Review of Literature
REVIEW OF LITERATURE

The review of literature pertaining to the present study entitled “Effect of supplementation of vitamin E and selenium and their combinations on the growth, immune response, production performance and biochemical profile of layer chickens” is discussed under the following headings:

2.1 DISEASES PREVALENT IN LAYER CHICKENS
2.2 TYPES OF STRESSES ENCOUNTERED IN POULTRY BIRDS
2.3 FREE RADICALS AND LIPID PEROXIDATION
2.4 NATURAL ANTIOXIDANT DEFENCE MECHANISMS
2.5 SIGNIFICANCE OF DIETARY ANTIOXIDANTS IN POULTRY
2.6 VITAMIN E AND ITS BIOLOGICAL SIGNIFICANCE
2.7 SELENIUM AND ITS BIOLOGICAL SIGNIFICANCE
2.8 VITAMIN E AND SELENIUM SUPPLEMENTATION IN POULTRY

2.1 DISEASES PREVALENT IN LAYER CHICKENS

Health and disease control remain high priority areas in the management of commercial poultry (Yegani, 2005). Infectious disease outbreak and increased mortality could adversely affect the egg production. Diseases of the chicken are mostly infectious in nature and therefore wide variability in losses due to such diseases is expected in egg type layers (Usman and Diarra, 2008). Some important diseases in egg type layers cause mortality and or reduced productivity.

2.1.1 Newcastle Disease (ND)

ND is a highly contagious and destructive disease present in endemic form with frequent outbreaks in commercial poultry (Ananth et al., 2008). The
highly contagious and lethal form of ND is known as ViscerotropicVelogenic Newcastle Disease (VVND), Exotic Newcastle Disease, or Asiatic Newcastle Disease. It is reported to be caused by a single stranded, enveloped, non-segmented RNA virus i.e. Avian Paramyxovirus serotype-1 (APMV-1) belonging to the genus *Avulavirus* of family Paramyxoviridae (Mayo, 2002).

The disease is characterized by a sudden onset of clinical signs which include hoarse chirps, watery discharge from nostrils, labored breathing, facial swelling, paralysis, trembling and twisting of the neck. Mortality ranges from 10 to 80 percent depending on the pathogenicity. In adult laying birds, symptoms include decreased feed and water consumption and a dramatic drop in egg production (Shankar, 2008).

### 2.1.2 Infectious Bursal Disease (IBD)

IBD is one of the most important naturally occurring viral diseases of chickens worldwide. The causative agent, Infectious Bursal Disease Virus (IBDV), belongs to the family Birnaviridae (Khatri and Sharma, 2007). It is considered as AIDS of the chicken, because it adversely affects the chicken’s immune system. The disease affects primarily bursa of fabricius and other lymphoid organs to a lesser degree (Arshad *et al.*, 2005). Infection was found to cause direct mortality upto 90% of a flock in case of very virulent strain outcome (Lukert and Saif, 2003).

### 2.1.3 Infectious coryza

Infectious coryza is an important bacterial disease of chickens characterized by respiratory complications, swollen head syndrome, nasal discharge and severe drop in egg production (Usman and Diarra, 2008). The etiological agent responsible for the diseases is *Haemophilus paragallinarum* (Gayathri *et al.*, 2009). Conditions of poor hygiene, chilly environment and adverse climate exposure are the predisposing factors for the onset of this disease. Mortality from coryza was usually found to be low, but infections
were found to decrease egg production and increase the incidence and/or severity of other diseases.

2.1.4 Infectious Bronchitis (IB)

IB is a common endemic respiratory viral disease of chickens, second only in importance to Newcastle Disease and Avian Influenza. It was reported to be caused by an avian coronavirus and primarily resulting in respiratory diseases and loss of egg production, with some strains reportedly causing nephritis, proventriculitis and pectoral myopathy (Ganapathy, 2009). Infectious Bronchitis Virus (IBV) is a significantly and economically important pathogen of avian species (Origlia et al., 2009). IBV was observed to have a great economic effect on the layer industry, as it affected egg production and quality (Chousalkar et al., 2007).

2.1.5 Marek’s disease

Marek’s disease is a lymphoproliferative disease of chickens that is caused by a highly cell-associated oncogenic α-herpesvirus, Marek’s disease virus (Heidari et al., 2008). Lymphoproliferative neoplasms occur in various organs and tissues, including the viscera, peripheral nerves, skin, gonads, and musculatures. Marek’s disease virus is found to be restrictively produced in the feather follicle epithelial cells gaining access to the external environment via infected cells or as infectious enveloped cell-free virus particles (Heidari et al., 2007).

2.1.6 Avian Influenza (Al)

Al is an important poultry disease that had emerged with higher mortality in the recent decades. It is an infectious disease of birds caused by type A strains of the influenza virus (Broor, 2005). Al is categorized as mild or highly pathogenic. The mild form is found to produces listlessness, loss of appetite, respiratory distress, diarrhoea, transient drops in egg production and low mortality. The highly pathogenic form was reported to produce facial swelling, blue comb and wattles and dehydration with respiratory distress (Shankar, 2008). The 'avian
influenza' or 'bird flu' threat during early 2006 in India had resulted in losses to the tune of over Rupees 2200 crores to the Indian poultry industry (Usha et al., 2009).

2.1.7 Mycoplasmosis

Mycoplasmosis caused by Mycoplasma gallisepticum, M. synoviae, M. meleagridis and M. iowae is a widespread disease affecting poultry production. Mycoplasmas are important avian pathogens responsible for chronic respiratory diseases and infectious synovitis or bursitis of chickens and turkeys, which result in large economic loss for the world poultry industry (Buim et al., 2009). Some mycoplasm species were reported to adversely affect egg production, egg quality and hatching, in addition to increase in mortality and reduction in feed efficiency and weight gain (Silva et al., 2008).

2.1.8 Salmonellosis

Salmonellosis is one of the most common food borne bacterial diseases in the world. Salmonella, one of the bacterial species, are abundantly found in most of the areas of the poultry premises where chances of contamination are greater. These organisms usually contaminate feed and drinking water resulting in poor economic gains and higher mortality. Chickens can be infected with many different serovars of Salmonella. Some serovars such as S.Pullorum and S.Gallinarum are host specific for chickens (Foley et al., 2008).

2.1.9 Coccidiosis

Coccidiosis a protozoan disease, caused by various species of the genus Eimeria is one of the major problems of the chicken industry, characterized by blood tinged feces, ruffled feathers, loss of appetite, poor growth and reduced egg production (Usman and Diarra, 2008). Seven species of Eimeria are generally accepted to be the causative agents of avian coccidiosis, namely E. acervulina, E. brunetti, E. maxima, E. mitis, E. necatrix,
E. praecox and E. tenella. E. tenella was found to be the most prevalent and pathogenic species followed by E. maxima and E. acervulina throughout the world (Ayaz et al., 2003).

2.1.10 Yolk sac infection

It is one of the most common bacterial infections of chicken observed during the first few weeks of a chicken's life. Drowsiness, minimal mobility, vent pasting and lack of interest of feeding in the chicken characterize yolk sac infection. There may be several predisposing factors such as poor hygiene and stressful conditions leading to this anomaly because, it is a general bacterial infection. Yolk sac infection occurs mainly due to bacterial contamination of the eggshell at the farm shortly after the egg is laid, while the cuticle is still moistened (Saif et al., 2003).

2.1.11 Escherichia coli (E. coli)

E. coli are one of the most common and important avian bacterial pathogens and infections caused by E. coli are responsible for significant economic losses to the poultry industry (Salehi et al., 2008). E. coli continue to result in significant morbidity and mortality in domestic poultry causing a variety of diseases either alone or as an adjunct to other pathogens (Anand et al., 2006). Colisepticemia, coligranuloma, air sac disease, coliform salpingitis, coliform cellulites, swollen-head syndrome, coliform peritonitis, coliform osteomyelitis / synovitis and coliform omphalitis are the different forms of E. coli infections in poultry (Barnes et al., 2003).

2.1.12 Egg prolapse and cannibalism

When a hen lays an egg, the lower part of the oviduct is momentarily everted through the cloaca. Prolapse occurs when the hen cannot retract the oviduct and a part of it remains outside the body. This condition is most common in overweight, older hens and in early laying pullets with low body weight. Egg prolapse has become one of the major issues in egg type layers during the past few years (Usman and Diarra, 2008).
Cannibalism usually occurs when the birds are stressed by a poor management practice. Once becoming stressed, one bird begins picking the feathers, toes, heads, and vents of other birds. Mortality caused by cannibalism continues to be a major problem in the layer industry (Hartini et al., 2002).

2.1.13 Aflatoxicosis

Poultry has commonly been considered highly susceptible to aflatoxins (Diaz et al., 2008). Contamination of aflatoxin in feed causes aflatoxicosis in poultry and it is characterized by weakness, anorexia with lower growth rate, poor feed utilization, decreased egg production, increased susceptibility to environmental and microbial stressors and increased mortality. Aflatoxins are mycotoxins produced by the toxigenic fungi mainly by Aspergillus flavus and Aspergillus parasiticus (Hussain et al., 2008).

2.2 TYPES OF STRESSES ENCOUNTERED IN POULTRY BIRDS

Stress is probably one of the most common causes of immunosuppression in poultry. Under commercial conditions, the chicken is exposed to a variety of stressors that may adversely influence the immune system. Short-term stressors prevent the available nutrients towards resistance to challenges from bacteria, parasites and toxins and leave the bird susceptible to viral challenge. Chronic stressors leave the birds more susceptible to challenge by infectious agents (Yegani, 2005).

A list of possible stresses in poultry production includes the time between an egg being laid and its cooling down for storage, egg storage before incubation, temperature, humidity and carbon dioxide concentration fluctuations during incubation, day 19 of embryonic development, hatching time, delay in collecting chicks from incubator, transportation from hatchery to farm, sub-optimal temperatures in the poultry house, high levels of ammonia and carbon dioxide in poultry house as a result of inadequate ventilation, disease challenge, vaccination, induced molting with feed withdrawal,
mycotoxins in the feed, heavy metals and other toxicants in the feed, oxidized fat in the diet, extensive preventive medication and vitamin A excess in the diet (Surai, 2006).

The list of potential stresses can vary from one poultry farm to another, but overproduction of free radicals and the critical need for antioxidant protection are common factors.

2.3 FREE RADICALS AND LIPID PEROXIDATION

The animal body is under constant attack from free radicals, formed as a natural consequence of the body's normal metabolic activity and as a part of the immune system's strategy for destroying invading microorganisms (Surai et al., 2006). Free radicals are not only destructive to the living cells but also reduce the quality of animal products through oxidation (Al-Mamun et al., 2007).

Free radicals are chemical species containing one or more unpaired electrons and therefore are extremely reactive (Fernandez et al., 2009). This unstable configuration creates energy which is released through reactions with adjacent molecules, such as proteins, lipids, carbohydrates and nucleic acids.

Free radicals can initiate autocatalytic reactions so that molecules with which they react are themselves converted into free radicals to propagate the chain of damage (Rahman, 2007). Most biologically-relevant free radicals are derived from oxygen and nitrogen, the so-called reactive oxygen species (ROS) and reactive nitrogen species (RNS) (Surai, 2007).

2.3.1 Reactive oxygen species (ROS)

Reactive oxygen species (ROS) are small, highly reactive, oxygen-containing molecules that are naturally generated in small amounts during the body's metabolic reactions and can react with and damage complex cellular molecules such as fats, proteins, or DNA (Wu and Cederbaum, 2003).
ROS are produced both endogenously and exogenously. The endogenous sources of ROS include mitochondria, cytochrome P450 metabolism, peroxisomes and inflammatory cell activation (Inoue et al., 2003). ROS can also be produced by a host of exogenous sources such as xenobiotics, chlorinated compounds, environmental agents, metals (redox and nonredox), ions and radiation (Valko et al., 2006). ROS include free radicals such as superoxide (O₂⁻⁻) and hydroxyl (‘OH) and nonradical species such as hydrogen peroxide (Dworakowski et al., 2006).

It has been established that ROS can be both harmful and beneficial in biological systems depending on the environment (Lopaczynski and Zeisel, 2001; Glade, 2003). Beneficial effects of ROS include defense against infectious agents and function in a number of cellular signaling systems. In contrast, at high concentrations, ROS can mediate damage to cell structures, including lipids and membranes, proteins and nucleic acids; this damage is often referred as “oxidative stress” (Poli et al., 2004).

2.3.1.1 Hydrogen peroxide (H₂O₂)

Hydrogen peroxide is produced continuously in all cells. It diffuses within and in between cells. Hydrogen peroxide is unique in that it can be converted to the highly damaging hydroxyl radical or be catalyzed and excreted harmlessly as water. Hydrogen peroxide is not very reactive. It does not readily oxidize most proteins, lipids or DNA. Nevertheless, it can be cytotoxic at micromolar concentrations (Lenzen, 2008).

2.3.1.2 Superoxide radical (O₂⁻⁻)

O₂⁻⁻ is one of the strongest reactive oxygen species among the free radicals that are generated first after oxygen is taken into living cells (Al-Mamun et al., 2007). O₂⁻⁻ serves as the raw material for many reactive oxygen species members like H₂O₂ and ‘OH radicals following its catalysis by superoxide dismutase enzymes and also by autocatalysis (autodismutation).
reactions. Most of the $O_2^{\cdot-}$ radicals are formed in the mitochondrial and microsomal electron transport chain (Singh et al., 2004).

2.3.1.3 Hydroxyl radical (‘OH)

Hydroxyl radicals are highly reactive and consequently short-lived. It is formed in vivo upon high energy irradiation (e.g. x-rays) by hemolytic cleavage of water or from hydrogen peroxide in a metal catalyzed process. ‘OH is the most reactive oxygen radical known, reacting instantaneously with molecules in its immediate vicinity, which explains its great destructive power (Lenzen, 2008).

2.3.2 Reactive nitrogen species (RNS)

RNS are various nitric oxide-derived compounds, including nitroxy anion, nitrosonium cation, higher oxides of nitrogen, S-nitrosothiols and dinitrosyl iron complexes (Martinez, 2009).

$NO^-$ is a free radical which plays an important role in host defense and homeostasis when generated at a low level and for a brief period of time, but becomes genotoxic and mutagenic when generated at higher concentrations for prolonged periods (Hershkovich et al., 2007).

$NO^-$ reacts slowly with most biological molecules, but is highly reactive with other free radicals. $NO^-$ reacts faster with $O_2^{\cdot-}$ than with heme compounds or even faster than the reaction of $O_2^{\cdot-}$ with SOD. The reaction of $NO^-$ with $O_2^{\cdot-}$ is important because the biological actions of $NO^-$ and $O_2^{\cdot-}$ are prevented and because peroxynitrite (ONOO$^-$) is formed, which is in itself significantly toxic.

2.3.3 Oxidative stress

Oxidative stress that occurs in the cells, as a consequence of an inequity between the pro-oxidant/antioxidant systems, causes injury to biomolecules such as nucleic acids, proteins, structural carbohydrates and lipids. As a result of stress, feed consumption, growth rate, feed efficiency, egg
quality, fertility and chick quality were reported to decline (El-Lethey et al., 2000). Under stressful conditions, the requirement of antioxidants such as vitamin E (α-tocopherol) was suggested to increase to protect tissues from lipid peroxidation (Eid et al., 2006).

2.3.4 Lipid peroxidation

Lipid peroxidation occurs as a consequence of increased oxidative stress primarily due to disruption of pro-oxidant/antioxidant balance (Kurien and Scofield, 2006).

Lipid peroxidation proceeds by a chain reaction that includes initiation, propagation and termination. Initiation occurs when an oxidant gives rise to an initiating lipid peroxyl radical (LOO•) by reaction with either a lipid (LH) or pre-existing lipid hydroperoxide (LOOH). Propagation is cycled through rounds of LOO• abstraction of the bis-methylene hydrogen atoms of a poly unsaturated fatty acyl chain to generate new LOO•'s (after O₂ addition) that results in the net conversion of lipids to LOOHs. Lipid peroxidation termination involves the reaction of two LOO• to form non-radical products or the reaction of one LOO• with another terminating radical to generate non-propagating radical species (Catala, 2006).

2.4 NATURAL ANTIOXIDANT DEFENCE MECHANISMS

Living organisms protect themselves from harmful effects of free radicals by antioxidant defense mechanisms. The term “antioxidant” refers to any molecule capable of stabilizing or deactivating free radicals before they attack the cells (Rahman, 2007).

Biological antioxidants react with free radicals or precursor metabolites converting them into less reactive molecules and preventing or delaying oxidation of biological molecules. The protective antioxidant compounds are located in organelles, subcellular compartments or the extracellular space enabling maximum cellular protection to occur (Surai, 2007).
Antioxidants are classified as enzymic and non-enzymic antioxidants. Enzymic antioxidants are superoxide dismutase (SOD), glutathione peroxidase (GPx) and catalase (CAT). Non-enzymic antioxidants are α-tocopherol, β-carotene, vitamin A, ubiquinols, ascorbate, melatonin, glutathione, cysteine, ceruloplasmin, hemoglobin, bilirubin, albumin and minerals such as zinc, selenium and chromium. Enzymic and non-enzymic antioxidants work synergistically and in combination with each other to protect the cells and organ systems of the body against free radical damage (Karamouz et al., 2009).

2.4.1 Enzymic antioxidants

2.4.1.1 Superoxide dismutase (SOD)

SOD plays an important role in protecting the cells against the potentially deleterious effect of superoxide radicals (Mahmoud and Hijazi, 2007). SOD is one of the most effective intracellular enzymic antioxidants and it catalyzes the conversion of superoxide anions to dioxygen and hydrogen peroxide (Rahman, 2007).

Superoxide dismutase exists in several isoforms, which differ in the nature of active metal centre, amino acid composition, co-factors and other features. There are three forms of SODs present in humans: cytosolic Cu, Zn-SOD, mitochondrial Mn-SOD and extracellular-SOD (Landis and Tower, 2005).

2.4.1.2 Catalase (CAT)

CAT is an enzyme which is present mainly in the peroxisomes of mammalian cells. It is a tetrameric enzyme consisting of four identical, tetrahedrally arranged subunits of 60 kDa, each containing in its active center a heme group and NADPH (Scibior and Czeczot, 2006).

Catalases are ubiquitous antioxidant enzymes and irrespective of their origin catalyze the same basic reaction, the breakdown of hydrogen peroxide into water and oxygen (Chelikani et al., 2005).
2.4.1.3 Glutathione peroxidase (GPx)

GPx is one of the important antioxidant enzymes that protects the cell against oxidative damage (Yang et al., 2009). It is present both in the cytosol and mitochondria of various mammalian tissues. In animals, this enzyme is found in two forms namely selenium dependent GPx and selenium independent GPx (Li et al., 2004).

The selenium dependent enzyme contains selenocysteine at the active site and catalyzes the reduction of peroxides as well as organic hydroperoxides with glutathione as its hydrogen donor (Fu et al., 2007). The selenium independent GPx is active with organic hydroperoxides only and is identical with some glutathione transferase.

2.4.2 Non-enzymic antioxidants

2.4.2.1 Vitamin C

Vitamin C is an important and powerful water-soluble antioxidant and thus works in aqueous environments of the body. Its primary antioxidant partners are vitamin E and the carotenoids. Therefore vitamin C also works along with the antioxidant enzymes. Vitamin C co-operates with vitamin E to regenerate α-tocopherol from α-tocopherol radicals in membranes and lipoproteins (Kojo, 2004) and also raises intracellular glutathione levels thus playing an important role in protein thiol group protection against oxidation (Naziroglu and Butterworth, 2005).

2.4.2.2 Reduced glutathione (GSH)

GSH is a multifunctional intracellular antioxidant abundant in cytosol, nuclei and mitochondria and is considered to be the major thiol-disulphide redox buffer of the cell. The primary biological function of glutathione is to act as non-enzymic reducing agent to assist in keeping cysteine thiol side chains in a reducing state on the surface of proteins and to block free radical damage (Chavan et al., 2005).
It also acts as a co-factor for several detoxifying enzymes, participates in amino acid transport across plasma membrane, scavenges hydroxyl radical and singlet oxygen directly and regenerates vitamins C and E back to their active forms (Masella et al., 2005).

2.5 SIGNIFICANCE OF DIETARY ANTIOXIDANTS IN POULTRY

Poultry possesses limited natural resistance against colonization or infection by pathogenic organisms. For this reason, the poultry industry is suggested to rely on the use of antibiotics to improve the health and productivity of birds (Abdukalykova and Ruiz-Feria, 2006).

However, the use of antibiotics in animal production is under severe public scrutiny because livestock production practices have been linked to the development of antibiotic-resistant bacteria within the human population (Phillips et al., 2004).

Hence, antibiotics are being taken out of poultry diets around the world (Dibner and Richards, 2005). The poultry industry is hence compelled to find alternatives to the use of antibiotics in order to maintain and enhance markets for poultry products. It has been known that nutrition plays a significant role in the modulation and function of the immune response of chickens (Kidd, 2004).

It has been suggested that antioxidant/pro-oxidant balance is responsible for a regulation of major physiological functions in the body. The antioxidant/pro-oxidant balance could be modulated by sub-optimal diets and poor nutrient intakes, or positively affected by dietary supplementation (Surai, 2007).

As birds are exposed to increased stress factors, such as higher growth rates, increased infection pressure, antioxidant defense systems are in danger of losing control. This gives increased health risk to the birds as well as a progressive deterioration in the quality of animal products and losses in productivity (Mellor, 2001).
The most important step in preventing oxidative damage and balancing antioxidant defense in the animal body can be to enhance the antioxidant capacity by optimising the dietary intake of antioxidants. The causes and consequences of lipid peroxidation are shown in Figure 1.

**FIGURE 1**

**LIPID PEROXIDATION - CAUSES AND CONSEQUENCES**

**Antioxidant defense**
- Natural antioxidants in the feed: Vitamins A, E, C, carotenoids
- Synthetic antioxidants in the feed: BHT, ethoxyquin etc.
- Diet optimization
- Optimized environmental conditions
- Disease prevention and treatment (vaccines, antibiotics, coccidiostats etc.)

**Stress conditions**
- Nutritional stress: Toxins, high PUFA, deficiencies of vitamin E, Se, Mn, Zn, Fe overload
- Environmental stress: Temperature, humidity, hyperoxia, radiation, UV, microwave etc.

**ANTIOXIDANT SYSTEMS**
- Vitamins A, E, C; carotenoids, ubiquinol, uric acid, antioxidant enzymes [SOD, GSHPx, catalase]

**FREE RADICAL GENERATION**
- Electron-transport chain, phagocytes, xanthine oxidase etc.

**Lipid peroxidation, damage to lipids, proteins, DNA**
- Injuries to heart, vascular, brain, nervous & muscle system
- Membrane damage
- Decreased nutrient absorption

**DECREASE OF PRODUCTIVE & REPRODUCTIVE PERFORMANCES**

- Meat quality and shelf life reduction
- Immune incompetence

**ACRONYMS**
- BHT - Butylated hydroxytoluene
- SOD - Superoxide dismutase
- GSHPx - Glutathione peroxidase
- PUFA - Poly unsaturated fatty acid

*(Mellor, 2001)*
Animal feeds contain a range of different compounds that possess antioxidant activities including vitamin E, carotenoids, vitamin C, glutathione, selenium, zinc, copper, iron and manganese (Surai and Dvorska, 2002).

It is important to realize that all antioxidants in the body function in concert to provide antioxidant defense. If the diet is balanced and sufficiently provided with dietary antioxidants and antioxidant nutrients, then even low doses of such antioxidants are effective. On the other hand, under conditions of oxidative stress where free radical production dramatically increases, without external help it is difficult to prevent damage to major organs and systems. This external help takes the form of increased dietary supplementation of natural antioxidants, especially vitamin E and selenium (Surai, 2007).

2.6 VITAMIN E AND ITS BIOLOGICAL SIGNIFICANCE

Vitamin E is recognized as an essential nutrient for all species of animals, as well as humans. Vitamin E has been discovered in 1922 by Evans and Bishop as 'factor X' in vegetable oils essential for reproduction in female rats, which was later called as 'vitamin E' (Sure, 1924). It is chemically known as \( \alpha \)-tocopherol.

2.6.1 Chemical structure of vitamin E

The term vitamin E is used to describe eight lipophilic, naturally occurring compounds that include four tocopherols and four tocotrienols designated as \( \alpha \), \( \beta \), \( \gamma \) and \( \delta \) (Zingg, 2007). Of these, \( \alpha \)-tocopherol is the most biologically active and most widely distributed form of vitamin E. Tocotrienols are many times more potent as antioxidants than are tocopherols, but they are poorly assimilated by digestion, are poorly distributed to tissues in blood and are rapidly metabolized and eliminated from the body (Packer et al., 2001).

Recently, novel natural vitamin E analogues have been discovered. Vitamin E analogues occur in low amounts, such as the \( \alpha \)-tocomonoenol in palm oil and marine derived tocopherol in some marine organisms with a single
unsaturated bond at the end of the side chain, which is assumed to be the result of cold-water adaptation (Ng et al., 2004; Yamamoto et al., 2001). The phosphorylated form of α-tocopherol, α-tocopheryl phosphate, occurs naturally in foods and in animal as well as in human tissues (Gianello et al., 2005), acting possibly as a storage molecule, as a transport form, or as “lipid messenger” for the modulation of signal transduction and gene expression (Munteanu et al., 2004; Negis et al., 2005).

2.6.2 Absorption, transport, distribution and metabolism of vitamin E

All forms of vitamin E are absorbed apparently to the same extent in the intestine and secreted into the circulation in chylomicrons. Inadequate fat intake limits vitamin E absorption (Bruno et al., 2006). Lipoprotein lipase (LPL) hydrolyzes the chylomicron triglyceride and transfers fatty acids, as well as vitamin E to the tissues. During the formation of chylomicron remnants in the plasma compartment, some of the vitamin E is transferred to high density lipoproteins (HDLs) and subsequently to other lipoproteins.

The chylomicron remnants are taken up by the liver, where the α-tocopherol transfer protein (α-TTP) salvages α-tocopherol (α-T) from the lysosomal degradation pathway and returns it to the circulating lipoproteins, principally very low density lipoproteins (VLDLs). During lipoprotein catabolism in the circulation, α-tocopherol is redistributed among the various lipoproteins. Lipoproteins are taken up by the liver and peripheral tissues by various receptors and thus tocopherols are delivered to tissues (Traber, 2007).

α-TTP selectively binds α-tocopherol, as compared to other vitamin E forms and facilitates its secretion from the liver into the plasma for distribution to the tissues (Panagabko et al., 2002). The mechanism by which α-TTP facilitates the transfer of α-tocopherol to the plasma membrane for incorporation into VLDL, and/or HDL, has been attempted (Mustacich et al., 2007).
FIGURE 2
ABSORPTION AND METABOLISM OF VITAMIN E

Dietary & Supplemental Vitamin E

INTESTINE

Fatty acids & vitamin E to tissues

Vit. E

LIVER

INTESTINE

Vitamin E

Chylomicrons

Vitamin E

Remnants

Circulating Lipoproteins

Tissue Vitamin E Uptake

HDL

α-T

(Vit. E)

α-T (Vit. E)

VLDL

Liver Uptake

MDR 1

MDR 2/3

Bile Canaliculus

Xenobiotics

Phosphatidyl Choline

MDR 2/3

Glucuronide

CEHC

α-Tocopherol

P450s

Traber, 2007

LPL - Lipoprotein lipase
α-T - α-Tocopherol
α-TTP - α-Tocopherol transfer protein

MDR 1 - Multidrug-resistance gene product 1
MDR 2/3 - Multidrug-resistance gene product 2/3

CEHC - Carboxy ethyl hydroxy chroman
Excess α-tocopherol and the other tocopherol and tocotrienol analogues were found to be extensively metabolized before excretion (Pfluger et al., 2004). Mustacich et al. (1998) observed that in the liver, excess α-tocopherol and other vitamin E forms are excreted into bile via the multidrug-resistance gene products (MDR 2/3), e.g., P-glycoprotein [ATP-binding cassette (ABC) and other transporters].

Excess vitamin E is also metabolized similar to xenobiotics as studied by Birringer et al. (2002) by a cytochrome P450 (CYP)-mediated process to carboxy ethyl hydroxy chromans (CEHCs) that can be glucuronidated or sulfated and excreted in bile or urine. High α-tocopherol concentrations in the liver seemed to up-regulate various xenobiotic pathways, including CYP3A and MDR1 (Figure 2).

2.7 SELENIUM AND ITS BIOLOGICAL SIGNIFICANCE

Selenium was discovered by Berzelius in 1817 and has been recognized as an essential trace element for animals including humans (Hatfield et al., 2006). It was demonstrated that trace amounts of selenium protected against liver necrosis in vitamin E deficient rats and hence established its nutritional essentiality (Schwarz and Foltz, 1957).

Selenium is a required nutrient and essential trace mineral for all domestic animals, including poultry. It is an essential component of tissues and enzymes involved in the cellular antioxidant protection, because metabolic processes in the organism are susceptible to oxidative attack (Peric et al., 2007).

2.7.1 Properties and chemical forms of selenium

Selenium has both metallic and non-metallic properties and is considered a metalloid. Selenium is very similar to sulphur in its physical and chemical properties (Zeng, 2009). Selenium has an atomic number of 34 and
atomic weight of 78.96. It belongs to Group 16VIA with oxygen, sulfur, tellurium and polonium and its neighbours are arsenic and bromine.

Selenium is found in two forms in nature: inorganic and organic. Inorganic selenium refers to different minerals such as selenite, selenate and selenide and organic selenium is related to aminoacids, methionine and cysteine (Kralik et al., 2009). The compounds available for use as selenium supplements include the inorganic forms, sodium selenite, sodium selenate; and the organic forms, selenomethionine, Se-methylselenocysteine and high-selenium yeast (Schrauzer, 2001).

Organic selenium in the form of selenomethionine is a predominant form of this element in feed ingredients and therefore digestive system of animals including chickens has adapted itself during evolution to this form of the element (Skrivan et al., 2008). Selenite, a common form of selenium used in diets, is not found naturally and as a result is less effective in terms of assimilation from feed and its incorporation into the body (Surai, 2002). Furthermore, a pro-oxidative activity of sodium selenite has been established through reactions with reduced glutathione (Wycherly et al., 2004).

2.7.2 Selenoproteins

Selenium has been found to participate in various physiological functions; most importantly as an integral part of a range of selenoproteins. Recent evidence strongly suggested that there are at least 25 selenoproteins in the mammalian selenoproteome (Kryukov et al., 2003) and this would suggest more genes that encode selenoproteins.

The majority of the selenoproteins contain a single selenocysteine residue per polypeptide chain (Tujebajeva et al., 2000). The known selenoproteins have numerous functions, but many of the selenoproteins still have unknown functions (Table1).
### TABLE 1
**SELENOPROTEINS AND THEIR ROLE IN THE MAINTENANCE OF HOMEOSTASIS IN ANIMALS**

<table>
<thead>
<tr>
<th>Selenoproteins</th>
<th>Function</th>
<th>Reference</th>
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<tbody>
<tr>
<td><strong>Glutathione peroxidases (GPx)</strong></td>
<td></td>
<td></td>
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<tr>
<td>GPx-1 (cytosolic)</td>
<td>Reduces reactive molecules and free radicals; complements action of vitamin E</td>
<td>Rotruck et al. (1973); Flohe et al. (1973)</td>
</tr>
<tr>
<td>GPx-2 (gastrointestinal)</td>
<td>Antioxidant; reduces ingested lipid hydroperoxides</td>
<td>Chu et al. (1993)</td>
</tr>
<tr>
<td>GPx-3 (plasma)</td>
<td>Selenium carrier</td>
<td>Takahashi et al. (1987)</td>
</tr>
<tr>
<td>Phospholipid hydroperoxide GPx (spermatozoa and testis)</td>
<td>Directly reduces phospholipids and cholesterol hydroperoxides</td>
<td>Ursini et al. (1985)</td>
</tr>
<tr>
<td>GPx- sperm nucleus</td>
<td>Protamine thiol peroxidase; responsible for disulfide cross-linking; necessary for sperm maturation and male fertility</td>
<td>Behne et al. (1988, 1997)</td>
</tr>
<tr>
<td>GPx-6</td>
<td>Homologue of GPx-1</td>
<td>Kryukov et al. (2003)</td>
</tr>
<tr>
<td><strong>Thioredoxin reductases (TrxR)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cytosolic TrxR (TrxRI)</td>
<td>Reduces protein thiols and thioredoxin; provides reducing equivalents to several redox-dependent systems</td>
<td>Tamura and Stadtman (1996)</td>
</tr>
</tbody>
</table>
TABLE 1 (Contd...)

<table>
<thead>
<tr>
<th>Selenoproteins</th>
<th>Function</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Testicular TrxR (TrxR2)</td>
<td>Reduces protein thiols and thioredoxin; provides reducing equivalents to several redox-dependent systems</td>
<td>Gasdaska et al. (1999); Lee et al. (1999); Miranda-Vizuete et al. (1999); Watabe et al. (1999)</td>
</tr>
<tr>
<td>Mitochondrial TrxR (TrxR3)</td>
<td>-do-</td>
<td>Sun et al. (1999)</td>
</tr>
</tbody>
</table>

Iodothyronine deiodinases (ID)

| ID Type-I | Converts thyroxine (T4) to tri-iodothyronine (T3) | Behne et al. (1988, 1990); Arthur et al. (1990); Berry et al. (1991) |
| ID Type-II | Converts T4 to T3 and reverse T3 (rT3) to T2 | Croteau et al. (1996); Salvatore et al. (1996) |
| ID Type-III | Antioxidant in brain; converts T3 to rT3 and rT3 to T2; protects developing brain from excess T3 | Kaplan (1986); Croteau et al. (1995); Mortimer et al. (1996) |

Other selenoproteins

| Sel N,T,X | Unknown | Kryukov et al. (2003); Lescure et al. (1999) |
| Sel P | Selenium carrier; antioxidant | Herrman (1977); Motsenbocker and Tappel (1982) |
2.7.3 Absorption and bioavailability of selenium

Inorganic and organic forms of selenium in animal diets are absorbed by different mechanisms. Organic selenium is more thoroughly resorbed and more efficiently metabolised than its inorganic equivalent, which is poorly resorbed and acts more as a pro-oxidant provoking glutathione oxidation and oxidative damage to the DNA (Wycherly et al., 2004).

Selenium is highly absorbable with no homeostatic control mechanism for its absorption (Vasundara, 2006). Selenite is absorbed from the intestine by a simple diffusion process, whereas selenate is actively absorbed in the ileum by co-transport with sodium ions.
Selenomethionine contained in forages and grains is absorbed in the small intestine through the Na\(^+\)-dependent neutral amino acid transport system. A major portion of selenium is stored in the liver and to a lesser extent in the kidney and muscle. Small amounts exist in plasma and other organs (Patrick, 2004).

### 2.7.4 Metabolism of selenium

After absorption, dietary selenium of either inorganic or organic form is either used in the synthesis of selenoproteins, stored or excreted. The metabolism of selenium is dynamic (Ganther, 2001). The biologically active selenoproteins, of which around 20 have been identified, are primarily redox enzymes that contain a selenocysteine residue at the active site. Though metabolic fates of dietary inorganic and organic selenium forms differ, both must be converted to the common selenide form (hydrogen selenide), before selenium can be incorporated specifically into the active site of one of the selenoproteins (Figure 3) (Daniels, 1996).

Selenate is reduced to selenite and selenite is reduced through formation of selenodiglutathione to selenide. Selenomethionine is activated initially by adenosylation, demethylated and converted to selenocysteine through selenohomocysteine and selenocystathione in analogy to methionine. Pyridoxine-dependent enzymes are involved in the activation of selenomethionine and in this way vitamin B6 status and selenomethionine metabolism are related. The selenocysteine formed is then degraded in the liver to serine and selenide (Schrauzer, 2000).

Hydrogen selenide is a key metabolite that acts as a precursor for selenoprotein synthesis and is the excreted form of selenium. The hydrogen selenide is methylated and later excreted as dimethylselenium via the lungs while most of the excess selenide is excreted via urine in the form of the trimethylselenonium ion (Behne and Kyriakopoulos, 2001).
FIGURE 3
METABOLISM OF DIETARY SELENIUM IN ANIMALS

General body proteins

Dietary selenoproteins

Selenomethionine

Selenocysteine

Selenodeglutathione GS-Se-GS

Selenide H₂Se

Excretion

CH₂SeH Methyl selenol

(CH₃)₂Se Dimethyl selenol

(CH₃)₂Se⁺ Trimethylselenonium

Excreted via urine

Excreted via lungs

Selenophosphate

Selenocysteine

Selenoprotein

Selenate

Reduction

Selenite

Selenoprotein synthesis

ATP

AMP + P₁

Selenocysteine synthase

Growing peptide chain

Selenoprotein mRNA reads the Secys insertion sequence

(Schrauzer, 2000)
2.7.5 Health and product quality benefits from selenium

Selenium is an essential trace mineral for all animals and humans. It is important in antioxidant defense systems, but it has a narrow margin of safety. The importance of selenium in animal nutrition has been linked to a number of health and product quality benefits provided that selenium is supplied in the right form and dosage, which includes improved antioxidant defense systems leading to better resistance against disease, enhanced immune responses, superior resistance to viral diseases, maintenance of thyroid function, improved fertility in breeding animals, enhanced selenium content of meat and eggs, improved meat colour, reduced drip loss, protection against colon, prostate and breast cancer, reduced incidence of cardiovascular diseases and better tolerance to stress (Ganpute and Manjunatha, 2003).

2.7.6 Selenium toxicity

Selenium is toxic to poultry when used in high doses. However it is necessary to stress that selenium toxicity can usually be only seen when the dose exceeds at least ten fold the physiological requirement. In general, adequate selenium supplementation is considered to be a crucial factor in maintaining the high productive and reproductive characteristics of commercial poultry (Surai, 2002).

2.7.7 Selenium deficiency diseases

Selenium deficiency diseases have long been recognized in livestock, because most feed raw materials are deficient in this mineral. Selenium deficiency has been associated with many health problems in farm animals namely pancreatic atrophy, exudative diathesis, encephalomalacia and muscular dystrophy in chicks, impaired feathering in poultry, reduced egg production, increased dead-in-shell chicks, poor egg hatchability, low chick weights at hatching, suboptimal immunocompetence, mulberry heart disease in young pigs, white muscle disease in young ruminants, retained placenta and weak or stillborn calves, reproductive failure and infertility in all species (McCartney, 2006).
2.8 VITAMIN E AND SELENIUM SUPPLEMENTATION IN POULTRY

2.8.1 Body weight and growth rate

It was reported that body weight of broilers was significantly higher in vitamin E (250 mg/kg) and vitamin E plus organic selenium supplemented groups (250 mg + 0.2 mg/kg) than that of control and vitamin E plus inorganic selenium supplemented groups (250 mg + 0.2 mg/kg) (Salman et al., 2007).

The basal diet supplemented with 0.3 mg/kg selenomethionine was found to increase the body weight of broiler chickens by about 3% compared to control and sodium selenite supplemented group (Skrivan et al., 2008).

It has been reported that body weight of sel-plex (0.2 ppm) fed broilers was increased as compared to control and sodium selenite treatment groups (0.2 ppm). The combination of sodium selenite (0.1 ppm) and sel-plex (0.1 ppm) did not improve the body weight over the sel-plex fed group. It was concluded that the combination of sodium selenite and sel-plex was no more effective than sel-plex alone (Upton et al., 2008).

In contrary, Yoon et al. (2007) reported that broiler chicks fed supplemental selenium from organic (selenium yeast A and B) or inorganic (sodium selenite) sources did not affect the final body weight and average daily gain, although the final body weight of birds seemed to be greater with selenium yeast A than selenium yeast B.

The effect of dietary supplementation of Ocimum sanctum leaf powder (0.25% and 0.5%) and organic selenium (0.3 ppm) and their combination on the growth rate was studied in broiler chickens. It was concluded that supplementation of Ocimum sanctum leaf powder and organic selenium did not significantly influence the growth rate of broilers (Reddy et al., 2007).

2.8.2 Feed consumption and feed efficiency

The inclusion of vitamin E (250 mg/kg) in combination with either organic or inorganic selenium (0.2 mg/kg) in the diet of broilers had no effect on feed...
intake. But it was shown that feed conversion ratio was more efficient in the vitamin E plus organic selenium (250 mg + 0.2 mg/kg) supplemented group compared to the vitamin E and vitamin E plus inorganic selenium supplemented groups (Salman et al., 2007).

It was observed that broiler chicks fed supplemental selenium from organic (selenium yeast A and B) or inorganic (sodium selenite) sources had lower feed intake and increased feed efficiency during the first three weeks, but not during the following three weeks (Yoon et al., 2007).

Japanese quail exposed to high ambient temperature was fed on a basal diet or the basal diet supplemented with either three levels of vitamin C (0, 250 and 500 mg of L-ascorbic acid/kg of diet) or three levels of vitamin E (0, 250 and 500 mg of dl-α-tocopheryl acetate/kg of diet). It was reported that feed intake was not affected by vitamin C and E supplementation under thermo-neutral conditions. However, it was increased with vitamin C or E supplementation either singly or in combination in heat-stressed quail (Sahin et al., 2009).

2.8.3 Immune response

The effect of arginine and vitamin E (40, 80, and 400 IU / kg feed) on humoral and cellular immunity of broiler chickens was investigated and was found that antibody titres to sheep red blood cells were higher in arginine and vitamin E supplemented groups. The interaction between arginine and vitamin E was not significant. Moreover, the antibody titres were significantly higher in birds supplemented with 80 mg (4.66 ± 0.22) of vitamin E than 40 and 400 mg (3.33 and 2.83 ± 0.23) supplemented groups respectively (Abdukalykova and Ruiz-Feria, 2006).

The effect of increasing dietary selenium on immune responses was studied in growing (0 to 6 weeks) Japanese quail. The birds were fed the basal diet which contained 0.2 mg selenium/kg and the two experimental diets were supplemented with 0.5 and 1.0 mg selenium/kg and it was found that there
were significantly higher antibody titres against inoculated sheep red blood
cells in both the supplemented groups (Biswas et al., 2006).

The humoral immune response of broilers fed diets supplemented with zinc (0, 40, and 400 mg/kg) and vitamin E (0, 12, and 120 mg/kg), either separately or in combinations was evaluated against Newcastle Disease. Newcastle antibody levels in serum samples were analyzed using hemagglutination inhibition (HI) and enzyme-linked immunosorbent assay (ELISA). The interaction between the highest dietary levels of zinc and vitamin E resulted in higher antibody levels in ELISA after 14, 28, 35 and 41 days. Increasing levels of zinc and vitamin E supplemented separately in the diets provided greater levels of hemagglutinating antibodies in HI at 14, 28, 35 and 41 days of age (Cardoso et al., 2006).

Broiler chicks were supplemented with different concentrations of vitamin E (0-200 mg/kg) and selenium (0-0.2 mg/kg diet) either alone or in combinations in the maize-soybean diets from 1 to 42 days of age. It was reported that chicks receiving supplements of 200 mg vitamin E/kg and 0.2 mg selenium/kg recorded significantly higher HI antibody titres against Newcastle Disease Virus (NDV) vaccine (Singh et al., 2006).

Immunity was assessed as antibody production to Infectious Bronchitis Virus (IBV), Newcastle Disease Virus (NDV) and Sheep Red Blood Cell (SRBC) in broilers fed different levels of dietary vitamin E (0, 50, 75, IU / kg diet) and L-ascorbic acid (0, 500 and 1000 ppm in drinking water). Antibody titre for primary and secondary responses to SRBC was increased by 50 IU / kg vitamin E. Humoral immunologic response showed that antibody titre to NDV and IBV were highest in groups receiving 75 IU / kg diet vitamin E. Secondary responses to SRBC and antibody titre to NDV and IBV were improved by supplementing 500 ppm ascorbic acid in drinking water. This data suggested that optimum growth and humoral immune response might be achieved at supplemental level of vitamin E of 50 IU / kg and vitamin C at 500 ppm (Nameghi et al., 2007).
Weber et al. (2008) studied the effect of toxin contaminated feed (2.35 mg/kg) and/or vitamin E supplementation in drinking water (10.5 mg/animal/day) for 14 days on the hemagglutination inhibition titres against Newcastle Disease Virus in repeatedly vaccinated (on 23 days of age) broiler chicken. It was found that hemagglutination inhibition titres were significantly increased in the vitamin E supplemented group as compared to the vaccinated toxin control group, but only on day 7.

2.8.4 Biochemical profile

Antioxidant vitamins (vitamin E -100mg/kg/day and vitamin C - 200 mg/kg/day) supplementation significantly increased HDL-cholesterol in the broiler chickens compared to the control group and also caused significant elevation in serum HDL/LDL cholesterol ratio. Serum triglyceride and LDL-cholesterol levels were found to be decreased, but no significant changes were noted in total cholesterol (Ozturk et al., 2000).

It was reported that combination of dietary lycopene and vitamin E supplementation significantly reduced serum cholesterol concentrations in Japanese quails (Sahin et al., 2006).

The effects of vitamin E and heat stress on the metabolism of laying hens was investigated and was concluded that heat stress (35°C) lead to significant increase in plasma total protein, albumin, total lipid and cholesterol concentrations. It was also observed that extra vitamin E supplementation had no effect on the metabolism of laying hens (Yardibi et al., 2009).

Lin et al. (2005) indicated that maternal supplementation with high levels of vitamin E (120-160 mg/kg) enhanced antioxidant capability and depressed oxidative stress in chicks. Plasma vitamin E concentrations were increased linearly with the increase in supplemental vitamin E, but those in egg yolk reached a plateau at 120 mg/kg supplemental vitamin E. The malondialdehyde (MDA) concentration and reactive oxygen species of chick brain were
decreased linearly with an increase in supplemental vitamin E. Chicks of pullets given 120 mg/kg supplemental vitamin E had higher activities of liver catalase and those given 160 mg/kg supplemental vitamin E had higher activities of brain superoxide dismutase.

Ahmed et al. (2007) studied the effect of oral administration of enzymes and vitamins on hematological and biochemical parameters in broilers for a period of 21 days. They found that red blood corpuscles, packed cell volume and hemoglobin contents were increased significantly in the treated groups as compared to that of control group while serum glutamate oxaloacetate transaminase and serum glutamate pyruvate transaminase activities were decreased significantly in all the treated groups as compared to that of the control group.

The effect of dietary supplementation of the Ocimum sanctum leaf powder (0.25% and 0.5%) and organic selenium (0.3 ppm) and their combination was studied on lipid peroxidation in broiler chickens. It was concluded that dietary supplementation of Ocimum sanctum at 0.5% level and its combination with selenium significantly decreased lipid peroxidation levels with concomitant increase in glutathione levels in plasma (Reddy et al., 2007).

The basal diet supplemented with 0.3 mg/kg selenomethionine indicated a significant increase in the body weight of broiler chickens by about 3%. Breast muscle selenium concentration was increased by both selenium sources and this effect was more pronounced in the selenomethionine treatment. Dietary selenium supplementation increased the α-tocopherol content of breast meat from 25.9 mg/kg in the control to 33.2 mg/kg when selenomethionine supplementation was used. The extent of lipid oxidation, as measured by malondialdehyde formation in breast meat was lower in broilers fed with supplemented selenium diets than in the control group (Skrivan et al., 2008).
It was reported that blood selenium content of laying hens was significantly higher in all selenium supplemented groups compared to the control. The concentration of copper in egg albumen and egg yolk was significantly lower in the experimental group that received selenium-yeast at an amount of 0.9 mg/kg compared to the sodium selenite experimental group. The concentration of zinc was significantly lower and concentration of iron was significantly higher in the group supplemented with sodium selenite. A significantly higher concentration of calcium in egg yolk was detected in groups receiving selenium yeast compared to those given only sodium selenite. Supplementation of selenium did not significantly affect the concentration of phosphorus in egg yolk (Arpasova et al., 2009a).

2.8.5 Egg production and egg quality characteristics

Andi et al. (2006) established that Haugh units of eggs that indicate the freshness of eggs were higher in hens fed 40, 60, 80, 100 and 120 IU of vitamin E/kg feed at 35 weeks of age.

Japanese quails were fed a basal diet or basal diet supplemented with lycopene (100 mg/kg diet), vitamin E (250 mg dl-α-tocopheryl-acetate/kg diet) or a combination of lycopene and vitamin E (100 mg/kg lycopene plus 250 mg dl-α-tocopheryl-acetate/kg diet) and it was found that separately or as a combination, supplemental lycopene and vitamin E increased serum and egg yolk vitamin E content (Sahin et al., 2006).

It has been showed that egg production in laying hens in both poultry conditions (heat stress and normal) was increased significantly with the supplementation of dietary vitamin E (Bolukbasi et al., 2007).

The sources of selenium - both the organic and inorganic forms were added into the basal diet at 0, 0.2, 0.5, and 1.0 mg/kg and the effects of a commercial inorganic selenium source (sodium selenite) were compared with a commercial organic selenium source (selenium enriched yeast) on whole-egg
selenium concentrations in laying hens. The results showed that the addition of selenium from either source caused a significant increase in whole-egg selenium concentrations. There was a more significant increase in the selenium concentrations in eggs from hens fed selenium enriched yeast than those from hens fed sodium selenite (Pan et al., 2007).

The effects of selenite or zinc-L-selenium methionine in broiler breeder diets were evaluated on egg production. It was noticed that the hens fed 0.3 ppm of organic selenium during the first period produced significantly more eggs, whereas no difference in egg production was found in the second period (Reis et al., 2009).

The effect of vitamin C (L-ascorbic acid) and vitamin E (α-tocopherol acetate) supplementation was evaluated on egg production in the Japanese quail exposed to high ambient temperature. Birds were kept in wire cages in a temperature-controlled room at either 22°C (thermo-neutral) or 34°C (heat stress) for 8 hours/day (09:00 to 17:00 hours; until the end of the study) and fed on a basal diet or the basal diet supplemented with either one of the three levels of vitamin C (0, 250 and 500 mg of L-ascorbic acid/kg of diet) or three levels of vitamin E (0, 250 and 500 mg of dl-α-tocopheryl acetate/kg of diet). Egg production was not affected by vitamin C and E supplementation under thermo-neutral conditions. However, egg production was increased with the vitamin C or E supplementation either singly or in combination in heat-stressed quail (Sahin et al., 2009).

It was reported that the average weight of the whole egg, egg albumen and egg yolk in birds receiving selenized yeast-enriched diets was higher than those of eggs from control and sodium selenite-supplemented groups. The Haugh units score revealed a higher quality of egg albumen in the groups of birds fed the selenized yeast supplemented diet (Arpasova et al., 2009a).

Higher GPx activity was observed in the blood of hens supplemented with 0.4 ppm of organic selenium in the diet than the hens those received...
0.2 ppm of selenium. Furthermore, the hens supplemented with 0.4 ppm selenium had a significantly higher content of selenium in egg yolk and albumen (Gajcevic et al., 2009).