

New Castel Disease In Ethiopia: A Review

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INTRODUCTION

Livestock production is a major component of Ethiopian economy. In developing countries like Ethiopia, livestock production goes well beyond food production; sales of livestock and their products provide immediate cash income to farmers and foreign exchange for the countries (FAO, 1990).

Poultry production will assist in poverty alleviation and the improvement of food security. The increased availability of village chickens and eggs should result in an improved intake of protein by the population and increased access to cash and other sources. Chickens are often essential elements of female-headed and poor households. This is particularly important contribution in areas where child malnutrition is common. Malnutrition wider implications for development because protein-energy malnutrition in children inhibits their growth, increase their risk of morbidity, affect their mental development, and reduce their subsequent school performance and labour productivity (Alders and Spradbrow, 2001).

Chickens in traditional village poultry systems provide scarce animal protein in the form of meat and eggs, and are available for sale or barter in societies where cash is not abundant. They are generally owned and managed by women and children. Chickens also fulfill a range of other functions for which it is difficult to assign monetary value. They are active in pest control, provide manure, are required for special festivals and to meet social obligations, they are essential for many traditional ceremonies and traditional treatment of illness (Robyn and Spreadbrow, 2001).

Raising poultry has along line of tradition in Ethiopia and the production system shows a clear distinction between the traditional subsistence, low input system compared with small and large-scale commercial systems, which use relatively advanced technology.

Indigenous birds are raised under traditional; or "backyard" conditions without any input and are difficult to monitor. They comprise up to 99% of the total estimated, 56.6 million chickens in Ethiopia, while 1% are an exotic breed maintained under intensive management system (Hagos *et al.*, 2003).

Most poultry improvement programs in developing countries including Ethiopia have been directed towards the introduction of specialized exotic breeds or cross breeding and management intensification. Genetic improvement could be achieved either through selection and controlled mating or by introducing exotic chickens (Aklilu, 2000).

There are many constrains to village chicken production including a range of bacteria and other viral disease, internal and external parasites, poor nutrition and predation (Robyn and spradbrow, 2001).

The major constraint to productions of village chickens in many developing countries is Newcastle disease. In these countries circulating strains of NCD virus are capable of causing 100% mortality in unprotected flocks, outbreaks of NCD are unpredictable and discourage villagers from paying proper attention to the husbandry and welfare of their chickens. The Importance of NCD is indicated by the fact that NCD has a local name in many countries.

Newcastle disease is a serious and commonly fatal viral poultry disease, which is present all over the world. In many tropical and subtropical countries, virulent strains of NCD virus are endemic (Barman, 2002). In most developing countries, NCD is the most important infections disease affecting village chickens and causes great economic losses (Alders and Spradbrow, 2001). The disease is caused by a paramyxovirus, which is readily inactivated by formaline, alcohol, merthiolate. NCD virus may persist in undispersed chicken faeces for more than six months but under village conditions the virus can survive outside the host for more than one month.

NCD viruses occur in three pathotypes: Lentogenic, mesogenic, and velogenic. The most virulent (velogenic) isolates are further sub-divided into neurotropic and viscerotropic. In chickens NCD is characterized by lesions in the brain or gastrointestinal tract, morbidity rates near 100%, and mortality rates as high as 90% in susceptible chickens. Neurological symptoms or severe depression are the most obvious clinical signs of NCD

Such as loss of appetite, anorexia, yellowish or greenish diarrhea and paralysis and some unvaccinated birds may be found dead with no detected sign of prior illness (Serkalem *et al.*, 2005).

The prevailing management system, which involves exposure to wild birds, selling or giving away of sick birds, absence of vaccination programs and unrestricted contact between the different flocks are believed to facilitate the rapid spread of infection and persistence of the disease among the village chickens (Tadelle and Yelma, 2004).

NCD virus possesses two surface proteins that are important to the identification and behavior of the virus. The first, hemagglutinin /neuraminidase (HN) is important in the attachment and release of the virus from the host cells, in addition to its serologic identification. The other very important surface protein is the fusion (F) protein, which has a critical role in the pathogenesis of the disease.

Vaccination against NCD is routinely practiced throughout the world. In Ethiopia, intensive poultry farms vaccinate their poultry routinely, but poultry in extensive production systems are not vaccinated routinely due to lack of proper knowledge and it is difficult to transport and maintain conventional thermolabile vaccines in ambient temperatures ranging from 24^oc to 36^oc (Alders and Spradbrow, 2002).

Reports from six African countries, indicated that the mortality caused by NCD ranges from 50 – 100%. In addition, Spreadbrow (1991) indicated that NCD is the most devastating disease of village poultry responsible for heavy losses. It seriously interferes with the development of the poultry industry since farmers consider the disease as a serious constraint to investing in poultry (Tadelle and Yilma, 2004).

In Ethiopia, poultry diseases are considered to be the most important factor responsible for reducing both the number and productivity of chickens. Poultry diseases such as New castle disease, coccidiosis, and nutritional deficiency are considered to be the most endemic and the ones to incur huge economic losses. Study was conducted in 5 selected sites in central Ethiopia (Jeldu, sebeta, Awash-melka-kontire, Debreberhan and Nazareth.) has shown that is one of the major infectious disease which constrain poultry production (Hagos *et al.*,2003; Serkalem *et al.*,2005).

Definition

SYNONYMS: Avian Pneumoencephalitis, Ranikhet Disease, Pseudo-fowl pest, Pseudo poultry plagues, Avian pest, Avian distemper (Fikre, 2003).

Newcastle disease is an acute, rapidly spreading, viral disease of domestic poultry and other birds in which the respiratory signs (coughing, sneezing, rales) are often accompanied or followed by nervous manifestations and infections with some strains result in diarrhea and swelling of the head (Fraser 1986).

2.2 The history of NCD in the world

The disease was first encountered by Kraneveld in Java and reported in 1926. In 1927, Doyle described the disease in a flock of chickens in Newcastle on-Tyne, England and announced the cause to be a virus. By 1940, Newcastle disease had been recognized in the Philippines, Asia, Australia, and Africa. Later it appeared in continental Europe. Its identification in California in 1944 marked the first recognition of the malady in the western Hemisphere. It is widely scattered in the United States. Although the virus frequently produces, grave epizootics and high mortality rates among fowls in the Eastern hemisphere (Gillespie and Timely, 1981).

The word ‘poultry’ refers to all domesticated birds kept for the production of eggs and meat for human consumption. The term includes chickens, Turkeys, ducks, geese, quails, guinea fowls and other domesticated birds; poultry were domesticated later than sheep, pigs and cattle. Domesticated fowl from Asia in 2005 BC are probably the main the source of modern stocks. Domesticated geese were established in Egypt in 1500 BC and the earliest evidence of Turkey domestication was in Mexico in 2500 BC. Ducks were domesticated in China in 2500 BC. Poultry were initially domesticated for religious, culture and entertainment reasons and only later thought of as a food source (Aklilu, 2000).

History of Newcastle disease in Ethiopia

There is no clear record about the introduction of the virus to the country, however, NCD first occurred in and around seaports of the country and spread to the interior of the country along transport routes. The first documented outbreak of NCD in Ethiopia dates back to 1971 and reported from a small poultry farm in Asmera, Eritrea, located closed to a seaport and the province of the country. The first NCD virus reported was a velogenic type, which was classified according to the virulence of the strain and caused about 80% mortality (Kebreab *et.al.*, 2001). In the following years the disease spreads fast to other parts of the country. In 1972, outbreaks had been reported in Addis Ababa and in 1974 at the then Alemaya college of Agriculture poultry farm (Tadelle and Yilma, 2004) and also in 1995, NCD outbreaks in the surrounding areas of Debrezeit, Nazareth and Addis Ababa killed almost 50% of the local birds.

Velogenic strains of NCD virus are widely distributed throughout the country. It is possible to say that, currently, there are no low risk area remaining in Ethiopia. The disease has become endemic in village poultry

population and thus it recurs every year inflicting heavy losses. This disease entity has different local names in different areas and the most common one is "Fengle" which, means sudden dorsal prostration and signifies the acuteness and severity of the disease. However, the epidemiology of NCD in village poultry in Ethiopia including Tigray is poorly understood and there is no appropriate preventive strategy designed against the disease. This is due to lack of a disease monitoring capacity in the veterinary services. Farmers start to consider, therefore, losses due to the disease as normal and natural and they fail to report outbreaks to the veterinary authorities (Tadelle and Yilma, 2004).

In Tigray as the documents of the MOA of Tigray shows NCD outbreak has been occurring since 1991 and thus it recurs every year until now in village poultry population. (MOA, annual report 2005).

The national veterinary institute that was established in 1939 and has been fully engaged since then in the production of vaccine for prophylactic uses, started producing live NCD vaccine in 1974, an inactivated inject able oil emulsion NCD vaccine is also being produced since 1986/87. The institute has plans in the future to produce Newcastle clone vaccine (Kebreab *et al.*, 2001; Tadelle and Yilma, 2004).

Etiology

NCD is caused by a paramyxovirus genus Rubulavirus belonging to the paramyxoviridae family together with morbillivirus (causative agents of measles, rinder pest, canine distemper) and Pneumovirus (Fikre, 2003).

They possess two surface proteins that are important to the identification and behavior of the virus. The first, hemagglutinin/ neuraminidase (HN), is important in the attachment and release of the virus from the host cells in addition to its serologic identification. The other very important surface protein is the fusion (F) protein, which has a critical role in the pathogenesis of the disease (Serkalem *et al.*, 2005). There are at least nine known types of avian paramyxoviruses based on the antigenic make up of the hem agglutinin. NCD virus is the proto type virus for type 1 avian paramyxoviruses. On the basis of their antigenic relatedness in hemagglutination inhibition (HI) tests, paramyxovirus is divided in to nine serotypes (PMV1, 2,3...9). NCD virus belongs to PMV-1.

NCD virus can be classified into five patho types based on the clinical signs induced in infected chickens.

1. Viscerotropic velogenic Newcastle disease, (VVNCD) Characterized by an acute lethal infection of chickens of all ages, Hemorrhagic lesions of the digestive tracts are frequently present. It causes high mortality of 50-100% among adult birds. It is also called Asiatic NCD virus.
2. Neurotropic velogenic NCD virus characterized by an acute lethal infection of chickens of all ages. It produces respiratory and nervous signs.
3. Mesogenic NCD virus causes respiratory and some time, nervous signs with low mortality reduced egg production. Deaths are seen only in young birds.
4. Lentogenic NCD virus causes mild or impairment respiratory infections. Deaths confined to young chickens, which are commonly, used as live vaccines. It is also called hitcher's form.
5. Asymptomatic enteric form causes inapparent intestinal infection (Alders and spradbrow, 2001; Fikre, 2003).

Members of the family paramyxoviridae are enveloped RNA viruses, which possess non-segmented, single stranded genomes of negative polarity. They undergo capsid assembly in the cytoplasm and envelopment at the surface of infected cells (Jordan, 1990).

PMV-1 a single stranded RNA virus with an envelope bearing spikes, containing the components the initiate haemagglutinin (HA). The envelope itself contains components (2 glycoproteins and 7 polypeptides) that initiate the virus neutralizing antibodies in the host. PMV-1 is the most important pathogen for poultry. The viral envelope is sensitive to lipid solvents such as chloroform, ether, or alcohol and all known viricidal chemicals will destroy NCD virus with reasonable rapidity. However, proteinaceous matter may not be protective, but nullify the action of disinfectants.

Environmental conditions especially warm temperatures and solar radiation, facilitates the destruction by chemicals. NCD virus is destroyed by exposure to ultraviolet light rays and all virus activity is destroyed within 1 /one/ minute at 100⁰ c. At 56⁰c, the destruction of infectivity, HA activity and immunogenicity occur with in periods of 5 minutes to 6 hours. At 37⁰c, hours to days may be required to induce those changes. At 20⁰c and 8⁰c, months to years may pass before all reactivity in the virus is lost, and freezing temperatures inactivate NCD virus (Barman, 2002).

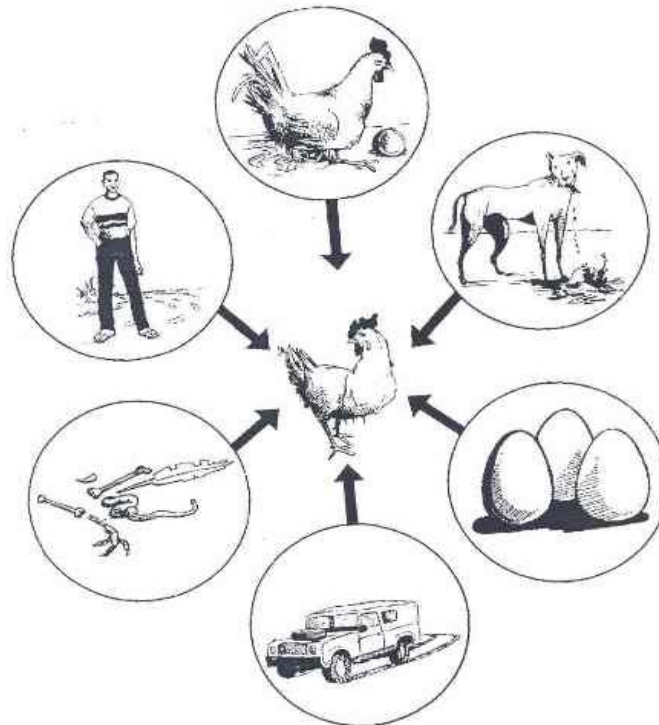
Epidemiology

Occurrence

Newcastle disease has worldwide distribution. In all poultry raising countries. NCD is endemic in many countries of Asia, the middle EAST, Africa, and Central and South America (Beard, 1989).

Transmission

NCD virus can infect through the respiratory tract, the ocular mucous membranes, and the digestive tract, although this usually requires very high doses of virus depending on the virulence of the strain (Alders and Spradbrow, 2001).



The mode of transmission from bird to bird is clearly dependent on the organs in which the virus multiplies. Birds showing respiratory disease presumably shed virus in aerosols of mucus, which may be inhaled by 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 inhalation of small infectious particles produced from dried faeces. Such considerations may drastically affect the rate of spread. Viruses transmitted by respiratory route in a community of closely situated birds, i.e. in an intensive broiler house, may spread with alarming rapidity. Viruses excreted in the faeces and transmitted chiefly by the oral/ fecal route may spread extremely slowly. Especially if birds are not in direct contact, i.e. caged layers (Jordan, 1990). NCD can be transmitted from one village to another via people, vehicles, animals, baskets, hoes, cages and infected products (egg shells, feathers, bones intestines, etc) (Alders and Spradbrow, 2001).

Host range and reservoir

The vast majority of birds are susceptible to infection with NCD virus, although the disease may vary enormously from one species to another. Chickens are highly susceptible, but other poultry species can be infected by NCD virus, and may play a role in the spread of NCD virus in extensively managed poultry. These birds include ducks, geese, turkeys, doves, and guinea fowl. Such birds can become infected with NCDV, shed the virus, and act as a source of infection to chickens, even if they do not develop clinical signs (Barman, 2002).

Panzootics

The history of NCD is marked by at least three panzootics in domestic birds. The first began with the emergence of the disease in fowl in the mid-1920s and spread slowly from the Far East throughout the world, despite the extremely rapid spread within some countries. The second panzootic appeared to emerge in fowl in the Middle East in the late 1960s and spread much faster than the first, reaching all continents and most countries by 1973. Several authors associated this rapid spread with the movement of infected psittacine birds as a consequence of the international pet bird market. A third panzootic was associated with a mainly neurotropic and enteric disease in pigeons. It too appeared to emerge in the Middle East and between the late 1970s and the mid-1980s spread throughout the world in racing, show, meat and feral pigeons; in some countries spread to other birds and poultry occurred (Jordan, 1990).

Seasonality of NCD outbreaks

Human activity influences the occurrence of NCD. In Asia, when seed rice is required for the seedbeds in paddy

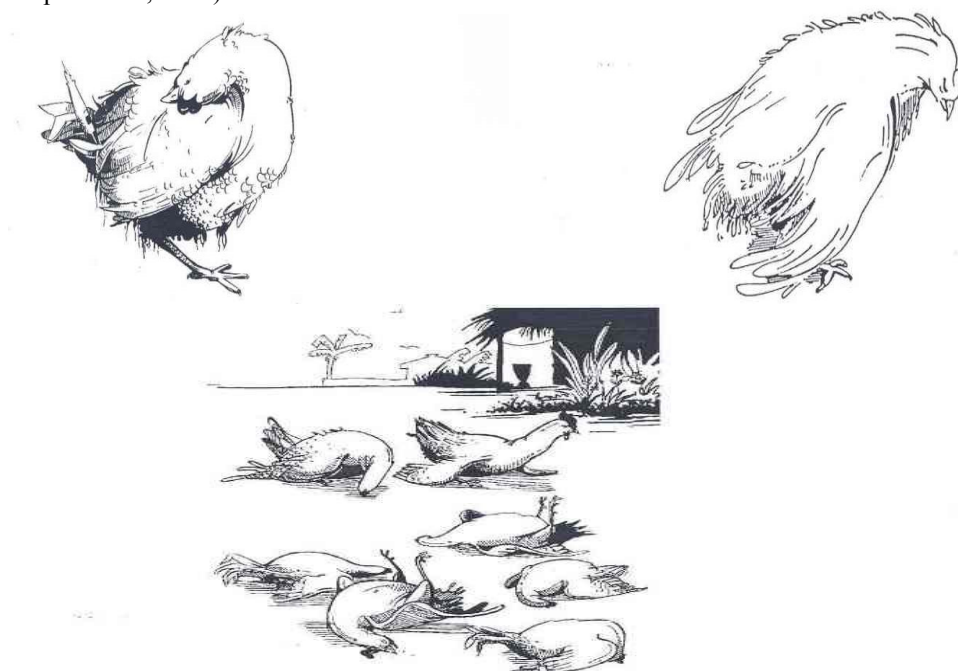
rice fields, chickens are sold to raise the funds to purchase seed. They also recognize that the disease may appear any time following the introduction of a diseased bird. In many areas the villagers recognize the season when NCD will occur, or they recognize the early cases, and they dispose of their chickens by sale, thus initiating or sustaining outbreaks. Most commonly NCD outbreak occurs from August to October and again in January (Alders and Sprad brow, 2001).

A key to the successful spread of NCD virus is the ability of the virus to survive in the dead host or excretions. In infected carcasses NCD virus may survive for several weeks at cool ambient temperatures or several years if held frozen. Faeces, in which virus may be present in high titers, also represents an excellent medium for the survival of NCD virus and even at 37⁰c infectivity has been retained for over a month (Jordan, 1990).

Clinical signs of NCD

Clinical signs vary considerably according to the virulence and tropism of the NCD virus involved, the species of bird, the age of host, the immune status of the host and environmental conditions. As a result, none may be regarded as a specific sign of NCD. The incubation period for NCD after natural exposure varies from 2 to 15 days.

- Chickens infected with virulent NCD virus strains may die without showing any signs of illness.
- The chicken flutes its feathers and appears to have its coat dragging on the ground.
- Lethargy and inappetence
- Respiratory signs such as mild rales and snick can be detected by careful observation.
- Severe respiratory distress and gasping
- Swelling of the head and neck
- Greenish diarrhea
- Marked decrease in egg production some times deformed eggs may be produced.
- Nervous signs of tremor, torticollis, convulsions and paralysis of wings and legs will not be seen until the disease is advanced.
- Mortality may be very high, often reaching 50 to 100%
- Other domestic poultry such as turkeys and pigeons may also be affected. Normally ducks are resistant to the disease but on occasions, ducklings may be affected (Gordon and Jordan, 1982; Alders and Spradbrow, 2001).



Post mortem findings

No gross lesions are pathognomonic for any form of Newcastle disease. NCD can be suspected if the following lesions are encountered, particularly in combination /and when the flock history is also consistent with an NCD outbreak/.

-Congestion and mucous exudates in the trachea

- Congestion of the lungs /heavier than normal; lungs sinks in water (formaline);
- Hemorrhages of the mucosa of the Proventriculus;
- Hemorrhagic and necrotic ulceration of lymphoid patches of the intestine, Caecal tonsils and bursa of Fabricius
- Congested ovarian follicles in chickens in lay.
- Where nervous signs have been dominant, examinations of the CNS have resulted in reports of neuronal degeneration, Perivascular Cuffing of Lymphocyte Cells and Proliferation of the endothelial cells (Jordan, 1990; Alders and Spradbrow, 2001).

Pathogenesis

The pathogenicity of NCD Virus Strains Varies greatly with and/or within the host. Chickens are highly susceptible but Turkeys, ducks and geese may be infected and show few or no clinical signs, even with strains lethal for chickens. In chickens, the pathogenicity of NCDV is determined chiefly by the Strain of the virus, although the dose, route of administration, age of the chicken and environmental conditions all have an effect. In general the younger the chicken, the more acute disease. With virulent viruses in the field, Younger chickens may experience sudden deaths without major clinical signs while in older birds the disease may be more protracted and with characteristic clinical signs. Breed or genetic stock appears to have very little effect on the susceptibility of chickens to the disease (Fikre, 2003).

Diagnosis

History, clinical signs and lesions may suggest ND but laboratory confirmation should always be pursued. The serological tests such as hemagglutination and hemagglutination inhibition (HI), agar gel diffusion, ELISA tests can be used (Fikre, 2003).

Samples for laboratory examination

Tissue sample

Since virulent NCD virus strains are normally thermolabile, it is important to send samples properly packaged with ice packs.

Fresh Samples – Samples of Spleen, lung and the entire head should be wrapped in plastic and placed in to cool box with ice or ice packs.

Where it is not possible to keep the samples cold or when it is not certain that samples will arrive at the laboratory within 24 hours. Samples of Spleen, lung, and entire head (or brain) and long bones should be conserved in 50% glycerin (glycerol) in saline and kept as cold as possible during dispatch (Chauhan, 1993).

Serum Sample

The reliability of any serological test depends to a large extent on the quality of the samples submitted. Haemolysed or contaminated samples will often give unreliable results. Blood from domestic chickens is usually collected from a wing vein. A separate needle should be used for each animal to avoid the risk of mechanically transmitting infectious agents from one animal to another.

Blood samples for serology must not be frozen until the serum has been separated from the clot. Serum samples can be submitted frozen, provided that there are no blood cells present in the sample (Alder and Spradbrow, 2001).

Virus isolation

Sampling from live birds of any species for virus isolation should consist of both cloacal swabs (or faeces) and tracheal swabs, regardless of the clinical signs. From dead birds intestines, intestinal contents and tracheas should be sampled, together with organs and tissues obviously affected or associated with the clinical signs, example the brain if nervous signs are present.

Samples should be placed in phosphate buffered isotonic saline containing antibiotics at PH 7.0-7.4 (checked after the addition of antibiotics); faeces and minced tissues as 10-20% W/V suspensions. The exact mixture of antibiotics does not appear to be critical and may be varied to meet local conditions. However, high concentrations are usually necessary, especially for faeces and cloacal swabs.

An example; penicillin 10,000 U/ml, streptomycin 10 mg/ml, gentamicin 250 mg/ml, mycostatin 5000 U/ml with 50 mg/ml oxytetracycline When chlamydia infection may be a possibility. Samples should be left at room temperature for 2 hours in the antibiotic solution or up to 3 days at 4°C (Jordan, 1990; Chauhan, 1993).

NCDVs will grow in a large range of cell systems and these may be chosen where local conditions preclude the use of specific pathogen free embryonated fowls eggs. However, the latter, at 8 to 10 days of age are the preferred method for the isolation of NCDV. Supernatant obtained after light centrifugation of the samples in antibiotic solution should be inoculated into the allantoic cavity of a minimum of 5 eggs and held at 37°C until dead or dying, or for 5 to 7 days. Eggs should be chilled at 4°C and the amino allantoic fluid

harvested and tested for haemagglutination of chickens red blood cells. Negative fluids should receive at least one more passage through eggs (Jordan, 1990). NCDV may be confirmed by HI test using specific NCDV anti serum.

Differential diagnosis

Newcastle disease can be confused with another viral disease, i.e., avian influenza. Some important thumb rules to differentiate the two in an ordinary laboratory are:- (1). The avian influenza virus can haemagglutinate rabbit erythrocytes, whereas NCD virus does not, and (2). Avian influenza virus does not produce disease in pigeons, whereas NCD virus can. It is also differentiated from infectious laryngotracheitis, fowl cholera and coryza (Beard, 1989).

Virus characterization

Because of the wide range of patho types and the almost universal use of live NCD Vaccines, more isolation and identification of NCDV are inadequate for disease control purposes and further characterization is usually required.

At present, conventional characterization of NCDV isolates involves an assessment of virulence. Three main techniques are employed (Jordan, 1990; Fikre, 2003).

- (1) **Mean death time (MDT) in eggs;** This involves the inoculation of at least five 9 to 10 day old embryonated fowls eggs from a specific pathogen-free source at each dilution in a 10' fold series and calculating the mean time for the embryos to die at the highest dilution at which 100 % mortality is recorded. The MDT has been used to group NCDV strains and isolates into three categories. Velogenic (MDT less than 60 hours), Mesogenic (MDT 61-90 hours) and Lentogenic (MDT greater than 90 hours).
- (2) **Intracerebral pathogenicity index (ICPI) in day old chicks.** This involves the inoculation of virus derived from fresh infectious allantoic fluid into the brain of ten 1-day old chicks from specific pathogen-free parents. Each bird is examined at 24 hours intervals or 8 days and scored; 0 if normal, 1 if sick and 2 if dead. The index is the mean score per bird per observation over the 8-day period. The most virulent viruses give ICPI values approaching the maximum score of 2.0, while lentogenic viruses give value close to 0.0.
- (3) **Intravenous Pathogenicity index (IVPI) in 6-week old chickens.** This test involves the intravenous inoculation of virus derived from fresh infections allantoic fluid into ten 6-week-old specific pathogen-free chickens. Each bird is examined at 24 hour intervals for ten days and scored: 0 if normal, 1 if sick, 2 if paralyzed and 3 if dead. The index is the mean score per bird per observation over the 10-day period. The most virulent viruses give IVPI values approaching 2. While lentogenic and most mesogenic viruses give values of 0.

The levels at which these values will be considered significant in the diagnosis of NCD will be dependent on such parameters as legislative control and trade policies in different countries and the virulence of the live vaccines, if any, in use in the country or geographical area (Jordan, 1990).

Control and prevention

Treatment has no effect on the virus. But antibiotics for 3 to 5 days to prevent secondary infection (E. coli), fresh feed and water, optimum temperature and adequate ventilation should be provided (Fikre, 2003).

Legislation

Most countries free of NCD have legislation aimed at preventing the introduction of disease by infected birds or contaminated product. Such legislation may be extremely complicated and variable as it depends on the disease situation, internal control policies and vaccination status in both the importing and the exporting country.

Legislative powers may also be applied to control disease within a country. Many countries have a slaughter policy for birds with NCD, with compulsory disposal of all birds and produce on site. Restrictions may also be imposed by law on the movement of poultry and produce within a defined affected area. In some countries ring vaccination, may be obligatory following an outbreak, while in other countries, prophylactic vaccination of poultry is require by law (Jordan, 1990). All legislative control measures require careful definition and inevitably dependent on nationally effective diagnosis, monitoring and enforcement

Vaccination

Basically, there are three types of commercially available vaccines for NCD: Live lentogenic, Live mesogenic and inactivated (Jordan, 1990).

Routine vaccination with low virulent live vaccines and/or inactivated (Killed) oil emulsion vaccines (administered parentally as a final vaccines) can be used. The effectiveness of NCD vaccines in the control of the disease depends on the virulence of the field strain, the type and state of the vaccine, the immunological state

of the birds and the method of the vaccine application.

Application of good sanitation and isolation of birds of different age groups. NCDV vaccine is usually given at 10 and 35 days of age and repeated every three (3) months. The commonly used vaccines in Ethiopia are hatchiner's B1, and lasota, which, are produced at the National veterinary institute (NVI), Debrezeit (Fikre, 2003).

Hygiene and disease security

Under field condition vaccination alone is insufficient to bring about effective control of NCD and must be accompanied by good hygiene. In poorly managed, overcrowded, badly ventilated conditions with inevitable underlying bacterial infections even the mildest live vaccine strains may produce disease sufficiently severe to mimic NCDV of high pathogenicity. Good hygiene complemented by good management is therefore of critical importance at all times and not merely during an epizootic of disease.

Hygiene measures should involve both maintaining the birds in a healthy environment and imposing some degree of biosecurity (Jordan, 1990).

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