Newcastle Disease Virus in Poultry: Current Status and Control Prospects

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ABSTRACT

Since its first appearance in Java Island, Indonesia, in 1926, Newcastle disease has significantly impacted the global poultry industry, leading to substantial economic losses. The disease has rapidly spread worldwide, becoming endemic in many countries where agriculture is the primary source of national income. The present study aimed to present a comprehensive review of the recent literature on the Newcastle disease virus to contribute to understanding the virus and its control measures in poultry and provide an updated perspective on current knowledge. All strains of the Newcastle disease virus are classified under a single serotype; however, they are grouped into two classes and have been found to have emerging genetic diversity. Although various molecular diagnostic protocols have been developed, many have limitations. Nucleotide variability in the *F* gene of the Newcastle disease virus seems to explain the false-negative results provided by different real-time reverse transcription polymerase chain reaction protocols. Vaccination combined with biosecurity measures has been shown to limit the devastating effect of the Newcastle disease virus. However, the current vaccines are not effective enough to prevent viral shedding and infection of vaccinated animals. The efficacy of the vaccine strains utilized for decades is being scrutinized, raising questions about their effectiveness over time. The development of reverse genetics offers promising prospects for exploring new generations of attenuated vaccines capable of protecting poultry against clinical diseases and infections, such as Newcastle disease.

Keywords: Diagnosis, Genotype, Newcastle disease, Pathogenicity, Poultry, Vaccination

INTRODUCTION

Newcastle disease, or fowl plague, is a highly contagious viral poultry disease affecting various avian species with varying degrees of susceptibility (Shakal et al., 2020). The virus is easily transmitted, leading to its rapid spread worldwide within three decades after its initial appearance in Indonesia in 1926. Newcastle disease is currently endemic in many countries worldwide (Azizah et al., 2021). Based on pathogenic studies, the virus strains can be classified into five pathotypes, namely asymptomatic enteric, lentogenic, mesogenic, viscerotropic, and neurotropic velogenic strains (Abdisa and Tagesu, 2017; Getabalew et al., 2019). Velogenic strains are capable of causing 100% mortality in unprotected poultry farms, leading to trade restrictions and embargoes in countries where the disease occurs (Dzogbema et al., 2021; Ali et al., 2022). The disease can harmfully affect developing countries where agriculture is the main source of household income and food (Fellahi and Boudouma, 2021). Newcastle disease is a notifiable disease by the World Organization for Animal Health (WOAH). It is defined as any infection caused by an isolate of *Avian Paramyxovirus type 1* with an intracerebral pathogenicity index (ICPI) in day-old chicks of 0.7 or greater and having several basic amino acids at the C-terminal part of the F2 protein and phenylalanine at residue 117 of the N-terminal part of the F1 protein (WOAH, 2022).

Despite the use of vaccines to limit the devastating effects of the virus, Newcastle disease continues to affect the poultry industry worldwide, causing epidemics and significant economic losses (Hu et al., 2022). Although current vaccines can prevent the outbreak of the disease and death from the disease when administered properly, the virus can infect vaccinated animals and replicate, leading to its spread via feces and saliva to other birds (Miller and Koch, 2013; Bello et al., 2018; Mahamud et al., 2022). Recent years have seen a renewed interest in research on the virus due to its pathogenic potential and its use as a vaccine vector in both human and animal health (Jamil et al., 2022; Naz et al., 2022). Advances in molecular biology and genomic sequencing have greatly expanded the understanding of the biology and replication of the Newcastle disease virus (NDV). Furthermore, the development of reverse genetics offers prospects for the construction of a new generation of an attenuated vaccine capable of controlling both the clinical disease and its infection with regard to the modular nature of transcription, low frequency of recombination, and the absence of DNA phase during replication (Fellahi and Boudouma, 2021).

The objective of this review was to provide a synthesis of recently published studies in the field. This review included the original studies conducted on the NDV, its virulence and pathogenicity mechanisms, genetic diversity, clinical and lesional picture resulting from its infection, and new methods and tools for laboratory diagnosis. The investigated articles were limited to those searched in the Google Scholar search engine. This review also examined the prevention and control methods currently in use and prospect.

Taxonomy and structure of Newcastle disease virus

Newcastle disease is caused by virulent strains of *Avian orthoavulavirus 1* (AOAV-1), formerly known as *Avian avulavirus 1* (AAvV-1) and commonly known as *Avian paramyxovirus 1* (APMV-1) or NDV. Newcastle disease virus belongs to the genus *Orthoavulavirus*, subfamily *Avulavirinae*, family *Paramyxoviridae* and order *Mononegavirales*. Isolated avian paramyxoviruses are classified into 21 serotypes designated APMV-1 to APMV-21. Each virus belongs to a viral species that are dispersed among the genera *metaavulaulavirus*, *orthoavulavirus,* and *paraavulavirus* (Amarasinghe et al., 2019; Dimitrov et al., 2019).

Newcastle disease virus is a pleomorphic enveloped virus sized approximately 200-300 nm in diameter. Its envelope is derived from the plasma membrane of the infected cell, with an outer face, where 8-12 nm long spicules are inserted, corresponding to the HN glycoprotein and the F protein. The inner side of the envelope is lined with a matrix protein. The viral genome is a non-segmented, single-stranded RNA of negative polarity. The RNA genome of NDV has a molecular weight of 5.2 to 5.7 X 106 Daltons. Genome sizes vary between 15186 and 15198 nucleotides (Ali et al., 2022). The nucleoprotein combines with the RNA to form a tubular nucleocapsid with a diameter of 18 nm, and the nucleoprotein, phosphoprotein, and large protein are tightly bound to the genomic RNA (Mao et al., 2022).

Viral proteins

The NDV genome consists of six genes, encoding six different proteins. The genes arranged in order of 3'-NP-P-M-F-HN-L-5' encode for nucleoprotein, phosphoprotein, matrix protein, fusion protein, hemagglutinin-neuraminidase protein, and large polymerase protein, respectively (Phale, 2018; Nurzijah et al., 2022). The nucleoprotein has a length of 489 amino acids and a molecular weight of 55 kDaltons. It surrounds the genomic RNA and protects it from nucleases, while phosphoprotein is made up of 395 amino acids and forms multiple bands with a molecular weight ranging from 50 to 55 kDaltons, which is essential in viral replication and transcription. Matrix protein (M) is a non-glycosylated protein associated with the inner surface of the envelope. It consists of 364 amino acids and has a molecular weight of about 40 kDaltons. It plays an important role in viral assembly and envelope stabilization. Glycoprotein (F) consists of 553 amino acids and fuses the viral envelope with the membrane of the target cell. Glycoprotein (HN) is a 577 amino acid long polypeptide with a molecular weight of 74 kDaltons. It is a protein with many functions related to the attachment of the virion to the target cell receptor, enzymatic cleavage of sialic acid, and promotion of viral fusion. The large protein is the largest in the viral genome consisting of 2204 amino acids and has a molecular weight of 250 kDaltons. This protein synthesizes viral mRNA and assists in the replication of genomic RNA (Ganar et al., 2014; Phale, 2018; Fellahi and Boudouma, 2021). Accessory proteins V and W are proteins derived from the *P* gene translated from alternative mRNAs produced by RNA editing during *P* gene transcription. The V protein consists of 239 amino acids with a molecular weight of 36 kDaltons. It is an interferon antagonist and plays an important role in NDV virulence. Finally, the role of the W protein in the NDV replication cycle has not yet been established (Ganar et al., 2014; Nurzijah et al., 2022).

Viral replication

The spread of NDV in the infected organism occurs in several steps. It begins with the penetration of the virus into the host cell via surface glycoproteins (HN and F). The HN protein binds to sialic acid-containing cell receptors on the host cell surface, while the F protein mediates viral entry into the host cell. After the fusion of the viral envelope with the host cell membrane, the viral nucleocapsid is released into the cytoplasm, where the transcription and translation phase begin. During this phase, the negative-sense RNA genome is transcribed into positive-sense mRNA, which is then translated into viral proteins. The mRNA of individual genes is synthesized and used as a template for the synthesis of negative-sense genomic RNA. Transcription of genomic RNA into messenger RNA is carried out by the viral transcriptase (P and L) associated with the nucleocapsid. After the transcription and translation phase, the newly formed viral particles undergo budding and release. The M protein of NDV is essential for the assembly and budding of viral particles (Mao et al., 2022). Its nuclear localization could promote viral replication, increase viral RNA synthesis, and inhibit host cell replication (Duan et al., 2019). The F protein synthesized as a non-functional precursor F0 is cleaved into F1 and F2 by host cell proteases. Viral proteins synthesized in the host cell are transported and incorporated at the cell membrane. Once the nucleocapsids are incorporated into the modified regions of the cell membrane, new viral particles bud at the cell surface, taking with them part of the cytoplasmic membrane. Finally, the HN protein catalyzes the cleavage of sialic acid in the cell receptor and allows the release of the virus (Ganar et al., 2014; Mao et al., 2022).

Infection can also occur by receptor-mediated endocytosis (Sánchez-Felipe et al., 2014). Cellular cholesterol may be required for optimal cell entry into the NDV infection cycle (Martín et al., 2012).

Resistance, virulence, and pathogenicity

The NDV can survive for long periods at room temperature (18 to 23.5°C), especially in feces (Getabalew et al., 2019; Dzogbema et al., 2021). However, it is inactivated at an elevated temperature of 56°C for 3 hours or 60°C for half an hour. It is also inactivated by an acidic pH ≤ 2. Finally, it is sensitive to detergents, lipid solvents, formaldehyde, and oxidizing agents (Getabalew et al., 2019).

The pathogenicity of NDV differs depending on the virus strain. It is well known that activation of the fusion protein (F) by host cell proteases is the primary determinant of virulence (Ganar et al., 2014). An inactive precursor glycoprotein (F0) is produced during replication. It must be cleaved into F1 and F2 polypeptides to exhibit virulence and become infectious (Worku and Teshome, 2020). The cleavability of the F0 molecule is directly related to the virulence of the virus. However, it can be influenced by the nature of proteases present in host tissues and organs. Molecular studies of the F0 glycoprotein site have revealed that the sequence of the F cleavage site between positions 112 and 116 primarily determines the virulence of NDV isolates (Puro and Sen, 2022). Virulent viruses are cleaved by furin-like proteases found in many tissues and organs, allowing them to cause lethal systemic infections. In contrast, trypsin-like proteases can only cleave low-virulence viruses in limited areas, such as the respiratory and intestinal tracts (Phale, 2018). According to Heiden et al. (2014), regions of the F protein modulate virulence other than the polybasic cleavage site.

The development of reverse genetics over the past two decades has allowed researchers to modify NDV genetically and study the individual contribution of genes and genome regions concerning its virulence (Dortmans et al., 2011). According to Samal et al. (2011), the conserved glutamine residue at position 114 in the cleavage site of the F protein is important in NDV replication and pathogenicity. In addition, the replacement of isoleucine at position 118 with valine around the fusion cleavage site reduces the pathogenicity of NDV. Additionally, mutation of the F protein glycosylation site can lead to a hyper fusogenic virus that could increase the virulence of NDV strains (Samal et al., 2012). Mutation of the cytoplasmic domain of the F protein can lead to the creation of a new hyperfusogenic virus that may increase viral replication and pathogenicity (Samal et al., 2013). Other studies have found that the amino acid residue located on site 430 of the HN protein of NDV can influence viral fusion capacity by promoting F protein cleavage (Chen et al., 2021). In addition, ubiquitination on lysine 247 of the NDV matrix protein enhances viral replication and virulence by promoting nuclear-cytoplasmic trafficking (Peng et al., 2022).

Genetic diversity and geographic distribution

Several classification schemes have been developed to identify and differentiate strains of NDV. Initially, these schemes were based on the biological properties of the virus (Diel et al., 2012). However, a new classification system has been proposed by Dimitrov et al. (2019), which is based on phylogenetic topology, genetic distances, branch support, and epidemiological independence of the virus. This system maintains two classes (I and II) of NDV, identifies three new class II genotypes, and reduces the number of subgenotypes. The class I NDV isolates are all grouped into a single genotype and three subgenotypes, as they exhibit strong genetic relatedness. These isolates have been identified in wild and domestic birds from Africa, Asia, Europe, and America (Rauw et al., 2009). It is worth noting that NDV isolates belonging to class I are generally considered to be of low virulence to chicken, with a few exceptions, such as an isolate that caused a severe epidemic in Northern Ireland in the early 1990s (Bello et al., 2018), as well as the JS10-A10 and 9a5b strains generated by consecutive experimental passages through chicken (Rehman et al., 2018).

Class II NDV isolates include viruses of varying susceptibilities, including lentogenic, mesogenic, and velogenic strains, resulting in nearly twenty genotypes due to ongoing virus emergence and evolution (Bello et al., 2018). The most prevalent genotype among all class II viruses in waterfowl is genotype I, which is apathogenic like nearly all class I viruses. Genotype I is further divided into three subgenotypes, namely 1a, 1b, and 1c (Rauw et al., 2009). Genotype II is neurotropic but includes viruses of different pathogenicity (lentogenic, mesogenic, and velogenic). Some viruses belonging to this genotype, including B1, LaSota, VG/GA, and strains not commonly isolated, are used as vaccines (Miller et al., 2010). Genotype III has been isolated mainly in Southeast Asia, Australia, Japan, the United Kingdom, Zimbabwe, Singapore, and China. All NDV isolates belonging to genotype III are virulent, and their isolates have been found in chicken and domestic waterfowl (Dimitrov et al., 2016). Genotype IV is associated with Newcastle disease epizootics in Europe after World War II (Wehmann et al., 2003). Genotype V includes four subgenotypes (Va-Vd) and was responsible for an epizootic wave that started in Western Europe and spread to Yugoslavia during the 1970s (Wehmann et al., 2003). This genotype has also been isolated in Central and North America and Africa (Denis et al., 2015). Genotype VI has 11 subgenotypes (VIa-VIk) and has been described in pigeons in Asia, Africa, Europe, and South America (Dimitrov et al., 2016; Bello et al., 2018). Genotype VII is commonly found in Asia and Africa (Xue et al., 2017; Naguib et al., 2021) and has been associated with the most recent Newcastle disease epizootics

worldwide (Miller et al., 2015). This genotype has complex genetic diversity since it can be subdivided into 9 subgenotypes (VIIa-VIIi; Bello et al., 2018). Genotype VIII has been isolated from South Africa, South Asia, and western China (Cao et al., 2013; Denis et al., 2015; Megahed et al., 2020). Virulent and low-virulent isolates of genotype IX have been described in several asymptomatic wild bird species in China (Duan et al., 2014). Virulent strains of genotype X have been identified in Taiwan, Argentina, and the USA. Genotype XI is also virulent, but its distribution is largely restricted to the island of Madagascar (Maminiaina et al., 2010). Genotype XII has been isolated in South America and China (Liu et al., 2013; Chumbe et al., 2017), while XIII genotype includes three subgenotypes (XIIIa-XIIIc) and has been identified in Asia, Europe, and Africa (Denis et al., 2015; Das and Kumar, 2016; Ana et al., 2020). Genotype XIV includes two subgenotypes and is highly virulent, and has only been found in domestic birds in Africa (Snoeck et al., 2013; Samuel et al., 2013). Genotype XV was isolated in China and included virulent and vaccine strains (Bello et al., 2018). Genotype XVI is strongly related to genotype IV and has been isolated in Europe, Africa, and Asia (Bello et al., 2018). Finally, genotypes XVII and XVIII have two subgenotypes, and they are highly virulent and are found in West and Central Africa (Snoeck et al., 2013; Bello et al., 2018; Souley et al., 2021).

Host species

Newcastle disease virus is capable of infecting over 200 different bird species, but the disease infection consequence varies greatly depending on the host species and the virus strain (Rauw et al., 2009). Newcastle disease affects chickens more severely than turkeys, which typically show few clinical signs (WOAH, 2022). Pheasants, partridges, quail, and guinea fowl are also susceptible (Getabalew et al., 2019). Pigeons (Colombiformes) can also be infected with pigeon paramyxovirus (PPMV-1, Ramsubeik et al., 2023). Ducks and geese (Anseriformes) may be infected but show little to no clinical signs (Rahman et al. 2018). Teal, swans, wild geese, double-crested cormorants, white pelicans, and gulls are susceptible, but they are generally resistant and show little clinical signs of apathy and anorexia (Rasamoelina et al., 2016). Parrots, ravens, sparrows, and kingbirds are susceptible (Rahman et al., 2018). Generally, wild birds and waterfowl are considered reservoir hosts (Getabalew et al. 2019). Newcastle disease can also infect a range of non-avian species. Evidence of Newcastle disease infection and detection have been reported in cattle, sheep, mink, hamsters, mice, rabbits, camels, pigs, monkeys, and humans (Ul-Rahman and Shabbir, 2019; Shabbir et al., 2021; Ul-Rahman et al., 2022). Workers in poultry production and processing and vaccine production laboratories are at the greatest risk (Shabbir et al., 2021).

Mode of dissemination

Transmission of NDV can occur through inhalation or ingestion of virus particles. In the case of natural infection, the virus multiplies in the respiratory and/or digestive tract of infected poultry, which then excretes the virus through airborne or fecal routes (Brown and Bevins, 2017). These released virus particles are inhaled by healthy birds or affect their mucous membranes. Viruses released in feces may also contaminate feed and drinking water and thus be ingested by other birds in the poultry house (Rauw et al., 2009). Non-bird species, such as cats, dogs, foxes, and rodents, may play a role in virus transmission because they may spread the virus for up to 72 hours in their feces after ingesting contaminated poultry (Rasamoelina et al., 2016). Similar to insects, rodents, and reptiles may also be potential vectors of NDV, as their susceptibility to infection has been reported (Rasamoelina et al., 2016). Semi-captive or free-living wild birds may act as natural reservoir hosts for NDV and play a considerable role in the spread of the virus (Rahman et al., 2018).

Clinical signs and lesions

Pathogenicity of the virus strain, host species, host age, secondary infections, stress, environmental conditions, host immune status, viral dose, and route of exposure may play a role in determining incubation time and disease severity (Getabalew et al., 2019; WOAH, 2022). Infection with lentogenic NDV virus strains can range from unapparent respiratory or gastrointestinal illness to mild disease in adult chickens. Infection with mesogenic isolates can lead to the development of nonfatal respiratory disease, decreased egg production, and rare nervous signs, but mortality is generally low (Abdisa and Tagesu, 2017). Infection with highly virulent velogenic viruses can cause mortality in highly susceptible flocks of up to 100% (WOAH, 2022). Viscerotropic velogenic strains cause acute infection of the gastrointestinal mucosa and result in hemorrhagic lesions and death. Clinical signs resulting from chicken infection include general ill health, respiratory disturbances, greenish or whitish diarrhea, muscle tremors, and paralysis of the extremities. Edema may also be seen around the eyes (Caroline, 2022). Neurotropic velogenic strains of NDV primarily cause respiratory distress followed by neurological disease and decreased egg production (Abdisa and Tagesu, 2017). Mortality in chickens infected with velogenic NDVs can reach 100% (Alazawy and Al Ajeeli, 2020).

Newcastle disease virus lesions are primarily characterized by hemorrhages and ulcerations in the proventriculus and intestine (Rauw et al., 2009). Infection with viscerotropic velogenic NDV can result in severe clinical signs in chickens, including dehydration and emaciation of the chicken carcass, hemorrhagic lesions, and necrotic ulcerations at

the tips of the proventricular glands, in the intestine, caecal tonsils, and spleen parenchyma. Additionally, the lungs may show congestive, edematous, and hemorrhagic lesions (Mariappan et al., 2018; Sonkusale et al., 2023). In the respiratory form of the disease in chicken, lesions are marked initially by congestive or hemorrhagic tracheitis that becomes complicated after a few days of consecutive colibacillosis evolution into fibrinous tracheitis, fibrinous pneumonia, aerosaculitis, pericarditis and perihepatitis (Fellahi and Boudouma. 2021). The involvement of the central nervous system in NDV infection can be observed through anatomical manifestations, such as progressive encephalitis, neuronal necrosis, neuronal phagocytosis, the presence of clusters of cells of microglial morphology in the gray matter of the brain, cerebellum and spinal cord, axonal degeneration, and demyelination lesions (Ecco et al., 2011).

Diagnose

Observed clinical signs cannot provide a reliable basis for diagnosis, but can be used as an element of suspicion to guide the diagnosis (Abdisa and Tagesu, 2017). Lesions may also be confused with those of other diseases, including Avian Influenza (Ganar et al., 2014). However, a definitive diagnosis of Newcastle disease is based on the direct detection of viral antigens, which can be achieved by virus isolation from swabs of live animals or organs taken from cadavers. Virus culture is readily performed on 9-11-day-old embryonated chicken eggs or a wide range of cells, including chicken embryo fibroblast cells, chicken embryo hepatocytes, African vervet monkey kidney cells, and chicken embryo reticulum cells (WOAH, 2022). After incubation at 37°C for 4-7 days, the inoculated embryonated chicken eggs are refrigerated at +4°C. The allantoic-amniotic fluid from the eggs is then tested for NDV using the hemagglutination test. Confirmation of NDV infection can be done by performing a hemagglutination inhibition test or by using molecular methods (WOAH, 2022). Agar gel immunodiffusion technique, fluorescent antibody test, hemolysis test, or identification of viral particle morphology by electron microscopy can also be used to identify NDV, but none of them provide information on the pathotype of ND (Cattoli et al., 2011).

Serological diagnosis provides indirect evidence of NDV through antibodies that demonstrate infection. The hemagglutination inhibition (HI) test is the most widely used reference and confirmatory test for Newcastle disease serology (WOAH, 2022). The test is based on the agglutination of chicken red blood cells caused by the hemagglutinin of the viral envelope and the inhibition of hemagglutination by specific antibodies. Enzyme-linked immunosorbent assays (ELISAs) are also commonly used for the detection and quantification of antibodies. The antibody detection technique consists of binding antibodies to viral antigens on a microtiter plate. The viral antigens are captured by other detection antibodies produced in another species against those of the chicken and coupled to an enzyme that catalyzes the reaction to cause a color change. The plate is then read on a spectrophotometer (Dzogbema et al., 2021). The HI test correlates well with the ELISA test (Getabalew et al., 2019; WOAH, 2022). An intracellular cytokine labeling technique was developed using Pinette et al. (2014) to quantify the immune response mediated by T cells. There are other serological tests, such as the neutralizing antibody test, the immunofluorescence test, and the colloidal gold immune technique. However, the limitation of serological tests lies in the cross-reactivity between NDV and other homologous avian paramyxoviruses, which can give false positive results (Mao et al., 2022).

Techniques based on molecular biology methods have been developed for the isolation and identification of NDV strains (Dzogbema et al., 2021; Moa et al., 2022). Several laboratory protocols have been developed, including gel-based reverse transcription polymerase chain reaction (RT-PCR), real-time RT-PCR, restriction enzyme-based methods, and rapid sequencing. In addition, nested PCR, fluorogenic probe-based real-time RT-PCR, ligase chain reaction (LCR), intercalated DNA (SYBR Green), or light-extended fluorogenic primer (LUX) should also be mentioned. All of these assays have shown promising results, but most of them have some limitations and their ability to detect different virus types needs validation (Cattoli et al., 2011; Mao et al., 2022). Since the NDV *F* gene is the primary determinant of NDV pathogenicity, RT-PCR protocols to pathotype NDV generally target this gene (Putri et al., 2017; Abd Elfatah et al., 2021; Mao et al., 2022). The growth of the gene sequence database over the last decade has revealed the high degree of nucleotide variability in the NDV *F* gene, which is mainly responsible for mismatches between oligonucleotides (primers and/or probes) and cDNA templates. This may well explain the false negative results provided by different real-time RT-PCR protocols (Cattoli et al., 2011). Another real-time reverse transcription isothermal loop amplification (RT-LAMP) method has recently been developed. This assay is faster and more sensitive than real-time RT-PCR but had specificity only for NDV genotype VII (Selim et al., 2022).

Pathogenicity testing

Based on calculations of the intracerebral pathogenicity index (ICPI) in day-old chicks, the intravenous pathogenicity index (IVPI) in six-week-old chickens, or the mean time to death in embryos. The NDV isolates can be characterized as virulent (velogenic), moderately virulent (mesogenic), or less virulent (lentogenic). However, ICPI appears to be the more accurate and sensitive method compared to the other two methods (Dortmans et al., 2011). The calculation of ICPI is done after intracerebral infection of day-old chicks with a score ranging from 0 to 2, where 0 is assigned to a normal chick, 1 to a sick chick, and 2 to a dead chick. This score is assigned daily to each chick for eight

days. Finally, the virulence of the virus strain is determined by the average of the scores. Strains are velogenic when the ICPI value is greater than 1.5. They are mesogenic when the ICPI value is between 0.7 and 1.5. Finally, they are lentogenic when the ICPI value is less than 0.7. The IVPI is calculated in the same way as the ICPI, but the six-week-old birds are injected intravenously. The presence of a velogenic strain is indicated by an IVPI greater than 2.5 (Al-Shammari et al., 2020). Mean time to death refers to the average time in hours required for all inoculated embryos to die. If the embryos die within 60 hours, it is an indication of the presence of velogenic strains, while survival of the embryos beyond 90 hours indicates the presence of lentogenic strains (Cattoli et al., 2011). *In vitro*, NDV induces plaque formation in embryonic fibroblast culture, the size and morphology of which varies according to the virulence of the viral strain. The amino acid sequence of the F protein cleavage site can also be used in differentiating the virulence of NDV isolates (Awad et al., 2020).

Control

Control of Newcastle disease must include strict biosecurity that will prevent velogenic virus strains from coming into contact with poultry and adequate administration of effective vaccines (Dimitrov et al., 2017). In addition, serological monitoring of the immunological status of vaccinated flocks should be performed to assess the antibody response to the administered vaccines (Ahmed and Odisho, 2018). Traditional vaccines (live and inactivated vaccines) have been widely used for several decades (Dimitrov et al., 2017). However, another generation of vaccines, such as recombinant vaccines and antigen-matching vaccines, have recently been adopted in some countries, and other vaccine approaches are in the experimental phase (Dimitrov et al., 2017; Nurzijah et al., 2022). The vaccine strains used are obtained from the naturally apathogenic, low pathogenic, or medium pathogenic strains (Dimitrov et al., 2017; Hu et al., 2022). These vaccine strains belong to genotype I (Ulster and V4), genotype II (Lasota and B1), genotype III (Mukteshwar), genotype IV (Herts/33, UK), genotype V (Anhinga, USA), genotype VIII (AF22440), and genotype XV (vaccine strains, China, Bello et al., 2018).

With the development of genetic engineering, NDV vaccine strains have emerged as promising vectors for developing effective multipurpose vaccines against pathogens that infect both animals and humans (Kim and Samal, 2016; Choi, 2017). For example, the use of an NDV virus-like particle-based vaccine expressing NDV F protein and influenza virus matrix 1 (M1) protein could protect chickens from a lethal challenge, while significantly reducing viral shedding and can also be used as a DIVA strategy to differentiate infected from vaccinated animals (Park et al., 2014). An experiment conducted by Izquierdo-Lara et al. (2019) indicated that vaccine strains paired with wild type XII strains conferred better protection to vaccinated animals and significantly reduced viral shedding. Similarly, Miller et al. (2013) in an experimental setting showed that by increasing the homology of the vaccine to a wild-type virus, humoral antibody efficacy levels could be increased. As the level of humoral antibodies increases in vaccinated birds, the number of infected birds and the amount of virus shed decreases. Results of an experiment conducted by Pandarangga et al. (2022) also revealed that viral shedding in chickens is reduced when vaccinated with strains homologous to the infectious challenge strain compared to the vaccine using the heterologous strain (LaSota).

Furthermore, in an intranasal vaccination trial in chickens, Zhao et al. (2016) found that a DNA vaccine containing the NDV *F* gene encapsulated in hollow nanoparticles induced higher serum antibody (IgA) titers. Results of a comparative study by Zhao et al. (2012) also showed that a live vaccine (LaSota strain) encapsulated in chitosan nanoparticles (NDV-CS-NP) induced better protection to immunized specific pathogen-free chickens, compared to the live LaSota strain vaccine and the inactivated NDV vaccine. An encapsulated inactivated NDV vaccine in chitosan nanoparticles has also been successfully developed (Mohammadi et al., 2021). These studies lay the foundation for the future development of mucosal vaccines encapsulated in nanoparticles. Plant-derived vaccines are in development and offer potential advantages over traditional vaccines, including a significant reduction in viral shedding and the ability to differentiate infected birds from vaccinated birds (Nurzijah et al., 2022; Smith et al., 2023).

CONCLUSION

Newcastle disease is a health threat to the global poultry industry. It is endemic in many developing countries. In disease-free countries, NDV outbreaks are occasional, but when they occur, they result in significant trade restrictions and embargoes. Newcastle disease is sometimes misdiagnosed because serological and molecular diagnostic tests for NDV have certain limitations as a result of cross-reactivity of NDV with other homologous avian paramyxoviruses and variability in the nucleotide sequence of the NDV *F* gene. To prevent ND, only sanitary and medical prophylactic measures can be used effectively to enhance the immune capacities of birds and reduce ambient viral pressure. The international reports show that vaccinated poultry can be infected with wild viruses. The NDV genotypes are emerging, which can make control strategies complex and often difficult, hence the need to use high-quality and up-to-date vaccines. Research priorities should therefore focus on improving diagnostic tools and developing better vaccines.

DECLARATIONS

Acknowledgments

Authors' contributions

This work was carried out with the contribution of all authors. Ahamidou Moustapha drafted the manuscript. Essodina Talaki, Akourki Adamou, and Moumouni Ousseini revised the manuscript. All authors read and approved the final version of the manuscript.

Competing interests

The authors have not declared any conflict of interest.

Ethical consideration

The authors of the current study checked for ethical issues, including plagiarism, consent to publish, misconduct, double publication and/or submission, and redundancy.

Funding

Data availability and materials

Data from the study are available according to a reasonable request.

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