AN ABSTRACT OF THE THESIS OF

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TITLE: SELENIUM DEFICIENCY IN THE EQUINE

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Whole blood selenium levels were measured in 207 horses, representing 41 ranches and 11 regions throughout Oregon. Age, sex, diet, and history of disease were recorded. Diet was divided into three categories; local feed only, commercial feed, and Se supplemented feed. Region ($p<.01$), diet type ($p<.01$), and a region x diet interaction ($p<.01$) affected blood Se levels. For horses fed only local diets, region and ranch were found to affect Se levels also ($p<.05$). Least squares means for selenium among regions ranged from .045 ppm to .461 ppm Se. Feeding local diets resulted in lowest blood Se levels (.058 ± .006); feeding commercial diets resulted in intermediate Se levels (.129 ± .012); and feeding Se supplemented diets resulted in highest Se levels (.206 ± .012). Age ($p<.07$) affected Se levels (positive relationship), whereas sex did not. Disease was recorded as presence or absence of disease of any sort and also categorized into three classifications; muscle-related diseases, reproductive-related diseases, and all others. Selenium was found to be negatively associated with overall incidence of disease ($p<.05$). Low Se level was associated with higher incidence

of reproductive-related disease, but there was no association of selenium level with muscle-related or other diseases.

In a study involving ten selenium supplemented (.15 ppm) pony mares and ten low selenium mares (.02 ppm) and their foals, it was shown that diet affected ($p<.01$) both mare Se and glutathione peroxidase but month of gestation affected only glutathione peroxidase. Week of lactation influenced ($p<.01$) mare blood Se, with the Se supplemented mares showing a decline in blood Se levels. Milk Se levels were low and not affected by either diet or week of lactation. Diet of the dam influenced the levels of foal blood Se, serum glutamic oxaloacetic transaminase (SGOT) and creatine phosphokinase (CPK). Foals from low Se mares had increased levels of the muscle-related enzymes, SGOT and CPK, until seven weeks of age, when the levels decreased to the levels of the foals from Se supplemented mares. Mare diet had no effect on foal weight or height.

SELENIUM DEFICIENCY IN THE EQUINE

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SELENIUM DEFICIENCY IN THE EQUINE

Introduction

In 1818, Berzelius discovered the element selenium. Little attention was paid to this trace mineral until the 1930's when scientists became aware of its toxic properties. High levels of selenium are associated with alkali disease and blind staggers in livestock.

In 1957, Schwarz discovered the nutritional importance of selenium. He found that Se prevented liver necrosis in vitamin E deficient rats. For several years it was not known how selenium exerted its beneficial influence. Then in 1973, Rotruck and associates discovered it to be an integral component of the enzyme glutathione peroxidase. Glutathione peroxidase acts to convert naturally occurring peroxides to less harmful alcohols or water. Vitamin E is closely interrelated with selenium. It decreases the formation of harmful peroxides (Scott, 1976).

Selenium deficiency in animals may depend on several factors (Underwood, 1962; Van Vleet, 1980):

- Se content of the soil which the feed is grown on
- Acidity of the soil
- Use of sulfate fertilizers
- Heavy irrigation or rainfall
- Exposure to high levels of some heavy metals
- Type of hay fed

- Stress to the animal
- Unsaturated fatty acids in the diet
- Vitamin E status of the animal

Selenium deficiency has been associated with a variety of health problems in livestock, listed below (NRC, 1976; Van Vleet, 1980). In cattle and sheep, these include skeletal and cardiac myopathy (White Muscle Disease), placental retention, unthriftiness, reduced fertility and abortion. In swine, hepatosis dietetica, mulberry heart disease and gastric ulcers are observed as well as skeletal myopathy. In poultry, Se deficiency has been associated with exudative diathesis, neuromuscular disease, pancreatic fibrosis and slowed growth.

The forages of the Pacific Northwest are known to be generally low in selenium (Carter et al., 1968). Little is known of the Se status of Oregon horses. We have conducted a survey to determine baseline blood Se levels of horses throughout Oregon and to investigate the association of blood Se concentrations with disease incidence.

For the equine, the case for selenium deficiency is not so clear cut as with other animals. A disease similar to White Muscle Disease has been observed in foals raised in low Se areas (Gabbedy and Richards, 1970; Wilson et al., 1976). Also, it has been reported that Se - vitamin E injections are beneficial for the prevention and treatment of a muscle stiffness problem known as tying-up (Hill, 1962). Blackmore et al. (1979) found that at one racetrack, horses which were not performing as well as expected had low levels of Se

in the blood. They also had increased levels of gamma glutamyl transferase which would indicate possible liver damage. So there are indications that Se does play a nutritionally important role in the equine. However, none of the disorders described above have been experimentally induced by feeding low Se diets.

While many horses appear to be healthy on low Se diets, it is possible that they suffer subclinical deficiency problems. For this reason, we have conducted an experiment with mares and their foals to examine these possible subclinical disorders. Other objectives of this experiment were to investigate mare Se levels during gestation and lactation and also milk and foal blood Se concentrations.

Selenium Status of Oregon Horses and Association of Blood Selenium Level With Disease Incidence

Summary

Whole blood selenium levels were measured in 207 horses, representing 41 ranches and 11 regions throughout Oregon. Age, sex, diet, and history of disease were recorded. Diet was divided into three categories; local feed only, commercial feed, and Se supplemented feed. Region ($p<.01$), diet type ($p<.01$), and a region x diet interaction ($p<.01$) affected blood Se levels. For horses fed only local diets, region and ranch were found to affect Se levels also ($p<.05$). Least squares means for selenium among regions ranged from .045 ppm to .461 ppm Se. Feeding local diets resulted in lowest blood Se levels (.058 ± .006); feeding commercial diets resulted in intermediate Se levels (.129 ± .012); and feeding Se supplemented diets resulted in highest Se levels (.206 ± .012). Age ($p<.07$) affected Se levels (positive relationship), whereas sex did not. Disease was recorded as presence or absence of disease of any sort and also categorized into three classifications; muscle-related diseases, reproductive-related diseases, and all others. Selenium was found to be negatively associated with overall incidence of disease ($p<.05$). Low Se level was associated with higher incidence of reproductive-related diseases, but there was no association of selenium level with muscle-related or other diseases.

Introduction

Many areas of the US, including the Pacific Northwest, have been shown to be deficient in the trace mineral selenium (Kubota et al., 1967). During the past several years much research has been done on the health problems associated with Se deficiencies in livestock (Van Vleet, 1980). Cases of selenium-related disorders have been reported in horses (Hill, 1962; Gabbedy and Richards, 1970; Blackmore et al., 1979), however it is possible that factors other than Se deficiency may play a role in these health problems.

Little is known of the selenium status of Oregon horses. Many horseowners feed only locally grown feeds, which would tend to be low in selenium. Others feed a mixture of local feed and commercial feeds from outside sources, while a few supplement their horses' diets with preparations containing sodium selenite.

The objectives of this survey were: 1) to establish baseline blood Se levels of horses throughout Oregon; 2) to investigate factors which might affect Se levels of horses and 3) to investigate the association of blood Se levels with incidence of disease.

Experimental Procedure

Whole blood selenium levels were recorded for 207 horses representing 41 ranches and 11 regions throughout Oregon. Blood samples were collected from horses into heparinized tubes by jugular venipuncture. Samples were collected July 1980 through January 1981. Maylin et al. (1980) reported no seasonal fluctuations of Se levels in horses, so time of year was not considered critical.

Age, sex, diet and history of disease were recorded. Diet was divided into three categories; local feed only, commercial feed and Se supplemented feed. Disease was recorded as presence or absence of health problems of any sort and also categorized into three classifications; muscle-related disorders, reproductive-related disorders and all others. Muscle-related disorders observed were muscle stiffness and tying-up. Reproductive-related disorders were failure to cycle, failure to conceive and abortion.

Whole blood was analyzed for selenium by the automated fluorometric procedure described by Brown and Watkinson (1977).

Data was analyzed by least squares analysis (Harvey, 1975) using three separate models. In the first model, region and diet were main effects, adjusting for age and sex. In the second analysis, source of variation of ranch was included in the first model using records obtained for horses on local diet only. The third model used selenium as an independent variable.

Results and Discussion

Factors Affecting Selenium Levels - For horses fed only locally grown feeds, the Se levels were low ($<.05$ ppm) except in the Harney Basin and Ontario areas (Figure 1). The "normal" blood Se for horses is considered to be .10 to .16 ppm (Stowe, 1967). Region of state influenced ($p<.01$) the level of Se in the blood (Table 1). The selenium level in animal tissue is known to be dependent on the content of Se in the animal's feed (Underwood, 1962). A survey of

forages in the Pacific Northwest (Carter et al., 1968) reveals that most areas of this region are low in Se (< .10 ppm). Oregon forages are generally considered to be inadequate in Se except for the Harney Basin in east central Oregon and an area along the eastern border in the vicinity of Ontario. Ancient lakes, which at one time covered the Harney Basin may have received some runoff waters from seleniferous areas near their eastern margin, accounting for the relatively high Se content of the soil in this area. In the Ontario area, sediments of fairly high Se content are exposed where recent volcanic deposits of low Se content are deeply truncated by the Snake river (Kubota et al., 1967).

For horses fed only local diets, ranch as well as region affected ($p<.05$) Se levels, possibly due to differences in feed type or agronomic practices such as irrigation or fertilization. Feed type, irrigation and fertilization have all been shown to affect Se levels in animals (Schwarz, 1964; Allaway et al., 1967; Lakin and Davidson, 1967).

Diet type ($p<.01$) and a ranch x diet interaction ($p<.01$) also influenced blood Se levels (Table 1). Feeding locally grown diets resulted in lowest blood Se levels; feeding commercial diets resulted in intermediate Se levels and feeding Se supplemented diets resulted in highest Se levels (Table 2). Even though the commercial diets were not supplemented with selenium as such, the blood Se levels of horses consuming these feeds were in the range considered normal, possibly due to high Se in feeds shipped in from other regions. These results are similar to findings in New York horses (Maylin et al., 1980).

Age influenced blood Se levels as older horses tended ($p<.07$) to have higher levels of Se. Other researchers have found this same trend in humans and rats (Pinto and Bartley, 1969; Zeinally et al., 1975).

Sex of the horse was found not to influence Se levels. It should be noted that all males in the survey were geldings. Studies with rats have shown no difference in glutathione peroxidase (a selenium containing enzyme) levels between castrated males and sham-operated males (Pinto and Bartley, 1969). The same studies also showed that glutathione peroxidase levels in females were about 60% higher than in similarly treated males. It is postulated that these sex related differences are due to the female sex hormones.

Association of Blood Selenium Level with Disease Incidence - Selenium deficiencies have been associated with a host of diseases in various species (Underwood, 1962). In the present survey, low blood Se levels were associated ($p<.05$) with a higher incidence of overall disease. Muscular degeneration and reproductive disorders seem to be of particular importance for selenium deficient livestock (Muth et al., 1958; Hartley and Grant, 1961). In this study, when overall incidence of disease was categorized into reproductive-related disorders, muscle-related disorders and all others, a negative association ($p<.06$) was found between Se levels and incidence of reproductive-related disorders. Low blood Se levels have been reported in a limited number of mares with reproductive disorders (Maylin et al., 1980). No association was found between Se levels

and incidence of muscle-related disorders, possibly due to the fact that only a few (4) cases of muscle problems were observed.

This study shows that the Se content of the horse's blood will vary from region to region. Feeding commercial feeds in low Se areas may result in adequate Se levels, but this will depend on where the commercial feed is grown. If horseowners are unsure of the Se status of their horses, it would probably be wise to have samples of blood analyzed for selenium content.

The survey also reveals the association of low Se levels with disease incidence. Although these health problems were not observed under controlled conditions, the study does indicate that selenium is probably a nutritionally important mineral for the equine and should be studied further.

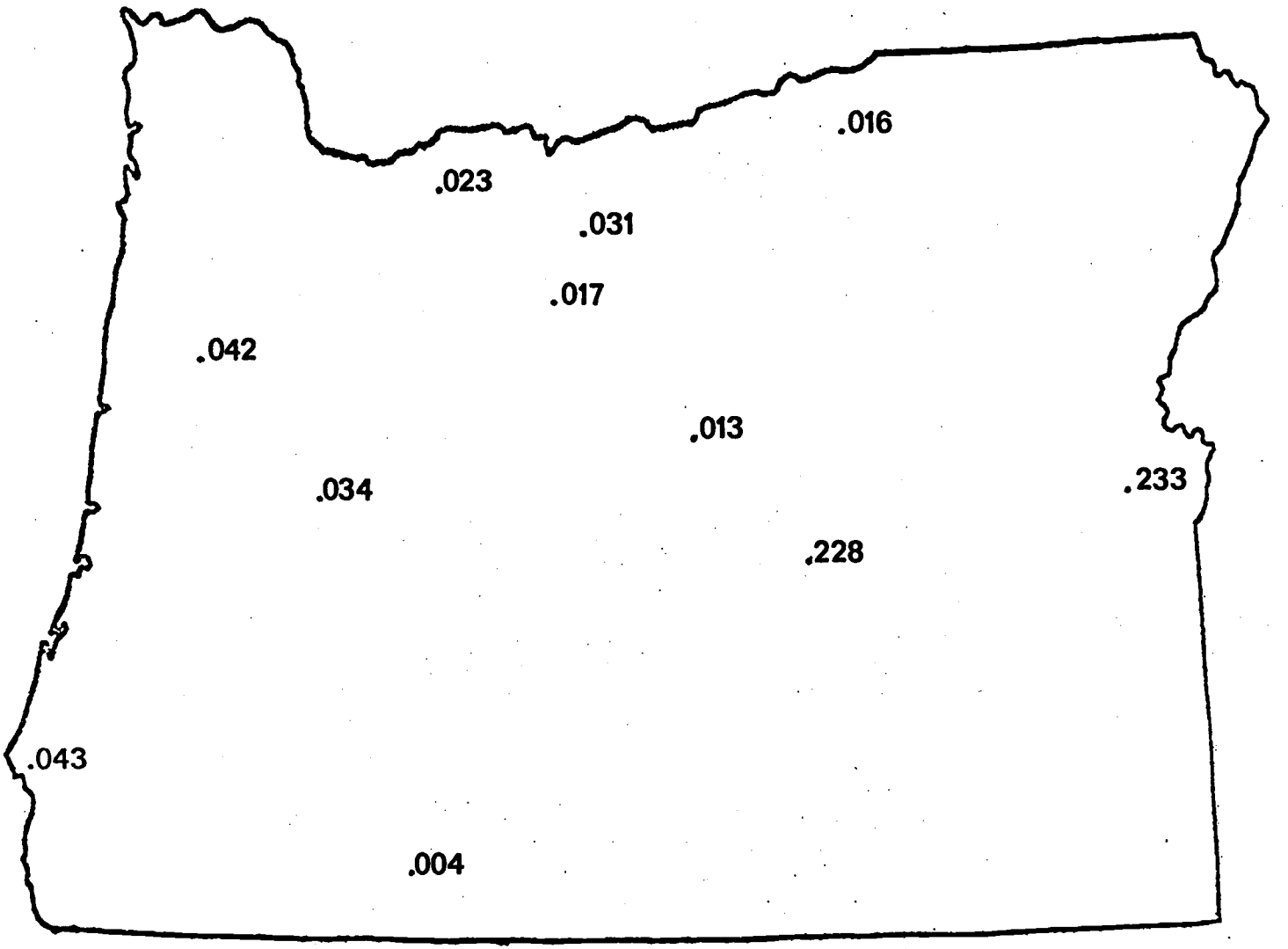

Figure 1. Least-squares means for blood Se values (ppm) of horses on local feeds in 11 regions of Oregon. The only groups with adequate Se levels are in the Harney Basin (.228 ppm Se) and Ontario (.233 ppm Se) regions. Overall SE = .005. (n ranges from 7-17 horses per region.

Table 1. Least-squares means and analysis of variance results for selenium and disease traits of horses throughout Oregon.

Independent Variable	Trait				
	Se(ppm)	Disease(%)	Muscle(%)	Repro.(%)	Others(%)
(Model 1) Least-squares means	.131	17.20	2.56	7.23	8.47
Region	**				
Diet	**				
Region x diet	**				
Age				**	
Sex					
(Model 2) Ranch					
(Model 3) Blood Se (ppm)		*			

P .10
* P .05
** P .01

Table 2. Least-squares means for whole blood selenium values of Oregon horses with three types of diet.

Diet	L.S.M. (ppm)	S.E.
Local	.058	.006
Commercial	.129	.012
Se supplemented	.206	.012

All L.S.M.'s significantly different (p .01)

Selenium Levels in Horses During Gestation and Lactation and the Effect of Selenium Deficiency on Foals

Summary

Ten pony mares were maintained on a low Se diet (.02 ppm Se) and ten were on a diet supplemented with sodium selenite (.15 ppm Se). Whole blood Se and glutathione peroxidase levels were monitored throughout gestation. Diet affected ($p<.01$) both Se and glutathione peroxidase but month of gestation did not. Monitoring mare blood and milk Se during the first 11 weeks of lactation indicated week of lactation influenced mare blood Se ($p<.01$), with the Se supplemented mares showing a decline in blood Se levels. Milk Se levels were low ($<.03$) and not affected by either diet or week of lactation. Diet of the dam influenced the levels of foal blood Se, serum glutamic oxaloacetic transaminase (SGOT) and creatine phosphokinase (CPK). Foals from low Se mares had increased levels of the muscle-related enzymes, SGOT and CPK, until seven weeks of age, when the levels decreased to the levels of the foals from Se supplemented mares. In this study, mare diet had no effect on foal weight or height.

Introduction

The nutritional importance of selenium has been well documented for many species (Van Vleet, 1980). However, in the equine, few controlled experiments have been done with this trace mineral. Selenium-vitamin E preparations are sold for the prevention and treatment of various muscle-related disease entities (nutritional muscular dys-

trophy and tying-up). But the research which would document that Se deficiencies lead to these musculoskeletal diseases consists mainly of case reports which are not under controlled experimental conditions.

Little is known of the mare's needs for Se during gestation and lactation. In this study we have followed the mare's blood Se level throughout gestation and lactation. Blood levels of the selenium dependent enzyme, glutathione peroxidase, were also recorded throughout gestation. Other objectives were to monitor milk Se and foal blood Se during the 11 weeks after foaling.

Selenium is known to be particularly important for the newborn animal (Schubert et al., 1961). In the lamb and calf, deficiencies often lead to severe muscular dystrophy problems (White Muscle Disease). For the most part, young foals in Se deficient areas do not appear to be affected with severe symptoms of muscular damage. However, it is possible they suffer from less acute symptoms of Se deficiency. For this reason, the levels of the enzymes, serum glutamic oxaloacetic transaminase (SGOT) and creatine phosphokinase (CPK) were investigated. Elevations in these enzymes could indicate subclinical damage.

Experimental Procedure

Twenty pony mares of unknown genetic origin were randomly assigned to two treatment groups; a low selenium group (Se(-)) and a selenium supplemented group (Se(+)). All mares were fed the same preconditioning diet for three months prior to being fed the experimental diet. The preconditioning diet consisted of mixed grass

hay (.02 ppm Se) and approximately 1 kg of barley, corn and mill run grain mixture (.035 ppm Se) daily. The ten mares assigned to the Se supplemented group were given sodium selenite injections im, 4 mg Se/200 kg BW, at two months and one month prior to being fed the experimental diet, to ensure rapid increase of Se levels.

The experimental diet for all mares consisted of free choice mixed grass hay (.02 ppm Se), approximately 1 kg whole oats (.002 ppm Se) and free choice trace mineralized salt blocks. Mares on the Se supplemented diet received .15 ppm Se in the diet as sodium selenite. This was administered by top dressing approximately 100 g barley pellets which had been premixed with sodium selenite. Ponies on the low Se diet received low selenite barley pellets. Feeding of the experimental diet began at the time of breeding. All mares were synchronized for heat using 50 mg progesterone im daily for ten days with a 2.5 mg injection of prostaglandin $F_{2\alpha}$ on day seven. Mares were artificially inseminated using one pony stud for all mares. Mares which didn't conceive on the first cycle were inseminated on the second and third cycle if necessary. During the last trimester of pregnancy and throughout lactation, alfalfa hay (.02 ppm Se) was substituted for half of the grass hay in order to increase protein levels.

Mare blood samples were collected by juglar venipuncture into heparinized tubes monthly throughout gestation and analyzed for Se and glutathione peroxidase.

Mare blood was sampled at parturition and one week after foaling, then bi-weekly for ten weeks. Mare milk, foal blood and foal height

and weight were also taken at these times. All blood and milk samples were analyzed for selenium. Foal blood was analyzed for SGOT and CPK in addition to selenium.

Whole blood and milk were analyzed for Se by an automated fluorometric procedure (Olson, 1969 with modifications by Brown and Watkinson, 1977).

For the glutathione peroxidase assay, the heparinized tubes of blood were placed on dry ice immediately upon collection and stored at -20°C until assay. The assay used was an enzyme coupled procedure described by Paglia and Valentine (1967), with modifications.

SGOT and CPK determinations were made according to routine methods.[1,2]

The data from the lactation period were analyzed by least-squares analysis of variance (Harvey, 1975) with the following mathematical model:

$$Yijkl = \quad + D_i + M_{ij} + W_k + DW_{ik} + e_{ijkl}$$

where

Y_{ijkl} = the observed value of a given dependent variable;

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[1]Sigma Technical Bulletin No. 505. The quantitative colorimetric determination of glutamic-oxaloacetic and glutamic-pyruvic transaminases at 490-520 nm in serum, plasma or cerebrospinal fluid. St. Louis, MO. 15 pp.

[2]CPK Stat-Pack kit from Calbiochem. LA, CA.

μ = the overall mean;

D_i = the fixed effect of the i^{th} diet;

M_{ij} = the random effect of the j^{th} mare within the i^{th} diet;

W_k = the fixed effect of the k^{th} week;

DW_{ik} = the interaction of the i^{th} diet with the k^{th} week and

e_{ijkl} = the random error.

Data from the gestation period were analyzed in a similar manner except the main effect of pregnancy class (pregnant vs non-pregnant) was included as well as resulting interactions.

Results and Discussion

Results show diet affected ($p<.01$) mare blood selenium levels during both gestation and lactation. Wide variation ($p<.01$) among mares receiving the same diet occurred only during lactation. The Se levels of Se(+) mares tended to increase during the first part of gestation and then level off (Figure 2). This increase probably occurred because mares were not yet at threshold Se levels at the time of conception. Stowe (1967) proposed a urinary selenium threshold mechanism in horses promoting Se excretion when serum Se levels approach .14 to .16 ppm. It was not until month two of gestation that the Se(+) mares reached this level. The non-pregnant mares tended to follow the same pattern of increase and leveling off as did the pregnant mares. In a study feeding horses 1 mg Se daily, Maylin and associates (1980) also found that it takes two to three months to reach these levels.

Neither Se(+) nor Se(-) mares exhibited a decline in Se level during the last part of gestation as has been reported for other species (Behne et al., 1978; Vrzgula et al., 1980). During gestation, perhaps the mare has some mechanism which would either enhance uptake of Se or inhibit Se excretion, thereby preventing the decrease in blood Se which is seen in other animals.

Mare blood glutathione peroxidase levels, monitored throughout pregnancy were affected by diet ($p<.01$), month ($p<.01$) and a diet x month interaction ($p<.01$). The overall least squares means for Se(-) mares was 14 $nm \cdot min^{-1} \cdot mg^{-1}$ and 81.4 $nm \cdot min^{-1} \cdot mg^{-1}$ for Se(+) mares. A correlation of .84 was found between blood glutathione peroxidase and selenium. The regression is $y = .0015 \chi + .0194$. Anderson (1978) and Caple et al. (1978) have shown correlations of .83 and .98, respectively.

There was a decline ($p<.01$) in blood Se levels in Se(+) mares during lactation (Figure 2). This is in contrast to findings for other animals which show an initial drop in Se and then a gradual increase to pre-gestation levels (Behne et al., 1978). Stress is known to increase the need for Se in animals (Allen et al., 1975) and it is possible that the stress of lactation is responsible for the decline seen in this study.

Diet had no significant effect on mare milk selenium. Overall least square means for Se(-) and Se(+) mares were .0125 ppm and .0165 ppm respectively. Week of lactation was not important for milk Se levels (Figure 2). Bergsten et al. (1970) and Maylin et al. (1980) also report low levels of Se in the milk of mares. They suggest that

Se is eliminated mainly via pathways other than through milk. The Se levels in cows milk also remain low until much higher levels of selenite are added to the diet (Conrad and Moxon, 1979). It should be noted that less Se is available to the newborn when selenium is administered as sodium selenite rather than organically incorporated selenium in the form of brewers grains (Conrad and Moxon, 1979; Allen and Miller, 1980).

Seven foals were born to Se(+) mares and four foals to the Se(-) mares, hereafter referred to as Se(+) foals and Se(-) foals. Early in gestation, one of the low Se mares aborted and died of a prolapsed uterus. Diet of the mare significantly affected foal blood selenium levels ($p<.01$). Group means for foals born to Se(-) and Se(+) mares were .012 ppm Se and .071 ppm Se, respectively. While the levels of the Se(-) foals were similar to those of their dams, those of the Se(+) foals were only about half of the values found in their dams (Figure 4). The Se(+) foals were born with much higher blood Se levels than the Se(-) foals, indicating there is placental transfer of Se to some extent in the equine. Even though the Se(+) foals were receiving only low amounts of Se from mare's milk and were given no supplemental Se, their Se levels decreased only slightly ($p<.10$) over the 11 weeks measured possibly indicating a low rate of Se turnover in the foal.

Diet of the mare did not affect foal height or weight. There was wide variation ($p<.01$) among foals within treatment group for these traits. Even when mare size was taken into account, there was no effect of diet on foal weight or height. Stowe (1967), however,

did show increased weight gains when foals were supplemented with 2 ppm Se.

Creatine phosphokinase and serum glutamic oxaloacetic transaminase are considered to be good indicators of muscle damage in selenium related myopathies. Elevations in SGOT can be seen with muscle or liver damage. Elevations in CPK are specific for muscle damage and the enzyme levels remain elevated for shorter periods after muscle damage than with SGOT. Analysis of variance results show diet of mare to be important for foal SGOT ($p<.05$) and CPK ($p<.06$) with the levels of both enzymes being generally higher in the Se(-) foals. Foal age did not appear to affect SGOT and only slightly affected CPK ($p<.10$). However, this may be misleading, for when age was plotted as a linear regression function, age was important for both SGOT ($p<.05$) and CPK ($p<.01$). As seen in Figure 5, both SGOT and CPK for Se(-) foals were elevated during the first five weeks of life and then decreased to the levels of the Se(+) foals around week seven. Least-squares differences reveal the levels of both enzymes were significantly different between treatment groups for weeks one through five but not different thereafter. Other animals have been shown to recover from mild cases of WMD and it has been postulated that the recovery may be associated with the increased function of gut microflora beginning at this time (Whanger et al., 1970).

The elevated levels of SGOT and CPK in the Se(-) foals were not nearly as great as those seen with White Muscle Disease in lambs and calves. The foals appeared "normal" outwardly and probably the increased enzyme levels indicate subclinical muscle damage.

Figure 2. Whole blood Se levels for pregnant and non-pregnant mares on either low Se diet (Se(-)) or Se supplemented diet (Se(+)). Values were recorded from two months prior to gestation through the eleventh week of lactation. Overall SE = .006. (n = 5 non-preg. Se(-), 4 preg. Se(+), 3 non-preg. Se(+) and 7 preg Se(+).

Figure 2.

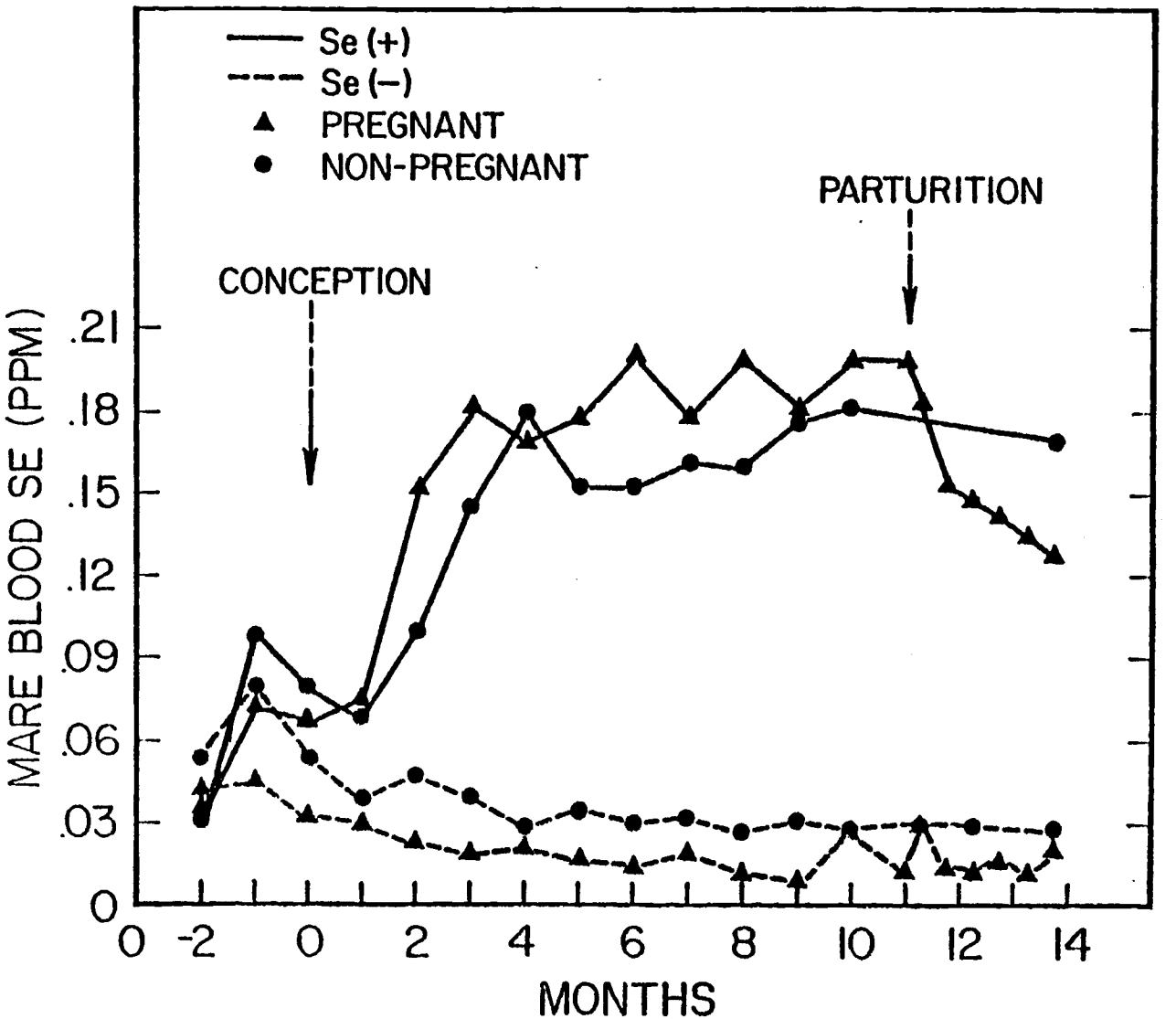

Se (+)
Se (–)
PREGNANT
NON-PREGNANT
PARTURITION
CONCEPTION
MARE BLOOD SE (PPM)
.21
.18
.15
.12
.09
.06
.03
0
0 -2 0 2 4 6 8 10 12 14
MONTHS

Figure 3. Milk Se concentrations of 4 Se deficient and 7 Se supplemented mares through 11 weeks of lactation. Overall SE = .002 Se(-) and .003 Se(+).

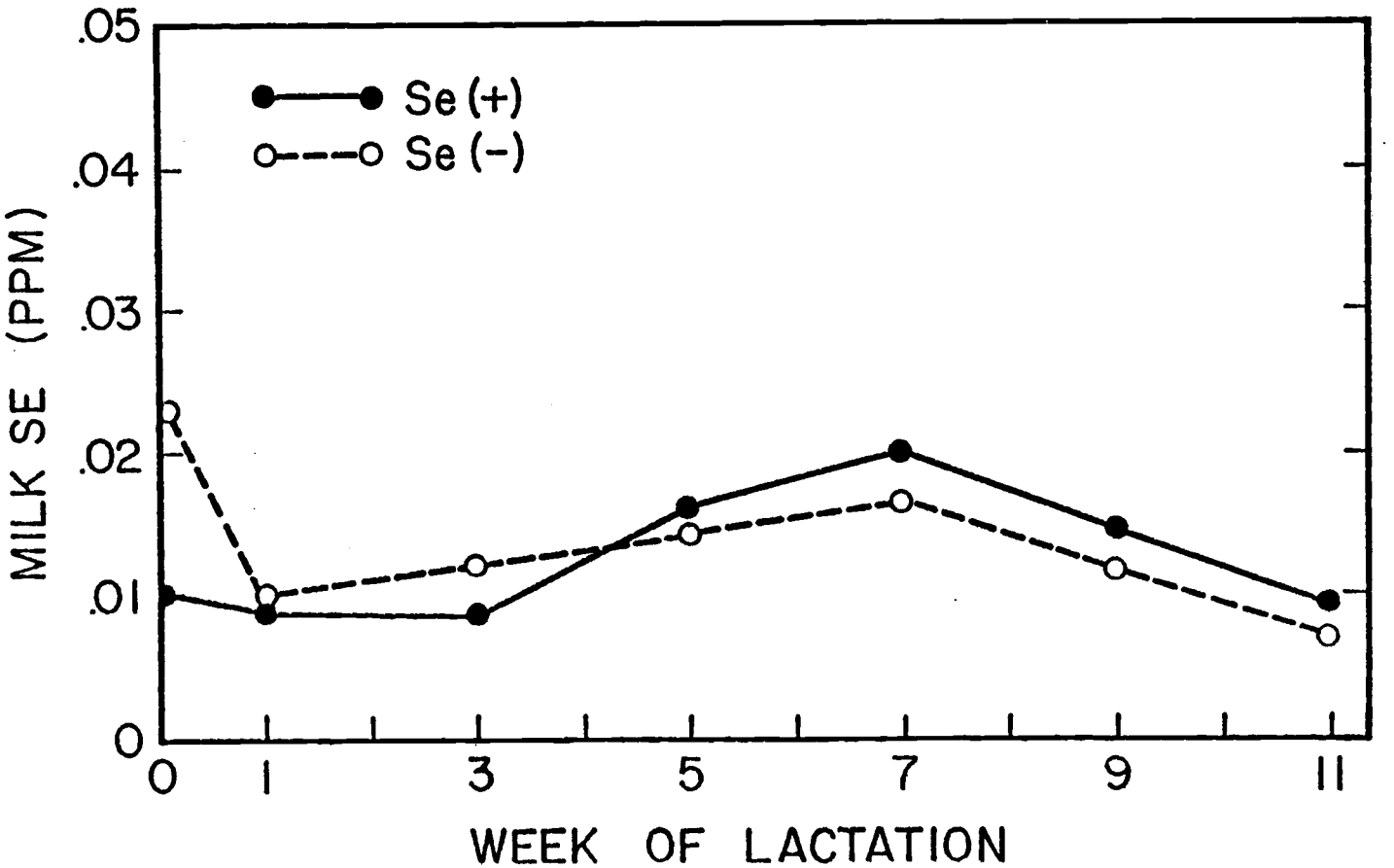


Figure 4. Whole blood selenium levels for 4 Se(-) foals and 7 Se(+) foals through 11 weeks of age. Overall SE= .003 Se(-) and .002 Se(+).

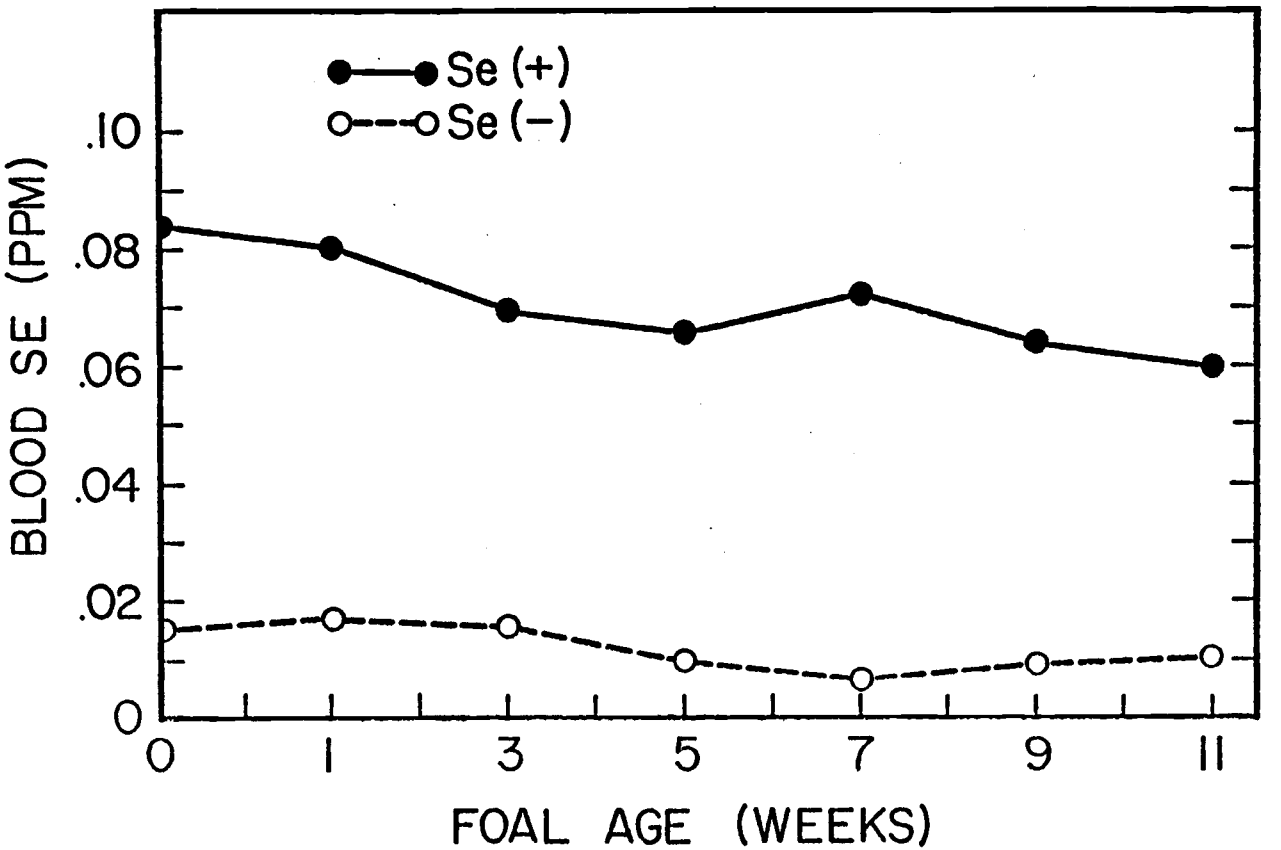
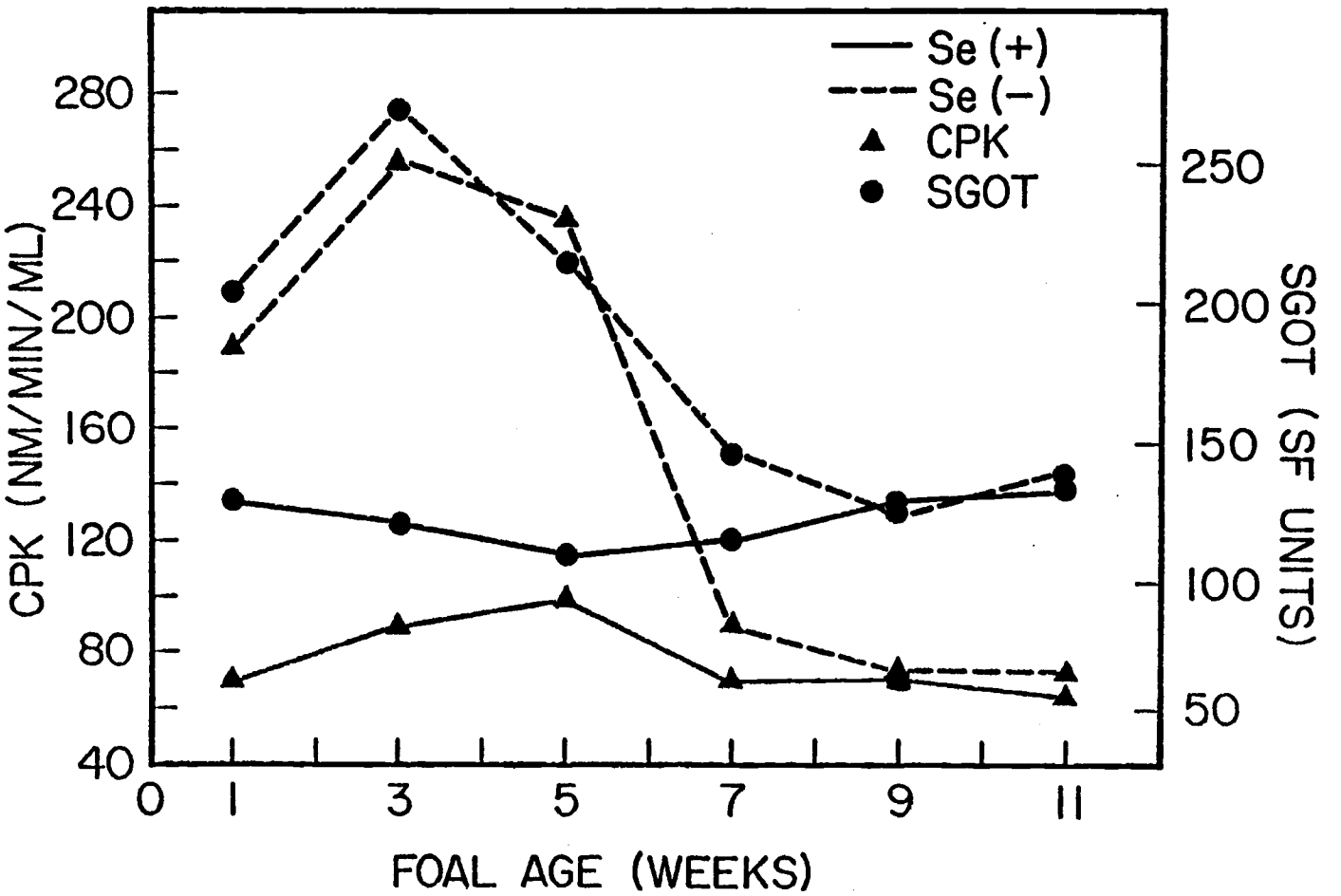


Figure 5. Creatine phosphokinase (CPK) and serum oxaloacetic transaminase (SGOT) values of 4 Se(-) and 7 Se(+) foals through 11 weeks of age. Overall SE = 17.9 for CPK and 11.3 for SGOT.



Summary and Conclusions

Although few controlled experiments have been done showing the nutritional importance of selenium in horses, it is felt that Se is indeed an important nutrient for the equine. Our survey data shows an association of low Se levels with disease incidence, particularly reproductive-related disorders and the experimental results from the mare and foal study indicate possible subclinical muscle damage in the foal.

The selenium status of the horse may depend on several factors. The area where the feed is grown probably plays the most significant role in determining blood Se levels. Outside sources of selenium such as in commercial feeds or Se supplements must be taken into consideration. We have found that age also has some influence on equine Se levels in that older horses tend to have higher Se concentrations. We have observed that the Se levels of the pregnant mare remain constant throughout gestation but tend to decrease during lactation. The young growing animal is particularly vulnerable to Se deficiency problems. Not only is this the time when the horse (as with other young animals) is most prone to Se related disease symptoms, but this is also the time when it is least apt to receive adequate amounts of Se, for the mare's milk is generally low in selenium. So just when the animal needs Se the most, it is least likely to get the element. For this reason, it is suggested that the newborn foal be given an injection of selenium - vitamin E.

While it is true that many horseowners in this area successfully raise horses without giving supplemental selenium, it is felt that the subtle subclinical problems associated with Se deficiency represent an economic loss. Horses may have individual needs for Se, with heredity and other factors possibly playing a role in this. One horse may perform satisfactorily with low levels of Se while another would show a greater need for this mineral. If the horseowners are unsure of the Se status of their horses, blood sampling for Se analysis is recommended.

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