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## Vitamin E and Its Impact on Poultry Health and Production: An Update

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**ABSTRACT:** The main goals of recent poultry production sectors are to enhance the immune response of the birds, improve the performance, reduce mortalities and reduce stressors. These goals are achievable with dietary supplementation of vitamins. Vitamin E is one of the fat-soluble vitamin that has been used from last decades for different poultry production types. The inoculation level of vitamin E in the diet of poultry depends on several factors. Low or high level of vitamin E can induce severe adverse economic losses in poultry industry. Vitamin E has been regarded as a potent chain-breaking antioxidant as well as immuno-stimulator for both cell-mediated and humoral immunity. Vitamin E is added to the diet of broilers, layers and breeders especially those under heat stress conditions. In broilers, vitamin E can improve the health conditions, feed efficiency and immunity. However, in layers and breeders, it enhances the egg's quantity and quality as well as the fertility; respectively. Moreover, vitamin E proves its efficacy in modifying the carcass trait or meat quality of broilers. Therefore, this review article aimed to investigate the forms and inoculation levels vitamin E, the role of this vitamin in the biological process as well as its effect on different poultry production types, carcass quality and hematological parameters.

**Keywords:** Birds, vitamin E, antioxidant, immunity, carcass quality

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## INTRODUCTION

The best and efficient strategy to enhance the production performance, disease resistance and the immune response of poultry is the nutrients modification of poultry farming (Liu et al., 2014). Vitamins are organic substances with a complex nature that are found in a very small amount in feed. They are very important for the performance and body's physiological functions. Vitamins supplied to poultry ration to improve the viability, reduce stressors and enhance the growth performance parameters and antioxidant properties (Attia et al., 2017; Surai, 2020). Vitamins are divided into fat and water soluble types.

Vitamin E is considered as a fat-soluble vitamin that has been discovered in 1920s (Evans and Bishop, 1922). It is crucial for humans and animals and poultry species. The natural form of vitamin E (D $\alpha$ -tocopherol) is the most common and superior form in being retained in serum and tissues (Yang et al., 2009). The inoculation levels of vitamin E in poultry diets is recommended by NRC (NRC, 1994), however, the ideal levels are still controversial due to several factors (Kuttappan et al., 2012). Either deficiency or excess level of vitamin E is associated with severe adverse economic losses in poultry industry.

Vitamin E is necessary for the functions of immune, reproductive, nervous, respiratory, muscular and circulatory systems. Moreover, dietary supplementation of this vitamin is a common in poultry practice to improve both cell-mediated and humoral immunity (Konieczka et al., 2017; Pompeu et al., 2018) as well as counteract the deteriorative effects of oxidative stress (Surai et al., 2019; Pirgozliev et al., 2020). It has been recorded that vitamin E plays an important role for broilers production (Pal, 2017; Pitargue et al., 2019) as well as for layers and breeders production and reproduction (Asl et al., 2018; Nawab et al., 2018; Aamir et al., 2019). The carcass trait and meat quality are also positively affected by inoculation of vitamin E in broiler diet during rearing (Fellenberg and Speisky, 2006; Rey et al., 2015; Pitargue et al., 2019).

Accordingly, the objectives of the current review article were to investigate the forms and inoculation levels vitamin E, the role of this vitamin in the biological process as well as its effect on different poultry production types, carcass quality and hematological parameters.

## Forms of vitamin E

Chroman-6-ols collectively tocopherols (tocopherols and tocotrienols) are emerged as vitamin E molecules. However, 8 substances have been detected to have the activity of vitamin including 4 tocopherols ( $\alpha$ -,  $\beta$ -,  $\gamma$ - and  $\delta$ -tocopherols) and 4 tocotrienols ( $\alpha$ -,  $\beta$ -,  $\gamma$ - and  $\delta$ -tocotrienols) (Panda and Cherian, 2014); only tocopherol can meet the requirements of animals to vitamin E. They present in the fat sources of the diet as they absorbed in the intestine after ingestion and transformed into non-esterified form (Colombo et al., 1998). The form  $\alpha$ -tocopherol is considered as the most common studied form of vitamin E, while tocotrienols form is still under investigations. Vitamin E is commonly added to poultry diet in the natural form (D $\alpha$ -tocopherol) that being retained in the blood and tissue (Yang et al., 2009). The form of vitamin E determines its bioavailability. Vitamin E is measured in international units by defining one mg of all-rac- $\alpha$ -tocopherol acetate as 1 IU, as D- $\alpha$ -tocopherol has a bioactivity of 1.49 IU (Machlin, 1991).

## Recommended vitamin E levels in poultry diets

Several factors as bird's physiological and metabolic functions as well as the environmental stressors can control the actual requirements for vitamin E. The fatty acid contents, pelleting and storage conditions of the diets can mainly affect on the required amount of vitamin E. In addition, genetic variations among birds that lead to differences in vitamin E absorbability and degradation in the intestinal tract. Therefore, the ideal inclusion levels of vitamin E in poultry diets are still controversial (Kuttappan et al., 2012).

Poultry cannot synthesize vitamin E. Birds can obtain their requirements for this vitamin from fat sources and then stored by the body, so there is no need to be consumed daily (Colombo, 2010). Vitamin E is regarded as one of the most expensive vitamins for poultry. Under normal conditions, the standard recommended dose of vitamin E for poultry according to NRC ranges from 5-25 IU/kg of feed (NRC, 1994). To meet the poultry requirements, 10 IU/kg of the ration is also suitable. Studies of Rebolé et al. (2006); Singh et al. (2006); Hashizawa et al. (2013); Habibian et al. (2014) and Ismail et al. (2014) successfully used the basal level of vitamin E as adequate or to marginally exceed the minimum requirements of broilers. However, vitamin E requirements may increase especially in broilers to alleviate the negative effects of high temperature condition that affects on feed efficiency (Guo et al., 2003; Niu et al., 2009). Liu et al. (2014)

suggested that using of vitamin E as 25 times up to NRC requirement to enhance the antibody titer in turkeys. It has been suggested that poultry fed on 100 mg vitamin E/kg diet may prevent vitamin E deficiency (Aamir et al., 2019). The recommended dietary level of vitamin E to maintain bird's fertility differs according to the age, breed and the health of the bird as well as the composition of vitamin E. However, a concentration of 10 mg /kg of diet vitamin E is beneficial to maintain the fertility (Biswas et al., 2007; Hooda et al., 2007; Pekmezci, 2011; Khan et al., 2012b).

### **Deficiency of vitamin E in poultry**

Vitamin E deficiency produces severe adverse economic losses in the poultry industry. There are some interaction of vitamin E and other nutritional elements as polyunsaturated fatty acids (PUFAs), sulfur-containing amino acids and selenium. The deficiency of vitamin E with PUFAs is associated with nutritional encephalomalacia in chicks, while with selenium and sulfur-containing amino acids deficiencies induce exudative diathesis and enzootic muscle dystrophy; respectively (Beck, 2007; Guetchomet et al., 2012; Michalczuk et al., 2016). Reproductive disorders, hock disorders and retardation of growth are also forms of vitamin E deficiency in poultry (Niu et al., 2009). Besides, depletion of lymphocytes (Dietert et al., 1983) and growth depressant effect of thymus, bursa and spleen (Marsh et al., 1986) have been recorded as a result of vitamin E deficiency.

### **Hypervitaminosis with vitamin E in poultry**

High dietary levels of vitamin E resulting in reticulocytosis, decreased hematocrit value, lowered thyroid activity and increased vitamins D and K requirement in chicks (March et al., 1973). Also, supplementation with high levels of tocopherol alleviated hypervitaminosis with vitamin A in chicks (Mc Cuaig and Motzok, 1970; Sklan and Donoghue, 1982). Though, decreases the level of vitamin A in the blood and liver may adversely affect on the bone ash and plasma calcium level of birds (Aburto and Britton, 1998).

### **The role of vitamin E in the biological process**

#### **Antioxidant**

Vitamin E plays a major antioxidant role by prevention of lipid peroxidation of PUFAs in plasma membranes of cells and sub-capsular organs (Fusco et al., 2007; Khan et al., 2012a; Surai and Kochish, 2019), therefore protecting cells from free radicals

toxicity (free radicals scavenger) during normal metabolic status and inflammation (Colombo, 2010; Khan, 2011; Rizvi et al., 2014). Vitamin E can mediate free radicals signal transduction and finally modulates the genes expression that are regulated by free radical signaling (Packer and Suzuki, 1993). In addition, it has a negative effect on the production of reactive oxygen species (ROS) which activate unsaturated phospholipids and critical sulfhydryl group oxidation (Traber and Atkinson, 2007). Particularly, phospholipid membranes are more prone to oxidative stress, being positively correlated with the degree of PUFAs. Vitamin E has been classified as a reducing agent for ROS molecules. Nowadays, the bioactive contents of some phytobiotics plant react synergistically with vitamin E to enhance the antioxidant potential of vitamin E (Sonam and Guleria, 2017).

#### **Immunity**

It has been documented that vitamin E is essential for the ontogeny of the bird's immune response (Gore and Qureshi, 1997; Silva et al., 2011). Vitamin E significantly increased Sephadex-elicited inflammatory exudate cells as well as the macrophages percentage of chickens in a dose of 10 IU (Gore and Qureshi, 1997). Dietary vitamin E increases the T helper cells, and in turn improves responsiveness to immunologic stimuli (Erf et al., 1998). The dietary level of vitamin E may alter the innate cellular oxidative immunity (Perez-Carbajal et al., 2010). Besides, vitamin E is regarded as immuno-potentiator via delaying the production of ROS in lipid membranes (Pekmezci, 2011; Tufarelli and Laudadio, 2016; Aslet et al., 2018). It has been shown that vitamin E reduces the generation of MDA, decreases the total antioxidant capacity levels in the liver which is consistent with enhancing hepatic  $\alpha$ -tocopherol content; resulting in improvement of the antioxidant capacity (inhibit lipid peroxidation) of immunosuppressed broilers (Cheng et al., 2017).

Vitamin E acts on the immune organs either directly or indirectly through the affection of metabolic and endocrine parameters (Gershwin et al., 1985; Marsh et al., 1986; Leshchinsky and Klasing, 2001; Lohakare et al., 2005; Pompeu et al., 2018). As an antioxidant, vitamin E may reduce plasma concentrations of corticosterone (Puthongsiriporn et al., 2001). It can modulate cyclooxygenase and lipoxygenase pathways which reflects on the synthesis of leukotrienes and prostaglandins (Leshchinsky and Klasing, 2001, 2003).

It is not exactly known whether vitamin E directly increases production of antibodies by altering B cells or indirectly through T cells (Lee and Han, 2018).

As vitamin E acts as an antioxidant, it may prevent the oxidation of arachidonic acid involved in the biosynthesis pathway of prostaglandins which has immunosuppressive effects at elevated levels (Sheffy and Schultz, 1979). Modulation of arachidonic acid metabolism via cyclo-oxygenase and lipoxygenase pathways lead to synthesis of prostaglandins and leukotriens, respectively (Leshchinsky and Klasing, 2001). The inhibition of lipid peroxidation and protection of mitochondria and microsomes of the liver against oxidative stress may be another possible immunomodulatory role of vitamin E (Leshchinsky and Klasing, 2001). In addition, Gore and Qureshi (1997) suggested that higher levels of vitamin E may maintain the integrity of macrophage membrane that needed for phagocytosis. Broilers fed on excess vitamin E showed an increase in the phagocytosis process of peritoneal macrophages as a result of increasing the expression of Fc receptors of antibodies on macrophages membranes (Konjufca et al., 2004). Elevated numbers of macrophages displayed an increased ability to opsonize sheep red blood cells (SRRCS). Khan et al. (2014) suggested that vitamin E may affect macrophage cell viability and function by regulating levels of free radicals to maintain normal cell functions.

It has been appeared that vitamin E can boost both cell mediated and humoral immune response to various antigens. It enhances IFN- $\gamma$  production, induces proliferation of immune cells and modulates chemotaxis and bactericidal properties of polymorphonuclear cells (Boxer, 1986). Quantitatively and qualitatively augments of lymphocyte and monocyte mediated responses have been shown after dietary supplementation with vitamin E. For instance, feeding of broiler chickens with 80 IU/kg or 40 IU/kg of vitamin E following vaccination with infectious bursal disease virus (IBDV) vaccination induced significant increase in peripheral blood CD4<sup>+</sup> and CD8<sup>+</sup> T cells (Abdukalykova et al., 2008). Similarly, an increase in lymphocytes populations of the thymus as well as the number of plasma cells in spleen, cecal tonsils and ileum of broiler chickens have been observed following feeding on higher levels of vitamin E (Khan et al., 2008). Dalia et al. (2018) detected that inclusion of vitamin E (100 mg/kg) along with inorganic selenium (0.3 mg/kg) effectively improved the immune system through regulation of some cytokines expression and

immunoglobulin levels.

Vitamin E can benefit the immune response of poultry via anti-inflammatory effects. It has an essential role in balancing cytokine responses, which could be critical in cases of inflammation. It has been found that broiler chickens fed on 220 IU/kg of vitamin E showed significant decrease in the level of IL-6 mRNA in spleen (Kaiser et al., 2012) as vitamin E controls inflammatory responses when pro-inflammatory cytokine production is elevated. Broilers fed on vitamin E supplemented feed (100 mg/kg) and kept under heat stress showed significant decrease in liver expression IL-6 and heat shock protein 70 (Jang et al., 2014). Recent study of Pitargue et al. (2019) revealed that broiler chickens received vitamin E showed decrease in inflammatory (IFN- $\gamma$ , IL-1 $\beta$  and IL-6) and anti-inflammatory (IL-4, IL-10 and TGF- $\beta$ ) cytokines in the intestine. Inclusion of arginine in a vitamin E-supplemented diet in broiler chickens enhanced responses to phytohemagglutinin as assessed by the cutaneous basophil hypersensitivity test (Abdukalykova and Ruiz-Feria, 2006).

The findings of Lin and Chang (2006) suggested that moderate supplementation of vitamin E may enhance immune responses to selective antigens in breeders. Supplementation with vitamin E at level of (100IU/kg) to the diet of broilers breeders resulting in enhancing the immune response to bronchitis virus vaccine (Khan et al., 2014). In the same context, broiler chickens supplemented with 200IU/kg of vitamin E and 0.2 mg/kg of selenium and vaccinated with Newcastle disease virus (NDV) vaccine developed significant higher vaccine-specific antibodies when compared with control (Singh et al., 2006). Similarly, Ismail et al. (2014) demonstrated significant increase in the titers of antibodies against NDV and avian influenza disease virus in the plasma of vitamin E supplemented broiler chickens (300 mg/ kg diet). Significant elevation of antibody titers was observed in broilers after primary and secondary immunization with SRBCS and feeding on vitamin E (Niu et al., 2009; Habibian et al., 2014). Chickens infected with IBDV and fed on vitamin E (178 IU/kg) showed reduced mortalities and high body weight gain (McIlroy et al., 1993).

Broiler chickens received a diet containing 100 IU/kg of vitamin E and infested with *E. tenella* oocysts revealed significant resistance indicated by decreasing in mortalities and increasing in body weight gain (Colnago et al., 1984). Perez-Carbajal et al. (2010)



demonstrated that supplementation of chickens with vitamin E and arginine improved the phagocytic activity of heterophils and monocytes. Similar effects were also seen when chickens were challenged with *Salmonella enterica* serovar Typhimurium (Liu et al., 2014). Recently, Liu et al. (2019) assessed the effects of feeding laying hens on 30 IU/kg of vitamin E on antibody levels, pro-inflammatory cytokines and mortalities after challenge with *Salmonella enteritidis*. The results proved increasing IgA, IgM and IgY levels, while decreasing in IL-1 $\beta$ , IL-6 and mortalities at 2 weeks post-challenge.

Feeding of vitamin E to breeder hens can passively transferred antibody-mediated response against diseases in their progeny. When broiler breeder hens supplemented with vitamin E (150 IU/kg) or (450 IU/kg) in feed before inoculation with *Brucella abortus* antigens, their chicks that received more vitamin E showed higher antigen-specific antibody titers (Jackson et al., 1978). Supplementation of breeders on 0.03% total vitamin E in their diet for 3 weeks prior to immunization with Newcastle disease virus vaccine induce high antibody levels in their progeny at 1 and 7 days old as compared with controls (Haq et al., 1996). Inoculation of 10 mg of vitamin E in embryonated chicken eggs increased cellular and humoral immunity in newly hatched chickens with (Gore and Qureshi, 1997). In addition, these chicks showed higher phagocytic activity when inoculated with sheep red blood cells at 7 days of age as well as higher antibody titers to SRBC were also detected at 14 and 21 days of age. The same research also tested the effect of inoculating three doses of vitamin E into embryonated turkey eggs 3 days prior hatching. The results revealed that inoculation of 20 and 30 IU of vitamin E resulting in significant reduction of hatchability, while 10 IU induced slight higher hatchability. Furthermore, 7 days old turkey poults showed higher level of IgM antibodies against sheep red blood cells than controls at 7 and 14 days post-inoculation. The number of phagocytic macrophages at 7 weeks post hatch were also significantly higher in the group inoculated with 10 IU of vitamin E.

## The effect of vitamin E supplementation in poultry production

### Broilers

Improvement in feed efficiency has been recorded in broilers after feeding on vitamin E at levels of 60, 90 and 120 IU/kg of diet (Serman et al., 1992). Addi-

tion of both vitamins E and C at levels of 150 mg/kg and 200 mg/kg ration, respectively enhanced chicken's growth and immune response to vaccination (Rajmane and Ranade, 1994). Moreover, improvement in broilers feed efficiency has been observed after addition of 75 ppm of vitamin E/kg in diet (Aravind et al., 2001). Villar-Patino et al. (2002) recorded an enhancement of the live body weight of broilers supplemented by 75 mg of vitamin E/kg of diet.

Erf et al. (1998) reported that inoculation of vitamin E at levels beyond those needed to enhance the optimal growth is efficient for increasing the immuno-competence of growing broilers. It has been documented that supplementation of vitamin E induced significant increase in the relative weight of spleen which indirectly has a benefit for the broilers' immune system (BasmacioğluMalayoğlu et al., 2009). Konieczka et al. (2017) detected an increase in the relative weight of bursa Fabricius of chickens supplemented with dietary 300 IU/kg vitamin E as compared to those fed diets containing 50 IU/kg. Moreover, vitamin E at 100 and 200 mg/kg of the diet could improve the performances and have immune potentiating effect in broiler chickens (Desoky, 2018). Vitamin E has a significant role in enhancing the health conditions through the positive influence on both humoral and cell-mediated immune response of birds (Zhao et al., 2011; Lu et al., 2014; Rizvi et al., 2014). Moreover, it can induce protective immunity in broiler chickens through amelioration of the immuno-suppressive effect of lipopolysaccharide (Zhang et al., 2010), *Escherichia coli*, *Eimeriatenella*, T2 toxins (Jaradat et al., 2006), as well as heat stress (Niu et al., 2009).

It has been documented that heat stress increases the serum and liver concentrations of malondialdehyde (MDA), while vitamin E decreases the production of MDA in the liver by acting against lipid peroxidation and cell damage (McDowell, 2012) and results in the enhancement of the bird's performance (Sahin and Kucuk, 2001; Sahin et al., 2001). Several reports showed the positive effect of vitamin E supplementation alone or with other elements on broiler performance under heat stress condition. Both vitamin E and vitamin C at levels of 250 mg/kg of the diet induced the highest productive performance of Japanese quails reared under heat stress (Sahin and Kucuk 2001; Sahin et al., 2003). Habibian et al. (2014) confirmed that heat stressed broiler chickens supplemented with combined levels of vitamin E and selenium at 250 mg/kg and 0.5 mg/kg, respectively

showed an improvement of both health and immune response to sheep red blood cells (SRBCs). Broiler chickens fed on dietary vitamin E at 30-50mg/kg under heat stress pressure showed reduced lipid peroxidation that can be detected by reduced levels of MDA (Dalólio et al., 2015). Furthermore, dietary concentration of zinc at 30-60 mg/kg has synergistic positive action with vitamin E on the productive performance of broilers under heat stress climate (Kim et al., 1998; Salgueiro et al., 2000).

Other literatures revealed no effect of vitamin E on broiler performance. Sosnowka-Czajka et al. (2005) found that dietary supplementation of broilers with both 40 mg/kg of vitamin C and 70 mg/kg of vitamin E failed to increase the resistance of birds to high temperature stressor. This result may be due to low doses of the vitamins or presence of factor interfere with the vitamins bioavailability. Feeding of dihydroquercetin (antioxidant) or vitamin E improved different parameters of antioxidant status of broiler chickens, although it did not affect growth performance parameters and energy or nutrient availability (Pirgozliev et al., 2020).

### Layers

Laying hens supplemented by 6% semi-refined sunflower oil and 150 mg/kg vitamin E showed significant increase in egg production performance (Narimany-Rad et al., 2011). A concentration of 60 IU vitamin E /kg feed revealed an increase in egg production, yolk and albumin weights, and vitelline membrane strength of layer chickens (Parolini et al., 2015). In addition, vitamin E at levels 125-300 mg/kg has been found to minimize the egg production losses, eggshell density and feed efficiency (Cherian, 2015). Dietary supplementation of 125 to 300 mg vitamin E /kg feed improved the feed efficiency rate, egg production and egg shell thickness of layers (Karadas et al., 2017).

It has been observed that addition of vitamin E to the diets of layer hens appeared to be beneficial especially during the heat stress, probably, due to its concurrent function as fertility factor (Bollingier-Lee et al., 1999; Sahin et al., 2002a; Attia et al., 2016). Numerous studies have investigated the beneficial effects of vitamin E supplementation in laying hens under heat stressed conditions. For example, Kirunda and Scheideler (2001) found that vitamin E supplementation in the diet of heat stressed hens was able to alleviate egg quality deterioration. Ciftci et al. (2005) found that vitamin E can improve the egg quantity

and quality of laying chickens reared under heat stress conditions. It has been demonstrated that vitamin E at 250 mg/ fed of layer hens may decrease the harmful stress effects of high temperature (Chung et al., 2005). Besides, dietary concentration of zinc at 30-60 mg/kg has synergistic positive action with vitamin E on the health and egg production of laying hens (Onderci et al., 2003; Sahin and Kucuk, 2003; Kucuk et al., 2008). Sahin and Kucuk (2001) observed a greatest performance of Japanese quails after supplementation with a combined treatment with vitamin C (200 mg) and vitamin E (250 mg) under chronic heat stress. Also, supplementation with 150 mg vitamin C and/or 150 mg vitamin E to the diet improved the production performance in heat stressed layer chickens (Joachim Ajakaiye et al., 2011). Dietary vitamin E and vitamin C at levels of 65 IU/kg and 1, 000 ppm; respectively enhanced the *in vitro* lymphocyte proliferations of layer hens under bad environmental conditions (Jiang et al., 2013).

The role of vitamin E in improving the egg production under heat stress may be through the protection of liver from lipid peroxidation and damage of cell membrane that resulted in increasing in plasma egg yolk precursors as very low density lipoprotein and vitellogenin (Bollingier-Lee et al., 1999). Addition of vitamin E to diets containing high levels of PUFAs may prevent feed oxidation as well as may contribute to egg formation as these evidenced by increasing in the egg/bird/day and improving the feed intake and efficiency. Moreover, vitamin E protects the tissue from lipid peroxidation due to production of ROS and consequently affects the egg quality in layers (Lin et al., 2004; Khan et al., 2017).

### Breeders

There are several factors that have a great hazardous effect on the semen and sperm quality (Rengarajet al., 2015; Nawabet al., 2018). Antioxidant feed supplementation reduces these effects by lipid peroxidation (Richard et al., 2008). As a result of neutralization of free radicals and inhibition of lipids membranes oxidation, vitamin E is regarded as chain-breaking antioxidant (Raederstorff et al., 2015). Vitamin E reduces the production of ROS molecules in the cells at their initial phase with destruction of thousands of PUFAs molecules (Anwar et al., 2016). It has been found that ROS damages hydroxyl radical, superoxide anion radical, singlet oxygen and hydrogen peroxide that produced during aerobic cellular metabolism (Anwar et al., 2016). Nevertheless, these oxidative

radicals induced destruction of healthy cells if they are not eliminated. Thus, it is necessary to add vitamin E to poultry ration to increase antioxidant metabolites in sperms and semen and consequently helps in improving the quality and motility of sperms (Khan et al., 2017). Vitamin E reduces the defects in the DNA of sperm through decreasing free radicals production and consequently increase the semen volume, sperm motility and sperm capacity in fertilizing eggs (Anwar et al., 2016). Biswas et al. (2009) demonstrated that birds supplemented with high doses of vitamin E (100 mg/kg diet) showed good quality semen and spermatozoa in comparison with those received 10 mg/kg of the vitamin. At a level of 20 mg/kg diet of breeder chickens, vitamin E significantly enhanced the immune response of SRBCs in comparison with levels of 0, 80 and 160 mg/kg diet (Lin and Chang, 2006).

### Carcass trait

Lipid oxidation is very important process by which deterioration of meat products can occur as it is initiated at the membrane level in the intracellular phospholipid fractions (Buckley et al., 1995; Cortinas et al., 2005). Generally, supplementing birds with high levels of antioxidants in the diets enhances the oxidative stability, sensory quality, shelf life and consequently acceptability of meat (Buckley and Morrissey, 1992). It has been demonstrated that the peroxidation process begins just after slaughter, so the rate of meat spoilage is dependent on the concentration of vitamin E in the tissue (Morrissey et al., 1994). Vitamin E, in the form of  $\alpha$  tocopheryl, is regarded as the major antioxidant defense and the lipid-soluble antioxidant that delays and breaks the lipid peroxidation chain in cell membranes, prevents hydroperoxides formation (Halliwell, 1987) and improves the quality of poultry meat (Pompeu et al., 2018). The level of dietary  $\alpha$ -tocopheryl acetate in the poultry feed determines its level in the muscle and consequently the oxidative stability of meat (Carreras et al., 2004; Goñi et al., 2007). Previous studies of Gao et al. (2010) and Rey et al. (2015) have suggested that  $\alpha$  tocopherol retained in serum and tissues and improved the meat quality of broiler chickens. Increasing the levels of  $\alpha$  tocopherol levels in poultry diets significantly improved the feed conversion rate, average body weights, and net income/bird (Kennedy et al., 1992). In addition, the higher levels of  $\alpha$  tocopherol resulting in high tissue concentrations, improvement of the cells membranes structure as well as an increase the oxidative stability

of meat and meat products (Bartov and Frigg, 1992; Sheehy et al., 1993). It has been found that addition of  $\alpha$  tocopherol to turkey's ration can improve meat oxidative stability leading to improving the flavour and colour (Sheldon et al., 1997). Supplementing chickens with 20 mg vitamin E/kg diet doubled the storage time in freezer, however 40 mg vitamin E/kg diet extended storage time by one day in refrigerated broiler carcasses (Coetzee and Hoffman, 2001). The MDA production in the broilers muscles decreased by addition of vitamin E to the ration, which reflects on the lipid peroxidation during the storage of chicken meat (Yesilbag et al., 2011). Brandon et al. (1993) suggested that feeding on 200 mg  $\alpha$  tocopheryl acetate/kg of ration (i.e. 20 times higher than the NRC requirement) for at least 4-5 weeks, is essential to obtain the protective benefit of the vitamin in processed meat. However, Nobakht (2012) demonstrated that inclusion of broilers fat until 6% has no adverse effects on performance and carcass percent while, supplementing diet with 150 mg/kg of vitamin E is not recommended.

### Hematology

Vitamin E significantly protected erythrocytes against high levels of hydrogen peroxide (Calabrese et al., 1985). In addition, vitamin E prevents oxidation of unsaturated fatty acids as linoleic acid on the membranes of erythrocytes (Bast et al., 1991) so, the deficiency of this vitamin increases erythrocytes' hemolysis (Levander et al., 1977). The effect of different dietary levels of vitamin E (100, 200 and 300) ppm on erythrocyte osmotic fragility and some biochemical parameters were studied in broiler chickens for 7 weeks observation period and the results showed significant decrease in erythrocyte osmotic fragility (Arslan et al., 2001).

Excessive supplementation of vitamin E decreases the plasma cholesterol (Bell, 1971; Clegg et al., 1976) and triglyceride levels and consequently inhibits atherosclerosis in poultry (Donaldson, 1982; Smith et al., 1989). Francini et al. (1988) demonstrated that the addition of vitamin E at level of 325 ppm in the diet of broilers resulting in decrease in cholesterol and triglyceride levels till 49 days of age. Decreasing the level of cholesterol was also found in turkeys fed with vitamin E on the 42nd day (Francini et al., 1990). Ismail et al. (2014) found no effects on T4, total lipids, total cholesterol and high density lipoprotein cholesterol after inoculation of high levels vitamin E (200, 300 and 400 mg/kg diet) in the diets of broiler



chickens. However, plasma level of T3 increased significantly in response to high level of vitamin E (400 mg/kg diet). Serum concentration levels of T3 and T4 were higher in birds treated with dietary vitamin E (Sahin et al., 2001, 2002b).

It has been declared that dietary supplementation of birds with vitamin E in high levels increased alkaline phosphatase (ALP) levels. Arslan et al. (2001) demonstrated statistical significant decrease in ALP level by the 7th week after treatment of broilers with 100, 200 and 300 ppm of vitamin E. In the same line, no significant difference was also found in the plasma ALP levels of turkeys supplemented with 30, 90, 180, 360 ppm of vitamin E/kg of ration, however, an increase in plasma ALP levels were increased with increasing the bird's age (Francini et al., 1990).

In addition, the total protein, calcium (Ca), phosphorus (P), aspartate aminotransferase (AST) or alanin aminotransferase (ALT) was not affected by the treatment with vitamin E by the 5th and 7th weeks of age (Arslan et al., 2001). In the same context, no significant differences were found in Ca and P in broilers treatment with 100 and 200 mg/kg of vitamin E (Desoky, 2018). One hundred, day old broilers fed on 25 and 10, 000 IU of vitamin E/kg of the diet showed decrease in plasma P and Ca levels (Murphy et al., 1981). But Francini et al. (1988) found an increase in the levels of ALP, Ca and P in birds treated with excess vitamin E and proposed this result to the osteoblastic activity (Francini et al., 1988).

Although AST level has been increased with the increase in the dietary level of vitamin E in turkey

poult, but decreased in older birds (140 days old) (Francini et al., 1990). Desoky (2018) observed significant decline in AST and ALT activities in the group of broilers fed with vitamin E at 200 mg/kg.

Dietary supplementation with vitamin E (250 mg/kg) for Japanese quails under heat stress induce significant increase in lymphocytes (L) numbers and white blood cells counts, whereas, heterophils (H) numbers and H/L ratio was decreased (Abdel Maksoud, 1999; Ipek et al., 2007).

It has been shown that broilers fed on vitamin E at levels of 100 and 200 mg/kg had significant increase in hemoglobin, total proteins and albumin, significant decrease in the level of glucose and while no significant differences were found in globulin (Desoky, 2018)

## CONCLUSION

As can be seen, vitamins E has measurable effects as an efficient antioxidant and immuno-stimulant agent. Addition of vitamin E in poultry diet is essential for growth and health parameters as well as maintenance and enhancement of immune system function in broilers. In addition, supplementing layers and breeders with vitamin E has positive effects on the quantity and quality of eggs as well as fertility and hatchability. Improvement of carcass trait and blood parameters are also the other beneficial aspects of vitamin E.

## CONFLICT OF INTEREST

The author declares that there are no conflicts of interest.

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