

The spread of non OIE-listed avian diseases through international trade of chicken meat: an assessment of the risks to New Zealand

This paper (No. 04112015-00069-EN) has been peer-reviewed, accepted, edited, and corrected by authors. It has not yet been formatted for printing. It will be published in December 2015 in issue 34 (3) of the *Scientific and Technical Review*

S.P. Cobb ^{(1)*} & H. Smith ⁽²⁾

(1) Ministry for Primary Industries, P.O. Box 2526, Wellington 6140, New Zealand

(2) 10 Reading Street, Greytown 5712, New Zealand

*Corresponding author: stephen.cobb@mpi.govt.nz

Summary

Twelve avian diseases are listed by the World Organisation for Animal Health (OIE), although more than 100 infectious diseases have been described in commercial poultry. This article summarises a recent assessment of the biosecurity risks posed by non-listed avian diseases associated with imports of chilled or frozen chicken meat and meat products into New Zealand. Following the guidelines described in Section 2 of the OIE *Terrestrial Animal Health Code*, avian adenovirus splenomegaly virus, avian paramyxovirus-2 (APMV-2), *Bordetella avium*, *Mycoplasma* spp., *Ureaplasma* spp., *Ornithobacterium rhinotracheale*, *Riemerella anatipestifer*, and *Salmonella arizonae* have been identified as hazards. However, of all the non-listed avian diseases discussed here, only APMV-2 and *S. arizonae* are assessed as being risks associated with the commercial import of chicken meat into New Zealand. Specific control measures may have to be implemented to mitigate such risks. This conclusion is likely to reflect both the high-health status of New Zealand poultry and the threat posed by these infectious agents to New Zealand's unique population of native psittacine species.

Keywords

Avian disease – Biosecurity – Chicken meat – Import risk analysis – International trade – New Zealand – Paramyxovirus – Poultry – *Salmonella arizonae*.

Introduction

The risk of spreading diseases listed by the World Organisation for Animal Health (OIE) through the international trade in poultry meat has been discussed previously (1). However, more than 100 infectious diseases have been associated with poultry. This article outlines the findings of a recent import risk analysis undertaken in New Zealand to assess the biosecurity risks associated with imports of chilled or frozen meat and meat products derived from chickens (*Gallus gallus*) (2).

For the purposes of this article, the commodity is defined as chilled or frozen meat and meat products derived from chickens (*G. gallus*) that have passed ante-mortem and post-mortem inspection in slaughter and processing plants which operate under Good Manufacturing Practices (GMP) and Hazard Analysis and Critical Control Point (HACCP) programmes (e.g. based on Codex Alimentarius guidelines for the control of *Campylobacter* and *Salmonella* in chicken meat, CAC/GL 78-2011).

The risk analysis methodology follows the guidelines described in the OIE *Terrestrial Animal Health Code (Terrestrial Code)* (3), as illustrated in Figure 1. The first step in this process is hazard identification. The diseases/agents of interest are those that are not known to be in New Zealand, which could be transmitted in chicken meat or meat products and could infect domestic, feral, or wild animals or humans. In this case, the preliminary hazard list was compiled from the 12th edition of *Diseases of Poultry* (2008), edited by Y.M. Saif, and published by Blackwell (4).

****Fig. 1. Here****

The authors' assessments of the risks associated with chicken pathogens determined to be exotic to New Zealand are summarised below.

Viruses

Arboviruses

Hazard identification

Arboviruses replicate in bloodsucking arthropods and are transmitted by bite to vertebrate hosts. Over 100 arboviruses have been isolated from avian species or ornithophilic vectors but only five are associated with disease in domestic poultry (4). The only way arthropod vectors can be infected is by sucking blood as they do not feed on meat and cannot be infected from it. Arboviruses are not identified as a hazard in the commodity.

Astroviruses

Hazard identification

Avian nephritis virus (ANV) is present in New Zealand (5). However, chicken astroviruses (CAstV) have been described which are serologically distinct from ANV (6). The pathogenic role of CAstV is unclear although infectivity is associated with the intestine and is unlikely to be present in imported commodities. Astroviruses are not identified as a hazard.

Avian adenovirus splenomegaly virus

Hazard identification

Avian adenovirus splenomegaly virus (AASV) of chickens is a strain of turkey adenovirus A (7). Serological evidence indicates that AASV is widespread in chickens in many countries, with as many as 50% of chickens in up to 46% of flocks affected (8, 9). In spite of this widespread seroconversion, clinically affected birds with viral inclusions are rare (10) and AASV may not be a primary pathogen (11).

The pathogenesis of AASV infection in chickens is poorly understood. The spleen is the major site of viral replication and macrophages and B lymphocytes are the primary target cells (12, 13, 14). Infected cells have also been identified in the intestine, bursa of Fabricius, caecal tonsils, thymus, liver, kidney, peripheral blood leukocytes, lung, and spleen. A short transitory viraemia accompanies the appearance of virus in these tissues (13).

However, AASV is not identified as a hazard in chicken meat or chicken meat products. Although evisceration removes the vast majority of infectivity from chicken carcasses, visceral remnants may remain following automated processing (15). AASV is identified as a hazard in imported whole chicken carcasses.

Entry assessment

The spleen is the most commonly and consistently affected organ and hence is the most infectious tissue (13, 16). Given the anatomical location of the spleen, remnants would be unlikely to remain in chicken carcasses following automated evisceration.

Viraemia occurs only at the peak of clinical disease and coincides with marked splenic pathology (splenomegaly and mottling), which is likely to be detected at slaughter. Faecal contamination during slaughter might result in limited contamination of the skin of an infected bird but, unlike bacteria of public health concern, viruses do not multiply on the carcass surface. Furthermore, the commodity should originate from slaughter and processing plants which operate effective GMP and HACCP programmes, significantly reducing the likelihood of cross-contamination.

The likelihood of AASV entry in imported whole chicken carcasses is assessed to be negligible.

Risk estimation

Since the likelihood of entry is assessed to be negligible, AASV is not assessed to be a risk.

Avian enterovirus-like viruses

Hazard identification

Enterovirus-like viruses (ELVs) have a worldwide distribution (17). Although there are no reports of ELVs in New Zealand, losses due to ‘runting and stunting’ occur periodically (5). Gross and histopathological lesions are restricted to the intestines. The jejunum and ileum are the major sites of virus localisation and replication, and viral antigen is most abundant in the enterocytes situated just above the crypt opening (18, 19, 20). The pathogenic role of ELVs is not completely understood. However, infectivity is restricted to the intestinal tract and there is no evidence for infectivity elsewhere in the carcass. ELVs are not identified as a hazard.

Avian hepatitis E virus

Hazard identification

The primary causative agent of hepatitis–splenomegaly syndrome is avian hepatitis E virus (HEV) (21, 22). Avian HEV antigen is located primarily in the liver and spleen of experimentally inoculated adult chickens (23). The liver and spleen are removed from all commodities considered in this risk analysis. There is no evidence of HEV replication in muscle tissue. HEV is not identified as a hazard.

Avian paramyxoviruses

Hazard identification

Eleven serogroups of avian paramyxovirus (APMV) are recognised; APMV-1 to APMV-11. Newcastle disease (infection with APMV-1) is an OIE-listed disease that has been previously discussed (1). Although APMV-2, APMV-4 and APMV-6 have also been associated with domestic chickens (24, 25), APMV-4 and APMV-6 are both recognised in New Zealand (26) so are not identified as hazards.

Avian paramyxovirus -2 viruses have been widely reported. The presence of APMV-2 in Israel and Italy was associated with the

importation of poultry products from North America, although subclinical infection of migratory passeriformes has also been suggested as a means of international spread (27, 28, 29).

Infection with APMV-2 leads to shedding from the respiratory and intestinal tracts (30). There is limited information concerning the epidemiology of avian paramyxoviruses other than APMV-1. Given the similarities in infection and replication between APMV-1 and other avian paramyxoviruses, it has been suggested that the same mechanisms of introduction and spread apply (31).

Replication of APMV-2 is limited to the intestinal and respiratory tracts. Although respiratory and intestinal tissues are removed from chicken carcasses, remnants of these may remain after processing. APMV-2 is identified as a hazard in imported whole chicken carcasses.

Entry assessment

Infection with APMV-2 may be associated with mild respiratory signs, so infected flocks may not be detected during routine ante- and post-mortem inspection. Infected tissues would be limited to any remnants of respiratory or intestinal tissues that remained after processing. The likelihood of entry is assessed to be very low.

Exposure assessment

The heat sensitivity of APMV-2 is likely to be similar to that of APMV-1, so scraps of cooked chicken meat would be unlikely to contain viable virus. It is assumed that any respiratory or intestinal tissue remnants in imported chicken carcasses would be discarded as raw tissue before cooking and could therefore become accessible to poultry.

The virus has been isolated from captive or free-ranging passeriformes, hanging parrots, mynahs, *Neophema* spp., lovebirds, and African grey parrots (32). Free-living avian species may become infected with APMV-2, either following exposure to an infected

backyard flock or through the consumption of uncooked kitchen waste.

In New Zealand, commercial egg producers are required to have a risk management programme (RMP) that describes how their products are processed to meet the requirements of domestic legislation. Such commercial producers should not feed food scraps to their birds, whereas non-commercial poultry flocks containing 100 or fewer birds (such as backyard flocks) are not required to have an RMP and the feeding of uncooked waste food from retail and catering outlets is recognised. At one time, a voluntary agreement was in place among feed manufacturers to prevent the feeding of poultry meat to poultry in New Zealand. However, this has now been discarded by at least one large feed manufacturer (2).

Surveys of commercial poultry farms show a high rate of compliance with biosecurity measures to prevent the introduction of exotic and endemic disease agents, especially in broiler farms (33, 34). There have been no reports describing the spread of APMV-2 infection from backyard flocks to commercial poultry.

There is a low likelihood of backyard poultry, free-living avian species, or commercial poultry being exposed to APMV-2.

Consequence assessment

Infection of chickens with APMV-2 has been associated with mild respiratory signs. Most APMV-2 infections of passeriformes are mild and self-limiting but infection of psittacines can lead to severe clinical signs, including pneumonia, mucoid tracheitis, diarrhoea and high mortality (32). The introduction of APMV-2 would be associated with non-negligible consequences to the New Zealand poultry industries and wildlife.

Risk estimation

Since the likelihood of entry, likelihood of exposure, and consequences are assessed to be non-negligible, the risk is estimated to be non-negligible and APMV-2 is assessed to be a risk.

Fowl adenoviruses

Hazard identification

Most fowl adenoviruses (FAdV) have a poorly defined role as pathogens, with the exception of strains that cause quail bronchitis and hydropericardium syndrome (HPS) (35). HPS is associated with group I, serotype 4 avian adenovirus (FAdV-4) (36, 37, 38).

Although chickens are the natural hosts of FAdV-4 and the virus may be found in many visceral organs, the risk of virus transmission through poultry meat appears to be small. Flocks infected with significant adenoviruses would show evidence of disease and accordingly would be unlikely to be slaughtered for human consumption. Additionally, adenoviruses do not multiply in carcass meat and high doses are required to induce mortality in birds older than one week of age (39, 40). Group I avian adenoviruses are not identified as a hazard.

Marek's disease virus

Hazard identification

Marek's disease virus (MDV) is common in New Zealand poultry (41, 42). However, based on clinical signs and pathology, New Zealand is considered free from the more virulent strains of MDV (2). Cell-free infectious MDV is associated with the epithelial cells of feather follicles, so virus could be present on the skin of infected chickens at slaughter. However, as imports will originate from slaughter and processing plants which operate effective GMP and HACCP programmes, this will remove dust and dander present on the skin surface. Viral inactivation occurs with storage at 4°C for two weeks (43). Chilling chicken meat during transportation to New Zealand would further reduce the amount of virus present on the skin, and so MDV is not identified as a hazard.

Multicentric histiocytosis

Hazard identification

The aetiological agent of multicentric histiocytosis (MH) has not been identified, although MH has been associated with subgroup J of the avian leukosis virus (44). Experimental studies have shown that infectivity for MH is concentrated in the spleen, which is removed from the commodities being considered here. Carcasses at slaughter presenting with hepatosplenomegaly and multiple white plaques or nodules are likely to be condemned (45). Moreover, the transmission of infection requires close contact with an infected bird due to the fragility of the virus. MH is not identified as a hazard.

Bacteria

***Aegyptianella* spp.**

Hazard identification

Aegyptianella spp. are obligate intracellular organisms. Infections are limited to erythrocytes with no infectivity being found in other body tissues. The disease is found mainly in free-range poultry and is transmitted by fowl ticks in the genus *Argas* (46). *Argas* spp. ticks are not found in New Zealand (47, 48). Even if these ticks were present, they would have to feed on an infected bird, rather than meat products, before they could transmit the disease. *Aegyptianella* spp. are not identified as a hazard.

Bordetella avium

Hazard identification

Bordetella avium is a Gram-negative, non-fermentative, motile, strictly anaerobic bacillus, previously described as *Alcaligenes faecalis* (49). Turkeys are the most common host of *B. avium*, although the organism has been recovered from other avian species (50, 51, 52). Strains of *B. avium* recovered from turkeys and chickens are similar and cross-infection can occur between these species (50).

Bordetella avium infection of chickens results in respiratory disease, with similar but less severe clinical signs and lower mortality than the same disease in turkeys (50). Following infection, *B. avium* attaches to and causes lesions in the upper respiratory tract. There is no evidence of this agent elsewhere. *B. avium* is not identified as a hazard in those commodities that exclude upper respiratory tract material. Although respiratory tract tissues are removed from carcasses, remnants of these tissues may remain after processing. *B. avium* is therefore identified as a hazard in imported whole chicken carcasses.

Entry assessment

Infection with *B. avium* may be associated with mild disease unless concomitant infections are present. It is unlikely that infected flocks would be detected during ante-mortem inspection. After infection, *B. avium* is only found in upper respiratory tract tissues and these are removed from the birds at slaughter. However, it has been previously estimated that some upper respiratory tract tissue remains in around 0.2% of processed chicken carcasses (53). The likelihood of *B. avium* entry in imported chicken carcasses is assessed to be very low.

Exposure assessment

Bordetella avium is susceptible to heat (54), so there is a negligible likelihood of *B. avium* persisting in scraps of meat after domestic cooking. However, it is assumed that some respiratory tract remnants may be discarded as raw tissue before cooking and could be accessible to backyard poultry or wild birds.

Simmons and Gray (55) demonstrated that disease could be transmitted to poults through direct or indirect contact with an infected bird. However, disease was not transmitted when nasal mucus, faeces or a suspension of triturated nasal turbinates from clinically ill poults were inoculated into susceptible poults by the nasal or oral routes. It is reasonable to conclude that ingestion of raw scraps of meat discarded from imported chicken carcasses would not transmit infection. The likelihood of exposure is assessed to be negligible.

Risk estimation

Since the likelihood of exposure is assessed to be negligible, the risk is estimated to be negligible and *B. avium* is not assessed to be a risk.

***Borrelia* spp.**

Hazard identification

Borrelia anserina causes borreliosis in a number of avian species, including chickens, although borreliosis is no longer considered to be a disease of commercial poultry farming. Natural transmission requires the presence of *Argas* spp. ticks, which act as the disease reservoir and primary vector (46). *Argas* spp. ticks are not found in New Zealand (47, 48). Even if these ticks were present, they would have to feed on an infected bird, rather than meat products, before they could transmit disease. *B. anserina* is not identified as a hazard.

***Brachyspira* spp.**

Hazard identification

Avian intestinal spirochaetosis is associated with colonisation of the large intestine by *Brachyspira* spp. Intestinal spirochetes colonise the caeca and rectum, but not the small intestine. *Brachyspira pilosicoli* has been associated with spirochetaemia in humans but this has not been reported in other species (56). Infectivity is confined to the lower intestinal tract, which is removed from the commodities under consideration. The agents of avian intestinal spirochaetosis are not identified as a hazard.

Long-segmented filamentous organisms

Hazard identification

Long-segmented filamentous organisms (LSFOs) are Gram-positive, anaerobic, spore-forming bacteria found in the ileum and jejunum of poultry (46). *Candidatus* Arthromitus has been proposed as a name for this group of organisms (57). LSFOs attach to the intestinal epithelium, embed in the apical cytoplasm of enterocytes, replace

microvilli, and produce strong stimulation of the mucosal immune system (58). The pathogenic role of LSFOs is unclear. It is likely that these organisms may not be pathogens but overgrowths associated with enteric disease (46, 59). Nevertheless, LSFOs have only been identified in the small intestine, which is removed from the commodities under consideration here. LSFOs are not identified as a hazard.

***Mycoplasma* spp. and *Ureaplasma* spp.**

Hazard identification

Mycoplasma gallinaceum, *M. gallinarum*, *M. gallisepticum*, *M. glycyphilum*, *M. iners*, *M. iowae*, *M. imitans*, *M. lipofaciens*, *M. pullorum*, *M. synoviae* and *Ureaplasma gallorale* have been isolated from chickens (60, 61). There have also been sporadic reports of 'non-avian' mycoplasmas infecting chickens, although these are isolated cases and the associated mycoplasma species are not considered to be significant pathogens of chickens (61, 62, 63, 64, 65, 66, 67). No records could be found of the recovery of *M. glycyphilum*, *M. imitans*, *M. iowae*, *M. lipofaciens*, *M. pullorum*, or *Ureaplasma* spp. from New Zealand poultry.

Unpublished pathogenicity studies have indicated that *M. glycyphilum* may cause caecal enlargement and possible stunting of chickens (68, 69). *Mycoplasma imitans* has been isolated from chickens (70) but its clinical significance has not been established (71).

As a result of eradication efforts, *M. iowae* is rarely encountered in commercial poultry (61). The natural host of *M. iowae* is the turkey (72), although it has also been found in chickens (73, 74). Experimentally, *M. iowae* can cause mortality in chicken embryos, as well as stunting, poor feathering, leg abnormalities, and airsacculitis in chickens. Few reports describe these clinical signs in chickens under field conditions (72).

Mycoplasma lipofaciens has occasionally been isolated from the respiratory tract of healthy chickens (75, 76, 77). Experimental infection has caused some chicken embryo mortality (76, 78).

Mycoplasma pullorum is frequently isolated from healthy birds, including chickens (79, 80), but is not considered to be pathogenic (81).

Ureaplasma gallorale has been recovered from healthy poultry on a number of occasions (75, 82, 83) but little is known about the spread of infection and pathogenicity of this organism (60). Experimental studies have produced mixed results. In one study, inoculation failed to produce clinical signs or macroscopic lesions (84) and, in another, inoculation produced mild upper respiratory clinical signs with serofibrinous airsacculitis and peritonitis (60).

Mycoplasma glycyphilum, *M. imitans*, *M. lipofaciens*, *M. pullorum*, and *U. gallorale* have been isolated from healthy chickens and there is little evidence that they have a pathogenic role in natural infections. When experimental infections have resulted in clinical disease, the organisms are confined to the upper respiratory tract tissues. *Mycoplasma iowae* has been isolated from the upper respiratory tract, oviduct, and hock joint of chickens.

Mycoplasma spp. and *Ureaplasma* spp. are identified as a hazard in carcasses that may contain remnants of upper respiratory tract tissue, reproductive tract tissue, or abdominal viscera.

Entry assessment

Mycoplasma infections are rarely associated with marked clinical signs unless accompanied by concurrent infections or environmental stressors. Subclinically infected birds are unlikely to be detected during ante- and post-mortem inspection.

Mycoplasma spp. and *Ureaplasma* spp. are fragile in the environment (61). They are readily killed by disinfectants and do not survive for prolonged periods outside the host (61, 85). In one study,

M. gallisepticum persisted for seven to 28 days at 4°C, seven to 14 days at room temperature, and less than seven days at 30°C and 37°C.

It is thought that mycoplasmas may be able to exist for a longer period within animal tissues (86). Chandiramani *et al.* (87) recovered *M. gallisepticum* from the muscle tissue of intravenously inoculated chickens for up to 49 days at 6°C, three days at 20°C, and less than one day at 37°C; and from whole carcasses for up to four weeks when stored under conditions varying between 2°C and 24°C. In contrast, Peters *et al.* (88) demonstrated the organism in the respiratory tract, brain, kidney, and spleen from three to 30 days following intratracheal inoculation of turkeys but were unable to isolate the organism from skeletal muscle.

Although experimental infections have been associated with widespread dissemination, *Mycoplasma* spp. and *Ureaplasma* spp. are principally localised in the respiratory and reproductive tissues after natural infection. As noted above, remnants of these tissues may remain in carcasses after automated processing. The likelihood of the entry of exotic *Mycoplasma* spp. or *Ureaplasma* spp. in imported whole chicken carcasses is assessed to be very low.

Exposure assessment

The growth range for a number of *Mycoplasma* spp. is 24°C to 42°C, with rapid inactivation above 53°C (89). There is a negligible likelihood of *Mycoplasma* spp. or *Ureaplasma* spp. persisting in scraps of chicken carcass after domestic cooking.

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 (67, 80, 85). Additionally, *M. iowae* has been shown to spread via the oral route under experimental conditions (90). The oral dose sufficient to initiate infection is not known and there are no field observations to support this as a natural pathway (72).

Fresh or frozen poultry meat products for human consumption are not ordinarily considered risks for *M. gallisepticum* infection (71). Goldberg *et al.* (91) were unable to isolate any of the mycoplasmas usually associated with clinical problems in domestic poultry from wild birds, and the infection of wild birds with *M. iowae*, with subsequent spread to poultry, has never been reported. The likelihood of exposure is assessed to be negligible.

Risk estimation

Since the likelihood of exposure is assessed to be negligible, the risk is estimated to be negligible and exotic *Mycoplasma* spp. and *Ureaplasma* spp. are not assessed as a risk.

Ornithobacterium rhinotracheale

Hazard identification

Ornithobacterium rhinotracheale is a Gram-negative, non-motile, highly pleomorphic, rod-shaped, non-sporulating bacterium. The organism is closely related to *Riemerella anatipestifer* and *Coenonia anatine* and was previously designated as a *Pasteurella*-like and *Kingella*-like organism (92). In experimental studies, infection with *O. rhinotracheale* alone is associated with minimal pathological lesions. The severity of lesions is enhanced by co-infection with other respiratory pathogens (93, 94). Nevertheless, a number of studies have shown that *O. rhinotracheale* alone is capable of causing respiratory disease in chickens (95, 96, 97, 98).

Infection with *O. rhinotracheale* is associated with a short incubation period, with seroconversion seen in four-week-old chickens within four days of experimental infection (94). Infection with *O. rhinotracheale* may be accompanied by marked clinical signs in live birds and significant post-mortem pathology, which are likely to be detected during ante-mortem and post-mortem inspection. However, the severity of clinical signs, duration of disease, and mortality rate of *O. rhinotracheale* outbreaks are extremely variable (92).

Following infection, lesions and infectivity are restricted mainly to the respiratory tissues (92). *Ornithobacterium rhinotracheale* is not identified as a hazard in those commodities that exclude respiratory tract material. Although respiratory tract tissues are removed from chicken carcasses, remnants of these tissues may remain after processing. *Ornithobacterium rhinotracheale* is identified as a hazard in imported whole chicken carcasses.

Entry assessment

The clinical signs associated with *O. rhinotracheale* infection are extremely variable (92) so it is unlikely that infected flocks would be reliably detected during ante-mortem inspection. After infection, *O. rhinotracheale* is found primarily in the respiratory tract. These tissues are removed from the birds at slaughter although it has been estimated that some upper respiratory tract tissue remains in around 0.2% of processed chicken carcasses (53). The likelihood of *O. rhinotracheale* entry in imported chicken carcasses is assessed to be non-negligible.

Exposure assessment

Ornithobacterium rhinotracheale is closely related to *Riemerella anatipestifer*, which is inactivated after one hour at 60°C (99). There is a negligible likelihood of *O. rhinotracheale* persisting in scraps of chicken meat after domestic cooking. However, it is assumed that respiratory tract remnants may be discarded as raw tissue before cooking and so be accessible to backyard poultry or wild birds.

Van Empel *et al.* (93) demonstrated that injecting *O. rhinotracheale* directly into air sacs resulted in a significant decrease in the daily weight gain of turkeys and that aerosol challenge of turkeys resulted in severe airsacculitis but no growth retardation. Sprenger *et al.* (96) reproduced clinical disease in turkeys using intra-tracheal inoculation with a pure culture of the organism and demonstrated that this route was more effective than intravenous inoculation with a pure culture.

As there is no evidence for the spread of *O. rhinotracheale* other than by the respiratory route, it is considered that ingesting scraps of chicken meat discarded from imported carcasses would not transmit infection and the likelihood of exposure is assessed to be negligible.

Risk estimation

Since the likelihood of exposure is assessed to be negligible, the risk is estimated to be negligible and *O. rhinotracheale* is not assessed to be a risk.

***Planococcus* spp.**

Hazard identification

There is one report of *Planococcus halophilus* causing an outbreak of necrotic hepatitis in chickens in Pakistan, associated with extreme environmental conditions and feed contamination. Affected birds were reported to have obvious liver lesions at necropsy (100). Because there have been no further reports of poultry infections with this organism, and the imported commodity must pass ante-mortem and post-mortem inspection in slaughter and processing plants that operate effective GMP and HACCP programmes, *P. halophilus* is not identified as a hazard.

Riemerella anatipestifer

Hazard identification

Riemerella anatipestifer is a Gram-negative, non-motile, non-spore-forming rod (101). The organism was originally named *Pfeifferella anatipestifer* (102), then *Moraxella anatipestifer* (103) and *Pasteurella anatipestifer*. Subsequent molecular investigations have placed this organism in the genus *Riemerella* (104). Infection with *R. anatipestifer* is primarily a disease of ducks and geese, although disease outbreaks have also been reported in chickens (105).

The severity of disease varies widely, depending on the strain of the organism, the infectious dose, the age of the host, and the route of

exposure (101, 106). Transmission occurs via the respiratory route or through skin wounds, although an arthropod vector (*Culex* mosquitoes) has also been suggested (107). Infection is followed by an incubation period of two to five days before clinical signs are seen, including listlessness, ocular and nasal discharge, coughing, sneezing, diarrhoea, ataxia, coma, and death, with a mortality rate of between 5% and 75% (101).

Infection with *R. anatipestifer* may be accompanied by marked clinical signs in live birds and significant post-mortem pathology. Imported chicken meat is derived from birds that have passed ante-mortem and post-mortem inspection. Although inspection is likely to detect clinically affected individuals, birds infected two to five days before slaughter or those exhibiting less marked clinical signs could go undetected. *Riemerella anatipestifer* is therefore identified as a hazard.

Entry assessment

Infection with *R. anatipestifer* may be associated with lesions that are barely noticeable on gross examination, including caseous pus between the dermis and underlying musculature. Broth cultures of *R. anatipestifer* remain viable for two to three weeks if stored at 4°C (101, 108). The likelihood of entry is assessed to be non-negligible.

Exposure assessment

Riemerella anatipestifer is susceptible to cooking temperatures (99, 109). There is a negligible likelihood of *R. anatipestifer* persisting in scraps of chicken meat after domestic cooking.

Hendrickson and Hilbert (102) found that feeding pure cultures of *R. anatipestifer* to ducklings over a ten-day period did not transmit infection and they were only able to reproduce disease using intravenous inoculation. Similarly, Asplin (110) demonstrated that infection could be readily transmitted through wounds, scratches, fissures, or punctures of the skin but was unable to infect ducks using a culture suspension of *R. anatipestifer* given orally.

Graham *et al.* (111) were able to transmit disease to young ducks when *R. anatipestifer* was administered intraperitoneally, intravenously, intra-tracheally, or (occasionally) intra-conjunctivally. However, installing *R. anatipestifer* into the crop did not result in infection.

Dougherty *et al.* (112) reported that they were able to transmit disease to ducks using intra-tracheal and intraperitoneal inoculations of suspensions of ground spleen, liver, and serosal exudates.

Hatfield and Morris (113) inoculated 16-day-old ducks with 10^9 colony-forming units (CFU) of *R. anatipestifer*, given intramuscularly, intranasally, or orally. Intramuscular challenge resulted in clinical signs and mortality in all birds within three days; intranasal challenge resulted in clinical signs (but no deaths) in two of the 12 inoculated birds, and no disease signs or deaths were observed in orally challenged ducks.

Sarver *et al.* (106) inoculated ducks with *R. anatipestifer*, using a range of challenge doses (0.5×10^2 CFU to 0.5×10^6 CFU), given via the subcutaneous, intravenous, oral, and nasal routes. Whilst inoculation via the intravenous and subcutaneous routes was associated with significant mortality at all challenge doses, there were no deaths associated with oral inoculation using a dose of either 0.5×10^2 CFU or 0.5×10^4 CFU, and only one death (from 11 inoculated individuals) recorded after oral inoculation with a dose of 0.5×10^6 CFU.

Considering the above evidence, there is a negligible likelihood of *R. anatipestifer* being transmitted to susceptible species through the ingestion of uncooked chicken meat scraps. The likelihood of exposure is assessed to be negligible.

Risk estimation

Since the exposure assessment for *R. anatipestifer* is negligible, the risk estimation is negligible and this organism is not assessed to be a risk.

***Salmonella arizonae* serovar 18Z₄Z₃₂**

Hazard identification

Avian arizonosis occurs throughout the world and has been associated with considerable losses in commercial turkey operations (114, 115). However, *Salmonella arizonae* has never been reported in animals or birds in New Zealand (53).

Salmonella arizonae is most frequently seen in turkeys, although infections of chickens have been described (116, 117). In a survey of 1,308 serotyped isolates of *S. arizonae*, 826 cultures were associated with turkeys and 87 with chickens (118).

A survey of chicken and turkey livers at slaughter found no evidence of *S. arizonae* in either of these species (119). However, Izat *et al.* (120) recovered *S. arizonae* from frozen poultry carcasses at retail in the United States. Similarly, Jiménez *et al.* (121) reported the recovery of *S. arizonae* from chicken carcasses immediately after slaughter. A survey of chickens in Brazil recovered *S. arizonae* from 3% of healthy birds and 10% of clinically sick birds (122). On the basis of available information and expert opinion, a Canadian study classified *S. arizonae* as a hazard likely to be associated with processed poultry (123).

Salmonella arizonae primarily localises in the intestinal tract of adult birds, although widespread dissemination of the organism has also been described (15). This organism has been recognised in commercial chickens. *Salmonella arizonae* is exotic to New Zealand and is therefore identified as a hazard in chicken meat.

Entry assessment

In an infected flock, shedding of *S. arizonae* can be expected to stop by the time birds reach slaughter weight. However, long-term carriers of infection are described and surveys of poultry meat have identified extremely low rates of *S. arizonae* contamination in frozen chicken carcasses at retail (120). The likelihood of entry is assessed as low.

Exposure assessment

Salmonella arizonae may be present on scraps of raw chicken meat generated during domestic processing so there is a non-negligible likelihood that backyard poultry could be exposed to this organism if fed raw meat scraps. Except for a few distinctively thermoresistant strains, salmonellae are susceptible to destruction by heat (124). There is a negligible likelihood that backyard poultry would be exposed to *S. arizonae* in scraps of imported chicken meat after cooking.

Salmonella arizonae has been recovered from a variety of avian species, and it is assumed that free-living avian species in New Zealand could be exposed to this organism either from an infected backyard flock or through consumption of uncooked chicken meat.

As described above, the feeding of uncooked waste food from retail and catering outlets is recognised, and a voluntary agreement between feed manufacturers to prevent the feeding of poultry meat to poultry in New Zealand has been discarded. Although there is a generally high rate of compliance with biosecurity measures to prevent the introduction of exotic and endemic disease agents into New Zealand poultry farms (33, 34), rodents and reptiles are common sources for introducing this organism into commercial flocks. Therefore, the likelihood of commercial poultry being exposed to *S. arizonae* is assessed to be non-negligible.

Consequence assessment

Salmonella arizonae is known to infect chickens and turkeys. Infection of chickens is not considered to be economically important although infection has been associated with considerable losses in commercial turkey operations (125). *Salmonella arizonae* has been recovered from a variety of free-living avian species with no associated clinical disease (126, 127). However, *S. arizonae* has been described as the cause of fatal hepatitis in a captive psittacine (128).

Reptiles are commonly associated with *S. arizonae* and it is considered to be part of their normal intestinal microflora in many

species (129). However, *S. arizonae* may act as an opportunistic pathogen in individuals with a depressed immune response (128). If *S. arizonae* were to become established in New Zealand, human infection could occur after exposure to reptiles, wild birds, pet birds, or poultry. *Salmonella arizonae* has been associated with a variety of disease syndromes in immunocompromised humans, including gastroenteritis, septicaemia, and localised infections (129).

The introduction of *S. arizonae* in the commodity would be associated with non-negligible consequences for the New Zealand poultry industries, wildlife, and human health. The consequences of introduction are assessed to be non-negligible.

Risk estimation

Since the likelihood of entry, likelihood of exposure, and consequences are assessed to be non-negligible, the risk is estimated to be non-negligible and *S. arizonae* is assessed to be a risk.

Parasites

Cestodes and trematodes

Hazard identification

Cestode infestation is associated with free-range rearing or backyard flocks and is rare in intensively reared poultry. The usual sites for adult cestode attachment are the duodenum, jejunum and ileum, and gravid proglottids are shed daily from adult worms in the intestinal tract. Trematodes require a mollusc intermediate host to complete their life cycle and some species also require the presence of a second intermediate host (130).

With the exception of *Collyriclum faba*, all cestodes and trematodes associated with chickens deposit their eggs in the faeces of infected birds. Immature *C. faba* migrate to the subcutaneous tissue of infected birds where they form cysts and pass eggs into the environment through an opening in the cyst wall. These then complete their life

cycle by passing through snails then dragonfly larvae. This parasite is only found in birds with access to marshy areas (131).

The intestinal tract is removed from all commodities considered here. Commercially reared chickens would be unlikely to be raised in wet marshy areas required for the persistence of *C. faba*. Cestodes and trematodes are not identified as a hazard.

***Eimeria* spp.**

Hazard identification

A wide range of coccidian parasites of chickens could be considered exotic to New Zealand. However, as the oocysts of all these parasites are deposited in the faeces of infected birds and the intestinal tract is removed from all commodities considered here, *Eimeria* spp. are not identified as a hazard (132).

***Haemoproteus* spp.**

Hazard identification

Over 128 species of the protozoan parasite *Haemoproteus* have been identified in birds, with the overwhelming majority of *Haemoproteus* spp. associated with wild bird species (133, 134). Transmission of *Haemoproteus* spp. occurs via biting dipteran flies of the families *Hippoboscidae* and *Ceratopogonidae* (134). The only way arthropod vectors can be infected with *Haemoproteus* spp. is by sucking blood, since they do not feed on meat and cannot be infected by it. Tissue-based parasites are unlikely to be transmitted in chicken meat because of their complex life cycles requiring specific hosts. *Haemoproteus* spp. are not identified as a hazard.

***Leucocytozoon* spp.**

Hazard identification

At least 67 valid species of the genus *Leucocytozoon* are recognised, 66 of which are found in birds (135). *Leucocytozoon* spp. have a two-host life cycle. Sporogony occurs in insects (*Simuliid* flies and

Culicoid midges), followed by the migration of sporozoites into the salivary glands of the insect. The only way that arthropod vectors can be infected with *Leucocytozoon* spp. is through sucking blood, as they do not feed on meat and cannot be infected from it. Tissue-based parasites are unlikely to be transmitted in chicken meat because of their complex life cycles requiring specific hosts (136). *Leucocytozoon* spp. are not identified as a hazard.

Nematodes and acanthocephalans

Hazard identification

Although a wide range of nematodes and acanthocephalans of chickens could be considered exotic to New Zealand, since the eggs of all these parasites are deposited in the faeces of infested birds, and the intestinal tract is removed from all commodities considered here, nematodes and acanthocephalans are not identified as a hazard (137, 138).

***Plasmodium* spp.**

Hazard identification

All species of *Plasmodium* are transmitted by mosquitoes. Around 65 species of *Plasmodium* have been described, although the species considered specific for domestic fowl are found mostly in Asia, Africa, and South America (134). The life cycle of *Plasmodium* spp. depends on host/mosquito interaction and the only way that arthropod vectors can be infected is by sucking blood as they do not feed on meat and cannot be infected from it. *Plasmodium* spp. are not identified as a hazard.

***Sarcocystis* spp.**

Hazard identification

Avian sarcocystosis has a worldwide distribution but is rare in domestic poultry (134). *Sarcocystis horvathi* (*S. gallinarum*) is associated with chickens (139). Sarcocysts have an obligatory two-

host life cycle (140). Modern poultry production systems prevent the occurrence of sarcocystosis as the avian intermediate host is not exposed to the oocyst-contaminated excreta of the definitive host (134). Heavy infections with sarcocysts are normally picked up during routine meat inspection and freezing reduces the viability of sarcocysts during storage and transport (141). *Sarcocystis* spp. are not identified as a hazard in the commodity.

Conclusions

The international trade in animals and animal products presents a potential risk to the importing country because of the possible presence of pathogens in the imported commodity, which might threaten the resources (human, animal, or environmental) of the importing country, although this risk should not be used as an unjustifiable barrier to trade.

Animal health import risk analysis is a tool that provides an objective and defensible method of identifying and managing the disease risks associated with the importation of animals, animal products, animal genetic material, feedstuffs, biological products, and pathological material.

Of all the non-listed avian diseases discussed above, the authors' assessment concludes that only APMV-2 and *S. arizonae* pose non-negligible risks associated with the commercial import of chicken meat into New Zealand. This conclusion may be seen partly as a reflection of the high-health status of New Zealand poultry. Furthermore, New Zealand has a number of iconic endemic psittacine species (e.g. kea, kaka, and kakapo) and several of these species are considered to be vulnerable, endangered, or critically endangered. Hence, both APMV-2 and *S. arizonae* may also pose risks to this unique population.

The OIE *Terrestrial Code* provides recommendations and principles for conducting transparent, objective, and defensible risk analyses for international trade. The Sanitary and Phytosanitary (SPS) Agreement requires World Trade Organization (WTO) Members to base their

sanitary measures on international standards, guidelines, and recommendations, where they exist. The SPS Agreement recognises the OIE as the international organisation responsible for the development and promotion of international standards, guidelines, and recommendations for animal health and zoonoses. However, WTO Members may choose to adopt measures that result in a higher level of protection than that provided by international standards, although these must be based on the outcome of an import risk analysis.

Acknowledgements

The authors would like to acknowledge all those who contributed to New Zealand's import risk analysis for chicken and duck meat; colleagues in the Ministry for Primary Industries for providing an internal review, and David E. Swayne, Nigel Horrox, and Kerry Mulqueen, who agreed to act as external expert reviewers.

References

1. Cobb S.P. (2011). – The spread of pathogens through trade in poultry meat: overview and recent developments. *In* The spread of pathogens through international trade in animals and animal products (S. MacDiarmid, ed.). *Rev. Sci. Tech. Off. Int. Epiz.*, **30** (1), 149–164. Available at: <http://web.oie.int/boutique/extrait/12cobb1149164.pdf> (accessed on 7 October 2015).
2. Ministry for Primary Industries (New Zealand) (2013). – Import risk analysis: chicken and duck meat for human consumption. Ministry for Primary Industries, Wellington, New Zealand. Available at: mpi.govt.nz/document-vault/2815 (accessed on 8 January 2015).
3. World Organisation for Animal Health (OIE) (2013). – Chapter 2.1. Import risk analysis. *In* Terrestrial Animal Health Code, 22nd Ed. OIE, Paris. Available at: www.oie.int/index.php?id=169&L=0&htmfile=chapitre_1.2.1.htm (accessed on 1 May 2014).

4. Guy J.S. & Malkinson M. (2008). – Arbovirus infections. *In* Diseases of poultry (Y.M. Saif, ed.), 12th Ed. Blackwell Publishing, Ames, Iowa, 414–425.
5. Howell J. (1992). – Viral diseases and the New Zealand poultry industry. *Surveillance (Wellington)*, **19** (2), 15–17. Available at: www.sciquest.org.nz/node/46557 (accessed on 11 October 2015).
6. Baxendale W. & Mebatsion T. (2004). – The isolation and characterisation of astroviruses from chickens. *Avian Pathol.*, **33** (3), 364–370.
7. Smyth J.A. & McNulty M.S. (2008). – *Adenoviridae*. *In* Poultry diseases (M. Pattison, P.F. McMullin, J.M. Bradbury & D.J. Alexander, eds), 6th Ed. Elsevier/Butterworth-Heinemann, Philadelphia, 367–381.
8. Domermuth C.H., Harris J.R., Gross W.B. & DuBose R.T. (1979). – A naturally occurring infection of chickens with a hemorrhagic enteritis/marble spleen disease type of virus. *Avian Dis.*, **23** (2), 479–484. doi:10.2307/1589578.
9. Domermuth C.H., Weston C.R., Cowen B.S., Colwell W.M., Gross W.B. & DuBose R.T. (1980). – Incidence and distribution of ‘avian adenovirus group II splenomegaly of chickens’. *Avian Dis.*, **24** (3), 591–594. doi:10.2307/1589794.
10. Fitzgerald S.D., Reed W.M., Langheinrich K.A., Porter A.S. & Lumbert L.A. (1994). – A retrospective immunohistochemical study of type II avian adenoviral infection in turkey, pheasant, and chicken tissues. *Avian Dis.*, **38** (1), 78–85. doi:10.2307/1591840.
11. Hess M. (2000). – Detection and differentiation of avian adenoviruses: a review. *Avian Pathol.*, **29** (3), 195–206. doi:10.1080/03079450050045440.
12. Veit H.P., Domermuth C.H. & Gross W.B. (1981). – Histopathology of avian adenovirus group II splenomegaly of chickens. *Avian Dis.*, **25** (4), 866–873. doi:10.2307/1590061.

13. Fasina S.O. & Fabricant J. (1982). – Immunofluorescence studies on the early pathogenesis of hemorrhagic enteritis virus infection in turkeys and chickens. *Avian Dis.*, **26** (1), 158–163. doi:10.2307/1590034.

14. Gómez-Villamandos J.C., Carranza J., Sierra M.A., Carrasco L., Hervás J., Blance A. & Fernández A. (1994). – Hemorrhagic enteritis by adenovirus-like particles in turkeys: a possible pathogenic mechanism. *Avian Dis.*, **38** (3), 647–652. doi:10.2307/1592093.

15. Ministry of Agriculture and Forestry (MAF) (New Zealand) (2010). – Import risk analysis: turkey meat. MAF, Biosecurity New Zealand, Wellington, New Zealand. Available at: mpi.govt.nz/document-vault/2822 (accessed on 8 January 2015).

16. Silim A. & Thorsen J. (1981). – Hemorrhagic enteritis: virus distribution and sequential development of antibody in turkeys. *Avian Dis.*, **25** (2), 444–453. doi:10.2307/1589936.

17. Guy J.S., McNulty M.S. & Hayhow C.S. (2008). – Avian enterovirus-like viruses. In *Diseases of poultry* (Y.M. Saif, ed.), 12th Ed. Blackwell Publishing, Ames, Iowa, 356–361.

18. Swayne D.E., Radin M.J. & Saif Y.M. (1990). – Enteric disease in specific-pathogen-free turkey poults inoculated with a small round turkey-origin enteric virus. *Avian Dis.*, **34** (3), 683–692. doi:10.2307/1591264.

19. Hayhow C.S. & Saif Y.M. (1993). – Experimental infection of specific-pathogen-free turkey poults with single and combined enterovirus and group A rotavirus. *Avian Dis.*, **37** (2), 546–557. doi:10.2307/1591685.

20. Hayhow C.S., Saif Y.M., Kerr K.M. & Whitmoyer R.E. (1993). – Further observations on enterovirus infection in specific-pathogen-free turkey poults. *Avian Dis.*, **37** (1), 124–134. doi:10.2307/1591465.

21. Payne C.J., Ellis T.M., Plant S.L., Gregory A.R. & Wilcox G.E. (1999). – Sequence data suggests big liver and spleen disease virus (BLSV) is genetically related to hepatitis E virus. *Vet. Microbiol.*, **68** (1–2), 119–125.

22. Haqshenas G., Shivaprasad H.L., Woolcock P.R., Read D.H. & Meng X.J. (2001). – Genetic identification and characterization of a novel virus related to human hepatitis E virus from chickens with hepatitis–splenomegaly syndrome in the United States. *J. Gen. Virol.*, **82** (10), 2449–2462.

23. Clarke J.K., Allan G.M., Bryson D.G., Williams W., Todd D., Mackie D.P. & McFerran J.B. (1990). – Big liver and spleen disease of broiler breeders. *Avian Pathol.*, **19** (1), 41–50.

24. Shortridge K.F., Alexander D.J. & Collins M.S. (1980). – Isolation and properties of viruses from poultry in Hong Kong which represent a new (sixth) distinct group of avian paramyxoviruses. *J. Gen. Virol.*, **49** (2), 255–262.

25. Alexander D.J. (1993). – Paramyxovirus infection. *In* Virus infections of birds (J.B. McFerran & M.S. McNulty, eds). Elsevier Science Publishers, Amsterdam, 321–340.

26. Stanislawek W.L., Wilks C.R., Meers J., Horner G.W., Alexander D.J., Manvell R.J., Kattenbelt J.A. & Gould A.R. (2002). – Avian paramyxoviruses and influenza viruses isolated from mallard ducks (*Anas platyrhynchos*) in New Zealand. *Arch. Virol.*, **147** (7), 1287–1302.

27. Alexander D.J. (1980). – Avian paramyxoviruses. *Vet. Bull.*, **50** (9), 737–752.

28. Zhang G.Z., Zhao J.X., Wang H.W., Yang A.M., Bu C.Y. & Wang M. (2006). – Isolation, identification, and comparison of four isolates of avian paramyxovirus serotype 2 in China. *Avian Dis.*, **50** (3), 386–390.

29. Zhang G.Z., Zhao J.X. & Wang M. (2007). – Serological survey on prevalence of antibodies to avian paramyxovirus serotype 2 in China. *Avian Dis.*, **51** (1), 137–139.

30. Alexander D.J. & Senne D.A. (2008). – Newcastle disease, other avian paramyxoviruses, and pneumovirus infections. *In Diseases of poultry* (Y.M. Saif, ed.), 12th Ed. Blackwell Publishing, Ames, Iowa, 75–115.

31. Alexander D.J. (2000). – Newcastle disease and other avian paramyxoviruses. *In Diseases of poultry: world trade and public health implications* (C.W. Beard & M.S. McNulty, eds.). *Rev. Sci. Tech. Off. Int. Epiz.*, **19** (2), 443–462. Available at: www.oie.int/doc/ged/D9310.PDF (accessed on 7 October 2015).

32. Ritchie B.W. (1995). – *Paramyxoviridae*. *In Avian viruses: function and control* (B.W. Ritchie & K. Carter, eds). Wingers Publishing, Inc., Lake Worth, Florida, 253–283.

33. Rawdon T., Thornton R., McKenzie J. & Gerber N. (2007). – Biosecurity risk pathways in New Zealand's commercial poultry industry. *Surveillance*, **34** (3), 4–9. Available at: www.sciquest.org.nz/node/47506 (accessed on 11 October 2015).

34. Rawdon T., Tana T., Frazer J., Thornton R. & Chrystal N. (2008). – Biosecurity risk pathways in the commercial poultry industry: free-range layers, pullet-rearers and turkey broilers. *Surveillance (Wellington)*, **35** (4), 4–9. Available at: www.sciquest.org.nz/node/47565 (accessed on 12 October 2015).

35. Adair B.M. & Fitzgerald S.D. (2008). – Group I adenovirus infections. *In Diseases of poultry* (Y.M. Saif, ed.), 12th Ed. Blackwell Publishing, Ames, Iowa, 252–266.

36. Hess M., Raue R. & Prusas C. (1999). – Epidemiological studies on fowl adenoviruses isolated from cases of infectious hydropericardium. *Avian Pathol.*, **28** (5), 433–439. doi:10.1080/03079459994443.

37. Ganesh K. & Raghavan R. (2000). – Hydropericardium hepatitis syndrome of broiler poultry: current status of research. *Res. Vet. Sci.*, **68** (3), 201–206.

38. Schonewille E., Singh A., Gobel T.W., Gerner W., Saalmuller A. & Hess M. (2008). – Fowl adenovirus (FAdV) serotype 4 causes depletion of B and T cells in lymphoid organs in specific pathogen-free chickens following experimental infection. *Vet. Immunol. Immunopathol.*, **121** (1–2), 130–139.

39. McFerran J.B. & Smyth J.A. (2000). – Avian adenoviruses. *In Diseases of poultry: world trade and public health implications* (C.W. Beard & M.S. McNulty, eds.). *Rev. Sci. Tech. Off. Int. Epiz.*, **19** (2), 589–601. Available at: www.oie.int/doc/ged/D9436.PDF (accessed on 7 October 2015).

40. Mazaheri A., Prusas C., Voss M. & Hess M. (2003). – Vertical transmission of fowl adenovirus serotype 4 investigated in specified pathogen-free birds after experimental infection. *Arch. Geflügelkde*, **67** (1), 6–10.

41. McCausland I.P. (1972). – Diseases of the domestic fowl in northern New Zealand. *N.Z. Vet. J.*, **20** (9), 160–166.

42. Horner G.W. & James M.P. (1975). – Isolation of Marek's disease virus from affected chickens. *N.Z. Vet. J.*, **23** (6), 102–104.

43. Calnek B.W. & Adldinger H.K. (1971). – Some characteristics of cell-free preparations of Marek's disease virus. *Avian Dis.*, **15** (3), 508–517. doi:10.2307/1588727.

44. Hafner S. & Goodwin M.A. (2008). – Multicentric histiocytosis. *In Diseases of poultry* (Y.M. Saif, ed.), 12th Ed. Blackwell Publishing, Ames, Iowa, 591–593.

45. Takami S., Goryo M., Masegi T. & Okada K. (2005). – Systemic spindle-cell proliferative disease in broiler chickens. *J. Vet. Med. Sci.*, **67** (1), 13–18.

46. Barnes H.J. & Nolan L.K. (2008). – Other bacterial diseases. *In* Diseases of poultry (Y.M. Saif, ed.), 12th Ed. Blackwell Publishing, Ames, Iowa, 952–970.

47. McKenna P.B. (1996). – The tick fauna of New Zealand. *Surveillance (Wellington)*, **23** (4), 27. Available at: www.sciquest.org.nz/node/46963 (accessed on 11 October 2015).

48. Loth L. (2005). – Review of exotic tick interceptions in New Zealand since 1980. *Surveillance (Wellington)*, **32** (3), 7–9.

49. Jackwood M.W. & Saif Y.M. (2008). – Bordetellosis (turkey coryza). *In* Diseases of poultry (Y.M. Saif, ed.), 12th Ed. Blackwell Publishing, Ames, Iowa, 774–788.

50. Simmons D.G., Davis D.E., Rose L.P., Gray J.G. & Luginbuhl G.H. (1981). – *Alcaligenes faecalis*-associated respiratory disease of chickens. *Avian Dis.*, **25** (3), 610–613. doi:10.2307/1589991.

51. Hinz K.H., Glünder G. & Römer K.J. (1983). – A comparative study of avian *Bordetella*-like strains, *Bordetella bronchiseptica*, *Alcaligenes faecalis* and other related nonfermentable bacteria. *Avian Pathol.*, **12** (2), 263–276.

52. Raffel T.R., Register K.B., Marks S.A. & Temple L. (2002). – Prevalence of *Bordetella avium* infection in selected wild and domesticated birds in the eastern USA. *J. Wildl. Dis.*, **38** (1), 40–46.

53. Ministry of Agriculture and Forestry (MAF) (New Zealand) (1999). – Import risk analysis: chicken meat and chicken meat products; Bernard Matthews Foods Ltd turkey meat preparations from the United Kingdom. MAF Regulatory Authority, Wellington, New Zealand. Available at: mpi.govt.nz/document-vault/2816 (accessed on 8 January 2015).

54. Arp L.H. & McDonald S.M. (1985). – Influence of temperature on the growth of *Bordetella avium* in turkeys and *in vitro*. *Avian Dis.*, **29** (4), 1066–1077. doi:10.2307/1590461.

55. Simmons D.G. & Gray J.G. (1979). – Transmission of acute respiratory disease (rhinotracheitis) of turkeys. *Avian Dis.*, **23** (1), 132–138. doi:10.2307/1589680.

56. Hampson D.J. & Swayne D.E. (2008). – Avian intestinal spirochaetosis. *In Diseases of poultry* (Y.M. Saif, ed.), 12th Ed. Blackwell Publishing, Ames, Iowa, 922–940.

57. Snel J., Heinen P.P., Blok H.J., Carman R.J., Duncan A.J., Allen P.C. & Collins M.D. (1995). – Comparison of 16S rRNA sequences of segmented filamentous bacteria isolated from mice, rats, and chickens and proposal of ‘*Candidatus Arthromitus*’. *Int. J. Syst. Bacteriol.*, **45** (4), 780–782.

58. Yamauchi K.E. & Snel J. (2000). – Transmission electron microscopic demonstration of phagocytosis and intracellular processing of segmented filamentous bacteria by intestinal epithelial cells of the chick ileum. *Infect. Immun.*, **68** (11), 6496–6504.

59. Goodwin M.A., Cooper G.L., Brown J., Bickford A.A., Waltman W.D. & Dickson T.G. (1991). – Clinical, pathological, and epizootiological features of long-segmented filamentous organisms (bacteria, LSFOs) in the small intestines of chickens, turkeys, and quails. *Avian Dis.*, **35** (4), 872–876. doi:10.2307/1591623.

60. Stipkovits L. & Kempf I. (1996). – Mycoplasmoses in poultry. *Rev. Sci. Tech. Off. Int. Epiz.*, **15** (4), 1495–1525. Available at: www.oie.int/doc/ged/D9107.PDF (accessed on 7 October 2015).

61. Bradbury J.M. & Morrow C. (2008). – Avian mycoplasmas. *In Poultry diseases* (M. Pattison, P.F. McMullin, J.M. Bradbury & D.J. Alexander, eds), 6th Ed. Elsevier/Butterworth-Heinemann, Philadelphia, 220–234.

62. Ongor H., Kalin R., Karahan M., Cetinkaya B., McAuliffe L. & Nicholas R.A.J. (2008). – Isolation of *Mycoplasma bovis* from broiler chickens in Turkey. *Avian Pathol.*, **37** (6), 587–588.

63. Khiari A.B., Landoulsi A., Aissa H., Mlik B., Amouna F., Ejlassi A. & Mardassi B.B. (2011). – Isolation of *Mycoplasma meleagridis* from chickens. *Avian Dis.*, **55** (1), 8–12. doi:10.1637/9621-936511-DIGEST.1.

64. Adler H.E. (1958). – A PPLO slide agglutination test for the detection of infectious sinusitis of turkeys. *Poult. Sci.*, **37** (5), 1116–1123. doi:10.3382/ps.0371116.

65. Yamamoto R. & Bigland C.H. (1964). – Pathogenicity to chicks of *Mycoplasma* associated with turkey airsacculitis. *Avian Dis.*, **8** (4), 523–531. doi:10.2307/1587939.

66. Yamamoto R., Bigland C.H. & Ortmayer H.B. (1965). – Characteristics of *Mycoplasma meleagridis* sp. n., isolated from turkeys. *J. Bacteriol.*, **90** (1), 47–49.

67. Chin R.P., Yan Ghazikhanian G. & Kempf I. (2008). – *Mycoplasma meleagridis* infection. In *Diseases of poultry* (Y.M. Saif, ed.), 12th Ed. Blackwell Publishing, Ames, Iowa, 834–845.

68. Forrest M. & Bradbury J.M. (1984). – *Mycoplasma glycyphilum*, a new species of avian origin. *J. Gen. Microbiol.*, **130** (3), 597–603.

69. Loria G.R., Ferrantelli E., Giardina G., Vecchi L.L., Sparacine L., Oliveri F., McAuliffe L. & Nicholas R.A.J. (2008). – Isolation and characterization of unusual *Mycoplasma* spp. from captive Eurasian Griffon (*Gyps fulvus*) in Sicily. *J. Wildl. Dis.*, **44** (1), 159–163.

70. Ganapathy K. & Bradbury J.M. (1999). – Pathogenicity of *Mycoplasma imitans* in mixed infection with infectious bronchitis virus in chickens. *Avian Pathol.*, **28** (3), 299–237.

71. Levisohn S. & Kleven S.H. (2000). – Avian mycoplasmosis (*Mycoplasma gallisepticum*). In *Diseases of poultry: world trade and public health implications* (C.W. Beard & M.S. McNulty, eds.). *Rev.*

Sci. Tech. Off. Int. Epiz., **19** (2), 425–442. Available at: www.oie.int/doc/ged/D9309.PDF (accessed on 7 October 2015).

72. Bradbury J.M. & Kleven S.H. (2008). – *Mycoplasma iowae* infection. In Diseases of poultry (Y.M. Saif, ed.), 12th Ed. Blackwell Publishing, Ames, Iowa, 856–862.

73. Yoder Jr H.W. & Hofstad M.S. (1962). – A previously unreported serotype of avian mycoplasma. *Avian Dis.*, **6** (2), 147–160. doi:10.2307/1587854.

74. Benčina D., Mrzel I., Tadina T. & Dorrer D. (1987). – *Mycoplasma* species in chicken flocks with different management systems. *Avian Pathol.*, **16** (4), 599–608.

75. Priante E.S., Flores C.L. & Muñiz A.O. (2011). – First isolation and identification of *Ureaplasma* spp and *Mycoplasma lipofaciens* in commercial hens in Mexico. *Rev. Mex. Cienc. Pecu.*, **2** (1), 85–92.

76. Bradbury J.M., Forrest M. & Williams A. (1983). – *Mycoplasma lipofaciens*, a new species of avian origin. *Int. J. Syst. Bacteriol.*, **33** (2), 329–335.

77. Benčina D., Dorrer D. & Tadina T. (1987). – *Mycoplasma* species isolated from six avian species. *Avian Pathol.*, **16** (4), 653–664.

78. Lierz M. (2009). – Role of unusual mycoplasmas in poultry – *M. lipofaciens* as an example. In Proc. VI International Symposium on Avian Corona- and Pneumoviruses and complicating pathogens, Rauschholzhausen, Germany, 14–17 June 2009. VVB Laufersweiler Verlag, Giessen, Germany, 371–375.

79. Lo Y.T., Lan Y.C., Chern R.S. & Lin M.Y. (1994). – Isolation and identification of *Mycoplasma* spp. in ducks in Taiwan. *Taiwan J. Vet. Med. Anim. Husb.*, **63** (1), 60–64.

80. Kleven S.H. & Ferguson-Noel N. (2008). – Other mycoplasmal infections. *In Diseases of poultry* (Y.M. Saif, ed.), 12th Ed. Blackwell Publishing, Ames, Iowa, 862–864.

81. Poveda J.B., Carranza J., Miranda A., Garrido A., Hermoso M., Fernandez A. & Domenech J. (1990). – An epizootiological study of avian mycoplasma in southern Spain. *Avian Pathol.*, **19** (4), 627–633.

82. Harasawa R., Koshimizu K., Pan I.-J. & Barile M.F. (1985). – Genomic and phenotypic analyses of avian ureaplasma strains. *Jpn. J. Vet. Sci.*, **47** (6), 901–909. doi:10.1292/jvms1939.47.901.

83. Koshimizu K., Harasawa R., Pan I.J., Kotani H., Ogata M., Stephens E.B. & Barile M.F. (1987). – *Ureaplasma-Gallorale* sp. nov. from the oropharynx of chickens. *Int. J. Syst. Bacteriol.*, **37** (4), 333–338.

84. Koshimizu K., Kotani H., Magaribuchi T., Yagihashi T., Shibata K. & Ogata M. (1982). – Isolation of ureaplasmas from poultry and experimental infection in chickens. *Vet. Rec.*, **110** (18), 426–429.

85. Ley D.H. (2008). – *Mycoplasma gallisepticum* infection. *In Diseases of poultry* (Y.M. Saif, ed.), 12th Ed. Blackwell Publishing, Ames, Iowa, 807–834.

86. Nagatomo H., Takegahara Y., Sonoda T., Yamaguchi A., Uemura R., Hagiwara S. & Sueyoshi M. (2001). – Comparative studies of the persistence of animal mycoplasmas under different environmental conditions. *Vet. Microbiol.*, **82** (3), 223–232.

87. Chandiramani N.K., Van Roekel H. & Olesiuk O.M. (1966). – Viability studies with *Mycoplasma gallisepticum* under different environmental conditions. *Poult. Sci.*, **45** (5), 1029–1044.

88. Peters R.L., Faber J.E., Keenum W.D. & Devolt H.M. (1966). – Distribution of *Mycoplasma gallisepticum* in selected tissues and organs of artificially infected turkeys. *Poult. Sci.*, **45** (5), 913–923.

89. Mitscherlich E. & Marth E.H. (1984). – Microbial survival in the environment. Bacteria and Rickettsiae important in human and animal health. Springer-Verlag, Berlin, Heidelberg. doi:10.1007/978-3-642-69974-0.

90. Shah-Majid M. & Rosendal S. (1987). – Oral challenge of turkey poults with *Mycoplasma iowae*. *Avian Dis.*, **31** (2), 365–369. doi:10.2307/1590887.

91. Goldberg D.R., Samuel M.D., Thomas C.B., Sharp P., Krapu G.L., Robb J.R., Kenow K.P., Korschgen C.E., Chipley W.H., Conroy M.J. & Kleven S.H. (1995). – The occurrence of mycoplasmas in selected wild North American waterfowl. *J. Wildl. Dis.*, **31** (3), 364–371.

92. Chin R.P., van Empel C.M. & Hafez H.M. (2008). – *Ornithobacterium rhinotracheale* infection. In Diseases of poultry (Y.M. Saif, ed.), 12th Ed. Blackwell Publishing, Ames, Iowa, 765–774.

93. Van Empel P., van den Bosch H., Goovaerts D. & Storm P. (1996). – Experimental infection in turkeys and chickens with *Ornithobacterium rhinotracheale*. *Avian Dis.*, **40** (4), 858–864. doi:10.2307/1592310.

94. Van Empel P., Vrijenhoek M., Goovaerts D. & van den Bosch H. (1999). – Immunohistochemical and serological investigation of experimental *Ornithobacterium rhinotracheale* infection in chickens. *Avian Pathol.*, **28** (2), 187–193.

95. Travers A.F., Coetzee L. & Gummow B. (1996). – Pathogenicity differences between South African isolates of *Ornithobacterium rhinotracheale*. *Onderstepoort J. Vet. Res.*, **63** (3), 197–207.

96. Sprenger S.J., Back A., Shaw D.P., Nagaraja K.V., Roepke D.C. & Halvorson D.A. (1998). – *Ornithobacterium rhinotracheale* infection in turkeys: experimental reproduction of the disease. *Avian Dis.*, **42** (1), 154–161. doi:10.2307/1592588.

97. Van Veen L., van Empel P. & Fabri T. (2000). – *Ornithobacterium rhinotracheale*, a primary pathogen in broilers. *Avian Dis.*, **44** (4), 896–900. doi:10.2307/1593063.

98. Odor E.M., Salem M., Pope C.R., Sample B., Primm M., Vance K. & Murphy M. (1997). – Isolation and identification of *Ornithobacterium rhinotracheale* from commercial broiler flocks on the Delmarva Peninsula. *Avian Dis.*, **41** (1), 257–260. doi:10.2307/1592468.

99. Harry E.G. & Deb J.R. (1979). – Laboratory and field trials on a formalin inactivated vaccine for the control of *Pasteurella anatipestifer* septicaemia in ducks. *Res. Vet. Sci.*, **27** (3), 329–333.

100. Abdel-Gabbar K.M.A., Dewani P., Junejo B.M. & Gabbar K.M.A.A. (1995). – Possible involvement of *Planococcus halophilus* in an outbreak of necrotic hepatitis in chickens. *Vet. Rec.*, **136** (3), 74.

101. Sandhu T.S. (2008). – *Riemerella anatipestifer* infection. In *Diseases of poultry* (Y.M. Saif, ed.), 12th Ed. Blackwell Publishing, Ames, Iowa, 758–764.

102. Hendrickson J.M. & Hilbert K.F. (1932). – A new and serious septicaemic disease of young ducks with a description of the causative organism, *Pfeifferella anatipestifer*. *Cornell Vet.*, **22**, 239–252.

103. Bruner D.W. & Fabricant J. (1954). – A strain of *Moraxella anatipestifer* (*Pfeifferella anatipestifer*) isolated from ducks. *Cornell Vet.*, **44** (4), 461–464.

104. Segers P., Mannheim W., Vancanneyt M., De Brandt K., Hinz K.H., Kersters K. & Vandamme P. (1993). – *Riemerella anatipestifer* gen. nov., comb. nov., the causative agent of septicemia anserum exsudativa, and its phylogenetic affiliation within the Flavobacterium-Cytophaga rRNA homology group. *Int. J. Syst. Bacteriol.*, **43** (4), 768–776.

105. Rosenfeld L.E. (1973). – *Pasteurella anatipestifer* infection in fowls in Australia. *Aust. Vet. J.*, **49** (1), 55–56.

106. Sarver C.F., Morishita T.Y. & Neressian B. (2005). – The effect of route of inoculation and challenge dosage on *Riemerella anatipestifer* infection in Pekin ducks (*Anas platyrhynchos*). *Avian Dis.*, **48** (1), 104–107.

107. Cooper G.L. (1989). – *Pasteurella anatipestifer* infections in California turkey flocks: circumstantial evidence of a mosquito vector. *Avian Dis.*, **33** (4), 809–815. doi:10.2307/1591165.

108. Bangun A., Tripathy D.N. & Hanson L.E. (1981). – Studies of *Pasteurella anatipestifer*: an approach to its classification. *Avian Dis.*, **25** (2), 326–337. doi:10.2307/1589926.

109. Ministry of Agriculture and Forestry (MAF) (New Zealand) (2006). – Import risk analysis: cooked duck meat from Australia. MAF Biosecurity New Zealand, Wellington, New Zealand. Available at: mpi.govt.nz/document-vault/2821 (accessed on 8 January 2015).

110. Asplin F.D. (1956). – Experiments on the transmission of septicaemic disease of ducklings. *Vet. Rec.*, **68** (9), 588–590.

111. Graham R., Brandly C.A. & Dunlop G.L. (1938). – Studies on duck septicemia. *Cornell Vet.*, **28**, 1–8.

112. Dougherty E., Saunders L.Z. & Parsons E.H. (1955). – The pathology of infectious serositis of ducks. *Am. J. Pathol.*, **31** (3), 475–487.

113. Hatfield R.M. & Morris B.A. (1988). – Influence of the route of infection of *Pasteurella anatipestifer* on the clinical and immune responses of White Pekin ducks. *Res. Vet. Sci.*, **44** (2), 208–214.

114. Mayeda B., McCapes R.H. & Scott W.F. (1978). – Protection of day-old poult against *Arizona hinshawii* challenge by

preincubation streptomycin egg treatment. *Avian Dis.*, **22** (1), 61–70. doi:10.2307/1588721.

115. Crespo R., Jeffrey J.S., Chin R.P., Senties-Cué G. & Shivaprasad H.L. (2004). – Genotypic and phenotypic characterization of *Salmonella arizonae* from an integrated turkey operation. *Avian Dis.*, **48** (2), 344–350.

116. Edwards P.R., West M.G. & Bruner D.W. (1947). – Arizona group of paracolon bacteria. *Kentucky Agricult. Experim. Station Bull.*, **499** (1), 3–32.

117. Silva E.N., Hipolito O. & Grecchi R. (1980). – Natural and experimental *Salmonella arizonae* 18:z4,z32 (Ar. 7:1,7,8) infection in broilers. Bacteriological and histopathological survey of eye and brain lesions. *Avian Dis.*, **24** (3), 631–636. doi:10.2307/1589799.

118. Edwards P.R., McWhorter A.C. & Fife M.A. (1956). – The Arizona group of Enterobacteriaceae in animals and man. *Bull. WHO*, **14** (3), 511–528.

119. Sadler W.W., Yamamoto R. & Corstvet R.E. (1965). – Biological survey of market poultry livers. *Poult. Sci.*, **44** (4), 993–998.

120. Izat A.L., Kopek J.M. & McGinnis J.D. (1991). – Research note: incidence, number, and serotypes of *Salmonella* on frozen broiler chickens at retail. *Poult. Sci.*, **70** (6), 1438–1440.

121. Jiménez S.M., Tiburzi M.C., Salsi M.S., Pirovani M.E. & Moguilevsky M.A. (2003). – The role of visible faecal material as a vehicle for generic *Escherichia coli*, coliform, and other enterobacteria contaminating poultry carcasses during slaughter. *J. Appl. Microbiol.*, **95** (3), 451–456.

122. de Avila F.A. & Moreira E.C. (1976). – Isolamento e identificação de germes do grupo Arizona de aves doentes e aparentemente sadias em Belo Horizonte, Brazil [Isolation and

identification of the Arizona organism from sick and healthy chickens in Belo Horizonte, Brazil]. Arquivos da Escola de Veterinária da Universidade Federal de Minas Gerais, **28** (3), 189–191.

123. Bisailon J.-R., Feltmate T.E., Sheffield S., Julian R., Todd E., Poppe C. & Quessy S. (2001). – Classification of grossly detectable abnormalities and conditions seen at post-mortem in Canadian poultry abattoirs according to a hazard identification decision tree. *J. Food Protec.*, **64** (12), 1973–1980.

124. Gast R.K. (2008). – Paratyphoid infections. *In Diseases of poultry* (Y.M. Saif, ed.), 12th Ed. Blackwell Publishing, Ames, Iowa, 636–665.

125. Shivaprasad H.L. (2008). – Arizonosis. *In Diseases of poultry* (Y.M. Saif, ed.), 12th Ed. Blackwell Publishing, Ames, Iowa, 665–674.

126. Windingstad R.M., Trainer D.O. & Duncan R. (1977). – *Salmonella enteritidis* and *Arizona hinshawii* isolated from wild sandhill cranes. *Avian Dis.*, **21** (4), 704–707. doi:10.2307/1589429.

127. Winsor D.K., Bloebaum A.P. & Mathewson J.J. (1981). – Gram-negative aerobic, enteric pathogens among intestinal microflora of wild turkey vultures (*Cathartes aura*) in West Central Texas. *Appl. Environ. Microbiol.*, **42** (6), 1123–1124.

128. Orós J., Rodríguez J.L., Fernández A., Herráez P., Espinosa de los Monteros A. & Jacobson E.R. (1998). – Simultaneous occurrence of *Salmonella arizonae* in a sulfur crested cockatoo (*Cacatua galerita galerita*) and iguanas. *Avian Dis.*, **42** (4), 818–823. doi:10.2307/1592723.

129. Guckian J.E., Byers E.H. & Perry J.E. (1967). – Arizona infection of man. *Arch. Internal Med.*, **119** (2), 170–175.

130. McDougald L.R. (2008). – Cestodes and trematodes. *In Diseases of poultry* (Y.M. Saif, ed.), 12th Ed. Blackwell Publishing, Ames, Iowa, 1056–1066.

131. Taylor M.A., Coop R.L. & Wall R.L. (2007). – Parasites of poultry and gamebirds. *In* Veterinary parasitology, 3rd Ed. Blackwell Publishing, Ames, Iowa, 459–557.

132. McDougald L.R. & Fitz-Coy S.H. (2008). – Coccidiosis. *In* Diseases of poultry (Y.M. Saif, ed.), 12th Ed. Blackwell Publishing, Ames, Iowa, 1068–1085.

133. Levine N.D. & Campbell G.R. (1971). – A check-list of the species of the genus *Haemoproteus* (Apicomplexa, Plasmodiidae). *J. Protozool.*, **18** (3), 475–484.

134. Bermudez A.J. (2008). – Miscellaneous and sporadic protozoal infections. *In* Diseases of poultry (Y.M. Saif, ed.), 12th Ed. Blackwell Publishing, Ames, Iowa, 1105–1117.

135. Hsu C.K., Campbell G.R. & Levine N.D. (1973). – A checklist of the species of the genus *Leucocytozoon*. *J. Protozool.*, **20** (2), 195–203.

136. Biosecurity Australia (2008). – Generic import risk analysis report for chicken meat, final report. Biosecurity Australia, Canberra, Australia. Available at: www.agriculture.gov.au/SiteCollectionDocuments/ba/memos/2008/2008_33c.pdf (accessed on 8 January 2015).

137. Yazwinski T.A. & Tucker C.A. (2008). – Nematodes and acanthocephalans. *In* Diseases of poultry (Y.M. Saif, ed.), 12th Ed. Blackwell Publishing, Ames, Iowa, 1025–1056.

138. McDougald L.R. (2008). – Internal parasites. Introduction. *In* Diseases of poultry (Y.M. Saif, ed.), 12th Ed. Blackwell Publishing, Ames, Iowa, 1025.

139. Levine N.D. (1986). – The taxonomy of *Sarcocystis* (Protozoa, Apicomplexa) species. *J. Parasitol.*, **72** (3), 372–382.

140. Odening K. (1998). – The present state of species-systematics in *Sarcocystis* Lankester, 1882 (Protista, Sporozoa,

Coccidia). *Syst. Parasitol.*, **41** (3), 209–233.
doi:10.1023/A:1006090232343.

141. MacDiarmid S.C. (1991). – The importation into New Zealand of meat and meat products: a review of the risks to animal health. Ministry of Agriculture and Forestry, Wellington, New Zealand. Available at: mpi.govt.nz/document-vault/2811 (accessed on 8 January 2015).

Fig. 1
Overview of import risk analysis process

