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REVIEW OF THE MECHANISMS OF VIRUS-INDUCED IMMUNOSUPRESSION IN POULTRY

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Abstract*. A healthy immune system is the basis of successfully poultry farming. The industry suffers economic losses due to prolonged immunosuppression mediated by several viruses: Marek's disease virus (MDV), chicken infectious anemia virus (CIAV), infectious bursal disease virus (IBDV), reoviruses, some retroviruses as well as their associations. Two categories of causes of viral immunosuppression have been identified: apoptosis and/or necrosis of lymphoid cells and changes caused by the virus in the regulation of the immune response due to disruption of the cytokine profile. In many cases, the actual molecular interactions between virus proteins and host cells are poorly understood. Future research should focus on better understanding the interplay between viruses, immunocompetent cells, and cytokine regulation. Recent developments in the understanding of the immunotoxic and immunosuppressive effects of viruses may potentially offer a way to prevent these conditions.*

Key words*:* immunosuppressive viruses, pathogenesis of immunosuppressive effect of viruses, Marek's disease virus, chicken infectious anemia virus, infectious bursal disease virus

The review is aimed to characterize the pathogenetic mechanisms of action of viruses that cause immunosuppressive disorders in poultry. Poultry health is a combination of the practice of proper maintenance and bioprotection of livestock, which includes, among other things, vaccination. The success of immunization depends entirely on the state of the bird's immune system. But the innate ability to form an immune response depends on many external and internal factors.

Weakening of the functional state of the immune system - immunosuppression - is defined as a state of temporary or permanent dysfunction of the immune response, which occurs as a result of damage to the immune system and leads to increased susceptibility to diseases. Often, the dysfunction of the immune system is the result of infection of cells of the immune system, which leads to a violation of their function against primary and secondary infections. Immunosuppression can be caused by pathogens such as chicken infectious anemia virus, infectious bursal disease virus, reoviruses, and some retroviruses (Schat, 2014; Umar, 2017).

Depression of the humoral immune response is due to impairment of soluble factors such as complement and chemokines in innate immunity and antibodies or cytokines in adaptive immunity. In a cellular immune response, immunosuppression is a consequence of dysfunction of T cells, B cells, heterophils, monocytes, macrophages, and natural killer cells. Control of immunosuppression includes, in general, biosecurity methods, maintenance of appropriate breeding conditions, and vaccination of breeding animals and their offspring (Kaboudi, 2020)

There are two main ways of negative impact of viruses on the immune system. The first is direct destruction (apoptosis) of immunocompetent cells as a result of virus reproduction. This process of complete loss of part of the immune functions is often called immunodeficiency. The second way is changes caused by the virus in the regulation of the immune response due to disruption of the cytokine profile.

The indirect influence or destruction of microbial diversity and integrity of the intestinal microbiota is also described. Commensal bacteria provide protection against pathogens through direct competition and production of antibodies and activation of various cytokines to modulate innate and adaptive immune responses. Violation of microbial homeostasis (dysbacteriosis) is associated with various pathological conditions that contribute to the emergence of acute viral infections in chickens. A group of scientists studied interactions between microbiota and intestinal viruses. The viruses inhibited the production of various interferons (IFNs), interleukins, and virus-specific IgA and IgG, which affected the severity of viral infections in chickens. The results suggest that acute viral infection reduces the abundance of commensal bacteria such as *Lactobacillus, Bifidobacterium, Firmicutes* and *Blautia* spp. populations and enhances the colonization of pathobionts, including *E. coli, Shigella* and *Clostridia* spp. in the body of infected chickens (Abaidulla, 2019).

Infectious bursal disease (IBD), chicken infectious anemia (CIA), and Marek's disease (MD) are the major viral diseases that interfere with vaccine-derived immunity. A common feature is a lymphocytolytic infection capable of suppressing both humoral and cellular immune function. These viruses cause similar changes: atrophy and depletion of lymphoid organs, reproduction in blood monocytes. Each of these viruses has its own specific mechanisms of influence on the immune system (Schat, 2014).

One of the most dangerous lymphotropic viruses is the *Infectious bursal disease virus* (IBDV), which has 2 serotypes and several pathotypes. Only serotype 1 causes immunosuppression and disease in chickens. It has long been recognized that serotype 1 strains replicate in B cells that express surface immunoglobulin M (s-IgM). Replication of the virus leads to cell apoptosis and atrophy of the bursa of Fabricius. Apoptosis of immature B-cells leads to severe immunosuppression with impaired humoral immunity (decrease in antibody synthesis) (Schat, 2014). Important questions remain unsolved, for example: why does part of the bird recover in conditions of suppression of the immune response, what role does cellular immunity and interleukins play? The role of interleukins chIL-2 and chIL-7 was demonstrated by scientists who studied the immunogenicity of the DNA vaccine against IBDV. The bicistronic gene vector chIL-2/chIL-7 was used in the vaccine for co-immunization of chickens together with the DNA vaccine. chIL-2 and chIL-7 have been shown to significantly increase IBDV-specific antibody titers, T-cell proliferation, and IFN-γ production, resulting in a definitive enhancement of vaccine-induced protection (Huo, 2019).

In an experiment on the study of T-lymphocytes of bursa of Fabricius, chickens free from specific pathogens were exposed to a pathogenic strain of IBDV. The virus rapidly destroyed the B cells in the bursa of Fabricius. Moreover, virus replication was accompanied by infiltration of T cells in the bursa. Flow cytometric analysis of single-cell suspensions of bursal tissue showed that T cells were first detected 4 days after virus inoculation. On day 7 after inoculation, 65% of bursal cells were T cells, and only 7% were B cells. Virus-induced bursal T cells produced elevated levels of interleukin-6-like factor and nitric oxide-inducing factor *in vitro*. Spleen and bursa cells from IBDV-infected chickens had increased interferon-gamma gene expression compared to virus-free chickens. In IBDV-infected chickens, bursal T cells proliferated in vitro after stimulation with purified IBDV. The results suggest that intrabursial T cells and T cell-mediated responses may be important in viral clearance and promoting recovery from infection (Kim, 2000).

The discovery of new variants of IBDVs requires studying the effect of these strains on the immune response to vaccine viruses. A group of Chinese scientists investigated the effect of a new variant of IBDV (strain SHG19) on immunization with Newcastle disease (ND) vaccine (strain LaSota) in broilers and laying hens. In commercial broilers, hemagglutination-inhibiting antibody titers against the LaSota strain were reduced by previous infection with the new IBDV variant. The new IBDV variant severely damaged key immune organs (bursa and spleen) and

B lymphocytes in the bursa were severely destroyed, which was the main cause of immunosuppression during Newcastle disease vaccination (Fan, 2020).

Another study compared the virulence of two IBDV strains: the highly virulent UK661 and the classical strain F52/70. There was no difference in peak virus replication in the bursa of Fabricius, but the expression of chicken IFNα, IFNβ, MX1 and IL-8 was significantly lower in the bursa of birds infected with strain UK661 compared with F52/70. Data demonstrate that the UK661 strain induced the expression of lower levels of antiviral type I IFN and proinflammatory genes than the classical strain in vitro and in vivo, and this was partially due to strain-dependent differences in the viral protein VP4 (Dulwich, 2020).

Study of IBDV's pathogenesis was focused mainly on primary lymphoid organs. Whether IBDV infection can produce alterations in intestinal lymphoid tissues (ILT) as well as the composition of the microbiota is poorly understood. In the study, there was a significant reduction in total caecal mucosal thickness in birds infected with a highly virulent strain of the virus (vvIBDV) compared to virus-free controls. vvIBDV infection also resulted in increased numbers of T-lymphocytes and macrophages and decreased numbers of B-lymphocytes in the lamina propria and tonsils of the cecum. The results suggest that vvIBDV infection had a significant effect on ILT and resulted in modulation of gut microbiota composition, which may lead to greater susceptibility of affected birds to gut-borne pathogens (Li, 2018).

The use of such an indicator of immunity as the ratio of bursa to body weight (BVT) is described. Infection caused by IBDV was detected in 43% of poultry farms in Saskatchewan, one of the Canadian provinces. Infected herds had a very low (0.06) bursa-to-body weight ratio compared to a high ratio of IBT (0.17) in uninfected flocks, indicating significant immunosuppression in the former (Zachar, 2016).

Chicken infectious anemia virus (CIAV or CAV), due to its ubiquitous presence in chicken flocks, small size, and resistance to physical and chemical treatments, can be present as a contaminant in other viruses, especially if these agents multiply in chicken embryos. Thus, it can become an additional complicating factor in studies of immunosuppressive properties of other pathogens (Schat, 2014).

The pathogenesis and immunosuppression caused by CIAV have been reviewed in several papers. One of the viral proteins (VP2) performs several roles in viral replication, such as causing apoptosis of infected cells. Rapidly dividing cells are most susceptible to CIAV: hemocytoblasts in the bone marrow, T cell precursors in the thymus, or T cells dividing in response to antigenic stimulation. Infection of hemocytoblasts leads to a decrease in the number of erythrocytes, platelets and granulocytes. The loss of the latter two cell types is important because platelets and granulocytes are important effector cells during bacterial infections and, as a consequence, secondary bacterial infections are often associated with CIAV-induced immunosuppression (Schat, 2014; Tongkamsai, 2019).

To evaluate the pathogenicity of epidemic CAV (CIAV) strains from China, two representative strains CAV-JL16/8901 and CAV-HeN19/3001 and the reference strain Cux-1 were selected for animal experiments. Thymic atrophy and yellowing of the bone marrow were observed in all chickens infected with the isolates and the control strain. It has been proven that epidemic strains in China are more pathogenic than the reference strain Cux-1 (Li, 2021).

The virulence of three CIAV strains was studied in 10-week-old specific pathogen-free (SPF) chickens. The main manifestations of immunosuppression were significantly reduced indices of thymus, bursa and antibody levels. The new strain SD15 not only demonstrated higher pathogenicity, but also the potential ability to disrupt the age-related resistance of older chickens to CAV (Fang, 2023).

Control of CIAV is based on vaccination of breeding animals and maternal antibodies. However, a subclinical manifestation of anemia may occur after a decrease in the level of maternal antibodies. IBDV infection affects the innate immune response during viral replication

and the humoral immune response as a consequence of the destruction of B-cell populations (Schat, 2014).

Associations of chicken anemia virus with other viruses and the influence of coinfections on the work of the immune system were also studied.

Chicken anemia virus (CAV) and *Gyrovirus homsa* 1 (GyH1) belong to the genus *Gyrovirus*. The two viruses cause similar clinical manifestations in chickens, aplastic anemia and immunosuppression. CAV and GyH1 often co-infect chickens. The scientists established a CAV and GyH1 co-infection model in SPF chickens to investigate the synergy between CAV and GyH1. CAV and GyH1 were found to significantly inhibit weight gain, increase mortality, and inhibit erythropoiesis in coinfected chickens. Severe atrophy of immune organs and depletion of lymphocytes was observed in co-infected chickens. These results demonstrate that CAV and GyH1 synergistically contribute to immunosuppression, increasing the potential health risk in the poultry industry (Yang, 2023).

A study was conducted to investigate the possible co-infections of MDV and CIAV in poultry flocks in Nigeria. Coinfections of MD and CIAV were registered in 100% (6/6) and 27.7% (5/18) of material samples from broilers and laying hens, respectively (Adedeji, 2024).

Avian leukemia virus subgroup J (ALV-J) and chicken infectious anemia virus (CIAV) are widely recognized as significant immunosuppressive pathogens that commonly co-infect chickens (Cui N, 2017). When studying the synergistic pathogenic effect, scientists infected SPF chickens and created a model of coinfection of ALV-J and CIAV in HD11 cells. The result of the study showed that ALV-J and CIAV can synergistically promote the secretion of IL-6, IL-10, IFN-α and IFN-γ and apoptosis in HD11 cells, thereby synergistically enhancing pathogenicity and immunosuppression (Xu, 2024).

Marek's disease virus (MDV), or *Gallid herpesvirus* 2, causes T-lymphocyte tumors in chickens. It attracted the attention of virologists and immunologists as highly successful vaccines became available to protect against the disease in the early 1970s. Since then, the virus has evolved from virulent to highly virulent (vv) and, more recently, to vv+ pathotypes. This increase in pathogenicity has not only increased the incidence of tumors, but has also been associated with some new neurological syndromes. In addition, vv+ strains caused more severe damage to lymphoid organs than virulent and vv pathotypes, likely resulting in increased immunosuppression. Genetic resistance to MD is a promising alternative strategy to enhance current disease control measures (vaccination and treatment). Four virus genes DEG, FGA, ALB, FN1, and F13A1, which are reported to contribute to virus invasion or immunosuppression, were found to be significantly upregulated in susceptible line 72 but downregulated in resistant line 63 birds. These results provide new resources for future research to further elucidate the genetic mechanism conferring resistance to MD (Dong, 2017).

Leukemia viruses (ALV) and *Rous sarcoma viruses* (REV) belong to *Retroviridae*. REV and exogenous ALV can be transmitted congenitally or horizontally. Chicks hatched from congenitally infected eggs are tolerant and unable to produce neutralizing antibodies to the virus. Some ALV-J isolates can cause thymic and bursal atrophy and reduce the antibody response to HDV vaccines in chicks infected at day-old and vaccinated 7 days later. However, no reduction in response was found when chicks were vaccinated at 30 days of age. In addition, ALV-J infection did not reduce the antibody response to the IBDV vaccine. The immunosuppressive effects of REV infection on cell-mediated immunity have been studied by several groups of scientists, mostly using mitogen stimulation assays. Immunosuppressive effects are associated with the development of T-reg suppressor cells (Schat, 2014).

In the scientific literature there are references to the immunosuppressive properties of some other poultry viruses.

Avian reovirus infections can cause tenosynovitis and other diseases in chickens or lead to subclinical infection. Reovirus infection reduces the responses of peripheral blood monocytes and splenocytes to mitogens. Poultry enteritis and mortality syndrome (PEMS) is

characterized by diarrhea, growth inhibition, and immunosuppression with a multifactorial etiology. Turkey reovirus is associated with some aspects of PEMS, including reduced thymus and bursal mass (Schat, 2014).

Virulent avian *Adenoviruses*, which also have immunosuppressive capacity, have been described. Newcastle disease can damage lymphoid tissues and macrophages. Pneumoviral infections of birds disrupt the mucociliary functions of the upper respiratory tract and increase secondary bacterial infections (Hoerr, 2010).

In the early 1930s, infectious bronchitis (IBV) was first characterized as a respiratory disease of young chickens; later the disease was also described in older chickens. IBV is subject to constant changes in the genome due to mutations and recombinations, which leads to changes in clinical and pathological manifestations. Over the years, different IBV genotypes associated with reproductive, renal, gastrointestinal, muscular, and immunosuppressive manifestations have emerged. The evolution of new IBV strains over the past nine decades against the vaccine-induced immune response and changes in clinical and pathological manifestations highlight the need for rational development of intervention strategies based on a deep understanding of the interaction of IBV with the host (Najimudeen, 2020).

One solution to prevent immunosuppressive conditions is to improve the ability of birds to resist viruses through genetic selection while maintaining industry standards. However, more research is needed to improve the understanding of the interaction between immune cells and viral proteins in order to effectively develop new vaccines that protect birds against new highly pathogenic strains, can regulate cytokine expression and have the ability to activate cells of the innate immune system. Future research should focus on developing vaccines that prevent cytotoxic effects and dramatic changes in cytokine expression and activate innate, cellular, and humoral immune responses.

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ОГЛЯД МЕХАНІЗМІВ ВІРУС-ІНДУКОВАНОЇ ІМУНОСУПРЕСІЇ ПТИЦІ

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Резюме. *Здорова імунна система є основою птахівництва. Галузь зазнає економічних збитків через тривалу імуносупресію, опосередковану кількома видами вірусів: вірусом хвороби Марека (MDV), вірусом інфекційної анемії курей (CIAV, вірусом інфекційної бурсальної хвороби (IBDV), реовірусами та ретровірусами, а також взаємодії між ними. Було визначено дві категорії причин вірусної імуносупресії: апоптоз та/або некроз лімфоїдних клітин і зміни, спричинені вірусом в регуляції імунної відповіді через порушення цитокінового профілю. У багатьох випадках фактичні молекулярні взаємодії між білками вірусу та клітинами-хазяїнами недостатньо вивчені. Майбутні дослідження повинні зосередитися на кращому розумінні взаємодії між вірусами, імунокомпетентними клітинами та цитокіновою регуляцією. Останні розробки в розумінні імунотоксичних та імуносупресивних ефектів вірусів можуть потенційно запропонувати спосіб запобігання цим станам.*

Ключові слова: віруси, що викликають імуносупресію, патогенез імуносупресивної дії вірусів, вірус хвороби Марека, вірус інфекційної анемії курей, вірус інфекційної бурсальної хвороби.

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