Immunosuppression in chickens – what is it?

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Immunity is the ability to stop an infection. Immunosuppression is a status where the immunity is reduced. Humoral (antibodies) and/or cell immunity may be depressed.

Immunosuppression can be due to infectious agents, to improper feeding balance (deficiencies), to a lack of biosecurity, to management failures (stress), or to a combination of them. Each of these possible causes must be seriously worked out to prevent the consequences of immunosuppression on profitability.

How to recognise immunosuppression

Immunosuppression may affect both health and performances. Increased mortality, uneven growth, decreased body weight, higher feed conversion, higher medication costs and higher rate of condemnations at slaughter (when compared to previous flocks) are common findings in immunosuppressed birds.

Immunosuppressed birds will typically show long and complicated vaccine reactions, and will be more easily predisposed to respiratory diseases with secondary bacterial infection.

A loss of medication efficacy can be met. 'Unusual' infections may occur, for example gangrenous dermatitis, anaemia, or inclusion body hepatitis.

Lastly, immunosuppressed birds will show a lower antibody response to vaccines than expected. However, some other causes may mimic an immunological disorder without any link with any direct damage of the immune system, like the emergence of a new serotype of infectious bronchitis virus (for example a variant).

It can make the available vaccines less effective, and it may induce an increased incidence of E. coli secondary infections.

Increased losses may also be linked to the development of resistances to frequently used antibiotics.

Performance results must be correlated with the examination of the main lymphoid tissues to establish a complete and practical evaluation of the immune system.

Basically, the organs or cells that can be damaged are:

- The bursa of Fabricius.
- The thymus.
- The spleen.
- The bone marrow.
- The lymphoid cell aggregates along the gut, the trachea, the oesophagus, for instance, the Harderian gland, the caecal tonsils and the Peyer's patches.
- The circulating lymphocytes themselves.

During the embryonic development, the immature B and T lymphocytes are present in the bursa and the thymus, respectively.

During the third week of embryonation, the B and T cells migrate from the bursa and thymus to the peripheral lymphoid system, including spleen and bone marrow. Lymphoid cell aggregates start to be colonised at hatch.

The causes of immunosuppression may be infectious agents or non-infectious agents.

Infectious agents

- Infectious bursal disease virus (Gumboro disease virus). When infection occurs before two weeks of age, it induces a severe impairment of the immune system (for instance, with the current US variants).
- Chicken infectious anaemia virus.
- Marek's disease virus.
- Reovirus.
- Reticuloendotheliosis virus.
- Subgroup J avian leukosis virus (responsible for myeloid leukosis).
- Newcastle disease virus.
- Infectious bronchitis virus.
- Avian pneumovirus (responsible for swollen head syndrome).
- Mycoplasma Spp.
- Eimeria Spp.

Such a list is certainly not exhaustive.

Non-infectious agents

- Genetic breed. Some meat type birds are more susceptible to respiratory pathogens because they have a too small heart weight compared to the total weight. For instance, it has been demonstrated that fast growing turkeys are more susceptible to cholera.
- Feed imbalance, for example vitamin deficiency:
  - **Vitamin E** is stabilising the cell membranes. Free radicals or oxidative compounds can be ingested through poorly stabilised fats, oils and animal byproducts. They are able to damage the cell membranes. The membranes of the rapidly multiplying cells of the immune system are particularly exposed to the consequences of free radicals. Vitamin E is able to remove these oxidative molecules.
  - **Vitamin C** helps in reducing the effects of stress, and speeds up the healing

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Table 1. Mechanisms of immunosuppression in some viral poultry diseases.

Virus	Effect upon			
	B lymphocytes	T lymphocytes	Macrophages	Others
Gumboro disease	Depletion of bursal and peripheral populations			
Marek's disease	Destruction in early stages of virus multiplication	Transformation in tumoral cells		
Reovirus	Destruction by virus multiplication			
Chicken infectious anaemia	Depletion of all cell lines			
Newcastle disease, avian influenza			Decrease in phagocytic activity	Damage in the trachea ciliae

process by collagen formation enhancement. Vitamin C is also important for the formation of white blood cells.

• **Vitamin A** seems to be more directly involved in antibody function. Studies have shown that vitamin A deficient birds produced less Newcastle antibodies and a lower T-cell response.

● Mycotoxins. On their own, mycotoxins like aflatoxins are directly immunosuppressive. The fungi that may contaminate the feed ingredients are typically represented by three genera – aspergillus, penicillium, and fusarium.

They produce mycotoxins that are commonly contaminating feed ingredients employed in the poultry industry. Maize, wheat, rice, and peanut meal are most frequently implicated.

• **Aflatoxin B1** (produced by aspergillus) has been shown to impair the immune cells function by reducing the amount of antibodies following infection or vaccination, and by reducing the activity of phagocytic cells.

Aflatoxin is also responsible for lymphoid depletion and necrosis in the bursa of Fabricius, spleen and thymus. It is also hepatotoxic.

• **Ochratoxin A** (produced by penicillium) is associated with a generalised impaired humoral and cell mediated immune response. It is also nephrotoxic.

• **Trichotecene mycotoxins**, also called fusariotoxins (for instance, T-2 toxin) are produced by fusarium.

First, they are strong tissue irritants and alter mucosal membranes integrity. Secondly, they inhibit protein synthesis and consequently interfere with antibody production.

The most prevalent mycotoxins in feed ingredients used in the poultry sector are the aflatoxins and the trichotecenes.

● Ammonium or dust. They act by damaging the respiratory system. The target concentration of ammonia should be less than 10ppm. In layer houses, it should be less than 25ppm (see Table 2).

● Stress – cold or heat stress, lack of access to drinking water, density, poor ventilation, noise, light intensity, etc. Long term stress is responsible for the release of steroids that are immunosuppressive.

It has been demonstrated that chilled birds have a lower antibody response and a lower cell mediated immunity.

Following force moulting stress, breeder hens may shed more salmonella and may experience a recurrence of a previous disease like cholera or colibacillosis.

● Poor management can be responsible for higher morbidity and mortality rates, poorer feed conversion, and an increased susceptibility to several diseases.

For instance, poor litter conditions may increase the bacterial load on the skin. Sudden dietary changes, or improper use of antibiotics may disrupt the gut microflora, making it less able to absorb the nutrients and to compete against the harmful bacteria.

These factors alone may be responsible for immunosuppression, but most of the time several of them may act synergistically.

Agent	Responsible for atrophy of		
	Bursa of Fabricius	Thymus	Other
Gumboro disease virus	X	X	
Chicken infectious anaemia virus	X	X	X (bone marrow: aplasia)
Marek's disease virus	X	X	
Reovirus	X	X	
Reticuloendotheliosis virus	X	X	
Newcastle disease virus	X		
Mycoplasma	X	X	
Ammonia	X		
Mycotoxins	X	X	X (spleen: necrosis)
Heat stress		X	X (spleen: atrophy)

Table 3. Agents responsible for atrophy of some immune organs.

Preventing immunosuppression

First try to identify the causative agent(s) through a sound diagnosis, like Gumboro disease virus, reovirus, or Marek's disease virus. The post mortem findings are rarely straightforward, as summarised in Table 3.

In addition to post mortem examination, the diagnosis will usually require histopathology analysis – sample bursa, spleen, liver, sciatic nerve, thymus, brain for instance.

Chicken infectious anaemia virus infection will rather be evaluated using serology.

Any results must be evaluated cautiously, and must obviously be confronted to the flock's performances.

As an example, microscopic bursal lesions can be found in many flocks as early as four weeks of age; consequently, the presence of lesions in the bursa is not necessarily an indication of health problems in the flock.

Check the day old chick quality, with a special focus towards mycoplasma vertically transmitted infection.

Revise management practices, starting with biosecurity, good sanitation and litter management.

We have to keep in mind that the improvements made in genetics for a better production yield should be accompanied in parallel with a continuous upgrading in husbandry practices.

For instance, the ammonia control is always a balance between good litter management and proper ventilation.

Check the diet composition and quality. Check the water quality and, if necessary, add 2-3ppm active chlorine.

Also check if the chlorinated water is actually available in all drinkers.

To minimise the spreading risk of any disease to susceptible birds, always travel from youngest to oldest age birds.

Implement a relevant vaccination programme. This includes to chose the right product strengths, to vaccinate at the right time, to deliver every vaccine using the most appropriate administration route, and to check the actual vaccine solution intake by the birds (by using a dye for instance).

Adequate vaccination of the breeder flocks is necessary as well, as Gumboro disease, chicken anaemia and reovirus vaccination in breeders will provide protection to the progeny ('passive immunity') for the critical first weeks of age.

A continuous control of the immunosuppression causes is of paramount importance to protect the integrity and the function of the immune system. This will, in turn, give better flock health, better performances and a better response to any vaccination or infection. ■

Table 2. The target concentration of ammonia.

Ammonia level	Effect
10 ppm	Damage of the trachea ciliae and excessive mucus production
10-40ppm	Reduction in E. coli clearance from the respiratory system, reduction of feed intake, reduction of BB ratio, airsacculitis
40-100ppm	Damage of the eyes, dehydration, tracheitis