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EVALUATING THE EFFECTS OF MATERNAL AND PROGENY DIETARY SUPPLEMENTATION OF SELENIUM YEAST AND VITAMIN E ON THE PERFORMANCE OF BROILER-BREEDER HENS AND PERFORMANCE AND MEAT QUALITY OF PROGENY

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EVALUATING THE EFFECTS OF MATERNAL AND PROGENY DIETARY SUPPLEMENTATION OF SELENIUM YEAST AND VITAMIN E ON THE PERFORMANCE OF BROILER-BREEDER HENS AND PERFORMANCE AND MEAT QUALITY OF PROGENY

DISSERTATION

A dissertation submitted in partial fulfillment of the
requirements for the degree of Doctor of Philosophy in the
College of Agriculture at the University of Kentucky

by

Anthony David Quant
Lexington, Kentucky

Director: Dr. Anthony J. Pescatore, Extension Professor of Animal and Food Sciences
Lexington, Kentucky
2012

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ABSTRACT OF DISSERTATION

EVALUATING THE EFFECTS OF MATERNAL AND PROGENY DIETARY SUPPLEMENTATION OF SELENIUM YEAST AND VITAMIN E ON THE PERFORMANCE OF BROILER-BREEDER HENS AND PERFORMANCE AND MEAT QUALITY OF PROGENY

The objectives of these experiments were to evaluate the effects of selenium (Se) and vitamin E (Vit.E) supplementation in maternal and progeny diets on the performance of breeder hens and the performance and meat quality characteristics of progeny.

Inclusion of Se, as Se yeast, in the diets of developing broiler breeder pullets resulted in greater Se accumulation of Se ($P<0.01$) in liver, pancreas, and breast tissues than when Se yeast was not provided. Improving the overall Se status of breeder pullets in the early stages may help maintain adequate tissue Se concentrations during egg production.

Maternal supplementation of Se yeast and Vit.E increased the liver and breast Se concentration ($P<0.05$) of newly hatched chicks compared to the chicks originating from hens not receiving dietary Se. At 7d of age, Se yeast supplementation in either the chick or maternal diet increased breast and liver Se concentrations ($P<0.01$). At 14d of age, breast and liver Se concentrations remained the highest for chicks supplemented with Se yeast ($P<0.01$), however there was no effect of maternal Se supplementation. Vitamin E supplementation in either the chick or maternal diets did not affect the liver Vit.E concentrations of chicks at 7 or 14d of age.

Supplementing broiler diets with Se yeast and Vit.E improved the meat quality characteristics of raw and marinated breast fillets. The Se content of breast meat from broilers fed Se yeast was higher ($P<0.01$) than those from broilers that were not fed Se yeast. Antioxidant supplementation improved the drip loss ($P<0.05$) and oxidative stability ($P<0.10$) of raw breast fillets after 7d of refrigerated storage. Marination appeared to increase the susceptibility for lipid oxidation of the marinated breast fillets. Dietary supplementation of Se yeast and Vit.E reduced lipid oxidation ($P<0.01$) of marinated breast fillets after prolonged refrigerated storage, thus improving oxidative stability.

Overall, dietary supplementation of Se yeast can increase the accumulation of Se in the tissues of broiler breeder hens and their subsequent progeny. Improvements in the

avian antioxidant system may have beneficial effects on the performance of broiler breeder hens, broilers, and the meat quality characteristics of broiler breast fillets.

KEYWORDS: Selenium, Vitamin E, broiler breeders, progeny, meat quality

Anthony David Quant

July 6th, 2012

EVALUATING THE EFFECTS OF MATERNAL AND PROGENY DIETARY SUPPLEMENTATION OF SELENIUM YEAST AND VITAMIN E ON THE PERFORMANCE OF BROILER-BREEDER HENS AND PERFORMANCE AND MEAT QUALITY OF PROGENY

By

Anthony David Quant

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This work is dedicated to my great grandfather Elmer W. Rieke, my grandfather Wallace D. Hagen, and my grandmother Alice B. Quant.

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF FILES

Quant.Dissertation.pdf

CHAPTER 1. Literature review

1.1. Introduction

Selenium (Se) is a controversial element as it is characterized as both a toxic element and as an essential nutrient. Selenium was first identified for its toxic properties by the Swedish chemist Jons Jakob Berzelius in 1817, as it was the cause of worker illness in a local sulphuric acid plant (Oldfield, 2002). It was not until 1957 that Se was identified as a nutrient by Klaus Schwarz, who demonstrated it was required in the prevention of liver necrosis in rats (Schwarz and Foltz, 1957). It was later discovered that Se deficiency was associated with a variety of animal diseases in addition to the rat, especially in livestock (Oldfield, 2002).

Alongside vitamin E (Vit.E), Se plays a critical role in the avian antioxidant system by reducing oxidative damage within the body. It was first identified by Rotruck et al. (1973) that Se is an integral component of the glutathione peroxidase enzymes (GSH-Px). This family of enzymes is responsible for the removal of damaging hydroperoxides and organic peroxides (Surai, 2006). Selenium is also involved in the recycling of Vit.E through the joint action of GSH-Px and Se-dependent thioredoxin reductase (TrxR), and ascorbic acid (May et al., 1997; Surai, 2006). The combined action of Se-dependent GSH-Px on peroxides and the free radical chain breaking action of Vit.E provides excellent antioxidant protection within the avian body (Surai, 2006).

The plane of maternal nutrition has a considerable effect on the health and subsequent performance of the progeny because all of the nutrients required for embryonic development of the progeny are transferred to the egg from the hen (Hamal et al., 2006). During incubation, the nutrients that have been deposited in the egg are assimilated into the tissues of the developing chick. The hatching process is a time of high oxidative stress, therefore the status of the chick’s antioxidant system at the time of hatch is a key determinant in the hatchability and survivability of the chick (Surai, 1999; Surai, 2006). This further emphasizes the importance of antioxidant supplementation in the maternal diets, as the antioxidant status of the progeny is reflected by that of the dam (Surai, 2006). Previous research has indicated that maternal Se supplementation

effectively improved the Se status of the chicks at hatch, with lasting maternal effects throughout the early stages of growth (Pappas et al., 2005; Surai et al., 2006).

Antioxidant supplementation has been shown to affect the meat quality characteristics of poultry meat, specifically regarding water holding capacity (WHC) and oxidative stability. Water holding capacity (as measured by percent drip loss) can be affected by oxidative damage which compromises the integrity of the cellular membranes, and allowing for uncontrolled movement of water between and outside of the various compartments of the muscle (Edens et al., 1996). It has been reported that supplementing broiler diets with Se can reduce oxidative damage through the effect of GSH-Px, therefore reducing the drip loss of poultry meat (Edens, 1996; Choct et al., 2004; Upton et al., 2008; Jiang et al., 2009; Perić et al., 2009). Lipid peroxidation is one of the primary causes of rancidity in raw and cooked meat products under refrigerated storage. Previous studies have reported improved oxidative stability (i.e. reductions in lipid peroxidation) in poultry meat products when broiler have been supplemented with Se, Vit.E, or both (Sheldon et al., 1997; Guo et al., 2001; Chekani-Azar et al., 2010; Kim et al., 2010; Narciso-Gaytan et al., 2010; Voljč et al., 2011). The improvements in oxidative stability described in the previous research reports indicates that antioxidant supplementation in broiler diets may effectively prolong the onset of rancidity in poultry meat products, thereby extending the shelf life.

Therefore, the research presented herein was aimed at evaluating the effects of Vit.E and Se yeast supplementation in the diets of broiler breeder hens and broilers. Chapter 2 describes the evaluation of Se yeast and Vit.E on body weight (BW) uniformity and tissue Se and Vit.E deposition in the tissues of developing broiler breeder pullets, as well as the subsequent egg production performance of the hens through 40 weeks of production. Chapter 3 describes the evaluation of the transfer of Se and Vit.E from the dam to the progeny as well as the interactive effects of maternal and progeny dietary supplementation of Se and Vit.E on the growth performance of the progeny. Finally, chapter 4 describes the evaluation of the effects of supplementing broiler diets with Se and Vit.E on the meat quality characteristics of raw and marinated breast fillets.

The information generated from these experiments will hopefully further characterize the essentiality of Se as a nutrient for poultry.

1.2. The avian antioxidant system

1.2.1. The need for antioxidant defense

Aerobic respiration in the mitochondria of animals, plants, and microorganisms generates energy by reducing molecular oxygen to water. In the natural process of aerobic respiration, small amounts of partially reduced oxygen are produced and these products are called free radicals (Aruoma et al., 1991; Surai, 2006). A free radical refers to any molecule with an unpaired electron in its outer shell, and these molecules are energetically unstable and highly reactive. Halliwell and Gutteridge (1999) introduced the terms reactive oxygen species (ROS) and reactive nitrogen species (RNS) for oxygen and nitrogen radicals as well as their non-radical derivatives such as hydrogen peroxide (H_2O_2) or peroxynitrite ($ONOO^-$; Table 1.1).

Table 1.1. Reactive oxygen and nitrogen species.[1]

Radicals	*Non-radicals*
Alkoxyl, $RO^\bullet$	Hydrogen peroxide, H_2O_2
Hydroperoxyl, $HOO^\bullet$	Hypochlorous acid, HOCl
Hydroxyl, $^\bullet OH$	Ozone, O_3
Peroxyl, $ROO^\bullet$	Singlet oxygen, $^1O_2$
Superoxide, $O_2^\bullet$	Peroxynitrite, $ONOO^-$
Nitric oxide, $NO^\bullet$	Nitroxyle anion NO^-
Nitrogen dioxide, $NO_2^\bullet$	Nitrous acid, HNO_2

[1]Adapted from Halliwell and Gutteridge (1999).

The major free radical produced during normal metabolism is the superoxide ($O_2^{\bullet -}$) radical (Surai, 2006). This radical can inactivate certain enzyme systems through the formation of unstable complexes with transition metals within the enzyme (Halliwell and Gutteridge, 1999). Interestingly, $O_2^{\bullet -}$ by itself is not extremely reactive as it does not readily cross the lipid bilayer (Surai, 2006). Superoxide is, however, a precursor to more damaging species such as $ONOO^-$ or $^\bullet OH$, which have been shown to be damaging to a wide variety of biological molecules including proteins, deoxyribonucleic acids (DNA), and lipids (Halliwell and Gutteridge, 1999; Benzie, 2000; Surai, 2006). The $^\bullet OH$ is the

most reactive species of free radicals, as it reacts with anything it contacts and causes extensive intracellular damage (Benzie, 2000; Surai, 2006). In many cases, $^{\bullet}OH$ triggers the chain reaction in the peroxidation of lipids (Surai, 2006). Hydroxyl radicals are formed in the presence of $O_2^{\bullet-}$ and H_2O_2 via the Haber-Weiss reaction below (Benzie, 2000):

$$H_2O_2 + O_2^{\bullet-} \rightarrow {}^{\bullet}OH + OH^- + O_2$$

The most significant impact free radicals have on cellular metabolism is through their participation in lipid peroxidation (Surai, 2006). The peroxidation of lipids is a chain reaction that proceeds in three stages (Burton and Traber, 1990). The first step (Reaction 1) is the initiation phase, which is the formation of carbon-centered lipid radical from a precursor molecule (initiator) such as $^{\bullet}OH$.

Reaction 1: $RH \rightarrow R^{\bullet}$

This is followed by the propagation phase (Reactions 2 and 3) where in the presence of oxygen, the lipid radical rapidly reacts with oxygen, yielding a highly reactive $ROO^{\bullet}$ that can then attack another lipid molecule. When the $ROO^{\bullet}$ attacks another lipid, the original $ROO^{\bullet}$ is converted to a hydroperoxide (ROOH) and a new carbon-centered lipid radical is formed.

Reaction 2: $R^{\bullet} + O_2 \rightarrow ROO^{\bullet}$

Reaction 3: $ROO^{\bullet} + RH \rightarrow ROOH + R^{\bullet}$

Lipid peroxidation is therefore a propagating chain reaction that can potentially cause a substantial amount of damage to cells (Burton and Traber, 1990; Surai, 2006). This process continues until the termination phase (Reaction 4) when the $ROO^{\bullet}$ reacts with another $ROO^{\bullet}$ to form inactive products (Burton and Traber, 1990).

Reaction 4: $ROO^{\bullet} + ROO^{\bullet} \rightarrow$ inactive products

1.2.2. Three levels of antioxidant defense

Living organisms have developed a specific endogenous defense system to protect cells from the damaging actions of ROS and RNS which is generally referred to as the

antioxidant system (Halliwell and Gutteridge, 1999). The avian antioxidant system includes natural fat soluble antioxidants (vitamins A, E, carotenoids, and ubiquinones), water soluble antioxidants (ascorbic acid, taurine), antioxidant enzymes (GSH-Px, catalase, and superoxide dismutase), and the thiol redox system (thioredoxin; Surai, 2006). These antioxidant compounds are located within organelles, in subcellular compartments or in the extracellular space allowing for optimum protection against oxidative damage throughout the body (Surai, 2006).

It has been proposed that the avian antioxidant system is comprised of three different levels of defense (Surai, 2006). The first level of defense is responsible for preventing the formation of damaging free radicals through the removal free radical precursors such as H_2O_2 or by inactivating catalysts. The key players in the first level of antioxidant defense includes the antioxidant enzymes superoxide dismutase (SOD), GSH-Px, and catalase (CAT), as well as metal-binding proteins which act on the cellular level. Superoxide dismutase successfully reduces the superoxide radical into H_2O_2 and oxygen, while GSH-Px and CAT reduce H_2O_2 to water and oxygen. Metal-binding proteins such as transferrin or metallothionenin prevent hydroxyl radical formation by binding transition metals which prevents their participation in radical reactions (Surai, 2006).

The first level of antioxidant defense alone is not sufficient to completely prevent free radical formation in the body. Therefore, the second level of antioxidant defense comes into play and this level is responsible for the prevention and restriction of chain formation and propagation that is caused by free radicals. The second level consists of the chain-breaking antioxidants vitamin A, vitamin C, vitamin E (Vit.E), and carotenoids which act to prevent peroxidation by minimizing the propagation chain length (Surai, 2006). The third and final level of antioxidant defense focuses on the repair or removal of damaged molecules and consists of lipases, proteases, transferases, and DNA repair enzymes (Surai, 2006).

It is believed that all of the antioxidants within the avian antioxidant system work together in harmony and that there is a delicate balance that exists between the generation

of free radicals and free radical scavenging (Surai, 2006). This balance can be affected by several exogenous factors including nutrition, environmental conditions, and disease status. In addition, inclusion of both natural and synthetic antioxidants in the diet along with optimal levels of key trace minerals (i.e. Se, Mn, Cu, and Zn) may improve the efficiency and effectiveness of the avian antioxidant system at preventing damage from free radicals (Surai, 2006).

1.3. Selenium

1.3.1. History of selenium

Selenium was first discovered in 1817 by the Swedish chemist Jons Jakob Berzelius while investigating the source of worker illness in a local sulphuric acid plant (Oldfield, 2002). Berzelius originally suspected the origins of this illness may have been due to toxic properties of arsenic or tellurium. He later analyzed sludge from the vats of sulfuric acid and identified that is was a new element in which he named “selenium” after the Greek moon goddess Selene (Oldfield, 2002). Consequently, much of the following research associated with Se was directed at preventing or coping with the toxicity of this element.

Selenium was known solely as a toxic element for well over 100 years after its discovery as much of the following research associated with Se was directed at preventing or coping with the toxicity of this element. It was not until 1957 when the role of Se as an essential nutrient was discovered by the German biochemist Klaus Schwarz (Schwarz and Foltz, 1957). In his studies, Schwarz had discovered that rats fed diets containing torula yeast as the dietary protein source developed necrosis of the liver. However, the necrosis disappeared when the protein source was substituted with brewer’s yeast (*Saccharomyces cerviseae*) and deduced that brewer’s yeast contained an essential nutrient that was not found in torula yeast. The unknown substance was referred to as “Factor 3” due to the fact that Vit.E and *L*-cysteine (known as Factors 1 and 2) had already been recognized for their abilities to alleviate liver necrosis. Fractional analysis of the brewer’s yeast determined that Se was an integral component of Factor 3, and it was later shown that that as little as 13.33 μg of sodium selenite (SS, Na_2SeO_3) was

capable of preventing necrotic livers in rats (Schwarz and Foltz, 1957). This discovery by Schwarz and Foltz (1957) identified the first disease associated with Se deficiency, and later lead to the recognition of Se as an essential nutrient (McCoy and Westin, 1969). In the following years, it was discovered that Se deficiency was associated with several animal diseases in addition to liver necrosis including white muscle disease in lambs and calves, hepatosis dietetica in swine, and exudative diathesis and pancreatic degeneration in poultry (Oldfield, 2006).

Early research clearly demonstrated that Se played a critical role in the prevention of several metabolic diseases, however, its biochemical function as a nutrient in the animal body was still relatively unknown. In 1973, Rotruck et al. (1973) discovered the erythrocytes of Se-deficient rats were practically lacking in GSH-Px activity, an enzyme responsible for metabolizing damaging hydroperoxides. Further analysis revealed that that Se was an integral component of GSH-Px. This discovery confirmed the essentiality of Se as a nutrient and that only a small amount of dietary Se is required to provide metabolic benefits through its involvement in the GSH-Px enzyme systems.

1.3.2. Dietary sources of selenium

Selenium is a chemical element belonging to group VI in the periodic table with an atomic number of 34 and an atomic mass of 78.96. It has similar properties to those of sulfur and tellurium, and therefore is commonly found associated with sulfur in various organic and inorganic compounds (Leeson and Summers, 2001). In nature, Se can be found in two different forms where it is bound to organic or inorganic substrates (Surai, 2006). Inorganic Se exists in three different oxidation states, Se^{-2} (selenide), Se^{+4} (SeO_3^{-2}, selenite) or Se^{+6} (SeO_4^{-2}, selenate) and can be found complexed with different minerals in these forms.

The Se concentration in forages and grains depend on the Se concentration and biological availability of Se in the soil (Combs and Combs, 1984). The content of Se in soils varies significantly depending on geographical location (Leeson and Summers, 2001; Reilly, 1996). Regions containing large amounts of sedimentary rocks and shale are

generally high in Se, while regions with lower concentrations of Se contain volcanic rock, granite, limestone, and sandstone (Surai, 2006). In North America, regions with soils deficient in Se include the pacific northwestern, northeastern, and very southeastern United States, as well as north central and eastern Canada (Combs and Combs, 1984). Plants absorb Se in soils either in the form of selenite or selenate and incorporate it into selenoamino acids by replacing the sulfur group in methionine and cysteine with Se, yielding selenomethionine (SeMet) and selenocysteine (SeCys) (Figure 1.1; Schrauzer, 2000, 2003). Indeed, Se is found primarily in the organic forms as SeMet (more than 50% of total Se) in animal feed ingredients (grains, oil seeds, forages, etc.), where Se is in the Se^{-2} oxidation state (Surai, 2006). In animal nutrition, the most important chemical forms of Se are the inorganic forms selenite and selenate, and the organic forms SeMet and SeCys (Wolffram, 1999).

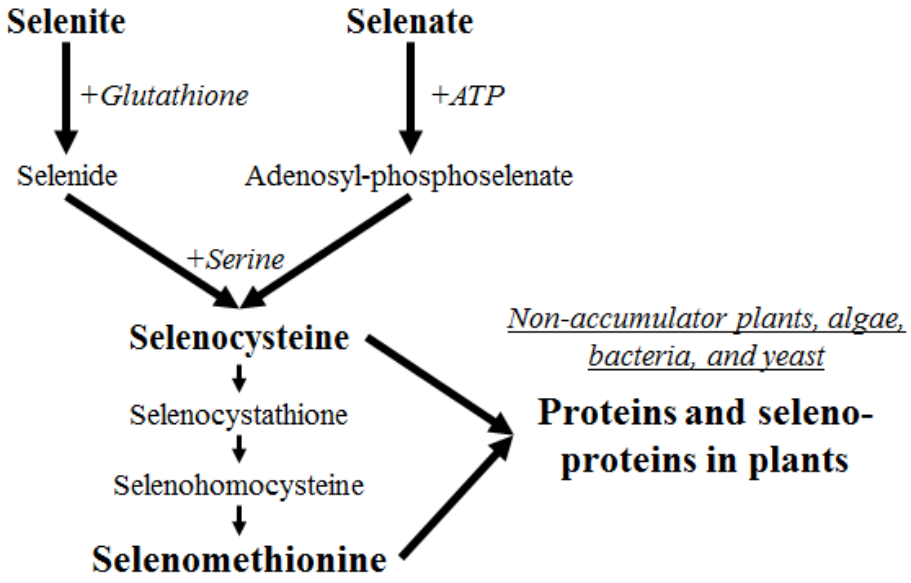

Figure 1.1. Biosynthesis of selenomethionine in plants, algae, bacteria, and yeast (Adapted from Schrauzer, 2000, 2003).

Selenium is commonly included in the trace mineral premixes in poultry diets, with inorganic sources such as SS being most common. The inclusion of organic sources of Se such as Se-enriched yeast or algae (specifically *chollera*) in poultry diets has received considerable attention in recent history. Selenium-enriched yeast is the most

common form of organic Se fed to poultry and is produced by growing *Saccharomyces cerevisiae* in a nutrient medium that is rich in Se. According to Schrauzer (2000), Se-enriched yeast may contain up to 90% of the total Se as SeMet.

1.3.3. Selenium storage and metabolism

The bioavailability of Se may be defined as the amount of Se that is effectively absorbed by the intestinal tract that is metabolically available to the animal for normal physiological processes (Wolffram, 1999). It has been established that there is a positive correlation between tissue Se accumulation and the functional activity of Se-containing compounds such as GSH-Px in the avian body (Surai, 1999; Wolffram, 1999; Leeson and Summers, 2001). However the bioavailability of Se can be affected by several factors including its chemical form, physiological state of the animal, and the bioavailability and metabolism of other nutrients (Surai, 2006).

Absorption of Se differs considerably between the two main forms of dietary Se. Sodium selenite is absorbed by the digestive tract by simple diffusion (Wolffram et al., 1988). In contrast, SeMet is actively transported across the intestinal brush border membrane by the same absorption mechanisms associated with methionine (Wolffram et al., 1989; Leeson and Summers, 2001). Overall, both the inorganic and organic forms of Se appear to be relatively well absorbed by the small intestine, suggesting that intestinal absorption of Se alone does not limit Se bioavailability (Vendeland et al., 1992; Wolffram, 1999).

The bioavailability of dietary Se is dependent upon the conversion of absorbed Se into a form that is biologically active or stored (Tapiero et al., 2003). Once absorbed, Se is transported throughout the body by plasma bound proteins and is incorporated into a number of selenoproteins with specific functions. These selenoproteins include the GSH-Px enzymes, TrxR, and various other selenoproteins including selenoproteins P and W (Wolffram, 1999). It has been well established that organic Se (specifically SeMet) is more bioavailable than inorganic sources because SeMet is non-specifically substituted for methionine into bodily proteins, especially into skeletal muscle and liver tissue

(Daniels, 1996; Leeson and Summers, 2001; Surai, 2006). Thus, SeMet makes up a considerable portion of the total pool of Se in the body (Daniels, 1996). Two specific Se pools in the body have been identified (Janghorbani et al., 1990; Daniels, 1996). The first pool, termed the exchangeable metabolic pool, consists of all of the forms of Se derived from inorganic sources such as SS and includes endogenously synthesized selenoproteins such as GSHPx and selenoamino acids. All of the functionally important seleno-compounds are metabolized in this pool (Daniels, 1996). The second Se pool contains all of the SeMet containing proteins, however the function has not been identified aside from contributing to the body's overall Se stores (Daniels, 1996).

Glutathione peroxidase is one of the most important selenoproteins in the avian body, as it plays an integral role in the first line of antioxidant defense by removing the precursors of free radicals (Surai, 2006). There are 6 different members within the GSH-Px family of enzymes that vary in size, distribution in the body, and function; however, it has been well established that all types are involved in the reduction of damaging peroxides (Surai, 2006; Lei et al., 2007). Four of the members of this family of enzymes are Se-dependent and include cystolic (GSH-Px1), gastrointestinal (GSH-Px2), extracellular (GSH-Px3), and phospholipid hydroperoxide (GSH-Px-4; Brigelius-Flohé, 1999; Roy et al., 2005; Surai, 2006). Cystolic GSH-Px is the most abundant selenoenzyme in the body (expressed in most tissues), whereas GSH-Px2 is expressed primarily in the gastrointestinal tract (Lei, 2007). Extracellular GSH-Px is primarily expressed in the kidneys; however it exists in a variety of other tissue types in lower concentrations. Phospholipid hydroperoxide GSH-Px, as its name suggests, is the only member of this enzyme family that is capable of reducing phospholipid peroxides and it is expressed in renal and epithelial cells as well as in the testes (Lei et al., 2007; Surai, 2006). The remaining known members of this family of enzymes are GSH-Px5 and GSH-Px6, both of which are not Se-dependent but possess similar properties to Se-dependent GSH-Px enzymes in their capacity to catalyze peroxides (Lei et al., 2007).

The role of GSH-Px as an antioxidant is to reduce hydrogen peroxides and organic peroxides through its SeCys-containing active site, selenol (ESeH), utilizing

reduced glutathione (GSH) as an electron donor (Figure 1.2; Seis et al., 1997; Mugesh and Singh, 2000; Roy et al., 2005; Surai, 2006). Selenol is oxidized to selenic acid (ESe-OH), which then reacts with a molecule of reduced GSH to form a selenosulfide adduct (ESeSG). Then a second reduced GSH reacts with ESeSG yielding oxidized glutathione (GSSG) and the active form of the enzyme ($E\text{-}Se^{-}+H^{+}$) which can then catalyze the hydroperoxide (Seis et al., 1997; Roy et al., 2005; Surai, 2006). To summarize the entire process, two units of GSH are oxidized to glutathione disulfide and water, and the hydroperoxide is reduced to its corresponding alcohol (Roy et al., 2005).

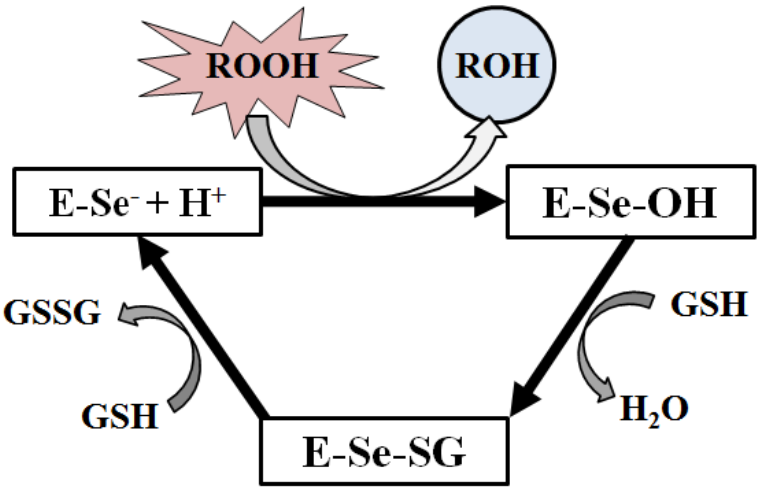

Figure 1.2. Proposed catalytic mechanism of the reduction of hydroperoxides by GSH-Px (Adapted from: Seis et al., 1997; Roy et al., 2005).

Thioredoxin reductase is another selenoenzyme that plays a key role in the avian antioxidant system. The Trx system plays a variety of roles in the body including antioxidant defense, redox regulation, gene regulation, DNA synthesis regulation, and protein biosynthesis (Surai, 2006). There are three known forms of TrxR in the body: TrxR1 exists primarily in the cytosol, TrxR2 in the mitochondria (Powis et al., 2000), and TrxR3 in the testes (Sun et al., 1999). As an antioxidant, TrxR can directly reduce hydrogen peroxide, lipid hydroperoxides, and ascorbyl free radicals (Arnér and Holmgren, 2000; Surai, 2006). In addition, TrxR can reduce Trx, GSH, dehydroascorbic acid, and selenite, which could be beneficial for cellular antioxidant defense in terms of the reactivation of oxidatively inactivated proteins (May et al., 1997; Surai, 2006). The

general recycling of antioxidants in the body is partially dependent on the ability of TrxR to reduce dehydroascorbic acid to ascorbate, and links TrxR to Se and Vit.E within the entire scope of the avian antioxidant system (May et al., 1997).

1.3.4. Selenium requirements of poultry

Considering the narrow range between essentiality and toxicity of Se in livestock, most countries have imposed very strict limitations on the concentration of supplemental Se in livestock diets. This is true for the United States of America, as the maximum level of supplemental Se was set at 0.3 ppm for all major food producing animals by the Food and Drug Administration (FDA, 1987a,b). Overall, the Se requirement for poultry is relatively low, ranging from 0.06 to 0.20 ppm, and the true requirement depends largely on the physiological state of the bird (NRC, 1994). Cantor and Scott (1974) first reported that egg production was maximized when Se was included at 0.1 ppm in layer diets, while also increasing the Se concentration of the eggs when compared to the unsupplemented control. Further studies have indicated that Se supplementation in layer diets effectively increases egg Se concentrations (Combs and Scott, 1979; Payne et al., 2005; Utterback et al., 2005; Pan et al., 2007; Pavlovič et al., 2009; Čobanová et al., 2011). The Se status of broiler breeder hens is extremely important, as a lowered Se status of the breeder can reduce egg production in the breeders as well as reduce the hatchability of the subsequent offspring (Surai, 2006). Indeed, several studies have reported that Se supplementation in breeder diets can improve chick hatchability as well as improve the Se status of the newly hatched chick (Surai, 2000; Paton et al., 2002; Pappas et al., 2005; Pappas et al., 2006; Surai et al., 2006; Skřivan et al., 2008; Wang et al., 2010; Wang et al., 2011; Wu et al., 2011; Yuan et al., 2011). Maternal Se supplementation may not only aid in protecting the developing chick from damaging free radicals, but also stimulate the development and function of the avian immune system (Pappas et al., 2005; Surai et al., 2006).

Supplementing Se in broiler diets has not been shown to improve growth performance (Edens et al., 2001; Payne and Southern, 2005), however it has been shown to improve the meat quality characteristics of the subsequent poultry meat products.

These benefits include improved WHC, oxidative and color stability (Edens, 1996; Choct et al., 2004; Upton et al., 2008; Jiang et al., 2009; Perić et al., 2009). In general, Se is included in the trace mineral premixes of most poultry diets at a level of 0.3 ppm.

1.3.5. Selenium deficiency in poultry

Selenium deficiency is responsible for the development of several diseases in poultry due to the impairment of the avian antioxidant system. In fact, most of these diseases involve combined deficiencies of both Se and Vit.E, which can result in dysfunctions of the brain, muscles, liver, pancreas, and nervous, cardiovascular, immune, and reproductive systems (Combs and Combs, 1984; Van Vleet and Ferrans, 1992; Surai, 2006). Classical diseases resulting from Se deficiency in poultry include exudative diathesis (ED) in chicks, nutritional pancreatic atrophy (NPA), nutritional muscular dystrophy (NMD), and nutritional encephalomalacia (NE) (Surai, 2006). Given the interrelationship of Se and Vit.E in the avian antioxidant system, it is not surprising that many of these syndromes will respond to the administration of either Se or Vit.E. (Van Vleet and Ferrans, 1992).

Exudative diathesis is likely the most extensively researched disease relating to Se deficiency (Noguchi et al., 1973; Combs and Scott, 1974; Hassan et al., 1990; Barthlomew et al., 1998, Huang et al., 2011). This condition appears in chickens that are deficient in both Se and Vit.E and shows signs of severe edema as a result of increased capillary permeability caused by extensive lipid peroxidation and increased mortality (Noguchi et al., 1973; Barthlomew et al., 1998). This capillary leakage results in the subcutaneous accumulation of a viscous, bluish-green fluid of the breast and wings in chicks (Noguchi et al., 1973; Huang et al., 2011). Exudative diathesis is associated with low tissue levels of Se, GSH-Px, and Vit.E (Hassan et al., 1990). Although this disease is responsive to both Se and Vit.E supplementation, it is recognized primarily as a Se-deficiency syndrome because adequate levels of Se would promote the action of Se-dependent GSH-Px which reduces the damaging effects of peroxides on cellular membranes (Noguchi et al., 1973). Low levels of Vit.E supplementation do not appear to effectively prevent ED (Hassan et al., 1990), however high levels may be effective by

acting as a secondary defense against peroxidation within the membrane (Noguchi et al., 1973).

Nutritional muscular dystrophy is characterized by the degeneration of muscle fibers in poultry, most noticeable in the breast and thigh muscles, as well as myopathy of the gizzard and heart (Surai, 2006). Nutritional encephalomalacia is associated with peroxidative dysfunction in poultry and is characterized by ataxia, sudden prostration, extension of the legs, flexed toes, and twisting of the head, accompanied by uncontrollable muscle spasms (Fuhrmann and Sallmann, 1995; Surai, 2006). Although NMD and NE may be largely due to deficiencies in Vit.E, Se could play a positive role through improvements in the antioxidant system by optimizing the Se supply in the avian body (Surai, 2006).

Nutritional pancreatic atrophy is the only clearly defined disease that is due to Se deficiency alone, uncomplicated by deficiencies of Vit. E or any other nutrients (Combs and Combs, 1984; Whitacre et al., 1987). This condition is characterized by pancreatic lesions and atrophy, and is accompanied by very low levels of GSH-Px activity in the liver (Whitacre et al., 1987). In most Se-deficiency syndromes, deficiency symptoms can be relieved by the administration of Vit.E through the nutritional sparing of Se by Vit.E. However, this is not the case with NPA as prevention of this syndrome is not effective at dietary levels of Vit.E less than ~250 international units (IU)/kg (Whitacre et al., 1987).

In addition to the diseases mentioned above, Se and/or Vit.E deficiency can also display symptoms of reduced fertility, egg production, hatchability and increased embryonic mortality as well as decreased immunocompetence and impairment of thyroid hormone metabolism (Surai, 2006). Currently, Se deficiency is relatively rare due to the widespread inclusion of Se in poultry diets; however subtle symptoms of marginal Se status may appear including reduced oxidative stability and immunocompetence (Wolffram, 1999).

1.3.6. Selenium toxicity in poultry

The toxic properties of Se were well known long before it was first discovered as an essential nutrient. Selenium can be toxic to poultry when the dose exceeds 10 times the physiological requirement (Surai, 2002) and these effects have been well documented in the literature. Excessive dietary Se levels can reduce growth, egg production and hatchability, as well as affect reproductive performance by reducing fertility, hatchability, and cause embryonic abnormalities (Leeson and Summers, 2001). In truth, the margin between the beneficial and toxic effects of Se is rather small, therefore careful attention must be given to the formulation and mixing of Se in the trace mineral premixes of poultry diets. This was confirmed in a recent case study where breeding pheasants received diets that were abnormally high in Se (~9.3 ppm) as a result of poor cleaning of feed mill equipment after a Se premix was prepared from a concentrated source (Latshaw et al., 2004). The authors noted that this amount of Se was enough to reduce egg production and increase hen mortality in just 4 days. Necropsy of the expired hens revealed severe degenerative cardiomyopathy and hepatic necrosis. Detrimental effects on hatchability and embryonic development were also observed, most notably were severe deformities in the beak and eyes. The authors concluded that distinctive signs of Se toxicity include decreased egg production, increased mortality, and increased aggression and cannibalism in adult birds, and decreased hatchability and severe deformations in the beak and eyes of chicks.

1.4. Vitamin E

1.4.1. History of vitamin E

The discovery of Vit.E can be largely attributed to the work of Dr. Henry A. Mattill, a distinguished professor and researcher at the University of Rochester and the University of Iowa (Wolf, 2005). In 1920, Dr. Mattill evaluated whether milk could be an adequate food source for rats throughout their life span or whether it was lacking in a particular factor necessary for normal performance of physiological function (Mattill and Conklin, 1920). In this study, rats were fed a diet containing whole milk and the authors observed that growth performance declined after 50d and the females were sterile. These experiments were followed up by research evaluating various other milk products (i.e.

butterfat, dried whole milk) as well as well as experiments using purified diets that were adequate in vitamins A, B, and C, all of which produced similar results (Evans and Bishop, 1922; Mattill and Stone, 1923). Vitamin E was officially discovered by Evans and Bishop (1922), who concluded that natural foodstuffs as opposed to purified diets contained a particular substance that was essential for reproduction and this substance was later referred to as substance "X" by Mattill et al. (1924). Substance "X" was coined as "Vitamin E" by Barret Sure later in 1924, because vitamins A, B, C, and D were already known (Sure, 1924).

Vitamin E's properties as an antioxidant were first alluded to by Mattill et al. in 1927, citing that animal fats that are high in unsaturated fatty acids may be subjected to autoxidation, leading to the destruction of Vit.E. In contrast, the author also suggested that vegetable oils (especially wheat germ oil) contained a compound that inhibits autoxidation, thus preserving Vit.E. Interestingly, Mattill did not realize that this "antioxidizer" he referred to could in fact be the vitamin itself (Wolf, 2005). In 1931, Cummings and Mattill suggested that the physiological role of Vit.E in the body resides in its antioxidant properties and that the oxidation of Vit.E may protect other substances from oxidation. The authors concluded that Vit.E therefore controls the progress of oxidation in tissues through its antioxidant properties (Cummings and Mattill, 1931). Vitamin E was first isolated and characterized by Evans et al. (1936) who referred to Vit.E as a "tocopherol." The name tocopherol was derived from the Greek word *phero* meaning "to bring," and *tocos* meaning "childbirth" (Evans, 1962). In the following years, several forms of Vit.E were discovered and its role as an antioxidants was further characterized (Wolf, 2005).

1.4.2. Dietary sources of vitamin E

Vitamin E is the primary fat-soluble antioxidant in the avian antioxidant system and is obtained exclusively from the diet (i.e. not synthesized by the bird). There are eight different forms of Vit.E. from plant origin that are similar in structure, but differ in biological activity: α-, β-, γ-, and δ-tocopherol and the corresponding tocotrienols (Table 1.2; Traber and Arai, 1999; FAO, 2001). Tocotrienols differ from tocopherols as they

have an unsaturated side chain, whereas tocopherols have a saturated phytyl tail with three chiral centers which occur naturally in the *RRR* configuration (Traber and Arai, 1999; Singh et al., 2005). Of these different forms, D-α-tocopherol has the greatest biological activity. One international unit (IU) of Vit.E is equivalent to the activity of 1 mg of D-α-tocopherol acetate (NRC, 1998).

Table 1.2. Approximate biological activity of naturally occurring tocopherols and tocotrienols.[1]

Compound	Biological activity compared to *d*-α-tocopherol, %
d-α-tocopherol	100
d-β-tocopherol	50
d-γ-tocopherol	10
d-δ-tocopherol	3
d-α-tocotrienol	30
d-β-tocotrienol	5
d-γ-tocotrienol	Unknown
d-δ-tocotrienol	Unknown

[1]Adapted from FAO (2011).

The primary sources of Vit.E in animal diets are the tocopherols that exist in green plants and their derived oils (Leeson and Summers, 2001). To a lesser extent, Vit.E can also be found in animal fats such as lard or tallow. The Vit.E concentration of animal fats and vegetable oils commonly included in livestock diets as well as the relative contribution of tocopherols and tocotrienols to the total Vit.E concentration is detailed in Table 1.3. (Chow, 1985). It is important to note that oxidation, resulting from extreme storage conditions, can rapidly destroy the naturally occurring Vit.E in oils or feedstuffs, therefore it is often difficult to accurately predict the Vit.E content of these sources (NRC, 1998). Aside from plant sources, Vit.E is also commercially available in synthetic form as *all rac*-α-tocopherol (containing d-α-tocopherol acetate or dl-α-tocopherol acetate) which consists of the 8 possible stereoisomers in equal proportions (Leeson and Summers, 2001; Singh, 2005). The α-tocopherol acetate form of Vit.E is used because the ester bond protects Vit.E from oxidation during processing and storage of diets, as well as prior to absorption when ingested (Villaverde et al., 2008)

Table 1.3. Concentrations of vitamin E, tocopherols, and tocotrienols in fats and oils.[1]

Item	Total vitamin E (mg/100g)	Tocopherols (%)			Tocotrienols (%)			
		α	γ	δ	α	β	γ	δ
Animal Fats								
Lard	0.6-1.3	>90	<5		<5			
Butter	1-5	>90	<10					
Tallow	1.2-2.4	>90	<10					
Vegetable oils								
Soybean	56-160	4-18	58-69					
Cottonseed	30-81	51-67	33-49					
Corn	53-162	11-24	76-89					
Coconut	1-4	14-67		<17	<14	<3	<53	<17
Peanut	20-32	48-61	39-52					
Palm kernel	33-73	28-50		<9	16-19	4	34-39	<9
Safflower	25-49	80-94	6-20					
Olive	5-15	65-85				15-35		

[1]Adapted from Chow (1985).

1.4.3. Vitamin E storage and metabolism

The absorption of Vit.E appears to occur mainly through micellar formation in the small intestine (Leeson and Summers, 2001). Vitamin E absorption is dependent on the presence of bile acids, which are required for micelle formation (Bjørneboe et al., 1990; Kaydon and Traber, 1993). Absorption into the enterocytes occurs passively along with fatty acids as a lipid-bile-lipase micelle; therefore any of the factors affecting micellar formation can also influence the absorption of Vit.E (Kayden and Traber, 1993; Leeson and Summers, 2001). Once vitamin E is absorbed into the intestinal cells, the ester is hydrolyzed and about 99% of the now active Vit.E is secreted into chylomicrons (Bjørneboe et al., 1990; Kaydon and Traber, 1993). Unlike retinol or cholesterol, α-tocopherol is not reesterified following absorption; therefore, Vit.E is stored in tissues as α-tocopherol and not the ester form (Bjørneboe et al., 1990; Villaverde et al., 2008). Chylomicrons are a class of lipoproteins that transport lipids (and Vit.E in this case) to the liver via the portal vein and then to peripheral tissues (Leeson and Summers, 2001).The uptake of Vit.E in these tissues occurs during catabolism of the chylomicrons by lipoprotein lipase, which hydrolyzes triglycerides, releasing free fatty acids and Vit.E (Bjørneboe et al., 1990; Kaydon and Traber, 1993; Villaverde et al., 2008). Storage of

Vit.E occurs in the liver and in adipose tissue throughout the avian body (Leeson and Summers, 2001).

Vitamin E has several different metabolic functions in the avian body; however it is widely believed that its main function is through its role as the primary chain breaking antioxidant that inhibits the propagation of free radical chain reactions (Burton and Traber, 1990; Kaydon and Traber, 1993; Leeson and Summers, 2001; Traber and Atkinson, 2007, Villaverde et al., 2008). In addition to its role as antioxidant, Vit.E is also involved in normal tissue respiration, normal phosphorylation reactions, the metabolism of nucleic acids, ascorbic acid synthesis, ubiquinone synthesis, sulphur AA metabolism, and maintaining an active immune system (Leeson and Summers, 2001).

The Vit.E that is integrated into cellular membranes exerts its antioxidant effects by intercepting peroxyl radicals (ROO•) more rapidly than can polyunsaturated fatty acids (Burton and Traber, 1990). Its antioxidant property is due to the free OH- group on the aromatic ring of Vit.E, which donates a H^+ to the free radical yielding a stable, non-damaging, reduced free radical (Figure 1.3; Burton and Traber, 1990). The resulting α-tocopherol radical is stable enough to stop the chain reaction and is instead removed completely from the propagation cycle when reacted with another peroxyl radical, yielding a non-radical product. When Vit.E donates its H^+ (oxidized Vit.E), it is inactive as an antioxidant until it is reduced to the active form of Vit.E through reaction with an active free OH^- on its aromatic ring (Chow, 1991).

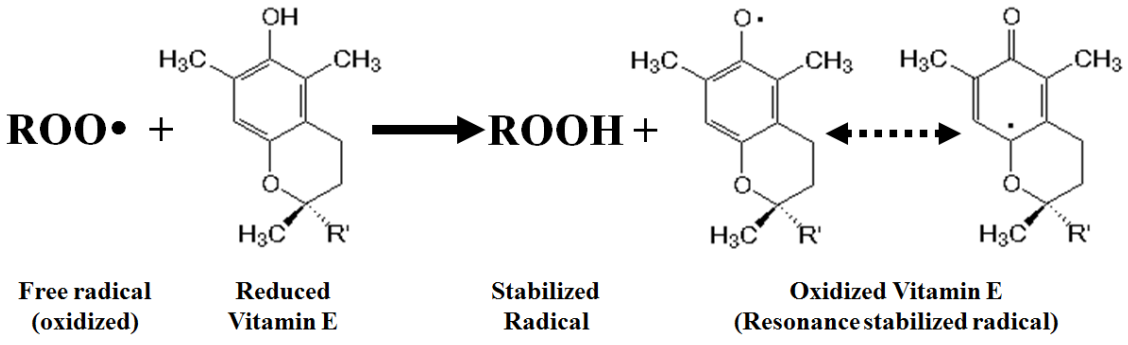

Figure 1.3. Mechanism of inactivation of free radicals by vitamin E (adapted from Burton and Traber, 1990).

In order for Vit.E to continually exert its antioxidant effects, the oxidized or radical form of Vit.E must be converted back to the active form through the action of other antioxidants. In the event that oxidized Vit.E is not recycled back to the reduced form, the molecule is therefore lost (excreted) and additional Vit.E must be obtained from the diet. Recycling of Vit.E in the avian body requires the oxidative decarboxylation of 6-phosphogluconate (6PG) to ribulose-5-phophate (R5P) in the pentose phosphate pathway, and the seleno-dependent enzymes GSH-Px and TrxR (Figure 1.4; Chow, 1991). Thioredoxin reductase reduces dehydroascorbic acid (DAA) back to reduced ascorbic acid (AA), which in turn recycles the oxidized α-tocopherol back to the reduced (active) form. Absence of the TrxR facilitated reduction of oxidized DAA to AA would result in minimal recycling of Vit.E in the body (Chow, 1991).

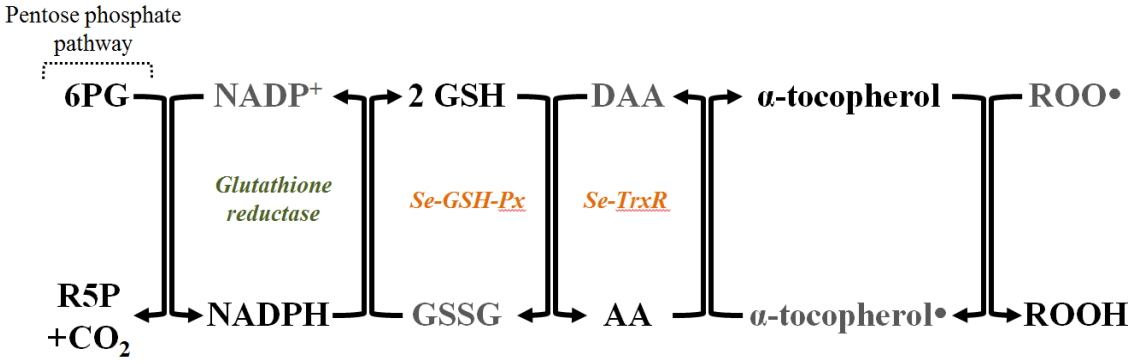

Figure 1.4. Proposed mechanism for the recycling of α-tocopherol. 6PG represents 6-phosphogluconate; R5P, ribulose-5-phosphate; NADP or NADPH, reduced or oxidized nicontinamide adenine dinucleotide; Se-GSH-Px, selenium dependent glutathione peroxidase; Se-Trx-R, selenium dependent thioredoxin reductase; DAA, dehydroascorbic acid; AA, reduced ascorbic acid. (Adapted from Chow, 1991).

1.4.4. Vitamin E requirements of poultry

The Vit.E requirement of poultry is quite variable as it depends on the relative concentration and type of fat in the diet (Vit.E is fat soluble), the presence of pro-oxidant compounds, as well as the Se concentration of the diet. Under ideal conditions, the Vit.E requirement is quite low, as the NRC (1994) recommends only 5 IU Vit.E/kg diet for laying hens, and 10 IU Vit.E/kg diet for broilers. In general, Vit.E supplementation at

levels well above the NRC (1994) recommendations have not been shown to affect growth performance or feed conversion of broilers (Hossain et al., 1998; Coetzee and Hoffman, 2001) or egg production and feed intake in layers or breeders (Hossain et al., 1998). However, previous research has indicated that Vit.E supplementation can improve the meat quality characteristics of poultry meat. This includes improvements in prolonging the onset of lipid oxidation and rancidity in meat, as well as improvements in color stability (Sheldon et al., 1997; Cotzee and Hoffman, 2001; Grau et al., 2001; Kim et al., 2010).

1.4.5. Vitamin E deficiency

As mentioned previously, Vit.E and Se share many of the same deficiency symptoms due to chronic impairment of the avian antioxidant system. The main deficiencies related to Vit.E are NE, ED, and NMD in addition to reductions in growth performance, egg production, and hatchability. In a commercial setting, the occurrence of these deficiency symptoms are rare and depend on the environmental and dietary conditions exposed to the birds (Leeson and Summers, 2001). Nutritional encephalomalacia develops if the diets are devoid of Vit.E, Se, or both, or if the diets are extremely high in unsaturated fatty acids (Surai, 2006). Exudative diathesis requires a deficiency in both Vit.E and Se for deficiency symptoms to appear (Bartholomew et al., 1998; Leeson and Summers, 2001). Nutritional muscular dystrophy, while extremely rare, develops as a result of deficiencies in both Vit.E and sulfur AA (Leeson and Summers, 2001).

1.4.6. Vitamin E toxicity

Vitamin E is considered one of the least toxic vitamins in livestock diets and therefore reports of toxicity have been scarce. Symptoms relating to Vit.E toxicity are most likely related to antagonisms with the other fat soluble vitamins such as A, D, or K (Leeson and Summers, 2001). This was confirmed by March et al. (1973) who reported that feeding 2,200 IU/kg Vit.E in chick diets resulted in reduced growth performance and bone calcification. The authors suggested that excess dietary Vit.E may have increased the chick's requirement for vitamin D. In addition to these symptoms, feeding above

4,000 IU/kg Vit.E has been shown to decrease the pigmentation of the beak, shanks, and feet, while above 8,000 IU/kg caused birds to develop waxy feathers (Nockels et al., 1976). In commercial practice, dietary supplementation of poultry diets with Vit.E is relatively low due in part to the high cost of synthetic Vit.E. The upper range of Vit.E inclusion in poultry diets would be considered between 100-200 IU/kg, and at these levels there have not been any consequential effects on the metabolism of any of the other fat soluble vitamins (Bartov, 1997; Leeson and Summers, 2001).

1.5. Maternal nutrition of the broiler breeder hen

1.5.1. Feeding the developing breeder

Early nutrition of the developing broiler breeder (BB) pullet requires a different approach when compared to that of fast growing broilers or egg layers. Unlike layers which are capable of regulating their feed intake based on their energy requirements, modern day BB strains, if fed on an *ad libitum* basis, will over consume feed beyond what is required for achieving energy homeostasis, thus resulting in an overweight bird (Richards et al., 2010). It is critical to control the growth and body weight (BW) of the developing broiler breeder pullet as it has been shown that overweight hens display reduced egg production, fertility, hatchability as well as increased likelihood of producing eggs with shell abnormalities or multiple yolks (Hocking, 1993; Bruggemann et al., 1999; Leeson and Summers, 2005; Pishnamazi et al., 2008). In practice, BW gain is limited throughout development (pre-lay) and egg production through the use of various feeding regimens that restrict feed intake (Leeson and Summers, 2000; Leeson and Summers, 2005; Richards et al., 2010). In general, the dietary energy level for developing BB pullets ranges from 2,750 to 2,950 Kcal ME/kg diet, while the levels of the other essential nutrients (protein, vitamins, minerals) will vary depending on the genetic strain of birds used and their growth potential (Leeson and Summers, 2005). Controlling the growth rate of developing BB pullets so that they reach their target BW prior to lay is an important management tool used to ensure optimum performance during the laying cycle.

The use of feed restriction regimens allows for controlled growth of the developing BB pullets while also maintaining ideal flock uniformity (80% of birds within

± 15% of the targeted mean weight; Leeson and Summers, 2005). Broiler breeder flocks with higher BW uniformity generally achieve peak egg production sooner and will have greater peak egg production than flocks of lower BW uniformity (Leeson and Summers, 2000; Leeson and Summers, 2005; Pishnamazi et al., 2008; Abbas et al., 2010). There are two commonly used feeding regimens used in commercial systems to control the pullet growth: skip-a-day feeding and continuous daily feeding. Skip-a-day feeding programs administer feed to the birds every other day, therefore by offering a relatively large quantity of feed every other day, it provides ample time and opportunity for all birds to eat (Leeson and Summers, 2005). The alternative to skip-a-day feeding would be continuous daily feeding, where the birds are administered feed on a daily basis. In practice, the feed allowance for continuous daily feeding is generally 2.2 times lower than the skip-a-day allowance in order to achieve equivalent performance (Leeson and Summers, 2005). No matter the regimen utilized, feed intake is gradually increased as age increases. At the time of lay, adult BB hens must remain on a restricted feeding program to maintain their optimum BW and are generally fed on a daily basis.

1.5.2. Breeder nutrition and the developing chick

Maternal nutrition plays a crucial role in the subsequent development and hatching of the progeny. The hen is responsible for depositing all of the nutrients required by the developing embryo into the egg prior to ovipositon (Wilson, 1997). Not just the concentration, but the also the forms of these deposited nutrients ultimately determine the success of the developing embryo and its hatching into a healthy chick (Vieira, 2007). Any nutritional deficiencies in the hen diet may directly translate into abnormal development of the embryo.

Adequate energy and protein levels in the BB diet are essential for optimal chick development and hatchability. Diets low in crude protein and energy can result in a reduction in egg albumin which may increase embryonic death and decrease hatchability of viable chicks (Lopez and Leeson, 1994). Aside from protein and energy, the vitamin and mineral status of the BB hen can have a major impact on bone development, growth, immunity and resistance to disease of the progeny (Wilson, 1997; Kidd, 2003). Adequate

levels of calcium, phosphorus, and vitamin D in BB diets will promote optimum bone development and hatchability of the subsequent progeny. However, any alterations in the previously mentioned diet, above that of adequate levels, are unlikely to have a significant positive impact on the early growth and survivability of the progeny (Kidd, 2003). Much of the research regarding mineral transfer to the progeny has focused on trace minerals. The trace minerals are deposited in the yolk and then transferred to the yolk sac which serves to store and regulate the transfer of these nutrients to the developing embryo (Richards, 1997). These trace minerals are utilized by the developing chick in cellular metabolism as cofactors in multiple enzymatic reactions during development including bone development, energy metabolism, and immune function (Wilson, 1997; Kidd, 2003).

Embryonic development and hatching is a time of high oxidative stress for the chick and is further compounded by the fact that the tissue lipids of the chick contain high levels of PUFA's (Speake et al., 1998; Surai, 2006). Therefore, it is critical to supply the BB hen with antioxidants such as Vit.E and Se to ensure the development of the progeny's antioxidant system during embryogenesis and in early postnatal development (Surai, 2006). As a fat soluble vitamin, Vit.E is deposited primarily in the yolk of the egg (Surai, 2006). In general, Se is deposited into the yolk of the egg; however the source of Se in the maternal diet can have a major impact on where Se is deposited into the egg (Latshaw and Biggert, 1981; Paton et al., 2002; Surai, 2006). Organic Se sources such SeMet are incorporated into the albumen proteins, while Se from inorganic sources (i.e. SS) tend to accumulate in the yolk (Latshaw and Biggert, 1981). Paton et al. (2002) evaluated Se deposition into the eggs of BB hens fed graded levels of either SeMet or SS. Supplementation of SS at 0.1, 0.2., and 0.3 ppm increased egg Se compared to the unsupplemented control, however there were no differences between inclusion levels. When SeMet was fed, egg Se continued to increase as inclusion levels increased and egg Se was greater when SeMet was fed compared to SS. Overall, the authors reported that SeMet was more efficiently deposited into the egg than was SS. Skřivan et al. (2008) reported that maternal SeMet supplementation was more effective at sparing Vit.E in the egg, as egg Vit.E concentrations were higher for SeMet than when SS was fed.

Increased Se and Vit.E concentrations in BB eggs could increase the antioxidant activity of the developing embryo and help defend against peroxidation (Surai, 2006; Wang et al., 2010).

Inclusion of SeMet in the maternal diet of BB hens has been shown to improve embryo viability, hatchability, and growth of the subsequent progeny (Pappas et al., 2005; Pappas et al., 2006; Wu et al., 2010; Wang et al., 2011). This was likely due to the fact that maternal SeMet supplementation increased tissue Se at hatch, which is reflected in higher GSH-Px activity and thus improved antioxidant activity during the stressful process of hatching (Surai, 2000; Pappas et al., 2005; Surai, 2006; Wang et al., 2011). As egg-derived antioxidants (i.e., Vit.E, β-carotene) are quickly depleted after the hatching process, antioxidant enzymes such as GSH-Px become the crucial line of defense against peroxidation post-hatch (Surai et al., 1998; Wang et al., 2011). In addition, Pappas et al. (2005) indicated that the effect of maternal SeMet supplementation remained apparent through 14d post-hatch. Therefore maternal Se supplementation represents an effective method for enhancing the antioxidant defenses of the chick post-hatch and through early stages of growth (Pappas et al., 2005; Surai, 2006; Wang et al., 2011).

1.6. Factors affecting poultry meat quality

1.6.1. Harvest and the conversion of muscle to meat

At the time of harvest, the bird expires within few minutes of exsanguination, however, its muscle cells continue to metabolize and react to their environment for several hours after the cessation of death (Sams, 1999). Cellular oxygen is quickly depleted; therefore the cell depends exclusively on anaerobic metabolism for the production of adenosine triphosphate (ATP) as its energy source (Sams, 1999; Sams 2001). Lactic acid is produced as an end product of anaerobic metabolism, and the buildup of lactic acid results in a drop in muscle pH from about 7 (at the time of death) to about 5.7 (physiological pH of poultry meat; Sams, 2001). The consumption of ATP continues to decline after death (due to its availability) and the onset of rigor mortis develops due to insufficient ATP to dissociate actin and myosin, consequently forming the complex actinomyosin (Lawrie, 1999; Sams, 1999). Within 1.5 to 2.5 hours of death,

the broiler carcasses are rapidly chilled to below 4 ºC via water immersion chilling or air chilling (Sams, 1999; Savell et al., 2005). This serves to reduce microbial growth as well as increase carcass firmness and the juiciness of the meat. The time delay between death and chilling is important as rapid chilling of the carcass prior to the onset of rigor mortis can result in a toughening of the meat. This process is referred to as "cold shortening," and is caused by contractions which shorten the sarcomere, thus increasing the toughness of the meat (Bilgili et al., 1989; Sams, 1999; Savell et al., 2005). Following a 1 to 3 hour chill, the carcasses can then be deboned into the respective cuts of meat and then packaged for sale (Sams, 2001).

1.6.2. Pre-slaughter factors affecting meat quality

In practice, feed and water is withdrawn from broilers for a period of 8 to 12 hours before processing to ensure that the bird's digestive tract is completely empty to prevent fecal contamination of the carcass during processing (Northcutt, 2001). This is a requirement in the poultry industry because the United States Department of Agriculture (USDA) has a strict zero tolerance policy of fecal contamination of harvested broiler carcasses (USDA, 1998). It is important to stay within this time frame of feed withdrawal as longer durations may result in decreased carcass yields (Northcutt, 2001).

Other pre-slaughter factors that can affect poultry meat quality and carcass yields include heat and transportation stress. Exposure to high temperatures and extreme stress during transportation prior to slaughter have been shown to reduce the WHC of poultry meat while also increasing the paleness of the meat color (McKee and Sams, 1997; Northcutt, 2001; Alvarado and Sams, 2002). These stressors result in accelerated post mortem glycolysis which further decreases the pH of the meat well below normal (Sams, 1999). The rate at which pH declines is a critical factor in the WHC of meat. Decreases in muscle pH reduce the net charge of the proteins within the muscle, therefore reducing the number of charges available to bind water. In addition, the combination of the rapid pH decline and high carcass temperatures post mortem can cause protein denaturation, further reducing WHC (Offer, 1991, Sams, 1999). The meat from broilers stressed in this

manner is referred to as pale, soft, and exudative (PSE) meat (Sams, 1999; Northcutt, 2001).

1.6.3. Diet composition and poultry meat quality

The composition and quantity of carcass fat is directly related to the amount and type of fat in poultry diets (Leeson, 1999). Therefore the fatty acid profile of both poultry fat and eggs can be manipulated by simple changes in the diet. This effect was confirmed by Narciso-Gaytán et al. (2010), who reported that the FA composition of chicken breast fillets reflected the FA composition of the fat source in the diet, as soybean and palm kernel oil increased polyunsaturated fatty acids (PUFA) in the breast fillets. Fish oils, which are rich in the omega-3 fatty acids eicosapentenoic acid (EPA) and decosahexaenoic acid (DHA), have been supplemented in broiler and layer diets with successful increases in these specific fatty acids in poultry meat (López-Ferrer et al., 2001; Rymer and Givens, 2005; Mirghelenj et al., 2009; Rymer and Givens, 2009).

The vitamin and mineral content of poultry meat can be affected by the relative dietary concentration and sources of these nutrients. The bioavailability of the minerals incorporated into poultry diets directly affects the level of tissue accretion, and in general, mineral proteinates and inorganic mineral sulphates are more bioavailable to the animal than are mineral oxides (Leeson and Summers, 2005). Regarding meat quality, a deficient intake of trace minerals and vitamins (such as Vit.E) have been related to higher levels of lipid oxidation which can have detrimental effects on WHC, oxidative stability, and color stability of meat products (Leeson, 1999; Surai, 2006). In contrast to vitamins, minerals and dietary fat, the composition of proteins in poultry meat are relatively unaffected by any manipulation of protein levels in the diets. Feeding poultry diets deficient in particular amino acids in turn results in decreased growth and other performance traits without any change in the resulting amino acid profile of the proteins (Leeson, 1999).

Rancidity in meat is caused by lipid peroxidation (or lipid oxidation) and is considered one of the most important factors contributing to the deterioration of the quality characteristics of meat (Fellenberg and Speisky, 2006). This especially becomes

an issue when high levels of PUFA's are included in the diets as this can increase the degree of unsaturation in the membrane lipids in muscle, which could reduce the overall oxidative stability of the meat (Morrissey et al., 1998; Leeson, 1999; Fellenberg and Speisky, 2006; Surai, 2006). Processing and storage of the fat sources used in poultry diets is also extremely important, as unstablized fat will eventually oxidize yielding undesirable odors and flavors in meat products due to the accumulation of aldehydes, ketones, and hydroxyl acids (Leeson, 1999). With this in mind, the use of antioxidants such as Vit.E and Se in poultry diets presents a viable solution for controlling lipid oxidation in meat products, thus improving shelf life and meat quality characteristics.

Improvements in the avian antioxidant system have been shown to have beneficial effects on WHC (reduced drip loss), oxidative stability and color stability (Surai, 2006). Several investigators have reported reductions in breast fillet drip loss when broilers received organic Se in the diet (Edens, 1996; Choct et al., 2004; Upton et al., 2008; Jiang et al., 2009; Perić et al., 2009). Edens (1996) suggested that synergism between Vit.E and Se-dependent GSH-Px may reduce oxidative damage that otherwise could compromise the integrity of cellular membranes allowing for uncontrolled movement of water between the various compartments (i.e., increasing drip loss).

Overall color stability of fresh meat declines over time under storage conditions, because myoglobin is oxidized to metmyoglobin due to oxygen exposure during storage. It has been suggested that the presence of antioxidant compounds may improve the color stability of raw meat by retarding the formation of metmyoglobin (Monahan et al., 1994; Fernández -Lopez, 2005). For example, Kim et al. (2010) reported that a* (redness) values of the breast fillets were reduced (indicator of fresh meat color) after extended refrigerated storage from broilers supplemented with 50 or 100 IU/kg Vit.E, Se yeast, or in combination when compared with the unsupplemented control.

Improvements in the oxidative stability of poultry meat can delay the onset of rancidity, thus extending its shelf life. The determination of thiobarbituric acid reactive species (TBARS) levels in meat products has become a widely accepted method for

quantifying the production of reactive oxygen species and thus, assessing the oxidative stability of meat (Gutteridge, 1984; Schmedes and Holmer, 1989). Previous studies have reported improvements in the oxidative stability of poultry meat when diets were supplemented with Se (Mikuski et al., 2009; Chekani-Azar et al., 2010), Vit.E (Maraschiello et al., 1999; Cotzee and Hoffman, 2001; Guo et al., 2001; Ryu et al., 2005; Sheldon et al., 1997; Rebolé et al., 2006; Narciso-Gaytan et al., 2010; Voljč et al., 2011), or in combination (Ryu et al., 2005; Kim et al., 2010). Chekani-Azar et al. (2010) reported that Se yeast was more effective than SS at reducing TBARS values in breast fillets through 8d of storage. The authors indicated that tissue Vit.E levels increased due to Se yeast supplementation and it is likely that the sparing effect of Se on Vit.E may have contributed to the improvement in oxidative stability of the breast fillets. Coetzee and Hoffman (2001) reported that including up to 160 mg/kg Vit.E in broiler diets resulted in d 8 TBARS that were nearly identical to that of the d 0 values for the control group (no Vit.E supplementation).

1.6.4. Marination

Marination is a widespread technique that is utilized in the poultry industry to enhance the flavor, quality, and functional characteristics of poultry meat (Smith and Young. 2007). Up to 50% of the total raw poultry meat produced is marinated, and market forms of marinated poultry include whole birds, cut-up parts, boneless meat, and ground meat (Smith and Acton, 2001; Smith and Young. 2007). The primary ingredients found in a typical marinade include salt, phosphates, and water, where the salt and phosphates act to improve WHC and tenderness of meat (Allen et al., 1998; Barbut et al., 1988; Xiong and Kupski, 1999a,b; Lyon et al., 2005; Saha et al., 2009; Gorsuch and Alvarado, 2010). The salt and phosphates included in typical marinade solutions work synergistically to increase WHC by increasing meat pH and changing ionic strength, leading to expansion or “swelling” of the myofibril lattices and increased capacity to bind water (Bendall, 1954; Xiong and Kupski, 1999a,b; Alvarado and McKee, 2007). Increasing the WHC of poultry meat should translate to decreased cooking losses, therefore enhancing the sense of juiciness to the consumer (Smith and Acton, 2001).

Previous research has also suggested that the sodium and phosphate ions in marinade solutions can improve the tenderness of poultry meat (Paladino and Ball, 1979; Goodwin and Maness, 1984; Saha et al., 2009; Petracci et al., 2012). Marination has also been shown to minimize the detrimental meat quality attributes of PSE poultry meat, as increasing meat pH improved WHC and tenderness of pale breast fillets (Gorsuch and Alvarado, 2010). Marination brines can be applied through a variety of techniques including soaking, injection, or vacuum tumbling, the latter being the most popular method used for boneless skinless breasts (Smith and Acton, 2001; Smith and Young, 2007).

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CHAPTER 2. Effects of dietary selenium yeast and vitamin E supplementation to developing broiler breeder pullets on body weight, flock uniformity, tissue antioxidant concentration, and subsequent hen production performance

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2.1. Introduction

Pullet body weight (BW) and flock uniformity at the time of sexual maturation are the two most important criteria in any broiler breeder development program. Broiler breeder pullets are reared using feed restriction programs to carefully control pullet growth to ensure optimum BW at the time of sexual maturity (Gous et al., 2000). Determining flock uniformity provides an estimate for the BW variability at a particular age. In general, the "uniform" flock is characterized by a minimum of 80% of the pullets with a BW within ±15% of the flock average (Cobb-Vantress, 2008). In general, higher BW uniformity during the developmental period can be reflected in improved hen day production during the laying cycle (Hudson et al., 2001). Decreased uniformity or considerable deviations from the target BW at the time of photostimulation may reduce the long term production performance of a broiler breeder flock (Hudson et al., 2001; Zuidhof et al., 2007).

Vitamin E and Se are two key components of the avian antioxidant system that together, reduce oxidative damage by detoxifying damaging free radicals in the avian body (Surai, 2006). Maternal nutrition in avian species is of critical importance because all of the required nutrients for embryo development are pre-deposited by the hen in the egg, which may ultimately affect the health and performance of the chick posthatch (Pappas et al., 2008). The process of hatching is a time of oxidative stress for the chick, therefore optimal Se and Vit.E status will likely be reflected in the chick's defense

against oxidative damage during this time. Previous studies have indicated that inclusion of Se in maternal diets has been shown to increase tissue Se concentrations and GSH-Px activity of the progeny posthatch (Surai et al., 1999; Pappas et al., 2005). The influence of pullet development diets on the nutrient status of the breeding hen at the onset of lay has not been evaluated. Furthermore, information in the literature is lacking regarding the effects of Se and Vit.E supplementation in the diets of developing breeder pullets on tissue antioxidant status and subsequent egg production performance. Therefore, the objective of this experiment was to evaluate the effects of dietary supplementation of Se yeast and Vit.E in developing broiler breeder pullet diets on BW, BW uniformity, tissue antioxidant concentrations, and hen egg production through 40 weeks of production.

2.2 Materials and methods

Experiments were conducted under protocols approved by the University of Kentucky Institutional Animal Care and Use Committee.

2.2.1. Animals and Treatments

A total of 640 Cobb500^{TM} broiler breeder pullets were allotted to 4 dietary treatments in a randomized complete block design utilizing a 2 x 2 factorial arrangement. Birds were placed in 2.44 x 1.83 m floor pens with dry wood shavings as bedding providing 4.47 m^2 of floor space with 5 blocks of pullets (32 pullets/pen, 20 pens total) per dietary treatment. Dietary treatments consisted of a corn-soybean meal basal diet without added Se or Vit.E, the basal diet supplemented with 0.3 mg/kg Se as selenium yeast (Sel-Plex®, Alltech, Inc., Nicholasville, KY), or 30 IU/kg Vit.E per kg as all-rac-α-tocopheryl acetate (Rovimix® E50-Adsorbate, DSM Nutritional Products, Parsippany, NJ), or both (Table 2.1). Diets were formulated to meet or exceed the National Research Council (1994) estimated requirements for metabolizable energy, crude protein, vitamins and minerals other than Se and Vit.E. The starter diet was fed from 0 to 8 weeks of age, the grower diet from 9 to 21 weeks of age, and the breeder diet from 22 to 61 weeks of age.

Pullets received light according to the following schedule: 22h of light through 2 weeks of age, 8h of light from 3 through 21 weeks of age, and 16h of light after 21 weeks of age. Birds were provided with *ad libitum* access to water and were limit-fed daily in trough feeders (243.8 x 10.2 cm) according to the Cobb Breeder Management Guide for fast feathering birds (Cobb-Vantress, 2008).

2.2.2. Performance measurements

Pullets were weighed weekly in groups from 2 to 21 weeks of age. Estimates of flock uniformity were obtained weekly by randomly selecting and weighing 10 pullets/pen (50 pullets/treatment) from 2 to 21 weeks of age. Flock uniformity was determined on a pen basis and was the percentage of pullets that had a BW within ±15% of the flock average at a given age (running average of 3 week intervals). Egg production data was collected for determination of hen day production (HDP%) through 40 weeks of production (21 through 61 weeks of age).

2.2.3. Sample collection and tissue analysis

At 14, 18, 21, and 26 weeks of age, one pullet per pen as randomly selected and killed by asphyxiation using argon gas followed by cervical dislocation for subsequent collection of breast, liver and pancreas for analysis of Se content. At 26 weeks of age, an additional bird was killed and breast and liver samples were collected for analysis of total tissue α-tocopherol content. Collected samples were placed on ice and frozen at -80 °C until analyzed. Frozen samples destined for Se analysis were weighed and then placed in a freeze dryer (Labconco FreeZone Plus 6, Labconco Corp., Kansas City, MO) for 4 days to a constant weight by lyophilization. Upon removal from the freeze dryer, all samples were weighed once again for determination of dry matter content and placed into plastic bags. Samples then were finely ground using a mortar and pestle prior to Se analysis.

Tissue Se concentration (dry matter basis) was determined using an inductively coupled plasma mass spectrometer (ICP-MS; Agilent 7500cx, Santa Clara, CA, USA). Prior to ICP-MS analysis, samples were digested in 10 mL nitric acid and 1 mL 30% concentrated hydrogen peroxide in sealed Teflon bombs using a microwave digestion

system (MARSXpress, CEM Corporation, Matthews, NC) and appropriately diluted. A commercial standard, NIST SRM 1577c (bovine liver), was used as a reference material to assure accuracy of the obtained results (See Appendix 1 for full description). Week 26 breast and liver samples were analyzed for total tissue α-tocopherol concentrations using the procedures described by Liu et al. (1996).

2.2.4. Statistical analysis

Data for this experiment were analyzed using the Proc GLM function of SAS® (SAS, Cary, NC) and the main effects of dietary Se, Vit.E, and the interaction were determined. The replicate pen of broiler breeder hens served as the experimental unit. Fisher's least significant difference test was used to determine significance between means with a significance set at $P \leq 0.05$.

2.3. Results

Through 21 weeks of age, pullet BW did not differ between dietary treatments (Table 2.2). However, there were significant main effects of Se to reduce pullet BW at 3 ($P \leq 0.05$) and 6 ($P < 0.05$) weeks of age. In general, mean BW for all treatment groups were above the specified target BW through 21 weeks of age. Figure 2.1 illustrates the average weight above the weekly specified target BW for the developing pullets. There were significant main effects of Se to reduce the deviation from the specified target weight at 3 ($P \leq 0.05$) and 6 ($P < 0.05$) weeks of age. Flock uniformity decreased below 80% for all treatments after 12 weeks of age (Figure 2.2) There was a reduction in uniformity for the Vit.E and Vit.E + Se yeast treatments at week 9 compared with the control ($P < 0.05$), with a significant main effect of Vit.E ($P < 0.05$) to reduce uniformity. Overall flock uniformity declined as pullets approached the age of photostimulation.

The tissue Se concentration data is detailed in Table 2.3. Selenium yeast supplementation significantly increased ($P < 0.01$) liver, pancreas, and breast Se concentrations at week 14, 18, 21, and 26 (main effect, Se: $P < 0.01$). As a result of the onset of egg production, a marked decrease in liver Se levels was observed between 21 and 26 weeks of age for all treatment groups. The Vit.E concentrations of breast and liver

tissues collected at week 26 are displayed in Table 2.4. There were no differences between dietary treatments on the Vit.E concentrations of breast or liver tissues. However, there was a significant main effect of Se*Vit.E to increase breast Vit.E concentrations ($P < 0.05$).

The results for HDP% through 40 weeks of production is illustrated in Figure 2.3. Peak production appeared to occur for all treatment groups near 10 to 12 weeks of production (31 to 33 weeks of age). Overall, egg production was numerically lower for the Vit.E treatment group compared to the other treatments. At week 12, HDP% was significantly lower for hens fed Vit.E ($P < 0.05$) compared with those fed the control, Se yeast alone, or Vit.E + Se yeast. In addition, there were significant main effects of Se to increase HDP% at weeks 10, 12, 32, and 36 ($P < 0.05$).

2.4. Discussion

Pullet BW and flock uniformity at the time of photostimulation plays a key role in determining to reproductive efficiency and success of a broiler breeder flock. A reduction in flock uniformity is associated with variations in the degree of sexual maturity, where heavier pullets achieve sexual maturity earlier than do lighter pullets (Yuan et al., 1994). The quality of the settable eggs can be affected by flock uniformity, as heavy pullets tend to produce fewer eggs with a higher incidence of double yolked eggs, while lighter pullets tend to produce eggs that vary considerably in size (Petitte et al., 1982; Abbas et al., 2010). In addition, a negative relationship exists between pullet BW at the time of sexual maturity and subsequent egg production due to asynchrony in the ovulation process (Yu et al., 1992; Renema and Robinson, 2004; Zuidhof et al., 2007). Overall, broiler breeder flocks that have higher uniformity generally reach peak egg production earlier, with higher peaks than flocks with poor uniformity (North, 1980).

Selenium yeast and Vit.E supplementation in developing broiler breeder pullet diets did not affect overall pullet BW prior to sexual maturity. It is unclear as to why there were significant main effects of Se to reduce pullet BW at weeks 3 and 6 weeks. These effects could be interpreted as beneficial as the average pullet BW throughout the

entire 21 week developmental period was well above the target BW (Cobb-Vantress, 2008). However, there was no distinct pattern regarding weight deviations above the targeted weight among treatments beyond 6 weeks of age. Achieving the target BW at the time of sexual maturity is very important because even an overweight flock with a very high degree of uniformity may still have reduced reproductive performance due heavier BW (Hudson et al., 2001).

In addition, overall flock uniformity declined through 21 weeks of age, and all treatments dropped below 80% uniformity after 12 weeks of age. It is unclear why the control treatment displayed greater uniformity at week 9 when compared with the Vit.E or Vit.E + Se yeast treatments. Numerically, the Vit.E treatment appeared to consistently have lower uniformity throughout the 21 week developmental period compared to the other treatment groups. Information in the literature is lacking regarding the effects of Se or Vit.E on developing pullet BW uniformity. The pullets were fed on a daily basis, therefore it is possible that the heavier, dominant birds may have consumed more than their allotted amount of feed and the lighter birds may have consumed less feed, despite the fact that all birds could feed at the same time (North, 1980). It is well known feeding programs such as skip-a-day feeding has been shown to improve BW uniformity because the relatively larger meal administered every other day provides more time and opportunity for the smaller, more timid birds, to consume feed (North, 1980). Therefore, it is possible that the feeding program utilized in this experiment may have negatively affected BW uniformity. Nonetheless, overall BW uniformity at the time of photostimulation was below the minimum qualifications (80%) for a “uniform” flock (Cobb-Vantress, 2008).

There was a significant main effect of Se supplementation to increase liver, pancreas, and breast Se concentrations through 26 weeks of age in pullets fed diets containing Se yeast (Se yeast and Vit.E + Se yeast) compared to those fed the control diet or Vit.E alone. Information in the literature is limited regarding Se supplementation on the tissue Se status of developing broiler breeder pullets. These results are in agreement with previous studies evaluating Se yeast supplementation on tissue levels of broilers

(Pappas et al., 2012), egg-laying hens (Pan et al. 2007; Pavlović et al., 2009; Čobanová et al., 2010), and broiler breeder hens (Leeson et al., 2008; Wang et al., 2011). Upon the onset of egg production (26 weeks of age), liver Se concentrations were reduced by 45 and 60% for the control and Vit.E treatments, respectively, compared with values at the time of photostimulation (21 weeks of age). Liver Se concentrations of birds fed Se yeast alone or Vit.E + Se yeast were only reduced by 33 and 38%, respectively. The weeks preceding the onset of egg production represents a period of intense development of the female reproductive system (Yu and Marquardt, 1974). A considerable reduction in liver Se reserves from 21 to 26 weeks of age suggests that Se may be mobilized from the liver in response to a possible demand from the developing reproductive tract and follicles. Nonetheless, broiler breeder pullets fed dietary Se yeast had higher tissue Se concentrations at the beginning of the laying cycle than pullets fed diets that did not contain supplemental Se yeast.

It has been reported that maternal supplementation of Se yeast can improve embryo viability, hatchability, and growth of the subsequent progeny (Pappas et al., 2005; Pappas et al., 2006; Pappas et al., 2008; Wu et al., 2010; Wang et al., 2011). Indeed, dietary inclusion of Se in maternal diets has been shown to increase tissue Se concentrations and GSH-Px activity of the progeny, thus improving the chick's antioxidant status during the stressful process of hatching (Surai et al., 1999; Surai, 2000; Pappas et al., 2005; Surai, 2006; Wang et al., 2011).

In the laying phase of this experiment, all hens reached peak egg production between 10 and 12 weeks of production and at levels comparable to level specified in the Cobb Breeder Management Guide (Cobb-Vantress, 2008). At 12 weeks of production, HDP% was reduced for the Vit.E treatment group ($P < 0.05$) compared with the control, Se yeast alone, and Vit.E + Se yeast treatment. Hens receiving Vit.E appeared to have numerically lower HDP% than compared with the other treatment groups throughout the 40 week production period. At the time of photostimulation, hens in the Vit.E alone treatment group displayed higher BW and lower uniformity than the other treatment groups. In combination, these effects may have resulted in the reduced HDP%

performance by the Vit.E group. The overall HDP% of hens fed dietary Se yeast were numerically higher than that of the Vit.E treatment, which is supported by significant main effects of Se at weeks 10, 12, 32, and 36. Research is lacking on the effects of dietary Vit.E and Se supplementation on the production performance of broiler breeders. From these results, it appears that dietary treatment does not appear to improve nor reduce production performance. These results are in agreement with Wang et al. (2011), who reported that dietary supplementation of 0.3 ppm Se yeast did not affect egg production of broiler breeder hens.

In the post-peak production phase, HDP% for all treatments was lower than the targeted level of egg production specified in the Cobb Breeder Management Guide (Cobb-Vantress, 2008). Since there was no overall distinct pattern in the responses between dietary treatments, it is possible that breeder management may have affected long term production performance. It has been reported in the literature that highly uniform flocks yield optimal production performance (Hudson et al., 2001; Zuidhof et al., 2007). Since heavier pullets may achieve sexual maturity sooner than lighter pullets, the length of the lay period is also shorten resulting in a decrease in total egg production (Yuan et al., 1994; Hudson et al., 2001; Zuidhof et al., 2007). In this experiment, hen BW at the time of photostimulation was at least 180 g above the target weight, which qualified the pullets as overweight. In addition, overall BW uniformity at 21 weeks was below the specified “80%” minimum for a uniform flock, indicating relatively poor flock uniformity. Therefore, the combination of overweight birds and poor BW uniformity at the time of sexual maturation may have increased the variation of egg production on a bird to bird basis within each pen due to the wider range in the size of these hens.

In summary, Se yeast and Vit.E did not appear to impact broiler breeder pullet BW or uniformity during the developmental phase. This experiment highlights the importance of maintaining optimal pullet BW and uniformity prior to photostimulation as production performance was below the genetic potential in the post-peak phase. Inclusion of Se, as Se yeast, in the diets of developing broiler breeder pullets resulted in greater Se accumulation of Se in liver, pancreas, and breast tissues than when Se yeast was not

provided in the diet. Improvements in the overall Se status of breeder pullets in the early stages may help maintain adequate tissue Se concentrations during egg production.

2.5. Tables and figures

Table 2.1. Composition of broiler breeder basal diets (as-fed basis)

	% of diet		
Ingredients	Starter	Grower	Breeder
Ground corn	60.00	69.13	70.15
Soybean meal (48% CP)	25.00	16.00	20.50
Wheat middling's	10.95	11.00	–
Ground oyster shells	–	–	3.00
Ground limestone	1.20	1.20	4.00
Dicalcium phosphate	1.70	1.70	1.30
Salt	0.45	0.35	0.43
Vitamin mix[1]	0.25	0.25	0.25
Trace mineral mix[2]	0.25	0.25	0.25
DL-methionine	0.20	0.12	0.12
Total	100.00	100.00	100.00
Calculated nutrient composition[3]			
ME, kcal/kg	2,839	2,926	2,850
Crude protein, %	18.98	15.36	15.97
Lysine, %	0.99	0.74	0.80
TSAA, %[4]	0.82	0.65	0.66
Threonine, %	0.72	0.57	0.61
Tryptophan, %	0.23	0.18	0.19
Calcium, %	0.92	0.90	1.89
Phosphorus (available), %	0.46	0.44	0.35
Selenium, ppm	0.13	0.12	0.04
Vitamin E, IU/kg	27.50	30.13	24.07

[1] Supplied per kilogram of diet: Vitamin A, 8,000 IU; Vitamin D_3, 3,000 IU; Vitamin K, 3 mg; Thiamin, 2 mg; Riboflavin, 10 mg; Pantothenic acid, 12 mg; Niacin, 40 mg; Pyridoxine, 4 mg; Biotin, 0.2 mg; Folic acid, 0.75 mg; Vitamin B-12, 0.015 mg; Choline, 500 mg; Ethoxyquin, 125 mg.
[2] Supplied per kilogram of diet: Copper, 24 mg; Iodine, 0.89 mg; Manganese, 281.35 mg; Zinc, 83.33 mg; Cobalt, 20.19 mg.
[3] Calculated based on the NRC (1994) estimated values.
[4] TSAA: Total sulfur amino acids, methionine + cysteine.

Table 2.2. Average body weight (g) of developing broiler breeder pullets through 21 weeks of age.[1]

	Diet					*P*-values		
Age, wk	Control	Se yeast	Vit.E	Vit.E + Se yeast	SEM	Se	Vit.E	Se*Vit.E
3	507	485	506	490	8.9	0.05	0.83	0.74
6	963	932	948	928	9.5	0.02	0.33	0.58
9	1,253	1,235	1,253	1,248	11.1	0.30	0.56	0.55
12	1,457	1,454	1,455	1,466	9.4	0.67	0.62	0.47
15	1,710	1,737	1,733	1,736	15.3	0.35	0.48	0.42
18	2,092	2,110	2,120	2,125	19.4	0.54	0.28	0.75
21	2,599	2,598	2,638	2,648	21.9	0.84	0.06	0.79

[1] Values represent the mean of 5 pens/treatment.

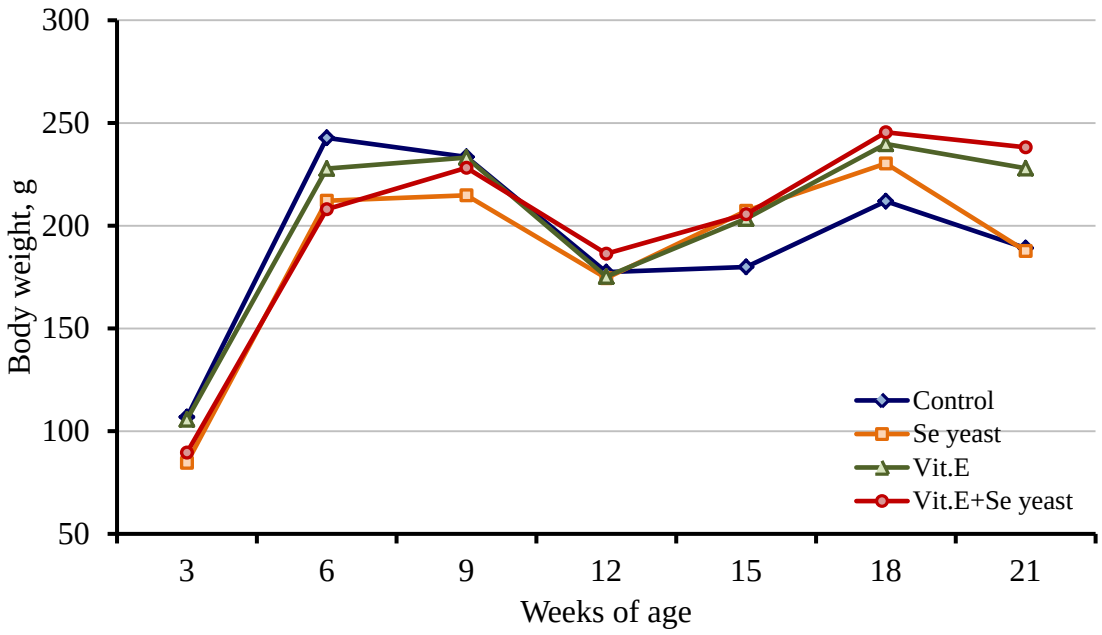

Figure 2.1. Average weight (g) above the weekly specified target BW for the developing broiler breeder pullets through 21 weeks of age. Displayed values represent the mean of 5 pens/treatment. There were significant main effects of Se to reduce the deviation from the specified target weight at 3 ($P \leq 0.05$) and 6 ($P < 0.05$) weeks of age.

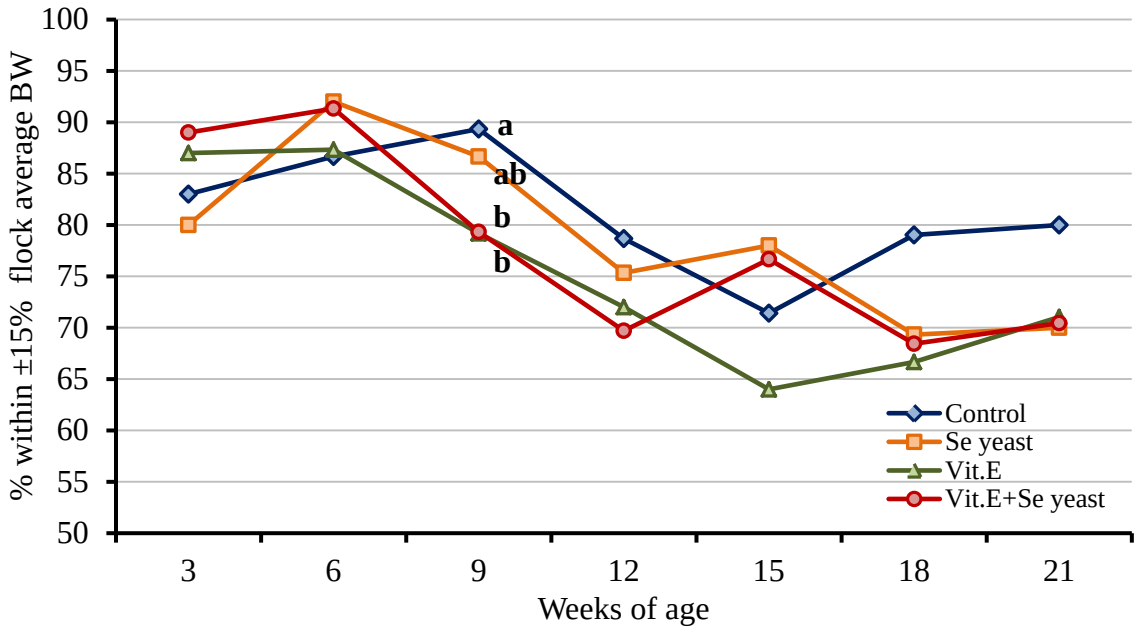

Figure 2.2. Pullet body weight (BW) uniformity (% within ± 15% of flock average BW) through 21 weeks of age. Displayed values represent the mean of 5 pens/treatment. At 9 weeks of age, pullets receiving the Vit.E alone and Vit.E + Se yeast treatments displayed lower BW uniformity ($P < 0.05$) than hens on the control treatment. There was a significant main effect of Vit.E to decrease BW uniformity at 9 weeks of age ($P < 0.01$).

Table 2.3. Selenium concentration of liver, pancreas, and breast tissues (DM basis).[1]

	Diet					*P*-value		
Item	Control	Se yeast	Vit.E	Vit.E + Se yeast	SEM	Se	Vit.E	Se*Vit.E
Liver Se, ppm								
Wk 14	1.62^{B}	3.82^{A}	1.71^{B}	3.70^{A}	0.17	<0.01	0.90	0.55
Wk 18	2.08^{B}	3.57^{A}	2.06^{B}	3.47^{A}	0.11	<0.01	0.59	0.74
Wk 21	1.99^{B}	3.62^{A}	2.03^{B}	3.63^{A}	0.13	<0.01	0.87	0.92
Wk 26	1.10^{B}	2.41^{A}	0.81^{B}	2.26^{A}	0.16	<0.01	0.19	0.69
Pancreas Se, ppm								
Wk 14	0.95^{B}	2.27^{A}	0.87^{B}	2.12^{A}	0.09	<0.01	0.23	0.70
Wk 18	0.84^{C}	2.02^{A}	0.82^{C}	1.81^{B}	0.07	<0.01	0.10	0.18
Wk 21	0.81^{B}	2.01^{A}	0.77^{B}	2.20^{A}	0.07	<0.01	0.29	0.11
Wk 26	0.59^{B}	1.84^{A}	0.66^{B}	2.13^{A}	0.14	<0.01	0.22	0.44
Breast Se, ppm								
Wk 14	0.35^{B}	1.25^{A}	0.35^{B}	1.27^{A}	0.03	<0.01	0.83	0.70
Wk 18	0.44^{B}	1.30^{A}	0.42^{B}	1.26^{A}	0.03	<0.01	0.40	0.80
Wk 21	0.44^{C}	1.36^{B}	0.45^{C}	1.46^{A}	0.03	<0.01	0.06	0.10
Wk 26	0.38^{B}	1.40^{A}	0.40^{B}	1.28^{A}	0.05	<0.01	0.34	0.22

[A-B] Means within the same row with different superscript letters are significantly different ($P < 0.01$).
[1] Values represent the average of 5 pens/treatment (n = 1 sample/pen).

Table 2.4. α-tocopherol concentrations (μg/g) of breast and liver samples at 26 weeks of age.[1]

	Diet					*P*-values		
Item	Control	Se yeast	Vit.E	Vit.E + Se yeast	SEM	Se	Vit.E	Se*Vit.E
Breast	0.43	0.38	0.34	0.50	0.04	0.15	0.66	0.02
Liver	3.77	3.63	3.75	4.03	0.46	0.88	0.69	0.66

[1] Values represent the average of 5 pens/treatment (n = 1 sample/pen).

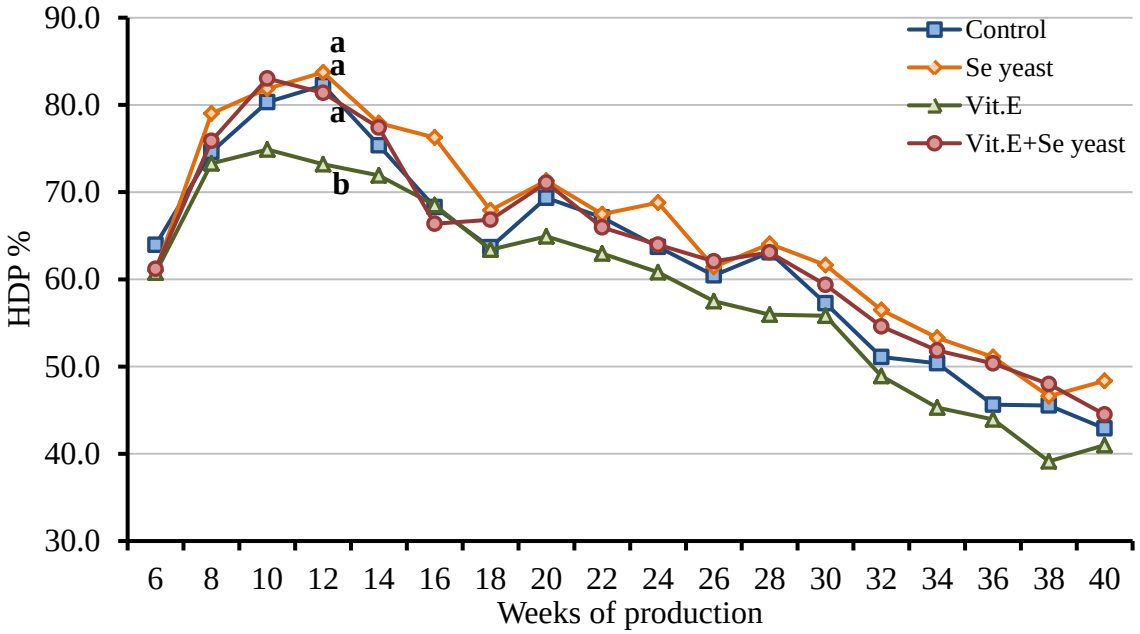

Figure 2.3. Average hen daily production (HDP) percentages through 40 weeks of production. Displayed values represent the mean of 5 pens/treatment. At 12 weeks of production, hens fed Vit.E alone displayed lower HDP% ($P < 0.05$) that the control, Se yeast alone, and Vit.E + Se yeast treatments. There were significant main effects of Se to increase HDP% at weeks 10, 12, 32, and 36 ($P < 0.05$).

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CHAPTER 3. Effects of selenium yeast and vitamin E supplementation in maternal and progeny diets on tissue antioxidant nutrients and growth performance of broilers

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3.1. Introduction

Vitamin E (Vit.E) and selenium (Se) are the two main components of the avian antioxidant system that work in synchrony to reduce oxidative damage by detoxifying free radicals in the avian body (Surai, 2006). Selenium plays an integral role in this system as a component of the antioxidant enzymes glutathione peroxidase (GSH-Px) and thioredoxin reductase (Surai, 2006). The Se-dependent GSH-Px enzymes are responsible for preventing oxidative damage through the removal of lipid- and hydro-peroxides which are naturally formed during metabolism (Jaeschke, 1995). Selenium occurs naturally in feedstuffs, predominantly as selenomethionine (SeMet; Combs and Combs, 1984); however, it is common practice to supplement poultry diets with inorganic (sodium selenite) or organic (Se yeast) Se sources to avoid deficiency symptoms (Surai, 2006).

Antioxidant protection is of critical importance to the young chick during hatching and the first few days post-hatch since these are periods of high oxidative stress. At this time, the combination of increased oxidative metabolism at hatch with the transition to pulmonary respiration can lead to the production of free radicals (Surai et al., 1999). In addition, chick tissues contain relatively high levels of polyunsaturated fatty acids, which are extremely susceptible to peroxidative damage by free radicals (Surai et al., 1996). It is well established that the most effective way of increasing the antioxidant status of the developing embryo is through inclusion of Se and Vit.E in the maternal diets (Surai, 2000; Paton et al., 2002; Pappas et al., 2005; Surai et al., 2006). Surai (2000)

noted that the maternal carryover of Se and Vit.E to the progeny was also accompanied by significant increases in GSH-Px activity in the liver and muscle of the progeny. Indeed, previous research has indicated that there is a highly significant, positive correlation between Se concentration and GSH-Px activity in poultry tissues (Surai et al., 1999; Surai, 2000; Pappas et al., 2005).

The benefits of maternal derived antioxidants in the developing embryo do not terminate at hatch, but instead, are retained to varying extents in the progeny tissues throughout the early stages of growth (Surai, 2000; Pappas et al., 2005; Surai et al., 2006). Surai (2000) indicated that Se yeast supplementation in the maternal diets resulted in elevated liver GSH-Px activity of the progeny for at least 5d post-hatch. Pappas et al. (2005) reported that Se yeast supplementation in broiler breeder hen diets increased progeny tissue Se concentrations for 3-4 weeks after hatching. In Japanese quail, Surai et al. (2006) demonstrated persistent effects of maternal Se yeast supplementation to increase tissue Se of the progeny through 2 weeks post-hatch. Information is lacking however, on the effects of maternal Se or Vit.E supplementation on the tissue Vit.E concentrations of the progeny post-hatch. Interestingly, previous research has not evaluated the effects of both Se and Vit.E supplementation in the maternal diets on the tissue antioxidant status of the progeny. Therefore, two experiments were conducted to evaluate the effects of supplementing maternal and progeny diets with Se yeast and Vit.E on the maternal transfer of Se and Vit.E to the progeny, as well as the effectiveness of maternal or progeny supplementation on tissue antioxidant status and their subsequent growth performance.

3.2. Materials and methods

Experiments were conducted under protocols approved by the University of Kentucky Institutional Animal Care and Use Committee.

3.2.1. Animals and Treatments

Breeders. A total of 640 Cobb500^{TM} broiler breeder pullets and 80 roosters were randomly allotted to 4 dietary treatments in a 2 x 2 factorial design. Birds were

placed in 2.44 x 1.83 m floor pens with dry wood shavings as bedding providing 4.47 m^2 of floor space with 5 blocks of pullets (32 pullets/pen, 20 pens total) and 1 block of roosters (20 roosters/pen, 4 pens total) per dietary treatment. Diets consisted of a corn-soybean meal basal diet without added Se or Vit.E, the basal diet supplemented with 0.3 mg/kg Se as selenium yeast (Sel-Plex®, Alltech, Inc., Nicholasville, KY), or 30 IU/kg Vit.E per kg as all-rac-α-tocopheryl acetate (Rovimix® E50-Adsorbate, DSM Nutritional Products, Parsippany, NJ), or both. Pullets and roosters received light according to the following schedule: 22h of light through 2 weeks of age, 8h of light from 3 through 22 weeks of age, and 16h of light at 22 weeks of age. Birds were provided with *ad libitum* access to water and were limit-fed daily in trough feeders (243.8 x 10.2 cm) according to the Cobb Breeder Management Guide for fast feathering birds (Cobb-Vantress, 2008). At the time of light stimulation, males were added and the number of hens per pen was reduced to a ratio of 13 females to 1 male, which was maintained throughout the entire laying cycle for the 5 blocks of hens.

Eggs were collected from broiler breeder hens at 44 and 67 weeks of age for Exp. 1 and 2, respectively. The collected eggs were allotted within a Natureform NOM 45 incubator (NatureForm Hatchery Systems, Jacksonville, FL) according to maternal diet and incubated at 37.5 °C and 55% RH. Eggs were transferred to the same model NatureForm hatcher at 18d and held at 36.5 °C and 65% RH. At hatch, chicks were placed in 1.22 x 1.83 m floor pens with dry wood shavings as bedding providing 2.23 m^2 of floor space and were allotted to 4 dietary treatments in a randomized complete block design, with block corresponding to the pen location within the broiler facility. Chicks were allocated within each block to account for all maternal and chick diet interactions (16 pens/block). Chicks were provided with *ad libitum* access to feed and water and feed was administered in tube feeders.

Chicks. Both experiments used Cobb500™ broilers that were placed as straight run. Dietary treatments consisted of a corn-soybean meal basal diet without added

Se or Vit.E (Table 3.1), the basal diet supplemented with 0.3 mg/kg Se yeast, or 30 IU/kg Vit.E, or both. Diets were formulated to meet or exceed the NRC (1994) estimated requirements for metabolizable energy, crude protein, vitamins and minerals other than Se and Vit.E. In Experiment 1, 768 1-d old chicks were randomly allotted to 48 pens of 16 birds per pen with 3 blocks. In Experiment 2, 448 1-d old chicks were randomly allotted to 32 pens of 14 birds per pen with 2 blocks.

3.2.2. Performance measurements

Chicks were weighed at the time of placement (0d of age) and then weekly through 49 weeks of age on a pen basis and average daily gain (ADG) was calculated. Feed intake data was also collected on a weekly basis for calculation of average daily feed intake (ADFI) and feed conversion ratio (ADG/ADFI) as determined by gain:feed (G:F).

3.2.3. Sample collection and chemical analysis

Liver and breast samples were collected from chicks at 0, 7, and 14d of age for subsequent analysis. Sampled chicks were randomly selected from each pen and killed by asphyxiation using argon gas followed by cervical dislocation. Due to the relatively small size of the chicks at hatch, breast and liver samples from 3 chicks were composited into one sample, with a total of 3 samples collected per treatment in Exp. 1, and 2 samples collected per treatment in Exp. 2. At 7 and 14d of age, two chicks were sampled per pen; liver and breast samples were collected from the first chick for Se analysis, and liver samples were collected from the second chick for analysis of total α-tocopherol content. All collected samples were placed on ice and frozen at -80 °C until analyzed. Frozen samples destined for Se analysis were weighed and then placed in a freeze dryer (Labconco FreeZone Plus 6, Labconco Corp., Kansas City, MO) for 4 days to a constant weight by lyophilization. Upon removal from the freeze dryer, all samples were weighed once again for determination of dry matter content and placed into plastic bags. Samples then were finely ground using a mortar and pestle prior to Se analysis.

Tissue selenium concentration (dry matter basis) was determined using an inductively coupled plasma mass spectrometer (ICP-MS; Agilent 7500cx, Santa Clara, CA, USA). Prior to ICP-MS analysis, samples were digested in 10 mL nitric acid and 1 mL 30% concentrated hydrogen peroxide in sealed Teflon bombs using a microwave digestion system (MARSXpress, CEM Corporation, Matthews, NC) and appropriately diluted. A commercial standard, NIST SRM 1577c (bovine liver), was used as a reference material to assure accuracy of the obtained results (See Appendix 1 for full description). In Exp. 1, progeny liver samples collected at 7 and 14d of age were analyzed for total tissue α-tocopherol concentrations using the procedures described by Liu et al. (1996).

3.2.4. Statistical analysis

These experiments utilized a split-plot arrangement, with maternal diet as the whole-plot factor and chick diet as the sub-plot factor. Statistical analysis was conducted using the GLM procedure of SAS® (SAS, Cary, NC). Replicate pen of broilers served as the experimental unit. All interactions for Se and Vit.E of the maternal (mSe, mVit.E, and mSe*mVit.E) and chick (cSe, cVit.E, and cSe*cVit.E) diets were analyzed. Fisher's least significant difference test was used to determine significance between means with a significance set at $P \leq 0.05$.

3.3. Results

3.3.1. Experiment 1

Liver and breast Se concentrations at hatch (0d of age) were significantly increased ($P < 0.05$) by Se yeast supplementation in the maternal diets (Table 3.2). This resulted in significant main effects of mSe to increase progeny liver ($P < 0.05$) and breast ($P < 0.01$) Se concentrations. Overall, maternal supplementation of Se yeast resulted in a 2.2 and 2.4 fold increase in progeny liver and breast Se concentrations, respectively, compared to the treatments that did not include Se. At 7 and 14d of age, Se yeast supplementation in the progeny diets significantly increased liver and breast Se concentrations ($P < 0.01$), with significant main effects of cSe ($P < 0.01$). There were significant main effects of mSe to increase liver ($P < 0.01$) and breast ($P < 0.05$) Se concentrations through 7d of age, however these effects disappeared at 14d. There were

interactive main effects for 14d liver Se concentrations for mSe*cVit.E, mSe*cSe*cVit.E, and mVit.E*mSe*cSe ($P < 0.05$); however, there were no discernible patterns to explain these effects (Appendix 2, Table A.2.1.).

There were no differences between progeny treatment diets on the liver Vit.E concentrations at 7 or 14d of age (Table 3.3). However at 7d, cVit.E supplementation tended to increase liver Vit.E concentrations and there was a significant main effect of mSe*mVit.E to increase liver Vit.E concentrations at 7d ($P < 0.05$). In addition, there was a significant interactive effect of mSe*cSe*cVit.E to increase liver Vit.E at 14d ($P < 0.01$; Appendix 3, Table A.2.2.).

Average daily gain was not different among progeny treatments during the starter (0-21d) or grower (22-49d) phases, as well as overall from 0-49d of age (Table 3.4). There was a significant main effect of cSe to increase ADG during the starter phase ($P < 0.05$). At 14d however, ADG was significantly increased for the Vit.E + Se yeast treatment ($P < 0.05$) compared with the other dietary treatments. There were significant main effects of cSe to increase ADG at 7 ($P \leq 0.05$) and 14d ($P < 0.01$). There was also a significant interactive effect of mVit.E*cSe*cVit.E to increase ADG ($P < 0.05$; Appendix 2, Table A.2.3.).

Average daily feed intake was not affected by progeny treatment diet during the starter phase (Table 3.5). During the grower phase however, ADFI was greater for the Se yeast treatments ($P < 0.05$) compared to Vit.E alone, with significant main effects of cSe to increase ADFI. On a weekly basis, ADFI was significantly higher for the Vit.E + Se yeast treatment at 28d ($P < 0.05$) compared to treatments not supplemented with Se yeast. There were significant main effects of cSe to increase ADFI at 28 ($P < 0.01$), 35 ($P < 0.01$), and 42d ($P < 0.05$), as well as overall from 0 to 49d ($P < 0.05$). A significant interaction of mVit.E*cSe existed at 14d ($P < 0.05$) for ADFI, however there was no discernible pattern explaining this effect. There was also a significant main effect interaction of mSe*cVit.E to increase ADFI at 49d ($P < 0.05$; Appendix 2, Table A.2.4.).

During the starter phase, G:F did not differ between progeny treatment diet, however, there was a significant main effect of cSe to improve G:F at 21d ($P < 0.05$; Table 3.6). Gain:feed during the grower phase and overall from 0 to 49d was reduced for the Vit.E + Se yeast treatment ($P < 0.05$) compared to the other treatment diets. On a weekly basis, G:F was significantly reduced for the Vit.E + Se yeast treatment ($P < 0.01$) compared to the other treatments at 28 and 35d. There were significant main effects of cSe to reduce G:F at 28 and 35d, and from 22-49d. There were also significant main effects of cVit.E to reduce G:F at 28 ($P < 0.01$), 35 ($P < 0.01$), and 49d ($P < 0.05$), as well as from 22-49d ($P < 0.01$) and 0-49d ($P < 0.05$; Appendix 2, Table A.2.5.).

3.3.2. Experiment 2

Liver and breast Se concentrations at hatch were significantly increased ($P < 0.01$) by Se yeast supplementation in the maternal diets (Table 3.7), resulting in significant main effects of mSe to increase tissue Se levels ($P < 0.01$). Overall, maternal supplementation of Se yeast in Exp. 2 significantly increased progeny liver and breast Se concentrations 3.4 and 2.4 fold, respectively, compared to the treatments that did not include Se. At 7 and 14d of age, Se yeast supplementation in the progeny diets significantly increased liver and breast Se concentrations ($P < 0.01$), with significant main effects of cSe ($P < 0.01$). There were significant main effects of mSe ($P < 0.01$) and mSe*mVit.E ($P < 0.01$) to increase breast Se concentrations at 7d, however these effects were not present at 14d. Multiple interactive effects existed for liver Se concentrations that were consistent with the presence of significant main effects from the maternal or chick diets. These interactive effects included mVit.E*cVitE, mVit.E*cSe, and mVit.E*mSe*cVit.E at 7d ($P < 0.05$), and mVit.E*cSe, mSe*cVit.E, and mSe*cSe*cVit.E ($P < 0.05$) at 14d ($P < 0.05$; Appendix 2, Table A.2.6.).

Average daily gain in Exp. 2 was significantly increased by Se yeast supplementation during the starter phase ($P < 0.05$), with a significant main effect of cSe to increase ADG ($P < 0.05$; Table 3.8). During the grower phase and overall through 49d of age, ADG was not different among progeny treatment diets, however there was a significant main effect of cSe to increase ADG from 0 to 49d ($P \leq 0.05$). On a weekly

basis, ADG was increased by Se yeast supplementation at 14d ($P < 0.01$), with a significant main effect of cSe to increase ADG ($P < 0.01$). In addition, there was a main effect of cSe to increase ADG at 21d ($P < 0.05$). There were multiple interactive effects for ADG including mSe*cSe at 21d ($P < 0.05$), mSe*cVitE at 35d ($P < 0.05$), and mVitE*cSe, mSe*cSe, and mVitE*mSe*cSe at 42d ($P < 0.05$; Appendix 2, Table A.2.7.). These interactive effects did not display discernible patterns explaining their effects and in general they were consistent with the presence of significant main effects from either the maternal or progeny diets.

Average daily feed intake in Exp. 2 was not different between progeny diets through 49d of age (Table 3.9). There were significant main effects for mSe to increase ADFI at 35d ($P \leq 0.05$), 49d ($P < 0.05$), and overall from 22-49d of age ($P < 0.05$), and for mVit.E to reduce ADFI at 49d ($P < 0.05$). The interative effects did not display patterns explaining the presence of these effects, but were present for mVitE*mSe*cSe at 7d ($P < 0.05$), mSe*cVitE ($P < 0.05$), mSe*cSe ($P < 0.01$), mSe*cSe*cVitE ($P < 0.05$) at 14d, mSe*cVit.E ($P < 0.05$) at 42d, and mSe*cSe, and mSe*cSe*cVit.E ($P < 0.05$) from 0-21d of age (Appendix 2, Table A.2.8.).

Gain:feed was affected by progeny diet through 49d of age (Table 3.10). During the starter phase, G:F was significantly increased by Se yeast supplementation ($P < 0.01$), with a main effect of cSe to increase G:F ($P < 0.01$). Gain:feed was not affected by progeny diet during the grower phase. Overall through 49d of age, G:F was improved by Se yeast supplementation ($P < 0.05$) compared to the control diet, with a significant main effect of cSe to improve G:F ($P < 0.01$). On a weekly basis, Se yeast supplementation resulted in improved G:F at 14d ($P < 0.01$) compared with the control or Vit.E alone treatments. At 21 and 28d, the treatment containing Se yeast alone displayed greater G:F ($P < 0.05$) than the control of Vit.E treatments. This resulted in significant main effects of cSe to improve G:F at 14, 21, and 28d ($P < 0.01$). As with ADG and ADFI, there were multiple interactive effects that were consistent with the presence of significant main effects from the maternal or chick diets. These include mVit.E*cVit.E ($P \leq 0.05$), mVit.E*cSe ($P < 0.05$), mSe*cVit.E ($P < 0.01$), and mSe*cSe ($P < 0.05$) at 14d,

mVitE*cSe*cVit.E, mSe*cVit.E, and mSe*mVit.E*cVit.E at 28d ($P < 0.05$), mSe*cSe at 42d ($P < 0.05$), mSe*cVit.E at 49d ($P < 0.05$), and overall for mSe*cVit.E ($P \leq 0.05$) from 0-21d, mSe*cVit.E from 22-49d ($P < 0.05$), and mSe*cVit.E from 0-49d of age ($P < 0.01$; Appendix 2, Table A.2.9.).

3.4. Discussion

The results from these experiments confirm that inclusion of Se yeast in broiler breeder hen diets can significantly increase the tissue Se status of the newly hatched chick. These results are in agreement with previous studies that have evaluated the maternal transfer of Se to the subsequent progeny (Surai, 2000; Paton et al., 2002; Pappas et al., 2005; Surai et al., 2006; Skřivan et al., 2008). Although GSH-Px activity was not determined in these experiments, it has been well established in the literature that maternal supplementation of Se yeast increases the activity of this enzyme in the progeny at the time of hatch (Combs and Scott, 1979; Surai, 2000; Pappas et al., 2005). In fact, Surai (2000) reported that feeding a combination of Se yeast and Vit.E in maternal diets further increased the GSH-Px activity in the livers of newly hatched chicks, however this was not accompanied by an additional increase in tissue Se. It has been demonstrated in previous studies that there is a highly significant, positive correlation between Se concentration and GSH-Px activity in most tissues (Surai et al., 1999; Surai, 2000; Pappas et al., 2005). Dietary supplementation of Se in the maternal diets becomes extremely important because the Se-dependent GSH-Px enzyme comprises about 61% of the total antioxidant enzyme activity in the liver of the newly hatched chick (Surai et al., 1999).

Based on the results from these experiments and from previous reports, it is clear that the developing embryo is capable of effectively utilizing and incorporating the Se that has been deposited in the egg. The dietary source of Se therefore, has a tremendous impact on the amount and location of Se that is deposited into the egg. Comparisons of inorganic (sodium selenite) and organic (Se yeast) sources have indicated that organic sources, where Se exists primarily as selenomethionine (SeMet), are more effective at increasing total egg Se than inorganic sources (Paton et al., 2002; Payne et al., 2005;

Surai et al., 2006; Pan et al., 2007; Skřivan et al., 2008; Čobanová et al., 2011). This is due not only to increased deposition of Se in the egg yolk, but in egg albumen proteins as well, suggesting a greater incorporation of SeMet (Paton et al., 2002; Surai et al., 2006; Čobanová et al., 2011). When feeding Se yeast, the hen is able to utilize additional metabolic pathways for incorporating Se into the egg through non-specific integration of SeMet into albumen proteins in place of methionine (Paton et al., 2002; Surai, 2006).

Hatching is considered a time of high stress for the chick due to increased oxidative metabolism and the transition to pulmonary respiration, which can lead to the overproduction of damaging free radicals (Surai et al., 1999). This is further complicated by the fact that a substantial portion of the lipids in the tissues of the developing embryo are polyunsaturated, which are highly susceptible to oxidation by free radicals (Speak et al., 1998; Surai et al., 1999). Therefore, the status of the antioxidant system of the developing chick is a key determinant in embryo viability, and the subsequent hatchability and performance of the chick post hatch (Surai et al., 1999). It has been reported in the literature that maternal Se supplementation is required to maintain normal hatchability through improvements in GSH-Px activity (Cantor and Scott, 1974; Combs and Scott, 1979; Renema, 2004). Supplementing Se in the starter diets of the newly hatched chicks can further enhance the post-hatch expression of GSH-Px (Cantor and Tarino, 1982; Pappas et al., 2005).

There were reductions in overall tissue Se concentrations between 0 and 7d of age in both experiments. However, there were significant main effects of maternal Se supplementation to increase liver and breast Se concentrations through 7d of age. When progeny were maintained on diets not supplemented with Se yeast, maternal Se supplementation increased 7d progeny liver and breast Se concentrations 1.5 and 1.4-fold, respectively, in Exp. 1, and 2.0 and 1.3-fold in Exp. 2 (data not shown). These results are in agreement with Pappas et al. (2005) and Surai et al. (2006) who demonstrated that supplementing maternal diets with Se yeast increases the Se status of chicks post-hatch. In both Exp. 1 and 2 however, the maternal effects of Se supplementation disappeared after 7d, which does not agree with the results from the previous authors. Pappas et al.

(2005) reported that the effects of maternal Se yeast supplementation to increase progeny tissue Se concentrations persisted for 3-4 weeks after hatching. In Japanese quail, Surai et al. (2006) demonstrated persistent effects of maternal Se yeast supplementation to increase tissue Se through 2 weeks post-hatch. Based on the results presented herein, it appears that the effects of a high Se progeny diet may rapidly overtake the beneficial effects of a high Se maternal diet on progeny tissue Se concentrations in a little as 7d. Nonetheless, there were persistent effects of maternal Se supplementation in Exp. 1 and 2 to increase progeny tissue Se concentrations through 7d which may improve the antioxidant defenses during this stressful period. It has been reported that improved tissue Se status of the progeny is highly correlated with increased GSH-Px activity in these tissues (Surai, 2000; Pappas et al., 2005).

It has been well established that Se supplementation has a 'sparing' effect on the Vit.E reserves in the egg and in the tissues of broilers (Surai, 2000; Surai, 2006; Skřivan et al., 2008). This assumption is based on the fact that Se, as a component of multiple Se-dependent GSH-Px enzymes, improves antioxidant activity through active removal of damaging lipid peroxides within the cells, thus sparing the requirement of Vit.E for this purpose (Surai, 2000). Interestingly, there were no differences among dietary treatments on progeny liver Vit.E concentrations at 7 or 14d of age in Exp. 1. At 7d, liver Vit.E tended to increase in response to progeny Vit.E supplementation, which was paired with a significant main effect of maternal Se and Vit.E supplementation to increase progeny liver Vit.E concentrations. The presence of a significant interaction of mSe*cSe*cVit.E to increase 7d liver Vit.E concentrations suggests that there may be a sparring effect of maternal and progeny Se supplementation on Vit.E stores.

In previous studies, the effect dietary supplementation of Vit.E in hen or broiler diets has been shown to increase egg or tissue α-tocopherol concentrations relative to the dietary inclusion level (Hossain et al., 1998; Marasheillo et al., 1999; Surai, 2000; Surai and Sparks, 2000; Guo et al., 2001; Young et al., 2003; Lauzon et al., 2008; Voljč et al., 2011). It is important to note that in most of the previous studies, Vit.E was supplemented at considerably higher levels (>50 IU Vit.E/kg diet) compared with the 30 IU Vit.E/kg

diet that were used in the experiments presented herein. In addition, the basal diets in these experiments were not deficient in Vit.E (19.71 and 20.43 IU Vit.E/ kg diet in Exp. 1 and 2, respectively), therefore the sparing effect of Vit.E from Se yeast supplementation may only be apparent when the dietary Vit.E concentration in the diet is low. Surai (2000) did not show a sparing effect on Vit.E by Se yeast supplementation (0.4 ppm) when 100 IU Vit.E was also included in the diet, suggesting that increased tissue Vit.E from high levels of Vit.E supplementation was not further compounded by the presence of dietary Se yeast.

In the literature, it has been well established that Vit.E supplementation in broiler diets does not affect the growth performance or feed conversion of broilers, provided there is not a deficiency of this nutrient (Seier and Bragg, 1973; Guo et al., 2001; Choct and Naylor, 2004; Ryu et al., 2005; Lauzon et al., 2008; Niu et al., 2009; Kim et al., 2010; Yesilbag et al., 2011). The effects of dietary Se supplementation to broilers however, are not as well defined, as previous reports vary on whether or not Se improved broiler performance. Multiple references have indicated that supplementing broiler diets with Se (specifically Se yeast) does not affect the growth performance or feed conversion of growing broilers (Edens et al., 2000; Edens et al., 2001; Choct and Naylor, 2004; Payne and Southern, 2005; Özkan et al., 2007; Yoon et al., 2007; Kim et al., 2010). In contrast, Ševčíková et al (2006) reported that the inclusion of 0.3 ppm Se yeast in broiler diets improved live BW at 21 and 42d of age compared to the unsupplemented control. Upton et al. (2007) reported that 0.2 ppm Se yeast supplementation in broiler diets significantly improved broiler BW and feed conversion from 28 to 42d of age compared to diets containing 0.2 ppm sodium selenite or no Se supplementation.

The growth performance results from these experiments support the previous research as there were varied performance responses between Exp. 1 and 2. There were significant main effects of Se supplementation of the progeny diets to improve ADG from 0-21d of age in both Exp. 1 and 2. These results are in general agreement with the results of Ševčíková et al. (2006) who indicated Se yeast supplementation improved live BW at 21d of age, which indicated that Se yeast improved ADG during this period.

Selenium yeast supplementation did not appear to improve growth performance from 22-49d in both experiments. The main effect of progeny Se to improve overall ADG from 0-49d in Exp.2 was likely due to the improvements in ADG during the starter phase.

Similar to previous reports, broiler ADFI in Exp. 1 and 2 was not affected by Se or Vit.E supplementation through 21d of age. It is unclear why there was a main effect of Se yeast to increase ADFI from 22-49d (and consequently, overall from 0-49d), as there have not been any previous reports indicating that Se yeast supplementation increases ADFI. Average daily feed intake throughout Exp. 2 was not affected by Se yeast or Vit.E supplementation, which is in agreement with previous reports in the literature.

The effect of progeny Se supplementation to increase ADFI from 22-49d in Exp. 1, resulted in a main effect of progeny Se to reduce G:F during this period. In contrast, G:F was improved by Se yeast supplementation from 0-21 and 0-49d in Exp. 2 due to improvements in ADG with no changes in ADFI. The results from Exp. 2 are in agreement with the results from Ševčíková et al (2006) and Upton et al. (2008) who indicated that Se yeast supplementation improved G:F when compared to the unsupplemented control. There were multiple significant interactive effects between maternal and the chick treatments that did not display a clear pattern explaining the significance of these effects. In general, the interactive effects appeared to be consistent with the existence of significant main effects from the maternal or chick diets alone.

In summary, Se yeast supplementation in the maternal diets considerably improved the Se status of the progeny at hatch, displaying persistent maternal effects to increase the Se concentrations in progeny through 7d of age. The beneficial effects of maternal Se supplementation appear to be overtaken by Se inclusion in the progeny diets after 7d, as feeding Se yeast to the progeny increased liver and breast Se concentrations through 14d of age. The effects of Se yeast and Vit.E supplementation on growth performance are still unclear, as performance was relatively unaffected by dietary treatments in Exp. 1, however Se yeast supplementation improved early growth performance and feed efficiency in Exp. 2. Further research is required to fully elucidate

the effects of Se and Vit.E supplementation on growth performance. Nonetheless, Se yeast supplementation in both the maternal and progeny diets effectively improves the antioxidant status of the newly hatched chick and young broilers through increases in tissue Se concentrations.

3.5. Tables and figures

Table 3.1. Composition of the progeny basal diets for Exp. 1 and 2 (as-fed basis).

	% of diet	
Ingredients	Starter	Grower
Ground corn	54.82	57.47
Soybean meal (48% CP)	36.00	32.50
Choice white grease	4.80	5.55
Ground limestone	1.50	1.20
Dicalcium phosphate	1.70	2.00
Salt	0.48	0.46
Vitamin mix[1]	0.25	0.25
Trace mineral mix[2]	0.25	0.25
L-lysine	–	0.10
DL-methionine	0.20	0.22
Total	100.00	100.00
Calculated nutrient composition[3]		
ME, kcal/kg	3,090	3,154
Crude protein, %	22.23	20.77
Lysine, %	1.25	1.22
Methionine, %	0.55	0.55
TSAA, %	0.90	0.88
Threonine, %	0.87	0.81
Tryptophan, %	0.27	0.25
Calcium, %	1.05	1.00
Phosphorus (available), %	0.45	0.50
Selenium, ppm	0.05	0.05
Vitamin E, IU/kg	19.71	20.43

[1] Supplied per kilogram of diet: Vitamin A, 8,000 IU; Vitamin D_3, 3,000 IU; Vitamin K, 3 mg; Thiamin, 2 mg; Riboflavin, 10 mg; Pantothenic acid, 12 mg; Niacin, 40 mg; Pyridoxine, 4 mg; Biotin, 0.2 mg; Folic acid, 0.75 mg; Vitamin B-12, 0.015 mg; Choline, 500 mg; Ethoxyquin, 125 mg.
[2] Supplied per kilogram of diet: Copper, 24 mg; Iodine, 0.89 mg; Manganese, 281.35 mg; Zinc, 83.33 mg; Cobalt, 20.19 mg.
[3] Calculated based on the NRC (1994) estimated values.

Table 3.2. Progeny liver and breast Se concentrations (ppm) in Exp. 1 on d 0, 7, and 14.[1]

	Liver Se, ppm			Breast Se, ppm		
Means	0d	7d	14d	0d	7d	14d
Maternal diet						
Control	0.93^{a}	0.71^{b}	0.87	0.55^{b}	0.63^{bc}	0.65
Se yeast	2.21^{a}	0.94^{A}	1.01	1.49^{a}	0.79^{a}	0.72
Vit.E	0.90^{b}	0.68^{B}	0.99	0.53^{b}	0.56^{c}	0.65
Vit.E + Se yeast	1.90^{a}	0.96^{A}	0.94	1.13^{a}	0.75^{ab}	0.66
SEM	0.14	0.07	0.06	0.07	0.05	0.05
Progeny diet						
Control	–	0.60^{C}	0.50^{B}	–	0.55^{B}	0.38^{B}
Se yeast	–	1.15^{A}	1.35^{A}	–	0.86^{A}	0.99^{A}
Vit.E	–	0.60^{C}	0.48^{B}	–	0.53^{B}	0.37^{B}
Vit.E + Se yeast	–	0.94^{B}	1.49^{A}	–	0.79^{A}	0.94^{A}
SEM	–	0.07	0.06	–	0.05	0.03
——— *P*-values (main effects) ———						
Maternal diet						
Se	0.01	<0.01	0.46	<0.01	0.02	0.23
Vit.E	0.34	0.98	0.71	0.12	0.31	0.39
Se*Vit.E	0.42	0.67	0.19	0.16	0.76	0.31
Progeny diet						
Se	–	<0.01	<0.01	–	<0.01	<0.01
Vit.E	–	0.15	0.35	–	0.39	0.37
Se*Vit.E	–	0.14	0.24	–	0.70	0.46

[a-b] Means within the same column with different superscript letters are significantly different ($P < 0.05$).

[A-B] Means within the same column with different superscript letters are significantly different ($P < 0.01$).

[1] Day 0 values represent the mean of 3 samples per treatment; day 7 and 14 values represent the mean of 12 pens/treatment (n=1 sample/pen).

Table 3.3. Progeny liver α-tocopherpol concentrations (μg/g) in Exp. 1 on d 7 and 14.[1]

	Liver α-tocopherol, μg/g	
Means	7d	14d
Maternal diet		
Control	2.85	4.57
Se yeast	2.50	4.04
Vit.E	2.65	4.36
Vit.E + Se yeast	2.94	4.05
SEM	0.14	0.16
Progeny diet		
Control	2.68	4.13
Se yeast	2.53	4.29
Vit.E	2.86	4.31
Vit.E + Se yeast	2.87	4.58
SEM	0.14	0.17
——— *P*-values (main effects) ———		
Maternal diet		
Se	0.78	0.35
Vit.E	0.33	0.68
Se*Vit.E	0.03	0.47
Progeny diet		
Se	0.61	0.23
Vit.E	0.08	0.20
Se*Vit.E	0.57	0.75

[1] Values represent the mean of 12 pens/treatment (n=1 sample/pen).

Table 3.4. Average daily gain (g/d) through 49d of age of the broilers in Exp. 1.[1]

	Weekly averages							Phase averages		
Means	7d	14d	21d	28d	35d	42d	49d	0-21d	22-49d	0-49d
Maternal diet										
Control	13.6	34.7[b]	55.5	73.6	86.7	78.7	76.5	34.3	78.5	59.6
Se yeast	14.6	38.0[a]	57.3	75.0	85.5	81.0	77.8	36.4	79.7	61.1
Vit.E	14.7	36.3[ab]	57.9	75.6	85.0	72.1	72.7	36.0	75.9	58.8
Vit.E + Se yeast	13.6	35.1[b]	54.2	73.4	87.7	78.6	83.6	34.0	80.6	60.6
SEM	0.4	0.7	1.3	1.9	2.3	2.4	3.8	0.7	2.0	1.3
Progeny diet										
Control	13.8	34.8[B]	54.8	74.3	87.7	77.9	81.1	34.2	79.9	60.3
Se yeast	14.0	36.2[B]	57.1	76.6	89.5	77.9	81.6	35.5	81.1	61.5
Vit.E	13.7	34.6[B]	55.6	73.6	85.1	76.3	72.5	34.4	76.5	58.4
Vit.E + Se yeast	15.1	38.5[A]	57.2	73.1	82.6	78.3	75.4	36.7	77.2	59.9
SEM	0.4	0.7	1.3	1.9	2.3	2.4	3.8	0.7	2.0	1.3
P-values (main effects)										
Maternal diet										
Se	0.96	0.38	0.50	0.83	0.79	0.20	0.26	0.97	0.33	0.39
Vit.E	0.95	0.56	0.79	0.90	0.93	0.19	0.85	0.73	0.76	0.73
Se*Vit.E	0.10	0.08	0.08	0.33	0.49	0.52	0.37	0.07	0.55	0.94
Progeny diet										
Se	0.05	<0.01	0.14	0.61	0.88	0.69	0.66	0.02	0.63	0.32
Vit.E	0.21	0.17	0.72	0.28	0.05	0.80	0.06	0.30	0.08	0.19
Se*Vit.E	0.11	0.08	0.79	0.46	0.37	0.69	0.77	0.45	0.92	0.93

[a-b] Means within the same column with different superscript letters are significantly different ($P < 0.05$).
[A-B] Means within the same column with different superscript letters are significantly different ($P < 0.01$).
[1] Represents the mean of 12 pens/treatment.

Table 3.5. Average daily feed intake (g/d) through 49d of age of the broilers in Exp. 1.[1]

	Weekly averages							Phase averages		
Means	7d	14d	21d	28d	35d	42d	49d	0-21d	22-49d	0-49d
Maternal diet										
Control	22.7	59.6	85.6	113.3	140.7	145.4	193.0	56.0	148.1	108.6
Se yeast	23.3	54.2	81.6	115.2	146.4	147.7	196.6	53.0	151.5	109.3
Vit.E	25.1	57.9	88.0	122.4	147.7	144.3	192.4	57.0	151.7	111.1
Vit.E + Se yeast	21.5	55.0	79.9	112.7	143.1	146.2	197.0	52.2	149.7	107.9
SEM	1.6	1.7	2.3	2.7	3.0	2.5	4.3	1.4	2.6	1.7
Progeny diet										
Control	23.3	55.8	86.7	111.4[b]	140.8	144.4	195.4	55.3	148.0[ab]	108.3
Se yeast	22.2	55.8	80.9	118.6[ab]	147.9	148.5	199.0	53.0	153.5[a]	110.4
Vit.E	23.0	55.6	84.1	110.9[b]	139.7	141.5	187.2	54.2	144.8[b]	106.0
Vit.E + Se yeast	24.0	59.5	83.4	122.7[a]	149.6	149.2	197.2	55.6	154.7[a]	112.2
SEM	1.6	1.7	2.3	2.7	3.0	2.5	4.3	1.4	2.6	1.7
	P-values (main effects)									
Maternal diet										
Se	0.34	0.06	0.23	0.36	0.91	0.69	0.62	0.15	0.90	0.75
Vit.E	0.83	0.80	0.93	0.44	0.71	0.80	0.99	0.97	0.86	0.89
Se*Vit.E	0.21	0.51	0.67	0.19	0.32	0.96	0.95	0.70	0.62	0.62
Progeny diet										
Se	1.00	0.26	0.17	<0.01	<0.01	0.03	0.13	0.76	<0.01	0.02
Vit.E	0.64	0.31	0.98	0.51	0.92	0.67	0.25	0.56	0.71	0.89
Se*Vit.E	0.52	0.26	0.28	0.40	0.64	0.47	0.47	0.20	0.41	0.23

[a-b] Means within the same column with different superscript letters are significantly different ($P < 0.05$).

[1] Represents the mean of 12 pens/treatment.

Table 3.6. Gain:feed through 49d of age of the broilers in Exp. 1.[1]

	Weekly averages							Phase averages		
Means	7d	14d	21d	28d	35d	42d	49d	0-21d	22-49d	0-49d
Maternal diet										
Control	0.63	0.59^{b}	0.66	0.65	0.62	0.54	0.39	0.62	0.53^{A}	0.55^{ab}
Se yeast	0.65	0.71^{a}	0.70	0.65	0.59	0.55	0.40	0.69	0.53^{A}	0.56^{a}
Vit.E	0.61	0.63^{b}	0.67	0.62	0.58	0.50	0.38	0.64	0.50^{B}	0.53^{b}
Vit.E + Se yeast	0.66	0.64^{ab}	0.68	0.65	0.61	0.54	0.42	0.66	0.54^{A}	0.56^{a}
SEM	0.04	0.03	0.02	0.01	0.01	0.01	0.02	0.02	0.01	0.01
Progeny diet										
Control	0.62	0.63	0.64	0.67^{A}	0.62^{A}	0.54	0.41	0.63	0.54^{A}	0.56^{A}
Se yeast	0.65	0.66	0.71	0.65^{A}	0.61^{A}	0.52	0.41	0.67	0.53^{A}	0.56^{A}
Vit.E	0.63	0.63	0.67	0.66^{A}	0.61^{A}	0.54	0.38	0.64	0.53^{A}	0.55^{AB}
Vit.E + Se yeast	0.65	0.65	0.69	0.60^{B}	0.55^{B}	0.52	0.38	0.66	0.50^{B}	0.53^{B}
SEM	0.04	0.03	0.02	0.01	0.01	0.01	0.02	0.02	0.01	0.01
	P-values (main effects)									
Maternal diet										
Se	0.34	0.03	0.32	0.14	0.87	0.09	0.10	0.13	<0.01	0.03
Vit.E	0.89	0.48	0.78	0.23	0.64	0.07	0.57	0.66	0.12	0.35
Se*Vit.E	0.74	0.05	0.64	0.18	0.02	0.21	0.19	0.37	<0.01	0.22
Progeny diet										
Se	0.60	0.37	0.02	<0.01	<0.01	0.27	0.84	0.12	<0.01	0.25
Vit.E	0.90	0.81	0.86	<0.01	<0.01	0.99	0.05	0.86	<0.01	0.05
Se*Vit.E	0.85	0.98	0.18	0.02	0.10	1.00	0.90	0.60	0.21	0.22

[a-b] Means within the same column with different superscript letters are significantly different ($P < 0.05$).
[A-B] Means within the same column with different superscript letters are significantly different ($P < 0.01$).
[1] Represents the mean of 12 pens/treatment.

Table 3.7. Progeny liver and breast Se concentrations (ppm) in Exp. 2 on d 0, 7, and 14.[1]

	Liver Se, ppm			Breast Se, ppm		
Means	0d	7d	14d	0d	7d	14d
Maternal diet						
Control	0.80^{B}	0.56^{B}	1.17^{ab}	0.61^{B}	0.56	0.70
Se yeast	2.41^{A}	0.66^{B}	1.28^{a}	1.41^{A}	0.71	0.73
Vit.E	0.66^{B}	0.60^{B}	0.97^{c}	0.65^{B}	0.65	0.62
Vit.E + Se yeast	2.49^{A}	0.95^{A}	1.09^{bc}	1.46^{A}	0.75	0.65
SEM	0.06	0.06	0.06	0.08	0.05	0.04
Progeny diet						
Control	–	0.55^{B}	0.51^{B}	–	0.47^{B}	0.37^{B}
Se yeast	–	0.88^{A}	1.66^{A}	–	0.88^{A}	0.92^{A}
Vit.E	–	0.41^{B}	0.54^{B}	–	0.46^{B}	0.43^{B}
Vit.E + Se yeast	–	0.93^{A}	1.81^{A}	–	0.85^{A}	0.98^{A}
SEM	–	0.06	0.06	–	0.05	0.04
			P-values (main effects)			
Maternal diet						
Se	<0.01	0.08	0.42	<0.01	<0.01	0.68
Vit.E	0.69	0.17	0.21	0.62	<0.01	0.29
Se*Vit.E	0.13	0.25	0.95	0.95	<0.01	0.96
Progeny diet						
Se	–	<0.01	<0.01	–	<0.01	<0.01
Vit.E	–	0.52	0.15	–	0.67	0.20
Se*Vit.E	–	0.12	0.31	–	0.82	0.94

$^{a-b}$ Means within the same column with different superscript letters are significantly different ($P < 0.05$).

$^{A-B}$ Means within the same column with different superscript letters are significantly different ($P < 0.01$).

[1] Day 0 values represent the mean of 2 samples per treatment; day 7 and 14 values represent the mean of 8 pens/treatment (n=1 sample/pen).

Table 3.8. Average daily gain (g/d) through 49d of age of the broilers in Exp. 2.[1]

	Weekly averages							Phase averages		
Means	7d	14d	21d	28d	35d	42d	49d	0-21d	22-49d	0-49d
Maternal diet										
Control	14.0	28.5^{b}	52.3	75.3	75.2	75.8^{A}	34.6	31.6	65.2^{a}	50.8^{ab}
Se yeast	15.0	33.5^{a}	54.2	76.4	79.5	81.6^{A}	38.0	34.2	68.9^{a}	54.0^{a}
Vit.E	14.6	31.8^{a}	58.5	77.5	75.8	63.2^{B}	18.9	35.0	58.8^{b}	48.6^{b}
Vit.E + Se yeast	14.8	32.5^{a}	54.6	79.2	80.3	75.4^{A}	35.2	34.0	67.6^{a}	53.1^{a}
SEM	0.4	1.0	1.9	3.0	2.0	2.6	5.8	1.0	2.0	1.3
Progeny diet										
Control	14.5	29.8^{B}	51.9	74.7	77.4	72.0	26.7	32.1^{b}	62.7	49.6
Se yeast	14.5	33.5^{A}	58.1	78.4	78.0	77.6	32.3	35.4^{a}	66.6	53.2
Vit.E	14.4	29.2^{B}	52.6	74.7	76.7	74.6	34.6	32.1^{b}	65.1	51.0
Vit.E + Se yeast	15.0	33.7^{A}	56.9	80.6	78.8	71.8	33.3	35.2^{a}	66.1	52.9
SEM	0.4	1.0	1.9	3.0	2.0	2.6	5.8	1.0	2.0	1.3
					P-values (main effects)					
Maternal diet										
Se	0.14	0.03	0.62	0.42	0.09	0.07	0.10	0.37	0.07	0.09
Vit.E	0.53	0.18	0.17	0.19	0.73	0.07	0.12	0.13	0.17	0.40
Se*Vit.E	0.27	0.05	0.21	0.84	0.94	0.41	0.22	0.10	0.33	0.70
Progeny diet										
Se	0.42	<0.01	0.02	0.13	0.51	0.61	0.71	0.01	0.25	0.05
Vit.E	0.57	0.79	0.92	0.71	1.00	0.56	0.45	0.94	0.63	0.69
Se*Vit.E	0.51	0.67	0.63	0.71	0.72	0.14	0.56	0.93	0.48	0.51

[a-b] Means within the same column with different superscript letters are significantly different ($P < 0.05$).
[A-B] Means within the same column with different superscript letters are significantly different ($P < 0.01$).
[1] Represents the mean of 8 pens/treatment.

Table 3.9. Average daily feed intake (g/d) through 49d of age of the broilers in Exp. 2.[1]

	Weekly averages							Phase averages		
Means	7d	14d	21d	28d	35d	42d	49d	0-21d	22-49d	0-49d
Maternal diet										
Control	20.9	75.8^{A}	87.1	127.5	129.0	166.8	128.8^{ab}	61.2	138.0	105.1
Se yeast	22.0	64.9^{B}	83.8	128.1	137.0	170.3	146.3^{a}	56.9	145.4	107.5
Vit.E	21.4	74.3^{A}	87.3	132.9	135.2	150.2	107.8^{b}	61.0	131.5	101.3
Vit.E + Se yeast	21.6	64.1^{B}	88.6	136.6	142.8	161.4	131.7^{ab}	58.1	143.1	106.7
SEM	0.4	2.4	2.9	5.4	5.1	6.6	8.0	1.5	4.5	2.8
Progeny diet										
Control	21.0	73.9	86.2	130.5	131.9	169.5	124.9	60.4	139.2	105.4
Se yeast	21.6	67.0	83.4	127.9	135.3	162.9	131.0	57.4	139.3	104.2
Vit.E	21.9	70.7	90.0	132.9	136.7	157.5	127.0	60.9	138.6	105.3
Vit.E + Se yeast	21.4	67.4	87.1	133.9	140.2	158.7	131.7	58.6	141.1	105.8
SEM	0.4	2.4	2.9	5.4	5.1	6.6	8.0	1.5	4.5	2.8
P-values (main effects)										
Maternal diet										
Se	0.38	0.11	0.89	0.57	0.05	0.18	0.02	0.29	0.03	0.19
Vit.E	0.95	0.83	0.73	0.14	0.10	0.06	0.03	0.88	0.17	0.39
Se*Vit.E	0.50	0.94	0.76	0.68	0.95	0.43	0.56	0.81	0.45	0.56
Progeny diet										
Se	0.82	0.06	0.35	0.88	0.52	0.69	0.52	0.10	0.77	0.90
Vit.E	0.38	0.58	0.23	0.46	0.36	0.24	0.86	0.55	0.90	0.81
Se*Vit.E	0.15	0.47	0.99	0.76	1.00	0.57	0.93	0.78	0.79	0.76

[a-b] Means within the same column with different superscript letters are significantly different ($P < 0.05$).
[A-B] Means within the same column with different superscript letters are significantly different ($P < 0.01$).
[1] Represents the mean of 8 pens/treatment.

Table 3.10. Gain:feed through 49d of age of the broilers in Exp. 2.[1]

	Weekly averages							Phase averages		
Means	7d	14d	21d	28d	35d	42d	49d	0-21d	22-49d	0-49d
Maternal diet										
Control	0.67	0.39^{B}	0.61	0.59	0.58	0.46	0.27	0.52^{b}	0.48	0.49
Se yeast	0.68	0.52^{A}	0.65	0.59	0.58	0.48	0.26	0.60^{a}	0.47	0.50
Vit.E	0.68	0.44^{B}	0.67	0.58	0.56	0.42	0.17	0.58^{a}	0.45	0.48
Vit.E + Se yeast	0.69	0.52^{A}	0.62	0.59	0.57	0.47	0.25	0.59^{a}	0.47	0.50
SEM	0.02	0.02	0.02	0.01	0.01	0.02	0.04	0.01	0.01	0.01
Progeny diet										
Control	0.69	0.44^{B}	0.60^{b}	0.57^{b}	0.59	0.43	0.19	0.54^{B}	0.45	0.47^{b}
Se yeast	0.67	0.51^{A}	0.70^{a}	0.61^{a}	0.58	0.48	0.24	0.62^{A}	0.48	0.51^{a}
Vit.E	0.66	0.42^{B}	0.60^{b}	0.57^{b}	0.56	0.47	0.27	0.53^{B}	0.47	0.49^{ab}
Vit.E + Se yeast	0.70	0.51^{A}	0.66^{ab}	0.60^{ab}	0.56	0.45	0.25	0.60^{A}	0.47	0.50^{a}
SEM	0.02	0.02	0.02	0.01	0.01	0.02	0.04	0.01	0.01	0.01
P-values (main effects)										
Maternal diet										
Se	0.78	0.06	0.87	0.91	0.64	0.14	0.24	0.08	0.23	0.09
Vit.E	0.76	0.54	0.60	0.61	0.03	0.16	0.14	0.29	0.16	0.60
Se*Vit.E	0.88	0.56	0.30	0.96	0.50	0.45	0.16	0.16	0.22	0.89
Progeny diet										
Se	0.46	<0.01	<0.01	<0.01	0.67	0.34	0.65	<0.01	0.22	<0.01
Vit.E	0.97	0.51	0.38	0.61	0.24	0.58	0.26	0.47	0.55	0.77
Se*Vit.E	0.12	0.62	0.36	0.78	0.76	0.07	0.33	0.72	0.20	0.17

[a-b] Means within the same column with different superscript letters are significantly different ($P < 0.05$).
[A-B] Means within the same column with different superscript letters are significantly different ($P < 0.01$).
[1] Represents the mean of 8 pens/treatment.

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CHAPTER 4. Effects of dietary selenium yeast and vitamin E supplementation to broilers on breast selenium concentrations and meat quality characteristics of raw and marinated breast fillets

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4.1. Introduction

The inclusion of organic selenium (Se) in poultry diets has received considerable attention because recent research has indicated that improvements in the avian antioxidant system have beneficial applications in all stages of production (Surai, 2006). Regarding meat quality, several investigators have reported reductions in breast fillet drip loss when broilers received organic Se in the diet (Edens, 1996; Choct et al., 2004; Upton et al., 2008; Jiang et al., 2009; Perić et al., 2009; Wang et al., 2010). Dietary supplementation of organic Se in broilers has also been shown to improve feathering and carcass yields (Choct et al., 2004; Upton et al., 2008).

Several studies investigating the effects of dietary vitamin E (Vit.E) supplementation to broilers have indicated improvements in prolonging the onset of lipid oxidation and rancidity in meat samples (Sheldon et al., 1997; Grau et al., 2001; Kim et al., 2010). Coetzee and Hoffman (2001) reported that feeding up to 160 mg/kg Vit.E in broiler diets resulted in thiobarbituric acid reactive substances (TBARS) values after 8d of storage that were nearly identical to that of the control group (no Vit.E supplementation) on d 0, thus prolonging the onset of rancidity. Organic Se supplementation often results in a ‘sparing’ effect of Se on Vit.E metabolism in poultry, increasing the Vit.E stores in the body (Surai 2000; Skřivan et al., 2008). Few studies have investigated the supplementation of organic Se together with other antioxidants such

as Vit.E on raw poultry meat quality and research is needed involving marinated poultry meat.

Marination is a widespread technique utilized by the poultry industry to enhance the quality characteristics of poultry meat (Alvarado and McKee, 2007; Smith and Young 2007). Typical commercial marinades contain a mixture of water, sodium chloride (NaCl) and phosphate salts (i.e., pyrophosphate or tripolyphosphate) that may be applied to the meat by a variety of methods. It has been well established that marination of poultry meat improves its water-holding capacity. The presence of NaCl and phosphates in the marinade solution promotes muscle fiber expansion (swelling) through electrostatic repulsion, allowing for increased water binding in the myofibril lattices (Xiong and Kupski, 1999a,b; Alvarado and McKee, 2007; Smith and Young, 2007). Despite the basic nature of NaCl and phosphates in marinade solutions, information is lacking regarding the oxidative stability of uncooked marinated chicken meat under storage conditions. Furthermore, information in the literature is lacking regarding the effects of dietary Se and Vit.E supplementation on the meat quality characteristics of marinated poulty meat products. Therefore, the objective of these experiments was to evaluate the effects of supplementing broiler diets with organic Se and Vit.E on the meat quality characteristics of raw and marinated breast fillets.

4.2. Materials and methods

Experiments were conducted under protocols approved by the University of Kentucky Institutional Animal Care and Use Committee.

4.2.1. Animals and Treatments

Breeders. A total of 640 Cobb500^{TM} broiler breeder pullets and 80 roosters were randomly allotted to 4 dietary treatments in a 2 x 2 factorial design. Birds were placed in 2.44 x 1.83 m floor pens with dry wood shavings as bedding providing 4.47 m^2 of floor space with 5 blocks of pullets (32 pullets/pen, 20 pens total) and 1 block of roosters (20 roosters/pen, 4 pens total) per dietary treatment. Diets consisted of a corn-soybean meal basal diet without added Se or Vit.E, the basal

diet supplemented with 0.3 mg/kg Se as selenium yeast (Sel-Plex®, Alltech, Inc., Nicholasville, KY), or 30 IU/kg Vit.E per kg as all-rac-α-tocopheryl acetate (Rovimix® E50-Adsorbate, DSM Nutritional Products, Parsippany, NJ), or both. Pullets and roosters received light according to the following schedule: 22h of light through 2 weeks of age, 8h of light from 3 through 22 weeks of age, and 16h of light after 22 weeks of age. Birds were provided with *ad libitum* access to water and were limit-fed daily in trough feeders (243.8 x 10.2 cm) according to the Cobb Breeder Management Guide for fast feathering birds (Cobb-Vantress, 2008). At the time of light stimulation, males were added and the number of hens per pen was reduced to a ratio of 13 females to 1 male which was maintained throughout the entire laying cycle for the 5 blocks of hens.

Eggs were collected from broiler breeder hens at 44 and 67 weeks of age for Exp. 1 and 2, respectively. The collected eggs were allotted within a Natureform NOM 45 incubator (NatureForm Hatchery Systems, Jacksonville, FL) according to maternal diet and incubated at 37.5 °C and 55% RH. Eggs were transferred to the same model NatureForm hatcher at 18d and held at 36.5 °C and 65% RH. At hatch, chicks were placed in 1.22 x 1.83 m floor pens with dry wood shavings as bedding providing 2.23 m^2 of floor space and were allotted to 4 dietary treatments in a randomized complete block design, with block corresponding to the cage location within the broiler facility. Chicks were allocated within each block to account for all maternal and chick diet interactions (16 pens/block). Chicks were provided with *ad libitum* access to feed and water and feed was administered in tube feeders.

Chicks. Both experiments used Cobb500™ broilers that were placed as straight run. Dietary treatments consisted of a corn-soybean meal basal diet without added Se or Vit.E (Table 4.1), the basal diet supplemented with 0.3 mg/kg Se yeast, or 30 IU/kg Vit.E, or both. Diets were formulated to meet or exceed the NRC (1994) estimated requirements for metabolizable energy, crude protein, vitamins and minerals other than Se and Vit.E. In Exp. 1, 768 1-d old chicks were randomly

allotted to 48 pens of 16 birds per pen with 3 blocks. In Exp. 2, 448 1-d old chicks were randomly allotted to 32 pens of 14 birds per pen with 2 blocks.

4.2.2. Carcass yield and sample collection

In Exp. 1 and 2, broilers were slaughtered at 49 and 56d of age (for the raw and marinated portions of each experiment, respectively). Carcass yield measurements were obtained at 49d, which included weight without giblets (WOG), front half (breast, ribs, corresponding back, and wings), and back half (pelvis, thigh, drumstick) percentages (expressed as a percentage of the live weight). Following a 3h chill, each carcass was deboned and boneless, skinless, breast fillets (pectoralis major) were collected and chilled on ice before analysis of meat quality characteristics. Additional breast samples were collected on d 49 and stored at -80 °C for subsequent analysis of breast Se concentration (Exp. 1 and 2) and fatty acid profile (Exp. 2).

4.2.3. Chemical analyses

The Se concentration of the breast muscle samples were analyzed according to the fluorometric assay of Olson et al. (1975), with modifications described by Cantor and Tarino (1982; See Appendix 3 for full description). Breast tissue samples were minced and manually macerated to ensure homogeneity. Tissue samples were then weighed into digestion tubes and digested in nitric and perchloric acids. The fatty acid profiles of breast fillets in Exp. 2 were determined using methods 965.49, 969.33, and 996.96 described in the AOAC (2006).

4.2.4. Marination procedure

Breast fillets for the marinated portions of Exp. 1 and 2 were collected from broilers at 56d of age in both experiments. Breast fillets were marinated in a solution containing 3.2% sodium pyrophosphate and 4% NaCl. Before marination, breast fillets were placed on a stainless steel rack to drain any excess moisture. Fillets were then placed in netted ham socks according to pen of origin, labeled, and placed in the marinade solution to soak for approximately 13h in a 2 ºC walk in cooler. The pH and initial temperature of the marinade were 9.70 and 19.95 °C for Exp. 1, and 9.77 and

20.55 °C for Exp. 2. After marination, breast fillets were removed from the marinade and placed on a stainless steel rack to drain excess moisture. Samples were collected before and after marination in Exp. 1 for determination of breast fillet pH.

4.2.5. Drip loss

Drip loss was measured using the suspension method (NPPC, 2000). Breast fillets were weighed before placement in sealed plastic bags and stored at 2 °C. After 3 and 7d of storage, each sample was weighed and percent drip loss was calculated (expressed as a percentage of the initial weight).

4.2.6. Oxidative stability

Determination of the dietary treatment effect on lipid oxidation of raw breast fillets over 7d (Exp. 1) and 12d (Exp. 2) was assessed by measuring TBARS according to procedures similar to Schemedes and Holmer (1989). Breast fillets were placed on a Styrofoam tray with a moisture pad and then covered with polyvinyl chloride (PVC) overwrap and stored at 2 °C under 1300 lux fluorescent lighting. For Exp. 1, samples were collected on d 0, 2, 5, and 7, and on d 0, 5, 7, 10, and 12 for Exp. 2. A 5 g meat sample was collected and the unfrozen sample was homogenized in a blender with 22.5 mL trichloroacetic acid (TCA) solution (11%) and filtered through Whatman #1 filter paper (in duplicate). Then 1 mL of the filtrate was mixed with 1 mL thiobarituric acid (TBA) aqueous solution (20 mM) and incubated at 25 °C. A blank was also prepared using 2 mL TCA/H_2O mixture (TCA/H_2O: 1:1 v/v) and 2 mL TBA solution. After 20h of incubation, samples were read at 532 nm on a Shimadzu UV-2401 PC spectrophotometer (Shimadzu Inc., Columbia, MD) and TBARS values were expressed as mg of malondialdehyde (MDA) equivalents/kg of meat using the method described by Witte et al. (1970).

4.2.7. Color score analysis

The color changes in the breast fillets in Exp. 1 were measured objectively for the Commission Internationale de l'Eclairage (CIE) values of lightness (L*), redness (a*), and yellowness (b*) using a HunterLab Miniscan XE Plus colorimeter (Hunter

Associates Laboratories, Inc., Fairfax, VA) using illuminant D-65/10° (1-cm aperture), calibrated against a white and black tile covered with PVC overwrap. Breast fillets were placed on a Styrofoam tray with a moisture pad and then covered with PVC overwrap and stored at 2 °C under 1300 lux fluorescent lighting. Color measurements were measured in triplicate on the ventral surface of each breast fillet through the PVC overwrap on d 0, 2, 5, and 7.

4.2.8. pH determination

Samples were collected for determination of pre- and post-marination breast fillet pH in Exp. 1. Approximately 10 g of sample was homogenized with 50 mL of deionized water and the pH was determined using a pH meter (Accumet pH meter, Model AR 25, Fisher Scientific Co., Fair Lawn, NJ).

4.2.9. Cooking loss

The cooking loss (%) was measured as the moisture lost from cooking the breast fillets was measured in Exp. 1. Post harvest, breast fillets were weighed and then cooked using a George Foreman clam-shell grill (Salton Inc., Columbia, MO), with an upper and lower surface temperature of 170–180° C (Suman et al., 2004, 2005) to an internal temperature of 72 °C which was monitored using an internal temperature probe. After cooking, breast fillets were weighed and cooking loss (CL) was determined on a weight basis according to the following equation:

$$\mathrm{CL} = \frac{(uncooked\ weight - \ cooked\ weight)}{uncooked\ weight} \times 100$$

4.2.10. Tenderness

Tenderness of the cooked breast fillets was determined in Exp. 1 by measuring the shear force of the cooked breast fillets. Breast fillets were allowed to reach to room temperature (25 °C) before analysis. Three sample cores were obtained from each cooked breast fillet using a 1.27-cm diameter hollow drill, cut perpendicular to the fiber orientation of the muscle through the thickest part of each fillet. Each sample core was analyzed for shear force value using a Shimadzu EZ-S texture analyzer (Shimadzu Corp.,

Kyoto, Japan) set at a speed of 50 mm/min. The mean value of the three cores represented the value for each pen (n = 1 breast/pen).

4.2.11. Statistical analysis

These experiments utilized a split-plot arrangement, with maternal diet as the whole-plot factor and chick diet as the sub-plot factor. Statistical analysis was conducted using the GLM procedure of SAS® (SAS, Cary, NC). Replicate pen of broilers served as the experimental unit. All interactions of maternal and chick dietary Se and Vit.E were analyzed, although only main effects from the chick diets (Se, Vit.E, interaction) are presented. Fisher's least significant difference test was used to determine significance between means with a significance set at $P \leq 0.05$ and a tendency set at $P \leq 0.10$.

4.3. Results

4.3.1. Experiment 1

There was a significant main effect of Se supplementation ($P < 0.01$) to increase breast Se concentrations in broilers fed diets containing Se yeast (Se yeast alone or Vit.E + Se yeast) compared to those fed the control diet or Vit.E diet (Figure 4.1). Dietary treatments had no effect on carcass yields (Table 4.2).

In the raw portion of Exp. 1, dietary treatments did not affect cooking loss or shear force values of the cooked breast fillets (Table 4.3). The drip loss and oxidative stability results for the raw portion of Exp. 1 are detailed in Table 4.4. Drip loss of the raw breast fillets under typical storage conditions were affected by dietary treatments as the birds receiving dietary Se yeast (Se yeast alone or Vit.E + Se yeast) displayed lower ($P < 0.05$) drip loss values than the control and Vit.E treatments after 3 and 7d of storage, with significant main effects for Se supplementation at 3 and 7d ($P < 0.01$). Oxidative stability of the raw breast fillets in Exp. 1 was affected by antioxidant supplementation as TBARS values for the Vit.E + Se yeast treatment were significantly lower ($P < 0.05$) than those fed the control diet at d 2 and 5d of storage, and tended to be lower ($P < 0.10$) after 7d of storage.

There was a significant main effect of dietary Se supplementation on breast fillet L* values on d 7 ($P < 0.054$), indicating that Se supplementation resulted in higher L* values (Table 4.5). No effects of dietary treatments on the a* and b* values of breast fillet through 7d of storage were observed.

In the marinated portion of Exp. 1, breast fillet pH values were slightly increased as a result of marination, however the difference was not significant (Table 4.6). Dietary treatments had no effect on pre- or post-marination breast fillet pH. There were no effects of dietary treatments on cooking loss and shear force values of the cooked marinated breast fillets. However, there was a significant main effect of Se supplementation to reduce the percent cooking loss of marinated breast fillets ($P < 0.05$).

The drip loss and oxidative stability results for the marinated portion of Exp. 1 are detailed in Table 4.7. Dietary treatments had no effect on drip loss of the marinated breast fillets through 3 and 7d of storage. Overall oxidative stability of the marinated breast fillets was affected by dietary treatments, as the TBARS values from all antioxidant-supplemented treatments (Se, Vit.E or both) were lower ($P < 0.01$) than those from the control at d 2, 5, and 7 of storage, with significant main effects for Se, Vit.E, and their interaction at d 2, 5, and 7 ($P < 0.01$). At 7d of storage, the lowest TBARS response was observed when Se yeast and Vit.E were fed in combination, compared to the control treatment or feeding Se yeast or Vit.E alone.

4.3.2. Experiment 2

There was a significant main effect of Se yeast supplementation to increase ($P < 0.01$) the breast Se concentrations of broilers fed diets containing Se yeast (Se yeast and Se yeast + Vit.E) compared to those fed the control diet or Vit.E alone (Figure 4.2). There were no differences between treatments on WOG, front half, and saddle percentages of the harvested broilers in Exp. 2 (Table 4.8). However, significant main effects were observed for Se on front half and saddle percentages ($P < 0.05$), indicating that Se supplementation increased front half and decreased saddle percentages.

The drip loss and oxidative stability results for the raw portion of Exp. 2 are detailed in Table 4.9. Drip loss of the raw breast fillets at 3 and 7d of storage was not affected by dietary treatments in Exp. 2. The TBARS values of the raw breast fillets increased as storage duration increased however there were no differences between dietary treatments through 12d of storage.

The drip loss and oxidative stability results for the marinated portion of Exp. 2 are detailed in Table 4.10. Drip loss of the marinated breast fillets at 3d were not significantly different between treatments, however there was a significant main effect of Se supplementation to reduce drip loss ($P < 0.05$). At 7d, drip loss tended to be lower ($P < 0.10$) for the Se yeast and Vit.E + Se yeast treatments compared to the control, due to a significant main effect of Se supplementation ($P < 0.05$). Oxidative stability was affected by dietary treatments in the marinated portion of Exp. 2. The TBARS values of marinated breast fillets for all antioxidant-supplemented treatments were significantly lower ($P < 0.01$) than those from birds fed the control diet at 5, 7 and 10d of storage. At 12 d of storage, the TBARS values for the treatments containing Vit.E alone or Vit.E + Se yeast were significantly lower ($P < 0.01$) than both the control and Se yeast alone treatments. Significant main effects were observed for Se from d 5 through d 10 ($P < 0.05$), for Vit.E from d 5 through d 12 ($P < 0.01$), and their interaction on d 5 ($P < 0.05$) and d 7 ($P < 0.10$).

The fatty acid composition of the breast fillets in Exp. 2 is detailed in Table 4.11. There was a significant main effect for Se*Vit.E on the elaidic acid concentration, indicating higher percentage of elaidic acid in response to Se and Vit.E supplementation. Otherwise, dietary treatments had no effect on the fatty acid concentration of breast fillets in Exp. 2.

4.4. Discussion

Supplementation of Se yeast in these experiments significantly increased Se accumulation in the breast tissue. These results are in agreement with previous studies utilizing organic sources of Se (Downs et al., 2000; Bou et al., 2005; Surai et al., 2006;

Ševčíková et al., 2006; Chekani-Azar et al., 2010; Pappas et al., 2012). It has been well established in the literature that the bioavailability of organic Se (Se yeast) is greater than inorganic sources (i.e., sodium selenite), because it is utilized differently by the chicken (Cantor and Tarnino, 1982; Wolffram, 1999; Choct et al., 2004; Payne and Southern, 2005; Surai et al., 2006; Özkan et al., 2007; Yoon et al., 2007; Wang et al., 2010). Sodium selenite (SS) is passively absorbed in the small intestine and later incorporated into selenoenzymes or excreted, whereas Se yeast (predominately selenomethionine) is actively absorbed by the same transport pathways as methionine and later incorporated (non-specifically) into tissues in place of methionine (Wolffram, 1999). Vitamin E functions as the primary chain-breaking antioxidant in the avian body, by scavenging free radicals and inhibiting the propagation of lipid oxidation (Jensen et al., 1995; Surai, 2006). Selenium plays an integral supportive role to Vit.E as a component of several glutathione peroxidases (GSH-Px), which are responsible for the cellular removal of the precursors of free radicals (i.e., hydroperoxides) (Surai, 2006; Puvača and Stanaćev, 2011). In addition, Se is involved in the recycling of Vit.E through the selenoenzyme thioredoxin reductase, which recycles ascorbic acid and, in turn, promotes the recycling of Vit.E (Surai, 2002; Surai, 2006; Skřivan et al., 2008). In fact, it has been shown that the action of these Se-dependent enzymes often display a “sparing” effect on the Vit.E stores in poultry by reducing the demand on Vit.E for the inhibition of lipid peroxidation, ultimately rendering more Vit.E available to the animal (Surai, 2000; Surai, 2006; Skřivan et al., 2008).

Previous reports on the effect of antioxidant supplementation on carcass yields vary. The results from Exp. 1 indicate that Se yeast and Vit.E supplementation had no effect on WOG, front half, and saddle percentages. These results are in agreement with previous reports that Se supplementation does not affect broiler carcass yields (Downs et al., 2000; Choct and Naylor, 2004; Deniz et al., 2005; Payne and Southern, 2005; Ševčíková et al., 2006). Other investigators have reported that Se yeast supplementation increased drumstick (Choct et al., 2004; Upton et al., 2008) and thigh yields (Choct et al., 2004). In contrast, the results from Exp. 2 indicate that Se supplementation increased front half and decreased saddle percentages. It remains unclear from these results whether

Se supplementation affects carcass yields; however, Surai (2002) suggested that any effects on meat yield from Se supplementation may be due to alterations in thyroid hormone metabolism in the bird. Reports of the effects of Vit.E supplementation on carcass yields are absent in the literature.

Very little data exist on the cooked characteristics of breast fillets originating from broilers supplemented with Se or Vit.E. Overall, tenderness and moisture loss as a result of cooking (cooking loss) are not affected by Se supplementation (Jiang et al., 2009; Miezeliene et al., 2011). Our results are in agreement with these reports as there were no differences in tenderness or cooking loss in Exp. 1.

It has been well established in the literature that the water holding capacity (as measured by percent drip loss) of chicken meat is improved by Se yeast supplementation (Edens, 1996; Choct et al., 2004; Deniz et al., 2005; Upton et al., 2008; Jiang et al., 2009; Perić et al., 2009; Wang et al., 2010). Choct and Naylor (2004) compared feeding 0.1 ppm Se as SS and Se yeast with 50 and 100 IU/kg Vit.E and reported that Se yeast with 50 and 100 IU Vit.E significantly reduced 24h drip loss compared with SS. Edens (1996) suggested that synergism between Vit.E and GSH-Px may reduce oxidative damage that otherwise could compromise the integrity of cellular membranes allowing for uncontrolled movement of water between the various compartments (i.e., increasing drip loss). Interestingly, Payne and Southern (2005) reported no differences in drip loss between control (no added Se) and Se yeast treatments. The results for Exp. 1 support previous findings that Se yeast supplementation reduces drip loss of raw breast fillets. In contrast to Exp. 1, there was no effect of Se yeast and Vit.E supplementation on 3 and 7 d drip loss of the raw breast fillets for Exp. 2. Note that the drip loss at 3 d for Exp. 2 was extremely low (less than 1.25%) for all treatments; any dietary treatment effects may not have been apparent at these low levels.

Lipid peroxidation is one of the primary causes of rancidity in raw and cooked meat products under refrigerated storage. The determination of TBARS levels in meat products has become a widely accepted method for quantifying the production of reactive

oxygen species and thus, assessing the oxidative stability of meat (Gutteridge, 1984; Schmedes and Holmer, 1989). Previous studies have reported improvements in the oxidative stability of poultry meat when diets were supplemented with Se (Mikuski et al., 2009; Chekani-Azar et al., 2010), Vit.E (Maraschiello et al., 1999; Guo et al., 2001; Ryu et al., 2005; Sheldon et al., 1997; Rebolé et al., 2006; Narciso-Gaytan et al., 2010; Voljč et al., 2011), or in combination (Ryu et al., 2005; Kim et al., 2010). Chekani-Azar et al. (2010) reported that Se yeast was more effective than SS at reducing TBARS values in breast fillets through 8d of storage. The authors indicated that tissue Vit.E levels increased due to Se yeast supplementation and it is likely that the sparing effect of Se on Vit.E may have contributed to the improvement in oxidative stability of the breast fillets. Coetzee and Hoffman (2001) reported that including up to 160 mg/kg Vit.E in broiler diets resulted in d 8 TBARS that were nearly identical to that of the d 0 values for the control group (no Vit.E supplementation). Recently, Kim et al. (2010) evaluated supplementing broiler diets with Vit.E (50, 100, and 200 IU/kg) and Se (0.3 ppm Se yeast) alone and in combination on the oxidative stability of thigh muscle during prolonged refrigerated storage. The authors reported that oxidative stability through 7d did not differ from the unsupplemented control when 50 IU/kg Vit.E and Se yeast were fed alone. However, oxidative stability was significantly lower for the control when compared to feeding 100 IU/kg Vit.E alone or in combination with Se yeast, or 200 IU/kg Vit.E alone. After 10d of storage, the authors reported that supplementation of Se and Vit.E alone, or in combination, reduced thigh muscle TBARS values compared with the control. Similar results were reported by Ryu et al. (2005), indicating that supplementation of 100 IU/kg Vit.E and 8 ppm Se (SS) alone effectively improved the oxidative stability of chicken breasts stored for up to 12d. In contrast, Pappas et al. (2011) reported that feeding Se yeast at 0.3 and 3 ppm in broiler diets was not effective at reducing TBARS values at 7d. The presence of Vit.E (80 IU/kg) in the vitamin premix may have masked any effects of Se. In our experiments, TBARS levels increased as the duration of storage increased for all dietary treatments. Our results for Exp. 1 are consistent with those of Kim et al. (2010) as Se yeast and Vit.E supplementation alone reduced 7d TBARS values of raw breast fillets, with the greatest reduction observed when Se yeast and Vit.E were fed in combination. It is unclear why TBARS values were not affected by dietary treatment in

the raw portion of Exp. 2. It appears that the combination of both Se yeast and Vit.E in broiler diets may be more effective at reducing lipid oxidation in poultry meat under prolonged storage conditions than feeding either Se yeast or Vit.E alone (Surai, 2002; Pappas et al., 2012).

Overall color stability of fresh meat declines over time under storage conditions, because myoglobin is oxidized to metmyoglobin due to oxygen exposure during storage. It has been suggested that the presence of antioxidant compounds may improve the color stability of raw meat by retarding the formation of metmyoglobin (Fernandez-Lopez, 2005). Previous studies evaluating antioxidant supplementation in broiler diets have reported mixed results on the color stability of poultry meat. Ryu et al. (2005) reported that supplementation of Se up to 0.8 mg/kg (as SS) or 100 IU/kg Vit.E did not affect color stability in broiler breast fillets. In contrast, Kim et al. (2010) reported that a* (redness) values of the breast fillets were reduced at 7 and 10d of refrigerated storage for broilers supplemented with 50 or 100 IU/kg Vit.E, Se yeast, or in combination when compared with the unsupplemented control. The results from Exp. 1 are consistent with Ryu et al. (2005) as L*, a*, and b* values of the raw breast fillets were unaffected by dietary treatment through 7d of storage. The significant main effect of Se to increase L* values at 7d is unclear. Changes in L* values are unrelated to the chemical status of myoglobin (McKenna et al., 2005). Instead L* values depend upon the amount of reflected light that is scattered and absorbed which can be affected by the moisture content of the meat (Miezeliene et al., 2011). Any reductions in superficial moisture content of meat as a result of lipid oxidation may result in paler (increased L* value) colored meat (Ripoll et al., 2011). Selenium supplementation effectively reduces lipid oxidation in meat products, therefore it was unexpected that our results indicated an increase in L* values at 7d as a result of Se supplementation. Further investigation may be required to evaluate the effects of antioxidant supplementation on the color stability of breast fillets.

The effects of Se yeast and Vit.E on breast fatty acid (FA) profile are somewhat variable. In the literature, changes in FA profile resulting from Se or Vit.E inclusion

appears to be dependent on the FA profile of the experimental diets. High levels of unsaturated FA in broiler diets may increase the degree of unsaturation in the membrane lipids in muscle, which could reduce the overall oxidative stability of the meat (Morrissey et al., 1998). This effect was confirmed by Narciso-Gaytán et al. (2010), who reported that the FA composition of chicken breast fillets reflected the FA composition of the fat source in the diet, as soybean and palm kernel oil increased polyunsaturated and saturated FA in the breast fillets. Interestingly, the authors did not report any effect of Vit.E supplementation or interactive effect of Vit.E and fat source on the FA composition of breast filets. Pappas et al. (2011) reported a linear increase in long-chain polyunsaturated FA and a linear decrease in linolenic and α-linoleic acids as dietary Se yeast supplementation increased. The authors suggested that higher levels of dietary Se may have reduced the degradation of long-chain polyunsaturated FA by peroxidation. In this experiment, there was a significant main effect of Se*Vit.E to increase the elaidic acid concentration in breast meat. Elaidic acid is the trans isomer form of oleic acid and contributes to the total unsaturated FA content of chicken breasts, however the individual role has not been characterized in poultry. Overall, our results were similar to those of Bou et al. (2005), who indicated no effect of Se yeast supplementation at 0.2 mg/kg on the FA profile of breast fillets.

The effects of antioxidant supplementation in broiler diets on the meat quality of marinated breast meat have not been previously established. Marination of chicken meat with salt and phosphates has been evaluated extensively in the literature and has been shown to increase water-holding capacity, decrease cooking losses, and improve meat tenderness (Xiong and Kupski, 1999a,b; Alvarado and McKee, 2007; Smith and Young, 2007). Water-holding capacity, and thus the moisture lost from cooking, is influenced by the pH of the myofibrillar proteins actin and myosin (Alvarado and McKee, 2007). During the development of rigor, the accumulation of lactic acid in the meat results in a decline in meat pH, ultimately reducing the number of reactive groups available on the myofibrillar proteins for water binding (Hedrick et al., 1976). Reductions in water-holding capacity may also occur as the pH approaches the isoelectric point of actin and myosin, minimizing net charges and interfilamental space where water could be bound

(Hedrick et al., 1976; Alvarado and McKee, 2007). The presence of salt and phosphates in marinade solutions may increase the pH of meat, leading to expansion or “swelling” of the myofibril lattices and increased capacity to bind water (Bendall, 1954; Xiong and Kupski, 1999a,b; Alvarado and McKee, 2007).

In our experiments, it was difficult to accurately determine the marinade uptake of the marinated breast fillets. Average marinade uptake was less than 1.5% across all dietary treatments in Exp. 1 and 2, and some pens displayed no change or negative changes (less than 1%) in final breast weight. Given that the breast fillets were soaked in the marinade for 13 h in these experiments, it is possible that actual marinade uptake was underestimated as it was not possible to measure the amount of purge loss from the fillets during marination, which may have then been replaced by the marinade. Conventional marination techniques utilize short-term marination, durations (< 1h), which are generally accompanied by agitation, or application of vacuum pressure, or both (Alvarado and McKee, 2007; Smith and Young, 2007).

It is clear that marinade uptake may have been underestimated as marination affected breast pH (increased from 5.8 to 6.0) and overall breast fillet drip loss 7d of storage (<1.1%; data not shown). Given that effects of marination with sodium pyrophosphate and NaCl considerably improve the water-holding capacity of chicken breast fillets (Alvarado and McKee 2007; Smith and Young 2007), the minimal improvements in drip loss typically associated with antioxidant supplementation may have been overshadowed in our Exp. 1. In Exp. 2 however, drip loss of the marinated breast fillets at d 3 and 7 was reduced as a result of Se supplementation (main effect, Se). In addition, cooking loss of the marinated breast fillets was improved by Se yeast supplementation in Exp. 1 (main effect, Se). Dietary treatment did not affect the shear force values of the marinated breast fillets. These results differ from previous reports that suggest that marination with NaCl and phosphates improve cooking losses and tenderness (Young and Lyon, 1997; Lemos et al., 1999; Alvarado and McKee, 2007; Petracci et al., 2012).

The present experiments indicate that the oxidative stability of marinated breast fillets was affected through 7 and 12d of refrigerated storage. It appears that from these results, the application of a marinade solution with a basic pH (>7) may increase the susceptibility for lipid peroxidation in marinated chicken breasts. This hypothesis was based on the fact that the highest TBARS response (mg of MDA equivalents/kg meat) at 7d for the marinated breast fillets was 3.8- and 8.4-fold greater that for the raw breast fillets in Exp. 1 and 2, respectively; however, statistical comparisons were not conducted comparing the raw and marinated portions of these experiments. Few studies have evaluated the oxidative stability of marinated meat products. Wettasinghe and Shahidi (1997) reported that the addition of NaCl during processing increased lipid oxidation in cooked pork patties, suggesting that NaCl promotes the release of iron ions from heme-compounds, which have been known to catalyze lipid oxidation. In contrast, early studies suggest that marination with phosphates may decrease lipid oxidation in cooked and frozen leg (Brotsky, 1976; Ang and Young, 1987), and breast meat (Ang and Hamm, 1986). Ang and Hamm (1986) theorized that the antioxidant activity they observed from marination in a phosphate solution may be due to its function as a metal-sequestering agent. Nonetheless, our data suggest that marination in a solution containing NaCl and sodium pyrophosphate may increase oxidative conditions. Supplementation of Se yeast and Vit.E (alone or in combination) effectively reduced the susceptibility for lipid oxidation through 7d of storage. The synergistic relationship between Se and Vit.E in the avian antioxidant system and their ability to reduce lipid oxidation was especially apparent after 7d of storage, when the combination of Se yeast and Vit.E resulted in the greatest protection against oxidative damage.

In conclusion, supplementing broiler diets with Se yeast effectively increased the deposition of Se in broiler breast fillets. The oxidative stability of raw breast fillets was improved through 7d of storage by dietary inclusion of Se yeast and Vit.E. The combination of 0.3 ppm Se yeast and 30 IU/kg Vit.E in broiler diets appeared to be most effective at protecting against lipid oxidation in marinated chicken breast fillets. The synergistic relationship between Se and Vit.E in the avian antioxidant system was especially apparent in the marinated breast fillets after 7d of storage, when the

combination of Se yeast and Vit.E resulted in the greatest protection against oxidative damage.

4.5. Tables and figures

Table 4.1. Composition of broiler basal diets for Exp. 1 and 2 (as-fed basis).

	% of diet	
Ingredients	Starter	Grower
Ground corn	54.82	57.47
Soybean meal (48% CP)	36.00	32.50
Choice white grease	4.80	5.55
Ground limestone	1.50	1.20
Dicalcium phosphate	1.70	2.00
Salt	0.48	0.46
Vitamin mix[1]	0.25	0.25
Trace mineral mix[2]	0.25	0.25
L-lysine	–	0.10
DL-methionine	0.20	0.22
Total	100.00	100.00
Calculated nutrient composition[3]		
ME, kcal/kg	3,090	3,154
Crude protein, %	22.23	20.77
Lysine, %	1.25	1.22
Methionine, %	0.55	0.55
TSAA, %	0.90	0.88
Threonine, %	0.87	0.81
Tryptophan, %	0.27	0.25
Calcium, %	1.05	1.00
Phosphorus (available), %	0.45	0.50
Selenium, ppm	0.05	0.05
Vitamin E, mg/kg	13.14	13.62

[1] Supplied per kilogram of diet: Vitamin A, 8,000 IU; Vitamin D_3, 3,000 IU; Vitamin K, 3 mg; Thiamin, 2 mg; Riboflavin, 10 mg; Pantothenic acid, 12 mg; Niacin, 40 mg; Pyridoxine, 4 mg; Biotin, 0.2 mg; Folic acid, 0.75 mg; Vitamin B-12, 0.015 mg; Choline, 500 mg; Ethoxyquin, 125 mg.
[2] Supplied per kilogram of diet: Copper, 24 mg; Iodine, 0.89 mg; Manganese, 281.35 mg; Zinc, 83.33 mg; Cobalt, 20.19 mg.
[3] Calculated based on the NRC (1994) estimated values.

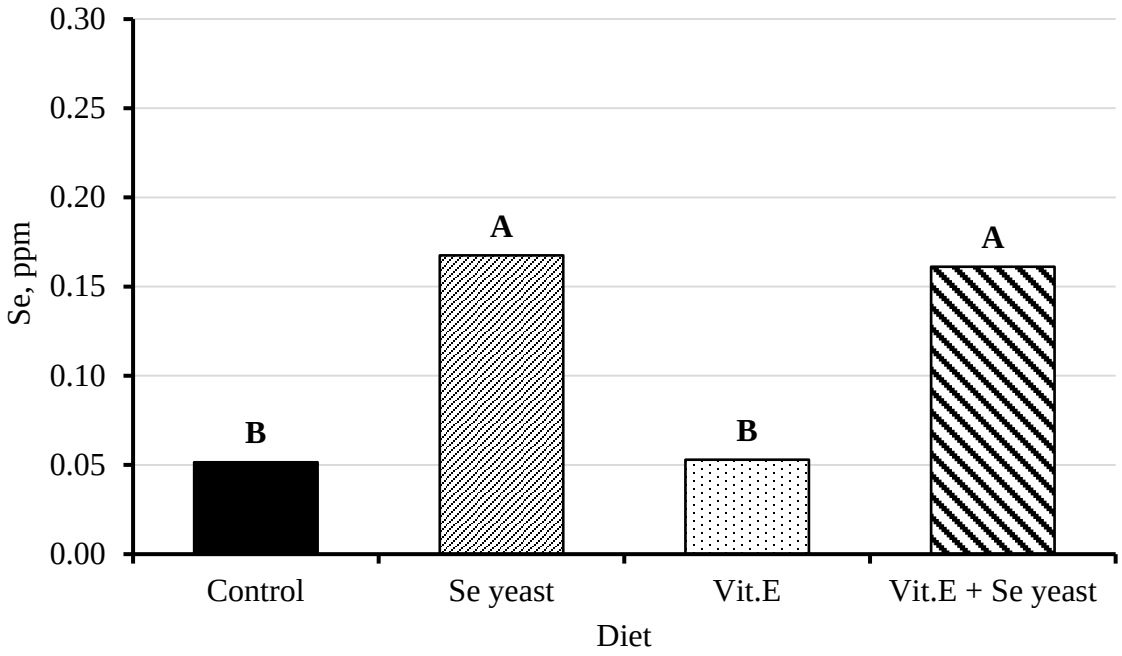

Figure 4.1. Breast Se (ppm) concentration in Exp. 1 (12 pens/treatment, n = 1 breast/pen). Means without a common letter differ significantly ($P < 0.01$). A significant main effect was observed for Se supplementation ($P < 0.01$).

Table 4.2. Carcass yields of harvested broilers in Exp. 1.[1]

Item	Diet				SEM	*P*-value		
	Control	Se yeast	Vit.E	Vit.E + Se yeast		Se	Vit.E	Se*Vit.E
WOG, %[2]	75.64	76.51	76.42	75.94	0.50	0.70	0.84	0.19
Front half, %[3]	47.80	48.02	47.88	47.51	0.37	0.84	0.57	0.44
Saddle, %[3]	27.84	28.50	28.54	28.43	0.33	0.42	0.36	0.26

[1] Values represent the average of 12 pens/treatment with 3 birds sampled/pen.
[2] Weight without giblets: carcass weight expressed as a percentage of the live weight.
[3] Expressed as a percentage of the live weight.

Table 4.3. Cooked characteristics of raw breast fillets in Exp. 1.[1]

Item	Diet				SEM	*P*-value		
	Control	Se yeast	Vit.E	Vit.E + Se yeast		Se	Vit.E	Se*Vit.E
Cooking loss, %[2]	19.52	19.13	19.95	19.24	1.11	0.62	0.81	0.89
Shear force, kg	1.44	1.47	1.36	1.30	0.09	0.89	0.17	0.58

[1] Values represent the average of 12 pens/treatment (n = 1 breast /pen).
[2] Expressed as a percentage of the uncooked breast fillet weight.

Table 4.4. Drip loss and TBARS values of raw breast fillets through 7 d of storage in Exp. 1.[1]

	Diet					*P*-value		
Item	Control	Se yeast	Vit.E	Vit.E + Se yeast	SEM	Se	Vit.E	Se*Vit.E
Drip loss, %[2]								
d 3	2.25[a]	1.66[b]	2.60[a]	1.88[b]	0.22	<0.01	0.20	0.76
d 7	3.92[a]	3.20[b]	4.60[a]	3.31[b]	0.35	<0.01	0.27	0.42
TBARS[2]								
d 0	0.002	0.003	0.003	0.002	0.001	0.85	0.72	0.30
d 2	0.009[ab]	0.006[ab]	0.010[a]	0.006[b]	0.001	0.02	0.68	0.57
d 5	0.014[a]	0.013[ab]	0.010[b]	0.010[b]	0.001	0.53	<0.01	0.83
d 7*	0.031[a]	0.022[ab]	0.025[ab]	0.017[b]	0.003	0.03	0.12	0.89

[a-b] Means within the same row with different superscript letters differ significantly ($P < 0.05$).

* $P = 0.06$.

[1] Values represent the average of 12 pens/treatment (n = 1 breast /pen).

[2] Drip loss expressed as a percentage of the initial weight (d 0).

[3] TBARS values are expressed as mg MDA/kg meat.

Table 4.5. Color stability of raw breast fillets through 7 d of storage in Exp. 1.[1]

	Diet					*P*-value		
Item	Control	Se yeast	Vit.E	Vit.E + Se yeast	SEM	Se	Vit.E	Se*Vit.E
L* Lightness								
d 0	62.30	63.61	62.32	62.95	0.91	0.30	0.73	0.71
d 2	64.74	66.13	65.08	65.34	0.66	0.22	0.73	0.40
d 5	63.06	64.52	63.37	63.46	0.77	0.32	0.63	0.38
d 7	61.25	63.51	62.03	63.08	0.77	0.04	0.82	0.44
a* Redness								
d 0	7.44	7.00	7.45	7.80	0.36	0.90	0.26	0.29
d 2	7.05	6.42	7.01	7.21	0.29	0.46	0.21	0.17
d 5	6.87	6.28	6.69	7.03	0.33	0.72	0.41	0.17
d 7	6.87	6.36	6.68	6.89	0.29	0.61	0.55	0.22
b* Yellowness								
d 0	20.00	19.27	19.97	20.31	0.38	0.62	0.20	0.17
d 2	18.94	18.73	18.89	19.09	0.33	1.00	0.64	0.54
d 5	18.98	19.02	19.12	19.60	0.36	0.48	0.33	0.55
d 7	20.00	19.21	19.49	19.83	0.33	0.50	0.87	0.10

[1] Values represent the average of 12 pens/treatment (n = 1 breast /pen).

Table 4.6. Breast pH and cooked characteristics of the marinated breast fillets in Exp. 1.[1]

Item	Diet				SEM	*P*-value		
	Control	Se yeast	Vit.E	Vit.E + Se yeast		Se	Vit.E	Se*Vit.E
pH								
Pre-marination	5.82	5.83	5.82	5.80	0.04	0.83	0.65	0.71
Post-marination	6.10	6.03	6.04	6.05	0.03	0.51	0.59	0.29
Cooking loss, %[2]	17.05	15.27	16.37	15.32	0.66	0.04	0.64	0.58
Shear force, kg	1.25	1.24	1.39	1.30	0.08	0.54	0.25	0.60

[1] Values represent the average of 12 pens/treatment (n = 1 breast /pen).
[2] Expressed as a percentage of the uncooked breast fillet weight.

Table 4.7. Drip loss and TBARS values of marinated breast fillets through 7 d of storage in Exp. 1.[1]

Item	Diet				SEM	*P*-value		
	Control	Se yeast	Vit.E	Vit.E + Se yeast		Se	Vit.E	Se*Vit.E
Drip loss, %[2]								
d 3	0.43	0.51	0.52	0.41	0.08	0.81	0.93	0.24
d 7	0.96	0.97	1.02	0.89	0.12	0.62	0.93	0.55
TBARS[2]								
d 0	0.002	0.003	0.003	0.002	0.001	0.85	0.72	0.30
d 2	0.019^{A}	0.010^{B}	0.004^{B}	0.004^{B}	0.003	0.08	<0.01	0.09
d 5	0.054^{A}	0.029^{B}	0.016^{B}	0.016^{B}	0.006	0.04	<0.01	0.03
d 7	0.118^{A}	0.062^{B}	0.050^{BC}	0.026^{C}	0.009	<0.01	<0.01	0.09

[A-B] Means within the same row with different superscript letters differ significantly ($P < 0.01$).
[1] Values represent the average of 12 pens/treatment (n = 1 breast /pen).
[2] Drip loss expressed as a percentage of the initial weight (d 0).
[3] TBARS values are expressed as mg MDA/kg meat.

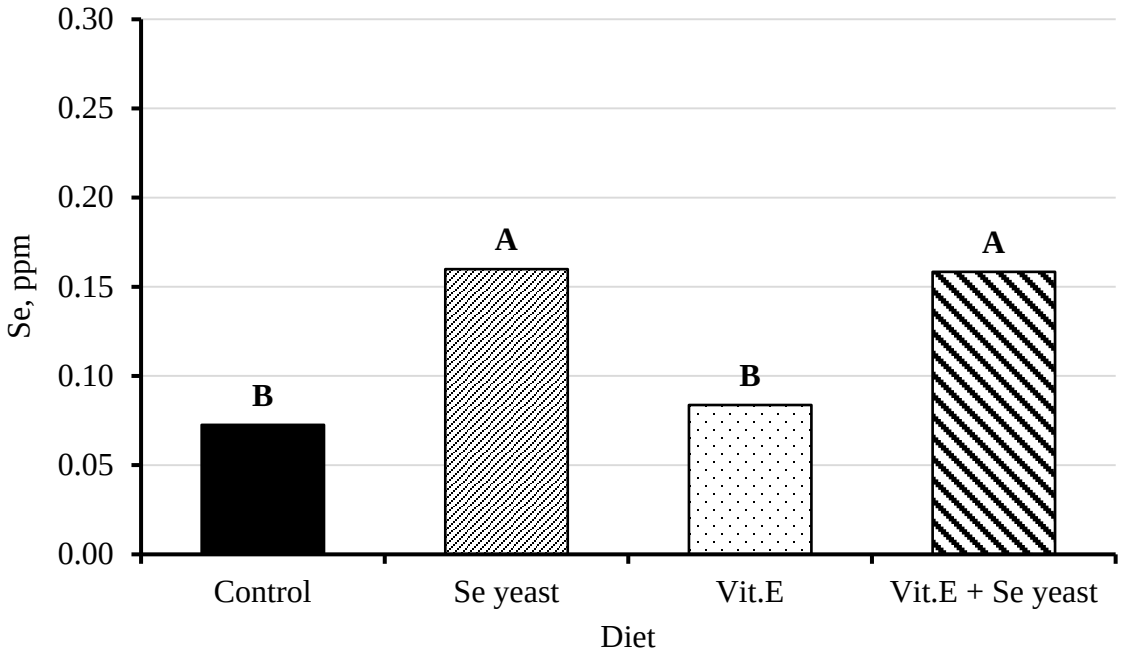

Figure 4.2. Breast Se (ppm) concentration in Exp. 2 (8 pens/treatment, n = 1 breast/pen). Means without a common letter differ significantly ($P < 0.01$). A significant main effect was observed for Se supplementation ($P < 0.01$).

Table 4.8. Carcass yields of harvested broilers in Exp. 2.[1]

Item	Diet				SEM	*P*-value		
	Control	Se yeast	Vit.E	Vit.E + Se yeast		Se	Vit.E	Se*Vit.E
WOG, %[2]	74.67	75.13	74.51	74.76	0.32	0.29	0.43	0.75
Front half, %[3]	44.62	45.65	44.73	45.55	0.37	0.03	0.99	0.78
Saddle, %[3]	30.05	29.47	29.78	29.21	0.24	0.03	0.29	0.99

[1] Values represent the average of 8 pens/treatment with 2 birds sampled/pen.
[2] Weight without giblets: carcass weight expressed as a percentage of the live weight.
[3] Expressed as a percentage of the live weight.

Table 4.9. Drip loss and TBARS values of raw breast fillets through 12d of storage in Exp. 2.[1]

Item	Diet				SEM	*P*-value		
	Control	Se yeast	Vit.E	Vit.E + Se yeast		Se	Vit.E	Se*Vit.E
Drip loss, %[2]								
d 3	0.81	0.79	1.23	0.98	0.16	0.41	0.09	0.51
d 7	2.75	2.34	3.23	2.43	0.45	0.20	0.53	0.67
TBARS[2]								
d 0	0.005	0.004	0.004	0.006	0.001	0.74	0.52	0.20
d 5	0.012	0.012	0.009	0.010	0.002	0.72	0.21	0.72
d 7	0.017	0.017	0.014	0.016	0.002	0.83	0.44	0.66
d 10	0.027	0.029	0.021	0.025	0.004	0.51	0.29	0.82
d 12	0.023	0.026	0.019	0.020	0.003	0.48	0.12	0.73

[1] Values represent the average of 8 pens/treatment (n = 1 breast /pen).
[2] Drip loss expressed as a percentage of the initial weight (d 0).
[3] TBARS values are expressed as mg MDA/kg meat.

Table 4.10. Drip loss and TBARS values of marinated breast fillets through 12 d of storage in Exp. 2.[1]

	Diet					*P*-value		
Item	Control	Se yeast	Vit.E	Vit.E + Se yeast	SEM	Se	Vit.E	Se*Vit.E
Drip loss, %[2]								
d 3	0.80	0.45	0.63	0.48	0.10	0.03	0.48	0.35
d 7	1.22[a]	0.70[b]	0.89[ab]	0.71[b]	0.14	0.03	0.27	0.24
TBARS[2]								
d 0	0.009	0.009	0.009	0.009	0.001	0.48	0.78	0.81
d 5	0.105[A]	0.068[B]	0.037[C]	0.040[C]	0.007	0.04	<0.01	0.02
d 7	0.143[A]	0.090[B]	0.055[BC]	0.051[C]	0.012	0.04	<0.01	0.06
d 10	0.153[A]	0.102[B]	0.054[C]	0.048[C]	0.013	0.05	<0.01	0.12
d 12	0.214[A]	0.154[A]	0.071[B]	0.072[B]	0.021	0.18	<0.01	0.17

[a-b] Means within the same row with different superscript letters are different ($P = 0.07$).
[A-C] Means within the same row with different superscript letters differ significantly ($P < 0.01$).
[1] Values represent the average of 8 pens/treatment (n = 1 breast /pen).
[2] Drip loss expressed as a percentage of the initial weight (d 0).
[3] TBARS values are expressed as mg MDA/kg meat.

Table 4.11. Fatty acid composition of breast fillets in Exp. 2.[1]

Item	Diet				SEM	*P*-value		
	Control	Se yeast	Vit.E	Vit.E + Se yeast		Se	Vit.E	Se*Vit.E
Fat (by acid hydrolysis), %	1.27	1.30	1.37	1.09	0.18	0.51	0.75	0.42
Moisture, %	74.58	74.71	74.78	74.55	0.29	0.87	0.95	0.54
Fatty acid profile, % of total fat								
Myristic (14:0)	1.17	1.18	1.14	1.27	0.07	0.28	0.66	0.40
Myristoleic (14:1)	0.46	0.47	0.49	0.48	0.06	0.89	0.83	0.75
Pentadecanoic (15:0)	0.34	0.31	0.32	0.36	0.04	1.00	0.78	0.37
Palmitic (16:0)	18.30	18.68	18.08	19.06	0.73	0.37	0.91	0.69
Palmitoleic (16:1)	2.94	3.08	2.89	2.84	0.26	0.85	0.60	0.72
Heptadecanoic (17:0)	0.38	0.46	0.45	0.40	0.03	0.63	1.00	0.05
Heptadecenoic (17:1)	1.03	0.99	1.07	1.00	0.09	0.54	0.75	0.88
Stearic (18:0)	9.70	8.64	8.90	8.86	0.97	0.58	0.78	0.61
Elaidic (18:1*t*9)	0.96[b]	1.17[a]	1.13[a]	1.08[ab]	0.05	0.13	0.46	0.02
Oleic (18:1*n*9)	27.02	28.56	25.57	27.07	1.79	0.41	0.43	0.99
Linoleic (18:2)	12.22	12.63	12.26	11.24	0.79	0.71	0.41	0.39
Linolenic (ω18:3)	0.59	0.67	0.64	0.63	0.06	0.55	0.90	0.40
Arachidic (20:0)	0.17	0.20	0.23	0.18	0.03	0.83	0.44	0.15
Docosenoic (20:1*n*9)	0.42	0.33	0.37	0.38	0.05	0.49	0.99	0.31
Arachidonic (20:4*n*6)	4.55	4.21	4.23	4.13	0.57	0.70	0.73	0.83
Eicosapentaenoic (20:5 ω3; EPA)	0.32	0.25	0.32	0.35	0.05	0.64	0.32	0.33
Docosapentaenoic (22:5 ω3; DPA)	0.91	0.83	0.92	0.77	0.11	0.30	0.82	0.77
Docosahexaenoic (22:6 ω3; DHA)	0.79	0.69	0.74	0.67	0.11	0.45	0.75	0.92
Lignoceric (24:0)	0.17	0.16	0.18	0.19	0.03	0.91	0.39	0.80
Nervonic (24:1*n*9)	1.54	1.38	1.35	1.48	0.17	0.93	0.76	0.40

[a-b] Means within the same row with different superscript letters are different ($P = 0.06$).

[1] Values represent the average of 8 pens/treatment (n = 1 breast /pen).

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CHAPTER 5. Summary and conclusion

The research presented in this dissertation was conducted to evaluate maternal and progeny dietary supplementation of Se yeast and Vit.E. It has been well established in the previous literature that fortification of poultry diets with antioxidants such as Se and Vit.E can have beneficial effects at all stages of growth and reproduction, as well as promoting optimal meat quality characteristics in poultry meat products.

The purpose of the research in chapter 2 was to evaluate the effects of Se yeast and Vit.E supplementation in the diets of developing broiler breeder pullets on BW uniformity and tissue Se and Vit.E concentrations prior to the onset of lay. The second objective in this experiment was to evaluate the effects of dietary Se and Vit.E on the subsequent egg production performance of hens during the laying cycle. Dietary supplementation of Se or Vit.E to the developing pullets did not affect the overall BW uniformity throughout the developmental period. The average BW for the entire flock was well above the targeted BW during each week of development, with deviations between dietary treatments throughout the entire period with no distinct pattern explaining the variation. In the developing broiler breeder flock, it is important to maintain high flock uniformity during development and to achieve the target BW at the time of sexual maturity in order to promote optimum reproductive performance during the lay cycle.

Overall throughout the 40 week laying cycle, the hens supplemented with Vit.E alone in the diet had reduced HDP% compared to hens receiving the other treatment diets. Hens receiving dietary Se yeast did not differ in HDP% compared to the unsupplemented control. Consequently, HDP% in the post-peak production phase of the laying cycle was lower for all treatment diets compared to the targeted level of egg production. This may have been due to the fact that at the time of photostimulation, pullets were overweight (at least 180g above the target BW) and overall BW uniformity was below the “80%” required for a uniform flock. The combination of these two factors may have reduced HDP% in the later stages of the laying cycle. Nonetheless, Se yeast and Vit.E

supplementation during pullet development and the laying cycle did not improve production performance.

Selenium yeast supplementation in the pullet diets significantly elevated the Se concentrations in the liver, breast, and pancreas throughout the developmental period. Interestingly, liver Se concentrations were reduced between the time of photostimulation (21 weeks) and the onset of egg production (26 weeks) for all treatment groups. This is not surprising as the broiler breeder hen experiences intense development of the reproductive tract in the weeks preceding the onset of egg production, and the observed reduction in liver Se stores suggests that Se may have been mobilized to the hen's developing reproductive tract during this time. Nonetheless, Se yeast supplementation effectively improved the antioxidant status of breeder pullets at the beginning of the laying cycle, which may help maintain adequate tissue Se concentrations during the early stages of production. This is extremely important as the hen's plane of nutrition directly affects the concentration of nutrients deposited into the egg that become available to the developing embryo. Improving antioxidant status of the developing embryo through fortification of the maternal diets with antioxidants such as Se or Vit.E may improve the hatchability, survival, and subsequent health and performance of the progeny.

The purpose of the research in chapter 3 was to evaluate the effect of maternal and progeny dietary supplementation of Se and Vit.E on the maternal transfer of nutrients to the progeny as well as maternal and progeny dietary effects on the growth performance of the progeny. The results from the experiments in chapter 3 indicate that Se supplementation in maternal diets is an effective way of increasing the antioxidant status of the progeny at hatch as the progeny liver and breast Se concentrations were significantly higher than of progeny originated from unsupplemented hens. Previous reports in the literature have reported that the beneficial effects of maternal Se supplementation on progeny tissue Se concentrations persist through 3-4 weeks post-hatch. This was not the case in the experiments presented in chapter 3, as all maternal effects disappeared in the progeny after 7d of age. The effects of feeding progeny diets supplemented with Se appeared to rapidly overtake the beneficial effects of a high Se

maternal diet on progeny tissue Se concentrations in a little as 7d. Selenium supplementation in the progeny diets increased the liver and breast Se concentrations through 14d of age. It has been well established that there is a highly positive correlation between Se concentration and GSH-Px activity in poultry tissues. Therefore based on these results, it can be inferred that increasing tissue Se concentrations through supplementation of Se in either maternal or progeny diets may increase the antioxidant status of the progeny tissues. Previous research has indicated that dietary Se supplementation has a sparing effect on the Vit.E stores in poultry tissues, however, information is lacking in the literature regarding the effects of maternal Se supplementation on progeny Vit.E levels post-hatch. The results presented in chapter 3 did not indicate that maternal or progeny Se yeast supplementation affected progeny liver Vit.E concentrations at either 7 or 14d of age. Nonetheless, these experiments suggest that Se yeast supplementation in maternal and progeny diets effectively increases the tissue Se status of the offspring.

The purpose of the research presented in chapter 4 was to evaluate the effects of Se yeast and Vit.E supplementation in broiler diets on the subsequent meat quality characteristics of raw and marinated breast fillets. Previous research has indicated that feeding Se yeast and/or Vit.E in broiler diets improves the water holding capacity (drip loss), oxidative stability, and color stability of raw poultry meat during prolonged refrigerated storage. In chapter 4, the results indicated that Se yeast improved raw breast fillet drip loss at 3 and 7d of storage, and Se yeast and Vit.E supplementation improved oxidative stability through 7d of storage. Color stability however, was not affected by Se yeast or Vit.E treatments through 7d of storage. Overall, these results indicate that Se yeast can improve the water holding capacity and extend the shelf life of breast fillets by prolonging the onset of rancidity.

Marination is a technique widely utilized in the poultry industry to enhance the quality characteristics of poultry meat. Typical industry marinades contain a mixture of NaCl and phosphate salts, yielding a marinade solution with a basic pH (ph: 7-10). Despite the basic nature of NaCl and phosphates in marinade solutions, it is interesting to

note that information in the literature is lacking regarding the oxidative stability of uncooked marinated chicken meat under storage conditions. In addition, information is lacking regarding the effects of dietary antioxidant supplementation in broiler diets on the meat quality characteristics of marinated poultry meat. The results from chapter 4 suggest that marinating broiler breast fillets in a basic marinade solution may increase the susceptibility for lipid oxidation. In Exp. 1 and 2 in chapter 4, the response for lipid oxidation for the marinated breast fillets from the control treatment was approximately 4- and 7-fold higher, respectively, when compared to the unmarinated breast fillets. Supplementation of Se yeast and Vit.E (alone or in combination) in the broiler diets effectively reduced the susceptibility for lipid oxidation through 7 and 12d of storage in Exp. 1 and 2, respectively, compared to the unsupplemented control. These results suggest that Se yeast and Vit.E supplementation may significantly reduce the level of lipid oxidation in marinated poultry meat under commercial storage conditions, ultimately prolonging the onset of rancidity and extending shelf life. To date, this research is the first to suggest that marination of poultry meat in basic marinade solutions may increase the susceptibility to lipid oxidation, therefore this is an area of research that will required further investigation in the future.

Overall, the research presented in this dissertation illustrates the importance of dietary antioxidant supplementation throughout all life stages in poultry (from the developing broiler breeder hen, all the way to the meat quality of the progeny). Chapter 2 and 3 indicate that Se supplementation is important for improving the Se status of the broiler breeder hen, which has a direct impact on the Se status of the progeny. Antioxidant protection is not only important for reducing oxidative stress in the highly productive broiler breeder hen, but for the developing embryo as well because the process of embryo development and then hatching of the chick are times of high oxidative stress. The results from chapter 4 demonstrated that Se and Vit.E are required for maintaining optimal poultry meat quality, specifically water holding capacity and oxidative stability, during prolonged refrigerated storage in both raw and marinated breast fillets.

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Appendicies

Appendix 1. Microwave digestion and inductively coupled plasma mass spectrometry (ICP-MS) for selenium analysis in chapter 3 and 4.

Sample preparation guideline is according to U.S. EPA method and ICP-MS was operated by Dr. Jason Unrine from the Laboratory of Environmental Chemistry and Toxicology Analysis of University of Kentucky. All the tissue samples weighed and then placed in a freeze dryer (Labconco FreeZone Plus 6, Labconco Corp., Kansas City, MO) for 4 days to a constant weight by lyophilization. Upon removal from the freeze dryer, all samples were weighed once again for determination of dry matter content and placed into plastic bags. Samples then were finely ground using a mortar and pestle prior to Se analysis. Tissue Se concentration (dry matter basis) was determined using an inductively coupled plasma mass spectrometer (ICP-MS; Agilent 7500cx, Santa Clara, CA, USA). Prior to ICP-MS analysis, samples were digested in 10 mL nitric acid and 1 mL 30% concentrated hydrogen peroxide in sealed Teflon bombs using a microwave digestion system (MARSXpress, CEM Corporation, Matthews, NC) and appropriately diluted. A commercial standard, National Institute of Standards and Technology (NIST) SRM 1577c (bovine liver), was used as a reference material to assure accuracy of the obtained results.

Digestion procedure:

1. Obtain ground, freeze-dried sample
2. Weigh 0.25 g of sample into each digestion vessel and record sample weight and ID (total of 38 experimental samples, 1 NIST bovine liver, and one blank per sample run).
3. Add 10 mL of 70% trace mineral grade HNO_3 to each vessel and seal with the appropriate cap assembly. Place the carousel of sealed vessels into the microwave.
4. Select the digestion program “EPA method 3052” on the microwave and hit start. This method ramps the temperature up to 180 ºC over 30 minutes and this temperature is then held at 180 ºC for 10 minutes.

5. Once the digestion has completed, allow the samples to cool to a pressure less than 80 PSI and carefully remove the carousel of digestion tubes from the microwave and place in the fume hood.
6. Carefully vent and open each sample and add 1 mL of 30% to each tube. Seal each tube and place the carousel back into the microwave.
7. Select the same digestion program as in step 4 and hit start on the second round of digestion.
8. Repeat step 5.
9. Once the vessels have cooled, carefully vent and open each tube and transfer the solution into a 50 mL, pre-labeled centrifuge tube. Rinse each digestion vessel and cap with deionized water into the specified centrifuge tube. Dilute the sample to a weight of 50 g, record its exact weight, seal and place the tube in storage.
10. Place empty digestion vessels into the sink and wash immediately according to the cleaning procedures required by the laboratory.

ICP-MS analysis:

Before analysis, the operating conditions of the ICP-MS instrument need to be optimized on a daily basis. In particular, the nebulizer gas flow rate, the ion lens voltage(s) and the ICP RF power must be adjusted to yield the highest signal intensities possible while still maintaining low levels of oxides and doubly-charged ion production (both should be less than ~ 3 %). Once the instrument has been optimized, appropriate interference standards should be analyzed to allow interference correction factors for molecular and doubly-charged ion interferences to be determined and entered into the instrument software. Finally, the unknown samples may be analyzed. The quality of the results may be evaluated on the basis of within-run statistics such as standard deviations, the reproducibility of repeat analysis, the measured analyte concentrations in total procedural blanks and the accuracy of results for any standard reference materials analyzed with the unknown samples.

Inductively coupled plasma mass spectrometry (Agilent 7500cx, Santa Clara, CA, USA) was employed for Se analysis and operated by Dr. Jason Unrine from Laboratory

of Environmental Chemistry and Toxicology Analysis of University of Kentucky and followed the ICP-MS protocol. The instrument allows for a maximum of 180 samples to be analyzed at one time. Samples were prepared by taking a 1 mL subsample of the diluted, acid-digested sample that was then added to at 15 mL centrifuge tube and diluted again to 4x (1 mL sample + 3 mL distilled de-ionized H_2O). Approximately 20 µL of internal standard was added to each sample as well. Duplicate samples were created every 30 samples. In addition, duplicate spiked samples were prepared which included a identical duplicate sample that was spiked with 5 µL of the initial calibration verification (ICV) Se standard.

Result:

- Calculate the Method Detect Limit (MDL; 3 times the standard deviation of procedural blanks).
- Calculate recovery rate for NIST, laboratory control sample, and average relative percentage of difference (RPD) for duplicate samples.
- Calculate spike recovery average.
- Calculate the sample mineral concentration.

Appendix 2. Tissue nutrient composition and growth performance tables in Chapters 2, 3, and 4.

Table A.2.1. Chick liver and breast Se concentrations (ppm) on 0, 7, and 14d of age in Exp. 1 of Chapter 3.[1]

	Liver Se, ppm			Breast Se, ppm		
Means	0d	7d	14d	0d	7d	14d
Maternal diet						
Control	0.93^{a}	0.71^{b}	0.87	0.55^{b}	0.63^{bc}	0.65
Se yeast	2.21^{a}	0.94^{A}	1.01	1.49^{a}	0.79^{a}	0.72
Vit.E	0.90^{b}	0.68^{B}	0.99	0.53^{b}	0.56^{c}	0.65
Vit.E + Se yeast	1.90^{a}	0.96^{A}	0.94	1.13^{a}	0.75^{ab}	0.66
SEM	0.14	0.07	0.06	0.07	0.05	0.03
Chick diet						
Control	–	0.60^{C}	0.50^{B}	–	0.55^{B}	0.38^{B}
Se yeast	–	1.15^{A}	1.35^{A}	–	0.86^{A}	0.99^{A}
Vit.E	–	0.60^{C}	0.48^{B}	–	0.53^{B}	0.37^{B}
Vit.E + Se yeast	–	0.94^{B}	1.49^{A}	–	0.79^{A}	0.94^{A}
SEM	–	0.07	0.06	–	0.05	0.03
	P-values (main effects)					
Maternal diet						
mSe	0.01	<0.01	0.46	<0.01	0.02	0.23
mVit.E	0.34	0.98	0.71	0.12	0.31	0.39
mSe*mVit.E	0.42	0.67	0.19	0.16	0.76	0.31
Chick diet						
cSe	–	<0.01	<0.01	–	<0.01	<0.01
cVit.E	–	0.15	0.35	–	0.39	0.37
cSe*cVit.E	–	0.14	0.24	–	0.70	0.46
Interactions						
mSe*cSe	–	0.83	0.90	–	0.87	0.97
mSe*cVit.E	–	0.08	0.03	–	0.08	0.14
mSe*cSe*cVit.E	–	0.94	0.02	–	0.64	0.50
mVit.E*cSe	–	0.88	0.53	–	0.49	0.16
mVit.E*cVit.E	–	0.67	0.37	–	0.41	0.79
mVit.E*cSe*cVit.E	–	0.39	0.75	–	0.69	0.59
mSe*mVit.E*cSe	–	0.70	0.04	–	0.79	0.29
mSe*mVit.E*cVit.E	–	0.72	0.66	–	0.38	0.56
mSe*mVit.E*cSe*cVit.E	–	0.50	0.33	–	0.45	0.19

[a-b] Means within the same column with different superscript letters are significantly different ($P < 0.05$).
[A-B] Means within the same column with different superscript letters are significantly different ($P < 0.01$).
[1] Day 0 values represent the mean of 3 samples/treatment; day 7 and 14 values represent the mean of 12 pens/treatment (n=1 sample/pen). Abbreviations: mSe, maternal dietary Se; mVit.E, maternal dietary Vit.E; cSe, chick dietary Se; cVit.E, chick dietary Vit.E.

Table A.2.2. Chick liver α-tocopherol concentrations (μg/g) on d 7 and 14 in Exp. 1 of Chapter 3.[1]

	Liver α-tocopherol, μg/g	
Means	7d	14d
Maternal diet		
Control	2.85	4.57
Se yeast	2.50	4.04
Vit.E	2.65	4.36
Vit.E + Se yeast	2.94	4.05
SEM	0.14	0.16
Chick diet		
Control	2.68	4.13
Se yeast	2.53	4.29
Vit.E	2.86	4.31
Vit.E + Se yeast	2.87	4.58
SEM	0.14	0.17
——— *P*-values (main effects) ———		
Maternal diet		
mSe	0.78	0.35
mVit.E	0.33	0.68
mSe*mVit.E	0.03	0.47
Chick diet		
cSe	0.61	0.23
cVit.E	0.08	0.20
cSe*cVit.E	0.57	0.75
Interactions		
mSe*cSe	0.64	0.33
mSe*cVit.E	0.87	0.42
mSe*cSe*cVit.E	0.25	0.01
mVit.E*cSe	0.76	0.97
mVit.E*cVit.E	0.64	0.69
mVit.E*cSe*cVit.E	0.35	0.82
mSe*mVit.E*cSe	0.27	0.91
mSe*mVit.E*cVit.E	0.84	0.81
mSe*mVit.E*cSe*cVit.E	0.78	0.04

[1] Values represent the mean of 12 pens/treatment (n=1 sample/pen).
Abbreviations: mSe, maternal dietary Se; mVit.E, maternal dietary Vit.E; cSe, chick dietary Se; cVit.E, chick dietary Vit.E.

Table A.2.3. Average daily gain (g/d) results through 49d of age of the broilers in Exp. 1 in Chapter 3.[1]

	Weekly averages							Phase averages		
Means	7d	14d	21d	28d	35d	42d	49d	0-21d	22-49d	0-49d
Maternal diet										
Control	13.63	34.70[b]	55.49	73.57	86.68	78.68	76.50	34.34	78.54	59.60
Se yeast	14.64	37.98[a]	57.27	74.99	85.51	81.03	77.83	36.37	79.66	61.11
Vit.E	14.70	36.29[ab]	57.87	75.59	85.03	72.11	72.66	36.01	75.90	58.81
Vit.E + Se yeast	13.64	35.05[b]	54.15	73.40	87.66	78.59	83.64	34.04	80.56	60.62
SEM	0.4	0.7	1.3	1.9	2.3	2.4	3.8	0.7	2.0	1.3
Chick diet										
Control	13.84	34.84[B]	54.84	74.26	87.73	77.91	81.11	34.19	79.90	60.31
Se yeast	14.00	36.16[B]	57.10	76.59	89.47	77.91	81.64	35.45	81.07	61.52
Vit.E	13.71	34.56[B]	55.64	73.57	85.07	76.32	72.52	34.40	76.45	58.42
Vit.E + Se yeast	15.07	38.45[A]	57.20	73.14	82.62	78.27	75.36	36.73	77.24	59.88
SEM	0.4	0.7	1.3	1.9	2.3	2.4	3.8	0.7	2.0	1.3
	P-values (main effects)									
Maternal diet										
mSe	0.96	0.38	0.50	0.83	0.79	0.20	0.26	0.97	0.33	0.39
mVit.E	0.95	0.56	0.79	0.90	0.93	0.19	0.85	0.73	0.76	0.73
mSe*mVit.E	0.10	0.08	0.08	0.33	0.49	0.52	0.37	0.07	0.55	0.94
Chick diet										
cSe	0.05	<0.01	0.14	0.61	0.88	0.69	0.66	0.02	0.63	0.32
cVit.E	0.21	0.17	0.72	0.28	0.05	0.80	0.06	0.30	0.08	0.19
cSe*cVit.E	0.11	0.08	0.79	0.46	0.37	0.69	0.77	0.45	0.92	0.93
Interactions										
mSe*cSe	0.55	0.26	0.62	0.73	0.83	0.36	0.52	0.41	0.56	0.75
mSe*cVit.E	0.69	0.50	0.58	0.82	0.62	0.11	0.03	0.56	0.08	0.10
mSe*cSe*cVit.E	0.19	0.07	0.24	0.75	0.15	0.95	0.75	0.12	0.62	0.95
mVit.E*cSe	0.56	0.94	0.51	0.89	0.64	0.48	0.49	0.82	0.73	0.72
mVit.E*cVit.E	0.12	0.46	0.39	0.44	0.38	0.75	0.62	0.64	0.42	0.42
mVit.E*cSe*cVit.E	0.04	0.30	0.86	0.64	0.83	0.53	0.49	0.56	0.71	0.85
mSe*mVit.E*cSe	0.80	0.96	0.60	0.60	0.73	0.78	0.18	0.64	0.59	0.56
mSe*mVit.E*cVit.E	0.91	0.88	0.76	0.82	0.28	0.68	0.69	0.80	0.99	0.94
mSe*mVit.E*cSe*cVit.E	0.11	0.37	0.85	0.15	0.67	0.33	0.97	0.41	0.47	0.66

[a-b] Means within the same column with different superscript letters are significantly different ($P < 0.05$).
[A-B] Means within the same column with different superscript letters are significantly different ($P < 0.01$).
[1] Represents the mean of 12 pens/treatment. Abbreviations: mSe, maternal dietary Se; n ıal dietary Vit.E; cSe, chick dietary Se; cVit.E, chick dietary Vit.E.

Table A.2.4. Average daily feed intake (g/d) results through 49d of age of the broilers in Exp. 1 in Chapter 3.[1]

	Weekly averages							Phase averages		
Means	7d	14d	21d	28d	35d	42d	49d	0-21d	22-49d	0-49d
Maternal diet										
Control	22.7	59.6	85.6	113.3	140.7	145.4	193.0	56.0	148.1	108.6
Se yeast	23.3	54.2	81.6	115.2	146.4	147.7	196.6	53.0	151.5	109.3
Vit.E	25.1	57.9	88.0	122.4	147.7	144.3	192.4	57.0	151.7	111.1
Vit.E + Se yeast	21.5	55.0	79.9	112.7	143.1	146.2	197.0	52.2	149.7	107.9
SEM	1.6	1.7	2.3	2.7	3.0	2.5	4.3	1.4	2.6	1.7
Chick diet										
Control	23.3	55.8	86.7	111.4[b]	140.8	144.4	195.4	55.3	148.0[ab]	108.3
Se yeast	22.2	55.8	80.9	118.6[ab]	147.9	148.5	199.0	53.0	153.5[a]	110.4
Vit.E	23.0	55.6	84.1	110.9[b]	139.7	141.5	187.2	54.2	144.8[b]	106.0
Vit.E + Se yeast	24.0	59.5	83.4	122.7[a]	149.6	149.2	197.2	55.6	154.7[a]	112.2
SEM	1.6	1.7	2.3	2.7	3.0	2.5	4.3	1.4	2.6	1.7
	P-values (main effects)									
Maternal diet										
mSe	0.34	0.06	0.23	0.36	0.91	0.69	0.62	0.15	0.90	0.75
mVit.E	0.83	0.80	0.93	0.44	0.71	0.80	0.99	0.97	0.86	0.89
mSe*mVit.E	0.21	0.51	0.67	0.19	0.32	0.96	0.95	0.70	0.62	0.62
Chick diet										
cSe	1.00	0.26	0.17	<0.01	<0.01	0.03	0.13	0.76	<0.01	0.02
cVit.E	0.64	0.31	0.98	0.51	0.92	0.67	0.25	0.56	0.71	0.89
cSe*cVit.E	0.52	0.26	0.28	0.40	0.64	0.47	0.47	0.20	0.41	0.23
Interactions										
mSe*cSe	0.33	0.03	0.35	0.28	0.55	0.91	0.94	0.08	0.65	0.30
mSe*cVit.E	0.34	0.31	0.98	0.33	0.33	0.17	0.27	0.43	0.19	0.37
mSe*cSe*cVit.E	0.11	0.98	0.67	0.92	0.49	0.63	0.47	0.39	0.85	0.89
mVit.E*cSe	0.92	0.55	0.21	0.48	0.88	0.70	0.86	0.36	0.98	0.72
mVit.E*cVit.E	0.77	0.28	0.15	0.78	0.97	0.12	0.02	0.25	0.16	0.10
mVit.E*cSe*cVit.E	0.48	0.43	0.30	0.87	0.86	0.50	0.24	0.53	0.51	0.71
mSe*mVit.E*cSe	0.83	0.74	0.02	0.45	0.12	0.28	0.92	0.13	0.34	0.17
mSe*mVit.E*cVit.E	0.12	0.19	0.17	0.78	0.58	0.94	0.72	0.71	0.95	0.94
mSe*mVit.E*cSe*cVit.E	0.66	0.68	0.92	0.48	0.05	0.09	0.42	0.96	0.14	0.19

[a-b] Means within the same column with different superscript letters are significantly different ($P < 0.05$).

[1] Represents the mean of 12 pens/treatment. Abbreviations: mSe, maternal dietary Se; mVit.E, maternal dietary Vit.E; cSe, chick dietary Se; cVit.E, chick dietary Vit.E.

Table A.2.5. Gain:feed results through 49d of age of the broilers in Exp. 1 in Chapter 3.[1]

	Weekly							Phase averages		
Means	7d	14d	21d	28d	35d	42d	49d	0-21d	22-49d	0-49d
Maternal diet										
Control	0.63	0.59[b]	0.66	0.65	0.62	0.54	0.39	0.62	0.53[A]	0.55[ab]
Se yeast	0.65	0.71[a]	0.70	0.65	0.59	0.55	0.40	0.69	0.53[A]	0.56[a]
Vit.E	0.61	0.63[b]	0.67	0.62	0.58	0.50	0.38	0.64	0.50[B]	0.53[b]
Vit.E + Se yeast	0.66	0.64[ab]	0.68	0.65	0.61	0.54	0.42	0.66	0.54[A]	0.56[a]
SEM	0.04	0.03	0.02	0.01	0.01	0.01	0.02	0.02	0.01	0.01
Chick diet										
Control	0.62	0.63	0.64	0.67[A]	0.62[A]	0.54	0.41	0.63	0.54[A]	0.56[A]
Se yeast	0.65	0.66	0.71	0.65[A]	0.61[A]	0.52	0.41	0.67	0.53[A]	0.56[A]
Vit.E	0.63	0.63	0.67	0.66[A]	0.61[A]	0.54	0.38	0.64	0.53[A]	0.55[AB]
Vit.E + Se yeast	0.65	0.65	0.69	0.60[B]	0.55[B]	0.52	0.38	0.66	0.50[B]	0.53[B]
SEM	0.04	0.03	0.02	0.01	0.01	0.01	0.02	0.02	0.01	0.01
	P-values (main effects)									
Maternal diet										
mSe	0.34	0.03	0.32	0.14	0.87	0.09	0.10	0.13	<0.01	0.03
mVit.E	0.89	0.48	0.78	0.23	0.64	0.07	0.57	0.66	0.12	0.35
mSe*mVit.E	0.74	0.05	0.64	0.18	0.02	0.21	0.19	0.37	<0.01	0.22
Chick diet										
cSe	0.60	0.37	0.02	<0.01	<0.01	0.27	0.84	0.12	<0.01	0.25
cVit.E	0.90	0.81	0.86	<0.01	<0.01	0.99	0.05	0.86	<0.01	0.05
cSe*cVit.E	0.85	0.98	0.18	0.02	0.10	1.00	0.90	0.60	0.21	0.22
Interactions										
mSe*cSe	0.85	0.45	0.15	0.82	0.92	0.37	0.57	0.29	0.30	0.99
mSe*cVit.E	0.92	0.57	0.39	0.89	0.44	0.33	0.08	0.59	0.11	0.43
mSe*cSe*cVit.E	0.17	0.96	0.94	0.85	0.07	0.74	0.92	0.60	0.86	0.79
mVit.E*cSe	0.52	0.13	0.22	0.22	0.94	0.35	0.29	0.17	0.19	0.09
mVit.E*cVit.E	0.90	0.19	0.61	0.85	0.89	0.56	0.93	0.42	0.83	0.77
mVit.E*cSe*cVit.E	0.71	0.60	0.49	0.34	0.69	0.69	0.59	0.73	0.75	0.69
mSe*mVit.E*cSe	0.80	0.95	0.01	0.95	0.05	0.68	0.06	0.22	0.03	0.02
mSe*mVit.E*cVit.E	0.16	0.28	0.29	0.84	0.46	0.60	0.65	0.68	0.82	0.88
mSe*mVit.E*cSe*cVit.E	0.38	0.72	0.83	0.13	0.16	0.98	0.51	0.55	0.55	0.41

[a-b] Means within the same column with different superscript letters are significantly different ($P < 0.05$).
[A-B] Means within the same column with different superscript letters are significantly different ($P < 0.01$).
[1] Represents the mean of 12 pens/treatment. Abbreviations: mSe, maternal dietary Se; mVit.E, maternal dietary Vit.E; cSe, chick dietary Se; cVit.E, chick dietary Vit.E.

Table A.2.6. Chick liver and breast Se concentrations (ppm) on 0, 7, and 14d of age in Exp. 2 of Chapter 3.[1]

	Liver Se, ppm			Breast Se, ppm		
Means	0d	7d	14d	0d	7d	14d
Maternal diet						
Control	0.80^{B}	0.56^{B}	1.17^{ab}	0.61^{B}	0.56	0.70
Se yeast	2.41^{A}	0.66^{B}	1.28^{a}	1.41^{A}	0.71	0.73
Vit.E	0.66^{B}	0.60^{B}	0.97^{c}	0.65^{B}	0.65	0.62
Vit.E + Se yeast	2.49^{A}	0.95^{A}	1.09^{bc}	1.46^{A}	0.75	0.65
SEM	0.06	0.06	0.06	0.08	0.05	0.04
Chick diet						
Control	–	0.55^{B}	0.51^{B}	–	0.47^{B}	0.37^{B}
Se yeast	–	0.88^{A}	1.66^{A}	–	0.88^{A}	0.92^{A}
Vit.E	–	0.41^{B}	0.54^{B}	–	0.46^{B}	0.43^{B}
Vit.E + Se yeast	–	0.93^{A}	1.81^{A}	–	0.85^{A}	0.98^{A}
SEM	–	0.06	0.06	–	0.05	0.04
	P-values (main effects)					
Maternal diet						
mSe	<0.01	0.08	0.42	<0.01	<0.01	0.68
mVit.E	0.69	0.17	0.21	0.62	<0.01	0.29
mSe*mVit.E	0.13	0.25	0.95	0.95	<0.01	0.96
Chick diet						
cSe	–	<0.01	<0.01	–	<0.01	<0.01
cVit.E	–	0.52	0.15	–	0.67	0.20
cSe*cVit.E	–	0.12	0.31	–	0.82	0.94
Interactions						
mSe*cSe	–	0.14	0.80	–	0.97	0.55
mSe*cVit.E	–	0.66	0.03	–	0.86	0.09
mSe*cSe*cVit.E	–	0.21	0.05	–	0.30	0.54
mVit.E*cSe	–	0.03	0.02	–	0.23	0.13
mVit.E*cVit.E	–	0.04	0.81	–	0.86	0.84
mVit.E*cSe*cVit.E	–	0.15	0.77	–	0.28	0.95
mSe*mVit.E*cSe	–	0.34	0.11	–	0.51	0.54
mSe*mVit.E*cVit.E	–	0.02	0.78	–	0.85	0.15
mSe*mVit.E*cSe*cVit.E	–	0.14	0.87	–	0.67	0.37

[a-b] Means within the same column with different superscript letters are significantly different ($P < 0.05$).
[A-B] Means within the same column with different superscript letters are significantly different ($P < 0.01$).
[1] Day 0 values represent the mean of 2 samples/treatment; day 7 and 14 values represent the mean of 8 pens/treatment (n=1 sample/pen).
Abbreviations: mSe, maternal dietary Se; mVit.E, maternal dietary Vit.E; cSe, chick dietary Se; cVit.E, chick dietary Vit.E.

Table A.2.7. Average daily gain (g/d) results through 49d of age of the broilers in Exp. 2 in Chapter 3.[1]

	Weekly averages							Phase averages		
Diet	7d	14d	21d	28d	35d	42d	49d	0-21d	22-49d	0-49d
Maternal diet										
Control	13.97	28.49^{b}	52.29	75.26	75.24	75.79^{A}	34.62	31.58	65.23^{a}	50.81^{ab}
Se yeast	14.99	33.45^{a}	54.20	76.36	79.50	81.61^{A}	38.04	34.21	68.88^{a}	54.02^{a}
Vit.E	14.60	31.83^{a}	58.49	77.47	75.77	63.18^{B}	18.92	34.97	58.84^{b}	48.61^{b}
Vit.E + Se yeast	14.80	32.46^{a}	54.55	79.23	80.32	75.43^{A}	35.21	33.94	67.55^{a}	53.14^{a}
SEM	0.39	0.96	1.94	2.96	2.01	2.64	5.75	1.01	1.99	1.29
Chick diet										
Control	14.45	29.84^{B}	51.87	74.66	77.41	72.01	26.65	32.05^{b}	62.68	49.55
Se yeast	14.50	33.54^{A}	58.10	78.38	78.02	77.59	32.28	35.38^{a}	66.57	53.20
Vit.E	14.41	29.15^{B}	52.61	74.67	76.65	74.62	34.59	32.06^{b}	65.13	50.96
Vit.E + Se yeast	15.00	33.69^{A}	56.94	80.61	78.75	71.79	33.28	35.21^{a}	66.11	52.87
SEM	0.4	1.0	1.9	3.0	2.0	2.6	5.8	1.0	2.0	1.3
				P-values (main effects)						
Maternal diet										
mSe	0.14	0.03	0.62	0.42	0.09	0.07	0.10	0.37	0.07	0.09
mVit.E	0.53	0.18	0.17	0.19	0.73	0.07	0.12	0.13	0.17	0.40
mSe*mVit.E	0.27	0.05	0.21	0.84	0.94	0.41	0.22	0.10	0.33	0.70
Progeny diet										
cSe	0.42	<0.01	0.02	0.13	0.51	0.61	0.71	0.01	0.25	0.05
cVit.E	0.57	0.79	0.92	0.71	1.00	0.56	0.45	0.94	0.63	0.69
cSe*cVit.E	0.51	0.67	0.63	0.71	0.72	0.14	0.56	0.93	0.48	0.51
Interactions										
mSe*cSe	0.94	0.68	0.04	0.37	0.45	0.04	0.35	0.13	0.21	0.54
mSe*cVit.E	0.47	0.36	0.99	0.86	0.02	0.28	0.13	0.69	0.94	0.84
mSe*cSe*cVit.E	0.57	0.95	0.67	0.72	0.62	0.73	0.67	0.74	0.94	0.96
mVit.E*cSe	0.45	0.27	0.62	0.36	0.67	0.03	0.89	0.89	0.81	0.87
mVit.E*cVit.E	0.94	0.46	0.54	0.37	0.50	0.35	0.20	0.52	0.46	0.66
mVit.E*cSe*cVit.E	0.46	0.95	0.43	0.37	0.97	0.22	0.97	0.53	0.91	0.91
mSe*mVit.E*cSe	0.73	0.09	0.97	0.57	0.97	0.03	0.89	0.61	0.27	0.25
mSe*mVit.E*cVit.E	0.28	0.82	0.53	0.16	0.36	0.20	0.65	0.64	0.64	0.80
mSe*mVit.E*cSe*cVit.E	0.16	0.23	0.47	0.16	0.68	0.05	0.11	0.31	0.15	0.34

[a-b] Means within the same column with different superscript letters are significantly different ($P < 0.05$).

[A-B] Means within the same column with different superscript letters are significantly different ($P < 0.01$).

[1] Represents the mean of 8 pens/treatment. Abbreviations: mSe, maternal dietary Se; mVit.E, maternal dietary Vit.E; cSe, chick dietary Se; cVit.E, chick dietary Vit.E.

Table A.2.8. Average daily feed intake (g/d) results through 49d of age of the broilers in Exp. 2 in Chapter 3.[1]

	Weekly averages							Phase averages		
Means	7d	14d	21d	28d	35d	42d	49d	0-21d	22-49d	0-49d
Maternal diet										
Control	20.9	75.8^{A}	87.1	127.5	129.0	166.8	128.8^{ab}	61.2	138.0	105.1
Se yeast	22.0	64.9^{B}	83.8	128.1	137.0	170.3	146.3^{a}	56.9	145.4	107.5
Vit.E	21.4	74.3^{A}	87.3	132.9	135.2	150.2	107.8^{b}	61.0	131.5	101.3
Vit.E + Se yeast	21.6	64.1^{B}	88.6	136.6	142.8	161.4	131.7^{ab}	58.1	143.1	106.7
SEM	0.4	2.4	2.9	5.4	5.1	6.6	8.0	1.5	4.5	2.8
Chick diet										
Control	21.0	73.9	86.2	130.5	131.9	169.5	124.9	60.4	139.2	105.4
Se yeast	21.6	67.0	83.4	127.9	135.3	162.9	131.0	57.4	139.3	104.2
Vit.E	21.9	70.7	90.0	132.9	136.7	157.5	127.0	60.9	138.6	105.3
Vit.E + Se yeast	21.4	67.4	87.1	133.9	140.2	158.7	131.7	58.6	141.1	105.8
SEM	0.4	2.4	2.9	5.4	5.1	6.6	8.0	1.5	4.5	2.8
	P-values (main effects)									
Maternal diet										
mSe	0.38	0.11	0.89	0.57	0.05	0.18	0.02	0.29	0.03	0.19
mVit.E	0.95	0.83	0.73	0.14	0.10	0.06	0.03	0.88	0.17	0.39
mSe*mVit.E	0.50	0.94	0.76	0.68	0.95	0.43	0.56	0.81	0.45	0.56
Chick diet										
cSe	0.82	0.06	0.35	0.88	0.52	0.69	0.52	0.10	0.77	0.90
cVit.E	0.38	0.58	0.23	0.46	0.36	0.24	0.86	0.55	0.90	0.81
cSe*cVit.E	0.15	0.47	0.99	0.76	1.00	0.57	0.93	0.78	0.79	0.76
Interactions										
mSe*cSe	0.51	0.00	0.75	0.92	0.99	0.53	0.70	0.04	0.92	0.56
mSe*cVit.E	0.35	0.01	0.53	0.39	0.08	0.03	0.51	0.07	0.06	0.04
mSe*cSe*cVit.E	0.17	0.04	0.12	0.34	0.54	0.67	0.79	0.03	0.45	0.24
mVit.E*cSe	0.35	0.26	0.50	0.37	0.38	0.39	0.48	0.32	0.91	0.75
mVit.E*cVit.E	0.51	0.06	0.93	0.89	0.89	0.89	0.73	0.33	0.90	0.74
mVit.E*cSe*cVit.E	0.19	0.13	0.91	0.58	0.56	0.24	0.54	0.50	0.30	0.28
mSe*mVit.E*cSe	0.02	0.69	0.08	0.60	0.58	0.06	0.17	0.21	0.10	0.09
mSe*mVit.E*cVit.E	0.07	0.10	0.38	0.86	0.87	0.72	0.76	0.61	0.92	0.98
mSe*mVit.E*cSe*cVit.E	0.60	0.09	0.10	0.88	0.42	0.84	0.21	0.04	0.44	0.25

[a-b] Means within the same column with different superscript letters are significantly different ($P < 0.05$).

[A-B] Means within the same column with different superscript letters are significantly different ($P < 0.01$).

[1] Represents the mean of 8 pens/treatment. Abbreviations: mSe, maternal dietary Se; mVit.E, maternal dietary Vit.E; cSe, chick dietary Se; cVit.E, chick dietary Vit.E.

Table A.2.9. Gain:feed results through 49d of age of the broilers in Exp. 2 in Chapter 3.[1]

	Weekly averages							Phase averages		
Diet	7d	14d	21d	28d	35d	42d	49d	0-21d	22-49d	0-49d
Maternal diet										
Control	0.67	0.39^{B}	0.61	0.59	0.58	0.46	0.27	0.52^{b}	0.48	0.49
Se yeast	0.68	0.52^{A}	0.65	0.59	0.58	0.48	0.26	0.60^{a}	0.47	0.50
Vit.E	0.68	0.44^{B}	0.67	0.58	0.56	0.42	0.17	0.58^{a}	0.45	0.48
Vit.E + Se yeast	0.69	0.52^{A}	0.62	0.59	0.57	0.47	0.25	0.59^{a}	0.47	0.50
SEM	0.02	0.02	0.02	0.01	0.01	0.02	0.04	0.01	0.01	0.01
Chick diet										
Control	0.69	0.44^{B}	0.60^{b}	0.57^{b}	0.59	0.43	0.19	0.54^{B}	0.45	0.47^{b}
Se yeast	0.67	0.51^{A}	0.70^{a}	0.61^{a}	0.58	0.48	0.24	0.62^{A}	0.48	0.51^{a}
Vit.E	0.66	0.42^{B}	0.60^{b}	0.57^{b}	0.56	0.47	0.27	0.53^{B}	0.47	0.49^{ab}
Vit.E + Se yeast	0.70	0.51^{A}	0.66^{ab}	0.60^{ab}	0.56	0.45	0.25	0.60^{A}	0.47	0.50^{a}
SEM	0.02	0.02	0.02	0.01	0.01	0.02	0.04	0.01	0.01	0.01
				P-values (main effects)						
Maternal diet										
mSe	0.78	0.06	0.87	0.91	0.64	0.14	0.24	0.08	0.23	0.09
mVit.E	0.76	0.54	0.60	0.61	0.03	0.16	0.14	0.29	0.16	0.60
mSe*mVit.E	0.88	0.56	0.30	0.96	0.50	0.45	0.16	0.16	0.22	0.89
Chick diet										
cSe	0.46	<0.01	<0.01	<0.01	0.67	0.34	0.65	<0.01	0.22	<0.01
cVit.E	0.97	0.51	0.38	0.61	0.24	0.58	0.26	0.47	0.55	0.77
cSe*cVit.E	0.12	0.62	0.36	0.78	0.76	0.07	0.33	0.72	0.20	0.17
Interactions	0.62	0.02	0.06	0.20	0.36	0.01	0.40	0.93	0.08	0.08
mSe*cSe	0.86	<0.01	0.61	0.30	0.91	0.22	0.04	0.05	0.01	<0.01
mSe*cVit.E	0.80	0.09	0.27	0.04	0.19	0.61	0.69	0.10	0.27	0.11
mSe*cSe*cVit.E	0.87	0.04	0.71	0.79	0.46	0.08	0.78	0.25	0.83	0.77
mVit.E*cSe	0.73	0.05	0.63	0.22	0.70	0.33	0.10	0.14	0.36	0.78
mVit.E*cVit.E	0.94	0.11	0.53	0.02	0.55	0.92	0.97	0.20	0.25	0.11
mVit.E*cSe*cVit.E	0.25	0.08	0.15	0.81	0.53	0.58	0.62	0.87	0.47	0.34
mSe*mVit.E*cSe	0.96	0.19	0.23	0.01	0.40	0.16	0.76	1.00	0.71	0.73
mSe*mVit.E*cVit.E	0.09	0.00	0.05	0.01	0.60	0.04	0.12	0.00	0.33	0.65
mSe*mVit.E*cSe*cVit.E	0.62	0.02	0.06	0.20	0.36	0.01	0.40	0.93	0.08	0.08

[a-b] Means within the same column with different superscript letters are significantly different ($P < 0.05$).
[A-B] Means within the same column with different superscript letters are significantly different ($P < 0.01$).
[1] Represents the mean of 8 pens/treatment. Abbreviations: mSe, maternal dietary Se; mVit.E, maternal dietary Vit.E; cSe, chick dietary Se; cVit.E, chick dietary Vit.E.

Table A.2.10. Broiler breast Se concentrations in Exp. 1 of Chapter 4.[1]

Broiler diet	Breast Se, ppm
Control	0.05[B]
Se yeast	0.17[A]
Vit.E	0.05[B]
Vit.E + Se yeast	0.16[A]
SEM	0.01
———— *P*-values (main effects) ————	
Maternal diet	
mSe	0.13
mVit.E	0.03
mSe*mVit.E	0.05
Broiler diet	
cSe	<0.01
cVit.E	0.61
cSe*cVit.E	0.43
Interactions	0.68
mSe*cSe	0.17
mSe*cVit.E	0.58
mSe*cSe*cVit.E	0.14
mVit.E*cSe	<0.01
mVit.E*cVit.E	<0.01
mVit.E*cSe*cVit.E	0.87
mSe*mVit.E*cSe	0.69
mSe*mVit.E*cVit.E	0.34
mSe*mVit.E*cSe*cVit.E	0.68

[A-B] Means within the same column with different superscript letters differ significantly ($P < 0.01$).

[1] Values represent the mean of 12 pens/treatment (n=1 sample/pen). Abbreviations: mSe, maternal dietary Se; mVit.E, maternal dietary Vit.E; cSe, chick (broiler) dietary Se; cVit.E, chick (broiler) dietary Vit.E.

Table. A.2.11. Carcass yields of harvested broilers in Exp. 1 of Chapter 4.[1]

Broiler diet	WOG, %[2]	Front half, %[3]	Back half, %[3]
Control	75.64	47.80	27.84
Se yeast	76.51	48.02	28.50
Vit.E	76.42	47.88	28.54
Vit.E + Se yeast	75.94	47.51	28.43
SEM	0.50	0.37	0.33
	P-values (main effects)		
Maternal diet			
mSe	0.13	0.68	0.21
mVit.E	0.52	0.45	0.99
mSe*mVit.E	0.47	0.54	0.24
Broiler diet			
cSe	0.70	0.84	0.42
cVit.E	0.84	0.57	0.36
cSe*cVit.E	0.19	0.44	0.26
Interactions			
mSe*cSe	0.06	0.24	0.11
mSe*cVit.E	0.45	0.87	0.20
mSe*cSe*cVit.E	0.25	0.55	0.02
mVit.E*cSe	0.74	0.67	0.99
mVit.E*cVit.E	0.59	0.42	0.09
mVit.E*cSe*cVit.E	0.30	0.65	0.30
mSe*mVit.E*cSe	0.38	0.90	0.24
mSe*mVit.E*cVit.E	0.39	0.69	0.40
mSe*mVit.E*cSe*cVit.E	0.78	0.99	0.67

[1] Values represent the average of 12 pens/treatment with 3 birds sampled/pen. Abbreviations: mSe, maternal dietary Se; mVit.E, maternal dietary Vit.E; cSe, chick (broiler) dietary Se; cVit.E, chick (broiler) dietary Vit.E.

[2] Weight without giblets: Carcass weight expressed as a percentage of the live weight.

[3] Expressed as a percentage of the live weight.

Table A.2.12. Cooked characteristics of the raw breast fillets in Exp.1 of Chapter 4.[1]

Broiler diet	Cooking loss, %[2]	Shear force, kg
Control	19.52	1.44
Se yeast	19.13	1.47
Vit.E	19.95	1.36
Vit.E + Se yeast	19.24	1.30
SEM	1.11	0.09
	P-values (main effects)	
Maternal diet		
mSe	0.61	0.94
mVit.E	0.51	0.91
mSe*mVit.E	0.96	1.00
Broiler diet		
cSe	0.62	0.89
cVit.E	0.81	0.17
cSe*cVit.E	0.89	0.58
Interactions		
mSe*cSe	0.28	0.14
mSe*cVit.E	0.57	0.97
mSe*cSe*cVit.E	0.72	0.29
mVit.E*cSe	0.24	0.95
mVit.E*cVit.E	0.61	0.37
mVit.E*cSe*cVit.E	0.33	0.83
mSe*mVit.E*cSe	0.24	0.87
mSe*mVit.E*cVit.E	0.48	0.72
mSe*mVit.E*cSe*cVit.E	0.31	0.96

[1] Values represent the average of 12 pens/treatment with 3 birds sampled/pen. Abbreviations: mSe, maternal dietary Se; mVit.E, maternal dietary Vit.E; cSe, chick (broiler) dietary Se; cVit.E, chick (broiler) dietary Vit.E.

[2] Expressed as a percentage of the uncooked breast fillet weight.

Table A.2.13. Drip loss and TBARS values of raw breast fillets through 7d of storage in Exp. 1 of Chapter 4.[1]

	Drip loss, %[2]		TBARS[3]			
Broiler diet	3d	7d	0d	2d	5d	7d*
Control	2.25[a]	3.92[a]	0.002	0.009[ab]	0.014[a]	0.031[a]
Se yeast	1.66[b]	3.20[b]	0.003	0.006[ab]	0.013[ab]	0.022[ab]
Vit.E	2.60[a]	4.60[a]	0.003	0.010[a]	0.010[b]	0.025[ab]
Vit.E + Se yeast	1.88[b]	3.31[b]	0.002	0.006[b]	0.010[b]	0.017[b]
SEM	0.22	0.35	0.001	0.001	0.001	0.003
	P-values (main effects)					
Maternal diet						
mSe	0.45	0.49	0.79	0.05	0.04	0.50
mVit.E	0.04	0.01	0.99	0.23	<0.01	0.46
mSe*mVit.E	0.04	0.02	0.54	0.18	0.14	0.26
Broiler diet						
cSe	<0.01	<0.01	0.85	0.02	0.53	0.03
cVit.E	0.20	0.27	0.72	0.68	0.01	0.12
cSe*cVit.E	0.76	0.42	0.30	0.57	0.83	0.89
Interactions						
mSe*cSe	0.21	0.22	0.32	0.86	0.71	0.26
mSe*cVit.E	0.97	0.81	0.10	0.91	0.72	0.02
mSe*cSe*cVit.E	0.72	0.69	0.89	0.26	0.25	0.97
mVit.E*cSe	0.09	0.25	0.47	0.37	0.35	0.98
mVit.E*cVit.E	0.53	0.46	0.17	0.17	0.64	0.47
mVit.E*cSe*cVit.E	0.44	0.23	0.98	0.18	0.42	0.38
mSe*mVit.E*cSe	0.90	0.57	0.12	0.78	0.92	0.23
mSe*mVit.E*cVit.E	0.20	0.17	0.94	0.76	0.13	0.32
mSe*mVit.E*cSe*cVit.E	0.37	0.50	0.40	0.67	0.87	0.28

[a-b] Means within the same column with different superscript letters differ significantly ($P < 0.05$).

* $P = 0.06$

[1] Values represent the mean of 12 pens/treatment (n=1 sample/pen). Abbreviations: mSe, maternal dietary Se; mVit.E, maternal dietary Vit.E; cSe, chick (broiler) dietary Se; cVit.E, chick (broiler) dietary Vit.E.

[2] Drip loss expressed as a percentage of the initial weight (0d).

[3] TBARS values are expressed as a mg MDA/kg meat.

Table. A.2.14. Color stability of raw breast fillets through 7d of storage in Exp. 1 of Chapter 4.[1]

	L*				a*				b*			
Broiler diet	0d	2d	5d	7d	0d	2d	5d	7d	0d	2d	5d	7d
Control	62.30	64.74	63.06	61.25	7.44	7.05	6.87	6.87	20.00	18.94	18.98	20.00
Se yeast	63.61	66.13	64.52	63.51	7.00	6.42	6.28	6.36	19.27	18.73	19.02	19.21
Vit.E	62.32	65.08	63.37	62.03	7.45	7.01	6.69	6.68	19.97	18.89	19.12	19.49
Vit.E + Se yeast	62.95	65.34	63.46	63.08	7.80	7.21	7.03	6.89	20.31	19.09	19.60	19.83
SEM	0.91	0.66	0.77	0.77	0.91	0.66	0.77	0.77	0.38	0.33	0.36	0.33
	P-values (main effects)											
Maternal diet												
mSe	0.54	0.61	0.44	0.35	0.74	0.48	0.48	0.32	0.13	0.43	0.22	0.34
mVit.E	0.90	0.67	0.40	0.51	0.11	0.16	0.76	0.24	0.32	0.84	0.79	0.91
mSe*mVit.E	0.83	0.37	0.75	0.97	0.83	0.29	0.07	0.06	0.02	0.14	0.66	0.52
Broiler diet												
cSe	0.30	0.22	0.32	0.04	0.90	0.46	0.72	0.61	0.62	1.00	0.48	0.50
cVit.E	0.73	0.73	0.63	0.82	0.26	0.21	0.41	0.55	0.20	0.64	0.33	0.87
cSe*cVit.E	0.71	0.40	0.38	0.44	0.29	0.17	0.17	0.22	0.17	0.54	0.55	0.10
Interactions												
mSe*cSe	0.51	0.34	0.26	0.39	0.44	0.22	0.24	0.38	0.18	0.17	0.23	0.17
mSe*cVit.E	0.03	0.13	0.26	0.11	0.39	0.42	0.99	0.43	0.74	0.12	0.25	0.28
mSe*cSe*cVit.E	0.37	0.38	0.93	0.64	0.13	0.27	0.37	0.25	0.06	0.94	0.91	0.38
mVit.E*cSe	0.92	0.84	0.66	0.55	0.05	0.03	0.11	0.09	0.36	0.04	0.28	0.25
mVit.E*cVit.E	0.66	0.68	0.39	0.61	0.97	0.63	0.29	0.44	0.76	0.89	0.90	0.65
mVit.E*cSe*cVit.E	0.60	0.77	0.71	0.51	0.60	0.79	0.49	0.45	0.40	0.80	0.75	0.75
mSe*mVit.E*cSe	0.18	0.08	0.05	0.26	0.16	0.04	0.09	0.67	0.02	0.06	0.05	0.12
mSe*mVit.E*cVit.E	0.27	0.36	0.12	0.37	0.45	0.57	0.31	0.29	0.62	0.98	0.93	0.59
mSe*mVit.E*cSe*cVit.E	0.26	0.25	0.10	0.32	0.95	0.65	0.81	0.47	0.55	0.78	0.68	0.06

[1] Values represent the average of 12 pens/treatment (n=1 sample/pen). Abbreviations: mSe, maternal dietary Se; mVit.E, maternal dietary Vit.E; cSe, chick (broiler) dietary Se; cVit.E, chick (broiler) dietary Vit.E.

Table A.2.15. Breast pH and cooked characteristics of the marinated breast fillets in Exp. 1 of Chapter 4.[1]

Broiler diet	pH		Cooking loss, %[2]	Shear force, kg
	Pre-marination	Post-marination		
Control	5.82	6.10	17.05	1.25
Se yeast	5.83	6.03	15.27	1.24
Vit.E	5.82	6.04	16.37	1.39
Vit.E + Se yeast	5.80	6.05	15.32	1.30
SEM	0.04	0.03	0.66	0.08
	P-values (main effects)			
Maternal diet				
mSe	0.71	0.46	0.65	0.06
mVit.E	0.68	0.39	0.17	0.02
mSe*mVit.E	0.19	0.18	0.81	0.46
Broiler diet				
cSe	0.83	0.51	0.04	0.54
cVit.E	0.65	0.59	0.64	0.25
cSe*cVit.E	0.71	0.29	0.58	0.60
Interactions				
mSe*cSe	0.65	0.70	0.64	0.51
mSe*cVit.E	0.54	0.41	0.01	0.32
mSe*cSe*cVit.E	0.30	0.46	0.69	0.34
mVit.E*cSe	0.71	0.51	0.68	0.45
mVit.E*cVit.E	0.46	0.41	0.75	0.87
mVit.E*cSe*cVit.E	0.32	0.79	0.24	0.11
mSe*mVit.E*cSe	0.08	0.81	0.44	0.80
mSe*mVit.E*cVit.E	0.33	0.38	0.79	0.94
mSe*mVit.E*cSe*cVit.E	0.84	0.59	0.40	0.64

[1] Values represent the average of 12 pens/treatment (n=1 sample/pen). Abbreviations: mSe, maternal dietary Se; mVit.E, maternal dietary Vit.E; cSe, chick (broiler) dietary Se; cVit.E, chick (broiler) dietary Vit.E.
[2] Expressed as a percentage of the uncooked breast fillet weight.

Table A.2.16. Drip loss and TBARS values of marinated breast fillets through 7d of storage in Exp. 1 of Chapter 4.[1]

	Drip loss, %[2]		TBARS[3]			
Broiler diet	3d	7d	0d	2d	5d	7d
Control	0.43	0.96	0.002	0.019^{A}	0.054^{A}	0.118^{A}
Se yeast	0.51	0.97	0.003	0.010^{B}	0.029^{B}	0.062^{B}
Vit.E	0.52	1.02	0.003	0.004^{B}	0.016^{B}	0.050^{BC}
Vit.E + Se yeast	0.41	0.89	0.002	0.004^{B}	0.016^{B}	0.026^{C}
SEM	0.08	0.12	0.001	0.003	0.006	0.009
	P-values (main effects)					
Maternal diet						
mSe	0.87	0.19	0.79	0.35	0.63	0.61
mVit.E	0.95	0.63	0.99	0.95	0.36	0.98
mSe*mVit.E	0.78	0.75	0.54	0.13	0.11	0.17
Broiler diet						
cSe	0.81	0.62	0.85	0.08	0.04	<0.01
cVit.E	0.93	0.93	0.72	<0.01	<0.01	<0.01
cSe*cVit.E	0.24	0.55	0.30	0.09	0.03	0.09
Interactions						
mSe*cSe	0.10	0.24	0.32	0.92	0.77	0.69
mSe*cVit.E	0.62	0.74	0.10	0.40	0.47	0.59
mSe*cSe*cVit.E	0.78	0.27	0.89	0.52	0.86	0.52
mVit.E*cSe	0.61	0.38	0.47	0.47	0.84	0.59
mVit.E*cVit.E	0.96	0.74	0.17	0.93	0.89	0.90
mVit.E*cSe*cVit.E	0.27	0.61	0.98	0.93	0.65	0.75
mSe*mVit.E*cSe	0.76	0.72	0.12	0.44	0.56	0.89
mSe*mVit.E*cVit.E	0.37	0.72	0.94	0.55	0.59	0.31
mSe*mVit.E*cSe*cVit.E	0.43	0.10	0.40	0.64	0.96	0.18

[A-B] Means within the same column with different superscript letters differ significantly ($P < 0.01$).

[1] Values represent the mean of 12 pens/treatment (n=1 sample/pen). Abbreviations: mSe, maternal dietary Se; mVit.E, maternal dietary Vit.E; cSe, chick (broiler) dietary Se; cVit.E, chick (broiler) dietary Vit.E.

[2] Drip loss expressed as a percentage of the initial weight (0d).

[3] TBARS values are expressed as mg MDA/kg meat.

Table A.2.17. Broiler breast Se concentrations (ppm) in Exp. 2 of Chapter 4.[1]

Broiler diet	Breast Se, ppm
Control	0.07[b]
Se yeast	0.16[a]
Vit.E	0.08[b]
Vit.E + Se yeast	0.16[a]
SEM	0.01
——————— *P*-values (main effects) ———————	
Maternal diet	
mSe	0.68
mVit.E	0.52
mSe*mVit.E	0.55
Broiler diet	
cSe	<0.01
cVit.E	0.59
cSe*cVit.E	0.48
Interactions	
mSe*cSe	0.48
mSe*cVit.E	0.33
mSe*cSe*cVit.E	0.97
mVit.E*cSe	0.80
mVit.E*cVit.E	0.71
mVit.E*cSe*cVit.E	0.23
mSe*mVit.E*cSe	0.71
mSe*mVit.E*cVit.E	0.54
mSe*mVit.E*cSe*cVit.E	0.82

[a-b] Means within the same column with different superscript letters differ significantly ($P < 0.05$).

[1] Values represent the mean of 8 pens/treatment (n=1 sample/pen). Abbreviations: mSe, maternal dietary Se; mVit.E, maternal dietary Vit.E; cSe, chick (broiler) dietary Se; cVit.E, chick (broiler) dietary Vit.E.

Table. A.2.18. Carcass yields of harvested broilers in Exp. 2 of Chapter 4.[1]

Broiler diet	WOG, %[2]	Front half, %[3]	Back half, %[3]
Control	74.67	44.62	30.05
Se yeast	75.13	45.65	29.47
Vit.E	74.51	44.73	29.78
Vit.E + Se yeast	74.76	45.55	29.21
SEM	0.32	0.37	0.24
	P-values (main effects)		
Maternal diet			
mSe	0.16	0.18	0.28
mVit.E	0.21	0.29	0.69
mSe*mVit.E	0.06	0.63	0.01
Broiler diet			
cSe	0.29	0.03	0.03
cVit.E	0.43	0.99	0.29
cSe*cVit.E	0.75	0.78	0.99
Interactions			
mSe*cSe	0.03	0.23	0.19
mSe*cVit.E	0.62	0.53	0.76
mSe*cSe*cVit.E	0.01	<0.01	0.26
mVit.E*cSe	0.57	0.26	0.31
mVit.E*cVit.E	0.54	0.72	0.80
mVit.E*cSe*cVit.E	0.51	0.95	0.34
mSe*mVit.E*cSe	0.31	0.64	0.52
mSe*mVit.E*cVit.E	0.81	0.95	0.68
mSe*mVit.E*cSe*cVit.E	0.36	0.95	0.26

[1] Values represent the average of 8 pens/treatment with 2 birds sampled/pen. Abbreviations: mSe, maternal dietary Se; mVit.E, maternal dietary Vit.E; cSe, chick (broiler) dietary Se; cVit.E, chick (broiler) dietary Vit.E.

[2] Weight without giblets: Carcass weight expressed as a percentage of the live weight.

[3] Expressed as a percentage of the live weight.

Table A.2.19. Drip loss and TBARS values of raw breast fillets through 12d of storage in Exp. 2 of Chapter 4.[1]

	Drip loss, %[2]		TBARS[3]				
Broiler diet	3d	7d	0d	5d	7d	10d	12d
Control	0.81	2.75	0.005	0.012	0.017	0.027	0.023
Se yeast	0.79	2.34	0.004	0.012	0.017	0.029	0.026
Vit.E	1.23	3.23	0.004	0.009	0.014	0.021	0.019
Vit.E + Se yeast	0.98	2.43	0.006	0.010	0.016	0.025	0.020
SEM	0.16	0.45	0.001	0.002	0.002	0.004	0.003
	P-values (main effects)						
Maternal diet							
mSe	0.61	0.67	0.34	0.33	0.72	0.46	0.39
mVit.E	0.88	0.90	0.69	0.20	0.04	0.15	0.05
mSe*mVit.E	0.29	0.46	0.85	0.22	0.05	0.87	0.50
Broiler diet							
cSe	0.41	0.20	0.74	0.72	0.83	0.51	0.48
cVit.E	0.09	0.53	0.52	0.21	0.44	0.29	0.12
cSe*cVit.E	0.51	0.67	0.20	0.72	0.66	0.82	0.73
Interactions							
mSe*cSe	0.29	0.84	0.66	0.52	0.40	0.42	0.83
mSe*cVit.E	0.78	0.74	0.24	0.71	0.45	0.64	0.85
mSe*cSe*cVit.E	0.89	0.65	0.11	0.43	0.52	0.50	0.57
mVit.E*cSe	0.16	0.56	0.69	0.51	0.52	0.77	0.97
mVit.E*cVit.E	0.45	0.66	0.93	0.29	0.61	0.48	0.81
mVit.E*cSe*cVit.E	0.21	0.57	0.90	0.94	0.46	0.41	0.43
mSe*mVit.E*cSe	0.32	0.42	0.54	0.56	0.73	0.67	0.62
mSe*mVit.E*cVit.E	0.40	0.69	0.29	0.99	0.56	0.73	0.77
mSe*mVit.E*cSe*cVit.E	0.27	0.46	0.43	0.37	0.34	0.49	0.41

[1] Values represent the mean of 8 pens/treatment (n=1 sample/pen). Abbreviations: mSe, maternal dietary Se; mVit.E, maternal dietary Vit.E; cSe, chick (broiler) dietary Se; cVit.E, chick (broiler) dietary Vit.E.
[2] Drip loss expressed as a percentage of the initial weight (0d).
[3] TBARS values are expressed as a mg MDA/kg meat.

Table A.2.20. Drip loss and TBARS values of marinated breast fillets through 12d of storage in Exp. 2 of Chapter 4.[1]

	Drip loss, %[2]		TBARS[3]				
Broiler diet	3d	7d	0d	5d	7d	10d	12d
Control	0.80	1.22^{a}	0.009	0.105^{A}	0.143^{A}	0.153^{A}	0.214^{A}
Se yeast	0.45	0.70^{b}	0.009	0.068^{B}	0.090^{B}	0.102^{B}	0.154^{A}
Vit.E	0.63	0.89^{ab}	0.009	0.037^{C}	0.055^{BC}	0.054^{C}	0.071^{B}
Vit.E + Se yeast	0.48	0.71^{b}	0.009	0.040^{C}	0.051^{C}	0.048^{C}	0.072^{B}
SEM	0.10	0.14	0.001	0.007	0.012	0.013	0.021
			P-values (main effects)				
Maternal diet							
mSe	0.52	0.61	0.07	0.61	0.72	0.73	0.87
mVit.E	0.38	0.32	0.42	0.53	0.81	0.88	0.58
mSe*mVit.E	0.67	0.58	0.86	0.80	0.60	0.61	0.70
Broiler diet							
cSe	0.03	0.03	0.48	0.04	0.04	0.05	0.18
cVit.E	0.48	0.27	0.78	<0.01	<0.01	<0.01	<0.01
cSe*cVit.E	0.35	0.24	0.81	0.02	0.06	0.12	0.17
Interactions							
mSe*cSe	0.80	0.94	0.13	0.23	0.12	0.13	0.14
mSe*cVit.E	0.81	0.42	0.59	0.32	0.20	0.08	1.00
mSe*cSe*cVit.E	0.99	0.88	0.81	0.91	0.57	0.97	0.31
mVit.E*cSe	0.65	0.44	0.20	<0.01	0.03	0.09	0.06
mVit.E*cVit.E	0.55	0.32	0.53	0.04	0.45	0.72	0.42
mVit.E*cSe*cVit.E	0.19	0.13	0.98	0.10	0.57	0.90	0.45
mSe*mVit.E*cSe	0.86	0.82	0.08	0.79	0.93	0.63	0.33
mSe*mVit.E*cVit.E	0.11	0.10	0.98	1.00	0.55	0.79	0.90
mSe*mVit.E*cSe*cVit.E	0.77	0.77	0.05	0.71	0.65	0.37	0.25

[1] Values represent the mean of 8 pens/treatment (n=1 sample/pen). Abbreviations: mSe, maternal dietary Se; mVit.E, maternal dietary Vit.E; cSe, chick (broiler) dietary Se; cVit.E, chick (broiler) dietary Vit.E.
[2] Drip loss expressed as a percentage of the initial weight (0d).
[3] TBARS values are expressed as a mg MDA/kg meat.

Table A.2.21. Fat and moisture percentages of the breast fillets in Exp. 2 of Chapter 4.[1]

Broiler diet	Fat, %	Moisture, %
Control	1.27	74.58
Se yeast	1.30	74.71
Vit.E	1.37	74.78
Vit.E + Se yeast	1.09	74.55
SEM	0.18	0.29
	P-values (main effects)	
Maternal diet		
mSe	0.08	0.55
mVit.E	0.80	0.95
mSe*mVit.E	0.27	0.99
Broiler diet		
cSe	0.51	0.87
cVit.E	0.75	0.95
cSe*cVit.E	0.42	0.54
Interactions		
mSe*cSe	0.23	0.34
mSe*cVit.E	0.92	0.29
mSe*cSe*cVit.E	0.14	0.22
mVit.E*cSe	0.34	0.39
mVit.E*cVit.E	0.18	0.04
mVit.E*cSe*cVit.E	0.10	0.40
mSe*mVit.E*cSe	0.95	0.84
mSe*mVit.E*cVit.E	0.46	0.92
mSe*mVit.E*cSe*cVit.E	0.44	0.79

[1] Values represent the average of 8 pens/treatment (n=1 sample/pen). Abbreviations: mSe, maternal dietary Se; mVit.E, maternal dietary Vit.E; cSe, chick (broiler) dietary Se; cVit.E, chick (broiler) dietary Vit.E.

Table A.2.22. Fatty acid composition of breast fillets in Exp. 2 of Chapter 4.[1]

Broiler diet	14:0	14.1	15:0	16:0	16:1	17:0	17:1	18:0	18:1 *t*9	18:1 *n*9	18:2	ω18:3	20:0	20:1 *n*9	20:4 *n*6	20:5 ω3	22:5 ω3	22:6 ω3	24:0	24:1 *n*9
Control	1.17	0.46	0.34	18.30	2.94	0.38	1.03	9.70	0.96[b]	27.02	12.22	0.59	0.17	0.42	4.55	0.32	0.91	0.79	0.17	1.54
Se yeast	1.18	0.47	0.31	18.68	3.08	0.46	0.99	8.64	1.17[a]	28.56	12.63	0.67	0.20	0.33	4.21	0.25	0.83	0.69	0.16	1.38
Vit.E	1.14	0.49	0.32	18.08	2.89	0.45	1.07	8.90	1.13[a]	25.57	12.26	0.64	0.23	0.37	4.23	0.32	0.92	0.74	0.18	1.35
Vit.E + Se yeast	1.27	0.48	0.36	19.06	2.84	0.40	1.00	8.86	1.08[ab]	27.07	11.24	0.63	0.18	0.38	4.13	0.35	0.77	0.67	0.19	1.48
SEM	0.07	0.06	0.04	0.73	0.26	0.03	0.09	0.97	0.05	1.79	0.79	0.06	0.03	0.05	0.57	0.05	0.11	0.11	0.03	0.17
P-values (main effects)																				
Maternal diet																				
mSe	0.96	0.42	0.28	0.03	0.31	0.63	0.13	0.84	0.67	0.16	0.46	0.49	0.83	0.47	0.44	0.26	0.49	0.33	0.50	0.42
mVit.E	0.48	0.74	0.39	0.24	0.24	0.86	0.46	0.81	0.57	0.83	0.42	0.98	0.54	0.66	0.47	0.45	0.77	0.90	0.79	0.55
mSe*mVit.E	0.28	0.92	0.51	0.03	0.56	0.91	0.47	0.72	0.29	0.73	0.11	0.68	0.22	0.19	0.53	0.62	0.58	0.62	0.27	0.49
Broiler diet																				
cSe	0.28	0.89	1.00	0.37	0.85	0.63	0.54	0.58	0.13	0.41	0.71	0.55	0.83	0.49	0.70	0.64	0.30	0.45	0.91	0.93
cVit.E	0.66	0.83	0.78	0.91	0.60	1.00	0.75	0.78	0.46	0.43	0.41	0.90	0.44	0.99	0.73	0.32	0.82	0.75	0.39	0.76
cSe*cVit.E	0.40	0.75	0.37	0.69	0.72	0.05	0.88	0.61	0.02	0.99	0.39	0.40	0.15	0.31	0.83	0.33	0.77	0.92	0.80	0.40
Interactions																				
mSe*cSe	0.79	0.62	0.71	0.15	0.19	0.50	0.24	0.07	0.58	0.10	0.17	0.75	0.01	0.67	0.27	0.37	0.27	0.25	0.04	0.06
mSe*cVit.E	0.96	0.16	0.80	0.73	0.68	0.55	0.88	0.90	0.18	0.98	0.42	0.52	0.19	0.58	0.53	0.75	0.91	0.58	0.87	0.51
mSe*cSe*cVit.E	0.70	0.69	0.65	0.59	0.20	0.90	0.73	0.86	0.95	0.44	0.46	0.24	0.91	0.59	0.37	0.55	0.50	0.55	0.69	0.51
mVit.E*cSe	0.34	0.75	0.52	0.37	0.12	0.60	0.40	0.28	0.85	0.52	0.75	0.89	0.21	0.91	0.29	0.69	0.70	0.69	0.13	0.15
mVit.E*cVit.E	0.28	0.43	0.49	0.49	0.34	0.72	0.47	0.79	<0.01	0.29	0.92	0.22	0.98	0.54	0.25	0.47	0.13	0.19	0.42	0.29
mVit.E*cSe*cVit.E	0.28	0.84	0.76	0.97	0.94	0.45	0.86	0.06	0.07	0.61	0.28	0.37	0.04	0.18	0.94	0.77	0.85	0.87	0.32	0.69
mSe*mVit.E*cSe	0.99	0.38	0.89	0.72	0.66	0.47	0.99	0.48	0.49	0.71	0.47	0.96	0.13	0.84	0.44	0.75	0.15	0.48	0.94	0.76
mSe*mVit.E*cVit.E	0.44	0.35	0.15	0.05	0.49	0.02	0.16	0.06	0.13	0.63	0.47	0.34	0.08	0.40	0.40	0.42	0.52	0.85	0.42	0.34
mSe*mVit.E*cSe*cVit.E	0.82	0.35	0.98	0.65	0.88	0.21	0.60	0.62	0.67	0.58	0.54	0.80	0.56	0.48	0.57	0.88	0.73	0.77	0.91	0.49

[a-b] Means within the same column with different superscript letters differ significantly ($P < 0.05$).

[1] Values represent the mean of 8 pens/treatment (n=1 sample/pen). Abbreviations: mSe, maternal dietary Se; mVit.E, maternal dietary Vit.E; cSe, chick (broiler) dietary Se; cVit.E, chick (broiler) dietary Vit.E.

Appendix 3. Procedure for determination of selenium concentration in Chapter 4.

Selenium was determined using the method described by Olson et al. (1975) with modifications by Cantor and Tarino (1982). Samples destined for analysis were weighed (± 0.5 g, wet tissue basis) and digested overnight in concentrated perchloric and nitric acid (trace mineral grade). After partial acid digestion, the samples were then wet-ashed in culture tubes using microkjeldahl digesters. The samples were then titrated to a pH of 1.5 with NaOH and HCl, and buffered with ammonium hydroxide EDTA. Finally, the samples were reacted with 5.0 mL of 2,3-diaminonapthalene (DAN). The Se within each sample binds to DAN forming a fluorescent Se-DAN complex (piazselenol), which was then extracted from the solution using cyclohexane. The cyclohexane extract was then measured by fluorescence (360 nm excitation, 520 nm emission). The data were then regressed against a standard curve which accounted for the base level of fluorescence which would be detected in absence of Se in the samples. The regressed data were used to calculate the original concentration (ppm) of Se in the sample based on the weight of the sample. The commercial standard NIST SRM 1577c (bovine liver) was used as a reference material to assure accuracy of the obtained results.

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Vita

Anthony Quant was born August 3rd, 1984 in Owensboro, KY to Bill and Alane Quant. In August 1988, Anthony moved to Erlanger, KY, where he attended grade school and later graduated from Lloyd Memorial High School in 2002. Anthony attended Centre College in Danville, KY in the fall of 2002, where he was a member of the varsity football team and a member of Phi Delta Theta fraternity. He earned his bachelor's degree in Biology from Centre in 2006.

Anthony was given the opportunity to participate in an internship program during the winter of his senior year in college, where he worked as a research intern at Christensen Family Farms in Sleepy Eye, MN. It was the experiences from working with this large swine producer that fueled Anthony's desire to pursue a M.S. degree in swine nutrition. Anthony completed his M.S. degree in December of 2008 at the University of Kentucky under the advisement of Dr. Merlin Lindemann. His thesis research evaluated the optimum standardized ileal digestible trptophan to lysine ratio in growing pigs fed U.S.-type and non-U.S.-type feedstuffs. In addition to his thesis research, Anthony also conducted research regarding the effects of supplemental antibiotic growth promoters on nutrient digestibility of finishing pigs and evaluated the optimum isoleucine to lysine ratio in growing pigs fed wheat-based diets. While pursuing his Master's degree, Anthony also served as a teaching assistant for the undergraduate level Domestic Animal Biology and Swine Production classes.

After completing his M.S. degree, Anthony chose to continue on to pursue his doctorate in poultry nutrition at the University of Kentucky under the advisement of Dr. Anthony Pescatore. During his time as a Ph.D. student, Anthony married the love of his life and best friend, Ms. Brandy Jacobs in September of 2012.