

Hygiene Protocols for the Prevention
and Control of Diseases
(Particularly Beak and Feather Disease)
in Australian Birds

Newcastle Disease



Australian Government

Department of the Environment and Heritage

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Newcastle Disease

Definition: Newcastle disease (ND) is an acute, mild to severe, highly infectious and pathogenic disease of birds caused by a paramyxovirus. Depending on the viral strain, exposure to ND virus (NDV) may lead to a subclinical infection or a range of syndromes including respiratory, intestinal and nervous signs, with mortalities up to 100%.

The natural hosts of NDV are domestic poultry, including chickens, turkeys, ducks, geese, pigeons, quail, pheasants, guinea fowl and ostriches, and many species of captive caged birds and wild birds (Alexander 2000). Susceptibility varies between species, with chickens the most likely to show clinical ND, and water birds the least likely to be affected clinically (Kaleta and Baldauf 1988).

ND takes its name from the town of Newcastle-upon-Tyne, where the first outbreak was reported in 1926.

Synonyms: ND; exotic ND; Asiatic ND; VVND; pneumoencephalitis; *Maladie de Newcastle*; *Enfermedad de Newcastle*; *Pneumoencephalitis Avium*; *Pseudopestis Avium*; Ranikhet disease. It is often incorrectly called Newcastle's disease. The modern poultry industry is highly susceptible to NDV, since over the last 40 years, the industry has developed into a highly integrated production system, the interconnections and interrelationships of which provide a means for the rapid spread of this devastating disease should it enter the system. Since all avian species are probably susceptible to NDV, and the different species differ in their susceptibilities to a particular NDV strain, any species can be a source of virus for any other species.

Epidemiology: Can cause a conjunctivitis in humans.

Since the emergence (or recognition) of this apparently new disease of chickens, there have been three panzootics. The **first panzootic** began with the emergence of ND in chickens in England and South East Asia in 1926. The disease had disappeared from England by 1928. Two waves of spread have been recognised. The first occurred in the Far East and Eastern Europe between 1926 and 1942, and the second in Europe, Africa and the Americas in the late 1940's and early 1950's. It has been postulated that between 1926 and the early 50's the disease circulated in South-East Asia and that chance introductions from this source to other countries occurred, e.g., England in 1926, Australia in 1930 and Kenya in 1935. The slow spread of the disease was attributed to the underdeveloped state of the poultry industry, consisting mainly of backyard flocks with no international trade.

The **second panzootic** apparently arose in the late 1960's in the Middle East. This panzootic spread to other continents faster than the first, due to the international air trade in psittacine species, which rapidly spread the virus.

The **third panzootic** is the current neurotrophic ND which occurs mainly in racing pigeons. The NDV involved shows marked variation from classical strains and has been

isolated from flocks of domestic fowl in Great Britain, Germany, Saudi Arabia, Austria and Uganda, turkeys in Israel, ducks in Switzerland, and psittacine species in Germany (Alexander *et al*, 1985).

Aetiology: NDV is placed in the genus *Rubulavirus* of the subfamily *Paramyxovirinae* and the family *Paramyxoviridae*. The genus *Rubulavirus* contains avian paramyxoviruses 1-9, with Newcastle disease virus being avian paramyxovirus 1 (APMV-1, PMV-1). Although there are different pathotypes of NDV, antigenically they are the same. They each vary in their spreadability, transmissibility, immunogenicity, virulence, heat susceptibility, ability to elute erythrocytes, and so on.

Transmission: There are two main reservoirs of NDV. Avirulent virus is mainly associated with waterfowl, and highly virulent viruses with tropical birds, especially psittacine species. Some avian Orders have a high level of susceptibility to NDV (*Phasianiformes* [gallinaceous birds, pheasants]; *Psittaciformes* [parrot-like birds]; *Struthioniformes* [ratites]; and *Columbiformes* [pigeons and doves]), others have an intermediate susceptibility (*Strigiformes* [owls]; *Falconiformes* [falcons]; *Accipitriformes* [eagles]; *Ciconiiformes* [storks]; *Sphenisciformes* [penguins]; and *Passeriformes* [sparrows and song birds]), while others have minimal susceptibility (*Anatiformes* [waterfowl]; *Pelicaniformes* [pelicans, shags]; *Ralliformes* [coots]; *Lariformes* [gulls]; and *Carianiformes* [cranes]). This grouping reflects a general rather than a specific predictability.

Thus birds living in close contact with water appear to be resistant to developing clinical signs after infection, and it has been postulated that such birds have had a long phylogenetic relationship with NDV, since the virus survives well in sea or fresh water, and waterfowl would have received considerable exposure during their evolution. Granivorous and fructivorous birds are moderately susceptible to NDV, while omnivorous birds are most sensitive. Gregarious species are more likely to develop ND than solitary species. Among mammals, man is the only species in which infection has occurred naturally, and this usually results in a mild conjunctivitis. The ability of the virus to multiply in mammalian hosts is limited, and mammals play little or no part in the natural spread of the disease, except during an outbreak when they might passively transfer virus.

NDV spreads along three avenues: the domestic poultry industry with its vertical integration; intra- and intercontinental avian migration, and the pet bird trade. The virus is **not** transmitted vertically. Some embryos will be infected before being enclosed by the shell, but these die before hatching. It is thus theoretically possible to hatch ND-free eggs from a viraemic and shedding flock.

The most important manner of spread is the movement of domestic poultry, including day-old chicks, hatching eggs, live and dead birds and poultry offal, the migratory movements of infected wild avian species, the mechanical transfer of virus by rodents and wind transmission of the virus. Spread of the virus is facilitated by its high resistance to adverse environmental conditions, its wide avian host range, and its ability to persist in poultry carcasses and offal. Under modern conditions of poultry management, airborne dissemination of virus has become of particular importance in local spread of the virus, particularly in houses utilising exhaust fans.

Signs

Poultry: The clinical signs of ND in susceptible chickens vary, depending on the virulence and tissue tropism of the virus. Disease caused by viscerotropic velogenic ND (VVND) virus starts suddenly and progresses rapidly. Birds stop eating, have ruffled feathers, and become listless. The combs and wattles become cyanotic and oedematous, and there is a serous to mucopurulent ocular discharge and conjunctivitis. A greenish-yellow diarrhoea is commonly present. Rales, sneezing, coughing, nasal discharge, and laboured breathing with gaping and extended head and neck, may be seen. Nervous signs (tremors, torticollis, opisthotonus, incoordination) are usually seen only in older birds, and then only when the disease in the flock is advanced. Egg production usually ceases. Morbidity and mortality may reach 100%. Infection with mesogenic ND viruses results in a less severe disease, with respiratory and nervous signs predominating and severe effects of egg production. Younger birds are more severely affected (Alexander, 2003; Allan *et al.*, 1978).

Lentogenic ND viruses may cause mild respiratory signs and egg production drops, with negligible to low mortality in young chickens. Infection with avirulent ND viruses is unapparent, with seroconversion being the only evidence of infection. However, avirulent ND viruses have been implicated in respiratory disease complexes in broiler chickens.

Lesions: The predominant lesions in an outbreak of VVND in the domestic fowl are focal diphtheritic, necrotic or haemorrhagic lesions throughout the alimentary tract. Velogenic and mesogenic pathotypes cause serous to catarrhal or haemorrhagic exudation in the trachea, with congestion and oedema of the lungs and airsacculitis. The ovary is usually flaccid and contains congested, discoloured, degenerating follicles. Yolk may be present in the abdominal cavity. Subcutaneous oedema may be present over the head, eyelids, comb, wattles and neck, but this is not as marked as that seen in outbreaks of avian influenza in the domestic fowl. The conjunctivae may be swollen, oedematous and haemorrhagic, so much so that they protrude over the lids. Infection with lentogenic strains usually causes only a mild tracheitis and airsacculitis.

Microscopic lesions seen in the domestic fowl include hyalinisation of capillaries and arterioles, hyaline thrombosis and necrosis of endothelial cells with associated oedema and haemorrhage. Following infection with VVND virus, necrotic haemorrhagic foci develop in lymphoid aggregates throughout the gastrointestinal tract. Central nervous lesions are most commonly seen in the cerebellum, brain stem, mid-brain and spinal cord, and consist of neuronal degeneration, gliosis, endothelial cell hypertrophy and perivascular lymphocytic accumulation. Lesions in the trachea vary with the virulence of the virus strain. They range from deciliation and degeneration of epithelial cells to epithelial hyperplasia and infiltration of the *lamina propria* by lymphocytes. Degeneration and necrosis of lymphoid tissues, together with vascular damage and haemorrhage may be present in parenchymatous organs.

Pigeons: In 1981, an infectious disease with nervous signs was first seen in Mediterranean racing pigeons. The earliest reports of the disease were from Italy in 1981. In 1982, there were reports in the Continental lay press of the disease. It is likely, however, that the disease

started in Iraq in 1977 and spread to Egypt by 1981. It was probably misdiagnosed as pigeon herpesvirus encephalitis, since the pigeon NDV variant was later grown from cultures of herpesviruses isolated from diseased pigeons in Iraq in 1977. The disease was introduced into Belgium in 1981 by two racing pigeons which were imported from Italy. The disease spread rapidly in Belgium and Germany because of the highly organised pigeon racing and extensive trade in these birds. In April 1983, the Ministry of Agriculture, Fisheries and Food placed a ban on the racing and importation of pigeons from the Continent to Great Britain. In spite of this, the disease was diagnosed in a loft in Cornwall in June of 1983 and was widespread in lofts in Great Britain six months later. The disease spread internationally and the virus has been isolated from pigeons in most of Europe, as well as in Israel, Egypt, The Sudan, Uganda, South Africa, Hong Kong, Japan, Canada and the United States of America (Alexander *et al.* 1985).

The incubation period of pigeon ND varies from a few days to several weeks, so that in any outbreak, new clinical cases continue to appear in the loft for up to 5-8 weeks after it appeared in the loft. Initially, pigeons drink excessively, with intestinal signs (watery to haemorrhagic diarrhoea) appearing first followed by nervous signs (head tremor, torticollis, paralysis of wing(s) and/or leg(s), and loss of visual acuity, with affected pigeons pecking at and missing grain). If the disease is contracted during moulting, then poor feathering may result. Respiratory signs are always absent. Pigeons may return to health after a convalescence of up to 6 months, but can have a persistent diarrhoea (chronic enteritis for several months. All viruses isolated from diseased racing pigeons were similar and of PMV-1 serotype.

The pigeon variant NDV caused ND in domestic fowl in Great Britain in 1984. At first, there appeared to be no direct or indirect contact between these poultry flocks and pigeons. Subsequent investigation revealed that the flocks had been fed grain which had been stored in the Liverpool docks, and that this grain was contaminated by pigeon carcasses and faeces. Pigeon PMV-1 was isolated from these carcasses and from the grain itself. In Great Britain it is common to feed egg-laying bird rations which are merely mixed at the mill, so that the virus was not heated at any stage. The majority of the flocks affected received such a ration, and other outbreaks probably resulted from secondary spread. The source of four outbreaks could not be determined. During 1983 192 racing pigeon lofts were confirmed as infected with NDV. In 69% of these outbreaks, pigeon racing was implicated in the spread of the disease, while some outbreaks were traced to visits by owners to infected lofts. Vaccination of birds against NDV had been banned in Great Britain in 1981. Because of the pigeon ND, vaccination of pigeons using an inactivated oil emulsion vaccine was permitted from September 1983. In flocks effectively vaccinated, no outbreaks occurred. Of 866 outbreaks of the disease in pigeon lofts in Great Britain since, 92% occurred in unvaccinated flocks, and 7% in either inadequately vaccinated flocks or flocks vaccinated after clinical signs appeared in the loft (Alexander *et al.* 1985).

Game Birds and Turkeys

Pheasants, quail and partridges are more susceptible to NDV than guinea fowl and turkeys. These species have been associated with outbreaks of ND in the domestic fowl. Turkeys may be long-term carriers of the virus, since NDV has been isolated from a turkey kept in isolation for 12 months. Generally the occurrence of ND in game birds and waterfowl follows an outbreak of the disease in the domestic fowl.

Wild Waterfowl

At times other than during annual migration, waterfowl are unlikely to receive significant exposure to NDV. However, when they become infected, they do not develop clinical signs, and remain carriers and shed it for long periods. Most sampling of waterfowl has been undertaken just prior to or during annual migration, when the population density is high and the birds have spent significant periods in the one area. In this aquatic environment, intestinal infection and transmission by the faecal-oral route would spread NDV (and other pathogens). During migration, weakened and diseased waterfowl are unlikely to complete their migration, and will either die or fall victim to predators. Evolutionary environmental adaptations have ensured that exposure to pathogens is minimised. At breeding, most birds pair off and tend to separate from other pairs, tend to build a new nest each year, minimising exposure to pathogens, they remove the droppings of offspring, and also any weakened and underdeveloped offspring. Fledglings also tend to stay with their own parents, and avoid living for long periods at the one site. Breeding, hatching and rearing usually coincides with a period of abundant food supply, thus ensuring that the offspring receive optimal nutrition. Humoral antibody will presumably effectively protect offspring in a stable environment. Any pathogens that infect the environment are rapidly inactivated by ultraviolet light.

In Western Australia, NDV was isolated from aquatic species, with a high prevalence in pelagic species. All NDV isolates from these birds were avirulent for the domestic fowl. Aquatic birds also carry and excrete avirulent NDV, and lentogenic viruses circulate among them. Velogenic viruses have not been isolated from wild aquatic birds, although they can carry and excrete velogenic viruses in captivity. It is possible that migratory waterfowl might shed NDV into the water supply of domestic poultry, but it is unlikely that such viruses will be virulent.

Domestic Pet Birds

The development of clinical signs in pet and free-living non-aquatic birds is the same as that observed in the domestic fowl. As in the domestic fowl, the outcome of infection depends on the virulence of NDV, dosage and method of entry of the virus; the age of the challenged bird and whether it has innate resistance; whether the bird has passive or active antibody; and whether the bird is undergoing stress at the time of challenge.

The transport of psittacine species has been associated with many of the outbreaks of ND in domestic fowl that occurred throughout the world in 1970-1971. Because of the relationship between VVND viruses and the rapid international movement of psittacine species, many countries imposed quarantine restrictions on the importation of such birds.

It is not known how exotic birds become infected with VVND virus. As with waterfowl, environmental adaptations ensure that exposure to pathogens is minimised. However, once birds are captured and either imported or smuggled into a country, their caging results in a breakdown of these protective mechanisms, and infectious agents accumulate. Infection in transit when birds from different sources are mixed must also be considered. Modern air transport ensures the rapid transport of such birds to their destination and so many birds will be incubating viral diseases on their arrival at a quarantine station and show no clinical signs.

Recovered psittacine birds may carry and excrete NDV for months (Kaletea and Baldauf 1988).

Diagnosis

Although a clinician may strongly suspect ND in a flock of domestic fowl, the disease cannot be unequivocally diagnosed clinically or pathologically, because of the similarities of many of the clinical signs to those of other poultry diseases. Final diagnosis is based on isolation and characterisation of the virus, which must include pathotyping, since there is a wide diversity in the disease-causing potential of NDV isolates. Pathotyping is necessary, since there have been instances of the isolation of non-pathogenic, vaccinal or endemic strains from birds with clinical disease typical of virulent strains, as well as of the isolation of pathogenic strains from asymptomatic birds. The main objective of characterising NDV isolates is to assess their pathogenicities for the domestic fowl. Suitable specimens for isolation are tracheal swabs, as well as tissues taken from the respiratory and alimentary tracts at autopsy. The virus will grow readily in embryonated eggs of the domestic fowl, and a wide range of avian and mammalian cell cultures. Isolation of NDV is confirmed by haemagglutination inhibition by monospecific antisera.

Once the virus is identified, various pathotyping tests may be undertaken: the mean death time (MDT) in 9-11 day-old embryonated eggs of the domestic fowl; the intracerebral pathogenicity index (ICPI) in one day-old chicks of the domestic fowl; and the intravenous pathogenicity index (IVPI) in six week old chicks of the domestic fowl. Using these methods, ND viruses are broadly grouped into three pathotypes: velogenic (highly pathogenic); mesogenic (moderately pathogenic); and lentogenic (mildly pathogenic to non-pathogenic). Velogenic viruses are further subdivided according to the predominant clinical syndrome produced in infected domestic fowl, into viscerotropic velogenic (VVND), neurotrophic velogenic and pneumotropic velogenic strains.

Reverse-transcription polymerase chain reaction (RT-PCR) and sequencing of the cleavage site may be used to determine pathogenicity of NDV isolates (Alexander 1997). Panels of mouse monoclonal antibodies have also been used to establish antigenic profiles of NDV isolates (Alexander *et al.* 1997).

The HI test is most widely used serological test (Alexander 2000). APMV-1 may show some antigenic cross-reactions in HI tests with APMV-3 and APMV-7 (Alexander 1997), but these can be resolved by the use of suitable antigen and antiserum controls.

ND must be differentiated from AE, encephalomalacia, thiamine deficiency, MD (neural form), virulent avian influenza, avian cholera.

Treatment:

None. ND is an OIE listed disease agent and is notifiable in all Australian states and territories. Outbreaks of ND occurred in Australia in 1930, 1932, 1998, 1999, 2000 (Westbury 2001), and also in 2002 in NSW.

Control:

Government quarantine. Vaccination

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