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The spread of pathogens through trade in poultry meat: overview and recent developments

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Summary

Increasing international trade in poultry meat presents an opportunity for the global dissemination of poultry disease. However, it would be very unfortunate if expanding world trade resulted in animal diseases being used as unjustified non-tariff trade barriers.

For those avian diseases currently listed by the World Organisation for Animal Health, the current evidence suggests that only highly pathogenic avian influenza, Newcastle disease, and (for chicken meat) infectious bursal disease should be considered likely to be spread though trade in this commodity.

Keywords

Agreement on the Application of Sanitary and Phytosanitary Measures – Avian influenza – Highly pathogenic avian influenza – Import risk analysis – Infectious bursal disease – International trade – Low pathogenic avian influenza – Newcastle disease – Poultry meat.

Introduction

In 1970, 521,000 tonnes of poultry meat (approximately 3.5% of global production) were traded internationally. Since then there has been a considerable increase in the global production of poultry meat and the proportion exported worldwide. In 2004, 9,700,000 tonnes were traded internationally, equivalent to 12% of the total global production (190), and by 2008 this quantity had risen to 10,500,000 tonnes (65).

Although this trade may present an opportunity for the global dissemination of poultry disease, this risk should not be used as an unjustified trade barrier (27, 183). The World Trade Organization (WTO) Agreement on the Application of Sanitary and Phytosanitary Measures (SPS Agreement) allows for sanitary measures to be applied to traded commodities to the extent necessary for the protection of human, animal, or plant life or health. Under the SPS Agreement, such control measures should be based on an assessment, appropriate to the circumstances, of the risks to human, animal, or plant life or health which takes into account the risk assessment techniques developed by the relevant international organisations (198). The principal aim of import risk analysis is to provide importing countries with an objective and defensible method of assessing the disease risks associated with the importation of animals and animal products. The analysis should be transparent so that the exporting country is provided with clear reasons for the imposition of import conditions or refusal to import (195).

More than 100 diseases have been associated with commercial poultry. This review is restricted to a discussion of the likelihood of avian diseases listed by the World Organisation for Animal Health (OIE) being spread through the international trade in poultry meat. For further information, the reader is referred to a number of published import risk analyses that examine the risks associated with both listed and unlisted diseases (26, 111, 120, 121, 122).

For disease to spread in poultry meat, the aetiological agent must be able to:

– infect poultry species

– disseminate to those tissues likely to be present in traded commodities

– persist in these tissues during the processing and handling conditions to which poultry meat products are likely to be subject.

Disease is then only likely to spread from traded poultry meat if the aetiological agent is able to establish infection in a naïve recipient by the oral route (i.e. the feeding of raw or cooked scraps of poultry meat generated from the imported commodity). Therefore, the following factors should be considered germane to an assessment of the likelihood of disease spread through the international trade in poultry meat:

– which species of poultry are recognised as being susceptible to natural infection

– the distribution of carcass lesions after infection

– the likelihood of infectivity being present in the muscle of infected birds

– the ability to transmit infection by the oral route.

These factors are discussed below for each OIE-listed avian disease.

Avian influenza

The introduction of avian influenza (AI) in domestic poultry can result in widespread disease with high mortalities, leading to disruption of the poultry industry and export trade in poultry products. The direct and indirect economic costs associated with H5N1 AI in Asia from late 2003 to mid-2005 were estimated to exceed US$10 billion (175).

Avian influenza viruses are most frequently recorded in waterfowl, which are considered to be the biological and genetic reservoirs of all AI viruses and the primordial reservoir of all influenza viruses for avian and mammalian species (139, 170, 187). Wild birds, particularly migratory waterfowl, may introduce AI viruses into commercial poultry (70, 75), but have very little or no role in secondary spread (129). Infection of wild birds with AI usually produces no mortality or morbidity (175), although recent highly pathogenic AI (HPAI) H5N1 viruses have been associated with deaths in several wild bird species in Asia (41, 59, 165, 188).

Avian influenza infections have been reported in most domesticated Galliformes and Anseriformes, as well as in emus, ostriches, rhea, and Psittaciformes (55), although chickens and turkeys represent an abnormal host for influenza infection (171). Avian influenza is rare in commercial integrated poultry systems in developed countries but, when infection does occur, it can spread rapidly (175).

Sporadic cases of AI infection of humans have been described, although these are rare and typically present with conjunctivitis, respiratory illness, or flu-like symptoms. Recent Asian H5N1 human cases have been closely associated with exposure to infected live or dead poultry (175). However, surveys of people in four Thai villages (52) and a Cambodian village (186) found no evidence of neutralising antibodies against H5N1, despite frequent direct contact with poultry likely to be infected with this virus.

Low pathogenicity AI (LPAI) infection of domestic poultry can result in mild to severe respiratory signs, possibly accompanied by huddling, ruffled feathers, lethargy, and, occasionally, diarrhoea. Layers may show decreased egg production. High morbidity and low mortality are normal for LPAI infections (175). Intra-tracheal inoculation of poultry with LPAI may result in localised infection of the respiratory tract, with histological lesions and viral antigen distribution restricted to the lungs and trachea, although pancreatic necrosis is also reported in turkeys (37, 124, 163, 179). Intravenous inoculation of poultry with LPAI results in swollen and mottled kidneys with necrosis of the renal tubules and interstitial nephritis, and high viral titres in kidney tissues (163, 167, 168, 173, 176, 177, 178, 180). However, this renal tropism is strain-specific and is most consistently associated with experimental intravenous inoculation studies (175), although Alexander and Gough (6) did report the recovery of H10N4 LPAI from the kidneys of hens presenting with nephropathy and visceral gout. Salpingitis associated with a non-pathogenic H7N2 virus was described by Ziegler *et al.* (199).

In contrast, most cases of HPAI infection of domestic poultry are associated with severe disease, with some birds being found dead before clinical signs are noticed. Clinical signs such as tremors, torticollis, and opisthotonus may be seen for three to seven days before death. Morbidity and mortality are usually very high (175). Infection results in necrosis and inflammation of multiple organs, including the cloacal bursa, thymus, spleen, heart, pancreas, kidneys, brain, trachea, lungs, adrenal glands, and skeletal muscle (124, 141, 172). Histopathological lesions described include diffuse non-suppurative encephalitis, necrotising pancreatitis, and necrotising myositis of skeletal muscle (1). Viral infection of the vascular endothelium is suggested as the mechanism for the pathogenesis of HPAI infections in poultry, especially the central nervous system lesions (97, 98). Viral antigen can be detected in several

organs, most commonly the heart, lungs, kidneys, brain, and pancreas (124).

An early study found that AI virus persisted in refrigerated muscle tissue for 287 days, although feeding meat or blood from a viraemic bird to a susceptible bird did not transmit infection (150). Swayne and Beck (174) demonstrated that LPAI virus could not be found in the blood, bone marrow, or breast or thigh meat of experimentally infected poultry, and that feeding breast or thigh meat to a susceptible bird did not transmit infection. However, experimental infection of poultry with HPAI results in detectable virus in blood, bone marrow, and breast and thigh meat. An H5N2 isolate was found to achieve only low viral titres in muscle tissue ($10^{2.2-3.2}$ EID_{50} virus/g) and feeding this meat to susceptible birds did not transmit infection, whereas an H5N1 isolate achieved a much higher titre in muscle tissue ($10^{7.3}$ EID_{50} virus/g), which was sufficient to achieve transmission in a feeding trial. The authors concluded that the potential for LPAI virus to be present in the meat of infected chickens was negligible, while the potential for HPAI virus to be present in meat from infected chickens was high. However, it should also be noted that Kishida *et al.* (94) have described frequent isolations of LPAI (H9N2) from the meat and bone marrow of chicken carcasses imported from China into Japan, although extensive virus replication in bone marrow and muscle was not observed when chickens were experimentally infected with these isolates.

Newcastle disease

Newcastle disease (ND) is defined by the OIE as an infection of poultry caused by a virus (NDV) of avian paramyxovirus serotype 1 (APMV-1) that meets the criteria for virulence described in the OIE *Terrestrial Animal Health Code* (the *Terrestrial Code*) (197). It has been suggested that the spread of ND from one bird to another is primarily through aerosols or large droplets, although the evidence to support this is lacking (7). During infection, large amounts of virus are excreted in the faeces and this is thought to be the main method of spread for avirulent enteric avian paramyxovirus (APMV) infections, which are unable to replicate outside the intestinal tract (9).

In situ hybridisation studies (32) using four-week-old chickens experimentally infected with APMV-1 isolates, revealed widespread viral replication in the spleen, caecal tonsil, intestinal epithelium, myocardium, lungs, and bursa following challenge with viscerotropic velogenic strains. Neurotropic velogenic strains are associated with viral replication in the myocardium, air sacs, and central nervous system. Challenge with mesogenic viral strains is followed by viral replication in the myocardium, air sacs, and (rarely) in splenic macrophages. Lentogenic isolates result in minimal transient viral replication, confined to the air sac at five days post exposure and the myocardium at five to ten days post exposure.

Birds slaughtered for meat during disease episodes may be an important source of virus, and most organs and tissues have been shown to carry infectious virus at some time during infection with virulent NDV (3).

Infected meat has been shown to retain viable virus for over 250 days at –14°C to –20°C (7), and dissemination by frozen meat has been described historically as an extremely common event (100). Modern methods of preparing poultry carcasses and legislation on feeding untreated swill to poultry may have reduced the risk from poultry products, although the possibility of spread in this way nonetheless remains (5).

The NDV titre in the muscle of infected chickens is approximately 10^4 EID_{50} per gram and the oral infectious dose of NDV in a three-week-old chicken has been shown to be 10^4 EID_{50} (4). Tissue pools of muscle, liver, spleen, lungs, kidneys, and bursa, collected at two, four, seven, and nine days after experimental infection, are infectious for three-week-old birds (108). On the basis of these findings, it can be concluded that poultry meat is a suitable vehicle for the spread of NDV and that poultry, especially in backyard or hobby flocks, can be infected by the ingestion of uncooked contaminated meat scraps.

Infectious bursal disease

Two serotypes of infectious bursal disease virus (IBDV) are recognised (IBDV-1 and IBDV-2) (113). Very virulent (vv) strains of IBDV-1 (vvIBDV) have been described (42).

Chickens are the only animals known to develop clinical disease and distinct lesions when exposed to IBDV (60). Serotype 1 and 2 viruses have been isolated from chickens (113). Infection of chickens with IBDV-1 can lead to diarrhoea, anorexia, depression, ruffled feathers, trembling, and death. Flock mortality may be as high as 20% to 30%, although mortality rates of 90% to 100% have been associated with vvIBDV. The cloacal bursa is the primary target organ and infection leads initially to cloacal oedema and hyperaemia, which is followed by atrophy around five days after infection. Microscopic lesions in other lymphoid tissues are described (60). Studies commissioned by the New Zealand Ministry of Agriculture and Forestry and the Chief Veterinary Officer of Australia demonstrated that IBDV-1 is recoverable from the muscle tissue of chickens for two to six days post infection (120).

Although Sivanandan *et al.* (166) reported bursal necrosis and atrophy in specific-pathogen-free (SPF) chickens

experimentally infected with an IBDV-2 isolate, Ismail *et al.* (83) found that five different IBDV-2 isolates – including the isolate used by Sivanandan *et al.* (166) – caused no gross or microscopic lesions in SPF chickens and had no significant impact on the bursa-to-body-weight ratio, when compared to uninfected controls.

Although there has been serological evidence of IBDV-1 exposure in some commercial turkey flocks, this has been limited to flocks derived from parent hens vaccinated with IBDV-1 vaccines (16, 44, 84). A survey of 32 turkey flocks in England found antibodies against IBDV-2 in 29 flocks, while no turkey flocks had antibodies against IBDV-1, despite the widespread infection of chickens in England with IBDV-1 (57). Giambrone *et al.* (67) experimentally inoculated turkey poults with an IBDV-1 isolate that had been passaged through turkeys six times, in order to increase pathogenicity in this species. The resulting infections were subclinical with no morbidity, mortality, or gross lesions observed. However, microscopic changes were seen in the lymphoid organs of infected poults and similar changes were seen in non-inoculated poults housed with the experimentally infected birds. Similarly, experimental infection of turkey poults with IBDV-1 was shown to result in microscopic changes in the bursa of Fabricius and impairment of the immune system, although these changes were only partial and in no way comparable to those seen in chickens infected with IBDV-1 (140). More recently, Oladele *et al.* (132) experimentally inoculated chickens, turkeys, and ducks with IBDV-1 and found that all three species could be infected with the virus, although there was no bursal damage and minimal viral replication in ducks and turkeys. The authors concluded that the chicken host has a facilitating inherent 'factor' which permits maximal replication of IBDV, compared with turkeys and ducks.

Experimental infection of day-old turkey poults with IBDV-2 results in no clinical disease or histological changes in the bursa, spleen, or thymus, although suppression of the cellular immune system and a decrease in the plasma cell population of the Harderian gland have been described (131).

Infection with IBDV-2 is not associated with any clinical disease in turkeys or experimentally infected chickens, and there are no reports of IBDV-2 causing disease in free-living avian species. There would be little justification for applying sanitary measures for IBDV-2 to the international trade in poultry meat.

The available evidence suggests that IBDV-1 should not be considered as a hazard likely to be associated with turkey or duck meat, although trade in chicken meat should be considered a potential vehicle for the spread of IBDV-1 (120).

Turkey rhinotracheitis

Turkey rhinotracheitis (TRT) infection is transmitted to susceptible turkeys through direct contact or, experimentally, by inoculation through the intranasal or intra-tracheal routes, with nasal mucus obtained from infected birds (8, 112). Following disease introduction, the spread within a country is significantly influenced by the density of the poultry industry (87).

Histopathological studies have shown that the main sites of TRT virus (TRTV) replication in experimentally infected chickens and poults are the epithelial cells of the turbinates and the lung (116). An early study of experimentally infected, 30-week-old turkeys demonstrated virus localisation in the turbinates and trachea, while lungs, air sacs, spleen, ovary, liver, kidneys, and hypothalamus all tested negative for the presence of the virus (88). Similarly, Pedersen *et al.* (138) detected virus in the turbinates, sinus, trachea, and lungs of experimentally infected four-week-old poults, and found that turbinate tissues were significantly more productive sources of virus and viral RNA than either lung or tracheal specimens.

Cook (48) concluded that the short persistence time of TRTV in turkeys and the restricted tissue distribution of the virus help to minimise the risk of transmission through carcasses or processed products. The virus may, however, be present in respiratory tissue remnants that could persist in imported turkey carcasses after automated processing. Nonetheless, as there is no evidence of transmission other than through direct contact with infected birds, imported turkey meat should not be considered a vehicle for the spread of TRT.

Marek's disease

Marek's disease virus (MDV) replicates in feather follicle epithelial cells (34) and MDV associated with feathers and dander is infectious (18, 35, 36). Naïve poultry are infected through exposure to infectious dust or dander, either directly or via aerosols, fomites, or personnel (157). Viral shedding begins two to three weeks after infection (91) and can continue indefinitely (193).

Marek's disease virus is cell-associated in tumours and in all body organs, except in the feather follicle, where enveloped infectious virus is excreted and spread either by direct contact or by the airborne route (149). Virus could persist in the skin (in feather follicles) of poultry carcasses, although processing removes dust and dander and is likely to significantly reduce the amount of virus present on the skin surface. The virus is unlikely to be present in meat (137).

Avian infectious bronchitis

Avian infectious bronchitis is primarily a disease of chickens. It has been suggested that other avian hosts for infectious bronchitis virus (IBV) do exist, but the virus only causes disease in chickens (39).

This virus multiplies primarily in the respiratory tract. After experimental exposure to IBV in aerosols, the concentration of virus is greatest in the trachea, lungs, and air sacs, with lesser amounts recovered from the kidneys, pancreas, spleen, liver, and bursa of Fabricius (78). Nephropathic strains of IBV are also recognised (51), which may cause significant mortality without respiratory lesions (200). Strains of IBV can also replicate in many parts of the alimentary tract without associated enteric disease (10, 38, 89).

Prolonged virus excretion from infected birds has been described and both the kidneys and caecal tonsil have been suggested as locations for persistent IBV infection, although the kidney is considered to be the more likely site (53).

Infectious bronchitis virus is spread by airborne or mechanical transmission and the movement of live birds has been suggested as a potential source for the introduction of the virus (81). Ignjatovi and Sapats (81) suggested that processed poultry meat, which has undergone treatment at temperatures above 56°C for 30 to 45 minutes and is destined for human consumption, should be considered a low risk for IBV. However, given the distribution of the virus in infected birds, it would be difficult to justify imposing sanitary measures against IBV on any chicken meat products that exclude respiratory or renal tissues.

Avian infectious laryngotracheitis

The chicken is the primary natural host of infectious laryngotracheitis virus (ILTV) (69). Young turkeys can be experimentally infected with ILTV (192) but older turkeys are considered resistant (160). Natural infection of turkeys with ILTV has also recently been described (142). Reports have also described ILTV infection of pheasants (50, 79, 92).

Strains of ILTV show a great degree of variation in their virulence, with some being associated with high morbidity and mortality (106, 147, 159) while others are associated with mild-to-inapparent infections (161, 182).

Infectious laryngotracheitis virus infection of an individual can occur via the eye, nasal cavity, sinus, or respiratory tract (20). Transmission of ILTV via the oral cavity is theoretically possible, although this would require exposure of the nasal epithelium and is therefore highly inefficient (155). Following infection, viral replication is limited largely to the conjunctival and respiratory epithelium with no evidence of viraemia (14, 77).

Infectious laryngotracheitis virus can be recovered from the tracheal tissues and secretions for six to eight days after infection (14, 77, 148). It may also spread to the trigeminal ganglia (14), which are considered to be the main sites of latency for ILTV (189).

Provided poultry meat is not contaminated with respiratory tissues, the spread of ILTV through international trade should be considered unlikely (15).

Duck virus hepatitis

Duck virus hepatitis (DVH) is caused by a picornavirus with a worldwide distribution (194). Natural disease outbreaks are limited to young ducklings, although turkey poults have been successfully infected in experimental trials (194). Chickens are refractory to experimental challenge (153, 158).

The transmission of infection through aerosols and oral inoculation has been described (71, 146). Following infection, clinical signs develop rapidly and death may occur within three to four days. Morbidity can reach 100%, with 95% mortality in ducklings less than one week old. Once ducklings reach five weeks, morbidity and mortality may be low or negligible (194).

Electron microscopic studies have demonstrated structural muscle changes associated with an increase in the metabolic activity in ducklings infected with DVH, although no viral particles can be seen in the muscle tissue at any time during infection (2). Gross lesions can consistently be seen in the liver of infected birds, which may be accompanied by congestion in the spleen or kidneys. Histopathological lesions are confined to the liver (61). From the available evidence, the transmission of DVH in duck meat should be considered unlikely.

Fowl cholera

All bird species are thought to be susceptible to infection with *Pasteurella multocida*. Turkeys are considered to be more susceptible than chickens and clinical disease is most commonly associated with young mature turkeys (68). The

introduction of chronically infected carriers is thought to be the major source of infection in flocks, with the organism being found in the nasal clefts of infected birds (144, 145). The spread of *P. multocida* within a flock occurs via contaminated nasal, oral, and conjunctival excretions (68), although the organism is also (rarely) found in faeces (152).

The route of infection for *P. multocida* is via the mucous membranes of the pharynx and upper respiratory tract. Birds that are orally inoculated with virulent strains of *P. multocida* do not become infected (80).

Pasteurella multocida may become disseminated throughout the carcasses of birds that die with acute fowl cholera. The organism has been isolated from the blood of naturally infected chickens for up to 49 days before death and can remain viable for two months at 5°C to 10°C (73). However, *P. multocida* is a fairly delicate organism, which is easily inactivated by common disinfectants, sunlight, drying, or heat (46).

Christensen and Bisgaard (46) state that no country can be considered free from fowl cholera, because *P. multocida* has a broad habitat, including the mucosal surfaces of a wide range of domestic and wild birds and mammals. Further, they state that processed poultry products are not considered to present a major risk of infection transmission, due to the delicate nature of *P. multocida*. It would be difficult to justify the imposition of sanitary measures against *P. multocida* on imported poultry meat.

Pullorum disease and fowl typhoid

Chickens are the natural host for *Salmonella* Gallinarum-Pullorum, although rare outbreaks have been described in turkeys (74, 76, 82, 86, 117). Brant (31) commented that pullorum disease was a major problem as the young turkey industry grew but subsequent measures in a number of countries have virtually eliminated the disease. Pullorum disease and fowl typhoid are rare in modern commercial poultry companies, although epizootics do still occur (85, 156).

Mortality from pullorum disease usually occurs in the first two to three weeks of life, although a proportion of birds become chronic carriers (25), whereas fowl typhoid tends to cause disease in older chickens, although high mortality in young chicks has been described in the older literature (17, 19, 99, 117). Losses due to pullorum disease are reported to vary from 0% to 100%, whereas fowl typhoid is associated with losses from 10% to 93% (47, 164).

Pullorum disease and fowl typhoid are systemic infections and *S.* Gallinarum-Pullorum can be recovered from most of the internal organs of infected chickens, including the liver, spleen, caeca, lungs, heart, ventriculus, pancreas, yolk sac, synovial fluid, and reproductive organs (164). However, chicken meat contamination with *S.* Gallinarum-Pullorum has only been described in environments with poor hygiene practices (115). Poultry meat from birds that have passed ante-mortem and post-mortem inspection in slaughter and processing plants which operate effective Good Management Practice and Hazard Analysis and Critical Control Point programmes is unlikely to act as a vehicle for the spread of *S.* Gallinarum-Pullorum.

Avian mycoplasmosis (*Mycoplasma gallisepticum*)

Mycoplasma gallisepticum has a worldwide distribution (101) and naturally occurs primarily in gallinaceous birds, especially commercial chickens and turkeys (102). *Mycoplasma gallisepticum* has also been recovered from pheasants, chukar partridges, peafowl, and Japanese quail (22, 49, 127, 151), as well as from ducks (23), geese (24, 33), a yellow-naped Amazon parrot (29), greater flamingos, and white pelicans (58). In 1994, *M. gallisepticum* was recognised as the cause of peri-orbital swelling and conjunctivitis in free-ranging house finches in the United States (62, 104, 105, 109, 110). Conjunctivitis associated with *M. gallisepticum* infection was subsequently also reported in a blue jay, a purple finch, and goldfinches in the United States (72, 103), and in evening grosbeaks and pine grosbeaks in Canada (119).

Isolates and strains of *M. gallisepticum* vary widely in their relative pathogenicity (102). Low-passage strains of the R strain of *M. gallisepticum* (R_{low}) are pathogenic and capable of adhesion and cell invasion, whereas high-passage strains (R_{high}) are avirulent (118, 135). After infection, R_{low} strains can be recovered from internal organs, whereas R_{high} strains cannot (126). The upper respiratory tract and conjunctiva are generally accepted as the portals of entry for naturally acquired *M. gallisepticum* infections and the organism is considered to be a surface parasite of the respiratory tract and conjunctiva (102).

Mycoplasma gallisepticum can be cultured from suspensions of tracheal or air sac exudates, turbinates, lungs, or sinus exudate and has also been recovered from the oviduct and cloaca of infected birds (11, 54, 114, 130). More virulent strains are more likely to be recovered from a wider range of tissues, including the bursa, spleen, liver, and kidneys following experimental infection (185). *Mycoplasma gallisepticum* can be found predominantly in the respiratory

tissues, although more virulent strains may disseminate more widely (43, 181).

It is generally accepted that organisms belonging to the *Mollicutes* class are unstable and die rapidly in liquid media. However, it is also known that mycoplasmas can persist for a long period within or on animal tissues (128). Chandiramani *et al.* (40) intravenously inoculated chickens with 3×10^9 to 1.2×10^{10} *M. gallisepticum* organisms and demonstrated that the organism could be recovered from muscle tissue for up to 49 days, if stored at 6°C, and from whole carcasses for up to four weeks, when stored at temperatures ranging between 2°C and 24°C.

Horizontal transmission of *Mycoplasma* spp. occurs either through aerosol or infectious droplet transmission, resulting in localised infection of the upper respiratory tract or conjunctiva, or through venereal transmission (45, 96, 102). Fresh or frozen poultry meat products produced for human consumption are not ordinarily considered risks for *M. gallisepticum* infection (101).

Avian mycoplasmosis (*Mycoplasma synoviae*)

Chickens and turkeys are considered to be the natural hosts of *Mycoplasma synoviae* (95), although the organism has also been described in ducks (23), geese (24), guinea fowl (136), pigeons (21), Japanese quail (21), pheasants (30), and partridges (143).

Chickens usually become infected at 4 to 16 weeks old and turkeys at 10 to 24 weeks old (162), with transmission occurring via the respiratory tract. Infected birds typically develop synovitis of the tendon sheaths, joints, and keel bursa, which may progress to a caseous exudate extending from tendon sheaths and joints into muscle and air sacs (93, 95). Airsacculitis may be seen in the respiratory form of the disease (64, 154). *Mycoplasma synoviae* can be recovered from these lesions in the early stages of disease but viable organisms may no longer be present once chronic disease has developed. Birds remain carriers for life (95). Following experimental infection, *M. synoviae* can be recovered from the trachea and sinus and, although gross lesions may be seen in other organs (liver, spleen, and kidney), the organism is only consistently recovered from these sites following intravenous inoculation (66, 90).

Given the limited tissue distribution following natural infection, *M. synoviae* is unlikely to be transmitted through international trade in fresh or frozen poultry meat products produced for human consumption.

Avian chlamydiosis

There are eight known serovars of *Chlamydophila psittaci*. Highly virulent strains of *C. psittaci* cause acute disease epidemics, resulting in the deaths of 5% to 30% of affected birds, while less virulent strains cause slowly progressive epidemics. Highly virulent serovar D strains are most often isolated from turkeys (191) and are especially noted to be a risk for veterinarians and poultry workers (13).

Transmission of *C. psittaci* occurs through the inhalation of contaminated material, with large numbers of chlamydiae found in the respiratory tract exudate and faeces of infected birds (12). Page (133) was unable to transmit infection following oral inoculation of turkeys, using a *C. psittaci* dose of 340,000 mouse LD_{50}. Transmission via arthropod vectors has also been suggested (56, 134) and there is evidence for limited vertical transmission (107). *Chlamydophila psittaci* is an obligate intracellular organism that has been described as an 'energy parasite' since it depends on the host cell for adenosine triphosphate (ATP) and other high-energy metabolites (125).

Following experimental inoculation of turkeys with four strains of chlamydiae, primary replication was found to occur throughout the respiratory tract after two to seven days, with subsequent replication throughout the intestinal tract, especially in the jejunum, caecum, and colon (184). An earlier study (133) quantified the tissue distribution of *C. psittaci* in turkeys following aerosol exposure and found that the organism multiplied primarily in the lungs, air sac system, and pericardium, although infectivity was also detected in other tissues (including the kidneys), and in muscle tissue after 120 h. For diagnostic purposes, the best tissues from which to recover the organism are the air sacs, spleen, pericardium, heart, liver, and kidneys (13). Proper handling using a transport medium is necessary to prevent loss of infectivity (169).

Infectivity is concentrated in the respiratory tissues and intestinal tract but some infectivity can be detected in muscle and renal tissues. Although infection with a highly virulent strain would be likely to result in carcass condemnation, slaughterhouse inspection might be unlikely to detect birds infected with less virulent strains or in the early stages of infection.

Meat after rigor usually has a pH of between 5.4 and 5.6 because of the conversion of muscle glycogen to lactic acid. The ultimate pH of uncooked poultry meat can be expected to fall within the range of 5.7 to 6.0 (63). The optimal pH for the survival of rickettsiae is 7.0 (28), and the pH range for the growth of *C. psittaci* is limited to 6.5 to 7.5 (123). *Chlamydophila psittaci* would not, therefore, be expected to survive in the normal pH range of poultry meat. Furthermore, *C. psittaci* is an obligate

intracellular organism which depends on the host cell for ATP and other high-energy metabolites (125), and the recovery of the organism for diagnostic purposes requires proper handling using a transport medium to prevent loss of infectivity (169). Taking all these factors into account, trade in poultry meat should not be considered a vehicle for the international transmission of *C. psittaci*.

Conclusions

There are few diseases where the causative agent should be considered likely to be present in poultry meat. Highly pathogenic AI, NDV, and (for chicken meat) IBDV-1 should be considered the most significant pathogens that might reasonably be expected to be spread through international trade. The *Terrestrial Code* contains recommendations to effectively manage the risk associated with both HPAI (196) and NDV (197) in poultry meat. As yet, the OIE has been unable to formulate appropriate measures to manage the risk associated with IBDV-1 in chicken meat.

Although there is no evidence for other OIE-listed diseases being associated with poultry meat, the likelihood of carcass contamination or non-muscle tissue remnants being present in processed carcasses should be noted when considering the risks associated with such imports.

According to OIE risk analysis methodology (195), the overall risk estimation for a commodity reflects the likelihood of entry (i.e. those issues discussed in this article), together with the likelihood of exposure and the consequences of exposure. When assessing whether or not sanitary measures are justified for any disease agent likely to be associated with poultry meat, those factors specific to the importing country that might influence exposure and consequences should also be considered. For poultry meat imported for human consumption, these factors may include:

– the likelihood of raw or cooked waste being generated from the imported commodity

– the access of wild birds to this waste

– the ability of wild bird species to act as vectors

– biosecurity practices in the domestic commercial poultry industry

– the extent of backyard poultry flocks and their likely contact with commercial units

– the national presence of (or freedom from) disease in avian species

– domestic legislation on feeding food waste to poultry species. ■

La propagation des agents pathogènes au travers des échanges de viande de volaille : état de la situation et évolutions récentes

S.P. Cobb

Résumé

L'intensification du commerce international de viande de volaille ouvre de nouvelles voies au risque de dissémination mondiale des maladies aviaires. Il serait néanmoins regrettable que ce développement des échanges se traduise par un recours abusif à l'argument sanitaire pour dresser des barrières non tarifaires au commerce.

D'après les données scientifiques disponibles, les seules maladies aviaires de la liste de l'Organisation mondiale de la santé animale dont le risque de propagation à travers le commerce de ces marchandises doit être pris en compte sont l'influenza aviaire hautement pathogène, la maladie de Newcastle et la bursite infectieuse (pour ce qui concerne la viande de poulet).

Mots-clés

Accord sur l'application des mesures sanitaires et phytosanitaires – Analyse du risque à l'importation – Bursite infectieuse – Échanges internationaux – Influenza aviaire – Influenza aviaire faiblement pathogène – Influenza aviaire hautement pathogène – Maladie de Newcastle – Viande de volaille.

■

Descripción general y evolución reciente de la diseminación de patógenos por el comercio de carne aviar

S.P. Cobb

Resumen

El creciente comercio internacional de carne aviar trae consigo el riesgo de diseminación a escala planetaria de enfermedades de las aves de corral. Sin embargo, sería muy lamentable que la intensificación del comercio llevara al uso de las enfermedades animales como pretexto para imponer injustificadamente barreras no tarifarias al comercio.

Por lo que respecta a las enfermedades aviares actualmente inscritas en las listas de la Organización Mundial de Sanidad Animal, de los datos científicos hoy disponibles se desprende que sólo se debería considerar probable la propagación a resultas del comercio de este tipo de artículos de la influenza aviar, la enfermedad de Newcastle y (tratándose de carne de pollo) la bursitis infecciosa.

Palabras clave

Acuerdo sobre la Aplicación de Medidas Sanitarias y Fitosanitarias – Análisis del riesgo de importación – Bursitis infecciosa – Carne aviar – Comercio internacional – Enfermedad de Newcastle – Influenza aviar – Influenza aviar altamente patógena – Influenza aviar levemente patógena.

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