THE EFFECT OF ORGANIC SELENIUM SUPPLEMENTATION AND DIETARY ENERGY MANIPULATION ON MARES AND THEIR FOALS: SELENIUM CONCENTRATIONS, GLUTATHIONE PEROXIDASE ACTIVITY, FOALING PARAMETERS AND FOAL PHYSICAL CHARACTERISTICS

A Thesis

by

BRADY JOHN KARREN

Submitted to the Office of Graduate Studies of
Texas A&M University
in partial fulfillment of the requirements for the degree of

MASTER OF SCIENCE

August 2008

Major Subject: Animal Science

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Approved by:

Chair of Committee,	Josie Coverdale
Committee Members,	Pete Gibbs
	Brett Scott
	Rosemary Walzem
Head of Department,	Gary Acuff

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ABSTRACT

The Effect of Organic Selenium Supplementation and Dietary Energy Manipulation on Mares and Their Foals: Selenium Concentrations, Glutathione Peroxidase Activity, Foaling Parameters and Foal Physical Characteristics. (August 2008)

Brady John Karren, B.S., Brigham Young University

Chair of Advisory Committee: Dr. Josie Coverdale

Quarter Horse mares (n=28, 465-612 kg BW, 6-19 yrs of age) were used to investigate the effect of organic selenium (Se) supplementation (Selenosource, Diamond V Mills, Inc. Cedar Rapids, IA (SeM)) and DE manipulation on plasma, muscle, and colostrum Se concentrations, plasma glutathione peroxidase (Gsh-Px) activity, foaling parameters, and physical characteristics in mares and their foals. Mares were arranged in a 2x2 factorial with two levels of nutrition, pasture (100% NRC DE) or pasture plus grain (120% NRC DE) (fed at 0.75% BW (0.63 ppm Se)) and two levels of Se supplementation (0 or 0.3 mg/kg DM) equaling four treatment groups: pasture (P), pasture+grain (PG), pasture+grain+Se (PGS), or pasture+Se (PS). Mares were blocked by expected foaling date and randomly assigned to dietary treatment within block. Body condition score (BCS), BW, and rump fat (RF) were observed every 14 d beginning at d 0. Mare and foal plasma and muscle sampling began on d 0 (birth in foals). Plasma continued every 14 d and muscle every 28 d until parturition (d 56 in foals). Upon parturition, foaling parameters consisting of times: water break to birth, birth to placenta

expulsion, foal standing, and nursing were recorded. Colostrum quality was determined via refractometer and colostrometer analysis, and placenta weight, foal birth weight, whither and hip height and body length were recorded. Maternal SeM supplementation influenced ($P<0.05$) mare and foal plasma, muscle and colostrum Se concentrations. Increased maternal DE influenced ($P<0.05$) mare and foal plasma and foal muscle Se, mare BW, BCS, and RF. However, mare muscle Se was unaffected ($P>0.05$) by DE. Mare and foal plasma Gsh-Px, foal physical characteristics, and foaling parameters were unaffected by treatment ($P>0.05$). Greater ($P<0.02$) colostrum refractometer values (Brix%) for P, PS mares were noted and PGS, P mares had shorter gestational lengths (nutrition x SeM interaction ($P<0.05$)). These data indicate that maternal DE manipulation and SeM supplementation influences mare and foal Se status, mare BW and colostrum quality (Brix%), but not plasma Gsh-Px activity. Additionally, nutrition and SeM supplementation may affect gestational length. However, despite treatments there was no difference in foaling parameters or foal physical characteristics.

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Finally, thanks to my mother and father for their encouragement and to my wife for her patience and love.

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CHAPTER I

INTRODUCTION

Review of Literature

Selenium (Se) has long been accepted as an important mineral to all living things. It was discovered by Swedish chemist, Berzelius in 1818 (Ullrey, 1992). Since then, many studies have investigated animal diets containing various levels of Se. Initial studies investigated dietary requirements to prevent deficiency symptoms. Selenium deficiency in animals may lead to white muscle disease, or nutritional muscular dystrophy (Muth et al., 1958; Lofstedt, 1997), suppression of the immune system (Boyne et al., 1979; Knight and Tyznik, 1990; Stabel et al., 1989), or hamper the body's ability to destroy free radicals. This defense against free radicals is accomplished via the enzyme glutathione peroxidase (Gsh-Px) (Figure 1).

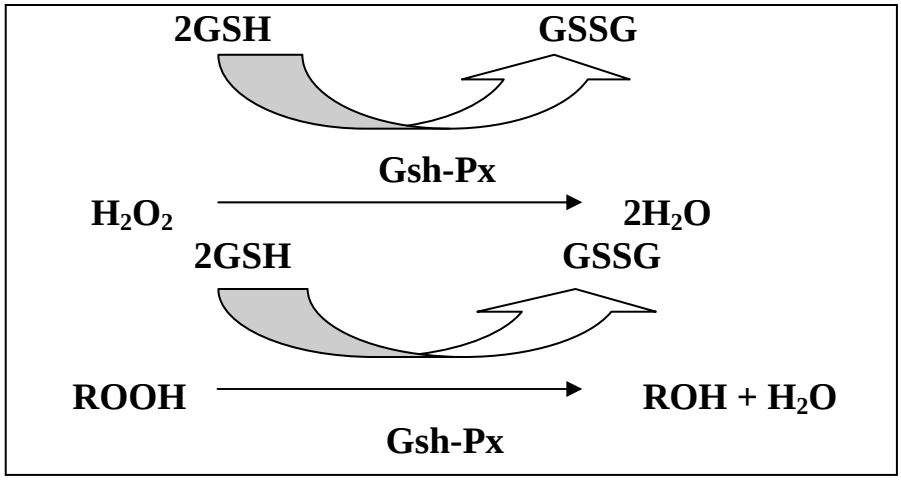

Figure 1. Mechanism of glutathione peroxidase catabolism of free radicals

This thesis follows the style of The Journal of Animal Science.

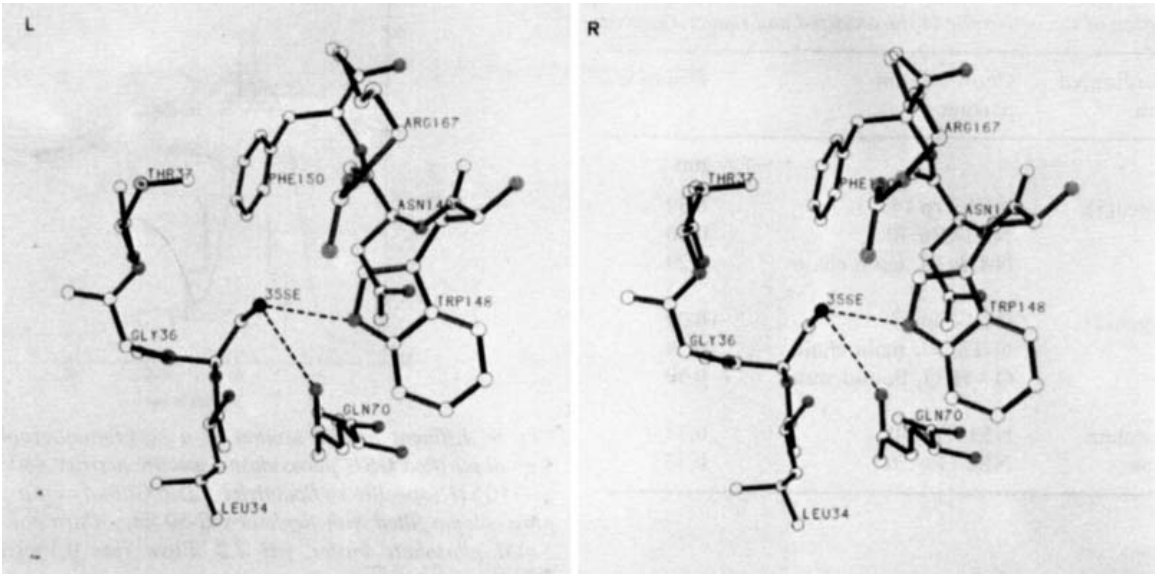

Figure 2. Depiction of glutathione peroxidase subunit structures (L and R)
Adapted from Epp et al., 1983

Because the Gsh-Px structure contains four seleno-cysteine molecules at its core, Se is necessary for formation (Figure 2) (Rotruck et al., 1973). Therefore, the amount of Se in the body is directly correlated with the activity of Gsh-Px, as evidenced by the linear increase in Gsh-Px activity in bovine mammary cells when increasing amounts of organic Se were provided (Miranda et al., 2007). Selenium status of an animal also plays a vital role in reproduction. A lack of Se in the diet can result in abortion of offspring. Orr and Blakley (1997) reported that 22 of 43 aborted fetuses of cows on pasture sent to the Department of Veterinary Pathology at the Western College of Veterinary Medicine for analysis died of myocardial necrosis, lymphocytic myocarditis, and myocardial fibrosis due to Se deficiency. Additionally, Tengelsen et al. (1997)

observed that out of 290 mares from Kentucky, Michigan, and England which aborted fetuses, approximately 32 were due to Se deficiency.

Although Se is necessary for many biological systems, toxicity can occur. Symptoms of Se toxicity in horses can include blind staggers, and alkali disease which is characterized by alopecia and cracking of the hooves around the coronet band. Because Se is both a required nutrient and an intoxicant, the Food and Drug Administration (FDA) and the National Research Council (NRC) have established guidelines for Se supplementation. The FDA has recently recommended that 0.3 mg Se/kg DM intake, regardless of source, can be safely supplemented in the diet of all livestock species. This supplemental Se can be safely added to the equine recommended daily allowance of 0.10 mg/kg DM (NRC, 2007). The equine NRC (2007) states the maximum tolerance of dietary Se for a horse is 5 mg/kg DM, and that total daily dietary Se intake should not exceed 2 mg/kg DM. Currently, two of the most widely used sources of supplemental Se are Se bound to a salt as sodium-selenate or selenite (NaSe) and Se bound to the amino acid methionine as selenized yeast (SeY) or selenomethionine (SeM). Sodium-selenate or selenite are classified as inorganic sources of Se while SeM is considered an organic source. Recent research has focused on the absorption, storage, and bioavailability of inorganic versus organic forms of trace mineral supplements in several livestock species.

Recent studies have elucidated positive effects of Se supplementation in an organic form when compared to inorganic. Juniper et al. (2008) reported that there were no adverse effects to cattle and sheep that consumed 10 times the recommended Se

allowance when SeY was fed, indicating that Se supplemented from an organic source may be less likely to result in toxicity. Another study found that it required 4, 6, or 8 mg Se/kg BW of SeY fed to sheep to induce the same toxicosis type side effects as 2, 3, or 4 mg Se/kg BW as NaSe (Tiwary et al., 2006). Furthermore, Kim et al. (2001) determined that Se toxicity symptoms in pigs (alopecia and separation of hooves at the site of the coronary band occurred at ≥ 10 ppm NaSe but not until ≥ 15 ppm SeY was fed. Therefore, organic Se appears to be a safer alternative to inorganic Se supplementation. In addition to a reduced risk of toxicity, organic Se supplementation may be preferential to inorganic sources with respect to absorption and bioavailability.

Selenium Supplementation: Absorption in Blood Components

Selenium supplementation, regardless of source, results in increased plasma Se concentrations in horses (Shellow et al., 1985, Wichtel et al., 1998). However, when compared to inorganic supplemental Se, organic sources of dietary Se are more effective at increasing plasma Se concentrations in cattle (Awadeh et al., 1998; Pehrson and Ortman , 1999; Ortman and Pehrson, 1999; Gunter et al., 2003; Guyot et al., 2007), sheep (Oh et al., 1976; Rock et al., 2001; Davis et al., 2008), pigs (Parsons et al., 1985; Mahan et al., 1999; Kim and Mahan, 2001; Mateo et al., 2007), and horses (Pagan et al., 1999). This may be the case because SeM appears to be absorbed in greater amounts and more rapidly in all three segments of the small intestine than selenite or selenate (Vendeland et al., 1992). Additionally, because SeM is Se bound to the amino acid methionine, SeM is transported in the blood by amino acid transport mechanisms (Pagan et al., 1999). The ability of SeM to utilize amino acid transport mechanisms, rather than

relying on normal Se transport such as RBC utilization may partially facilitate the observed increases in plasma Se concentrations. One of the few studies to observe the effect of organic Se supplementation on horse plasma Se concentrations was performed by Richardson et al. (2006). These researchers utilized 18 eighteen month old horses fed similar amounts of SeM, NaSe or no supplemental Se at all. By day (d) 28 of the trial horses supplemented with Se had greater plasma Se concentrations, and horses supplemented with SeM had the greatest concentrations. However, at the conclusion of the study (d 56) there was no statistical difference in plasma Se concentrations between SeM and NaSe supplemented horses. The lack of definitive data from this study may be due to the relatively short time that horses were supplemented with Se. Therefore further research is needed to more fully elucidate the relationship between SeM supplementation and increased plasma Se concentrations in horses.

Similar to plasma Se concentrations, serum Se concentrations are also influenced by source of supplemental Se. When sheep were supplemented with similar amounts of organic or inorganic Se, those receiving organic Se had greater serum Se values (Tiwary et al. 2006, Davis et al. 2008). This was also the case in pigs (Mahan et al., 1999; Mahan, 2000; Yoon and McMillan, 2006; Mateo et al., 2007) and cattle (Awadeh et al., 1998; Weiss and Hogan, 2005). Horse serum Se concentrations also respond favorably to organic Se supplementation.

Janicki et al. (2001) observed that when mares were supplemented with either organic or inorganic Se, serum Se concentrations were greater when an organic source of Se was supplemented. These researchers supplemented 15 pregnant mares with 1 or 3

mg Se/d as NaSe, 3 mg Se/d as SeM. Immediately following parturition, and at 4 and 8 weeks post-parturition, serum Se concentrations were greater for mares fed organic Se than either 1or 3 mg NaSe supplemented mares.

Erythrocyte and whole blood Se concentrations respond to organic Se supplementation similarly to serum. However, erythrocyte Se concentrations do not appear to be as influenced by NaSe supplementation as they are by SeM supplementation (Tiwary et al., 2006). When cattle were supplemented with either inorganic or organic Se, those receiving SeM had greater erythrocyte Se concentrations (Gunter et al., 2003; Pehrson et al., 1999). Additionally, cattle whole blood seems to respond preferentially to organic Se rather than NaSe. Researchers that supplemented cattle with SeM and NaSe reported greater whole blood Se concentrations for cattle receiving an organic Se supplement (Awadeh et al., 1998; Pehrson et al., 1999; Gunter et al., 2003; Juniper et al., 2008). This was also true in sheep (Van Ryssen et al., 1989; Tiwary et al., 2006; Davis et al., 2008) and ppigs (Kim and Mahan, 2001).

While whole blood and erythrocyte Se interactions with SeM data are limited in horses, inorganic Se supplementation has been shown to increase whole blood and/or erythrocyte Se concentrations in horses (Shellow et al., 1985; Lee et al., 1995; Wichtel et al., 1998; Avellini et al., 1999). Pagan et al. (1999) is one of the few studies to investigate the relationship of SeM supplementation and whole blood and erythrocyte Se concentrations in horses. It was observed in their study that horses supplemented with either inorganic or organic Se had increased whole blood Se concentrations. However, there was no statistical significance related to either source of Se supplementation

pertaining to superiority of one source over the other. Therefore, further research is needed to more fully elucidate this relationship.

Selenium Supplementation: Glutathione Peroxidase Activity

A lack of Se can hamper the body's ability to destroy free radicals. Free radicals, such as hydrogen peroxide and lipid peroxides, are formed during oxidative metabolism when oxygen interacts with certain molecules creating highly reactive atoms with unpaired numbers of electrons. Free radicals are also produced when phagocytic cells in the bloodstream ingest foreign particles. During phagocytosis large amounts of oxygen are consumed and hydrogen peroxide, singlet oxygen ($^1O_2$), and superoxide (O_2^{--}) are created. While these radicals are utilized as an oxidative burst directed at pathogens, an excess could negatively affect the phagocyte or the rest of the body (Boyne and Arthur, 1979).

Once these highly reactive radicals are formed, or escape, they can react with important cellular components such as DNA, or the phospholipid bilayer which comprises the cellular membrane. Cells may have decreased functionality or die if this occurs. Part of the body's defense against free radicals is that radicals are catalyzed by a system of enzymes, of which Gsh-Px is primary. Glutathione peroxidase contains four seleno-cysteine molecules at its core and it must have Se to function (Rotruck et al., 1973), therefore, the amount of functional Gsh-Px in the system is corollary with the amount of systemic Se. As a result, Se deficient animals have less cellular protection against oxidative stress and cell membrane breakdown or death caused by free radicals.

Selenium supplementation can aid the body by providing the necessary components for Gsh-Px production. Organic Se supplementation in multiple species has resulted in greater whole blood, serum, or plasma Gsh-Px activity than either inorganic Se supplementation or no Se supplementation at all (Oh et al., 1976; Reffett et al. 1988; Enjalbert et al., 1999; Rock et al., 2001; Janicki et al., 2001; Mahan, 2000).

Cows receiving supplemental Se as either SeY or NaSe had greater whole blood or erythrocyte Gsh-Px activity than cows receiving no supplemental Se (Hoffman et al., 1978; Pehrson and Ortman 1999; Gunter et al. 2003; Rowntree et al., 2004; Juniper et al. 2008). Furthermore, cows fed supplemental Se as SeY seem to have greater whole blood or erythrocyte Gsh-Px activities than NaSe supplemented cows (Gunter et al. 2003; Juniper et al. 2008). Sheep Gsh-Px activities tend to react to Se supplementation through feed similarly to those in cattle. Lambs receiving an artificial milk replacer and supplemented with Se as NaSe had greater erythrocyte Gsh-px activities than those receiving no Se supplementation (Oh et al. 1976). This same effect was also noted in ewes receiving supplemental inorganic Se (Ullrey et al. 1978). Qin et al. (2007) similarly studied the effect of Se supplementation on blood Gsh-Px activity with the addition of comparing SeY and Se enriched probiotics with NaSe. Qin concluded that the whole blood Gsh-Px activity was greater in all Se supplemented sheep than control animals. Moreover, it was reported that sheep supplemented with organic Se had greater whole blood Gsh-Px activity than those supplemented with sodium selenite. The same is true in pigs (Meyer et al., 1981; Goehring et al., 1984; Adkins and Ewan, 1984; Kim and Mahan, 2001; Mahan and Peters, 2004).

Results of Se Gsh-Px interaction trials in horses have been conflicting. Brady et al. (1978) reported no difference between NaSe supplemented and non-supplemented horses Gsh-Px activity. Shellow et al. (1985) also found little evidence suggesting a relationship between dietary Se as NaSe and Gsh-Px activity in the horse. When blood analysis data was subjected to statistical manipulation there was no significance for the Se Gsh-Px relationship. However it should be noted that the authors felt this lack of evidence may be attributed to the possibility that the Gsh-Px levels of the horses had already reached a plateau prior to the study and therefore could not react to any supplemental Se. When the effect of SeM and NaSe on Gsh-Px activity in horses was compared there were conflicting results. Richardson et al. (2006) noted that at d 28 of their study horses supplemented with SeM had greater Gsh-Px activities. However, upon completion of the project (d 56) it was observed that horses receiving NaSe had greater Gsh-Px activities. Janicki et al. (2001) reported that when they fed fifteen pregnant mares a basal diet plus Se supplementation, there was an effect of Se on Gsh-Px. Though the effect was not seen on the mares themselves, there was a significant difference in whole blood Gsh-Px activity in their foals. At six weeks post-parturition foals from mares receiving either 1 or 3 mg/Se d had greater Gsh-Px activities then foals from mares receiving 1 mg/Se d. While the work of those researchers begins to reveal the effect of SeM on Gsh-Px activities, the data is still insufficient to be considered definitive. Further research is needed to fully elucidate this relationship.

Selenium Supplementation: Selenium Absorption and Tissue Accretion

When an animal consumes Se it is absorbed by the brush border enzymes of the small intestine (Vandeland et al., 1994). Once it is absorbed in the small intestine it passes through the cells to the blood stream. From the blood stream it can be utilized in a number of different ways. Selenium can stay in the blood stream as free Se, be utilized within the erythrocytes, or be relegated to Gsh-Px synthesis. Additionally it can be absorbed and stored in colostrum or milk, or the organs and musculature of the body (Mahan, 2000). Multiple species which were supplemented with organic Se had greater tissue Se concentration than those supplemented with inorganic forms of Se (Mahan et al., 1999; Mateo et al., 2007; Lawler et al., 2004; Qin et al., 2007) however this was not the case in horses (Richardson et al., 2006).

Lawler et al. (2004) reported that steers fed organic Se had a higher total tissue Se concentration than those fed inorganic Se. More specifically, a sample of the semitendonosus muscle from an animal fed NaSe had a 1.55 ppm concentration while a sample from an animal fed SeY had a Se concentration of 4.41 ppm. Skrˇivanova´ et al. (2007) similarly reported that calves fed supplemental SeY had greater muscle Se concentrations than calves supplemented with NaSe or no Se supplementation at all. Conversely, O'Grady et al. (2001) determined that there was no significant effect of supplemented Se in muscle Se concentration. However, the authors hypothesized that this could be the case because of the basal diet containing enough Se to saturate muscle tissue.

As was the case in cattle, sheep also tend to have greater tissue Se concentrations when they are supplemented with SeY versus NaSe or no Se supplementation at all. Lambs supplemented with SeY had greater liver, kidney cortex, heart, and muscle Se concentrations than those supplemented with NaSe (Taylor, 2005; Tiwary et al. 2006; Qin et al. 2007). Furthermore, when ewes were supplemented with SeM versus NaSe they had greater liver, heart, pancreas, muscle and wool Se concentrations than those fed NaSe (Van Ryssen et al., 1989).

The ability of supplemental Se to increase tissue Se concentrations is not relegated to ruminants alone. Pigs which were supplemented with Se had greater liver, kidney, spleen, heart, lung, brain, spinal cord, and muscle Se values. And pigs which were supplemented with SeY had greater tissue Se concentrations than those supplemented with NaSe (Ku et al. 1973; Mahan and Moxon 1978; Mateo et al. 2007).. While organic Se supplementation appears to influence tissue Se accretion in cattle, sheep, and pigs, there has not been, to date, an interaction seen in horses. Following an exhaustive search, Richardson et al. (2006) is the only study found by this author to investigate this relationship. Richardson et al. (2006) et al. (2006) randomly assigned (within sex) eighteen 18-month old horses to one of three treatments. Group one (control) received no supplemental Se, (0.15 mg of Se/kg of total diet DM); group 2 received control plus inorganic Se (0.45 mg of Se/kg of total diet DM from NaSe); and group 3 received control plus organic Se (0.45 mg of Se/kg of total diet DM from SeY). Muscle biopsies were taken at day 0 and day 56 and stored for analysis. Upon analysis it

was determined that there was no difference in Se source over the 56 day period. Further research is needed to fully elucidate this relationship.

Selenium Supplementation: Colostrum Selenium Concentrations

Young animals are among those most affected by a lack of Se in the diet. Because of the threat of white muscle disease, oxidative stress during parturition, and passive immunity, Se plays an important role in the diet of neonates. Although Se status of offspring may be a function of the maternal gestational diet (Mahan et al., 1977; Enjalbert et al., 1999), it is imperative that neonates receive an adequate supply of Se from birth until they are on feed. As with all mammals, livestock glean nutrition from their mother's colostrum and subsequent milk until they are able to consume feedstuffs.

The dependency of foals on mother's milk during early life has led to supplementing dams with Se in order to ensure that offspring have sufficient Se in their diet. The Se that is supplemented to the dams can then be imparted to the offspring via colostrum and milk if supplementation increases colostrum and milk Se concentrations. Se supplementation of dams in multiple species has increased the Se concentration of colostrum and milk (Lee et al., 1995; Awadeh et al., 1998; Mahan, 2000; Guyot et al., 2007). Furthermore, Se supplementation with SeM has resulted in greater colostrum and milk Se concentrations than NaSe supplementation. Cows which received Se supplementation as SeM had greater colostrum and milk Se concentrations than those supplemented with NaSe (Ortman and Pehrson, 1999; Knowles et al., 1999; Pehrson and Ortman, 1999; Givens et al., 2004; Guyot et al., 2007).

Similarly, sheep supplemented with NaSe and SeM had increased colostrum Se concentrations. Furthermore, those sheep supplemented with SeM had greater colostrum Se concentrations (Rock et al., 2001). When sows were supplemented with Se during gestation they had greater colostrum and milk Se concentrations (Loudenslager et al. 1986). And when SeY was added to the equation, sows receiving SeY had greater colostrum and milk Se concentrations than those receiving NaSe (Mahan and Kim, 1996; Mahan, 2000; Kim and Mahan 2001; Yoon and McMillan 2006).

While there have been numerous studies investigating the influence of supplemental Se on colostrum and milk Se concentrations in other species, the literature is somewhat limited in regards to horses. Lee et al. (1995) observed mares under practical management conditions and utilized information from a number of different stables to get a sense of the relationship of Se in mares to colostrum and milk Se concentrations. It was concluded that mares which received adequate Se had greater colostrum and milk Se concentrations than mares receiving Se insufficient feedstuffs. Janicki et al. (2001) observed the effect of SeY versus NaSe on colostrum and milk Se concentrations. These researchers collected colostrum samples prior to foal suckling and milk samples every two weeks until 56 days post-partum. They reported that mares fed organic Se had greater colostrum and milk Se concentrations than either control or NaSe supplemented mares. However, after an exhaustive search this is the only study the author could find investigating organic Se supplementation to mares and further research is needed for definitive answers to this question.

Selenium Supplementation of Dam: Offspring Selenium and Glutathione Peroxidase Activity

Although Se has many positive effects for animals of all ages, it is especially important to young animals. While reservoirs of vitamins and minerals can be mobilized and transferred to the fetus (Ghany-Hefnawy et al., 2007) it is thought to be more advantageous to feed supplemental sources to dams for fetal use (Mahan and Vallet, 1997). Ghany-Hefnawy et al. (2007) reported that there was a significant correlation between the maternal and fetal liver Se concentration. While Hostetler and Kincaid (2004) observed that when sows were fed a low-Se diet, fetal livers were also low in Se, and although fetal liver Gsh-Px was not affected, there was evidence of increased oxidative stress in the fetus. Due to the correlation between the dam's dietary Se intake and the Se status of the offspring, it is important to discuss the influence of maternal Se supplementation and the subsequent affect on the offspring.

Calves of cows supplemented with Se have greater whole blood and plasma Gsh-Px activity than those from cows receiving no Se supplementation whatsoever (Enjalbert et al. 1999; Rowntree et al. 2004). Guyot et al. (2007) similarly observed that calves from cows receiving supplemental Se had greater whole blood Gsh-Px activities. Furthermore, these researchers determined that calves from cows supplemented with SeY versus NaSe had greater plasma Se concentrations. Additionally, Rock et al. (2001) noted that lambs of ewes which were fed SeY had greater concentrations of Se and Gsh-Px activities in blood than lambs of ewes supplemented with NaSe. Research in non-

ruminants reported that piglets of sows supplemented with SeY exhibited greater tissue Se concentrations (Mahan and Kim, 1996; Mahan and Peters, 2004).

Lee et al. (1995) utilized information from a number of different stables in a practical management environment to investigate the relationship of maternal Se status and that of the foal. Thirty mares from 9 different stables were observed and Se intake was estimated by their regular feed composition. It was concluded that blood Se concentration of foals at parturition were highly correlated with maternal blood Se concentration. And foals of mares receiving adequate Se had greater plasma Se concentrations. Janicki et al. (2001) determined that at 12 hours of age foals born from mares supplemented with SeY had greater serum Se concentrations than foals from mares supplemented with 1 mg Se as NaSe. However, to the knowledge of the author, this has been the only study to investigate the effects of organic Se supplementation on mares and their foals. Therefore, more research is needed to further elucidate the benefits, or lack thereof, of organic Se supplementation to mares and their foals.

Maternal Energy Restriction and Selenium

Maternal Se status during gestation plays an integral role in the Se status of offspring (Kinkaid and Hodgson, 1989; Ghany-Hefnawy et al., 2007), so to does the maternal plane of nutrition. When ewes were relegated to either 50 or 100% of NRC recommended dietary requirements, lambs of restricted ewes had less growth at one month of age than their 100% counterparts (Hyatt et al., 2007). Furthermore, when ewes were subjected to the same treatments in another study, lambs of nutrient restricted ewes had lower liver weights at birth than lambs from ewes receiving adequate nutrition

(Hyatt et al., 2008). While dams may be able to divert tissue bound minerals to the developing fetus (Mahan and Vallet, 1997) to curtail some of these deleterious effects, supplemental trace minerals (ie. Se) may assist the dam in providing for fetal development. Cronjé et al. (2006) determined that lambs from ewes fed a poor quality diet suffered a loss of weight and tended to have lower immune responses. However, lambs from sheep fed a poor quality diet but supplemented with Se were able to maintain their weight, although no weight gain was recorded. While nutrient or energy restriction appears to influence dams and their offspring, excess nutrition may also be influential. Wallace et al. (1999) reported that ewes which received ~ 300 g/d of a diet designed for rapid growth, versus those receiving ~ 55 g/d of the same diet, exhibited a major restriction in placental growth and lower offspring birth weights.

While level of maternal nutrition is corollary to fetal development and offspring physical characteristics, an interaction of nutrition and Se supplementation may also be influential. Reed et al. (2007) investigated the effect of maternal plane of nutrition and Se supplementation in a study with 36 pregnant ewe lambs. Treatments consisted of adequate nutrition (100% NRC recommendations), restricted nutrition (60%), adequate Se (6 µg/kg of BW), and high Se (80 µg/kg of BW) as SeY. It was concluded that maternal jejunal RNA:DNA and maternal BW, stomach complex, small intestine, large intestine, liver, and kidney mass were less in restricted than control ewes. Fetuses from restricted ewes had lower BW, empty carcass weight, crown-rump length, liver, pancreas, perirenal fat, small intestine, and spleen weights compared with controls. Fetuses from high Se ewes were heavier and had higher empty carcass, heart, lung,

spleen, total viscera, and large intestine weights compared with adequate Se ewes. Nutrient restriction resulted in lower protein content (mg) and protein: DNA in fetal jejunum. Fetal muscle DNA concentration was greater in restricted ewes fed high Se compared with other treatments. Fetal muscle RNA concentration and heart RNA content were greater in fetuses from high Se versus adequate Se ewes.

While the work of Reed et al. (2007) et al. (2007) begins to clarify the effect of maternal DE intake and maternal trace mineral supplementation on offspring, there is a shortage of studies pertaining to this matter. Furthermore, there was no data examining this relationship in horses. It is apparent that further research needs to be performed to elucidate the advantages or disadvantages that maternal DE manipulation and maternal trace mineral supplementation may provide for dams and their offspring.

CHAPTER II

MATERIALS AND METHODS

Horses and Treatments

Twenty-eight Quarter Horse mares from the Texas A&M University Horse Center were utilized in a randomized complete block design. Mares were housed at the Texas A&M University Horse Center and maintained according to farm protocol. Horses ranged from 6 to 19 years of age and weighed between 465 and 612 kg. Care, handling, and sampling of animals were approved by the Texas A&M University Animal Care and Use Committee.

Mares were blocked by expected foaling date and randomly assigned within block to dietary treatments. Dietary treatments were arranged as a 2 x 2 factorial with two levels of nutrition (pasture or pasture plus grain) and two levels of selenium (Se) supplementation (0 or 0.3 mg selenomethioine /kg DM). This resulted in four treatment groups: pasture (P), pasture + selenium (PS), pasture + grain (PG), and pasture + grain + selenium (PGS). Pasture diets (P and PS) provided approximately 100% NRC DE requirements while grain supplemented diets (PG and PGS) provided approximately 120%. Because pasture intake was not measured, total dietary intake was assumed to be 2% BW (DM basis) (Aiken et al., 1989) with concentrate supplemented mares consuming 1.25% BW in pasture and 0.75% BW in concentrate (Rouquette et al., 1987). Based on this assumed intake, P mares consumed approximately 0.19 mg Se/kg DMI, PS mares consumed 0.49 mg Se/kg DMI, PG mares consumed 0.35 mg Se/kg DMI, and PGS mares consumed 0.65 mg Se/kg DMI. All dietary treatments were initiated at the

third trimester of pregnancy, approximately 110 d prior to the estimated foaling date and terminated at parturition.

All mares had continual access to bermudagrass (*Cynodon Dactylon*) pastures (Table 1), water, and trace mineralized salt (containing no added Se) throughout the study. Multiple adjacent pastures were utilized at the facility; however blocks were maintained on the same pasture and rotated between pastures as part of farm protocols.

Treatments PG and PGS received supplemental grain (13% CP pellet, Producers Co-Op, Bryan TX.) at 0.75% of BW (AF) (Table 1). Treatments PS and PGS received supplemental Se in the form of selenomethionine (Selenosource, Diamond V Mills, Inc. Cedar Rapids, IA) (Table 1) at 0.3 mg/kg DM. Grain and Se treatments (PG, PS and PGS) were fed in individual 3.0 x 2.9 m stalls twice daily. Selenium was mixed with a minute amount of sweet feed (12% CP sweet feed, Producers Co-Op, Bryan TX.) stored in individual containers (237 ml plastic storage container, Fisher Scientific, Waltham, MA) and offered to mares to ensure ingestion of Se supplement. Body weight was recorded every two weeks and diets were adjusted accordingly.

Table 1. Nutrient composition of Coastal Bermuda Grass (*Cynodon Dactylon)* pasture, concentrate, and supplemental selenium (Se)[1] (DM basis)

Nutrient	Pasture	Concentrate	Selenosource[2]
CP %	14.3	15.37	25.00
Fat (EE) %	2.6	2.45	3.00
ADF %	36.41	16.77	-
Crude Fiber %	-	-	5.50
TDN %	60.14	72.27	-
Ca %	0.48	0.98	0.20
P %	0.41	0.56	0.90
Se ppm	0.19	0.63	600.00
Organic Se ppm	-	-	588.00
DE (Mcal/kg)[3]	2.17	3.14	-

[1]. Supplemental selenium provided as Selenosource (Diamond V Mills, Inc. Cedar Rapids, IA)
[2]. Selenosource values provided by manufacturer
[3]. DE calculated based on NRC, 2007

Sample Collection

At the same time each collection day, body condition score **(**BCS), body weight (BW), rump fat (RF), blood, muscle, placenta, and colostrum samples were obtained from each mare throughout the trial. Body condition score, BW, RF measurements, and blood samples were collected every 14 d and muscle samples every 28 d from the start of the third trimester (d 0) until parturition. At parturition, placenta and colostrum samples were collected and colostrum quality recorded. Foal blood and muscle samples were similarly collected (blood every 14 d and muscle every 28 d until d 56) with day 0 being birth. Furthermore, foaling parameters (time to stand and time to nurse) were recorded at parturition. Foal physical characteristics such as BW, body length, hip

height, and whither height were determined at 12 hrs of age along with the first muscle sample.

Blood Samples

Beginning at the start of the third trimester (d 0) and every 22 wks until parturition, blood samples were collected from mares prior to the morning feeding via jugular venipuncture. Samples were harvested into evacuated tubes containing lithium heparin (Becton-Dickinson, Franklin Lakes, NJ.) and placed on ice until centrifugation. Samples were centrifuged at 2700 g and 10˚C for 20 min (ALC, PM140R, Thermo Fisher Sci., Waltham, MA.), plasma was harvested and stored at -20˚C until further analysis for Se and Gsh-Px. Foal blood samples were similarly collected and processed. Sampling began at birth and continued every 14 d until 56 days of age.

Parturition Observations and Colostrum Samples

When signs of parturition were apparent, mares were housed in individual 3.7 x 7.3 meter stalls and kept under supervision. At parturition; time of water break to birth, time of birth to placental expulsion, foal time to stand, and foal time to nurse was recorded and placenta was weighed. Following parturition, and prior to nursing, approximately 100 ml of colostrum was collected into a conical vial (VWR Int., West Chester, PA.) and stored at -20˚C until analysis for Se concentration. Refractometer (Equine Colostrum Refractometer, Animal Reproduction Systems, Chino, CA.) and colostrometer (Equine Colostrometer, Lane Manufacturing, Denver, CO.) readings were also obtained to determine colostrum quality.

Muscle Biopsies

Muscle biopsies began at d 0 and were repeated every 28 d until parturition for mares (d 56 in foals). On collection day, immediately pre-procedure, the sample site was sterilely prepped by shaving with a # 40 blade, then scrubbing 3x with chloradine (Chloradine Scrub 4%, RX Veterinary Products, Grapevine TX.) and alcohol (Isopropyl Alcohol 70%, Aaron Industries, Lynwood CA.). Following sterile preparation, 2.5 ml (1.5 ml in foals) of lidocaine (Lidocaine HCL 2%, RX Veterinary Products, Grapevine TX.) was injected subcutaneously. After approximately 5 min, samples (approximately 1g each) were collected as described by Snow and Guy (1976) from the right-middle gluteal muscle via purcutaneous needle biopsy (5 mm Biopsy Needle, Popper and Sons Inc. New Hyde Park, NY.). Incision sites were cleaned and a liquid suture was applied. Immediately after collection samples were placed in cryogenic vials (polypropylene 5ml low temp freezer vials, VWR Int., West Chester PA.) and snap-frozen in liquid nitrogen. Muscle samples were stored at -60˚C until analysis for Se.

Body Condition Score, Body Weight, and Rump Fat

Beginning at d 0, and every 14 d thereafter until parturition**,** BCS was determined by four individuals (two constant, and two rotating) utilizing the system described by Henneke et. al. (1983). Following BCS scoring, BW was determined by a digital scale (CAS Corp. Seoul, Rep. of Korea). Finally, RF was measured via ultrasonic images on the left hip at a point 5 cm dorsal of halfway between the coccygeal vertebrae one and the ischium (Westervelt et. al., 1976). An image was generated using an Aloka 55D-500V ultrasound (Aloka inc., Tokyo, Japan) and RF measured and recorded.

Forage and Concentrate Samples

Forage samples randomly obtained from all pastures and concentrate samples collected weekly throughout the trial were subsequently dried in a forced air oven (Lindberg/Blue M, Asheville, NC.) at 60˚C for 96 hrs. Samples were then ground for homogenization to 2 mm (Thomas model 4 Wiley Mill, Thomas Scientific, Swedesboro, NJ.). Ground samples were analyzed by a commercial laboratory (SDK Laboratories, Hutchinson, KS.) for nutrient (DM, CP, ADF, TDN, Fat (EE)) and mineral (Ca, P, Se) content (Table 1).

Sample Analysis

Plasma Glutathione Peroxidase (Gsh-Px) Analysis. Plasma Gsh-Px activity was determined by a commercially available colorimetric assay kit (Bioxytech, Foster City, CA.). Sample analysis began by mixing the sample with an NAD phosphate (NADPH) reagent (comprised of NADPH, glutathione, and glutathione reductase), and the manufacturer provided assay buffer in a semi-micro acrylic cuvette (VWR Int., West Chester PA.). Immediately after placing the cuvette in the spectrophotometer, tert-butyl hydroperoxide was added to the solution and was subsequently reduced by glutathione (this reduction is catalyzed by the organic peroxide contained in the sample). The oxidized form of glutathione produced by the preceding reaction was recycled back to glutathione via glutathione reductase and hydronium ions from NADPH, yielding glutathione and $NADP^+$. The oxidation of NADPH to $NADP^+$ was directly correlated to a decrease in absorbance at 340 nm and is analogous with Gsh-Px activity. Therefore, one unit of Gsh-Px activity is equal to a one unit decrease of NADPH. The extinction

coefficient 6220 M-1 cm-1 at 340 nm was used to calculate NADPH concentration. Mare plasma was diluted 1:10 (vol/vol) in assay buffer in order to measure values within the manufacturers recommended range (0.035 to 0.15 $A_{340/min}$) while foal plasma required no dilution.

This assay provides an indirect measure of plasma glutathione peroxidase activity and therefore must be expressed per unit of plasma protein. Plasma protein concentrations were determined using a commercial kit (Sigma-Aldrich Co., Saint Louis, MO.). The commercially available kit utilized pyrogallol red-molybdate complexes which bind to basic amino acid groups of protein molecules. Once the amino acid groups are bound to the pyrogallol red-molybdate complexes, spectrophotometry can be utilized to ascertain the protein concentration of the sample via an increase in absorbance of light at 600 nm. The increase in absorbance at 600 nm is directly proportional to the protein concentration in the sample. The spectrophotometric readings required by the Gsh-Px and plasma protein assays were produced via a Pharmacia LKB Ultrospec III spectrophotometer (Amersham Biosciences, Piscataway, NJ.)

Muscle, Colostrum, and Plasma Selenium Analysis. Muscle, colostrum, and plasma selenium concentrations were determined by North Dakota State University (Fargo, ND.) using atomic absorption spectrophotometery (AA Spec) with a hydride generator (A Analyst 800, PerkinElmer, Waltham, MA.). Sample preparation for AA Spec analysis consisted of weight analysis of sample followed by preparatory digestion. Samples were thawed and apportioned to 0.5 grams aliquots. Samples were then placed in 100 ml tall form beakers and 10 ml of 40% magnesium nitrate solution, 2 ml of 6 M

hydrochloric acid, and 10 ml of 16 M nitric acid were added and samples swirled gently until wet. Beakers were covered with a watch glass and left overnight (approximately 15 hours) on a very low reflux. The following morning, watch glasses were removed and samples were evaporated to dryness. Samples were then ashed 470˚C for 12-16 hrs in a muffle oven. Following ashing samples were allowed to cool prior to the addition of 10 ml of 12 M hydrochloric acid, samples were swirled gently until solids were dissolved. Watch glasses were replaced onto the beakers then placed on a hotplate for 15 min. Dissolved they were poured into individual 25 ml volumetric flask, rinsed with deionized distilled water and diluted to volume. Accurately diluted samples were then poured into 100 ml beakers. Samples were then transferred to scintillation vials, capped tightly, and stored until analysis.

Statistical Analysis

Data were analyzed as a 2 x 2 factorial design and values are presented as LS means ± standard error. Foaling parameters, colostrum Se concentrations, refractometer, and colostrometer data were analyzed using the PROC GLM procedures of SAS (SAS Inst. Inc., Cary, NC) while plasma and muscle Se and plasma Gsh-Px were analyzed using PROC MIXED. The model contained effects for block (early, mid-1, mid-2, and late), nutrition (P vs G), level of Se (none vs supplement), and nutrition x SeM interaction. Main effects were considered significant when $P < 0.05$ and a trend toward significance when $P < 0.10$.

CHAPTER III

RESULTS

Mare BW, BCS, and Rump Fat

Mare BW, BCS, and RF were all influenced by dietary treatment (Table 2). Dietary energy intake (nutrition) influenced mare BW with PG and PGS mares being heavier ($P < 0.01$) than P or PS mares. Mares in the P group exhibited a mean weight gain of 43 kg from d0 to d112, while PS gained 41 kg, PG 61 kg, and PGS 44 kg. There was no influence of SeM supplementation ($P > 0.88$) or an interaction between nutrition and SeM ($P > 0.18$) with respect to mare BW. Similarly, nutrition resulted in PG and PGS mares achieving greater mean BCS and RF ($P < 0.0001$ and $P < 0.02$, respectively) compared to P and PS mares. While SeM supplementation had no effect ($P > 0.44$ and $P > 0.98$, respectively) on mare BCS and RF, the interaction between SeM and nutrition tended to influence mare BCS and RF ($P > 0.06$ and $P > 0.08$, respectively) with SeM supplementation increasing PGS mares BCS and RF values while PS mares exhibited a decrease in these values.

Table 2. Effect of dietary energy manipulation and selenomethionine (SeM) supplementation on LS mean mare BW, BCS, and rump fat (RF) values

	Treatment[1]				Main Effect[2]		
Measurement	P	PS	PG	PGS	Nut	SeM	Inter
BW (kg)	572 (± 13.88)	554 (± 12.97)	603 (± 16.41)	624 (± 12.99)	P < 0.002	P > 0.88	P > 0.18
BCS[3]	4.9 (± 0.27)	4.6 (± 0.26)	5.8 (± 0.32)	6.6 (± 0.26)	P < 0.0001	P > 0.44	P > 0.06
RF (mm)[4]	1.01 (± 0.12)	0.78 (± 0.12)	1.11 (± 0.14)	1.33 (± 0.12)	P < 0.02	P > 0.98	P > 0.08

[1]Dietary treatments: P = pasture only (Coastal Bermudagrass pasture), PS = pasture plus SeM supplementation, PG = pasture plus concentrate supplementation, and PGS = pasture plus concentrate plus SeM supplementation.
[2]Nut = influence of nutrition (PG + PGS vs. P + PS), SeM = influence of selenomethionine (PGS + PS vs. PG + P), Inter = interaction between Nut and SeM (PGS + P vs. PG + PS).
[3]Determined using methods described by Henneke et al., 1983.
[4]Determined using methods described by Westervelt et al., 1976.

Mare Plasma Se and Plasma Gsh-Px

Mare plasma Se concentrations (Table 3) were influenced by nutrition ($P < 0.02$) (Figure 3) with PG and PGS mares having greater concentrations than P and PS mares. Selenomethionine supplementation likewise influenced ($P < 0.01$) plasma Se concentrations (Figure 3) with PS and PGS mares having greater values than P or PG mares. Additionally, there was a tendency towards influence ($P > 0.08$) of a SeM x nutrition interaction with PS mares exhibiting a greater increase in plasma Se concentrations than PGS mares. Despite changes in mare plasma Se concentrations, mare plasma Gsh-Px activities (Table 3) were not influenced by nutrition ($P > 0.89$), SeM supplementation ($P > 0.55$), or SeM x nutrition interactions ($P > 0.25$) (Figure 4).

Table 3. Effect of dietary energy manipulation and selenomethionine (SeM) supplementation on LS mean mare plasma, muscle, and colostrum selenium (Se) concentrations and plasma glutathione peroxidase (Gsh-Px) activities

	Treatment[1]				Main Effect[2]		
Measurement	P	PS	PG	PGS	Nut	SeM	Inter
Plasma Se (µg/mL)	0.2242 (±0.006)	0.2548 (±0.005)	0.2525 (±0.007)	0.2599 (±0.006)	P < 0.02	P < 0.01	P < 0.08
Muscle Se (µg/mg)	0.3170 (±0.023)	0.3616 (±0.021)	0.2821 (±0.028)	0.4188 (±0.023)	P > 0.64	P < 0.01	P < 0.07
Colostrum Se (µg/mL)	0.1790 (±0.029)	0.2548 (±0.030)	0.2030 (±0.035)	0.3192 (±0.027)	P > 0.16	P < 0.01	P > 0.51
Plasma Gsh-Px (mU/mg protein)	6.8459 (±0.397)	7.5622 (±0.366)	7.3721 (±0.468)	7.1352 (±0.379)	P > 0.89	P > 0.55	P > 0.25

[1]Dietary treatments: P = pasture only (Coastal Bermudagrass pasture), PS = pasture plus SeM supplementation, PG = pasture plus concentrate supplementation, and PGS = pasture plus concentrate plus SeM supplementation.

[2]Nut = influence of nutrition (PG + PGS vs. P + PS), SeM = influence of selenomethionine (PGS + PS vs. PG + P), Inter = interaction between Nut and SeM (PGS + P vs. PG + PS).

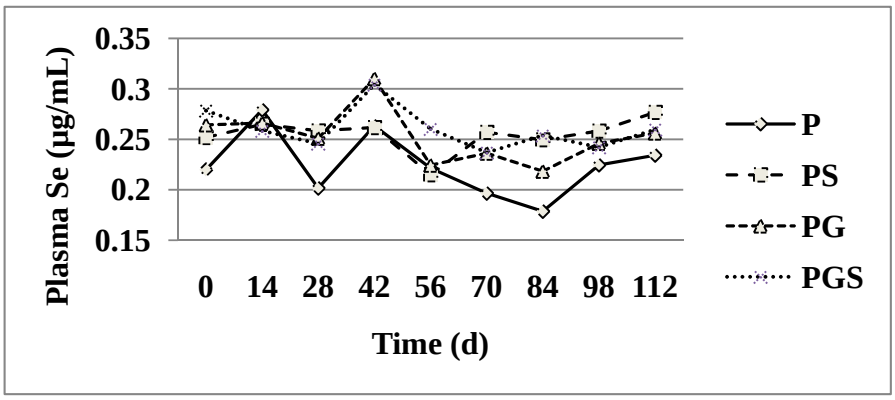

Figure 3. The effect of selenomethionine supplementation and dietary energy manipulation on mare plasma Se concentrations (P = pasture only (Coastal Bermudagrass pasture), PS = pasture plus SeM supplementation, PG = pasture plus concentrate supplementation, and PGS = pasture plus concentrate plus SeM supplementation).

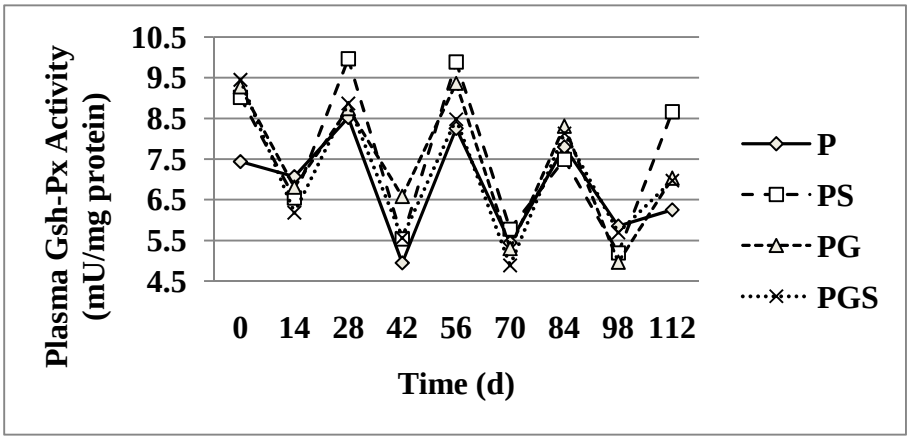

Figure 4. The effect of selenomethionine supplementation and dietary energy manipulation on mare plasma Gsh-Px activity (P = pasture only (Coastal Bermudagrass pasture), PS = pasture plus SeM supplementation, PG = pasture plus concentrate supplementation, and PGS = pasture plus concentrate plus SeM supplementation).

Mare Muscle and Colostrum Se and Colostrum Quality

Mare muscle Se concentrations (Table 3) were altered ($P < 0.01$) by SeM supplementation (Figure 5) with PS and PGS mares having greater muscle Se concentrations than P or PG mares. Furthermore, there was a tendency towards a

nutrition x SeM interaction (P > 0.07) with PGS mares exhibiting a greater muscle Se concentration increase than PS mares. However, there was no influence of nutrition (P > 0.64) on mare muscle Se concentrations.

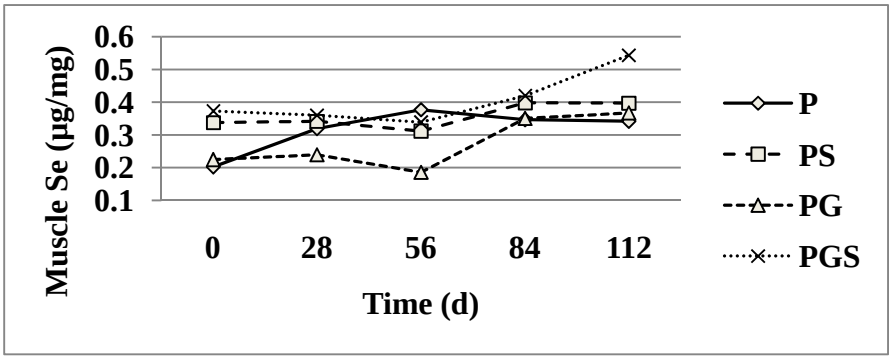

Figure 5. The effect of selenomethionine supplementation and dietary energy manipulation on mare muscle Se concentrations (P = pasture only (Coastal Bermudagrass pasture), PS = pasture plus SeM supplementation, PG = pasture plus concentrate supplementation, and PGS = pasture plus concentrate plus SeM supplementation).

Colostrum Se concentrations (Table 3) were influenced (P < 0.01) by SeM supplementation, with mares from the PS and PGS groups having greater colostrum Se concentrations than P or PG mares. While colostrum Se concentrations were not influenced by nutrition (P > 0.1) or nutrition x SeM interactions (P > 0.5). Colostrum quality, as determined by a colostrometer, did not differ with nutrition, SeM supplementation, or nutrition x SeM interactions (P > 0.9). However, there was an influence (P < 0.02) of nutrition with P and PS mares having greater brix percentages (refractometer) than PG or PGS mares (Table 4).

Table 4. Effect of dietary energy manipulation and selenomethionine (SeM) supplementation on LS mean mare colostrum quality via colostrometer and refractometer analysis

	Treatment[1]				Main Effect[2]		
Measurement	P	PS	PG	PGS	Nut	SeM	Inter
Refractometer (Brix %)	32 (±1.99)	32 (±2.74)	26 (±2.11)	26 (±2.11)	P < 0.02	P > 0.91	P > 0.94
Colostrometer (mg/mL)	1.08 (±0.01)	1.08 (±0.01)	1.07 (±0.01)	1.09 (±0.01)	P > 0.89	P > 0.39	P > 0.23

[1]Maternal dietary treatments: P = pasture only (Coastal Bermudagrass pasture), PS = pasture plus SeM supplementation, PG = pasture plus concentrate supplementation, and PGS = pasture plus concentrate plus SeM supplementation.
[2]Nut = influence of maternal nutrition (PG + PGS vs. P + PS), SeM = influence of maternal selenomethionine (PGS + PS vs. PG + P), Inter = interaction between maternal Nut and SeM (PGS + P vs. PG + PS).

Foaling Parameters

There was no effect of plane of nutrition (P > 0.10), SeM supplementation (P > 0.15), or nutrition x SeM interaction (P > 0.23) on the following foaling parameters (Table 5): time of water break to birth (min), time of birth to placental expulsion (min), foal time to stand (min), foal time to nurse (min), placenta weight (kg), placenta weight expressed as percent average pre-parturition mare BW, placenta weight expressed as percent average foal birth BW, foal birth weight (kg), foal hip height (cm), whither height (cm), or foal body length (cm). However, PGS and P mares had a difference in gestational length via a nutrition x SeM interaction (P < 0.02). Mares in the PGS and P groups had shorter average gestational lengths (336.6 days ± 2.7, 342.7 days ± 3.2 respectively), while PG and PS mares had longer average gestational lengths (349.2 days ± 3.6, 346.3 days ± 2.7 respectively).

Table 5. Effect of dietary energy manipulation and selenomethionine (SeM) supplementation on LS mean mare foaling parameters and foal physical characteristics

	Treatment[1]				Main Effect[2]		
Measurement	P	PS	PG	PGS	Nut	SeM	Inter
Time Water Break to Birth (min)	19.8 (±4.1)	13.7 (±4.3)	13.2 (±4.3)	14.2 (±3.7)	P > 0.45	P > 0.55	P > 0.41
Time Birth to Placental Expulsion (min)	81.4 (±23.1)	60.9 (±22.6)	89.7 (±23.1)	65.1 (±23.1)	P > 0.79	P > 0.33	P > 0.92
Foal Time to Stand (min)	47.9 (±11.1)	58.6 (±11.7)	38.9 (±11.7)	58.3 (±10.3)	P > 0.67	P > 0.21	P > 0.71
Foal Time to Nurse (min)	93.3 (±11.2)	92.2 (±11.8)	83.0 (±11.8)	89.9 (±10.4)	P > 0.57	P > 0.80	P > 0.73
Placenta Weight (kg)	4.2 (±0.41)	4.3 (±0.40)	3.9 (±0.40)	4.5 (±0.35)	P > 0.93	P > 0.39	P > 0.61
Placenta Weight as % Mare BW	0.7 (±0.00)	0.8 (±0.00)	0.6 (±0.00)	0.7 (±0.00)	P > 0.42	P > 0.20	P > 0.87
Placenta Weight as % Foal BW	8.6 (±0.01)	9.3 (±0.01)	8.8 (±0.01)	9.5 (±0.00)	P > 0.65	P > 0.18	P > 0.97
Foal Birth Weight (kg)	48 (±2.40)	47 (±2.19)	46 (±2.89)	48 (±2.19)	P > 0.97	P > 0.76	P > 0.44
Foal Birth Weight as Percent Mare BW (%)	8.0 (±0.00)	8.0 (±0.00)	7.5 (±0.00)	7.6 (±0.00)	P > 0.10	P > 0.88	P > 0.85
Foal Body Length (cm)	75.9 (±2.15)	76.2 (±1.84)	74.3 (±2.43)	78.3 (±1.84)	P > 0.90	P > 0.32	P > 0.39
Foal Whither Height (cm)	95.8 (±1.72)	94.5 (±1.57)	93.4 (±2.07)	94.9 (±1.57)	P > 0.56	P > 0.93	P > 0.42
Foal Hip Height (cm)	99.1 (±1.92)	96.9 (±1.75)	95.3 (±2.31)	96.3 (1.75)	P > 0.27	P > 0.76	P > 0.42

[1]Dietary treatments: P = pasture only (Coastal Bermudagrass pasture), PS = pasture plus SeM supplementation, PG = pasture plus concentrate supplementation, and PGS = pasture plus concentrate plus SeM supplementation.
[2]Nut = influence of nutrition (PG + PGS vs. P + PS), SeM = influence of selenomethionine (PGS + PS vs. PG + P), Inter = interaction between Nut and SeM (PGS + P vs. PG + PS).

Foal Plasma and Muscle Se and Plasma Gsh-Px

Foal plasma and muscle Se concentrations and plasma Gsh-Px activities (Table 6) reacted to maternal dietary treatments differentially. Maternal plane of nutrition influenced foal plasma (Figure 6) and muscle (Figure 7) Se concentrations ($P < 0.04$ and 0.02 respectively) with foals of PG and PGS mares having greater Se concentrations than foals of P and PS mares. Similarly, maternal SeM supplementation influenced foal plasma (Figure 6) and muscle (Figure 7) Se concentrations ($P < 0.01$ and $P < 0.01$ respectively) with foals from PS and PGS mares having greater values than foals of P and PG mares. Additionally, there was a maternal nutrition x SeM interaction ($P < 0.01$) in regards to foal muscle Se concentrations with foals from PGS mares exhibiting a greater increase in muscle Se concentrations than foals from PS mares. However, maternal nutrition x SeM interactions did not influence ($P > 0.34$) foal plasma Se concentrations. Furthermore, foal plasma Gsh-Px activities were not influenced ($P > 0.12$) by maternal plane of nutrition, SeM supplementation of the dam, or maternal nutrition x SeM interactions (Figure 8).

Table 6. Effect of maternal dietary energy manipulation and selenomethionine (SeM) supplementation on LS mean foal plasma and muscle selenium (Se) concentrations and plasma glutathione peroxidase (Gsh-Px) activities

	Treatment[1]				Main Effect[2]		
Measurement	P	PS	PG	PGS	Nut	SeM	Inter
Plasma Se (µg/mL)	0.1181 (±0.006)	0.1496 (±0.006)	0.1375 (±0.007)	0.1575 (±0.005)	P < 0.04	P < 0.01	P > 0.34
Muscle Se (µg/mg)	0.3815 (±0.022)	0.4249 (±0.021)	0.3530 (±0.026)	0.5733 (±0.021)	P < 0.02	P < 0.01	P < 0.01
Plasma Gsh-Px (mU/mg protein)	8.3341 (±0.483)	7.7179 (±0.458)	8.0612 (±0.571)	7.1039 (±0.452)	P > 0.37	P > 0.12	P > 0.73

[1]Maternal dietary treatments: P = pasture only (Coastal Bermudagrass pasture), PS = pasture plus SeM supplementation, PG = pasture plus concentrate supplementation, and PGS = pasture plus concentrate plus SeM supplementation.
[2]Nut = influence of maternal nutrition (PG + PGS vs. P + PS), SeM = influence of maternal selenomethionine (PGS + PS vs. PG + P), Inter = interaction between maternal Nut and SeM (PGS + P vs. PG + PS).

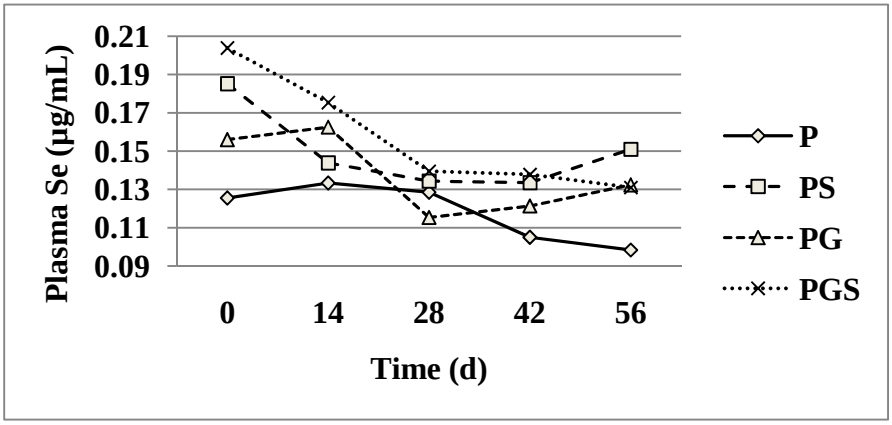

Figure 6. The effect of maternal selenomethionine supplementation and dietary energy manipulation on foal plasma Se concentrations (P = pasture only (Coastal Bermudagrass pasture), PS = pasture plus SeM supplementation, PG = pasture plus concentrate supplementation, and PGS = pasture plus concentrate plus SeM supplementation).

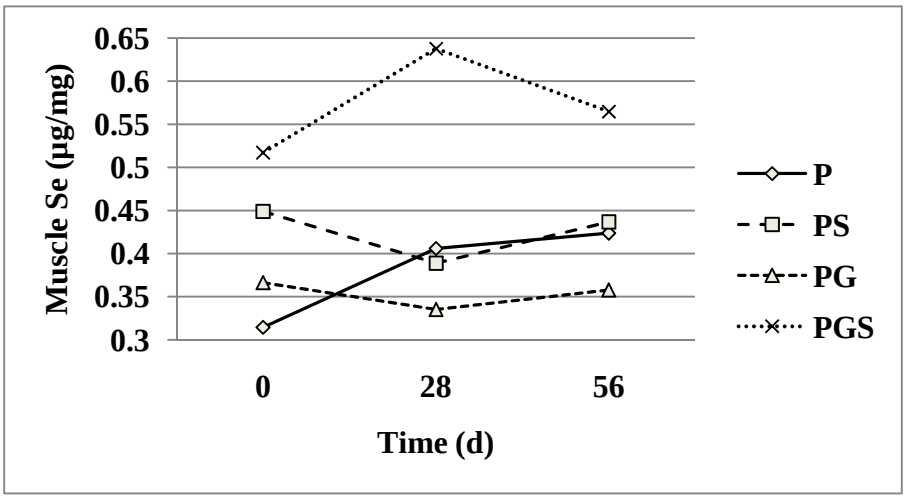

Figure 7. The effect of maternal selenomethionine supplementation and dietary energy manipulation on foal muscle Se concentrations (P = pasture only (Coastal Bermudagrass pasture), PS = pasture plus SeM supplementation, PG = pasture plus concentrate supplementation, and PGS = pasture plus concentrate plus SeM supplementation).

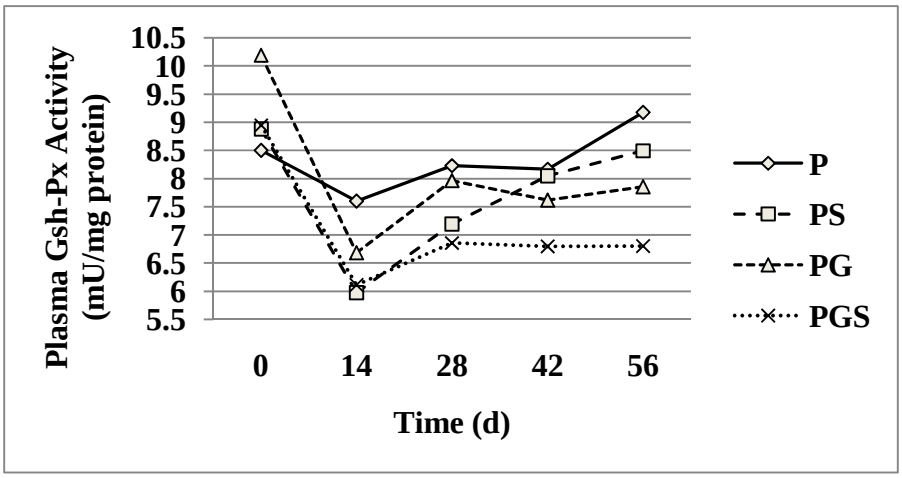

Figure 8. The effect of maternal selenomethionine supplementation and dietary energy manipulation on foal plasma Gsh-Px activity (P = pasture only (Coastal Bermudagrass pasture), PS = pasture plus SeM supplementation, PG = pasture plus concentrate supplementation, and PGS = pasture plus concentrate plus SeM supplementation).

CHAPTER IV

DISCUSSION

Prior work to address Se supplementation in horses agrees that dietary Se supplementation (regardless of source) does influence plasma/serum and muscle Se status (Shellow et al., 1985; Wichtel et al. 1998; Janicki et al., 2001; Richardson et al., 2006). Previous studies investigating organic Se supplementation in horses gave inconsistent and inconclusive outcomes. In some studies organic Se supplementation resulted in an increase in plasma/serum Se concentrations, while others have reported no influence.

Janicki et al. (2001) reported that SeM supplementation increased mare serum Se concentrations to a greater extent than supplementation with inorganic Se. While the work of Richardson et al. (2006) observed no difference in the effect of SeM or NaSe on plasma Se concentrations. The difference between these two studies may be explained partially by the pre-study Se status of the animal. Richardson et al. (2006) reported that initial plasma Se concentrations were below the reference range of 0.13 to 0.16 µg/mL reported by Stowe and Herdt (1992) but above Se deficient concentrations of 0.008 to 0.05 µg/mL (Lee et al., 1995). While Janicki et al. (2001) did not report initial serum Se concentrations; the study was performed in Kentucky which has historically been a Se deficient state. However, dietary Se concentrations were not reported for feedstuffs utilized by Janicki et al. (2001) and this may be an additional source of variability between these studies.

Plasma Se concentrations in the current study were influenced by SeM supplementation with final mean concentrations ranging from 0.22 (P mares) to 0.26 (PGS mares) µg/mL. These values are well above those reported by Richardson et al. (2006), which observed final plasma Se concentrations of 0.096 to 0.169 µg/mL. The difference between these concentrations may be in part due to the length of SeM supplementation. While the current study provided supplemental SeM for an average of 112 days, Richardson et al. (2006) supplemented horses for only 56 days. However, when comparing a similar length of SeM supplementation (56 days), horses in the current study continued to show greater plasma Se concentrations than those in the Richardson et al. (2006) study. Furthermore, it should be noted that while plasma Se concentrations appeared to plateau by d 28 in previous studies (Shellow et al., 1985; Richardson et al., 2006), plasma Se concentrations in the current study peaked by day 42, experienced a slight decline, then continued to increase until day 112. However, the current study observed this effect in mature, pregnant mares while previous studies utilized 18 month old geldings and non-pregnant females (Richardson et al., 2006). Therefore, these differences may be due in part to an effect of physiological status (growth, pregnancy, etc.) or age of animals utilized. Sunde et al., (2005) noted that the dietary Se requirement was less for gestating rats than that for growing rats however; Se requirements for growth in horses have not been determined (NRC, 2007) and further research is needed to provide definitive requirements for horses. Additionally, amount of Se provided may be a source of variation between the current work and that of Janicki et al. (2001) and Richardson et al. (2006). While Janicki et al. (2001) provided

supplemental Se at 1 or 3 mg Se/d, Richardson et al. (2006) supplemented Se at the rate of 0.45 mg Se/kg DM, and the current study provided 0.3 mg Se/kg DM supplemental Se. Although Janicki et al. (2001) did not report total dietary Se intake (feedstuff plus supplemental Se) Richardson et al. (2006) reported total dietary Se intakes of 0.15 and 0.60 mg Se/kg DM, while the current study provided 0.19, 0.35, 0.49, and 0.69 mg Se/kg DM.

Furthermore, the current study manipulated DE intake and this aspect was not investigated in the work of either Janicki et al. (2001) or Richardson et al. (2006). Because the current study reports that DE manipulation and nutrition x SeM interactions do influence mare plasma Se, this may be another possible explanation of the differences observed between these studies. While plane of nutrition, SeM supplementation, and nutrition x SeM interactions appear to influence mare plasma Se, they do not seem to influence mare plasma Gsh-Px activity.

Previous studies observing Se supplementation and its influence on Gsh-Px activity in horses have been inconclusive. Janicki et al. (2001) reported that SeM supplementation of mares resulted in an increase in whole blood Gsh-Px activity in their foals. Conversely, the current study and the work of Richardson et al. (2006) are in agreement that SeM supplementation did not affect plasma/whole blood Gsh-Px activities. Richardson et al. (2006) reported that final plasma Gsh-Px activities ranged from 10.0 to 12.3 mU/mg protein. The current study observed lower final plasma Gsh-Px activities of 6.8 to 7.5 mU/mg protein, however it is important to note that Richardson et al. (2006) used immature horses while the current study used pregnant

mares. Janicki et al. (2001) did not report Gsh-Px values for the pregnant mares used in their study. The lack of pertinent data makes it difficult to determine the effect of physiological status on Gsh-Px activities in horses. However, the lack of increased Gsh-Px activity observed by Richardson et al. (2006) and the current study, and the increase noted by Janicki et al. (2001) may be partially due to pre-study Se status. Sunde et al. (2005) noted a plateau in plasma Gsh-Px activity which correlated with dietary Se levels in rats. This is in agreement with the work of Shellow et al. (1985) who reported that a plateau in Gsh-Px activity, via adequate pre-study Se levels of horses, may have been the reason they did not observe an increase in Gsh-Px activities with Se supplementation. Therefore, horses utilized in the current study, and those utilized by Richardson et al. (2006), may have reached a plasma Gsh-Px plateau prior to the beginning of the studies due to the adequate Se in those environments. However, because Janicki et al. (2001) failed to report initial mare Se status values, it is difficult to ascertain whether this is the case definitively. It should also be noted that while Janicki et al. (2001) and Richardson et al. (2006) investigated the effect of SeM supplementation on whole blood Gsh-Px, the current study did not. Although Richardson et al. (2006) did not observe an effect of treatment on either plasma or whole blood Gsh-Px, it may be a possible explanation of the difference observed between the current study and that of Janicki et al. (2001). Additionally, amount of dietary Se and DE manipulation may be sources of differentiation between the results of these studies. Although plasma Gsh-Px was unaffected by nutrition or selenomethionine supplementation in the current study, mare muscle Se concentrations appear to react differently.

Mare muscle Se concentrations in the current study were influenced by SeM supplementation. Conversely, Richardson et al. (2006) reported that Se supplementation (regardless of source) did not affect muscle Se concentrations. Mean final muscle Se concentrations in the current study ranged from 0.282 (PG mares) to 0.418 (PGS mares) μg/mg and increased with increasing levels of dietary Se. Final muscle Se concentrations reported by Richardson et al. (2006) ranged from 0.079 to 0.101 μg/mg. Amount of dietary Se provided may be a factor in the difference observed between these studies. However, mares in the current study which received 0.35 mg Se/kg DM daily had a final mean muscle Se concentration of 0.282 μg/mg. While Richardson et al. (2006) reported that horses receiving 0.46 mg Se/kg DM daily had a final muscle Se concentration of 0.101 μg/mg.

Another possible explanation is the amount of time that Se supplementation occurred. Richardson et al. (2006) supplemented horses for a total of 56 days, while the current study supplemented for an average of 112 days. Mare muscle Se concentrations were also influenced by nutrition x SeM interactions in the current study. While Richardson et al. (2006) did not investigate this relationship; it may be an explanation of the differences observed between these studies. Furthermore, age and physiological status of horses may be a source of differentiation. Richardson et al. (2006) utilized 18 month old immature horses while the current study utilized mature pregnant mares. However, further research is needed to further clarify this relationship.

Similar to mare muscle Se concentrations, Colostrum Se concentrations in the current study were influenced by SeM supplementation. Colostrum Se concentrations

ranged from 0.179 to 0.319 μg/mL. This finding agrees with the work of Janicki et al. (2001) which likewise reported in increase in colostrum Se concentrations with SeM supplementation. Additionally, Janicki et al. (2001) reported that Se from an organic source was more effective in increasing colostrum Se concentrations than inorganic sources. While the current study did not observe the difference between organic and inorganic Se, it was concluded that Se supplementation was more effective in raising colostrum Se concentrations than no Se supplementation or simply providing adequate Se through increased nutrition.

Additionally, the relationship of SeM supplementation and the plane of nutrition seems to influence mare gestational length. In the current study there was a nutrition x SeM interaction which resulted in shorter gestational lengths for mares receiving supplemental SeM and increased DE, as well as mares receiving no supplementation whatsoever. This relationship has also been noted by researchers at North Dakota State University when investigating the effect of varying levels of DE and SeM supplementation in sheep (personal communication, Dr. Carrie Hammer). However, further research is needed to more fully elucidate this relationship.

Selenomethionine supplemententation and DE manipulation not only influence the Se status of mares, there is also an influence of maternal dietary treatments on foals. Previous studies have reported that offspring Se status is correlated with maternal Se status during gestation (Lee et al., 1995, Mahan and Kim, 1996). Foal plasma Se concentrations in the current study were influenced by maternal DE intake and SeM supplementation with average mean concentrations ranging from 0.118 to 0.157 μg/mL.

This finding agrees with the work of Janicki et al. (2001) which reported that foals from mares supplemented with SeM had greater serum Se concentrations at 12 hrs and 2, 4, 6, and 8 weeks of age. This relationship has also been noted in cows (Ortman and Pehrson, 1999; Guyot et al., 2007), sheep (Rock et al., 2001; Reed et al., 2007) and pigs (Loudenslager et al. 1986; Mahan and Kim, 1996). However, further research may be beneficial in order to define requirements in horses.

While foal plasma Se concentrations are influenced by maternal DE intake and SeM supplementation, foal plasma Gsh-Px activities appear to react differently. Although previous studies regarding maternal DE manipulation and organic Se supplementation and their effect on foal plasma Gsh-Px activity are somewhat limited. Janicki et al. (2001) reported that at 6 and 8 weeks of age, foals from mares supplemented with 3 mg Se/d (regardless of source) had greater whole blood Gsh-Px activities than foals from mares supplemented with 1 mg Se/d as NaSe. Conversely, foals from mares in the current study exhibited no increase in plasma Gsh-Px activity. Mean foal plasma Gsh-Px activities ranged from 7.1 to 8.3 mU/mg protein and were not influenced by treatment. While Janicki et al. (2001) investigated Gsh-Px activities in whole blood and the current study utilized plasma, Richardson et al. (2006) found no appreciable difference between either medium. Furthermore, while pre-study maternal Se status and the influence of DE manipulation may be possible explanations of the differences observed between the current study and the work of Janicki et al. (2001), additional research is needed to further clarify this relationship.

Although the relationship of maternal SeM supplementation and foal plasma/serum Se concentrations has been previously investigated, literature regarding foal muscle Se concentrations is limited. This is the first report of which the author is aware that reports the effect of maternal DE manipulation and SeM supplementation on foal muscle Se status. Foal muscle Se concentrations were influenced by maternal SeM supplementation and maternal DE intake. Mean muscle Se concentrations ranged from 0.353 to 0.573 μg/mg. These results suggest that supplemental SeM provided to mares in the third trimester of pregnancy can improve the tissue Se status of foals. These findings are in agreement with results of work in other non-ruminant species (Mahan and Kim, 1996; Kim and Mahan, 2001; Mahan and Peters, 2004). The ability to improve tissue Se concentrations prenataly is of particular importance because it is doubtful that foals receive sufficient Se through mares' milk alone due to the relatively low concentration of Se in mares' milk (Breedveld et al., 1988). Therefore, if foals have increased Se stores upon birth it is likely that they will be able to utilize stores to meet Se needs in the first few months of life. This is in accordance with the work of Janicki et al. (2001) who reported increased serum Se concentrations in foals from SeM supplemented mares through two months of age. However, further research is needed to define this relationship.

In contrast to foal muscle Se concentrations, foaling parameters and foal physical characteristics do not appear to be influenced by maternal dietary treatments. Foaling parameters and foal physical characteristics consisting of: time of water break to birth, time of birth to placental expulsion, foal time to stand, foal time to nurse, placenta

weight, placenta weight expressed as percent average pre-parturition mare BW, placenta weight expressed as percent average foal birth BW, foal birth weight, foal hip height, whither height, and foal body length were not affecting by treatments in the current study. This is in agreement with the work of Kubiak et al. (1988) who reported that when mares were fed to fatness (BCS of 8.5 to 9) or kept in a moderate body condition there was no effect on foaling parameters. Furthermore, when mares were provided with lower quality diets and relatively insufficient DE, there was no influence on foal birth weight (Guay et al., 2002; Heidler et al., 2004). These results suggest that when mares are overfed, or perhaps more importantly when underfed, maternal dietary nutrition may be preferentially partitioned to the foal. However, further research is needed to fully elucidate this relationship.

CHAPTER V

CONCLUSIONS

The results of this study indicate that maternal plane of nutrition and SeM supplementation in the third trimester of pregnancy does influence Se status, BW, BCS, and colostrum Se concentration of mares. While SeM supplementation does not affect mare physical characteristics, it may be beneficial in increasing mare Se status and colostrum Se concentrations. Furthermore, providing Se via SeM supplementation in addition to increased DE may be more effective in increasing mare Se status than providing increased Se through greater nutrition or SeM supplementation alone. However, SeM supplementation and increased nutrition, or these factors collectively, do not appear to be beneficial in increasing mare plasma Gsh-Px activities in an area known to be adequate in Se. Similarly, these treatments do not appear to be beneficial or detrimental to foal physical characteristics or behavior.

However, maternal plane of nutrition and SeM supplementation does influence foal plasma and muscle Se status. Although these treatments are beneficial in increasing foal Se status, it does not seem to be effective in increasing foal plasma Gsh-Px activities in an area known to be adequate in Se. While maternal plane of nutrition and SeM supplementation individually are effective in increasing foal plasma Se concentrations, collectively they are not. However, foal muscle Se concentrations are influenced by the interaction of maternal plane of nutrition and SeM supplementation in addition to these factors individually.

In conclusion, SeM supplementation is beneficial in increasing the Se status of mares in the third trimester of pregnancy and their foals. Increased DE intake is likewise beneficial for mares and their foals. However, feeding mares at 100% NRC recommendations versus 120% is not deleterious to mares or foal physical characteristics and foal behavior. Furthermore, providing excess DE to mares in the third trimester does not result in a definite increase in colostrum quality. However, further research is needed to fully elucidate these relationships in horses.

LITERATURE CITED

Adkins, R. S., and R. C. Ewan. 1984. Effect of selenium on performance, serum selenium concentration and glutathione peroxidase activity in pigs. J. Anim. Sci. 58:346-350.

Aiken, G. E., G. D. Potter, B.E. Conrad, and J. W. Evans. 1989. Voluntary intake and digestion of coastal bermuda grass hay by yearling and mature horses. Equine Vet. Sci. 9 (5):262-264.

Avellini, L., E. Chiaradia, and A. Gaiti. 1999. Effect of exercise training, Se and vitamin E on some free radical scavengers in horses (*Equus caballus*). Comp. Biochem. Physiol. Part B 123:147–154.

Awadeh, F. T., R. L. Kinkaid, and K. A. Johnson. 1998. Effect of level and source of dietary selenium on concentrations of thyroid hormones and Immunoglobulins in beef cows and calves. J. of Anim. Sci. 76:1204-1215.

Boyne, R. and J. R. Arthur. 1979. Alterations of Neutrophil function in selenium-deficient cattle. J. Comp. Path. 89:151-158.

Brady, P. S., P. K. Ku, and D. E. Ullrey. 1978. Lack of effect of Se supplementation on response of the equine erythrocyte glutathione system and plasma enzymes to exercise. J. Anim. Sci.47:492–496.

Breedveld L, Jackson S. G., Baker J. P. 1988. The determination of a relationship between the copper, zinc and selenium levels in mares and those in their foals. J. Equine. Vet. Sci. 8:378-382.

Cronjé, M. C., J.B.J. van Ryssen, and R.J. Coertze. 2006. Potential benefit of trace element supplementation of sheep on a low level of nutrition. S. Afri. J. Anim. Sci. 36:20-26.

Davis, P. A., L.R. McDowell, N.S. Wilkinson, C.D. Buergelt, R. Van Alstyne, R.N. Weldon c, T.T. Marshall a, E.Y. Matsuda-Fugisaki. 2008. Comparative effect of various dietary levels of Se as sodium selenite or Se yeast on blood, wool, and tissue Se concentrations of wether sheep. Small Rum. Res. 74:149–158.

Enjalbert, F., P. Lebreton, O. Salat, and F. Schelcher. 1999. Effects of Pre- or Postpartum Selenium Supplementation on Selenium Status in Beef Cows and Their Calves. J. Anim. Sci. 77:223–229.

Ghany-Hefnawya,A. E., R. L´opez-Arellano, A. Revilla-V´azquez, E. Ram´ırez-Bribiesca, J. T´ortora-P´erez. 2007. The relationship between fetal and maternal selenium concentrations in sheep and goats. Small Rum Res 73: 174–180.

Givens, D. I., R. Allison, B. Cottrill, and J. S. Blake. 2004. Enhancing the selenium content of bovine milk through alteration of the form and concentration of selenium in the diet of the dairy cow. J. Sci. Food Agric. 84:811–817.

Goehring, T. B., I. S. Palmer, O. E. Olson, G. W. Libal, and R. C. Wahlstrom. 1984. Effects of seleniferous grains and inorganic selenium on tissue and blood composition and growth performance of rats and swine. J. Anim. Sci. 59: 725-732.

Guay, K. A., H. A. Brady, V. G. Allen, K. R. Pond, D. B. Wester, L. A. Janecka, and N. L. Heninger. 2002. Matua bromegrass hay for mares in gestation and lactation. J. Anim. Sci. 80:2960–2966.

Gunter, S. A., P. A. Beck, and J. M. Phillips. 2003. Effects of supplementary selenium source on the performance and blood measurements in beef cows and their calves. J. Anim. Sci. 81:856-864.

Guyot, H., P. Spring, S. Andrieu, and F. Rollin. 2007. Comparative responses to sodium selenite and organic selenium supplements in Belgian Blue cows and calves. Livestock Science. 111: 259–263.

Heidler, B., J.E. Aurich, W. Pohl, and Chr. Aurich. 2004. Body weight of mares and foals, estrous cycles and plasma glucose concentration in lactating and non-lactating Lipizzaner mares. Theriogenology. 61:883–893.

Henneke, D. R., G. D. Potter, J. L. Kreider, and B. F. Yeates. 1983. Relationship between condition score, physical measurements and body fat percentage in mares. Equine Vet. J. 15 (4):371-372.

Hoffman, C., B. Rivinus, and L. Swanson. 1978. Effect of intramuscular administration of selenium and vitamin E in dairy heifers on erythrocyte glutathione peroxidase activity and blood selenium levels. J. of Anim. Sci. 47:192-197.

Hostetler, C. E., and R. L. Kincaid. 2004. Gestational changes in concentrations of selenium and zinc in the porcine fetus and the effects of maternal intake of selenium. Bio Trace Element Res. 97: 57-70.

Hyatt, M. A., H. Budge, D. Walker, T. Stephenson, and M. E. Symonds. 2007. Effects of maternal parity and late gestational nutrition on mRNA abundance for growth factors in the liver of postnatal sheep. Am. J. Physiol. Regul. Integr. Comp. Physiol. 292: R1934–R1942.

Hyatt, M. A., E A Butt, H Budge, T Stephenson and M E Symonds. 2008. Effects of maternal cold exposure and nutrient restriction on the ghrelin receptor, the GH–IGF axis, and metabolic regulation in the postnatal ovine liver. Reproduction. 135: 723–732.

Janicki, K. M., L. M. Lawrence, T. Barnes, and C. I. O'Connor. 2000. The effect of dietary selenium source and level on broodmares and their foals. J. Anim. Sci. 78: 172.

Janicki, K. M., L. M. Lawrence, T. Barnes, and C. J. Stine. 2001. The effect of dietary Se source and level on Se concentration, glutathione peroxidase activity, and influenza titers in broodmares and their foals. Pages 43–44 in Proc. 17th ENPS Symp., Lexington, KY.

Juniper, D. T., R. H. Phipps, D. I. Givens, A. K. Jones, C. Green, and G. Bertin. 2008. Tolerance of ruminant animals to high dose in-feed administration of a selenium-enriched yeast. J. Anim. Sci. 86:197–204.

Kim, Y. Y., and D. C. Mahan. 2001. Comparative effects of high dietary levels of organic and inorganic selenium on selenium toxicity of growing-finishing pigs. J. Anim. Sci. 79:942–948.

Knight, D. A., and W. J. Tyznik. 1990. The effect of dietary Se on humoral immunocompetence of ponies. J. Anim. Sci. 68:1311–1317.

Knowles, S. O., N. D. Grace, K. Wurms, and J. Lee. 1999. Significance of amount and form of dietary selenium on blood, milk, and casein selenium concentrations in grazing cows. J. Dairy Sci. 82:429–437.

Ku, P. K., E. R. Miller, R. C. Wahlstrom, A. W. Groce, J. P. Hitchcock, and D. E. Ullrey. 1973. Selenium supplementation of naturally high selenium diets for swine. J. Anim. Sci. 37:501-505.

Kubiak, J. R., J. W. Evans, G. D. Potter. P. G. Harms, and W.L. Jenkins. 1988. Parturition in the multiparous mare fed to obesity. Equine Vet. Sci. 8: 135-140.

Lawler, T. L., J. B. Taylor, J. W. Finley, and J. S. Caton. 2004. Effect of supranutritional and organically bound selenium on performance carcass

characteristics, and selenium distribution in finishing beef steers. J. of Anim. Sci. 82:1488-1493.

Lee, J., E.S. McAllister, and R.W. Scholz. 1995. Assessment of selenium status in mares and foals under practical management conditions. J Equi Vet Sci. 15: 240-245.

Lofstedt, J. 1997. White muscle disease of foals. Vet. Clin. North Am. Equine Pract. 13:169–185.

Loudenslager, M. J., P. K. Ku, P. A. Whetter, D. E. Ullrey, C. K. Whitehair, H. D. Stowe, and E. R. Miller. 1986. Importance of diet of dam and colostrum to the biological antioxidant status and parenteral iron tolerance of the pig. J. Anim. Sci. 63:1905-1914.

Mahan, D. C., A. L. Moxon, and M. Hubbard. 1977. Efficacy of inorganic selenium supplementation to sow diets on resulting carry-over to their progeny. J. Anim. Sci. 46:738-746.

Mahan, D. C., and A. L. Moxon. 1978. Effects of adding inorganic or organic selenium sources to the diets of young swine. J. Anim. Sci. 47:456-466.

Mahan, D. C., and Y. Y. Kim. 1996. Effect of inorganic or organic selenium at two dietary levels on reproductive performance and tissue selenium concentrations in first-parity gilts and their progeny. J. Anim. Sci. 74:2711–2718.

Mahan, D. C., and J. L. Vallet. 1997. Vitamin and mineral transfer during fetal development and the early postnatal period in pigs. J. Anim. Sci. 75:2731–2738.

Mahan, D. C., T. R. Cline, and B. Richert. 1999. Effects of dietary levels of Se-enriched yeast and sodium selenite as Se sources fed to growing-finishing pigs on performance, tissue Se, serum glutathione peroxidase activity, carcass characteristics, and loin quality. J. Anim. Sci. 77:2172–2179.

Mahan, D. C. 2000. Effect of organic and inorganic selenium sources and levels on sow colostrum and milk selenium content. J. Anim. Sci. 78:100–105.

Mahan, D. C., and J. C. Peters. 2004. Long-term effects of dietary organic and inorganic selenium sources and levels on reproducing sows and their progeny. J. Anim. Sci. 82:1343–1358.

Mateo, R. D., J. E. Spallholz, R. Elder, I. Yoon, and S. W. Kim. 2007. Efficacy of dietary selenium sources on growth and carcass characteristics of growing-finishing pigs fed diets containing high endogenous selenium. J. Anim. Sci. 85:1177–1183.

Meyer, W. R., D. C. Mahan and A. L. Moxon. 1981. Value of dietary selenium and vitamin e for weanling swine as measured by performance and tissue selenium and glutathione peroxidase activities. J. Anim. Sc. 52:302-311.

Miranda, S. G., Y. J. Wang, N. G. Purdie, V. Osborne, B. L. Coomber and J. P. Cant. 2007. Modulation of cellular activity of glutathione peroxidase by L-selenomethionine in primary cultures of bovine mammary gland epithelial cells. J. Anim. Sci. Suppl. 1 85:61-62.

Muth, O. H., J. E. Oldfield, R. F. Remmert, and J. R. Schuber. 1958. Effect of selenium and vitamin E on white muscle disease. Science (Wash., DC) 128:1090-1091.

National Research Council. 2007. Nutrient Requirements of Horses. 6th revised edition. Natl. Acad. Press, Washington, D. C.

O'Grady, M. N., F. J. Monahan, R. J. Fallon, and P. Allen. 2001. Effects of dietary supplementation with vitamin E and organic selenium on the oxidative stability of beef. J. Anim. Sci. 79:2827–2834.

Oh, Sang-Hwan, R. A. Sunde, A. L. Pope and W. G. Hoekstra. 1976. Glutathione peroxidase response to selenium intake in lambs fed a torula yeast-based, artificial milk. J. Anim. Sci. 42 (4):977-983.

Orr, J. P. and B. R. Blakley. 1997. Investigation of the selenium status of aborted calves with cardiac failure and myocardial necrosis. J. Vet. Daign. Invest. 9:172-179.

Ortman, K. and B. Pehrson. 1999. Effect of selenate as a feed supplement to dairy cows in comparison to selenite and selenium yeast. J. of Anim. Sci. 77:3365-3370.

Pagan, J. D., P. Karnezos, M.A.P. Kennedy, T. Currier, and K.E. Hoekstra. 1999. Effect of selenium source on selenium digestibility and retention in exercised thoroughbreds. Pages 135-140 in: Proc. 16th Equine Nutr. And Physiol. Soc. Symp. Raleigh, NC.

Parsons, M. J., P. K. Ku, D. E. Ullrey, H. D. Stowe, P. A. Whetter, and E. R. Miller.1985. Effects of riboflavin supplementation and selenium source on selenium metabolism in the young pig. J. Anim. Sci. 60 (2):451-461.

Pehrson, B., K. Ortman, N. Madjid and U. Trafikowska. 1999. The influence of dietary selenium as selenium yeast or sodium selenite on the concentration of selenium in the milk of suckler cows and on the selenium status of their calves. J. Anim. Sci. 77:3371-3376.

Qin, S., J. Gao, and K. Huang. 2007. Effects of different selenium sources on tissue selenium concentrations, blood gsh-px activities and plasma interleukin levels in finishing lambs. Biol. Trace Element Res. 116:91-102.

Reed, J. J., T. L. Neville, K. A. Vonnahme, P. P. Borowicz, J. B. Taylor, D. A. Redmer, J. S. Luther, C. J. Hammer, L. P. Reynolds and J. S. Caton. 2007. Effects of maternal nutrition and selenium supply on visceral organ mass of pregnant ewe lambs. J. Anim. Sci. Suppl. 1 85:361.

Reffett, J. K., J. W. Spears, and T. T. Brown Jr. 1988. Effect of dietary selenium and vitamin E on the primary and secondary immune response in lambs challenged with parainfluenza virus. J. Anim. Sci. 66:1520-1528.

Richardson, S. M., P. D. Siciliano, T. E. Engle, C. K. Larson, and T. L. Ward. 2006. Effect of selenium supplementation and source on the selenium status of horses. J. Anim. Sci. 84:1742-1748.

Rock, M.J., R.L. Kincaid, and G.E. Carstens. 2001. Effects of prenatal source and level of dietary selenium on passive immunity and thermometabolism of newborn lambs. Small Rum. Res. 40:129-138.

Rowntree, J. E., G. M. Hill, D. R. Hawkins, J. E. Link, M. J. Rincker, G. W. Bednar and R. A. Kreft, Jr. 2004. Effect of Se on selenoprotein activity and thyroid hormone metabolism in beef and dairy cows and calves. J. of Anim. Sci. 82:2995-3005.

Roquette, F. M., G. W. Webb, and G. D. Potter. 1987. Influence of pasture and feed on growth and development of yearling Quarter Horses. Pages 14-19 in: Proc. 9th Equine Nutr. And Physiol. Soc. Symp. Fort Collins, CO.

Rotruck, J. T., A. L. Pope, H. E. Ganther, A. B. Swanson, D. G. Hafeman, and W. G. Hoekstra. 1973. Selenium: Biochemical role as a component of glutathione peroxidase. Science. 179:588-590.

Shellow, J. S., S. G. Jackson, J. P. Baker, and A. H. Cantor. 1985. The influence of dietary Se levels on blood levels of Se and glutathione peroxidase activity in the horse. J. Anim. Sci. 61:590–594.

Skr˘ivanova´, E., M. Marounek, S. De Smet, and K. Raes. 2007. Influence of dietary selenium and vitamin E on quality of veal. Meat Science. 76:495–500.

Snow, D. H., and P. S. Guy. 1976. Percutaneous needle muscle biopsy in the horse. Equine Vet. J. 8:150–155.

Stowe, H. D., and T. H. Herdt. 1992. Clinical assessment of selenium status of livestock. J. Anim. Sci. 70:3928–3933.

Stabel, J. R., J. W. Spears, T. T. Brown Jr., and J. Brake. 1989. Selenium effects on glutathione peroxidase and the immune response of stressed calves challenged with *Pasteurella Hemolytica.* J. Anim. Sci. 67:557-564.

Sunde, R. A., J. K. Evenson, K. M. Thompson, and S. W. Sachdev. 2005. Dietary selenium requirements based on glutathione peroxidase-1 activity and mRNA levels and other Se-dependent parameters are not increased by pregnancy and lactation in rats. J. Nutr. 135:2144–2150.

Taylor, J. B. 2005. Time-dependent influence of supranutritional organically bound selenium on selenium accumulation in growing wether lambs. J. Anim. Sci. 83:1186–1193.

Tengelsen, L. A., B. Yamini, T. P. Mullaney, T. G. Bell, J. A. Render, J. S. Patterson, B. A. Steficek, S. D. Fitzgerald, F. A. Kennedy, M. R. Slanker, J. A. Ramos-Vara. 1997. A 12-year retrospective study of equine abortion in Michigan. J Vet. Diagn. Invest. 9:303-306.

Tiwary, A. K., B. L. Stegelmeier, K. E. Panter, L. F. James, J. O. Hall. 2006. Comparative toxicosis of sodium selenite and selenomethionine in lambs. J. Vet. Diagn. Invest. 18:61–70.

Ullrey, D. E., M. R. Light, P. S. Brady, P. A. Whetter, J. E. Tilton, H. A. Henneman, and W. T. Magee. 1978. Selenium supplements in salt for sheep. J. Anim. Sci. 46:1515-1521.

Ullrey, D. 1992**.** Basis for regulation of selenium supplements in animal diets. J. Anim. Sci. 70:3922-3927.

Vendeland, S. C., J. A. Butler, and P. D. Whanger. 1992. Intestinal absorption of selenite, selenate, and selenomethionine in the rat. J. Nutr. Biochem. 3:359-365.

van Ryssen, J. B. J., J. T. Deagen, M. A. Beilstein, and P. D. Whanger. 1989. Comparative metabolism of organic and inorganic Se by sheep. J. Agric. Food Chem. 37:1358–1363.

Wallace, J. M., D. A. Bourke, R. P. Aitken, and M. A. Cruickshank. 1999. Switching maternal dietary intake at the end of the first trimester has profound effects on placental development and fetal growth in adolescent ewes carrying singleton fetuses. Biol.of repro. 61**:**101–110.

Weiss, W. P., and J. S. Hogan. 2005. Effect of selenium source on selenium status, neutrophil function, and response to intramammary endotoxin challenge of dairy cows. J. Dairy Sci. 88:4366–4374.

Wichtel, J. J., N.D. Grace, and E.C. Firth. 1998. The effect of injectable barium selenate on the selenium status of horses on pasture. New Zealand Vet. J. 46: 186-190.

Westervelt, R. G., J. R. Stouffer, H. F. Hintz, and H. F. Schryver. 1976. Estimating fatness in horses and ponies. J. Anim. Sci. 43 (4):781-785.

Yoon, I. and E. McMillan. 2006. Comparative effects of organic and inorganic selenium on selenium transfer from sows to nursing pigs. J. Anim. Sci. 84:1729–1733.

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