Oxidative stress is defined as the imbalance between prooxidants and antioxidants (in favor of the pro-oxidants). Reactive oxygen species (ROS) are produced in the body continuously as a part of normal metabolism. Under normal physiological conditions a healthy balance exists between prooxidants and antioxidants. However, when the defense system gets disturbed due to fast growth, inadequate diet supply, disease, or other stressors, body is not able to synthesize the enzymes needed to destroy ROS or repair the damage. In the intensive system of poultry production birds are exposed to several stressors which may result in oxidative stress and lead to poor performance. Antioxidants in the animal body work together as the so called “antioxidant system” to prevent damaging effects of free radicals and toxic products of their metabolism. Vitamin E is considered as a very potent antioxidant in biological systems and found to be beneficial in counteracting the adverse effect of oxidative stress. The knowledge of antioxidant defense systems will serve as guiding principle for establishing most effective nutrition support to minimize oxidative stress. Such an approach will enhance bird health and welfare, product quality and will increase economic returns and the sustainability of poultry production. The current review describes the role of vitamin E as an antioxidant in alleviating oxidative stress in poultry.

Key words: oxidative stress, poultry, vitamin E

Introduction

The economic and nutritional demands of food production necessitate raising large number of birds in confinement during a short period of time. During intensive production processes, birds are exposed to several factors that can enhance oxidative stress. Some of these factors include diet, environmental and management-related. Diet factors includes: fat level and type of fatty acids, content of antioxidants (e.g. vitamin A, E and C, Zn, Cu, Mn and Se), storage, mycotoxin contamination, and hypervitaminosis (Papas, 1999; Iqbal et al., 2002; Panda et al., 2008a). Environmental factors include: atmospheric temperature, humidity, dust, ammonia, radiation, hyperoxia and bacterial or viral exposure. These conditions along with fast growth and high metabolic rate in modern birds can stimulate free radical generation and enhance oxidative stress. Once the free radical production exceeds the ability of the antioxidant system to neutralize them, lipid peroxidation develops and causes damage to unsaturated lipids in cell membranes, amino acids in proteins, and nucleopeptides in DNA resulting in disruption of membrane and cell integrity (Surai, 2000). All of these result in adverse effects on the productive and reproductive performance of the animal. Modern poultry production greatly depends on understanding of the relationship between diet, bird health and product quality (Fig. 1). The current review describes the role of vitamin E as an antioxidant in alleviating oxidative stress in poultry.

What is Oxidative Stress?

Oxidative stress is caused by the imbalance between prooxidants and antioxidants at either cellular or individual level (Voljc et al., 2011). During the normal respiration process, oxygen is progressively reduced to yield water. However, the incomplete reduction of oxygen during this process leads to formation of chemical entities that have powerful oxidizing properties and are known as reactive oxygen species (ROS). ROS are constantly produced in vivo during the course of physiological metabolism in living tissues. When ROS surpasses the ability of the antioxidant system of an organism to remove them, oxidative stress occurs (Surai, 2003). Oxidative stress constitutes an important factor of biological damage and is regarded as the cause of several pathological conditions that affect poultry growth and development (Avanzo et al., 2001; Iqbal et al., 2001). The concept of oxidative stress is becoming very important in nutritional research. In poultry, oxidative stress may occur due to several factors such as: 1) feed (high concentration of...
polyunsaturated fatty acids [PUFA], contamination with fungal toxins, prolonged storage, antioxidant deficiency) (Chee et al., 2005; Lin et al., 2006), 2) environmental (heat, high stocking density, transportation, vaccination) (Sahin et al., 2003; Panda et al., 2008a), and 3) pathological conditions (ascites, fatty liver haemorrhagic disease syndrome, arthritis, coccidiosis) (Bottje and Wideman, 1995; Papas, 1999; Iqbal et al., 2002). When an animal is challenged by oxidant compounds, there is an induction of protective antioxidant systems which enable the animal to detoxify foreign compounds.

The bird’s antioxidant system includes both enzymatic and non enzymatic defenses (Surai, 1999, 2000). The principal enzymatic systems include superoxide dismutase, glutathione peroxidase, glutathione reductase, and catalase (Fang et al., 2002). Non enzymatic antioxidants defense systems include molecules (e.g., glutathione, vitamin A and E, and carotenoids) and other elements such as selenium. When the ROS surpasses the ability of the antioxidant systems, lipid peroxidation is accelerated and accumulation of ROS is increased in serum and tissues which may damage biological membranes leading to cellular injury and dysfunctions and thus reduces productivity and suffers from diseases (Engberg et al., 1996; Halliwell and Gutteridge, 1999; Sahin and Kucuk, 2003; Tavarez et al., 2011). The tissue status of endogenous antioxidant enzymes are considered as markers for evaluating oxidative stress (Surai, 2003; Panda et al., 2008a). Vitamin E is the main chain-breaking antioxidant in biological systems (Surai, 2003) and prevents free radical damage to tissues, especially PUFA (Vlojc et al., 2011).

Oxidation and Free Radicals (FR) Formation

Oxygen is the primary oxidant in metabolic reactions designed to obtain energy from the oxidation of a variety of organic molecules. It is required for normal respiration in animals, however, at the same time oxygen is potentially a toxic substance and this has frequently been described as the oxygen paradox (Miller and Brzezinska, 1993). During the normal respiration processes of animals oxygen is progressively reduced to yield water. However, incomplete reduction of oxygen in the body lead to the formation of chemical entities known as free radicals (FR) which are atoms, molecules or any compounds containing one or more unpaired electrons. They are highly unstable and reactive, and are capable of damaging biologically relevant molecules such as DNA, proteins, lipids and carbohydrates. The FR is often known as reactive oxygen species (ROS) and is defined as oxygen-containing, reactive chemical species (Table 1). There are two types of ROS; those of free radicals, which contain one or more unpaired electron(s) in their outer molecular orbit such as superoxide, nitric oxide and hydroxyl radicals, and non-radical ROS, which do not have unpaired electron(s) but are chemically reactive and can be converted to radical ROS such as hydrogen peroxide, ozone, peroxynitrate and hydroxide (Trachootham et al., 2009). The most important effect of free radicals on the cellular metabolism is due to their participation in lipid peroxidation reactions. The
lipid peroxidation is a chain reaction and many cycles of peroxidation may result in substantial damage to the cells. Under normal physiological conditions, usually a healthy balance exists between prooxidants and antioxidants in the body. However, during stress, the balance gets disturbed and there is over production of free radicals causing oxidative stress leading to potential tissue damage. During stress, the hypothalamus-pituitary adrenal axis is activated and glucocorticoid hormone is released from the adrenal cortex. Glucocorticoid is essential for maintain life and normal growth. However, excessive glucocorticoid causes growth inhibition, immune system suppression and induction of oxidative stress (Tanguchi et al., 2001). Thus minimizing stress is important in poultry production. High dose vitamin E minimizes the elevation of plasma corticosterone due to stress (Taniguchi et al., 2001). When stress is more severe, the pro-oxidant systems outbalance the anti-oxidant systems, potentially producing oxidative damage to lipids, proteins, carbohydrates, and nucleic acids, ultimately leading to cell death in severe oxidative stress. Such uncontrolled oxidative reactions can lead to damage of cellular tissues and can lead to cell death, severe oxidative stress and metabolic diseases (Surai, 2000; Tavarez et al., 2011). Therefore, controlling auto-oxidation is very important to maintain feed and food quality and to enhance bird health and welfare.

Antioxidant Defense

All the living organisms have specific antioxidant defense mechanism to deal with ROS (Halliwell and Gutteridge, 1999). They are natural antioxidants which make them possible to survive in oxygen rich environment (Halliwell, 1991). This mechanism is generally known as antioxidant system and includes natural antioxidant vitamins described earlier. The antioxidant system of living cell consists of three major levels of defense (Surai, 1999) and the first step is responsible for prevention of radical formation by removing precursors of free radicals or by inactivating catalysts (Fig. 2). The second level of defense is to prevent and restrict chain formation and propagation and which consist of chain breaking antioxidant like vitamin E. The chain breaking antioxidant inhibits peroxidation by keeping the chain length of propagation reaction as small as possible (Surai, 2003). The third level of defense is excision and repair of damaged parts of molecules which include lipolytic (lipases), proteolytic (peptidases or proteases) and other enzymes (DNA repair enzymes, polymerase, ligases, phospholipases, nucleases). The antioxidant compounds are located in organelles, subcellular compartments or the extracellular space providing maximum cellular protection.

Antioxidants

An antioxidant is a molecule that inhibits the oxidation of other molecules. There are thousands of compounds in nature which possess antioxidant properties. Some of them are synthesized in the body (ascorbic acid, glutathione) while others are supplemented with feed (vitamin E, carotenoids, Se etc.). Antioxidant enzymes those are synthesized in the body require metal co-factors (Se for glutathione peroxidases (GSH-Px) and thioredoxin reductase (TR), Zn, Cu and Mn for superoxide dismutases (SOD), and Fe for catalase). The synthesis of the antioxidant enzymes required sufficient concentrations of the element in the diet. Deficiency of any of the above metals causes oxidative stress and allows damage to biological molecules and membranes. The antioxidant/prooxidant balance in the body are responsible for maintaining health, productive and reproductive performances of birds. Sub-optimal concentration of the nutrients in the diet may adversely affect the antioxidant-prooxidant balance resulting in oxidative stress. Poultry in intensive systems of farming frequently exposed to oxidative stress which may result in damage to body proteins, lipids and DNA (McCall and Frei, 1999) and can lead to poor performance and health (Lykkesfeldt and Svendsen, 2007). Therefore, optimizing the dietary intake of antioxidant nutrients is an important step.
in balancing oxidative damage and antioxidant defense in the animal body. That vitamin E is one such antioxidant which play a potent role in antioxidant defense in poultry is discussed below.

Vitamin E

Vitamin E, a fat soluble vitamin is a biological chain-breaking antioxidant that protects cells and tissues from lipoperoxidative damage induced by free radicals (Mcdowell, 1989). Vitamin E is essential for the integrity and optimal function of the reproductive, muscular, circulatory, nervous and immune systems (Leshchinsky and Klasing, 2001). Eight naturally occurring substances have been found to have vitamin E activity: four tocopherols (\(\alpha\), \(\beta\), \(\gamma\), \(\delta\)-tocopherols) and four tocotrienols (\(\alpha\), \(\beta\), \(\gamma\), \(\delta\)-tocotrienols). The \(\beta\), \(\gamma\) and \(\delta\)-tocopherols can act as an antioxidant but not retained well in the body tissues and biologically less active than \(\alpha\)-tocopherol (Surai, 2003). One IU of vitamin E is defined as 1 mg of all-rac-\(\alpha\)-tocopherol acetate. The only stereoisomer of \(\alpha\)-tocopherol found in the nature is RRR-\(\alpha\)-tocopherol, which is the most biologically effective form of vitamin E in animals (Halliwell and Gutteridge, 1999) having a bioactivity of 1.49 IU (Machlin, 1991).

**Vitamin E to Counteract Oxidative Stress in Poultry**

Vitamin E is receiving considerable attention in poultry nutrition due to its functional role as a dietary antioxidant to combat oxidative stress. Vitamin E is supplemented to the diet to maintain and enhance performance in layers, broilers, broiler breeders, and turkey (Sunder et al., 1997; Panda et al., 2009). The results obtained varied depending upon the level and duration of feeding diets supplemented with vitamin E, genetic stocks, age, assessment criteria, stress conditions and managemental aspects (Panda et al., 2009; Surai, 2000). Studies conducted in poultry on the role of vitamin E in alleviating oxidative stress is described below.

**Vitamin E in Chicken Embryo Development**

The avian embryo develops in a close system using the nutrients that are available within the egg before hatching. During chicken embryo development, considerable accumulation of highly PUFAs occurs within the embryonic tissue (Noble and Speake, 1997; Cherian et al., 1997) and the rate of oxidative metabolism increases dramatically over the hatching period (Freeman and Vince, 1974). At hatching, the chick is suddenly exposed to atmospheric oxygen and has a dramatic increase in metabolic rate. Thus, oxidative stress may be a problem during the last days of pre-hatch and first day of post-hatch life. It has been reported that vitamin E content in newly hatched chicks was dramatically depleted during the first nine days to about 5% of the day one (Surai and Ionov, 1994). This indicates that antioxidant may play a vital role during embryonic stage to initial few days of chick’s life. Vitamin E is transported from the yolk to the embryonic tissues during embryonic development (Gaál et al., 1995; Surai et al., 1996; Cherian and Sim, 2003). It prevents lipid peroxidation of PUFA within the cell, thus protecting the cell against the toxicity of free radicals (Khan, 2011). Supplementation of higher vitamin E in the maternal diet increased the concentration of vitamin E in the em-

<table>
<thead>
<tr>
<th>Vitamin E (IU/kg diet)</th>
<th>Lipid peroxidase (nmol/MDA/mg protein)</th>
<th>Glutathione peroxidase (U/mg)</th>
<th>RBC catalase (U/g/Hb)</th>
</tr>
</thead>
<tbody>
<tr>
<td>10</td>
<td>1.81(^a)</td>
<td>217(^d)</td>
<td>284</td>
</tr>
<tr>
<td>30</td>
<td>1.71(^b)</td>
<td>235(^e)</td>
<td>279</td>
</tr>
<tr>
<td>150</td>
<td>1.59(^c)</td>
<td>269(^h)</td>
<td>290</td>
</tr>
<tr>
<td>300</td>
<td>1.31(^d)</td>
<td>289(^a)</td>
<td>288</td>
</tr>
<tr>
<td>SEM</td>
<td>0.02</td>
<td>3.20</td>
<td>6.49</td>
</tr>
</tbody>
</table>

\(^a,b,c,d\) Means with different superscripts in a column differ significantly (\(P<0.05\)) (Panda et al., 2009)
bryonic tissue of the chick (Surai, 1999; Cherian and Sim, 2003). Surai (2000) studied the effect of various concentrations of Se (0.2 and 0.4 mg/kg) and vitamin E (40, 100 and 200 mg/kg) in maternal laying hen diet and concluded that higher concentration of Se and vitamin E increased the concentration of antioxidant enzymes and decreased susceptibility of lipid peroxidation in day old chick.

Vitamin E during Early Postnatal Development in Chicks

The time from hatching to the onset of receiving nutrition is also a critical period in the development of poultry. In the commercial poultry practice, chicks are accessed to feed and water after 48–72 hour of hatching (Bhanja et al., 2009). Thus, under practical condition the time between placements of chick right from hatch to growing site is a stressful condition (Panda et al., 2008b). In addition, birds are subjected to other practices such as transportation, chilling/over heating, vaccination, and high density of rearing. With so many challenges, the bird has to grow and produce optimum performance within the shorter span of 38–40 days. Nutrition has been identified as one source of early exposure that might affect early development and later phenotype performance (Cherian, 2011). Panda et al. (2009) studied the effect of higher supplementation of vitamin E (10, 30, 150 and 300 IU/kg diet) during early post natal period in broiler chicks and observed a linear decrease in the lipid peroxidase activity and linear increase in glutathione peroxidase (GSH-Px) activity by enhancing the levels of vitamin E from 10 to 300 IU per kg diet. Thus, it is suggested that higher level of vitamin E in the diet of young chicks could be beneficial in alleviating oxidative stress and thereby improving performance and immunity in broilers. He further concluded that vitamin E requirement for better immunity and antioxidant status is higher than that for growth performance.

Dietary Fatty Acids and Vitamin E

Vegetable oils are commonly added to poultry feed for increasing the energy density of the diet. Oils high in PUFAs are more susceptible to lipid peroxidation. The oxidation products decrease the nutrient content of the diet by reacting with proteins, lipids, and fat-soluble vitamins. Lipid oxidation can be reduced with the addition of antioxidant in the poultry feed (Cabel and Waldrop, 1989; Anjum et al., 2004). Many authors have reported that feeding high PUFA diet to broilers resulted in an increase plasma and tissue thiobarbituric acid reactive substances (TBARS), which is an indicator of oxidative stress (Sheehy et al., 1994; Cherian et al., 1996; Tavarez et al., 2011). Taking into account the fatty acid composition of feed, Mugli (1994) recommended vitamin E @ 0.13 IU/g of oleic acid, 0.9 IU/g of linoleic acid and 1.34 IU/g of linolenic acid. Leeson and Summer (2001) suggested 3 IU of vitamin E for each g of PUFA in 1 kg of poultry feed. These above recommendations were made by assessing oxidative stress in live birds.

Polyunsaturated Fatty Acids, Vitamin E and Oxidative Stability

Dietary PUFA are important for normal biological function and are involved in different physiological processes (Shahidi and Finely, 2001). Recently supplementation of poultry feeds with PUFA, especially, n-3 PUFA is a common practice to increase their concentrations in eggs and meat. Broilers and layers tend to deposit PUFAs in meat or eggs making the products susceptible to oxidation (Cherian et al., 1996; Crespo and Gracia, 2002; Cortinas et al., 2004). The susceptibility of meat to lipid peroxidation depends on several factors like proportion of PUFA in lipid bilayers, amount of ROS produced and the level of antioxidants that are supplied through nutritional or endogenous origin (Voljc et al., 2011). The most important phase of lipid oxidation in meat occurs during handling, processing, storage and cooking. During these processing conditions, Fe is released from high molecular compounds (hemoglobin, myoglobin, ferritin, and hemosederin) and become available to compounds with lower molecular weight (amino acids, nucleotides and phosphates) with which it forms chelates. These chelates are primarily responsible for catalysis of lipid oxidation in biological tissues (Morrissey et al., 1998). Vitamin E is essential for improving the oxidative stability of broiler meat (Grau et al., 2001; Eder et al., 2005). Supplementation of 200 IU of all-rac-α-tocopherol per kg diet reduced the lipid peroxidation in fresh meat samples (Voljc et al., 2011) confirming earlier reports that higher concentration of vitamin E is needed to prevent oxidative stress in birds as well as oxidative damage to meat. In another study Guo et al. (2001) reported a lower level of vitamin E for prevention of oxidative damage to meat probably due to lower level of PUFA in their diet. This finding further confirmed that the levels of vitamin E supplementation in the diet depend on the PUFAs content of the diet.

Vitamin E and Heat Stress

Heat stress is a major stressor for poultry due to their relatively high metabolic rate, efficient insulation due to feathers, lack of sweat glands and high deep body temperature (Sahin and Kucuk, 2003; Panda et al., 2008a). It is a major economic concern in poultry production due to its adverse effects on feed consumption, growth rate, egg production, hatchability, mortality, and health of birds (Bartlett and Smith, 2003; Panda et al., 2007; Gu et al., 2012). Heat stress is one major source of oxidative stress which stimulates the release of corticosterone and catecholamines and initiates lipid peroxidation in cell membranes (Freeman and Crapo, 1982). It is suggested that vitamin E (DL-α-tocopheryl acetate) can reduce the negative effects of corticosterone induced by stress (Watson and Petro, 1982). Vitamin E is mainly found in the hydrocarbon part of the membrane lipid bilayer towards the membrane interface and in close proximity to oxidase enzymes, which initiate the production of free radicals (McDowell, 1989; Sahin and Kucuk, 2001). Vitamin E therefore protects cells and tissues from oxidative
damage induced by free radicals (Sahin and Kucuk, 2001). Sahin et al. (2002) reported that Japanese quails diet supplemented with dietary vitamin E (250 mg/kg) had a significant reduction in malondialdehyde (MDA) in serum and tissue. Panda et al. (2007) reported that supplementation of vitamin E at 125 mg per kg diet significantly reduced the concentration of lipid peroxide and increased the concentration of glutathione peroxidase in serum of heat stressed laying hen (Table 4).

When living organisms are exposed to thermal stresses, the synthesis of most proteins is reduced, but a group of highly conserved proteins known as heat shock protein (HSP) are rapidly synthesized. One of the most important functions of HSP (HSP-70) is to protect organisms from the toxic effect of stressors (Zulkifli et al., 2003; Al-Aqil and Zulkifli, 2009). HSP70 protects the intestinal mucosa from heat-stress injury by improving antioxidant capacity of broilers and inhibiting lipid peroxidation (Gu et al., 2012). Sahin et al. (2009) reported that vitamin E supplementation (500 mg/kg diet) may alleviate heat-stress related effects on egg production and heat shock protein (Hsp70) expression in Japanese quails.

**Vitamin E and Ascites**

Ascites is a metabolic disorder of fast growing broilers and is characterized by accumulation of lymph fluid in the peritoneal spaces. It is caused by an imbalance between the oxygen requirement of the tissues and the oxygen supply to body tissues (Julian, 1993; Wideman et al., 2013). Results of many studies suggested that peroxidative damage may play an important role in the pathogenesis of ascites, and free radicals may lead to endothelial damage in both heart, and lung cells (Enkvetchakul et al., 1993; Bottje et al., 1997; Arab et al., 2006). During ascites, activated white blood cells generate a variety of ROS into the surroundings tissue, which in turn alter tissue antioxidant status. A lower level of vitamin E was found in liver and lungs tissues of broilers suffering from ascites compared to the control diets (Enkvetchakul et al., 1993). Vitamin E has a beneficial effect in lowering oxidative stress in broilers with ascites (Bottje et al., 1997). It is also suggested that high vitamin E supply can alleviate oxidative stress in pulmonary hypertension syndrome (PHS) (Iqbal et al., 2002) and can be beneficial in reducing ascites mortality in broilers (Bottje et al., 1997).

**Vitamin E and Mycotoxin Contamination**

Mycotoxicosis is the most devastating and wide spread non infectious disease affecting all species of livestock and poultry. Mycotoxins are the secondary metabolites of fungi like Aspergillus, Penicillium and Fusarium. Feeding diets infested with fungi and contaminated with mycotoxins affects not only feed intake, body weight gain and feed efficiency but also affects the health and productivity (Jand et al., 2005). Mycotoxins in the feed significantly reduce the antioxidant assimilation from the feed and increase their requirement to prevent damaging effects of free radicals and toxic products of their metabolism produced as a result of mycotoxin exposure (Surai, 2003). Several studies have reported that mycotoxins can generate free radicals causing lipid peroxidation and damage to other biological molecules including lipids, proteins and DNA which results in oxidative stress in poultry (Hoehler et al., 1997; Dvorska et al., 2001). Vitamin E is important for its antioxidant functions in the body. To maintain its tissue level adequate supplementation would be necessary during mycotoxicosis. Higher supplementation of antioxidants protect against toxic action of mycotoxins by inhibiting or preventing lipid peroxidation (Hoehler and Maraquadt, 1996, Verma and Nair, 2001). Thus combination of antioxidant like vitamin E with toxin binders could be one of the im-

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### Table 4. Effect of supplemental vitamin E on antioxidant status of heat stressed Layers

<table>
<thead>
<tr>
<th>Antioxidant enzymes</th>
<th>Supplemental vitamin E in diets (mg/kg)</th>
<th>SEM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lipid peroxidase (nmolMDA/mg protein)</td>
<td>1.92&lt;sup&gt;a&lt;/sup&gt; 1.74&lt;sup&gt;b&lt;/sup&gt; 1.66&lt;sup&gt;c&lt;/sup&gt; 1.68&lt;sup&gt;a&lt;/sup&gt; 1.66&lt;sup&gt;c&lt;/sup&gt; 0.02</td>
<td></td>
</tr>
<tr>
<td>Catalase (K/g hemoglobin)</td>
<td>222.7&lt;sup&gt;c&lt;/sup&gt; 238.5&lt;sup&gt;b&lt;/sup&gt; 252.8&lt;sup&gt;a&lt;/sup&gt; 259.8&lt;sup&gt;a&lt;/sup&gt; 252.6&lt;sup&gt;a&lt;/sup&gt; 3.03</td>
<td></td>
</tr>
<tr>
<td>Glutathione peroxidase (unit/ml)</td>
<td>219.5&lt;sup&gt;a&lt;/sup&gt; 222.9&lt;sup&gt;b&lt;/sup&gt; 246.9&lt;sup&gt;c&lt;/sup&gt; 236.93&lt;sup&gt;b&lt;/sup&gt; 240.9&lt;sup&gt;ab&lt;/sup&gt; 2.03</td>
<td></td>
</tr>
</tbody>
</table>

<sup>a,b,c</sup> Means with different superscripts in a row differ significantly (P<0.05) (Panda et al., 2007)

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### Table 5. Effect of α-tocopherol supplementation on liver vitamin E concentration during mycotoxicosis in chicks

<table>
<thead>
<tr>
<th>α-tocopherol (IU/kg)</th>
<th>Control</th>
<th>T-2 toxin (4 ppm)</th>
<th>Ochratoxin (2.5 ppm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>14</td>
<td>2.6</td>
<td>2.1</td>
<td>1.6</td>
</tr>
<tr>
<td>100</td>
<td>40</td>
<td>24</td>
<td>16</td>
</tr>
<tr>
<td>1000</td>
<td>234</td>
<td>167</td>
<td>110</td>
</tr>
</tbody>
</table>

Hoehler and Maraquadt (1996)
portant strategies to counteract mycotoxicosis in poultry.

**Vitamin E and Semen Quality**

In cockerels, lipids form an integral part of the sperm membrane and are involved in a series of biochemical and functional changes (maturation, capacitation and acrosome reaction), required in the process of fertilization (Koyanagi et al., 1988). The high proportion of PUFAs in avian spermatozoa is associated with increased susceptibility to ROS and lipid peroxidation (Khan, 2011). These ROS are involved in many physiological functions of spermatozoa, however, excessive production of the same may result in oxidative stress (Agarwal et al., 2003). The production of ROS is increased during stressful conditions. Oxidative stress reduces gamete numbers, decreases sperm motility and increases percentage of dead cells (Alkan et al., 1997; Sikka, 2001). Vitamin E supplementation has been resulted in improvement in semen quality which was attributed to prevention of oxidation (Niki, 1993) and inhibition of lipid peroxidation of sperm (Fujihara and Howarth, 1978). It has been reported that 88% of chicken semen vitamin E is located in the spermatozoa. Depending upon dietary supplementation the concentration of vitamin E in chicken semen varies from 0.46 μg/ml (no vitamin E supplementation) up to 1.04 μg/ml (vitamin E supplementation - 200 mg/kg) as reported by Surai et al. (1997). Vitamin E effectively scavenges free radicals producing stable ROOH groups. An increased level of vitamin E (160 mg/kg) in the chicken spermatozoa has been associated with a reduction in susceptibility to Fe³⁺-induced lipid peroxidation (Lin et al., 2005).

**Conclusions**

The economic and nutritional demands of food from poultry necessitate the raising of large number of birds in relatively small areas with high rates of productivity. During this intensive system of production birds are exposed to considerable stress which leads to overproduction of free radicals. The free radical generation and lipid peroxidation are responsible for the development of disease conditions leading to poor performance and product quality. Among the natural antioxidants, vitamin E is considered to be important because of its role in maintaining avian health and productive and reproductive performance of poultry. Vitamin E is important in protection against the development of oxidative stress. It prevents oxidative injury of PUFAs, thiol rich protein constituents of cellular membranes and the cytoskeleton and nucleic acid and thereby maintains the structural and functional integrity of sub cellular organelles.

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