Compassion in World Farming

Biosecurity and factory farming

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Summary of evidence and arguments

One of the main arguments made in support of the factory farming of animals is that it provides greater biosecurity and hence minimises infectious disease outbreaks on farm and the risk of zoonotic diseases affecting the human population.

Our survey of scientific and industry evidence shows on the contrary that:

- Large, intensive farms are more likely to be infected by pathogens (i.e. they have a higher risk of infectious disease) than small, free-range and backyard farms
- The most dangerous of farm animal zoonoses, the highly pathogenic avian influenza (HPAI) viruses, have evolved within large-scale, concentrated poultry production sites, not in backyard farms or in wild bird populations, and are most often spread farm-to-farm by human activities
- The risk of infectious disease in poultry and pig production has been heightened by the spread of factory farming worldwide

- The crowded and stressful conditions that animals experience in factory farms makes them less able to resist disease individually and more likely to spread pathogens among themselves and to the outdoor environment.

The known facts and trends in animal disease show that factory farming tends to increase rather than decrease the risk of infectious animal disease. Over the last 50 years factory farming has become the dominant system of commercial animal production in developed countries and has spread globally in pig and poultry production. Over the same time the number of outbreaks of infectious animal disease has greatly increased rather than reduced. FAO has noted the increasing 'frequency and impact of emerging and re-emerging diseases' and that 'intensification of poultry production renders the industry more susceptible to threats of poultry diseases.'

Between 1959 and 1978 (a 20-year timespan) there were 4 primary outbreaks of highly pathogenic avian influenza (HPAI) in poultry globally, all in developed countries. Between 1979 and 1998 (20 years during which factory farming became the norm) there were 3 times as many global outbreaks of HPAI, all except 2 in developed countries.

In order to see why factory farming is fostering disease rather than preventing it, we need to examine the term 'biosecurity' itself. The factory farming model of biosecurity is equivalent to 'bioexclusion' of pathogens and 'biocontainment' of disease by keeping animals indoors and preventing contact between the animals and the outside environment. Factory farms have a very poor record of achieving either bioexclusion or biocontainment.

In fact, biosecurity has a much broader meaning encompassing animal, human and environmental health (often expressed as 'One Health'). FAO has stated that 'Biosecurity is defined as a holistic concept of direct relevance to the sustainability of agriculture, food safety, and the protection of the environment, including biodiversity'.[1] The conditions experienced by animals in factory farms and the externalities such as zoonoses, environmental pollution, antibiotic overuse, and climate impacts clearly run counter to biosecurity properly understood.

It is a major problem for animal and human health is that, typically, official guidance for animal health does not so far question the model of factory farming – ie the concentration of tens or hundreds of thousands of animals confined together in the smallest feasible space. In some US states the average poultry operation holds over 500,000 confined birds, a self-evident risk to animal health and environmental pollution from wastes.

Because factory farming is taken by the global animal health authorities as the standard model for modern animal husbandry, the authorities have to fall back on ever more stringent prescriptions for avoiding cross-contamination or contact with the environment. Because of this bias, animal health authorities misguidedly tend to see small-scale free-range farming as non-standard and not consistent with the enclosed model of the factory farm that they are more familiar with.

OIE records show that around 40 percent of the HPAI H5N1 outbreaks in domestic poultry between late 2005 and early 2007 occurred in poultry units of 10,000 birds or more But on average less than 10 percent of flocks consisted of more than 10,000 birds at that date, showing a much higher relative risk of infection for industrial farms.

In a major HPAI outbreak in Minnesota in 2014/15, a total of 232 poultry farms were affected but only 21 of these were backyard farms. In the Netherlands outbreak of HPAI in 2003, 17% of industrial farms but only 0.1% of backyard flocks were infected.

A 2017 EFSA report shows that only 7% of outdoor laying hen units and 3% of outdoor meat turkey units got infected with AI virus, compared to 20% and 28% of the indoor laying hen and meat turkey units were infected, respectively.

One of the potentially disastrous results of factory farming has been the evolution of Highly Pathogenic Avian Influenza (HPAI) viruses within industrial poultry production. HPAI viruses did not exist in wild birds before they evolved in poultry and they do not persist in wild bird populations. The virus was transferred from domestic poultry to wild birds around 2005, having emerged in the poultry industry around 1997. A number of HPAI viruses (H5N1, H5N8, H7N8 and others) are now circulating and mutating in the global poultry industry and are now a potential cause of human pandemics.

A 2018 review of the known cases of evolution of low pathogenic avian flu (LPAI) viruses to HPAI within poultry makes clear that the evolution of HPAI viruses almost always takes place within large, intensive farms, rather than in small farms. Between 1959 and 2015, 37 of 39 known conversion events took place in intensive commercial units, mostly in developed countries, and only 2 took place in backyard rural flocks.

Common foodborne pathogens are prevalent on factory farms. Again, the facts contradict the industry argument that confined animals are less likely to be infected. Campylobacter is found in 37.5% of chickenmeat samples in the EU (2018), almost all originating in factory farms (over 90% of EU chicken production being industrialised). Studies of salmonella prevalence show that large farms are a risk factor for infection and that hens confined in cages are between twice and several times more likely to be infected.

The record of the global pig industry over recent years also demonstrates the failure of the industrial model of biosecurity to control infectious disease. The H1N1 virus, which caused the first global pandemic of the 21st century, emerged in industrial pig production in Mexico in 2009. African Swine Fever (ASF) has spread globally since 2007 and since 2014 has reached Eastern Europe and Asia. China killed 12 million pigs to control ASF between August 2018 and early March 2019. The viral diseases termed Postweaning Multisystemic Wasting Disease (PMWS) and Porcine Reproductive and Respiratory Syndrome (PRRS or ‘blue ear’) both emerged in the 1980s and 1990s and swept the international pig industry in Europe, North America and Asia.

Numerous scientists have pointed out the association between intensive animal production and disease risk. European virologists have pointed out that ‘The unprecedented crowded conditions are undoubtedly major drivers of the emergence and spread of pathogens in domestic animal populations, allowing their increasingly more frequent cross-species transmission to humans.’ US scientists have noted that ‘A major impact of modern intensive production systems is that they allow the rapid selection and amplification of pathogens, thus there is increasing risk for disease entrance and/or dissemination.’

Far from being biosecure environments, the conditions of factory farms are the perfect breeding ground for infectious diseases. Animals are crowded together in sheds or feedlots in tens or even hundreds of thousands, and are under stress from a number of factors that are likely to weaken their immune systems. Insects, rodents and wild birds that are potential disease vectors are attracted to the extraordinary quantities of manure and feed on site.

Why does the ‘industrial biosecurity’ strategy not work? The reason comes back to the fact that large intensive farms are more likely than small and traditional farms to experience outbreaks of devastating outbreaks of infection. This is entirely consistent with the known facts of epidemiology that a large, concentrated, indoor population of susceptible hosts (a factory farm) is much more likely to propagate infection than a small and less concentrated outdoor population in a well-managed backyard or traditional farm.

In spite of a wealth of evidence, the industry and regulators often continue to attribute the continuing disease problems to insufficient biosecurity rather than to the inherent flaws in the industrial animal production model itself.

1. What is 'biosecurity' - and what should it be?

One of the main arguments made in support of factory farming is that it allows for greater biosecurity and hence minimises infectious disease outbreaks on farm and the risk of zoonotic diseases to the human population.

In most cases this does not accord with the known facts about animal disease. Over the last 50 years factory farming has spread globally and is the dominant system of animal production in industrial countries. The FAO has said of the same period of time, in its Emergency Prevention System (EMPRES) bulletin, 'the frequency and impact of emerging and re-emerging animal diseases have increased over the past decades'[2], Similarly, in relation to poultry, the most intensively-farmed species, FAO says that 'intensification of poultry production renders the industry more susceptible to threats of poultry diseases, including Newcastle disease or Gumboro, and in some cases diseases that can affect human health, such as H5N1 highly pathogenic avian influenza and Salmonella.'[3]

In the case of highly pathogenic avian influenza (HPAI), scientists now know that the viruses causing this disease evolved in the huge concentrations of animals in factory farms.

Between 1959 and 1978 (a 20-year timespan) there were 4 primary outbreaks of HPAI in poultry globally, all in developed countries. Between 1979 and 1998 (20 years during which factory farming became the norm) there were 3 times as many global outbreaks of HPAI, all except 2 in developed countries. [4, 5]

In order to see why factory farming is fostering disease rather than preventing it, we need to examine the term 'biosecurity' itself. The factory farming industry often implies that biosecurity is equivalent to keeping animals indoors and preventing contact between the animals and the outside environment. This view of biosecurity is focussed on 'bioexclusion' (of pathogens) and 'biocontainment' (of pathogens passing between farm units)[6]. As this briefing will show, factory farming very often fails on even this narrow definition of biosecurity.

In fact, biosecurity has a much broader meaning encompassing animal, human and environmental health, and the conditions of factory farming typically run counter to biosecurity properly understood.

The FAO has been a leader in developing the approach to biosecurity, which is now applied to plant, animal and human health, food security, biodiversity conservation and environment, trade and economic development, and security considerations such as terrorism.[7]

A 2005 FAO discussion paper, Biosecurity in Food and Agriculture, states that:

"Biosecurity is defined as a holistic concept of direct relevance to the sustainability of agriculture, food safety, and the protection of the environment, including biodiversity".[8]

A 2014 FAO definition is that, 'Biosecurity is a strategic and integrated approach that encompasses the policy and regulatory frameworks [including instruments and activities] that analyse and manage risks in the sectors of: food safety; animal life and health; plant life and health, including associated environmental risks.' [9]

In relation to animal health, FAO defines biosecurity to mean activities taken to prevent or manage diseases, but adds that this should involve:

'a strategic and integrated approach to biosecurity as a holistic concept that is of direct relevance in meeting consumer expectations in relation to the safety of their food supply, preventing and controlling zoonotic aspects of public health, ensuring the sustainability of agriculture, safeguarding terrestrial, freshwater and marine environments, and protecting biodiversity.[10]

Even more clearly, FAO stated in 2009:

'Biosecurity covers three main sectors: food safety; plant life and health; animal life and health. The biosafety within biosecurity approach, encompassing all policy and regulatory frameworks to manage biological risks associated with food and agriculture (including relevant environmental risks), is necessary to protect: 1) agricultural production systems, agricultural producers and their associated interests; 2) human health and consumer confidence in agricultural products; and 3) the environment.'[11]

The integration of public health, environmental sustainability and consumer expectations in FAO's statement is crucial to our understanding of biosecurity. It runs counter to a narrow factory-farm view of biosecurity as bioexclusion (even if that could be successful). At a first glance we could identify several aspects of 'biosecurity' as advocated for concentrated animal farming operations that are damaging to human and environmental health and even to global food security.

One of the most obvious of these is over-use of antibiotics that is made necessary by the very conditions of factory farms, causing antibiotic resistance in human medicine.

The integrated view of biosecurity, in response to concerns over emerging disease, zoonosis and challenges to environmental health caused by pesticides and antibiotics, has developed into the concept of 'One Health'. The 'One Health' view that animal and human health must be seen as related is now mainstream in the FAO, WHO, the US CDC and others. The UK's 2019 Veterinary Medicines Directorate report on the monitoring of antibiotic use and antibiotic resistance in people and animals is titled the 'One Health report'[12]. It is also interesting that One Health was initially proposed by a wildlife conservation organisation.[13]

Some useful definitions of 'One Health' are:[14]

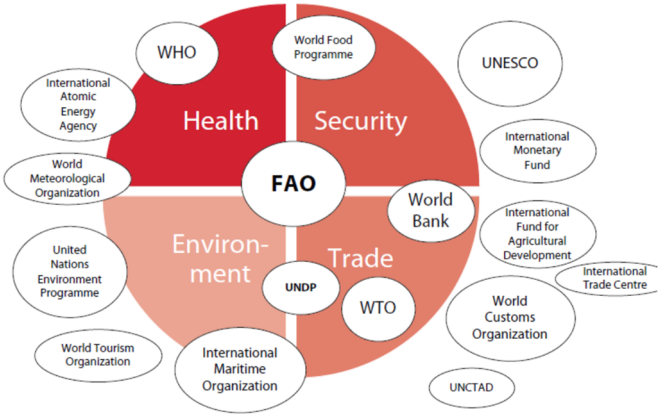
(i) 'One Health is the integrative effort of multiple disciplines working locally, nationally, and globally to attain optimal health for people, animals, and the environment.' (AVMA, 2008)

(ii) One Health is 'a broad and holistic paradigm that addresses and frames the complex interactions between human health, livestock, pet and wildlife health, environmental health, ecosystems services, climate, water, sanitation, food systems, energy, human development, equity and justice. (Global Risk Forum, Davos, 2015)

(iii) The Royal Veterinary College states that 'The One Health approach recognises the relationship between health and disease at the human, animal and environment interfaces and has become an important focus in both medical and veterinary science. It promotes a "whole of society" treatment of health hazards and a systemic change of perspective in the

management of risk.' The RVC now runs a Master's degree in 'One Health: ecosystems, humans and animals'.[15]

Figure 1. Illustration of global organisations and fields related to biosecurity, centred on the FAO. Source IPPC 2016.[16]



Source and copyright: International Plant Protection Convention (IPPC), 2016[17] https://www.ippc.int/static/media/files/irss/2016/09/09/Review_of_biosecurity_approaches_FINAL_report.pdf

2. A regulatory bias towards factory farming

How do concepts of integrated biosecurity translate into the actual advice given to the animal production industry?

A major problem in the detailed official guidance for animal health is that the animal health authorities do not question the model of factory farming – ie the concentration of large numbers of animals confined together in the smallest feasible space, either in sheds or pens. This unfortunately applies to the advice of the OIE, the World Organisation for Animal Health, as factory farming has spread globally on the Western industrial model, as well as to Defra and other national authorities. Because factory farming is taken by the global animal health authorities as the standard model for modern animal husbandry, the authorities have to fall back on ever more stringent prescriptions for avoiding cross-contamination or contact with the environment. Because of this bias, animal health authorities tend to see small-scale free-range farming as non-standard and not consistent with the enclosed model of the factory farm that they are more familiar with.

An important recent exception to the official bias is the policy statement of the American Public Health Association(APHA) in November 2019 that 'calls for a moratorium on the establishment of new CAFOs and expansion of existing CAFOs until regulation and enforcement conditions are in place to adequately protect the public's health.' [18] A CAFO is a Concentrated Animal Feeding Operation (housed or feedlot). A large CAFO holds at least 1,000 beef cattle, 700 dairy cows, 2,500 pigs, 125,000 broilers or 82,000 laying hens, although the average size of such facilities is much larger. The average cattle feedlot holds over 4,000 animals and in in some US states the average poultry operation holds over 500,000 birds.[19]

APHA points out that the average numbers in CAFOs are much larger. The average cattle feedlot holds 4,300 animals, and in some states the average poultry operation exceeds

500,000 birds. Even in ordinary (non-CAFO) units in the US, the average number of pigs per farm is now over 1,000, compared to less than 40 pigs on average per farm 50 years ago.[20] Defra's Guidance on disease prevention focusses on hygiene and the avoidance of cross-contamination by pathogens. For illustration, Defra's advice on most likely sources of disease spread includes[21]:

- animals moving between and within farms and, in particular, the introduction of new animals
- direct or close contact with neighbours' animals
- sharing vehicles, machinery, equipment, feed and bedding between farms
- movement of people, especially workers, between and within farms
- farm visitors – people, pets, equipment and vehicles
- contamination by wildlife, vermin and wild birds
- animals drinking from contaminated rivers and streams.

Given the factory farm model, these recommendations are important, since we know that cross-contamination by farmers/farm workers has been a major cause of disease spread in many cases. 'Secondary spread' was responsible for multiple outbreaks of highly pathogenic avian influenza in Hungary and southwest France in 2016 (H5N8)[22] and in Minnesota in 2015[23] (H5N2) and Indiana[24] in 2016 (a novel H7N8). Farm-to-farm secondary spread by human activities is probably more common than reported. Infections may take a long time to be noticed, or may be ignored; in January 2018 Hong Kong food surveillance inspectors found the H5N6 HPAI virus in chilled imported poultry meat in a food shop[25], a scenario that suggests that the virus had many opportunities for transmission before it arrived in the food shop.

But the essential point is that factory farms create conditions where pathogens are more likely to thrive and therefore any contact between factory farmed animals and others is more likely to pose a risk of disease transmission.

3. How factory farming is associated with infectious animal diseases

The evidence is that factory farming tends to increase rather than decrease the risk of infectious disease. Firstly, confinement of large numbers of animals indoors has not up to now succeeded in preventing outbreaks of infectious disease. Secondly, the conditions of factory farming actually make animals more susceptible to disease and make infections more likely to spread.

This survey by CIWF survey of the scientific literature shows that:

(i) Intensive farms are more likely to be infected by pathogens than small, free-range and backyard farms

(ii) Highly pathogenic avian influenza (HPAI) viruses have evolved within large-scale, concentrated poultry production sites, not in backyard farms or in the environment.

(iii) The problem of infectious disease in poultry and pig production has been made greater by the spread of factory farming worldwide

(iv) The crowded and stressful conditions that animals experience in factory farms makes them less able to resist disease individually and more likely to spread pathogens among themselves and to the outdoor environment.

4. Intensive farms are more likely to become infected

Several examples show that large-scale industrialised farms are actually more rather than less likely to become infected and that their 'biosecurity' measures do not keep infections out.

Several studies of highly pathogenic bird influenza (HPAI) show that large, enclosed farms and backyard farms both get infected, but that the proportion of large, enclosed commercial farms that are infected is much higher. In other words, the risk of infection is considerably higher for large, enclosed farms, despite their claims to high biosecurity.

In many cases this higher risk can be quantified. The Pew Pro-Poor Livestock Initiative has pointed out that: 'HPAI H5N1 for instance has been reported to have caused outbreaks in large scale industrial poultry units with supposedly high biosecurity standards in South Korea (a 300,000 bird unit), Russia (two 200,000 bird units) and Nigeria (a 50,000 bird unit) in 2006, and in the UK (a 160,000 turkey unit) in 2007. Moreover, large(r) industrial-type flocks appear to be overrepresented in the list of HPAI H5N1 outbreaks reported to OIE vis-à-vis outbreaks in backyard / village flocks in relation to their respective shares of total national flocks.'[26]

Around 40 percent of the HPAI H5N1 outbreaks in domestic poultry reported to OIE between late 2005 and early 2007 occurred in poultry units of 10,000 birds or more (more than 25 percent occurred in units of more than 10,000 birds), while, even in many OECD countries, e.g. Germany, France, UK and Belgium, less than 10 percent of flocks consist of more than 10,000 birds.' [27]

In the early HPAI outbreaks in Italy (1999-2000), Netherlands (2003) and Canada (2004), the number and the proportion of industrial farms in the area that were infected was very much higher than the number and proportion of backyard flocks infected. In the Netherland, 17% of the industrial farms got infected compared to 0.1% (1 in 1000) of the backyard flocks. In Canada, 5% of the industrial and 2% of the backyard farms were infected. In Italy 382 or 12% of the industrial farms in the area got infected, whereas only 10 backyard flocks were infected (no data on proportion in this case, possibly because too many backyard flocks to count). [28]

5. Why industrial animal units present higher risks

The main risk for factory farms is their scale and concentration of animals, which biosecurity measures struggle to counteract.

'Larger facilities are often assumed to implement more advanced biosecurity measures, but the intensity of their operations also pose higher risks for infection and pathogen propagation. Over one cycle of 10,000 broilers for example, around 42 tons of feed and

100,000 l of water have to be supplied to the birds, and unless stringent measures are taken these remain potential routes of introduction, while around 20 tons of waste will be produced requiring disposal.

Measurement of aerosol emissions from a broiler operation revealed a million-fold elevated concentration of aerosolized invisible dust near a poultry barn fan as compared to outdoor air in a semi-rural area. These particles have the potential to remain suspended in the air for up to several days, and, depending on prevailing winds, poultry barn dust could be found several kilometers from its source. [29]

'Other pathogens have been shown to readily move in and out of poultry and swine houses. Pathogen entry was demonstrated in a recent study of C*ampylobacter*-free broiler flocks, housed in sanitized facilities, using standard biosecurity measures, and fed C*ampylobacter*-free feed and water. Seven out of ten flocks became colonized with *Campylobacter* by the time of slaughter and two flocks were colonized by *Campylobacter* strains genetically indistinguishable from strains isolated from puddles outside of the facility prior to flock placement[30].'

A study in Denmark found that as many as 30,000 flies may enter a broiler facility during a single flock rotation in the summer months[31]. HPAI outbreak in Kyoto, Japan in 2004, found that flies caught in proximity to broiler facilities where the outbreak took place, carried the same strains of H5N1 influenza virus as found in chickens of an infected poultry farm[32].'

The scale of the operations means that in practice the biosecurity protocols that are intended to isolate animals in factory farms from the outside environment are not maintained. Whenever surveyed, failures to follow the protocols are widespread, even in countries where compliance might be expected to be higher[33].

6. The disease risk for different types of farm

In relation to outbreaks in poultry, backyard farms that keep small numbers of birds in free-range conditions are frequently blamed for lack of 'biosecurity' and for contact with wildfowl, but the evidence that they attract infection is weak. Typically, the number of backyard flocks far outnumber large commercial farms, so the proportion of all backyard farms infected turns out to be much lower than the proportion of large and indoor farms. In that case it's clear that the risk of infection is actually higher for industrial than for traditional farms.

For example, the British Veterinary Poultry Association has pointed out that in the 2016/17 UK infections with H5N8, nearly half of the 13 poultry infections were in backyard poultry[34], without taking into account the much higher number of backyard holdings compared with commercial poultry farms. In fact, in that outbreak, no outdoor commercial layer or broiler flocks were infected, despite the fact that over 55% of the UK laying hen flock and 88% of the commercial holdings were free range or organic[35].

During the 2014/2015 outbreaks of HPAI in Minnesota, 232 poultry farms were affected but of these only 21 were backyard farms[36]. The large majority were commercial turkey farms, many of them integrated with major turkeymeat producers[37] . Equipment and vehicle sharing between farms was common, including feed trucks and poultry transport vehicles and loaders and one third of farms admitted to seeing wild birds inside the turkey barns![38].

An EFSA-commissioned report of 2017 claims that free range production increases the risk of infection with AI. The report based this view on the fact that, of 54 analysed outbreaks of AI (both HP and low-pathogenic, LP), 29 were in free range flocks and 25 in indoor housed

commercial flocks. This difference in prevalence is small, but further examination in fact shows that waterfowl accounted for the majority of the outdoor flocks, and when egg laying hens and meat turkeys are considered, the comparison looks different. Only 7% of outdoor laying hen units and 3% of outdoor meat turkey units got infected, compared to 20% and 28% of the indoor laying hen and meat turkey units were infected, respectively.[39]

Given that approximately half of all egg laying units in the EU are outdoor [40], one would expect these figures to be equal, or, if free range holdings carried a greater risk, for outdoor units to be the higher percentage. The EFSA report concludes "These data do not provide a strong indication of free-range (outdoor) farming as an indirect risk for introduction of avian influenza, in particular for Gallinaceous (turkeys and chickens) species.'

The timeline of H5N8 infections in 2014 also shows a pattern of large and enclosed farms being infected. In January 2014 the HPAI version of H5N8 was reported in a Korean farm holding 21,000 breeding ducks (followed by around 30 more outbreaks in chickens, ducks and geese), followed by poultry infections in Japan (April 2004, in a 100,000 broiler farm), Germany ('semi-closed' turkey farm), Netherlands (150,000 confined hens), UK (confined breeding ducks). The European H5N8 viruses were closely related to the Korean one[41].

From December 2014 to June 2015 the US was hit by 'the most costly HPAI outbreak to date' (3.3 billion USD)[42], with the loss of 48 million birds and 'strong evidence of farm to farm transmission'[43]. These outbreaks of both H5N8 and (more often) H5N2, affected mainly turkeys and laying hens in the midWest. One group of scientists also noted the 'common adverse environmental conditions' such as 'stress of egg production, higher density of birds, concomitant infections, or immunosuppression' which made the birds more susceptible to infection in farm conditions than when studied in a laboratory[44].

At the start of 2018 there were HPAI outbreaks in, for example, a farm holding 43,000 birds in Iraq, a farm holding 51,000 broilers in Japan, a farm of 54,000 birds in Afghanistan, farms of 190,000 hens, 82,000, 99,000 hens and 45,000 hens in China, of 55,000 birds in Inner Mongolia, and farms of over 100,000 birds each in the Philippines – illustrating the global reach of industrialised poultry and the viruses.

Evidence from Thailand also shows that industrial poultry units were more likely to become infected during an epidemic. During the H5N1 outbreaks in 2004 in Thailand, backyard flocks, which consisted of 30 birds per flock on average, constituted approximately three-quarters of all flocks whereas commercial broiler units consisted of 3,500 birds on average and accounted for only 2% of all flocks but 60% of the total poultry population. Active surveillance by the Thai government (taking swabs from 230,000 flocks in 50,000 villages) found that around 0.2% (2 per 1000) of commercial layer and broiler units were infected, whereas only 0.05% (5 per 10,000) of the backyard flocks were infected[45].

It is more than plausible that human activities are responsible for at least some instances of long-distance transport of virus that the industry finds easier to attribute to wild migratory birds and to the perceived biosecurity deficiencies of small-scale free-range husbandry. A supporting report for EFSA's 2017 Scientific Opinion on HPAI counted 345 HPAI outbreaks in EU 2005-2015 of which 79 (23%) were recorded as 'secondary' (buying animals, transport vehicles, contact with neighbouring holding)[46]. We can probably assume that in reality 'secondary' spread accounted for a lot more than 23%, since this is something farmers might be unwilling to admit.

The H5Nx viruses (eg H5N8, H5N6, etc, referred to as clade 2.3.4.4) seem to be especially suited to spread through intensive chicken production and poultry trade systems. Globally their presence coincides with areas of intensive chicken density (ie density of their hosts), intensive crop production and human density. Scientists have concluded this from a global mapping of infected areas and a number of environmental conditions (eg distance to water, temperature, extensive or intensive chicken density, tree cover, cultivated areas, human urban density, duck density)[47].

6.1 Salmonella and Campylobacter

The same pattern is seen in farm infections with other pathogens. Salmonella and Campylobacter are common foodborne infections that caused nearly 246,600 confirmed human cases of Campylobacteriosis and nearly 91,900 human cases of Salmonellosis in the EU in 2018[48].

Chicken meat is responsible for most of the human cases of Campylobacter infection. In 2018 EFSA reported an average of 37.5% of samples of chicken meat were positive for Campylobacter in the EU, together with 28% of turkeymeat samples and 5.8% of pigmeat samples[49]. The results for retail chicken meat were 59.79% positive in UK, 51.84% in Germany, 37.63% in Netherlands, 66.67% in Spain and 0.00% in Sweden[50].

From the countries that reported to EFSA, samples from live broilers on farm were 65.3% positive in Greece, 55.5% positive in Austria, 24% positive in Denmark 22.2% positive in Italy, 100% positive in Czech Republic, 3.5% positive in Finland.[51]

Industrial, enclosed farming is often proposed as the most ‘biosecure’ approach for minimising the foodborne pathogens Salmonella and Campylobactor in poultry and pigs because it should prevent the pathogen entering the housing and because housing is easier to disinfect that the outdoor environment. In particular this is used as an argument for keeping laying hens in cages. This argument is widely used by the industry, and is often taken as self-evident, but the evidence does not support it.

A 2017 Swedish study assessed the risk of the expansion of outdoor production systems for poultry, prompted by consumer and welfare demands. This concluded that there was no statistically significance difference between Salmonella levels in indoor and outdoor flocks (laying hens and broilers) and no increased risk of human exposure in outdoor poultry production[52]. A 2015 US review identified as risk factors for eggs being contaminated with Salmonella a flock size of more than 30,000 hens and an egg production rate higher than 96%, suggesting that the concentration of birds and pressure for maximum output per hen increase risk[53]. The consensus of opinion is that less intensive production systems with higher welfare do not increase the risk of Salmonella infection and on balance are likely to have a lower risk of infection.[54]

A number of studies show that hens confined in cages are actually more likely to be infected[55]. A Belgian study of 2008 found that cage production was actually the main risk factor for Salmonella in laying hens and that the risk was 10 times higher in cages[56]. A 2010 baseline study in Great Britain found that Salmonella was present in 25.8% of farms using cages, compared to 7.1% of free-range farms and 5.4% of organic (free-range) farms.[57] A German study similarly found that the proportion of flocks positive for Salmonella was 46.3% in conventional cage systems compared to 21.9% in free range systems.[58]

Salmonella may be introduced into the sheds on the clothing or vehicles of workers and others entering the farm. Wild birds are less likely to carry infection into poultry sheds than often thought, as only 0.2% of healthy wild birds have been found to carry Salmonella[59].

Contaminated feed or water is another major source of infection. The major 2010 S. Enteritidis outbreak in the USA that lead to nearly 2,000 human cases and recall of over 500 million eggs was related directly to contamination in the feed mill. Salmonella in laying hens in the UK and other countries has largely been reduced by the use of vaccination.[60]

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Broiler production is the most intensive and industrialised in the EU (and globally). It's clear that industrial methods do not provide biosecurity in the case of Campylobacter. An additional possible reason for the very high prevalence of Campylobacter infection in broilers is that genetic selection of the modern broiler has made these chickens more susceptible to infection. [63]

6.2 Swine flu and other diseases of the pig industry

The record of the global pig industry over recent years also demonstrates the failure of the industrial model to achieve biosecurity.

In the US, the proportion of small herds (i.e. with fewer than 5,000 animals) has fallen from over 70% in 1994 to less than 20% in 2008. The modern large herds are maintained by a constant supply of new young pigs, probably fully susceptible to diseases such as swine influenza. Whereas swine influenza in the US was a seasonal disease, like human influenza, there is now year-round transmission in pigs[64]. This creates a constant opportunity for infection of workers. Worryingly, the HPAI H5N1 that originated in poultry in 1996-1997 (often fatal to people) has been found in pigs in China and Indonesia[65], explaining why pigs have been considered a 'mixing vessel' for reassortment of influenza viruses because they can be infected with both human and avian influenza strains[66].

As Singapore experienced the boom in pig production in the 1980s, the Central Veterinary Laboratory reported that on one estate the number of pigs had doubled to 400,000 and the density of pigs on big commercial units had increased by 2/3 (up to 2500 pigs per hectare), 'recurrent viral diseases have emerged' with 'widespread viral persistence within a dense population of susceptible hosts.' The most common and infectious ones were Aujeszky's disease virus (Herpesviridae), swine fever virus (togaviridae) and gastroenteritis virus (coronaviridae)[67].

The multiple reassortant events, which occur when a pig is simultaneously infected by more than one virus, gave rise to the first pandemic of the 21st Century. Initially termed 'swine-origin' H1N1, the virus that was later declared to be a pandemic H1N1 virus first emerged in Mexico in 2009[68].

African Swine Fever was identified in Kenya in 1929 and today the virus can kill 30%-100% of infected pigs.[69] As well as domestic pigs, the virus is carried by wild boar, by flies and by ticks. From 2007 it spread to multiple countries across Asia and Europe. The virus can be spread by live or dead pigs, pigmeat, contaminated feed, farm equipment and clothes of personnel[70].

Since 2014 ASF has spread rapidly in Eastern Europe, from there to China and Southeast Asia (Mongolia, Vietnam, Cambodia, Korea, Lao, Myanmar, Timor-Leste, Indonesia) and has now reached Oceania. In China 12 million pigs were killed between August 2018 and early March 2020[71]. China's pig production is expected to have declined by around 20% in each of the last 2 years as a result of the ASF virus[72]. By late March it had infected commercial pigs farms in Romania, Bulgaria, Russia, Poland (where nearly 24,000 pigs were culled on one farm, including 17,000 piglets and 7,000 sows[73]) and Ukraine. Infected wild boar have been found in western Poland, the Baltic states, Hungary, Slovakia and Russia[74]. In May 2020 the ASF virus was reported in India and a fattening pig unit of 10,000 pigs in Poland was infected after buying piglets from the first infected farm.[75]

Other new diseases have decimated the pig industry in recent years; Postweaning Multisystemic Wasting Syndrome (PMWS, partly caused by the porcine circovirus PCV) emerged during the 1990s and spread over North America, Europe and Asia; Porcine Reproductive and Respiratory Syndrome (caused by the PRRS virus, also called 'blue ear') also arose from the late 1980s, first identified in the US, and has become the most important disease in intensively kept pigs in North America, Europe and Asia. Its spread is most often due to the movement of infected animals, and the virus is found in faeces, urine, semen (artificial insemination is widely used in pigs) and on farm equipment and supplies, and has been found in insects[76].

The record serves to show that, at best, the industrial model of 'biosecurity' is often ineffective in protecting animals from infection and preventing the spread of the disease between farms. Further, the evidence shows that industrial farming has actually created new epidemic diseases and provides the conditions to make infectious diseases among the animals more likely.

7. How industrial poultry production increases virulence in viruses

Far from protecting poultry from infections, the intensification of the poultry industry can make viruses more virulent. There are several characteristics of industrial poultry production that facilitate the evolution of HPAI viruses from LPAI viruses and facilitate the continuing circulation of the HPAI virus in poultry and the environment. An important one is that industrial production gives the virus a constant supply of thousands of new hosts. This means that the virus can be highly virulent and still be transferred to a new host before the current one dies. The more times a virus encounters the immune system of a new host, the more it has to mutate in order to infect each new host, and the faster and further it will mutate. This means that industrial poultry production is a system that selects for virulent mutations.

An example is Marek's Disease, which used to produce relatively mild illness, but according to scientists has 'become substantially more virulent over the last 60 years. This evolution was presumably a consequence of changes in virus ecology associated with the intensification of the poultry industry.' The virus can afford to be more virulent because industrial chicken production provides it with a rapid supply of new hosts. The authors comment, 'These results illustrate the dramatic impact anthropogenic change can potentially have on pathogen virulence.' [77]

In the case of HPAI, the high-pathogenic viruses actually originated among the thousands of birds crowded together in poultry sheds. A 2016 study in the journal *Epidemics* tells us, 'It is well known that highly pathogenic avian influenza (HPAI) viruses emerge through mutation of precursor low pathogenic avian influenza (LPAI) viruses in domestic poultry populations.'[78]

Genetic studies have shown that HPAI viruses are not separate strains (ie having a separate origin from LPAI viruses) but that they arise from LPAI (ie non-pathogenic) strains. The current understanding is that HPAI viruses typically arise by mutation from LPAI viruses introduced into terrestrial poultry – chickens, turkeys, quail. When a LPAI H5 or H7 virus gets into terrestrial poultry, the LPAI may within weeks or months unpredictably mutate into a LPAI virus[79].

A background report in support of EFSA's 2017 Scientific Opinion on Avian Influenza concludes that 'The mutation of low pathogenic avian influenza (LPAI) virus to highly pathogenic avian influenza (HPAI) virus is a major factor in the disease burden from avian influenza in poultry flocks.'[80].

Scientists have concluded that the mutation from LPAI to HPAI takes place when the LPAI virus has crossed from wild birds to poultry[81] , and particularly in gallinaceous domestic poultry, such as chickens[82]. One reason for this conclusion is that HPAI viruses found in wild birds (although only rarely) were most often near to sites of HPAI outbreaks in poultry or were close in time to HPAI poultry outbreaks[83].

Before the 1990s, chickens were rarely infected by bird flu and surveillance in slaughter plants in Hong Kong in the 1970s found it much more often in ducks than in chickens. Scientists say the assumed evolution of H5N1 is from aquatic birds (ducks) to partially aquatic birds (geese) to humans, and that 'the global intensification of the poultry industry was a key element in adapting avian influenza viruses to terrestrial species.'[84]

The H5N1 virus needed to evolve in order to infect chickens because bird flu in waterfowl is transmitted via the faecal-water-oral route and chickens don't swim. During its evolution to infect terrestrial animals the virus may also have become more airborne and more able to infect via the respiratory route (rather than the typical faecal-oral route in water)[85].

8. Can the emergence and transmission of HPAI be solved by biosecurity?

The evolution takes place either by conversion of LP to HP viruses or by reassortment between different genetic segments of low and highly pathogenic viruses already circulating with the flock.[86]

Between 1959 and 2015 the authors identified 39 LP to HP conversion events and 127 reassortment events where a new HPAI virus emerged within poultry flocks Only 2 of the conversion 39 events took place in backyard rural flocks and 37 took place in intensive commercial units. In addition, a majority of these events took place in high-income countries, where over 95% of chickens are raised intensively for egg and meat production. Over 90% of the reassortment events took place in Asia in countries where intensive production systems were expanding at the expense of backyard systems.

Among examples where the LPAI virus has been identified in the poultry before the HPAI virus emerged are: Pennsylvania in 1983, Mexico in 1994, Italy in 1999, Chile[87] in 2002, Pakistan in 2003 and Canada in 2004. In all these cases the mutation to virulence occurred in an intensive confinement system – for example 600,000 broiler breeders on the farm in Chile and 700,000 caged laying hens at the index farm in Pakistan. Because LPAI may not cause unusual symptoms of illness in domestic chickens, it's possible that LPAI viruses may be circulating in a flock for some time without being detected[88].

In January 2016, a new HPAI H7N8 emerged in a commercial turkey farm in Indiana, followed by secondary spread to another 9 farms (together more than 400,000 birds were

killed or culled). Genetic analysis suggested that the HPAI H7N8 evolved from a LPAI H7N8 virus that was assumed to be introduced into the first farm from diving ducks using the Mississippi flyway in autumn 2015[89].

Scientists who studied the emergence and spread of the H5N1 virus from its origin in 1996 in Guangdong to its spread globally in 2003-4 have concluded that, 'The geographic extension and the genetic evolution of the virus since 1996 had probably taken place without any link with wild birds.' In 2004, 2200 wild birds were tested in Hong Kong and none found positive for H5N1. The only wild birds in Asia found sick were victims of the virus circulating in domestic birds.' [90]

Knowing that HPAI viruses evolve inside crowded poultry farms when the domestic birds are already circulating LPAI viruses derived from wild birds, we might conclude that the emergence of HPAI viruses in poultry could be prevented by sealing off the poultry indoors to prevent all contact with wild birds. But this has been proved time and time again to be ineffective in preventing either the initial infection or the spread of viruses. FAO stated in a 2017 article on the epidemiology of AI that, once the virus is introduced to a flock, 'the virus is spread from flock to flock by the usual methods involving the movement of infected birds, contaminated equipment, egg flats, feed trucks, and service crews, to mention a few.' [91]

Why does the 'industrial farm biosecurity' strategy not work? The reason comes back to the fact that large intensive farms are more likely than small and traditional farms to experience outbreaks of devastating outbreaks of infection. This is entirely consistent with the known facts of epidemiology that a large, concentrated, indoor population of susceptible hosts (a factory farm) is much more likely to propagate infection than a small and less concentrated outdoor population (a backyard or traditional farm).

9. Why factory farming creates the conditions for infectious disease

The industrial model of animal production has created the need for protective biosecurity just because it is an inherently unhealthy system for keeping animals.

Far from being biosecure environments, the conditions of factory farms are the perfect breeding ground for infectious diseases. Animals are crowded together in sheds or feedlots in tens or even hundreds of thousands, and are under stress from a number of factors that are likely to weaken their immune systems[92]. They are under stress from the environment (including social stress)[93] and physiological stress from fast growth or continuous reproduction[94], and stress affecting their immune systems due to the many pathogens circulating in intensive production systems[95].

Numerous scientists have pointed out the association between intensive animal production and disease risk. European virologists have pointed out that 'The unprecedented crowded conditions are undoubtedly major drivers of the emergence and spread of pathogens in domestic animal populations, allowing their increasingly more frequent cross-species transmission to humans.'[96]

The US Council for Agriculture, Science and Technology has stated that 'In intensive agriculture, larger quantities of raw materials and products flow within a country and between countries. A major impact of modern intensive production systems is that they allow the rapid selection and amplification of pathogens, thus there is increasing risk for disease entrance and/or dissemination.' [97]

The Pro-Poor Livestock Initiative report from Johns Hopkins School of Public Health has concluded that 'The proximity of thousands of confined animals increases the

likelihood of transfer of pathogens within and between these populations, with consequent impacts on rates of pathogen evolution.'

In spite of a wealth of evidence, the industry and regulators often continue to attribute health problems to insufficient biosecurity rather than to the inherent flaws in the industrial animal production model itself.

10. Biosecurity and wild animals

It is often stated by the industry and some regulators that infectious disease in farmed animal is kept going because there is a 'reservoir' of the pathogen in wild animals. This has been particularly put forward as the reason for the apparently endless circulation of HPAI viruses among poultry worldwide. However, the evidence is almost the reverse. HPAI viruses originate in domestic poultry, not in wild birds, and were transmitted from poultry to wild birds. Without the poultry industry, any HPAI affecting wild birds would rapidly die out. If there is a 'reservoir' of HPAI viruses, it is the poultry industry.

Confirming this conclusion, the FAO has stated in a technical update on the H5N1 outbreaks, 'In intensive surveillance schemes in Europe, North America, Asia, and Africa encompassing some 750,000 samples, very few healthy wild birds have been found to harbour [HPAI] H5N1 viruses. They are not considered the reservoir of this virus.' [98]

Before the Asian HPAI H5N1 outbreaks in chickens (from December 2003), there had been no evidence of HPAI in wild birds except for a few found dead near outbreaks in poultry, in spite of numerous surveys over many years, and the infection route was from domestic to wild birds. On the occasions that wild individuals were infected by HPAI it would not have caused a major outbreak because of the lower densities of hosts, so the virus could not be passed on to a sufficient number of healthy birds before the sick one died[99].

It's now generally accepted that HPAI viruses were originally passed from poultry to wild birds. What is not known is how likely it is that wild birds infected by poultry go on to infect other poultry either locally or by long distance migration. FAO experts on bird flu in wildlife have stated: 'spill back of a highly virulent strain into wild birds is rare'[100], but it equally plausible that it happens frequently.

Many of the disease outbreak reports to OIE state the source of the infection as 'unknown'. Often the source of HPAI is said to be 'wild birds' and the source of African Swine Fever assumed to be 'wild boars'. Often, these attributions of wild animal sources must be the most convenient assumption rather than having been scientifically proved.

In some cases, outbreaks occur near or on a known wild bird migratory flyway, and at the right time of year for migration. This seems may be the case for some outbreaks in Europe following outbreaks in central Asia and in the US midWest following the arrival of infected wild birds near the Pacific flyway. But this is not always the case. So it is plausible that wild birds are infected by 'spillover' from poultry sites and carry the virus either all the way or by relay along their migration routes, and that any infected wild bird can pass the infection on before the virus kills it.

FAO has concluded that by 2010, 'H5N1 HPAI has only been detected in 63 of the over 200 countries or territories worldwide. This suggests that wild birds are not the primary spreader of H5N1 HPAI but that human movement of virus through trade, marketing, and fomites likely accounts for the significant spread of the virus'. [101]

As we have already noted, there is much evidence of farm-to-farm transmission by human activities. During the 2014/2015 outbreaks in Minnesota, 232 poultry farms were affected but

of these only 21 were backyard farms[102]. The large majority were commercial turkey farms, many of them integrated with major turkeymeat producers[103] . Equipment and vehicle sharing between farms was common, including feed trucks and poultry transport vehicles and loaders and one third of farms admitted to seeing wild birds inside the turkey barns![104].

Surveillance of wild birds after the 2014-2015 HPAI outbreaks in North America left the role of wild birds as a mystery. The scientists concluded: 'Virologic surveillance in 2014/15 and over the previous 43 years failed to detect HPAIVs in wild aquatic birds before or after the poultry outbreak, supporting the premise that there are unresolved mechanisms preventing wild aquatic birds from perpetuating HPAIVs'. [105] The researchers termed the non-appearance of H5Nx in wild birds an 'enigma' and conclude [italics added:

'The recent North American experience with HP H5Nx IAVs suggests that these viruses are not well fitted to persist in wild bird populations and likely *will disappear without an endemic poultry source*. *It is likely that various poultry production systems and/or practices facilitate the perpetuation of HP IAVs, which can then spill over into wild birds and other host species.* This observation is consistent with the apparent disappearance in Western Europe of HP H5N1 following detection in 2005, 2006, and 2007 and of H5N8 in 2014.'[106]

No-one really knows whether migrating birds are a major means of spreading HPAIs long distance (for example from Asia to Europe) or whether the virus arrives via trade in feed, live birds, meat, travel by people, etc. Often (but not always) a HPAI is found in both poultry and dead wild birds either at the same time or in roughly the same area. The authorities tend to assume this means that wild birds have infected the poultry, but (because surveillance of disease in wild birds is very difficult) it is equally possible that the HPAI evolved among the poultry first (possibly unnoticed) and was transmitted to wild birds in the vicinity.

It seems plausible that long-distance transmission of HPAI can sometimes happen through wild bird migration, for example (eg the timing of the wild bird outbreak of H5N8 in September 2016 in southern Russia followed by the appearance of H5N8 in Hungary and later throughout Europe during the autumn/winter of 2016/2017). But the migratory birds almost certainly became infected by contact with poultry sites where the virus was circulating. The precise mechanism of transmission remains unclear. The only clear conclusion is that the industrial-scale poultry industry that arose globally in the late 20th century has upset the longstanding balance between bird flu viruses and birds (wild or domestic) and that industrial-style biosecurity measures have not protected against it. HPAI is a creation of the poultry industry that transformed relatively harmless LPAI bird flu into a lethal disease is circulating apparently endlessly in poultry and that infects wild birds who come in contact with it.

11. Conclusion: The factory farm model is a risk to biosecurity

Biosecurity properly understood is an integrated concept that includes animal and public health, the health of the environment, biodiversity, food safety and food security. Industrial animal production, which has become standard practice throughout the world, presents high and unnecessary risks across all these categories and is not even able to prevent diseases emerging from factory farms by its own protocols of biosecurity[107].

The 'industrial farm' model of biosecurity is put forward as the solution for the problem of farmed animal disease. The record serves to show that, at best, the industrial model of 'biosecurity' is often ineffective in protecting animals from infection and preventing the spread of the disease between farms. Further, the evidence shows that industrial farming has actually created new epidemic diseases and provides the conditions to make infectious diseases among the animals more likely.

12 Additional sections on African Swine Fever and Porcine Reproductive and Respiratory Syndrome

Biosecurity and factory farming

ASF and PRRS sections only

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Contents:

Summary of ASF and PRRS sections

African Swine Fever ASF: The extraordinary spread of ASF globally, from Africa to Europe to China and southeast Asia, illustrates the failure of 'industrial biosecurity' in regions of dense, concentrated and intensive pig production, where we have provided the virus with a constant supply of naïve hosts.

The 'new era' of ASF started in 2007 with its re-introduction to Georgia via infected pigmeat, from where the highly virulent Georgia strain of ASFV reached the Caucasus by 2008, from 2014 spreading to Ukraine, Russia, Lithuania, Latvia, Estonia, Poland, Bulgaria and Romania by 2017, thence to eastern Siberia, China in 2018, and Vietnam, Cambodia and North and South Korea, and much of south east Asia by the end of 2019.

The long distance introductions of ASF virus took place mainly in infected pigmeat or other pig products. The virus reached Irkutsk from the nearest known source thousands of kilometers away, and 1000km from the border with China, the next country to be infected.

ASF virus spread regionally between neighbouring countries and between farms is typically by human activities: EFSA has stated that 'In the vast majority of introductions, direct contact with infected domestic pigs or wild boar could be excluded as the likely route of introduction', compared with indirect contact via fomites or the environment.' ASF is also known to be transmissible by ticks and by wild boar. ASFV can persist for weeks in blood, faeces and urine excreted in the environment by infected pigs for more than a week.

Once ASF has been introduced to a country or region (through meat, pig products, feed, etc.), viral spread between farms or backyards depends essentially on how human activities enable those groups of susceptible hosts to be connected.

A number of ASF outbreaks that occurred in large commercial farms in Russia and Lithuania have been explained by potential gaps in terms of compliance with the biosecurity rules,

such as improper disinfection of clothing and boots, or contaminated food brought onto the premises, or by farmers hunting wild boar and handling potentially infected carcases.

Feed is an important route of transmission. Swill-feeding with infected meat is a widely cited cause, the likely cause of 62% of the first reported cases in China. The ASF virus can be easily transported in feed and water, as the level of virus needed for infection is low (1kg of contaminated feed).

Experiments show that ASF virus in plant-based feed products (including soymeal) could survive a trans-Atlantic shipping of up to 30 days (mean half-life in feed ingredients was 12 days). Millions of tonnes of feed ingredients are imported annually from countries where the virus is active.

Trading and transport of pigs among either small or large producers is cited as another major cause of local spread of ASF.

In Romania a 3-farm industrial pig business, the 2nd largest pig farm in Europe, was infected probably via water taken from the Danube. In some countries there are reports of late or non-reporting, selling or burying known infected pigs and dumping them in rivers.

Although the authorities tend to emphasise small and backyard farms as a biosecurity risk, the evidence shows that all sizes and types of farm have been infected, and large-scale industrial pig farms have not been able to keep out the virus. The earliest reported infected farm in China held 19,000 pigs. In Sardinia, well-managed and registered free-roaming ('open') farms were found to be at lower risk than unregistered enclosed farms.

The evidence for both ASF and PRRS viruses is that there are two main disease risks that apply to all pig keeping and are common to different farm types:

i) density of pigs in an area and proximity between groups/herds
(ii) poor or illegal practices, especially swill-feeding of untreated pigmeat and transport/trading of infected pigs.

FAO has said that, 'The higher density of pigs in small-scale confined production systems leads to a higher risk of pathogen circulation among herds' and that 'Large integrated farm systems [and] regions with large livestock populations have a higher regional disease challenge'. Because the number of backyard or small units is so much higher than the number of industrial farms in many of the infected countries, the large confined farms may actually be at higher relative risk.

Wild boar, feral pigs, bushpigs and warthogs can become infected with ASF virus, which can also be transmitted by ticks, and in ASF's original African setting there has long been what is referred to as a 'sylvatic cycle' of infection between wildlife and domestic pigs.

Wild boar are clearly not a possible cause of transcontinental transport of the virus, but after the virus was imported into European domestic pig populations, a small minority of wild boar have also become infected and are frequently cited as a risk factor.

As is found for HPAI in wild birds, very few live (hunted) wild boar are found with the disease, even though they are quite often found dead with it. Very few live wild boar carry the virus (e.g. 2.4% in Estonia, a heavily infected country, often less than 1%) and there is 'limited persistence of ASFV in wild boar populations' because the disease is so severe and limits their movement and so limits transmission to immediate individuals.

There may be indirect transmission via infected carcases of wild boar or domestic pigs. In areas where both infected farms and infected dead boar have both been found, it is equally possible that the boar were infected by the farms. A study from 2014 to 2017 in Lithuania found more than 10-fold increase in the prevalence in both farmed pigs and in hunted wild boar in 2017 compared to 2014, but found no correlation between the density of wild boar and the number of recorded ASF cases in either wild boar or pigs.

Wild boar hunts are a considerable, often ignored, factor in keeping ASF circulating in parts of Europe. Hunting and supplementary feeding of wild boar (for hunting or deterrence from crops) is common in Europe, and has greatly encouraged the growth of wild boar populations, as has 'Massive developments of industrial agriculture and favourable landscape changes provided additional feeding resources and shelter' and presumably limiting their natural habitat.

Porcine reproductive and respiratory syndrome: PRRS was a new pathogen of pigs first identified in 1987 in the US, and shortly after in Japan, Germany and Netherlands. OIE says that 'Within a few years it became a pandemic.' Vaccines exist which OIE says are effective at controlling outbreaks, and scientists have recently been working on gene-editing to breed PRRS-resistant pigs. The original two virus types (I and II) have within 20 years spread globally, in Europe, North America, South America, China (2006/7) and the rest of Asia.

PRRS emerged and was first identified in countries where pig farming was already industrialised, such as the US where a standard pig breeding operation holds 5,000 sows, and the 'biosecurity' of modern, intensive farms has not been able to keep it out. Of China, in spite of the consolidation of the industry after 1990 and the disappearance of many small and medium sized farms, FAO said in 2018 that 'Notwithstanding changes in biosecurity protocols implemented on commercial farms, the commercial industry appeared incapable of controlling the spread of swine diseases such as PRRS in 2006'.

PRRS virus is spread via the movement of infected pigs, together with infected faeces, urine and semen, pig meat, milk, saliva, colostrum, on vehicles and other fomites and potentially by insects, or airborne (live virus up to 4.7km from an infected group of pigs is confirmed).

Pig density is almost certainly the highest risk for PRRS spread once infection has been introduced to an area via human activities. All types of farm have been infected, including backyards and villages in China and Vietnam, but there is good evidence that large, intensive farms have been greatly affected by it, and that their 'industrial biosecurity' has failed to keep the virus out.

In Hungary in 2015, 61% of large-scale fattening farms were infected, and in Canada in 2006-2007 74% of pig breeding sites were infected. In Illinois in a PRRS epidemic in the early years of the virus, herds with total confinement housing were 3 times as likely to be infected, and farms buying in semen were twice as likely to be infected, compared to others. The median number of sows in infected herds was twice the median number of sows in uninfected herds. Overall, 'larger herd size was associated with an increased risk of PRRSV infection and clinical PRRS.'

In 2008-2009 in England, higher pig density and earlier weaning were identified as risk factors, both indicators of intensification. PRRS was confirmed on 35% of 147 farrow-to-finish farms studied, leading to the conclusion that 'there was an 'association between high pig density areas and higher prevalence of infection, suggesting that current control measures are not effective.'

In the US, around half of the total pig production in in the 3 states of Iowa, North Carolina and Minnesota, and scientists point out that this region 'would not only be exposed to higher amounts of emission of airborne viral particles from potentially infected herds, but also have higher rates of traffic and transportation besides other unmeasured PRRSV exposures (e.g. comingling of producers and opportunities for cross-contaminations during manure spread, culling, slaughterhouse and other events.'

African Swine Fever ASF

1. How ASF spread globally from Africa

The extraordinary spread of ASF globally illustrates the failure of 'industrial biosecurity' in regions of concentrated and intensive pig production, where we have provided the virus with a constant supply of naïve hosts.

From being identified in Kenya in the early 1920s, the virus spread to central and west Africa, Madagascar and Mauritius. It was transmitted to Portugal in 1957, via infected pig products fed to pigs[108]. From Portugal ASF spread to Spain, Malta, Italy, France, Belgium and the Netherlands, but was eradicated from all these except Sardinia by the 1990s. ASF also spread (and subsequently reported as eradicated) to Cuba (presumably from Spain), Haiti, Dominican Republic and Brazil[109].

The 'new era' of ASF started in 2007 after its re-introduction to Georgia [110] (again presumed to be in infected pigmeat[111]). From there the virus spread to other Caucasian countries (Armenia, Azerbaijan[112]) including the Russian Federation (2008-2009[113]). In 2014 the virus reached Ukraine, Belarus, and, consequently, European Union countries[114]: Lithuania, Latvia, Estonia, and Poland, Bulgaria, and was the start of further transcontinental spread. Because of the presence of wild boar in these regions, they were often considered responsible for the introduction and spread.

ASF reached Romania in 2017. Romania is an example of the very rapid spread possible in 2018 through pig populations where numerous backyard farms holding just a few pigs still coexist with industrial ones. In the Southeast region, a backyard farm and then a wild boar were first confirmed infected in June 2018 and within 4 months ASF had been confirmed in 943 outbreaks in backyard pigs, in 61 dead and 11 hunted wild boar, in 15 commercial farms and one slaughterhouse[115]. In 2019 EFSA's risk assessment for south east Europe concluded that the chances of the disease spreading 9 additional countries in the Balkans within one year of introduction was very high (66-100%).[116]

Since 2017 the highly virulent Georgia (2007) strain of the ASF virus spread to Siberia, to China (2018), Mongolia, Vietnam (2019), Cambodia (2019) and North Korea (2019)[117]. By the end of May 2020 OIE reported a total of 7,117 ongoing ASF outbreaks worldwide, of which 440 were new notifications[118]. By mid 2020 ASF was present in most of southeast Asia, including India, South Korea (2019), Malaysia and Indonesia[119].

ASF virus was reported in eastern Siberia (Irkutsk) March 2017, 'thousands of miles away from previously reported outbreaks and at approximately 1000 km from the border with China,' [120] and was reported in China just 17 months later.

The spread of ASF virus in China from August 2018 was very rapid. By April 2019 over 100 cases had been officially reported, covering almost all the provinces, autonomous regions and municipalities (except for Taiwan, Hong Kong and Macao)[121]. 33 cases were reported in rapid succession between 3rd August and 8th October 2018, affecting farms of all sizes, a

few backyards, and towards the end of the period, some villages. The first reported case was a farm holding over 19,000 pigs, and the 7th held over 14,600 pigs. Out of the 33 cases, the first 13 were on a 'farm' (presumably meaning commercial) and one slaughterhouse. Cases 14, 21,22 and 24 are described as 'backyards' and at the end in October cases 28 to 32 are described as in a 'village', and the last was a 'farm' with over 900 pigs[122].

In early 2019 ASF reached Vietnam, another major pig-producing country, where 'The most probable source and major cause of transmission across the countries' was thought to be illegal movements of meat and pigs over the border with China (250 km away from the first known infection[123].

2. How is ASF virus transmitted?

ASF is spread primarily by human activities that carry the virus on pigmeat, feed and water, semen, equipment, clothing and vehicles. EFSA's epidemiological analysis of 2018 for the European Union stated that although it was rarely possible to identify the exact route of infection, 'In the vast majority of introductions, direct contact with infected domestic pigs or wild boar could be excluded as the likely route of introduction. Inadequate biosecurity is likely to have contributed to introduction of ASF into domestic farms via indirect contact through contaminated fomites [objects that carry infection] or environment.'

A 2019 review of 11 instances of introduction of ASF in countries or regions since 1957, only 2 introductions (Russia, Poland and Estonia) are assumed to have wild boar as the transmission route, the rest being attributed to pig products[124].

In Romania, contact with infected water (farms near the Danube) was a major risk. A 3-farm industrial pig business in Braila county[125, 126] (Tebu Consult, reportedly owned by foreign investors[127]), the largest pig farm in Romania and second largest in Europe, was infected in August 2018. News reports at the time stated that 140,000 pigs had been culled at that business, and that in total 8 large pig farms had been infected.[128]

In some cases insects may have been involved, as no other explanation seemed to fit. EFSA reported that 'In several localities, the outbreaks appeared for the first time in farmers' backyards with two or three pigs, where the owners were elderly people who had no contact with the sylvatic environment [ie possible wild boar contact], the field, the forest and even the neighbours. They did not buy food, used only products from their own household, nor did they handle swill in pig feed.'[129]

News reports also cited some behaviour of people, such as burying pigs dead from ASF, dumping ASF-infected dead pigs in the Danube rather than notifying the infection to the veterinary authorities, and sending known diseased pigs to slaughter, as suspected or known risks, together with local reports of political scandals surrounding the outbreaks.[130]

Research published in 2019 shows that ASF virus can be easily transported in animal feed and water that is used on pig farms. The level of virus that causes infection in liquid is very low, although a bit higher in feed. The scientists said that 'The likelihood of infection increased dramatically after even 10 exposures [to the feed], or consumption of 1 kilogram of contaminated feed. Modelling multiple exposures increases the applicability of our experimental data to what would occur at the farm.'[131]

Millions of kilograms of feed ingredients are imported from countries where African swine fever virus is circulating[132]. Contaminated feed as a transmission vehicle for introducing transboundary animal diseases onto high-biosecurity swine operations has been recognized as a major risk factor since the introduction of porcine epidemic diarrhea virus into the United

States in 2013[133]. In 2019 scientists showed that ASF virus in plant-based feed ingredients could survive trans-Atlantic shipping of up to 30 days. The virus half-life was 14 days in complete feed and 10-13 days in soybean meal, with a mean for all feed ingredients of 12.2 days[134].

Historical outbreaks, including the introduction of ASF into the Caucasus region in 2007 and subsequent spread into Russia, have been attributed to swill-feeding of untreated pigmeat, since ASF virus survives in pigmeat for months and is resistant to temperature. China and Siberia were infected with the same viral strain identified in Georgia in 2007, with outbreaks occurring thousands of kilometres away from any source. ASF virus spread from Shenyang in northern China to Wenzhou, south of Shanghai, in around 3 weeks. In 2014 the introduction and spread of ASF in Latvia was similarly caused by feeding contaminated crops to pigs[135].

ASFV can persist for weeks in blood, faeces and urine excreted in the environment by infected pigs for more than a week. A number of ASF outbreaks that occurred in large commercial farms in Russia and Lithuania have been explained by potential gaps in terms of compliance with the biosecurity rules, such as improper disinfection of clothing and boots, or contaminated food brought onto the premises, or by farmers hunting wild boar and handling potentially infected carcases.[136]

Chinese animal health officials investigating the rapid spread in their country identified using ASF-infected meat from restaurants in pig swill as the cause of 13 of the first 21 officially reported cases. They also cited failure to disinfect trucks transporting pigs to slaughter or other farms, plus the reluctance of official veterinarians or farmers to report the disease, due to fear of being held accountable or economic losses[137]. Other Chinese scientist speculate that the virus entered China from an Eastern European country in smuggled pigmeat or offals[138].

3. The risks in industrial versus backyard production

It is common to see small pig farms (fewer than 10 pigs) cited as a risk factor, 'as it was assumed that these small farms would often implement suboptimal biosecurity measures'[139] . However, it is clear that both large and small farms are affected and the largest and presumably most modern farms also fail to keep out the virus. As the number of small or backyard farms far outnumber the large commercial ones, the large farms are probably at even greater relative risk.

Once ASF has been introduced to a country or region (through meat, pig products, feed, etc.), viral spread between farms or backyards depends essentially on how human activities enable those groups of susceptible hosts to be connected (through feed, water, trade, equipment, etc.). There is very little definite information on the path of transmission in any particular case. Apart from the (usually speculative) contacts with wild boar, we can draw two main conclusions about the main risks of disease spread:
(i) density of pigs in an area and proximity between groups/herds, especially infected ones, and
(ii) poor or illegal practices, especially swill-feeding of untreated pigmeat and transport/trading of infected pigs ('emergency sales'[140]).

Some sources make the assumption that poor practice is almost inevitable in backyard pig-keeping. However, on examination, the weaknesses often come down to specific practices, rather than to the nature of small farms themselves. A study of ASF spread in Georgia cited the trade and marketing of pigs by small farmers, and the use of middlemen, as an important route of transmission, together with swill-feeding[141]. A study of Sardinia, where most pig

farming is still traditional, found the lowest risk in registered ('censed', ie better managed) open farms where the pigs use communal land, compared to unregistered closed farms[142].

In Romania and Estonia, key risks were identified as the distance between farms and the number of outbreaks within 2km of a farm.[143] In Russia, 'since a single point source may easily develop into an epizootic event', the risks included the density of the local pig population, density of the road network, distance to the nearest infection, the movement of infected pigs, and the presumed circulation between domestic pigs and wild boar which prolonged the outbreaks[144].

The higher density in confined pig herds amplifies the risk of infection. OIE and FAO's 2010 report on Good Practices for Biosecurity in the Pig Sector has a tendency to categorise the practice of scavenging free-roaming pigs as having numerous health risks, but admits that 'The higher density of pigs in small-scale confined production systems leads to a higher risk of pathogen circulation among herds'. OIE adds that 'Large integrated farm systems usually create a large population of animals in a region' and that 'regions with large livestock populations have a higher regional disease challenge'. [145]

The records show that in different cases and countries the ASF was first officially reported in a backyard, a dead wild boar, or a large farm. South Korea reported its first outbreaks in mid-September 2019 when a total of nearly 4,000 pigs were culled on 3 farms owned by the same person by 16 September 2019. This report stated that there had been no swill feeding and fences [presumably against wild boar] had been installed[146]. Vietnam reported its first ASF case in January 2019 in a backyard farm with 20 sows, with illegal movements of meat and animals suspected[147].

An FAO rapid risk assessment for China, a few months before ASF arrived there, pointed out that 'Chinese pig production today is very different from the 1970s, when backyard farming supplied 70 percent of the internal market'. Since the 1990s, production consolidation began, 'modern, intensive swine farms developed rapidly' and medium and small farms were closed. Only 27% of total pig production now comes from backyards[148]. From the record of both the PRRS virus and the ASF virus in China, this move to 'modern, intensive production' did not succeed in preventing epidemic disease spread.

4. Are wild boar a biosecurity risk?

Wild boar, feral pigs, bushpigs and warthogs can become infected with ASF virus, which can also be transmitted by ticks, and in ASF's original African setting there has long been what is referred to as a 'sylvatic cycle' of infection between wildlife and domestic pigs.

Wild boar are clearly not a possible cause of transcontinental transport of the virus, but after the virus was imported into European domestic pig populations, a small minority of wild boar have also become infected. Doubtless they sometimes transmit the virus to local farms (sometimes via hunters) but the connection is often not clear and wild boar populations are not easy to study (or even count[149]). But we can also assume that the wild boar explanation would be more palatable to both for farmers and officials than human failings or the high density of potential hosts in the pig population.

As we have seen with HPAI in poultry, in some areas where wild boar live in proximity to pig farms and backyard pig keepers, some wild boar have become infected and are cited as a risk factor for the spread of ASF locally. In Romania, a key risk factor in non-commercial backyard farms has been identified as the number of outbreaks in the vicinity, either other farms or in wild boar, while for commercial farms the only risk factor was the distance to the

nearest domestic pig outbreak[150]. The arrival of ASF in Lithuania in 2014 has been 'linked' to the movement of infected wild boar from Belarus[151].

A 2016 study pointed out that while the ASF virus is likely to transmit between wild boars by direct contact or via infected carcases of either boars or pigs, 'it remains unclear whether ASFV can be sustained in these wild boar populations. For example, … recent analyses showed that there was no spacetime interactions among ASF cases in wild boars in south-west areas of Russia, suggesting the limited persistence of ASFV in wild boar populations.'[152]

A Polish study of wild boar movements (dispersal of yearlings, home range size) over 2 years 2014-2015 found that the steady spread of the ASF virus did not even correspond to where the wild boar were moving. The scientists concluded that the disease was so severe and lethal that it 'quickly hampers extensive movements and restricts disease transmission to only the most immediate individuals', followed by indirect transmission via carcases[153].

As is found for HPAI in wild birds, very few live (hunted) wild boar are found with the virus, even though they are quite often found dead with it. In Estonia, less than 2.4% of hunted wild boar tested positive for ASF, and there has been no increase in the proportion infected since 2016.[154] The evidence is that infection was mainly going the other way – there was an 18-fold increase in the probability of ASF-positive wild boar for each unit increase in the density of pigs per local administrative unit. [155]

EFSA's 2020 update on ASF in the European Union reported that the area affected by ASF expanded progressively in 2019, in a southwestern direction. However, there was no increase in the small proportion of hunted wild boar that were positive for ASF. For most of the 11 countries affected, fewer than 3% and for some less than 1% of the samples submitted from hunted wild boar from 2014 to 2019 were positive, although the proportion was 5% for Latvia[156]. The infection in wild boar appeared to be moving at between 3 and 12 km per year, but EFSA added that 'Human-mediated spread, both in pigs and wild boar, remains important.' [157]

In Latvia, wild boar were equally blamed for the arrival in January 2014 and persistence of ASF. All the 32 reported outbreaks in pigs were in areas where infected wild boar were living but in 16 out of 28 outbreaks classified as 'primary' the infection was attributed to swill feeding, while 12 were attributed to wild boars[158] – possibly a catch-all explanation rather than one scientifically traced. The prevalence of ASF in hunted wild boar was very low (average 1.5% of hunted boars), but interestingly was highest (2.3 – 2.9%) in the regions where there were clusters of infected pig farms[159]. From this it seems clear that the two sources of infection were driving each other, but there is no reason to think that the primary source was the wild boar rather than the infected pigs (swill feeding is also implicated [160]).

Overall, the reported evidence suggests that the growing ASF virus prevalence on farms, driven by human activities, may equally well have been driving the local wild boar infections. A study from 2014 to 2017 in Lithuania found more than 10-fold increase in the prevalence in both farmed pigs and in hunted wild boar in 2017 compared to 2014, but found no correlation between the density of wild boar and the number of recorded ASF cases in either wild boar or pigs. An outbreak on an industrial farm holding 19,000 pigs occurred in 2014 and on another holding 25,000 pigs in 2017[161].

The increase in wild boar populations has been directly and indirectly encouraged by human activities in some European countries, including protecting wild boar for hunting. A FAO/OIE/EU review of wild boar ecology in 2019 points out that 'Massive developments of industrial agriculture and favourable landscape changes provided additional feeding resources and shelter' to wild boar, plus 'large scale reintroduction efforts, facilitated by

protection, predator control and supplementary winter feeding', or summer feeding of piglets, sometimes at specially constructed feeding stations.[162]

Supplementary feeding is commonplace everywhere is northern and eastern Europe, although not well documented, and is done for a number of reasons, such as keeping wild boar away from crops, or attracting them to hunting locations. There is a clear correlation between the number of feeding sites and the size of the wild boar hunting bag. Research shows that 'supplementary feeding on the scale currently practiced in many European countries is excessive and significantly contributes to the increase in wild boar populations', say these ecologists. [163]

Wild boar hunts turn out to be a considerable factor in keeping the disease going in some parts of Europe. Game managers want to provide their clients with 'easy and predictable encounters with wild boars', and use supplementary feeding to achieve this. This results in gatherings of wild boar at feeding stations, increased infection between them, opportunities to scavenge infected carcases either at feeding stations or elsewhere, and the possibility of infected carcases being left out or taken home by hunters. The ecologists recommend terminating hunting in infected areas. [164]

5. Appendix to ASF section: CDC interview on reasons for spread of ASF, with Dr Niederwerder[165]

[Megan Niederwerder] The virus that's currently circulating is a highly virulent strain, which means that the mortality rates, after pigs are infected, really approach 100 percent. So, it's very…it has a high fatality rate. The disease that it causes is very severe. It often causes high fever in pigs, then they become lethargic, depressed, can have hemorrhage in the skin, as well as hemorrhagic diarrhea. And so, the disease is very, very severe as far as mortality rate, but also with regards to the disease that it actually causes for clinical signs in pigs. The other important thing about African swine fever virus is that it's only a pig disease. It does not affect humans.
[Sarah Gregory] Ah, that is important, because I'm sure a lot of humans were getting quite nervous now.
[Megan Niederwerder] Yeah.
In 2013, porcine epidemic diarrhea virus was introduced into the U.S. and that, was really, historically, the last major transboundary animal disease introduced into U.S. swine population. And several epidemiological investigations after PEDV was introduced revealed the risk of feed and feed ingredients serving as a potential route for, not only new transboundary diseases to be introduced into swine populations, but also as a potential route for these viruses to spread to susceptible populations within the country.

In fact, the U.S. imports several million kilograms of feed ingredients from countries such as China. And those feed ingredients are in…are, of course, in the country of origin—China, in this case—and then shipped overseas and delivered to feed mills, where the feed is actually manufactured.

And so, the risk is, is if those feed ingredients become contaminated, and then are shipped to the country, they can serve as a source of virus when introduced into the complete feed and then, of course, after the complete feed is manufactured at the feed mill, it is widely distributed to several barns where pigs then consume the feed.

Porcine reproductive and respiratory syndrome PRRS

6. The global spread of PRRS

According to the OIE, PRRS, a 'new pathogen of pigs', was first recognised in 1987 in the US, in 1980 in Japan, in 1990 in Germany and the virus (an *Arterivirus*) was identified in the Netherlands in 1991[166]. OIE says that 'Within a few years it became a pandemic.'[167] It was officially notified in China in May 2007, among nearly 3,000 'fattening pigs' in 'backyards and small-scale farms'[168], but reportedly had killed 1 million pigs during the previous year.[169] In 2008 pigs in the Philippines were found to be infected with both a highly virulent strain of the PRRS virus (PRRSV) but also with Ebola-Reston, which itself had been previously found in monkeys from the Philippines in outbreaks in the 1990s[170].

Vaccines exist and are said by OIE to be effective in controlling outbreaks and preventing economic losses[171]. But scientists have found that developing effective vaccines is difficult because the virus evolves rapidly and has many variants[172] and that vaccines have not yet been able to prevent the virus spreading[173]. There are also reports of some vaccines apparently causing the emergence of highly virulent variants[174]. In addition, various scientific groups have claimed success in breeding PRRS-resistant pigs through gene-editing[175] [176].

Initially, Type 1 virus emerged in Europe and Type 2 in North America, but both have spread globally. Most PRRSV isolates from South America and much of Asia are of Type 2 and it is assumed these viruses were introduced through the movement of swine or semen. Highly virulent Type 2 viruses have emerged in China and South-East Asia (highly pathogenic PRRSV).[177]

7. How PRRS is transmitted

The most common cause of PRRS virus transmission is through the movement of infected pigs, together with infected faeces, urine and semen, pig meat, milk, saliva, colostrum on vehicles and other fomites and potentially by insects, or airborne[178] [179]. Trials in Minnesota, one of the world's most dense pig production areas, have shown that PRRS virus can be transported long-distance by aerosols; PRRS virus was collected in 4.7km from an experimentally infected group of only 300 pigs[180].

8. Pig density and intensification favour PRRS spread

The record of PRRS that the 'biosecurity' of industrial farms was no more able to keep the virus out than backyards. The FAO stated in 2018 of the risk to China that 'Notwithstanding changes in biosecurity protocols implemented on commercial farms, the commercial industry appeared incapable of controlling the spread of swine diseases such as PRRS in 2006.' [181] Ironically, FAO wrote this when ASF virus had already reached eastern Siberia (Irkutsk), 'thousands of miles away from previously reported outbreaks and at approximately 1000 km from the border with China,' [182] and ASF was reported in China itself 5 months after FAO issued its warning

As with other livestock infectious diseases, pig density is almost certainly the highest risk for PRRS spread once infection has been introduced to an area via human activities. All types and sizes of pig farm are affected. PRRS emerged and flourished in industrial countries such as the US, where a standard pig breeding operation holds 5,000 sows[183] . In China it was first officially reported in villages and small farms[184]. In Hungary in 2015, 61% of large-scale

fattening farms studied had PRRS-positive pigs (the disease was then reportedly eradicated from large-scale fattening farms by 2019 through strict testing, quarantine and slaughter measures)[185].

A Canadian study of 54 pig breeding sites during 2006-2007 found 74% of sites infected, and the 4 main risk factors were identified as large pig inventory, proximity to the nearest pig site, together with two additional factors related to human activities (absence of a shower and free access to the front of the site by the rendering truck) [186].

In the US, around half of the total pig production is in the 3 states of Iowa, North Carolina and Minnesota[187], and the Minnesota/Iowa region is among the most dense pig regions in the world. Scientists notes that it 'would not only be exposed to higher amounts of emission of airborne viral particles from potentially infected herds, but also have higher rates of traffic and transportation besides other unmeasured PRRSV exposures (e.g. comingling of producers and opportunities for cross-contaminations during manure spread, culling, slaughterhouse and other events'[188].

A study of risk factors carried out during a PRRS epidemic in Illinois in the early years of the disease (1990s) found the median number of sows in infected herds was twice the median number of sows in uninfected herds, but the largest risk factor found was whether or not the herd was in 'total confinement' housing. 54% of herds with total confinement showed clinical PRRS (epidemic abortions, preweaning mortality, etc.), compared to 26% of herds without total confinement (odds ratio 3.3). The odds ratio for PRRSV infection was also high for herds buying in semen (OR 2.5). [189]

The Illinois scientists concluded that 'larger herd size was associated with an increased risk of PRRSV infection and clinical PRRS. It is possible that the larger number of animals represents a composite of demographic and management practices', including the increased animal density increasing the opportunities for transmission, a larger potential source of virus shedding, and possibly could increase the presence of flies and birds, infected needles, boots and hands of personnel[190].

A 2017 review of PRRS in the three main pig producing US states noted that 'The concentration of such a large population in a relatively small area resulted in high density, and consequently, relatively high vulnerability of the industry to the introduction and spread of infectious diseases.' The most important risk identified for PRRS was pig density. Farms located in 'managed' or cultivated areas were at higher risk that farms in areas of vegetation or trees [191]. Bushes, vegetation, trees and sloping land were found to reduce risk, thought to be because they acted as shields against incoming virus[192].

Similarly, in England PRRS was confirmed on 35% of 147 farrow-to-finish farms in 2008-2009, with a higher proportion of infected farms in areas with high pig density (more than 15000 pigs within 10 km radius from the farm). As well as high density, farms that weaned piglets later (28 days) were at lower risk than farms that weaned at 21-27 days (and we can assume that quicker weaning means these farms were more intensively managed). The study concluded that there was an 'association between high pig density areas and higher prevalence of infection, suggesting that current control measures are not effective.'[193]

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