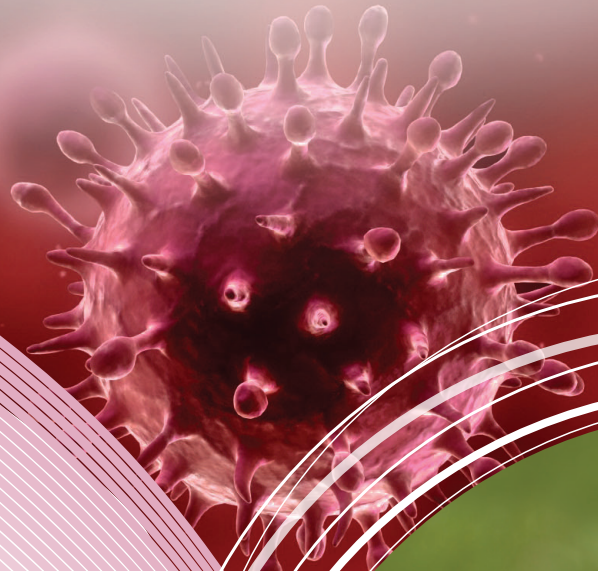


ZOONOTIC DISEASES, HUMAN HEALTH AND FARM ANIMAL WELFARE



CONTENTS

02	SUMMARY
03	INTRODUCTION
03	THE STUDIES
04	ESCHERICHIA COLI (E. COLI)
05	CAMPYLOBACTER
07	SALMONELLA
09	AVIAN AND SWINE INFLUENZA
11	CONCLUSIONS
12	POLICY RECOMMENDATIONS
13	REFERENCES
16	ACKNOWLEDGEMENTS

SUMMARY

There is a major threat to humanity and it comes from the very food we eat – a terrible consequence of our modern farming systems. Some diseases that infect animals can also be passed on to humans. These are known as zoonotic diseases.

As farming methods have become more intensive, there is an increasing number of animals reared in confined spaces. This is combined with breeding and feeding approaches designed to increase production. It is often at the expense of the animals' welfare but it's also putting human health at risk. It increases the risk of certain diseases, which can lead to serious illness in humans and may be fatal. As we consume more animal products, particularly chicken and pig meat, there is greater risk of exposure to these illnesses.

Salmonella, *E. coli* and *Campylobacter* are all bacteria that can cause food poisoning. We can get ill when we eat contaminated meat, eggs and dairy. It is not only what we eat that puts us at risk; influenza viruses that affect poultry and pigs on farms can give rise to a 'flu that infects humans and can lead to rapid, widespread disease.

This briefing by Compassion in World Farming and the World Society for the Protection of Animals is based on longer reports written by experts. It examines some of the most common food poisoning bacteria, as well as the viruses avian and swine influenza; assessing the causes and risks to us and farm animals.

Governments, Inter-Governmental Organisations (IGOs) and the food production industry must urgently work together to implement the following recommendations:

- **Ensure health** – by developing farming policies for humane sustainable food supplies that ensure the health of animals and people. This includes using animal breeds, diets and management conditions that minimise stress and optimise animal welfare and immunity.
- **Surveillance and vaccination** – helping minimise the spread of disease.
- **Limit transportation time** – ensuring animals are slaughtered humanely on or near to the farm where they were raised.
- **Invest in research and knowledge transfer** – helping support farmers to develop and implement higher welfare livestock systems.
- **Reduce non-therapeutic antibiotic use** – limiting the risk of antibiotic resistance.
- **Encourage consumers to eat less and higher welfare meat** – reducing the risk of exposure to food infected with *Salmonella*, *Campylobacter* or *E. coli*.

KEY FINDINGS

Both *Salmonella* and *E. coli* infections are often greater in intensive farm production conditions. Fast-growing birds may be more susceptible to *Campylobacter* infection, believed to be the most important foodborne pathogen. The bacteria can pass from the bird's gut into the meat of the chicken, greatly increasing the risk of infection to humans.

Long distance transport of animals increases the risk of infection for all three bacteria. Risk of exposure to foodborne pathogens generally increases with increased consumption and lower welfare animal products.

The risk of new strains of influenza that can infect humans is of serious concern, now and in the future. Farm animal numbers have risen rapidly and large-scale concentration of poultry and pigs has become increasingly common, alongside long distance transport. This increases the risk of new strains of influenza viruses emerging and spreading.

INTRODUCTION

Zoonotic diseases are a major global threat to public health and animal welfare. Animal products contaminated with bacteria such as *Salmonella*, *Campylobacter* and *E. coli* are responsible for large numbers of foodborne human infections, which can be fatal. Influenza viruses circulating in farmed animals periodically give rise to a human disease pandemic, often with devastating consequences.

Farming methods have changed dramatically in recent decades. Production has become increasingly industrialised, with larger numbers of animals stocked at higher densities, coupled with breeding and feeding strategies aimed at maximising production. These changes have a huge impact on the welfare of farmed animals we rear for food and can increase the risks to people and animals from some zoonotic diseases.

THE STUDIES

In 2012, Compassion in World Farming, with support from The Tubney Charitable Trust and the World Society for the Protection of Animals, commissioned leading experts to compile a series of reports examining the public health threat posed by some of the major zoonotic diseases and the effects of farming systems on this threat. This report summarises their findings and provides our policy recommendations.



ESCHERICHIA COLI (E. COLI)

Key message

The high stocking densities and diet commonly found in intensive fattening systems for beef cattle increase the risk of *E. coli* infection. Cattle fed on grass and reared extensively are less likely to carry the bacteria and are likely to have better welfare. Long journeys for slaughter can lead to increased shedding and spreading of bacteria, as well as poor animal welfare.

Background

Most strains of *E. coli* do not cause disease, but live naturally in the intestines of animals and humans, where many are probably beneficial to health as a key part of our gut flora. However, a small number of *E. coli* strains can cause disease in people, usually intestinal infection. The most common is Enterohaemorrhagic *E. coli* (EHEC), particularly the O157:H7 strain, which is responsible for the majority of UK and North American cases¹. Although infection with pathogenic *E. coli* is relatively rare, it is the serious and sometimes fatal nature of the disease that gives it such high importance².

Sources of infection and effects of farming systems

Cattle, sheep and pigs may carry EHEC without developing disease. The bacteria may then contaminate meat or animal products from faecal

material in the lower gut or on the hide of animals at slaughter. Cattle, and therefore beef, are the main source of EHEC^{3,4}, particularly minced or ground beef, due to its large surface area that supports the bacteria and, because it may be produced from multiple cuts of meat from several animals⁵. Cross contamination between raw and cooked meat is a particular risk. When this happens in the food industry, the consequences can be catastrophic.

The use of feedlots as an intensive system to fatten beef cattle prior to slaughter seems to be a particular risk for EHEC infection. Transmission from one animal to another is more likely as a result of high stocking densities in feedlots. Also, feedlot cattle are fed a diet of grain to fatten them for slaughter quickly. This diet promotes the growth of *E. coli*, including EHEC, in the hindgut, leading to increased colonisation and shedding of EHEC, which can then spread to other animals⁶. Cattle fattened in feedlots may also be under considerable environmental stress in hot and crowded conditions, which can also lead to increased shedding of bacteria in faeces⁷. Long transport times and poor conditions while awaiting slaughter or at markets may further increase shedding.

Cattle fed on grass and reared in more extensive systems are less likely to carry EHEC. Traditional grass and forage diets are higher in plant compounds such as tannins and phenolics that inhibit *E. coli* growth. Cattle are also typically reared with lower stocking densities. Grass-fed beef is considered to be a superior product in terms of flavour and this production system is more welfare-friendly for an animal that has evolved to eat grass⁸.



Photo © Inmagine

Intensively farmed cattle have a higher risk of carrying *E. coli* compared to pasture-reared herds.



Grass and forage based diets help inhibit the growth of *E.coli* in cattle.

Levels of infection in animals and people

The difference in the way cattle are reared (commonly intensively in the US) is reflected in the different levels of infection between the US and UK. Studies of beef cattle in the US indicate that EHEC may be present in the intestines or on the hides of 20-28% of cattle at slaughter^{9,10} and in 43% of meat samples after processing¹¹. Levels in the UK are lower, with 4.7% of cattle, 0.8% of sheep and 0.3% of pigs colonised¹². The US has around 73,000 human EHEC cases a year, compared to fewer than 1,000 in England and Wales, which is a substantial difference, even when the difference in population is taken into account.

Control options and future prospects

Raising and feeding cattle on grass rather than grain would help limit the risk of EHEC infection. A number of interventions have also been proposed to reduce EHEC in cattle. Vaccination may be possible^{13, 14, 15}. However, modelling suggests that elimination is unlikely and some reduction is the best we can realistically achieve¹⁶. Therefore, keeping cattle on pasture is likely to be the best way to help minimise risk.

A related strain that causes disease in poultry is Avian Pathogenic *E. coli* (APEC). This is an increasing problem in intensive meat chicken production and is considered to cause the loss of at least 10 million animals per year in the UK. There is concern that APEC could possibly evolve into a zoonotic disease, infecting humans in the future¹⁷.

CAMPYLOBACTER

Key message

Lower levels of stress in free-range chicken production systems, as well as slower-growing breeds, may balance the disadvantages of environmental contamination. Avoiding thinning of chicken flocks and ensuring humane handling during catching and transport have an important role in minimising acute stress, which could reduce levels of *Campylobacter*.

Background

Campylobacter is the single biggest identified cause of bacterial infectious intestinal disease in people in much of the developed world. Recently, the World Health Organisation declared it the most important foodborne pathogen. Symptoms of acute *Campylobacter* infection vary from mild diarrhoea lasting 24 hours to severe illness lasting more than a week. Around 1% of cases go on to develop long-term complications.



The practice of 'thinning' poultry makes birds more susceptible to *Campylobacter*.

Sources of infection and effects of farming systems

Poultry are the main source of *Campylobacter* infection and are estimated to be responsible for up to 80% of cases in the EU¹⁸. The biggest risk is chicken meat (including chicken liver). Levels of surface contamination of *Campylobacter* on chicken carcasses from gut contents at slaughter are high, probably due to the speed of slaughtering and the fact that chicken carcasses and portions are generally wrapped, keeping meat surfaces moist, which facilitates *Campylobacter* survival. Cross-contamination in catering is also an important risk factor identified¹⁹. Unlike other meat products, *Campylobacter* is also found deep inside chicken muscle (meat) and liver, rather than just on the surface and is discussed later. This internal contamination of edible tissues poses a major public health threat, as the bacteria may survive cooking better.

Acute stress

An important risk factor for housed birds is the practice of 'thinning'^{20, 21}. Intensive chicken houses are often stocked to maximise the number of birds that can be produced from a given floor space. At approximately five weeks of age, around 30% of birds are removed for slaughter at a lighter weight, with the remainder being kept for around another week so they are heavier. Infection can be introduced during catching of the birds by people and machines coming in from outside. The birds remaining in the house will be stressed making them more susceptible to infections like *Campylobacter*.

Feed is withdrawn from poultry flocks prior to slaughter to reduce the risk of faecal contamination of carcasses during the slaughter process. However, fasting tends to increase the number of *Campylobacter* in the gut^{22,23} and the stress caused by feed withdrawal may pre-dispose birds to *Campylobacter* infection and increased shedding.

Acute stress (for example due to catching and transport) leads to physiological changes that can reduce the levels of potentially protective bacteria in the intestines, alter the permeability of the gut wall and potentially increase the growth

rate and shedding of *Campylobacter*²⁴. Levels of *Campylobacter* are higher in birds that have been caught and transported compared to ones from the same flock left on the farm²⁵.

The farm environment

The most important source of *Campylobacter* infection is the farm environment²⁶. Wild animals may act as an indirect source of flock infection through environmental contamination. Spread of infection can be very rapid in a newly-infected flock²⁷.

There is a need for the risk from extensive systems to be properly assessed. Industry figures currently show that there seems to be little difference in the frequency of *Campylobacter* in housed and extensive flocks. It needs to be established whether extensively reared birds pose the same public health risk as ones reared inside. If risk is based solely on contamination of carcass surfaces, then such birds may be a risk. However expert assessment suggests that, if other factors such as contamination of edible tissues are taken into account, the risk from extensively reared birds may be lower.

Slower-growing birds

Birds reared outside are more likely to have higher welfare and use a slower-growing breed of chicken than intensive production systems. Research suggests that *Campylobacter* in these birds is more likely to remain in the gut rather than penetrating the meat²⁸. Chronic stress (for example due to a poor production environment) has also been shown to lead to immunosuppression in chickens, rendering birds less able to resist infection²⁹. This may make it more likely that *Campylobacter* is able to spread to muscle and organs such as the liver.

Chickens reared for meat are continuously being selected to grow and put on weight ever more quickly. Slower-growing breeds, of the type used in higher welfare systems, are generally healthier and may be at lower risk of *Campylobacter* infection³⁰.

There is an urgent animal welfare and public health need to determine the effects of selection for rapid growth in chickens on the gut environment and muscle penetration as well as disease resistance.



Levels of infection in animals and people

Current estimates indicate that around 75% of chickens on sale in the EU are infected with *Campylobacter* and 1% of the human population of the EU is infected with *Campylobacter* each year. It is estimated that there are 700,000 cases and over 100 deaths in the UK each year due to *Campylobacter* infection.

In most developed countries, the number of *Campylobacter* cases has been increasing over the past 20 years. Improved diagnosis may play some part in this, although most clinical laboratories have not significantly changed their techniques over this time period. It is difficult to escape the conclusion that the rising tide of cases is associated with increased chicken consumption. If the UK is used as an example, chicken was perceived to be a luxury item in the 1960s, often eaten only once or twice a year. The introduction of industrial-scale production and birds with much faster growth rates has dramatically reduced the price of chicken so that it is now seen as an everyday food.

Control options and future prospects

The international poultry industry faces a major challenge in trying to control *Campylobacter*. It is likely that the EU will establish baseline figures and targets for member states in the near future.

Past work has shown that *Campylobacter* control is possible for housed birds by strict observance of biosecurity by farm staff³¹. The current high levels of *Campylobacter* in chickens on sale clearly indicate that either biosecurity is not being properly applied and/or that measures that were once successful no longer work as well, possibly because the modern fast-growing meat chicken is more susceptible to infection. Application of biosecurity measures in higher welfare indoor systems, with lower stocking densities and slower-growing birds, may be more successful. It may be possible to breed chickens that are resistant to *Campylobacter*. These would be likely to grow more slowly than current fast-growing commercial strains. Other potential control measures, such as vaccination, are being researched.

SALMONELLA

Key message

Poultry production systems with higher welfare do not increase the risk of *Salmonella* infection and are in fact likely to have a lower risk. Biosecurity, testing and management, including vaccination, are the best ways of controlling *Salmonella* in every production system.

Background

Salmonella is a major worldwide problem for both animal and public health. Most of the 2,500 strains of *Salmonella enterica* can infect a wide range of animal species and are capable of causing diarrhoea in humans. Throughout the world, the most important foodborne *Salmonella* strains are *Salmonella* Typhimurium and *Salmonella* Enteritidis, both in terms of number of cases and the severity of infection caused. *Salmonella* infection can sometimes be fatal.

Sources of infection and effects of farming systems

The majority of human *Salmonella* infections come from contaminated food, especially poultry meat, eggs and pig meat. It is thought around 20% of human cases of *Salmonella* infection in the EU are due to consumption of pork or pork products³². Chickens may carry *Salmonella* with little or no ill effect to the animal³³. Pigs may also be infected without showing signs of disease, although young pigs may develop diarrhoea in much the same way as humans.

The industrial nature of both production and slaughter make the spread of infection relatively easy in poultry. Carcasses are frequently contaminated by gut contents during slaughter. In laying hens, eggs may become infected within the reproductive tract. Faecal contamination of eggs after laying may also occur, which appears to be a problem in intensive, cage-based systems³⁴.

It has been suggested that *Salmonella* should be easier to prevent in animals housed indoors than in free-range production, using good biosecurity to prevent the entry of infection. Recent studies suggest the risk of wild birds introducing infection on free-range farms has been overstated, with less than 0.2% of healthy wild birds being infected³⁵. Also, poultry are more susceptible to infection in flocks with poor welfare and spread of infection is likely to be greater in more intensive production.

Larger flock sizes, particularly with birds of mixed ages, increase the levels of *Salmonella*^{36, 37}. Several studies show that caged birds have much higher levels of *Salmonella*^{38, 39, 40, 41}. In some cases, the likelihood of infection has been found to be ten times higher in caged birds than in free-range hens. However, not all studies agree and some have found that cage systems can have lower or equivalent levels of *Salmonella* compared to free-range or floor-housed hens^{42, 43}. The balance of opinion is that production systems with higher welfare do not increase the risk of *Salmonella* infection and on balance are likely to have a lower risk of infection⁴⁴.

In some countries, such as the US, hens may be subjected to forced moulting to trigger a new cycle of egg laying. This involves reducing or withdrawing food for up to two weeks. It has profound effects on chicken welfare and particularly on the immune system, which may result in increased susceptibility to both intestinal and egg infection with *Salmonella*^{45, 46, 47}. This practice leads to an increased public health risk as well as a period of high physical and psychological stress for the birds⁴⁸.

Mixing of young pigs from separate pens, sheds or farms is considered to be a major factor in the spread of *Salmonella* infection. Gut contents may contaminate meat with *Salmonella* at slaughter if pigs are carrying the bacteria⁴⁹. Stress in infected pigs, particularly from lengthy journeys to slaughter, may increase shedding of *Salmonella* in faeces and therefore the spread of the bacteria at the time of slaughter^{50, 51}.

Levels of infection in animals and people

The US Centers for Disease Control and Prevention estimates it has over 1.2 million cases of human *Salmonella* infection a year, compared to around 50,000 cases in the UK as estimated by the Health Protection Agency. There are around 80 to 100 deaths caused by *Salmonella* infection each year in the UK.

Less than 1% of UK laying flocks and 3% of meat chicken carcasses are infected with *Salmonella*⁵². United States Department of Agriculture (USDA) figures suggest as much as 23% of US poultry meat is infected with *Salmonella*. Prevalence of *Salmonella* infection in pigs at slaughter is estimated to be 10% in the US⁵³ and 22% in the UK⁵⁴.

Control options and future prospects

The development of improved testing and control, including vaccination, has been successful in significantly reducing *Salmonella* in laying hens in many countries, including the UK. Vaccination is not yet widely used in the control of *Salmonella* in pigs and commercially available vaccines do not really offer the protection needed. Vaccination of meat chickens reared for slaughter is not considered feasible due to the cost⁵⁵ and the young age of the birds at slaughter⁵⁶. Production systems need to be used that provide higher welfare for laying hens. Practices, such as forced moulting, should not be permitted.

Recent European legislation has formalised controls throughout Europe. Baseline surveys of *Salmonella* in breeding flocks, layer and meat chicken flocks, turkeys and pigs were made by the European Food Safety Authority (EFSA). Each member state was required to develop and implement a series of National Control Plans for *Salmonella* and set out targets for reduction. Surveillance and control measures in the US are considerably less rigorous. Vaccination is used by around 50% of US egg producers compared to over 99% in the UK.

For the future, there is increasing concern about the emergence of *Salmonella* strains that are resistant to multiple antibiotics, potentially making the treatment of infections in animals and people more difficult.

AVIAN AND SWINE INFLUENZA

Key message

Lower levels of stress and sunlight (which kills the virus) in extensive chicken and pig production may balance the disadvantages from the risk of the virus being spread by the wind or wild birds, particularly ducks, to extensively farmed animals. Despite the common perception that industrial poultry has a lower risk of spreading the disease compared to free-range or backyard farms, research suggests this is not the case. Long distance transport, which has a negative impact on animal welfare, should be avoided to reduce the risks of new pandemics. Stringent biosecurity is considered the best way of controlling the spread of the disease in every farming system.

Background

Avian and swine influenza are caused by influenza A viruses. There are many different subtypes, categorised according to two types of protein that project from the surface of the virus: HA and NA. Avian influenza has the potential to cause rapid and widespread mortality in domestic chickens and turkeys. Usually, influenza infection in poultry causes mild disease, referred to as low pathogenicity avian influenza (LPAI), but two subtypes (H5 and H7) can mutate to a highly pathogenic form (high pathogenicity avian influenza, HPAI) in poultry. There is particular concern about H5N1 HPAI, which has affected flocks in over 60 countries.

Swine influenza typically causes respiratory disease in pigs with a rapid onset of fever, loss of appetite and coughing. It is rarely a fatal illness; animals may lose a considerable amount of weight, which has economic consequences, but they usually recover within 7 to 10 days⁵⁷. Avian and swine influenza viruses can sometimes infect and cause disease in people, causing worldwide concern. Occasionally, a new strain emerges that can be transmitted easily from person-to-person and a pandemic can result, often with devastating consequences.

Sources of infection and effects of farming systems

HPAI viruses are rarely transmitted from poultry to people, but the occurrence seems to be on the increase in line with increasing numbers of reported outbreaks of HPAI in poultry. The World Health Organisation reports 615 laboratory-confirmed cases of human infection with H5N1 HPAI across 15 countries between 2003 and 1st February 2013, resulting in 364 deaths. There have been some isolated incidents of human-to-human transmission of HPAI H5N1, but to date there has been no sustained human-to-human transmission.

Pigs can be infected with both avian and human influenza strains and may provide a 'mixing' vessel, allowing novel combinations of HA and NA genes to emerge⁵⁸. This is called 'reassortment'. In this way, pigs may act as an intermediate host in the introduction of novel influenza subtypes into the human population. When a virus emerges with HA and NA proteins not previously encountered by the majority of people, and the virus is able to transmit from person-to-person, then a pandemic can result.



Intensive farms concentrate large numbers of animals close together. They also tend to be concentrated in specific geographic areas. They may be close to large cities that they supply or in regions where cereal crops, used for poultry and pig feed, are cultivated. Intensive poultry and pig units are often concentrated in the same area⁵⁹, potentially enhancing the risk of transmission of avian influenza to pigs, in which reassortment may occur. Transport of live pigs over long distances facilitates the mixing of swine influenza viruses that can lead to multiple reassortments and give rise to new pandemics⁶⁰.

Housing animals indoors may reduce the risk of a new virus being spread on the wind and introduced into a facility from other facilities, or wild birds in the vicinity; however, once a virus is inside an animal house, crowding of animals will facilitate animal-to-animal transmission⁶¹. Also, stress can have a negative impact on the ability of animals to raise a robust immune response to infection. Influenza viruses are inactivated by exposure to the ultraviolet rays in sunshine so the virus may survive for longer indoors⁶². Unless there is an extremely efficient ventilation system, there will be a greater accumulation of virus in the indoor environment. Testing of air samples during an outbreak has shown that the virus can be found in the air outside infected barns⁶³.

It is often assumed that large commercial units are more likely to have stringent disease prevention measures, in part because of the greater risks of disease spread associated with intensive farming. However, studies have called this assumption into question. A thorough analysis of data from Thailand suggests that commercial poultry production is not associated with any reduction in risk of H5N1 HPAI occurring compared with backyard farms⁶⁴.

Modern large pig herds are maintained by the frequent introduction of young animals. A consequence of this is that whereas swine influenza in the US was a seasonal disease, like human influenza, there is now year-round transmission in pigs⁶⁵. This creates a constant opportunity for infection of stockpeople, who in turn may spread infection to the wider population. The likelihood of this may be enhanced in intensive farm units where contracted labourers are employed. They travel from their homes, often in larger communities, to work on the farm, potentially increasing the interactions between farm workers and other members of the general population⁶⁶.

Human pandemics

When a new influenza virus emerges that can be transmitted easily between people, the resulting pandemic can have very serious impacts and, in some cases, cause large numbers of deaths. The most notable example in human history is that of the 'Spanish flu' pandemic during 1918-19, which is estimated to have killed 50 million people⁶⁷. A further two viral pandemics occurred in the 20th Century: one caused by an H2N2 virus in 1957 and one by an H3N2 virus in 1968⁶⁸. These two pandemic viruses appear to have arisen by reassortment between avian and pre-existing human viruses⁶⁹.

Multiple reassortment events taking place in pigs gave rise to the first human pandemic of the 21st Century. Initially termed 'swine-origin' H1N1, the virus that was later declared to be a pandemic H1N1 virus first emerged in Mexico in 2009.

Control options and future prospects

There are numerous biosecurity procedures that can be adopted to minimise the risk of influenza, although none totally eliminate it. Heightened surveillance of people working with poultry and pigs could enable early detection of an emerging potential pandemic virus. Vaccination of people working in intensive poultry and pig units (including veterinarians) against a potential influenza pandemic has been proposed⁷⁰. However, it is difficult to predict exactly what will be the next pandemic virus and vaccinating a farm worker with a strain that provides only partial immunity could lead to infection without any clinical signs. This increases the risk that they continue with their daily lives and pass on the infection to others. Vaccinating farm workers against regular seasonal human influenza minimises the risk of reassortment of human and animal influenza strains.

Poultry flocks can be vaccinated against influenza, and increasingly this is practised in some areas^{71, 72}. Vaccination of pigs has also become increasingly widespread. However, as with vaccination of people, use of an imperfect vaccine may mean that infection occurs without clinical signs, leading to unseen transmission. It is important to monitor vaccination programmes adequately and to update vaccine strains as necessary. There is concern that widespread use of vaccination could potentially drive the selection of variant viruses⁷³.

There is a fear that antiviral drugs available for treating influenza in people may be being mis-used in poultry, thus leading to the emergence of drug-resistant strains. Drugs may then become ineffective for the treatment of human infections from H5N1 virus in the future.

CONCLUSIONS

The industrialisation of livestock farming has led to a dramatic increase in the number of animals, especially poultry and pigs, reared for food. This has been accompanied by an equally dramatic rise in our consumption of meat, particularly chicken meat. This increased consumption leads to more opportunities for exposure to foodborne pathogens and is consistent with the increased number of cases reported. Chicken meat, and products like hamburgers made from minced or ground meat, pose a greater risk because pathogens are not restricted to the surface of the food and may be better able to survive cooking.

The crowding together of large numbers of animals at high stocking densities can facilitate the spread of disease. In addition, animals reared intensively may be more susceptible to infection due to immunosuppression. This is the result of chronic stress induced by the production conditions and/or the use of animals highly selected for rapid growth rates. In many cases, these factors appear to lead to a greater risk of infection in intensive systems, despite the potentially greater risk of exposure to bacteria and viruses from the natural environment in animals reared outdoors.

It appears that the risk of *Salmonella* and *E. coli* infection is often greater in intensive production conditions. *Campylobacter* levels in chicken is a serious concern for human health and the application of biosecurity measures in higher welfare indoor systems, with lower stocking densities and slower-growing birds

may be successful in reducing the risk. Further research is urgently needed to clarify the implications of animal breeds for the risks associated with *Campylobacter*, and to avoid solutions being put forward that have negative consequences for human health as well as animal welfare.

The explosion in farm animal numbers, along with the geographical concentration of large-scale poultry and pig production and the transport of animals over long distances, facilitates the emergence of new strains of influenza viruses that can give rise to human pandemics, with potentially devastating consequences.

Zoonotic diseases carried by farmed animals pose a major threat to public health and animal welfare.

Important tools in the battle against zoonotic diseases include:

- Using animal breeds, diets and management conditions that minimise stress and optimise animal welfare and immunity.
- Limiting transport times.
- Surveillance, vaccination programmes and increased food hygiene procedures.



Photo © iStockphoto

Long distance transport increases the risk of zoonotic diseases.

POLICY RECOMMENDATIONS

Governments, Inter-Governmental Organisations (IGOs) and the food production industry must urgently work together to implement the following recommendations:

- **Ensure health** – by developing farming policies for humane sustainable food supplies that ensure the health of animals and people. This includes using animal breeds, diets and management conditions that minimise stress and optimise animal welfare and immunity.
- **Surveillance and vaccination** – helping minimise the spread of disease.
- **Limit transportation time** – ensuring animals are slaughtered humanely on or near to the farm where they were raised.
- **Invest in research and knowledge transfer** – helping support farmers to develop and implement higher welfare livestock systems.
- **Reduce non-therapeutic antibiotic use** – limiting the risk of antibiotic resistance.
- **Encourage consumers to eat less and higher welfare meat** – reducing the risk of exposure to food infected with *Salmonella*, *Campylobacter* or *E. coli*.

“Animals need, and deserve, to be in higher welfare farming systems. This report shows that intensive farming is not just bad for animals, it is also bad for our health. The risk of food poisoning is already a real problem and the potential for an influenza pandemic risks causing serious devastation. We need humane sustainable agriculture to secure healthy food now and in the future.”

**Dil Peeling, BVSc MSc MRCVS
Director of Campaigns, Compassion in World Farming**

“In case after case, conditions that are bad for welfare such as close confinement and high stocking densities are associated with dangerous diseases, while higher welfare systems such as grazing are as good or better for animal health. Safeguarding farm animal and human health is best achieved by safeguarding other aspects of animal welfare.”

**Dr. Michael C. Appleby, PhD
Chief Scientific Adviser, World Society for the Protection of Animals**

REFERENCES

- ¹ Karmali, M.A., Gannon, V. & Sargeant, J.M. (2010) Verocytotoxin-producing *Escherichia coli* (VTEC). *Veterinary Microbiology*, **140**: 360-370.
- ² Pennington, H. (2010) *Escherichia coli* O157. *Lancet*, **376**: 1428-1435.
- ³ Wales, A.D., Woodward, M.J. & Pearson, G.R. (2005) Attaching-effacing bacteria in animals. *Journal of Comparative Pathology*, **132**: 1-26.
- ⁴ Karmali, M.A., Gannon, V. & Sargeant, J.M. (2010) Verocytotoxin-producing *Escherichia coli* (VTEC). *Veterinary Microbiology*, **140**: 360-370.
- ⁵ Pennington, H. (2010) *Escherichia coli* O157. *Lancet*, **376**: 1428-1435.
- ⁶ Callaway, T.R., Carr, M.A., Edrington, T.S., Anderson, R.C. & Nisbet, D.J. (2009) Diet, *Escherichia coli* O157:H7, and cattle: a review after 10 years. *Current Issues in Molecular Biology*, **11**: 67-79.
- ⁷ Brown-Brandl, T.M., Berry, E.D., Wells, J.E., Arthur, T.M. & Nienaber, J.A. (2009) Impacts of individual animal response to heat and handling stresses on *Escherichia coli* and *E. coli* O157:H7 fecal shedding by feedlot cattle. *Foodborne Pathogens and Disease*, **6**: 855-864.
- ⁸ Gott, H., Beckett, W., Turner, R. & Leopard, D. (2011) *Hawksmoor at home*. Preface Publishing, London.
- ⁹ Elder, R.O., Keen, J.E., Siragusa, G.R., Barkocy-Gallagher, G.A., Koohmaraie, M. & Laegreid, W.W. (2000) Correlation of enterohemorrhagic *Escherichia coli* O157 prevalence in feces, hides, and carcasses of beef cattle during processing. *Proceedings of the National Academy of Sciences of the United States of America*, **97**: 2999-3003.
- ¹⁰ Walker, C., Shi, X., Sanderson, M., Sargeant, J. & Nagaraja, T.G. (2010) Prevalence of *Escherichia coli* O157:H7 in gut contents of beef cattle at slaughter. *Foodborne Pathogens and Disease*, **7**: 249-255.
- ¹¹ Elder, R.O., Keen, J.E., Siragusa, G.R., Barkocy-Gallagher, G.A., Koohmaraie, M. & Laegreid, W.W. (2000) Correlation of enterohemorrhagic *Escherichia coli* O157 prevalence in feces, hides, and carcasses of beef cattle during processing. *Proceedings of the National Academy of Sciences of the United States of America*, **97**: 2999-3003.
- ¹² Pennington, H. (2010) *Escherichia coli* O157. *Lancet*, **376**: 1428-1435.
- ¹³ Stevens, M.P., Roe, A.J., Vlisidou, I., et al. (2004) Mutation of *tox B* and a truncated version of the *efa-1* gene in *Escherichia coli* O157:H7 influences the expression and secretion of locus of enterocyte effacement-encoded proteins but not intestinal colonization in calves or sheep. *Infection and Immunity*, **72**: 5402-5411.
- ¹⁴ Allen, K.J., Rogan, D., Finlay, B.B., Potter, A.A. & Asper, D.J. (2011) Vaccination with type III secreted proteins leads to decreased shedding in calves after experimental infection with *Escherichia coli* O157. *Canadian Journal of Veterinary Research*, **75**: 98-105.
- ¹⁵ Vilte, D.A., Larzabal, M., Garbaccio, S. et al. (2011) Reduced faecal shedding of *Escherichia coli* O157:H7 in cattle following systemic vaccination with gamma intimin C and EspB proteins. *Vaccine*, **29**: 3962-3968.
- ¹⁶ Zhang, X.S. & Woolhouse, M.E. (2011) *Escherichia coli* O157 infection on Scottish cattle farms: dynamics and control. *Journal of the Royal Society, Interface / The Royal Society*, **8**: 1051-1058.
- ¹⁷ Tivendale, K.A., Logue, C.M., Kariyawasam, S. et al. (2010) Avian-pathogenic *Escherichia coli* strains are similar to neonatal meningitis *E. coli* strains and are able to cause meningitis in the rat model of human disease. *Infection and Immunity*, **78**: 3412-3419.
- ¹⁸ EFSA Panel on Biological Hazards (2011) Scientific Opinion on *Campylobacter* in broiler meat production: control options and performance objectives and/or targets at different stages of the food chain. *EFSA Journal*, **9**(4): 2105.
- ¹⁹ Hillers, V.N., Medeiros, L., Kendall, P., Chen, G., DiMascola, S. (2003) Consumer food-handling behaviors associated with prevention of 13 foodborne illnesses. *Journal of Food Protection*, **66**:1893-1899.
- ²⁰ Patriarchi, A., Fox, A., Maunsell, B., Fanning, S., Bolton, D. (2011) Molecular characterization and environmental mapping of *Campylobacter* isolates in a subset of intensive poultry flocks in Ireland. *Foodborne Pathogens and Disease*, **8**: 99-108.
- ²¹ Allen, V.M., Weaver, H., Ridley, A.M., Harris, J.A., Sharma, M., Emery, J., Sparks, N., Lewis, M. & Edge, S. (2008) Sources and spread of thermophilic *Campylobacter* spp. during partial depopulation of broiler chicken flocks. *Journal of Food Protection*, **71**: 264-70.
- ²² Northcutt, J.K., Buhr, R.J., Berrang, M.E., Fletcher, D.L. (2003) Effects of replacement finisher feed and length of feed withdrawal on broiler carcass yield and bacteria recovery. *Poultry Science*, **82**:1820-4.
- ²³ Northcutt, J.K., Berrang, M.E., Dickens, J.A., Fletcher, D.L., Cox, N.A. (2003) Effect of broiler age, feed withdrawal, and transportation on levels of coliforms, *Campylobacter*, *Escherichia coli* and *Salmonella* on carcasses before and after immersion chilling. *Poultry Science*, **82**:169-73.
- ²⁴ Cogan, T.A., Thomas, A.O., Rees, L.E., Taylor, A.H., Jepson, M.A., Williams, P.H., Ketley, J. & Humphrey, T.J. (2007) Norepinephrine increases the pathogenic potential of *Campylobacter jejuni*. *Gut*, **56**: 1060-1065.

- ²⁵ Stern, N.J., Clavero, M.R., Bailey, J.S., Cox, N.A. & Robach, M.C. (1995) *Campylobacter* spp. in broilers on the farm and after transport. *Poultry Science*, **74**: 937-941.
- ²⁶ Ellis-Iversen, J., Ridley, A., Morris, V., Sowa, A., Harris, J., Atterbury, R., Sparks, N. & Allen, V. (2012) Persistent environmental reservoirs on farms as risk factors for *Campylobacter* in commercial poultry. *Epidemiology and Infection*, **140**:916-924.
- ²⁷ Shreeve, J.E., Toszeghy, M., Pattison, M. & Newell, D.G. (2000). Sequential spread of *Campylobacter* infection in a multipen broiler house. *Avian Disease*, **44**: 983-988.
- ²⁸ Cogan, T.A., Thomas, A.O., Rees, L.E., Taylor, A.H., Jepson M.A, Williams, P.H., Ketley, J. & Humphrey, T.J. (2007) Norepinephrine increases the pathogenic potential of *Campylobacter jejuni*, *Gut*, **56**: 1060-1065.
- ²⁹ Pandiri, A.R., Gimeno, I.M., Mays, J.K., Reed, W.M. & Fadly, A.M. (2012) Reversion to subgroup J avian leukosis virus viremia in seroconverted adult meat-type chickens exposed to chronic stress by adenocorticotrophin treatment. *Avian Disease*, **56**(3): 578-582.
- ³⁰ Bull, S.A., Thomas, A., Humphrey, T., Ellis-Iversen, J., Cook, A.J., Lovell, R. & Jorgensen, F. (2008) Flock health indicators and *Campylobacter* spp. In commercial housed broilers reared in Great Britain. *Applied and Environmental Microbiology*, **74**: 5408-5413.
- ³¹ Gibbens, J.C., Pascoe, S.J., Evans, S.J., Davies, R.H. & Sayers, A.R. (2001) A trial of biosecurity as a means to control *Campylobacter* infection of broiler chickens. *Preventive Veterinary Medicine*, **48**: 85-99.
- ³² EFSA (2010) Scientific Opinion on a Quantitative Microbiological Risk Assessment of *Salmonella* in slaughter and breeder pigs. *EFSA Journal*, **8**: 1547.
- ³³ Chappell, L., Kaiser, P., Barrow, P., Jones, M.A., Johnston, C. & Wigley, P. (2009) The immunobiology of avian systemic salmonellosis. *Veterinary Immunology and Immunopathology*, **128**:53-59.
- ³⁴ Wales, A.D., Cook, A.J. & Davies, R.H. (2011) Producing *Salmonella*-free pigs: a review focusing on interventions at weaning. *The Veterinary Record*, **168**: 267-276.
- ³⁵ Hughes, L.A., Shopland, S., Wigley, P., et al. (2008) Characterisation of *Salmonella enterica* serotype Typhimurium isolates from wild birds in northern England from 2005 - 2006. *BMC veterinary research*, **4**: 4.
- ³⁶ Van Hoorebeke, S., Van Immerseel, F., De Vylder, J., et al. (2010) The age of production system and previous *Salmonella* infections on-farm are risk factors for low-level *Salmonella* infections in laying hen flocks. *Poultry Science*, **89**: 1315-1319.
- ³⁷ Van Hoorebeke, S., Van Immerseel, F., Schulz, J., et al. (2010) Determination of the within and between flock prevalence and identification of risk factors for *Salmonella* infections in laying hen flocks housed in conventional and alternative systems. *Preventive Veterinary Medicine*, **94**: 94-100.
- ³⁸ Molbak, K. & Neimann, J. (2002) Risk factors for sporadic infection with *Salmonella enteritidis*, Denmark, 1997-1999. *American Journal of Epidemiology*, **156**: 654-661.
- ³⁹ Namata, H., Meroc, E., Aerts, M., Faes, C., Abrahantes, J.C., Imberechts, H. & Mintiens, K. (2008) *Salmonella* in Belgian laying hens: an identification of risk factors. *Preventive Veterinary Medicine*, **83**: 323-336.
- ⁴⁰ Chemaly, M., Huneau-Salaun, A., Labbe, A., Houdayer, C., Petetin, I. & Fravallo, P. (2009) Isolation of *Salmonella enterica* in laying-hen flocks and assessment of eggshell contamination in France. *Journal of Food Protection*, **72**: 2071-2077.
- ⁴¹ Huneau-Salaun, A., Marianne, C., Sophie, le B., et al. (2009) Risk factors for *Salmonella enterica* subsp. *enterica* contamination in 519 French laying hen flocks at the end of the laying period. *Preventive Veterinary Medicine*, **89**: 51-58.
- ⁴² Schaar, U., Kaleta, E.F. & Baumbach, B. (1997) [Prevalence of *Salmonella enteritidis* and *Salmonella typhimurium* in laying hen flocks battery and on floor housing. Comparative studies using bacteriological and serological demonstration methods]. *Tierärztliche Praxis. Ausgabe G, Grosstiere/ Nutztiere*, **25**: 451-459.
- ⁴³ Mollenhorst, H., van Woudenberg, C.J., Bokkers, E.G. & de Boer, I.J. (2005) Risk factors for *Salmonella enteritidis* infections in laying hens. *Poultry Science*, **84**: 1308-1313.
- ⁴⁴ Van Hoorebeke, S., Van Immerseel, F., Haesebrouck, E., Ducatelle, R. & Dewulf, J. (2011) The influence of the housing system on *Salmonella* infections in laying hens: a review. *Zoonoses and Public Health*, **58**: 304-311.
- ⁴⁵ Holt, P.S. & Porter, R.E. Jr. (1993) Effect of induced molting on the recurrence of a previous *Salmonella enteritidis* infection. *Poultry Science*, **72**: 2069-2078.
- ⁴⁶ Holt, P.S. (2003) Molting and *Salmonella enterica* serovar *enteritidis* infection: the problem and some solutions. *Poultry Science*, **82**: 1008-1010.
- ⁴⁷ Golden, N.J., Marks, H.H., Coleman, M.E., Schroeder, C.M., Bauer, N.E. Jr. & Schlosser, W.D. (2008) Review of induced molting by feed removal and contamination of eggs with *Salmonella enterica* serovar *Enteritidis*. *Veterinary Microbiology*, **131**: 215-228.

- ⁴⁸ Golden, N.J., Marks, H.H., Coleman, M.E., Schroeder, C.M., Bauer, N.E., Jr. & Schlosser, W.D. (2008) Review of induced molting by feed removal and contamination of eggs with *Salmonella enterica* serovar Enteritidis. *Veterinary Microbiology*, **131**: 215-228.
- ⁴⁹ Davies, P.R. (2011) Intensive swine production and pork safety. *Foodborne Pathogens and Disease*, **8**: 189-201.
- ⁵⁰ Pullinger, G.D., van Diemen, P.M., Carnell, S.C., Davies, H., Lyte, M. & Stevens, M.P. (2010) 6-hydroxydopamine-mediated release of norepinephrine increases faecal excretion of *Salmonella enterica* serovar Typhimurium in pigs. *Veterinary Research*, **41**: 68.
- ⁵¹ Verbrugge, E., Boyen, F., Van Parys, A., et al. (2011) Stress induced *Salmonella* Typhimurium recrudescence in pigs coincides with cortisol induced increased intracellular proliferation in macrophages. *Veterinary Research*, **42**: 118.
- ⁵² EFSA (2010) Quantitative estimation of the public health impact of setting a new target for reduction of *Salmonella* in laying hens. *EFSA Journal*, **8**: 1546.
- ⁵³ Davies, P.R. (2011) Intensive swine production and pork safety. *Foodborne Pathogens and Disease*, **8**: 189-201.
- ⁵⁴ EFSA (2010) Scientific Opinion on a Quantitative Microbiological Risk Assessment of *Salmonella* in slaughter and breeder pigs. *EFSA Journal*, **8**: 1547.
- ⁵⁵ Barrow, P.A. (2007) *Salmonella* infections: immune and nonimmune protection with vaccines. *Avian Pathology: Journal of the W.V.P.A.*, **36**: 1-13.
- ⁵⁶ Beal, R.K., Wigley, P., Powers, C., Hulme, S.D., Barrow, P.A. & Smith, A.L. (2004) Age at primary infection with *Salmonella enterica* serovar Typhimurium in the chicken influences persistence of infection and subsequent immunity to re-challenge. *Veterinary Immunology and Immunopathology*, **100**: 151-164.
- ⁵⁷ Van Reeth, K. (2007) Avian and swine influenza viruses: our current understanding of the zoonotic risk. *Vet Research*, **38**(2): 243- 260.
- ⁵⁸ Webster, R. G., Sharp, G. B. et al. (1995) Interspecies transmission of influenza viruses. *American Journal of Respiratory and Critical Care Medicine*, **152**: 25-30.
- ⁵⁹ Otte, J., Roland-Holst, D. et al. (2007) Industrial livestock production and global health risks. Food and Agriculture Organization of the United Nations, *Pro-Poor Livestock Policy Initiative Research Report*.
- ⁶⁰ Smith, G. J., Vijaykrishna, D. et al. (2009) Origins and evolutionary genomics of the 2009 swine-origin H1N1 influenza A epidemic. *Nature*, **459**(7250): 1122-1125.
- ⁶¹ Gilchrist, M. J., Greko, C. et al. (2007) The potential role of concentrated animal feeding operations in infectious disease epidemics and antibiotic resistance. *Environ Health Perspect*, **115**(2): 313-316.
- ⁶² Weber, T. P. & Stilianakis, N. I. (2008) Inactivation of influenza A viruses in the environment and modes of transmission: a critical review. *Journal of Infection*, **57**(5): 361-373.
- ⁶³ Power, C.A. (2005) An investigation into the potential role of aerosol dispersion of dust from poultry barns as a mode of transmission during an outbreak of avian influenza (H7:N3) in Abbotsford, BC in 2004. *Bulletin of the Aquaculture Association of Canada*, **105**: 7-14.
- ⁶⁴ Graham, J. P., Leibler, J. H. et al. (2008) The animal-human interface and infectious disease in industrial food animal production: rethinking biosecurity and biocontainment. *Public Health Rep*, **123**(3): 282-299.
- ⁶⁵ Myers, K. P., Olsen, C. W. et al. (2006) Are swine workers in the United States at increased risk of infection with zoonotic influenza virus? *Clinical Infectious Diseases*, **42**(1): 14-20.
- ⁶⁶ Gray, G. C., Trampel, D. W. et al. (2007) Pandemic influenza planning: shouldn't swine and poultry workers be included? *Vaccine*, **25**(22): 4376-4381.
- ⁶⁷ Taubenberger, J. K. & Morens, D. M. (2006). 1918 Influenza: the mother of all pandemics. *Emerging Infectious Diseases*, **12**(1): 15-22.
- ⁶⁸ Kilbourne, E. D. (2006) Influenza pandemics of the 20th century. *Emerging Infectious Diseases*, **12**(1): 9-14.
- ⁶⁹ Kawaoka, Y., Krauss, S. et al. (1989). Avian-to-human transmission of the PB1 gene of influenza A viruses in the 1957 and 1968 pandemics. *Journal of Virology*, **63**(11): 4603-4608.
- ⁷⁰ Saenz, R. A., Hethcote, H. W. et al. (2006). Confined animal feeding operations as amplifiers of influenza. *Vector Borne Zoonotic Diseases*, **6**(4): 338-346.
- ⁷¹ Peiris, J. S., de Jong, M. D. et al. (2007). Avian influenza virus (H5N1): a threat to human health. *Clinical Microbiology Review*, **20**(2): 243-267.
- ⁷² Swayne, D. E. & Kapczynski, D. (2008). Strategies and challenges for eliciting immunity against avian influenza virus in birds. *Immunology Review*, **225**: 314-331.
- ⁷³ Wuetrich, B. (2003). *Chasing the fickle swine flu*. *Science*, **299**: 1503-1504.

ACKNOWLEDGEMENTS

This report is based on longer reports by Dr Paul Wigley* on 'Salmonella in poultry and pig production' and 'Zoonotic *Escherichia coli* in cattle production and other livestock'; Professor Tom Humphrey* on 'Campylobacter in poultry' and Dr Janet Daly** on 'Avian Influenza' and 'Swine Influenza'.

*Institute for Infection and Global Health, University of Liverpool
** Faculty of Medicine & Health Sciences, The University of Nottingham

Funding for this research has been provided by a partnership of three organisations:
Compassion in World Farming, The Tubney Charitable Trust and the World Society for the Protection of Animals.
This report is available to download from ciwf.org/ZoonoticDiseases

May 2013.



River Court, Mill Lane,
Godalming, Surrey, GU7 1EZ, UK
Email: research@ciwf.org
Tel: +44 (0) 1483 521 950
Web: ciwf.org

Registered Charity No. 1095050.
Printed on 100% recycled paper.



5th floor, 222 Grays Inn Road,
London WC1X 8HB, UK
Email: wspa@wspa-international.org
Tel: +44 (0) 207 239 0500
Web: wspa-international.org

Registered Charity No. 1081849.