

Review

Nutritional Balance Matters: Assessing the Ramifications of Vitamin A Deficiency on Poultry Health and Productivity

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Abstract: Vitamin A, a critical micronutrient, plays a vital role in maintaining poultry health and maximizing productivity. This comprehensive review paper conducts a thorough analysis of the consequences of vitamin A deficiency in domestic fowl. It delves into the physiological functions of vitamin A and investigates how hypovitaminosis A impacts growth, immune function, reproduction, and overall poultry performance. Additionally, the review explores effective strategies for preventing and managing vitamin A deficiency, such as dietary adjustments and supplementation, while addressing the specific requirements for vitamin A intake. The implementation of these strategies holds immense importance in optimizing poultry management practices and achieving peak performance in poultry production. A profound understanding of the prevalence and factors contributing to clinical and subclinical vitamin A deficiency in domestic fowl is essential for ensuring the efficiency of poultry farming operations. Recognizing the pivotal role of vitamin A and applying the appropriate measures empowers poultry farmers to enhance the health outcomes and overall performance of their flocks.

Keywords: vitamin A; retinol; supplementation; poultry health; requirement; deficiency; performance



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1. Introduction

The well-being and productivity of poultry are closely tied to their nutritional status. Adequate nutrition is essential for maintaining optimal health, growth, and overall productivity in poultry flocks. Nutritional imbalances can lead to various health issues, reduced performance, and economic losses for poultry producers [1]. Among the many essential micronutrients, vitamins play a crucial role in supporting vital physiological functions. Understanding the significance of vitamins, particularly vitamin A (retinol), in poultry health and productivity is paramount for optimizing poultry management practices [2,3].

Vitamins are organic compounds that are required in small quantities but are essential for normal metabolic processes in domestic fowl [4]. They act as cofactors for various enzymatic reactions and are involved in critical physiological functions such as growth, reproduction, immune response, and vision. Among the different vitamins, vitamin A stands out as a key player in maintaining optimal health and productivity in animals [5]. It plays a vital role in vision, embryonic development, bone growth, immune function, and maintaining the integrity of epithelial tissues.

Exclusively animal-derived feeds such as fish liver oil and fish meal are the primary sources of vitamin A. However, there are certain plant pigments called carotenoids (e.g., β -carotene) that have the potential to exhibit vitamin A activity via metabolic conversion [2,6]. It is important to note that only a small portion of the carotenoids found in nature can be converted into vitamin A by mammals and birds [7]. The efficiency of this conversion depends on various factors such as the type and dietary concentration of protein and fat, total carotene load, animal species, and the bird's vitamin A supply status [8,9]. Consequently, synthetic retinol, primarily in the form of retinyl acetate, is commonly applied in animal nutrition to meet the dietary requirement for vitamin A [3].

Vitamin A deficiency can have severe consequences on poultry health, compromising growth, reproduction, immune competence, and overall productivity [10]. Acute hypovitaminosis A is rare among poultry populations because retinol in the form of retinyl acetate is usually supplemented using premixes in domestic fowl feed [11,12]. However, subclinical vitamin deficiencies may occur more frequently [13]. Various factors contribute to the occurrence of hypovitaminosis A, such as incorrect premix and feed formulations, improper storage conditions for premixes and feeds, inadequate feed management practices, as well as contextual criteria. Poultry raised in intensive production systems may be particularly susceptible to suboptimal levels of micronutrients due to the presence of negative environmental factors and stressors [14,15]. These factors include high ambient temperature, high stocking density, microbial challenges, and hygienic issues. Under such conditions, the vitamin A requirement for poultry may increase [4,16].

This review paper aims to assess the implications of vitamin A deficiency on poultry health and productivity. Its specific objectives are as follows:

1. Describe the critical contributions of vitamin A to the metabolism, growth, immunity, antioxidant capacity, and reproduction of avian species.
2. Identify the key factors that contribute to vitamin A deficiency in domestic fowl.
3. Identify the main clinical manifestations of vitamin A deficiency in poultry.
4. Discuss effective intervention strategies for preventing and managing vitamin A deficiency in poultry populations.
5. Address considerations for meeting the vitamin A requirements of poultry.

By addressing these objectives, this research synthesis aims to enhance our understanding of the significance of nutritional balance, particularly vitamin A, in optimizing poultry health and productivity. Such knowledge can inform effective management strategies to mitigate the detrimental effects of hypovitaminosis A in poultry populations, thereby benefiting both poultry producers and consumers.

2. The Crucial Contributions of Vitamin A: Metabolism, Growth, Immunity, Antioxidant Capacity, and Reproduction

Vitamin A plays a pivotal role in the intricate physiology of poultry, showcasing a myriad of essential functions. At the forefront, its three forms, known as retinol (the alcohol), retinal (the aldehyde), and retinoic acid (all-trans retinoic acid, ATRA) [17], assume a critical role in a range of metabolic processes, fostering growth, bolstering immunity, and even supporting the delicate realm of reproduction [18].

(1) Digestion and Metabolism:

Vitamin A digestion and metabolism in poultry are complex processes that ensure the efficient utilization of this essential micronutrient. In domestic fowl, vitamin A is obtained via the diet in the form of supplemented retinyl esters [3]. After ingestion, the feed undergoes enzymatic hydrolysis in the digestive system, where pancreatic enzymes such as esterases and lipases break down the retinyl esters into free retinol [19].

To facilitate absorption in the small intestine, the free retinol is incorporated into mixed micelles, which are small structures formed in the presence of bile salts and phospholipids [20]. These micelles aid in the absorption of retinol by the absorptive cells of the intestinal lining, known as enterocytes [7]. The enterocytes take up the retinol via specific transport proteins [21]. Once absorbed, retinol enters the bloodstream and is transported to the liver.

In the liver, retinol undergoes esterification, a process where it is conjugated with fatty acids to form retinyl esters. These esterified forms of vitamin A are stored in hepatic stellate cells within the liver, serving as a reservoir for the release of retinyl esters into circulation as needed [22].

Within target tissues, such as the eyes, skin, immune system, and reproductive organs, retinyl esters are hydrolyzed back into retinol [17]. This conversion is facilitated by cellular enzymes, and the released retinol can be further metabolized into its active form, ATRA [23].

ATRA acts as a ligand for nuclear receptors, regulating the expression of genes involved in various physiological processes, including vision, growth, reproduction, and immune function [24].

Any excess retinol that is not immediately needed can partly be eliminated via biliary excretion [25–27] or reesterified and stored in hepatic stellate cells [22]. The initial step of biliary excretion involves the secretion of retinol and its metabolites into the bile, enabling their eventual removal from the body via feces.

(2) Growth and Development:

ATRA plays a crucial role in regulating the ontogeny of poultry. It exerts its effects via various mechanisms that primarily focus on promoting growth and supporting organ development [28]. By binding to specific retinoic acid receptors (RARs) in target cells, ATRA acts as a transcriptional regulator, activating the transcription of genes involved in cell differentiation, proliferation, and tissue development [23]. This process ensures proper cell specialization and the formation of specific tissues and organs during embryonic development [29].

In addition, ATRA influences skeletal development in poultry by promoting the synthesis and mineralization of bone tissue. It regulates the expression of genes associated with bone growth and remodeling, resulting in the formation of a robust skeletal system [30]. Moreover, ATRA affects the development and function of the vital organs involved in growth. It stimulates the growth and maturation of intestinal villi, thereby enhancing nutrient absorption and digestion [31,32]. In the respiratory system, ATRA plays a role in lung development and surfactant production, facilitating efficient gas exchange [33,34].

Furthermore, ATRA supports growth and development by stimulating the production of growth factors such as insulin-like growth factor (IGF) and transforming growth factor-beta (TGF- β) [35]. These growth factors play crucial roles in tissue maturation, repair, and overall development.

(3) Immune Function:

Vitamin A, specifically in ATRA, plays a vital role in supporting a strong immune system in domestic fowl. ATRA binds to specific nuclear receptors RARs and retinoid X receptors (RXRs) present in the immune cells [36]. The binding of ATRA to these receptors triggers a sophisticated cascade of processes that regulates the transcription of genes and the synthesis of proteins, ultimately impacting immune function [37].

One of the primary ways in which vitamin A (via ATRA) regulates gene expression is by influencing the differentiation and maturation of immune cells [38]. Retinoic acid signaling facilitates the activation of cistrons, which are essential for lymphocyte development and triggering [39]. It plays a role in promoting the differentiation of T cells into various effector subsets, including helper T cells (such as Th1, Th2, and Th17) and regulatory T cells (Tregs) [40–42]. These subsets of T cells have critical functions in coordinating immune responses and maintaining immune balance. Additionally, ATRA aids in the maturation of B cells and enhances the production of specific antibodies by regulating the genes involved in antibody synthesis [43].

Furthermore, vitamin A plays a significant role in regulating the expression of genes associated with the integrity and barrier function of epithelial cells. ATRA signaling stimulates the production of proteins that maintain the structural resilience of epithelial cells, such as tight junction proteins and mucins [44–47]. These proteins contribute to the physical barrier that prevents the entry of pathogens into the body. By regulating the expression of cistrons involved in epithelial cell function, vitamin A helps fortify the first line of defense against infections.

Another important mechanism by which vitamin A impacts immune function is through its regulation of cytokine production. Cytokines are signaling molecules that coordinate immune responses and inflammation. ATRA influences the expression of the genes encoding cytokines and their receptors, thereby modulating the immune response [48,49]. For example, ATRA enhances the production of interleukin-10 (IL-10), an anti-inflammatory

cytokine that helps mitigate excessive immune responses [50]. Moreover, vitamin A also promotes the expression of cistrons responsible for antimicrobial peptides, which are vital components of innate immunity [51,52].

(4) Antioxidant Capacity:

Antioxidants play a crucial role in maintaining the health of poultry, and vitamin A is particularly noteworthy due to its remarkable antioxidant properties [53,54].

Vitamin A exerts its antioxidant effects via various mechanisms. One of its principal functions is retinol's ability to scavenge free radicals [55], although there is some controversy surrounding this mode of action [56]. Free radicals are highly reactive molecules that can cause damage to cell membranes, proteins, and DNA, resulting in cellular dysfunction and oxidative stress. By neutralizing these free radicals, retinol potentially helps protect the integrity of cellular components and maintain optimal cellular function [57].

Apart from its possible direct scavenging activity, vitamin A, in the form of ATRA, supports the activity of various antioxidant enzymes, including superoxide dismutase (SOD) and catalase (CAT), through its effect on gene expression [58]. When ATRA binds to RARs, it initiates a cascade of molecular events that ultimately lead to changes in cistron transcription. The ATRA–RAR complex recruits co-activators that facilitate the expression of nearby genes [59]. In the case of antioxidant enzymes such as SOD and CAT, ATRA enhances the transcription of their respective cistrons [60,61]. This increased gene expression results in the synthesis of higher levels of SOD and CAT proteins. SOD is an important enzyme that catalyzes the conversion of superoxide radicals ($O_2^{\bullet-}$) into hydrogen peroxide (H_2O_2) [62], while CAT converts hydrogen peroxide into water and oxygen [63]. These enzymes play a crucial role in neutralizing harmful reactive oxygen species within the cells.

By enhancing the activity of SOD and CAT, vitamin A strengthens the antioxidant defense system of poultry birds [64]. Increased levels of SOD and CAT enable more efficient removal of reactive oxygen species, reducing redox imbalance and preventing damage to cellular components such as proteins, lipids, and DNA [65]. This additional layer of protection against oxidative stress contributes to the overall health and well-being of avian species.

(5) Reproduction:

Vitamin A plays a crucial role in the reproductive processes of domestic fowl. Its mechanism of action involves the regulation of gene expression, specifically through its effects on the retinoic acid signaling pathway. In the context of reproductive organs, ATRA influences the development and function of the testes, ovaries, and oviducts [66]. By binding to RARs and RXRs in these tissues, ATRA regulates the expression of genes involved in the growth, differentiation, and maturation of reproductive cells [67]. In males, ATRA promotes normal sperm production by stimulating the proliferation and differentiation of spermatogonia, which are the precursor cells of sperm [68]. It also plays a role in the maturation and motility of spermatozoa. In females, ATRA is essential for oocyte maturation and fertilization [69]. It helps regulate the production and release of mature oocytes from the ovaries, as well as the transport and viability of the oocytes within the oviducts.

Furthermore, ATRA influences embryonic development and hatchability in poultry [70,71]. During embryogenesis, ATRA controls the expression of genes involved in cell proliferation, differentiation, and morphogenesis [72]. It plays a crucial role in the development of various organ systems, including the cardiovascular system, nervous system, and reproductive system [73,74].

(6) Vision:

The essential role of vitamin A in poultry vision stems from its multiple important functions [2]. One of its primary roles is to support and maintain overall eye [75]. Vitamin A in the form of retinal is vital for the synthesis of rhodopsin, a light-sensitive pigment

found in the retina, which is necessary for vision in low-light conditions [76]. On the other hand, ATRA promotes the development and differentiation of photoreceptor cells, allowing poultry to effectively perceive and process visual information [77]. Moreover, vitamin A helps protect the cornea and conjunctiva, preventing dryness and facilitating the production of a clear fluid by the lacrimal glands in birds [75,78]. This fluid helps maintain optimal vision clarity.

In conclusion, vitamin A is an essential micronutrient for avian species, serving critical functions in their physiology. It regulates gene expression, supports growth and organ formation, strengthens the immune system, and protects against oxidative damage. Additionally, it plays a pivotal role in reproductive processes, influencing organ development and contributing to successful embryonic development and hatchability. Overall, ensuring adequate vitamin A intake is crucial for the health and well-being of poultry.

3. Causes of Vitamin A Deficiency: Identifying Key Factors

Instances of severe vitamin A deficiency in poultry operations have significantly decreased in recent times, with only a few isolated cases observed [79]. This improvement can be attributed to increased awareness among farmers worldwide who are adopting scientific farming practices and implementing effective measures to address this issue. However, subclinical vitamin A deficiency can still be encountered [13].

Several key factors may contribute to vitamin A deficiency in poultry diets, with each factor playing a significant role in identifying the causes and implementing effective solutions. These key elements include:

- (1) **Inadequate feed formulation:** One prominent factor contributing to vitamin A deficiency in poultry diets is inadequate feed formulation [80]. When the formulation of poultry feed does not include sufficient quality sources of preformed vitamin A, it fails to provide the necessary levels of this essential micronutrient. To address this issue, careful consideration should be given to the feed formulation process, including the use of least-cost feed formulation methods [81,82]. It is crucial to ensure the inclusion of stable and highly bioavailable commercial sources of vitamin A to prevent deficiencies.
- (2) **Mishandling and improper storage of poultry premixes and feed** can contribute to the deficiency of essential vitamins, including vitamin A. Over time, exposure to light, heat, and oxygen can lead to the degradation of vitamins in the premixes and feed [83]. For instance, some of the vitamin A sources available on the market, particularly those that are less stable, may lose their vitamin A activity completely if stored at elevated temperatures within a short period (Figure 1) [84]. Therefore, it is crucial to store premixes and feed in cool, dry, and dark conditions for the shortest possible time to maintain optimal levels of vitamin A. Regular quality checks and timely replenishment of premixes and feed stocks are also vital to preserve the potency of vitamin A and prevent its degradation. Furthermore, paying meticulous attention to the thermal treatment of feed is of the utmost importance [85]. Considering the specific example of pelleting, it is crucial to avoid subjecting the feed to excessively high temperatures for prolonged periods of time, as this can result in significant losses in vitamin A activity [86]. Under such conditions, implementing an additional safety margin for vitamin activity becomes sensible.
- (3) **Factors affecting absorption and utilization:** Factors that affect the absorption and utilization of fat-soluble vitamins should not be overlooked [3]. Gut health disorders, parasitic infections, imbalances, or deficiencies of other nutrients (dietary fat, other fat-soluble vitamins etc.), stress, mycotoxins in feed, and certain diseases can impair the absorption of retinol in the digestive tract [87]. To mitigate these factors, implementing measures to promote good gut health, such as using intestinal health promoters or ensuring proper sanitation practices, is important [88]. These strategies optimize vitamin A absorption and utilization in poultry.

- (4) **Bioavailability:** The bioavailability of vitamin A sources is often overlooked, but it plays a crucial role [89]. Unlike other vitamins, vitamin A in the form of retinyl acetate is commonly formulated in small solid beadlet particles by different suppliers (Figure 2). This can result in variations in stability and bioavailability among the vitamin A products available on the market [84]. Experienced formulators can easily create highly stable vitamin A products that can withstand challenging conditions with the help of special formulation aids. However, the main challenge lies in the digestive tract of poultry, where these stable formulations must release retinyl acetate in the intestinal lumen. If the formulations are too stable, they may have reduced or no biological value at all [90]. Therefore, it is essential to strike a delicate balance between the overall stability and bioavailability of a vitamin A commercial product. This balance ensures the product's ability to withstand harsh storage conditions in premixes and high pelleting temperatures in feed, while also facilitating easy release in the digestive tract.

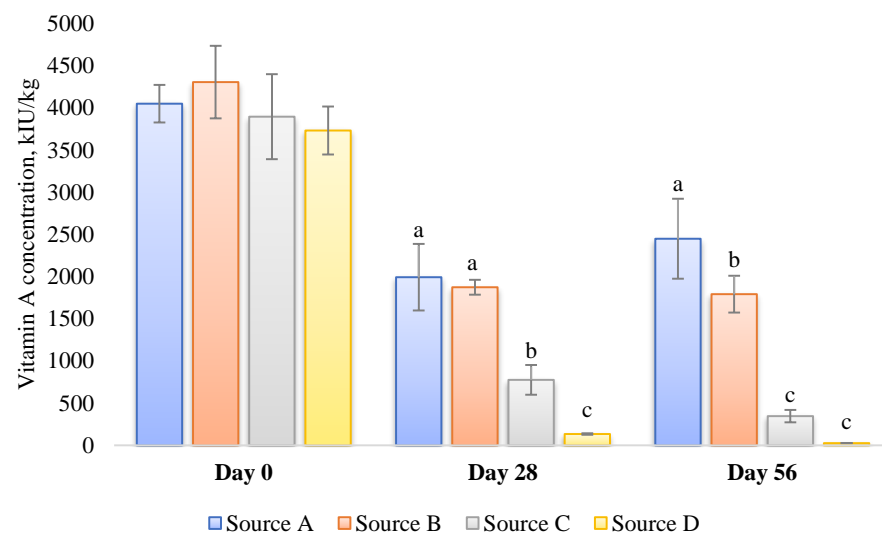


Figure 1. Stability of four different commercial vitamin A sources stored for up to 56 days in vitamin–mineral premix (incl., choline chloride) for broilers at 35 °C and 60–70% r.h. [84]. Values are presented as mean \pm SD ($n = 3$). The premixes were prepared to contain 4.4 Mio IU vitamin A per kg. ^{a–c} Within a time frame (0, 28, or 56 d), values not sharing a common superscript letter are significantly different ($p < 0.05$).

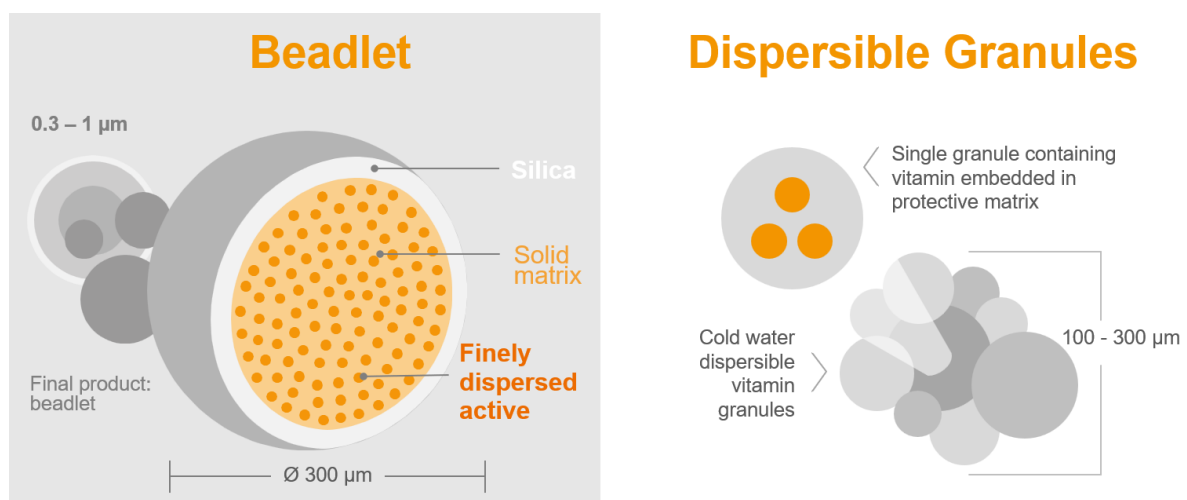


Figure 2. Microencapsulation: Delivering a diverse range of products tailored to target applications.

In conclusion, identifying the causes of vitamin A deficiency in poultry diets requires a comprehensive approach. Addressing inadequate feed formulation, ensuring proper storage and handling practices, sourcing high-quality feed ingredients, and mitigating factors that hinder absorption and utilization are all vital steps in preventing and managing vitamin A deficiency in domestic fowl.

4. Identifying Clinical Manifestations of Vitamin A Deficiency

In 1920, Haring et al. [91] documented multiple outbreaks of a previously unknown disease among flocks of pullets in California, USA. The symptoms exhibited by the affected birds were similar to those of “roup” (respiratory diseases), but there were distinctive characteristics that allowed for a differential diagnosis. Flock owners reported a loss of approximately 10% of their fowls within a span of two to six weeks. Subsequent examination of several affected flocks revealed that 10 to 20% of the birds exhibited typical lesions. Notably, all observed flocks consisted of birds in their first year of laying or pullets that had not yet reached laying age. This marked the first official diagnosis and reporting of vitamin A deficiency in poultry.

Hypovitaminosis A in domestic fowl can manifest via various clinical signs and symptoms, indicating the impact it has on their health and productivity. Early reports suggest that the symptoms of vitamin A deficiency in turkeys and ducks are generally similar to those in chickens. However, Hinshaw and Lloyd [92] and Wolbach and Hegsted [93] have found that the disease is much more severe in the former two species. The following are some common signs observed in poultry affected by hypovitaminosis A:

(1) Reduced growth, high mortality, and poor feed conversion:

Poultry suffering from vitamin A deficiency often display stunted growth rates and inefficient feed conversion, affecting various species such as chickens, turkeys, ducks, geese, and Japanese quail [11,94–99]. This can be attributed to the crucial role of vitamin A in cell differentiation, tissue repair, and protein synthesis, all of which are essential for optimal growth. The importance of vitamin A in promoting growth becomes evident even at the embryonic stage, as several biologically active retinoids, including ATRA, 9-cis-retinoic acid, 4-oxo-all-trans-retinoic acid, and 3,4-didehydro-all-trans-retinoic acid, have been found in the developing embryo [100].

In a study conducted by Fu et al. [101], the impact of vitamin A deficiency on the expression levels of the retinoic acid receptor- β (RAR β) gene was investigated in various tissues of Japanese quail. The results revealed a significant reduction in the mRNA levels of RAR β 2/ β 4 in the brain, liver, heart, lungs, and kidneys when vitamin A was lacking. These findings indicate that vitamin A plays a vital role in the proper functioning and growth of these organs.

Fu et al. [102] also conducted a study on Japanese quail, providing evidence that vitamin A depletion significantly decreased the mRNA levels of IGFBP-5 (insulin-like growth factor binding protein 5) in a tissue-specific manner. Interestingly, this reduction occurred before the observed decrease in body weight. These findings strongly suggest that the expression of certain IGFBPs *in vivo* is influenced by the vitamin A status and imply that the insulin-like growth factor system facilitates the physiological effects of vitamin A on the growth of avian species.

Moreover, Uni et al. [96] reported that the absence of vitamin A in the diet disrupts the normal growth rate in chickens by affecting the functionality of the small intestine and altering the proliferation and maturation of cells in the small intestinal mucosa. In the case of growing ducks, vitamin A deficiency leads to a significant retardation of endochondral bone growth, resulting in disproportional growth of the axial skeleton [93].

(2) Decreased immune response:

Vitamin A plays a crucial role in maintaining a robust immune system in poultry. Its deficiency compromises the integrity and function of various immune cells, reducing their ability to combat infections and diseases [36,38]. Poultry that lack sufficient vitamin A

are more susceptible to bacterial, viral, and parasitic infections, which ultimately leads to increased morbidity and mortality rates [3,103,104].

When the diet lacks vitamin A, it damages the epithelium, the surface layer of the mucous membranes, making it easier for bacteria to enter the poultry's body. Therefore, the value of vitamin A lies in maintaining the "first line of defense", which includes the epithelial structures [105]. In a study conducted by Fan et al. [45], it was found that vitamin A deficiency suppressed the immunity of the airway in neonatal chicks by decreasing the concentrations of IgA and mucin. Additionally, histological observations in vitamin-A-deficient turkeys by Cortes et al. [11] revealed squamous metaplasia of the mucosal epithelium in various organs, such as the oral mucosa, esophagus, sinuses, nasal glands, bronchi, proventriculus, and the bursa of Fabricius. Aye et al. [106] also reported similar symptoms.

Leutskaya and Fais [107] conducted in vivo and in vitro studies, which demonstrated that the antibody content in chickens is influenced by the amount of vitamin A in their diet. Chickens given a high dose of vitamin A (1000 IU of retinyl palmitate daily) had two to five times more antibodies in their serum compared to those not receiving vitamin A (0 IU of retinyl palmitate). Furthermore, administering an additional dose of vitamin A (4000 IU of retinyl palmitate daily) after reimmunization with an antigen from *Ascaridia* further elevated the antibody levels, especially when given on the third day. In vitro experiments using spleen cells showed that the addition of retinyl palmitate to the incubation medium enhanced antibody synthesis. Moreover, introducing retinyl palmitate into vitamin A-deficient chicken spleen cells restored antibody synthesis to the levels observed in control cells. These findings suggest that vitamin A plays a role in the immune process by influencing the synthesis of immunoglobulins.

Bang et al. [108] reported that chickens raised on a diet deficient in vitamin A for 30 days exhibited depletion of lymphocyte and plasma cell populations in their nasal, paranasal, and bursal lymphoepithelial tissues. Infection with Newcastle disease virus further depleted the plasma cells, weakened the inflammatory response, and caused abnormal changes in epithelial tissues, particularly in the bursae. Six days after virus inoculation, the chickens on the diet lacking vitamin A had completely depleted lymphocyte populations in the bursae. These findings highlight the critical role of vitamin A in maintaining immune cell populations and proper tissue function. In laying hens, the absence of this important fat-soluble vitamin in their feed accentuates their susceptibility to fowl pox infection, decreases productivity, and increases mortality rates [79].

Moreover, even a slight deficiency of vitamin A (500 IU of vitamin A per kg of feed) in chickens infected with *Ascaridia galli* led to an elevated excretion of *A. galli* eggs [109]. Additionally, female worms residing in chickens that were fed a diet deficient in vitamin A showed higher levels of egg production at week 5 after infection. These findings clearly highlight the significance of vitamin A in domestic fowl for regulating infections caused by parasitic roundworms.

(3) Ocular abnormalities:

One of the most noticeable signs of vitamin A deficiency in poultry is eye abnormalities. Birds may experience issues such as conjunctivitis, corneal ulcers, and excessive "tearing" [78]. In severe cases, blindness can occur, affecting their ability to navigate and feed properly [110,111].

In a study conducted by Waters [112], pigeons were used to investigate the effect of vitamin A deficiency on dark adaptation. The researcher measured the b-wave height in the electroretinogram and the minimum light intensity required to generate an electrical response as indicators. The results showed that vitamin A deficiency caused a decrease in retinal sensitivity. However, this sensitivity could be restored to normal levels using vitamin A supplementation. Another study by Fu et al. [113] revealed that vitamin A plays a crucial role in maintaining the avian pineal gland's responsiveness to light. Vitamin A deficiency was found to reduce the amplitude of the nighttime surge of pineal melatonin. In

a study conducted by Feng et al. [98], xerophthalmia, a condition characterized by dryness of the conjunctiva and cornea, was observed in ducks due to vitamin A deficiency.

(4) Reproductive disorders:

Vitamin A plays a crucial role in the reproductive processes of poultry, and its deficiency can have significant consequences. Studies have shown that inadequate levels of vitamin A can lead to a decrease in egg production, lower hatchability rates, and an increase in embryonic mortality [94,110]. Additionally, it can cause abnormalities in the reproductive organs, which can result in infertility or reduced fertility in breeding flocks [114].

Research conducted by Thompson et al. [115] revealed that mature cocks fed a diet deficient in vitamin A experienced a reduction in testicular size, loss of spermatids, and degeneration of the seminiferous epithelium. In the case of laying hens, when they were fed vitamin A-free diets for a period of 32 weeks, an increase in the number of atretic follicles was observed [116]. These atretic follicles exhibited moderate to severe hemorrhage. Furthermore, severe ovarian degeneration can occur in laying hens, leading to a significant decline in egg production [117].

(5) Neurological disorders

According to Maden et al. [118], quail embryos that lack vitamin A exhibit significant defects in their central nervous system, including the absence of the posterior hindbrain. Ectopic apoptosis, or abnormal cell death, is observed in two regions: the mesenchyme during the early somite stage and the neuroepithelium of the presumptive posterior hindbrain during a later somite stage. The injection of retinol before gastrulation prevents these apoptotic events, indicating that vitamin A plays a role in preventing cell death. The upregulation of the gene *Msx-2* in the apoptotic neuroepithelium suggests its involvement, while *Bmp-4* remains unaffected. This phenomenon, referred to as positional apoptosis, occurs due to the misspecification of the rostrocaudal position during the development of axial identity and segmentation.

In a study by Wolbach and Hegsted [93] involving the feeding of vitamin-A-deficient diets to growing ducks, it was observed that all birds became paralyzed within 10 days. Gross examination of the deficient birds revealed brain herniations, which were later confirmed using histological analysis. Furthermore, Howell and Thompson [119] reported that feeding Japanese quail a vitamin-A-deficient diet for 25 days resulted in the compression of the brain and spinal cord, accompanied by early nerve fiber degeneration.

In conclusion, vitamin A deficiency in domestic fowl negatively impacts their productivity and immune response, leading to poor growth rates, high mortality, susceptibility to infections, and various health issues, including eye abnormalities, reproductive disorders, and neurological maladies (Table 1). Thus, ensuring sufficient vitamin A levels is essential for poultry health.

Table 1. Some disorders associated with vitamin A deficiency in domestic fowl.

Disorder	Poultry Species	Manifestation	Reference
Immune deficiency	Chick	Reduced T lymphocyte activity after a challenge	[120]
	Chick, turkey poult	Reduced antibody production and T cell proliferative response after a challenge	[121,122]
	Chick	Decreased cytotoxic T lymphocyte activity after a vaccination	[103]
	Chick	Reduced growth of the bursa of Fabricius and thymus	[123]
	Turkey	Squamous metaplasia and hyperkeratinization of glandular epithelium in bursa of Fabricius	[11]
	Broiler chicken	Lowered haemagglutination inhibition titers after a vaccination	[124]
	Broiler chicken	Fewer intraepithelial lymphocytes expressing surface markers CD3, CD4, CD8, $\alpha\beta$ TCR, and $\gamma\delta$ TCR after a challenge; higher shedding of <i>Eimeria</i> oocysts and lower levels of interferon- γ	[104]

Table 1. Cont.

Disorder	Poultry Species	Manifestation	Reference
Immune deficiency	Broiler chicken	Elevated excretion of <i>A. galli</i> eggs	[109]
	Broiler chicken	Lower geometric antibody mean titer	[125]
	Broiler chicken	Increased viral replication and reduced serum immunoglobulin G levels	[126]
	Broiler chicken	Reduced cutaneous basophil hypersensitivity and heterophil and lymphocyte counts	[127]
	Broiler chicken	Reduced cytokine production	[128]
	Broiler breeder	Reduction in antibody titers after a vaccination	[129,130]
	Goose	Lower serum immunoglobulin A and G levels	[131]
	Goose	Lower immune organ weight, immune organ index, and immunoglobulin content	[132]
	Laying hen	Lower plasma immunoglobulin G content and mRNA expression of interleukin-10 in the spleen	[133]
Reproductive dysfunction	Laying hen	Decrease in egg production and lower hatchability rates	[94]
	Turkey breeder	Decrease in egg production, lower fertility and hatchability rates, and an increase in embryonic mortality	[110]
	Broiler breeder	Reduced ovarian stromal and white follicle weights along with oviduct weight and length; reduction in insulin-like growth factor 1 receptor transcripts in the ovarian stroma and the walls of yellow follicles, follicle stimulating hormone receptor expression in the walls of white and yellow follicles, and luteinizing hormone receptor and growth hormone receptor transcripts in the walls of yellow follicles; expression of apoptosis gene transcripts in the ovary; reduced egg weight	[114]
	Cock	Reduction in testicular size, loss of spermatids, and degeneration of the seminiferous epithelium	[115]
	Laying hen	Increase in the number of atretic follicles, including moderate to severe hemorrhages	[116]
	Laying hen	Reduction in egg number, egg mass, and albumen quality	[134]
	Laying hen	Reduction in egg number, egg mass, and albumen quality	[134]
Abnormalities in digestive, respiratory, cardiovascular and urinary system	Turkey breeder	Pustules in the mouth and esophagus, urates in the ureters and kidneys	[110]
	Turkey	Squamous metaplasia and hyperkeratinization of glandular epithelium in the esophagus, proventriculus, and nasal glands	[11]
	Turkey	Pustules in the mouth and pharynx, urates in the intestinal mesentery and pericardium	[135]
	Turkey	Squamous metaplasia in the oesophagus, hyperuricemia	[136]
	Chicken	Gross lesions characterized by the presence of white to yellowish caseous, coalescing nodules in the mucous glands and ducts of the pharynx and esophagus	[137,138]
	Goose	Reduced villus height and width, crypt depth, and muscular layer thickness in the duodenum, jejunum, and ileum	[131]
	Chick	Metaplasia of the nasal respiratory epithelium to a stratified squamous form	[119]
	Turkey	Squamous metaplasia affecting the epithelia of the digestive, respiratory, and urinary tracts	[35,106]
	Turkey	Squamous metaplasia affecting the epithelia of the digestive, respiratory, and urinary tracts	[35,106]
Neurological maladies	Chick	Constriction of the central nervous system and the occurrence of Wallerian degeneration specifically in the ventral and lateral columns of the cervical spinal cord	[119]
	Laying hen	Herniation of cerebral tissue, thinning of the cerebellar flocculi, ridging of the lumbosacral spinal cord, and early Wallerian degeneration in the cervical cord	[139]
	Chicken	Constriction of the brain and spinal cord	[140]
	Chick	Pin-pointed areas of degeneration in the brain stem, the base of the cerebellum, the optic chiasma, and the cerebrum	[141]
	Chick	The brains exhibited dispersed pyknotic neurons, predominantly concentrated in the optic tectum and the Purkinje cell layer of the cerebellum	[142]
	Japanese quail	Abnormalities in neural tissue	[119]

Table 1. Cont.

Disorder	Poultry Species	Manifestation	Reference
Ophthalmic anomalies	Turkey	Increased lacrimation, inflammation of the nictitating membrane	[92]
	Pigeon	Decrease in retinal sensitivity	[112]
	Chicken	Conjunctivitis, corneal ulcers, and excessive lacrimation	[78]
	Japanese quail	Diminished sensitivity of the pineal gland to light stimuli	[113]
	Duck	Keratoconjunctivitis sicca	[98]
	Turkey	Development of watery eyes followed by the accumulation of caseated matter in the eyes and blindness	[110]
Skeletal disorders	Chick	Pathological changes in the epiphyseal cartilage zone of the femur and in the periosteum of the vertebrae; the vertebral periosteum exhibited heightened osteoblast activity and excessive cartilage formation	[119]
	Japanese quail	Overgrowth of periosteum	[143]
	Duck	Altered endochondral bone growth leading to disproportional axial skeleton development	[93]
	Laying hen	Lesions in the bone tissue	[139]
Hindered growth and development	Chick	Hindered potential for growth	[94]
	Broiler chick	Reduced growth performance	[121]
	Turkey poult	Stunted growth	[122]
	Broiler chicken	Reduced growth performance	[128]
	Duck	Decreased growth	[98]
	Broiler chicken	Diminished growth	[144]

5. Addressing Effective Intervention Strategies

Vitamin A deficiency is a significant concern in poultry production, as it can have detrimental effects on both the health and productivity of birds [3]. In order to address this issue, various intervention strategies can be used to prevent and treat vitamin A deficiency in poultry. This chapter will explore these strategies, including dietary modifications, supplementation, injection, and oral application, while also evaluating their efficacy, practicality, and potential challenges.

(1) Dietary Modifications:

Dietary modifications can be a debatable approach to preventing vitamin A deficiency in domestic fowl. One method involves adjusting poultry diets to include ingredients like some varieties of yellow corn and alfalfa meal, which are sources of β -carotene [6]. By incorporating these β -carotene-containing components into the diet, poultry can potentially fulfill a portion of their vitamin A requirement [8]. However, the practicality of using dehydrated alfalfa meal in broiler rations is often limited due to its low energy and protein content and high fiber concentration [145]. In laying hen diets, dehydrated alfalfa is sometimes used to enhance the color of egg yolks because of its high lutein and zeaxanthin content (non-provitamin A carotenoids) [146].

Compared to swine (7–40:1) and ruminants (6–8:1), poultry have a better but still variable conversion rate of β -carotene into vitamin A (2–6:1) [8]. Nevertheless, mammals generally have higher absorption of β -carotene, while birds and fish more effectively absorb oxygenated xanthophylls (e.g., lutein and zeaxanthin) [147]. For example, chickens are generally known to be poor absorbers and accumulators of carotenoids [8,148]. Furthermore, the β -carotene content as well as its bioaccessibility in corn and alfalfa can vary significantly, and their storage stability is relatively low [149–151].

Moreover, the effectiveness of dietary modifications in addressing vitamin A deficiency largely depends on the availability and affordability of ingredients such as corn, alfalfa meal, and corn gluten. While β -carotene has the potential to serve as a source of vitamin A in animals, its contribution is hindered by factors such as the variable β -carotene content

in raw materials, limited and unpredictable absorption and conversion rates into vitamin A, low stability, and the influence of animal age, genetics, physiological state, stress, and health-related factors [152]. As a result, accurately estimating the amount of vitamin A that can be obtained from β -carotene in poultry diets presents a challenge. For further exploration of this subject, readers are encouraged to consult the comprehensive research conducted by Green and Fascetti [8].

(2) Supplementation:

Another universally used and proven intervention strategy is the addition of synthetic sources of vitamin A to poultry diets, or occasionally to water [3]. This approach involves incorporating preformed sources of vitamin A, such as retinyl acetate, into the feed or water supply of the birds [18]. Its objective is to maintain a consistent level of vitamin A throughout a large-scale poultry production system. By supplementing with vitamin A, a direct and reliable source of this essential micronutrient is provided, bypassing any limitations or uncertainties associated with dietary modifications [97]. To ensure an even distribution within the feed, commercial vitamin A sources are typically added via vitamin- or vitamin-mineral premixes, considering the low inclusion levels per metric ton of feed [84]. In cases where quick fortification is necessary, such as during heat or other forms of stress, special water-dispersible formulations or vitamin mixes can be utilized to deliver retinol (usually as retinyl acetate or retinyl palmitate) via drinking water [153].

The supplementation of vitamin A has demonstrated significant effectiveness in preventing and treating hypovitaminosis A for the past seventy years [12]. It provides birds with a consistent and readily available source of this crucial micronutrient. Additionally, it enables precise control over the levels of vitamin A in the diet, ensuring optimal supplementation without the risk of excessive intake. However, it is important to pay attention to the stability of different commercial vitamin A sources during premix and feed storage and handling, as they may degrade under certain conditions [84].

(3) Injection:

Retinol, typically in the form of liquid retinyl propionate or sometimes retinyl palmitate, or vitamin mixes, can be administered to individual birds via injection [154], either intramuscularly (i.m.) in areas like the breast, legs, or tail, or subcutaneously (s.c.) in the inguinal crease or cervix [155,156]. Intravenous injection (i.v.) is possible in poultry depending on their size as well [155], but it typically requires a skilled professional to perform the procedure safely and effectively. However, these methods are not practical for large flocks and are occasionally used for larger fowl such as ostriches or valuable birds in breeding flocks [156]. One major advantage of this approach is its faster onset of action compared to supplementing via feed or water [152]. Additionally, when birds are unable to consume feed and water adequately due to illness, injection may be the only viable option.

(4) Oral application:

An alternative method, which is not commonly employed, involves the oral administration of retinol (typically as retinyl palmitate) or vitamin mixes to individual birds. Similar to the injection method, this approach ensures precise delivery of the desired dosage to the specific bird.

In conclusion, addressing hypovitaminosis A in poultry production requires effective intervention strategies. Dietary modifications, such as incorporating β -carotene-rich ingredients, have limitations due to variable conversion rates, low stability, and the influence of various factors. Supplementation using synthetic sources of vitamin A offers a reliable and consistent solution, allowing precise control over the micronutrient levels in the diet. Injection of retinol provides a faster onset of action and is suitable for individual birds, but not practical for large flocks. Alternatively, oral application can deliver the desired dosage accurately to specific birds, although it is not commonly utilized. Each strategy has its advantages and considerations, and their efficacy, practicality, and potential challenges should be carefully evaluated in addressing hypovitaminosis A in poultry.

6. Balancing Vitamin A Intake and Hypervitaminosis Risks

Hypervitaminosis A is a condition that can be induced in animals due to excessive consumption of retinol. Although vitamin A is vital for proper nutrition, maintaining a balanced intake is crucial to avoid adverse effects. The susceptibility of poultry to hypervitaminosis A can be influenced by various factors such as breed, age, the dosage and form of vitamin A, the method and frequency of administration, as well as factors affecting the absorption of fat-soluble vitamins [20]. Exposure of birds to high levels of vitamin A can have detrimental effects on their physiological functions [3].

Wolbach and Hegsted [157] administered 1,000,000 IU of vitamin A per kg of feed to chicks, which is approximately 667 times higher than the latest requirement estimate of NASEM [158] for growing chickens. The authors reported a decrease in feed intake, reduced growth rate, and impaired bone development in response to the high dosage for several weeks.

In a separate study, Veltmann and Jensen [159] administered either 330 IU or 660 IU of vitamin A per gram of body weight to broilers for 21 days, corresponding to 330,000 IU or 660,000 IU per kg of body mass. The researchers observed reduced body weight and feed intake in birds that were subjected to excessive vitamin A (330 IU or 660 IU/g) compared to the control group without vitamin A overdose. Additionally, the study found that vitamin A overdosing led to a decrease ($p < 0.05$) in serum P concentration, while serum Ca levels were not significantly affected ($p > 0.05$). Furthermore, the epiphyseal plates of broilers subjected to vitamin A overdose were wider compared to the control group.

A study conducted by Yuan et al. [130] explored the impacts of varying levels of vitamin A supplementation on broiler breeders. Their findings indicated that an increase from 5000 IU/kg to 20,000 IU/kg of feed resulted in an elevation of NDV (Newcastle disease virus) antibody titers following vaccination. However, a subsequent increase to 35,000 IU/kg led to a decrease in this parameter. In contrast, Coskun et al. [160] found no significant effects of dietary supplementation with vitamin A at levels up to 24,000 IU/kg on the immune response of Hisex Brown laying hens.

Sklan et al. [121,122] conducted studies on the impact of increasing doses of vitamin A, up to 44,000 IU/kg feed following a challenge, on growth and antibody production in broiler chickens and turkeys. Their research revealed that both antibody production and the T cell proliferative response peaked at around 20,000 IU of vitamin A supplementation. However, when the supplementation level was further increased to 44,000 IU/kg feed, a decline in these parameters was observed, suggesting a bell-shaped response. These findings were later corroborated by Guo et al. [128] in their study on broilers.

Recent research has suggested the development of resistance to hypervitaminosis A due to high intake of vitamin A. Bozhkov et al. [161] administered vitamin A daily at a dose of 300 IU/100 g of body weight to experimental animals. They observed that vitamin A accumulated in the liver, reaching a concentration of 250–300 mg/g. Subsequent administrations of vitamin A led to a decrease in its liver content, indicating a potential resistance mechanism.

It is crucial to note that the negative effects of hypervitaminosis A on physiological functions in domestic fowl are usually observed when animals are exposed to excessively high levels of the vitamin for extended periods. Such excessive intake is uncommon under normal feeding conditions [12]. Therefore, maintaining proper nutritional management and regularly monitoring vitamin A levels in animal diets are essential to prevent the development of hypervitaminosis A and maintain optimal physiological functions.

7. Fulfilling Vitamin A Requirements

Ensuring the well-being and productivity of domestic fowl requires meeting their vitamin A requirements, as they cannot synthesize this micronutrient metabolically *de novo*. Therefore, their dietary intake is crucial. With regard to this matter, it is important to differentiate between requirement estimates and allowances for vitamin A provided by scientific committees such as the National Academies of Sciences, Engineering, and

Medicine (NASEM, formerly National Research Council) [158] and the Gesellschaft für Ernährungsphysiologie (GfE) [162], as well as recommendations from breeding companies (Aviagen, Cobb-Vantress, Lohmann etc.) [163–166]. The following comparative aspects regarding vitamin nutrition should be considered:

- (1) The NASEM and GfE are reputable scientific organizations that offer evidence-based guidance and requirement estimates for various nutrients, including vitamins. They conduct thorough reviews of scientific literature and consult experts to develop their specifications. Breeding companies, on the other hand, specialize in specific aspects of poultry production, focusing on developing and breeding poultry strains that excel in certain traits such as growth rate, feed conversion, and disease resistance. Their recommendations are often based on their own research, experience, and published scientific data.
- (2) The primary goal of NASEM and GfE is to provide scientifically rigorous and unbiased recommendations to meet the basic nutritional requirements of poultry. They may consider factors such as physiological needs, growth rates, reproductive performance, and environmental conditions. Breeding companies aim to develop and provide specific genetic lines or breeds of poultry that perform well under practical conditions. Their recommendations for vitamins and other nutrients are tailored to support the genetic potential of their poultry strains and achieve maximal performance in terms of growth, feed conversion, and overall production efficiency.
- (3) NASEM and GfE base their requirement estimates and allowances on extensive scientific research and systematic reviews of the available literature. They evaluate a wide range of sources, including published studies, experimental data, and meta-analyses, to establish nutrient requirement estimates for different poultry species and production stages. Breeding companies conduct their own research, often using proprietary data and genetic selection strategies. Their recommendations are specific to the genetic lines they develop and are typically derived from their internal studies, field trials, and the performance data of their poultry strains.
- (4) The requirement estimates and allowances of NASEM and GfE are widely accessible to the public and scientific community. They publish reports and guidelines that provide detailed information on the nutrient requirements for poultry and other animal species, including vitamins and other essential nutrients. Recommendations from breeding companies may be proprietary and shared only with their customers or collaborators.

Poultry producers often consult both sources to ensure a balanced approach that considers both scientific knowledge and the specific genetic potential of their flocks [3]. However, an urgent update of NASEM requirement estimates is needed since their last update was in 1994, and there have been significant genetic improvements in the performance of broilers, turkeys, and laying hens strains. The Nutrient Requirements of Poultry: 10th Revised Edition committee is preparing an update to the 1994 Ninth Edition to address this need.

In modern times, it is increasingly recognized that the vitamin A requirements for commercial poultry production may exceed the previously established levels for healthy birds in controlled research settings by organizations like NASEM [167]. Stress, infection, and illness can significantly increase the vitamin A needs of birds, which must be considered in practical farming situations [87,168]. For instance, the most recent recommendations for broilers from Aviagen and Cobb, dating from 2022, aim to ensure sufficient vitamin levels in commercial farming operations while probably accounting for potential vitamin losses during storage and processing by including a certain safety margin (Table 2).

Table 2. Vitamin A guidelines for poultry: requirement estimates (NASEM), allowances (GfE), and recommendations (remaining sources).

Source	Vitamin A Requirement, IU/kg Feed			
	Broilers	Laying Hens	Broiler Breeders	Turkeys
NASEM [158]	1500	3750 ⁴	n/a	5000
GfE [162]	2500	4500	4500	n/a
Brazilian tables [169]	7053–13,538	9000–12,216	11,000	n/a
FEDNA [170]	7000–10,000	9000–10,000	9000–10,000	8000–13,000
Cobb [163]	10,000–13,000	n/a	10,000–13,000	n/a
Aviagen ¹ [164,165]	10,000–13,000	n/a	10,000–12,000	5000–12,000
Hubbard [171]	10,000–15,000	n/a	12,000–14,000	n/a
Hendrix (Hybrid turkeys) [172]	n/a	n/a	n/a	9500–12,500
EW Group (laying hens) ² [166,173,174]	n/a	8000–13,000	n/a	n/a
Hendrix (laying hens) ³ [175–178]	n/a	10,000–13,000	n/a	n/a

¹ Valid for Ross, Arbor Acres, and Indian River broilers breeds as well as Nicholas and B.U.T. medium and heavy turkey lines; ² valid for Lohmann, Hy-Line, and H&N Nick layer breeds; ³ valid for ISA, Dekalb, Shaver, Bovans, Babcock, and Hisex layer breeds; ⁴ at 100 g of feed per hen daily; n/a = not applicable.

The appropriate level of vitamin A in poultry diets depends on several factors, including the age of the birds. Young fowl have higher requirements for vitamin A due to their rapid growth and development [179]. For example, broiler chicks (ROSS, Cobb, Hubbard, Arbor Acres, Indian River breeds) in the starter phase typically require 10,000 to 13,000 IU per kilogram of feed, according to breeding companies' recommendations. This level supports their rapid growth and helps develop a robust skeletal structure and musculature. In the finisher phase, there is a slight decline in the vitamin A content of the feed, ranging from 9000–10,000 IU/kg. Similar trends can be observed for turkeys (Nicholas and B.U.T. medium and heavy lines, Hybrid turkeys (Hendrix)), with the recommended levels ranging from 11,000 to 12,500 IU/kg feed in the starter phase and declining to 5000–9500 IU/kg feed by week 17 post-hatching. For laying and breeding hens (Hubbard, Cobb, Hy-Line W-36, Babcock, Dekalb, Hisex, H&N Nick, ISA, Lohmann), vitamin A plays a significant role in their reproductive performance and egg production. The recommended level generally falls between 8000 and 14,000 IU per kilogram of feed for optimal performance, eggshell quality, fertility, and hatchability.

The breed or genetic strain of the birds is another factor that influences their vitamin A requirements [180]. Some breeds may have specific requirements due to variations in metabolic processes. Breed-specific recommendations or consultation with poultry nutrition experts is important to determine the appropriate vitamin A levels for specific breeds. However, it should be noted that these differences may have declined over the last decades due to breeding consolidation efforts [181].

The reproductive status of poultry also impacts their vitamin A requirements. Breeding birds have higher vitamin A needs to support fertility and reproductive functions [182]. Adequate levels of vitamin A are necessary for the development and maturation of reproductive organs, sperm production, and embryonic development [168]. Including higher levels of vitamin A within the recommended ranges in the diets of breeding birds helps ensure optimal reproductive performance [114,134].

The overall health and condition of the birds are additional factors to consider when determining vitamin A requirements. Stressful conditions, such as disease outbreaks or environmental challenges, can increase the vitamin A needs of poultry [87]. Vitamin A plays a critical role in supporting immune function and reducing susceptibility to infections during periods of stress [121,122,126]. Providing adequate levels of vitamin A during such situations is important for maintaining bird health and minimizing disease incidence.

Ultimately, careful formulation of poultry diets is crucial to meet the recommended levels of retinol and consider factors such as the source and stability of vitamin A to optimize its bioavailability for the birds. Visual observations, body weight measurements,

feed intake monitoring, and periodic blood or tissue analysis can be used to determine whether adjustments in vitamin A supplementation are necessary to meet the specific needs of the birds.

8. Conclusions

Based on the information provided in the scientific review paper, the following conclusions can be drawn:

- (1) Adequate nutrition, including sufficient vitamin A, is essential for maintaining optimal health, growth, and productivity in poultry flocks.
- (2) Vitamin A plays a crucial role in supporting various physiological functions in domestic fowl, including growth and development, immune function, antioxidant capacity, reproduction, and vision.
- (3) Vitamin A deficiency in poultry diets can result from inadequate feed formulation and the mishandling and improper storage of premixes and feed, factors affecting absorption and utilization.
- (4) Clinical manifestations of vitamin A deficiency in domestic fowl include reduced growth, high mortality, poor feed conversion, decreased immune response, ocular abnormalities, reproductive disorders, and neurological pathologies.
- (5) Effective intervention strategies for preventing and treating vitamin A deficiency in poultry encompass a range of approaches, including dietary modifications, supplementation, injection, and oral application. It is essential to evaluate the efficacy, practicality, and potential challenges associated with these strategies based on the specific circumstances.
- (6) Meeting the vitamin A requirements of poultry is crucial for their well-being and productivity. Differentiating between the requirement estimates and allowances provided by scientific committees and recommendations from breeding companies is important when addressing vitamin A nutrition.

Overall, understanding the significance of vitamin A in poultry health and productivity is essential for optimizing poultry management practices and preventing the negative consequences of vitamin A deficiency.

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