Ministry for Primary Industries
Manatū Ahu Matua

Rapid Risk Assessment: Pasteurised eggs: Addendum to 2008 Import Risk Analysis

Prepared for the Ministry for Primary Industries

ISBN No: 978-1-77665-508-3 (online)

November 2015

New Zealand Government

Growing and Protecting New Zealand

Disclaimer

While every effort has been made to ensure the information in this publication is accurate, the Ministry for Primary Industries does not accept any responsibility or liability for error of fact, omission, interpretation or opinion that may be present, nor for the consequences of any decisions based on this information.

Requests for further copies should be directed to:

Publications Logistics Officer
Ministry for Primary Industries
PO Box 2526
WELLINGTON 6140

Email: brand@mpi.govt.nz
Telephone: 0800 00 83 33
Facsimile: 04-894 0300

This publication is also available on the Ministry for Primary Industries website at
http://www.mpi.govt.nz/news-and-resources/publications/

Rapid Risk Assessment: Pasteurised eggs: Addendum to 2008 Import Risk Analysis

November 2015

This page is intentionally blank

Ministry for Primary Industries
Pastoral House
25 The Terrace
PO Box 2526
Wellington 6011
New Zealand

Tel: 64 4 894 0100
Fax: 64 4 894 0731

Biosecurity Science, Food Science and Risk Assessment,
Ministry for Primary Industries

Ministry for Primary Industries
Manatū Ahu Matua

Rapid Risk Assessment: Pasteurised eggs: Addendum to 2008 Import Risk Analysis

November 2015

Approved for general release

Christine Reed
Biosecurity Science, Food Science and Risk Assessment,
Ministry for Primary Industries

This page is intentionally blank

Contents

Contributors to this risk analysis

Authors

Stephen Cobb	Manager (Animals and Aquatics), Biosecurity Science, Food Science and Risk Assessment	MPI Wellington
Abraham Growcott	Adviser (Animals and Aquatics), Biosecurity Science, Food Science and Risk Assessment	MPI Wellington

Internal peer review

Lucy Johnston	Senior Adviser, Animal Imports Team Animal & Animal Products Directorate	MPI Wellington

1. Executive Summary

A rapid risk assessment was requested to identify additional biosecurity risks that may be associated with pasteurized egg products imported under the import health standards *Pasalbic.aus* and *Poueggic.aus* that have less stringent processing conditions than the commodity definition used in the 2008 import risk analysis for egg powders.

No additional hazards were identified for pasteurized eggs which are subject to the processing conditions specified in *Poueggic.aus*.

Although the less stringent conditions specified in *Poualbic.aus* for pasteurised egg white from Australia may not be sufficient to inactivate *Escherichia coli* or infectious bronchitis virus, further assessment did not identify either of these to be hazards in the commodity.

2. Introduction

The import risk analysis (IRA) for egg powder (MAF 2008a) examined the risks associated with imported egg powders that have been subject to 60°C for a period of no less than 3.5 minutes (egg powders and egg yolk powders) or a final heat treatment of greater than 54.4°C for no less than 7 days (egg albumen powders). On the basis of this commodity definition, exotic group 1 avian adenoviruses (FAdV-4) were assessed to be a risk in whole egg, egg yolk, and egg albumen powders and exotic avian influenza viruses were assessed to be a risk in egg albumen powders.

Import health standards for pasteurised egg white from Australia (*Pasalbic.aus*) and for pasteurised egg from Australia (*Poueggic.aus*) relate to commodities that are subject to different processing conditions than those described in the 2008 IRA. As a result, a rapid risk assessment has been requested to assess if any additional risks are likely to be associated with pasteurised egg white heated to 55°C for no less than 9.5 minutes (*Pasalbic.aus*) or pasteurised egg heated to 64°C for no less than 2.5 minutes (*Poueggic.aus*).

3. Preliminary hazard list

The 2008 IRA (MAF 2008a) examined a preliminary hazard list of 41 diseases that may be transmitted either on or in eggs. 31 of these diseases were potentially exotic to New Zealand, and of these the following were identified to be transmitted through the egg contents:

Angara disease (Adenoviridae)
Arizonosis (*Salmonella* Arizonae)
Avian influenza (Orthomyxoviridae)
Avian leukosis/sarcoma (Retroviridae)
Avian paramyxovirus types 2 & 3 (Paramyxoviridae)
Campylobacteriosis (*Campylobacter jejuni* and others)
Colibacillosis (*Escherichia coli* 0111, 0157:H7 and others)
Fowl typhoid (*Salmonella* Gallinarum)
Group 1 adenovirus infections (Adenoviridae)
Infectious bronchitis (Coronaviridae)
Mycoplasmosis (*Mycoplasma iowae*)
Newcastle disease (Paramyxoviridae)
Ornithobacteriosis (*Ornithobacterium rhinotracheale*)
Paratyphoid salmonellae (*Salmonella* Enteritidis etc.)
Psittacosis (*Chlamydophila psittaci*)
Pullorum disease (*Salmonella* Pullorum)

Table 1 summarises the available evidence indicating if these disease agents are likely to be inactivated by the processing conditions currently required for pasteurised egg white from Australia (*Pasalbic.aus*) and for pasteurised egg from Australia (*Poueggic.aus*).

Table 1. Stability of preliminary hazards identified in the 2008 IRA

Disease Agent	Likely to be inactivated by exposure to 55°C for no less than 9.5 minutes?	Reference	Likely to be inactivated by exposure to 64°C for no less than 2.5 minutes?	Reference
Avian adenoviruses	**No**	McFerran and Adair (2003)	**No**	McFerran and Adair (2003)
Although adenoviruses are generally regarded as being inactivated by exposure to temperatures of 56°C for 30 minutes, avian adenoviruses show more variability and appear to be more heat resistant with some strains surviving exposure to 60°C or even 70°C for 30 minutes (McFerran and Adair 2003).				
Avian Influenza virus	**No**	Swayne and Beck (2004)	Yes	Swayne and Beck (2004)
Swayne and Beck (2004) demonstrated that a low pathogenicity avian influenza virus strain (LPAI/NY/H7N2) had a D-value of <20 seconds in homogenized whole egg at 61°C, and a D-value of 0.5 days in dried egg white at 55°C. However, this study also demonstrated that inactivation of a high pathogenicity AI strain (HPAI/PA/83) in dried egg white would require exposure to 54.4°C for 21.38 days.				
Salmonella spp.	Yes	USDA (2005); CFR (2012)	Yes	Mitscherlich and Marth (1984a)
The United States Code of Federal Regulations 590.570 (CFR 2012) (based on USDA 2005) requires egg white to be pasteurised at 55.5°C for a minimum of 6.2 minutes. Studies on liquid whole egg artificially inoculated with sixteen different Salmonella serotypes and subjected to a range of pasteurisation temperatures demonstrated that none of these organisms were able to survive exposure to 60°C beyond a period of 2.9 minutes (Mitscherlich and Marth 1984a).				
Paramyxoviruses	Yes	Foster and Thompson (1957)	Yes	OIE (2012)
Studies into the thermal stability of avian paramyxoviruses have shown that within 5 minutes at 56°C, infectivity, haemagglutinative activity, and immunogenicity are destroyed (Foster and Thompson 1957). Article 10.9.21 of the *Code* describes exposure to 65°C for 39.8 seconds as sufficient to achieve a 7-log reduction in Newcastle disease virus (OIE 2012).				
Retroviruses	Yes	Dougherty (1961)	Yes	Dougherty (1961)
Avian retroviruses have been shown to be rapidly inactivated at high temperatures. Rous sarcoma virus has a half-life of 8.5 minutes at 50°C and 0.7 minutes at 60°C (Dougherty 1961).				
Escherichia coli	**No**	Doyle and Schoeni (1984)	Yes	Doyle and Schoeni (1984)
Although *E.coli* does not possess an unusual resistance to heat (Charimba *et al.* 2010), Doyle and Schoeni (1984) determined the D value for *E.coli* in ground beef to be 9.6 seconds at 64.3°C and 2390 seconds (approximately 40 minutes) at 54.4°C.				
Campylobacter spp.	Yes	Lori *et al.* (2007)	Yes	Lori *et al.* (2007)
Campylobacter spp. are generally regarded as being more sensitive to heat than other foodborne bacterial pathogens. Treatment at 55°C inactivates approximately 3.5 log CFU of *C. coli* or 2.5 log CFU of *C. jejuni* within 10 minutes. Incubation at 65°C for 2 minutes reduces the viable survivors of *C. coli* and *C. jejuni* strains by about 7 and 3.5 log CFU, respectively (Lori *et al.* 2007).				
Infectious bronchitis virus	**No**	See below	Yes	See below
Studies have shown that most strains of infectious bronchitis virus are inactivated at temperatures of 56°C after 15 minutes and at 45°C after 90 minutes (Cavanagh and Naqi 2003). However, studies on the SARS coronavirus have shown it to be completely inactivated by exposure to 56°C for 60 minutes or longer (Kariwa *et al.* 2004), although the thermal stability of the virus is increased by the presence of protein (Rabenau *et al.* 2005). More recent studies have shown that live IBV can be recovered from samples containing egg albumen following trestment at 56°C for over 15 minutes (Jackwood *et al.* 2010).				
Mycoplasma spp.	Yes	Mitscherlich and Marth (1984b; 1984c)	Yes	Mitscherlich and Marth (1984b; 1984c)
Sealed cultures of *Mycoplasma agalacticae* subsp. *agalacticae* have been shown to survive less than 8 minutes at 53°C (Mitscherlich & Marth 1984b) and cultures of *Mycoplasma gallisepticum* innoculated into eggs were found to be inactivated after 10 minutes at 54°C (egg white) or after 10 minutes at 56°C (egg yolk) (Mitscherlich & Marth 1984c).				
Ornithobacterium rhinotracheale	Yes	Mitscherlich and Marth (1984d)	Yes	Mitscherlich and Marth (1984d)
Ornithobacterium rhinotracheale will grow at temperatures up to 42°C, and it is likely that, as with most other vegetative forms of bacteria, it is quickly inactivated at temperatures much higher than that. A closely related bacterium, *Haemophilus paragallinarum,* is destroyed at 50°C in 2 minutes (Mitscherlich and Marth 1984d)				
Chlamydophila psittaci	Yes	Page (1959)	Yes	Page (1959)
Exposure of infectious tissue homogenates of *Chlamydophila psittaci* to 56°C for 5 minutes is sufficient for inactivation of the organism (Page 1959).				

3.1. PRELIMINARY HAZARD LIST CONCLUSION

Based on the evidence cited above, exotic group 1 avian adenoviruses (FAdV-4) may be a hazard in pasteurised egg heated to 64°C for no less than 2.5 minutes. The 2008 IRA (MAF 2008a) assessed FAdV-4 to be a risk in imported egg powders that have been subject to 60°C for a period of no less than 3.5 minutes and the risk management options presented to manage this risk are also appropriate for pasteurised egg heated to 64°C for no less than 2.5 minutes.

FAdV-4, avian influenza (AI) viruses, *Escherichia coli* and infectious bronchitis virus (IBV) may be hazards in pasteurised egg white heated to 55°C for no less than 9.5 minutes. The 2008 IRA (MAF 2008a) assessed FAdV-4 and AI viruses to be a risk in egg albumen powders subject to a final heat treatment of greater than 54.4°C for no less than 7 days. The risk management options presented in the 2008 IRA (MAF 2008a) to manage FAdV-4 and AI are also appropriate for pasteurised egg white heated to 55°C for no less than 9.5 minutes.

E. coli and IBV may be hazards in egg white heated to 55°C for no less than 9.5 minutes and require further risk assessment.

4. *Escherichia coli*

4.1. HAZARD IDENTIFICATION

4.1.1. Aetiological agent

Escherichia coli are classified according to the Kauffmann scheme on the basis of their somatic (O), flagellar (H), and capsular (K) antigens. More than 180 O, 60 H, and 80 K antigens are currently described (Stenutz *et al.* 2006). Localised or systemic infections are caused by avian pathogenic *E. coli* (APEC) (Barnes *et al.* 2008).

4.1.2. OIE list

Not listed.

4.1.3. New Zealand status

Colibacillosis has been described in New Zealand poultry (Ross 1984; Orr 1994; Orr 1995; Orr 1998) and has been associated with various disease manifestations including omphalitis, peritonitis, salpingitis, airsacculitis, colisepticaemia, coligranuloma, synovitis, and opthalmitis (Black 1997).

4.1.4. Epidemiology

E.coli is a common inhabitant of the intestinal tract of most mammals and birds. Potentially pathogenic strains of *E.coli* may be found in the intestines of around 13% of healthy chickens (Harry and Hemsley 1965).

Colibacillosis is responsible for significant economic losses in poultry flocks throughout the world. A survey of a poultry processing plant in the United Kingdom found 43% of broiler carcase rejections were due to colisepticaemia (Yogaratnam 1995) and *E.coli* was also found to be responsible for the majority of infections resulting in the condemnation of broiler carcases in Switzerland (Jakob *et al.* 1998). Post-mortem examination of poultry from 503 farms in Belgium demonstrated disease due to APEC in 153 farms (Vandemaele *et al.* 2002), a survey of 100 broiler farms in Jordan found 88% of airsaccultitis cases were due to *E. coli*

(El-Sukhon *et al.* 2002), and colibacillosis was found to be one of the most common diseases affecting Californian turkey flocks (Christiansen *et al.* 1996).

APEC isolates are generally considered to act as opportunistic pathogens and avian colibacillosis is thought to be a secondary disease. However, clones of APEC exist that are well adapted as pathogens and may not always require the presence of a primary predisposing infection (Barnes *et al.* 2008). APEC can be distinguished from commensal *E.coli* strains based on the ability to cause mortality in embryos or chicks, and this is regarded as the best single test for discriminating APEC from commensal *E. coli* strains (Gibbs *et al.* 2003; Gibbs and Wooley 2003; Gibbs *et al.* 2004). However, virulence assays do not account for predisposing host or environmental factors which may enable a less virulent isolate to cause disease under natural conditions (Nolan *et al.* 2002).

Surveys to determine which *E.coli* serotypes are present in poultry show that the predominant serotypes vary with geographic region (Sharada *et al.* 2001; Rosario *et al.* 2004) although Barnes *et al.* (2008) have described the most common serotypes identified as O1, O2, O35, O36, and O78.

Most APEC isolates are only pathogenic to chickens although *E.coli* O157 has been identified in both chickens (Pilipčinec *et al.* 1999) and ducks (Samadpour *et al.* 2002; Leclercq and Mahillon 2003). Chicken meat has also been recognised as a source of *E.coli* with virulence and antimicrobial resistance factors (Doyle and Schoeni 1987; Griffin and Tauxe 1991; Johnson *et al.* 2003).

All ages of poultry are susceptible to colibacillosis although disease is reported more often and with more severe clinical signs in developing embryos and chicks (Harry 1957; Goren 1978; Montgomery *et al.* 1999; Johnson *et al.* 2001). Infections can be predisposed by other infectious agents such as infectious bronchitis virus (Williams Smith *et al.* 1985; Nakamura *et al.* 1996) or haemorrhagic enteritis virus (Newberry *et al.* 1993; van den Hurk *et al.* 1994), or by environmental factors such as dust or high levels of ammonia (Oyetunde *et al.* 1978; Nagaraja *et al.* 1984). The incidence of colibacillosis has been shown to be related to the number of primary infections birds are exposed to before being challenged with *E. coli* (Pierson *et al.* 1996).

New strains of *E.coli* can be introduced into a flock through contact with other animals or their faeces (Barnes *et al.* 2008). Avian-adapted strains may be acquired from free-living waterfowl (Fallacara *et al.* 2001; Fallacara *et al.* 2004; Cole *et al.* 2005) or passerine species

(Morishita *et al.* 1999). Houseflies (*Musca domestica*) have also been associated with the transmission of *E.coli* (Rochon *et al.* 2004; Rochon *et al.* 2005).

The most frequent pathology in poultry associated with *E. coli* is systemic infection (Stordeur *et al.* 2002). Clinical signs of avian colibacillosis are highly variable, including localised infections (omphalitis, cellulitis, diarrhoea, vaginitis, salpingitis, and orchitis) and systemic diseases (colisepticaemia, airsaccultitis, meningitis, synovitis, and polyarthritis) (Barnes *et al.* 2008). Although primary enteritis is a common manifestation of *E.coli* infections in mammals, it is considered rare in poultry (Barnes 2008). Morbidity and mortality are highly variable depending on the type of disease associated with infection (Barnes *et al.* 2008).

It is difficult for bacteria such as *E. coli* to move across an intact good quality egg shell although small defects may provide means for bacteria on the shell surface to penetrate and move into the egg contents (De Reu *et al.* 2008). Studies on table eggs frequently identify *E.coli* on the surface of eggs but report infrequent *E. coli* contamination of egg contents (Chousalkar *et al.* 2010; Loongyai *et al.* 2011). However, *E. coli* will penetrate intact shells of eggs incubated in liquid cultures containing high doses of bacteria (Chousalkar *et al.* 2010).

4.1.5. Hazard identification conclusion

Colibacillosis is recognised in New Zealand poultry and has been associated with a variety of disease presentations. The clinical manifestation of colibacillosis is likely to be determined by underlying host, infectious, or environmental factors. There is no evidence to suggest that strains of APEC found overseas are any more virulent than the strains encountered in this country. *E. coli* is not identified as a hazard.

5. Infectious bronchitis virus

5.1. HAZARD IDENTIFICATION

5.1.1. Aetiological agent

Family *Coronaviridae*; Genus *Coronavirus*; Species *Avian Infectious Bronchitis Virus*

The *Coronaviridae* family is divided into two genera, *Coronavirus* and *Torovirus*, but only viruses of the *Coronavirus* genus have been reported to infect birds (Cook 2008). The coronaviruses have been assigned to three groups. The coronaviruses which infect mammals, including humans, belong to groups 1 and 2. Group 3 includes the type species infectious bronchitis virus (IBV) as well as turkey coronavirus (TCoV) pheasant coronavirus (PhCoV) and other emerging avian coronaviruses (Cavanagh 2005).

IBV does not constitute a single homogenous antigenic type. The prototype virus is Massachusetts M41 but a plethora of IBV strains exist and new IBV variants continue to be recognised (Dhinakar Raj and Jones 1997; Cook 2008).

5.1.2. OIE list

Avian infectious bronchitis is an OIE-listed disease.

5.1.3. New Zealand status

Serological testing confirms that IBV is widespread in New Zealand (Watts 2010) and 28 strains have been identified using RT-PCR (Ramneek *et al.* 2005).

In many countries, some of the most frequently used IBV vaccines have been developed from strains isolated either in Europe or in the USA. Vaccine is used in New Zealand although the only live vaccines available are derived from local field strains (Howell 1992).

5.1.4. Epidemiology

IBVs are ubiquitous in countries where poultry are reared intensively (Wit *et al.* 2010). There is extensive antigenic variation and differences in virulence and tropism between strains from different geographic regions (McMartin 1993; McFarlane and Verma 2008). The IBVs in

Australia belong to a distinct lineage with two genotypically distinct groups. Group I comprises nephropathogenic strains of varying pathogenicity and group II contains three mildly pathogenic respiratory strains (Sapats *et al.* 1996; Ignatovic *et al.* 2006). New Zealand IBV strains are closely related to the group I serotypes from Australia and are distinct from the European and North American strains (Ramneek *et al.* 2005; McFarlane and Verma 2008; Wit *et al.* 2010).

Avian IB is predominantly a respiratory infection, however three clinical manifestations of IBV infection are observed in the field; respiratory disease, reproductive disorders and nephritis (Ignjatovic and Sapats 2000).

The majority of IBV strains around the world, with the exception of Australia and New Zealand, produce prominent respiratory disease (Ignjatovic and Sapats 2000). The QX strain of IBV was first detected in China between 1995 and 2004 and subsequently spread through Russia and Europe (Irvine *et al.* 2010). The Australian group I strains include the vaccinal strain Vic S as well as the highly nephropathogenic N1/62 (synonym 'T') strains responsible for mortalities of up to 96%. The Australian group II strains only replicate in the trachea and do not induce mortalities (Sapats *et al.* 1996; Ignatovic *et al.* 2006). In New Zealand, the presence of virus may lead to impaired productivity although it is generally not linked to clinical disease (Ramneek *et al.* 2005). The respiratory effects of all New Zealand field strains appear to be mild, but nephritis and outbreaks of uraemia due to IBV have been reported (Lohr 1977; Howell 1992) and infected layers can have a drop in egg production of up to 12% (Findon 1987).

IBV is highly infectious to chickens of all ages, and under natural conditions will spread between houses within 1 to 2 days, and between farms within 3 to 4 days (Ignjatovic and Sapats 2000).

The virus is present in considerable titers in tracheal mucus and faeces during the acute and recovery phases of disease, respectively. The virus may be shed for more than 20 weeks after clinical recovery and can persist in the intestinal tract for several months (Alexander and Gough 1977; Cook 2008). Spread occurs horizontally by aerosol or by ingestion of faeces or contaminated feed or water. The most common source of infection is direct chicken-to-chicken contact, but indirect transmission via mechanical spread, sometimes over long distances, also occurs (OIE 2008). The virus can survive for a considerable time in faeces.

The upper respiratory tract is the initial site of IBV replication, regardless of strain, following which a viraemia occurs, disseminating the virus to other epithelial surfaces, including the kidney, oviduct, testes, bursa of Fabricius, and alimentary tract (McMartin 1993; Cavanagh 2007).

Respiratory signs are the first and most common clinical manifestation in birds of all ages and include tracheal rales, gasping, sneezing and watery nasal discharge (Cook 2008). In uncomplicated cases these signs are short-lived (10–14 days) and mortalities are generally low. Frequently infection is complicated by secondary infections with organisms such as infectious bursal disease virus, *Mycoplasma gallisepticum*, or *Escherichia coli*. IBV interacts synergistically with these organisms and co-infection is considered the main cause of mortality in older birds (Lopez and McFarlane 2006; Cavanagh 2007).

Nephropathogenic IBV (NIBV) strains initially cause some respiratory signs followed by signs due to kidney damage and mortalities up to 30% (Ignjatovic and Sapats 2000; Cook 2008). Infection of the oviduct can lead to permanent damage in immature birds and a drop in egg production (exceeding 50%) and quality in hens. There are also reports of variant strains causing pectoral myopathy (Dhinakar Raj and Jones 1997) and proventriculitis (Benyeda *et al.* 2010). The QX strain of IBV has been associated with a wide range of clinical problems including respiratory disease, wet litter, mortality due to kidney damage, proventriculitis, and decreased flock performance. Infection of layers with the QX strain has been associated with 'blind' or 'false-layer' syndrome due to oviduct lesions (Irvine *et al.* 2010).

Latent infection (from both vaccine and field strains) can establish in the kidney and caecal tonsils for up to 163 days or longer, with subsequent erratic shedding of virus via both faeces and aerosol (Ignjatovic and Sapats 2000; Cavanagh and Gelb 2008;) and often with long pauses (up to 42 days) between episodes of shedding (Naqi *et al.* 2003). Virus re-excretion is not generally accompanied by clinical signs and may serve as a source of infection to susceptible chickens (Dhinakar Raj and Jones 1997).

Cavanagh and Naqi (2003) stated that there were reports of virus isolations from eggs up to 43 days after recovery from infection, even though chickens have been hatched from infected flocks and raised free of IBV. However, this statement on IBV transmission in the egg has been removed from the latest (12th) edition of this text (Cavanagh Gelb 2008). Furthermore, this unreferenced statement by Cavanagh and Naqi (2003) is the only reference that could be found to transmission of this virus in the egg (MAF 2008b). It would be reasonable to

conclude that there is insufficient evidence to suggest that poultry eggs are a vehicle for the transmission of IBV (Cobb 2011).

5.1.5. Hazard identification conclusion

Avian infectious bronchitis virus (IBV) multiplies primarily in the respiratory tract. Following 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 (Hofstad and Yoder 1966). There is insufficient evidence to suggest that poultry eggs could be a vehicle for the transmission of IBV (Cobb 2011). IBV is not identified as a hazard.

References

Alexander DJ, Gough RE (1977). Isolation of avian infectious bronchitis virus from experimentally infected chickens. *Research in Veterinary Science* 23, 344-347

Barnes JH, Nolan LK, Vaillancourt J-P (2008). Colibacillosis. In *Diseases of Poultry* 12th Edition, Ed Saif YM, Blackwell Publishing, 691-732.

Benyeda Zs, Szeredi L, Mato T, Suveges T, Balka Gy, Abony-Toth Zs, Rusvai M, Palya V (2010). Comparative histopathology and immunohistochemistry of QX-like, Massachusetts and 793/B serotypes of infectious bronchitis virus infection in chickens. *Journal of Comparative Pathology* 143, 276-283.

Black A (1997). Bacterial and parasitic diseases of New Zealand poultry. *Surveillance* 24(4), 3-5.

Cavanagh D (2005). Coronaviruses in poultry and other birds. *Avian Pathology* 34, 439-448.

Cavanagh D (2007). Coronavirus avian infectious bronchitis virus. *Veterinary Research* 38, 281-297.

Cavanagh D, Naqi SA (2003). Infectious bronchitis. In *Diseases of Poultry* 11th Edition. Ed Saif YM. Iowa State Press, Ames, Iowa, 101-119.

Cavanagh D, Gelb J Jr (2008). Infectious Bronchitis. In *Diseases of Poultry* 12th Edition. Eds Saif YM, Fadly AM, Glisson JR, McDougald LR, Nolan LK and Swayne DE, Blackwell Publishing, 511-526.

CFR (2012). *Pasteurization of liquid eggs*. Available at: http://www.gpo.gov/fdsys/pkg/CFR-2012-title9-vol2/pdf/CFR-2012-title9-vol2-sec590-570.pdf

Charimba G, Hugo CJ, Hugo A (2010). The growth, survival and thermal inactivation of *Escherichia coli* O157:H7 in a traditional South African sausage. *Meat Science* 85, 89-95.

Christiansen KH, Hird DW, Snipes KP, Danaye-Elmi C, Palmer CW, McBride MD, Utterback WW (1996). California National Animal Health Monitoring System for meat turkey flocks – 1988-89 pilot study: management practices, flock health, and production. *Avian Diseases* 40, 278-284.

Chousalkar KK, Flynn P, Sutherland M, Roberts JR, Cheetham BF (2010). Recovery of *Salmonella* and *Escherichia coli* from commercial egg shells and effect of translucency on bacterial penetration of eggs. *International Journal of Food Microbiology* 142, 207-213.

Cobb SP (2011). The spread of pathogens through trade in poultry hatching eggs: overview and recent developments. *Revue Scientifique et Technique* 30, 165-175.

Cole D, Drum DJV, Stallknecht DE, White DG, Lee MD, Ayers S, Sobsey M, Maurer JJ (2005). Free-living Canada geese and antimicrobial resistance. *Emerging Infectious Diseases* 11, 935-938.

Cook J (2008). Coronaviridae. In *Poultry Diseases* 6th Edition, Eds Pattison M, McMullin PF, Bradbury JM and Alexander DJ, Elsevier/Butterworth-Heinemann, 340-349.

De Reu K, Messens W, Heyndrickx M, Rodenburg B, Uyttendaele M, Herman L (2008). Bacterial contamination of table eggs and the influence of housing systems. *World's Poultry Science Journal* 64, 5-19.

Dhinakar Raj G, Jones RC (1997). Infectious bronchitis virus: immunopathogenesis of infection in the chicken. *Avian Pathology* 26, 677-706.

Dougherty RM (1961). Heat inactivation of Rous sarcoma virus. *Virology* 14, 371-372.

Doyle MP, Schoeni JL (1984). Survival and growth characteristics of *Escherichia coli* associated with hemorrhagic colitis. *Applied and Environmental Microbiology* 48, 855-856.

Doyle MP, Schoeni JL (1987). Isolation of *Escherichia coli* O157:H7 from retail fresh meats and poultry. *Applied and Environmental Microbiology* 53, 2394-2396.

El-Sukhon SN, Musa A, Al-Attar M (2002). Studies on the bacterial aetiology of airsaccultitis of broilers in northern and middle Jordan with special reference to *Escherichia coli*, *Ornithobacterium rhinotracheale*, and *Bordetella avium*. *Avian Diseases* 46, 605-612.

Fallacara DM, Monahan CM, Morishita TY, Wack RF (2001). Fecal shedding and antimicrobial susceptibility of selected bacterial pathogens and a survey of intestinal parasites in free-living waterfowl. *Avian Diseases* 45, 128-135.

Fallacara DM, Monahan CM, Morishita TY, Bremer CA, Wack RF (2004). Survey of parasites and bacterial pathogens from free-living waterfowl in zoological settings. *Avian Diseases* 48, 759-767.

Findon G (1987). Some field observations on the antibody response to avian infectious bronchitis on Auckland layer farms. *New Zealand Veterinary Journal* 35, 211-214.

Foster NM, Thompson CH (1957). The comparative thermostability of four strains of Newcastle disease virus of widely varying virulence. *Veterinary Medicine* 52, 119-121

Gibbs PS, Maurer JJ, Nolan LK, Wooley RE (2003). Prediction of chicken embryo lethality with the avian *Escherichia coli* traits complement resistance, colicin V production, and presence of the increased serum survival gene cluster (*iss*). *Avian Diseases* 47, 370-379.

Gibbs PS, Wooley RE (2003). Comparison of the intravenous chicken challenge method with the embryo lethality assay for studies in avian colibacillosis. *Avian Diseases* 47, 672-680.

Gibbs PS, Petermann SR, Wooley RE (2004). Comparison of several challenge models for studies in avian colibacillosis. *Avian Diseases* 48, 751-758.

Goren E (1978). Observations on experimental infection of chicks with *Escherichia coli*. *Avian Pathology* 7, 213-224.

Griffin PM, Tauxe RV (1991). The epidemiology of infections caused by *Escherichia coli* O157:H7, other enterohemorrhagic *E.coli*, and the associated hemolytic uremic syndrome. *Epidemiologic Reviews* 13, 60-98.

Harry EG (1957). The effect on embryonic and chick mortality of yolk contamination with bacteria from the hen. *Veterinary Record* 69, 1433-1439.

Harry EG, Hemsley LA (1965). The association between the presence of septicaemia strains of *Escherichia coli* in the respiratory and intestinal tracts of chickens and the occurrence of coli septicaemia. *Veterinary Record* 77, 35-40.

Hofstad MS, Yoder Jr HW (1966). Avian infectious bronchitis-virus distribution in tissues of chicks. *Avian Diseases* 10, 230-239.

Howell J (1992). Viral diseases and the New Zealand poultry industry. *Surveillance* 19 (2), 15-17.

Ignjatovic J, Gould G, Sapats S (2006). Isolation of a variant infectious bronchitis virus in Australia that further illustrates diversity among emerging strains. *Archives of Virology* 151, 1567-1585.

Ignjatovic J, Sapats S (2000). Avian infectious bronchitis virus. *Revue Scientifique et Technique* 19, 493-508.

Irvine RM, Cox WJ, Ceeraz V, Reid SM, Ellis RJ, Jones RM, Errington J, Wood AM, McVicar CM, Clark MI (2010). Detection of IBV QX in commercial broiler flocks in the UK. *Veterinary Record* 167, 877-879.

Jackwood MW, Hilt DA, Sellers HS, Williams SM, Lasher HN (2010). Rapid heat-treatment attenuation of infectious bronchitis virus. *Avian Pathology* 39, 227-233.

Jakob HP, Morgenstern R, Albicker P, Hoop RK (1998). Konfiskationsursachen bei geschlachteten Broilern aus zwei grossen schweizerischen Mastorganisationen (Condemnation reasons of slaughtered broilers from two major Swiss producing companies). *Schweizer Archiv für Tierheilkunde* 140, 60-64.

Johnson JR, Murray AC, Gajewski A, Sullivan M, Snippes P, Kuskowski MA, Smith KE (2003). Isolation and molecular characterization of nalidixic acid-resistant extraintestinal pathogenic *Escherichia coli* from retail chicken products. *Antimicrobial Agents and Chemotherapy* 47, 2161-2168.

Johnson LC, Bilgili SF, Hoerr FJ, McMurtrey BL, Norton RA (2001). The influence of *Escherichia coli* strains from different sources and the age of broiler chickens in the development of cellulitis. *Avian Pathology* 30, 475-479.

Kariwa H, Fujii N, Takashima I (2004). Inactivation of SARS coronavirus by means of povidone-iodine, physical conditions, and chemical reagents. *Japanese Journal of Veterinary Research* 52, 105-112.

Leclercq A, Mahillon J (2003). Farmed rabbits and ducks as vectors for VTEC O157:H7. *Veterinary Record* 152, 723-724.

Lohr JE (1977). Studies on avian infectious bronchitis virus in New Zealand, I. Serotypes. *New Zealand Veterinary Journal* 25, 48 -51.

Loongyai W, Wiriya B, Sangsawang N (2011). Detection of *Salmonella* and *Escherichia coli* in egg shell and egg content from different housing systems for laying hens. *International Journal of Poultry Science* 10, 93-97.

Lopez JC, McFarlane R (2006). Environmental factors influence the prevalence of infectious bronchitis virus. *Proceedings of the 18th Australian Poultry Science Symposium,* Sydney, New South Wales, Australia, 127-130.

Lori S, Buckow R, Knorr D, Heinz V, Lehmacher A (2007). Predictive model for inactivation of *Campylobacter* spp. by heat and high hydrostatic pressure. *Journal of Food Protection* 70, 2023-2029.

McFarlane R, Verma R (2008). Sequence analysis of the gene coding for the S1 glycoprotein of infectious bronchitis virus (IBV) strains from New Zealand. *Virus Genes* 37, 351-357.

McFerran JB, Adair BM (2003). In *Diseases of Poultry,* 11th Edition. Ed. Saif YM, Iowa State Press, p216

McMartin D (1993). Infectious Bronchitis. In *Virus Infections of Birds,* Volume 4, Eds McFerran JB and McNulty MS, Elsevier Science Publishers BV, 249-274.

Ministry of Agriculture and Forestry (MAF) Biosecurity New Zealand (2008a). Import risk analysis: egg powders from all countries. MAF, Wellington, New Zealand. Available at: http://www.biosecurity.govt.nz/files/regs/imports/risk/egg-powder-ra.pdf

Ministry of Agriculture and Forestry (MAF) Biosecurity New Zealand (2008b). Import risk analysis: hatching eggs from chickens (*Gallus gallus*) from the European Union, Canada, the United States of America, and Australia. MAF, Wellington, New Zealand. Available at: www.biosecurity.govt.nz/files/regs/imports/risk/hatching-eggs-eu-ra.pdf

Mitscherlich E, Marth EH (1984a). *Microbial Survival in the Environment: Bacteria and Rickettsiae Important in Human and Animal Health.* Berlin, Springer-Verlag., p597

Mitscherlich E, Marth EH (1984b). *Microbial Survival in the Environment: Bacteria and Rickettsiae Important in Human and Animal Health.* Berlin, Springer-Verlag., p266

Mitscherlich E, Marth EH (1984c). *Microbial Survival in the Environment: Bacteria and Rickettsiae Important in Human and Animal Health.* Berlin, Springer-Verlag., p270

Mitscherlich E, Marth EH (1984d). *Microbial Survival in the Environment: Bacteria and Rickettsiae Important in Human and Animal Health.* Berlin, Springer-Verlag., p194

Montgomery RD, Boyle CR, Lenarduzzi TA, Jones LS (1999). Consequences to chicks hatched from *Escherichia coli*-inoculated embryos. *Avian Diseases* 43, 553-563.

Morishita TY, Aye PP, Ley EC, Harr BS (1999). Survey of pathogens and blood parasites in free-living passerines. *Avian Diseases* 43, 549-552.

Nagaraja KV, Emery DA, Jordan KA, Sivanandam V, Newman JA, Pomeroy BS (1984). Effect of ammonia on the quantitative clearance of *Escherichia coli* from lungs, air sacs, and livers of turkeys aerosol vaccinated against *Escherichia coli*. *American Journal of Veterinary Research* 45, 392-395.

Nakamura K, Imai K, Tanimura N (1996). Comparison of the effects of infectious bronchitis and infectious laryngotracheitis on the chicken respiratory tract. *Journal of Comparative Pathology* 114, 11-21.

Naqi S, Gay K, Patalla P, Mondal S, Liu R (2003). Establishment of persistent avian infectious bronchitis virus infection in antibody-free and antibody-positive chickens. *Avian Diseases* 47, 594-601.

Newberry LA, Skeeles JK, Kreider DL, Beasley JN, Story JD, McNew RW, Berridge BR (1993). Use of virulent hemorrhagic enteritis virus for the induction of colibacillosis in turkeys. *Avian Diseases* 37, 1-5.

Nolan LK, Giddings CW, Horne SM, Doetkott C, Gibbs PS, Wooley RE, Foley SL (2002). Complement resistance, as determined by viable count and flow cytometric methods, and its association with the presence of *iss* and the virulence of avian *Escherichia coli*. *Avian Diseases* 46, 386-392.

OIE (2008). Avian infectious bronchitis. *Manual of Diagnostic Tests and Vaccines for Terrestrial Animals.* Chapter 2.3.2. Available at: http://www.oie.int/fileadmin/Home/eng/Health_standards/tahm/2.03.02_AIB.pdf.

OIE (2012). Newcastle disease. *Terrestrial Animal Health Code*. Chapter 10.9. Available at: http://www.oie.int/index.php?id=169&L=0&htmfile=chapitre_1.10.9.htm

Orr M (1994). Animal health laboratory network. Review of diagnostic cases – October to December 1993. *Surveillance* 21(1), 3-4.

Orr M (1995). Animal health laboratory network. Review of diagnostic cases – October to December 1994. *Surveillance* 22(1), 3-5.

Orr M (1998). Review of diagnostic cases – April to June 1998. *Surveillance* 25(3), 15-17.

Oyetunde OOF, Thomson RG, Carlson HC (1978). Aerosol exposure of ammonia, dust and *Escherichia coli* in broiler chickens. *Canadian Veterinary Journal* 19, 187-193.

Page LA (1959). Thermal inactivation studies on a turkey ornithosis virus. *Avian Diseases* 3, 67-79.

Pierson FW, Larsen CT, Domermuth CH (1996). The production of colibacillosis in turkeys following sequential exposure to Newcastle disease virus or *Bordetella avium*, avirulent hemorrhagic enteritis virus, and *Escherichia coli*. *Avian Diseases* 40, 837-840.

Pilipčinec E, Tkáčiková L', Naas HT, Cabadaj R, Mikula I (1999). Isolation of verotoxigenic *Escherichia coli* O157 from poultry. *Folia Microbiologica* 44, 455-456.

Rabenau HF, Cinatl J, Morgenstern B, Bauer G, Preiser W, Doerr HW (2005). Stability and inactivation of SARS coronavirus. *Medical Microbiology and Immunology* 194, 1-6.

Ramneek, Mitchell NL, McFarlane RG (2005). Rapid detection and characterisation of infectious bronchitis virus (IBV) from New Zealand using RT-PCR and sequence analysis. *New Zealand Veterinary Journal* 53, 457-461.

Rochon K, Lysyk TJ, Selinger LB (2004). Persistence of *Escherichia coli* in immature house fly and stable fly (Diptera: Muscidae) in relation to larval growth and survival. *Journal of Medical Entomology* 41, 1082-1089.

Rochon K, Lysyk TJ, Selinger LB (2005). Retention of *Escherichia coli* by house fly and stable fly (Diptera: Muscidae) during pupal metamorphosis and eclosion. *Journal of Medical Entomology* 42, 397-403.

Rosario CC, López AC, Téllez IG, Navarro OA, Anderson RC, Eslava CC (2004). Serotyping and virulence genes detection in *Escherichia coli* isolated from fertile and infertile eggs, dead-in-shell embryos, and chickens with yolk sac infection. *Avian Diseases* 48, 791-802.

Ross AD (1984). Laboratory and field reports. Colibacillosis in turkey poults. *Surveillance* 11(1), 42-43.

Samadpour M, Stewart J, Steingart K, Addy C, Louderback J, McGinn M, Ellington J, Newman T (2002). Laboratory investigation of an *E. Coli* O157:H7 outbreak associated with swimming in battle ground lake, Vancouver, Washington. *Journal of Environmental Health* 64, 16-20.

Sapats SI, Ashton F, Wright PJ, Ignjatovic J (1996). Sequence analysis of the S1 glycoprotein of infectious bronchitis viruses: identification of a novel genotypic group in Australia. *Journal of General Virology* 77, 413-418.

Sharada R, Krishnappa G, Upendra HA (2001). Serological 'O' grouping and drug susceptibility of *Escherichia coli* strains from chicken. *Indian Veterinary Journal* 78, 78-79.

Stenutz R, Weintraub A, Widmalm G (2006). The structures of *Escherichia coli* O-polysaccharide antigens. *FEMS Microbiology Reviews* 30, 382-403.

Stordeur P, Marlier D, Blanco J, Oswald E, Biet F, Dho-Moulin M, Mainil J (2002). Examination of *Escherichia coli* from poultry for selected adhesion genes important in disease caused by mammalian pathogenic *E. coli*. *Veterinary Microbiology* 84, 231-241.

Swayne DE, Beck JR (2004). Heat inactivation of avian influenza and Newcastle disease viruses in egg products. *Avian Pathology* 33, 512-518.

USDA (2005). *Pasteurization of Liquid Egg Products and Shell Eggs*. Available at: http://www.fsis.usda.gov/pdf/se_risk_assess_annex_g_oct2005.pdf

Van den Hurk JV, Allan BJ, Riddell C, Watts T, Potter AA (1994). Effect of infection with hemorrhagic enteritis virus on susceptibility of turkeys to *Escherichia coli*. *Avian Diseases* 38, 708-716.

Vandemaele F, Vereecken M, Derijcke J, Goddeeris BM (2002). Incidence and antibiotic resistance of pathogenic *Escherichia coli* among poultry in Belgium. *Veterinary Record* 151, 355-356.

Watts J (2010). Poultry health surveillance. *Surveillance* 37(3), 20-21.

Williams Smith H, Cook JKA, Parsell ZE (1985). The experimental infection of chickens with mixtures of infectious bronchitis virus and *Escherichia coli*. *Journal of General Virology* 66, 777-786.

Wit JJ (Sjaak) de, Cook JKA, van der Heijden HMJF (2010). Infectious bronchitis virus in Asia, Africa, Australia and Latin America - History, current situation and control measures. *Brazilian Journal of Poultry Science* 12, 97-106.

Yogaratnam V (1995). Analysis of the causes of high rates of carcase rejection at a poultry processing plant. *Veterinary Record* 137, 215-217.