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Review Article

Maternal Immunity in chickens

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Abstract:

Maternal Derived Antibodies (MDA) or passive immunity is passed from hyper-immunized or naturally infected breeder hens to the progeny through the egg. Developing avian embryos and neonatal chicks are transiently protected against bacterial toxins, bacteria, parasites and viruses by maternal immunoglobulins (Ig) transferred via the yolk. Passive Immunity has relatively short duration, commonly 1-2 weeks and generally less than 4 weeks. The half-life time of MDA was estimated to be 3-4 days in broiler and 5-5 days in breeders. Its function is to protect young chicks during the first few weeks, when their immune system is not fully developed to proper react to an early challenge. while its disadvantage is it's interferes with vaccine efficacy in chicks as high levels of maternal antibodies, live vaccines administered during the first two weeks of life may be neutralize.

MDA has become an important factor in deciding vaccination policy in the poultry industry. The most common serological test used for Evaluation of maternal immunity is Enzyme Linked Immuno Sorbent Assay (ELISA). Other additional serological tests can be used to evaluate vaccine administration–virus neutralization (VN) will show the level of neutralizing or protective antibodies.

Nowadays, the vaccine technology derived to create subute and recombinant vaccines those can overcome MDA.

The objective of this mini review is to collect the published data to be available for students, practical poultry men and researches in the poultry industry.

Key words: Maternal immunity, Chickens, Function, Half life time, Evaluation, Duration.

Introduction

Maternal immunity, a form of passive immunity, plays a vital role for the one day old chicks. Birds transfer maternal antibodies (MAb) to their offspring through the egg yolk where the antibody is absorbed and enters the circulatory system. MAb are immunoglobulins that are transferred from vaccinated or naturally infected breeder hens to the progeny through the egg, which provide passive immunity to progeny and protect them against infectious agents that cannot be cleared by the immature immune system [1,2]. MAb may interfere with the vaccination efficacy in the chick, as Mab reduce the growth-suppressive costs of an innate immune response toward pathogens during early development of the immune system [3]. However, this passive immunity has relatively short duration. The level of MDA peaks at 3 to 4 days post hatch, and then gradually decreases to undetectable levels at 2 to 3 weeks of age [2].

Maternal immunity

The immune system of newly hatched chick is only partially mature and therefore is not capable of providing complete protection against pathogens upon its first encounter with the external environment after hatching. Transfer of maternal antibodies helps to protect the offspring until adaptive immune responses become fully effective. This was first described by Felix Klemperer at 1893, who observed that in birds, immunity against tetanus bacteria is transmitted via the egg yolk. It was later addressed by Brierley and Hemmings, who demonstrated the selective transport of antibodies from yolk sac to the circulation of the embryo and hatched chick. These and other studies laid the foundations for the concept that in birds maternal antibodies are deposited into the egg and subsequently absorbed by the developing embryo [4,5]. The phenomenon has been exploited in vaccination strategies by the poultry industry; point-of-lay pullets are usually boosted with vaccines to raise their circulating antibody levels during lay, so that protective maternal antibodies are transferred to the offspring.

IgY is selectively secreted from the circulation of the hen into the egg yolk [6,7]. The amount of IgY transferred across the follicular epithelium into the yolk is pro- portional to the IgY concentrations in serum and mediated by an active transport mechanism [2,8-11]. Antigen-specific IgY antibodies are found in the newly laid egg with a delay of 5–6 days in comparison with the serum antibody concentration. This is explained by the time required for follicular development and oviposition [7]. Yolk IgY concentra-

tions were reported to be in the range of 20–25 mg/ml, 7.9 mg/ml, and 10 mg/ml for the chicken, depending on the analytical method used [11-13].

The second step in maternal antibody transfer requires absorption of IgY across the yolk sac membrane into the embryonic circulation. This transport process begins at a slow rate around the 7th day of embryonic life and increases steeply during the 3 days before hatching to reach a capacity of 600 μg/day [8,11]. It has long been proposed that this process is Fc receptor-mediated since chicken IgY – but not mammalian IgG – is capable of being transported, and binding of IgY to the yolk sac membrane is pH dependent [14]. Recently, the receptor has been cloned and named FcRY [15].

The total amount of IgY absorbed by the embryo represents only 10% of that deposited into the egg yolk. The fate of the remaining 90% is not known, though most likely it is digested proteolytically along with the residual yolk contents [11]. This may explain the approximate 8-fold lower antigen-specific antibody titres in neonatal serum compared with those in yolk. Total serum IgY levels increase to their maximum value about 2 days after hatching, reaching 1–5 mg/ml, and subsequently they decline until de novo synthesis of IgY becomes evident. Comparable kinetics have been described for the duck, with peak levels at day 5 and minimum levels at day 14 after hatching [16].

While IgY is only found in the yolk, but not the albumen, the converse is true for IgM and IgA antibodies. Only minute amounts of these two isotypes can be detected in the yolk, with most being secreted into the egg white Rose and Orlans, reaching concentrations of 0.15 and 0.7 mg/ml for IgM and IgA, respectively [12,17,18]. During embryonic development these isotypes are distributed to the yolk sac and the amniotic fluid, but not transferred into the embryonic circulation [19-21].

Building up high maternal immunity

Maternally-derived antibody (MDA) has become an important factor in deciding vaccination policy in the poultry industry. Prime boost strategies, using live vaccines followed by inactivated vaccines, are utilized to protect the laying hen or breeder from disease throughout the laying period. A consequence of this is the transfer of high levels of antibody into the progeny. Originally, there were doubts in regard to the isotypes that could be transferred to the yolk [12,17]. Breeder chickens were given oil adjuvant Avian influenza (AI- H9N2) vaccines in 3-4 weeks before start of egg production to confer high maternal antibodies to their progeny that lasted for the 1st 3-4 weeks of age [22].

Advantage of maternal immunity

Maternal antibodies helps to protect the offspring until adaptive immune response becomes fully effective, the maternal/progeny relationship is important in the protection of young animals particularly against endemic infections [23]. MAb levels play crucial role in the health status of the modern day broiler-chicken industry because of the short lifespan of broiler chickens. Commercial broiler chickens are being protected against certain pathogens such as CIA, AEV, and Reovirus, solely by providing sufficient levels of maternal antibodies from their parent flock [24-26]. Maternal antibodies also play a major role in modulating early life live vaccine strategy for commercial broiler flocks [27].

In birds all antibodies are transferred via the egg, but the IgY which is the major maternal antibody transferred via the egg yolk. High titres of specific IgY are deposited into the yolk of the developing egg and have a transient, but essential, role protecting chicks from pathogens such as: Eimeria, Cryptosporidium, Salmonella and Campylobacter [28-32].

The importance of maternal antibody declines with the age of the chick and is considered most important during the first 3 weeks of age. Transfer of protective antibody into the yolk requires high levels of circulating antibody in the hen and naturally occurring maternal protection is probably limited to those infections currently in the environment at the time of lay. For example, the transitory nature of maternal protection was evident with E. maxima where significant protection occurred only in chicks obtained from the eggs laid between 17 and 30 days post-infection [12]. Nonetheless, the maternal protection route has been exploited in the control of coccidiosis and a commercial vaccine (CoxAbic®) has been developed, based on the maternal transfer concept [29].

The potential for exploiting maternal immunization is attractive in many ways, since large numbers of offspring can be protected by intensive immunization of a relatively few hens. Although this route of immunization deserves further exploitation, protection is transient and maternal transfer of antibody (either from natural ongoing infection or immunization) may also interfere with direct immunization of the chick [21].

Disadvantage of maternal immunity

One of factors which interfere with vaccine efficacy in poultry is MDA; Maternal immunity. In presence of high levels of maternal antibodies, live vaccines administered during the first two weeks of life may be neutralized [33]. Maternal antibody decrease the immunity after vaccination [27, 34].

Weaning of maternal immunity

Maternal antibody can persist for up to a month after hatching [2]. Maternal antibody titres in chickens are the highest directly after hatching, and decrease to zero in 3 or 4 weeks [35].

The half-life of the IgY in the serum of the hatched chick has been estimated to be between 3 and 6 days and in line with this MDA is catabolized over a period of 3–4 weeks in the progeny [21]. Duration of mAb in broiler chickens has been studied extensively for IBDV. Half-life of mAb against IBDV ranged from 3 to 8 d in several previous studies [10,27,36-38].

In newly hatched layer type chicks, maternally derived antibodies exhibit a linear or curvilinear decline with a mean half-life of 5 to 6 days. Fahey et al. reported a half-life of 6.7 days for IBDV-specific maternal antibodies. It is gene- rally thought that the half-life of maternally derived antibodies in broiler lines is much shorter, approximately 3 days [23,38]. Allen et al. and Darbyshire and Peters reported that the half-life of MAb for NDV and IBV was 4.5 and 5 to 6 d, respectively [39,40]. The half-life time of maternal antibodies against AI- H9N2 expressed as loss of one HI log 2 between groups was ranged from 3.3- 7.2 days; with average 5.1- 5.6 days. Half life time by ELISA titre was in average of 8.9 days. Correlations between HI and ELISA ranged from 0.83- 0.94 [22].

Interaction between maternal immunity and vaccination

A high level of MAb against IBDV neutralizes the vaccine virus and results in complete failure of immunization [41]. There is also recent evidence indicating that MAb decreases the efficacy of killed vaccine against AIV [35,42,43]. Therefore, estimating the half-life of MAb has become necessary and valuable information that can be used in designing vaccination programs for broiler chickens to minimize the cross reaction between MAb and the vaccination programs [27]. MAbs against NDV, as an example, have a favorable effect of decreasing the severity of live vaccine reaction [27].

How to overcome maternal immunity

For the primary vaccination of young chickens against viral poultry diseases such as ND and IBD, live vaccines are mostly used. Depending on the antibody titres and the virulence of vaccine viruses, it can be predicted at what age young chickens can be vaccinated efficiently [35,44].

MDAs specific to IBDV antibodies may interfere with early vaccination with live vaccines. Thus new technologies and second-generation vaccines

including rationally designed and subunit vaccines have been developed. Recently, live viral vector vaccines have been licensed in several countries and are reaching the market. Here, the current status of IBD vaccines is discussed [23]. MDAs substantially interfere with active immunity in post-hatch vaccination, although they provide early protection against disease through passive immunity in young chickens.

Previously, NDV strain TS09-C was demonstrated to be safe and immunogenic as in-ovo vaccine in specific-pathogen-free chickens, the maternally derived antibodies against NDV do not significantly interfere with the ability of the in-ovo vaccine strain TS09-C to induce protective cellular immunity [45]. NDV recombinants expressing the ILTV glycoproteins B and D have previously been demonstrated to confer complete clinical protection against virulent ILTV and NDV challenges in naive chickens, Overall results indicate that the presence of maternal antibodies to NDV and ILTV did not significantly interfere with the ability of the NDV LaSota strain-vectored ILTV gB and gD vaccine candidates to elicit protective immunity against infectious laryngotracheitis [46].

The immunogenicity of vaccines in young chicks with MDA depends on the vaccination scheme and the type of vaccine used in their parent flocks. The heterologous prime-boost in birds with MDA may at least partially overcome MDA interference on inactivated vaccine [47].

Maternal immunity and prevention of viral infections

MDA is very protective especially when the antibody response correlates with protection. Chicks with MDA can be protected against infection with IBDV, but MDA also modulates infections with MDV, NDV, IBV and Reovirus [26,40,48].

In the case of IBV, IgY MDA can be detected in the respira- tory mucus of hatched chicks [49]. This antibody can protect chicks against IBV challenge [40,50], although since MDA in the respiratory tract declines more rapidly than serum antibody, a rapid decline in protection provided by MDA to respiratory diseases [1,21].

Commercial broiler chickens are being protected against certain pathogens such as CAV [24] and AEV [25], by providing sufficient levels of maternal antibodies from their parent flock. Maternal antibodies also play a major role in modulating early life live vaccine strategy for commercial broiler flocks [27].

Evaluation of maternal immunity

Maternal antibody titers were measured by ELISA for AEV, AIV, CAV, IBDV, IBV, ILTV, Mycoplasma gallisepticum MG, Mycoplasma synoviae (MS), and Reovirus. The results are also quantitative for most antigens – giving Mean Titers, Geometric Mean Titers (GMT) and Coefficient of Variation (%CV) in the results. The desire is to achieve high GMT's and low %CV for the common antigens - IBDV, NDV, IBV and Reovirus. Poor vaccine administration of parent birds can raise the %CV and lower GMT of chicks sampled. This may be explained by high numbers of missed parent birds, vaccine leakage or improper location of injection. Maternal antibody titers for NDV were measured using a hemagglutination inhibition test. Half-life estimates of maternal antibody titers were 5.3, 4.2, 7, 5.1, 3.9, 3.8, 4.9, 4.1, 6.3, and 4.7 days for AEV, AIV, CAV, IBDV, IBV, ILTV, MG, MS, NDV, and Reo, respectively. The statistical analysis revealed significant differences among half-lives of maternal antibody titers against certain pathogens. Furthermore, all maternal antibody titers were depleted by 10 d of age except for IBDV [27].

Conclusion

The passive immunity is protective during the first 2 to 5 weeks for most of the viral diseases in chickens. The protective capacity and the duration depend on the level of MDAs transferred. Therefore, accurate and uniform immunization of the breeders is fundamental in order to provide high and uniform titers in day-old chicks. The MDAs interfere also with active immunization with live vaccines. Therefore, this should be considered in the set up of a vaccination program.

Abbreviations

- AEV: Avian encephalomyelitis virus
- AIV: Avian influenza virus
- CAV: Chicken anemia virus
- ELISA: Enzyme Linked Immuno Sorbent Assay
- IBD: Infectious bursal disease.
- ILTV: Infectious laryngotracheitis virus
- MAB: Maternal immunity
- MDA: Maternally-derived antibody
- NDV: Newcastle disease virus

Declarations

All data collected in this mini review are included in this published article.

Competing Interests

The author declares that they have no competing interests.

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