



FACTORY FARMS

A BREEDING GROUND FOR DISEASE

The pandemic threat of animal agriculture
By Dr Justine Butler, Viva!



Viva!

FACTORY FARMS – A BREEDING GROUND FOR DISEASE

The pandemic threat of animal agriculture

By Dr Justine Butler, Viva!

©Viva! 2022

Published by: Viva!, 8 York Court, Wilder Street, Bristol BS2 8QH

(+44) 0117 944 1000 | viva.org.uk | info@viva.org.uk

Registered charity 1037486

CONTENTS

Introduction	4
Factory farms – a largescale problem.....	5
Bird flu – waiting in the wings	8
Swine flu – piggy in the middle.....	13
BSE and Creutzfeldt-Jakob disease	16
Bovine TB – badgers are not the problem.....	18
Nipah virus – how pig farming provided a pathway to disease	20
Covid-19, Sars and Mers – driven by the hunger for meat	22
Antibiotic resistance – superbugs on the rise.....	26
<i>E. coli</i> and the deadly threat from O157	32
<i>Staphylococcus aureus</i> and MRSA	35
<i>Salmonella</i> – are eggs really safe?	38
<i>Campylobacter</i> – top of the food poisoning table	41
Final warning	45
References	47

INTRODUCTION

Factory farms provide the perfect breeding ground for disease. Animals are raised in closed, filthy, stressful and crowded, industrial facilities with little or no natural light. Their immunity is low as they have been bred for fast growth. It's the perfect setting for viruses and bacteria to mutate and spread. In fact, you would be hard pushed to provide a more ideal environment than a modern factory farm.

As the insatiable global demand for meat and dairy increases, so does the scale of animal agriculture, with the number of factory farms – and the number of animals confined in them – rising at ever-increasing levels. Alongside that, infectious diseases are emerging globally at an unprecedented rate (Rohr *et al.*, 2019). The rise in factory farms and diseases are directly connected: “Agricultural intensification has been proposed as a major underlying cause of pathogen emergence from wildlife and domestic animal populations into human populations” (Pulliam *et al.*, 2012).

Scientists have been warning us for years that the next pandemic could come from a factory farm. We've already seen BSE, bovine TB, foot and mouth, avian influenza (bird flu), swine flu, *Campylobacter*, *Salmonella*, antibiotic-resistant superbugs and more coming from factory farms. The next serious outbreak of an infectious disease leading to a pandemic may be a bird or swine flu virus or another coronavirus. It may be an antibiotic resistant superbug or some other previously unseen infectious disease. We are playing a dangerous game of Russian roulette and factory farming lies at the heart of it.

Factory farms play a critical role not only in the spread of diseases from animals to humans but also in the amplification of diseases. In other words, factory farms act as a Petri dish, providing an ideal environment for bacteria and viruses to mutate and develop into something more sinister – this has already happened and continues to do so all over the world.

The 2009 swine flu pandemic arose from a pig farm and antibiotic-resistant superbugs may kill more people than cancer by 2050 unless we stop factory farming now. There is no time to waste, three in four new infectious diseases come from animals and the frequency of their emergence is increasing. Viva!'s ground-breaking *Zoonoses* report investigates some of these zoonotic diseases, such as Covid-19, that have jumped from animals to humans. It includes measles, Ebola, AIDS, SARS, MERS, bird flu and antibiotic-resistant superbugs. Find out where they came from and what role wet markets and factory farms play and we could avert another pandemic.

We have an opportunity to change the way we eat, the way we treat animals and the way we treat the planet. Going vegan is the only way to remove the reservoir for disease. It would benefit our health, the environment and the animals too. A truly One Health approach for the planet!

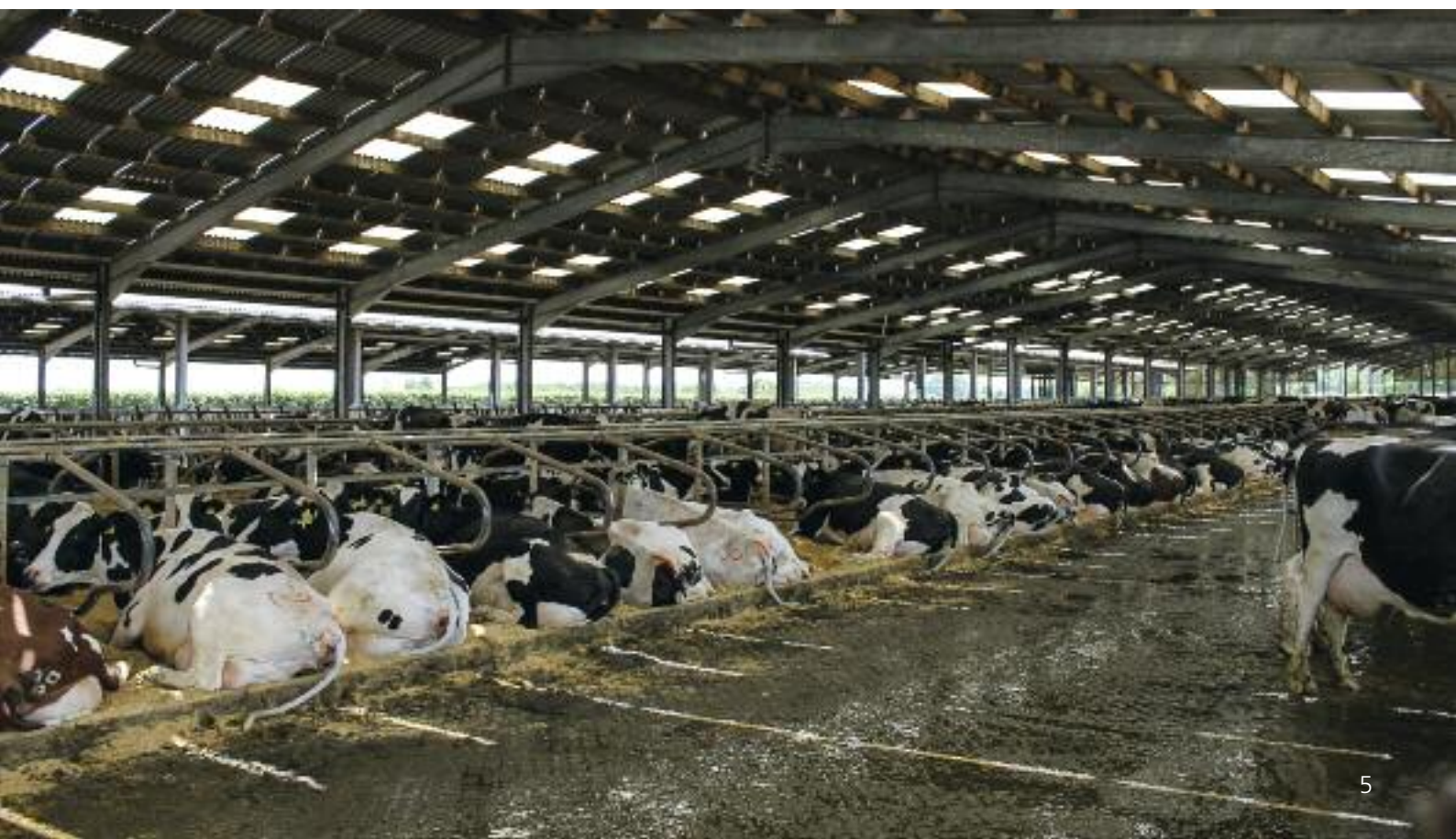
FACTORY FARMS – A LARGESCALE PROBLEM

Today, livestock accounts for 60 per cent of all mammal biomass on the planet. Cattle are the most abundant, far surpassing the biomass of all wild mammals combined, which make up a paltry four per cent (Bar-On *et al.*, 2018). There are 26 billion chickens in the world – more than three birds for every single person on the planet. Most farmed animals in the UK and many other countries are reared intensively in huge numbers: “In industrialised countries, the vast majority of chickens and turkeys are now produced in houses in which 15,000-70,000 birds are confined throughout their lifespan. Increasingly, pigs and cattle are also raised under similar conditions of confinement and high density” (Leibler *et al.*, 2009). These large factory farms increase the risk of disease: “Intensification of livestock production, especially pigs and poultry, facilitates disease transmission by increasing population size and density” (Jones *et al.*, 2013).

These highly intensified systems are a far-cry from the bucolic image of cows grazing on hilly pastures. Most farmed animals are confined, many in their thousands, in giant metal sheds condemned to a life where they will never feel the grass under their feet or the sun on

their face: “Thousands of animals of similar genotypes are raised for one purpose (such as pigs, layer hens, broiler chickens, ducks and turkeys) with rapid population turnover at one site under highly controlled conditions, often in confined housing, with nutrient dense and artificial feeds replacing access to forage crops” (Leibler *et al.*, 2009).

Experts at the European Medicines Agency (EMA) and European Food Safety Authority (EFSA) say: “Knowledge on the relationship between stress and animal disease is accumulating.” They say that animals reared in high-stress environments such as factory farms are more susceptible to infectious diseases (EMA and EFSA, 2017). They describe eight stressors that are present in modern livestock production units: heat, cold, crowding, mixing, weaning, limit-feeding, insufficient bedding, noise and restraint. All of these, they say, have been shown to alter the immune system of animals. More recently other stressors such as human-to-animal interaction (handling), a lack of enrichment and live transportation have been added. Taken together, the evidence shows that stress suppresses the immune system in birds and other animals leaving them more susceptible to disease.



FACTORY FARMS – A BREEDING GROUND FOR DISEASE

“On-farm stressors interfere with the normal behaviour of the animals and have been shown to alter the immune system of animals and susceptibility to diseases.”

(EMA and EFSA, 2017).

The “meatification” of agriculture, driven by the rising demand for meat and dairy, has increased the potential for new zoonotic diseases to emerge (Bernstein and Dutkiewicz, 2021). Another study found: “strong evidence that modern farming practices and intensified systems can be linked to disease emergence and amplification” (Jones *et al.*, 2013). Intensive livestock systems, they said, generally have high density populations of low genetic diversity, which may favour increased transmission and adaptation of disease pathogens. They describe how ventilation systems used in intensive livestock farming can spread disease by expelling pathogens such as *Campylobacter* and avian influenza virus into the environment, increasing the risk of transmission to wild and domestic animals.

In industrial farms, pathogens can move by a number of different unregulated and unrecognised pathways, such as on airborne dust, via insects, in animal waste utilised in agriculture and aquaculture, in contaminated water and on wild animals (Graham *et al.*, 2008).

Factory farms produce large quantities of waste that contain a variety of pathogens capable of surviving for months if left untreated. Open cesspits of pig waste (lagoons) may attract wild birds and waste spread on

land can come into contact with wild animals and contaminate water. Similarly, the use of animal waste in aquaculture leads to potential contact with wild birds (Jones *et al.*, 2013).

The international trade of live animals and animal-based food products is also contributing to the pandemic risk posed by food systems. Scientists say that: “the globalised nature of the food animal production industry and supply chain must be recognised for its role in augmenting rapid transmission of pathogens across long distances” (Leibler *et al.*, 2009).

Writing in Nature’s *Scientific Reports*, scientists from several US organisations presented a picture of the animal-human transmission networks likely to perpetuate future disease emergence. Their model (see below) shows how viruses with high host plasticity (a wide host range) are able to move between species and when domesticated animals become infected, the risk of human infection increases. They said: “Most human infectious diseases, especially recently emerging pathogens, originate from animals and ongoing disease transmission from animals to people presents a significant global health burden” (Kreuder *et al.*, 2015).

While some viruses jump from wild animals to humans, when their habitats are encroached upon for example, Kreuder *et al.* highlight the critical role farmed animals such as pigs and poultry play via direct contact with people, as well as the amplification of disease transmission in intensive animal production facilities (Kreuder *et al.*, 2015).

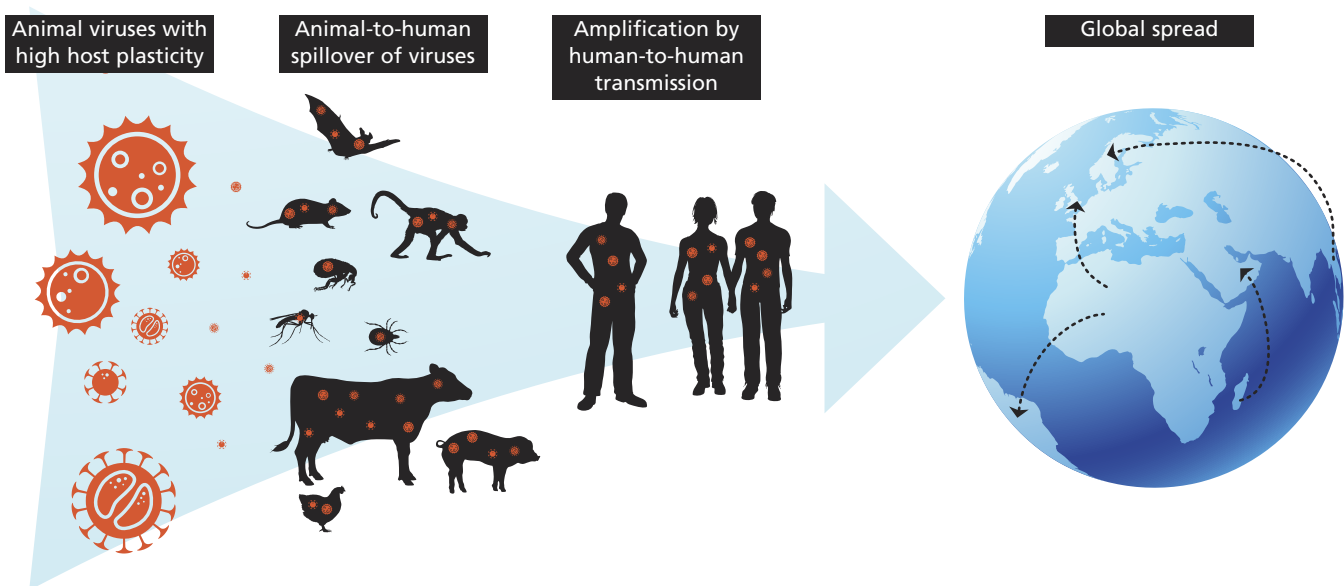


Figure 1. Pandemic potential of zoonotic viruses that spill over from animals to humans and spread by secondary transmission among humans.

Source: Kreuder *et al.*, 2015.



So, for the many disease pathogens (bacteria and viruses) shared by wildlife and farmed animals, it is the latter that play a critical role in facilitating direct contact with people, as well as providing an opportunity for the amplification of disease transmission in intensive animal production facilities.

Researchers from Johns Hopkins University in the US say that: “Many emerging infectious diseases in human populations are associated with zoonotic origins. Attention has often focused on wild animal reservoirs, but most zoonotic pathogens of recent concern to human health either originate in, or are transferred to, human populations from domesticated animals raised for human consumption.” They highlight the often-overlooked threat posed by factory farms: “Although not typically recognised as such, industrial food animal production generates unique ecosystems – environments that may facilitate the evolution of zoonotic pathogens and their transmission to human populations” (Leibler *et al.*, 2009).

Intensive agriculture is associated with more than a quarter of all infectious diseases and more than half of all zoonotic diseases.

A survey of the current research found that since 1940, intensive agriculture was associated with more than a quarter of all infectious diseases that emerged in humans and more than half of all zoonotic infectious diseases (that leapt from animals to humans) and suggested that these numbers will likely increase as more and more land around the world is converted to agricultural use (Rohr *et al.*, 2019).

Lead author Jason Rohr, Professor of Biological Sciences at the University of Notre Dame, said that certain steps can be taken to mitigate the proliferation of these pathogens: “Because we share fewer pathogens with plants, decreasing meat consumption might help slow the transfer of these zoonotic diseases. We already have enough food on the planet to feed everyone.” Rohr says, “the distribution of the food to the people who need it is a major issue.”

One of the main drivers of climate change – deforestation, and the conversion of former forests to intensive agriculture – is fuelling zoonotic transfer. Which means that the case for protecting and restoring the world’s wild landscapes is not just to mitigate climate change – it’s also to prevent epidemics before they even start (Ajani, 2020).

Scientists describe how industrial systems can increase animal and public health risks: “Domesticated animals raised to produce human food play a significant role in the emergence of zoonotic pathogens through two primary mechanisms: as a bridge between wild animal reservoirs and human populations, and as the locus of pathogen evolution itself” (Leibler *et al.*, 2009). This means that factory farms not only provide a pathway between animals and humans for disease, but the means for that disease to develop into something far deadlier too. We are literally handing these bugs a perfect opportunity to evolve into deadlier forms and spread to humans. An invitation to do their worst.

The science couldn’t be more clear in demonstrating how factory farms are breeding grounds for disease – they are the absolute opposite of social distancing!

BIRD FLU – WAITING IN THE WINGS

There are four types of influenza viruses: A, B, C and D.

Bird Flu is caused by influenza A viruses that do not normally infect people but have the potential to do so, and that is the worry. Most human infections with avian influenza A viruses have occurred following direct or close contact with infected poultry leading to concerns that a bird flu virus from a factory farm may cause the next pandemic.

Influenza A viruses are the only ones known to cause flu pandemics. A pandemic can occur when a new and different influenza A virus emerges that both infects people and can spread easily between them.

Influenza A viruses are divided into subtypes based on two proteins on the surface of the virus: hemagglutinin (H) and neuraminidase (N). There are 18 different H subtypes (H1 to H18) and 11 different N subtypes (N1 to N11). The H subtype determines the ability of the virus to bind to and enter cells, where multiplication of the virus then occurs. The N subtypes are responsible for the release of newly formed viruses from the cells. Some subtypes have low pathogenicity (the capacity to cause disease) and others have high pathogenicity.

While there are potentially 198 different influenza A subtype combinations, only 131 have been detected in nature. Only two influenza A subtypes are currently in general circulation among people: H1N1 and H3N2. Influenza A viruses can infect other species – including ducks, chickens, pigs, whales, horses, seals and cats.

Influenza B is only found in humans and causes seasonal flu outbreaks but not pandemics. During the flu season, type A and B viruses (and different subtypes of A) circulate and cause flu in people.

Influenza type C infections generally cause mild illness and are not thought to cause human flu epidemics.

Influenza D viruses primarily affect cattle and are not known to infect or cause illness in people.

Source: CDC, 2019.

Avian influenza (bird flu) viruses are among the most dangerous viruses that can affect humans, with a case fatality rate ranging from around 30 to 60 per cent. Seasonal flu kills around 0.1 per cent (one in 1,000) of those infected. In an average year, this means 7,000 to 9,000 people in the UK die of complications caused by seasonal flu. In a bad year, more than 20,000 people may die.

Some strains of bird flu can spread easily between birds, causing illness with a high death rate among poultry populations. In some cases, they can lead to severe illness and deaths in humans too, usually where there has been close contact with infected birds. While it is still relatively difficult for people to catch bird flu viruses from other people, there have been a limited number of reports of person-to-person infection. If a bird flu virus mutates and becomes as easy to catch as a common cold, seasonal flu or Covid-19, we will be in deep trouble.

Bird flu is a classic example of a zoonotic disease and most pandemics can be traced back to viruses containing genes of avian origin, including the 1918 Spanish flu, 1957 Asian flu, 1968 Hong Kong flu and 2009 swine flu pandemics. Prior to Covid-19, most scientists thought the next pandemic would be caused by an avian influenza virus emerging from a poultry or pig farm.



The viruses responsible for all these pandemics contained genes of avian origin but also contained elements of human-adapted viruses (like the ones that cause seasonal flu). Until recently there was only limited evidence that a wholly avian influenza virus could directly infect humans, but in 1997, 18 people were infected with the avian influenza virus H5N1 and six of them died (Taubenberger, 2006). Chickens in poultry markets were found to be the source of these purely avian H5N1 viruses (Shortridge *et al.*, 1998).

Some records suggest that bird flu, previously known as “fowl plague”, may have been infecting domestic birds as far back as the 1870s. However, it’s widely accepted that it has been infecting poultry at least since the late 1950s (Lycett *et al.*, 2008). Bird flu viruses are described as being low or highly pathogenic depending on how lethal they are to chickens. From 1959 to 1995, the emergence of highly pathogenic viruses was recorded on 15 occasions, but losses were minimal. In contrast, between 1996 and 2008, highly pathogenic viruses emerged at least 11 times and four of these outbreaks involved many millions of birds (Alexander and Brown, 2009).

Bird flu became the focus of intense international attention in 1996 when a highly pathogenic strain of H5N1 emerged in farmed geese in the Guangdong province in China, killing more than 40 per cent of the birds it infected. By 1997, it had spread to poultry farms and live-poultry wet markets in Hong Kong – where it infected 18 people, leading to six deaths. To try and stop the outbreak, the Government ordered the slaughter of more than 1.5 million chickens. Writing in the journal *Nature*, science writer Cassandra Willyard said: “On that occasion, there was no pandemic – no more human cases emerged. But in 2004, the World Health Organisation warned that the next pandemic could result in the deaths of up to seven million people worldwide” (Willyard, 2019).

H5N1 re-emerged in 2003. In addition to further human cases in Hong Kong, there were poultry outbreaks in mainland China and other countries in Southeast and East Asia. There have now been in the region of 15,000 poultry outbreaks and tens of millions of birds have died of H5N1 influenza, and hundreds of millions of birds have been slaughtered to limit the spread of disease.

To date, there have been almost 900 laboratory-confirmed cases of H5N1 in humans and 456 people – mostly teenagers and young adults – have died (WHO,

2021). That’s 53 per cent of those infected! Deaths have occurred in Azerbaijan, Bangladesh, Cambodia, Canada, China, Djibouti, Egypt, India, Indonesia, Iraq, Lao People’s Democratic Republic, Myanmar, Nepal, Nigeria, Pakistan, Thailand, Turkey and Vietnam.

The origins of bird flu

Wild water birds (including geese, swans, gulls, terns and sandpipers but especially ducks), are the natural hosts of low pathogenic bird flu viruses. Infection in these birds is not only typically low pathogenic but can be asymptomatic (Lycett *et al.*, 2008). Infected birds shed large quantities of virus in their faeces, saliva and nasal secretions (WHO, 2007) and so it is easily spread between them in water. It is thought that bird flu viruses have coexisted in perfect harmony with their natural hosts for hundreds, if not thousands, of years without substantial change (Webster *et al.*, 2006).

However, when viruses cross the species barrier (spillover) to new hosts, they adapt and change to suit their new host. This may result in a virus becoming more virulent (Kuiken, 2013). So, when a virus jumps from one species to another, it may mutate and become more deadly to its new host. This is exactly what happens when low pathogenic bird flu viruses spread from wild water birds to domestic poultry.

Bird flu viruses can spread through direct contact with secretions from infected birds, via airborne transmission or through faecal contamination of material, feathers or feed (ECDC, 2021). Large amounts of virus are secreted in faeces and may contaminate soil and water supplies. Contaminated equipment, vehicles, feed, cages or clothing – especially shoes – can spread the virus. It may spread between farms via contaminated dust particles or may be carried on other animals such as rodents. In Asia, wet markets or live bird markets, where live birds are sold, can be another source of spread and mixing of different viruses between bird species (ECDC, 2021).

So, low pathogenic viruses from wild waterbirds can spread either directly or indirectly to domestic birds, causing mild to severe disease. In poultry they can evolve into highly pathogenic viruses, causing severe disease and death. From poultry, viruses can be transmitted back to wild birds, where they can circulate asymptotically, or cause disease and death (Verhagen *et al.*, 2021).

Global production of poultry meat has increased rapidly

FACTORY FARMS – A BREEDING GROUND FOR DISEASE

over the last 50 years, growing 15-fold between 1961 and 2019 (FAOSTAT, 2021). In Europe over the last 20 years, poultry production has almost doubled and over the same period, highly pathogenic viruses have been detected in increasing numbers in domestic and wild birds (Verhagen *et al.*, 2021).

According to scientists: “it is the ever increasing poultry industry that provides the reassortment interface between wild and domestic avian species” (Kim *et al.*, 2009). In other words, as the global demand for chicken meat rises, more and larger poultry farms are required. The more farms there are, the higher the risk of contact between wild birds and poultry. The more infections there are, the higher the risk of a virus mutating to one with pandemic potential. Large-scale factory farms provide the perfect environment for these mutating viruses: closed, dimly lit (UV can harm viruses), crowded, stressful and unsanitary conditions. A perfect storm of our own making!

The poultry industry likes to blame the spread of bird flu on migratory birds. However, while wild birds may contribute to the local spread of the virus, human commercial activities, particularly those associated with poultry, are the major factors responsible for the global spread of bird flu (RSPB, 2021).

The 2007 H5N1 outbreak in Sussex, for example, was traced to the trading of hatching eggs, birds and poultry products between the UK and Hungary, highlighting the implications of these transfers for long-distance pathogen transmission (Leibler *et al.*, 2009). The poultry trade has also been implicated in the cross-border spread of H5N1 in Asia and Africa (Kilpatrick *et al.*, 2006).

Scientists say that around 40 per cent of the highly pathogenic H5N1 outbreaks in domestic poultry reported to the World Organisation for Animal Health between late 2005 and early 2007 occurred in poultry units of 10,000 birds (mainly chickens and turkeys) or more (Leibler *et al.*, 2009). However, in many countries, at that time, less than 10 per cent of flocks were that size. Again, providing evidence that factory farms are a breeding ground for disease.

The United Nations’ Scientific Task Force on Avian Influenza and Wild Birds agrees, saying that: “Typically, highly pathogenic avian influenza (HPAI) outbreaks are associated with intensive domestic poultry production.” The UN task force said that highly infectious bird flu viruses are not only transmitted by wild and migratory birds, but are also found on poultry farms, where they can be transmitted to wild animals. They said: “the risk of HPAI virus circulation by poultry production and trade remains significantly high” (Scientific Task Force on Avian Influenza and Wild Birds, 2016).



H5N1: kills one in two

A number of bird flu viruses have infected people including H5N1, H5N6, H5N8, H7N9 and H10N3. H5N1 is of particular concern. The World Health Organisation says that globally, from January 2003 to December 2021, there were 863 cases of human infection with H5N1 reported from 18 countries. The last case was reported in July 2021 (WHO, 2021). Of these 863 cases, 456 were fatal. So, this virus has killed 53 per cent of those infected; one in every two! It's thought that Covid-19 might have a case fatality rate of around one per cent. It's hard to imagine a pandemic killing 50 times more people.

Luckily, H5N1 is not easily caught or spread between humans, yet! There have been a handful of person-to-person infections among families caring for sick relatives. However, in an interview with the BBC, senior public health expert at the World Health Organisation, David Nabarro, warned that should it mutate and become more transmissible, a pandemic caused by H5N1 could result in a death rate of anywhere between five and 150 million people (BBC, 2005).

He's not the only one concerned: "If this virus acquires human-to-human transmissibility with its present fatality rate of 50%, the resulting pandemic would be akin to a global tsunami. If it killed those infected at even a fraction of this rate, the results would be catastrophic" (Webster *et al.*, 2006).

Bird flu in the UK 2021-2022

Bird flu outbreaks continue around the world without any signs of slowing. The UK has experienced an increasing number of outbreaks year on year. Between late October and the end of December 2021, there were 68 outbreaks in captive birds (mainly on commercial poultry farms) around the country, four times the number of outbreaks during the same period in the previous year. Avian influenza prevention zones (AIPZs) were introduced across Great Britain with additional housing measures put in force from November 2021 meaning all bird keepers (whether they have companion birds, commercial flocks or just a few birds in a backyard flock) were required by law to take a range of biosecurity precautions, including housing their birds indoors (Gov.uk, 2021). But it clearly didn't work as the spread of bird flu continued.

Outbreaks reported during the winter of 2021-2022 were caused by a highly pathogenic H5N1 virus. The UK Government said the risk to human health was low.



However, the Northern Ireland Department of Agriculture, Environment and Rural Affairs has warned that: "There is a possibility that were an individual to be infected with human and bird flu viruses there may be the potential for the virus to combine and produce a more dangerous variant which could spread from human to human" (Defra, 2021).

First UK person to catch H5 bird flu

In January 2022 the UK Health Security Agency confirmed avian influenza (bird flu) in a person in the South West of England. It's assumed they caught the virus from infected ducks kept in and around their home. The virus was identified as an H5 type but it's not yet confirmed to be H5N1.

The H5N1 story shows how farmed animals such as poultry can act simultaneously as a host in which a virus mutates into a deadlier form (pathogen evolution) and a vector, from which the virus then spreads to other animals and humans. Scientists say that: "H5N1 demonstrates how a viral challenge emerged from wildlife, adapted to domestic poultry, and after circulating in these populations, acquired limited ability to infect humans" (Leibler *et al.*, 2009).

Mutations leading to deadlier variants

During the Covid-19 pandemic, a number of new variants of the coronavirus emerged that had acquired the ability to spread more easily between people and cause more severe disease, doubling the risk of

FACTORY FARMS – A BREEDING GROUND FOR DISEASE



hospitalisation. When large numbers of people (or animals) are infected with a virus, the likelihood of a random mutation occurring that offers the virus an advantage increases greatly. A simple analogy would be to imagine five people playing on a fruit machine – the chances of one of them winning the jackpot are low. Now imagine 100 million people playing, then consider the fact that there are 26 billion chickens in the world, all providing the potential reservoir for a mutating virus to take a gamble.

How close is H5N1 to becoming a pandemic threat? One study suggests that it might only take five mutations for it to become airborne and able to spread via tiny droplets in a cough or sneeze (Herfst *et al.* 2012). Another suggests just four mutations may be all that is required (Imai *et al.* 2012). A third study, looking at surveillance data, found that two of the five mutations described are already common in H5N1 viruses in nature (Russell *et al.* 2012). So, some viruses might require only three more mutations to become more easily transmissible.

There are numerous examples of bird flu viruses mutating into ones that pose a greater threat. In the early 2000s, the North American H7N2 virus developed an increased affinity for human cell receptors suggesting that the virus had adapted following contact between infectious poultry and humans (Belser *et al.*, 2008).

Low pathogenic bird flu viruses have been seen mutating into highly pathogenic strains – but only ever in chickens (and other poultry), this has never been seen in water-birds. Many such ‘conversion events’ have been reported around the world in commercial poultry farms, representing a potential pandemic threat: “The probability of such a mutation is amplified in the setting of industrial poultry production due to the rapid viral

replication that occurs in an environment of thousands of confined, susceptible animals” (Leibler *et al.*, 2009).

In Mexico in 1994, a low pathogenic H5N2 virus mutated into a highly pathogenic one and spread to Guatemala in 2000 and to El Salvador in 2001, presumably via trade in poultry. Low pathogenic H5N2 is now endemic in domestic poultry populations in Central America (Lee *et al.*, 2004).

In northern Italy, the 1999-2000 H7N1 highly pathogenic epidemic in poultry, one of the most severe in Europe, was preceded by 199 reported outbreaks across poultry farms of low pathogenic H7N1 in the same region. Both the 2003 H7N7 epidemic in the Netherlands and the 2004 H7N3 epidemic in British Columbia, Canada, were caused by highly pathogenic viruses preceded by low pathogenic infections on the same farms. In all three epidemics, authorities noted the high geographic density of poultry farms, frequent contact among farms by trucks, and low levels of biosecurity practised by some operators contributing to the considerable spread of the viruses (Leibler *et al.*, 2009).

Scientists are questioning the wisdom of factory farming in the light of the threat they pose to human health: “food animal production is also inherently an economic activity, driven by financial incentives and profit motives. These economic forces exist in parallel with the biological pressures that moderate pathogen evolution and spread” (Leibler *et al.*, 2009). In other words, we need to rebalance our priorities and choose health over profit.

The best way to take control of the situation would be for huge numbers of people to stop eating poultry, pigs and other animals and remove the viral reservoir of factory farms.

SWINE FLU – PIGGY IN THE MIDDLE

Some researchers are even more worried about pigs than poultry. Professor Gregory Gray, an epidemiologist at Duke University in Durham, North Carolina in the US, describes pigs as ideal “mixing vessels” for flu viruses (Willyard, 2019). This, Gray says, is because pigs are susceptible to infection with flu viruses from other pigs, humans and birds.

If pigs are infected with more than one virus, the viruses can “mix and match” to produce a new strain, previously unseen. This mixing of genes is called “reassortment” and occurs commonly with flu viruses. It’s why we have a new seasonal flu vaccine every year – to keep up to date with the new strains circulating.

Reassortment led to the emergence of the viruses responsible for the 1957 Asian flu and 1968 Hong Kong flu pandemics, caused by the mixing of bird and human viruses, possibly in pigs (Nelson and Worobey, 2018).

The H1N1 virus responsible for the 2009 swine flu pandemic was definitely of swine origin. It had an unusual mix of genetic sequences from birds, humans and pigs. This came about following the mixing of live pigs through international trade, creating the ideal conditions for different viruses to mix with devastating consequences. Moving live pigs between Eurasia and North America facilitated the mixing of different swine flu viruses, leading to the genesis of a completely new one, never seen before. At the time, scientists said: “Recent

reports of widespread transmission of swine-origin influenza A (H1N1) viruses in humans in Mexico, the United States, and elsewhere highlight this ever-present threat to global public health” (Shinde *et al.*, 2009).

Before the 2009 pandemic, for at least 80 years, so-called “classical swine H1N1” viruses, containing elements from birds, humans and pigs (triple-reassortant viruses), had been circulating in North American pigs (CDC, 2009). Scientists say that between the 1930s and 1990s, H1N1 underwent little change. However, by the late 1990s, multiple strains and subtypes (H1N1, H1N2 and H3N2) of triple-reassortant swine flu viruses – containing gene segments from birds, humans and pigs – had emerged and become predominant among North American pigs (Shinde *et al.*, 2009).

The 2009 so-called “quadruple-reassortant” virus included additional segments from Eurasian pigs (CDC, 2009). Once this new virus infected humans, swine flu spread quickly around the world, reaching pandemic status in just two months (Willyard, 2019). The US Centers for Disease Control and Prevention (CDC) estimated that 150,000 to 575,000 people died from swine flu in the first year of the outbreak (Dawood *et al.*, 2012). Unlike other seasonal flu epidemics, 80 per cent of deaths occurred in people under 65 with the highest burden of infection occurring in those aged five to 24 years old (Le Sage *et al.*, 2021).



FACTORY FARMS – A BREEDING GROUND FOR DISEASE

Scientists think that most older people had some protection because they would have been exposed to similar flu viruses descended from the one that caused the 1918 Spanish flu pandemic – an old strain of H1N1 that their body still remembered. Younger people, on the other hand, may have only been exposed to H3N2 which offered no protection against the new swine flu virus. Swine flu has now become one of the seasonal flu viruses that circulate each winter. If you've had flu in the last few years, there's a chance it was caused by this virus.

Reassortment occurs frequently in nature and, although it rarely results in a virus with pandemic potential, scientists say that all three pandemics of the twentieth century may have been generated by a series of multiple reassortment events in pigs or humans.

The genesis of the 2009 swine flu pandemic followed a recognised evolutionary pathway. Initial transmission to humans appears to have occurred several months before the outbreak. It may have taken years for all the elements that made up the final virus to come together, highlighting the need for the systematic surveillance of flu in pigs as a means of identifying potentially pandemic strains before they cross into human populations. Scientists say that "...despite widespread influenza surveillance in humans, the lack of systematic swine surveillance allowed for the undetected

persistence and evolution of this potentially pandemic strain for many years" (Smith *et al.*, 2009).

The World Organisation for Animal Health, formerly the Office International des Epizooties (OIE), is an intergovernmental body that sets standards for reporting animal disease. They require that certain strains of avian influenza be declared, but pork producers do not need to report swine flu to the authorities. So, flu viruses in pigs often go undetected and unreported. According to Professor Gray: "Influenza A viruses are largely tolerated because they don't cause a big problem, at least not in the pigs" (Willyard, 2019).

H1N1 strains continue to circulate in pigs in the US and Asia. H3N2 viruses are now endemic in pigs in southern China, where they co-circulate with H9N2 viruses with the potential of reassortment with H5N1. In Spain, H1N1, H1N2 and H3N2 viruses circulate concurrently in pigs (Leibler *et al.*, 2009). A flu virus circulating in pigs reassorting with bird and human viruses to generate a new swine flu virus could represent a future influenza pandemic threat for humans, as highlighted by the last 2009 swine flu pandemic (He *et al.*, 2018).

Scientists say: "The emergence of variant influenza viruses that transmit efficiently from person to person and for which little population immunity exists may



increase the possibility of an influenza pandemic” (Epperson *et al.*, 2013).

Science writer Willyard warns: “Modern farms are particularly vulnerable to devastation from influenza. A large farm might hold tens of thousands of chickens or thousands of pigs in the name of efficient protein production, and this creates an opportunity for viruses such as influenza to mutate and spread. But there is an even greater fear: that these ever-changing viruses will give rise to the next human pandemic” (Willyard, 2019). Given the potential role pigs play in the emergence of novel flu viruses, placing intensive poultry and pig farms close together could further increase the risk (Leibler *et al.*, 2009).

The way the 2009 swine flu pandemic would occur was predicted in a review published just months before it began: “...recent events resulting in the establishment and isolation of reassorted, mammalian-adapted H2N3 viruses from pigs in the US should remind scientists, medical doctors, veterinarians and farmers that the creation of novel reassortant swine influenza viruses with zoonotic and pandemic potential could also happen in modern swine facilities in the backyard of a highly industrialised country in North America or Western Europe” (Ma *et al.*, 2008). Within a few months of this review being published, the world faced an influenza pandemic for the first time in 40 years. The next one could be a lot sooner according to some scientists.

The authors of this review also warned that: “Even though pigs can generate novel influenza viruses capable of infecting humans, at present it is difficult to predict which particular virus will cause the next human influenza pandemic. The index case (patient zero) probably linking a wild bird, chicken or domestic duck with a pig and/or a person could be anywhere in the world, but a Southeast Asian “wet market” is most likely to be the locale in which the next pandemic virus is generated” (Ma *et al.*, 2008). The wet market scenario described is the most likely location from which the coronavirus pandemic emerged a decade later but it could just as easily have been a bird flu virus.

A new animal virus could jump to humans tomorrow and start another pandemic, which could be far worse than the 2009 swine flu or Covid-19 pandemics. We are sitting on a ticking time bomb! The best way to prevent another pandemic is to end factory farming.



BSE AND CREUTZFELDT-JAKOB DISEASE

Creutzfeldt-Jakob disease (CJD) is a rare and fatal condition that affects the brain. It causes brain damage that worsens rapidly over time. Sporadic CJD is the most common type and the cause is unclear, but it is thought to happen when a normal brain protein changes abnormally by misfolding and turning into a prion. Prions are misfolded proteins with the ability to transmit their misfolded shape onto normal versions of the same protein. Prions undergo a structural change as part of the disease process, which, as well as making them infectious, also makes them very difficult to destroy (HSE, 2005).

Most cases of sporadic CJD occur in adults aged between 45 and 75. Sporadic CJD is very rare, affecting only one or two people in every million each year in the UK. In 2014, there were 90 recorded deaths from sporadic CJD in the UK (NHS, 2018).

In 1996, a new variant (vCJD) emerged in the UK in people, that was strongly linked to bovine spongiform encephalopathy (BSE), which had been first recognised in cattle a decade earlier. Many people remember in 1990, then agricultural minister John Gummer, cheerfully trying to feed his four-year-old daughter a beef burger (though she refused to eat it) in front of the world's press. It was at the height of the BSE outbreak that became known as mad cow disease.

The fatal neurodegenerative disease causes a spongy degeneration of the brain and spinal cord. During the 1986-1998 outbreak in the UK, more than 180,000 cattle were infected and 4.4 million were slaughtered. In 1990, fears were growing that BSE could infect humans, but Gummer hoped to convince the nation that British beef was perfectly safe. The only scientist to stand up to the Government and predict that BSE was likely to infect people was clinical microbiologist at the University of Leeds, Professor Richard Lacey, who worked with Viva! helping us to launch a BSE helpline.

In 1996, the worst fears were realised when the first case of the human form of the disease, vCJD, was confirmed in the UK. It led to a storm of news reports, changes in government policies regarding the beef industry, a ban on exports of meat, restrictions on blood donations and a widespread fear that anyone could be infected (Diack *et al.*, 2014).

BSE was caused by cattle being fed the remains of other cattle and sheep in a dietary supplement called meat and bone meal (MBM), which contained the infective agent. Animal by-products (from cattle and sheep) were rendered to produce MBM. Amplification of the disease in UK cattle occurred then as a consequence of the recycling of cattle by-products from infected cattle back to cattle through MBM (HSE, 2005).

Remember, cows are natural herbivores so it is very unnatural for them to eat the remains of other cows and sheep. The meat and bone meal had been produced by the rendering (industrial cooking) of carcasses of cattle infected with BSE and sheep infected with a similar disease, scrapie. The feeding of MBM to all farmed animals was banned in 1996 and an EU-wide ban has been in place since 2001.

The last death from vCJD in the UK occurred in 2016, making a total of 178 deaths recorded since 1995. The number of deaths per year peaked at 28 in 2000 and there have been no cases of vCJD in people born after the 1980s in the UK (HM Government, 2018).

A team of researchers at University College London studied Papua New Guineans with a related disease called kuru (a prion disease caused by cannibalism) in order to work out how long BSE may lurk in the body before it develops into CJD. All the kuru patients were born before the cessation of cannibalism in the late 1950s and it had been the practice in these communities to engage in the consumption of dead relatives as a mark of respect and mourning. They found that those, who had consumed the flesh of their relatives, succumbed to prion disease as much as half a century later (Collinge *et al.*, 2006).

This discovery raised concerns that vCJD, could also be incubating silently and could rear its head decades after infection. It was suggested that vCJD patients identified so far may have had particular genes which made them easy targets for the rogue protein. Professor John Collinge, who led the study, said: "Recent estimates of the size of the CJD epidemic based on uniform genetic susceptibility could be substantial underestimations." An editorial in *The Lancet* accompanying this study stated: "Any belief that CJD incidence has peaked and that we are now through the worst of this sinister disease must now be treated with extreme scepticism."



Other scientists share these concerns. The rate of cases diagnosed as sporadic CJD has doubled since the mid-1990s (National CJD research & surveillance unit, 2021). Part of this is due to better diagnosis, but that should have peaked by now and cases are still climbing. Until recently, vCJD had struck only people with a certain genetic makeup. However, in 2016, researchers confirmed a case in someone with different genes (Mok *et al.*, 2017). This could mean we have been misdiagnosing a new wave of cases that is already underway.

It wasn't until humans began dying from vCJD in the UK in 1996 that the unhealthy practice of feeding cows the remains of other dead cows and sheep was acknowledged as the probable cause of this fatal neurodegenerative disease. Animal feeding practices

have now been changed but vCJD has a long incubation period and cases may still emerge. Yet another example of how the barbaric practices associated with factory farming are linked to disease.

In animals, the occasional case of BSE still crops up. There have been five confirmed cases in the UK since 2014, the most recent in September 2021 when a cow tested positive, as part of surveillance screening, on a farm in Somerset. In response China and the Philippines have banned British beef imports and other countries may follow. It is unclear why cows are still getting BSE and the UK Animal and Plant Health Agency (APHA) has begun an investigation into this latest case and the World Organisation for Animal Health (OIE) has also been informed.

BOVINE TB – BADGERS ARE NOT THE PROBLEM

Bovine tuberculosis (bovine TB or bTB) is an infectious zoonotic disease caused by the bacterium *Mycobacterium bovis*. Unlike *Mycobacterium tuberculosis*, which is the main cause of TB in humans, *M. bovis* has a broad host range. It is most commonly found in cattle but can be carried by a variety of animals including badgers, foxes, rabbits, moles, deer, alpacas, llamas, goats, cats and dogs. It is a zoonotic disease – so humans can be infected too.

M. bovis can cause zoonotic TB in humans through ingestion, inhalation and, less frequently, by contact with mucous membranes and broken skin. Zoonotic TB is indistinguishable from TB caused by *M. tuberculosis*, which makes it difficult to accurately estimate the number of human TB cases caused by *M. bovis* infection caught from animals (de la Rua-Domenech, 2006).

Zoonotic TB used to be quite common, usually transmitted via the consumption of raw cow's milk but also – less commonly – through the consumption of raw or uncooked meat or direct physical contact with infected animals (WHO, 2018).

Nowadays, most of the 7,000 or so cases of human TB annually reported in the UK are due to *M. tuberculosis* spread from person to person. In the period 1990-2003, between 17 and 50 new cases of human *M. bovis* infection were confirmed every year in the UK, representing between 0.5 and 1.5 per cent of all confirmed TB cases, a proportion similar to that of

other industrialised countries (de la Rua-Domenech, 2006). Human infection with *M. bovis* in the UK has largely been controlled through pasteurisation of milk and systematic culling of cattle testing positive in compulsory tests (de la Rua-Domenech, 2006).

The Government's bTB Strategy, published in 2014, aims to achieve officially bovine tuberculosis free (OTF) status for England by 2038, whilst maintaining an economically sustainable livestock industry. However, this presents a challenge as they acknowledge that: "Bovine TB has the potential to spread to new herds and new areas via movement of cattle with undetected infection between OTF herds" (Defra, 2021a).

Between March 2020 and March 2021, 38,841 cattle were compulsorily slaughtered in Britain to try and control the disease (Defra, 2021b). In previous years, similar numbers (ranging from 36,000 to 45,000) were killed. Farmers are compensated for each cow testing positive and slaughtered and "dealing with the disease is costing the taxpayer over £100 million each year" (Defra, 2021a).

Badgers have been blamed as the primary reason for bTB spreading, the idea being that infected badgers spread the disease into dairy herds. Under pressure, because of hardship to farmers and the growing cost of the disease to the UK economy, the Government introduced badger culling in 2013. Since then, up to January 2021, 140,830 badgers have been killed (Badger Action Network, 2021). An increasing number of reviews and scientific reports have challenged the strategy, but the cull has continued.

In March 2020, the Government responded to an independent bTB strategy review, led by Professor Sir Charles Godfray, saying it would, "begin an exit strategy from the intensive culling of badgers." The aim was to move towards improved bTB testing, tighter biosecurity on farms, tougher restrictions on cattle movement and widespread vaccination of cattle and badgers.

However, just months later, in a major U-turn – largely eclipsed by news coverage of Covid-19 and Brexit – an extensive continuation of the badger-culling programme was announced with the cull being expanded to 11 new areas of England, including parts of Oxfordshire, Lincolnshire, Leicestershire and Derbyshire so that more than 60,000 badgers can be killed.



Conservation groups said the expansion was a betrayal of trust after the Government pledged to phase out the cull. Writing in the journal *National Geographic*, veterinary surgeon Dr Iain McGill, who spearheaded the investigation into the BSE crisis in the 1990s, said: "This is more disgraceful governmental deception. They are hinting for the second year that they are phasing out culling... [but] the reality is that culling will expand massively in the next two years, as pro-cull landowners rush to apply for their licenses. They'll be granted four-year licenses to cull, and can apply to cull for two more supplementary years after that. Thousands of badgers will continue to be targeted until at least 2028, and under "exceptional circumstances" thereafter. Sadly, this is most definitely not the end" (Jarvis, 2021).

Then, in May 2021, the Government announced that no new four-year cull licences will be issued after 2022. In a classic example of spin, this announcement came on the same day that they issued 10 new culling licences (the maximum number allowed).

As many as 38,462 badgers were killed in 2020, so under the existing and new licences issued up to 2022, many thousands more badgers could still be killed before the badger cull finally comes to an end. Badger Trust acting chief executive Dawn Varley said: "Mass culling licences will run to 2026 and, combined with those already in play, we estimate will lead to another 140,000 badgers still to be killed in addition to the 140,000 we have already lost."

Dr Jo Smith, CEO of Derbyshire Wildlife Trust, said: "The Government has failed to listen to the public who want to see an immediate end to the badger cull. If a further 130,000 animals are killed within the next five years, we could lose 60% of England's badgers" (The Wildlife Trusts, 2021).

Not only does the research suggest the role of badgers in spreading bTB has been overstated, culling them may even increase the spread of disease (Pope *et al.*, 2007). Research shows that culling badgers drives them to roam further afield, allowing them, if infected, to disperse bTB over a larger area. Varley says: "The culling policy is inhumane and unnecessary at best, and at worst it's a smokescreen and ineffective strategy to appease farmers."

Scientists say: "Given the increasing numbers of cattle herds being affected each year, physicians and other public health professionals must remember that zoonotic TB is not just a disease of the past" (de la Rúa-Domenech, 2006). Although, for most people, the risk of contracting *M. bovis* infection from animals appears to be very low, according to the Government: "the TB epidemic in cattle and badgers, with occasional spill-over into other wild and domestic species, represents a low but ongoing public health risk" (APHA, 2020). A significant risk remains for those consuming unpasteurised milk and dairy products and people who risk occupational exposure to infectious aerosols from infected animals and their carcasses (de la Rúa-Domenech, 2006).

The drive for cheap meat and milk comes at an increasingly high cost to the farmed animals, the farmers, the environment and native wildlife. Some farmers are beginning to turn away from livestock farming and look for new, more sustainable ways of farming. The Government needs to start supporting this move with guidance and incentives.

The most effective, kindest and cheapest way to eliminate the risk of bTB spreading to humans would be to end our exploitation and unnatural interaction with bovine animals, such as using cows for milk and beef. If we didn't farm cattle, we wouldn't have a bTB problem. There are now plenty of dairy and meat alternatives which play no part in the spread of disease, which require neither badgers nor cows to be killed and don't cost the taxpayer millions of pounds in pointless culls.

To find out more go to viva.org.uk/animals/other-animals/badgers



NIPAH VIRUS – HOW PIG FARMING PROVIDED A PATHWAY FOR DISEASE

The 1998-1999 Malaysian outbreak of Nipah virus exemplifies how factory farming provides a pathway for disease to spread and develop into a lethal threat to human health. Researchers from Johns Hopkins University say that, in the outbreak, domesticated pigs being farmed for meat served as a biological vector between wild fruit bats (the host species) and humans (Leibler *et al.*, 2009).

The Nipah virus is highly contagious in pigs and an infected pig may exhibit no symptoms, while others develop acute feverish illness, laboured breathing and neurological symptoms such as trembling, twitching and muscle spasms. In humans, symptoms also range from hardly any to severe acute respiratory infection and fatal encephalitis.

The Malaysian outbreak caused respiratory disease in pigs and a high death rate in humans killing 105 of the 265 patients. The World Health Organisation says that the case fatality rate is estimated at 40-75 per cent (WHO, 2018a).

Many of the humans infected were pig farmers and a respiratory illness was observed among pigs on some of their farms. An investigation revealed that in 1997, both pig and human cases had occurred on a single large intensive pig farm in northern Malaysia. At this farm, Nipah virus-infected fruit bats had been attracted to fruit trees planted around the pig farm. The virus spillover event occurred when pigs ate fruit contaminated with bat saliva or urine.

The spread of disease among the pigs was made easier by the high density of over 30,000 animals and by transporting them to other farms located in what later became the main outbreak area in south Malaysia. Agricultural intensification thereby provided the pathway for a virus circulating in fruit bats to infect pigs and then people. Researchers from the University of London describe how: "Pigs then acted as amplifier hosts for human infection" (Jones *et al.*, 2013).

Summing up the role of factory farms in disease, they said: "In intensive systems, genetic selection and management of livestock creates frequent contact opportunities, high animal numbers, and low genetic

diversity, providing opportunities for "wild" microorganisms to invade and amplify or for existing pathogens to evolve to new and more pathogenic forms" (Jones *et al.*, 2013).

In a paper published in *Interface*, a journal of the Royal Society, scientists describe two different stages of a deadly disease outbreak and a missed opportunity for early detection and prevention. They suggested that it was the Nipah virus jumping repeatedly from fruit bats to pigs that had deadly consequences and say that it was this repeated introduction that changed the infection dynamics in pigs and subsequently in humans. The initial introduction of the virus created a "priming" effect that allowed a secondary introduction to persist in what was then a partially immune population. As pigs born after the initial event gradually lost their maternal antibodies, they became susceptible and allowed Nipah virus persistence (Pulliam *et al.*, 2012).

The emergence of Nipah virus in Malaysia was thus the product of two drivers. First, agricultural intensification, in the form of commercial pig production and fruit orchards, creating the pathway for the repeated transmission of the virus from fruit bats to pigs. Second, the initial spillover primed the pig population for persistence of the pathogen on reintroduction, leading to the increased transmission among pigs and to humans. Once infected pigs were sold outside the region, the opportunities for greater human exposure, infection and disease followed (Daszak *et al.*, 2013).

According to this study, the discovery of historical cases after the identification of an emerging pathogen is common and has been seen before with Nipah virus, SARS, HIV and H5N1 influenza (Pulliam *et al.*, 2012).

The authors of this study support previous calls for increasing targeted global surveillance of livestock in regions of high wildlife biodiversity to improve the chances that "viral chatter" of wildlife origin is detected prior to a widespread epidemic in livestock or people. A more effective solution would be to stop factory farming altogether.



COVID-19, SARS AND MERS – DRIVEN BY THE HUNGER FOR MEAT

The coronavirus pandemic may have originated, it is thought, in a wet market rather than a factory farm, but the risk factors in such places are very similar. The increasing global demand for meat from both wild and domesticated animals poses a serious pandemic threat – whether it comes from a wet market or a factory farm (Cheng *et al.*, 2020).

Evolutionary biologist Rob Wallace of the Agroecology and Rural Economics Research Corps in St Paul, Minnesota told the *Guardian*: “We can blame the object – the virus, the cultural practice – but causality extends out into the relationships between people and ecology” (Spinney, 2020).

Even when zoonotic diseases come from wildlife, factory farming often has a role to play. In China, for example, as industrialised farming has expanded, small-scale farmers have moved over to the wildlife market. They have also been geographically pushed into remote areas inhabited by bats, for example. We’ve seen the same thing with people catching Ebola from chimpanzees caught and slaughtered as “bushmeat.”

The severe acute respiratory syndrome, or SARS, outbreak in 2002-2003 was the first global pandemic of the 21st century followed by Middle East respiratory syndrome, or MERS, in 2012. SARS, MERS and Covid-19 are all caused by coronaviruses believed to originally come from bats (the so-called natural hosts). However, it is most likely that these viruses were only able to

infect humans after mutating in an intermediate host animal.

The intermediate hosts for SARS and MERS were civets and camels, respectively. Civets are captured in the wild and brought to market to be sold for their meat. Camels are raised for milk, meat, fibre (wool and hair) as well as transport and racing. For Covid-19, various candidate intermediate hosts have been proposed including pangolins, which have been illegally sold in Chinese markets (Rabi *et al.*, 2020) and are the most trafficked animal in the world. Raccoon dogs, another candidate, are farmed for their fur and housed in large numbers with more than 14 million captive raccoon dogs in China (Freuling *et al.*, 2020).

If SARS-CoV-2, the virus that causes Covid-19, was transmitted from bats to humans directly – there was ample opportunity. Over 100 species of bat are eaten globally, including the horseshoe bats which naturally harbour coronaviruses closely related to SARS-CoV-2 (Burki, 2020).

Writing in *The Lancet*, scientists said: “Learning from the SARS outbreak, which started as animal-to-human transmission during the first phase of the epidemic, all game meat trades should be optimally regulated to terminate this portal of transmission” (Chan *et al.*, 2020). Wet markets in China were closed temporarily after the SARS outbreak, though this ban did not last long (Aguirre *et al.*, 2020).



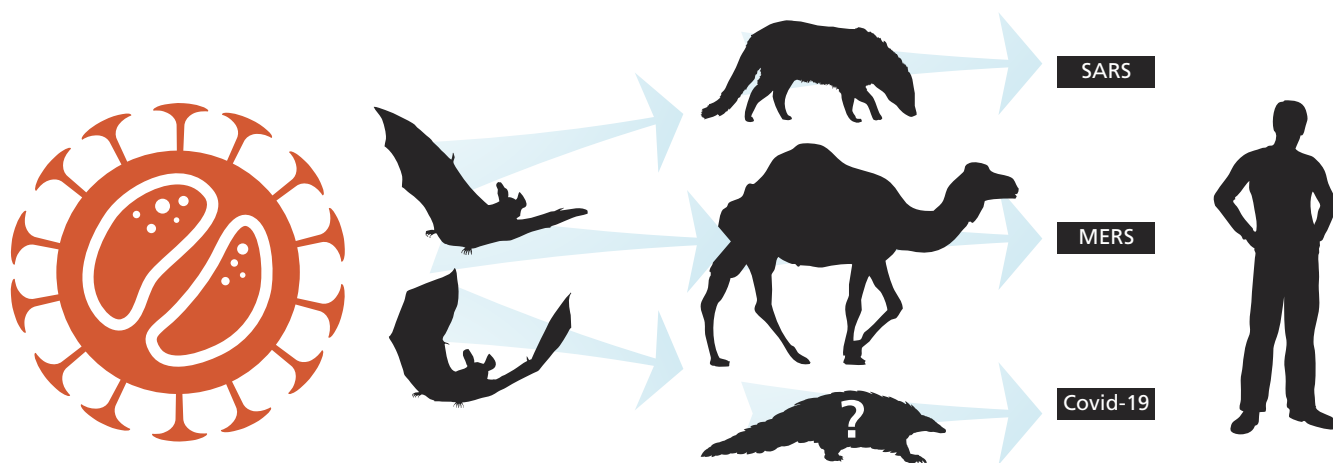


Figure 2. Animal origins of human coronaviruses.

In January 2020, following the outbreak of Covid-19, the Chinese government again announced a temporary ban on the sale of wild animal products at wet markets and then a permanent ban on wildlife trade in February 2020 with exceptions for fur farming and medicine. Unfortunately, these loopholes mean that many wild animals remain unprotected and could potentially lead to another pandemic caused by a zoonotic disease in the future.

By the end of March 2020, most of the wet markets in China were reopened without wild animals or wild meat. The World Health Organisation responded with the recommendation that wet markets only be reopened on the condition that they conform to stringent food safety and hygiene standards. The latest statement makes clear their belief that live animal markets pose a big risk in creating novel viruses in the



FACTORY FARMS – A BREEDING GROUND FOR DISEASE

future. In April 2020, the Chinese government unveiled plans to further tighten restrictions on wildlife trade.

In April 2021, the World Health Organisation, World Organisation for Animal Health (OIE) and the UN Environment Programme (UNEP) joined forces to call for a complete ban on the sale of live caught wild animals in food markets across the world. They called on countries to abolish the trade of “live caught wild animals of mammalian species” (UN NEWS, 2021) in a bid to stop a repeat of the Covid-19 pandemic. They also want to see improvements in standards of hygiene and sanitation including handwashing, pest control and waste management. In a joint statement they said: “Traditional markets, where live animals are held, slaughtered and dressed, pose a particular risk for pathogen transmission to workers and customers alike” (WHO, 2021a).

Wet markets place people, live and dead animals in constant close contact. Animals including chickens, ducks, pigs, snakes, civets, pangolins, bats, raccoon dogs, beavers, foxes, dogs and many other animals are

sold, skinned and slaughtered in front of customers, sending a cocktail of microorganisms into the air. The dreadful, cramped conditions and mix of wild and domestic creatures, alongside the throngs of people provides an ideal environment for zoonotic diseases to jump from animals to humans. Another pandemic is in the making.

Most of the wild animals traded in China are live, says Chris Walzer, a veterinary physician with the Wildlife Conservation Society in New York City. “You bring all those naturally distant species to one location, so there are more chances to incubate and generate a new virus” says QiuHong Wang, a virologist at the Ohio State University in Wooster (Lewis, 2021).

In December 2019, a cluster of cases of pneumonia were reported from Wuhan City in China. The cause was a coronavirus, similar to the SARS-CoV-1 virus that caused the SARS pandemic. Soon to be called Covid-19, the disease spread fast across Asia to the US, Europe and so on, becoming a global pandemic in less than three months.



Covid infections in mink fur farms

Mink, among other animals, can act as a reservoir for coronaviruses. Farmed mink are highly susceptible to infection with SARS-CoV-2 – the virus that causes Covid-19. They can catch it from humans, pass it on to each other and spread it back to humans.

In April 2020, SARS-CoV-2 was reported from two mink fur farms in the Netherlands. Evidence suggests that the virus was transmitted to the mink via infected farm workers. Transmission from human-to-mink and mink-to-human has now been well-established. Once Covid-19 reaches a mink farm, scientists say, it spreads very rapidly among the animals and one worry is that infected mink farms can become a viral reservoir for new outbreaks in humans. More recently, infections in minks have been reported in Denmark, Netherlands, Italy, Spain, Sweden and the US (Larsen and Paludan, 2020).

Mink fur farms have provided the perfect environment for the virus to mutate and then spread back to humans raising concerns that a mutated version (or variant) might be even more deadly or may hamper the development of a vaccine. Since June 2020, over 200 human cases of Covid-19 have been identified in Denmark with SARS-CoV-2 variants associated with farmed minks, including 12 caused by a mutated variant (WHO, 2020).

The human-mink-human transmission shows the real danger of what virologists call “reverse spillover” and demonstrates what a potential danger factory farms pose: “It is a fundamental part of viral evolution that a virus will change over time and accumulate mutations, but one should be particularly concerned when viruses pass between species, including humans and animals” (Larsen and Paludan, 2020).

Concerned about new variants arising from mink farms, the Danish prime minister warned that in a worst-case scenario, they “could cause a second pandemic and Denmark could become the new Wuhan. In addition, vaccines under development might not be effective” (Larsen and Paludan, 2020).

The Danish government ordered the country’s entire mink herd – one of the world’s biggest – to be culled in November 2020. At that time, there was no legal basis for the mass cull, however it proceeded and some 17 million mink were culled. Then, in a grim twist to the tale, some of the mink buried in mass graves resurfaced

as gas from their decomposing bodies pushed them up out of the ground. Amid concerns that drinking water could become contaminated, it was decided that their bodies should be exhumed and incinerated. In May 2021, Denmark began digging up millions of the culled minks buried six months earlier. The silver lining is that the whole episode has been a death knell for one of the cruellest industries in the world, the fur industry. Denmark was the world’s biggest producer of mink fur exporting mainly to China and Hong Kong – but not anymore. The Netherlands, another top exporter, fast-tracked a plan to phase out fur farming, bringing the deadline forward from 2024 to 2020. France has announced that it will ban farming mink for fur by 2025 and Poland may follow suit. Fur farming is already banned in the UK and has been for 20 years.

It’s not just a case of banning fur farms and wet markets, although that can’t come soon enough. We need to stop factory farming too. “In the meantime, the scientific community should be vigilant about the possibility of return of another coronavirus in another one or two decades” (Cheng *et al.*, 2020).



ANTIBIOTIC RESISTANCE – SUPERBUGS ON THE RISE

During his 1945 Nobel Prize acceptance speech, Alexander Fleming, who discovered penicillin, warned of the danger of an over-reliance on antibiotics and the threat of bacteria developing antimicrobial resistance (AMR).

Antibiotics have been helping us fight infection since the 1940s. Before they were developed, even a small scratch could be fatal. Giving birth and having surgery were a lot riskier and sexually transmitted infections (STIs), such as syphilis and gonorrhoea, caused untold misery and could be a death sentence.

We now rely heavily on antibiotics to treat and prevent infection but what many people don't realise is that they are also widely used in agriculture and aquaculture. The overuse of antibiotics in humans and animals has led to the emergence of multidrug-resistant "superbugs." The UK government says: "As in humans, the sub-optimal use of antimicrobials in agriculture and veterinary practice contributes to the rise and spread of AMR all over the world" (HM Government, 2019).

Some types of bacteria that cause serious infections in humans have already developed resistance to most or all of the available treatments, and there are very few promising options in the research pipeline (WHO, 2017).

How does antibiotic resistance develop?

AMR happens because rapid and random DNA mutations occur naturally in bacteria – these may help them to prosper or have no effect. If a mutation helps a single bacterium survive antibiotic treatment while all the others die, that one will go on to reproduce, spreading and taking its new resistance gene with it like an accessory, enabling it to survive the hostile environment – the genetic equivalent of a stab-vest!

Use of antibiotics in animals as growth promoters

While the proper therapeutic use of antibiotics in animals is essential for treating infection, much of the use of antibiotics in livestock is not therapeutic. Soon after they were first introduced, it was found that antibiotics could promote growth when fed to farmed animals at low (sub-optimal) levels. This led to their widespread use and abuse to help fatten animals for the table. Antibiotic use in livestock now outweighs human consumption in many countries and in the US, for example, some 80 per cent of all antibiotics are used as growth supplements and to control infection in farmed animals (Martin *et al.*, 2015).

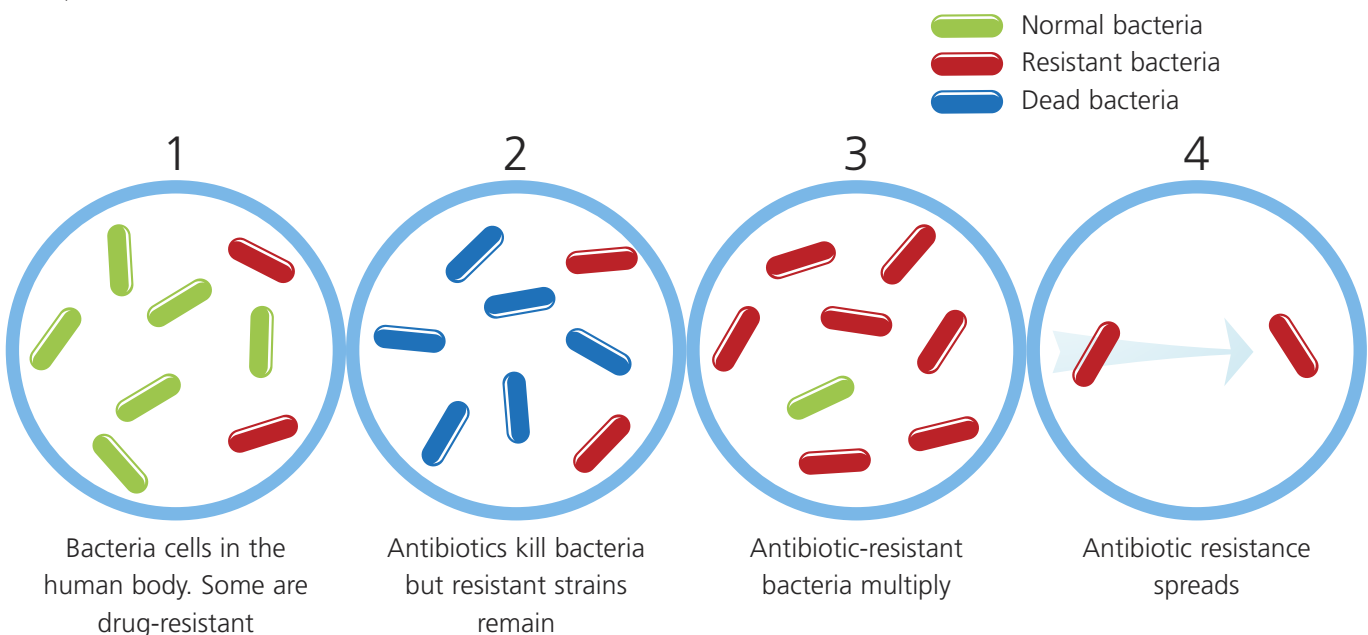


Figure 3. How antibiotic resistance occurs

Some countries have acted to reduce the use of antibiotics in livestock. In 2006, the European Union banned the use of antibiotics for growth promotion. However, mass medication of healthy animals is still used to prevent disease (metaphylaxis) when an infectious disease is present within the group, flock or herd (Baptiste and Kyvsgaard, 2017). The European Medicines Agency (EMA) and European Food Safety Authority (EFSA) say that metaphylaxis is frequently used in intensively reared animals and that: "There should be an aim to phase out preventive use of antimicrobials, except in exceptional circumstances" (EMA and EFSA, 2017).

Significant volumes of antibiotics continue to be used either prophylactically amongst healthy animals, to stop the development of an infection within a flock or herd, or simply for growth promotion, to speed up the pace at which animals gain weight. Both uses are particularly prevalent in intensive agriculture, where animals are kept in confined conditions (O'Neil, 2016). In 2017, the World Health Organisation recommended that farmers and the food industry stop using antibiotics routinely to promote growth and prevent disease in healthy farmed animals (WHO, 2017).

Why do factory farms pose such a risk?

Factory farms are the most disease-ridden places on Earth. They are used to confine hundreds, sometimes thousands, of genetically identical animals with no access to outdoor space or natural light. This highly stressful, barren environment can lead to severe behavioural problems including repetitive and aggressive behaviour like cage biting, feather pecking and cannibalism. So, antibiotics are frequently used in such places to prop up this cruel and deeply flawed system of animal food production.

Antibiotic-resistant superbugs from factory farms can spread in manure sludge from the farm into the environment, contaminating surface water, groundwater and soil. From there, they may infect people swimming, drinking or washing with contaminated water or consuming contaminated crops.

Antibiotic-resistant bugs in meat and dairy foods

People can also get infections from handling or eating meat, seafood, milk or eggs that are raw or undercooked and contaminated with resistant bacteria



(CDC, 2021). In 2018, a UK Foods Standards Agency survey looking at chicken and pork for sale in Britain's supermarkets found record levels of superbugs resistant to some of the strongest antibiotics (FSA, 2018). A quarter of all chicken samples tested were infected with *Campylobacter*. Of these, almost half were resistant to ciprofloxacin and more than half to tetracycline. Multidrug resistance was found in 8.9 per cent of the *Campylobacter coli* from chicken. The official line is that the risk of acquiring AMR bacterial infections from foods is low provided that they are cooked and handled hygienically, but thousands of people are still infected with these food poisoning bugs every year.

Antibiotic use in farmed animals in the UK

There have been some improvements in the UK with antibiotic sales for use in animals falling by 35 per cent (six per cent in humans) between 2013 and 2017 (UK-VARSS, 2019). In 2017, 36 per cent of antibiotics in the UK were sold for use in animals (the majority going to farmed animals with a smaller amount used in horses and companion animals), while 64 per cent were for human use (Veterinary Medicines Directorate, 2019). A lower proportion than in many other countries, but still a considerable amount weighing in at 226 tonnes in 2018 (UK-VARSS, 2019).

However, the Advisory Committee on Antimicrobial Prescribing, Resistance and Healthcare Associated

FACTORY FARMS – A BREEDING GROUND FOR DISEASE

Infection (APRHAI), the expert scientific advisory committee providing independent advice to the Department of Health and Social Care, says that during the same period, the number of antibiotic-resistant bloodstream infections in the UK increased by 35 per cent and continues to rise (APRHAI, 2019). It could be that the action being taken is too little, too late.

One of the top ten global public health threats

The World Health Organisation now says that AMR is one of the top ten global public health threats facing humanity (WHO, 2020b). It could be the cause of the next pandemic the world faces. When antibiotics fail, chest infections, urinary tract infections (UTIs), cuts, insect bites and even small scratches can develop into sepsis (blood-poisoning) which can be fatal if not treated quickly. In the UK, five people die from sepsis every hour and the numbers are rising (UK Sepsis Trust, 2020). The UK Sepsis Trust CEO, Dr Ron Daniels, says: "...nearly 40 per cent of *E. coli* – the bacteria that causes a huge number of infections – is now resistant to antibiotics and these organisms account for up to one third of episodes of sepsis, showing the vital need for responsible use of antimicrobial drugs" (UK Sepsis Trust, 2020a).

The idea of dying from a paper-cut or an insect bite is unthinkable but is fast becoming a possibility, thanks to the overuse and abuse of antibiotics. Professor Colin Garner, chief executive of Antibiotic Research UK, says: "We have been warning for some time that our antibiotics are so ineffective that we could reach the situation where people will once again die from an infected scratch or bite. That tragic moment may just have come. I personally got bitten recently by a horsefly and it is very painful. I am self-medicating with creams and an oral antihistamine tablet to ensure the bite site does not become infected" (Antibiotic Research UK, 2018).

The scale of the problem in people

Across Europe alone, an estimated 25,000 people die each year because of hospital infections caused by the following five resistant bacteria:

- *E. coli*
- *Klebsiella pneumoniae*
- *Enterococcus faecium*
- *Pseudomonas aeruginosa*
- Methicillin-resistant *Staphylococcus aureus* (MRSA)

Source: Public Health England, 2015.



E. coli bloodstream infections are increasing in the UK and internationally. The number of infections that were voluntarily reported to Public Health England increased by 44 per cent between 2003 and 2011, and after the introduction of mandatory reporting in July 2011, a further 28 per cent increase was seen by July-September 2016 (Vihta *et al.*, 2018). Between 2014 and 2018, the number of people affected in England rose from 53 to 70 per 100,000 population. An increasing trend in antibiotic resistance was also observed (Public Health England, 2019). Scientists suggest that *E. coli* bacteraemia rates have risen due to rising rates of antibiotic resistance (Schlackow *et al.*, 2012).

Public Health England's antimicrobial resistance (AMR) report, published at the start of World Antimicrobial Awareness Week in 2020, says that 21 out of every 100 people in England with bloodstream infections caused by a number of key bacteria are infected with antibiotic-resistant strains. This, they say, led to an estimated 65,000 antibiotic-resistant severe infections in 2019; equivalent to 178 new antibiotic-resistant infections per day (Public Health England, 2020).

Some of these infections come from animals. Lord Jim O'Neill's 2016 government-commissioned review on AMR describes the ways in which resistant bacteria in animals, created by the selective pressures of antibiotic use, could be transferred to humans. Transfer may occur, O'Neill says, through direct contact with animals, from undercooked or unpasteurised animal foods, or via the spread of resistant bacteria into environmental reservoirs, which may then transmit resistance genes to

human bacteria, or come into contact with humans directly. Thus, explaining the link between the use of antibiotics in agriculture and resistant infections in humans. The report says: "In light of this information, we believe that there is sufficient evidence showing that the world needs to start curtailing the quantities of antimicrobials used in agriculture now" (O'Neill, 2016).

O'Neill warns that the 700,000 global deaths caused by AMR each year will rise to 10 million by 2050 if no action is taken. This would mean antibiotic-resistant superbugs killing more people than cancer. The cost in terms of lost global production between now and 2050 would be an enormous £66 trillion if no action is taken (O'Neill, 2016).

In July 2018, O'Neill became Chair of Chatham House, the international think tank. With the eyes of the press on him, he said that antibiotic resistance should be listed as a cause of death on official certificates to help raise awareness of the growing superbug crisis.

O'Neill describes himself as a "resistance fighter" and says: "We need modern technology to discipline all seven billion of us to stop pressuring our medical practitioners to prescribe antibiotics when they aren't necessary, and the food industry needs to stop fattening animals and fish with antibiotics, in order to permanently reduce our excessive dependence on the few drugs that work and the new ones we hope to have in the future. Because all of us need to be resistance fighters" (Antimicrobial Resistance Fighter Coalition, 2021).

A failure to address the problem of antibiotic resistance could result in:



10M
DEATHS
PER YEAR
BY 2050

COSTING
£66
TRILLION
BETWEEN
NOW AND 2050



Rising meat consumption driving AMR

Dr Kazuaki Miyagishima, Director of the Department of Food Safety and Zoonoses at the World Health Organisation, says: “Scientific evidence demonstrates that overuse of antibiotics in animals can contribute to the emergence of antibiotic resistance.” Miyagishima says: “The volume of antibiotics used in animals is continuing to increase worldwide, driven by a growing demand for foods of animal origin, often produced through intensive animal husbandry” (WHO, 2017).

Due to this increasing global demand for meat, it’s predicted that antibiotic use in cattle, chicken and pigs worldwide will increase by 67 per cent by 2030 and nearly double in Brazil, Russia, India, China, and South Africa. This rise, scientists say, is likely to be driven by the growth in consumer demand for livestock products in middle-income countries and a shift to large-scale farms where antibiotics are used routinely (Van Boeckel *et al.*, 2015).

Colistin – using up our last resort

Colistin is regarded as a “last resort” antibiotic for humans but is still used widely in livestock, especially

pigs, in some parts of the world. In 2015, bacteria carrying colistin-resistance genes were identified in China. The genes are carried in such a way that resistant bacteria are able to transfer them to different species of bacteria, something not seen before. This so-called ‘horizontal gene transfer’ rang alarm bells among the scientific community everywhere as it not only heralded the breach of the last group of antibiotics available to humans but opened up the possibility of AMR spreading even faster (Liu *et al.*, 2016).

Screening in areas of China, where colistin had been routinely given to pigs, revealed resistant *E. coli* in more than 20 per cent of animals, 15 per cent of raw meat samples and one per cent of hospital patients (Liu *et al.*, 2016). In Bangladesh, *E. coli* with resistance to colistin have been found in water, street food, hand rinse samples of street food vendors and healthy human gut samples. The authors of this study say that this is alarming and sheds light on the potential health risk that colistin-resistant *E. coli* could pose to millions of people (Johura *et al.*, 2020).

Colistin-resistant bacteria have now been identified in over 50 countries, including the UK (Liu and Liu, 2018). The use of this antibiotic to treat infection in animals has since been voluntarily restricted by livestock industries in the UK, decreasing by 99 per cent

between 2015 and 2017 (Veterinary Medicines Directorate, 2019). The EMA suggests that substances which are of last resort for treatment of life-threatening disease in humans should be excluded from veterinary use (EMA, 2016).

It's unclear why there hasn't been a total ban on the use of this last resort antibiotic and the UK's policy to accept voluntary initiatives and farm assurance schemes contrasts with other countries, such as Sweden, Denmark and the Netherlands, where antibiotic use in animals is controlled through government legislation. China has now banned the use of colistin in animals as a growth promoter, falling in line with the EU, US, Brazil and, more recently, India. Scientists are unsure if this late action can curb the spread of resistance genes.

A huge step backwards

From January 2022, the EU banned the importation of all meat and dairy produced with antibiotic growth promoters, but it is still not clear whether the UK will implement this legislation too. The majority of meat and dairy imports in the UK come from the EU. However, the Government is keen to negotiate new trade deals with several countries, including the US, Canada, Australia and New Zealand. All four countries allow farmers to feed antibiotics routinely to livestock to make them grow faster (this practice is banned in the UK and the EU). In the US and Canada, for example, farm antibiotic use is about five times the level in the UK (Alliance to Save Our Antibiotics, 2020).

In their report *Farm antibiotics and trade deals – could UK standards be undermined?* The Alliance to Save Our Antibiotics says: "A failure to maintain and improve standards, coupled with a shift from importing EU-produced meat to importing cheaper meat from countries such as the US, Canada or Australia may have significant consequences for the levels of antibiotic resistance being spread via the food chain" (Alliance to Save Our Antibiotics, 2020). This would be a huge step backwards!

The World Health Organisation says that AMR is one of the main threats to modern medicine, with growing numbers of infections, such as pneumonia, tuberculosis, gonorrhoea and salmonellosis becoming harder to treat: "Without urgent action, we are heading for a post-antibiotic era, in which common infections and minor injuries can once again kill" (WHO, 2020a).

One health

There are stark parallels between the AMR crisis and climate change – both driven by the increasing global demand for cheap animal-based foods. The World Health Organisation talks about a 'One Health' solution because AMR does not recognise geographic or human-animal borders – we all share one planet (WHO, 2017a). The most effective way to tackle AMR and achieve optimal health for people, animals and our environment is to change the way we eat, reducing antibiotic use in humans and animals.

The contribution to AMR from agriculture is significant and may be growing. Of course, livestock industries are inevitably resistant to change but the obvious solution is to stop producing animal-based foods and go vegan. The widespread adoption of a vegan diet would remove the factory farms that are the breeding grounds for these superbugs.

AMR is a problem of our own making – a direct consequence of the inappropriate use of antibiotics in humans and animals, in a drive to produce cheap meat, fish and dairy foods on an industrial scale. Failure to act may result in the chilling prospect of an apologetic doctor saying to you: "Sorry but there's nothing we can do for you."





E. COLI AND THE DEADLY THREAT FROM O157

It's not just dangerous viruses lurking in factory farms, such places provide the perfect environment for pathogenic bacteria to spread and evolve too.

Escherichia coli (*E. coli*) are a type of bacteria that live in the intestines of many mammals, birds and reptiles.

They are a large and diverse group and although most strains are harmless, others can make you very ill, causing diarrhoea, urinary tract infections, respiratory illness or pneumonia.

Shiga toxins and haemolytic uraemic syndrome

There are over 700 types (serotypes) of *E. coli*, around 200 of them produce a toxic substance known as Shiga toxin and over 100 have been associated with human disease (ECDC, 2017). If Shiga toxins are released in the gastrointestinal tract by bacteria they can cross the epithelial barrier in the gut and kill cells lining blood vessels. For example, the vascular endothelium of the glomerulus (the kidney's filtering structure) appears to be a particular target. Damage to these cells can lead to haemolytic uraemic syndrome (HUS), a serious condition that can result in kidney failure. The most important Shiga toxin-producing *E. coli* (STEC) in

relation to public health is called STEC O157:H7. However, others have caused infections and outbreaks.

E. coli O157:H7 and other Shiga toxin-producing *E. coli* are found in the gut and faeces of many animals including: cows, sheep, goats, deer, moose, pigs, horses, dogs, cats, pigeons, chickens, turkeys and gulls (Chekabab *et al.*, 2013). In the UK, the predominant animal reservoirs are cows and sheep (Treacy *et al.*, 2019). Infections (colonisation) in animals normally does not cause illness; animals can appear relatively healthy but can spread *E. coli* O157:H7 to humans and other animals.

How is it spread?

Because *E. coli* O157:H7 is shed in the faeces of ruminants, it has been "implicated in outbreaks of human disease via the cross-contamination of foods and direct contact with the faeces of affected animals" according to the UK Food Standards Agency (FSA, 2013). *E. coli* O157:H7 can be spread in several ways including:

- Eating contaminated food (such as undercooked meat or raw leafy vegetables contaminated with animal faeces)

- Touching infected animals or coming into contact with their faeces
- Drinking water from inadequately treated water supplies
- Swimming or playing in contaminated water, such as ponds or streams
- Contact with people who have the illness, particularly if you do not wash your hands thoroughly after using the toilet or before handling food

Source: NHS Inform, 2020.

A zoonotic public health threat

E. coli O157:H7 first emerged as a zoonotic public health threat in 1982 when two outbreaks of an unusual gastrointestinal illness affected at least 47 people in Oregon and Michigan in the US. The illness was characterised by “severe crampy abdominal pain, initially watery diarrhoea followed by grossly bloody diarrhoea” (Riley *et al.*, 1983). The infections were linked to undercooked burgers eaten in a number of branches of a well-known fast-food restaurant chain.

The first outbreak in the UK occurred in 1983, when stools from three patients tested positive in a cluster of 11 cases of HUS in children from Wolverhampton in the West Midlands (Pennington, 2014). Throughout the 1980s, there were an increasing number of outbreaks of gastrointestinal disease and HUS associated with *E. coli* O157:H7 (Yara *et al.*, 2020).

E. coli O157:H7 is a public health concern due to the potential severity of disease, ranging from mild to more severe and death. Symptoms may include watery or bloody diarrhoea, fever, abdominal cramps, nausea and vomiting. In England, over one-third of cases are hospitalised (Butt *et al.*, 2021) and 5-15 per cent of patients with STEC infections may develop HUS (Byrne *et al.*, 2017), which is the most common cause of kidney failure in young children (WHO, 2018b).

Cases in the UK

E. coli O157:H7 is considered a significant gastrointestinal pathogen in the UK and around 700 cases of infection are reported annually in England (Byrne *et al.*, 2017). It can be easily spread because very few bacteria are required to cause infection (HSE, 2021). In 2018, there were over 600 laboratory-confirmed cases of *E. coli* O157:H7 in England and Wales, equating to one case per 100,000 population (Public Health England, 2021). Of those infected,

almost a third (28 per cent) were hospitalised and HUS occurred in 14 cases – most were under five years old. One death was reported (Public Health England, 2020).

In the same year, 567 cases of different serotypes were confirmed in England, the most common of which was *E. coli* O26. Out of 303 of these non-O157 cases, 75 were hospitalised, 24 developed HUS and four deaths were reported (Public Health England, 2020).

The effects of an outbreak can be devastating. During the 1994 West Lothian (Redhouse Dairy) milk-borne outbreak affecting a rural community west of Edinburgh, more than 100 people were involved. One child died early in the outbreak and 24 were admitted to hospital. Ten children developed HUS and six needed dialysis. A year after the outbreak, one child had had a kidney transplant and two still had no kidney function and were receiving dialysis; one had developed diabetes and the other required nasogastric feeding. Two other children had reduced kidney function. The dairy in question had obtained milk from feeder farms and pasteurised, bottled and delivered it to more than 1,000 customers and some retail outlets. *E. coli* O157:H7 was detected in the stools of 69 patients, bottling apparatus at the dairy, raw milk from a farm supplying the dairy and bovine faeces from the same farm. The medical, productivity loss, and outbreak control costs were estimated to be £3.2 million for the first year. Over the course of 30 years, the costs were projected to be £11.9 million (Pennington, 2014).

The main sources of outbreaks are raw or undercooked ground meat products (burgers), raw milk and raw milk products and the faecal contamination of raw vegetables (WHO, 2018b). Foodborne outbreaks in England have also been associated with contaminated raw or undercooked meat, or cooked meat which has been contaminated; raw milk and raw milk products and contaminated raw vegetables and salads (Butt *et al.*, 2021).

Faecal contamination of vegetables

Many of the earlier *E. coli* O157:H7 outbreaks were associated with undercooked meat or milk products but the balance has shifted to include increased exposure through the consumption of faecal-contaminated foods and drinks, such as salads and fruit juices, as well as direct contact with animals and their environment.

An increasing number of outbreaks are now linked to

FACTORY FARMS – A BREEDING GROUND FOR DISEASE

the consumption of contaminated fruits and vegetables (including sprouts, spinach, lettuce, coleslaw and salad). In many cases, the contamination comes from faecal residues in agricultural irrigation water or runoff. In the US, *E. coli* O157:H7 infections from faecal-contaminated fruits and vegetables increased from 11 per cent to 41 per cent from 1998 to 2007 (Chekabab *et al.*, 2013). Since 2018, outbreaks of *E. coli* linked to leafy greens – primarily faecal-contaminated romaine lettuce – have sickened 561 Americans, led to 262 hospitalisations and left seven dead (CDC, 2021a).

Contamination of fresh fruit and vegetables is associated with faecal contamination in agricultural irrigation water or runoff – the contamination comes from livestock farming. *E. coli* O157:H7 has also been found in ponds, streams, wells and water troughs, and has been found to survive for months in manure and water-trough sediments. Waterborne transmission has been reported, both from contaminated drinking-water and from recreational waters (WHO, 2018b).

E. coli from livestock faeces may persist for long periods of time on gates, stiles and other farmyard surfaces (Williams *et al.*, 2005). It may survive on grass pasture for at least five months and may remain infectious for even longer in livestock-associated water runoff. Scientists describe *E. coli* O157:H7 as “an underestimated environmental risk” (Chekabab *et al.*, 2013) providing further direct evidence of how factory farms are breeding grounds for disease.

“Whenever you have an *E. coli* outbreak in any produce, and you do a long enough investigation, eventually you will bump into a cow” says Richard Raymond, former undersecretary for the US Department of Agriculture (Carr, 2020).

Because of the vast numbers of farmed animals in the world, billions of tonnes of manure are produced every year. Physician, bestselling author and Honorary Scientific Advisor to Viva!, Dr Michael Greger, describes how: “Dairy cow and pig factories often dump millions of gallons of putrefying waste into massive open-air cesspits, which can leak and contaminate water used to irrigate our crops. That’s how a deadly faecal pathogen like *E. coli* O157:H7 can end up contaminating our spinach.”

The conditions on a typical factory farm, where large numbers of animals are confined in cramped spaces, facilitate the spread of bacterial pathogens such as *E. coli* and *Salmonella*. Bacteria are everywhere, but they

are even more widespread on livestock farms because there are so many animals and so much faeces. Of course, the livestock industry downplays the risks. Like the tobacco, asbestos and oil industries before them, there is a long history of industries subverting public health in the interests of profits.

The main pathways of infection

Because so many previous outbreaks were related to foodborne infection, the disease is often nicknamed the “burger bug” but environmental risk factors may now be even more important. The three main pathways of infection are foodborne, environmental (including direct contact with animals and their faeces and contaminated water supplies) and person-to-person transmission (Strachan *et al.*, 2006).

Person-to-person transmission may occur through the oral-faecal route, eg touching food with dirty hands. It may also occur during sex if *E. coli* are introduced to the genitals. This may be more frequent in women, because the vagina is close to the anus – where *E. coli* may come from. However, person-to-person transmission accounts for just 11 per cent of infections (Ameer *et al.*, 2021).

Prevalence in cattle

A report by Food Standards Scotland and the UK Food Standards Agency shows that the overall prevalence of *E. coli* O157:H7 in cattle is similar across Great Britain, and has remained relatively consistent in Scotland over the last decade with over 20 per cent of farms and 10 per cent of animals testing positive for *E. coli* O157:H7 based on faecal pat sampling. The report concluded that health authorities and the general public should continue to regard all cattle farms as potentially harbouring *E. coli* O157:H7 and continue to recognise the significance of cattle as a potential source of human infection (FSA, 2018a).

As the number of factory farms rises, and the numbers of animals in them increases, the risk of environmental contamination rises too and it follows that the risk of *E. coli* infections in humans will persist. Add to that the fact that the overuse of antibiotics is turning intensive farms into superbug factories, factory farming presents a multifaceted public health threat. We are playing a deadly game of What’s the time, Mr Wolf!



STAPHYLOCOCCUS AUREUS AND MRSA

Staphylococcus aureus is recognised worldwide as a pathogen causing many serious diseases in humans and animals. Since the 1960s, a strain resistant to penicillin and methicillin (and now, several other antibiotics), called methicillin-resistant *S. aureus* (MRSA) has spread as a human hospital-acquired pathogen throughout the world. More recently, community-acquired and livestock-associated MRSA have emerged and are an increasing source of concern.

S. aureus is a type of bacteria found on people's skin. They are usually harmless, but can cause serious infections that can lead to sepsis or death. In 1959, the antibiotic methicillin was introduced in the UK to treat infections caused by penicillin-resistant *S. aureus* but then in 1961 there were reports from the UK of strains that had acquired resistance to methicillin. MRSA isolates were soon recovered from other European countries and later from Japan, Australia and the US (Enright *et al.*, 2002).

Infections with methicillin-resistant *S. aureus* (MRSA) can be harder to treat than other bacterial infections as they are resistant to several widely used antibiotics (NHS, 2020).

S. aureus lives harmlessly on the skin of around one in 30 people – usually in the nose, armpits, groin or buttocks. Around one to three per cent of the population are colonised with MRSA and in most cases no treatment is necessary, as colonisation does not lead to any harmful infection. This is known as “carrying” MRSA.

You can get MRSA on your skin by touching someone who has it, sharing towels, sheets or clothes with someone who carries it, or touching surfaces or objects that have MRSA on them. Getting MRSA on your skin will not make you ill, and it may go away in a few hours, days, weeks or months without you noticing. But it could cause an infection if it gets deeper into your body (NHS, 2020).

MRSA can cause harm when it enters the body. It may cause skin infections, pimples or boils, or more serious potentially life-threatening problems such as wound or chest infections (pneumonia). In some cases, invasive MRSA infection may affect heart valves or bones and can lead to abscesses in organs, joint infections, or bloodstream infection (sepsis).

FACTORY FARMS – A BREEDING GROUND FOR DISEASE

Hospital patients are most at risk of this happening because they often have a way for the bacteria to get into their body, such as a wound, burn, feeding tube, drip into a vein or a urinary catheter. They may also have other serious health problems that mean their body is less able to fight off the bacteria. Healthy people, including children and pregnant women, are not usually at risk of MRSA infections (NHS, 2020).

MRSA remained largely confined to hospital settings, where it was first detected, until the 1990s (Aires-de-Sousa, 2017). Since then, there has been an explosion in so-called community-acquired MRSA, responsible for a large proportion of the increased disease burden observed in recent years (David and Daum, 2010). MRSA is no longer exclusive to hospitals and the prevalence of community-acquired infections in people that have had no contact with hospital environments is increasing.

Livestock-associated MRSA

In recent years, new livestock-associated strains of MRSA have also been emerging (Igrejas *et al.*, 2018). Farmed animals can be carriers, can become infected or can act as a reservoir for transmission to humans (Haag *et al.*, 2019). This versatile pathogen has established new reservoirs and is now widespread in the community and animals, especially livestock (Aires-de-

Sousa, 2017). Rising numbers of MRSA infection and colonisation in farmed animals illustrate how MRSA has become an important zoonotic pathogen.

Different strains of MRSA have emerged in livestock across different regions of the world. The predominant livestock-associated strain in Europe and North America is called ST398 (Pirolo *et al.*, 2019). MRSA ST398 has been reported in farmed animals from various countries. It is most prevalent in pigs, which are a major reservoir of ST398 in Western countries, including Italy, where a high prevalence has been documented in farms over the past decade (Pirolo *et al.*, 2019). In the Netherlands, the largest exporter of live pigs in Europe, up to 39 per cent of pigs carry MRSA in their nostrils or nasal passages (Krziwanek *et al.*, 2009).

The first report of MRSA in pigs was from the Netherlands in 2005 (Voss *et al.*, 2005). In this study, a pig, a six-month-old girl and her parents who lived on a pig farm, as well as other pig farmers, were all colonised by the same strain of MRSA ST398. Soon after the isolation of MRSA in Dutch pigs, ST398 was recovered from pigs in different European countries (Aires-de-Sousa, 2017). A large European survey looking for MRSA in the dust of pig holdings, detected it in 17 out of 26 countries. Higher rates were found in countries with higher densities of pig farming (Aires-de-Sousa, 2017).



In the UK, *S. aureus* is one of the three main bacteria that cause mastitis in dairy cows, along with *Streptococcus uberis* and *E. coli*. In Belgium, MRSA is an established cause of clinical mastitis in cows (Fergestad *et al.*, 2021). After the first report of MRSA in bovine mastitis in Belgium, increasing numbers of cases of MRSA in cows and cow's milk have been reported from all over the world and research shows that ST398 has also emerged in cattle and been found in beef samples (Aires-de-Sousa, 2017).

MRSA ST398 isolates have also been identified in Europe in healthy and diseased chickens as well as in turkeys. This finding, researchers say, indicates that the animal reservoir of MRSA ST398 is broader than previously thought. This, they add, may pose a public health hazard, since it has been shown that MRSA ST398 has zoonotic potential, causing infections in people in contact with carrier animals (Nemati *et al.*, 2008).

Why factory farms are a problem

Livestock-associated strains may evolve in factory farms because of the use of antibiotics used as feed additives for growth promotion in industrial livestock and poultry, for prevention of disease within a herd, or for treatment of an existing disease outbreak (Smith, 2015). Antibiotics used in livestock include many that are also important for human health, including tetracyclines, macrolides, penicillins and sulfonamides, among others (Smith, 2015). The inappropriate use of antibiotics in the treatment of human diseases and non-therapeutic use in animals may have played a significant role in the emergence of resistant strains. Such resistance may pose a great threat to public health if livestock-associated strains enter the community and healthcare settings (Mehndiratta and Bhalla, 2014).

Livestock-associated MRSA ST398 is now recognised as an occupational hazard for people who work in direct contact with livestock, especially for farmers, abattoir workers and veterinarians. But even just living close to a high-density livestock production facility (factory farm) can increase your risk of exposure (Pirolo *et al.*, 2019). One study found livestock-associated MRSA colonisation in 24-86 per cent of pig farmers, 31-37 per cent of dairy and beef farmers, and nine to 37 per cent of poultry farmers, as well as 44-45 per cent of pig-care veterinarians in European countries (Anjum *et al.*, 2019).

Antibiotic resistance generated in animal agriculture may spread to people in a number of different ways,



including: contact with contaminated meat products (via handling or ingestion); occupational contact (farmers, meat-packers, butchers etc) and secondary spread into the community from those who are occupationally exposed; entry into and transmission via hospitals and other healthcare facilities; or spread via environmental routes including air, water or manure in areas close to livestock farms or crop farms where manure has been used as fertiliser (Smith, 2015).

Although MRSA ST398 is associated with livestock, scientists say that it is a major public health threat to humans in close contact with livestock. A very high prevalence of MRSA ST398 was reported among pig farmers and pigs in Spain, and the data suggests animal-to-human transmission. The cross-transmission between animals and humans, scientists say, poses a high zoonotic threat (Dweba *et al.*, 2018).

MRSA ST398 has emerged in humans in Europe, China, Thailand and Canada, indicating a great potential for spread. Because the international meat and livestock market is so active, scientists warn that the stage is set for a rapid spread (Krziwanek *et al.*, 2009).

As we are fast-approaching a post-antibiotic era, the effectiveness of antibiotics used to treat human illnesses may be reduced if pathogens and resistance genes from the agricultural environment are repeatedly, but silently, being introduced into the human population (Smith, 2015).

SALMONELLA – ARE EGGS REALLY SAFE?

Salmonellosis is an infectious zoonotic disease usually caused by eating food contaminated with *Salmonella* bacteria. Typically, *Salmonella* food poisoning causes gastroenteritis, leading to stomach cramps, nausea, diarrhoea, vomiting and fever. Symptoms usually develop between 12 and 72 hours after becoming infected and illness usually lasts from four to seven days.

Salmonella generally causes a mild illness, but vulnerable groups like children under five, the elderly and those with weakened immune systems may experience more severe illness and may require hospitalisation (FSA, 2021). Invasive *Salmonella* infections, that spread into parts of the body such as the blood, are rare but can be severe and life-threatening. In the UK, although *Campylobacter* is the most common foodborne pathogen, *Salmonella* is responsible for the most hospital admissions.

Salmonella enterica is the most pathogenic species and can be divided into two main groups – typhoidal and nontyphoidal. *Salmonella* Typhi (not to be confused with *Salmonella* Typhimurium) and Paratyphi can cause typhoid fever and paratyphoid fever respectively. Both

tend to occur in developing countries with poor sanitation and infection is uncommon in the UK. They are both adapted to humans and are not considered to be zoonotic.

Salmonella enterica have more than 2,500 different serotypes (their names are not italicised). They are members of the same species but classified differently according to their distinctive surface structures. Nontyphoidal serotypes are more common and can cause gastrointestinal disease. They can infect a range of animals and are zoonotic. Two serotypes are responsible for around half of all salmonellosis cases in humans; *Salmonella* Enteritidis and *Salmonella* Typhimurium (HM Government, 2018).

Between 1981 and 1991, the incidence of salmonellosis in the UK rose by over 170 per cent due to an increase in *S. Enteritidis* infections. The UK was not alone; the World Health Organisation's *Salmonella* surveillance system showed that during the late 1980s, infections with *S. Enteritidis* increased on several continents, with North America, South America and Europe appearing to bear the brunt (O'Brien, 2013).



The problem with factory farms

Salmonella bacteria are found in the gut of many farmed and wild animals including poultry, pigs, cows, hedgehogs, snakes and lizards. Poultry, pigs and cows are a significant source of this food poisoning bug but they may show no signs of infection and infected foods usually look and smell normal. Factory farms provide the ideal environment for *Salmonella* bacteria to spread as they are shed in the faeces of infected animals. Research shows that poor ventilation, high levels of dust and overcrowding all help spread *Salmonella* among poultry and pigs.

A large-scale UK survey of egg-laying poultry farms found that cage farms were more than six times more likely than non-cage farms to be infected with the strain of *Salmonella* most commonly associated with food poisoning. Large farms with over 30,000 birds were more than 14 times as likely to test positive for *S. Enteritidis* as those with less than 3,000 birds (Snow *et al.*, 2010).

Salmonella in meat, eggs and dairy foods

Salmonella bacteria are usually found in raw or undercooked meat, raw eggs, milk and other dairy products. If contaminated meat is minced, as is the case with some chicken products, the bacteria become incorporated throughout the meat, which, if inadequately cooked, can lead to food poisoning. Poultry are especially likely to carry *Salmonella* which is why it is often found in raw eggs from chickens which have not been vaccinated (see below). Other foods like fruit, vegetables and shellfish can become contaminated through contact with animal waste that has been used on land or water.

In 1988, then junior health minister Edwina Currie angered farmers and egg producers by warning the British public on prime-time TV that: "Most of the egg production in this country, sadly, is now infected with *Salmonella*." Egg sales plummeted by 60 per cent and Currie was forced to resign. In 2001, a Whitehall report written months after Currie resigned was published, showing she had been largely correct. The study by the Ministry of Agriculture, Department of Health and the British Egg Industry Council said that Britain was experiencing a "salmonella epidemic of considerable proportions" in late 1988 (Wasley and Heal, 2019).

Measures introduced to control this epidemic included legislation, food safety advice and a voluntary, industry-

led vaccination scheme against *S. Enteritidis* and *S. Typhimurium* that began in broiler-breeder flocks in 1994 and in laying flocks in 1998 (O'Brien, 2013). Also in 1998, the egg industry launched the British Lion code of practice to try and rebuild consumer confidence by certifying the safety of eggs sold with that stamp.

Vaccines have now been used for over 20 years as one of the measures to reduce the risk of poultry infection with *Salmonella* (Defra, 2010). Most commercial egg-laying flocks in the UK are now vaccinated and eggs that carry the British Lion mark have been laid by hens vaccinated against *Salmonella*. In 2017, the Food Standards Agency declared Lion Mark eggs virtually free of *Salmonella* and Heather Hancock, then chairman of the Food Standards Agency, said: "The risk of *Salmonella* is now so low you needn't worry."

However, in 2019, the Bureau of Investigative Journalism revealed that more than 100 people had been poisoned by British eggs contaminated with *Salmonella* over the previous three years – 45 cases in 2019 alone. According to the bureau, 25 egg-laying poultry flocks tested positive in 2019, seven of them infected with the most serious strain. In 2018, 28 flocks tested positive for *Salmonella*, four of them with dangerous strains. Two egg-packing factories were also said to have been contaminated. The potentially fatal bacteria were traced to British poultry farms, despite government assurances that the risk had been virtually eliminated (Wasley and Heal, 2019).

Not so finger lickin' good!

Some outbreaks have been linked to imported chicken products. In 2021, an ongoing outbreak of *Salmonella* poisoning in the UK, dating back to early 2020, was linked to imported frozen raw chicken products. The UK Food Standards Agency and Public Health England joined forces in a multi-agency investigation into the rise in cases of *Salmonella* linked to frozen processed chicken products, such as nuggets, goujons, dippers, poppers and kiev. By 2021 at least one person had died and around 500 had been poisoned in the ongoing outbreak (FSA, 2021). Public Health England said that more than a third of the cases required hospital treatment and confirmed that *Salmonella* was recorded as a contributory cause of death in at least one case.

An investigation into the two strains of *Salmonella* found in the frozen raw breaded chicken products confirmed that they originated in Poland. The Food

FACTORY FARMS – A BREEDING GROUND FOR DISEASE

Standards Agency reminded consumers to take care when handling and cooking chicken products at home. They issued a number of product recalls from supermarkets to try and stop the spread, including a range of Southern-fried chicken products, nuggets, chip-shop curry chicken breast toppers and a take-home boneless bucket.

While the vaccination scheme may have reduced infection rates, vaccination is only partially protective and control of infection also depends on how the vaccine is given and good farm management to minimise contamination. In 2018, the Government's *Zoonoses report* found that *Salmonella* cases were rising. The report said that in 2017, 10,089 cases of laboratory confirmed salmonellosis were reported in the UK, 470 more than in the previous year (ACMSF, 2018). However, they estimate that for every laboratory-confirmed case there may be almost five cases in the community, suggesting the total number of cases in 2018 was closer to 50,000 (HM Government, 2018). The report said that thirteen foodborne outbreaks were reported in the UK in 2017 compared to 12 in 2016. Two were caused by *S. Enteritidis* and five by *S. Typhimurium* but other outbreaks were caused by different serotypes including *S. Adjame*, *S. Agona*, *S. Chester*, *S. Give*, *S. Infantis* and *S. Stanley*.

Vaccination schemes in intensive poultry farming could be contributing to larger problems. It has been suggested that vaccines against *S. Enteritidis* and *S. Typhimurium*, the targets of most vaccines, may be clearing the way for new serotypes to prosper. In commercial broilers in the US, for example, as *S. Enteritidis* has declined, *S. Heidelberg* and *S. Kentucky* (named after the state rather than the famous fried chicken brand) have become more predominant (Foley *et al.*, 2011). This may be leading us into unknown territory as the ecology and epidemiology of these serotypes isolated from poultry remain poorly understood (Shah *et al.*, 2017). Not so finger lickin' good!

Another significant concern is that antimicrobial resistance (AMR) has become increasingly seen among *Salmonella* serotypes that are frequently isolated from poultry (Shah *et al.*, 2017). In recent years, the development of AMR among foodborne pathogens such as *Salmonella* has led to longer and more expensive treatment in hospital, as antibiotics no longer work, and an increasing number of human deaths.

Recently, the increasing prevalence of multi-drug resistant *Salmonella*, with resistance towards clinically important antimicrobials like fluoroquinolones and third-generation cephalosporins, has become an emerging problem worldwide (Jajere, 2019).

Scientists say that reports of multi-drug resistant strains of the 12 most prevalent poultry-associated *Salmonella* serotypes (MPSSTS), including *S. Kentucky*, *S. Schwarzengrund*, *S. Hadar*, *S. Thomson*, *S. Sentfenberg* and *S. Enteritidis*, from different parts of the world are alarming. Some of these have been documented to spread internationally through contaminated foods as well as via travellers. The international spread of these resistant *Salmonella*, they warn, may pose a significant challenge to public health worldwide (Shah *et al.*, 2017).

Going vegan is no guarantee that you will avoid food poisoning altogether, but it certainly lowers the risk substantially. So, swap chicken for chickpeas and slash your risk of food poisoning!





CAMPYLOBACTER – TOP OF THE FOOD POISONING TABLE

Campylobacter is the main cause of food poisoning in the UK and it is estimated that there are more than half a million cases and 80,000 GP consultations every year in the UK (FSA, 2019). Specifically, *Campylobacter jejuni* accounts for around 90 per cent of all *Campylobacter* infections in humans (CDC, 2019a).

Symptoms include diarrhoea (may be bloody), vomiting, stomach pains, cramps, fever and generally feeling unwell. They usually develop within two to five days after becoming infected but can take as long as 10 days to appear. Illness usually lasts around seven days.

Most people with *Campylobacter* poisoning will recover fully but it can cause long-term and severe health problems in some. Like other food poisoning bugs, children under five and older people are most at risk because they may have weaker immune systems.

Like *E. coli* O157:H7, *Campylobacter* has a low infective dose, that means coming into contact with just a few bacteria can cause illness. This is especially important if

you are young or have an underlying illness. You can't see, smell it or even taste *Campylobacter* on food but if it affects you, the Food Standards Agency says, you won't forget it! (FSA, 2018a).

Poo is a problem – *Campylobacter* in farmed animals

Campylobacter bacteria are prevalent in farmed animals such as chickens, pigs, cattle and sheep, and have also been found in shellfish (WHO, 2020c). They are frequently carried in the faeces of chickens, pigs and cattle, but in animals, they don't normally cause disease. The main route of transmission to people is generally thought to be foodborne, via undercooked meat and meat products, as well as raw or faecal-contaminated milk. Most often, carcasses or meat are contaminated by *Campylobacter* from faeces during slaughtering (WHO, 2020c).

FACTORY FARMS – A BREEDING GROUND FOR DISEASE

The drive for cheap meat means that animal food processors are constantly striving to develop faster ways of processing carcasses. Mechanical evisceration (removal of internal organs) of poultry can result in the rupturing of their digestive tracts and the spilling of faecal matter onto the skin of the animal (Cho *et al.*, 2009). This is obviously a concern with poultry, as some people eat the skin. Companies selling this type of equipment boast how their machines can deal with high processing capacities, now heading towards being able to process 15,000 birds per hour.

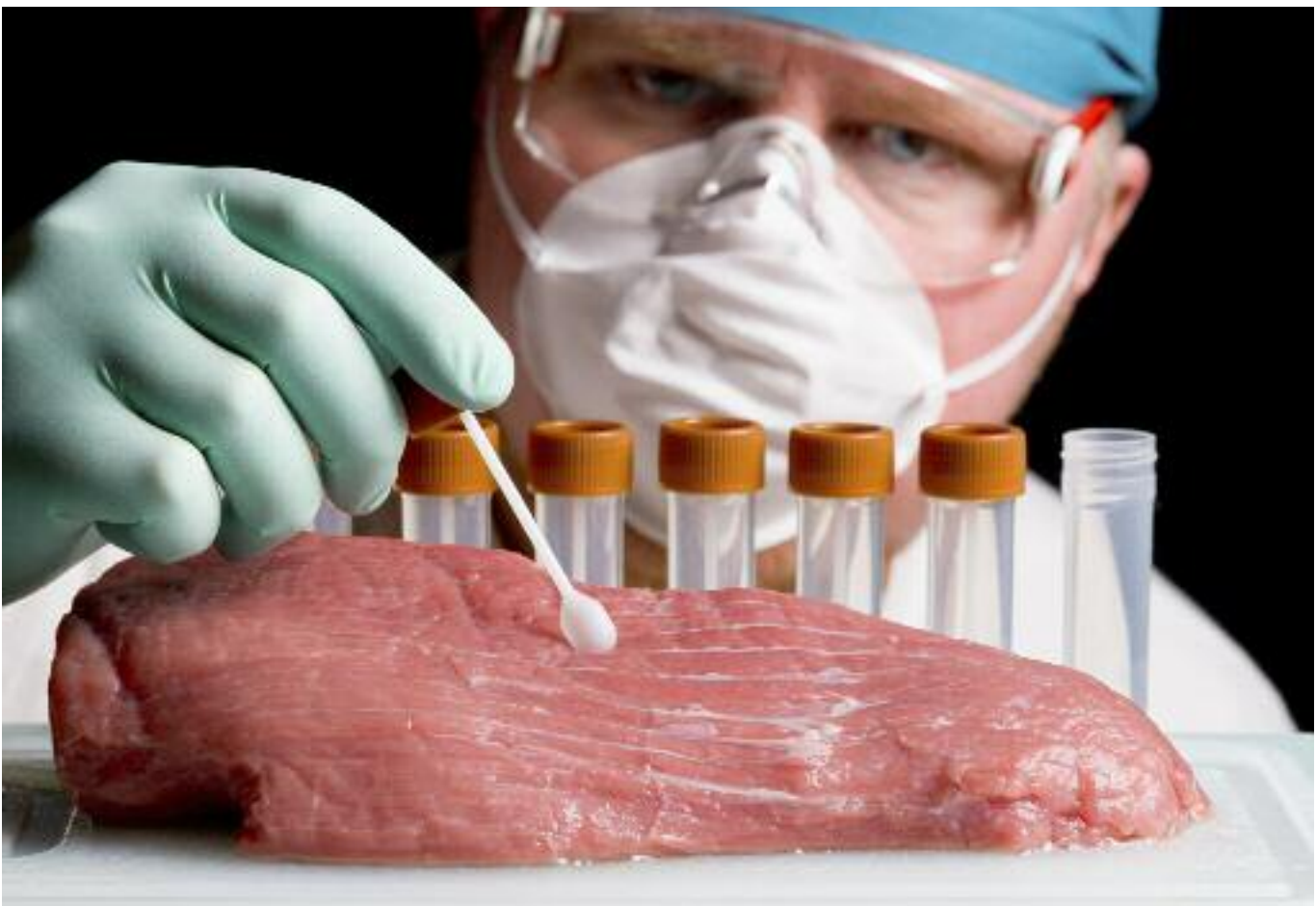
Chicken meat is known to be a major source of *Campylobacter* infection. The first UK government survey of *Campylobacter* contamination in retail chickens was published in 2015. The 12-month survey, carried out by the Food Standards Agency, looked at the levels of *Campylobacter* contamination in fresh whole chickens from large retail outlets, small independent shops and butchers. They tested over 1,000 samples and found that 73 per cent tested positive for *Campylobacter* (FSA, 2015).

A year later they repeated the survey, again testing just over 1,000 samples of fresh whole UK-produced chickens between January and March 2016. They found

Campylobacter in 50 per cent of samples. At first this looks like a drop, down from 76 per cent in the same quarter of the previous year. However, one reason these results were lower was because several retailers and their suppliers decided to remove neck skin from chickens *before* they were sold. The neck skin is the most contaminated part of the chicken and is the part of the bird that the Food Standards Agency had been testing. So, like-for-like comparisons were not possible (FSA, 2016).

In more recent tests, they reported that *Campylobacter* was found in 54 per cent of fresh, whole retail chicken in the UK, tested from August 2016 to July 2017 (FSA, 2019), 56 per cent of samples taken from August to October 2017 and 75 per cent of samples from smaller retailers tested between August 2017 to July 2018 (FSA, 2019). *Campylobacter* is not going away!

The Food Standards Agency puts *Campylobacter* at the top of their priority list as they consider it to be one of the biggest food safety problems affecting people in the UK. They tell people not to wash raw chicken, warning that splashed water droplets can spread *Campylobacter* bacteria on to human skin, work surfaces, clothing and cooking equipment.



Intensive farming practices increase the risk of zoonotic disease outbreaks. *C. jejuni* is not just a problem in poultry, it is also carried by 20 per cent of cattle worldwide. An international team of scientists, led by the Universities of Bath and Sheffield, investigated the evolution of this bacterium and found that the overuse of antibiotics, high animal numbers and low genetic diversity has led to the emergence of cattle-specific variants that can infect humans too, triggering a major public health risk (Mourkas *et al.*, 2020).

They found that cattle-specific strains of the bacterium emerged at the same time as the dramatic rise in cattle numbers in the 20th Century. Changes in how cows were farmed provided the opportunity for gene transfer between general and cattle-specific strains helping this pathogen cross the species barrier and infect humans. Combine this with the increased movement of animals through international trade, they say, intensive farming practices are providing the perfect environment for pathogens to spread globally (Mourkas *et al.*, 2020).

Around one in seven people suffer from an infection at some point in their life caused by *Campylobacter*. It causes three times more cases than *E.coli*, *Salmonella* and *Listeria* combined (Mourkas *et al.*, 2020). Professor Sam Sheppard, from the University of Bath, says: "There are an estimated 1.5 billion cattle on Earth, each producing around 30 kilograms of manure each day. If roughly 20 per cent of these are carrying *Campylobacter*, that amounts to a huge potential public health risk."

Sheppard says: "Over the past few decades, there have been several viruses and pathogenic bacteria that have switched species from wild animals to humans: HIV started in monkeys, H5N1 came from birds, now Covid-19 is suspected to have come from bats. I think this is a wake-up call to be more responsible about farming methods, so we can reduce the risk of outbreaks of problematic pathogens in the future" (University of Sheffield, 2020).

Another emerging problem is the potential for antibiotic-resistant superbugs to spread from livestock to humans. In 2018 the UK Foods Standards Agency reported that ciprofloxacin resistance was found in 54 per cent of *C. jejuni* samples from retail chicken (FSA, 2018b). The antibiotic ciprofloxacin is used in humans to treat serious infections, or infections for which other antibiotics have not worked. It belongs to a group of antibiotics called fluoroquinolones which are classified by the World Health Organisation as "critically important in human medicine" (WHO, 2011). The Alliance to Save Our Antibiotics has long campaigned for a total ban on the use of fluoroquinolones in poultry. However, their use is still permitted in UK poultry (and other farmed animals) and Red Tractor standards also allow their use in chickens and turkeys.

In 2017, over 56,000 cases of *Campylobacter* infections were reported in England and Wales, equivalent to 97 per 100,000 population (Public Health England, 2017). It's difficult to find out how many of these were caused by antibiotic-resistant strains and how the infections were transmitted. However, a freedom of information request submitted in 2016 by the Bureau of Investigative Journalism revealed that almost half of all human infections in 2015 with *Campylobacter* were resistant to ciprofloxacin (Alliance to Save Our Antibiotics, 2018).

Factory farming provides the perfect conditions for bacteria to adapt and spread from animals to humans, increasing the risk of epidemics. The ever-increasing drive to lower the cost of meat inevitably results in more intensive farming and processing methods. But as we are continually finding out, cheap meat comes at a high cost!



FINAL WARNING

Apparently, we are a nation of animal lovers, yet we continue to allow, even support, the systemic abuse of millions of farmed animals, year after year. Most of the meat people eat today comes from genetically uniform, immunocompromised, regularly drugged animals packed by the thousands into filthy sheds or stacked cages. The perfect environment for emerging diseases.

In Britain alone, over a billion farmed animals are killed every year in slaughterhouses. Every day in the UK over two-and-a-half million chickens are slaughtered for meat – that’s 30 deaths every second. Viva! has filmed in a number of intensive broiler “meat” chicken units, finding grim, windowless sheds crammed with thousands of miserable birds kept under artificial light. We estimate that there could have been up to quarter of a million chickens on one farm we visited.

Viva!’s recent investigations at Hogwood pig farm in Warwickshire and Flat House Farm in Leicestershire exposed extreme overcrowding, routine mutilation, cruel farrowing crates, sick and dying pigs abandoned in gangways, the dead left to rot amongst the living, painful lacerations from brutal assaults and live cannibalism. There are many, many other farms just like these, providing an ideal breeding ground for disease.

The next pandemic might come from a poultry farm or a pig farm – as the 2009 swine flu pandemic did. It may be caused by an antibiotic-resistant superbug or a virus. We are fast-approaching a post-antibiotic era where infections and minor injuries may mean a death sentence. It’s predicted that antibiotic-resistant infections might kill more people than cancer by 2050 if we carry on using so many antibiotics in animal agriculture.

Some avian influenza viruses kill more than 50 per cent of those infected. If one mutates to become more easily spread, like seasonal flu, we could be facing anything from five to 150 million deaths says David Nabarro, one of the most senior public health experts at the World Health Organisation.

More and more scientists are acknowledging the role that eating animals plays in the emergence of infectious diseases. Writing in the journal *Disaster Medicine and Public Health Preparedness*, scientists said that the world must now implement stricter rules on food

hygiene and consumption globally. They said: “International communities and organisations have strict rules against atomic bombs, chemical and biological weapons, wars, and many other traumatic events and all are more or less adhering to the rules. However, there are no such international rules on food consumption” (Farnoosh *et al.*, 2020). The best way to prevent the spread of contagious and deadly diseases, the authors of this study say, is to modify food culture worldwide. That means ending factory farming as well as wildlife markets.

If we ignore the facts, the crisis of the coronavirus pandemic will be repeated as the reckless exploitation of animals endangers lives around the world. Scientists have been warning us for years about the potential pandemic threat posed by food systems. It’s likely that Covid-19 came from a wet market in China. The dreadful, cramped conditions and mix of wild and domestic creatures alongside throngs of people, provided an ideal environment for an emerging zoonotic disease.

However, a pandemic risk resulting from the ill-treatment of animals lies closer to home – in a factory farm near you! As industrialised farming has spread around the world, diseases have followed. Densely packed sheds containing stressed animals, confined in filthy surroundings, with low immunity having been bred for fast growth. It’s an ideal environment for a mutating bacterium, virus or antibiotic-resistant superbug to emerge. It’s no surprise, therefore, that three in four new or emerging infectious diseases come from animals. Scientists say: “It is the *system* of factory farming that presents the relevant risks and we can never know in advance at which farm the next pandemic will originate” (Bernstein and Dutkiewicz, 2021).

In 2020, a report called *Preventing the Next Pandemic: Zoonotic diseases and how to break the chain of transmission*, a joint effort from the United Nations Environment Programme (UNEP) and the International Livestock Research Institute, warned that further outbreaks will emerge unless governments take active measures to prevent other zoonotic diseases from crossing into the human population. The message could not be starker – unless we address the causes of the coronavirus pandemic, the ongoing destruction of

FACTORY FARMS – A BREEDING GROUND FOR DISEASE

nature will lead to more animal diseases spreading to humans (UNEP and International Livestock Research Institute, 2020).

The report said that while wildlife is a common source of emerging infectious diseases, farmed animals are also original sources, transmission pathways and amplifiers of zoonotic diseases. Industrial farming of animals, especially pigs and chickens, is one of the primary risks for future spillover of zoonotic diseases. A key message in the report is that: “Pandemics such as the Covid-19 outbreak are a predictable and predicted outcome of how people source and grow food, trade and consume animals and alter environments” (UNEP, 2020).

Doreen Robinson, UNEP’s Chief of Wildlife told the *Guardian*: “Human activity is breaking down the natural buffer that once protected people from a number of pathogens. It’s critically important to get at the root causes, otherwise we will consistently just be reacting to things” (Carrington, 2020).

The UN report looks at the causes of the emergence and spread of Covid-19 and other zoonotic diseases, with the aim of helping policymakers prevent future outbreaks. It identifies a number of factors driving zoonotic disease outbreaks; the rising demand for animal protein, more intensive and unsustainable farming practices, greater exploitation of wildlife, surging global travel and the climate crisis.

The One Health approach shared by a number of health organisations recognises that the health of people is closely connected to the health of animals and our shared environment. The UNEP report builds on the One Health approach as the best hope for preventing future pandemics. Adopting this approach will, the report says, unite medical, veterinary and environmental experts, in order to help governments, businesses and society in

general achieve enduring health for people, animals and environments alike.

Despite all the warnings from international bodies like the World Health Organisation and UNEP, there remains a clear lack of political will to change the way we eat but people are making up their own minds – for the animals, the planet and their health. Market researchers Euromonitor International says that more consumers are shifting to vegan and vegetarian diets with those restricting animal-based foods (flexitarians) accounting for over 40 per cent of global consumers in 2020 (Euromonitor International, 2020).

As we emerge out of the coronavirus pandemic, many people are looking forward to getting back to normal. But if we’ve learnt anything, it’s that “normal” is what got us into this mess! Infectious-disease physician Dale Fisher told the BBC: “One of the worst things we can do is when this is over, we just go back to normal. If you don’t learn from it, then history will repeat itself.”

If one of the Government’s responsibilities is to protect public health, then they have an obligation to change the way animal foods are produced and end factory farming. Our relationship with animals and the environment can no longer just focus on exploitation. The way animal foods are produced is now considered to be a global threat and we have had our final warning. We must end factory farming now before it ends us.



REFERENCES

- ACMSF. 2018. Advisory committee on the microbiological safety of food epidemiology of foodborne infections group (efig). https://acmsf.food.gov.uk/sites/default/files/acm_1284_fig.pdf
- Aguirre AA, Catherina R, Frye H *et al.* 2020. Illicit Wildlife Trade, Wet Markets, and COVID-19: Preventing Future Pandemics. *World Medical Health Policy*. 10.1002/wmh3.348.
- Aires-de-Sousa M. 2017. Methicillin-resistant *Staphylococcus aureus* among animals: current overview. *Clinical Microbiology and Infection*. 23, 6, 373-380.
- Ajani A. 2020. Blame it on the farm too. <https://www.sierraclub.org/sierra/blame-it-farm-covid-coronavirus-agriculture-coronavirus-covid-19>
- Alexander DJ and Brown IH. 2009. History of highly pathogenic avian influenza. *Revue Scientifique et Technique*. 28 (1) 19-38.
- Alliance to Save Our Antibiotics. 2018. Government must ban all use of fluoroquinolone antibiotics in poultry as new FSA survey reveals record levels of resistance. <https://www.saveourantibiotics.org/news/press-release/government-must-ban-all-use-of-fluoroquinolone-antibiotics-in-poultry-as-new-fsa-survey-reveals-record-levels-of-resistance/>
- Alliance to Save Our Antibiotics. 2020. <https://www.saveourantibiotics.org/media/1864/farm-antibiotics-and-trade-could-uk-standards-be-undermined-asoa-nov-2020.pdf>
- Ameer MA, Wasey A, Salen P. 2021. *Escherichia Coli* (E Coli O157 H7). In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2021 Jan. PMID: 29939622.
- Anjum MF, Marco-Jimenez F, Duncan D *et al.* 2019. Livestock-Associated Methicillin-Resistant *Staphylococcus aureus* From Animals and Animal Products in the UK. *Frontiers in Microbiology*. 10, 2136.
- Antibiotic Research UK. 2018. News Release: Horsefly bite death threat wings its way to Britain. <http://www.antibioticresearch.org.uk/wp-content/uploads/2015/05/Horsefly.pdf>
- Antimicrobial Resistance Fighter Coalition. 2021. Resistance Fighter's story: I'm a resistance fighter. <https://antimicrobialresistancefighters.org/stories/story-lord-jim-o-neill>
- APHA. 2020. Bovine tuberculosis in England in 2019 Epidemiological analysis of the 2019 data and historical trends. https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/923195/tb-epidemiology-england-2019.pdf
- APRHAI. 2019. APRHAI 9th annual report. <https://www.gov.uk/government/publications/aprhais-annual-report-2017-to-2018/aprhais-9th-annual-report>
- Badger Action Network. 2021. 140,830 Badgers killed by culling since 2013. <https://badgeractionnetwork.org.uk/140830-badgers-killed-by-culling-since-2013/>
- Baptiste KE and Kyvsgaard NC. 2017. Do antimicrobial mass medications work? A systematic review and meta-analysis of randomised clinical trials investigating antimicrobial prophylaxis or metaphylaxis against naturally occurring bovine respiratory disease. *Pathogens and Disease*. 75 (7) ftx083.
- Bar-On YM, Phillips R and Milo R. 2018. The biomass distribution on Earth. *Proceedings of the National Academy of Sciences USA*. 115 (25) 6506-6511.
- BBC. 2005. Bird flu could kill 150m people. <http://news.bbc.co.uk/1/hi/world/asia-pacific/4292426.stm>
- Belser JA, Blixt O, Chen LM *et al.* 2008. Contemporary North American influenza H7 viruses possess human receptor specificity: Implications for virus transmissibility. *Proceedings of the National Academy of Sciences USA*. 105 (21)7558-7563.
- Bernstein J and Dutkiewicz J. 2021. A public health ethics case for mitigating zoonotic disease risk in food production. *Food Ethics*. 6 (2) 9.
- Burki T. 2020. The origin of SARS-CoV-2. *The Lancet Infectious Diseases*. 20 (9) 1018-1019.
- Butt S, Smith-Palmer A, Shand A *et al.* 2021. Evidence of on-going transmission of Shiga toxin-producing *Escherichia coli* O157:H7 following a foodborne outbreak. *Epidemiology and Infection*. 149, e147.
- Byrne L, Kaindama L, Bentley M *et al.* 2017. Investigation into a national outbreak of STEC O157:H7 associated with frozen beef burgers, UK, 2017. *Epidemiology and Infection*. 148:e215.
- Carr T. 2020. Unclean greens: how America's E coli outbreaks in salads are linked to cows. <https://www.theguardian.com/environment/2020/sep/01/unclean-greens-how-americas-e-coli-outbreaks-in-salads-are-linked-to-cows>
- Carrington D. 2020. Coronavirus: world treating symptoms, not cause of pandemics, says UN. <https://www.theguardian.com/world/2020/jul/06/coronavirus-world-treating-symptoms-not-cause-pandemics-un-report>
- CDC. 2009. Origin of 2009 H1N1 Flu (Swine Flu): Questions and Answers. https://www.cdc.gov/h1n1flu/information_h1n1_virus_qa.htm
- CDC. 2019. Types of Influenza Viruses. <https://www.cdc.gov/flu/about/viruses/types.htm>
- CDC. 2019a. *Campylobacter* (Campylobacteriosis). <https://www.cdc.gov/campylobacter/technical.html>
- CDC. 2021. Antibiotic / Antimicrobial Resistance. Food and food animals. <https://www.cdc.gov/drugresistance/food.html>
- CDC. 2021a. List of Selected Multistate Foodborne Outbreak Investigations. <https://www.cdc.gov/foodsafety/outbreaks/multistate-outbreaks/outbreaks-list.html>
- Chan JF, Yuan S, Kok KH *et al.* 2020. A familial cluster of pneumonia associated with the 2019 novel coronavirus indicating person-to-person transmission: a study of a family cluster. *The Lancet*. 395 (10223)514-523.

FACTORY FARMS – A BREEDING GROUND FOR DISEASE

- Chekabab SM, Paquin-Veillette J, Dozois CM *et al.* 2017. The ecological habitat of *E. coli* O157. *Journal of Microbiological Letters*. 341 (1) 1-12.
- Cheng ZJ, Qu HQ, Tian L, *et al.* 2020. COVID-19: Look to the Future, Learn from the Past. *Viruses*. 12 (11) 1226.
- Cho B, Kim MS, Chao K *et al.* 2009. Detection of fecal residue on poultry carcasses by laser-induced fluorescence imaging. *Journal of Food Science*. 74 (3) E154-9.
- Collinge J, Whitfield J, McKintosh E *et al.* 2006. Kuru in the 21st century—an acquired human prion disease with very long incubation periods. *The Lancet*. 367, 2068-2074.
- Daszak P, Zambrana-Torrel C, Bogich TL *et al.* 2013. Interdisciplinary approaches to understanding disease emergence: the past, present, and future drivers of Nipah virus emergence. *Proceedings of the National Academy of Sciences USA*. 110 (Suppl 1) 3681-3688.
- David MZ and Daum RS. 2010. Community-associated methicillin-resistant *Staphylococcus aureus*: epidemiology and clinical consequences of an emerging epidemic. *Clinical Microbiology Reviews*. 23 (3) 616-687.
- Dawood FS, Iuliano AD, Reed C *et al.* 2012. Estimated global mortality associated with the first 12 months of 2009 pandemic influenza A H1N1 virus circulation: a modelling study. *Lancet Infectious Diseases*. 12 (9) 687-695.
- Defra. 2010. Zoonosis report UK 2008. <https://webarchive.nationalarchives.gov.uk/20130402162428/http://archive.defra.gov.uk/foodfarm/farmanimal/diseases/atoz/zoonoses/documents/report-2008.pdf>
- Defra. 2021. Avian Influenza. <https://www.daera-ni.gov.uk/articles/avian-influenza-ai>
- Defra. 2021a. Bovine tuberculosis: call for views on possible future measures to accelerate disease eradication in England. https://consult.defra.gov.uk/bovine-tb-2020/bovine-tuberculosis-call-for-views-on-possible-fut/supporting_documents/2021%20Bovine%20TB%20Call%20for%20views.pdf
- Defra. 2021b. Great Britain bovine tuberculosis (TB) quarterly overview. https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/994069/bovine-tb-gb-quarterlyoverview-16jun21.pdf
- de la Rúa-Domenech R. 2006. Human *Mycobacterium bovis* infection in the United Kingdom: Incidence, risks, control measures and review of the zoonotic aspects of bovine tuberculosis. *Tuberculosis (Edinb)*. 86 (2) 77-109.
- Diack AB, Head MW, McCutcheon S *et al.* 2014. Variant CJD. 18 years of research and surveillance. *Prion*. 8 (4) 286-295.
- Dweba CC, Zishiri OT, El Zowalaty ME. 2018. Methicillin-resistant *Staphylococcus aureus*: livestock-associated, antimicrobial, and heavy metal resistance. *Infection and Drug Resistance*. 11, 2497-2509.
- ECDC. 2017. Facts about *Escherichia coli*. <https://www.ecdc.europa.eu/en/escherichia-coli-ecoli/facts>
- ECDC. 2021. Questions and answers on avian influenza. <https://www.ecdc.europa.eu/en/zoonotic-influenza/facts/faq-avian-influenza>
- EMA. 2016. CVMP strategy on antimicrobials 2016-2020. https://www.ema.europa.eu/en/documents/scientific-guideline/cvmp-strategy-antimicrobials-2016-2020_en.pdf
- EMA and EFSA. 2017. EMA and EFSA Joint Scientific Opinion on measures to reduce the need to use antimicrobial agents in animal husbandry in the European Union, and the resulting impacts on food safety. *EFSA Journal*. 15 (1) 4666.
- Enright MC, Robinson DA, Randle G *et al.* 2002. The evolutionary history of methicillin-resistant *Staphylococcus aureus* (MRSA). *Proceedings of the National Academy of Sciences USA*. 99 (11) 7687-7692.
- Epperson S, Jhung M, Richards S *et al.* 2013. Human infections with influenza A(H3N2) variant virus in the United States, 2011-2012. *Clinical Infectious Diseases*. 57 Suppl 1, S4-S11.
- FAOSTAT. 2021. <http://www.fao.org/faostat/en/#data>
- Farnoosh G, Zarei S, Hosseini Zijoud SS *et al.* 2020. Is there a guarantee that the crisis of COVID-19 will not be repeated? *Disaster Medicine and Public Health Preparedness*. 1-2. [published online ahead of print]. <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7156560/>
- Fergestad ME, De Visscher A, L'Abée-Lund T *et al.* 2021. Antimicrobial resistance and virulence characteristics in 3 collections of staphylococci from bovine milk samples. *Journal of Dairy Science*. S0022-0302 (21) 00568-3.
- Foley SL, Nayak R, Hanning IB *et al.* 2011. Population dynamics of *Salmonella enterica* serotypes in commercial egg and poultry production. *Applied and Environmental Microbiology*. 77 (13) 4273-4279.
- Freuling CM, Breithaupt A, Müller T *et al.* 2020. Susceptibility of Raccoon Dogs for Experimental SARS-CoV-2 Infection. *Emerging Infectious Diseases*. 26 (12) 2982-2985.
- FSA. 2013. Feasibility of introducing methods, in the UK, for reducing shedding of *E. coli* O157 in cattle. <https://www.food.gov.uk/research/microbial-risk-assessment-b12/feasibility-of-introducing-methods-in-the-uk-for-reducing-shedding-of-e-coli-o157-in-cattle>
- FSA. 2015. Year 1 of a UK-wide survey of campylobacter contamination on fresh chickens at retail (February 2014 to February 2015). <https://www.food.gov.uk/other/year-1-of-a-uk-wide-survey-of-campylobacter-contamination-on-fresh-chickens-at-retail-february-2014-to-february-2015>
- FSA. 2016. Campylobacter contamination in fresh whole chilled UK-produced chickens at retail: January – March 2016. https://www.food.gov.uk/sites/default/files/media/document/campy-survey-report-jan-mar-2016_0.pdf
- FSA. 2018. Surveillance study of antimicrobial resistance in bacteria isolated from chicken and pork sampled on retail sale in the United Kingdom. https://www.food.gov.uk/sites/default/files/media/document/amrinchickenandporkfinrepjuly18_fs101196.pdf
- FSA. 2018a. *Campylobacter*. <https://www.food.gov.uk/safety-hygiene/campylobacter>
- FSA. 2018b. Antimicrobial Resistance in *Campylobacter jejuni* and *Campylobacter coli* from Retail Chilled Chicken in the UK. <https://www.food.gov.uk/research/research-projects/antimicrobial-resistance-in-campylobacter-jejuni-and-campylobacter-coli-from-retail-chilled-chicken-in-the-uk>
- FSA. 2019. A microbiological survey of campylobacter contamination in fresh whole UK-produced chilled chickens at retail sale (Y2/3/4). <https://www.food.gov.uk/research/foodborne-diseases/a-microbiological-survey-of-campylobacter-contamination-in-fresh-whole-uk-produced-chilled-chickens-at-retail-sale-y234>

66. Wozniak M, Kulkarni A, Gnanapavan S, et al. 2016. A chicken and a pig: the impact of the H5N1 virus on the food chain.

<https://www.nature.com/articles/nrn2016012>

Gov.uk. 2021. Avian influenza (bird flu). Available from: <https://www.gov.uk/guidance/avian-influenza-bird-flu>

Graham JP, Leibler JH, Price LB *et al.* 2008. The animal:human interface and infectious disease in industrial food animal production: rethinking biosecurity and biocontainment. *Public Health Reports.* 123, 282-299.

Stapley AB, Fitzgerald LR, Peano A *et al.* 2019. *Biological Spectrum.* 7 (3).

He P, Wang G, Mo Y *et al.* 2018. Novel triple-reassortant influenza viruses in pigs, Guangxi, China. *Emerging Microbes and Infections.* 7 (1) 85.

Herfst S, Schrauwen EJ, Linster M *et al.* 2012. Airborne transmission of influenza A/H5N1 virus between ferrets. *Science.* 336 (6088) 1534-1541.

HM Government. 2018. Zoonosis report UK 2017.

https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/918089/UK_Zoonoses_report_2017.pdf

HM Government. 2019. Tackling antimicrobial resistance 2019-2024 – the UK's five-year national action plan.

https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/773130/uk-amr-5-year-national-action-plan.pdf

HSE. 2005. BSE – occupational guidance. <https://www.hse.gov.uk/pubns/web22.pdf>

HSE. 2021. Verotoxigenic *E. coli* (VTEC) – includes *E. coli* O157. <https://www.hse.gov.uk/agriculture/zoonoses-data-sheets/verotoxigenic-ecoli.pdf>

Igrejas G, Correia S, Silva V *et al.* 2018. Planning a One Health Case Study to Evaluate Methicillin Resistant Staphylococcus aureus and Its Economic Burden in Portugal. *Frontiers in Microbiology.* 9, 2964.

Imai M, Watanabe T, Hatta M *et al.* 2012. Experimental adaptation of an influenza H5 HA confers respiratory droplet transmission to a reassortant H5 HA/H1N1 virus in ferrets. *Nature.* 486 (7403)420-428.

Jajere SM. 2019. A review of Salmonella enterica with particular focus on the pathogenicity and virulence factors, host specificity and antimicrobial resistance including multidrug resistance. *Veterinary World.* 12 (4) 504-521.

Jarvis L. 2021. Badgers, cattle... and scapegoats? Why UK wildlife could remain in the firing line – despite new cull review.

<https://www.nationalgeographic.co.uk/environment-and-conservation/2021/01/badgers-cattle-and-scapegoats-is-controversial-science-putting>

Johura FT, Tasnim J, Barman I *et al.* 2020. Colistin-resistant *Escherichia coli* carrying mcr-1 in food, water, hand rinse, and healthy human gut in Bangladesh. *Gut Pathology.* 12, 5.

Jones BA, Grace D, Kock R *et al.* 2013. Zoonosis emergence linked to agricultural intensification and environmental change. *Proceedings of the National*

Academy of Sciences USA. 110 (21) 8399-8404.

Kilpatrick AM, Chmura AA, Gibbons DW *et al.* 2006. Predicting the global spread of H5N1 avian influenza. *Proceedings of the National Academy of Sciences USA.* 103 (51) 19368-19373.

Kim JK, Negovetich NJ, Forrest HL *et al.* 2009. Ducks: the "Trojan horses" of H5N1 influenza. *Influenza and other Respiratory Viruses.* 3 (4) 121-128.

Kreuder Johnson C, Hitchens P, Smiley Evans T *et al.* 2015. Spillover and pandemic properties of zoonotic viruses with high host plasticity. *Scientific Reports.* 5, 14830.

Krzywanek K, Metz-Gercek S, Mittermayer H. 2009. Methicillin-Resistant Staphylococcus aureus ST398 from human patients, upper Austria. *Emerging Infectious Diseases.* 15 (5) 766-769.

Kuiken T. 2013. Is low pathogenic avian influenza virus virulent for wild waterbirds?. *Proceeding. Biological Sciences.* 280 (1763) 20130990.

Larsen CS and Paludan SR. 2020. Corona's new coat: SARS-CoV-2 in Danish minks and implications for travel medicine. *Travel Medicine and Infectious Disease.* 38, 101922.

Le Sage V, Jones JE, Kormuth KA *et al.* 2021. Pre-existing heterosubtypic immunity provides a barrier to airborne transmission of influenza viruses. *PLoS Pathogens.* 18, 17(2) e1009273.

Lee CW, Senne DA, Suarez DL. 2004. Effect of vaccine use in the evolution of Mexican lineage H5N2 avian influenza virus. *Journal of Virology.* 78, 8372-8981.

Leibler JH, Otte J, Roland-Holst D *et al.* 2009. Industrial food animal production and global health risks: exploring the ecosystems and economics of avian influenza. *Ecohealth.* 6 (1) 58-70.

Lewis D. 2021. Can COVID spread from frozen wildlife? Scientists probe pandemic origins. *Nature.* DOI: [10.1038/d41586-021-00495-0](https://doi.org/10.1038/d41586-021-00495-0).

Liu YY, Wang Y, Walsh TR *et al.* 2016. Emergence of plasmid-mediated colistin resistance mechanism MCR-1 in animals and human beings in China: a microbiological and molecular biological study. *Lancet Infectious Diseases.* 16 (2) 161-168.

Liu Y and Liu JH. 2018. Monitoring Colistin Resistance in Food Animals, An Urgent Threat. *Expert Review of Anti-infective Therapy.* 16(6) 443-446.

Lycett SJ, Duchatel F, Digard P. 2008. A brief history of bird flu. *Philosophical Transactions of the Royal Society of London. Series B. Biological Sciences.* 24, 374 (1775) 20180257.

Ma W, Kahn RE, Richt JA. 2008. The pig as a mixing vessel for influenza viruses: Human and veterinary implications. *Journal of Molecular Genetic Medicine.* 3 (1) 158-166.

Martin MJ, Thottathil SE, Newman TB. 2015. Antibiotics Overuse in Animal Agriculture: A Call to Action for Health Care Providers. *American Journal of Public Health.* 105 (12) 2409-2410.

Mehndiratta PL and Bhalla P. 2014. Use of antibiotics in animal agriculture and emergence of methicillin-resistant Staphylococcus aureus (MRSA) clones: need to assess the impact on public health. *Indian Journal of Medical Research.* 140 (3) 339-344.

Mok T, Jaunmuktane Z, Joiner S *et al.* 2017. Variant Creutzfeldt-Jakob Disease in a Patient with Heterozygosity at PRNP Codon 129. *New England Journal of Medicine.* 376 (3) 292-294.

FACTORY FARMS – A BREEDING GROUND FOR DISEASE

- Mourkas E, Taylor AJ, M'ric G *et al.* 2020. Agricultural intensification and the evolution of host specialism in the enteric pathogen *Campylobacter jejuni*. *Proceedings of the National Academy of Sciences USA*. 117 (20) 12811-12816.
- National CJD research & surveillance unit (NCJDRSU). 2021. <https://www.cjd.ed.ac.uk/surveillance>
- Nelson MI and Worobey M. 2018. Origins of the 1918 pandemic: revisiting the swine 'mixing vessel' hypothesis. *American Journal of Epidemiology*. 187 (12) 2498-2502.
- Nemati M, Hermans K, Lipinska U *et al.* 2008. Antimicrobial resistance of old and recent *Staphylococcus aureus* isolates from poultry: first detection of livestock-associated methicillin-resistant strain ST398. *Antimicrobial Agents and Chemotherapy*. 52 (10) 3817-3819.
- NHS. 2018. Creutzfeldt-Jakob disease. <https://www.nhs.uk/conditions/creutzfeldt-jakob-disease-cjd/>
- NHS. 2020. MRSA. <https://www.nhs.uk/conditions/mrsa/>
- NHS Inform. 2020. *Escherichia coli* (*E. coli*) O157. <https://www.nhsinform.scot/illnesses-and-conditions/infections-and-poisoning/escherichia-coli-e-coli-o157>
- O'Brien SJ. 2013. The 'decline and fall' of nontyphoidal salmonella in the United Kingdom. *Clinical Infectious Diseases*. 56 (5) 705-710.
- O'Neill J. 2016. Tackling drug-resistant infections globally: final report and recommendations. London, UK: review on antimicrobial resistance. 1-84.
- Pennington TH. 2014. *E. coli* O157 outbreaks in the United Kingdom: past, present, and future. *Infection and Drug Resistance*. 7, 211-222.
- Pirola M, Visaggio D, Giofrè A *et al.* 2019. Unidirectional animal-to-human transmission of methicillin-resistant *Staphylococcus aureus* ST398 in pig farming; evidence from a surveillance study in southern Italy. *Antimicrobial Resistance and Infection Control*. 8, 187.
- Pope LC, Butlin RK, Wilson GJ *et al.* 2007. Genetic evidence that culling increases badger movement: implications for the spread of bovine tuberculosis. *Molecular Ecology*. 16 (23) 4919-2499.
- Public Health England. 2015. Health matters: antimicrobial resistance. <https://www.gov.uk/government/publications/health-matters-antimicrobial-resistance/health-matters-antimicrobial-resistance>
- Public Health England. 2017. *Campylobacter* data 2008 to 2017. <https://www.gov.uk/government/publications/campylobacter-infection-annual-data/campylobacter-data-2008-to-2017>
- Public Health England. 2019. Laboratory surveillance of *Escherichia coli* bacteraemia in England, Wales and Northern Ireland: 2018. [Laboratory surveillance of *Escherichia coli* bacteraemia in England, Wales and Northern Ireland: 2018 \(publishing.service.gov.uk\)](https://publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/821112/laboratory-surveillance-of-escherichia-coli-bacteraemia-in-england-wales-and-northern-ireland-2018.pdf)
- Public Health England. 2020. Shiga toxin-producing *Escherichia coli* (STEC) data: 2018. <https://www.gov.uk/government/publications/escherichia-coli-e-coli-o157-annual-totals/shiga-toxin-producing-escherichia-coli-stec-data-2018>
- Public Health England. 2021. STEC (Shiga toxin-producing *Escherichia coli*) serogroup O157 incidence rate/100,000. <https://fingertips.phe.org.uk/profile/health-protection/data#page/3/gid/1938133209/pat/6/par/E12000001/ati/102/are/E06000047/iid/93316/age/1/sex/4/cid/4/tbm/1/page-options/car-do-0>
- Pulliam JR, Epstein JH, Dushoff J *et al.* 2012. Agricultural intensification, priming for persistence and the emergence of Nipah virus: a lethal bat-borne zoonosis. *Journal of the Royal Society. Interface*. 9 (66) 89-101.
- Rabi FA, Al Zoubi MS, Kasasbeh GA *et al.* 2020. SARS-CoV-2 and Coronavirus Disease 2019: What We Know So Far. *Pathogens*. 9 (3)231.
- Riley LW, Remis RS, Helgerson SD *et al.*, 1983. Hemorrhagic colitis associated with a rare *Escherichia coli* serotype. *New England Journal of Medicine*. 308 (12) 681-685.
- Rohr JR, Barrett CB, Civitello DJ *et al.*, 2019. Emerging human infectious diseases and the links to global food production. *Nature Sustainability*. 2 (6) 445-456.
- Russell CA, Fonville JM, Brown AE *et al.* 2012. The potential for respiratory droplet-transmissible A/H5N1 influenza virus to evolve in a mammalian host. *Science*. 336 (6088) 1541-1547.
- RSPB. 2021. Avian influenza updates. <https://www.rspb.org.uk/birds-and-wildlife/advice/how-you-can-help-birds/disease-and-garden-wildlife/avian-influenza-updates/>
- Schlackow I, Stoesser N, Walker AS *et al.* 2012. Increasing incidence of *Escherichia coli* bacteraemia is driven by an increase in antibiotic-resistant isolates: electronic database study in Oxfordshire 1999-2011. *Journal of Antimicrobial Chemotherapy*. 67 (6) 1514-1524.
- Scientific Task Force on Avian Influenza and Wild Birds. 2016. Statement on H5H8 Highly Pathogenic Avian Influenza (HPAI) in poultry and wild birds. https://www.cms.int/sites/default/files/Scientific%20Task%20Force%20on%20Avian%20Influenza%20and%20Wild%20Birds%20H5N8%20HPAI_December%202016_FINAL.pdf
- Shah DH, Paul NC, Sicho WC *et al.* 2017. Population dynamics and antimicrobial resistance of the most prevalent poultry-associated *Salmonella* serotypes. *Poultry Science*. 96 (3) 687-702.
- Shinde V, Bridges CB, Uyeki TM *et al.* 2009. Triple-reassortant swine influenza A (H1) in humans in the United States, 2005-2009. *New England Journal of Medicine*. 360 (25) 2616-25.
- Shortridge KF, Zhou NN, Guan Y *et al.* 1998. Characterization of avian H5N1 influenza viruses from poultry in Hong Kong. *Virology*. 252 (2) 331-342.
- Smith G, Vijaykrishna D, Bahl J *et al.* 2009. Origins and evolutionary genomics of the 2009 swine-origin H1N1 influenza A epidemic. *Nature*. 459, 1122-1125.
- Smith TC. 2015. Livestock-associated *Staphylococcus aureus*: the United States experience. *PLoS Pathogens*. 11 (2) e1004564.
- Snow LC, Davies RH, Christiansen KH *et al.* 2010. Investigation of risk factors for *Salmonella* on commercial egg-laying farms in Great Britain, 2004-2005. *The Veterinary Record*. 166 (19) 579-586.

- Spioneyitu. 2020. Is factory farming to blame for coronavirus? www.theguardian.com/world/2020/mar/28/is-factory-farming-to-blame-for-coronavirus
- Strachan NJ, Dunn GM, Locking ME *et al.* 2006. Pathogenicity of *Escherichia coli* O157 in human beings. *Journal of Clinical Microbiology*. 112, 129-137.
- Taubenberger JK. 2006. The origin and virulence of the 1918 "Spanish" influenza virus. *Proceedings of the American Philosophical Society*. 150 (1) 86-112.
- The Wildlife Trusts. 2021. Government approves badger culling to 2026 and ignores public consultation. <https://www.wildlifetrusts.org/news/government-approves-badger-culling-2026-and-ignores-public-consultation>
- Treacy J, Jenkins C, Paranthaman K *et al.* 2019. Outbreak of Shiga toxin-producing *Escherichia coli* O157:H7 linked to raw drinking milk resolved by rapid application of advanced pathogen characterisation methods, England, August to October 2017. *Euro Surveillance*. 24 (16) 1800191.
- UK Sepsis Trust. 2020. About sepsis. <https://sepsistrust.org/about/about-sepsis/>
- UK Sepsis Trust. 2020a. Sepsis and antimicrobial resistance. <https://sepsistrust.org/sepsis-and-antimicrobial-resistance/>
- UK-VARSS. 2019. UK Veterinary Antibiotic Resistance and Sales Surveillance Report (UK-VARSS 2018). New Haw, Addlestone: Veterinary Medicines Directorate.
- UNEP and International Livestock Research Institute. 2020. Preventing the Next Pandemic: Zoonotic diseases and how to break the chain of transmission. Nairobi, Kenya.
- University of Sheffield. 2020. Intensive farming increases risk of epidemics, warn scientists. <https://www.sheffield.ac.uk/biosciences/news/intensive-farming-increases-risk-epidemics-warn-scientists>
- UN News. 2021. WHO and partners urge countries to halt sales of wild mammals at food markets. <https://news.un.org/en/story/2021/04/1089622>
- Van Boeckel TP, Brower C, Gilbert M *et al.*, 2015. Global trends in antimicrobial use in food animals. *Proceedings of the National Academy of Sciences USA*. 112 (18) 5649-5654.
- Verhagen JH, Fouchier RAM, Lewis N. 2021. Highly Pathogenic Avian Influenza Viruses at the Wild-Domestic Bird Interface in Europe: Future Directions for Research and Surveillance. *Viruses*. 13 (2) 212.
- Veterinary Medicines Directorate. 2019. UK One Health Report – joint report on antibiotic use and antibiotic resistance, 2013-2017. New Haw, Addlestone: Veterinary Medicines Directorate.
- Vihta KD, Stoesser N, Llewelyn MJ *et al.* 2018. Trends over time in *Escherichia coli* bloodstream infections, urinary tract infections, and antibiotic susceptibilities in Oxfordshire, UK, 1998-2016: a study of electronic health records. *Lancet Infectious Diseases*. 18 (10) 1138-1149.
- Voss A, Loeffen F, Bakker J *et al.* 2005. Methicillin-resistant *Staphylococcus aureus* in pig farming. *Emerging Infectious Diseases*. 11 (12) 1965-1966.
- Wasley A and Heal A. 2019. Bureau of Investigative Journalism. Exclusive: At least 100 cases of Salmonella poisoning from British eggs. <https://www.thebureauinvestigates.com/stories/2019-09-20/exclusive-at-least-100-cases-of-salmonella-poisoning-from-british-eggs>
- Webster RG, Peiris M, Chen H *et al.* 2006. H5N1 outbreaks and enzootic influenza. *Emerging Infectious Diseases*. 12 (1) 3-8.
- WHO. 2007. Review of latest available evidence on potential transmission of avian influenza (H5N1) through water and sewage and ways to reduce the risks to human health. https://www.who.int/water_sanitation_health/emerging/h5n1background.pdf
- WHO. 2011. Critically important antimicrobials for human medicine. https://apps.who.int/iris/bitstream/handle/10665/77376/9789241504485_eng.pdf
- WHO. 2017. Stop using antibiotics in healthy animals to prevent the spread of antibiotic resistance. <https://www.who.int/news/item/07-11-2017-stop-using-antibiotics-in-healthy-animals-to-prevent-the-spread-of-antibiotic-resistance>
- WHO. 2017a. One health. <https://www.who.int/news-room/q-a-detail/one-health>
- WHO. 2018. The challenges of preventing bovine tuberculosis. <https://www.who.int/bulletin/volumes/96/2/18-020218/en/>
- WHO. 2018a. Nipah virus. <https://www.who.int/news-room/fact-sheets/detail/nipah-virus>
- WHO. 2018b. *E. coli* – key facts. <https://www.who.int/news-room/fact-sheets/detail/e-coli>
- WHO. 2020. SARS-CoV-2 mink-associated variant strain – Denmark. <https://www.who.int/csr/don/06-november-2020-mink-associated-sars-cov2-denmark/en/>
- WHO. 2020a. Antibiotic resistance. <https://www.who.int/news-room/fact-sheets/detail/antibiotic-resistance>
- WHO. 2020b. Antimicrobial resistance. <https://www.who.int/news-room/fact-sheets/detail/antimicrobial-resistance>
- WHO. 2020c. Campylobacter. <https://www.who.int/news-room/fact-sheets/detail/campylobacter>
- WHO. 2021. Human infection with avian influenza A(H5) viruses. <https://www.who.int/docs/default-source/wpro---documents/emergency/surveillance/avian-influenza/ai-20210528.pdf>
- WHO. 2021a. Reducing public health risks associated with the sale of live wild animals of mammalian species in traditional food markets. <https://cdn.who.int/media/docs/default-source/food-safety/ig---121-1-food-safety-and-covid-19-guidance-for-traditional-food-markets-2021-04-12-en.pdf>
- Williams AP, Avery LM, Killham K, *et al.*, 2005. Persistence of *Escherichia coli* O157 on farm surfaces under different environmental conditions. *Journal of Applied Microbiology*. 98 (5)1075-1083.
- Willyard C. 2019. Flu of the Farm. *Nature*. 573 (7774) S62-S63.
- Yara DA, Greig DR, Gally DL *et al.* 2020. Comparison of Shiga toxin-encoding bacteriophages in highly pathogenic strains of Shiga toxin-producing *Escherichia coli* O157:H7 in the UK. *Microbial Genomics*. 6 (3) e000334.



Deadly biological weapons are being developed all over the world right now – and they're more destructive than any existing ones. But not a single government is prepared to try and stop them. There are thousands of laboratories involved and we can guarantee there's one not far from your home. These laboratories are called factory farms and they produce over 80 per cent of all the meat we eat.

Admittedly, these factory farms don't set out to try and kill us but a pursuit of 'return on investment', 'profitability' and 'intensification' has resulted in a major threat to humankind.

It's a result of cramming animals together in filthy, overcrowded sheds. They are bred for fast growth, are acutely stressed and therefore have low immunity. You couldn't have a better environment for bacteria and viruses to thrive and mutate into more virulent strains, while dosing the animals with antibiotics has already produced deadly superbugs. No wonder three in four new infectious diseases come from animals.

Scientists can't say where, and from which animal, a pandemic will emerge but they're sure it could happen.

This report explains why it is vital to end this abuse of animals and move towards a plant-based food system. It's a case of ending factory farming before it ends us!

Viva!

*Factory farms –
a breeding ground
for disease*

© Viva! 2022

£5

ISBN 978-1-9160003-6-0



9 781916 000360 >