

The Avian Immune System¹

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The avian immune system is divided into non-specific and specific immune mechanisms.

Non-specific immune mechanisms include the innate or inherent ways in which the chicken resists disease. This protective system is often not considered when designing a poultry health program. Many programs tend to rely primarily on vaccinations and/or antibiotics to maintain flock health. The importance of non-specific immune mechanisms should be realized. Examples include **Genetic factors** - birds may not have complementary receptors to allow many disease organisms to infect them. For example, some strains of chickens are genetically resistant to the lymphoid leukosis virus.

Body temperature—the high body temperature of the chicken precludes many diseases. Blackleg disease of cattle is not a problem in poultry. If the body temperature of the chicken is lowered, the disease may occur.

Anatomic features—many disease organisms cannot penetrate intact body coverings (skin and mucous membranes) or are trapped in the mucus secretions. Some nutritional deficiencies (biotin deficiency) or infectious diseases compromise the integrity of the body coverings, allowing penetration of disease organisms.

Normal microflora—the skin and gut normally maintain a dense stable microbial population. This stable microflora prevents invading disease organisms from gaining a

foothold. Improper use of antibiotics or poor sanitation can disrupt the balance of the microflora.

Respiratory tract cilia—parts of the respiratory system are lined with cilia which remove disease organisms and debris. If the air in the poultry house is of poor quality due to high levels of dust or ammonia, the ciliary system may be overwhelmed and become ineffective.

Other factors involved in innate resistance include nutrition, environment (avoid heat/cold stress), age (young/old animals are more susceptible to disease), inflammatory processes, metabolic factors, complement, and interferon.

The reason that good management practices are important in maintaining poultry health is better understood when the non-specific immune mechanisms are defined. For example: the overuse of antibiotics or poor sanitation may lead to a disruption of the normal microflora; poor nutrition may lead to deficiencies which allow disease organisms to penetrate the protective body coverings; selection of disease resistant strains of chickens may preclude or lessen the effects of certain diseases; and others.

Specific immune mechanisms (acquired system), on the other hand, are characterized by specificity, heterogeneity, and memory. This system is divided into cellular and non-cellular (humoral) components.

The non-cellular component includes immunoglobulins (antibodies) and the cells which produce them. Antibodies

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are specific (specificity) for the foreign material (antigen) to which they attach. The antibody against Newcastle disease virus will attach only to the Newcastle virus, not to the infectious bronchitis virus (heterogeneity). There are three classes of antibodies that are produced in the chicken after exposure to a disease organism: Ig M, Ig G, and Ig A. Ig M appears after 4-5 days following exposure to a disease organism and then disappears by 10-12 days. Ig G is detected after 5 days following exposure, peaks at 3 to 3 1/2 weeks, and then slowly decreases. Ig G is the important protective antibody in the chicken and is measured by most serological test systems. Thus, if you are interested in determining antibody titer levels following vaccination, you should collect sera after 3 to 3 1/2 weeks. If sera is evaluated prior to this time, the antibody titer levels are still increasing which makes interpretation of the vaccination program difficult. Ig A appears after 5 days following exposure. This antibody is found primarily in the mucus secretions of the eyes, gut, and respiratory tract and provides "local" protection to these tissues.

The cells which produce antibodies are called B-lymphocytes. These cells are produced in the embryonic liver, yolk sac and bone marrow. The cells move to the bursa of Fabricius (BF) after 15 days incubation through 10 weeks of age. The BF programs these cells which then move to the blood, spleen, cecal tonsils, bone marrow, Harderian gland, and thymus. Destruction of the BF at a young age by Gumboro disease or Marek's disease prevents programming of B-cells. Thus, the chicken will not be able to respond to diseases or vaccinations by producing antibodies.

When a disease organism enters the body, it is engulfed by a phagocytic-type cell, the macrophage. The macrophage transports the disease organism and exposes it to the B-lymphocytes. The B-cells respond by producing antibodies after day 5 following exposure. The lag period occurs because the B-cells must be programmed and undergo clonal expansion to increase their numbers. If the chicken is exposed a second time to the same disease, the response is quicker and a much higher level of antibody production occurs (memory). This is the basis for vaccinating. Antibodies do not have the capability to kill viruses or bacteria directly. Antibodies perform their function by attaching to disease organisms and blocking their receptors. The disease organisms are then prevented from attaching to their target cell receptors in the chicken. For example, an infectious bronchitis virus which has its receptors covered with antibodies will not be able to attach to and penetrate its target cells, the cells lining the trachea. The attached

antibodies also immobilize the disease organism which facilitates their destruction by macrophages.

The cellular component includes all the cells that react with specificity to antigens, except those associated with antibody production. The cells associated with this system, the T-lymphocytes, begin as the same stem cells as the B-cells. However, the T-lymphocytes are programmed in the thymus rather than the BF.

The T-lymphocytes include a more heterogeneous population than the B-cells. Some T-cells act by producing lymphokines (over 90 different ones have been identified); others directly destroy disease organisms; some T-cells act to enhance the response of B-cells, macrophages, or other T-cells (helpers); and others inhibit the activity of these cells (suppressors). The cellular system was identified when it was shown that chickens with damaged BF could still respond to and eliminate many disease organisms.

A chicken may become immune to a disease organism by producing antibodies itself or by obtaining antibodies from another animal. When the chicken produces its own antibodies following exposure to a foreign material, the process is called active immunity. This occurs after the bird is exposed to a vaccine or a field disease challenge. Active immunity is harmed by anything which damages the cellular or humoral immune systems.

When the chick receives pre-made antibodies from the hen through the egg, this is termed passive immunity. These antibodies are not produced by the chick. Maternal antibodies are present in the yolk, albumin, and fluids of the egg. If the hen has a high antibody titer level to a disease, the chick should also be immune for several weeks. However, since the immune system of the chick is not stimulated, there will be no antibodies produced by the chick and no memory cells. The flock manager must be aware of the maternal antibody levels in the chicks to schedule vaccinations. If chickens are vaccinated when maternal antibody titer levels are elevated, the vaccine may be buffered excessively resulting in a reduced response. Conversely, if vaccinations are delayed and maternal titer levels are low, a severe vaccine reaction may result.

In summary, the immune system of the chicken is very helpful in preventing disease and helping to insure maximum productive potential is realized. We must learn how to take advantage of all parts of the system when designing health programs.