



FSAS project FS245027 - Microbiological risks from the production and consumption of uneviscerated small game birds compared to eviscerated small game birds: A qualitative risk assessment

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Executive Summary

In this project we have developed a qualitative risk assessment in order to assess the microbiological risks from the production and consumption of uneviscerated small game birds, compared to eviscerated small game birds. The risk assessment considers nine different species of game birds: snipe, woodpigeon, woodcock, mallard, teal, widgeon, grey partridge, red-legged partridge and quail. The risk to the consumer from each species was assessed for six pathogens that were considered to be of most concern to human health from consumption of small game birds. These were *Campylobacter*, *Salmonella*, *Escherichia coli* (both antibiotic resistant strains, such as those with ESBL genes that confer resistance to cephalosporins, and toxicoinfectious strains such as VTEC O157), *Toxoplasma gondii*, *Chlamydophila psittaci* and *Listeria monocytogenes*. For each pathogen/species combination, four overall qualitative risk scores were estimated for the risk to individuals from consuming: eviscerated birds in the home, eviscerated birds outside of the home, uneviscerated birds in the home and uneviscerated birds outside the home. A number of factors contribute to the overall risk, including likelihood of human infection, severity of human infection and frequency of human consumption. Thus, the overall risk estimates are a qualitative indication of the relative level of risk associated with the pathogen/species combinations and do not represent an actual quantity. The overall risks are relative to each other, so a pathogen/species combination with a *Very Low* score would be considered less risky than one with a *Low* score.

The results of the risk assessment suggested that while large outbreaks of zoonotic infection among UK consumers due to small game bird production and consumption are unlikely, sporadic individual infections may occur due to combinations of 'rare-event hygiene-related issues' in the 'field-to-fork' chain (e.g. the lead shot puncturing the gut wall and the bird being hung for a long period of time at temperatures that would allow the growth of the pathogen) and/or inadequate cooking of the game bird in or outside the home. The overall risks to the UK consumer for the majority of the pathogens/species considered were *Very Low*. The highest risk was for *Campylobacter* due to consumption of eviscerated woodpigeon and mallard outside the home, which was assessed to be of *Low-Medium* risk. The high number of these two bird species consumed and the ability of *Campylobacter* to infect humans at low levels, combined with the tendency to eat gamebird meat 'pink' outside the home all contribute to the increase in risk for these pathogen/species combinations.

The evidence gathered did not suggest that there was much greater risk associated with consumption of uneviscerated game birds, compared with eviscerated game birds. In some pathogen/species combinations the evidence even suggested that the risk from eviscerated game birds may be slightly higher. This was due to the indication that the risk of cross-contamination resulting from the evisceration process would outweigh the reduction in number of pathogenic organisms due to removal of the viscera.

For uneviscerated birds, the highest combined risks to the consumer were *Medium* for mallard consumed outside the home and *Low-Medium* for woodpigeon and red-legged partridge consumed both in and outside the home. However, evidence suggests that the frequency of consumption of uneviscerated products for these species was *Negligible-Very Low* and thus the overall risk estimate for these birds was estimated to be *Very low*. If there is an increased frequency of consumption of these birds in the future, then this overall risk should be re-examined.

The wild game industry is not as stringently regulated as other farmed livestock industries and it is no surprise that the availability and quality of data are lacking in some areas. In general there was a suitable level of expert opinion knowledge available to assess the overall risk, but this is not always sufficient to get an accurate indication of the potential variability of UK wide processes. As such, we have highlighted the following areas in which insufficient knowledge has contributed to a data gap/deficiency:

- Small sample size studies of limited statistical design and representativeness, leading to great uncertainty on prevalence of pathogens in all species, in particular, woodcock and snipe
- The pathogen load in live game birds
- Information on the strains within bacterial species that are present in wild birds that are pathogenic to humans
- The numbers of birds that are processed through the different distribution pathways
- The numbers of consumers that shoot and process their own birds, and the quantity of such birds used in this way
- The frequency and volume of consumption of uneviscerated game birds
- The probability/level of pathogen cross-contamination at the various framework stages e.g. game larder, game handling establishment, and restaurant.
- The survival/ growth behaviour of pathogens (both in and on the carcass) during the framework pathway, in particular, during hanging of carcasses which is commonly practised for some bird species.
- Prevalence of pathogens on prepared game birds

The significance of these data gaps/deficiencies on the overall results should be considered when interpreting the findings of this risk assessment. Additionally, as this is a growing industry, it is recommended that the conclusions of this assessment are periodically revisited to assess whether significant changes have occurred that would affect the findings.

Based on the current level of knowledge, the conclusion from this risk assessment is that there are risks of zoonotic infection to the consumer associated with preparation and consumption of both eviscerated and uneviscerated small game birds. However, assuming a general level of compliance with regulations and basic hygiene practices, these risks are low

and are unlikely to be responsible for anything more than sporadic infection events in consumers.

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Glossary

AFO:	Authorised Food Officer
AGHE:	Approved Game Handling Establishment
BASC:	British Association for Shooting and Conservation
BTO:	British Trust for Ornithology
CA:	Countryside Alliance
DEFRA:	Department for Environment, Food and Rural Affairs
EC:	European Commission
EFSA:	European Food Safety Authority
FB:	Food business means any undertaking, whether for profit or not and whether public or private, carrying out any of the activities related to any stage of production, processing and distribution of food,
FBO:	Food business operator means the natural or legal persons responsible for ensuring that the requirements of food law are met within the food business under their control.
FSA:	Food Standards Agency
FSAS:	Food Standards Agency Scotland
GWCT:	Game and Wildlife Conservation Trust
HACCP:	Hazard Analysis Critical Control Point: a systematic preventative approach to food safety that identifies biological (and other) hazards in the production process and aims to reduce these risks to a safe level through the identification of critical control points and verification procedures
LA:	Local Authority
RSPB:	Royal Society for the Protection of Birds

1. Introduction

The production and consumption of wild game birds is a major industry in Scotland. Since the beginning of the 21st Century the wild game sector has evolved from what has historically been viewed as a minority sport to a food production industry in its own right. Wild game has often been viewed as a by-product of shooting, costing in the region of £45/bird to shoot translating to a value of a few pounds 'ready-to-eat' (ADAS 2005). The industry has recognised that for better prices it needs to consider game as a foodstuff, rather than a by-product, in terms of supplying a guaranteed quality product, something in which it had previously lagged behind other food sectors. Promotion by celebrity chefs, better marketing and increasing use of farmers' markets and mail order supply has meant that more people are now buying and eating wild game. At the same time, the low fat, healthy eating properties of game bird meat and its free range reputation have made it popular with today's consumer. Sales of game have increased by 64% since 2002 (Alliance 2008), concurrent with the initiation of the Countryside Alliance's Game-to-Eat campaign which is 'dedicated to the eating and enjoyment of British wild game'. In 2009 the game market was worth a projected £75 million (FSAS 2012b), of which feathered game made up approximately 28%.

Whilst the wild game industry has seen an expansion over recent years, research undertaken with a representative sample of UK adults estimates that only 5% claim to eat game 'fairly regularly' in season (Mintel 2008). This figure is slightly higher in Scotland (7%) equating to ~350,000 people. It is estimated that 200,000 people are involved in shooting wild game in Scotland (PACEC 2006), and it could be assumed that a high proportion of those consuming game are likely to be the hunters themselves and their families (FSAS 2012b).

The game meat supply chain differs from the conventional practices of the farmed meat industry. The slaughter process is less controlled than for domesticated species and the microbiological conditions of game meat can be compromised by primary production. Location of shot within the carcass, evisceration, hygiene and maintenance of the cold chain can all affect proliferation of contaminating organisms within game meat. New European Commission (EC) regulations, brought into force on January 1st 2006, to address these considerations, require that wild game is now produced, stored and processed to the highest standard possible (FSA 2011).

Wild game birds, like other livestock species, can carry, or be infected with, pathogens that can adversely affect the health of humans. Unlike farmed animals the dietary and migration habits of birds can influence their role in

the international spread of zoonotic infection (Kobayashi, Kanazaki *et al.* 2007); (Hubalek 2004); (Abulreesh 2007), although their population density and age mitigates against high-level carriage of foodborne bacterial pathogens within this population. Whilst it is possible for game birds to be clinically infected with some of these pathogens, and therefore identifiable to the hunter as a sick bird, the majority of infections are asymptomatic; it is these infections which pose the greatest risk to the consumer as they are more likely to pass through the food chain to the consumer undetected. Birds carrying pathogenic bacteria in their intestines can pose a direct risk of human infection via consumption of undercooked meat and can also disseminate pathogens into the food processing environment. The zoonotic risk posed by wild game birds as a food source is hard to quantify as there are few comprehensive studies on the presence of pathogens within this population. Such studies have frequently been conducted for farmed livestock e.g. VTEC O157 in cattle (Hussein 2007), *Salmonella* in pigs (Fosse, Seegers *et al.* 2009) and *Campylobacter* in chickens (Young, Rajic *et al.* 2009). Similar studies for the wild game sector would provide useful information to help identify appropriate control measures for zoonotic pathogens.

Prior to the introduction of the 2006 Food Hygiene Regulations, the Food Standards Agency (FSA) commissioned a qualitative risk assessment to address what the risks to human health from the handling/consumption of wild game meat were and how the proposed EC hygiene proposals would affect those risks (Coburn 2003). Non-negligible risks to human health from game birds were assessed to be associated with *Campylobacter jejuni*, with exposure through accidental ingestion during handling or consumption of contaminated meat, and *Chlamydomphila psittaci* from handling game birds. The report concluded that EC regulations requiring game meat plants to control hazards using Hazard Analysis Critical Control Point (HACCP) principles would result in a decrease in risk via a reduction in cross contamination.

Removal of the viscera is normal practice in the production of gamebirds. However, there is a specialised market for consumption of uneviscerated small game birds of certain species within the UK. Traditionally, small game birds such as woodcock and snipe have been cooked with the intestines intact and the viscera often ingested as part of the final dish. The viscera of birds infected with a pathogen may contain high concentrations of the organism and so consumption of the viscera could put the consumer at a higher risk of infection than consumption of an eviscerated bird. This risk would depend upon the cooking step, in any consideration, and whether this is sufficient to reduce the pathogen count to below that required for a dose response within the consumer. Conversely, the process of evisceration is known to be a risk for cross contamination by pathogens (at least in farmed livestock) to other birds, other food products within the food processing environment and to individuals carrying out the evisceration. It is not uncommon for the consumers to eviscerate wild game birds themselves, and the risk of intestinal rupture and consequent spillage of contents onto the carcass and operators' hands during this process is high.

There has been no formal assessment of the potential risks of human infection due to the production and consumption of uneviscerated small game birds and how these risks compare to consumption of eviscerated birds. Hence, there has been no formal consideration of what, if any, modifications to hygiene regulations might be required to control the risks to public health from the production and consumption of uneviscerated birds. Current EC regulations (853/2004 Annex 111) state that evisceration must be carried out, or completed, without undue delay upon arrival at the game handling establishment, unless the competent authority permits otherwise. Exemptions can, and do, occur at the discretion of the FSA for specific requests from Approved Game Handling Establishments (AGHE). Private and domestic consumption is also exempt from this regulatory stipulation.

These issues were factors in the decision for the Food Standard Agency for Scotland (FSAS) to put out a research call to address the risk question:

What are the microbiological risks to the consumer from the production and consumption of uneviscerated small game birds compared to eviscerated small game birds?

The risk assessment discussed here focuses on the microbiological risks to the consumer during production and consumption of eviscerated and uneviscerated small game birds both in the 'home' and 'outside the home'. For home consumption the consumer can have a more active role in preparation of the bird, possibly even shooting it themselves, but may also have purchased the bird from a local retailer or been given it as a gift or 'in-kind' payment for assisting at shooting events. For consumption outside the home, such as eating out at a restaurant or catered event, the consumer is not involved in the preparation.

It was decided at an early stage of the project to only consider the risk to the consumer and not to other people involved in the production/processing of the birds. However, if the consumer is directly involved in production/processing then this is considered.

This work aims to provide a qualitative assessment of the overall risk to consumer health and to highlight potentially appropriate steps in the chain where control measures could be implemented. In the course of the assessment we endeavour to highlight any relevant data gaps that may significantly affect the final risk estimates.

The risk assessment considers nine different species of game birds: snipe, woodpigeon, woodcock, mallard, teal, widgeon, grey partridge, red-legged partridge and quail. For the purpose of this project the term 'wild birds' will include birds that have been hatched/reared under controlled conditions before being introduced into the wild, in accordance with the definition in Regulation (EC) No 853/2004. 'Farmed birds' refer only to

those birds which remain on a commercial poultry farm until slaughter which, in this report, will only refer to quails. Whilst quail are regarded as farmed birds and not game from the point of view of production it is possible that they are regarded as game from the consumer's point of view and therefore treated as such when it comes to preparation and cooking. Wild game birds must have been killed by hunting if they are to be supplied for human consumption.

2. Materials and Methods

2.1. Overview

The risk assessment was conducted using elements from both the World Organisation for Animal Health (OIE) code for import risk analysis (OIE 2004) and the Codex Alimentarius Commission guidelines (CAC 1999) with the inclusion of a preliminary hazard identification stage. Under traditional OIE guidelines, there are three components of risk assessment: release assessment, exposure assessment and consequence assessment. While we are aware that Codex (CAC 1999) is more generally used for food safety risk assessments, we feel that the OIE pathway is more applicable to this qualitative risk assessment, as there is a distinct separation between the 'release' of the pathogen and the subsequent exposure of humans. The main points to consider at each stage of the assessment are:

(1) Hazard Identification - assessment of all relevant hazards to identify the major microbiological hazards that current knowledge suggests will be of public health concern due to the production and/or consumption of wild game birds (not including occupational hazards).

(2) Release assessment - assessment of the prevalence and microbiological load of the identified hazards in both eviscerated and uneviscerated wild game birds throughout the processing chain. The main factors include the bird species (which ones are natural hosts and pose a higher risk of being infected than other species) and the pathogenic load per bird.

(3) Exposure assessment - assesses the absolute risk of consumer exposure from contact with wild game birds for each hazard taking into account the pathways necessary for exposure of consumers to the hazard and the probability of the exposure occurring.

(4) Consequence assessment - assessment of the relative risk to public health from both eviscerated and uneviscerated small game birds for all hazards identified. The absolute risk to public health from consumption of all species is assessed, to set in context the relative difference in risk between eviscerated and uneviscerated birds.

For this risk assessment qualitative estimates were produced using the following definitions, which have been used in previous assessments (EFSA 2006)

Table 1: Definitions of qualitative risk assessment scores (EFSA, 2006)

Term	Definition
Negligible	So rare that it does not merit to be considered
Very Low	Unlikely to occur
Low	Rare, but may occur occasionally
Medium	Occurs regularly
High	Occurs very regularly
Very High	Is almost certain to occur

The information summarised in this report was collated from a range of different sources including:

- Email communication with scientific experts, policy makers and industry representatives
- European Food Safety Authority (EFSA) scientific opinions and risk assessments
- European Commission Regulations
- Scientific literature via searches in the ISI Web of Knowledge, Google search engines and references within other documents.

The focus of the risk assessment was primarily on Scotland, with a view that it would generally be applicable to the whole of the UK. Evidence and data from other countries have been used where appropriate and useful, or where Scottish data were lacking. Where published data were lacking, expert opinion was sought. Where we have been unable to find peer reviewed literature on the presence of pathogens in a particular bird species the search has been widened to include incidence in birds from the same family.

2.2. Hazard Identification/Pathogen Selection

2.2.1. Overview

The aim of the Hazard Identification step was to identify the major microbiological hazards that current knowledge suggests will be of public health concern due to the production and/or consumption of small game birds (not including occupational hazards). We first conducted a review to identify all the major microbiological hazards present in small game birds (the full list included 87 hazards and is provided in Appendix 3). We then conducted an analysis to detail the reasons for inclusion or exclusion in the full risk assessment. The hazard list and selection of final hazards was developed and agreed by the project steering group before final agreement by FSAS.

2.2.2. Compiling the full list of hazards

As a starting point we considered the hazards identified in a previous wild game risk assessment (Coburn 2003). We then conducted a literature search of peer reviewed publications using web of knowledge and non-peer reviewed articles using Google. Finally, we sought expert opinion from the project steering group and other experts. The project steering group and representatives from FSAS drew up a list of groups and individual experts involved in the wild game sector that could be expected to provide relevant information for the project. Where information was available, each hazard was categorised according to information on the type of clinical signs in the birds, the species affected, the level of infection (e.g. high morbidity/mortality), the potential for zoonotic transmission, presence in Great Britain, reported human cases, and human symptoms.

A recent EFSA scientific opinion on poultry meat inspection (EFSA 2012a), provided useful validation of the hazard list as it reported on a qualitative risk assessment that considered a large number of microbiological hazards and identified *Campylobacter* spp., *Salmonella* spp. and ESBL/AmpC gene-carrying bacteria as the most relevant biological hazards in the context of meat inspection for poultry. However, it was recognised that there are significant differences between poultry and small game bird production which needed to be taken into account when considering the relevance of the EFSA list to this project.

2.2.3. Selection of hazards for a short list

Figure 1 shows a decision tree outlining critical characteristics of the hazards that were used to determine whether they should be included in the short list or not.

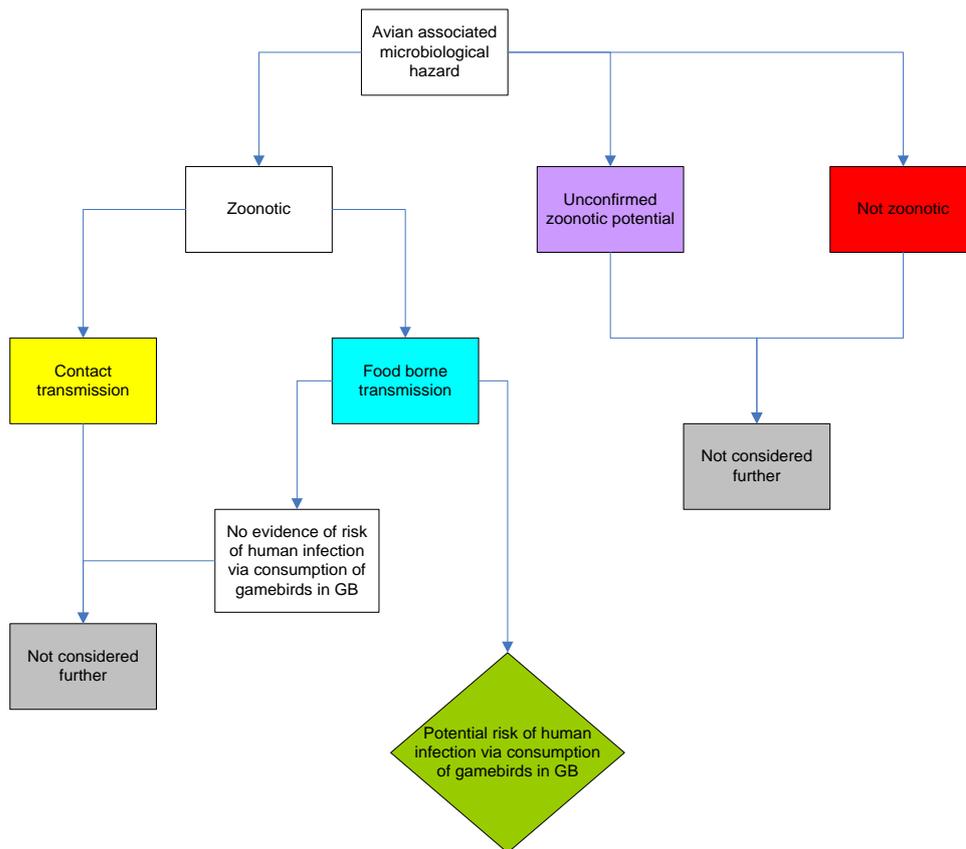


Figure 1: Decision tree outlining critical characteristics of the microbiological hazards when considering them for inclusion in the short list.

2.2.4. Selection of final list of hazards

The short list consisted of 18 hazards. Of these hazards, six were identified as being of most relevance to small game birds in Great Britain and therefore considered in the full risk assessment:

- *Campylobacter* spp.
- *Escherichia Coli* (toxicoinfectious strains including VTEC)
- *Escherichia Coli* with ESBL or AmpC based resistance to extended spectrum cephalosporins
- *Salmonella* spp.
- *Listeria monocytogenes*
- *Toxoplasma gondii*

After consultation with FSAS, it was agreed that the risk assessment would also look at *Chlamydophila psittaci*, to include a contact/inhalation pathogen, which may have different associated risks.

Table 2 shows the hazards on the short list and the reasons for inclusion/exclusion from the final list.

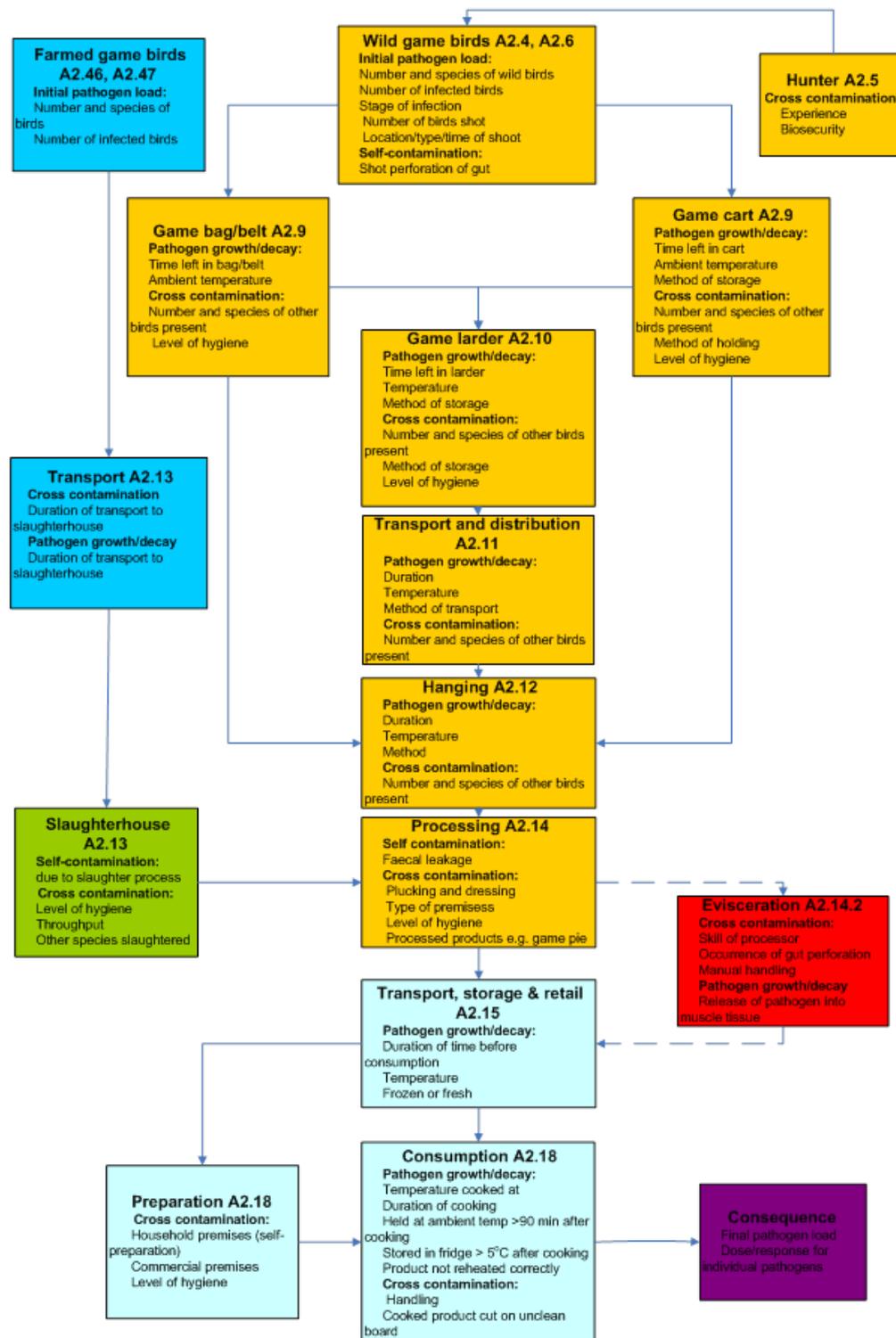
Table 2: Short list of microbiological hazards and reasons for inclusion/exclusion in full risk assessment. Hazards taken forward to the final risk assessment are highlighted in green.

Hazard	Inclusion/ exclusion characteristics
<i>Aeromonas hydrophila</i>	Generally ubiquitous in the environment with most human infections due to consumption of contaminated seafood or water.
<i>Bacillus cereus</i>	Ubiquitous pathogen; zoonotic transmission is usually as a result of improper hygiene during food preparation.
<i>Campylobacter</i> spp.	Organism is a frequent cause of infection in humans and has been found on gamebird carcasses.
<i>Clostridium botulinum</i> (Mostly Type C) poison	No cases of human infection of botulism since 2005 in the UK. Although <i>C. botulinum</i> is found in waterfowl, infection in humans is usually associated with preserved long life foods and different toxin types.
<i>Clostridium perfringens</i> (Type A and C)	Ubiquitous pathogen; zoonotic transmission is usually as a result of improper hygiene during food preparation.
<i>Escherichia coli</i> (toxicoinfectious strains including VTEC)	Organism is a serious cause of infection in humans and has been found on gamebird carcasses.
<i>Escherichia coli</i> (with extended spectrum cephalosporin resistance)	Occurrence is moderate to high in poultry and could be present in gamebirds, especially those that are reared industrially and exposed to antibiotic treatments. This would represent a source of human exposure.
<i>Listeria monocytogenes</i>	Organism is found in wild avian species and is able to grow at low temperatures with the potential to increase on the carcass surface during hanging, especially if moisture is present, and in the gut.
<i>Salmonella</i> spp.	Organism is a frequent cause of infection in humans and has been found on gamebird carcasses.
<i>Staphylococcus aureus</i>	Infection is usually transmitted to the cooked product by a human carrier and increases via subsequent temperature abuse. Animal strains are not usually associated with <i>S. aureus</i> food poisoning incidents.
<i>Yersinia enterocolitica</i>	Very few human cases of infection are reported annually and infection is usually associated with consumption of pig meat or

Hazard	Inclusion/ exclusion characteristics
	pig meat products.
<i>Yersinia pseudotuberculosis</i>	Relatively rare as a source of human infection. The origin of infection is typically difficult to identify, documented cases have been in milk and salad leaves.
<i>Capillaria</i> spp.	<i>C.philippinensis</i> is the only species of zoonotic significance and is found only occasionally in Europe in fish and fish eating birds. Human infection is mainly via consumption of raw or undercooked fish.
<i>Plesiomonas shigelloides</i>	Occurs mainly in tropical and sub-tropical areas and human infection is usually via consumption of contaminated water or raw shellfish.
<i>Toxocara canis</i>	Human infection is usually via accidental ingestion of eggs from canine faeces.
<i>Toxoplasma gondii</i>	Outdoor free ranging habitat of gamebirds is likely to expose them to infection via cat faeces. Reports of infection in free-range chickens (Dubey, 2010) show a risk of transmission to humans.

2.3. Model Framework

Figure 2 shows the detailed framework outlining the different potential pathways from the shot game bird to the consumer. The framework provides an outline of the pathway the game bird follows from the field to the consumer in terms of the stages and processes it undergoes. It does not specify any delineation with regards to 'in or outside the home'. This separation is dealt with in Appendix 1, where the processes are specifically dealt with in these two separate environments. Each section of the detailed framework is assigned a number, which is used as a reference throughout the document. Within each stage the figure shows the risk factors to be considered. These factors are subdivided depending on how they will affect consumer exposure either by increasing pathogen load or by providing potential for cross-contamination. For example, in the game larder, factors such as the time spent in the larder and the temperature could affect growth/decay of the pathogen, while factors such as presence of other birds and level of hygiene may affect the risk of cross-contamination. In some cases a risk factor may have multiple effects, for example, the heaping of birds in the game cart (method of holding) could result in both cross-contamination and growth of pathogens due to body heat maintained by the close proximity of the birds. Each risk factor was qualitatively assessed with regards to their contribution to consumer exposure and an overall risk for each pathway was determined.



Key	Description	Risk Assessment stage
	Farmed game birds only	Release Assessment
	Wild game birds only	Release Assessment
	Slaughterhouse	Release Assessment
	Evisceration	Release Assessment
	Both uneviscerated and eviscerated birds will be considered in these stages	Exposure Assessment
	Overall risk	Consequence Assessment

Figure 2: Detailed model framework including description of risk factors.

Figure 3 details the distribution pathway for the game bird industry and an indication of the percentage of wild game birds that follow each route. The wild game bird industry has a complex structure involving a variety of distribution pathways under different regulatory controls and inspection remits. In addition, the regulations themselves are complex and allow for exemptions and variable interpretation affecting both duration and temperature control within the risk framework (Figure 2). Compounding this complexity is the lack of knowledge on the actual numbers of birds entering the pathway and the subsequent numbers which go down the individual pathway routes. Data for the numbers of game birds which follow each supply route are based on evidence from interviews with industry representatives and obtained from previously published reports (FSAS 2012a); (FSAS 2012b); (PACEC 2006)) and should not be considered to be a completely accurate representation of current practices. The pathway indicated by the red line in Figure 3 shows those birds which are estate-shot, supplied to an AGHE for dressing and then redistributed via the estates for local or online sale.

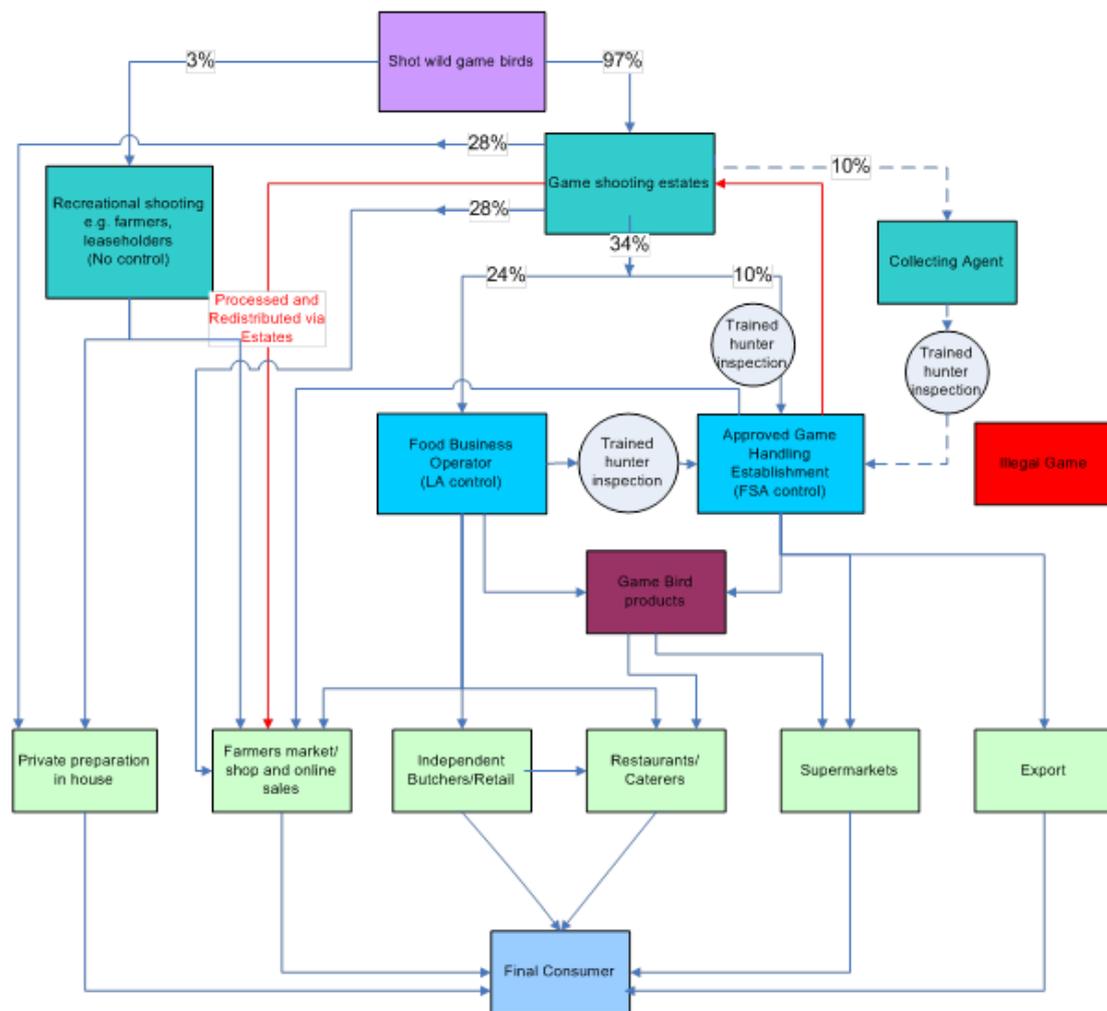


Figure 3: Distribution pathway for wild game birds

2.4. Format of assessment

2.4.1. Release & Exposure assessments

Data were collected for each pathogen/bird species combination, for each stage of the risk assessment (as outlined in Figure 2). These data include information on the survival, growth and cross-contamination of the pathogen at each stage (e.g. during storage in game larders or at retail, during transport and processing) and were used to assess the likelihood and degree of a change in prevalence and concentration of the pathogen during each stage of the pathway, in the medium in question (e.g. live bird, carcass, meat product).

Both pathogenic prevalence and concentration are important and should be considered independently; it is not only the presence of a pathogen that dictates whether a human will become infected but also the dose ingested. For example, if the prevalence is high, but the concentration is low then humans may not always get infected from consuming contaminated products depending on the dose response; the risk to human health in this scenario will be much lower than if both the prevalence and the concentration of the pathogen are high, or the dose response is low.

At the end of each stage we estimate two qualitative scores: for the prevalence and concentration of the pathogen. For the prevalence score we combine the prevalence score at the end of the previous stage with the information on the risk of a change in prevalence during the current stage. A similar method is followed for the concentration score. There are many different methods in the literature for combining qualitative scores in a risk assessment, such as the methods used in a previous risk assessment on wild game (Coburn, Snary *et al.* 2005), and the 'risk matrix' approach (Gale, Brouwer *et al.* 2010). The latter approach relies on the scores being treated like probabilities so they can be 'multiplied' together with the resulting probability being equal to or lower than the lowest probability. For this risk assessment we predominantly follow the methodology employed by (Coburn, Snary *et al.* 2005), but adapt as necessary when our framework differs.

For the first stage of the risk assessment we need to estimate the initial prevalence and concentration in the wild game populations from available data. Whilst data are available on the prevalence of pathogens in game birds, no reliable data on pathogenic load was found during the course of this project. Thus, estimates of initial pathogen concentrations are based on the qualitative data for prevalence and given the same qualitative score; an assumption accepted as a general rule of thumb by the project steering group since within-group prevalence and mean numbers of organisms carried are normally related and, given the absence of any better information. Nevertheless, this is still considered a significant gap.

From the evisceration stage onwards we also assess the risk of eviscerated and uneviscerated birds separately (prior to this stage we assume that there is no difference in risk).

2.4.2. Consequence assessment

At the end of the exposure assessment we have four routes which have scores for the prevalence and concentration on the product at the point of consumption: eviscerated birds in the home, eviscerated birds outside the home, uneviscerated birds in the home and uneviscerated birds outside the home.

These scores were then combined with data relating to the numbers of organisms required to cause an adverse effect on human health (i.e. dose-response), the frequency of consumption of the particular species and the severity of potential human illness (e.g. if a pathogen X is thought to cause the same number of human infections as pathogen Y, but those infections are more severe, we would consider there to be greater risk associated with pathogen Y) to determine an overall assessment of the consumer risk from handling and consumption of the wild game species. This process is outlined in Figure 4.

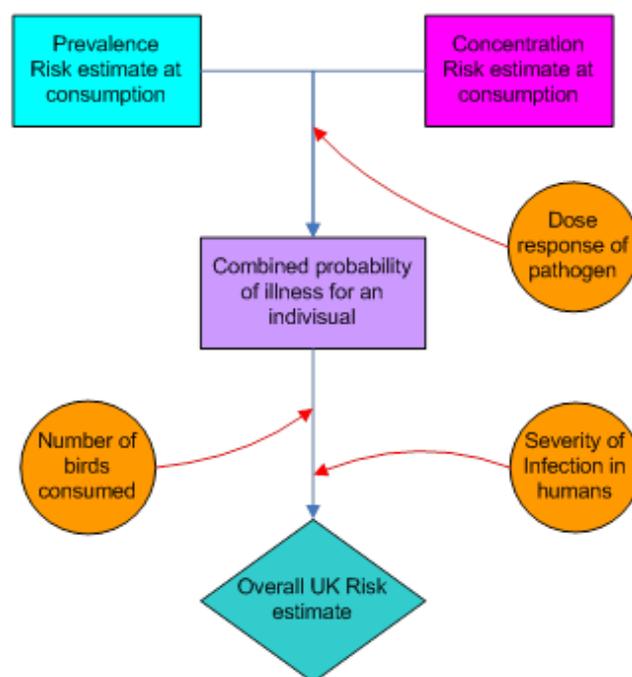


Figure 4: Schematic illustration of risk calculation from prevalence/concentration risk estimate at consumption to overall risk of human infection

The final outputs of the risk assessment are:

- The relative risk relating to human infection with pathogen y, from consumption of eviscerated small game bird species x.
- The relative risk relating to human infection with pathogen y, from consumption of uneviscerated small game bird species x.

Note that the overall risk estimates should be interpreted as a level of concern about consumer infection for a particular pathogen/species combination. The risks are relative to each other and do not actually represent a real life quantity such as probability or severity of illness or number of human cases per year, but these are all factors that contribute to the overall risk. The overall risk estimates can be compared against each other; if the overall risk estimate for one pathogen/species is higher than for another then the conclusion would be that there is greater risk associated with it. Similarly, if the uneviscerated risk estimate for a particular pathogen/species is higher than the eviscerated risk estimate then the conclusion would be that there is greater risk associated with the uneviscerated route.

3. Results

The overall risks for each pathogen/species combination are shown in Figure 5 - Figure 8. These results detail the risk to the consumer from preparation and consumption of eviscerated and uneviscerated products. Results detailing the change in risk throughout the whole pathway can be found in Appendix 1.

From these tables we can see that the highest risks to the consumer from eviscerated birds, after assessing the risk according to Figure 4, are:

- *Low-Medium Risk:*
 - *Campylobacter* due to consumption of woodpigeon and mallards outside the home
- *Low Risk:*
 - *Campylobacter* due to consumption of widgeon, red-legged partridge and quail outside the home
 - *Campylobacter* due to home consumption of woodpigeon, mallard, red-legged partridge and quail
 - *Toxoplasma gondii* due to consumption of mallard and red-legged partridge both in and outside the home

And from uneviscerated birds:

- *Very Low-Low Risk*
 - *Campylobacter* due to consumption of snipe and woodcock outside the home.

These figures suggest that the biggest risk to the consumer from wild game is from *Campylobacter* and *Toxoplasma gondii*. An increased risk of infection from these two pathogens was observed in mallard, red-legged partridge, quail, widgeon and woodpigeon. It is interesting to note that the first three

of these species have a high proportion of farm released birds whilst woodpigeon have a close association with human activities. The higher risk scores are likely to be skewed towards these birds because of:

- The increased prevalence of pathogens in these populations (although it is difficult to determine whether this is due to an increased number of studies in farmed birds because of their economic importance or whether this reflects a true increase in prevalence).
- The high number of birds consumed in these species groups

A *Low-Medium* risk is associated with *Campylobacter* in eviscerated woodpigeon and mallard consumed outside the home. These birds have a medium initial prevalence of *Campylobacter*, are eaten in large numbers and are more likely to be served 'pink' outside the home thereby not ensuring complete thermal inactivation of the bacteria at the time of consumption. The issue of undercooking is important when considering the fact that shot perforation of the gut can lead to microbial contamination of muscle tissue that would otherwise remain sterile (Geoff Mead pers. comm.) It is possible that the combination of muscle contamination and undercooking could result in a level of *Campylobacter* contamination high enough to cause infection in the consumer. The low dose response of *Campylobacter* in humans means that the risk of human infection is considered to be non-negligible.

Additionally, the risk of human infection with *Toxoplasma gondii* from eviscerated mallards and red-legged partridges was assessed as *Low*. This was a considered risk because of the high number of potentially infected birds consumed and the tendency to cook meat 'pink' which could result in tissue cysts retaining their viability after cooking. Although the dose response of *T. gondii* is unknown the severity of infection in humans in terms of longevity of symptoms is such that the risk is considered to be *Low* in these two species.

For woodcock and snipe the risk of infection from the pathogens considered, with the exception of *Campylobacter*, was estimated to be *Very low*. For *Campylobacter* the risk associated with eviscerated birds consumed both in and outside the home was considered to be *Very Low-Low*. Woodcock and snipe are wild, solitary birds and numbers consumed are small relative to woodpigeons, mallards and red-legged partridges. Expert opinion considers that these two species would have less exposure to pathogens than farm reared birds as they are considered to have little, if any, contact with humans or their environment.

Traditionally, woodcock have been eaten 'entire' as hunters view their habit of defecating before they fly as making their intestines 'safe' to eat. After roasting, the intestines are scooped out of the body cavity and fried with butter providing an additional exposure of the gut contents to high temperatures. Outside the home the overall risk of human infection with *Campylobacter* from snipe and woodcock was considered to be *Very Low-*

Low. The predilection for undercooking outside the home combined with the low infectious dose of *Campylobacter* and the known tendency of snipe and woodcock to be consumed uneviscerated increase the risk to the individual from *Very Low* to *Very Low-Low*. For all other uneviscerated birds the risk of human infection for all pathogens is *Very Low*. Whilst we have been unable to find any evidence of consumption of uneviscerated birds, other than of snipe and woodcock, this should not be taken as evidence of absence of such consumption occurring. The risk cannot therefore be considered as *Negligible* as the number of birds consumed remains unknown.

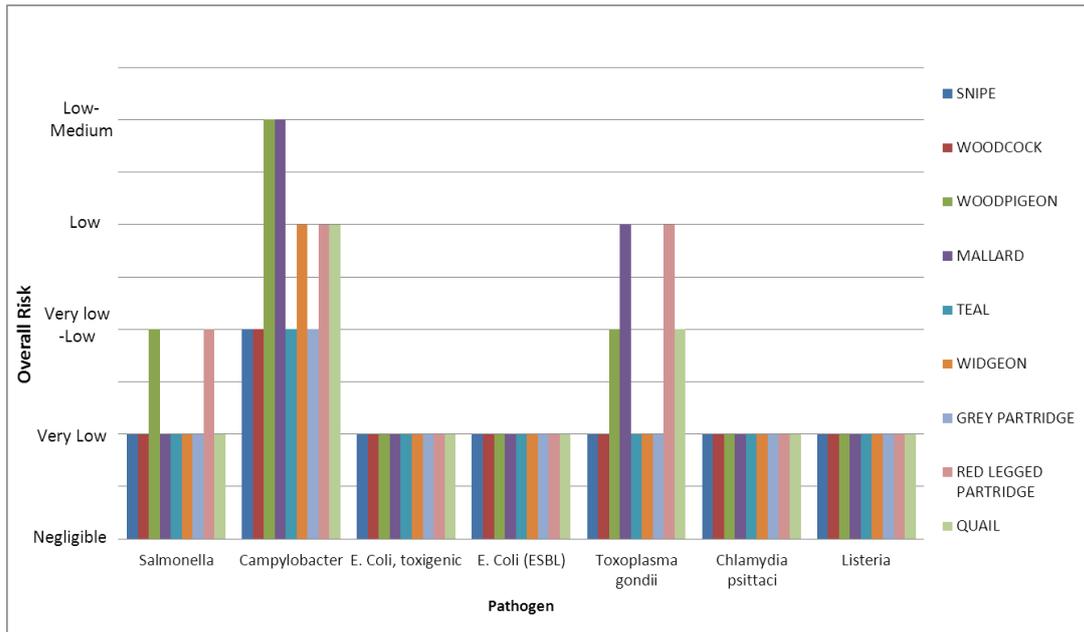


Figure 5: Graph showing risk of human infection from consumption of *eviscerated* small game birds outside the home for each pathogen and species.

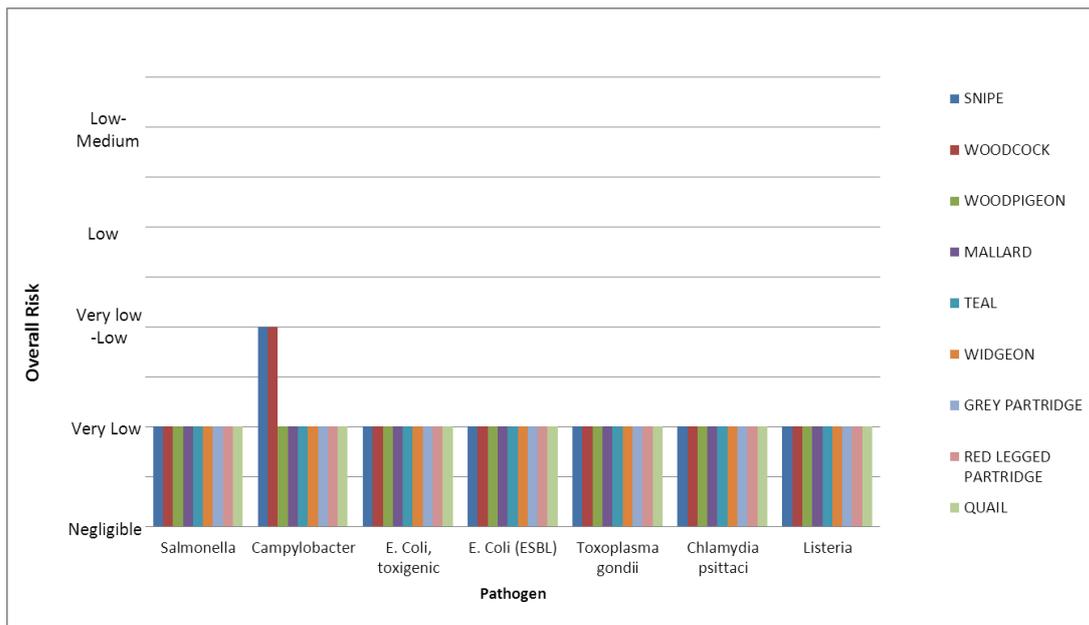


Figure 6: Graph showing risk of human infection from consumption of *uneviscerated* small game birds outside the home for each pathogen and species.

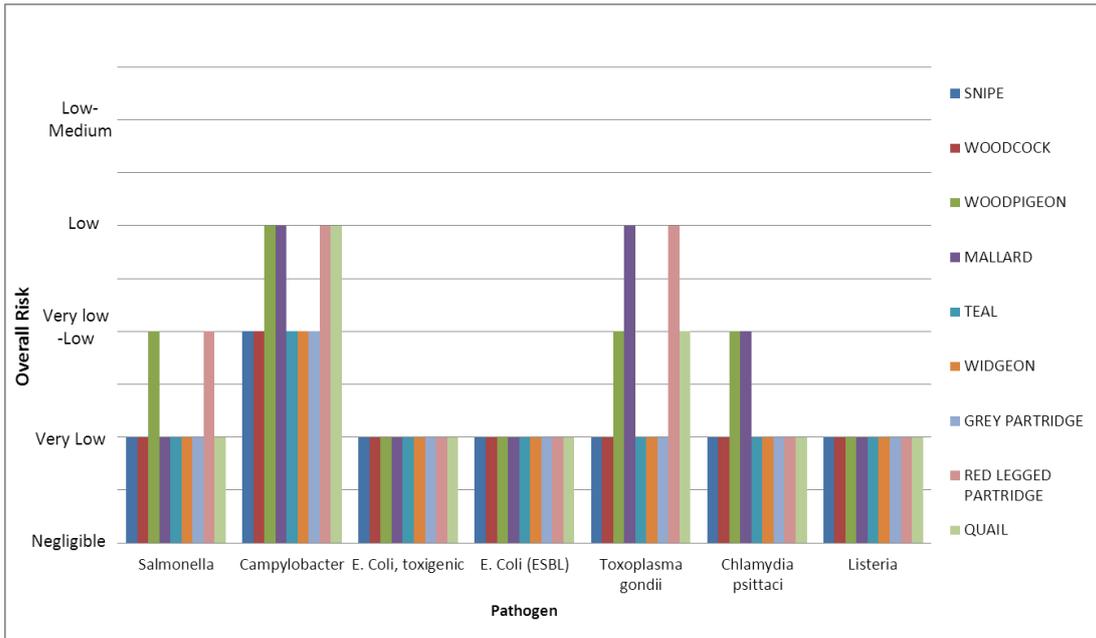


Figure 7: Graph showing risk of human infection from consumption of *eviscerated* small game birds in the home for each pathogen and species.

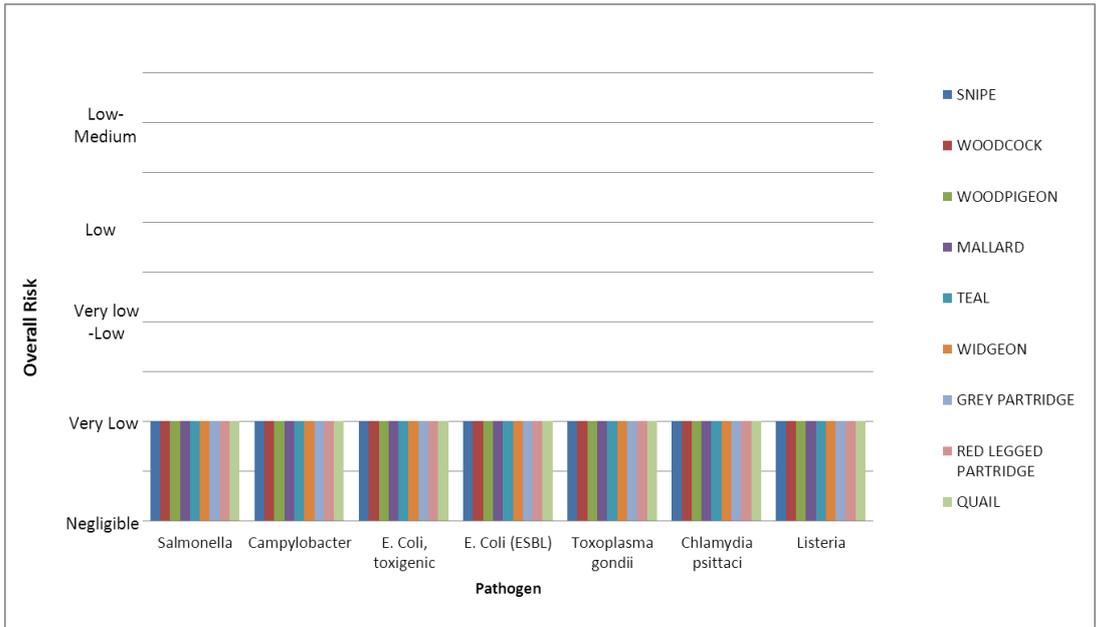


Figure 8: Graph showing risk of human infection from consumption of *unviscerated* small game birds in the home for each pathogen and species.

4. Discussion

4.1. Overall discussion

For this project we have developed a qualitative risk assessment framework in order to assess the microbiological risks from the production and consumption of both eviscerated and uneviscerated small wild game birds. This risk assessment is one of the first of its kind for the UK. Prior to this project it was not necessarily clear what the major risk factors relating to uneviscerated small game birds were and where the data gaps in the field-to-fork chain were. As such, a large part of this project involved gathering together information on the wild game industry from a large number of different sources and identifying those areas where data deficiencies exist (discussed in Section **Error! Reference source not found.**). The data gathering exercise suggested that, while a number of data gaps existed, carrying out the risk assessment would be a useful exercise. When interpreting the results of the risk assessment it should be appreciated that the data gaps will have an impact on their reliability. The identification of these gaps will help guide where future research is necessary.

The risk assessment suggested that the overall risks to the UK consumer for the majority of the pathogens/species considered were *Very Low*. The highest risk was for *Campylobacter* due to consumption of woodpigeon and mallards outside the home, which was assessed to be of *Low-Medium* risk. The low levels of overall risk were generally primarily due to a low frequency of consumption in the UK population, low prevalence of infection in the species and effective cooking to reduce the pathogen load before consumption. However, the individual risk to a consumer *if* a contaminated product was encountered could often be quite high, as the evidence suggested that for most pathogen/species combinations there was occasionally a risk of the concentration of the pathogen in some products, immediately prior to cooking, being high enough to cause human infection (although adequate cooking would still be expected to kill most of the pathogens). In these cases there is a risk of infection due to inadequate cooking or cross-contamination (e.g. to a salad that may accompany the meal or to other cooked meats/ready to eat products) prior to cooking. The assessment considers that a product could make it to the cooking stage with relatively high undetected pathogen loads, due to a series of unfortunate 'rare events' (e.g. a bird with a high initial concentration of pathogen has shot perforated gut, and is hung for long enough to allow growth of the pathogen within the muscle tissue,), or human error leading to inadequate implementation of control measures (e.g. the bird is stored at room temperature). In all cases, the fact that the pathogens are generally asymptomatic in the live bird and do not cause visible signs on the carcass, make them harder to detect unless they are specifically being tested for. The wild game industry is less regulated than other sectors, with many different distribution pathways. As such, the pathogens considered in this risk assessment are not routinely assessed (i.e. a test performed on the

carcass to determine if a pathogen is present), particularly when the consumer is responsible for the processing of the bird.

4.2. Eviscerated vs Uneviscerated

The evidence gathered did not suggest that there was much greater risk associated with consumption of uneviscerated game birds, compared with eviscerated game birds. In some pathogen/species combinations the evidence even suggested that the risk from eviscerated game birds may be slightly higher. This was due to the risk of cross-contamination resulting from the evisceration process outweighing the reduction in number of pathogenic organisms due to removal of the viscera. Additionally there was evidence that the cooking of uneviscerated birds was generally more likely to remove microbiological hazards, both due to the main method of cooking (uneviscerated birds are often thoroughly roasted) and also the propensity for serving eviscerated birds rare (a practice not thought to be so common for uneviscerated birds).

Average estimates of numbers of woodcock and snipe shot are 150,000 and 45,000 respectively. Whilst several AGHEs supply these birds uneviscerated, expert opinion (AGHEs, BASC) is that a large percentage of these birds are exported to the continent and that of those that are eaten in the UK approximately 10% are likely to be consumed uneviscerated. Internet searches have shown several London restaurants serving uneviscerated woodcock as a delicacy, although the frequency it appears on the menu is said to be very rare.

We were unable to find evidence of human consumption of uneviscerated birds other than woodcock and snipe in the UK. However, there was also not sufficient evidence to conclude that these birds were never consumed uneviscerated in the UK. There is anecdotal evidence of consuming squab (baby pigeons) and quail uneviscerated or *effilé* (partial evisceration where the heart, liver, lungs, gizzard, crop and kidneys are not removed from the carcass). If the viscera are not removed until after/during cooking, then there is still the possibility of cross-contamination up to this point, even if the viscera themselves are not actually consumed. We therefore estimated the frequency of uneviscerated preparation/consumption of these birds to be *Negligible-Very Low*.

For uneviscerated birds the highest combined risk to the individual (see Figure 4) were *Medium* for mallards consumed outside the home and *Low-Medium* for woodpigeon and red-legged partridge consumed both in and outside the home. However, as outlined above, we consider the frequency of consumption to be *Negligible-Very Low* and thus the overall risk estimate for these birds was estimated to be *Very low*. If there is (now or in the future) an increased frequency of consumption of these birds, then this overall risk should be re-examined; if consumption of the uneviscerated bird does occur then the risk of infection to the individual would be equivalent to the individual risks outlined above.

4.3. Assumptions

The wild game bird industry is a complex industry, involving a large number of individuals. There are many different ways in which a wild game bird may reach the consumer, some involving more input from the consumer than others. While we have endeavoured to cover the most common and/or risky routes as identified by experts, this risk assessment will not have considered every possibility.

A factor which has influenced the relative risks presented here is the assumption that there is a greater tendency to serve game undercooked or 'pink' outside the home than when cooked by the consumer in the home environment. This assumption is based on a combination of expert's opinions who considered that restaurants and catering establishments were more likely to serve gamebirds undercooked. Consumers cooking gamebirds within the home, however, were considered to more frequently use cooking methods such as roasting and casseroles that would be more likely to ensure a more thoroughly cooked product.

4.4. Data gaps/deficiencies

The assessed risks from the routes that are covered can only be as accurate as the data used to inform them. The wild game industry is not as regulated as other farmed livestock industries and it is no surprise that the availability and quality of data are lacking in some areas. In general there was a satisfactory degree of expert opinion knowledge available to assess the risk. Unfortunately, for some factors (particularly ones that require a numerical figure such as concentration of pathogens) expert opinion is a relatively poor substitute for hard data. As such, we have highlighted the following areas where data was deficient:

- Limited studies on prevalence of pathogens in game birds, in terms of small sample numbers and lack of data for birds such as woodcock and snipe, has introduced much uncertainty in the estimates of prevalence. Data for feral pigeons (*Columba livia* var *domestica*) has frequently been used as a proxy for woodpigeons (*Columba palumbus*). The true prevalence of pathogens in birds is likely to be affected by exposure and susceptibility. Furthermore, the effectiveness of detecting the true prevalence will depend on detection methods, sample sizes and sensitivity and specificity of the sampling methods used (Benskin, Wilson *et al.* 2009).
- The concentrations of pathogens in live game birds -there is a general lack of data regarding concentration. It would be useful to conduct a study to get an idea of both average levels of contamination in infected birds and the variation in contamination levels between infected birds. Further information relating to

duration of infection, and change in pathogen concentration over time post-infection, would also be interesting.

- The number of birds following each distribution pathway - Figure 3 outlines the distribution pathway and highlights the general lack of data on how many birds go down each route. It would be useful to conduct a survey to better estimate, for example, the number of people who shoot, process and eat wild game themselves.
- Frequency of consumption of wild game in and outside the home - there have been a number of studies in the past to determine the level of consumption of wild game, but it has been indicated that wild game is a fast growing industry and so such data will become out-dated fairly quickly. It is important to have a good idea of the current number of consumers as a significant increase in consumers could lead to a significant increase in risk.
- Frequency of consumption of uneviscerated bird species - is there definitely no preparation/consumption of wild game bird species other than woodcock and snipe?
- Probability/level of cross-contamination during processing - while there was evidence to suggest the potential for cross-contamination to be a factor during processing, there are little data to accurately assess the level of the associated risk (e.g. how many bacteria are transferred in a cross-contamination event). However, there are quantitative risk assessments for livestock species (e.g. EFSA QMRA for *Salmonella* in pigs) that can help to inform on this.
- Survival/ growth behaviour of pathogens during the framework pathway stages. Duration of hanging, in particular, could significantly affect the pathogen concentration both in and on the carcass depending on temperature and occurrence of shot gut perforation in the bird.
- Data on pathogenicity of *Salmonella* and *Campylobacter* strains found in wild birds with regards to species specific strains. Data on prevalence of the pathogens in the live birds rarely distinguishes between strains, but where this has been possible, not all strains found in wild game birds have been identified in humans and not all will cause serious clinical symptoms. For example, the phage types of *S. Typhimurium* and *S. Enteritidis* found in game birds are usually uncommon in people (Rob Davies pers. comm.).

4.5. Reliability of findings

Given the previous discussion surrounding data deficiencies and model assumptions, the results presented here are not (and never could be) 100% accurate. We do believe however, that they are a good indication of risk based on the current knowledge of wild game bird practices. Data have been collected from many experts across many different areas of the wild game industry (FSAS, Peer reviewed papers, AGHEs, BASC, Veterinarians etc...). As one of the first risk assessments of its kind for wild game birds,

the findings should provide a useful insight into the industry, highlighting the points in the farm-to-consumption chain that might be most risky and which pathogens or species could be of most concern. However, the overall risk estimates should be treated with a degree of caution. With regards to policy, this assessment should be used as one piece of evidence to be considered alongside other evidence.

To improve the accuracy/reliability of these results we recommend focussing attention on addressing the data deficiencies outlined in Section **Error! Reference source not found.**

4.6. Conclusions

The results of this risk assessment suggest that while large outbreaks of zoonotic infection among consumers due to wild game production and consumption are unlikely, sporadic individual infections may occur due to combinations of 'rare-event hygiene-related errors' in the field-to-fork chain and/or inadequate cooking of the game bird in or outside the home.

However, a number of data gaps/deficiencies were identified in the field-to-fork chain, which increases the level of uncertainty surrounding the results. Also, it is widely acknowledged that the gamebird sector is a growing industry; it is possible that production of farm-reared birds may become increasingly intensified to cope with the increased demand of gamebirds for release. The industrialisation of the gamebird sector could lead to similar problems currently affecting the poultry sector, as regards bacterial infections that are pathogenic to humans and the increase in use of important therapeutic antimicrobials during the rearing period that may select for resistant pathogens and opportunist organisms in the birds. It is therefore recommended that the conclusions of this assessment are periodically revisited to assess whether improved data are available to update the assessment or significant changes have occurred that would affect the findings.

Based on the current level of knowledge, the conclusions from this risk assessment are that while there are risks of zoonotic infection to the consumer associated with preparation and consumption of both eviscerated and uneviscerated small game birds, these risks are generally low and, assuming a general level of compliance with regulations and basic hygiene practices, are unlikely to be responsible for anything more than sporadic individual infection events in humans.

5. Acknowledgements

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6. Appendix 1: Detailed results

In this appendix we present full results figures, showing the qualitative scores for concentration and prevalence at each stage of the risk assessment for each bird species. We first present general results relevant across pathogens and then present the results in sections for each pathogen considered. Appendix 2 contains further detailed information and references for the data used in the assessment.

A1.1 General Considerations

A1.1.1 Game Bag/Cart

- Hunters often shoot multiple species of game at the same time. As such it is not uncommon for multiple bird species to be placed in the same game bag/cart (carts can hold up to 250 birds). Thus the potential opportunity for cross contamination between species is high.
- Shot perforation of the gut is relatively common. However, it is considered that pathogen release from the gut onto the exterior of the carcass will not commonly occur within the time period between shooting and transport to the game larder (Project steering group pers. comm.). Cross contamination of gut contents at this stage is therefore, not considered to be a risk.
- Penetration/biting of the retrieving dogs' teeth into the shot bird could lead to contamination with oral flora from the dog; however, this is likely to be a minor contributor to bird contamination
- Time spent in the bag can be as much as 3 hours (Colin Sheddon pers. comm.).
- While the opportunity for cross contamination is high, we consider the probability of it occurring to a significant extent is low.
- It is likely that growth of pathogenic bacteria within the intestines of dead birds will be limited as such bacteria appear to require residence within a live animal for significant growth to occur. In the dead bird, short term growth of pathogenic bacteria is soon overtaken by growth of commensal bacteria.

A1.1.2 Evisceration

- There is evidence to suggest that woodcock and snipe may be eaten uneviscerated.
- Squab (young pigeons) are mentioned in published literature as being consumed intact but these are generally farmed and are therefore not considered in this risk assessment.
- While there is no evidence to suggest that other bird species are eaten uneviscerated, there is insufficient evidence to conclude that they are *never* eaten uneviscerated.

- Therefore, we assume a *Negligible-Very low* level of consumption of uneviscerated game birds for these other species.
- Expert opinion suggests that birds that are to be consumed un-eviscerated, both in and outside the home, are likely to be treated with more attention paid to foodborne risks and cooked thoroughly, due to what has traditionally been an increased awareness of the potential risks to the consumer.
- Un-eviscerated birds are often roasted until the breast/leg meat is well done and thus cooked very thoroughly.

A1.1.3 Preparation & Consumption:

- There is evidence of a modern tendency to serve game meat from eviscerated birds ‘pink’ (underdone) in catering establishments, which increases the probability of pathogens surviving the cooking process. For larger birds this could result in the internal areas of the carcass not reaching sufficiently high temperatures for total pathogen elimination.
- There is a *Low-Medium* risk of products being held at room temperature for more than 90 minutes after cooking in the home environment (Worsfold and Griffith 1997).
- There is a *Low* risk of human error occurring which could result in non-compliance with proper procedures (e.g. refrigeration of cooked foods, unhygienic conditions), impacting on pathogen prevalence (Worsfold and Griffith 1997)
- Although thorough cooking kills most food poisoning bacteria the bird carcass may be handled many times before cooking occurs and any bacteria present may spread to other foods that may not be cooked before being consumed.
- These risks may increase the potential for survival and even growth of bacteria after the cooking process (particularly in the aforementioned ‘pink’ products). This is a particular concern if an occasion occurs when multiple risk associated events happen to the same product (e.g. not cooked properly, non-compliance with procedure and held at room temperature for 90 minutes or more post cooking).
- For all pathogens, but in particular *Listeria* and *T. gondii*, the risks to immunocompromised individuals and pregnant women should be considered higher than average, when taking into account the severity of symptoms as a result of infection in these two populations.
- Hunters, or those involved in the gamebird industry, may acquire higher resistance to infection by pathogens associated with gamebirds whilst those consuming gamebird meats in a catering establishment may be more vulnerable to infection if pathogens are present.

A1.1.4 Significant Data gaps and their impact on the overall risk

- There are no reliable data available for concentrations of any of the pathogens in the wild gamebird populations, or truly representative data for prevalence. Thus, initial pathogen concentrations are based on the same qualitative risks as for reported prevalence. If pathogen concentrations are significantly different than the prevalence then the risk would vary accordingly.
- Small reference samples in published studies, not always from the UK, results in great uncertainty for prevalence/concentration estimates.
- The 2006 Food Hygiene regulations set out a number of good practice guidelines (FSA 2011). There is no definitive evidence on how often these regulations are not adhered to, but there is anecdotal evidence that non-compliance does occur both accidentally and deliberately (FSAS 2012a). The trade of gamebirds through food hygiene regulation exemptions is unquantifiable and there is no accurate estimate of poached game; as such we assume a *Low* level of non-compliance. If the regulations are not complied with to the detriment of meat safety then the overall risk could increase.
- Prevalence data for woodpigeon is often based on feral pigeons. Although woodpigeons are increasingly found in urban areas and associated with agricultural practises resulting in exposure to maize silage and animal manure, it is possible that the prevalence has been over-estimated based on non-representative data.

A1.2 Salmonella

Figure 9 and Figure 10 show the results for *Salmonella* concentration and prevalence respectively. The data collected for the risk assessment (see Appendix 2) suggest that the initial background prevalence and concentration in all bird species is *Low* (<5%). While there are a number of potential risk factors during the production process there is little evidence to suggest a significant change in either prevalence or concentration of *Salmonella* before the plucking/evisceration stage. The removal of the intestines at this point, which provides the main reservoir of *Salmonella* within the carcass, will reduce the concentration of the pathogen to a *Very Low-Low* level in the eviscerated bird. Cross-contamination during the processes of plucking and evisceration and the ability of *Salmonella* to multiply can increase the prevalence of *Salmonella* at this stage, although the dry plucking that is most commonly used for shot game birds is likely to be less risky than wet plucking (Azanza and Ortega 2004). Whilst assumptions are made that the primary production of gamebirds complies

with the 2006 Food Hygiene regulations, in particular the practice of good hygiene and maintenance of the cold chain, slight variation in these conditions are not expected to alter the predicted risks.

During preparation it is expected that cooking of the eviscerated bird will further reduce the concentration of *Salmonella*. However, expert opinion suggests that there is a modern tendency to serve game meat 'pink' (underdone) outside the home. A risk to human infection would be present in any portion of contaminated meat that does not reach adequate temperatures for complete inactivation of *Salmonella*. In addition to this, there are *Low* risks of human error in not following proper procedures (e.g. prompt and correct refrigeration of cooked foods, unhygienic conditions etc...). These risks all give potential for survival and even growth of bacteria after the cooking process (particularly in the aforementioned 'pink' products). However, as the majority of products are cooked well we consider that the prevalence and concentration of *Salmonella* at the point of human consumption will be reduced to *Very Low*.

The estimated number of *Salmonella* infected game birds consumed is *Low*, whilst the dose response is relatively high for low fat foods. Consequently, using the risk assessment model in Figure 4, we consider that overall there is a *Very Low* risk of human infection for most species; while most products will not pose a risk we cannot rule out the possibility of a series of rare events along the chain (i.e. 'failures' of control measures/regulations) occurring and leading to a highly contaminated product being served to the consumer. This risk is greater from those products that are served 'pink' as complete thermal inactivation of the bacteria may not be achieved. The risk from woodpigeons and red-legged partridges consumed both in and outside the home is *Very Low-Low*, due to the higher frequency of birds consumed and the *Medium* risk of products being served 'pink'.

For *Salmonella* the combined risk to the individual (see figure 4) was considered to be *Very Low-Low* for un-eviscerated woodpigeons, mallards, red-legged partridges, quail and widgeons prepared and consumed outside the home. Most of these birds are relatively large (with the exception of quail) and it is possible that if the whole bird is cooked so that the breast meat is 'pink' the internal carcass temperature including the viscera may only reach ~55°C allowing for some *Salmonella* survival within the intestines. However, we could find no evidence that birds other than woodcock and snipe are eaten uneviscerated either in or outside the home so the overall risk of human infection from consumption of uneviscerated birds both in and outside the home was estimated to be *Very Low*.

Significant Data gaps and their impact on the overall risk:

- Limited studies for snipe, woodcock, quail and widgeon introduces much uncertainty in the prevalence/concentration estimates. We have predicted a *low* prevalence/concentration for these species based on published data for other birds. Small reference samples in published studies not always from the UK results in great uncertainty for prevalence/concentration estimates.

- Unknown pathogenicity of *Salmonella* isolates in gamebirds to humans. *Salmonella* in pigeons is suggested to be largely due to host adapted strains with little documented zoonotic transmission to humans. If isolates are non-pathogenic to humans then the risk could be reduced.

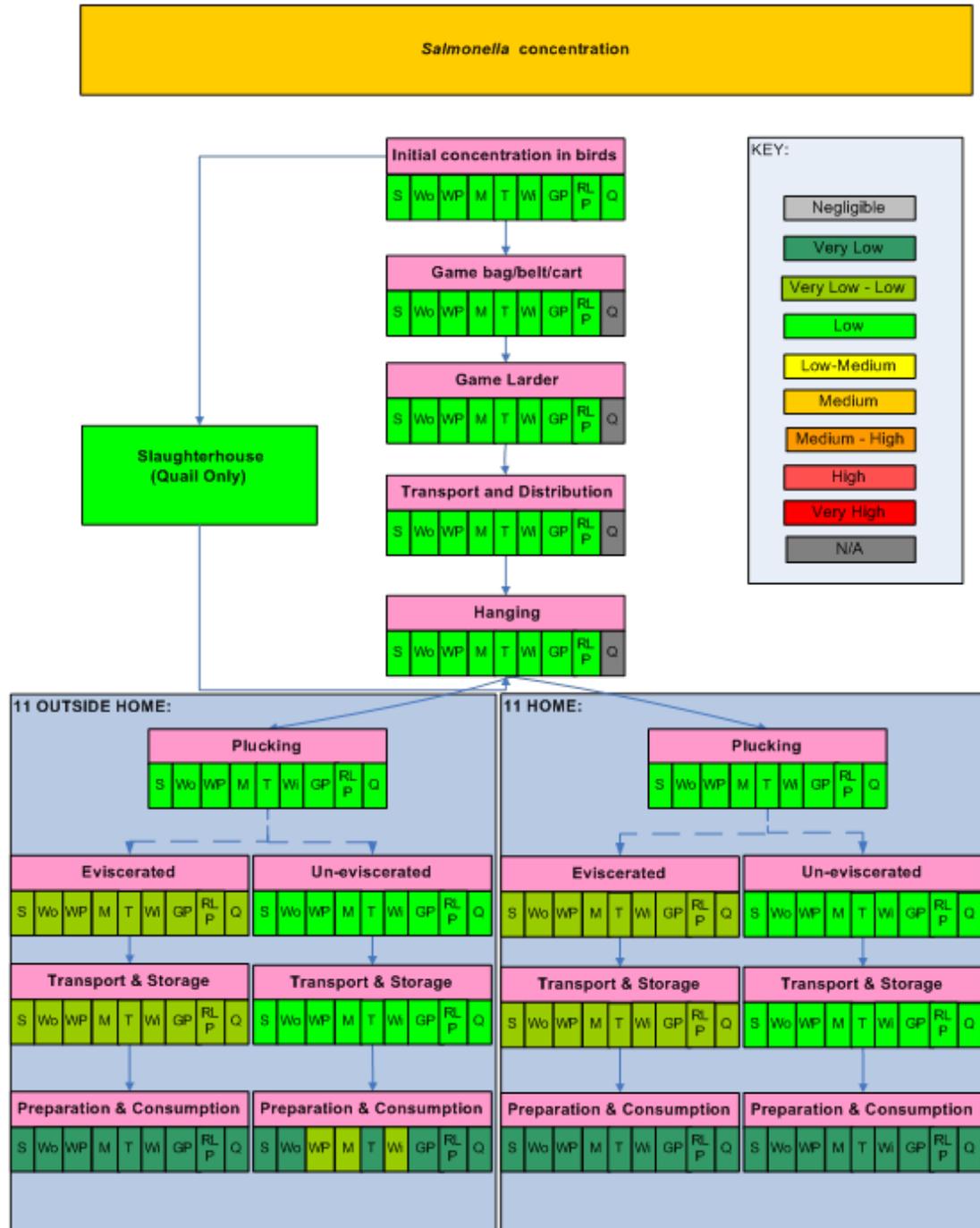


Figure 9: Qualitative scores for *Salmonella* concentration in small game bird species at each stage of the risk assessment.

Key: S= Snipe Wb= Woodcock, WP= Woodpigeon, M= Mallard, T= Teal, Wi= Widgeon, GP= Grey Partridge, RLP= Red-legged Partridge, Q= Quail

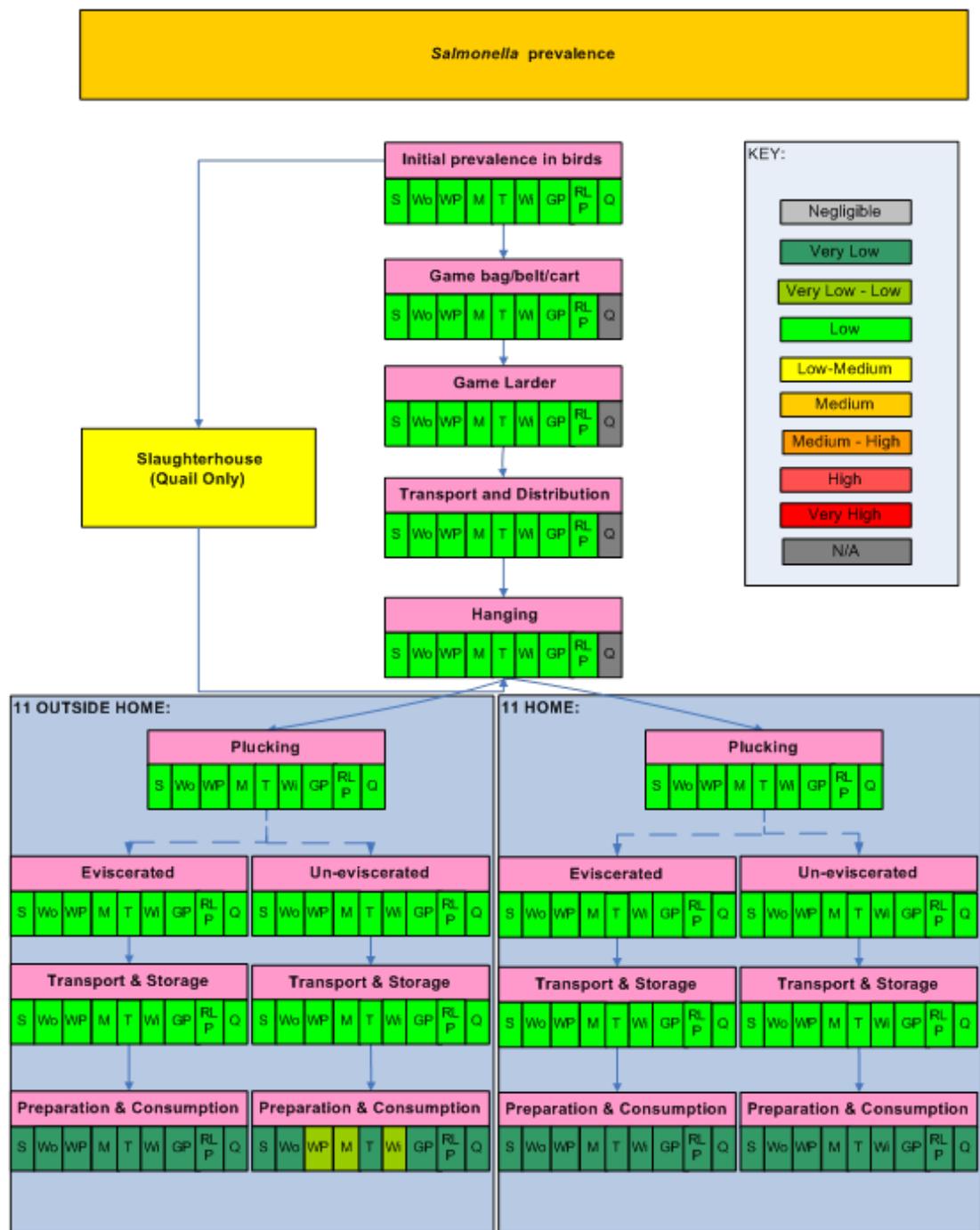


Figure 10: Qualitative scores for *Salmonella* prevalence in small game bird species at each stage of the risk assessment.

Key: S= Snipe Wo= Woodcock, WP= Woodpigeon, M= Mallard, T= Teal, Wi= Widgeon, GP= Grey Partridge, RLP= Red-legged Partridge, Q= Quail

A1.3 Campylobacter

Figure 11 and Figure 12 show the results for *Campylobacter* concentration and prevalence respectively. Data collected (see Appendix 2) suggests that the background prevalence in small game bird species is generally

considered to be relatively high, but there is a wide degree of variability in estimates between studies. *Campylobacter* is known to regularly colonise the intestines of birds as a commensal organism but small sample studies have resulted in wide-ranging estimates. The prevalence and concentration of *Campylobacter* in the intestine are considered to remain the same throughout initial storage and transportation. No significant cross contamination as a result of intestinal leakage is expected to occur in this time period (project steering group pers. comm.) and the organism can only grow above 30°C. As the shooting period for woodpigeons is not seasonal but occurs all year round any birds shot during the summer and not stored in cooled conditions could be exposed to high ambient temperatures and therefore be at risk from increased bacterial growth. Although there may be a slight decrease in pathogen concentration estimated at 1-3 logs (Rob Davies pers. comm.) over a 3-4 day time period due to *Campylobacter* die-off, the greatest reduction in pathogen concentration is likely to occur at the evisceration stage with the removal of the largest reservoir of the pathogen residing in the intestines.

The prevalence of *Campylobacter* is likely to increase during the plucking and evisceration process as a result of carcass cross contamination in particular during the release of pathogen at the evisceration stage although this will reduce over time particularly in dry storage (Rob Davies pers. comm.). The extent of cross contamination and, therefore, the increase in prevalence will rely on the efficiency of the evisceration technique. The use of high temperature waxing during plucking is likely to reduce surface contamination of the carcass. As consumption in the home is likely to be of frozen game birds out of season (FSAS 2012b) a further reduction in the concentration of *Campylobacter* is predicted as the pathogen is rapidly reduced at freezing temperatures. Thermal inactivation of *Campylobacter* begins at 46°C (Labbe and Garcia 2001). Thorough cooking, as is suggested to occur within the home, will reduce the pathogen and its concentration in all cooked eviscerated bird species, to a level that is considered to be *Very Low*. In the larger birds, that is, woodpigeon, mallard and widgeon, under cooking could result in pathogen survival and the concentration in these species is therefore considered to be *Very Low-Low*.

After multiplying the risk factors to take into account the relative consumption of each gamebird species, and the low infectious dose of *Campylobacter* (Kothary and Babu 2001) the overall risk of human infection with *Campylobacter* was considered to be *Low* for eviscerated woodpigeon, mallard, red-legged partridge and quail in the home. Outside the home, the risk of infection to the consumer is *Low-Medium* for eviscerated woodpigeon and mallard and *Low* for widgeon, red-legged partridge and quail. The risk values take into account the likelihood of inadequate cooking to ensure thorough removal of *Campylobacter* outside the home and the increased chance of cross contamination in high throughput kitchens.

The combined risk to the individual of *Campylobacter* infection from uneviscerated birds consumed in the home is considered to be *low-Medium* for woodpigeon, mallard and red-legged partridge and *Low* for widgeon and

quail. Outside the home the risk is considered to be *Medium* for mallard, *Low-Medium* for woodpigeon and red-legged partridge and *Low* for widgeon and quail. The *Very Low-Low* risk in the remainder of birds is considered as a result of their relatively small size allowing the internal cavity of the carcass to reach temperatures sufficient for further pathogen inactivation. However, due to the absence of evidence for consumption of uneviscerated birds, the overall risk of infection, with the exception of snipe and woodcock was reduced to *Very Low*. Outside the home the overall risk of infection for snipe and woodcock was considered to be *Very Low-Low*. The predilection for undercooking outside the home combined with the low infectious dose of *Campylobacter* and the known tendency of snipe and woodcock to be consumed uneviscerated increase the risk to the individual from *Very Low* to *Very Low-Low*.

The relatively high prevalence of *Campylobacter* on the uncooked carcass provides an additional risk via cross contamination of other ready-to-eat foods in the kitchen where cooking will not provide a further means of pathogen decrease. In the case of chickens the majority of human infections with *Campylobacter* are considered to arise from cross-contamination of other foods in the kitchen whilst preparing the bird for cooking and not as a result of consumption of the bird (Rob Davies pers. comm.). As the viscera forms a large reservoir of *Campylobacter*, evisceration of gamebirds even by experienced individuals, is likely to result in cross-contamination within the kitchen environment (Mead and Scott 1997) if carried out in this location.

Significant Data gaps and their impact on the overall risk:

- Multilocus sequence typing (MLST) data cannot confirm that *Campylobacter* species found in wild gamebirds are pathogenic to humans. Literature suggests that isolates found in gamebird intestines are rarely similar to those of humans unless the birds are associated with human activities. If isolates are non-pathogenic to humans then the risk could be reduced.
- Limited studies for snipe and woodcock introduces much uncertainty in the prevalence/concentration estimates. We have predicted prevalence/concentration for these species based on published data for other birds. If this prediction is an underestimate then the overall risk could be similarly underestimated

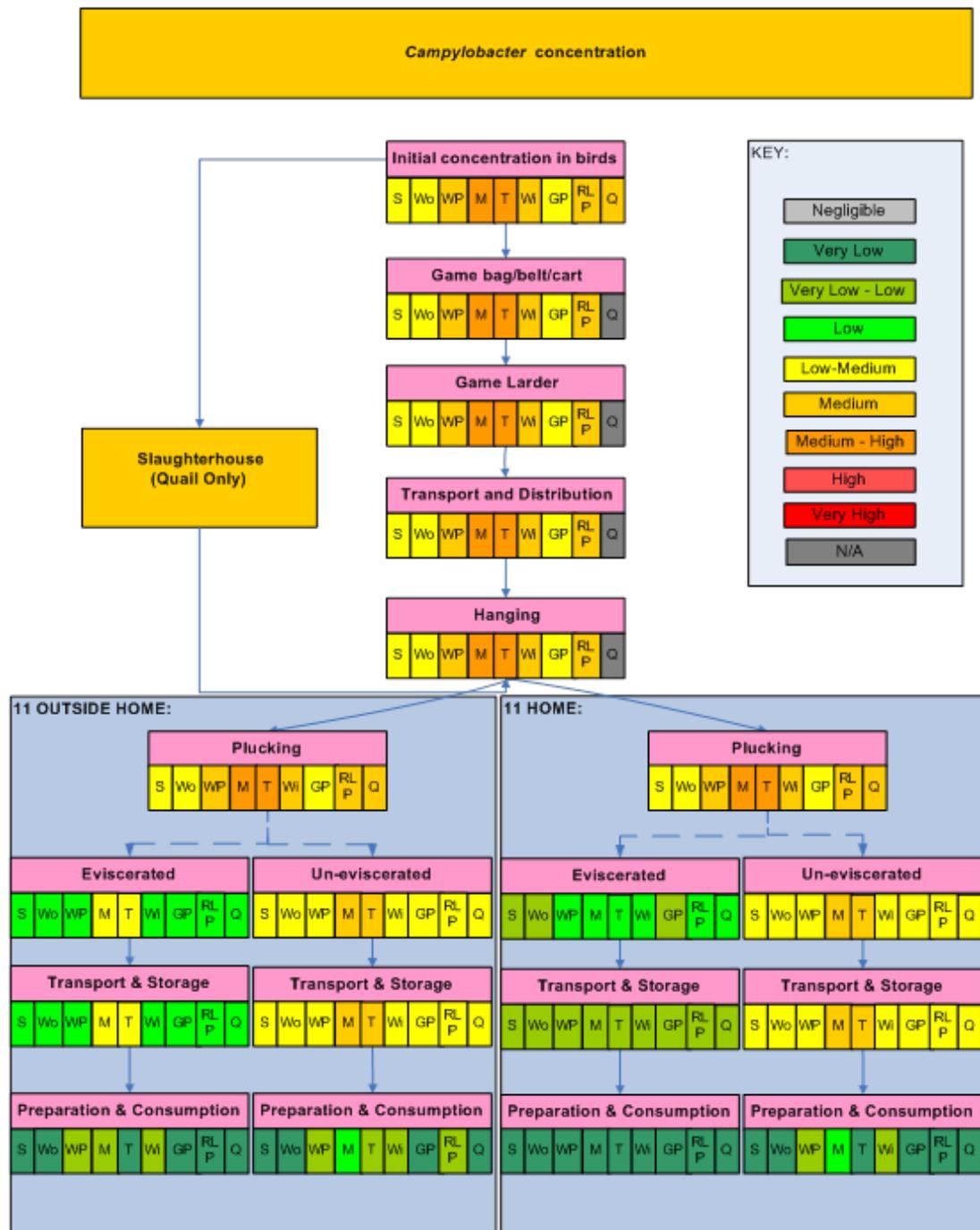


Figure 11: Qualitative scores for *Campylobacter* concentration in small game bird species at each stage of the risk assessment.

Key: S= Snipe Wo= Woodcock, WP= Woodpigeon, M= Mallard, T= Teal, Wi= Widgeon, GP= Grey Partridge, RLP= Red-legged Partridge, Q= Quail

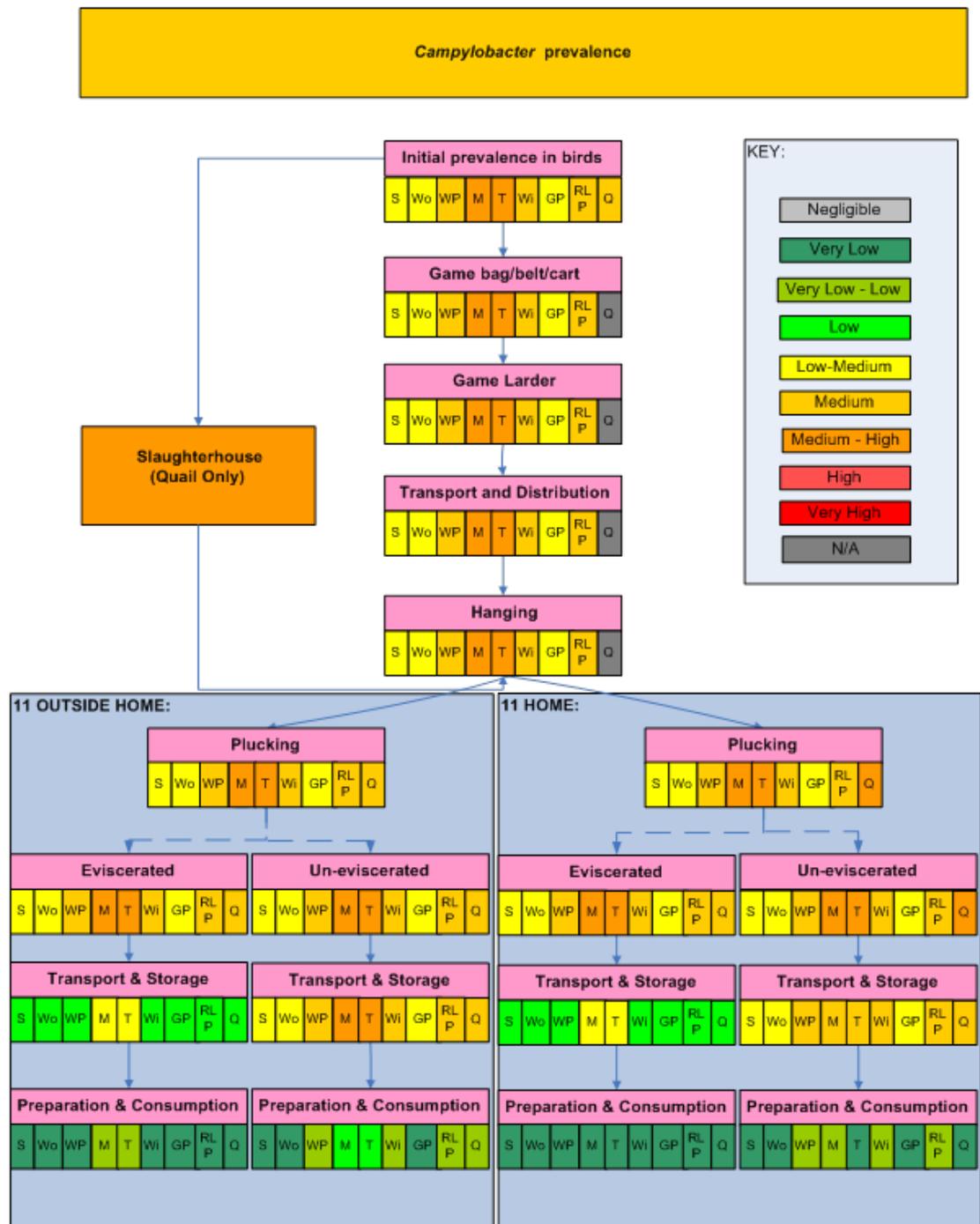


Figure 12: Qualitative scores for *Campylobacter* prevalence in small game bird species at each stage of the risk assessment.

Key: S= Snipe Wo= Woodcock, WP= Woodpigeon, M= Mallard, T= Teal, Wi= Widgeon, GP= Grey Partridge, RLP= Red-legged Partridge, Q= Quail

A1.4 *E. coli* (Toxicogenic)

Figure 13 and Figure 14 show results for *E. coli* (Toxicogenic) concentration and prevalence respectively. No data were available on the background prevalence and concentration of toxicogenic *E. coli* in most bird species with the exception of pigeons. Non-pathogenic *E. coli* are part of the normal gut

micro flora of birds but they can also be asymptomatic carriers of strains that are potentially zoonotic. Whilst wild birds can acquire the bacteria from feeding on rubbish dumps, sewage outlets and fertilised pastureland the small amount of evidence obtained suggests that prevalence within the game bird population is likely to be *Low*. VTEC has been identified in feral pigeons however prevalence in this species is still considered to be *Low*. Ruminants are recognised as being the main natural reservoir of VTEC (EFSA 2007) and cattle are considered to be the major animal source of VTEC that are virulent to humans.

The initial prevalence and concentration of toxigenic *E. coli* in the gamebird population as a whole is considered to be *Very Low*. The only evidence of the presence of this pathogen in gamebirds is from feral pigeons where the isolates were of unknown pathogenicity to humans. Whilst the risk pathway for toxigenic *E. coli* is similar to that of *Salmonella*, as the growth temperatures and survival at refrigeration and freezing are similar for both pathogens, the risk scores are considered to be *Very Low* for consumption of all species both in and outside the home. This score is the same for both eviscerated and uneviscerated birds.) This assumes that thorough cooking will establish complete inactivation of the *Very Low* concentration of *E. coli* in all gamebird species.

Significant Data gaps and their impact on the overall risk:

- Lack of data for prevalence in all game bird species with the exception of pigeon introduces much uncertainty in the prevalence/concentration estimates. We have predicted prevalence/concentration for these species based on published data for other birds. If this prediction is an underestimate then the overall risk could be similarly underestimated
- Unknown pathogenicity of *E. coli* isolates in birds to humans. If isolates are non-pathogenic to humans then the risk could be further reduced.

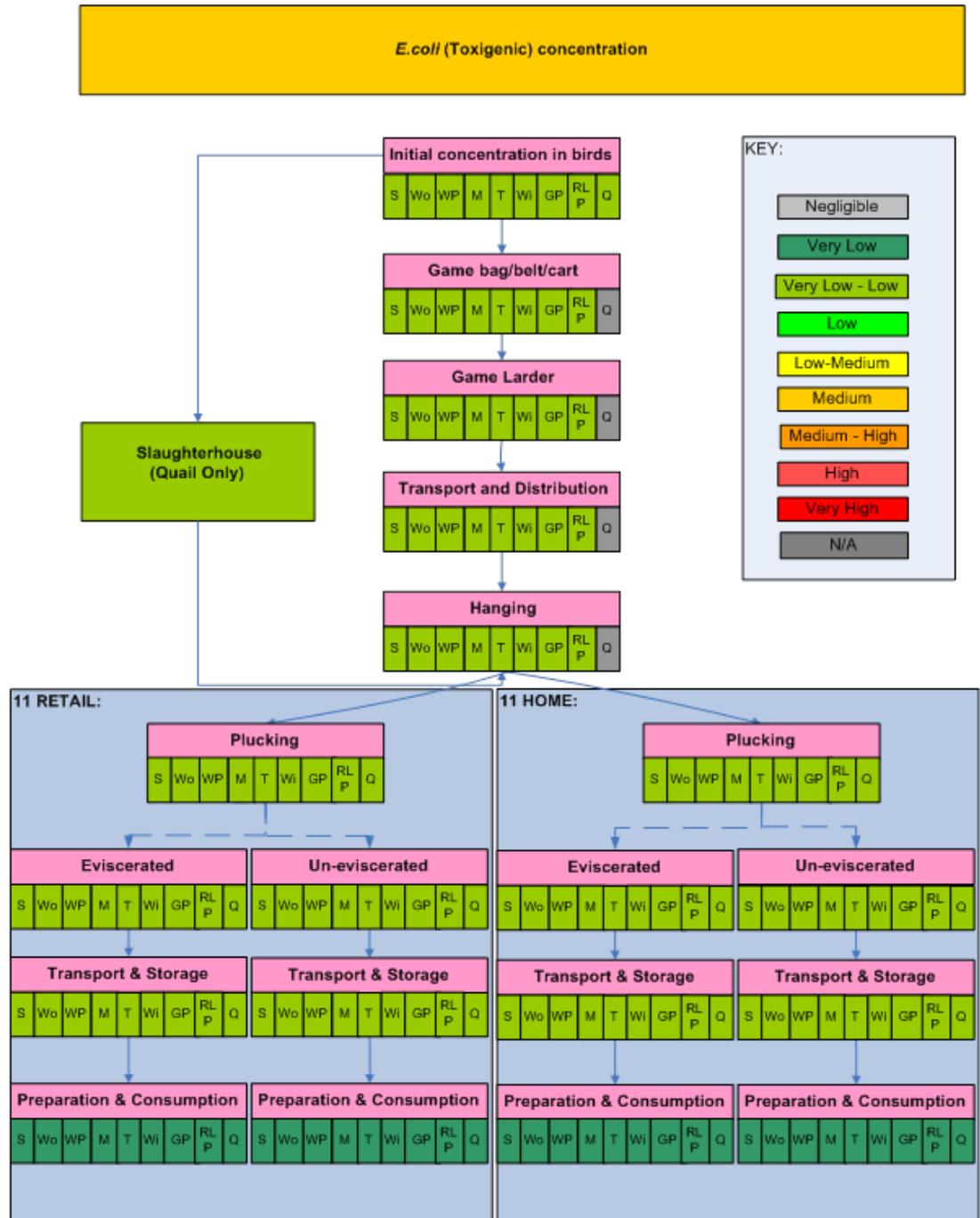


Figure 13: Qualitative scores for *E. coli* (Toxicogenic) concentration in small game bird species at each stage of the risk assessment.

Key: S= Snipe Wo= Woodcock, WP= Woodpigeon, M= Mallard, T= Teal, Wi= Widgeon, GP= Grey Partridge, RLP= Red-legged Partridge, Q= Quail

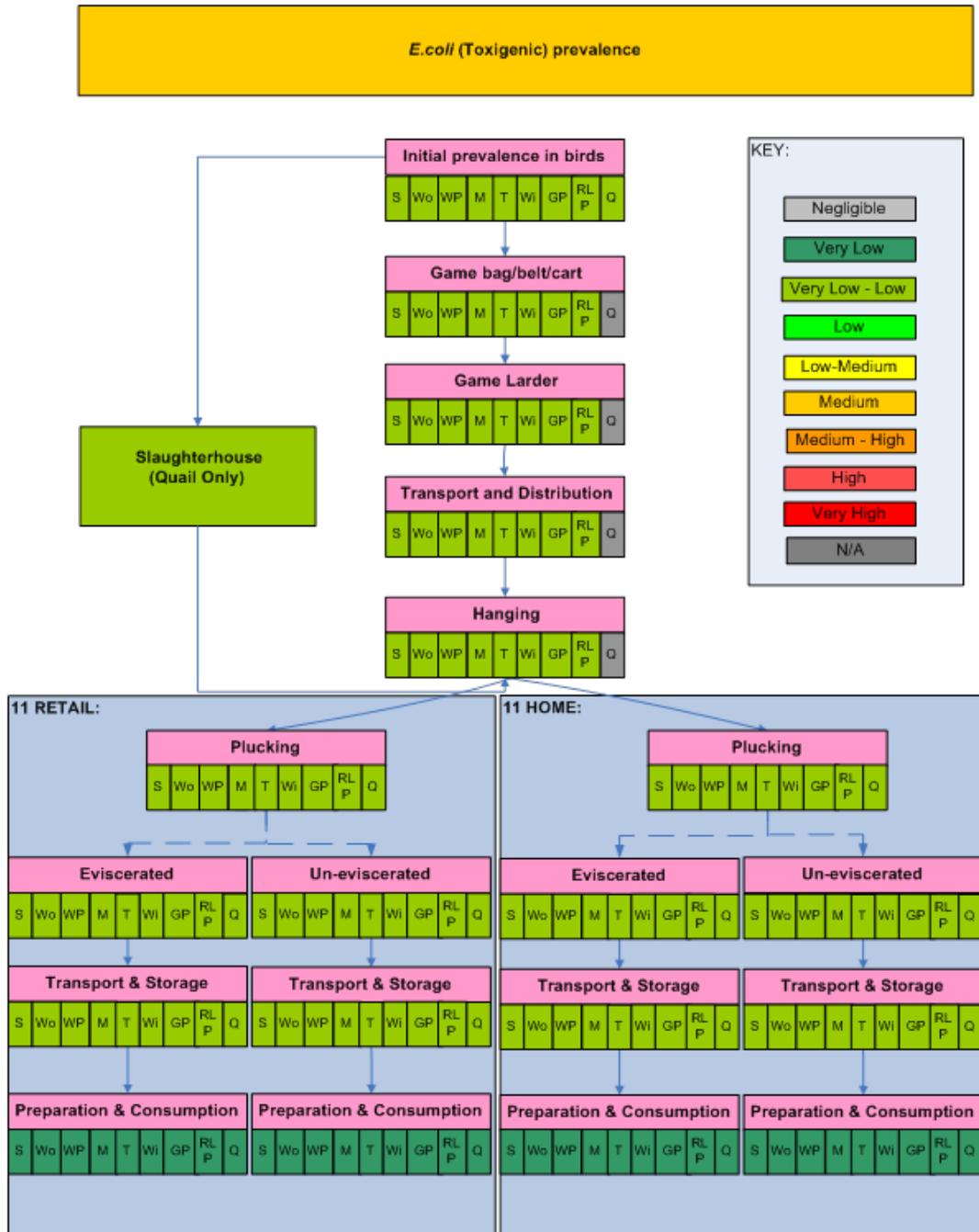


Figure 14: Qualitative scores for *E. coli* (Toxicogenic) prevalence in small game bird species at each stage of the risk assessment.

Key: S= Snipe Wo= Woodcock, WP= Woodpigeon, M= Mallard, T= Teal, Wi= Widgeon, GP= Grey Partridge, RLP= Red-legged Partridge, Q= Quail

A1.5 *E. coli* (ESBL)

Figure 15 and Figure 16 show the results for *E. coli* (ESBL) concentration and prevalence respectively. Data collected (see Appendix 2) suggest that background prevalence of *E. coli*-ESBL is low in most bird populations apart from farm reared red-legged partridges (Medium) and quail (Low-medium). Wild birds are considered to be vectors of resistant *E. coli* acquiring and

disseminating infection predominantly via faecal pollution of water courses (Guenther, Ewers *et al.* 2011). The use of antibiotics in farm reared game birds can also select for *E. coli*-ESBL in the birds. Eighty three per cent of shoots rely on released commercially reared pheasants, red-legged partridges and mallards which have been potentially exposed to antibiotics via treatment of parents or the birds themselves (GFA 2013). While there is no data on the off-label use of cephalosporins in game hatcheries, this is the main driver for the emergence of ESBL and AmpC type resistances in *E. coli* and other commensal flora of chickens and turkeys (EFSA 2011) However, it is considered unlikely that woodpigeons, snipe, woodcock, teal, grey partridge or widgeon would have been exposed directly to *E.coli*-ESBL contamination.

The risk pathway for *E. coli*-ESBL is similar to that of toxigenic *E. coli* as the growth temperatures and survival at refrigeration and freezing are similar. Consequently the risk scores are the same for consumption of both eviscerated and uneviscerated birds both in and outside the home (*Very Low*). Similarly to *Salmonella*, the risk of human infection from *E. coli*-ESBL infection resides more from the cross contamination of the kitchen environment from bacteria present within the bird rather than from consumption of the cooked bird itself (Depoorter, Persoons *et al.* 2012).

Significant Data gaps and their impact on the overall risk:

- Lack of data for prevalence in woodcock, snipe, teal and widgeon introduces much uncertainty in the prevalence/concentration estimates. We have predicted prevalence/concentration for these species based on published data for other birds. If this prediction is an underestimate then the overall risk could be similarly underestimated

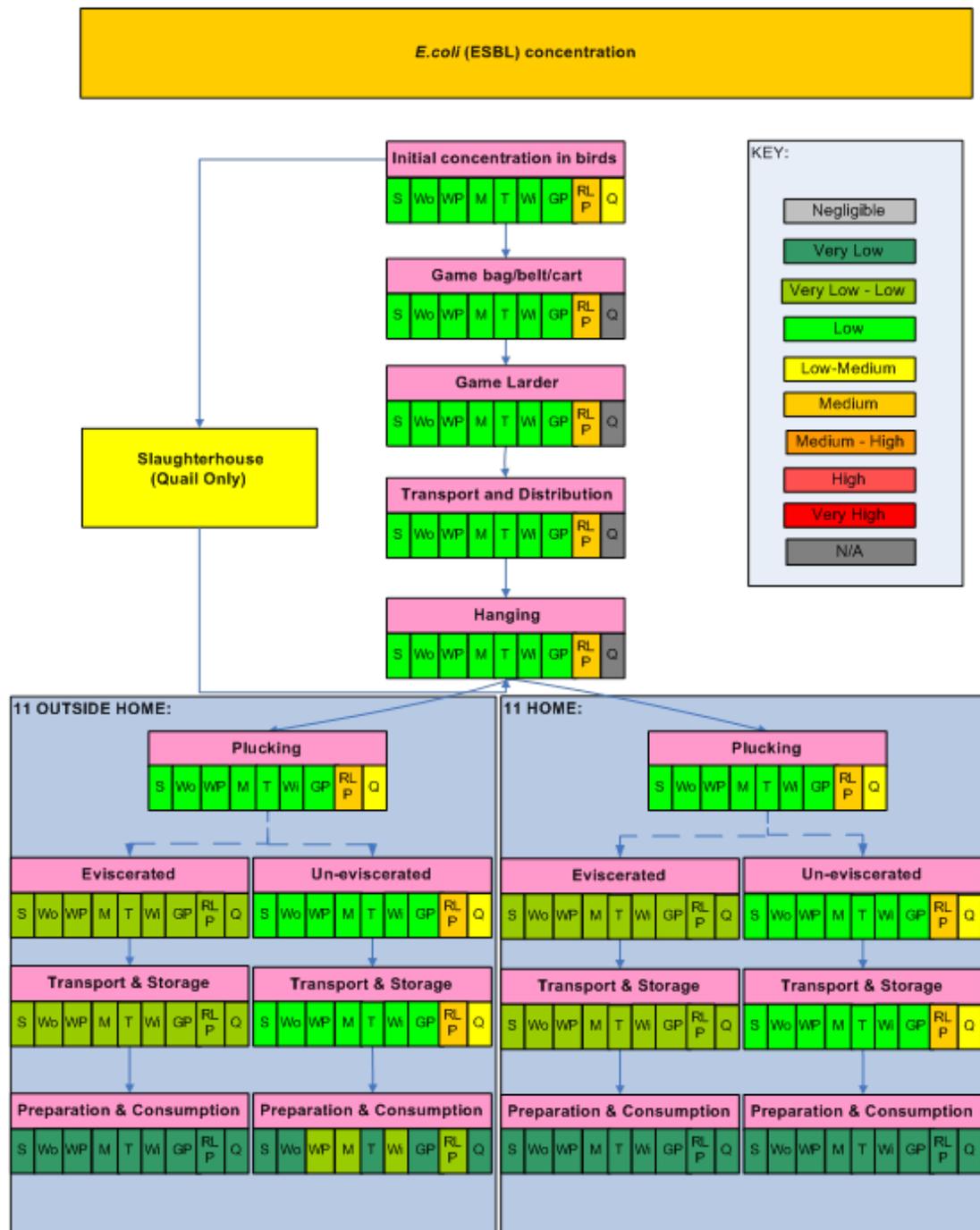


Figure 15: Qualitative scores for *E.coli* (ESBL) concentration in small game bird species at each stage of the risk assessment.

Key: S= Snipe Wo= Woodcock, WP= Woodpigeon, M= Mallard, T= Teal, Wi= Widgeon, GP= Grey Partridge, RLP= Red-legged Partridge, Q= Quail

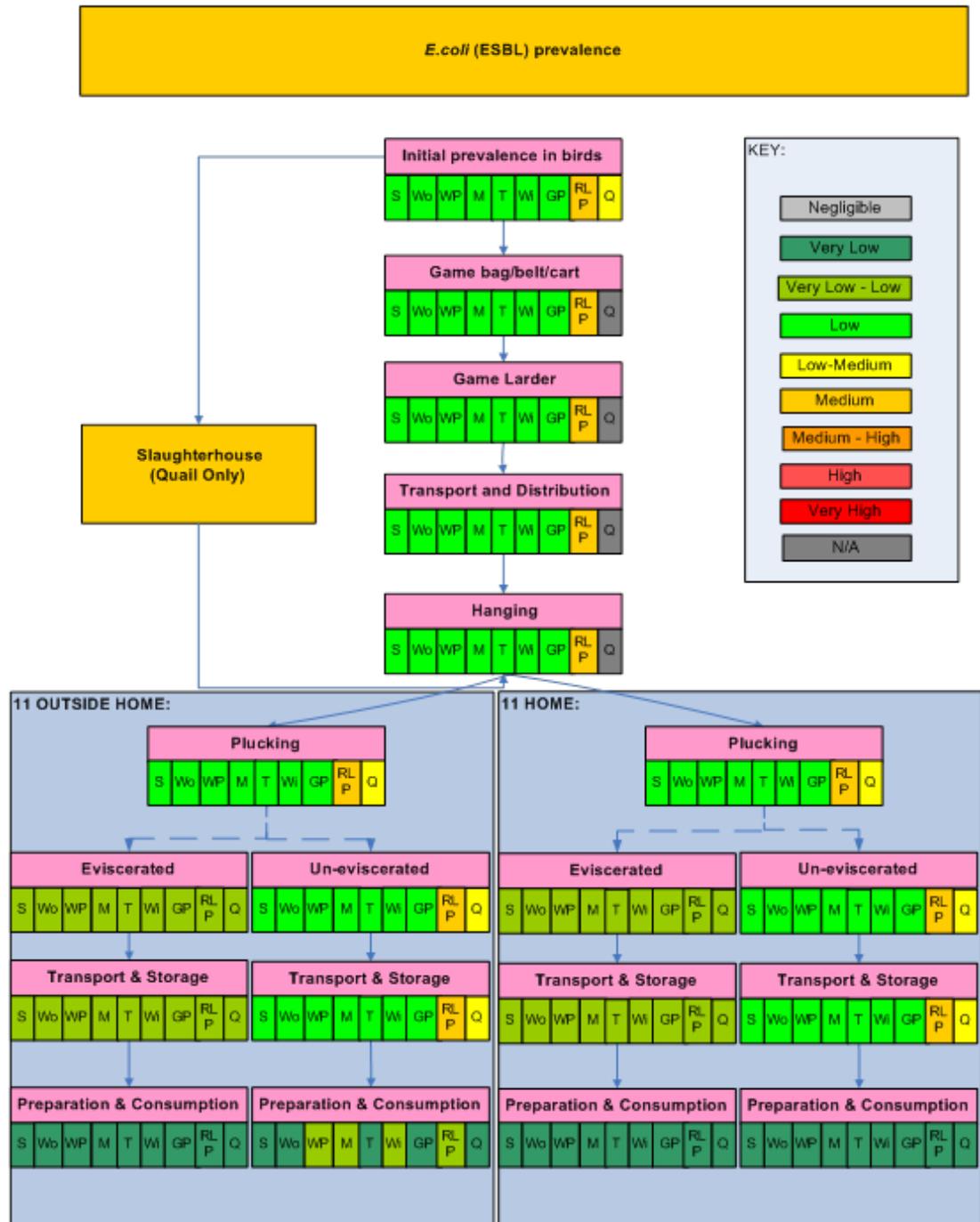


Figure 16: Qualitative scores for *E. coli* (ESBL) prevalence in small game bird species at each stage of the risk assessment.

Key: S= Snipe Wo= Woodcock, WP= Woodpigeon, M= Mallard, T= Teal, Wi= Widgeon, GP= Grey Partridge, RLP= Red-legged Partridge, Q= Quail

A1.6 *Chlamydophila psittaci*

Figure 17 and Figure 18 show the results for *Chlamydophila psittaci* concentration and prevalence respectively. Evidence collected (see Appendix 2) suggests that there is *High* background prevalence in woodpigeon and *Medium* prevalence in mallards. Data were not available for the other bird species. As many birds can be asymptomatic carriers it is estimated that there is a *Medium* prevalence in all other game bird species considered, with the exception of woodcock and snipe. The prevalence in these bird populations is estimated to be *Low-medium* because of their solitary 'wild' lifestyle allowing for less contact with other contaminated birds. *C. psittaci* can survive in a killed game bird for as long as the carcass is kept in an edible state (Alisdair Wood pers. comm. Cited in (Coburn 2003). As an intracellular bacterium it cannot replicate after total cell death has occurred in the bird. The storage temperature of the carcass is therefore irrelevant, as regards *C. psittaci* growth, after the carcass has cooled down. It is possible that a very low level of pathogen growth could occur within the first 2-3 hours of death; growth is considered negligible after that.

C. psittaci may be present on the surface of game birds so an increase in prevalence as a result of cross contamination is likely to occur especially where the birds are in close proximity to each other, for example, in the game bag. The critical points of *C. psittaci* concentration reduction are at the plucking and evisceration stage where infection is removed via the feathers, airsacs and lungs respectively (Deschuyffeleer, Tyberghien *et al.* 2012). After evisceration the concentration of *C. psittaci* is likely to be reduced to *Low* or *Very low* levels depending upon the original concentration.

C. psittaci is not currently known to be infectious via ingestion (EC 2002) therefore the risk of human infection via consumption in both the home and catering establishments is *Very Low*. However if the consumer plucks/eviscerates the bird within the home environment then there could be a *Very Low-Low* risk of consumer infection via inhalation depending on the initial concentration and prevalence of infection in the bird. The number of *C. psittaci* infected woodpigeons and mallards consumed are estimated to be very high so the risk of consumer infection via plucking and evisceration is therefore considered to be *Very Low-Low* for these two species. In the uneviscerated bird the risk of infection via inhalation is considered to be *Very Low* as the consumer may be exposed to a *Very Low* level of infection via plucking. The incidences of *C. psittaci* infection reported in abattoir workers (Deschuyffeleer, Tyberghien *et al.* 2012) are in environments where workers are subject to multiple exposures from high throughput of birds which are processed immediately after killing when *C. psittaci* organisms are more likely to be shed in high numbers.

Significant Data gaps and their impact on the overall risk:

- Infectious dose is unknown - low infectious dose could increase the overall risk
- Lack of data for prevalence in woodcock, snipe, partridge and quail introduces much uncertainty in the prevalence/concentration estimates. We have predicted prevalence/concentration for these species based on published data for other birds. If this prediction is an underestimate then the overall risk could be similarly underestimated
- Survival of *C. psittaci* in gamebirds after death is reliant on expert opinion. An over- or under-estimation of survival could impact on pathogen concentration and consequently on the overall risk.

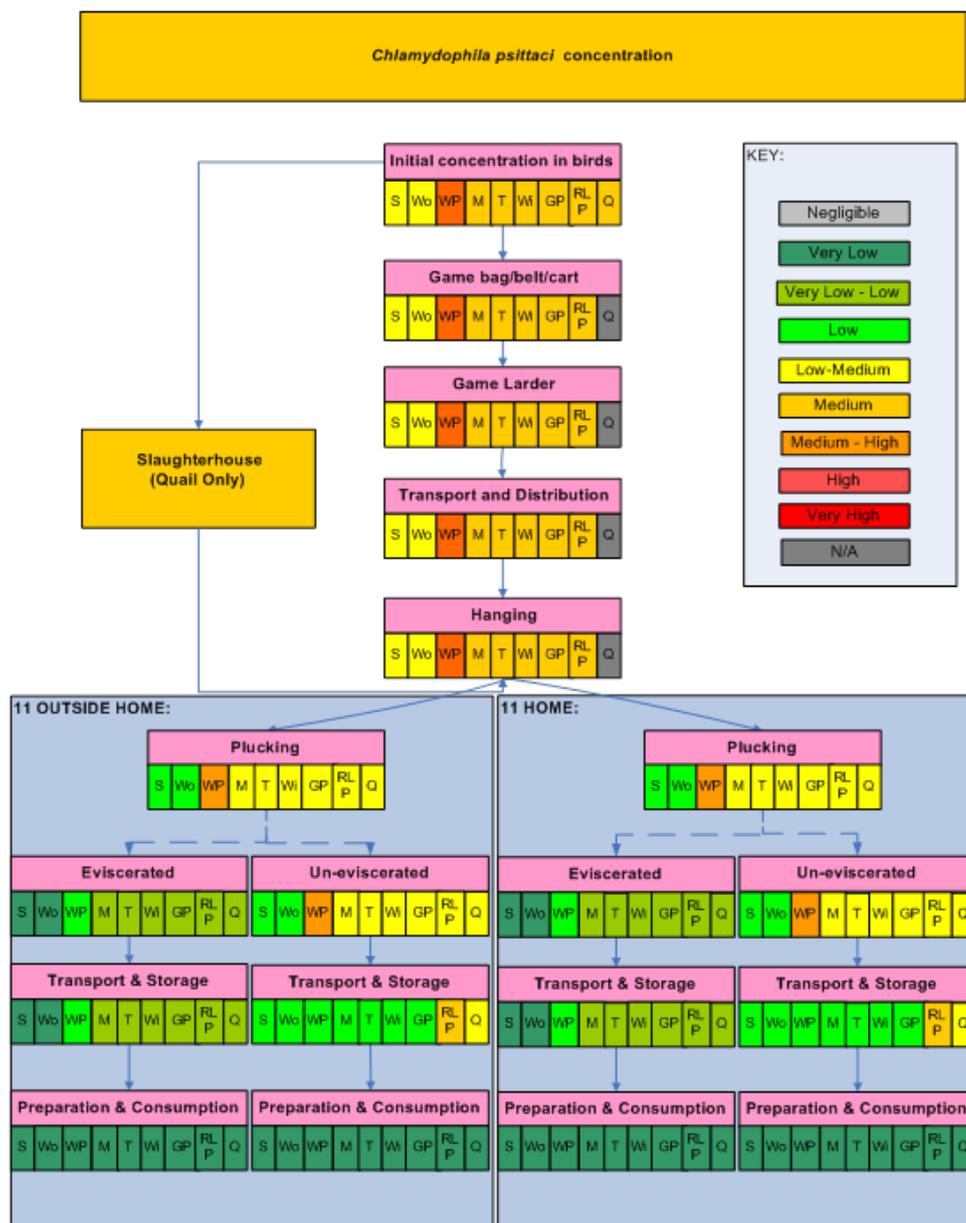


Figure 17: Qualitative scores for *Chamydophila psittaci* concentration in small game bird species at each stage of the risk assessment.

Key: S= Snipe Wo= Woodcock, WP= Woodpigeon, M= Mallard, T= Teal, Wi= Widgeon, GP= Grey Partridge, RLP= Red-legged Partridge, Q= Quail

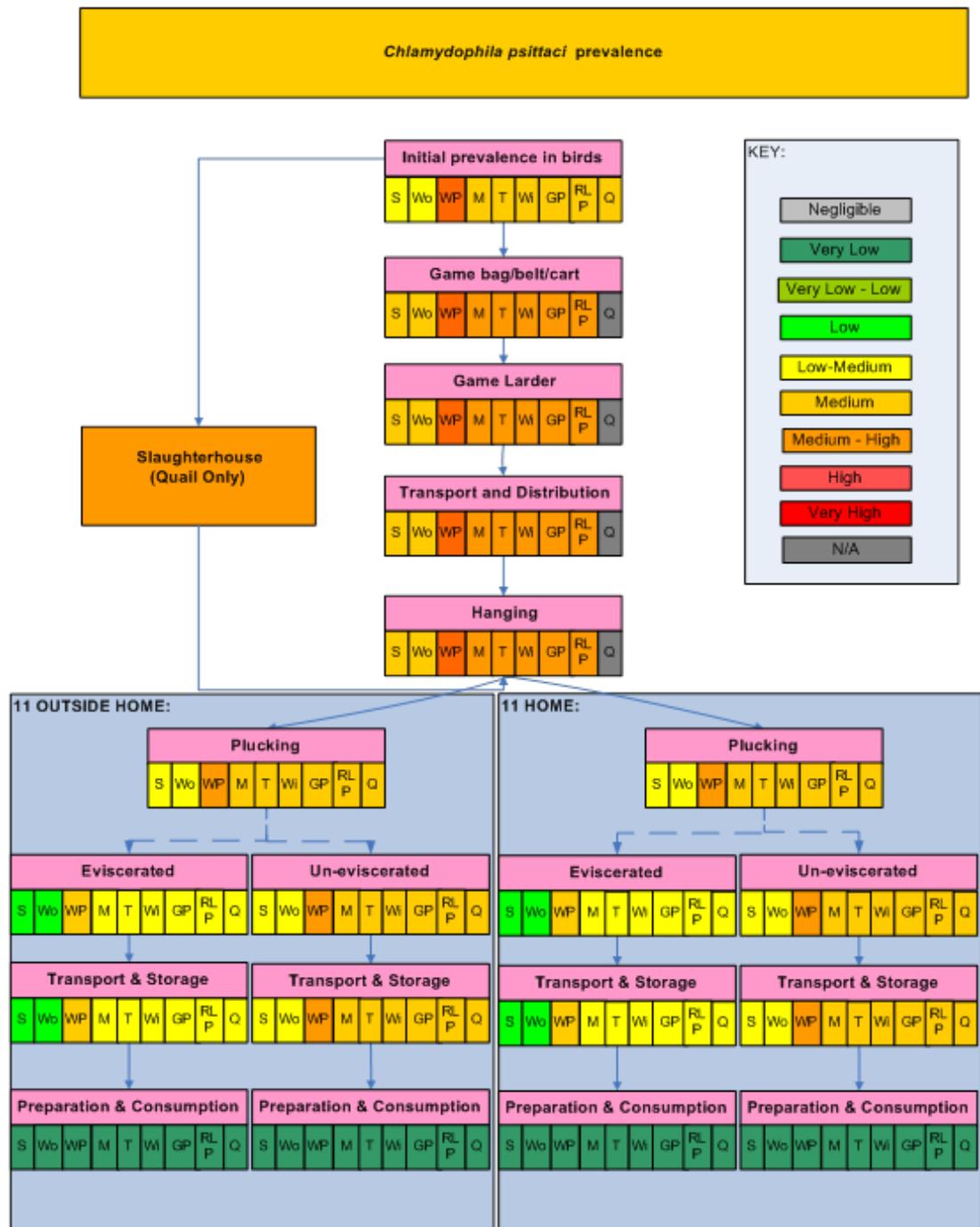


Figure 18: Qualitative scores for *Chlamydoiphila psittaci* prevalence in small game bird species at each stage of the risk assessment.

Key: S= Snipe Wo= Woodcock, WP= Woodpigeon, M= Mallard, T= Teal, Wi= Widgeon, GP= Grey Partridge, RLP= Red-legged Partridge, Q= Quail

A1.7 *Toxoplasma gondii*

Figure 19 and Figure 20 show the results for *Toxoplasma gondii* concentration and prevalence respectively. The data collected for the risk assessment (see Appendix 2) suggest that the initial background prevalence and concentration in all bird species ranges from *Low* in teal and widgeon to *Medium* in snipe, woodcock, mallard and quail. Pathogen growth throughout the pathway is considered to be absent, as *T. gondii* is present as tissue cysts within the carcass and therefore will not increase in the dead bird. Similarly there is no increase in prevalence throughout the pathway until the bird is slaughtered or eviscerated where the use of knives can result in cross contamination unless thorough cleaning is undertaken (Kapperud, Jenum *et al.* 1996).

The concentration of *T. gondii* is likely to be reduced at the evisceration stage as tissue cysts are predominant in the heart, brain and non-skeletal muscle of poultry. A further reduction in pathogen concentration is considered to occur in the home environment as the use of frozen storage to allow for gamebird consumption out of season will cause inactivation of any tissue cysts present. Freezing is less likely to occur in the pathway outside the home and the cysts will therefore remain viable.

During preparation it is expected that thorough cooking of the birds will reduce any remaining concentration of *T. gondii* to below levels likely to cause human illness. If tissue cysts exist in skeletal muscle, however, then cooking 'pink' will not result in inactivation and they will be ingested by the consumer as viable cysts going on to cause infection within the human host. Taking this into account, alongside the potential number of infected birds consumed and the long-term effect of *T. gondii* infection in the human host there is considered to be a *Low* risk of human infection from the consumption of mallard and red-legged partridge both in and outside the home. Here, the risk is increased to *Low* when considering the initial pathogen prevalence in these birds, the high number of infected birds consumed and the probability of cooking the meat 'pink'.

In the uneviscerated bird the combined risk to the individual is considered to be *Low* for woodpigeons, mallard, red-legged partridge and quail and *Very Low-Low* for grey partridge consumed outside home. A combined risk of *Low* is estimated for mallard and red-legged partridge and *Very Low-Low* for woodpigeon and quail consumed in the home. When taking into account the lack of evidence for consumption of these birds eaten uneviscerated the overall risk of infection considered to be reduced to *Very Low*. In a recent EFSA report on poultry meat inspection *T. gondii* was classed as a *low* risk hazard for poultry consumption in part because chicken meat is usually well cooked and most chickens are not exposed to vectors of toxoplasma, unlike gamebirds (EFSA 2012a).

Significant Data gaps and their impact on the overall risk:

- Infectious dose is unknown - low infectious dose could increase the overall risk
- Lack of data for prevalence in woodcock, widgeon and red-legged partridge introduces much uncertainty in the prevalence/concentration estimates. We have predicted prevalence/concentration for these species based on published data for other birds. If this prediction is an underestimate then the overall risk could be similarly underestimated
- Prevalence and concentration of tissue cysts in muscle tissue of game birds is unknown. It has been estimated to be low but if the true value is higher, the risk could alter accordingly



Figure 19: Qualitative scores for *Toxoplasma gondii* concentration in small game bird species at each stage of the risk assessment.

Key: S= Snipe Wo= Woodcock, WP= Woodpigeon, M= Mallard, T= Teal, Wi= Widgeon, GP= Grey Partridge, RLP= Red-legged Partridge, Q= Quail

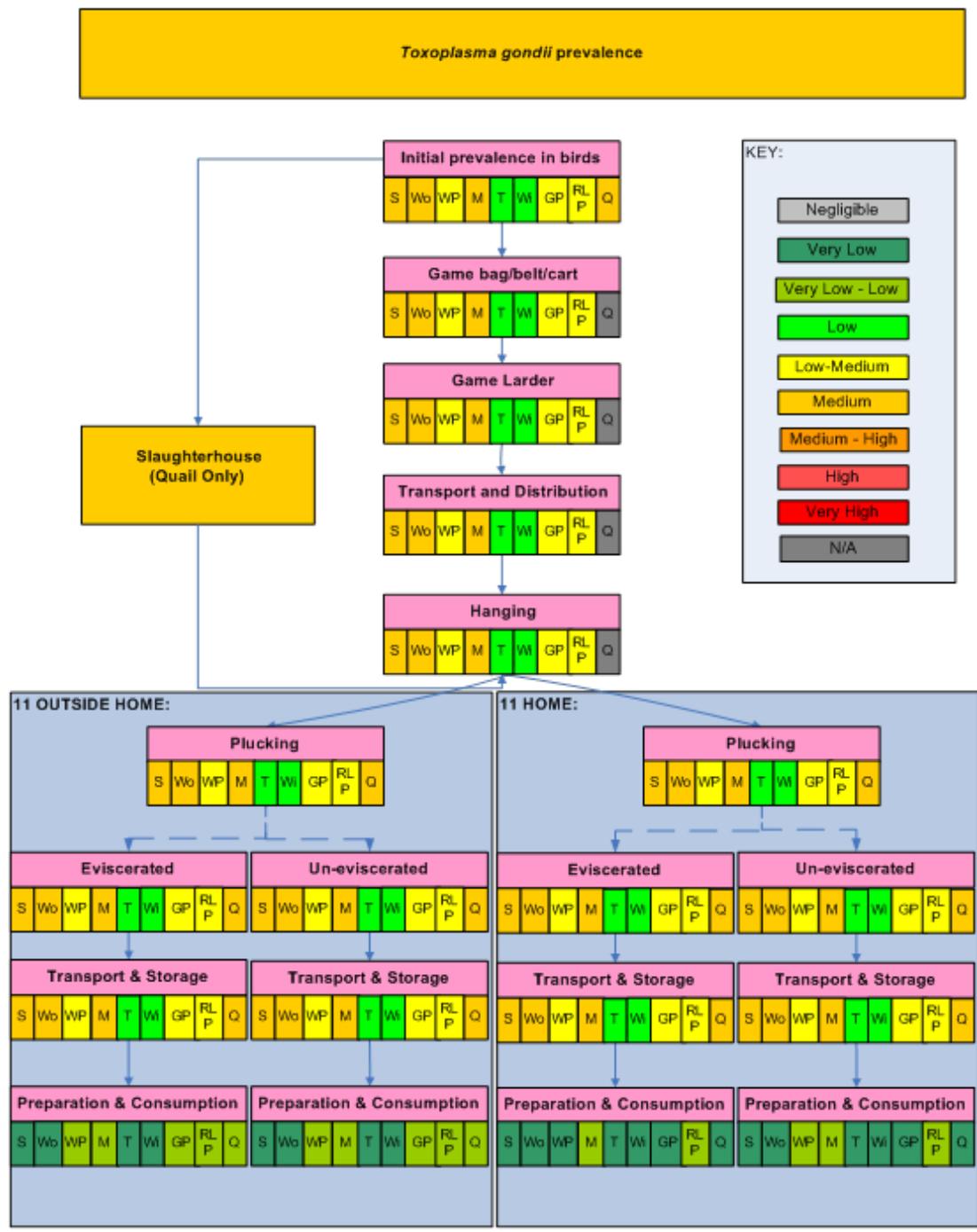


Figure 20: Qualitative scores for *Toxoplasma gondii* prevalence in small game bird species at each stage of the risk assessment.

Key: S= Snipe Wo= Woodcock, WP= Woodpigeon, M= Mallard, T= Teal, Wi= Widgeon, GP= Grey Partridge, RLP= Red-legged Partridge, Q= Quail

A1.8 Listeria

Figure 17 and 18 show the results for *Listeria* concentration and prevalence respectively. Data collected for the risk assessment (see Appendix 2) were limited but suggest that whilst birds can carry *Listeria* asymptotically in their intestines, prevalence is likely to be dictated by food and environmental contamination (Fenlon 1985). Literature has concentrated on birds known to be associated with human activities such as landfill sites and sewage outlets. Consequently the concentration and prevalence of *Listeria* in woodcock and snipe are estimated to be *Low* because of their solitary, 'wild' lifestyle and in the remaining bird species is estimated to be *Low-medium* because of their interaction with humans or other bird populations.

Listeria is a psychotropic pathogen and, as such, is capable of growth in cold environments. Whilst the probability of growth is estimated to be *Low-medium* throughout initial storage and transportation it is considered unlikely that growth will be sufficient to considerably increase the concentration of *Listeria* up to and including the Hanging stage. Pathogen concentration is likely to decrease at the evisceration stage when the intestines, containing the majority of *Listeria* infection are removed. Surface contamination via environmental contamination with this ubiquitous pathogen is considered to be reduced by the use of waxing at AGHEs.

Growth of *Listeria* can occur throughout the risk pathway. However if compliance with the Food Hygiene Regulations (2006) are undertaken then temperatures would be below 8°C and only limited growth would be expected. A slight reduction in pathogen concentration is likely to occur in the home where frozen storage, frequently for 3-6 months, will result in some bacterial inactivation.

The risk of human infection from consumption of eviscerated game birds of all species both in and outside the home is considered to be *Very Low*. The infectious dose of *Listeria* is estimated to be high (EC 1999). After preparation, it is not considered that the levels of *Listeria* present in the cooked game bird would be sufficient to pose a risk of infection.

With meats, human listeriosis is normally associated with post-processing contamination of cooked, ready-to-eat products that receive no further heating. However, the lack of knowledge on how *Listeria* could contaminate game meat must be considered as a data gap as the mechanism whereby *Listeria* contaminates foodstuffs is not fully understood. There is no data available to inform on whether gamebird carcasses would be contaminated with *Listeria* from processing machinery or the bird itself and whether such contamination could be significant in relation to public health (Geoff Mead pers. comm.). This is relevant for most pathogens, including *Listeria monocytogenes*, where there is a known association between human listeriosis and post-processing contamination of cooked, ready-to-eat meats.

In the un-eviscerated birds the overall risk of human infection was considered to be *Very Low* for all species prepared and consumed both in and outside the home.

Significant Data gaps and their impact on the overall risk:

- Lack of data for prevalence in all bird species with the exception of wood pigeons introduces much uncertainty in the prevalence/concentration estimates. We have predicted prevalence/concentration for these species based on published data for other birds. If this prediction is an underestimate then the overall risk could be similarly underestimated

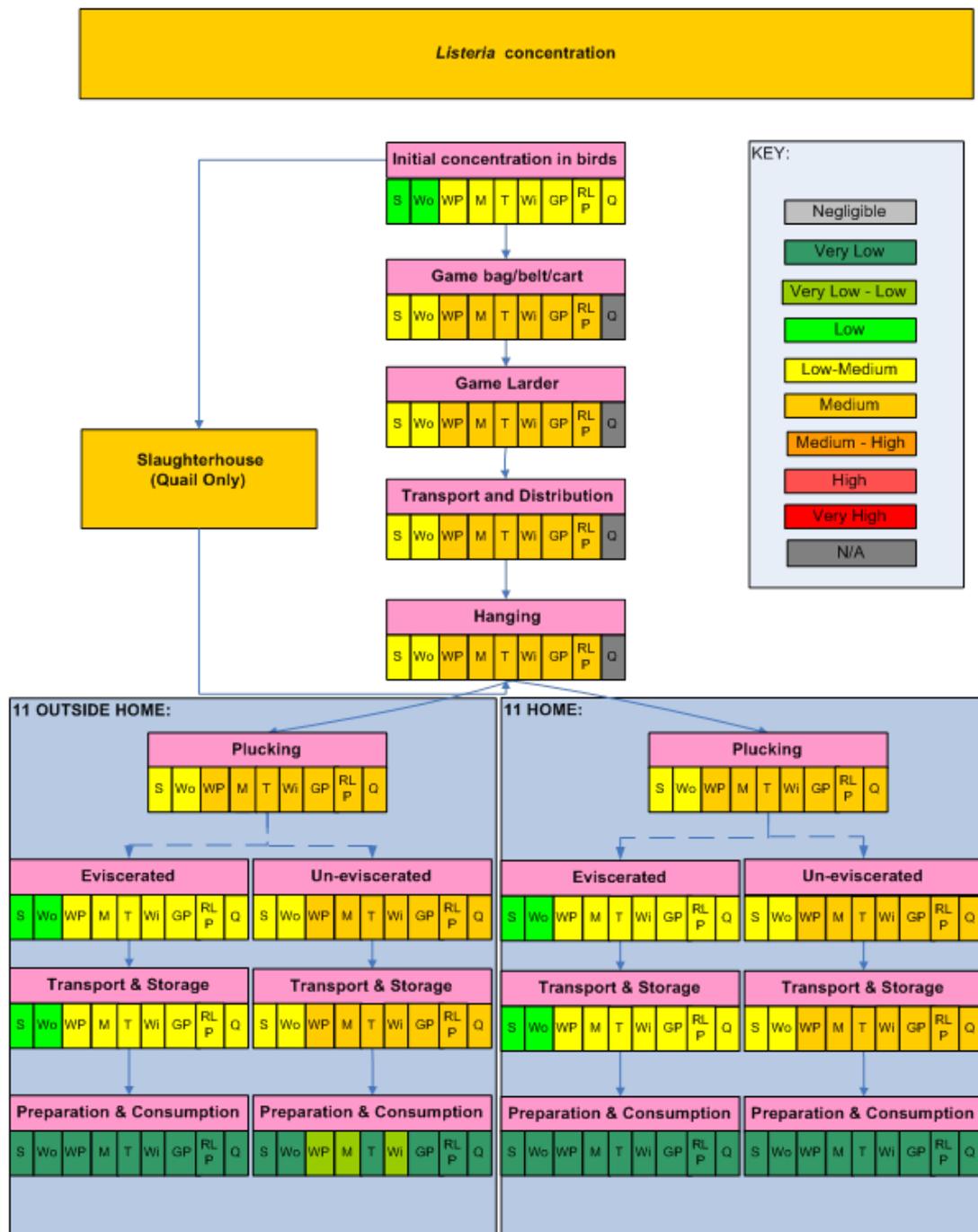


Figure 21: Qualitative scores for *Listeria monocytogenes* concentration in small game bird species at each stage of the risk assessment. Key:

S= Snipe Wo= Woodcock, WP= Woodpigeon, M= Mallard, T= Teal, Wi= Widgeon, GP= Grey Partridge, RLP= Red-legged Partridge, Q= Quail

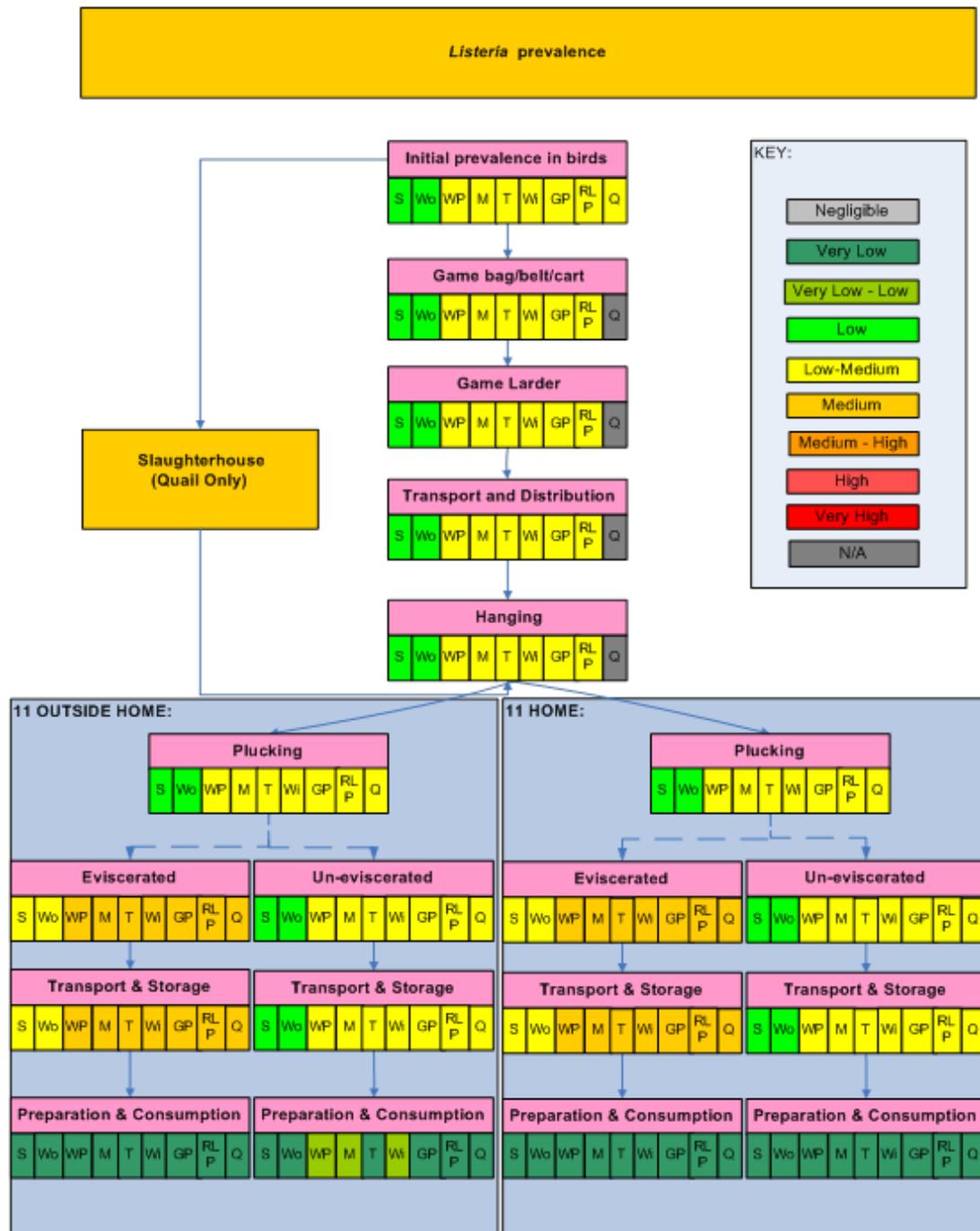


Figure 22: Qualitative scores for *Listeria monocytogenes* prevalence in small game bird species at each stage of the risk assessment.

Key: S= Snipe Wo= Woodcock, WP= Woodpigeon, M= Mallard, T= Teal, Wi= Widgeon, GP= Grey Partridge, RLP= Red-legged Partridge, Q= Quail

7. Appendix 2: Detailed derivation of qualitative scores.

A2.1 Overview

In this appendix we describe in detail the data used to determine the qualitative scores presented in the full results (Section 1.6). The Appendix is set out using the model framework as a guide (Figure 2); the numbers in the title of each subsection of the appendix relate to the number in the model framework.

A2.2 Regulations

The 2006 Food Hygiene Regulations covering the wild game bird industry require all game intended for human consumption to be handled hygienically from the point it is shot through to the point of sale or consumption including the use of any vehicles, game larders or collection centres used to transfer or store the wild game. The regulations are provided by the relevant parts of:

- Regulation 178/2002 - general food law requirements including establishing traceability of food producing animals.
- Regulation 852/2004 - the hygiene of foodstuffs
- Regulation 853/2004 - specific hygiene rules for food of animal origin
- Regulation 854/2004 - specific rules for the organisation of official controls on products of animal origin intended for human consumption

The EU regulations are implemented in Scotland by the Food Hygiene (Scotland) Regulations 2006 (SSI 2006/3) with two amendments in 2007 (SSI 2007/11) and 2010 (SSI 2010/69).

However, the regulatory requirements differ according to the supply chain of the 'primary product' incorporating a series of regulatory exemptions for small scale wild game operators. The logic of this relies on the small size of these exempt operators translating to a low threat to public health as the population at risk will be fewer. Yves LeCocq, former secretary general of the Federation of Associations for Hunting and Conservation of the EU (FACE), commented that it is when the game bird supply becomes industrialised that risks could occur. Similarly, Official Veterinarians (OV) have argued that the scale of operations of AGHEs justify the higher standards imposed (FSAS 2012a). LeCocq maintained that for the sourcing and killing of wild game responsibility for identifying possible health hazards could be vested in the hunter whilst hygiene legislation should provide for the handling, processing, transport and storage of the meat of wild game (Lecocq 1997). Regulations stipulating temperature and time requirements relating to carcass chilling should be flexible enough to take the practicalities of the primary production process into account. Legislation should contain realistic guidelines and rules for hygiene standards on design

of larders, collection centres and processing houses which are all essential links in the chain to ensure that game meat is safe from the point of origin to that of consumption.

Non-exempt operators must register as an Approved Game Handling Establishment (AGHE) with the FSA and be subject to official veterinary inspection. The OVs at AGHEs, however, are not responsible for checking the hygiene standards of the upstream supply chain; this is the responsibility of the Local Authority (LA) and there is a knock-on effect if the LA inspection is not being carried out efficiently. HACCP requirements place responsibility upon the AGHE to be confident that their suppliers handle the carcasses properly prior to delivery. The total enforcement activity that wild game represents for all LAs in one study (FSAS 2012a) was between 1-5% of activity with some LAs regarding wild game establishments to be of sufficiently low risk to be regulated without inspection.

Exemptions to the Food Hygiene Regulations allow operators to either remain unregistered or to register as a Food Business Operator (FBO) with the LA and be under their inspection remit:

- The 'Primary Producer' exemption applies to hunters supplying small quantities of the 'primary products' of hunting, *direct* to the final consumer and/or to local retailers, who also directly supply the final consumer. Local means supply to someone within the boundaries of the same local authority or adjoining local authorities but also allows mail order and internet sales. Such exempt producers do not need to register as an FBO, however, the Food Safety Act 1990 which makes it an offence to place unsafe food on the market still applies (Alliance 2008).
- The 'Hunter's' exemption and 'retail' exemption, exempts FBOs from registering their premises as an AGHE. Registration of their business must include their game larders, and any vehicle used for transporting wild game.

FBOs must accept primary responsibility for food safety and reinforce this responsibility using procedures based on HACCP principles and the application of good hygiene practice (FSA 2008). The HACCP approach requires that FBOs plan what needs to be done to maintain food safety and to follow this plan and monitor that the plan has been followed.

Private domestic supply of wild game encompasses game that the consumer has shot themselves, or has bought or been given, and will eat themselves. Although private direct sale from hunters to final consumers is regulated by general food law, in reality it is not possible to control enforcement. Furthermore expert opinion considers that a hunter's direct sale to the final consumer ensures the closest traceability and therefore the highest standards in terms of hygiene and quality of the product (FSAS 2012a).

Illegal trade is likely to be related to any game supply to, or purchase from, an unregistered wild game collection centre or carrier. In 2008 the National Game Dealers Association perceived that there was more illegal trade in the game industry than ever before (FSAS 2012a). There are on-going concerns about the levels of enforcements by LAs for FBOs with some game handling premises not being visited in the last 5 years by the LAs. AGHEs consider illegal supply from non-approved wild game operators to be widespread posing a risk to the industry. It should be taken into consideration that breaches of hygiene requirements have been suggested by anecdotal evidence but no concrete evidence. The scale of risk to public health is unknown as many of those claiming exemptions and avoiding inspection may not comply with the regulations and have a bare minimum of hygiene standards.

In 2007 the Government amended the Game Act to remove the requirement to hold a LA licence to deal in game or a game dealer's excise licence so it is no longer an offence for a person or estate to sell game to the public without having a licence to deal in game. It also enables local retailers e.g. butchers to sell game and venison without a game dealer's licence. The restriction on dealing game birds during the closed season was also removed allowing sales all year round providing the game was lawfully killed during the open season.

Official inspection at AGHEs is more geared towards animal health and quality issues, rather than microbiological contamination. However, whilst the inspection system is not designed for detecting microbiological risks in meat in general, not just in the wild game industry, birds obviously unfit for human consumption will be removed from the food chain at this point. The only effective way of controlling microbiological contamination is to apply good hygiene practice consistently. Interviewees for an FSAS (FSAS 2012a) study were of the opinion that the introduction of HACCP has considerably improved food hygiene standards, whilst inspection and testing cannot guarantee microbial safety, even though it should be carried out to verify the effectiveness of HACCP plans.

No legal limits exist for microbial numbers for carcasses or meat from wild game birds including total viable count (TVC), Enterobacteriaceae and *E. coli* which are commonly used as indicators of deficiencies in process hygiene.

In a recent review of food hygiene regulation in the Scottish Wild Game Sector commissioned by the FSAS the implementation and enforcement of the regulations since their introduction in 2006 was reviewed (FSAS 2012a). The implementations of these regulations have raised the hygiene standard in the Scottish wild game sector. According to a report from the Countryside Alliance Foundation published in 2008, the proportion of birds traceable to shoots has increased by a third, with over half of full-time keepered shoots guaranteeing traceability on all batches (Alliance 2008). Inspection of shot birds by a trained-person on the shoot had increased by 57% from 2006. In April 2008, a survey of 5,411 shoots conducted for the Game-to-Eat

campaign concluded that over half full-time keepered shoots had upgraded their game handling facilities with purpose-built chilled storage, up from one-third in 2006. Also, half of the aforementioned shoots had registered their game larder with the LA.

The report confirms that the food hygiene regulations put great emphasis upon self-regulation. They considered that a key issue in the implementation of the regulations is the evidence for a lack of consistency in the enforcement activity of LAs in relation to the FBOs. Interviewees from across the game bird industry found the regulatory exemptions to be complex and confusing.

The food hygiene regulations of 2006 have reinforced improvements to hygiene standards in the handling and processing of wild game largely through the requirement for trained hunter certification of supplies to AGHEs and management systems based on HACCP principles. However, legislation does not specify training courses for hunter's attendance, or the level of certification, and, most importantly, recreational hunters may not take up voluntary training. There is no legal requirement for a declaration to accompany small game to AGHE, though a trained person must have inspected the game and report any abnormal behaviour observed before killing or suspected environmental contamination to the AGHE. It is the responsibility of the AGHE operator to satisfy himself that those supplying him with game are suitably trained. The industry and the FSA agreed that a Vocationally Related Qualification (VRQ) is the most appropriate qualification to demonstrate that a hunter has the necessary knowledge required. However, other qualifications or training may be acceptable and guidance should be sought by AGHE operators to ensure that they meet the minimum legislative requirement (FSA 2005).

A partial generic HACCP guidance plan for producers of wild game meat either at an AGHE or under exemption has been provided by the FSA. This provides examples of the critical points where carcasses can be rejected for human consumption (FSA 2008) at each of the processing steps:

Acceptance of carcass:

- Carcass derived from unhealthy birds
- Contamination of carcass from excessive dirt on feathers
- Contamination of carcass from faecal material in body cavities due to 'belly' shots
- Growth of pathogenic bacteria on carcass due to too high temperature during transport or improper storage by supplier

Plucking:

- Contamination of carcass by pathogenic bacteria from plucking machine

Evisceration:

- Contamination of carcass by pathogenic bacteria/faecal material from ruptured stomach/intestines/crop

- Contamination of carcass by pathogenic material/faecal material from dirty knives/evisceration equipment
- Contamination of carcass by pathogenic bacteria on hands, arms, aprons of dressing staff

Chilling and chilled storage:

- Growth of pathogenic bacteria on carcass due to too high chilling and storage temperature
- Growth of pathogenic bacteria on carcass due to too slow chilling process/too long on chilling hall and close spacing of carcass during cooling.
- Contamination of carcass by pathogenic bacteria from dirty chill store/equipment.
- Contamination of carcass by pathogenic bacteria from store/handling staff

Inspection, cutting, trimming

- Contamination of meat by pathogenic material from other meat especially of other species
- Contamination of meat by pathogenic bacteria from knives, cutting tables etc.
- Contamination of meat by pathogenic bacteria from cutting staff

Package and dispatch

- Growth of pathogenic bacteria due to inadequate temperature control at dispatch
- Contamination of carcass by pathogenic bacteria from outer package during packing process
- Contamination of carcass by pathogenic bacteria from dirty vehicles
- Contamination of carcass by pathogenic bacteria from loading staff

It should be remembered that HACCP systems are only protective to the extent that the workforce and management are fully committed to their implementation (Jones, Parry *et al.* 2008). Managers of AGHEs stated that the process of game birds passing through AGHEs is in itself a form of quality control. Birds are checked on collection by the drivers and again at plucking, evisceration and packaging. Manual evisceration is often used in preference to automation as it is easier to check for any gut contamination; any gut breakage juices will cause 'greening' at the vent end.

A2.3 Pathogens and bird species

A2.3.1 Salmonella

Salmonella in man

In 2010 out of a total 99,020 confirmed cases of human salmonellosis in the EU the most frequently isolated *salmonellae* were: *S. Enteritidis* (45.0%) and *S. Typhimurium* (22.4%) (EFSA 2012b). In a susceptible host *Salmonella* replicates primarily in the mucosa of the digestive tract after oral challenge

and in some cases may spread to lymphoid tissue, the spleen, liver and various organs and tissues (Dibb-Fuller, Allen-Vercoe *et al.* 1999).

Man most commonly acquires *Salmonella* as a foodborne infection by the faecal-oral route. Raw or undercooked eggs, pig meat, poultry meat and environmental exposure are the most common vehicles of infection; other foods cross-contaminated during preparation, storage or serving may be involved (Friedman, Torigian *et al.* 1998) . Inadequate cooking has been cited as a contributing factor in 67% of *Salmonella* related outbreaks (Murphy, Osaili *et al.* 2004). The incubation period is 12 to 72 hours depending on the infectious dose, the *Salmonella* serotype, and specific host factors. The number of organisms ingested, the vehicle of infection and specific host factors are important in determining the outcome of exposure.

The infectious dose of *Salmonellae* can vary, depending on the bacterial strain ingested as well as on the immuno-competence of individuals (EC 2003). Data from outbreaks of foodborne disease indicate that infections can be caused by ingestion of as few as 10-45 cells (D'Aoust 1985) (Lehmacher, Bockemuhl *et al.* 1995) and that the infectious dose is lower when present in food with a high fat or protein content. An infectious dose of *S. Typhimurium* as low as 10 organisms was found in chocolate (Kapperud, Gustavsen *et al.* 1990)), and as low as 6 organisms for *S. Enteritidis* in ice cream servings (Hennessy, Hedberg *et al.* 1996). However, the infectious dose of *Salmonella* is generally considered to be relatively high, in the region of 10^4 cfu for most food types. Most patients develop a gastrointestinal illness with acute diarrhoea as the main symptom. Other common symptoms include abdominal pain or cramps, fever, chills, nausea, vomiting, pain in the joints, headache, myalgia, and general malaise. Infection is usually self-limiting although complications relating to bloodstream infections can occur. Between 2 % and 15 % of episodes of *Salmonella* gastroenteritis are followed by symptoms of self-limiting reactive arthritis.

There were 737 reported cases of *Salmonella* in Scotland in 2011 including 238 *S. Enteritidis* and 199 *S. Typhimurium* (HPS 2011). It should be noted, however, that private household outbreaks are not recorded in Scotland. For 2011 in England and Wales there were 8,534 total recorded *Salmonella* isolates of which 2,670 were *S. Enteritidis* and 2,239 were *S. Typhimurium* (Defra 2012). Under reporting of salmonellosis occurs with an estimated ratio of 1:5 between reported cases and actual incident rates within the community (Wheeler, Sethi *et al.* 1999);(Tam, Rodrigues *et al.* 2012). The most common food types associated with *Salmonella* outbreaks in 2011 were red meat and imported eggs (Defra 2012).

Birds and Salmonella

Avian *Salmonella* infections are important as both a cause of clinical disease in poultry and as a source of food-borne transmission of disease to humans. *Salmonella Pullorum* (pullorum disease) and *Salmonella Gallinarum* (fowl typhoid) are especially adapted to poultry mainly affecting hens and

turkeys. Birds can become infected with many different types of *Salmonella*, the most important, with regard to human illness, being *S. Enteritidis* and *S. Typhimurium*. Domestic fowl commonly harbour *S. Enteritidis* and other zoonotic serovars without it causing discernible illness in the birds, though this bacterium is the cause of food-borne outbreaks of salmonellosis in humans through the consumption of contaminated eggs (Guard-Petter 2001).

Young game birds reared in captivity can be exposed to *Salmonella* via manufactured feed as this bacterium is a common contaminant of animal feeding stuffs. On release of the birds into the wild, their natural diet may be less favourable for *Salmonella* colonisation of the gut (Geoff Mead pers. comm. cited in (Coburn 2003)).

S. Typhimurium is the serotype most commonly associated with wild birds (Benskin, Wilson et al. 2009) with *S. Enteritidis* being very rare in this population (Rob Davies pers. comm.). Asymptomatic *Salmonella* carriage in wild birds is known to occur and wild birds have been implicated as vectors on farms and in feed mills. Qualitative results from wild bird faeces on pig farms suggest average numbers of organisms are low (1-100/g faeces) (Rob Davies pers. comm.)

The incidence of *Salmonella* in wild birds tends to be low, often in just a few per cent of samples (reviewed in (Abulreesh 2007)). These studies suggest that wild birds may acquire *Salmonellae* after exposure to human or food animal contaminated environments, for example, refuse tips, farms and sewage sludge; birds that live away from such environments are unlikely to harbour *Salmonella* (Tizard 2004). There is limited data on the prevalence of *Salmonella* in wild game birds but a previous risk assessment judged the prevalence of *Salmonella* to be Low (Coburn 2003) and caused by a variety of serotypes, not all of which are associated with human illness.

Salmonella is common in intensively reared galliform birds (fowl-like birds including partridge and quail). The clinical symptoms associated vary considerably by age group and serotype. Infections with generalist serotypes rarely cause clinical disease in galliform birds and most become asymptomatic carriers. Infections with *S. Enteritidis* are typically asymptomatic in adult birds but can cause systemic disease in young birds.

Wild game are perceived as being healthier than intensively farmed animals with the health risk of food poisonings being low (FSAS 2012a) Public health authorities in Scotland have no record of any food borne disease outbreaks that can be definitively traced back to this source. There is some anecdotal evidence for the occurrence of food poisoning but generally the view is that game is of low biological hazard, is likely to be thoroughly cooked and has not been found to carry significant pathogens or to be identified as causing food poisoning.

Survivability characteristics

The survival ability of different *Salmonella* serotypes varies. *Salmonella* can grow in the temperature range 8°C - 43°C with optimum growth between 25°C and 41°C (Rob Davies pers. comm.). *Salmonella* is likely to survive refrigerated and frozen storage. Storing at refrigerated temperatures below 5°C throughout the chain of distribution, storage and retail sale is important, because *Salmonella* can multiply at temperatures exceeding 6°C (Oscar 2002). The lowest recorded temperature at which growth has occurred in a food product is 6.7°C (EC 2003). Survival of *Salmonella* Typhimurium DT104 on chicken skin was observed during 8 h of storage at 5 to 20°C and at 50°C, whereas growth was observed from 25 to 45°C and was optimal at 40°C with a lag time of 2.5 h and a specific growth rate of 1.1 log/h (Oscar 2009).

Freezer storage at -20°C or lower favours greater *Salmonella* survival than storage at refrigeration temperatures. The persistence of *Salmonella* at freezer temperatures is strain dependent and varies with food composition (Labbe and Garcia 2001)). White and Hall (White 1984)) showed that the numbers of *S. Typhimurium* decreased during frozen storage by approximately 99% after 168 days of storage, and by 90% for *S. Hadar* in a similar period. They also showed that the numbers of *S. Typhimurium* increased by 1.8 log cycles after 24 hours thawing at 20°C and by 2.93 log cycles after the same period at 27°C, cited in (WHO 2002).

The response of *Salmonellae* to heat can be quantified by means of the D-value and z-value. D-value is the time in minutes at a given temperature to achieve a 90% reduction in numbers of viable bacteria. The z-value is the temperature change to effect a 10-fold change in the D-value. *Salmonella* in chicken has been found to have a D-value of 0.176 minutes at 70°C and 0.286 minutes at 67.5°C (Murphy, Marks *et al.* 1999).

A2.3.2 Campylobacter

Campylobacter in man

Campylobacter is recognised as one of the main causes of bacterial foodborne disease in many developed countries. Campylobacteriosis in humans is caused by thermotolerant *Campylobacter* spp. The species most commonly associated with human infection are *C. jejuni* followed by *C. coli* and *C. lari*, although other *Campylobacter* species are also known to cause infection. Two independent studies found the cause of campylobacteriosis in the UK and Scotland to be 90-93% caused by *C. jejuni* with the remainder being mostly *C. coli* (Gillespie, O'Brien *et al.* 2002); (Sheppard, Dallas *et al.* 2009).

The infective dose of these bacteria is generally low although it can be dependent on the immune status of the individual. Illness has been reported with doses as low as 500 organisms (Newell 2002) whilst doses as high as 10⁹ cfu did not always cause illness. In a human volunteer study reported by

(Black 1993) there was no apparent dose relationship with illness. Infection normally has an incubation period averaging from two to five days. Examination of a bottle of bird-pecked milk, which was part of a batch implicated in an outbreak at a nursery, revealed contamination levels of approximately six cells per 500ml of milk (Riordan, Humphrey *et al.* 1993).

Where infection is attributable, human infections are most commonly associated with consumption of undercooked, contaminated poultry meat (Harris, Weiss *et al.* 1986). Cross-contamination of other foods by introducing contaminated poultry into the kitchen is the main route for human infection. In most case-control studies of *Campylobacter* infection the majority of cases remain unexplained. It has been suggested that between 20% and 40% of sporadic disease might be due to the consumption of chicken. Symptoms can be mild to severe with common clinical symptoms including watery, sometimes bloody diarrhoea, abdominal pain, fever, headache and nausea. Usually, infections are self-limiting and last only a few days. Infrequently, extraintestinal infections or post-infection complications such as reactive arthritis and neurological disorders occur. *C. jejuni* is also a recognised antecedent cause of Guillain-Barré syndrome (EFSA 2012b).

In 2011 there were 72,150 confirmed cases of *Campylobacter* in England and Wales (Defra 2012) and 6365 in Scotland (HPS 2011). It has been estimated that the ratio between incidence rates of *Campylobacter* in the community and reported to the GP is 7.2 (Tam, Rodrigues *et al.* 2012). This high level of under reporting is thought to be mainly down to self-management of symptoms. The total economic burden of campylobacteriosis has been estimated to be £500 million in the UK in terms of treatment costs, lost production and human welfare (FSA 2007).

Birds and Campylobacter

Campylobacter spp. can be isolated from the intestinal tract of most warm blooded animals, however, the favoured environment appears to be the intestinal mucosa of birds where *Campylobacter* colonize as a commensal organism (Hartog, Wilde *et al.* 1983) (Newell and Fearnley 2003). Birds are ideal hosts for *Campylobacter*, due to their relatively high body temperature (42°C), the optimum temperature for growth of this organism. The presence of *Campylobacter* species in normal healthy birds appears to be influenced by feeding behaviour with raptors, scavengers and ground-foraging guilds showing higher rates of colonisation (Waldenstrom, Broman *et al.* 2002); (Hughes, Bennett *et al.* 2009). Conversely, the intestines of birds whose diet consists of insects or grain show little, if any, presence of *Campylobacter*.

In a comparison of genotypes and serotypes of *C. jejuni* isolated from Danish wild birds and from broiler flocks and humans the serotype distribution in wildlife was significantly different from the known distribution in broilers and humans. Human and broiler isolates show a larger serotype overlap. Environmental sources, such as wild birds, are believed to be important

reservoirs of *Campylobacter* infection in broiler chicken flocks however, the relatively low number of wild bird strains with an inferred clonal relationship to human and chicken strains suggests that the importance of wildlife as a reservoir of infection is limited (Petersen, Nielsen *et al.* 2001).

The high prevalence of *C. jejuni*, *C. coli* and *C. lari* in healthy wild birds has identified them as a potential reservoir in nature and as a possible source for human infections. However, there is evidence that the *Campylobacter* clones isolated from birds may not be pathogenic to humans (Broman, Waldenstrom *et al.* 2004). Isolates from migrant birds often have subtypes similar to birds of the same species or feeding guild but are rarely similar to isolates from humans unless associated with human activities, for example, refuse dumps, bird feeders etc. (Broman, Waldenstrom *et al.* 2004). Multilocus sequence typing (MLST) has been used to quantify the relative contributions of different sources of human *Campylobacter* infection in Scotland in 2005-2006 (Sheppard, Dallas *et al.* 2009). Chickens were the dominant source of campylobacteriosis (76%) whilst the contribution of wild bird sources was low (<4%). Similarly, characterisation of *C. jejuni* isolates from wild bird populations revealed that wild birds carry both livestock-associated and unique strains of *C. jejuni*. However the absence of unique wild bird strains of *C. jejuni* in livestock suggests that the direction of infection is predominantly from livestock to wild birds (Hughes, Bennett *et al.* 2009).

Survivability characteristics

Campylobacter is unique amongst food poisoning bacteria in that it can only grow at above ambient temperatures so is unable to grow at temperatures normally used to store food. The temperature range for growth is 30 - 45 °C, with an optimum of 42 °C. Although survival at room temperature is poor, *Campylobacter* can survive up to 15 times longer at 2 °C than at 20 °C. Freezing and frozen storage at -20°C can cause a 100-fold reduction in numbers (Geoff Mead pers. comm.) but survival of *Campylobacter* is greater at colder temperatures, for example, -80°C (Rob Davies pers. comm.). Chicken was sold predominantly frozen in Iceland prior to 1996. Increased consumer demand led to the sale of chilled chicken after this time and coincided with human *Campylobacter* infections increasing from ~ 10 cases per 100,000 to a rate of 116/100,000 3 years later (WHO 2009).

Thermal inactivation of *C. jejuni* begins at 46°C (Labbe and Garcia 2001). *C. jejuni* was still present on undercooked turkey thighs with an internal temperature of 54°C. However, roasting, braising and stewing were effective methods of destroying *C. jejuni* on contaminated turkey thighs even when meat was undercooked (Acuff, Vanderzant *et al.* 1986). When deep muscle has been contaminated the organism, if present, may survive marginal cooking procedures.

As *Campylobacter* cannot grow at ambient and sub-ambient temperatures, the main risk in the domestic and catering kitchen will be associated with cross-contamination of raw foods to ready-to- eat foods, either directly or

indirectly from hands and work surfaces/kitchen utensils, and undercooking of contaminated raw foods (ACMSF 2005).

A2.3.3 *Escherichia coli* (Toxigenic)

E. coli (Toxigenic) in man

E. coli is a large and diverse group of bacteria. Commensal *E. coli* live harmlessly in the intestines of all animals and form a significant part of the healthy human intestinal microflora. However, some strains, whilst asymptomatic in animals, are pathogenic to humans through the presence of specific virulence factors. VTEC refers to both Vero cytotoxin-producing *Escherichia coli* and Verotoxigenic *Escherichia coli*, the most common being *E. coli* O157:H7. Shiga-toxin-producing *Escherichia coli* (STEC) are synonymous with VTEC. From a zoonotic point of view, VTEC is the *E. coli* pathogenicity group of most interest, as they are able to cause severe disease in humans when transmitted through the food chain or environment from their animal reservoirs.

The spectrum of disease in man caused by VTEC can range from mild to severe bloody diarrhoea to complications including haemolytic uremic syndrome (HUS) and thrombotic thrombocytopenic purpura (TTP). The typical incubation period is 3-4 days. 10% of patients develop HUS or other serious complications with 0.6% of cases proving fatal (HPA). Some people with VTEC have typical gastroenteritis symptoms which clear up within a week. Antibiotics are not recommended for treatment as they can increase the risk of further complications including HUS (HPA).

The infectious dose is lower than for many other enteric pathogens and has been reported to be fewer than 50 organisms (Wasteson 2001), and possibly as low as 10 (cited (Kudva *et al.* 1998)). Microbiological testing of meat from samples consumed by persons who became ill was suggestive of an infectious dose for *E. coli* O157:H7 of fewer than 700 organisms (Tuttle *et al.* 1999).

There were a total of 1407 reported cases of *E. coli* O157 in England and Wales in 2011 (Defra 2012) and 212 in 2010 in Scotland (HPS 2011); Scotland has the highest rate in the UK (Defra 2012). Cattle are the most important direct reservoir of VTEC O157 in the UK. Human infection can be as a result of: contact with infected animals or humans, exposure to contaminated water or faecally contaminated environment, consumption of contaminated foodstuffs.

Birds and E. coli (Toxigenic)

Commensal *E. coli* are part of the normal gut microbial flora of birds and most isolates are non-pathogenic. Although *E. coli* can be found on the skin and feathers of birds the most important reservoir of *E. coli* is the intestinal tract of animals, including poultry. Certain strains such as those designated

as avian pathogenic *E. coli* (APEC) spread into various internal organs and cause colibacillosis characterised by systemic, often fatal, disease in birds (Kabir 2010). Colibacillosis is an economically important disease which is prevalent throughout the world. Birds can also be asymptomatic carriers of VTEC *E. coli* in their intestines that are infectious to humans (Hughes, Bennett et al. 2009), but this is thought to relate to vector rather than carrier status in most cases.

Survivability characteristics

Growth can occur at +7°C (and possibly as low as + 4°C) to +44.5°C with an optimum for VTEC around +37°C. In laboratory media, the optimum temperature for growth of multiple isolates of 0157 was 40.2°C (Stringer, George *et al.* 2000). *E. coli* 0157:H7 can persist in frozen and chilled conditions although there is likely to be a reduction in pathogen number. When seeded into ground beef it has the ability to survive up to 9 months in frozen storage at -20°C (Kraft 1992). Heat treatment at 70°C for 2 minutes results in a 6-log reduction in *E. coli* 0157 numbers (Stringer, George et al. 2000).

A2.3.4 *E.coli* (Antibiotic resistant strains)

E.coli (Antibiotic resistant strains) in man

Extended - Spectrum Beta-Lactamases (ESBLs) are enzymes that can be produced by bacteria making them resistant to extended spectrum penicillins and cephalosporins widely used for high risk cases in hospitals (HPA). Extended-spectrum beta-lactamases producing *E. coli* (ESBL-E.coli) represent a major problem in human and veterinary medicine. The spread of ESBL-E.coli into the environment appears to be directly influenced by antibiotic practice and the level of resistant bacteria observed in wild animals appears to correlate well with the degree of association with human activity (Allen, Donato *et al.* 2010); (Skurnik, Ruimy *et al.* 2006). Horizontal transfer of resistance genes from clinical isolates or the intake of already resistant bacteria from human waste, sewage and domesticated animal manure is a probable cause. The common use of antibiotics in aquaculture of fish is important due to possible direct influences on waterbirds.

The total burden of human infection of ESBL-producing bacteria is not entirely known, nor is the prevalence of human faecal carriage. The data on frequency of occurrence in invasive infections in humans in Europe come from the European Antibiotic Resistance Surveillance System (EARS-Net: www.ecdc.europa.eu/en/activities/surveillance/EARSNet/Pages/index.asp) Human cases of bloodstream infections and infections of cerebrospinal fluid due to these bacteria have been increasingly reported from hospitals in Europe since the year 2000 . Infections with such resistant organisms may be more difficult to treat, and there is some evidence of increased severity compared with non-resistant *E. coli* infections (Schultsz and Geerlings 2012). Although there is no firm evidence at this time, various studies support the

theory that transfer of ESBL and/or AmpC-producing organisms from food animal production to humans is likely to be taking place (Lavilla, Gonzalez-Lopez *et al.* 2008). These include studies suggesting that *E. coli* isolates from poultry are genetically related to human pathogenic *E. coli*. (Johnson, Sannes *et al.* 2007); (Vincent, Boerlin *et al.* 2010)). In a recent study from the Netherlands, the results are suggestive of transmission of ESBL genes, plasmids and clones from poultry to humans, most probably through the food chain (Leverstein-van Hall, Dierikx *et al.* 2011). In Canada, Dutil *et al.* (Dutil, Irwin *et al.* 2010) reported on observed temporal links between the use of ceftiofur in chickens followed by the occurrence of resistant *E. coli* strains in chickens and humans. This occurrence of resistance decreased after reducing the use of this routine prophylactic medication and increased after it was re-introduced for economic reasons. A recent EFSA opinion (EFSA 2012a) indicated that transmission of ESBL genes, plasmids and clones from poultry to humans is most likely to have emerged following the routine use of ceftiofur mixed with Marek's disease vaccine injection or by spray in hatcheries for preventive treatment of day-old chicks.

It is difficult to precisely estimate the quantitative contribution of ESBL-/AmpC-carrying *E. coli* from poultry to human infections, largely relating to the different levels and sensitivities of monitoring and testing options and lack of harmonised methods for determining resistance and assigning its genetic background (EFSA 2012a). Accumulating evidence through specific studies in some countries has resulted in a medium- to high-risk categorization for this emerging hazard, based on expert opinion. Manges *et al.* (Manges and Johnson 2012) conducted a case-control study between April 2003 and June 2004 and they demonstrated that antimicrobial resistant, urinary tract infection (UTI) causing *E. coli* could have a food reservoir, possibly in poultry or pork. Uncontrolled, avian *E. coli* represents a serious animal welfare concern and risk to public health as it is a zoonotic organism with avian *E. coli* species known to adapt to humans.

DePoorter (2012) assessed human exposure to 3rd generation cephalosporin resistant *E. coli* through consumption of broiler meat in Belgium. They estimated that the probability of exposure to 1000 cfu of resistant *E. coli* or more during consumption of a meal containing chicken meat is ~ 1.5%, the majority of exposure being caused by cross-contamination in the kitchen. However the risk of this exposure to human health could not be estimated given a current lack of understanding of the factors influencing the transfer of these resistant genes from *E. coli* to the human intestinal bacteria and data on the further consequences on human health (Depoorter, Persoons *et al.* 2012)

Birds and E. coli (Antibiotic resistant forms)

Collibacillosis in birds can be controlled with antibiotic therapy and there has been a significant increase in drug resistant strains of *E. coli* in the poultry industry. Antibiotics can also be used in feed and water to control disease. Indiscriminate use of antibiotics has provided selective pressure for the emergence of drug-resistant strains of bacteria associated with poultry

products (Roy, Purushothaman *et al.* 2006). Transmission of R-plasmids from *E. coli* from poultry to human strains occurs very commonly in laboratory studies. Earlier studies revealed that use of fluoroquinolones in poultry was not appropriate due to the cross-resistance with fluoroquinolones used to treat human enteric infections. High resistance to chlortetracycline and oxytetracycline is of major concern because of the use of the same antibiotics in human medicine and poultry or in other food animals and the emergence of drug-resistant human pathogens.

Wildlife is not normally exposed to clinically used antimicrobial agents but can acquire antimicrobial resistant bacteria through the environment where water polluted with faeces appears to be the most important vector (Guenther, Ewers *et al.* 2011). Fluoroquinolone usage and resistant organisms are, however, very common in the industrial production and rearing of game birds such as pheasants and red-legged partridges (Rob Davies *pers. comm.*). Current literature indicates that wild birds could be the main wildlife hosts for ESBL-*E.coli* (Guenther, Ewers *et al.* 2011) in particular waterfowl/water related species and birds of prey. The dominance of waterfowl can be explained by faecal pollution of water by human or livestock sources.

At slaughter resistant strains from the gut can soil the carcass and contaminate it with multiresistant *E. coli*. The pathogen can go on to infect humans and may colonise the human intestinal tract (Kabir 2010).

Use of antibiotics in game bird rearing

Medicines specifically licensed for gamebirds in the UK are limited which could lead to the possibility of sub-optimal control of some diseases and parasites (Council 2008). According to the National Office of Animal Health (NOAH) for 2010 there are 9 products approved for game birds and 6 for ducks - compared to 115 for poultry. All antibiotics are prescription only medicines which can only be supplied by vets (POM-V). There are many products available for poultry market that vets can use for gamebirds under the cascade system. These products are prescribed by vets under their own responsibility and, as safety and efficacy data has not been generated specifically for game bird species, they carry compulsory withdrawal periods of 28 days for meat products if used for human consumption. Antibiotics may be prescribed for chicks if a definite infection is observed; *E. coli* is the most usual pathogen for which antibiotics are used. No single piece of legislation specifically regulates the breeding and rearing of birds for sport shooting. The code of practice for Scotland for the welfare of gamebirds reared for sporting purposes (Government 2011) applies to birds up to and including the period when they are confined to the release pen. Recommendations for best practice are, however, vague stating that 'Preventative use of medicines should only be carried out where appropriate and in conjunction with good husbandry practices'.

Survivability Characteristics

There are indications that the presence of multi-antibiotic resistant genes can render *E. coli* more sensitive to heat than non-resistant forms of the bacteria. Fluoroquinolone resistant strains showed a recovery rate of 83% from frozen swab samples (Lautenbach, Santana *et al.* 2008) and are considered to be able to survive at frozen temperatures.

A2.3.5 *Chlamydophila psittaci*

Chlamydophila psittaci in man

Chlamydophila psittaci is an obligate intracellular bacterium causing psittacosis (also known as ornithosis or avian chlamydiosis) in birds and is well-known as a zoonotic agent. Transmission to humans through inhalation of contaminated aerosols originating from feathers, faeces or the environment of birds is the most common route but handling of plumage, carcasses and tissues (via evisceration) of infected birds also present a zoonotic risk (EC 2002); (Deschuyffeleer, Tyberghien *et al.* 2012)). Transmission to consumers by ingestion has never been reported and data could not be identified linking the consumption of game bird meat to human disease. There are documented reports of outbreaks of psittacosis in workers at commercial poultry processing plants (PPP) (Meyer 1955); (Andrews, Major *et al.* 1981); (Newman, Palmer *et al.* 1992); (Dickx, Geens *et al.* 2010)). A recent risk assessment on the management of *C. psittaci* in PPPs found that most human infections are detected at reception, hooking, slaughtering, de-feathering and evisceration. On reception the living birds are actively shedding *C. psittaci* cells due to stress whilst during evisceration infected air sacs and lungs are exposed to the environment (Deschuyffeleer, Tyberghien *et al.* 2012).

There are seven different avian strains of *C. psittaci* each associated with a varying degree of virulence in animals and humans. The minimal infecting dose of the different strains for humans remains unknown. The disease in humans varies from a flu-like syndrome to a severe systemic disease with pneumonia and possibly encephalitis. The disease is rarely fatal in patients treated promptly. Infected humans typically develop headache, chills, malaise and myalgia, with or without signs of respiratory involvement; pulmonary involvement is common (Johnston, Eidson *et al.* 1999). It is thought that there is a significant amount of under diagnosis and under-reporting based on population sero-surveillance studies (HPA).

The numbers of reported cases of *C. psittaci* in humans are generally low. There was 5 reported case of *C. psittaci* recorded for Scotland in 2010 and 1 so far in 2011 (HPS 2011) and 41 for 2011 in England and Wales (Defra 2012). No information was available to suggest how many of the cases had close contact with birds. A psittacosis outbreak in Tayside Scotland was reported between December 2011 and February 2012. The outbreak involved three confirmed and one probable case and the epidemiological pattern suggested person-to-person spread as illness onset dates were consistent with the

incubation period and no single common exposure could explain the infections (HPA ; Defra 2012). The Swedish Institute for Communicable Disease Control is currently seeing an unusual increase of human *C. psittaci* infection in some areas of Sweden. Interviews with patients have revealed that many have tended bird feeders in their gardens. As shedding has been shown to increase when birds are stressed, the unusually cold weather this winter may have affected the shedding rate of bacteria thereby making wild birds more contagious (ProMed 2013).

Birds and Chlamydophila psittaci

C. psittaci can cause disease in a wide range of domestic birds, including game birds, and the disease can be particularly severe in ducks (Sandihill vets). Symptoms in birds are primarily respiratory in nature but many birds are asymptomatic carriers shedding the bacteria in faeces and respiratory tract secretions (Andrews & Major 1981). *C. psittaci* is endemic in nearly all bird species thereby posing a large potential zoonotic reservoir (Deschuyffeleer, Tyberghien et al. 2012). The strains isolated from wildbirds are not thought to be normally pathogenic for these hosts but the same strains can be highly virulent for domestic fowl and humans. There are 8 common serovars with corresponding host association, 1 with ducks and 2 with pigeons (EC 2002); psittacine birds and pigeons have the highest infection rates. Once within a flock, *C. psittaci* is primarily spread between birds by inhalation of desiccated droppings and secretions, both ocular and nasal, from infected birds, or through ingestion of contaminated faeces ((Takahashi, Takashima *et al.* 1988). The infection may be transmitted to fledglings in the nest by parent birds that are shedding the organism (Burnet and Rountree 1935) and there is evidence of transmission through eggs (Vanrompay, Mast *et al.* 1995).

Survivability characteristics

Chlamydia can survive in a killed game bird for as long as the carcass is kept 'fresh' in an edible but uncooked state (Alisdair Wood pers comm cited in (Coburn 2003). Chlamydia needs living cells to replicate, so it would not replicate to any extent after the death of an infected bird. It is possible that there may be a very limited chance for it to multiply while the carcass cools down, since some cells survive for a number of hours after death, but it cannot multiply after death in the way that food poisoning bacteria can (Alisdair Wood pers comm). Sparse evidence suggests that *C. psittaci*, if present, is likely to survive chilling and frozen storage. *C. psittaci* could still be isolated from turkey carcasses after 372 days at -20°C (Page 1959). Dilute suspensions (20%) of infected tissue homogenates are inactivated by incubation for 5 minutes at 56°C, 48hr at 37°C, 12 days at 22°C and 50 days at 4°C (Page 1959).

A2.3.6 *Toxoplasma gondii*

Toxoplasma gondii in man

Toxoplasmosis is a disease caused by the parasite *Toxoplasma gondii*, which can infect all mammal and bird species and is found throughout the world. *T. gondii* can be found in the faeces of infected cats, and in the meat of infected animals. All animals can pass on the infection if they enter the foodchain as the parasite can form microscopic cysts throughout the body where they remain for many years or decades. Cats are the only species in which *T. gondii* can undergo the sexual part of its life-cycle. The parasite multiplies in the cat's gut lining and is shed in the faeces in the form of microscopic eggs (oocysts). Once cats have been infected they can spread the parasite in their faeces for a few weeks. Oocysts can also persist in the soil for up to 18 months. This persistence of oocysts in the environment increases the probability of transmission to wildlife (Dabritz and Conrad 2010).

Humans can be infected with *T. gondii* by four major routes:

- Ingesting water, food or soil contaminated with the faeces of infected cats containing oocysts
- Transmission from a newly infected mother to the foetus
- Ingesting or handling undercooked or raw meat (mainly pork or lamb) that contains the tissue cyst form of the parasite
- Receiving organ transplants or, very rarely, blood products from donors very recently infected with toxoplasmosis

The relative importance of ingestion of oocysts from contaminated environments versus tissue cysts from the consumption of meat and offal is unclear. A US study reported that more than 70% of infections are related to unrecognized oocyst exposure (Boyer, Hill *et al.* 2011). However, the observed decline in toxoplasma seroprevalence noted in many developed countries over recent decades has been attributed largely to factors associated with risks from meat. Modern farming systems, including housed management, have resulted in a reduction in the incidence of tissue cysts in meat and more meat is now frozen prior to consumption. Whereas oocyst contamination of the environment is an important risk factor in infection, consumption of undercooked meat is likely to be an important risk factor for pregnant women and immune-compromised groups (ACMSF 2012).

Owing to the lifelong impact of symptoms related to toxoplasmosis, the burden of disease is high and *T. gondii* ranks highest in population burden (Disability Adjusted Life Year (DALY)) among 14 foodborne pathogens from both an individual and a population perspective (Havelaar, Haagsma *et al.* 2012). Studies have estimated that between 7-34% of people in the UK have been infected with *T. gondii* (HPA). However, as symptoms in healthy people are generally mild and non-specific, a significant proportion of cases probably go unnoticed. Accurate figures are not available but it is estimated that 350,000 people become infected with toxoplasma each year in the UK, of which 10-20% are symptomatic although a review of outbreaks of toxoplasmosis has suggested that up to a further 50% may experience mild symptoms (ACMSF 2012). On the basis of assessments made in the USA and Netherlands the costs of the relatively small proportion of cases with severe

disease make toxoplasmosis one of the most costly of gastro-intestinal infections.

The severity of *Toxoplasma* infection in humans is high. *Toxoplasma* ranked fourth in hospitalizations and third concerning deaths when compared to other foodborne pathogens in the USA (Mead, Slutsker *et al.* 1999) whilst, in France, it was the third cause of death due to foodborne infection (35 cases per year), preceded by *Salmonella* (92-535 cases) and *Listeria* (78 cases) (Vaillant, de Valk *et al.* 2005). Both the previous studies dealt with data collected from the late 1990s when the contribution of AIDS cases with toxoplasmosis was high.

The two groups of people most at risk of *Toxoplasma* infection are pregnant women and people with weakened immune systems. For the latter symptoms include headaches, confusion, seizures, chest pains, coughing up blood and breathing difficulties; the disease can be fatal in some cases. If a woman becomes infected with toxoplasma for the first time while she is pregnant, the infection can be passed on to her unborn child and cause congenital toxoplasmosis, including blindness and neurological abnormalities. The risk of transmission is greatest in late pregnancy; however, the severity of disease is greatest where the infection is transmitted to the unborn child in early pregnancy (ACMSF 2012). The latent form of the disease can also have behavioural effects in humans. The parasite has been linked to higher rates of car accidents, schizophrenia, and altered personality in affected humans (Elmore, Jones *et al.* 2010).

The infectious dose of *T. gondii* oocysts for humans is not known (Wainwright, Lagunas-Solar *et al.* 2007). Depending on the *Toxoplasma* strain, ingestion of as few as 10 sporulated oocysts may cause an infection in intermediate hosts, such as pigs (Dubey, Lunney *et al.* 1996). There were 23 reported cases of *Toxoplasma* in 2011 in Scotland with a peak of 69 in 2009 (HPS 2011) and 364 for 2011 in England and Wales (Defra 2012).

Risk factors for *T. gondii* infection may reflect differences in eating habits of consumers or different prevalence of infection in meat-producing animals in these regions. Thus, in Norway only 3% of slaughter pigs are infected with *Toxoplasma* (Skjerve, Waldeland *et al.* 1998), whereas 36% of slaughter pigs are infected in Poland (Bartoszcze, Krupa *et al.* 1991). In livestock, tissue cysts of *Toxoplasma* are most frequently observed in various tissues of infected pigs, sheep, and goats, less frequently in infected poultry, rabbits, and horses. Professional groups such as abattoir workers, butchers, and hunters may also become infected during evisceration and handling of meat (Buzby and Roberts 1997). In a European case-control study (Cook, Gilbert *et al.* 2000), eating raw or undercooked beef, lamb or pork, were significant risk factors. Consumption of other meats (including venison, horse, rabbit, whale and game bird) was also associated with an increased risk.

A case control study of risk factors for *T. gondii* infection in the United States found mussels, clams and oysters as a new risk factor for recent infections. They are filter feeders that can concentrate *T. gondii* infection

from seawater contaminated by oocytes that originate from cat faeces and travel to the coast via river systems (Jones, Dargelas *et al.* 2009). In the USA meat found to be contaminated with *T. gondii* was predominantly pork, lamb and venison. However, no risk factor could be identified for 48% of infections similar to findings in a European multicentre study which failed to identify risk in 14-49% of the cases, depending on the centre.

Birds and Toxoplasma gondii

A recent review by Dubey (Dubey 2010) concluded that the risk of ingestion of *T. gondii* cysts in meat from chickens from commercial indoor farms is low, but that a high prevalence of the parasite is found in backyard and free-range chickens. Edelhofer and Prossinger (Edelhofer and Prossinger 2010) found 36 % of free-range chickens in Austria to be infected with *Toxoplasma*. The risk of toxoplasmosis derived from the consumption of well-cooked poultry meat can be considered to be low, except in situations, such as barbequing or consumption of meat preparations, in which undercooking is more likely (EFSA 2012a). Based on the available limited evidence in Europe, chicken rarely contains viable oocysts. The presence of oocysts depends on age, time spent indoors, farm hygiene and the tissues concerned - non-skeletal-muscle is more frequently infected than skeletal muscle. Data from experimental infection studies suggest that cysts are located mainly in brain and heart tissue of poultry and rarely in muscle (ACMSF 2012). Low numbers of exposed poultry develop clinical symptoms such as encephalitis and neuritis.

The feeding behaviour of birds makes them highly susceptible to infection by oocysts. Exposure will depend on environmental contamination from infected cat faeces. There are currently 9 million pet cats residing in the UK and an estimated 1 million feral cats. In Scotland, specifically, there are 130,000 feral cats (GWCT) in addition to the Scottish Wildcat (*Felis silvestris grampia*) of which there are now thought to be approximately 35-100. They are confined to the Scottish Highlands primarily existing in the far north and west. In a survey of European wildcats in Scotland between 1982 and 1990 Toxoplasmosis was detected in all 42 wildcats tested (McOrist 1992). This suggests that this organism actively circulates in the wild and that wildcat faeces may act as a potential source of infection. The prevalence of *T. gondii* infection in feral cats has been estimated in many areas of the world, for example, 15.8% (n=456) in Seoul, Korea (Lee, Kim *et al.* 2011) 29.8% (n=96) in Prince Edward Island (Stojanovic and Foley 2011) 95.5% (n=180) in Cairo (Al-Kappany, Lappin *et al.* 2011) 22.2% (n=36) in Addis Ababa, Ethiopia (Dubey, Tiao *et al.* 2012) 84.7% (n=59) in Majorca (Millan, Cabezon *et al.* 2009). The difference in prevalence can be attributed to whether the study carried out antibody detection or faecal parasite isolation. It is assumed that feral cat colonies provide a reservoir for *T. gondii* infection and an opportunity for transmission between animals and humans. However, evidence suggests that the main risk factors associated with *T. gondii* seropositivity in wild birds were age and diet with the highest exposure in older animals and in carnivorous wild birds (Cabezon, Garcia-Bocanegra *et al.* 2011).

Survivability characteristics

Data on the survival of *T. gondii* during freezing and cooking is largely from experimentally infected animals. In both cases it is essential for the internal temperature of the meat to reach a critical point to render the parasite non-viable. In the case of freezing it is -12°C (Kotula, Dubey *et al.* 1991) and $+67^{\circ}\text{C}$ for cooking (Dubey, Kotula *et al.* 1990). Although freezing meat at -12°C or below for 24 hrs. will inactivate the cysts retail frozen meat display case temperatures in the USA have been found to be -7.6°C or higher (Hill, Benedetto *et al.* 2006).

Heating to 67°C or higher is considered sufficient to immediately kill tissue cysts (Dubey, Kotula *et al.* 1990). Survival of tissue cysts at lower temperatures depends on the duration of cooking. Some tissue cysts will remain infective if cooking procedures are used in which the meat is heated unevenly, for example microwave cooking (Lunden and Uggla 1992). The primary control factor for prevention of *T. gondii* infection via meat consumption is adequate cooking and prevention of cross-contamination (McCurdy, Takeuchi *et al.* 2006). In a case-control study in Norway, washing kitchen knives infrequently after preparation of raw meat was independently associated with an increased risk of primary infection during pregnancy (Kapperud, Jennum *et al.* 1996). Both tissue cysts and tachyzoites are killed by detergents so it is possible to avoid cross-contamination using good hygiene.

A2.3.7 *Listeria*

Listeria in man

There are several types of *Listeria* spp. but the most pathogenic is *L. monocytogenes* which is responsible for 98% of the human Listeriosis cases identified. The bacterium is ubiquitous in the environment, in soil, silage water and sewage, and can be shed by human and animal carriers. *Listeria* has been found in 47% of household supplies, in particular dishcloths and washing up brushes, and is often found in chilled or delicatessen products such as soft cheeses, pate and ready to eat meals and sausages. The bacterium has also been isolated from a range of raw foods including vegetables and uncooked meats. *L. monocytogenes* is a well-recognised zoonotic agent, but transmission to man is predominantly food-borne and/or associated with food processing rather than from infected animals.

The pathogen poses special problems for food handling and storage as standard refrigeration will not inhibit growth and the cells can survive for long periods in unfavourable conditions. *Listeria* can become established in food production environments surviving in biofilms and causing long-term contamination problems. The initial source of *L. monocytogenes* into the food production plant usually remains obscure (Autio 2004). Dissemination may be due to contaminated environment, workers or raw material (Lunden,

Bjorkroth *et al.* 2005) which in turn may be contaminated through agricultural practices and during handling and transportation (Beuchat 1996); (Oliver, Murinda *et al.* 2005); (Thevenot, Dernburg *et al.* 2006).

L. monocytogenes causes serious illness in humans. Although Listeriosis can occur in otherwise healthy adults and children, the most commonly affected populations include pregnant women, neonates, the elderly, and those persons who are immunosuppressed by medications or illness (EC 1999). *Listeria* infections in immunocompromised individuals most frequently result in meningitis, with or without septicaemia, or septicaemia alone. In pregnant women listeriosis most commonly produces a flu-like illness, characterised by fever, headache and myalgia. The most serious consequences of infections in pregnant women are to the foetus or newborn, resulting in miscarriage, stillbirth, or meningitis. Although the disease can be treated with antimicrobial drugs the use of these agents is not always successful; recurrent infections after appropriate antimicrobial treatment have also been reported. Healthy people can get a milder form of the illness (febrile gastroenteritis) which strikes within one day of eating contaminated food. The main symptoms are fever, diarrhoea, headache and stomach ache.

Because of the long incubation periods (1 to 90 days) shown by some human cases, incriminated food is rarely available from cases of listeriosis. In those instances where it is available, the levels of *L. monocytogenes* detected both from unopened foods and from food remnants obtained from the patients have usually been high ($>10^3$ /g) (EC 1999). In foods, the organism is usually present in relatively low numbers (<100 /g), far below the estimated infectious dose. A minimal infective dose has not been determined in human infection studies. Estimates vary from 10^2 cfu to 10^9 cfu depending on the immunological status of the host (Jemmi and Stephan 2006).

There were 14 reported cases of listeriosis in Scotland in 2011 (HPS 2011) and 147 for England and Wales (Defra 2012); it should be noted that there is under-reporting of listeriosis in Scottish private households. However, although the incidence is low the case fatality rate is reported to be between 20% and 40% (McLauchlin 1990) in the UK.

Birds and Listeria

Birds can carry *Listeria* asymptotically in their intestines; prevalence is likely to be dictated by food and the living environment. Birds most likely become infected by pecking *Listeria*-contaminated soil, faeces or dead animals. Avian listeriosis is far less common than *Listeria* in sheep, goats and cattle. Gulls have been suggested to be responsible for pasture contamination with *Listeria* sp. (Fenlon 1985). Quessy (Quessy and Messier 1992) collected cloacal swabs from ring-billed gulls 9.5% were infected with *Listeria monocytogenes*. Most frequent infection was found in rural areas suggesting it is widespread in farm soil and vegetation. Differences in environmental contamination may explain variation in prevalence in birds.

Survivability characteristics

L. monocytogenes is a psychotropic pathogen and is capable of growth at refrigerator temperatures. The growth range of the bacteria extends from - 0.4°C to +45°C with an optimum temperature of 37°C. At a temperature of 6°C the doubling time for *L. monocytogenes* growth on chicken legs was 19.3 hours (McClure, Beaumont *et al.* 1997).

In general, *L. monocytogenes* appears to be capable of survival on meat regardless of treatments such as freezing, surface dehydration, and simulated spray-chilling (Farber and Peterkin 1991) with poultry supporting growth better than other meat products (Glass and Doyle 1989). The cooking temperature to kill *Listeria* is 70°C for 2 minutes.

A2.4 Number and Species of wild birds

The species of birds considered for this project are small wild game birds (with the exception of quail). However, their size can vary from the small snipe to the larger mallard duck. Table 3 shows the comparative dressed and undressed weights of the different bird species based on information from Yorkshire Game and the RSPB website.

Table 3: Comparative weights of undressed and dressed game birds

Bird Species	Undressed wei	Dressed weight
Mallard	720g-1500g	500g-750g
Widgeon	720g-1500g	270g-330g
Woodpigeon	300-680g	250g-300g
Partridge (RL)	490g	230g-300g
Partridge (grey)	390g	230g-280g
Teal	350g	170g-230g
Woodcock	230g-400g	140g-170g
Quail	100g	100g
Snipe	85g-180g	50g

A2.4.1 Eurasian Woodcock (*Scolopax rusticola*)

The Eurasian woodcock are wading birds of the family Scolopacidae which inhabit damp woodland. They feed nocturnally around streams, pasture fields and on boggy ground preferring dense cover during the day. They are usually solitary birds and can migrate singly. Woodcock forage in soft soil and because they rely on probing into the ground to find food, they are vulnerable to cold winter weather when the ground remains frozen. In freezing weather woodcock may use intertidal mud for feeding. During extreme weather conditions woodcock have been found feeding in urban areas. They mainly eat earthworms, but also insects and their larvae, freshwater molluscs/crustaceans and some plant seeds.

Woodcock are considered to be a truly wild population of birds with no farmed introduction and, due to their solitary nature, little interaction with farmed/introduced birds. Woodcock were rare as breeding birds until the mid-19th century, when extensive planting of pheasant coverts was probably responsible for an increase in numbers (BASC 2013). However, the extent to which they currently interact with other game birds is unknown. The RSPB website states that they're often found in pheasant woods whereas expert opinion (Colin Trotman pers comm) maintains they are only rarely found there. Interaction with farmed game birds could affect their exposure to pathogens and antibiotic residues.

Breeding woodcock are currently most abundant in the north of England and the lower-lying areas of Scotland. In GB there is a resident breeding population estimated at 78,000 pairs and a total over-wintering population that could number 1.5 million (GWCT 2013). The resident woodcock population in the UK is increased by a large over-wintering migrant population from Scandinavia, Finland, the Baltic States and Russia. The bulk of migration coincides with the first full moon of November with a peak population from mid-November to January.

A2.4.2 Snipe (*Gallinago gallinago*)

The snipe is a medium sized wading bird of the family Scolopacidae commonly found throughout the UK as a breeding species with particularly high densities on the northern uplands of England and Scotland. Snipe is the smallest game species. They live in grass cover on wet moorland or marsh and pastures with easy access to soft, boggy ground and small shallow pools. They often nest near farmland with a high proportion of nests and chicks being trampled by livestock. Snipe feed by feeling for their prey deep down in the soil. Microscopic examination of their faeces revealed that earthworms, crane fly larvae and midge larvae were the most important food items of adult snipe, but they also ate small crustaceans, small amphibians and occasionally plant fibres and seeds. Earthworms and crane fly larvae accounted for 85% of the estimated dry weight ingested (GWCT 2013).

Like woodcock, snipe are considered to be a truly wild population of birds with no farmed introduction and little interaction with farmed/introduced birds. The UK population of snipe has undergone moderate declines overall in the past twenty-five years making it an RSPB amber list species. There are approximately 59,000 UK breeding resident snipe and 1 million UK wintering birds arriving from Northern Europe during October-March (RSPB 2013).

A2.4.3 Woodpigeon (*Columba palumbus*)

The woodpigeon of family Columbidae is the UK's largest and most common pigeon. They are found throughout the UK, most frequently in deciduous

woodland, but are also strongly associated with farmland and towns and cities where they frequent parks and gardens. The woodpigeon is the most serious bird pest to the farming industry in the UK and large numbers are shot for both sport and crop protection. The woodpigeon in the UK is largely non-migratory, hence crop damage and shooting occurs all year round. Woodpigeons are a flock bird that eat almost non-stop and because of their large size they generally forage on the ground. Their diet is mainly vegetarian, feeding on flowers, young leaves seeds, grains and berries but they also occasionally eat invertebrates. Woodpigeons are gregarious, often forming large flocks outside the breeding season. Although normally breeding in trees and bushes in woodland, increasing urbanisation has found the wood pigeon breeding in trees in gardens and parklands and in buildings.

The national UK population of wood pigeons has more than doubled over the past 25 years, largely due to the increase in oilseed rape production. There is currently estimated to be a breeding population of 8.2 million in the UK (BTO 2013).

A2.4.4 Duck (mallard, teal, wigeon)

Common teal (*Anas crecca*) are the UK's smallest dabbling ducks. They are thinly distributed as a breeding species in the UK with a preference for the northern moors and are most common as winter visitors. In winter, birds congregate in low-lying wetlands in the south and west of the UK - many of these are continental birds from around the Baltic and Siberia. They are found on both coastal and inland wetlands. Teal are highly gregarious, often forming large flocks. They are aquatic feeders of predominantly invertebrates or seeds. In winter, large numbers occur on lakes, ponds, marshes and to a lesser extent on coasts, estuaries and mudflats. In spring and summer their diet consists predominantly of animal matter such as molluscs, worms, insects and crustaceans whilst during winter it is mainly seeds, grasses and sedges and agricultural grain. Teal are a wild species and so numbers are unpredictable and governed by the weather.

There are 1,600-2,800 breeding pairs of teals annually in the UK and 210,000 migrant birds present from October to March (RSPB 2013).

The widgeon (*Anas Penelope*) is a medium sized duck which breeds in central and northern Scotland and in northern England. There are a large number of wintering birds from Iceland, Scandinavia and Russia found on the UK coast especially on the eastern side. It is a bird of open wetlands such as wet grasslands or marshes and in winter is found in estuaries, lakes and flooded grassland. They are highly gregarious and will form large flocks. The widgeon usually feeds by dabbling for plant food or grazing. The diet is vegetarian predominantly leaves, stems roots and seeds with insects also taken in the summer (birdweb 2013). The wigeon is similar to teal in that it is a wild species with an unpredictable supply.

There are 300-500 pairs of widgeons breeding annually, mostly in Scotland, and 440,000 birds wintering between October to March (RSPB 2013).

The mallard (*Anas platyrhynchos*) is a large heavy duck. It breeds throughout the UK in summer and winter in wetland habitats although it is scarcer in upland areas. Mallards in the UK may be resident breeders or migrants, many of the birds that breed in Iceland and northern Europe spend the winter here. Some mallard populations have habitats in the wilderness, while others may have more urban habitats. The diet is omnivorous consisting of seeds, stems and roots, insect larvae and other aquatic invertebrates such as insects, molluscs, worms, crustaceans and occasionally amphibians and fish especially in spring and summer.

Scotland lies on the main west-European flyway for migratory species of ducks with the main duck species on the UK shooting list being widgeon, teal and mallard. While all of Scotland carries these birds at some time during the year, the east coast is the principal area for migratory waterfowl, providing estuary feeding and shelter for large numbers from late September to March/April when birds return to their Scandinavian and Russian nesting grounds. The natural duck numbers are supplemented by reared mallard on many estates which are managed and fed on flight pond systems. Wild birds are also attracted by this artificial feeding taking advantage of the readily available food. There are about 50,400-127,100 breeding pairs of mallards in the UK and 680,000 overwintering migrant birds between October and March. The mallard population is boosted by the addition of farmed introduced birds released for shooting; in 2011 the number of released mallards in Scotland was 63,780 (AHVLA Poultry Register, unpublished data).

A2.4.5 Partridge: grey (*Perdix perdix*) and red-legged (*Alectoris rufa*)

The grey partridge of family Phasianidae is a British ground-nesting bird which inhabits grassland, arable farmland, hedgerows and field margins. New farming practices in the 1950s saw a dramatic decline in the native grey partridge. Partridge hens nest in thick grassy cover at the base of a field boundary. RSPB data show that they are present over most of England, south and east Scotland and much of the Republic of Ireland. The grey partridge is not shot in significant numbers and most activity is aimed at its conservation, though some greys are released. They are on the RSPB red list of species needing action to address population decline. The red-legged partridge, also of the family Phasianidae, is larger than the grey partridge and not under threat. The bird was introduced from Europe in the 18th century as a sporting quarry species. Red-legged partridges are native to France, Spain and Portugal and prefer dry habitats and hot summers which limit their natural breeding in the UK. The red-legged partridge is common throughout Scotland and is reared on many shoots. Its habitat is similar to the grey partridge i.e. chiefly open farmland. The diet of the grey partridge consists of leaves, seeds and insects whilst that of the red-legged partridge is predominantly seeds and roots.

Figures from the RSPB suggest that there are between 70-75,000 breeding pairs of grey partridge in the wild whilst the BASC estimate 145,000. The grey partridge is found in fewer numbers than the red-legged, numbers for which are estimated at 90,000- 250,000 pairs. Numbers are, however, boosted by reared and released birds, specifically for the hunting season. In the case of red-legged partridge around 90% are imported, the majority coming from France, though Spain and Poland also supply. They come in as fertile eggs or as day-olds in roughly a 50:50 ratio. The Game Farmers Association (GFA) put the figure for birds reared for release at 20-30 million, of which 16-17% are red-leg partridge and a few per cent are grey partridge. The number of partridge released for shooting in Scotland in 2011 was 509,285 (AHVLA Poultry Register, unpublished data).

A2.4.6 Farmed Game birds

Farmed game birds refer only to quail in this report. There is evidence of farmed pheasant and partridge (ADAS 2005) i.e. birds reared for meat in a similar way to broiler chicken production. Farmed 'wild' gamebirds are bred on farms in the UK or are imported as eggs or day-old chicks. They can be reared intensively on the farm or more extensively on the shoot before release for shooting when the season begins. After release there may still be some supply of feed or water. Increasingly more intensive methods are being used for both breeding and rearing with high stock densities at certain times of the season. Some gamebird farmers use commercial poultry systems to breed and rear larger numbers of birds (Council 2008). There is little official surveillance or monitoring of farmed gamebird premises. Whilst gamebird rearing and breeding has traditional roots the increased interest in this industry has attracted new enterprises and the introduction of larger breeding and rearing sites.

The release of farmed 'wild' game birds prior to the shooting season has the potential to introduce new parasites and disease into the wild population, moving pathogens to new areas and putting other native birds at risk. The prevalence of *E. coli* was found to be significantly higher in farm-reared red-legged partridges in Spain (Diaz-Sanchez, Mateo Moriones *et al.* 2012) whilst prevalence of *Campylobacter* did not differ significantly between wild or farm-reared groups. The artificial environment and intensive management of game birds in farms has been shown to increase the risk of infection by parasites (Villanua, Perez-Rodriguez *et al.* 2008) whilst the stress during transport to the release pens and the change in diet could potentially alter the digestive tract flora and encourage proliferation of pathogenic bacteria within the avian intestine.

A2.4.7 Quail (*Coturnix coturnix*)

The wild quail of the family Phasianidae is now protected in the UK and hunting has been banned since 1937. The native quail is the UK's only

migrant game bird, reaching the northern fringes of its breeding range here. They arrive in late April to May and remain until late summer. They are, therefore, rarely found in the UK during the shooting season as they have usually already migrated to a warmer climate.

All quail in the UK are farm produced and should be regulated as such. Farmed quail meat is available to purchase from supermarkets, butchers and mail-order companies. The main farmed breed is the Japanese quail (*Coturnix japonica*), bred as a small gamebird for meat. Producers in the UK tend to be hobby breeders and small producers. Between Jan 1st 2011 and Dec 31st 2011 there were 864,237 quail slaughtered in 11 slaughterhouses in England, Scotland and Wales (FSA, unpublished data). The main commercial company operating in this sector is Tom Barron Ltd, producing approx. 500,000 birds per annum under the Fayre Game label. The UK consumption of quail is around 10,000-12,000 birds/week which is met mainly from home production of about 10,000 birds/week with additional imports of 1,500 birds/week usually from France (SAC).

Quail can be bought live from quail farms available from 3 days of age to 'table ready' 8 weeks of age. These quail for meat can be sold live and unprocessed so could potentially be eaten uneviscerated.

A2.5 Shooting

The establishment of the quality and biological safety of the end product of shooting begins with the hunter at the point of kill. Inspection is an on-going process beginning before the bird is shot and continuing throughout handling (and dressing) until the carcass is consumed. Hunters should first be aware of any aspect of the bird's behaviour that might indicate abnormalities such as isolated individuals of normally flocking birds or inability to fly or unusual flight pattern. Aspects of appearance such as light body weight or physical damage not caused by hunting may also indicate abnormality. Once shot the hunter will usually inspect and remove damaged, abnormal or contaminated birds. If the bird is to be supplied to an AGHE for sale one member of the shooting party is required to hold a recognised hunting licence. This will confirm him/her as a 'trained person' i.e. someone who has sufficient knowledge of the pathology of wild game, and of the production and handling of wild game meat after hunting, to undertake an initial examination of wild game on the spot (FSA 2011). Alternately if the bird is for private consumption 'safe' meat will obviously be desired.

The main shooting period, with the exception of woodpigeon, commences in September and ends in February between reproduction periods. These months also correspond to the cooler periods of the year which will facilitate preservation of the carcasses and reduce risk of contamination.

There are several different types of shooting depending on the quarry and formality of the shooting environment. Driven shooting is usually highly organised where a group of shooters stand at given points and wait for game

to fly up, flushed out by a team of beaters and dogs towards them. The birds are mostly farmed and taken to release enclosures when they are in the early stages of maturity. Although the majority of the quarry will be farmed shooting of other wild birds will also occur where the opportunity arises. Alternately, walked up shooting is an informal style of shooting where the game is flushed ahead of the guns as they walk over the shooting ground. The type of quarry shot usually depends on the location and species availability.

Wildfowling is specifically the shooting of ducks, geese and wading birds including snipe and woodcock. Coastal wildfowling takes place on tidal sites where the birds are rarely farmed and are likely to be migratory. Times of hunting wildfowl usually take advantage of the morning and evening flights between feeding and roosting grounds. This form of shooting has the smallest number of participants nationally most of whom are usually highly experienced. Inland wildfowling occurs on inland sites where there are often natural or artificial flight ponds frequented by birds for roosting at night. These ducks are usually specifically bred in game farms for recreational shooting although wild ducks can take advantage of the food supplied on artificial ponds.

Different pellet size is used depending on the size of gamebird concerned. The main criterion is to place enough pellets in the pattern with enough striking energy to kill the chosen quarry humanely. The use of multiple shot pellets makes the wounding pattern highly variable. Bigger pellets contain more energy and can therefore kill from a greater distance. They also penetrate birds with thicker down. Smaller pellets are used more often for smaller game birds. All those involved in shooting wild game, either for their own consumption or for sale, will try to ensure a clean kill in order to avoid damage to the meat. A low end estimate of 3,500,000 birds shot each season are not being sold through game dealers, possibly as a result of heavy shot damage or other 'spoiling' of the carcass such as from dog bites. Pain et al. identify those involved in the shooting industry as more likely to consume these birds that would be most likely rejected by game dealers (FSAS 2012b).

Some AGHEs suggest that 15 - >80% of gamebirds have their intestines perforated by pellet but no obvious leakage as the guts still remain 'sealed'. Opinion states that it is very rare to see contamination of birds by gut leakage as the pellet is so small and when it hits the bird it has very low velocity and just remains within the meat. Leakage and bad perforation would only occur if the bird was shot from behind. Most birds are shot in the head and breast as they are driven birds and flying towards the shooter. Woodcock and snipe are shot, in particular, as a going away or crossing target and consequently the abdomen and intestines are likely to be better protected from shot penetration (Colin Sheddon pers. comm.). The presence of shot in the body cavity has proven to be an inaccurate indicator of perforated intestines and the presence of faecal material in the body cavity (Paulsen, Nagy *et al.* 2008). Faecal material was found in 10.9% of the shot

birds, whether shot in the abdominal area or not, compared to 0% in a control slaughtered group.

In a study on red-legged partridges, woodpigeons, woodcock and mallards purchased from supermarkets, game dealers/butchers and directly from shoots the mean number of whole pellets or large parts of pellets was 2.17 pellets per bird; 35% of birds contained no whole pellets or large fragments. There was significant positive correlation between the mean number of shot detected per bird and the mean body weight of the species (Pain, Cromie *et al.* 2010). The study illustrated that lead gunshot undergoes fragmentation on impact with the bird and that lead fragments cause contamination of their meat. They found the presence of variable numbers of tiny fragments observed on X-ray of the carcasses including the majority of those in which all gunshot had passed through the body.

Shot perforation of the gut can lead to microbial contamination of muscle tissue that would otherwise remain sterile, as well as leakage of gut contents into the body cavity. Accidental penetration by dog bites could also present as a risk for contamination, but the risk of this occurring is considered to be relatively low. There appears to be various degrees of gut perforation, from serious damage where visible leakage can be observed to minor perforation that is probably unlikely to be identified by visual inspection during dressing. Contaminated muscle tissue has been observed in birds with 'minor' gut perforation where the concentration of pathogen in the muscle tissue was estimated to be at least 100 cfu/g (Geoff Mead pers. comm.). It is likely that muscle would be contaminated with higher concentrations in the birds with serious gut damage. The combination of muscle contamination and undercooking could therefore lead to foodborne illness especially with *Campylobacter* which has a relatively low infective dose.

When retrieving quarry on shoots there are specific guidelines for the use of dogs to 'pick-up'. The BASC code of practice for quarry retrieval specifies that young or inexperienced dogs should not be taken on shoots without permission. They should be trained, under control and responsive to the owners instructions at all times and that should deliver game readily to hand without damage. A hard mouthed dog (one which damages game) should not be working in the shooting field. Only fully experienced gun dogs are used to retrieve wounded birds or 'runners'. The term 'soft mouth' refers to a behavioural tendency to pick up, hold and carry quarry gently. A 'hard mouthed' dog may not puncture a bird but can inflict crushing damage which can be felt by an experienced person as they examine the bird. The use of a well-trained 'soft mouthed' dog for quarry retrieval will therefore minimise the risk of accidental puncture marks and transfer of the dogs oral flora to the bird.

A2.6 Number of Birds shot

Estimates of number of game birds shot vary as there is no compulsory scheme collecting national statistics on game bag numbers. Data should therefore be treated with caution.

A2.6.1 Woodcock

- The shooting season for Woodcock is from October 1st to January 31st in England and Wales and September 1st to January 31st in Scotland. Woodcock, however, tend not to be available in sufficiently high numbers until early December although they are never abundant and always much sought after.
- They are hard to find and difficult to shoot, often making up part of a mixed bag of birds with partridges and pheasants. Swooping erratic flights make the woodcock a prized quarry.
- Woodcock bag counts have been published as: 100,000 -150,000 (Consultants 1997), 150,000 (International 2013) and 250,000 (combined with snipe (PACEC 2006)).

A2.6.2 Snipe

- The shooting season for snipe is from August 12th to January 31st.
- The snipe provides an interesting sporting target due to its erratic flight and small size.
- In Scotland snipe are usually shot as part of a mixed shoot and can be either driven from boggy areas or walked up.
- Estimates for the number of snipe shot annually have been 85,000 (Jackson 2004), 25,000 (Andrew Hoodless pers. comm. quoted in (Henderson 1993)) and 30,000 (Consultants 1997).

A2.6.3 Woodpigeon

- There is no official shooting season for the woodpigeon and it is legal to shoot the bird all year around. Pigeon shooting does, however, peak in the summer when crops are most abundant.
- The woodpigeon is one of the most popular species providing sporting shooting and often making up part of a mixed bag. Shooting is authorised for specific purposes such as: preventing serious damage to crops, vegetables, fruit and foodstuffs for livestock and for the purpose of preserving public health or public safety. More than 200,000 people hunt woodpigeon in the UK every year.
- The CRC estimated that between 5 and 7 million woodpigeons are shot each year (Consultants 1997). However, the actual number shot is difficult to estimate as kills are not always entered into gamebag records (Tapper 1992).
- In 2004, 3,600,000 pigeons were shot to protect crops of which 90% were consumed; the remainder were likely to be unfit for human consumption (PACEC 2006).

A2.6.4 Ducks

- The shooting season for ducks is September 1st to January 31st for inland shooting and September 1st to February 20th for coastal wildfowling.
- The annual numbers of duck shot has been estimated at 1 to 1.5 million (Consultants 1997) and 970,000 (PACEC 2006). This, however, is the number of total ducks shot including farmed mallards and all combined species.
- Consultation with AGHEs suggest that mallard is by far the most common species of wild duck shot in UK and that teal and widgeon each make up a maximum of 5% of total ducks shot.

A2.6.5 Partridge

- The shooting season for both types of partridge is from September 1st to February 1st. Most driven partridge shooting ends by the start of December though some birds will be shot throughout the season as part of a pheasant drive. Partridge are often introduced to pheasant shoots to add variety to the shooting and to extend the season.
- The bulk of red-legged partridges shot now derive from released stock. Since 1990 the numbers released per unit area have increased four-fold. The increase in released birds has meant the bag has also quadrupled over the last fifteen years. It is estimated that 6.5 million partridges (total) were released across the UK in 2004, and 2.6 million were shot (PACEC 2006).
- Grey partridge shooting only takes place if there is a sustainable surplus. It is not significant in shooting terms although some are released. Game bag records have shown that in the 40 years after the Second World War the numbers of grey partridges shot declined by 80% from a peak of 2 million. This was largely due to field area expansion, decline in the gamekeeping industry and a reduction in chick survival rate (GWCT 2013).

A2.7 Numbers of birds sold

The number of wild game birds sold is difficult to quantify and predicted values are highly variable. Small shoots will generally distribute all the birds among shoot members and give some away to friends or landowners. Larger shoots may sell their surplus birds to a game dealer or directly to local people. Excess birds shot may be sold to local butchers, game dealers and catering establishments. Selling game in-feather to a dealer has become an attractive option for shoots; it is quick, easy and convenient, and will keep costs to a minimum. Reputable dealers will take all the game which is available and, if he has advance notice of shoot dates, he may be able to collect at the end of each day, avoiding any need for the shoot to store

birds. A recent development involves continental game dealers doing the rounds of UK shoots, buying up large numbers of birds to take back across the Channel reportedly in chilled lorries.

The PACEC report estimated that 44% of birds were sold to game dealers whilst the remainder were consumed by shoot providers or taken for eating by shooters and their families, that is, for private and domestic consumption (PACEC 2006). A small percentage of birds may not pass as fit for human consumption. Of this 44% it is estimated that approximately 20% of small game birds pass through AGHE and 24% through FBOs in Scotland (FSAS 2012a). The remaining 56% may also involve local or mail/internet orders under regulatory exemptions and game that is killed and traded illegally. Almost all interviewees participating in the FSAS report agreed that the trade in wild game outside approved channels in Scotland is increasing. They concluded that the total volume of wild game entering the food chain is unclear, the trade through food hygiene regulation exemptions is unquantifiable and there is no accurate measurement of poached game. However, the Game-to-Eat campaign estimates that three quarters of game shot in GB is sold via game dealers while 14% is given away mostly to guns, beaters and other shoot helpers. 12% is sold by shoots direct to public or to local retail outlets (Alliance 2008).

Individual numbers of birds species sold vary considerably. Whilst approximately 37% of total partridge numbers, 45% of shot ducks and 55% of shot woodpigeons per provider are sold (PACEC 2006) only 11% woodcock (IUCN 2003) and 4% of snipe are sold (Consultants 1997). The proportion of birds fully traceable back to shoots has increased by a third, over half of fulltime keepered shoots now guarantee traceability on all batches.

A2.8 Background pathogen level in population

Information regarding the normal gastrointestinal bacterial flora is limited for the majority of wild bird species. Studies that have been carried out are restricted by small sample sizes and the constraints of using selective techniques to isolate specific pathogens. Whilst studies may give some indication of the frequency with which birds die from different infections they provide little or no information on the prevalence of the pathogens in apparently healthy individuals. Other considerations include: Data may not be from GB, small sample size and no information on the concentration of pathogens in gamebird populations.

Avian feeding ecology appears to be a key determinant of enterobacterial acquisition and correlations have been made between bacterial pathogens in the avian gut and those found in their foraging grounds, (Benskin, Wilson et al. 2009). Waterfowl which feed solely on vegetable matter appear to have low enteropathogen prevalence, while these bacteria are frequently found in waterfowl that feed on animals or strain mud to obtain nutrition (Luechtefeld, Blaser *et al.* 1980). Ground-foraging bird species eating filter-feeding molluscs living in sewage-contaminated habitats (Benskin, Wilson et

al. 2009), shoreline-foraging birds feeding on invertebrates (Waldenstrom, Broman et al. 2002) and wading birds feeding on bivalve molluscs (Fricker and Metcalfe 1984), have all been shown to have high carriage rates of *Campylobacter* species probably from ingestion of contaminated food. Bacteria can also be picked up by birds feeding from rubbish tips and foraging on pasture following the application of farm slurries and sewage sludge to land.

It has proved difficult to find published evidence on the number and prevalence of certain pathogens in some bird populations in particular woodcock and snipe. Expert opinion (Colin Trotman; Alan Pearson and Richard Byas) suggests that as these birds are truly wild populations the occurrence of pathogens is rare. Where data are lacking from both published data and expert opinion incidence in birds from the same family or feeding guild has been substituted where available. The scarcity of information in some instances must be taken into consideration when estimating the associated risks.

A2.8.1 Salmonella

In 2011 there were a total of 12 reports of *Salmonella* from laboratory submissions in game birds of which seven incidents were in partridges and the remainder were in pheasants (AHVLA 2011). Of these two were *S. Typhimurium*, one the monophasic variant 4,5,12:i:-, and one was *S. Enteritidis*. The AHVLA report also states there were 27 *Salmonella* incidents in ducks in 2011, although the species are not identified and are likely farmed ducks as 22 of the incidents arose from samples collected on farm and the remaining 5 from hatcheries. Sojka and Wray studied the incidence of *Salmonella* infection in animals in England and Wales, 1968-73. Of 1255 incidents involving 'poultry', 24 (1.9%) involved pigeon and 6 (0.5%) involved partridge (Sojka, Wray *et al.* 1975). Between 1986 and 1988 only 1% of red-legged partridges surveyed (n=702) and 0% of grey partridge (n=98) had salmonellosis (Beer 1989).

The majority of *Salmonella* infections in ducks appear to be asymptomatic although severe clinical disease has been observed in young animals. Of 449 free-living waterfowl sampled in central Ohio, one was positive for *Salmonella* (*S. Java*), a prevalence of 0.2% (Fallacara, Monahan *et al.* 2001). In a study of faecal shedding rate of *Salmonella* in free-living wild ducks, examination of 477 duck droppings during the winters of 1968/69 and 1969/70 gave isolation rates of just over 4%. The commonest serotype was *S. Typhimurium*. Teal had a 3.4% incidence (n=88) and widgeon had 0% (n=12). Prevalence probably increases when birds exist in close proximity, such as waterways where waterfowl congregate (Mitchell and Ridgwell 1971).

S. Typhimurium isolates from pigeons differ biochemically and antigenically from other isolates likely indicating host adaptation. *Salmonella* in pigeons appear to be due to host adapted subtype of *Typhimurium* and therefore

likely only pose a limited risk to humans. No zoonotic transmission from pigeons to humans has been documented in literature (Hoelzer, Switt *et al.* 2011). A Japanese study looked at the rectal contents and fresh droppings from 329 apparently healthy pigeons in central Japan, and identified *Salmonella* from only two (0.6%) specimens. The isolates were identified as *Salmonella* Typhimurium subserovar *copenhagen* (Kinjo *et al.* 1983). From January 1992 to the end of December 1993, *S.* Typhimurium was recovered from the viscera of 7 (3.3%) out of 209 pigeon carcasses and from 14 (4.5%) of 314 pigeon faeces samples, at a Scottish diagnostic laboratory (Pennycott 1994). Pigeon specific *Salmonellas* were found in only 4 (0.3%) of 1268 strains of *S.* Typhimurium isolated from humans in northern Germany, indicating that pigeon-specific *Salmonella* strains had only a small significance in the salmonellosis of humans (Wuthe & Wuthe 1980). In 2009 there were a total of 13 reports of *Salmonella* in pigeons 11 of which were *S.* Typhimurium. The other serovars reported were single incidents of *S.* Kedougou which was last reported from pigeons in 1994 and *Salmonella* 4,5,12:i:- (DT191a from an exotic pigeon at a zoo) which has never previously been reported from pigeons and which has been associated with 'feeder' mice and human outbreaks in UK and USA.

Wild birds may acquire *Salmonellae* after exposure to human-contaminated environments, or after scavenging on refuse tips and sewage sludge; birds that live away from such environments are unlikely to harbour *Salmonella* (Murray 2000), (Tizard 2004).

Studies on the incidence of *Salmonella* in snipe and woodcock are scarce. No *Salmonella* was isolated (Kobayashi, Kanazaki *et al.* 2007) from the one cloacal swab taken in a study from Tokyo Bay whilst one (n=28) woodcock was found positive for *Salmonella* Typhimurium in France (SAGIR data, ONCFS/FNC/FDC network)

A2.8.2 *Campylobacter*

A previous study on hazards in wild game birds (Coburn 2003) identified a high prevalence of *C. jejuni* in wild ducks (22%-67%), the range possibly reflecting regional variation in the habitats occupied by the birds. Fernandez *et al* (Fernandez, Gesche *et al.* 1996) suggested that waterfowl are natural reservoirs of *Campylobacter* that could play a role in the waterborne spread of the bacteria. Levels are usually lower among ducks feeding largely on vegetation compared with those straining the sediment of ponds (Fallacara, Monahan *et al.* 2001). However, the contribution of *Campylobacter* carriage in wild ducks to human infection is unknown.

A lower carriage rate in wild duck compared to farmed ducks has been demonstrated. However, studies on *Campylobacter* in wild duck faecal droppings are more likely to be influenced by extremes of temperature, moisture and ultra-violet light levels (Obiri-Danso and Jones 1999). In a comparison of farmed and wild mallard ducks the carriage rate of *Campylobacter* in wild ducks was 9.2% - 52.2% compared to 93.3% - 100% in

farmed ducks 2011 (Colles, Ali *et al.* 2011). *C. jejuni* was predominant (74.6%) among isolates from farmed ducks whilst *C. coli* was predominant from wild ducks (85.7%). Interestingly, 92.4% of *Campylobacter* isolates from farmed ducks were identified as sequence types commonly associated with human disease and farm animal sources. In contrast, only one similar isolate was found from wild ducks. They concluded that there is evidence of strong host association among *Campylobacter* genotypes.

Evidence of *Campylobacter* carriage in teal and widgeon is limited and contrasting. Hughes (Hughes, Bennett *et al.* 2009) found 1 out of 43 faecal samples from wigeons to be positive for *C. lari* whilst Gargiulo (Gargiulo, Sensale *et al.* 2011) found *C. jejuni* in 57% (n=70) and *C. coli* in 19% (n=70) of cloacal swabs from common teal. In an American study on migratory wildfowl the incidence of *C. jejuni* in cecal contents was: Mallard 34%, American widgeon 42% and green winged teal 16% (Luechtefeld, Blaser *et al.* 1980). The variation in isolation rates could be related to the dietary variation of the ducks and the sample origin i.e. cloacal, faecal or cecal.

Information on *Campylobacter* prevalence in woodpigeons is scarce. The one study on wood pigeons found a *C. jejuni* carriage rate of 36% (n=25) (de Boer, Seldam *et al.* 1983). Most research on *Campylobacter* carriage in pigeons has been done on the feral pigeon. Studies on carriage rates in the feral pigeon (Kinjo, Morishige *et al.* 1983) (Fenlon 1985); (Itoh, Saito *et al.* 1982) suggest that *C. jejuni* occurs commonly in the intestinal flora of pigeons with rates of 12.5%-54%. Pigeons probably constitute a natural reservoir of *C. jejuni*, however, it is not known whether the types of organism identified were pathogenic to humans. More recently, in a survey of feral pigeons in Madrid the prevalence of *C. jejuni* ranged from 35.7% to 86.4%. There were no clinical signs of disease so the birds were considered to be an asymptomatic reservoir (Vazquez, Esperon *et al.* 2010). The wood pigeon has become a more frequent visitor of urban landscapes over the past 30 years and its association with human activities has consequently increased.

Prevalence of *Campylobacter* in partridges has only been carried out for farmed game (de Boer, Seldam *et al.* 1983). 0% (n=8) of unspecified partridges had *C. jejuni* whereas commercially reared grey partridges had a *C. coli* carriage rate of 49.2% (n=240) and *C. jejuni* rate of 12.7% for cloacal swabs (Dipineto, Gargiulo *et al.* 2009). A study on the prevalence of *Campylobacter* in the intestinal flora of red-legged partridges from farm-reared, restocked and natural populations in Spain found a prevalence of *Campylobacter* of 23% (n=444) in all 3 groups; unfortunately there was no investigation into strains (Diaz-Sanchez, Mateo Moriones *et al.* 2012).

Data on *Campylobacter* in snipe and woodcock is scarce. Due to their wild migratory lifestyle very few scientific studies have been carried out on the prevalence of disease and they are considered by expert opinion to be 'healthier' than other game birds with less incidence of disease. The one snipe tested by Workman (Workman, Mathison *et al.* 2005) in Barbados was found to be positive for *C. jejuni*, however, the strain was genotypically

distinct from clinical strains and therefore not a likely source of human infection. 1 snipe and 1 woodcock were found positive for *Campylobacter* spp. in Sweden (Waldenstrom, Broman et al. 2002). In this study they were classed as shoreline foraging invertebrate feeders assessed as those that frequently feed at water edges or in shallow waters of habitats that commonly harbour *Campylobacter* spp. e.g. river mouths, seashores and sewage plants.

As quail are commercially reared poultry *Campylobacter* prevalence could be expected to be higher than in wild populations. Documented prevalence ranged from 14% to 41% in 3 flocks of farmed quail (McCrea, Tonooka et al. 2006). Experimental colonization of poultry flocks can reach levels of 10^9 colony-forming units (cfus) per gram of caecal contents (Wassenaar, van der Zeijst et al. 1993), although this is likely lower in the natural environment. The company 'Fayre Game' does not test their live or slaughtered quail for *Campylobacter* (ADAS 2007).

In summary although the prevalence of *Campylobacter* is high in game birds the relevance of these asymptomatic avian infections as regards potential human pathogens is largely unknown. Evidence suggests that the majority of *Campylobacter* strains found in game birds are not of the same subtypes as those found in human infections although where there is interaction between birds and humans e.g. farmed partridges and quail the subtypes may be similar.

A2.8.3 *E. coli* (Toxigenic)

Wild birds have the potential to act as vectors for VTEC, acquiring the bacteria as a result of feeding from rubbish dumps, sewage outlets and fertilised pastureland. VTEC strains have, however, only rarely been detected in wild birds with most studies showing a low frequency of VTEC positive birds. For example, one study isolated *E. coli* O157:H7 from only 0.34% (n=296) of wild bird (species unknown) faecal samples (Rice, Hancock et al. 2003). Significantly, a study in Gloucestershire, UK, showed that Rook *Corvus frugilegus* faeces were the source of *E. coli* O157 infection in two children and their mother; the father who worked as a forester had trapped and handled the birds and was the source of infection to his family (Ejidokun, Walsh et al. 2006).

A higher prevalence of VTEC has been identified in predominantly urban pigeon populations (Farooq, Hussain et al. 2009); (Grossmann, Weniger et al. 2005); (Kobayashi, Kanazaki et al. 2009). Of 160 pigeons sampled from the centre of Rome in 1997, 12.5% were found to be shedding VTEC. However, the isolates obtained were not known to be pathogenic to humans (Dell'Omo et al. 1998). In Italy, STEC was isolated from 10.8% (n=649) of pigeons sampled (Morabito, Dell'Omo et al. 2001), whilst in Norway, 0/50 isolated were VTEC (Cizek, Alexa et al. 1999). In surveys of VTEC O157:H7 in pigeons a carriage rate of 0.8% (n=504) was found in Napoli (Santaniello, Gargiulo et al. 2007), whilst In Japan, all 108 pigeon faecal samples

examined were negative for this strain (Tanaka, Miyazawa *et al.* 2005) . There is evidence of a child with diarrhoea and domestic pigeons in Germany having the same VTEC serotype O128:H2 (Sonntag, Zenner *et al.* 2005) although an American study indicated that pigeons may not be a major route of transmission of VTEC (Pedersen, Clark *et al.* 2006).

No data could be identified for VTEC in wild duck, however expert opinion suggests that its prevalence in wild waterfowl is likely to be very low due to their extensive habitat. Wild ducks that have contact with farm environments may have an increased risk of carrying VTEC on their exterior.

The French SAGIR network has reported 22 woodcock (n=216) as positive for *E.coli*, however no strain typing was undertaken. *E. coli* O157 was isolated from the faeces of 2.9% of shore birds (mainly gulls) on intertidal sediments may imply that snipe and woodcock could be exposed to the same environmental sources of VTEC (Wallace, Cheasty *et al.* 1997).

No data could be found concerning VTEC in partridges and quail. Occurrences of APEC were documented for both farmed bird species (Roy, Purushothaman *et al.* 2006).

VTEC is possible in game birds but the small amount of evidence obtained suggests that prevalence is likely to be low and that the identified serotypes are not usually implicated in human disease.

A2.8.4 *E. coli* (Antibiotic resistant)

Antimicrobial drug resistance is relatively commonplace in poultry, but has also been described in bacteria isolated from wild birds (Cole, Drum *et al.* 2005); (Middleton and Ambrose 2005) ; (Sjolund, Bonnedahl *et al.* 2008). Arctic birds are known to contain multi-drug-resistant bacteria, indicating that migration behaviour may be responsible for the introduction and transfer of drug-resistant bacteria to geographically remote areas (Sjolund, Bonnedahl *et al.* 2008). Although wild animals do not naturally come into contact with antibiotics, they can become infected with resistant bacteria disseminated by wild birds, and act as reservoirs and vectors of resistant bacterial pathogens, encouraging new health problems in wildlife populations to emerge, as well as novel reservoirs of zoonotic disease to form

83% of shoots in the UK rely on hand reared game released to supplement wild stocks and are therefore potentially exposed to the use of antibiotics. The Game Farmers Association (GFA 2013) put the figure for birds reared for release at 20-30 million, of which the majority (80%) are pheasants and most of the rest (16-17%) are red-leg partridge. The final few per cent are grey partridge and ducks. Quoting the GWCT as the source, the British Trust for Ornithology (BTO 2013) suggest that 20-22 million pheasants are released each summer, with more than 2 million surviving until spring. Thus, there is a wide range of estimates of the size of the industry and consequently the

number of birds which may be directly exposed to antibiotics. The use of commercially reared game is likely to result in an increase in exposure of these released gamebirds to antibiotics as opposed to true 'wild' game birds.

A2.8.5 *Chlamydophila psittaci*

Free-living wild birds are important as reservoirs of *C. psittaci* (Brand 1989). Both diseased birds and sub-clinically infected birds can shed chlamydia and are therefore a potential threat to both human and animal health (Brand 1989);(Franson and Pearson 1995);(Roberts and Grimes 1978). Evidence of exposure to chlamydia is most frequently reported in Charadriiformes, Passeriformes, and Anseriformes (Brand 1989);(Franson and Pearson 1995). However, disease development is more likely in Columbiformes e.g. pigeons (Kaleta and Taday 2003).

In a survey between 1974 and 1986, bird serum samples (submitted to aid diagnosis of a pre-existing condition, or for export certification) were tested at AHVLA for *C. psittaci* using either complement fixation test or direct culture. Forty seven per cent (n=1549) of pigeons, 23.3% (n=43) of wild ducks and 29% (n=62) of British game birds were found to be positive using the complement fixation test (Bracewell and Bevan 1986). For direct culture data from wood pigeons were linked with that from collared doves and resulted in 25% (n=52) positive samples whilst 23.5% (n=17) of British game birds were positive. They also showed isolation rates from different organs with *C. psittaci* cultured from 21 out of 103 samples of intestines. Spleen, trachea, lung, heart and liver also all gave positive results of between, 15 and 17% (Bevan and Bracewell 1986).

A high prevalence of *C. psittaci* exists within game birds, with wild pigeons having the highest prevalence (figures ranging from 30-90%). Kaleta and Taday (2003), looking at the number of bird species per order compared to number of bird species positive for chlamydia for Charadriiformes found 9% (n=194) to be positive compared to 21% (n= 157) for anatiformes and 5% (n=259) for phasianiformes (quails partridges and pheasants). Chlamydia found in 1 woodcock (SAGIR pers. comm.)

In a survey of feral pigeons in Madrid they concluded that *Chlamydia* was highly prevalent and that infected pigeons did not show signs of clinical disease thereby forming an asymptomatic reservoir. Prevalence ranged from 37% to 59.7% over 3 sampling periods (Vasquez, Esperon *et al.* 2010). However, the majority of data available are from studies on the feral pigeon used in this report as a substitute for the woodpigeon due to lack of data in this particular species. In a study of *C. psittaci* positive PCR tests on wild birds admitted to an RSPCA wildlife centre 1/25 woodpigeons and 3/8 feral pigeons were positive. An explanation for the high prevalence in feral pigeons was given as their propensity to live in colonies facilitating the transmission of infections between individuals and the high competition for food amongst these birds; they are commonly presented at the wildlife

hospital in a malnourished state which is known to contribute to susceptibility to infection (Sharples and Baines 2009).

A2.8.6 *Toxoplasma gondii*

Toxoplasma has been isolated from wild avian species belonging to several families in Spain (Cabazon, Garcia-Bocanegra et al. 2011) with the highest exposure found in carnivorous wild birds. Ground feeding birds are highly susceptible to oral infection with oocysts. Although subclinical *T. gondii* infections are prevalent in many avian species toxoplasmosis can be clinically severe in pigeons. A seroprevalence of 2.6% (n=1507) (Waap, Cardoso et al. 2012) and 11.86% (Cong, Huang et al. 2012) has been found from pigeons in Lisbon and China respectively. The distribution of *T. gondii* cysts in avian tissues was examined in 28 specimens of rooks (*Corvus frugilegus*). The presence of cysts was demonstrated mainly in the brain, liver, heart, skeletal muscles, spleen and sex organs (Literak, Hejlicek et al. 1992).

Waterborne transmission of *T. gondii* to humans has been implicated in numerous outbreaks in particular in British Columbia (Burnett, Shortt et al. 1998) and Brazil (de Moura, Bahia-Oliveira et al. 2006). Wildfowl would similarly be at risk from infection from infected water sources. Literak et al. (Literak, Hejlicek et al. 1992) found a high rate of infection in waterfowl including mallards. Mallard is the most frequently bagged species of waterfowl game and it was confirmed in 12%. Similar results have been found in China with 11.38% positive (Cong, Huang et al. 2012), Spain with 33.3% positive (Cabazon, Garcia-Bocanegra et al. 2011) and in Italy where 11.8% (n=17) mallard and 7.3% common teal (n=41) were seropositive (Mancianti, Nardoni et al. 2012).

There is little evidence of *T. gondii* infection in snipe and woodcock, 25% (n=8) prevalence in snipe (Mancianti, Nardoni et al. 2012) being the only data found. However, earth worms have been found to experimentally transmit *T. gondii* to eastern barred bandicoots in Tasmania (Bettiol, Obendorf et al. 2000) from passaging soil contaminated with *T. gondii* oocysts. As the diet of woodcock and snipe can be made up of up to 90% earthworms the possibility of infection via these vectors exists. In soil *T. gondii* oocysts have been reported to remain infective for up to 18 months (Frenkel 1975). Many millions of oocysts can be shed in the period a cat sheds the organism in its faeces. It has been calculated that the number of oocysts shed in a 20 g cat stool can be in the order of 2 to 20 million and after faecal decomposition, the local soil concentration can be as high as 100,000 oocysts/g (Frenkel 1975). Earthworms feeding on soil contaminated with decomposing cat faeces are known to take in *T. gondii* oocysts which can then be carried in their intestine and dispersed in their discarded alimentary casts (Frenkel, Ruiz et al. 1975).

The little evidence available on *T. gondii* infection in grey partridges suggests that they are highly susceptible to infection compared to other

birds with a prevalence of 18.7% (Literak, Hejlícek et al. 1992). Experimental infection confirmed this finding with grey partridges showing a higher susceptibility to infection compared to guinea fowl, turkeys and chickens (Sedlak, Literak et al. 2000).

Native quails (both Bobwhite and brown) in Egypt were found to have a prevalence of 22.4% and 28.8% respectively most likely from feeding from ground contaminated with oocytes (Shaapan, Khalil et al. 2011). After experimental infection of bobwhite and Japanese quail through oral inoculation of oocytes, infective stages were isolated from brains, hearts and skeletal muscles of all quail (Dubey, Ruff et al. 1993; Dubey, Goodwin et al. 1994).

In a recent EFSA report on poultry meat inspection *T. gondii* was classed as a low risk hazard for poultry consumption due to the fact that most commercial poultry is raised indoors and that chicken meat is usually well cooked. The prevalence rates obtained for most game birds indicate the potential risk of transmission to humans through consumption. As game birds are exposed to the outside environment and can be consumed underdone the risk of zoonotic transmission is non-negligible (EFSA 2012a).

A2.8.7 *Listeria monocytogenes*

Data on the incidence of *L. monocytogenes* in game birds is very scant. AHVLA VIDA records indicate that listeriosis is infrequently diagnosed in poultry - two cases were recorded during 2010, and there has been only one case per year from 2007-2009 (VIDA, 2010, unpublished data). Generally it appears that listeriosis in birds is rare with outbreaks often associated with other risk factors or infections, for example, concurrent IBV infection. Encephalitic listeriosis in red-legged partridges has been reported (AHVLA, 2011, unpublished data). In this incident infection was suggested to have been acquired at the hatchery or during transit and unknown risk factors enabled the disease to develop.

In an analysis of the potential risk that birds pose in spreading *Listeria* along the food chain a prevalence of 13.4% (n=996) in wild birds was found, but from just 2 species, crows and green-winged teals (Yoshida, Sugimoto et al. 2000). Only *L. innocua* was isolated from teals with a prevalence of 2.6%. No isolations were obtained from mallards (n=72) or from pigeons (n=135) in rural districts. McIlwain (1965) found 12.5% (n=8) of pigeons from pastureland to be positive indicating that the living environment may affect the incidence of *Listeria*. Similarly, a prevalence of 36% (n=212) of *L. monocytogenes* was found in birds from municipal landfill sites and urban areas in Helsinki (Hellstrom, Kiviniemi et al. 2008), with the landfill site showing a higher prevalence. The feeding environment can influence the prevalence of *L. monocytogenes* as illustrated by seagulls feeding on sewage having a higher prevalence than seagulls feeding elsewhere (Fenlon 1985). A *Listeria* prevalence of 20.3% was found in soil and plant samples (Weis and Seeliger 1975), 27.2% in wildlife feeding grounds and 17.3% in birds. One

pheasant and partridge showed septicaemia whereas the others harboured *Listeria* only in the intestinal tract suggesting that *L. monocytogenes* is an inhabitant of the normal intestine and there is a cycle between birds/animals and the soil/plant environment.

The environmental habitat combined with the feeding guild of certain bird species is likely to have an effect on the prevalence of faecal carriage of *L. monocytogenes*. Healthy wild birds commonly carry *Listeria* spp. asymptotically in their intestines, with the bacteria originating from the foods they eat and their environment. Birds do not harbour a distinct population of *L. monocytogenes* of their own but may have a role in disseminating *Listeria* in nature. The serotypes of *Listeria* found in birds include those commonly found to cause listeriosis in humans suggesting that birds might also disseminate the bacteria from their intestines into the food chain if they enter a food processing environment, but listeriosis has not been reported to be associated with contamination due to birds (Hellstrom, Kiviniemi et al. 2008). The presence of *L. monocytogenes* on carcasses of slaughtered birds is likely to be caused by cross contamination during processing (Escudero-Gilete, Gonzalez-Miret et al. 2007).

A2.9 Game bag/cart/belt

Food safety management starts straight after killing from when the birds are first picked up and moved. On most large shoots birds are retrieved and carried back to a game cart either by hand, in a specialist game carrier or in a game bag/belt. The majority of large shoots store birds hanging in a cart rather than a heap which is now apparently a trait of the past (Colin Sheddon pers. comm.). A game cart allows for free circulation of air and on most shoot days, with the exception of year-round shooting of woodpigeons, the ambient temperature will ensure some cooling.

Small shoots may retain the birds in the belt/bag until the end of the shoot. This could be up to 3 hours but they would usually be cooled initially by being placed in the net mesh compartment at the front of the bag. On small shoots birds are likely to be stored and handled carefully - each bird being relatively more important as it is more likely to be eaten by a member of the shooting party (Colin Sheddon pers. comm.).

A game bag can hold about 10 pheasants whilst some game carts can hold up to 250 birds. Most shoots aim to carry the game in a bag or by hand for as short a period as possible. On many rough shoots game will be hung in a suitable place and picked up later if this is feasible. As no chilling mechanism is available at this point the cooling of the bird carcass is reliant on good ventilation and the ambient temperature.

Whilst there is no shooting season for woodpigeons the majority of the shooting seasons run from around September to February, coinciding with bird migratory and breeding patterns. BASC, however, suggests that most commercial shoots don't commence until the beginning of October as birds

reared in the UK are not normally ready to shoot until this time. The average maximum temperature recorded during the shooting season in Scotland is 14.66°C for September (Figure 19); however, the average maximum temperature for the period between November and February does not rise above 8°C. For coastal wildfowling, where the preference is for shooting in the early morning or evening, the temperature is likely to be several degrees colder.

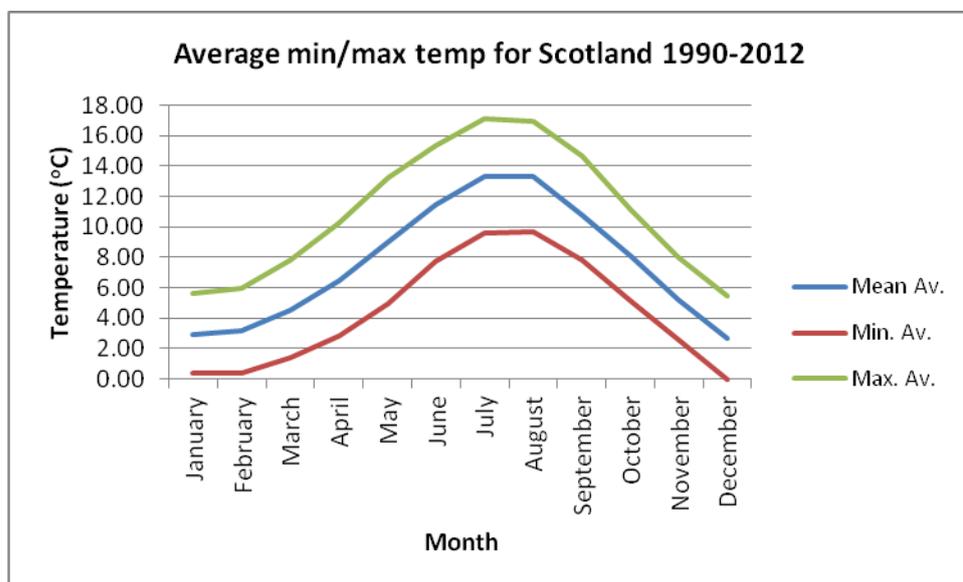


Figure 19: Average monthly min/max temperatures for Scotland 1990-2012 (Met. Office)

Practically all large shoots are likely to have a mixed bag. Shoots dominated by pheasants are still likely to have pigeons, partridges, or mallards in the game cart as incidental quarry. The bag of shoots targeted towards individual quarry such as coastal wildfowling or woodcock/snipe hunting are more likely to consist of just these species. Game carts are usually cleaned after each shoot although it is questionable whether a net-fronted game bag or specialist game carrier would need to be cleaned after each shoot due to the lack of contamination evident in these carriers (Colin Sheddon pers. comm.).

It is estimated that at an ambient temperature of 10°C it would take a period of 3 to 5 hours for the internal temperature of a bird carcass to reach the same value (Geoff Mead pers. comm.). Expert opinion (project steering group) suggests that the risk of cross-contamination between birds is likely to be minimal at this stage. Even though there is a high risk of contact between species, it is unlikely that pathogens from the gut will be transferred during such a short time period, even in the cases where the gut has been perforated.

A2.10 Game Larders

The term Game Larder refers to any premises or place (whether static or mobile) where killed wild game is kept either prior to being transported for further preparation at an AGHE or prior to preparation for sale to the final consumer or retail establishment. The primary functions of a larder is to provide initial cooling of the carcass and to provide temporary storage to hang wild game carcasses under hygienic, vermin and fly-proof conditions prior to despatch. Consequently the time period of storage can vary but expert opinion estimates that a shot gamebird will spend, on average, 12 - 72 hours stored in a game larder prior to dispatch to an AGHE or retail establishment.

Game estates may store shot game at a central game larder for collection and transport en masse. A game larder should have sufficient capacity to cater for throughput, protect game from contamination, be cleaned and disinfected and maintain the cold chain. A temperature of 4°C should be achieved for game birds within a reasonable period of time after killing. The cold chain should be maintained and game should be transported to a handling establishment ASAP after killing. In the UK, except at the very coldest times of the year and where storage and delivery times are short, active chilling in the game larder and the use of refrigerated vehicles to transport game from the larder to the approved game handling establishment, will be necessary to manage food risks. 38% of shoots with a full time keeper now have chillers installed, and 34% of shoots reported that their game dealers collected birds from them on the actual day of the shoot. The equivalent figure in 2002 was 6% so there appears to have been an improvement in this area (Alliance 2008). Although it still means that there is a degree of non-compliance with the regulations.

Growth of pathogens is possible if the game larder is unchilled. Storage at game larders represents a potential source of surface cross-contamination between game carcasses. An increased carcass microbial content will depend on whether or not the gut has been perforated by shot (Barnes *et al.* 1973). Carcass soiling with mud and faeces represents a potential source of carcass cross-contamination and contamination of meat, during transport and processing (David Inglis pers. comm. cited in (Coburn 2003).

In April 2008 Game-to-eat conducted a survey of 5,411 shoots to ascertain the level of improvement in game handling facilities. Results showed that two thirds of full-time kept shoots now have a chilling facility up from one third in 2006. Half of fulltime kept shoots have registered their game storage facility with their local authority (Alliance 2008). New regulations do not demand the installation of a chiller or refrigeration unit, but best practice will be to provide one.

Opinion states that there is still considerable non-compliance as shoot owners/managers can be reluctant to spend any money on larder facilities as the revenue from a shoot comes from paying customers shooting birds rather than the re-sale of shot game. Large commercial shoots will generally have something in place but these tend to be old shipping containers/lorry

bodies etc., the small farm shoots often hang birds in barns and hand them out at the end of the day.

However as game larders are not required to be registered as AGHEs and registration as an FBO is unlikely to be inspected by the LA on a regular basis it is largely unknown what proportion are chilled. Supermarkets are considered to collect and correctly store shot game (Coburn 2003). In a recent FSAS report some interviewees mistakenly said that it is a good practice to allow carcasses to cool down slowly to ambient temperature (FSAS 2012a). Whilst an increasing number of larders have improved their hygiene standards these are still not uniform and there is a considerable possibility of wild game being stored in non-refrigerated larders.

The key factor for hygienic standards of wild game is establishing and maintaining the cold chain, but this is difficult in practice and there is evidence that, in some cases, its operation is misunderstood. Temperature control is a generic means of food-pathogen control. Legislation requires that chilling of the carcass should begin within a 'reasonable period' after killing. The regulation avoids specifying the time between shooting and chilling to allow for longer lengths of time required for shoots in remote areas to access storage with chilling facilities.

A2.11 Transport and Distribution of birds

On small shoots the bag numbers are small and usually all consumed by the guns, beaters and local householders. Larger shoots usually sell any surplus birds on via an AGHE. A BASC survey showed that 80% of game consumed was obtained by shooting with the rest being bought, received as gifts or eaten in a restaurant.

Game birds are generally shot 12-24 hours before being transported for processing. Game bird carcasses may be laid in crates or suspended during transport. Some transportation practices increase the risk for carcass contamination. Contamination is a serious risk as birds and waterfowl may be laid in crates. The use of a chiller or refrigeration unit is best practice but may not be entirely necessary if storage times are short or ambient temperatures are low (Alliance 2008).

A recent FSAS report (FSAS 2012a) mapped 3 routes of Scottish wild game supply to the final consumer within which there is considerable variability since there is a large number and great diversity of suppliers.

- Supply from sporting estates passing primarily through AGHEs. The bulk of the primary production goes to an AGHE directly or through collecting agents, and the product of AGHEs enters the local/domestic market with a significant part exported. An increasingly common variation of this route of supply is for wild game to be processed by AGHEs and the game meat returned to the estates to be sold at local markets or by mail/internet sales. British Association for Shooting and Conservation estimates that 90 per cent

of the wild game birds shot in Scotland follow this first route of supply.

- Supply from estates to the final consumer or retailers under the 'hunter's' exemption. This route, which may pass through an estate larder, is for in-feather wild game or wild game meat supplied directly to the final consumer or to local retailers. Estates shooting between 50 and 150 birds a day tend to sell to farm shops/catering outlets, farmers' markets or through mail/internet sales. This scale of shooting is below the typical threshold of 200-300 birds a day that would be worthwhile for supply to an AGHE and exceeds the private consumption needs of a hunting party. British Association for Shooting and Conservation report that there is no information on the numbers of shoots of 50 or fewer birds a day and the destination of sales is unknown but they are assumed to go to private consumption.
- Supply from informal recreational shooting on farms or small properties. These recreational hunters do not usually have access to a larder. Sometimes, during spring and summer, game carcasses may be hung in small refrigerators.

The operators who are only involved in transportation should be registered with their LA as FBOs, but it is unclear to what extent this happens or if LAs examine their operations. Those collecting only in-feather game to deliver to AGHEs or exempt retailers for processing have to register as an FBO; meeting the traceability requirements of Regulation 178/2002; complying with general hygiene requirements for primary production and with the associated regulations specific provisions for the handling of large/small wild game. If these collecting agents also transport meat to AGHEs, or to exempt retailers for further processing, then they are obliged to also comply with the hygiene requirements as specified in Regulation 852/2004 Annex II, and have in place a food safety management system based on HACCP principles. Some estates are understood to have a direct relationship with collecting agents and it appears these are increasingly being used but their role in the supply chain is not clear.

It is estimated that 10 per cent of supply going to AGHEs is through collecting agents. It is alleged that collecting agents put good carcasses through AGHEs so as to command a better price, whilst the poorer quality carcasses, that may potentially pose a human health risk, bypass the system of official inspection and enter the food chain through regulatory exemptions. There is widespread concern within the wild game trade that collecting agents' standards are variable and that this is a weak link in the regulatory regime. In particular, respondents distinguished between the category of collecting agent that is working for a specific AGHE and those collecting agents who sell carcasses to various processors in response to offered price. This latter type of collecting agent is considered the most problematic part in the supply chain, where there are issues for traceability and maintenance of the cold chain. Although most collecting agents have

refrigerator vans and units, some have a disregard for temperature control since they mix carcasses at different temperatures, place carcasses in heaps, with few of their vehicles transporting carcasses on hanging rails. It was also suggested that the drivers of collecting vans may sign trained hunter's declarations (FSAS 2012a).

A2.12 Hanging

Hanging is a traditional procedure whereby gamebirds are hung, either by their feet or neck, usually in feather and uneviscerated for a certain length of time before consumption. The process produces a strong gamey flavour and tenderness in the meat of gamebirds which usually have relatively little fat content due to their lifestyle. The gamey flavour is suggested to occur due to autolytic changes occurring in the muscle. Normal 'spoilage' caused by bacteria, particularly enterobacteria which produce hydrogen sulphide, is inhibited from multiplying in the intestine of the hung game bird during storage. Evidence suggests that diet is largely responsible for this (Barnes 1979).

The effect of natural diet on bacterial growth in birds was illustrated in a study by Mead et al. Pheasants fed on a natural diet consisting of whole wheat, cabbage leaves, brambles and stinging nettles and chickens fed on normal broiler ration were killed by neck dislocation and hung in-feather at 10°C. Although bacteriologically there was little difference between the intestines of the two birds initially, the chickens developed greening within 5 days when the coli-aerogenes bacteria had multiplied throughout the small intestine, whilst in the pheasants hardly any growth occurred (Mead, Barnes *et al.* 1974). Pheasants reared on turkey starter rations throughout life are said to show greening like a chicken when hung and no gamey flavour develops. Many plants contain antimicrobial compounds (Nickell 1959). Stinging nettles which are eaten by some birds contain formic and acetic acids. These antimicrobial compounds may continue to exert their effect during hanging so that bacteria do not multiply in the small intestine.

Even when birds are hung at 15°C for several days the muscle will remain free from bacteria unless the gut has been perforated by shot. Multiplication of bacteria occurs only within the intestine (Barnes 1979). Most enterobacteria grow slowly at 10°C as refrigerated temperatures are near their minimal growth values. Growth will also depend on interactions with other factors such as pH and nutrients. In the caecum with its very large population of anaerobes multiplication of enterobacteria may increase possibly 10- or 100-fold. In the duodenum and small intestine where there are many fewer organisms and more available nutrients considerable multiplication can occur. It is unlikely that pathogenic bacteria will cross the intestine wall into muscle tissue during hanging unless the intestines are perforated by pellet shot to such a degree that leakage occurs. Natural decay of the intestinal wall would need to occur; and this would take up to 3 weeks depending on the holding temperature. During this time reduction

in fluid content of the gut and in the concentration of intestinal pathogens makes significant contamination less likely (Geoff Mead and Rob Davies pers. comm.).

Different views are held on the optimum duration of hanging according to personal taste although expert opinion states that modern tastes appear to tend towards little or no hanging. From this point of view, AGHEs usually supply game plucked and dressed with little or no hanging and birds are usually processed within 3 days of being shot. Private consumers however are more likely to hang gamebirds thereby increasing the time period over which pathogen multiplication could occur. Of those who consume game either shot by themselves or as a gift from others, 80% hang or store the birds before any form of preparation (FSAS 2012b). Generally this is for a few days, but can be for as long as 2 weeks. For a minority (10%) this can be for up to 4 weeks in duration (FSAS 2012b). It is possible for birds distributed via collecting agents or supplied for retail under regulatory exemptions to have been hung more than once.

The duration of hanging also appears to depend upon the species of bird. Woodcock and snipe are usually hung for 3-4 days and the flavour is strong and gamey. Recommended hanging time for partridge is 4-6 days in a cool place. The exceptions are quail, pigeon and wild duck which require little or no hanging.

A2.13 Transport to Slaughterhouse

Transport

Transport to slaughterhouse is only relevant for farmed quail as the other birds considered in this study are killed in the field. Transport of chickens to the slaughterhouse has been shown to increase the prevalence of birds positive for *Salmonella* and *Campylobacter* because of faecal contamination of skin and feathers by neighbouring birds during transport (Stern, Clavero *et al.* 1995). Asymptomatic shedding of *Salmonellae* in animals stressed through transport is also known to potentiate the surface contamination of meat (Labbe & Garcia 2001). Processing has also been shown to increase contamination in studies comparing on farm prevalence and to final product prevalence. No *Salmonella* was isolated from a commercial quail flock (McCrea, Tonooka *et al.* 2006) at farm level although swabs from transport crates were positive for *S. Heidelberg* and *S. Hadar*.

Transport cages are important sources of cross contamination (Berrang, Northcutt *et al.* 2003). Research shows that washing transport cages with water and leaving them to dry for 48 hours greatly lowers the levels of *Salmonella* found in the cages. Birds can pick up faeces and dust from cohort birds during transit to the slaughterhouse (Stern, Lyon *et al.* 1995). Consequently the skin and feathers of the birds can be contaminated with a variety of bacteria. Large numbers of bacteria are associated with feathers which can be soiled with dust, mud or faeces.

Slaughterhouse

The slaughterhouse presents a cross contamination risk for all pathogens including *T. gondii* from knives and work surfaces etc. (Kapperud, Jennum *et al.* 1996). The feathers, skin, crop and cloaca of birds brought to slaughter can be contaminated with *Salmonella* (Kotula and Pandya 1995). There can be a 20-40% increase in *Salmonella* both inside and outside the birds during movement based on chickens as moving the birds causes them to pass more faecal material. Once slaughtered the moist folds and crevices on the skin of the carcass give the bacteria a hospitable environment for growth and secure attachment (Stern, Lyon *et al.* 1995).

A2.14 Game Handling

A2.14.1 Plucking

Plucking can result in self-contamination of the bird due to manual manipulation resulting in faecal leakage. The Official Veterinary Surgeon (OVS) oversees the general operating of the licensed processing plants and is required to inspect 5% or 50 whichever is the greater, of each batch of birds to be exported. Immediately after post-mortem inspection the carcasses are chilled to below 4°C, or below -12°C if frozen. Birds are de-feathered 'dry'

using a de-feathering machine. Some processing plants immerse duck carcasses in hot wax to aid feather removal. Heads, legs and wings are cut off manually. Carcasses are eviscerated by hand or using evisceration forks. Body cavities may be further cleaned out by suction. Birds may be sold as whole bird carcass or may be portioned. If breast meat only is required, for example pigeon breast, birds are not de-feathered prior to the removal of the meat (David Inglis pers. comm. cited in (Coburn 2003)). In unlicensed plants producing birds for the domestic market only, carcasses and meat are not subject to official inspection at any stage of processing.

Personal observation by a risk assessor from a previous report (Coburn 2003) found evidence of failure to keep species separate from one another at unlicensed processing plants, and the processing of dirty carcasses. Under these circumstances, game birds could become contaminated via other species during processing. The effect of cross-contamination could result in spread of the organism, if present. This could result in more contaminated carcasses with lower numbers of organism on each.

Even on a plucked bird some feathers may remain attached to the carcass (John Longstreeth, pers. comm. cited in (Coburn 2003)). Defeathering can squeeze out faecal material from the feathers and gut by the action of the rubber fingers that mechanically remove the feathers resulting in self-contamination of the carcasses. The machines are difficult to clean and easily become contaminated during the slaughter of infected flocks; cross contamination can occur as a result.

The use of hot wax to remove feathers could significantly reduce surface pathogens. Some bacteria can be found in skin pores and other areas that might remain protected from the heat and wax. Additionally, although the wax temperature can be > 80°C it cools very quickly on the birds skin so the pathogens may not be exposed to high temperatures for the periods of time necessary to cause complete elimination of organisms.

A2.14.2 Evisceration

Evisceration is the process whereby the internal organs and intestines are removed from the game birds. The heart, liver and gizzard can be eaten and so are sold separately whilst others items of viscera are used for processing and manufacture of meat products. The inedible viscera consist of spleen, oesophagus, lungs, intestines and reproductive organs. A telephone survey of 8 AGHEs preferred to use manual evisceration techniques as they proved as quick and efficient as an automated line whilst also providing an opportunity for a further quality control step. The vent is removed and an incision is made from the vent hole to the tip of the breastbone. The bird is eviscerated by inserting a hand, or evisceration fork, into a vent incision and working the viscera away from the inside walls of the body. Proper technique for removing internal organs and other viscera are important for maintaining the quality of meat to be consumed. Poor evisceration techniques increase the risk of contamination from the alimentary tract. In

hung birds it is possible that autolysis could cause thinning of the intestine wall over a few days thereby making it more prone to rupturing during the process of evisceration.

The supply of small wild game birds such as woodcock, snipe and wood pigeon, often uneviscerated, from approved and exempt game-handling establishments to caterers and retailers, is suggested to be a quite widespread practice (FSAS 2012a). However, from a small telephone survey of AGHEs and consumers there is no evidence that any other bird species other than snipe and woodcock are consumed uneviscerated.

Evisceration with automated machines can rupture the intestines, causing faecal leakage to occur. Faecal contamination of the inner and outer surfaces of the carcass during evisceration is an important mode of contamination. There is more chance of spillage of intestinal contents onto the meat surface during the evisceration of birds than the processing of farm mammals. In considering commercial poultry processing evisceration is considered by several authors the most important source of microbial contamination since automated processing plant cannot completely avoid gut ruptures which are considered the most important source of several bacterial pathogens (Ziino, Giuffrida *et al.* 2008). The importance of this step was stressed with regard to the application of a HACCP system in the production of poultry meat. Faecal contamination and potential contamination from the crop or gizzard content are important considering the high frequency of *Salmonella* and *Campylobacter* isolations in these sites (Smith and Berrang 2006). Based on this evidence several authors have asserted that uneviscerated poultry could have better microbial characteristics and extended shelf life than eviscerated poultry (Mulder 2004). It has been shown that muscle tissue of uneviscerated game birds and poultry stored at refrigerated temperatures remained sterile for several days (Mead, Chamberlain *et al.* 1973).

Mead and Scott (1997) studied the spread of an enteric marker organism during evisceration of New York dressed (NYD) poultry in a simulated kitchen environment (Mead and Scott 1997). Five volunteers with experience of eviscerating game birds or poultry, each eviscerated 3 NYD chicken carcasses. In all cases, evisceration resulted in intestinal breakage, and faecal extrusion through cloaca, resulting in the spread of an enteric marker organism. The marker organism was identified on the chicken breast on 28/30 (93%) occasions, and on the back in 13/30 (43%) occasions. The volunteer's hands became heavily contaminated during the eviscerating process. Some of the marker organism spread beyond the cutting board supporting the view that evisceration of poultry in a domestic environment could lead to cross contamination of other foods.

Similarly, in a survey of enteric contamination in NYD and eviscerated chicken during storage at 3°C and 8°C results confirmed that evisceration greatly increased the faecal bacterial contamination of the internal body cavity surfaces. Bacteria migrated slowly into the muscles significantly affecting the muscle bacterial load after 14 days storage (Ziino, Giuffrida *et*

al. 2008). Average Total Viable Count (TVC), Enterobacteriaceae and *E. coli* counts for breast and thigh muscle from uneviscerated wild game birds were found to be within the limits set by EU legislation (Reg. (EC) No. 2073/2005) for freshly skinned carcasses of farmed ruminants and pigs. The EU limits apply to freshly slaughtered carcasses whilst the meat samples were from bird carcasses which had been stored for several days and which could also be contaminated via shot wounds (El-Ghareeb, Smulders *et al.* 2009)

Anecdotal evidence suggests that there has been some imported French quail meat to this country that has been supplied to markets and restaurants with heads on and only partially eviscerated (*effilé*) as chefs, particularly highly regarded chefs, enjoy working with game that is ‘entire’.

A2.15 Transport & Storage

A2.15.1 Transport

A sensitivity analysis of a risk assessment of salmonellosis in humans indicates that the probability of illness was highly sensitive to the growth of *Salmonella* during distribution and storage. Improper thawing was also a significant factor (FSANZ 2005).

Wild game meat may be sold directly by the processing plant or by farm shops or supermarkets. If there is insufficient demand for the wild game meat when it has been processed, some meat may be frozen and stored at the processing plant (Nacho Vinuela pers. comm. cited in (Coburn 2003)). Some farm shops process their own wild game. Supermarket wild game is selected from organised shoots by their suppliers and is generally collected from game estates within 24 hours. The meat is purchased directly from the processor and the final product is stored between 0 and 0.5°C. Game meats are normally on supermarket shelves within 4 days of being shot (Tom Richardson pers. comm. cited in (Coburn 2003)). Although the game season is quite short more and more frozen meat is available for all year round consumption.

An increasing trend is the supply of wild game bird meat by mail order. The Wild Meat Company products can be supplied fresh but may be frozen and refrozen this is deemed acceptable due to the perceived lack of pathogens in the original product. Major supermarkets all stock a variety of wild game during the season. High street butchers often sell game from local estates and farmers markets are a favourite for local game dealers to sell game. When game is out of season it is still available frozen.

A2.15.2 Storage

Bryan and McKinley (1974) measured the thawing temperatures of whole frozen turkey (Bryan and McKinley 1974). They recorded deep muscle and surface temperatures for a 20lb turkey. After 40 hours thawing in a refrigerator at 4°C the deep muscle temperature was only -2.8°C. At ambient temperature (24°C) the deep muscle temperature was 0°C after 9 hours and 10°C after 18 hours. The surface temperatures at ambient temperature were 10°C after 5 hours, and 16.6°C after 22 hours, demonstrating that surface temperatures can be relatively high by the time the turkey is thawed (cited in (WHO 2002) allowing opportunity for bacterial growth. However, a 20lb turkey is considerably larger and more difficult to thaw than a game bird (Geoff Mead pers. comm. cited in (Coburn 2003)).

Once wild game birds have been plucked or skinned almost two-thirds (60%) choose to freeze it prior to any other form of preparation, such as filleting. Nearly half (45%) of those freezing wild-game meat choose to freeze it for a period of more than six months, whilst 26% opt for a period of 2-3 months (FSAS 2012b). Freezing of wild-game meat for duration of six months ensures that the consumption of wild game meat can be continued out of the shooting season. Similarly, over two-thirds (70%) of respondents choose to freeze wild-game meat after the preparation stage. Within this group just over a quarter (26%) would freeze wild-game meat before any cooking, 13% after cooking, and the remainder (31%) both before and after the cooking. Although a high proportion of hunters freeze game for consumption out of season it is likely that they will consume mostly fresh gamebirds during the season.

Occasionally gamebirds, such as pheasants and grouse, can be frozen in feather allowing for sale outside of the shooting season time period by commercial companies. These birds are usually processed before sale or, very rarely, may be sold frozen in feather. Hunters may also use this form of storage for convenience. Depending on the exact conditions, freezing in feather would be expected to have little adverse effect on most bacterial pathogens but could be beneficial in reducing numbers of *Campylobacter* on the carcass surface. The combined effect of the initial freezing and frozen storage may be as much as a 100-fold reduction in numbers (Geoff Mead, pers. comm.)

A2.16 Pathogens at retail

A2.16.1 Salmonella

Differences in percentage of *Salmonella* (and *Campylobacter*) contaminated raw fresh and frozen chicken purchased at retail in the UK (FSA data from April-June 2001)). Fresh 4(56%) frozen 10.4 (31%) it was noted that freezing of chicken carcasses served to reduce *Campylobacters* on poultry meat by orders of magnitude. This contrasted with the situation in relation to *Salmonella* which was noted as being less sensitive to freezing. In the same study *Salmonella* prevalence was 6.6%. Thirty different serotypes were

found with *S. Kentucky* and *S. Bredeney* being most frequently found. Frequency of contamination was higher for frozen chicken (11.7%) than for chilled chicken (5.9%) (FSA 2009).

A2.16.2 *Campylobacter*

Campylobacter has been detected in fresh game bird meat samples (duck, grouse, guinea fowl, ostrich, pheasant, poussin) obtained in the UK at retail in a study undertaken between May and October 2004 (Little, Richardson *et al.* 2005). Samples were obtained from a variety of retail outlets mostly supermarkets (43% of samples) and licensed butchers (38% of samples) but also from outlets such as nonlicensed butchers, public houses, restaurants, market stalls, farm shops, convenience stores, hotels and take-aways. If the two recent UK retail surveys are combined the prevalence of *Campylobacter* in turkey and farmed game bird meat was significantly lower than in chickens and the average prevalence were 34, 42 and 61%, respectively. However, it should be noted that much fewer samples of retail turkey (214) and game bird meat (112) than chickens (1778) were sampled and thus may introduce uncertainty in the prevalence estimate. It is likely that as for chicken the prevalence will vary dependent on what samples are taken, location and time of year. The lower prevalence found in turkey and game bird meat in the combined UK surveys could suggest lower numbers of *Campylobacter* on turkey and game bird meat but there are no UK data available and only two reports have documented numbers of *Campylobacter* in non-chicken poultry samples. Portions of game bird meat samples exhibited higher contamination of *Campylobacter* compared to whole game bird samples.

In a UK survey of *Campylobacter* contamination of fresh chicken at retail between 2007-2008 prevalence was 65.2% with 52.9% being *C. jejuni*. Prevalence was higher for chilled chicken (47.6%) than frozen (13.6%) and levels of *Campylobacter* were significantly lower on frozen samples (FSA 2009).

A2.16.3 *E. coli*

A risk assessment on the attribution of human VTEC O157 infection from meat products has been carried out by AHVLA. It concluded that the prevalence of VTEC O157 in beef, lamb and pork joints was low (<0.04%) (Kosmider, Nally *et al.* 2010) with ground beef products, particularly beef burgers presenting the highest estimated risk.

A2.16.4 *T. gondii*

T. gondii has been found in retail meat: 4% (n=50) fresh pork meat in Spain (Bayarri, Gracia *et al.* 2012), 2.1% in pork meat in Mexico (Galvan-Ramirez, Madriz Elisondo *et al.* 2010), Zero prevalence in chicken and beef (Dubey, Hill *et al.* 2005) in USA but was found in pork. *T.gondii* was isolated from

the heart, brain and pectoral muscles of chickens in Guatemala (n=50) with a seroprevalence of 74% (Dubey, Lopez *et al.* 2005).

A2.16.5 Listeria

In a study on the presence of *Listeria* in retail chicken in Northern Ireland, *Listeria monocytogenes* was present in 18% (n=80) of retail packs of fresh chicken from supermarkets (Soultos, Koidis *et al.* 2003).

A2.1 Duration of time in stage

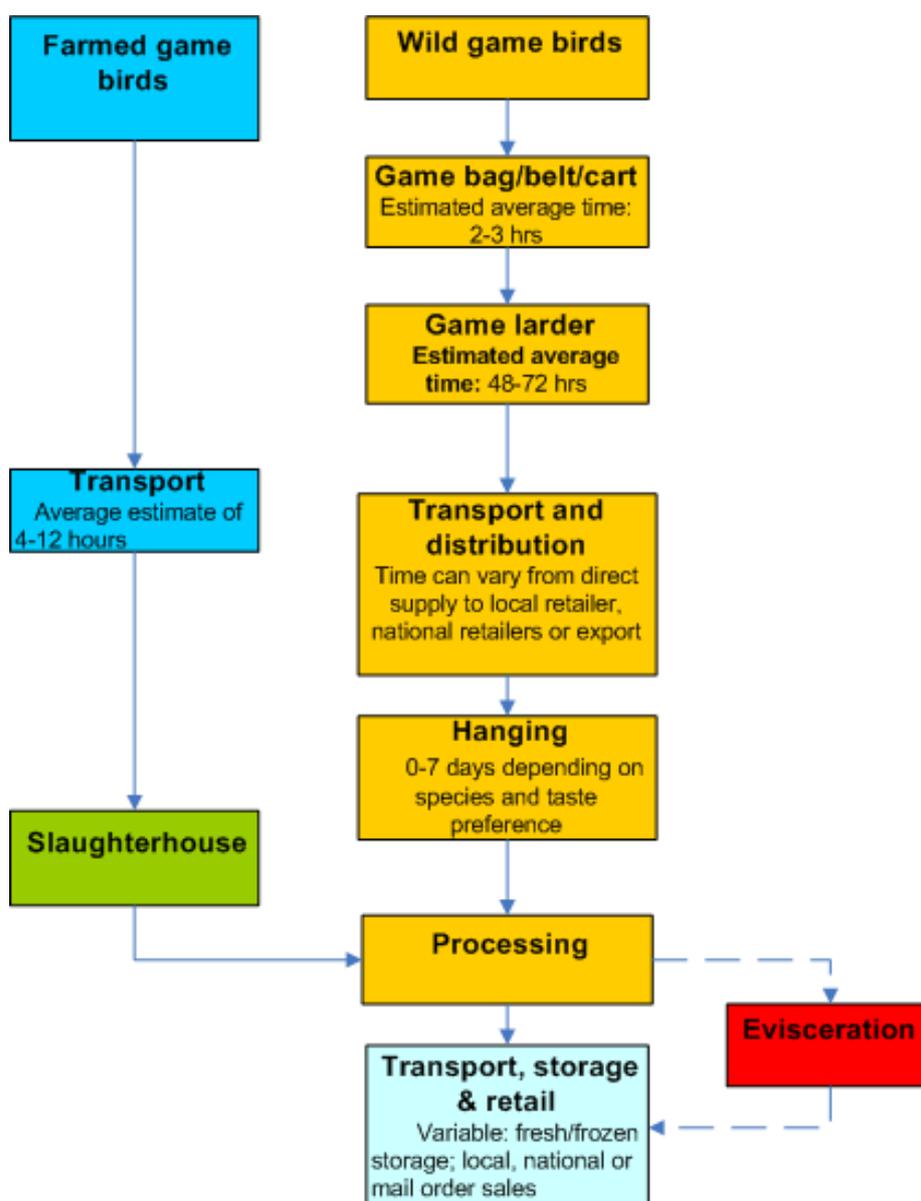


Figure 23 details information, where available, on the duration of time a bird would be expected to spend in each stage of the forest-to-fork chain. Information has been obtained from expert opinion in the industry, specifically Colin Sheddon (BASC) and AGHEs.

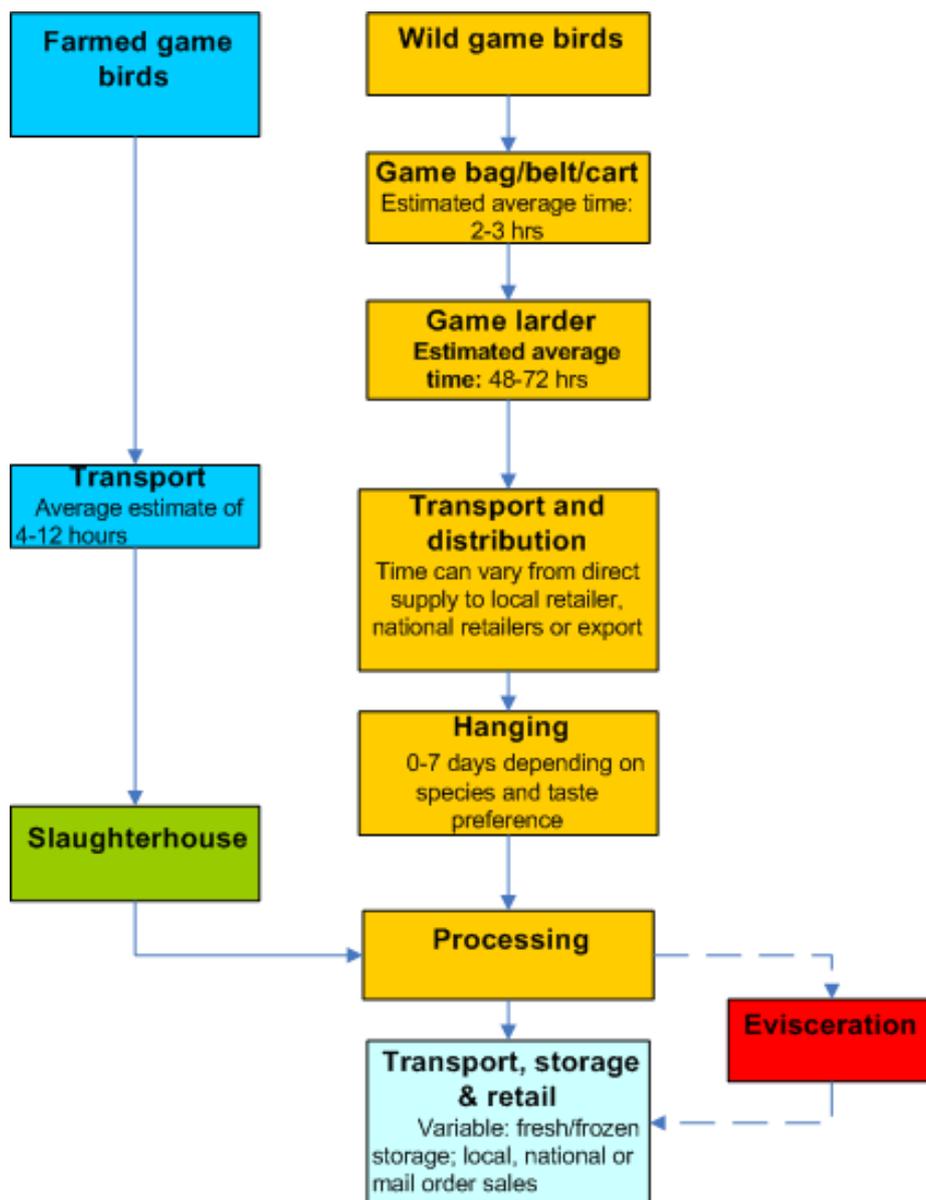


Figure 23: Estimate of average time a bird spends in each stage of the framework

A2.17 Game bird products

Meat that goes into pie mix and sausages is usually from birds that have been badly shot on one side and cannot be used for sale as a whole bird. There is not necessarily gut perforation and AGHEs will only cut away and use the good meat. When preparing wild-game meat themselves the majority discard any meat that is severely damaged as a result of shot, that is, wild-game meat which is not presentable, inedible or deemed unusable. In some instances where cuts of meat were badly damaged and not usable in their true form then the surrounding areas were cleaned from lead shot using the normal methods and used as wild game mix, generally taking the form of wild game pies, burgers or sausages (FSAS 2012b).

Diced game from the Wild Meat Company contains third venison, a third pheasant and partridge and a third combination of wild duck, pigeon, rabbit and hare. Their mixture depends on what is in season and what is being shot in good quantities at any particular time. The predominant use of diced game is for casseroles and pie making.

A potential growth area highlighted was thought to be through the sale of burgers and sausages. These are considered a 'familiar format' for everyday consumers as they are easy and quick for consumers to deal with. Coupled with the ease of freezing such formats, it was felt that consumption is likely to increase both out of season as well as during the shooting seasons.

In a Danish study looking at chicken products which are further processed, for example, sausages, prepared dishes, etc. due to the Danish legislation most plants, which produce these products, have implemented a HACCP-based quality assurance program to ensure microbiological 'safe' products. Moreover, the further processing often includes heat treatment, drying or smoking, which should eliminate the *Campylobacter* bacteria (Christensen 2001).

Restaurants and commercial organisations are more likely to be associated with terrines, smoking or salting and curing of wild-game meat.

An increase in pathogen prevalence can occur in game meat products as one contaminated bird can go on to contribute to several products. Critical factors will be:

- The final product, for example, game pie mix which has high probability of being well cooked or sausages, probability of being not thoroughly cooked especially if barbecued.
- Handling, for example, game pie mix has very little handling, sausages and burgers require more manual handling - increased risk of cross contamination between product and processor both ways
- Processing, for example, smoking and drying contributes to inactivation of pathogens but incorrect processing could result in pathogen survival and if no further cooking is carried out this will represent a risk.

A2.18 Preparation and consumption

A2.18.1 Household premises

Whilst there is little information on the preparation of wild game birds in either the home or catering kitchen, there is information relating to poultry. The critical points of preparation of game birds are therefore based on poultry due to the similarity between the anatomy of the products and the methods of cooking.

During normal cooking (roasting, frying, grilling) surfaces of poultry will reach temperatures at which *Salmonella* are killed. A risk for the consumer to be infected exists when eating undercooked products. In the UK (FSA 2005) undercooking of chicken was observed during consumer preparation (BBQ and stir-fry meals). Cross-contamination from raw products to cooked products or to ready-to-eat products via contaminated cutting-boards, kitchen utensils, dishcloths, hands etc. are also well known. However, in a study in UK no cross-contamination of *Salmonella* was detected when consumers prepared chicken in the home (FSA 2005).

Handling during preparation and cooking in the home or in professional food establishments is of critical importance for the prevention of salmonellosis. Gorman *et al.* (2002) found that *Salmonella* and other pathogens could be spread from fresh chicken to hands and food-contact surfaces in the domestic kitchen, such as the dish cloth, refrigerator handle, oven handle, counter top or draining board, during preparation of a traditional Sunday roast-chicken lunch (Gorman, Bloomfield *et al.* 2002).

The survival ability of *Campylobacter* means that there is a potential for cross-contamination in any food processing environment (Nolan *et al.* 1984, cited in (Acuff *et al.* 1986). In a series of experiments, *C. jejuni* could be recovered from either the hands or the fingernails of the food handler, hand soap and brushes in almost every case (Acuff *et al.* 1986). Cross-contamination during food-preparation in the home has been described as an important transmission route. Reducing the risk in the kitchen by avoiding cross-contamination, appropriate storage and handling and thorough cooking is essential.

Worsfold and Griffith (1997) studied food safety behaviour in the home. In general, if the organism is present there is plenty of opportunity for growth and cross-contamination, and particularly contamination of hands (Worsfold and Griffith 1997). The percentage of cooking steps that fail to heat food adequately is also not negligible; therefore if the organism is present there is a low probability that it remains viable. This coupled with the probable growth and cross-contamination of organism if present, means that it is quite likely to grow and spread.

Scott and Bloomfield looked at the survival and transfer of microbial contamination in a kitchen environment (Scott & Bloomfield 1990). On a clean laminate surface there was little survival at 4 and 24 hours, but under soiled conditions *E. coli* and *Salmonella* spp. survived in significant numbers up to 4 hours. In a quantitative risk assessment of human salmonellosis in Canadian chicken from retail to consumption (Smadi and Sargeant 2013) showed that concentration of *Salmonella* on chicken at retail and food hygiene practices in private kitchens such as cross-contamination due to not washing cutting boards, utensils or hands after handling raw meat along with inadequate cooking contributed most significantly to the risk of human salmonellosis. Here the pathway of interest was the retail to consumption pathway considering sources of contamination at different stages along the chain and their impact on human health. This part of the chain is less

controlled than other stages of the farm-to-fork continuum and the level of food contamination at the post retail level will be directly related to public health. The study looked at growth of *Salmonella* during transport from retail stores to consumers' homes and during refrigerated storage at consumers' homes. The average temperature on arrival at home was 10°C. Home refrigerated storage: 94% temperature was <5°C, maximum value of 5 days storage and average value of 2 days. Assuming that sufficient cooking should ensure elimination of bacteria it is only chicken that is inadequately cooked or exposed to cross-contamination at post cooking that might still carry *Salmonella* at the time of consumption. Cross contamination at serving - the potential to cross contaminate chicken with bacteria after cooking, hazardous as no further heat treatment will take place to inactivate *Salmonella*. Two main routes: raw chicken contaminates hands which contaminate cooked chicken and raw chicken contaminates cutting board or utensils which contact cooked chicken breast. A Canadian survey (Nesbitt, Ravel *et al.* 2012) found that 0.6% of food handlers did not wash their hands after handling raw meat. Table 4 shows the summarised data on transfer rate from cross contamination from published literature.

Table 4: Summary of transfer rates from cross contamination studies for *Salmonella* and surrogate bacteria from scientific literature. Values refer to weighted means in % (Smadi and Sargeant 2013)

Cross-contamination risk	Minimum	Mean	Maximum
Chicken to hands	1.135	6.54	26.06
Hands to food	0.145	8.93	52.95
Chicken to cutting board	3.02	7.5	30.96
Cutting board to food	10.49	19.4	42.38

The risk to human health due to consumption of contaminated gamebird meat with *Salmonella* depends on the set of circumstances existing during the consumption of each individual meal. There is no fixed risk but rather a 'range of risk' that could be attained by different consumers under different circumstances such as level of under cooking, degree of cross-contamination, susceptibility of the individual consuming the meat or a combination of these factors. The effect of different scenarios of risk mitigation strategies on the predicted probability of illness using @Risk simulation found that reducing the concentration of *Salmonella* at retail resulted in a greater reduction in predicted probability of illness (40% change) than reducing prevalence at retail (0% change). Reducing the probability of inadequate cooking occurring by 50%, however, gave only a 5% reduction in the probability of predicted illness, suggesting that cross contamination after cooking is likely to be important (Smadi and Sargeant 2013).

80% of the raw chickens brought into the home contained one or more intestinal disease microorganism (*Salmonella*, *Campylobacter*, *E. coli* and *S. aureus*). These microorganisms were found to cause cross-contamination in 12% dishcloths, 24% to hands, 4% refrigerator door handles, 20% oven door

handles 24% counter tops and 32% draining boards. The food preparer's hands have been cited as the main factor or contributory factor in up to 39% of domestic food poisoning outbreaks (Ryan, Wall *et al.* 1996).

DeWit, *et al.* (de Wit, Broekhuizen *et al.* 1979) used a marker organism to study cross-contamination resulting from the preparation of broilers. The cross-contamination rates showed that the more direct the contact between broiler and item, the greater the percentage of positive samples from that item. Washing reduces the incidence of cross-contamination, but not completely. In the preparation process other surfaces such as water taps and spice jars, also became contaminated, but to a lesser extent, indicating direct contamination from hands.

Zhao *et al.* (1998) found that chicken meat and skin inoculated with 10^6 cfu bacteria transferred 10^5 cfu to a chopping board and hands (Zhao, Zhao *et al.* 1998). Disinfection of the chopping board and hand washing reduced the numbers of bacteria by 1-2.8 logs.

Campylobacter has been found to be readily spread in the kitchen during preparation of raw foods such as chicken, and studies examining consumer behaviour in the kitchen have shown that practices likely to lead to cross-contamination of *Campylobacter* from raw foods, especially chicken, to ready-to-eat foods are common. One study involving the observation of 108 consumers from all socio-economic backgrounds making prescribed meals found 58% occurrence of the handler not washing their hands after handling raw meat/poultry. In the same study, one-third of consumers washed raw chicken, and 15% failed to cook foods to a temperature of at least 74°C . A questionnaire/interview-based study of 1,030 consumers assessing practices in relation to the handling of raw meat identified that the majority routinely washed raw meat, with whole chicken being the highest (80%) (ACMSF 2005).

In a study of the cross-contamination potential of *Campylobacter* during the preparation of Sunday lunch made from raw chicken, 25 participants were allowed to prepare a meal in their own kitchens. Of the 11 where *Campylobacter* was isolated from the raw chicken, the organism was recovered from hands (3), oven handles (2), counter tops (3) and the draining board (4) following preparation of the chicken (Gorman, Bloomfield *et al.* 2002). Levels of contamination with *Campylobacter* can be effectively reduced in the domestic kitchen by adherence to a prescribed cleaning regime using detergent, hot water and disinfectant. Some research has shown that using the former two alone is less effective on surface contamination (Cogan, Bloomfield *et al.* 1999). It is also clear that effective hand washing makes an important contribution to improving hygiene. A recent review determined that washing hands with soap could be expected to decrease the risk of diarrhoeal disease in the community by almost half (Curtis and Cairncross 2003).

A2.18.2 Outside the Home

34% of all food consumption now takes place outside the home (Alliance 2008). The majority of *Campylobacter* outbreaks in England in 2011 were associated with the consumption of poultry liver pate/parfait at food service premises. Evidence showed that chefs continue to use undercooked chicken livers in the preparation of these (Defra 2012).

In a case-control study of primary, indigenous, sporadic campylobacteriosis in England and Wales, however, consumption or handling of chicken cooked and eaten in the home was found to be protective. Similarly, in a study in New Zealand, recent consumption of baked or roast chicken seemed to be protective, although consumption of raw or undercooked chicken, or chicken from restaurants, was associated with illness. An earlier study in New Zealand also showed that eating at home was protective. There is scope for cross-contamination of other foods if infected poultry is introduced into the kitchen. Yet if cooked properly the contaminated chicken itself no longer poses a risk.

Investigation of the 50 outbreaks of campylobacteriosis in England and Wales between 1995 and 1999 identified 35 (70%) as foodborne transmission. Outbreaks mainly occurred in commercial catering premises (32/50, 64%) including 16 in restaurants, 10 in hotels, 4 in public houses or bars and 1 in each of a hall and canteen. The majority of the remainder occurred in schools (12%) and the armed services (8%). Of the 35 foodborne outbreaks, poultry products (13 chicken and 1 duck) were the most commonly identified likely vehicles. The reasons identified as contributing to the outbreaks included cross-contamination (18 outbreaks), inadequate heat treatment (10 outbreaks), and inappropriate storage (7 outbreaks) (ACMSF 2005). Of the 37 *Campylobacter* outbreaks reported in the EU summary report on zoonotic agents and food-borne outbreaks, 34 identified a source location of which 19 were catering establishments and 8 were from household/domestic kitchens. Broiler meat was the most commonly implicated food vehicle accounting for 17 outbreaks and two were related to consumption of duck liver pâté (EFSA 2013).

Food such as raw chicken entering a domestic or catering facility represents a significant cross-contamination and, in turn, risk of infection. With levels of over 100,000 cfu on some chicken carcasses, as little as 0.5% of the original contaminants need to be transferred to a ready-to-eat food to cause a potential infection. IID study concluded that even minor lapses in food hygiene practices could result in cross-contamination. Any attempt to reduce *Campylobacter* infections must address the high levels entering the food supply chain and kitchen, as well as the practices that should be in place in domestic and kitchen settings to destroy or prevent contamination with the organism. Indeed, a quantitative risk assessment of human campylobacteriosis associated with thermophilic *Campylobacter* spp. in chicken estimated that in order to achieve a 30-fold reduction in human disease, kitchen hygiene would have to improve by approximately 30-fold, whereas a reduction in the number of the *Campylobacter* on chicken

carcasses by 2-log cfu would achieve the same effect (Rosenquist, Nielsen *et al.* 2003).

A2.18.3 The consumer

Being a small bird, woodcock are often eaten uneviscerated. The head is left on, eyes removed and the beak used for trussing the bird. The innards should be at least 125°C to allow for thorough cooking although some recipes specify eating woodcock pink and slightly bloody. When woodcock and snipe are eaten uneviscerated, when they've been cooked if you open the vent the guts have disintegrated as they are so small. As soon as the heat hits it they frizzle up (AGHE pers. comm.).

It is commonly supplied traditionally i.e. plucked but uneviscerated and cooked and eaten with its innards intact. Some hunters prefer snipe to woodcock as it's possible to cook the innards properly with the smaller birds without ruining the breast by overcooking.

Woodpigeon is widely available and can be purchased from most supermarkets and numerous mail order companies. Most of the meat comes from the breast which is often served pink. There is no evidence to suggest that pigeons are eaten uneviscerated.

All three species of wild duck can be purchased from specialist mail order companies. Most of the meat comes from the breast which is often served pink. Widgeon has the most unpredictable flavour as this species grazes on both salt and freshwater marshes. There is no evidence to suggest that wild duck is eaten uneviscerated.

Recipes suggest that partridge meat is best eaten slightly pink. There is no evidence to suggest that partridge are eaten uneviscerated.

There is anecdotal evidence that those involved in game management (such as shooters, gamekeepers and game beaters) and their families eat higher quantities of wild-game meat than the general population. There is also a lack of information regarding the typical practices these individuals employ to prepare wild-game meat for consumption., i.e. how is the wild-game meat obtained (shot by a consumer, purchased from a shooter, obtained as a gift), which game is eaten in highest quantities (game birds, venison, etc.), how is meat dressed (is pellet/shot removed, is wounded tissue around the shot channel discarded), what common cooking and preparation techniques are involved (marinating, roasting, cooking, broiling) (FSAS 2012b).

The FSAS conducted an initial assessment of the currently available literature on the levels of wild game meat consumption and the practices involved in preparing the meat and came to the conclusion that there was very little information available (FSAS 2012b).

In the UK the National Diet and Nutrition Survey (NDNS 2010) states a mean daily consumption amongst the UK population of 0.681 g per day of game meat (approximately 250 g/year). Duck accounts for 70% of this average annual consumption of 250 g but it is not specified if this duck is wild or farmed. The mean consumption of game meat for those in the general population who reported eating game is 11.7 g per day or 82 g per week and the maximum recorded was 91.2 g daily or 638 g per week. It was estimated that high level consumers would eat an average 47.4 g daily or 331.5 g weekly. A High level consumer is defined as anybody that eats wild game meat at least once a week during the season. Amongst this group of consumers, wild-game meat is generally eaten no more than once or twice a week, so in the main it is not considered an everyday meal, even amongst high-level consumers.

In a report featuring consumer research undertaken with a representative sample of UK adults (Mintel 2008), 5% claim to eat game fairly regularly when in season, but there is no definition of '*fairly regularly*'. The figure for Scotland is 7%, equating to around 350,000 people. A survey carried out by the BASC amongst its members in the North-West of England and North-East Wales measured the frequency of consumption of game meat. Just under a quarter (23%) of households ate game meat (excluding venison and wild boar) once a week or more; In terms of seasonality, just under half (43%) of households typically consumed their game meat during the shooting season, while the rest (57%) consumed it all year round. It is widely accepted that high consumers are likely to be those associated with running shoots, shooters and their families.

Eighty nine per cent of shooters' households, including children, eat game meat (FSAS 2012b). Eighty per cent of the game consumed was obtained by shooting with the rest being bought, received as gifts or eaten in a restaurant. The market for game has grown substantially over the past few years and, according to Mintel, is likely to continue to grow, as it ties into a number of longer term trends, such as the interest in food origin, animal welfare and adventurous and authentic food. This may present limitations for the further development of commercial scale game production due to consumer perceptions relating to intensive production methods, feeding strategies and game management.

The consumer research (Mintel 2008) profiled game eaters in the following way:

- People with higher income and higher position in society (ABs) are the key consumers, as they are the most likely to appreciate gourmet food, and game. It fulfils their requirements for an interesting product with flavour.

- Third Age consumers (in the 45-54 age group) appear to be significant regular users of game meat. The low fat/healthy status of game meat is an important factor for this group.

- Younger consumers are the most concerned about whether they have the cooking skills to get the best out of this category of meat (they are

also the least likely to shop in butchers through which much game meat is still sold).

Whether eating in the home or eating out, it was thought the methods available for the cooking of wild game meat are wide and varied, and can vary depending on the type of meat. Generally, pan-frying was the cooking method of choice for most meat types, particularly amongst the larger wild-game meats available. For the smaller species however, such as partridge (72%), grouse (82%), woodcock (73%) and snipe (82%), there is more likelihood of them being roasted. There are some minor differences in methods between those adopted at home versus those when eating out. Although there seems to be increasing domestic numbers employing differing methods of preparation, restaurants and commercial organisations are more likely to be associated with potted terrines, smoking, or salting and curing of wild-game meat. Traditionally, roasting and pan-frying are considered to be the most popular cooking methods in a domestic environment, but more and more individuals are becoming adventurous and therefore more comfortable with new cooking methods providing a greater variety of styles of wild-game meat in the home.

Snipe or woodcock is normally eaten as a starter, and tends to be the whole bird.

□ Pigeon is also generally considered as a starter consisting of one or two breasts per person.

If a game bird has been bought in-feather, the carcass must first be defeathered and eviscerated by the food handler. These represent additional hazards to the food handler, similar to those encountered during game bird processing. Game birds sold in supermarkets are sold as fresh or frozen whole bird or portions. Preparation involves the handling of raw meat and hence cross-contamination could occur.

8. References:

- Abulreesh, H. H., Goulder, R., Scott, G. W (2007). "Wild birds and human pathogens in the context of ringing and migration." Ringling and migration **23**: 193-200.
- ACMSF (2005). "Second Report on *Campylobacter*."
- ACMSF (2012). "Risk Profile in relation to *Toxoplasma* in the food chain."
- Acuff, G. R., C. Vanderzant, M. O. Hanna, J. G. Ehlers, F. A. Golan and F. A. Gardner (1986). "Prevalence of *Campylobacter jejuni* in turkey carcass processing and further processing of turkey products." Journal of Food Protection **49**(9): 712-717.
- ADAS (2005). "The UK Game Bird Industry - A short Study."
- ADAS (2007). "Project B15019 - Review of current information on *Campylobacter* in poultry other than chicken and how this may contribute to human cases."
http://www.foodbase.org.uk/admintools/reportdocuments/34_68_Project_B15019_Campylobacter_in_poultry_other_than_chicken-.pdf.

- AHVLA (2011). "Salmonella in livestock production 2011." <http://www.defra.gov.uk/ahvla-en/publication/salm11/>.
- Al-Kappany, Y. M., M. R. Lappin, O. C. H. Kwok, S. A. Abu-Elwafa, M. Hilali and J. P. Dubey (2011). "SEROPREVALENCE OF *TOXOPLASMA GONDII* AND CONCURRENT *BARTONELLA* SPP., FELINE IMMUNODEFICIENCY VIRUS, FELINE LEUKEMIA VIRUS, AND *DIROFILARIA IMMITIS* INFECTIONS IN EGYPTIAN CATS." Journal of Parasitology **97**(2): 256-258.
- Allen, H. K., J. Donato, H. H. Wang, K. A. Cloud-Hansen, J. Davies and J. Handelsman (2010). "Call of the wild: antibiotic resistance genes in natural environments." Nature Reviews Microbiology **8**(4): 251-259.
- Alliance, C. (2008). "Making the most of your game." Fact sheet.
- Andrews, B. E., R. Major and S. R. Palmer (1981). "Ornithosis in poultry workers." Lancet **I**(8221): 632-634.
- Autio, T., Lindstrom, M., and Korkeala, H. (2004). "Research update on major pathogens associated with fish products and processing of fish." Food safety assurance and veterinary public health **2**: 115-134.
- Azanza, M. P. V. and M. P. Ortega (2004). "Microbiology of day-old chicks: a Philippine streetfood." Food Control **15**(4): 245-252.
- Barnes, E. M. (1979). "The intestinal microflora of poultry and game birds during life and after storage. Address of the president of the Society for Applied Bacteriology delivered at a meeting of the society on 10 January 1979." The Journal of applied bacteriology **46**(3): 407-419.
- Bartoszcze, M., K. Krupa and J. Roszkowski (1991). "ELISA for assessing *Toxoplasma gondii* antibodies in pigs." Zentralblatt fur Veterinarmedizin. Reihe B. Journal of veterinary medicine. Series B **38**(4): 263-264.
- BASC (2013). "The British association for Shooting & Conservation." <http://www.basc.org.uk/>.
- Bayarri, S., M. J. Gracia, C. Perez-Arquillue, R. Lazaro and A. Herrera (2012). "*Toxoplasma gondii* in Commercially Available Pork Meat and Cured Ham: A Contribution to Risk Assessment for Consumers." Journal of Food Protection **75**(3): 597-600.
- Beer, J. (1989). "Gamebird diseases in Great Britain." Pheasants in Asia: 265-274.
- Benskin, C. M. H., K. Wilson, K. Jones and I. R. Hartley (2009). "Bacterial pathogens in wild birds: a review of the frequency and effects of infection." Biological Reviews **84**(3): 349-373.
- Berrang, M. E., J. K. Northcutt, D. L. Fletcher and N. A. Cox (2003). "Role of dump cage fecal contamination in the transfer of *Campylobacter* to carcasses of previously negative broilers." Journal of Applied Poultry Research **12**(2): 190-195.
- Bettioli, S. S., D. L. Obendorf, M. Nowarkowski and J. M. Goldsmid (2000). "Pathology of experimental toxoplasmosis in eastern barred bandicoots in Tasmania." Journal of Wildlife Diseases **36**(1): 141-144.
- Beuchat, L. R. (1996). "Pathogenic microorganisms associated with fresh produce." Journal of Food Protection **59**(2): 204-216.
- Bevan, B. J. and C. D. Bracewell (1986). "Chlamydiosis in birds in Great Britain. 2. Isolations of *Chlamydia psittaci* from birds sampled between 1976 and 1984." The Journal of hygiene **96**(3): 453-458.

- birdweb (2013). "Birdweb." <http://www.birdweb.org/birdweb/>.
- Black, R. E., Perlman, D., Clements, M.L., Levine, M.M., Blaser, M.J. (1993). "Human Volunteer studies with *Campylobacter jejuni*." Online Information for the Defense Community: 207-215.
- Boyer, K., D. Hill, E. Mui, K. Wroblewski, T. Karrison, J. P. Dubey, M. Sautter, A. G. Noble, S. Withers, C. Swisher, P. Heydemann, T. Hosten, J. Babiarz, D. Lee, P. Meier, R. McLeod and G. Toxoplasmosis Study (2011). "Unrecognized Ingestion of *Toxoplasma gondii* Oocysts Leads to Congenital Toxoplasmosis and Causes Epidemics in North America." Clinical Infectious Diseases **53**(11): 1081-1089.
- Bracewell, C. D. and B. J. Bevan (1986). "Chlamydiosis in birds in Great Britain. 1. Serological reactions to chlamydia in birds sampled between 1974 and 1983." The Journal of hygiene **96**(3): 447-451.
- Brand, C. J. (1989). "Chlamydial infections in free-living birds." Journal of the American Veterinary Medical Association **195**(11): 1531-1535.
- Broman, T., J. Waldenstrom, D. Dahlgren, I. Carlsson, I. Eliasson and B. Olsen (2004). "Diversities and similarities in PFGE profiles of *Campylobacter jejuni* isolated from migrating birds and humans." Journal of Applied Microbiology **96**(4): 834-843.
- Bryan, F. L. and T. W. McKinley (1974). "Prevention of foodborne illness by time-temperature control of thawing, cooking, chilling and reheating turkeys in school lunch kitchens." Journal of Milk Food Technology **37**(8).
- BTO (2013). "British Trust for Ornithology." <http://www.bto.org/>.
- Burnet, F. M. and P. M. Rountree (1935). "Psittacosis in the Developing Egg." Journal of Pathology and Bacteriology **40**: 471-481.
- Burnett, A. J., S. G. Shortt, J. Isaac-Renton, A. King, D. Werker and W. R. Bowie (1998). "Multiple cases of acquired toxoplasmosis retinitis presenting in an outbreak." Ophthalmology **105**(6): 1032-1037.
- Buzby, J. C. and T. Roberts (1997). "Economic costs and trade impacts of microbial foodborne illness." World health statistics quarterly. Rapport trimestriel de statistiques sanitaires mondiales **50**(1-2): 57-66.
- Cabezón, O., I. García-Bocanegra, R. Molina-Lopez, I. Marco, J. M. Blanco, U. Hoefle, A. Margalida, E. Bach-Raich, L. Darwich, I. Echeverria, E. Obon, M. Hernandez, S. Lavin, J. P. Dubey and S. Almeria (2011). "Seropositivity and Risk Factors Associated with *Toxoplasma gondii* Infection in Wild Birds from Spain." Plos One **6**(12).
- Cabezón, O., I. García-Bocanegra, R. Molina-Lopez, I. Marco, J. M. Blanco, U. Hofle, A. Margalida, E. Bach-Raich, L. Darwich, I. Echeverria, E. Obon, M. Hernandez, S. Lavin, J. P. Dubey and S. Almeria (2011). "Seropositivity and Risk Factors Associated with *Toxoplasma gondii* Infection in Wild Birds from Spain." Plos One **6**(12).
- CAC, C. A. C. (1999). "Principles and guidelines for the conduct of microbiological risk assessment." FAO, Rome. CAC/GL-30.
- Christensen, B., Sommer, H., Rosenquist, H., Nielson, Niels. (2001). "Risk Assessment on *Campylobacter jejuni* in chicken products." The Danish Veterinary and Food Administration.
- Cizek, A., P. Alexa, I. Literak, J. Hamrik, P. Novak and J. Smola (1999). "Shiga toxin-producing *Escherichia coli* O157 in feedlot cattle and

- Norwegian rats from a large-scale farm." Letters in Applied Microbiology **28**(6): 435-439.
- Coburn, H., Snary, E., Wooldridge M (2003). "Hazards and Risks from Wild Game: A Qualitative Risk Assessment." FSA Project code MO1025.
- Coburn, H. L., E. L. Snary, L. A. Kelly and M. Wooldridge (2005). "Qualitative risk assessment of the hazards and risks from wild game." Veterinary Record **157**(11): 321-322.
- Cogan, T. A., S. F. Bloomfield and T. J. Humphrey (1999). "The effectiveness of hygiene procedures for prevention of cross-contamination from chicken carcasses in the domestic kitchen." Letters in Applied Microbiology **29**(5): 354-358.
- Cole, D., D. J. V. Drum, D. E. Stallknecht, D. G. White, M. D. Lee, S. Ayers, M. Sobsey and J. J. Maurer (2005). "Free-living Canada Geese and antimicrobial resistance." Emerging Infectious Diseases **11**(6): 935-938.
- Colles, F. M., J. S. Ali, S. K. Sheppard, N. D. McCarthy and M. C. J. Maiden (2011). "*Campylobacter* populations in wild and domesticated Mallard ducks (*Anas platyrhynchos*)." Environmental Microbiology Reports **3**(5): 574-580.
- Cong, W., S. Huang, D. Zhou, M. Xu, S. Wu, C. Yan, Q. Zhao, H. Song and X. Zhu (2012). "First report of *Toxoplasma gondii* infection in market-sold adult chickens, ducks and pigeons in northwest China." Parasites and Vectors **5**(110): (7 June 2012)-(2017 June 2012).
- Consultants, C. R. (1997). "Countryside sports, their economic, social and conservation significance. The standing conference on countryside sports. UK."
- Cook, A. J. C., R. E. Gilbert, W. Buffolano, J. Zufferey, E. Petersen, P. A. Jenum, W. Foulon, A. E. Semprini, D. T. Dunn and C. European Res Network (2000). "Sources of toxoplasma infection in pregnant women: European multicentre case-control study." British Medical Journal **321**(7254): 142-147.
- Council, F. A. W. (2008). "Opinion on the welfare of farmed gamebirds."
- Curtis, V. and S. Cairncross (2003). "Effect of washing hands with soap on diarrhoea risk in the community: a systematic review." Lancet Infectious Diseases **3**(5): 275-281.
- D'Aoust, J. Y. (1985). "Infective dose of *Salmonella* Typhimurium in Cheddar cheese." American Journal of Epidemiology **122**(4): 717-720.
- Dabritz, H. A. and P. A. Conrad (2010). "Cats and Toxoplasma: Implications for Public Health." Zoonoses and Public Health **57**(1): 34-52.
- de Boer, E., W. M. Seldam and H. H. Stigter (1983). "[*Campylobacter jejuni*, *Yersinia enterocolitica* and *Salmonella* in game and poultry]." Tijdschr Diergeneeskde **108**(21): 831-836.
- de Moura, L., L. M. G. Bahia-Oliveira, M. Y. Wada, J. L. Jones, S. H. Tuboi, E. H. Carmo, W. M. Ramalho, N. J. Camargo, R. Trevisan, R. M. T. Graca, A. J. da Silva, I. Moura, J. P. Dubey and D. O. Garrett (2006). "Waterborne toxoplasmosis, Brazil, from field to gene." Emerging Infectious Diseases **12**(2): 326-329.
- de Wit, J. C., G. Broekhuizen and E. H. Kampelmacher (1979). "Cross-contamination during the preparation of frozen chickens in the kitchen." The Journal of hygiene **83**(1): 27-32.

- Defra (2012). "Zoonoses Report UK 2011: ." <http://www.defra.gov.uk/publications/files/pb13851-zoonoses-2011.pdf>.
- Depoorter, P., D. Persoons, M. Uyttendaele, P. Butaye, L. De Zutter, K. Dierick, L. Herman, H. Imberechts, X. Van Huffel and J. Dewulf (2012). "Assessment of human exposure to 3rd generation cephalosporin resistant *E. coli* (CREC) through consumption of broiler meat in Belgium." International Journal of Food Microbiology **159**(1): 30-38.
- Deschuyffeleer, T. P. G., L. F. V. Tyberghien, V. L. C. Dickx, T. Geens, J. M. M. M. Saelen, D. C. G. Vanrompay and L. A. C. M. Braeckman (2012). "Risk Assessment and Management of *Chlamydia psittaci* in Poultry Processing Plants." Annals of Occupational Hygiene **56**(3): 340-349.
- Diaz-Sanchez, S., A. Mateo Moriones, F. Casas and U. Hoefle (2012). "Prevalence of *Escherichia coli*, *Salmonella* sp and *Campylobacter* sp in the intestinal flora of farm-reared, restocked and wild red-legged partridges (*Alectoris rufa*): is restocking using farm-reared birds a risk?" European Journal of Wildlife Research **58**(1): 99-105.
- Dibb-Fuller, M. P., E. Allen-Vercoe, C. J. Thorns and M. J. Woodward (1999). "Fimbriae- and flagella-mediated association with and invasion of cultured epithelial cells by *Salmonella* Enteritidis." Microbiology-Uk **145**: 1023-1031.
- Dickx, V., T. Geens, T. Deschuyffeleer, L. Tyberghien, T. Harkinezhad, D. S. A. Beeckman, L. Braeckman and D. Vanrompay (2010). "*Chlamydophila psittaci* Zoonotic Risk Assessment in a Chicken and Turkey Slaughterhouse." Journal of clinical microbiology **48**(9): 3244-3250.
- Dipineto, L., A. Gargiulo, L. M. D. L. Bossa, L. Rinaldi, L. Borrelli, A. Santaniello, L. F. Menna and A. Fioretti (2009). "Prevalence of thermotolerant *Campylobacter* in partridges (*Perdix perdix*)." Letters in Applied Microbiology **49**(3): 351-353.
- Dubey, J. P. (2010). "Toxoplasma gondii Infections in Chickens (*Gallus domesticus*): Prevalence, Clinical Disease, Diagnosis and Public Health Significance." Zoonoses and Public Health **57**(1): 60-73.
- Dubey, J. P., M. A. Goodwin, M. D. Ruff, O. C. Kwok, S. K. Shen, G. C. Wilkins and P. Thulliez (1994). "Experimental toxoplasmosis in Japanese quail." Journal of veterinary diagnostic investigation : official publication of the American Association of Veterinary Laboratory Diagnosticians, Inc **6**(2): 216-221.
- Dubey, J. P., D. E. Hill, J. L. Jones, A. W. Hightower, E. Kirkland, J. M. Roberts, P. L. Marcet, T. Lehmann, M. C. B. Vianna, K. Miska, C. Sreekumar, O. C. H. Kwok, S. K. Shen and H. R. Gamble (2005). "Prevalence of viable *Toxoplasma gondii* in beef, chicken, and pork from retail meat stores in the United States: Risk assessment to consumers." Journal of Parasitology **91**(5): 1082-1093.
- Dubey, J. P., A. W. Kotula, A. Sharar, C. D. Andrews and D. S. Lindsay (1990). "Effect of high temperature on infectivity of *Toxoplasma gondii* tissue cysts in pork." The Journal of parasitology **76**(2): 201-204.
- Dubey, J. P., B. Lopez, M. Alvarez, C. Mendoza and T. Lehmann (2005). "Isolation, tissue distribution, and molecular characterization of *Toxoplasma gondii* from free-range chickens from Guatemala." Journal of Parasitology **91**(4): 955-957.

- Dubey, J. P., J. K. Lunney, S. K. Shen, O. C. H. Kwok, D. A. Ashford and P. Thulliez (1996). "Infectivity of low numbers of *Toxoplasma gondii* oocysts to pigs." Journal of Parasitology **82**(3): 438-443.
- Dubey, J. P., M. D. Ruff, O. C. Kwok, S. K. Shen, G. C. Wilkins and P. Thulliez (1993). "Experimental toxoplasmosis in bobwhite quail (*Colinus virginianus*)." The Journal of parasitology **79**(6): 935-939.
- Dubey, J. P., N. Tiao, W. A. Gebreyes and J. L. Jones (2012). "A review of toxoplasmosis in humans and animals in Ethiopia." Epidemiology and Infection **140**(11): 1935-1938.
- Dutil, L., R. Irwin, R. Finley, L. K. Ng, B. Avery, P. Boerlin, A.-M. Bourgault, L. Cole, D. Daignault, A. Desruisseau, W. Demczuk, L. Hoang, G. B. Horsman, J. Ismail, F. Jamieson, A. Maki, A. Pacagnella and D. R. Pillai (2010). "Ceftiofur Resistance in *Salmonella* enterica serovar Heidelberg from Chicken Meat and Humans, Canada." Emerging Infectious Diseases **16**(1): 48-54.
- EC (1999). "Opinion of the scientific committee on veterinary measures relating to public health on *Listeria Monocytogenes*." European Commission.
- EC (2002). "Avian Chlamydiosis as a zoonotic disease and risk reduction strategies." European Commission.
- EC (2003). "Opinion of the Scientific Committee on Veterinary Measures relating to Public Health on Salmonellae in Foodstuffs."
- Edelhofer, R. and H. Prossinger (2010). "Infection with *Toxoplasma gondii* during Pregnancy: Seroepidemiological Studies in Austria." Zoonoses and Public Health **57**(1): 18-26.
- EFSA (2006). "Opinion on 'Migratory birds and their possible role in the spread of highly pathogenic Avian influenza'." The EFSA Journal **357**: 1-46.
- EFSA (2007). "Scientific Opinion of the Panel on Biological Hazards on a request from EFSA on monitoring of verotoxigenic *Escherichia coli* (VTEC) and identification of human pathogenic VTEC types." The EFSA journal **579**: 1-61.
- EFSA (2011). "Scientific Opinion on the public health risks of bacterial strains producing extended spectrum beta lactamases and/or AmpC beta lactamases in food and food producing animals." EFSA Journal **9**(8): 2322.
- EFSA (2012a). "Scientific opinion on the public health hazards to be covered by inspection of meat (poultry)." EFSA journal **10**(6): 2741.
- EFSA (2012b). "The European Union Summary Report on Trends and Sources of Zoonoses, Zoonotic Agents and Food-borne Outbreaks in 2010." EFSA journal **10**(3): 2597.
- EFSA (2013). "The European Union Summary Report on Trends and Sources of Zoonoses, Zoonotic Agents and Food-borne Outbreaks in 2011." EFSA Journal **11**(4): 3129.
- Ejidokun, O. O., A. Walsh, J. Barnett, Y. Hope, S. Ellis, M. W. Sharp, G. A. Paiba, M. Logan, G. A. Willshaw and T. Cheasty (2006). "Human Vero cytotoxigenic *Escherichia coli* (VTEC) O157 infection linked to birds." Epidemiology and Infection **134**(2): 421-423.

- El-Ghareeb, W. R., F. J. M. Smulders, A. M. A. Morshdy, R. Winkelmayr and P. Paulsen (2009). "Microbiological condition and shelf life of meat from hunted game birds." European Journal of Wildlife Research **55**(4): 317-323.
- Elmore, S. A., J. L. Jones, P. A. Conrad, S. Patton, D. S. Lindsay and J. P. Dubey (2010). "*Toxoplasma gondii*: epidemiology, feline clinical aspects, and prevention." Trends in Parasitology **26**(4): 190-196.
- Escudero-Gilete, M. L., M. L. Gonzalez-Miret, R. M. Temprano and F. J. Heredia (2007). "Application of a multivariate concentric method system for the location of *Listeria monocytogenes* in a poultry slaughterhouse." Food Control **18**(1): 69-75.
- Fallacara, D. M., C. M. Monahan, T. Y. Morishita and R. F. Wack (2001). "Fecal shedding and antimicrobial susceptibility of selected bacterial pathogens and a survey of intestinal parasites in free-living waterfowl." Avian Diseases **45**(1): 128-135.
- Farber, J. M. and P. I. Peterkin (1991). "*Listeria monocytogenes*, a food-borne pathogen." Microbiological reviews **55**(3): 476-511.
- Farooq, S., I. Hussain, M. A. Mir, M. A. Bhat and S. A. Wani (2009). "Isolation of atypical enteropathogenic *Escherichia coli* and Shiga toxin 1 and 2f-producing *Escherichia coli* from avian species in India." Letters in Applied Microbiology **48**(6): 692-697.
- Fenlon, D. R. (1985). "Wild birds and silage as reservoirs of *Listeria* in the agricultural environment." The Journal of applied bacteriology **59**(6): 537-543.
- Fernandez, H., W. Gesche, A. Montefusco and R. Schlatter (1996). "Wild birds as reservoir of thermophilic enteropathogenic *Campylobacter* species in Southern Chile." Memorias Do Instituto Oswaldo Cruz **91**(6): 699-700.
- Fosse, J., H. Seegers and C. Magras (2009). "Prevalence and Risk Factors for Bacterial Food-Borne Zoonotic Hazards in Slaughter Pigs: A Review." Zoonoses and Public Health **56**(8): 429-454.
- Franson, J. C. and J. E. Pearson (1995). "PROBABLE EPIZOOTIC CHLAMYDIOSIS IN WILD CALIFORNIA (*LARUS-CALIFORNICUS*) AND RING-BILLED (*LARUS-DELAWARENSIS*) GULLS IN NORTH-DAKOTA." Journal of wildlife diseases **31**(3): 424-427.
- Frenkel, J. K. (1975). "Epidemiology of toxoplasmosis." Journal of the American Veterinary Medical Association **167**(9): 862-862.
- Frenkel, J. K. (1975). "Toxoplasmosis in cats and man." Feline Practice **5**(1): 28-29, 32-33, 36-37, 40-41.
- Frenkel, J. K., A. Ruiz and M. Chinchilla (1975). "Soil survival of toxoplasma oocysts in Kansas and Costa Rica." The American journal of tropical medicine and hygiene **24**(3): 439-443.
- Fricker, C. R. and N. Metcalfe (1984). "*Campylobacters* in wading birds (Charadrii): incidence, biotypes and isolation techniques." Zentralblatt fur Bakteriologie, Mikrobiologie und Hygiene. 1. Abt. Originale B, Hygiene **179**(5): 469-475.
- Friedman, C. R., C. Torigian, P. J. Shillam, R. E. Hoffman, D. Heltzel, J. L. Beebe, G. Malcolm, W. E. DeWitt, L. Hutwagner and P. M. Griffin (1998). "An outbreak of salmonellosis among children attending a reptile exhibit at a zoo." Journal of Pediatrics **132**(5): 802-807.

- FSA (2005). "Determining exposure assessment and modelling risks associated with the preparation of poultry products in institutional catering and the home." http://www.foodbase.org.uk/admintools/reportdocuments/178-1-312_B01015.pdf.
- FSA (2005). "The wild game handling guide: Industry guidance on EU hygiene regulations relating to the supply of wild game for human consumption (outside approved premises)." <http://www.reading.ac.uk/foodlaw/pdf/uk-05048-wild-game-guidance.pdf>.
- FSA (2007). "Review of current information on *Campylobacter* in poultry other than chicken and how this may contribute to human cases."
- FSA (2008). "HACCP Guidance for those producing wild game meat for human consumption either at an approved game handling establishment or under exemption allowed by the food hygiene regulations."
- FSA (2009). "A UK survey of *Campylobacter* and *Salmonella* contamination of fresh chicken at retail sale." Food Survey Information sheet 04/09.
- FSA (2011). "The Wild Game Guide."
- FSANZ (2005). "Scientific Assessment of the public Health and Safety of Poultry Meat in Australia." Food Standards Australia New Zealand.
- FSAS (2012a). "Food Hygiene regulation in the Scottish Wild Game Sector."
- FSAS (2012b). "Habits and behaviours of high-level consumers of lead-shot wild-game meat in Scotland."
- Gale, P., A. Brouwer, V. Ramnial, L. Kelly, R. Kosmider, A. R. Fooks and E. L. Snary (2010). "Assessing the impact of climate change on vector-borne viruses in the EU through the elicitation of expert opinion." Epidemiology and Infection **138**(2): 214-225.
- Galvan-Ramirez, M. L., A. L. Madriz Elisondo, C. P. Rico Torres, H. Luna-Pasten, L. R. Rodriguez Perez, A. R. Rincon-Sanchez, R. Franco, A. Salazar-Montes and D. Correa (2010). "Frequency of *Toxoplasma gondii* in Pork Meat in Ocotlan, Jalisco, Mexico." Journal of Food Protection **73**(6): 1121-1123.
- Gargiulo, A., M. Sensale, L. Marzocco, A. Fioretti, L. F. Menna and L. Dipineto (2011). "*Campylobacter jejuni*, *Campylobacter coli*, and cytolethal distending toxin (CDT) genes in common teals (*Anas crecca*)."
Veterinary Microbiology **150**(3-4): 401-404.
- GFA (2013). "Game Farmers Association." <http://www.gfa.org.uk/>.
- Gillespie, I. A., S. J. O'Brien, J. A. Frost, G. K. Adak, P. Horby, A. V. Swan, M. J. Painter, K. R. Neal and S. Campylobacter Sentinel (2002). "A case-case comparison of *Campylobacter coli* and *Campylobacter jejuni* infection: A tool for generating hypotheses." Emerging Infectious Diseases **8**(9): 937-942.
- Glass, K. A. and M. P. Doyle (1989). "Fate of *Listeria monocytogenes* in processed meat products during refrigerated storage." Applied and Environmental Microbiology **55**(6): 1565-1569.
- Gorman, R., S. Bloomfield and C. C. Adley (2002). "A study of cross-contamination of food-borne pathogens in the domestic kitchen in the Republic of Ireland." International Journal of Food Microbiology **76**(1-2): 143-150.

- Government, T. S. (2011). "Code of practice for the welfare of gamebirds reared for sporting purposes."
- Grossmann, K., B. Weniger, G. Baljer, B. Brenig and L. H. Wieler (2005). "Racing, ornamental and city pigeons carry Shiga toxin producing *Escherichia coli* (STEC) with different Shiga toxin subtypes, urging further analysis of their epidemiological role in the spread of STEC." Berliner Und Munchener Tierarztliche Wochenschrift **118**(11-12): 456-463.
- Guard-Petter, J. (2001). "The chicken, the egg and *Salmonella* Enteritidis." Environmental Microbiology **3**(7): 421-430.
- Guenther, S., C. Ewers and L. H. Wieler (2011). "Extended-spectrum beta-lactamases producing *E. coli* in wildlife, yet another form of environmental pollution?" Frontiers in Microbiology **2**(December): 246- Article 246.
- GWCT (2013). "Game and Wildlife conservation trust." <http://www.gwct.org.uk/>.
- Harris, N. V., N. S. Weiss and C. M. Nolan (1986). "The role of poultry and meats in the etiology of *Campylobacter jejuni/coli* enteritis." American journal of public health **76**(4): 407-411.
- Hartog, B. J., G. J. A. d. Wilde and E. d. Boer (1983). "Poultry as a source of *Campylobacter jejuni*." Archiv fur Lebensmittelhygiene **34**(5): 116-122.
- Havelaar, A. H., J. A. Haagsma, M.-J. J. Manges, J. M. Kemmeren, L. P. B. Verhoef, S. M. C. Vijgen, M. Wilson, I. H. M. Friesema, L. M. Kortbeek, Y. T. H. P. van Duynhoven and W. van Pelt (2012). "Disease burden of foodborne pathogens in the Netherlands, 2009." International Journal of Food Microbiology **156**(3): 231-238.
- Hellstrom, S., K. Kiviniemi, T. Autio and H. Korkeala (2008). "*Listeria monocytogenes* is common in wild birds in Helsinki region and genotypes are frequently similar with those found along the food chain." Journal of Applied Microbiology **104**(3): 883-888.
- Henderson, I. G., Peach, W.J., Baille, S.R. (1993). "The hunting of snipe and woodcock in Europe: a ringing recovery analysis." British Trust of Ornithology Research report No. 115.
- Hennessy, T. W., C. W. Hedberg, L. Slutsker, K. E. White, J. M. BesserWiek, M. E. Moen, J. Feldman, W. W. Coleman, L. M. Edmonson, K. L. MacDonald, M. T. Osterholm, E. Belongia, D. Boxrud, W. Boyer, R. Danila, J. Korlath, F. Leano, W. Mills, J. Soler, M. Sullivan, M. Deling, P. Geisen, C. Kontz, K. Elfering, W. Krueger, T. Masso, M. F. Mitchell, K. Vought, A. Duran, F. Harrell, K. Jirele, A. Krivitsky, H. Manresa, R. Mars, M. Nierman, A. Schwab, F. Sedzielarz, F. Tillman, D. Wagner, D. Wieneke and C. Price (1996). "A national outbreak of *Salmonella* Enteritidis infections from ice cream." New England Journal of Medicine **334**(20): 1281-1286.
- Hill, A. E., S. M. C. Benedetto, C. Coss, J. L. McCrary, V. M. Fournet and J. P. Dubey (2006). "Effects of time and temperature on the viability of *Toxoplasma gondii* tissue cysts in enhanced pork loin." Journal of Food Protection **69**(8): 1961-1965.
- Hoelzer, K., A. I. M. Switt and M. Wiedmann (2011). "Animal contact as a source of human non-typhoidal salmonellosis." Veterinary Research **42**.

- HPA "Health Protection Agency website." <http://www.hpa.org.uk/>.
- HPS (2011). "Health Protection Scotland; Annual data tables for gastrointestinal & zoonoses: <http://www.hps.scot.nhs.uk/giz/annualdatatables.aspx#>."
- Hubalek, Z. (2004). "An annotated checklist of pathogenic microorganisms associated with migratory birds." Journal of Wildlife Diseases **40**(4): 639-659.
- Hughes, L. A., M. Bennett, P. Coffey, J. Elliott, T. R. Jones, R. C. Jones, A. Lahuerta-Marin, A. H. Leatherbarrow, K. McNiffe, D. Norman, N. J. Williams and J. Chantrey (2009). "Molecular Epidemiology and Characterization of *Campylobacter* spp. Isolated from Wild Bird Populations in Northern England." Applied and Environmental Microbiology **75**(10): 3007-3015.
- Hussein, H. S. (2007). "Prevalence and pathogenicity of Shiga toxin-producing *Escherichia coli* in beef cattle and their products." Journal of Animal Science **85**: E63-E72.
- International, B. (2013). "Eurasian Woodcock *Scolopax rusticola*." <http://www.birdlife.org/datazone/speciesfactsheet.php?id=2978>.
- Itoh, T., K. Saito, Y. Yanagawa, S. Sakai and M. Ohashi (1982). "*Campylobacter enteritis* in Tokyo. Includes poultry, swine and cattle." Campylobacter. Epidemiology, pathogenesis and biochemistry: 5-12.
- IUCN (2003). "Use of wild living resources in the United Kingdom - A review." <http://www.iucn-uk.org/portals/0/reports/livingresources.pdf>.
- Jackson, S. F. (2004). "Monitoring Methods for Non-Breeding Snipe." BTO Research paper No. 355.
- Jemmi, T. and R. Stephan (2006). "*Listeria monocytogenes*: food-borne pathogen and hygiene indicator." Revue Scientifique Et Technique-Office International Des Epizooties **25**(2): 571-580.
- Johnson, J. R., M. R. Sannes, C. Croy, B. Johnston, C. Clabots, M. A. Kuskowski, J. Bender, K. E. Smith, P. L. Winokur and E. A. Belongia (2007). "Antimicrobial drug-resistant *Escherichia coli* from humans and poultry products, Minnesota and Wisconsin, 2002-2004." Emerging Infectious Diseases **13**(6): 838-846.
- Johnston, W. B., M. Eidson, K. A. Smith, M. G. Stobierski and C. Psittacosis Compendium (1999). "Compendium of chlamydiosis (psittacosis) control, 1999." Journal of the American Veterinary Medical Association **214**(5): 640-646.
- Jones, J. L., V. Dargelas, J. Roberts, C. Press, J. S. Remington and J. G. Montoya (2009). "Risk Factors for *Toxoplasma gondii* Infection in the United States." Clinical Infectious Diseases **49**(6): 878-884.
- Jones, S. L., S. M. Parry, S. J. O'Brien and S. R. Palmeri (2008). "Are staff management practices and inspection risk ratings associated with foodborne disease outbreaks in the catering industry in England and Wales?" Journal of Food Protection **71**(3): 550-557.
- Kabir, S. M. L. (2010). "Avian Colibacillosis and Salmonellosis: A Closer Look at Epidemiology, Pathogenesis, Diagnosis, Control and Public Health Concerns." International Journal of Environmental Research and Public Health **7**(1): 89-114.

- Kaleta, E. F. and E. M. A. Taday (2003). "Avian host range of *Chlamydophila* spp. based on isolation, antigen detection and serology." Avian Pathology **32**(5): 435-462.
- Kapperud, G., S. Gustavsen, I. Hellesnes, A. H. Hansen, J. Lassen, J. Hirn, M. Jahkola, M. A. Montenegro and R. Helmuth (1990). "Outbreak of *Salmonella* Typhimurium infection traced to contaminated chocolate and caused by a strain lacking the 60-megadalton virulence plasmid." Journal of clinical microbiology **28**(12): 2597-2601.
- Kapperud, G., P. A. Jennum, B. StrayPedersen, K. K. Melby, A. Eskild and J. Eng (1996). "Risk factors for *Toxoplasma gondii* infection in pregnancy - Results of a prospective case-control study in Norway." American Journal of Epidemiology **144**(4): 405-412.
- Kinjo, T., M. Morishige, N. Minamoto and H. Fukushi (1983). "Prevalence of *Campylobacter jejuni* in feral pigeons." Nihon juigaku zasshi. The Japanese journal of veterinary science **45**(6): 833-835.
- Kobayashi, H., M. Kanazaki, E. Hata and M. Kubo (2009). "Prevalence and Characteristics of eae- and stx-Positive Strains of *Escherichia coli* from Wild Birds in the Immediate Environment of Tokyo Bay." Applied and Environmental Microbiology **75**(1): 292-295.
- Kobayashi, H., M. Kanazaki, Y. Shimizu, H. Nakajima, M. M. Khatun, E. Hata and M. Kubo (2007). "Salmonella isolates from cloacal swabs and footpads of wild birds in the immediate environment of Tokyo Bay." Journal of Veterinary Medical Science **69**(3): 309-311.
- Kosmider, R. D., P. Nally, R. R. L. Simons, A. Brouwer, S. Cheung, E. L. Snary and M. Wooldridge (2010). "Attribution of Human VTEC O157 Infection from Meat Products: A Quantitative Risk Assessment Approach." Risk Analysis **30**(5): 753-765.
- Kothary, M. H. and U. S. Babu (2001). "Infective dose of foodborne pathogens in volunteers: A review." Journal of Food Safety **21**(1): 49-73.
- Kotula, A. W., J. P. Dubey, A. K. Sharar, C. D. Andrews, S. K. Shen and D. S. Lindsay (1991). "Effect of freezing on infectivity of *Toxoplasma gondii* tissue cysts in pork." Journal of Food Protection **54**(9): 687-690.
- Kotula, K. L. and Y. Pandya (1995). "Bacterial contamination of broiler chickens before scalding." Journal of Food Protection **58**(12): 1326-1329.
- Kraft, A. (1992). "Psychotropic bacteria in foods."
- Labbe, R. G. and S. Garcia (2001). Guide to foodborne pathogens.
- Lautenbach, E., E. Santana, A. Lee, P. Tolomeo, N. Black, A. Babson, E. N. Perencevich, A. D. Harris, C. A. Smith and J. Maslow (2008). "Efficient recovery of fluoroquinolone-susceptible and fluoroquinolone-resistant *Escherichia coli* strains from frozen samples." Infection Control and Hospital Epidemiology **29**(4): 367-369.
- Lavilla, S., J. J. Gonzalez-Lopez, E. Miro, A. Dominguez, M. Llagostera, R. M. Bartolome, B. Mirelis, F. Navarro and G. Prats (2008). "Dissemination of extended-spectrum beta-lactamase-producing bacteria: the food-borne outbreak lesson." Journal of Antimicrobial Chemotherapy **61**(6): 1244-1251.
- Lecocq, Y. (1997). "A European perspective on wild game meat and public health." Revue Scientifique Et Technique De L Office International Des Epizooties **16**(2): 579-585.

- Lee, S.-E., N.-H. Kim, H.-S. Chae, S.-H. Cho, H.-W. Nam, W.-J. Lee, S.-H. Kim and J.-H. Lee (2011). "Prevalence of *Toxoplasma gondii* Infection in Feral Cats in Seoul, Korea." Journal of Parasitology **97**(1): 153-155.
- Lehmacher, A., J. Bockemuhl and S. Aleksic (1995). "Nationwide outbreak of human salmonellosis in Germany due to contaminated paprika and paprika-powdered potato chips." Epidemiology and Infection **115**(3): 501-511.
- Leverstein-van Hall, M. A., C. M. Dierikx, J. C. Stuart, G. M. Voets, M. P. van den Munckhof, A. van Essen-Zandbergen, T. Platteel, A. C. Fluit, N. van de Sande-Bruinsma, J. Scharinga, M. J. M. Bonten, D. J. Mevius and E. S. G. Natl (2011). "Dutch patients, retail chicken meat and poultry share the same ESBL genes, plasmids and strains." Clinical Microbiology and Infection **17**(6): 873-880.
- Literak, I., K. Hejlícek, J. Nezval and C. Folk (1992). "Incidence of *Toxoplasma gondii* in populations of wild birds in the Czech Republic." Avian Pathology **21**(4): 659-665.
- Little, C. L., J. F. Richardson, R. Owen, L. R. Ward, E. de Pinna and J. Threlfall (2005). "Report on the two year monitoring study of pathogens in raw meats, 2003-5." <http://www.food.gov.uk/multimedia/pdfs/committee/acm872report.pdf>.
- Luechtefeld, N. A., M. J. Blaser, L. B. Reller and W. L. Wang (1980). "Isolation of *Campylobacter fetus* subsp. *jejuni* from migratory waterfowl." Journal of clinical microbiology **12**(3): 406-408.
- Lunden, A. and A. Uggla (1992). "Infectivity of *Toxoplasma gondii* in mutton following curing, smoking, freezing or microwave cooking." International Journal of Food Microbiology **15**(3-4): 357-363.
- Lunden, J., J. Bjorkroth and H. Korkeala (2005). Contamination routes and analysis in food processing environments.
- Mancianti, F., S. Nardoni, L. Mugnaini and A. Poli (2012). "*Toxoplasma gondii* in Waterfowl: The First Detection of this Parasite in *Anas crecca* and *Anas clypeata* From Italy " Journal of Parasitology **epub ahead of print. doi: 10.1645/12-34.1**.
- Manges, A. R. and J. R. Johnson (2012). "Food-Borne Origins of *Escherichia coli* Causing Extraintestinal Infections." Clinical Infectious Diseases **55**(5): 712-719.
- McClure, P. J., A. L. Beaumont, J. P. Sutherland and T. A. Roberts (1997). "Predictive modelling of growth of *Listeria monocytogenes* - The effects on growth of NaCl, pH, storage temperature and NaNO₂." International Journal of Food Microbiology **34**(3): 221-232.
- McCrea, B. A., K. H. Tonooka, C. VanWorth, C. L. Boggs, R. Atwill and J. S. Schrader (2006). "Prevalence of *Campylobacter* and *Salmonella* species on farm, after transport, and at processing in specialty market poultry." Poultry Science **85**(1): 136-143.
- McCurdy, S. M., M. T. Takeuchi, Z. M. Edwards, M. Edlefsen, D.-H. Kang, V. E. Mayes and V. N. Hillers (2006). "Food safety education initiative to increase consumer use of food thermometers in the United States." British Food Journal **108**(9): 775-794.
- McLauchlin, J. (1990). "Human listeriosis in Britain, 1967-85, a summary of 722 cases. 2. Listeriosis in non-pregnant individuals, a changing

- pattern of infection and seasonal incidence." Epidemiology and Infection **104**(2): 191-201.
- McOrist, S. (1992). "Diseases of the European wildcat (*Felis silvestris* Schreber, 1777) in Great Britain." Revue scientifique et technique (International Office of Epizootics) **11**(4): 1143-1149.
- Mead, G. C., E. M. Barnes and C. S. Impey (1974). "Microbiological changes in the uneviscerated bird hung at 10 degrees C with particular reference to the pheasant." British Poultry Science **15**(4): 381-390.
- Mead, G. C., A. M. Chamberlain and E. D. Borland (1973). "Microbial changes leading to the spoilage of hung pheasants, with special reference to the clostridia." The Journal of applied bacteriology **36**(2): 279-287.
- Mead, G. C. and M. J. Scott (1997). "Spread of an enteric 'marker' organism during evisceration of New York dressed poultry in a simulated kitchen environment." British Poultry Science **38**(2): 195-198.
- Mead, P. S., L. Slutsker, V. Dietz, L. F. McCaig, J. S. Bresee, C. Shapiro, P. M. Griffin and R. V. Tauxe (1999). "Food-related illness and death in the United States." Emerging Infectious Diseases **5**(5): 607-625.
- Meyer, K. F. (1955). "Problems in the control of psittacosis and ornithosis." Proceedings 92nd Annu. Meet. Amer. vet. med. Ass., 1955: 412-419.
- Middleton, J. H. and A. Ambrose (2005). "Enumeration and antibiotic resistance patterns of fecal indicator organisms isolated from migratory Canada geese (*Branta canadensis*)." Journal of wildlife diseases **41**(2): 334-341.
- Millan, J., O. Cabezon, M. Pabon, J. P. Dubey and S. Almeria (2009). "Seroprevalence of *Toxoplasma gondii* and *Neospora caninum* in feral cats (*Felis silvestris catus*) in Majorca, Balearic Islands, Spain." Veterinary Parasitology **165**(3-4): 323-326.
- Mintel (2008). "Poultry and Game Meat UK."
- Mitchell, T. R. and T. Ridgwell (1971). "The frequency of salmonellae in wild ducks." Journal of medical microbiology **4**(3): 359-361.
- Morabito, S., G. Dell'Omo, U. Agrimi, H. Schmidt, H. Karch, T. Cheasty and A. Caprioli (2001). "Detection and characterization of Shiga toxin-producing *Escherichia coli* in feral pigeons." Veterinary Microbiology **82**(3): 275-283.
- Mulder, R. W. A. W. (2004). Managing the safety and quality of poultry meat.
- Murphy, R. Y., B. P. Marks, E. R. Johnson and M. G. Johnson (1999). "Inactivation of *Salmonella* and *Listeria* in ground chicken breast meat during thermal processing." Journal of Food Protection **62**(9): 980-985.
- Murphy, R. Y., T. Osaili, L. K. Duncan and J. A. Marcy (2004). "Thermal inactivation of *Salmonella* and *Listeria monocytogenes* in ground chicken thigh/leg meat and skin." Poultry Science **83**(7): 1218-1225.
- Murray, C. J. (2000). Environmental aspects of Salmonella.
- NDNS (2010). "National Diet and Nutrition Survey."
- Nesbitt, A., A. Ravel, R. Murray, R. McCormick, C. Savelli, R. Finley, J. Parmley, A. Agunos, S. E. Majowicz, M. Gilmour, A. Canadian Integrated Program and N. Canadian Public Hlth Lab (2012). "Integrated surveillance and potential sources of *Salmonella* Enteritidis in human cases in Canada from 2003 to 2009." Epidemiology and Infection **140**(10): 1757-1772.

- Newell, D. G. (2002). "The ecology of *Campylobacter jejuni* in avian and human hosts and in the environment." International journal of Infectious Diseases **6**(3): S16-S21.
- Newell, D. G. and C. Fearnley (2003). "Sources of *Campylobacter* colonization in broiler chickens." Applied and Environmental Microbiology **69**(8): 4343-4351.
- Newman, C. P., S. R. Palmer, F. D. Kirby and E. O. Caul (1992). "A prolonged outbreak of ornithosis in duck processors." Epidemiology and Infection **108**(1): 203-210.
- Nickell, L. G. (1959). "Antimicrobial activity of vascular plants." Economic Botany **13**(4): 281-318.
- Obiri-Danso, K. and K. Jones (1999). "Distribution and seasonality of microbial indicators and thermophilic campylobacters in two freshwater bathing sites on the River Lune in northwest England." Journal of Applied Microbiology **87**(6): 822-832.
- OIE (2004). "Handbook on Import Risk Analysis for Animals and Animal Products." Paris, France:OIE.
- Oliver, S. P., S. E. Murinda, L. T. Nguyen, H. M. Nam, R. A. Almeida and S. J. Headrick (2005). On-farm sources of foodborne pathogens: isolation from the dairy farm environment.
- Oscar, T. P. (2002). "Development and validation of a tertiary simulation model for predicting the potential growth of *Salmonella* Typhimurium on cooked chicken." International Journal of Food Microbiology **76**(3): 177-190.
- Oscar, T. P. (2009). "Predictive Model for Survival and Growth of *Salmonella* Typhimurium DT104 on Chicken Skin during Temperature Abuse." Journal of Food Protection **72**(2): 304-314.
- PACEC (2006). "The Economic and Environmental impact of sporting shooting." <http://www.shootingfacts.co.uk/pdf/pacecmainreport.pdf>.
- Page, L. A. (1959). "Thermal inactivation studies on a turkey ornithosis virus." Avian Diseases **3**: 67-79.
- Pain, D. J., R. L. Cromie, J. Newth, M. J. Brown, E. Crutcher, P. Hardman, L. Hurst, R. Mateo, A. A. Meharg, A. C. Moran, A. Raab, M. A. Taggart and R. E. Green (2010). "Potential Hazard to Human Health from Exposure to Fragments of Lead Bullets and Shot in the Tissues of Game Animals." Plos One **5**(4).
- Paulsen, P., J. Nagy, P. Popelka, V. Ledecy, S. Marcincak, M. Pipova, F. J. M. Smulders, P. Hofbauer, P. Lazar and Z. Dicakova (2008). "Influence of storage conditions and shotshell wounding on the hygienic condition of hunted, uneviscerated pheasant (*Phasianus colchicus*)." Poultry Science **87**(1): 191-195.
- Pedersen, K., L. Clark, W. F. Andelt and M. D. Salman (2006). "Prevalence of shiga toxin-producing *Escherichia coli* and *Salmonella* enterica in rock pigeons captured in fort Collins, Colorado." Journal of wildlife diseases **42**(1): 46-55.
- Petersen, L., E. M. Nielsen, J. Engberg, S. L. W. On and H. H. Dietz (2001). "Comparison of genotypes and serotypes of *Campylobacter jejuni* isolated from Danish wild mammals and birds and from broiler flocks and humans." Applied and Environmental Microbiology **67**(7): 3115-3121.

- ProMed (2013). "Psittacosis in Sweden." <http://www.promedmail.org/direct.php?id=20130322.1599238>.
- Quessy, S. and S. Messier (1992). "Prevalence of *Salmonella* spp., *Campylobacter* spp. and *Listeria* spp. in ring-billed gulls (*Larus delawarensis*)." Journal of Wildlife Diseases **28**(4): 526-531.
- Rice, D. H., D. D. Hancock and I. E. Besser (2003). "Faecal culture of wild animals for *Escherichia coli* O157 : H7." Veterinary Record **152**(3): 82-83.
- Riordan, T., T. J. Humphrey and A. Fowles (1993). "A point source outbreak of campylobacter infection related to bird-pecked milk." Epidemiology and Infection **110**(2): 261-265.
- Roberts, J. P. and J. E. Grimes (1978). "Chlamydia shedding by four species of wild birds." Avian Diseases **22**(4): 698-706.
- Rosenquist, H., N. L. Nielsen, H. M. Sommer, B. Norrung and B. B. Christensen (2003). "Quantitative risk assessment of human campylobacteriosis associated with thermophilic *Campylobacter* species in chickens." International Journal of Food Microbiology **83**(1): 87-103.
- Roy, P., V. Purushothaman, A. Koteeswaran and A. S. Dhillon (2006). "Isolation, characterization, and antimicrobial drug resistance pattern of *Escherichia coli* isolated from Japanese quail and their environment." Journal of Applied Poultry Research **15**(3): 442-446.
- RSPB (2013). "RSPB bird guide." <http://www.rspb.org.uk/wildlife/birdguide/>.
- Ryan, M. J., P. G. Wall, R. J. Gilbert, M. Griffin and B. Rowe (1996). "Risk factors for outbreaks of infectious intestinal disease linked to domestic catering." Communicable disease report. CDR review **6**(13): R179-183.
- Santaniello, A., A. Gargiulo, L. Borrelli, L. Dipineto, A. Cuomo, M. Sensale, M. Fontanella, M. Calabria, V. Musella, L. F. Menna and A. Fioretti (2007). "Survey of Shiga toxin-producing *Escherichia coli* O157 : H7 in urban pigeons (*Columba livia*) in the city of Napoli, Italy." Italian Journal of Animal Science **6**(3): 313-316.
- Schultsz, C. and S. Geerlings (2012). "Plasmid-Mediated Resistance in Enterobacteriaceae Changing Landscape and Implications for Therapy." Drugs **72**(1): 1-16.
- Sedlak, K., I. Literak, F. Vitula and J. Benaak (2000). "High susceptibility of partridges (*Perdix perdix*) to toxoplasmosis compared with other gallinaceous birds." Avian Pathology **29**(6): 563-569.
- Shaapan, R. M., F. A. M. Khalil and M. T. A. E. Nadia (2011). "Cryptosporidiosis and Toxoplasmosis in native quails of Egypt." Research Journal of Veterinary Sciences **4**(2): 30-36.
- Sharples, E. and S. J. Baines (2009). "Prevalence of *Chlamydophila psittaci*-positive cloacal PCR tests in wild avian casualties in the UK." Veterinary Record **164**(1): 16-17.
- Sheppard, S. K., J. F. Dallas, M. MacRae, N. D. McCarthy, E. L. Sproston, F. J. Gormley, N. J. C. Strachan, I. D. Ogden, M. C. J. Maiden and K. J. Forbes (2009). "*Campylobacter* genotypes from food animals, environmental sources and clinical disease in Scotland 2005/6." International Journal of Food Microbiology **134**(1-2): 96-103.
- Sjolund, M., J. Bonnedahl, J. Hernandez, S. Bengtsson, G. Cederbrant, J. Pinhassi, G. Kahlmeter and B. Olsen (2008). "Dissemination of

- multidrug-resistant bacteria into the Arctic." Emerging Infectious Diseases **14**(1): 70-72.
- Skjerve, E., H. Waldeland, T. Nesbakken and G. Kapperud (1998). "Risk factors for the presence of antibodies to *Toxoplasma gondii* in Norwegian slaughter lambs." Preventive Veterinary Medicine **35**(3): 219-227.
- Skurnik, D., R. Ruimy, A. Andremont, C. Amorin, P. Rouquet, B. Picard and E. Denamur (2006). "Effect of human vicinity on antimicrobial resistance and integrons in animal faecal *Escherichia coli*." Journal of Antimicrobial Chemotherapy **57**(6): 1215-1219.
- Smadi, H. and J. M. Sargeant (2013). "Quantitative Risk Assessment of Human Salmonellosis in Canadian Broiler Chicken Breast from Retail to Consumption." Risk Analysis **33**(2): 232-248.
- Smith, D. P. and M. E. Berrang (2006). "Prevalence and numbers of bacteria in broiler crop and gizzard contents." Poultry Science **85**(1): 144-147.
- Sojka, W. J., C. Wray, E. B. Hudson and J. A. Benson (1975). "Incidence of *Salmonella* infection in animals in England and Wales, 1968-73." Veterinary Record **96**(13): 280-284.
- Sonntag, A. K., E. Zenner, H. Karch and M. Bielaszewska (2005). "Pigeons as a possible reservoir of Shiga toxin 2f-producing *Escherichia coli* pathogenic to humans." Berliner Und Munchener Tierarztliche Wochenschrift **118**(11-12): 464-470.
- Soultos, N., P. Koidis and R. H. Madden (2003). "Presence of *Listeria* and *Salmonella* spp. in retail chicken in Northern Ireland." Letters in Applied Microbiology **37**(5): 421-423.
- Stern, N. J., M. R. S. Clavero, J. S. Bailey, N. A. Cox and M. C. Robach (1995). "CAMPYLOBACTER SPP IN BROILERS ON THE FARM AND AFTER TRANSPORT." Poultry Science **74**(6): 937-941.
- Stern, N. J., C. E. Lyon and M. T. Musgrove (1995). "Bacterial quality of broilers and alternative processing procedures." Journal of Applied Poultry Research **4**(2): 164-169.
- Stojanovic, V. and P. Foley (2011). "Infectious disease prevalence in a feral cat population on Prince Edward Island, Canada." Canadian Veterinary Journal-Revue Veterinaire Canadienne **52**(9): 979-982.
- Stringer, S. C., S. M. George and M. W. Peck (2000). "Thermal inactivation of *Escherichia coli* O157:H7." Symposium series (Society for Applied Microbiology)(29): 79S-89S.
- Takahashi, T., I. Takashima and N. Hashimoto (1988). "Shedding and transmission of *Chlamydia psittaci* in experimentally infected chickens." Avian Diseases **32**(4): 650-658.
- Tam, C. C., L. C. Rodrigues, L. Viviani, J. P. Dodds, M. R. Evans, P. R. Hunter, J. J. Gray, L. H. Letley, G. Rait, D. S. Tompkins, S. J. O'Brien and I. I. D. S. E. Comm (2012). "Longitudinal study of infectious intestinal disease in the UK (IID2 study): incidence in the community and presenting to general practice." Gut **61**(1): 69-77.
- Tanaka, C., T. Miyazawa, M. Watarai and N. Ishiguro (2005). "Bacteriological survey of feces from feral pigeons in Japan." Journal of Veterinary Medical Science **67**(9): 951-953.
- Tapper, S. C. (1992). "Game Heritage: an ecological review from shooting and gamekeeping records."

- Thevenot, D., A. Dernburg and C. Vernozy-Rozand (2006). "An updated review of *Listeria monocytogenes* in the pork meat industry and its products." Journal of Applied Microbiology **101**(1): 7-17.
- Tizard, I. (2004). "Salmonellosis in wild birds." Seminars in Avian and Exotic Pet Medicine **13**(2): 50-66.
- Vaillant, V., H. de Valk, E. Baron, T. Ancelle, P. Colin, M. C. Delmas, B. Dufour, R. Pouillot, Y. Le Strat, P. Weinbreck, E. Jougla and J. C. Desenclos (2005). "Foodborne infections in France." Foodborne Pathogens and Disease **2**(3): 221-232.
- Vanrompay, D., J. Mast, R. Ducatelle, F. Haesebrouck and B. Goddeeris (1995). "*Chlamydia psittaci* in turkeys: Pathogenesis of infections in avian serovars A, B and D." Veterinary Microbiology **47**(3-4): 245-256.
- Vasquez, B., F. Esperon, E. Neves, J. Lopez, C. Ballesteros and M. Jesus Munoz (2010). "Screening for several potential pathogens in feral pigeons (*Columba livia*) in Madrid." Acta Veterinaria Scandinavica **52**(45).
- Vazquez, B., F. Esperon, E. Neves, J. Lopez, C. Ballesteros and M. Jesus Munoz (2010). "Screening for several potential pathogens in feral pigeons (*Columba livia*) in Madrid." Acta Veterinaria Scandinavica **52**.
- Villanua, D., L. Perez-Rodriguez, F. Casas, V. Alzaga, P. Acevedo, J. Vinuela and C. Gortazar (2008). "Sanitary risks of red-legged partridge releases: introduction of parasites." European Journal of Wildlife Research **54**(2): 199-204.
- Vincent, C., P. Boerlin, D. Daignault, C. M. Dozois, L. Dutil, C. Galanakis, R. J. Reid-Smith, P.-P. Tellier, P. A. Tellis, K. Ziebell and A. R. Manges (2010). "Food Reservoir for *Escherichia coli* Causing Urinary Tract Infections." Emerging Infectious Diseases **16**(1): 88-95.
- Waap, H., R. Cardoso, A. Leitao, T. Nunes, A. Vilares, M. J. Gargate, J. Meireles, H. Cortes and H. Angelo (2012). "In vitro isolation and seroprevalence of *Toxoplasma gondii* in stray cats and pigeons in Lisbon, Portugal." Veterinary parasitology **187**(3-4): 542-547.
- Wainwright, K. E., M. Lagunas-Solar, M. A. Miller, B. C. Barr, I. A. Gardner, C. Pina, A. C. Melli, A. E. Packham, N. Zeng, T. Truong and P. A. Conrad (2007). "Physical inactivation of *Toxoplasma gondii* oocysts in water." Applied and Environmental Microbiology **73**(17): 5663-5666.
- Waldenstrom, J., T. Broman, I. Carlsson, D. Hasselquist, R. P. Achterberg, J. A. Wagenaar and B. Olsen (2002). "Prevalence of *Campylobacter jejuni*, *Campylobacter lari*, and *Campylobacter coli* in different ecological guilds and taxa of migrating birds." Applied and Environmental Microbiology **68**(12): 5911-5917.
- Wallace, J. S., T. Cheasty and K. Jones (1997). "Isolation of Vero cytotoxin-producing *Escherichia coli* O157 from wild birds." Journal of Applied Microbiology **82**(3): 399-404.
- Wassenaar, T. M., B. A. van der Zeijst, R. Ayling and D. G. Newell (1993). "Colonization of chicks by motility mutants of *Campylobacter jejuni* demonstrates the importance of flagellin A expression." Journal of general microbiology **139 Pt 6**: 1171-1175.
- Wasteson, Y. (2001). "Zoonotic *Escherichia coli*." Acta veterinaria Scandinavica. Supplementum **95**: 79-84.
- Weis, J. and H. P. Seeliger (1975). "Incidence of *Listeria monocytogenes* in nature." Applied microbiology **30**(1): 29-32.

- Wheeler, J. G., D. Sethi, J. M. Cowden, P. G. Wall, L. C. Rodrigues, D. S. Tompkins, M. J. Hudson, P. J. Roderick and E. Infect Intestinal Dis Study (1999). "Study of infectious intestinal disease in England: rates in the community, presenting to general practice, and reported to national surveillance." British Medical Journal **318**(7190): 1046-1050.
- White, C. H., L (1984). "The effect of temperature abuse on *Staphylococcus aureus* and salmonellae in raw beef and chicken substrates during frozen storage." Food Microbiology **1**: 29-38.
- WHO (2002). "Risk assessments of *Salmonella* in eggs and broiler chickens." World Health Organisation.
- WHO (2009). "Risk Assessment of *Campylobacter* spp. in broiler chickens."
- Workman, S. N., G. E. Mathison and M. C. Lavoie (2005). "Pet dogs and chicken meat as reservoirs of *Campylobacter* spp. in Barbados." Journal of clinical microbiology **43**(6): 2642-2650.
- Worsfold, D. and C. J. Griffith (1997). "Assessment of the standard of consumer food safety behavior." Journal of Food Protection **60**(4): 399-406.
- Yoshida, T., T. Sugimoto, M. Sato and K. Hirai (2000). "Incidence of *Listeria monocytogenes* in wild animals in Japan." Journal of Veterinary Medical Science **62**(6): 673-675.
- Young, I., A. Rajic, B. J. Wilhelm, L. Waddell, S. Parker and S. A. McEwen (2009). "Comparison of the prevalence of bacterial enteropathogens, potentially zoonotic bacteria and bacterial resistance to antimicrobials in organic and conventional poultry, swine and beef production: a systematic review and meta-analysis." Epidemiology and Infection **137**(9): 1217-1232.
- Zhao, P., T. Zhao, M. P. Doyle, J. R. Rubino and J. Meng (1998). "Development of a model for evaluation of microbial cross-contamination in the kitchen." Journal of Food Protection **61**(8): 960-963.
- Ziino, G., A. Giuffrida, C. Passafaro and A. Panebianco (2008). "Survey on enteric contamination in New York Dressed and eviscerated chicken during storage." Archiv fur Lebensmittelhygiene **59**(4): 124-129.

9. Appendix 3: Full List of Hazards

Key:

Not zoonotic
Low incidence, only potential zoonotic or not present in the UK
Contact transmission
Short listed

Hazard	Clinical signs	Species affected	Level of infection	Zoonotic Transmission	Present in GB or last occurred	Human cases	Human symptoms	Reference:
<i>Bacteria</i>								
<i>Aeromonas hydrophila</i>	conjunctivitis, sudden death	Rare but waterfowl can be affected	Low	Food borne transmission	Present	>500 cases / year (1998 data HPA)	Gastroenteritis	Janda and Abbott 2010
<i>Arcobacter</i> spp.	Subclinical	Chickens, turkeys, ducks	15% natural infection chickens	Potential zoonosis	Present	Increasing	Enteritis	Collado 2011, Ho 2006
<i>Avibacterium paragallinarum</i> (<i>Haemophilus paragallinarum</i>) (Infectious Coryza)	facial and wattle swelling, sneezing, inappetance	Chicken, pheasants, guinea fowl	High morbidity	Not zoonotic	Present	Not zoonotic	Not zoonotic	VLA website, Welchman 2010
<i>Bacillus cereus</i>	Apparently healthy	Chickens, game birds	Ubiquitous	Food borne transmission	Present	11 outbreaks affecting 104 people 2000 - 2010 (HPA data)	Diarrhoea, vomiting	HPA website

Hazard	Clinical signs	Species affected	Level of infection	Zoonotic Transmission	Present in GB or last occurred	Human cases	Human symptoms	Reference:
<i>Borrelia anserina</i> (Spirochaetosis)	Cyanosis, thirst, weakness, depression	Chicken, turkeys, ducks, pheasants, grouse	Up to 100% morbidity and mortality	Contact transmission (vector borne)	Not present in the UK	Not present in the UK	Relapsing fever	Porcella 2000
<i>Brucella</i>	Often subclinical or enteritis/diarrhoea	Seagulls (experimentally: pigeons, pheasants, ducks, geese)	Low	Currently predominantly via contact transmission. Food borne is only via raw milk/milk products	Cattle 1993	7 cases of brucellosis in 2009 ~ all non-UK nationality and not linked to an avian food source	Flu like symptoms, infection of CNS & heart	MacDiarmid 1983
<i>Burkholderia pseudomallei</i>	Melioidosis, can be fatal in exotics. Native birds are often unsusceptible	Documented mostly in captive exotic birds	Unknown in wild birds	Potential zoonosis	Not present in UK	Not present in the UK	Acute pulmonary infection	Hampton 2011
<i>Campylobacter</i> spp.	Apparently healthy	Pheasants, woodpigeons	12 - 28% pheasants, 12.5 -54% woodpigeons. Medium to high prevalence but higher if industrially reared	Food borne transmission	Present	70,298 laboratory confirmed cases 2010 (58 HPA reported outbreaks associated with poultry meat 1992-2010)	Diarrhoea/sickness. UK confirmed cases of 113/100,000 people with EU fatality rate of 0.22% for 2010)	Reich 2008
<i>Chlamydia psittaci</i>	Subclinical or scour	Ducks, pigeons, pheasant. Most common in psittacines	29% gamebirds, 47.3% pigeons (Bracewell & Bevan 1986)	Contact transmission (Respiratory/faeces)	Present	Likely to be underdiagnosed. 61 cases 2008 (HPA) largely due to domestic bird contact and declining since 2000	Flu like symptoms	Magnino 2009, Coburn 2003
<i>Clostridium botulinum</i> (Mostly Type C) poison	Paralysis of muscles	More common in aquatic birds, ducks. Also pheasants, chickens	15% mortality during outbreaks	Food borne transmission-	Present	Few cases reported in USA from consumption of fish. No cases since 2005 UK.	Botulism - weakness, respiratory failure	Jones 1996, Merck Vet manual

Hazard	Clinical signs	Species affected	Level of infection	Zoonotic Transmission	Present in GB or last occurred	Human cases	Human symptoms	Reference:
<i>Clostridium colinum</i>	Ulcerative enteritis, diarrhoea death	Captive quail mostly, also grouse, pheasant, partridges (red legged), pigeons.	Can be 100% mortality in captive quail.	Not zoonotic	Present	Not zoonotic	Not zoonotic	Beltran-Alcrudo 2008
<i>Clostridium difficile</i>	Apparently healthy	Chickens	0% in migratory passerines. 100% chickens	Potential zoonosis	Present	No direct evidence of zoonosis but has zoonotic potential	diarrhoea, colitis	Bandelj 2011, Hensgens 2012
<i>Clostridium perfringens</i>	Necrotic enteritis, rapid death	Mostly waterfowl, psittacine, chickens, wild birds	Upto 52% in commercial chickens	Food borne transmission	Present	53 cases from 3 outbreaks 2010	Gastroenteritis	Craven 2000
<i>Coxiella burnetii</i> (Q fever)	Subclinical	Pigeons, chickens, ducks, geese	Rare in birds	Contact transmission Aerosol (dried faeces)/foodborne via raw eggs/tick bites	Present	Rare from birds	flu-like symptoms	Stein 1999
<i>Escherichia coli</i> (toxicoinfectious strains including VTEC)	Can cause respiratory disease/diarrhoea/lameness or subclinical	All birds	Low 0.34% (Rice 2003)	Food borne transmission	Present	1182 laboratory confirmed cases 2011 (no specific data for game bird origin)	Diarrhoea/sickness	Dell'Omo 1998 Cizek 1999 Rice 2003
<i>Escherichia coli</i> (with Extended Spectrum Beta-Lactamases)	Subclinical	Chickens, turkeys, pigeons, waterfowl	Increasing	Food borne transmission	Present	Approximately 9-19% of reports of <i>E. coli</i> bacteraemia were non-susceptible to antibiotics.	Urinary tract infections. Can be fatal in elderly or immunocompromised hosts	HPA website, EFSA 2012, Overdeest 2011, Randall 2011
<i>Enterococcus</i> spp.	Enteric infections, septicemia, bacterial endocarditis	Chickens, ducks	High mortality in ducklings	Potential zoonosis	Present	Approximately 5,500 reports to HPA in 2010 mostly in children and the elderly	Urinary tract infections. Can cause bacteremia leading to endocarditis	Merck Vet manual, Sundsfjord 2001

Hazard	Clinical signs	Species affected	Level of infection	Zoonotic Transmission	Present in GB or last occurred	Human cases	Human symptoms	Reference:
<i>Erysipelothrix rhusiopathiae</i>	Sudden death	Ducks, wood pigeon, pheasant, quail	Rare in ducks and wood pigeons, occasional in pheasant and quail	Contact transmission	Present	Rare	Erysipelas, skin lesions	Twycross zoo website
<i>Helicobacter canadensis</i>	Apparently healthy	Wild geese	Wide distribution	Potential zoonosis	Present	Low - emerging pathogen	Diarrhoea	Fox 2000, Waldenstrom 2003
<i>Helicobacter pullorum</i>	Apparently healthy	Chickens	Wide distribution	Potential zoonosis	Present	Low - 4.3% in one study (Ceelen 2005)	Gastroenteritis, hepatic conditions	Fox 2000, Waldenstrom 2003
<i>Listeria monocytogenes</i>	Septicaemia, incoordination	Red legged partridges, duck, grouse, pigeon	5.2% loss described as rare	Foodborne transmission	Present	139 documented cases 2010 (HPA)	Flu-like symptoms, gastroenteritis	Gray 1958; McDiarmid 1961; AHVLA 2011
<i>Mycobacterium avium</i>	Weight loss/diarrhoea	All game birds especially red legged partridges and pheasants	2-4% woodpigeons, 2.5-89% captive pheasants. Prevalence in wild bird population is likely to be low.	Contact transmission (respiratory)	Present	Rare- primarily affects immunocompromised patients 1/100,000 USA to 1.92/100,000 NZ although incidents have been reduced with advent of new HIV drugs	Disseminated disease involving lymph nodes, CNS, liver, spleen. Cervical lymphadenitis in children	Tell 2001, Quaranta 1996, Dhama 2011
<i>Mycobacterium genavense</i>	Subclinical or emaciation	Chickens, wild birds in captivity	3% companion birds (Hoop 1996)	Contact transmission	Present	Rare	Progressive disease mostly in immunocompromised hosts	Tell 2001
<i>Mycobacterium tuberculosis</i>	Cutaneous growths on head and neck, granulomas in eyes	Psittacines only	Rare	Human to bird transmission with unproven bird to human transmission	Present	Not zoonotic	Not zoonotic	Washko 1998

Hazard	Clinical signs	Species affected	Level of infection	Zoonotic Transmission	Present in GB or last occurred	Human cases	Human symptoms	Reference:
<i>Mycoplasma gallisepticum</i>	Coughing, poor productivity, slow growth, inappetence	Chickens, turkeys, game birds, pigeons, pheasants	Common in commercial poultry farms	Not zoonotic	Present	Not zoonotic	Not zoonotic	http://www.cfsph.iastate.edu/Factsheets/pdfs/avian_mycoplasmosis_mycoplasma_gallisepticum.pdf
<i>Mycoplasma pullorum</i>	Upper respiratory disease	Pheasants, partridges, chickens, turkeys	Unknown in wild birds	Not zoonotic	Present	Not zoonotic	Not zoonotic	Bradbury 2001
<i>Ornithobacterium rhinotracheale</i>	Respiratory disease	Chickens, turkeys	High prevalence in commercial poultry farms	Not zoonotic	Present	Not zoonotic	Not zoonotic	Hafez 2010, Canal 2003
<i>Pasteurella multocida</i>	Nervous system signs, sudden death	Partridge, pheasant, grouse, quail predominantly waterfowl	Highest level of infection in N. American waterfowl but still low mortality levels ~2%.	Contact transmission - usually via cat and dog bites/scratches	Present	Relatively uncommon, 450 lab cases per year of which 70% are multocida. Avian isolates are generally non-pathogenic in mammals	Local wound infection, respiratory tract infection. 5 deaths since 1993	Botzler 1991; OIE-health standards
<i>Plesiomonas shigelloides</i>	Uncertain pathogenicity	Waterfowl	0.79% in birds (Bardon 1999)	Food borne transmission	Rare in the UK	Occurs mainly in tropical and subtropical areas	Diarrhoea, colitis, abdominal pain	Niskannen 2000, Gonzalez-Rey 2011
<i>Riemerella anatipestifer</i> (<i>Pasteurella anatipestifer</i>)	Listlessness, diarrhoea, fluid discharge from eyes	Ducks, reared pheasants and quail	Infrequent	Not zoonotic	Present	Not zoonotic	Not zoonotic	Twycross zoo website
<i>Salmonella</i> spp.	Subclinical or scour	All game birds, ducks	1% red legged partridge, 4.5% pheasant	Food borne transmission	Present	9,685 laboratory confirmed cases 2010	Salmonellosis, Diarrhoea/sickness	Beer 1989
<i>Salmonella</i> Arizona	Diarrhoea, paralysis, blindness	Turkeys	10-50% mortality	Food borne transmission	Not present in UK turkey population	50 reports since 1950	Predominantly affects immunosuppressed hosts	Hoag 2005

Hazard	Clinical signs	Species affected	Level of infection	Zoonotic Transmission	Present in GB or last occurred	Human cases	Human symptoms	Reference:
<i>Salmonella</i> Enteritidis	Subclinical or scour	Pheasants	Incidental	Food borne transmission	Present	2,444 isolates reported to HPA 2010	Salmonellosis, Diarrhoea/sickness	Hosie and Grant 1989
<i>Salmonella</i> gallinarum (Fowl typhoid)	ruffled feathers, weakness, diarrhoea	Chickens, turkeys, game birds	10-100% morbidity	Not zoonotic	Present	Not zoonotic	Not zoonotic	OIE
<i>Salmonella</i> pullorum	Subclinical or scour	Pheasants, game birds	10-80% morbidity, 30% mortality rate	Not zoonotic	Present	Not zoonotic	Not zoonotic	Pennycott & Duncan 1999
<i>Salmonella</i> Typhimurium	Subclinical or scour	Pigeons	0.6 - 4.5%	Food borne transmission	Present	1,959 isoaltes reported to HPA 2010	Acute intestinal pain and diarrhoea.	Kinjo 1983, Pennycott 1994
<i>Staphylococcus aureus</i>	Inflammation of skin on foot, arthritis	More common in captive birds - chickens and turkeys, pheasants	Ubiquitous organism	Food borne transmission	Present	10,070 cases voluntarily reported 2010	Severe vomiting, diarrhoea	HPA
Methicillin resistant <i>Staphylococcus aureus</i>	Dermatitis, feather plucking	Psittacines, pigeons	Rare but increasing	Contact transmission	Present	Approximately 1,481cases (HPA 2011 data)	Local wound infection, endocarditis, pneumonia, can be fatal	Saleha 2010, Schwarz 2004
<i>Streptococcus bovis</i>	Leg weakness and sudden death	Ducks, pigeons	10% in pigeons	Contact transmission	Present	Strongly associated with colorectal cancer (Al-Jashamy 2010)	Liver disease, endocarditis	Vanrobaeys 1997, de Herdt 1994
<i>Vibrio cholerae</i>	Hepatitis	chickens, ostriches	6% in gulls (lee 1982)	Only serotypes 01 and 0139 are responsible for cholera epidemics in man. Disseminators rather than transmitters	Mostly in developing countries. Not present in the UK	Not zoonotic	Not zoonotic	Lee 1982, Reed 2003

Hazard	Clinical signs	Species affected	Level of infection	Zoonotic Transmission	Present in GB or last occurred	Human cases	Human symptoms	Reference:
<i>Yersinia enterocolitica</i>	Usually asymptomatic	Pheasants, pigeons, wild birds	15.2% in pheasants (Kato 1985)	Food borne transmission	Present	19 cases 2010	Diarrhoea, abdominal pain, arthritis. UK confirmed cases of 0.09/100,000 people with no fatalities 2010	Galindo 2011
<i>Yersinia pseudotuberculosis</i>	ruffled feathers, weakness, diarrhoea	Turkeys, ducks wild birds	5-75% mortality	Food borne transmission	Present	14 other <i>Yersinia</i> spp cases 2010	Gastroenteritis only contributed to 1.7% of non <i>enterocolitica</i> isolates	Galindo 2011
Viruses								
Avian Encephalomyelitis	Drop in egg production (older) paralysis and tremors (younger)	Quail, pheasants, turkeys, chickens	5-60% morbidity	Not zoonotic	Present	Not zoonotic	Not zoonotic	Welchman 2009
Avian Hepevirus (Big Liver and Spleen Disease)	Egg drop, anaemia	Chickens	71% chickens seropositive (Huang 2002)	Potential zoonosis	Present	450 cases 2011 (>80% associated with foreign travel)	Hepatitis, flu like symptoms, vomiting	Peralta 2009, Vasickova 2007
Avian Influenza	diarrhoea, coughing, paralysis, sudden death	Ducks	0.67% in wild birds (AHVLA 2011)	Contact transmission	Present	Rare	Flu symptoms, severity of symptoms depends on strain	Kalthoff 2010
Adenovirus	Egg drop, Inclusion body hepatitis, marble spleen disease	Chickens, game birds, pheasants	Sporadic	Not zoonotic	Present	Not zoonotic	Not zoon	Kayali 2009
Avian Leukosis virus (Leukosis/Sarkoma group)	Emaciation, enlargement of abdomen	Chickens	Unknown in wild birds	Not zoonotic	Present	Not zoonotic	Not zoonotic	Payne 2000
Avian Rhinotracheitis	Dyspnoea, decreased appetite, nasal discharge	Chickens, turkeys, pheasants	10-100% morbidity, 1-10% mortality	Not zoonotic	Present	Not zoonotic	Not zoonotic	OIE, Dalton 2002

Hazard	Clinical signs	Species affected	Level of infection	Zoonotic Transmission	Present in GB or last occurred	Human cases	Human symptoms	Reference:
Circovirus	Feather/beak abnormalities, leucopaenia, fatalities.	Particularly affects psittacines. Pigeons	Increasing incidence in pigeons upto 75%	Potential zoonosis	Present	No significant sequence homology with animal circoviruses	Flu-like symptoms	Pennycott 2002, Biagini 2003
Crimean Congo hemorrhagic fever	Asymptomatic	Migratory birds	Only ostriches are susceptible	Contact transmission (Transmitted via ticks)	Not present in the UK	Majority of cases from workers in livestock industry	Infrequent infection but 30% mortality rate	WHO
Duck viral enteritis	Egg drop, closed eyes, dehydration, sudden death	Ducks	Morbidity 5-100%	Not zoonotic	Present - mostly in ornamental ducks	Not zoonotic	Not zoonotic	Dardiri 1975
Duck viral hepatitis	Falling, arching and sudden death	Ducks	100% morbidity	Not zoonotic	Present	Not zoonotic	Not zoonotic	Schultz 2004
Eastern Equine Encephalitis	Paralysis, tremors, ataxia	Pheasants, partridges, wild birds, pigeons, chickens, turkeys	High morbidity	Contact infection (Transmission via mosquito bite)	Not in the UK	Human cases usually associated with horses.	High fever, vomiting, convulsions, coma	Gibney 2011
Infectious Bronchitis (Coronavirus)	Depression, coughing, diarrhoea, loss of appetite	Chickens	50-100% morbidity	Not zoonotic	Present	Not zoonotic	Not zoonotic	Merck Vet manual,
Infectious Bursal Disease	Depression, unsteady gait, huddling	Chickens, turkeys, ducks	High morbidity	Not thought to be zoonotic	Present	Not zoonotic	Not zoonotic	Chettle 1989
Louping ill (Flavivirus)	Anorexia, muscle weakness	Red grouse	80% mortality in experimental infection	Contact transmission (via tick bites, aerosols, contact with infected animal tissue)	Present	45 documented cases worldwide, 26 from laboratory exposure, 12 infected carcasses.	Flu like illness	Davidson 1991
Marek's Disease (<i>Gallid Herpesvirus 2</i>)	Paralysis, loss of weight, vision impairment	Chickens, turkeys, quail	10-50% morbidity, low prevalence	Potential zoonosis	Present	Controversial zoonotic evidence	Possible link to human lymphoproliferative disorders	Karsten Tischer 2010

Hazard	Clinical signs	Species affected	Level of infection	Zoonotic Transmission	Present in GB or last occurred	Human cases	Human symptoms	Reference:
Paramyxovirus Type 1(Newcastle disease) (Notifiable)	Respiratory disease/diarrhoea	Partridge, quail, pheasant. Waterfowl subclinical, chickens	Morbidity varies with species and strain of virus	Contact transmission(Aerosol/contamination)	Last outbreak 2006	Rare	Conjunctivitis, sub-conjunctival haemorrhage	OIE
Reticuloendotheliosis	Can be subclinical, or may have leg weakness, diarrhoea	Chickens, turkeys, ducks, quail	Morbidity up to 25%	Not zoonotic		Not zoonotic	Not zoonotic	
West Nile Virus	Emaciation, inability to fly, sudden death	crows, jays, migratory birds, geese	Not present in the UK	Contact transmission (via mosquito bites)	Not present in UK	Not present in the UK	80% subclinical; 20% flu-like symptoms <1% more severe disease/death.	Reed 2003
<i>Parasites</i>								
<i>Ascaris</i> spp.	Diarrhoea, emaciation	Common in aviary housed birds, hawks,	20% in captive birds Patel 1999	Zoonotic evidence from domestic pigs	Present	Annual rates of 0.12/100,000 in England (HPA)	Usually asymptomatic but migrating larvae can cause obstructions.	Bendall 2011
<i>Capillaria</i> spp.	Wasting, poor growth, diarrhoea	Poultry, game birds, pigeons	Low	Food borne transmission	Present	<i>C.philippinensis</i> only species of significance - found occasionally in Europe	Diarrhoea, abdominal pain	Cross 1991, Ming-Jong 2004
<i>Centrocestus formosanus</i>	Subclinical	Chickens, fish eating birds	Not present in the UK	Potential zoonosis	Not present in the UK	Not present in the UK	Natural human infections have never been documented	Eun-Tak 2008
<i>Ceratophyllus columbae</i>	Asymptomatic	Pigeons	High	Contact transmission	Present	Rare	Skin irritation, dermatitis, erythema	Haag-Wackernage 2004
Coccidiosis (<i>Tyzzeria perniciososa</i>)	Depression, tucked appearance, sudden death	Ducks	Low	Avian strains are not normally zoonotic	Rare in the UK	Avian strains are not normally zoonotic	Avian strains are not normally zoonotic	Ruff 1987

Hazard	Clinical signs	Species affected	Level of infection	Zoonotic Transmission	Present in GB or last occurred	Human cases	Human symptoms	Reference:
<i>Cryptosporidium baileyi</i>	Swollen head, bulgy eyes, flight impairment	Red grouse, chickens, turkeys, ducks, quail	Unknown in wild birds	Not zoonotic	Present	Not zoonotic	Not zoonotic	McDougald 2008, Coldwell 2012
Cryptosporidiosis	Cough, low weight gain, diarrhoea	Chickens, turkeys, ducks, quail	Unknown in wild birds	Zoonotic potential	Present	3893 HPA reported infections 2010	abdominal pain, nausea and diarrhoea	Qi 2011
<i>Dermanyssus gallinae</i>	Egg spotting, anaemia	Poultry, pigeons	7.5 - 87.5%	Contact transmission	Present	High risk to Poultry workers or via bird nests in buildings	rash, pruritis, dermatitis	Akdemir 2009, Hamidi 2011, Sparagano 2009
<i>Echinostoma cinetorchis</i>	Apparently healthy	Chickens, water fowl	7% in ducks (Thi Lan Anh 2010)	Food borne transmission	Not present in the UK	Endemic in SE Asia	abdominal pain, nausea and diarrhoea	Graczyk 1998
<i>Francisella tularensis</i>	Subclinical	Grouse, quail, pheasant	Infrequent	Contact transmission	Not present in the UK	Not in the UK	Ulcers, sore throat, pneumonia	Padeshki 2010
<i>Giardia spp.</i>	watery faeces, ruffled feathers, death	Aquatic birds	Unknown in wild birds	Zoonotic potential	Present	~ 3000 lab reports annually of Giardia lamblia.	stomach cramps, diarrhoea	Majewska 2009
<i>Heterakis gallinae</i> (Caecal worm)	None	Poultry and game birds	High morbidity (transport host for <i>Histomonas</i>)	Not zoonotic	Present	Not zoonotic	Not zoonotic	Movsessian 1994
Hexamitiasis (now called <i>Spironucleus</i>)	Loss of weight, convulsions, inappetance	Pigeons, pheasants, pigeons and some game birds		Not zoonotic	Present	Not zoonotic	Not zoonotic	Beynon 2009
<i>Histomonas meleagridis</i>	Inappetance, emaciation, poor growth	Turkeys, chickens, pheasants, gamebirds	High morbidity and mortality	Not zoonotic	Present	Not zoonotic	Not zoonotic	Popp 2011
<i>Hypoderma conoideum</i>	Apparently healthy	Chickens, water fowl	20-30% in chickens and ducks	Food borne transmission	Not present in the UK	Endemic in SE Asia	abdominal pain, nausea and diarrhoea	Thi Lan Anh 2010

Hazard	Clinical signs	Species affected	Level of infection	Zoonotic Transmission	Present in GB or last occurred	Human cases	Human symptoms	Reference:
<i>Leucocytozoon</i> spp.	Loss of appetite, anaemia	Geese, ducks, chickens, turkeys	Mortality rates can be 90% in young birds	Not zoonotic	Not present in the UK	Not zoonotic	Not zoonotic	Hagihara 2004
<i>Sarcocystis</i> spp.	Often asymptomatic, can cause muscle weakness	Waterfowl	Frequent in American ducks	Not zoonotic	Very rare	Not zoonotic	Not zoonotic	Kutkiene 2011
<i>Schistosoma dermatitis</i>	Subclinical or scour	Ducks are natural host, snails intermediate host	Can exceed 50%	Avian associated not zoonotic	Present	Not zoonotic	skin rash	MacConnachie 2012, Kolarova 2007
<i>Syngamus trachea</i> (Gape worm)	gasping, coughing	chickens, game birds	High prevalence at high densities	Not zoonotic	Present	Not zoonotic	Not zoonotic	Draycott 2006
<i>Toxocara canis</i>	Apparently healthy	Accidental hosts	Unknown in wild birds	Food borne transmission	Present	Prevalence of 14% in USA	weight loss, respiratory symptoms, vomiting	Despommier 2003
<i>Toxoplasma gondii</i>	Apparently healthy	Chicken, pheasants, guinea fowl	36% prevalence in free range chickens	Food borne transmission	Present	Between 7-34% (HPA)	Generally mild in healthy hosts, can have longterm health affects	Dubey 2010
<i>Trichomonas gallinae</i> and <i>anseris</i>	Drizzling, loss of condition, open mouth	Pigeons and doves, turkeys, chickens	High morbidity	Not zoonotic	Present	Not zoonotic	Not zoonotic	Pennycott 1997
<i>Fungi</i>								
<i>Aspergillus fumigatus</i>	weakness, gasping, wasting	Ducks, game birds, waterfowl, pigeons	5-50% mortality	Contact transmission (inhalation of fungal spores)	Present	Only affects people with suppressed immune systems	asymptomatic in health individuals	Cacciuttolo 2009
<i>Candida albicans</i>	Poor appetite, slow growth, diarrhoea	Chickens, turkeys sometimes other birds	Low	Contact transmission	Present	875 laboratory reports in 2010 (HPA)	Thrush, can be severe in immunocompromised hosts	Cafarchia 2008
<i>Cryptococcus neoformans</i>	Healthy carriers	Pigeons, parrots, waterfowl	High prevalence in faeces	Association is via avian manure not directly with birds	Present	Opportunistic infection in immunosuppressed patients	Respiratory infection	Brizendine 2011

Hazard	Clinical signs	Species affected	Level of infection	Zoonotic Transmission	Present in GB or last occurred	Human cases	Human symptoms	Reference:
				i.e avian associated but not zoonotic				
<i>Histoplasma capsulatum</i>	Not susceptible	Most birds	High prevalence in faeces	Infection usually via soil not birds i.e avian associated not zoonotic	Present	50-200,000 cases annually in USA	mild flu-like symptoms	Luby 2005
<i>Microsporium gypseum</i>	Alopecia	Poultry, pigeons	Unknown in wild birds	Contact transmission	Present	More prevalent in children	Pruritus, ringworm	OIE
<i>Trichophyton gallinae</i>	Fungal infection affecting comb and wattle	Poultry, pigeons	Unknown in wild birds	Contact transmission	Present	Rarely zoonotic - 7 proven reported cases	Onychomycosis	OIE, Palacio 1992