Ecology and Epidemiology of Newcastle Disease

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Dennis J. Alexander

2.1 Introduction

The first outbreaks of the severe disease of poultry known as Newcastle disease (ND) occurred in 1926, in Java, Indonesia (Kraneveld 1926), and in Newcastle-upon-Tyne, England (Doyle 1927). The name "Newcastle disease" was coined by Doyle as a temporary measure because he wished to avoid a descriptive name that might be confused with other diseases (Doyle 1935). The name has, however, continued to be used, although when referring to ND virus (NDV), the synonym "avian paramyxovirus type 1" (APMV-1) is now often employed. Sometimes APMV-1 has been used to describe ND strains of low virulence, to avoid terming them ND viruses, as the definitions used by the World Organisation for Animal Health (Alexander 2008) and other international agencies reserve ND for virulent viruses.

Whether the outbreaks of 1926 marked the emergence of ND has been the subject of some discussion, as there are earlier reports of similar disease outbreaks in Central Europe before this date (Halasz 1912). Macpherson (1956), in reviewing the death of all the chickens in the Western Isles of Scotland in 1896, considered it probable that the cause was ND. It is possible, therefore, that ND did occur in poultry before 1926, but its recognition as a specifically defined disease of viral aetiology dates from the outbreaks during that year in Newcastle-upon-Tyne.

Later, it became clear that other, less severe infections were caused by viruses almost identical to the original virus. In the United States, a relatively mild respiratory disease, often with nervous signs, was first reported in the 1930s and subsequently termed "pneumoencephalitis" (Beach 1942). It was shown to be due to a virus indistinguishable from NDV in serological tests (Beach 1944). Since then, numerous isolations of viruses that produce an extremely mild disease or no evidence of disease in chickens have been made around the world, and it is now accepted that pools of such viruses are perpetuated in waterfowl and other wild birds.

2.2 Aetiology

The virus order Mononegavirales (i.e. the single-stranded, nonsegmented, negative-sense RNA viruses showing helical capsid symmetry) is formed from the virus families *Paramyxoviridae*, *Filoviridae* and *Rhabdoviridae*. The family *Paramyxoviridae* is divided into two subfamilies *Paramyxovirinae* and *Pneumovirinae* (Lamb et al. 2005). The subfamily Paramyxovirinae has five genera: *Rubulavirus,* which includes the mumps virus, mammalian para-influenza 2 and 4; *Respirovirus* containing mammalian para-influenza viruses 1 and 3; *Morbillivirus,* measles, distemper and rinderpest; *Henipavirus*, formed from the Nipah and Hendra viruses; and *Avulavirus*, formed from NDV and other avian paramyxoviruses (Lamb et al. 2005).

Nine serogroups of avian paramyxoviruses have been recognised: APMV-1 to APMV-9 (Alexander 1988a). Of these, NDV (APMV-1) remains the most important pathogen for poultry, but APMV-2, APMV-3, APMV-6 and APMV-7 are known to cause disease in poultry. The nomenclature used for isolates of influenza A virus has been adopted for avian paramyxoviruses so that an isolate is named by: (1) serotype, (2) species or type of bird from which it was isolated, (3) geographical location of isolation, (4) reference number or name and (5) year of isolation.

Antigenic variation of ND viruses (APMV-1) detectable by conventional haemagglutination inhibition (HI) tests has been reported, although such reports are rare and represent relatively minor variations (Arias-Ibarrondo et al. 1978; Hannoun 1977; Alexander et

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al. 1984). One of the most noted variations of this kind has been the virus responsible for the panzootic in racing pigeons. This NDV, often referred to as pigeon APMV-1 (PPMV-1), was demonstrably different from standard strains in haemagglutination inhibition tests, but not sufficiently different antigenically that conventional ND vaccines were not protective (Alexander and Parsons 1986). Antigenic variations between ND strains have been detected by monoclonal antibodies (mAbs) and have been used as an epidemiological tool (Alexander et al. 1997). Although use of mAb panels has shown that viruses grouped by their ability to react with the same mAbs share biological and epidemiological properties, this approach to understanding the epidemiology of ND has been largely replaced by phylogenetic analysis.

Genetic techniques have become established in the diagnosis of ND and as an epidemiological tool for distinguishing between virus strains (Aldous and Alexander 2001). Herczeg et al. (1999, 2001); Lomniczi et al. (1998) concluded from their phylogenetic analyses of NDV isolates that there were eight genetic lineages (I–VIII) and several sublineages within them. Aldous et al. (2003), in a study of 338 isolates of NDV representing a range of viruses of different temporal, geographical and host origins, concluded that the isolates divided into six broadly distinct groups (lineages 1–6). Lineages 3 and 4 were further subdivided into four sublineages (a–d) and lineage 5 into five sublineages (a–e). Essentially, lineages 1, 2, 4 and 5 correspond to the earlier defined lineages I, II, VI and VII, with comparable sublineages but the geno-groupings III, IV, V, VIII correspond to the sublineages 3a–3d. Lineage 6 represents a new geno-group.

Although the NDV isolates placed in geno-groups 1–5 (or I–VIII) are genetically quite close, viruses that were placed in geno-group 6 by Aldous et al. (2003), and later class I by Czeglédi et al. (2006), are very different from all the other NDV isolates, i.e. the class II viruses (Czeglédi et al. 2006). This has caused problems in molecular diagnosis, particularly as different primers are necessary for their detection in RT-PCR tests.

2.3 Host Range

Following a review of the available literature, Kaleta and Baldauf (1988) concluded that, in addition to the domestic avian species, natural or experimental infection with NDV has been demonstrated in at least 241 species from 27 of the 50 Orders of birds. It is highly probable that all bird species are susceptible to infection, but the outcome of infection in terms of disease varies considerably with different species.

2.3.1 Domestic Poultry

Virulent NDV strains have been isolated from all types of commercially reared poultry, ranging from pigeons to ostriches. The disease signs seen in different poultry infected with virulent NDV may show considerable variation. Ducks, for example, may not show clinical signs, while in other species the disease may be milder than in chickens and cause problems in initial diagnosis, e.g. in pheasants (Aldous et al. 2007). Ostriches may also cause problems in the initial suspicion of ND since, while they have been reported to show typical nervous signs, there is some difference in the severity of disease between young and adult birds (Alexander 2000).

Marginal domestic poultry may also play a significant role in the epidemiology of ND. For example, fighting cocks were involved in outbreaks of ND in the United States on several occasions. The most notable outbreak occurred in southern California in 2002–2003 (Kinde et al. 2003), where the widespread presence of ND in fighting cocks and the mobility and value of such birds not only posed considerable control problems but resulted in spread to 21 commercial table-egg farms and the slaughter of 3 million birds. The highest risk factors for infected commercial flocks were the farm employees and proximity to infected backyard game fowl.

2.3.2 Wild Birds

Isolates of NDV have been obtained frequently from wild birds, especially migratory feral waterfowl and other aquatic birds. Most of these isolates have been of low virulence for chickens and similar to viruses of the “asymptomatic enteric” pathotype.

Occasionally, virulent viruses have been detected in wild birds, but usually these were in birds found dead near infected poultry. The most significant outbreaks of NDV in feral birds have been those reported in double-crested cormorants (*Phalacrocorax auritus*) in North America since the 1990s. These out-

breaks began in 1990 in Canada, specifically, in Alberta, Saskatchewan and Manitoba (Wobeser et al. 1993). The disease re-appeared in 1992 in cormorants in mid-western Canada, around the Great Lakes and northern Midwest USA, in the latter case spreading to domestic turkeys (Mixson and Pearson 1992; Heckert 1993). Disease in double-crested cormorants was observed again in Canada in 1995 and in California in 1997; in both instances, NDV was isolated from dead birds (Kuiken 1998).

Antigenic and genetic analyses of the viruses isolated from the cormorants suggested that these viruses were very closely related despite the geographical separation of the hosts. Since these outbreaks covered birds that would follow different migratory routes it seems most probable that initial infection occurred at a mutual wintering area in the Southern USA or Central America. Allison et al. (2005) were able to isolate similar virulent NDV from double-crested cormorants over-wintering in the Florida Keys.

There had been earlier reports of ND in cormorants and related species in the late 1940s in Scotland (Blaxland 1951) and in 1975 in Quebec (Cleary 1977). It is therefore possible that cormorants represent an occasional or even continual reservoir of virulent NDV.

Interestingly, wild birds have been implicated in the introduction of virulent NDV into poultry in a number of outbreaks over the last 10 years. For example, it was concluded that the virus responsible for the outbreaks of ND in the UK in 1997 (Alexander et al. 1999) had most likely been introduced by migratory wild birds. The virus responsible for the outbreaks in free-living pheasants in Denmark in 1996 was closely related, as were isolates from a goosander in Finland in 1996 and, perhaps significantly, a cormorant from Denmark in 2001 (Jørgensen al. 1999; Alexander et al. 1999; P. Jørgensen, personal communication). Re-emergence of a genetically very closely related virus in pheasants in Great Britain and France in 2005 and the close proximity of the French (Loire Atlantique) farm to a lake led to the speculation that this virus may be established in some species of wild birds in Europe (Aldous et al. 2007).

2.3.3 Caged "Pet Birds"

Virulent NDV isolates have often been obtained from captive caged birds (Senne et al. 1983). Kaleta and Baldauf (1988) thought it unlikely that infections of recently imported caged birds resulted from enzootic infections in feral birds in the countries of origin. They considered that the infections more probably originated at holding stations before export, either as a result of enzootic NDV at those stations or of spread from nearby poultry, such as backyard chicken flocks. Panigrahy et al. (1993) described outbreaks of severe ND in pet birds in six states in the USA in 1991. Illegal importations were assumed to be responsible for the introductions of the virus.

One important consideration for psittacines has been the demonstration that infected birds have been shown to excrete virulent NDV intermittently for extremely long periods, in some cases for more than a year (Erickson et al. 1977), which further emphasises the role these birds may have in the introduction of NDV to a country or area.

2.3.4 Racing and Show Pigeons

In the late 1970s, an NDV strain showing some antigenic differences from classical strains appeared in pigeons. This strain, PPMV-1, probably arose in the Middle East. In Europe, it was first reported in racing pigeons in Italy in 1981 (Biancifiori and Fioroni 1983) and subsequently produced a true panzootic, spreading in racing and show pigeons throughout the world (Aldous et al. 2004). The disease in pigeons has been recognised for over 25 years but still seems to remain enzootic in racing pigeons in many countries, with regular spread to wild pigeons and doves and a continuing threat to poultry.

2.4 Molecular Basis of Viral Virulence

An understanding of the molecular basis that controls the virulence of NDV strains (Rott and Klenk 1988) has meant that it is now possible, using nucleotide sequencing techniques, to assess whether or not an isolate has the genetic makeup to be highly pathogenic for poultry (Collins et al. 1993). The viral F protein brings about fusion between the viral membrane and the cell membrane so that the viral genome enters the cell and replication can begin. The F protein is therefore essential for replication. However, during replication, NDV particles are produced with a precursor glycoprotein, F0, that has to be cleaved to F1 and F2 polypeptides, which remain

bound by disulphide bonds, for the virus particles to be infectious. This post-translational cleavage is mediated by host cell proteases.

The cleavability of the F0 molecule has been shown to be related directly to the virulence of the viruses in vivo. Numerous studies have confirmed the presence of multiple basic amino acids at the F0 cleavage site in virulent viruses. Usually the sequence is ^{113}RQK/RR*F^{117} in virulent viruses, but most have a basic amino acid at position 112 as well. In contrast, viruses of low virulence usually have the sequence ^{113}K/RQG/ER*L^{117}. Thus, there appears to be the requirement of a basic amino acid at residue 113, a pair of basic amino acids at 115 and 116 plus a phenylalanine at residue 117 if the virus is to be virulent for chickens. The presence of these basic amino acids at these positions means that cleavage can be effected by a protease or proteases present in a wide range of host tissues and organs; but for lentogenic viruses, cleavage can occur only with proteases recognising a single arginine, i.e. trypsin-like enzymes. Therefore, in host cells the replication of lentogenic viruses is restricted to areas with trypsin-like enzymes, such as the respiratory and intestinal tracts, whereas virulent viruses can replicate and cause damage in a range of tissues and organs, resulting in a fatal systemic infection.

That the virulence of NDV strains is governed by the F0 cleavage site is sufficiently accepted that it has been incorporated into the definition of ND adopted by the World Organisation for Animal Health (OIE):

"Newcastle disease is defined as an infection of birds caused by a virus of avian paramyxovirus serotype 1 (APMV-1) that meets one of the following criteria for virulence:

a) The virus has an intracerebral pathogenicity index (ICPI) in day-old chicks (Gallus gallus) *of 0.7 or greater.*
or

b) Multiple basic amino acids have been demonstrated in the virus (either directly or by deduction) at the C-terminus of the F2 protein and phenylalanine at residue 117, which is the N-terminus of the F1 protein. The term 'multiple basic amino acids' refers to at least three arginine or lysine residues between residues 113 and 116. Failure to demonstrate the characteristic pattern of amino acid residues as described above would require characterisation of the isolated virus by an ICPI test."

In this definition, amino acid residues are numbered from the N-terminus of the amino acid sequence deduced from the nucleotide sequence of the F0 gene, 113–116 corresponds to residues -4 to -1 from the cleavage site.' (Alexander 2008).

Various studies using cDNA clones of NDV and reverse genetics techniques have been undertaken to determine the precise minimum amino acid motif at the F0 cleavage site to confer virulence (Peeters et al. 1999). De Leeuw et al. (2003) generated a range of viruses with substituted amino acids at the F0 cleavage site. They concluded that virulence required F at position 117, R at 116, K or R at 115 and R not K at 113. Interestingly, all their generated mutants reverted to the virulent motifs ^{112}RRQRR*F^{117} or ^{112}RRQKR*F^{117} after a single passage in chicks.

Although it appears that the amino acid sequence of the F0 protein cleavage site is the primary influence on real or potential virulence of NDVs, it should be borne in mind that other factors associated with other virus genes and proteins may cause variations in virulence. For example, using reverse genetic techniques it has been demonstrated that the HN protein may influence virulence (Huang et al. 2004; Römer-Oberdörfer et al. 2006). Similarly, the V protein has been shown to inhibit apoptosis in infected cells and its absence also may affect virulence (Mebatsion et al. 2001).

2.5 Transmission

It is reasonable to conclude for NDVs that infection can take place by virus inhalation, ingestion (Alexander 1988b) or contact with mucous membranes, especially the conjunctiva. Spread from one bird to another therefore depends on the availability of the virus from the infected bird in an infectious form. Excretion of virus is dependent on the organs in which the virus multiplies and, as discussed above, this may vary with viral pathotype. Birds showing respiratory disease presumably shed virus in aerosols of mucus that may be inhaled by or contact susceptible birds. Viruses that are mainly restricted to intestinal replication may be transferred by ingestion of contaminated faeces, either directly or in contaminated food or water, or by the production of small infective particles produced from dried faeces that may be inhaled or impinge on mucous membranes. The method of virus transmission probably depends on

many environmental factors that may drastically affect the rate of spread. Viruses transmitted by the respiratory route in a community of closely situated birds, such as in an intensive broiler house, may spread with alarming rapidity. Viruses excreted in the faeces and transmitted chiefly by the oral/faecal route may spread extremely slowly, especially if birds are not in direct contact, e.g. in caged layers.

The significance of vertical transmission of NDVs, especially virulent viruses, which usually cause cessation of egg-laying in susceptible diseased birds, is not clear. There have been some reports of isolation of vaccinal virus from eggs laid by infected birds (e.g. Pospisil et al. 1991), and in one significant report Capua et al. (1993) were able to isolate virulent NDV from cloacal swabs taken from birds with high antibody titres to NDV and from eggs laid by those birds as well as from the hatched progeny.

2.6 Spread

Several reviews have addressed the way in which NDV may be introduced into a country or area and then subsequently spread from flock to flock (Lancaster 1966; Lancaster and Alexander 1975; Alexander 1988b).

As discussed above, pools of NDV, usually of low virulence for poultry, are maintained in wild bird populations and primary introduction in poultry populations may occur by direct or indirect contact with wild birds. There is good evidence from analyses of viruses isolated in Ireland in 1990 and during the outbreaks of ND in Australia beginning in 1998 that, on rare occasions, viruses of low virulence may mutate to high virulence (Alexander 2001; Westbury 2001). Virulent NDV has also been generated experimentally from low-virulence virus by passage in chickens (Shengqing et al. 2002). Also, as discussed above, virulent NDV may be present in wild birds and other sectors; primary introduction may come from contact with them.

Once in the poultry sector, secondary spread has been attributed to a number of different methods such as: (1) movement of live birds; (2) contact with other animals; (3) movement of people and equipment; (4) movement of poultry products; (5) airborne spread; (6) contaminated poultry feed; (7) contaminated water and (8) vaccines.

While some of these are self-explanatory, others require more careful examination or are less obvious. For example, it is clear that susceptible infected birds could be moved and therefore spread ND during the incubation period of the disease. What may be of greater importance is that clinically normal, vaccinated birds have been shown to excrete virulent virus following challenge (Alexander et al. 1999; Guittet et al. 1993; Parede and Young 1990) and thus represent a serious threat in terms of overt disease to unvaccinated birds that may come in contact with them either directly, e.g. by trade in birds, especially for backyard flocks, or indirectly.

The role of airborne spread of NDV also requires some consideration. In the past, spread of the virus in the air had been considered an important route in some outbreaks (Dawson 1973), but of no importance in others (Utterback and Schwartz 1973) even involving the same virus. Hugh-Jones et al. (1973) attempted to assess the survival of airborne virus and were able to detect virus at 64 m downwind, albeit in very low titres, in very large amounts of air sampled, but not 165 m downwind of an infected premises. These authors stressed the importance of environmental conditions, particularly relative humidity, on the likelihood of airborne spread. In recent years, airborne spread has not been an issue in reported outbreaks and there has nearly always been an alternative, more likely cause, particularly the movement of poultry and the agency of humans.

2.7 Distribution

The widespread use of NDV vaccines in commercial poultry throughout the world makes the true geographical distribution of ND difficult to assess. It is usually considered that virulent NDV is either enzootic or a cause of regular epizootics in poultry throughout most of Africa, Asia, Central America and parts of South America. In more developed areas, such as Western Europe, sporadic epizootics occur on a fairly regular basis despite the widespread use of vaccination. The OIE (2007) lists only five countries where the disease has never occurred (French Guiana, Guyana, New Caledonia, Samoa and Vanuatu), 58 countries with “demonstrated clinical disease” between July 2005 and June 2007, and a further 14 countries with “unresolved disease events”.

2.8 Human Health

The first report in which NDV was described to be a human pathogen was published by Burnet, in 1943. In a review of ND as a zoonosis, (Chang 1981) recorded 35 published reports of NDV infections of humans between 1948 and 1971. Since that time, there have been few additional publications, which probably reflects the lack of serious, lasting effects resulting from such infections and the fact that they are commonplace.

The most frequently reported and best substantiated clinical signs in human infections have been eye infections, usually consisting of unilateral or bilateral reddening, excessive lachrymation, oedema of the eyelids, conjunctivitis and subconjunctival haemorrhage (Chang 1981). Although the effect on the eye may be quite severe, infections are usually transient, lasting no more than a day or two, and the cornea is not affected. Reports of other clinical symptoms in humans infected with NDV are less well substantiated, but occasionally a more generalised infection resulting in chills, headaches and fever, with or without conjunctivitis, has been reported (Chang 1981).

Human infections with NDV have usually resulted from direct contact with the virus, infected birds or carcases of diseased birds. There have been no reports of human to human spread. The types of people known to have been infected with NDV include: laboratory workers (usually as a result of accidental splashing of infective material into the eye), veterinarians in diagnostic laboratories (presumably as a result of contact with infective material during postmortem examinations), workers in broiler processing plants and vaccination crews, especially when live vaccines are given as aerosols or fine dust. Pedersden et al. (1990) reported significantly higher antibody titres to NDV in people who had known associations with poultry.

2.9 Conclusion

Newcastle disease remains enzootic in poultry or other avian sectors, such as racing pigeons, in many areas of the world and thus represents a constant threat to most birds reared domestically. Every commercial flock of poultry reared is influenced in some way by measures aimed at controlling ND and spread of the virus. A large majority of the countries rearing poultry commercially rely on vaccination to keep ND under control, but the disease nevertheless represents a major limiting factor for increasing poultry production in many countries.

The greatest impact of ND may well be on village or backyard chicken production. In developing countries throughout Asia, Africa, Central America and some parts of South America, the village chicken is an extremely important asset in that it represents a significant source of protein in the form of eggs and meat. However, ND is frequently responsible for devastating losses in village poultry. Social and financial restraints mean that the control of ND in village chickens in developing countries is extremely difficult, if not impossible. This situation impinges on the further development of commercial poultry production and the establishment of trade links.

References

Aldous EW, Alexander DJ (2001) Technical review: detection and differentiation of Newcastle disease virus (avian paramyxovirus type 1). Avian Pathol 30:117–128

Aldous EW, Fuller CM, Mynn JK, Alexander DJ (2004) A molecular epidemiological investigation of isolates of the variant avian paramyxovirus type 1 virus (PPMV-1) responsible for the 1978 to present panzootic in pigeons. Avian Pathol 33(2):258-269

Aldous EW, Manvell RJ, Cox WJ et al (2007) Outbreak of Newcastle disease in pheasants (Phasianus colchicus) in south-east England in July 2005. Vet Rec 160(14):482-484

Aldous EW, Mynn JK, Banks J, Alexander DJ (2003) A molecular epidemiological study of avian paramyxovirus type 1 (Newcastle disease virus) isolates by phylogenetic analysis of a partial nucleotide sequence of the fusion protein gene. Avian Pathol 32(3):239–356

Alexander DJ (1988a) Newcastle disease virus-An avian paramyxovirus. In: DJ Alexander (ed) Newcastle Disease Kluwer Academic, Boston, MA, pp11-22

Alexander DJ (1988b) Newcastle disease: Methods of spread. In: DJ Alexander (ed) Newcastle disease. Kluwer Academic, Boston, MA, pp 256-272

Alexander DJ (2000) Newcastle disease in ostriches (Struthio camelus) – A review. Avian Pathol 29:95-100

Alexander DJ (2001) Gordon Memorial Lecture. Newcastle disease. Br Poult Sci 42(1):5–22

Alexander DJ (2008) Newcastle disease World Organisation for Animal Health Manual of Diagnostic Tests and Vaccines for Terrestrial Animals, 6th ed. Chapter 2.3.14. OIE, Paris, pp 576-589

Alexander DJ, Banks J, Collins MS et al (1999) Antigenic and genetic characterisation of Newcastle disease viruses isolated from outbreaks in domestic fowl and turkeys in Great Britain during 1997. Vet Rec 145(15):417-421

Alexander DJ, Manvell RJ, Banks J et al (1999) Experimental assessment of the pathogenicity of the Newcastle disease viruses from outbreaks in Great Britain in 1997 for chickens and turkeys and the protection afforded by vaccination. Avian Pathol 28:501-512

Alexander DJ, Manvell RJ, Lowings JP et al (1997) Antigenic diversity and similarities detected in avian paramyxovirus type 1 (Newcastle disease virus) isolates using monoclonal antibodies. Avian Pathol 26(2):399-418

Alexander DJ, Parsons G (1986) Protection of chickens against challenge with the variant virus responsible for Newcastle disease in 1984 by conventional vaccination. Vet Rec 118(7):176-177

Alexander DJ, Russell PH, Collins MS (1984) Paramyxovirus type 1 infections of racing pigeons: 1 characterisation of isolated viruses. Vet Rec 114(18):444-446

Allan WH, Lancaster JE, Toth B (1978) Newcastle disease vaccines—their production and use FAO Animal Production Series No 10 FAO, Rome, 163pp

Allison AB, Gottdenker NL, Stallknecht DE (2005) Wintering of neurotropic velogenic Newcastle disease virus and West Nile virus in double-crested cormorants (Phalacrocorax auritus) from the Florida Keys. Avian Dis 49(2):292–297

Arias-Ibarrondo J, Mikami T, Yamamoto H et al (1978) Studies on a paramyxovirus isolated from Japanese sparrow-hawks (Accipiter virgatus gularis). I. Isolation and characterization of the virus. Nippon Juigaku Zasshi 40:315-323

Beach JR (1942) Avian pneumoencephalitis. Proceedings of the Annual Meeting of the US Livestock Sanitary Association 46:203-223

Beach JR (1944) The neutralization in vitro of avian pneumoencephalitis virus by Newcastle disease immune serum. Science 100(2599):361-362

Beard CW, Hanson RP (1984) Newcastle disease. In: Hofstad MS, Barnes HJ, Calnek BW et al (eds) Diseases of poultry 8th ed. Iowa State University Press, Ames, pp 452-470

Biancifiori F, Fioroni A (1983) An occurrence of Newcastle disease in pigeons: virological and serological studies on the isolates. Comp Immunol Microbiol Infect Dis 6(3):247-252

Blaxland JD (1951) Newcastle disease in shags and cormorants and its significance as a factor in the spread of this disease among domestic poultry. Vet Rec 63:731-733

Burnet FM (1943) Human infection with the virus of Newcastle disease of fowl. Med J Aust 2:313–314

Capua I, Scacchia M, Toscani T, Caporale V (1993) Unexpected isolation of virulent Newcastle disease virus from commercial embryonated fowls' eggs. Zentralbl Veterinarmed B 40(9-10):609-612

Chang PW (1981) Newcastle disease. In: Beran GW (ed) CRC handbook series in zoonoses section B: Viral zoonoses, volume II. CRC, Baton Raton pp261-274

Cleary L (1977) Succès de reproduction du cormoran à aigrettes, Phalacrocorax auritus auritus, sur trois Îles du St Laurent, en 1975 et 1976. MSc Thesis, L'Université Laval, pp 1-68

Collins MS, Bashiruddin JB, Alexander DJ (1993) Deduced amino acid sequences at the fusion protein cleavage site of Newcastle disease viruses showing variation in antigenicity and pathogenicity. Arch Virol 128(3-4):363-370

Czeglédi A, Ujvàri D, Somogyi E et al (2006) Third genome size category of avian paramyxovirus serotype 1 (Newcastle disease virus) and evolutionary implications. Virus Res 120(1-2):36-48

Dawson, PS (1973) Epidemiological aspects of Newcastle disease. Bull OIE 79, 27-34

Doyle TM (1927) A hitherto unrecorded disease of fowls due to a filter-passing virus. J Comp Pathol Therapeut 40:144-169

Doyle TM (1935) Newcastle disease of fowls. J Comp Pathol Therapeut 48:1-20

Erickson GA, Maré CJ, Gustafson GA et al (1977) Interactions between viscerotropic velogenic Newcastle disease virus and pet birds of six species. I. Clinical and serologic responses, and viral excretion. Avian Dis 21(4):642-654

Guittet M, Le Coq H, Morin M et al (1993) Proceedings of the Tenth World Veterinary Poultry Association Congress, Sydney, p 179

Halasz F (1912) Contributions to the knowledge of fowlpest. Veterinary Doctoral Dissertation, Communications of the Hungarian Royal Veterinary School, Patria, Budapest pp 1-36

Hannoun C (1977) Isolation from birds of influenza viruses with human neuraminidase. Dev Biol Stand 39:469-472

Heckert RA (1993) Ontario. Newcastle disease in cormorants. Can Vet J 34(3):184

Herczeg J, Pascucci S, Massi P et al (2001) A longitudinal study of velogenic Newcastle disease virus genotypes isolated in Italy between 1960 and 2000. Avian Pathol 30:163-168

Herczeg J, Wehmann E, Bragg RR et al (1999) Two novel genetic groups (VIIb and VIII) responsible for recent Newcastle disease outbreaks in Southern Africa, one (VIIb) of which reached Southern Europe. Arch Virol, 144(11):2087–2099

Huang Z, Panda A, Elankumaran S et al (2004) The hemagglutinin-neuraminidase protein of Newcastle disease virus determines tropism and virulence. J Virol 78(8):4176-4184

Hugh-Jones M, Allan WH, Dark FA, Harper GJ (1973) The evidence for the airborne spread of Newcastle disease. J Hyg 71(2):325-339

Jørgensen PH, Handberg KJ, Ahrens P et al (1999) An outbreak of Newcastle disease in free-living pheasants (Phasianus colchicus). Zentralbl Veterinarmed B 46(6):381-387

Kaleta EF, Baldauf C (1988) Newcastle disease in free-living and pet birds. In: Alexander DJ (ed) Newcastle disease. Kluwer Academic, Boston, pp 197-246

Kinde H, Uzal F, Hietala S et al (2003) The diagnosis of exotic Newcastle disease in southern California: 2002-2003. Proceedings of the 46th Annual Conference of the American Association of Veterinary Laboratory Diagnosticians San Diego, CA, October 11-13 2003

Kraneveld FC (1926) A poultry disease in the Dutch East Indies. Nederlands-Indische Bladen voor Diergeneeskunde 38:448-450

Kuiken T (1998) Newcastle disease and other causes of mortality in double-crested cormorants (Phalacrocorax auritus). PhD Thesis University of Saskatchewan, 174 p

Lamb RA, Collins PL, Kolakofsky D et al (2005) Family Paramyxoviridae. In: Fauquet CM, Mayo MA, Maniloff J et al (eds) Virus taxonomy, Eighth Report of the International Committee on Taxonomy of Viruses. Elsevier, San Diego, pp 655-668

Lancaster JE (1966) Newcastle disease - a review 1926-1964. Monograph no 3, Canada Department of Agriculture, Ottawa

Lancaster JE, Alexander DJ (1975) Newcastle disease: virus and spread. Monograph no 11, Canada Department of Agriculture, Ottawa

de Leeuw OS, Hartog L, Koch G, Peeters BP (2003) Effect of fusion protein cleavage site mutations on virulence of Newcastle disease virus: non-virulent cleavage site mutants revert to virulence after one passage in chicken brain. J Gen Virol 84(Pt 2):475-484

Lomniczi B, Wehmann E, Herczeg J et al (1998) Newcastle disease outbreaks in recent years in Western Europe were caused by an old (VI) and a novel genotype (VII). Arch Virol 143(1):49-64

Macpherson LW (1956) Some observations on the epizootiology of Newcastle disease. Can J Comp Med 20(5):155-168

McFerran JB, McCracken RM (1988) Newcastle disease. In: Alexander DJ (ed) Newcastle Disease, Kluwer Academic, Boston, pp 161-183

Mebatsion T, Verstegen S, De Vaan LT et al (2001) A recombinant Newcastle disease virus with low-level V protein expression is immunogenic and lacks pathogenicity for chicken embryos. J Virol 75(1):420-428

Mixson MA, Pearson JE (1992) Velogenic neurotropic Newcastle disease (VNND) in cormorants and commercial turkeys FY 1992. In: Proceedings of the 96th Annual Meeting of the United States Animal Health Association, Louisville, Kentucky, 1992, pp 357-360

OIE (2007) List of countries by disease situation http://ww-woieint/wahid-prod/publicphp?page=disease_status_lists accessed 20th September 2007

Panigrahy B, Senne DA, Pearson JE et al (1993) Occurrence of velogenic viscerotropic Newcastle disease in pet and exotic birds in 1991. Avian Dis 37(1):254-258

Parede L, Young PL (1990) The pathogenesis of velogenic Newcastle disease virus infection of chickens of different ages and different levels of immunity. Avian Dis 34(4):803-808

Pedersden KA, Sadasiv EC, Chang PW, Yates VJ (1990) Detection of antibody to avian viruses in human populations. Epidemiol Infect 104:519-525

Peeters BP, de Leeuw OS, Koch G, Gielkens AL (1999) Rescue of Newcastle disease virus from cloned cDNA: evidence that cleavability of the fusion protein is a major determinant for virulence. J Virol 73(6):5001-5009

Pospisil Z, Zendulkova D, Smid B (1991) Unexpected emergence of Newcastle disease virus in very young chicks. Acta Vet Brno 60:263-270

Römer-Oberdörfer A, Veits J, Werner O, Mettenleiter TC (2006) Enhancement of pathogenicity of Newcastle disease virus by alteration of specific amino acid residues in the surface glycoproteins F and HN. Avian Dis 50(2):259-263

Rott R, Klenk HD (1988) Molecular basis of infectivity and pathogenicity of Newcastle disease virus. In: Alexander, DJ (ed) Newcastle disease, Kluwer Academic, Boston, pp 98-112

Senne DA, Pearson JE, Miller LD, Gustafson GA (1983) Virus isolations from pet birds submitted for importation into the United States. Avian Dis 27(3):731-744

Shengqing Y, Kishida N, Ito H et al (2002) Generation of velogenic Newcastle disease viruses from a nonpathogenic waterfowl isolate by passaging in chickens. Virology 301(2):206–211

Utterback WW, Schwartz JH (1973) Epizootiology of velogenic viscerotropic Newcastle disease in southern California, 1971-1973. J Am Vet Med Assoc 163(9):1080-1088

Westbury H (2001) Commentary Newcastle disease virus: an evolving pathogen. Avian Pathol 30:5-11

Wobeser G, Leighton FA, Norman R et al (1993) Newcastle disease in wild water birds in western Canada. Can Vet J 34(6):353-359