^a	10,071
<i>Strep suis</i>	1	1	1	1	0	2	2	2	7	2
<i>Taenia</i>	64	107	75	87	92	72	87	98	99 ^a	72
<i>Toxocara</i>	9	1	3	3	4	5	2	1	2	1
<i>Toxoplasma</i>	128	129	138	99	100	115	127	152	463 [*]	494 [*]
<i>Trichinella</i>	8 ⁶⁸	1	0	0	0	0	0	0	0	0
vCJD ⁶⁹	28	20	17	18	9	5	5	5	1	3
VTEC O157	1,139	1,049	852	874	926	1,169	1,287	1,120	1,237	1,306
West Nile Virus	0	0	0	0	0	0	1 ⁷⁰	1 ⁶⁹	0	0
Yersiniosis	66	66	44	95	70	64	62	73	62	61

* Includes Enhanced England & Wales data.

^a There has been an amendment to the 2008 figure printed in last year's report as data is derived from a dynamic database.

⁶⁴ Note: this is not a definitive list of zoonotic pathogens that are reported each year, but covers zoonotic diseases reported annually in the UK Zoonoses Report.

⁶⁵ Case of H7N3

⁶⁶ Cases of H7N2

⁶⁷ 111 confirmed with a further 28 probable and 5 possible in an outbreak in Scotland.

⁶⁸ Cluster of cases acquired from imported pork salami from Serbia.

⁶⁹ Defined as definite or probable cases.

⁷⁰ Infection imported from Canada

Appendix 3: Notifiable and Reportable diseases in animals which are potential zoonoses in the UK

Notifiable diseases are those where there is a statutory requirement to report a suspicion of a clinical case of disease.

Reportable diseases (in animals) are those where there is a statutory requirement to report laboratory confirmed isolation of organisms of the genera *Salmonella* and *Brucella* under the Zoonoses Order 1989. The report is to be made by the laboratory which isolated the organism from an animal derived sample.

Disease	Species	Last Occurred in UK ⁷¹	Notifiable to Animal Health in GB, Veterinary Service in NI	Reportable
Anthrax (<i>Bacillus anthracis</i>)	Cattle/other mammals	2006	✓	
Avian Influenza (HPAI)	Poultry/waterfowl	2008	✓	
Bovine Spongiform Encephalopathy	Cattle	Present	✓	
Brucellosis (<i>Brucella abortus</i>)	Cattle ⁷²	2004 GB/ 2009 NI ⁷³	✓	✓
Brucellosis (<i>Brucella melitensis</i>)	Sheep and goats	Never	✓	✓
Chlamydiosis	Sheep and goats	Present	⁷⁴ Ornithosis (including psitticosis) notifiable in NI in poultry	
Contagious Epididymitis (<i>B. ovis</i>)	Sheep and goats	Never	✓	✓
Equine Viral Encephalomyelitis	Horses	Never	✓	
<i>Echinococcus multilocularis</i> and <i>granulosus</i>	Dogs, and foxes	Present ⁷⁵		
Equine morbillivirus (Hendra)	Horses	Never	✓ (Not notifiable in NI)	
Glanders & Farcy (<i>Burkholderia mallei</i>)	Horses	1928	✓	

⁷¹ Figures taken are correct as at December 2009 and may be subject to change.

⁷² In the Zoonoses Order 1989 *Brucella* reporting relates to (a) "animal" meaning cattle (bull, cow, steer, heifer, calf), horse, deer, sheep, goat, pig or rabbit; and (b) "bird" meaning a domestic fowl, turkey, goose, duck, guinea-fowl, pheasant, partridge, quail or pigeon.

⁷³ Present in NI; outbreak in Scotland in 2003 and Cornwall, England in 2004.

⁷⁴ Legislative veterinary powers under The Psittacosis or Ornithosis Order 1953 (S.I. 1953 No. 38) give discretionary powers to serve notices to impose movement restrictions and require cleansing and disinfection of affected premises so Animal Health may be involved in the control of Psittacosis, even though it is not a notifiable disease in animals or birds.

⁷⁵ *E. granulosus* is present in the UK, *E. multilocularis* is not present in the UK.

Disease	Species	Last Occurred in UK ⁷⁶	Notifiable to Animal Health in GB, Veterinary Service in NI	Reportable
Newcastle disease and paramyxovirus infection	Poultry and pigeons	2006	✓	✓ (Not reportable in NI)
Rabies (Terrestrial)	Dogs and other mammals	197077	✓	
Rabies (EBLV)	Bats	200978	✓	
Rift Valley Fever	Cattle, sheep and goats	Never	✓	
Salmonella	All species	Present	Salmonellosis is poultry is notifiable in NI	✓
Trichinella	Pigs, horses and other mammals	Present ⁷⁹		
Tuberculosis (<i>Mycobacterium bovis</i>)	Domestic cattle, buffalo, bison and deer	Present ⁸⁰	✓ ⁸¹	✓
Vesicular stomatitis virus (VSV)	Cattle/ other mammals	Never	✓	
West Nile Virus	Horses	Never	✓	

⁷⁶ Figures taken are correct as at December 2009 and may be subject to change.

⁷⁷ A quarantine case was confirmed in 2008, however this does not affect the national disease status.

⁷⁸ European bat Lyssavirus type 2 was isolated from a Daubenton's bat in 2009.

⁷⁹ Trichinella is present in Northern Ireland and is not present throughout GB.

⁸⁰ Scotland has been officially free since October 2009, although sporadic incidents continue to be identified in cattle herds.

⁸¹ In addition to any bovines and deer with suspect clinical signs of tuberculosis, under the Tuberculosis (England) Order 2007, the Tuberculosis (Wales) Order 2006, and the Tuberculosis (Scotland) Order 2007 (as amended), there is a statutory requirement to notify to the local Animal Health office the presence of suspect TB lesions in the carcasses of any bovine animals or other farmed or companion (pet) mammals. Furthermore, identification of *Mycobacterium bovis* in samples taken from any mammal (other than man) is also notifiable to VLA Weybridge unless the organism was present in the sample as a result of an agreed research procedure. Notifying the suspicion of TB in a living domestic animal in the course of clinical examination, surgery, by radiography or in biopsy material is not mandatory (except for cattle or deer), but submission of clinical samples from such cases to VLA is encouraged.

Appendix 4: Laboratory confirmed cases of zoonotic disease in animals in GB, 2000-2009

	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009
Anthrax*	0	0	1	0	0	0	2	0	0	0
Avian Influenza ^{82*}	0	0	0	0	0	0	0	1 ⁸³	2 ⁸⁴	0
<i>Mycobacterium bovis</i> isolates in cattle ⁸⁵	NA	NA	NA	1,246	4,490	5,463	4,857	4,765	5,981 ^a	5,134
<i>Mycobacterium bovis</i> incidents in non-bovine animals	12	1	18	35	56	64	78	68	119	144
<i>Mycobacterium</i> species in non-bovine animals (excluding <i>M. bovis</i>)*	NA	NA	NA	18	25	55	138	104	77	122
<i>Brucella abortus</i>	0	0	0	0	0	0	0	0	0	0
<i>Brucella melitensis</i>	0	0	0	0	0	0	0	0	0	0
<i>Brucella sp</i> ***	0	0	6	6	5	2	0	30	6	5
BSE ^{86*}	1,430	1,187	1,137	611	343	225	114	67	37	12
Campylobacter**	300	170	168	223	284	150	170	217	155	152
Chlamydiosis (<i>Clamydophila abortus</i>) fetopathy**	764	426	506	559	390	473	462	532	349 ^a	373
Cryptosporidium**	1,780	994	1,086	1,237	1,156	1,229	1,146	841	1,330 ^a	1,346
Hydatid**	0	0	1	0	1	0	0	0	0	0
Leptospirosis**	400	163	217	93	38	46	45	93	39	5
Listeriosis**	175	119	150	210	214	193	200	134	196 ^a	175
Orf**	57	31	30	39	37	27	38	45	44	37
<i>Pasteurella multocida</i> **	422	254	435	587	511	471	452	347	322 ^a	327

* Confirmed cases are notifiable to Animal Health.

** Confirmed cases obtained through scanning surveillance/ VIDA database.

*** Confirmed cases statutorily reportable under Zoonoses Order 1989.

⁸² Only highly pathogenic strains of avian influenza are reportable to Animal Health, table shows number of incidents per year.

⁸³ H5N1 isolates were found in samples from one turkey farm in 2007.

⁸⁴ H7N7 isolates were reported from samples taken from an egg laying chicken farm in 2008, and isolates of H5N1 were reported in a cluster of 10 wild mute swans and 1 Canada goose found dead.

⁸⁵ This figure is different from the number of incidents. This is because laboratory confirmation is not sought for all individual reactors in an incident. However, where several reactors are tested from an incident, multiple isolations can result.

⁸⁶ Figures for BSE are obtained through scanning and targeted surveillance.

^a There has been an amendment to the 2008 figure printed in last year's report as the data is derived from a dynamic database.

	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009
Psittacosis (C. psittaci) **	28	16	23	17	9	5	2	2	1	3
Q fever **	15	5	6	3	3	6	7	4	5	3
Rabies 'classical' *	0	0	0	0	0	0	0	0	1 ⁸⁷	0
Rabies EBLV *	0	0	1	0	2	0	1	1	2	1
Salmonella (all types) ***	1,675	1,088	1,560	1,942	1,429	1,261	1,255	998	943 ^a	1,166
Streptococcus suis **	23	18	49	59	68	48	63	54	92 ^a	69
Swine Influenza **	20	8	9	21	10	20	11	9	17	14
Toxoplasma **	480	201	279	352	335	376	310	338	201	206
Trichinella ⁸⁸	0	0	1	0	0	0	2	0	0	0
Yersiniosis **	30	19	21	28	34	36	29	24	32	33

Note: this is not a definitive list of all zoonotic pathogens that are reported each year, but covers those for which data are available (notifiable/reportable and those recorded by VIDA system).

Survey Data

Survey data is available for Hantavirus and West Nile Virus, which are not routinely recorded and reported by VLA/SAC. See the quarterly reports of the GB Wildlife surveillance partnership:

www.defra.gov.uk/vla/reports/rep_surv_wildlife.htm

Outbreak Investigations

Isolations of VTEC are not routinely recorded and reported by VLA/ SAC. A list of outbreak investigations and further references can be found within the VTEC A-Z section of this report.

Unavailable data

Annual data for Toxocara and Taenia is unavailable as it is not recorded on the VIDA database.

⁸⁷ Rabies case was in a quarantined animal.

⁸⁸ Figures provided by FSA.

** Confirmed cases obtained through scanning surveillance/ VIDA database.

*** Confirmed cases statutorily reportable under Zoonoses Order 1989.

^a There has been an amendment to the 2008 figure printed in last year's report as the data is derived from a dynamic database.

Appendix 5: Relevant Legislation (covering statutory and non-statutory zoonoses)

Note that other associated legislation not listed below may exist in Devolved Administrations (Wales, Scotland and Northern Ireland).

Legislation marked with * was introduced in 2010 and reflects the situation at time of publication, not during 2009.

Human legislation

Control of Substances Hazardous to Health (COSHH) Regulations 1999
Health and Safety at Work etc Act 1974
Health and Services and Public Health Act 1968
Health and Social Care Act 2008
The Health Protection (Notification) Regulations 2010*
The Health Protection (Local Authority Powers) Regulations 2010 (for England and Wales)*
The Health Protection (Part 2A Orders) Regulations 2010 (for England and Wales)*
Public Health (Control of Disease) Act 1984 (as amended)
The Public Health (Ships) Regulations 1979
The Public Health (Aircraft) Regulations 1979
Reporting of Injuries, Diseases and Dangerous Occurrences Regulations (RIDDOR) 1995

Animal legislation

Animal Boarding Establishments Act 1963
Animal Boarding Establishment Regulations (NI) 1974
Animal Health Act 1981(as amended)
Diseases of Animals (NI) Order 1981 (as amended)
Animal Health and Welfare (Scotland) Act 2006
Animal Welfare Act 2006 (for England and Wales)
Welfare of Animals Act (NI) 1972
Anthrax Order 1981
Avian Influenza and Influenza of Avian Origin in Mammals (England) Order 2006 (equivalent NI legislation in 2007)
Avian Influenza and Influenza of Avian Origin in Mammals (Wales) (No 2) Order 2006
Avian Influenza and Influenza of Avian Origin in Mammals (Scotland) Order 2006
Breeding of Dogs Act 1973
Breeding and Sale of Dogs (Welfare) Act 1999
Brucellosis (England) Order 2000
Brucellosis (England and Wales) Order 1981 (as amended) (current Welsh legislation)
Dangerous Wild Animals Act 1976
Dogs Act 1906
EU Zoonoses Directive 2003/99/EC.
EU Zoonoses Regulation (EC) no 2160/2003

Infectious Diseases of Horse Order 1987
Litter (Animals Droppings) Order 1991
Pet Animals Act 1951 and 1983
Prevention of Damage by Pests Act 1949
Psittacosis or Ornithosis Order 1953
Rabies Control Order 1974
Rabies (Importation of Dogs, Cats and other Mammals) Order 1974 (as amended) –
Regulation EC 2075/2005, laying down specific rules and controls for Trichinella in
meat
Riding Establishments Act 1964 and 1979
Specified Animal Pathogens Order 2008
Specified Animal Pathogens (Wales) Order 2008
Specified Diseases (Notification and Slaughter) Order 1992
Specified Diseases (Notification) Order (NI) 2004
Transmissible Spongiform Encephalopathies (Wales) Regulations 2008
Transmissible Spongiform Encephalopathies (England) Regulations 2008
Transmissible Spongiform Encephalopathies (Scotland) Regulations 2008
Tuberculosis (England) Order 2007
Tuberculosis (Scotland) Order 2007 (as amended)
Tuberculosis (Wales) Order 2006
Tuberculosis (NI) Control order 1999 (as amended)
Tuberculosis (Scotland) Order 2007 (as amended)
Tuberculosis (Deer) (Order) 1989
Zoonoses (Monitoring) (England) Regulations 2007 (equivalent NI legislation in
2008)
Zoonoses (Monitoring) (Wales) Regulations 2007
Zoonoses (Monitoring) (Scotland) Regulations 2007
Zoonoses (Monitoring) (Scotland) Regulations 2007

Food

Animal By-Products Regulations 2005 (as amended)
Animal By-Products Regulations (NI) 2003
Animal By-Products (Wales) Regulations 2006
Animal By-Products (Scotland) Regulations 2003 as amended
EC Regulation 852/2004, 853/2004, 854/2004
Feed (Hygiene and Enforcement) (Wales) Regulations 2005 (as amended)
Food and Environment Protection Act 1985
Food Safety Act 1990
Food Safety (1991 Order) (commencement) Order (NI) 1991
The Food Hygiene (England) Regulations 2006 (and Amendment 2007)
Food Hygiene regulations (NI) 2006
The Food Hygiene (Wales) Regulations 2006 (as amended)
The General Food Regulations 2004 (as amended)
The Official Feed and Food Controls (Wales) Regulations 2006 (as amended)
Zoo Licensing Act 1981
Zoonoses Order 1989
Zoonoses order (NI) 1991

General

Anthrax Order (NI) 1969 (as amended)

Environmental Protection Act 1990

EU Directive 64/432/EEC as amended (EU Consolidated Text, CONSOLEG:
1964L0432

Rabies Control Order (NI) 1977

Riding establishment Regulations (NI) 1980

The Water Supply (Water Quality) Regulations 2000*

Transmissible Spongiform Encephalopathies (NI) 2008 (as amended)

Transmissible Spongiform Encephalopathies (Scotland) Regulations 2010*

Appendix 6: Animal population

Table 1: Number of livestock for each country in UK in 2009*

	England	Wales	Scotland	N. Ireland	UK
Cattle	5,484,083	1,129,968	1,812,405	1,599,025	10,025,481
Sheep	14,983,839	8,237,737	6,919,756	1,896,700	32,038,032
Pigs	3,872,413	22,303	396,041	433,539	4,724,296
Poultry	224,468,714	12,361,556	26,555,340	16,705,741	280,091,351
Goats	86,745	6,801	5,051	2,663	101,260
Farmed Deer	22,991	755	7,033	3,837	34,616
Horses	663,100	98,500	48,800	12,248	822,648

*Source: Radar Veterinary Surveillance database (Defra)

GB cattle data is from the GB Cattle Tracing System, accessed on 1st June 2009

GB sheep, pig, goat and farmed deer data is from the June Agricultural Surveys for 2009

GB Poultry data is from the GB Poultry Register, accessed in February 2010

UK Horse data comes from the UK National Equine Database, accessed on 22 January 2009

NI cattle, sheep, pig, poultry, goat and farmed deer data are provided by Department of Agriculture and Rural Development NI for 2009.

Note that figures in Table 1 are a snapshot of the population at a specific time during the year, as shown in the table footnotes.

For further information on data quality including accuracy and comparability contact: vetsurveillance@defra.gov.uk

Table 2: Number and percentage of pet owning households in the UK 2009*

Species	Percentage of total households	Approximate number of households (millions)
Dogs	23%	6.0
Cats	20%	5.2
Rabbits	2.8%	0.7
Birds (indoor)	1.8%	0.5
Hamsters	1.3%	0.3
Guinea Pigs	1.3%	0.3
Gerbils	0.5%	0.1
Snakes	0.4%	0.1
Lizards	0.4%	0.1
Horses/Ponies	0.3%	0.1
Tortoises/turtles	0.3%	0.1
Rats	0.3%	0.1

*Source: Pet Food Manufacturers' Association: www.pfma.org.uk/statistics/pet-statistics.htm

In 2009, approximately 43% of households in the UK owned a pet. Some households may own more than one type of pet.

Appendix 7: Further reading

General further reading

Advisory Committee on the Microbiological Safety of Food: Report on microbial antibiotic resistance in relation to food safety. The Stationery Office, ISBN 0 11 322283 1. <http://acmsf.food.gov.uk/infoguide/>

Defra - Zoonoses web pages

www.defra.gov.uk/foodfarm/farmanimal/diseases/atoz/zoonoses/index.htm

Defra Publications - Zoonoses Reports UK

www.defra.gov.uk/foodfarm/farmanimal/diseases/atoz/zoonoses/reports.htm

Food Standards Agency: A report on the study of Infectious Intestinal Disease in England, The Stationery Office, ISBN 0 11 322308 0

www.food.gov.uk/aboutus/publications/

Food Standard Agency – Foodborne Illnesses web pages

www.food.gov.uk/science/research/foodborneillness/microfunders/intestinal

Health Protection Agency - Zoonoses web pages

www.hpa.org.uk/Topics/InfectiousDiseases/InfectionsAZ/Zoonoses/

Health Protection Agency - Zoonoses newsletters

www.hpa.org.uk/Topics/InfectiousDiseases/InfectionsAZ/Zoonoses/ZoonosesNewsletters

HSE Agriculture Information Sheet 2 'Common zoonoses in agriculture' available free from HSE Books, tel. 01787 881165

www.hse.gov.uk/pubns/ais2.pdf

HSE Books: The Occupational Zoonoses, ISBN 0 1188 6397 5

www.hse.gov.uk/biosafety/information.htm#a7

Joint Agency Guidelines for the Investigation of Zoonotic Disease (England and Wales) www.hpa.org.uk/web/HPAwebFile/HPAweb_C/1240530336599

VLA - Non-Statutory Zoonoses Reports

www.defra.gov.uk/vla/reports/rep_surv_zoonoses.htm

Zoonoses (Palmer, Soulsby and Simpson) OUP ISBN 0 19 262380 X

Disease specific further reading:

Can also be found at the end of each A-Z section.

Appendix 8: Sources of data

Human infection

Surveillance centre	Surveillance area
Health Protection Agency Centre for Infections (HPA CfI), London	England and Wales
Health Protection Scotland (HPS), Glasgow	Scotland
Public Health Agency (PHA), Belfast	Northern Ireland
Communicable Disease Surveillance Centre, Public Health - Wales, Cardiff	Wales

Each of the surveillance centres in the UK (above) collect, collate and disseminate information from a variety of sources in the surveillance of infections in humans.

There are four main sources of data for zoonotic infection in the human population. These are:

- Notification of infectious diseases
- National surveillance schemes for laboratory-confirmed infections
- National surveillance schemes for general gastrointestinal outbreaks
- Enhanced surveillance for specific zoonoses

As discussed in the introduction, the notification of diseases will change in 2010 and a fuller description of the new systems in place will be given in the 2010 report.

Animal infection

The principle organisations in the UK collecting and collating surveillance information on zoonoses in animals are:

- Department for Environment, Food and Rural Affairs (Defra)
- Veterinary Laboratories Agency (VLA)
- Scottish Agriculture College (SAC)
- Meat Hygiene Service (MHS)
- Department of Agriculture and Rural Development, Northern Ireland (DARDNI)
- Agri-Food and Biosciences Institute, Northern Ireland (AFBI)

Information may also be available from universities, veterinary research organisations, and other private veterinary laboratories.

The sources of data on animal infections can be broadly divided up as follows:

- Reports of notifiable disease
- Laboratory reports of organisms specified in the relevant legislation
- Reports from statutory monitoring and control programmes

- Scanning surveillance reports from government veterinary laboratories on diagnostic samples submitted by private veterinary surgeons (mainly from farms)
- Reports from inspections carried out at abattoirs
- Reports from specific targeted surveillance and research studies
- Investigations carried out jointly with HPA into zoonotic incidents

The main characteristics of each of the sources of animal and human data have been detailed in previous UK Zoonoses reports and are available on the websites of each centre.

Appendix 9: List of Abbreviations/ Acronyms

ACMSF	Advisory Committee on the Microbiological Safety of Food
AFBI	Agri-Food and Biosciences Institute
AH	Animal Health
BIP	Border Inspection Posts
BSE	Bovine Spongiform Encephalopathy
bTB	Bovine Tuberculosis
CCDC	Consultant in Communicable Disease Control
CDC	Centres for Disease Control and Prevention (United States)
Cfi	Centre for Infections (HPA)
CJD	Creutzfeldt-Jakob Disease
DARD(NI)	Department of Agriculture and Rural Development (Northern Ireland)
Defra	Department for Environment, Food and Rural Affairs
DNA	Deoxyribonucleic acid
EAE	Enzootic Abortion of Ewes
EBLV(-2)	European Bat Lyssavirus (Type 2)
EC	European Commission
EFIG	Epidemiology of Foodborne Infections Group
EFSA	European Food Safety Authority
ELISA	Enzyme-Linked Immunosorbent Assay
EU	European Union
FDA	Food and Drug Administration
FSA	Food Standards Agency
GB	Great Britain (England, Wales, Scotland)
HAIRS	Human, Animal Infections and Risk Surveillance Group
HIV	Human immunodeficiency virus
HPA	Health Protection Agency
HPAI	Highly Pathogenic Avian Influenza
HPS	Health Protection Scotland
HSE	Health and Safety Executive
HUS	Haemolytic Uraemic Syndrome
ICT	Incident Control Team (multinational/ multiagency)
LACORS	Local Authority Co-ordinators of Regulatory Services
LPAI	Low Pathogenic Avian Influenza
MHS	Meat Hygiene Service
NaTHNaC	National Travel Health Network and Centre
NCP	National Control Programme for Salmonella in Poultry
NI	Northern Ireland
OBF	Officially Brucellosis Free
OCT	Outbreak Control Team
OIE	World Organisation for Animal Health

PCR	Polymerase Chain Reaction
PETS	Pet Travel Scheme
PFGE	Pulsed Field Gel Electrophoresis
PHA	Public Health Agency (Northern Ireland)
RADAR	Rapid Analysis & Detection of Animal-related Risks
pdmH1N1	Pandemic 2009 H1N1 Influenza
PT4	Phage Type 4
RIDDOR	Reporting of Injuries, Diseases and Dangerous Occurrences Regulations (HSE)
RT-PCR	Reverse Transcriptase-Polymerase Chain Reaction
SAC	Scottish Agriculture College
SGDIA	Surveillance Group on Disease and Infections in Animals
SI	Swine Influenza
SNH	Scottish Natural Heritage
TB	Tuberculosis
TSE	Transmissible Spongiform Encephalopathy
UK	United Kingdom (England, Wales, Scotland, Northern Ireland)
UKZADI	United Kingdom Zoonoses, Animal Diseases and Infections Group
UKZG	United Kingdom Zoonoses Group (forerunner of UKZADI)
US	United States
VIDA	Veterinary Investigation Diagnosis Analysis Database
VLA	Veterinary Laboratories Agency
VTEC	Verocytotoxigenic <i>Escherichia coli</i>
WAG	Welsh Assembly Government
WHO	World Health Organisation
ZNCP	Zoonosis National Control Programme for Salmonella in Pigs

Appendix 10: Acknowledgements

This report was produced by a small group formed under the Chairmanship of Dilys Morgan. The group contained representatives of, or received assistance from, the following organisations:

Department for Environment, Food and Rural Affairs (Defra)

Area 4A, Nobel House, 17 Smith Square, London SW1P 3JR

www.defra.gov.uk

Department of Agriculture and Rural Development (Northern Ireland) (DARDNI)

Dundonald House, Upper Newtownards Road, Belfast BT4 3SB www.dardni.gov.uk

Department of Health

Skipton House, 80 London Road, Elephant and Castle, London SE1 6LW

www.dh.gov.uk

Department of Health, Social Services & Public Safety (Northern Ireland)

Castle Buildings, Stormont, Belfast BT4 3SJ

www.dhsspsni.gov.uk

Food Standards Agency (FSA)

Aviation House, 125 Kingsway, London WC2B 6NH

www.food.gov.uk

Health Protection Agency (HPA)

Centre for Infections, 61 Colindale Avenue, London NW9 5EQ

www.hpa.org.uk/

Health Protection Scotland (HPS)

Clifton House, Clifton Place, Glasgow G3 7LN

www.hps.scot.nhs.uk

Leptospira Reference Unit

(HPA Collaborating Laboratory)

Department of Microbiology and Immunology, County Hospital, Hereford HR1 2ER

www.hpa.org.uk/ProductsServices/InfectiousDiseases/LaboratoriesAndReferenceFacilities/LeptospiraReferenceUnit/

Lyme Borreliosis Unit

Southampton HPA Laboratory Level B South Laboratory Block, Southampton

General Hospital, Southampton SO16 6YD

www.hpa.org.uk/ProductsServices/InfectiousDiseases/LaboratoriesAndReferenceFacilities/LymeBorreliosisUnit/

Public Health Agency (Northern Ireland)

18 Ormeau Avenue, Belfast, BT2 8HS

www.publichealth.hscni.net/

Public Health Wales

Communicable Disease Surveillance Centre, Health Protection Division, The Temple of Peace and Health, Cathays Park, Cardiff CF10 3NW

www.wales.nhs.uk/sitesplus/888

Scottish Agricultural College

West Mains Road, Edinburgh EH9 3JG

www.sac.ac.uk

Scottish Salmonella Reference Laboratory

North Glasgow University Hospitals NHS Trust, 133 Balornock Road, Glasgow G21 3UW

www.ssrl.scot.nhs.uk/

Scottish *E. coli* O157/VTEC Reference Laboratory (SERL)

Department of Clinical Microbiology, Western General Hospital, Crewe Road Edinburgh EH4 2XU

www.hps.scot.nhs.uk/reflab/RefLabDetail.aspx?id=13

Scottish Government, Rural Directorate

Pentland House, 47 Robb's Loan, Edinburgh EH14 1TY

www.scotland.gov.uk

Toxoplasma Reference Unit

(HPA Collaborating Laboratory)

Public Health Wales, Microbiology Swansea, Singleton Hospital, Sketty, Swansea SA2 8QA

www.wales.nhs.uk/sites3/page.cfm?orgId=457&pid=25359 and

www.hpa.org.uk/ProductsServices/InfectiousDiseases/LaboratoriesAndReferenceFacilities/ToxoplasmaReferenceLaboratory/

UK Cryptosporidium Reference Laboratory

(HPA Collaborating Laboratory)

Public Health Wales, Microbiology Swansea, Singleton Hospital, Sketty, Swansea SA2 8QA

www.wales.nhs.uk/sites3/page.cfm?orgId=457&pid=25284 and

www.hpa.org.uk/ProductsServices/InfectiousDiseases/LaboratoriesAndReferenceFacilities/CryptosporidiumReferenceUnit/

Veterinary Laboratories Agency (VLA)

New Haw, Addlestone, Surrey KT15 3NB

www.defra.gov.uk/vla/

Welsh Assembly Government (WAG)

Cathays Park, Cardiff, CF10 3NQ

www.wales.gov.uk