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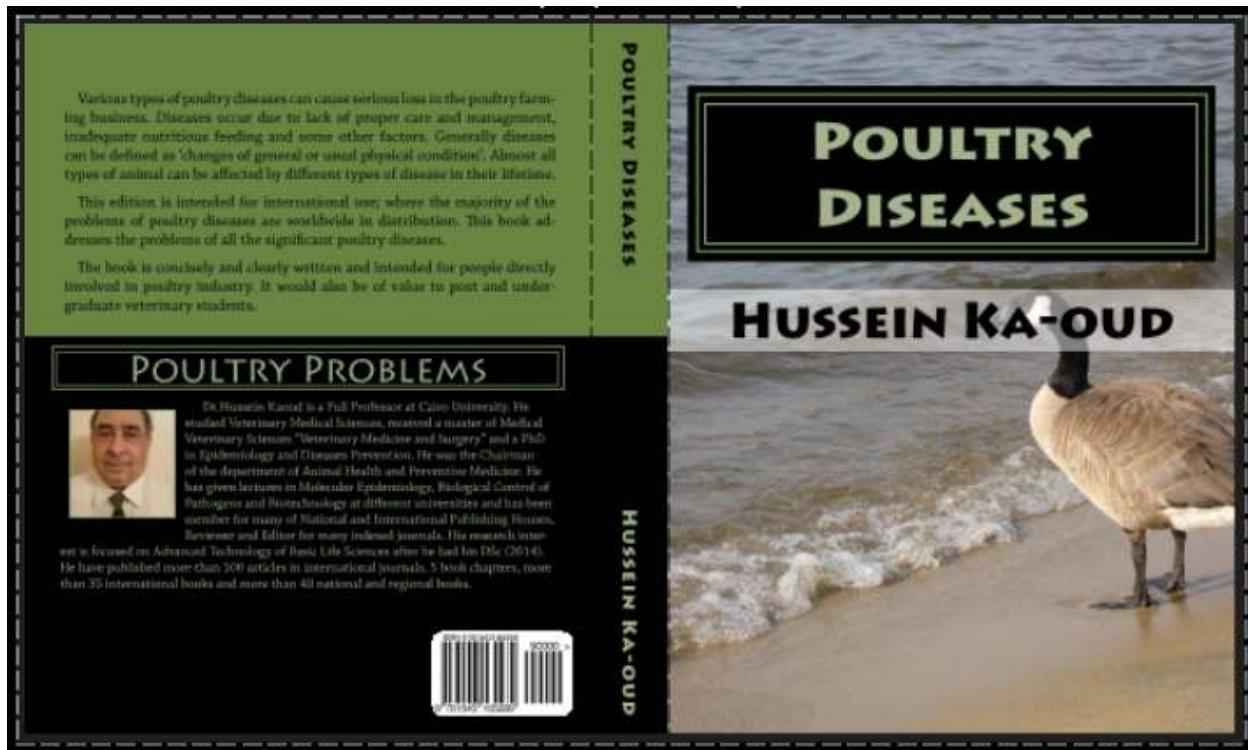
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Poultry Diseases

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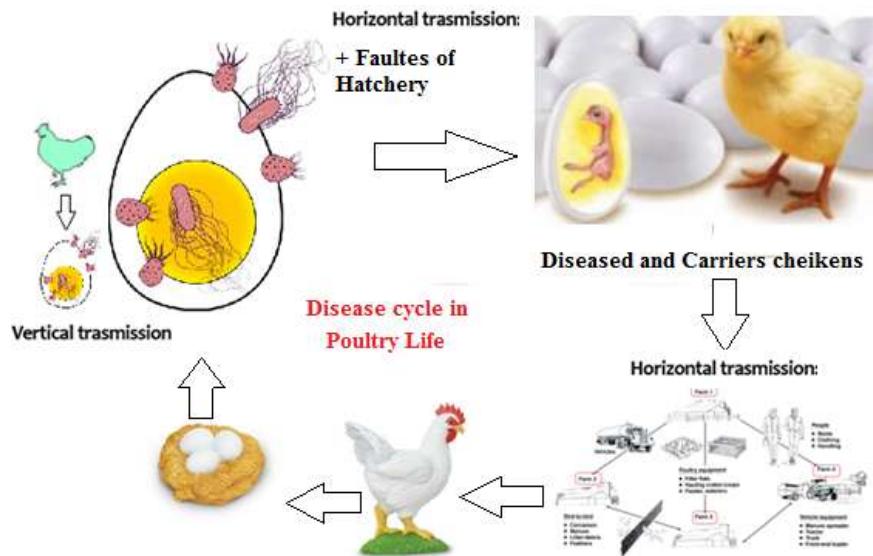
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Preface

Various types of poultry diseases can cause serious loss in the poultry farming business. Diseases occur due to lack of proper care and management, inadequate nutritious feeding and some other factors. Generally diseases can be defined as 'changes of general or usual physical condition'. Almost all types of animal can be affected by different types of disease in their lifetime.



Usually poultry producers face some problems during establishing a new poultry farming business such as lack of capital, location, housing, food management, diseases etc. Among all those problems, poultry diseases are most important to consider. Disease can destroy the whole farm and you might loss money seriously. The meat and egg production of poultry can suddenly decrease due to various types of diseases. Many poultry birds die every year throughout the world, due to various types of diseases. As a result of this, producers loss a huge amount of money. In a word the main reason of financial damage of poultry farming business is various types of diseases

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Vaccination

The poultry broiler industry is growing in size, with an annual production of approximately 40 billion birds worldwide. One of the major problems faced by the poultry industry is a loss of productivity due to disease, requiring prudent health management.

The use of veterinary vaccines has somewhat different requirements than the use of human vaccines. One of the limitations of veterinary vaccines is cost. Vaccines, particularly for poultry, must be cost-effective, particularly when they are required on a large scale of tens of billions of doses annually. Therefore, veterinary adjuvants must also be designed based on the economics of immunization.

Currently, the most common adjuvants found in licensed veterinary vaccines are aluminium salts and oil emulsions. Aluminium hydroxide (alum) adjuvants have been commonly used in many veterinary and human vaccines because of their safety. However, comparative studies in humans and animals show that alum is a weak adjuvant for the induction of antibody responses to recombinant protein vaccines. It has been shown in mice to bias towards Th2 rather than Th1 responses. Alum poorly induces cell-mediated immunity, particularly cytotoxic T-cell responses⁷, which is a significant drawback for its use in vaccines against intracellular parasites and some viruses. Additionally, alum adjuvants have a tendency to induce IgE-mediated immune responses and may promote IgE-mediated allergic reactions.

The use of oil-based adjuvants, in contrast, are limited by induction of side-effects and adverse site reactions. CFA is a typical example, having been shown to induce inflammation and ulceration at the site of injection as well as fever and sensitivity reactions. This is an important point to note when considering an adjuvant for veterinary use. Choosing the right adjuvant is important in terms of industry production requirements and is critically important to animal welfare. The poultry industry strongly relies on meat quality; site reactions which lead to carcass damage affect the meat quality, resulting in a loss of productivity. Site reactions induced by adjuvants such as alum or CFA also lead to animal distress and discomfort, which compromises the welfare of chickens. As these issues are of major importance to the consumer, it is vital to producers that site reactions be avoided. Poultry producers require effective adjuvants that promote protection and that do not cause any pain or distress to birds when administered with a vaccine. However, the currently used adjuvants do not fulfil these requirements. With this in mind, there is an increased need for effective, better vaccine adjuvants that avoid animal discomfort.

To design new adjuvants, their mechanism of action needs to be understood. An adjuvant is an agent that increases the antigenic response of an antigen. When incorporated into a vaccine formulation, adjuvants act to accelerate, extend or enhance the magnitude of a specific immune response to the vaccine antigen. The mechanism underlying adjuvant activity was somewhat poorly understood until the discovery and functional analysis of Toll-like receptors (TLR). TLR were first described in *Drosophila* and were found to play an important role in antifungal host defence. TLR recognize unique patterns of individual pathogen-associated molecular patterns (PAMP) and signal to induce the production of pro-inflammatory cytokines, thereby resulting in the activation of innate and adaptive immunity. It is well known that TLR are crucial in triggering both innate and adaptive immunity, and their involvement in immunostimulatory activities defines them as adjuvant receptors. With this in mind, many TLR ligands have been examined for their adjuvant activities in mammals. However, less is known about the existence and function of TLR in non-mammalian species. Because adjuvants interact with TLR, it is important to determine the conserved nature of TLR and the likelihood that the adjuvant will work across species. The recent release of the chicken genome has allowed the identification of a number of potential orthologues of the chicken TLR. Furthermore, the recent cloning and characterization of the avian homologue of TLR2 and TLR4 has revealed the similarities and differences between chicken and human TLR. It is crucial to know the role of TLR in chickens and how these receptors respond to adjuvants in order to develop adjuvants for use in chickens. Furthermore, the variation of TLR expression in different lines of chickens is of important consideration, particularly when the adjuvant response is dependent upon the level of TLR expression. The higher the level of expression, the greater the possible immunological response, and vice versa. As TLR signalling induces the production of pro-inflammatory cytokines and reactive intermediates, approaches using TLR signalling to enhance cytokines can be used as potential powerful adjuvants for initiating effective immune responses.

The use of recombinant cytokines as adjuvants is attracting extensive attention and they may be alternatives to existing adjuvants . Cytokines are naturally derived proteins that play a crucial role in controlling the immune system, and are produced in response to infection. In mammals, they provide signals that help to direct the immune response towards either an antibody-mediated or a cell-mediated response . Cytokines have been shown to be effective adjuvants in several studies. Early studies involving cytokines as adjuvants used mouse models to focus on IL-2 as a potential adjuvant, and they were shown to enhance the immune response to inactivated rabies vaccine and a herpes simplex virus antigen . IL-2 has been extensively studied as a vaccine adjuvant due to its pleiotrophic properties and crucial role in activating T-cell proliferation . The potential adjuvant effects of IL-2 have been demonstrated in vaccine model systems of cattle, guinea pigs , pigs and mice . Similarly, several human and animal studies involving the adjuvant efficacy of IFN- γ have been reported. IFN- γ has been shown to be an effective adjuvant in immunocompromised mice when delivered with a malaria vaccine or an influenza vaccine . Other mouse studies have demonstrated that cytokines such as IL-1 act as adjuvants in the upregulation of humoral and cellular responses to antigen . In livestock, cytokine therapy has been shown to be effective in several animal models. In cattle, the administration of recombinant bovine IL-2 or IFN- α and - γ greatly reduces mortality and the severity of clinical disease in vaccinated calves when challenged with bovine herpes virus-1 . Similarly, Nash and colleagues showed that IL-1 α and -1 β act as adjuvants in sheep to significantly enhance the secondary humoral immune response to an experimental antigen . These studies highlight the use of cytokines as adjuvants and therefore open up the avenue to assess such activities in non-mammalian species such as chickens.

Newcastle Disease Virus (NDV)

Vaccination

Basically, there are three types of commercially available vaccines for ND: live vaccines, inactivated vaccines and recombinant vaccines. Types of NDV vaccines and route of administration (eye drop, spray, drinking water and/or injection) vary depending on several factors. These factors include the level of maternal immunity, the virulence of the endemic NDV of a geographic location, the time between two successive vaccinations, and the lifespan of the birds.

Live NDV vaccines

Live vaccines can be divided into two categories based on virulence. More virulent vaccines are classified as mesogenic strains while less virulent strains are classified as lentogenic strains. Four mesogenic strains (Herts, Mukteswar, Komarov, and Roakin) have been used in many parts of the world but presently their use is restricted to areas in Africa, Southeast Asia and the Middle East where endemic velogenic

Recombinant NDV vaccines

The recombinant fowl pox virus, which expresses the HN and/or F NDV proteins, has been found to provide protection against virulent NDV challenge when it is inoculated intravenously, intramuscularly or by wing web in birds without pre-existing antibodies. However, only partial protection is achieved when the vaccines are administered orally or ocularly .

Furthermore, recombinant vaccines expressing the HN and/or F glycoprotein have also been reported with a vaccinia virus (W), a herpes virus of turkeys (HVT) , and a baculovirus . Recently, a recombinant fowlpox has been licensed and recommended for use with birds having no pre-existing antibodies to NDV

NDV is prevalent. These vaccines are pathogenic and are not recommended for use in chickens less than 8 weeks of age, or older chickens that have not been previously vaccinated against MDV.

Live lentogenic NDV vaccines are the most widely used. They have been shown to have a low pathogenicity in poultry while producing an adequate immune response. Four lentogenic NDVs (LaSota, Hitchner BI, F and V4) have been developed as vaccines. Of these, BI and LaSota are the most widely used. Both of these viruses replicate in the respiratory tract and induce local and systemic immunity. LaSota vaccines are more virulent and induce a stronger immune response than BI vaccines. Because of the greater potential for LaSota vaccines to cause respiratory disease, they are normally used for boosting NDV vaccines in chickens previously immunized with BI .

More recently, a non-pathogenic NDV has been developed for vaccine use in the USA. The VG/GA strain was isolated from commercial turkeys and found to provide protection in chickens against the lethal challenge of velogenic viscerotropic NDV (WNV), similar to that provided by the BI strain.

Inactivated vaccines

Inactivated oil emulsions of ND vaccines are found to provide uniform and long lasting protection without post-vaccinal respiratory reactions. Since inactivated vaccines must be injected, they are used primarily to vaccinate breeders and layers that have been vaccinated previously with one or more live NDV vaccines. The humoral immune response to inactivated NDV in breeders and layers is very high and of long duration, and provides a high level of maternal antibodies for their progeny.

The role of antibodies in early protection following vaccination against NDV has also been reported. Protective immunity against NDV is due primarily to antibodies. Circulating antibodies protect the host from reinfection, starting as early as four to six days after infection. In the early phase of vaccination IgM is involved and followed by IgG. Titers and protection peak three weeks after vaccination, and then gradually decline if there is no boosting. A number of reports have indicated that concurrent outbreaks of infectious bursal disease virus (IBDV), which has an immunosuppressive effect in chickens, reduces the serological response to ND vaccination as measured by the hemagglutination inhibition.

Maternally derived antibodies

Immunoglobulins in birds are deposited into the egg yolk and chicks absorb these antibodies into their developing systems. The yolk contains mainly IgG which becomes the circulating antibody in the chick, while the albumen contains predominantly IgA which is swallowed by the developing chick, thus coating its mucous membranes with the IgA . The levels of passively acquired maternal antibodies in the serum of a day-old chick are approximately the same as those found in the serum of the hen. The level of passively acquired maternal antibodies found in young chicks generally declines at a constant rate, and has a half-life of approximately 4 1/2 days . Maternal immunoglobulins (IgG) to NDV have also been detected in tears, the trachea and lungs.

Maternal antibodies play an important role in protecting the chick early in its life.

Local immunity

The development of a local antibody immune response to NDV has been demonstrated in both the respiratory and intestinal tracts. After vaccination, both IgA and IgG specific to NDV have been found in the trachea, lungs, saliva, lacrimal fluid, the Harderian glands and the intestine . The administration of NDV vaccines by the intra-ocular route results in the development of high levels of IgM, IgG and IgA antibodies in the Harderian gland and in lacrimal fluids due to NDV replication in the Harderian gland.

Chapter 2

Bacterial Diseases

Colibacillosis

Occurs as an acute fatal septicemia or subacute pericarditis and airsacculitis. It is a common systemic disease of economic importance in poultry and is seen worldwide.

Etiology

Escherichia coli is a gram-negative, rod-shaped bacterium normally found in the intestines of poultry and most other animals; although most are nonpathogenic, a limited number produce extraintestinal infections. Pathogenic strains are commonly of the O1, O2, and O78 serotypes, but serotypes O11, O15, O18, O51, O115, and O132 have also been reported for *E coli* isolates associated with cellulitis and colibacillosis. There is considerable diversity of serogroups among clinical isolates, and only a small percentage of these isolates belong to serotypes O1, O2, or O78. In fact, 18-29% of avian *E coli* isolates cannot be typed. Therefore, no single *E coli* serotype used as a bacterin can provide full protection against all of the serotypes that cause *E coli* infections. Virulence factors include the ability to resist phagocytosis, utilization of highly efficient iron acquisition systems, resistance to killing by serum, production of colicins, and adherence to respiratory epithelium. Virulent *E coli* are generally nontoxigenic, poorly invasive, and do not possess common adhesins.

Large numbers of *E coli* are maintained in the poultry house environment through fecal contamination. Initial exposure to pathogenic *E coli* may occur in the hatchery from infected or contaminated eggs, but systemic infection usually requires predisposing environmental factors or infectious causes. Mycoplasmosis, infectious bronchitis, Newcastle disease, hemorrhagic enteritis, and turkey

bordetellosis precede colibacillosis. Poor air quality and other environmental stresses may also predispose to *E coli* infections.

Systemic infection occurs when large numbers of pathogenic *E coli* gain access to the bloodstream from the respiratory tract or intestine. Bacteremia progresses to septicemia and death, or the infection extends to serosal surfaces, pericardium, joints, and other organs.

Clinical Findings and Lesions

Signs are nonspecific and vary with age, organs involved, and concurrent disease. Young birds dying of acute septicemia have few lesions except for enlarged, hyperemic liver and spleen with increased fluid in body cavities. Birds that survive septicemia develop subacute fibrinopurulent airsacculitis, pericarditis, perihepatitis, and lymphocytic depletion of the bursa and thymus. (Unusually pathogenic salmonellae produce similar lesions in chicks.) Although airsacculitis is a classic lesion of colibacillosis, whether it results from primary respiratory exposure or from extension of serositis is unclear. Sporadic lesions include pneumonia, arthritis, osteomyelitis, and salpingitis.

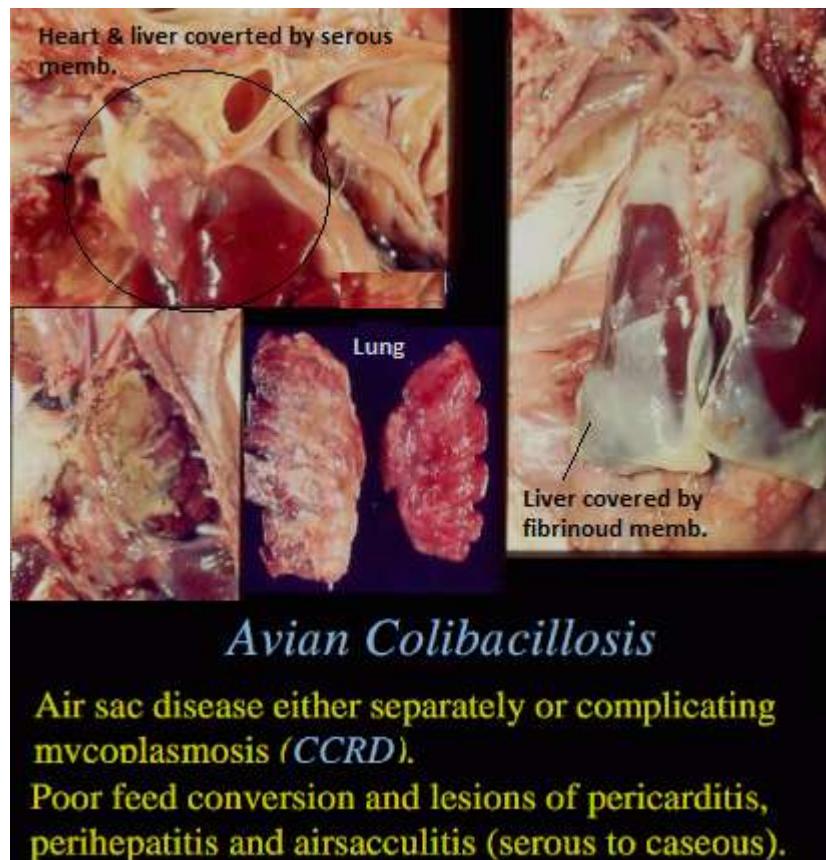


Fig.13: Lesions of Colibacillosis Disease.

Diagnosis