TISSUE SELENIUM AND SERUM TOCOPHEROL CONCENTRATIONS IN SELENIUM-VITAMIN E DEFICIENT PIGS FED PEAS (PISUM SATIVUM)¹

L. R. McDowell², J. A. Froseth³, R. C. Piper⁴, I. A. Dyer³ and G. H. Kroening⁵

Washington State University, Pullman 99163

SUMMARY

An experiment was conducted to study the effects of feeding growing pigs a diet low in selenium (Se) and vitamin E (E) with and without supplementation of various levels of Se and/or E on tissue Se and serum tocopherol concentrations. Nine of 10 pigs fed the unsupplemented cull pea diet had lesions attributed to a Se-E deficiency and eight of these pigs died during the 135-day experiment. No deaths, clinical signs or lesions attributed to Se-E deficiency were observed among pigs fed any of the supplemented diets. In pigs fed the unsupplemented basal ration, hepatic Se concentrations on a dry matter basis decreased from .50 to .16, renal cortical from 3.27 to 1.86, and blood from .46 to .05 ppm during 135 days. Supplementation of the basal ration with .10 ppm Se as sodium selenite resulted in an eightfold increase in hepatic, nearly a fivefold increase in renal cortical and a 25-fold increase in blood Se concentrations compared to pigs fed the unsupplemented basal ration. Supplementation of the basal ration with 100 ppm E resulted in increased renal cortical (P<.01) and blood (P<.05) Se concentrations. Hepatic, renal cortical, and blood selenium concentrations of .25, 2.5 and .1 ppm, (dry basis) respectively, were determined to be the critical levels below which clinical illness, death or lesions of Se-E deficiency could be expected. Selenium concentration in liver, renal cortex and blood each adequately portrayed the Se body status of pigs, while testicular Se did not. Pigs fed the unsupplemented basal ration had the lowest serum tocopherol concentrations.

(Key Words: Selenium, Vitamin E, Tissue Minerals, Serum Tocopherol, Growing Swine, Peas.)

INTRODUCTION

Clinical illness, lesions and death in pigs attributed to Se and/or E deficiency have been reported in the East, Midwest and Pacific Northwest regions of the United States. Low Se levels in corn-soybean meal diets in the Midwest (Michel et al., 1969; Trapp et al., 1970; Ullrey et al., 1970) and cull pea (Pisum sativum) diets in the Pacific Northwest (Piper et al., 1975) have been linked to outbreaks of Se-E deficiencies in swine. Tissue Se levels have been shown to be related to dietary Se levels in rats (Burk et al., 1968; Hurt et al., 1971; Cary et al., 1973), swine (Sharp et al., 1970a,b; Ewan, 1971; Groce et al., 1971, 1973b; Ku et al., 1972; Ruth and Van Vleet, 1974; Mahn et al., 1975; Mahn and Moxon, 1976; Wilkinson et al., 1976), chicks (Pond et al., 1971), sheep (Ewan et al., 1968; Lunde and Odegaard, 1972; Oh et al., 1976) and cattle Moxon et al., 1976; Perry et al., 1976a,b).

Tissue concentrations of tocopherols and Se have been used as indicators of the adequacy of these nutrients in the diet. Pigs with hepatitis dietetica and experimental E deficiencies had significantly lower tocopherol levels in heart, liver and spleen than control pigs (Wanntorp and Obel, 1957). Lindberg and Siren (1963, 1965) reported kidney and liver Se concentrations for “normal” pigs. Hepatic SE levels of .25 to .50 ppm and renal Se levels of 3.33 to 3.40 ppm, on a dry matter basis, were associated with nutritional muscular and hepatic dystrophy.

Selenium functions as a component of the...
enrichment of glutathione peroxidase (Rotruck et al., 1973). In swine, Ewan (1976) reported high correlations between tissue selenium content and glutathione peroxidase activity in liver (r=.86), kidney (r=.90) and spleen (r=.78). There is conflicting evidence on the influence of supplemental E on Se body stores. Yang et al. (1976) reports that both an excess and a deficiency of vitamin E can significantly depress the activity of glutathione peroxidase in liver and plasma of rats. Ewan (1971) noted that dietary E had no influence on tissue Se concentrations even though its addition to the ration completely prevented clinical signs and lesions of Se-E deficiency. Ku et al. (1972) also reported no apparent relationship between dietary α-tocopherol level and Se concentration of pork chops from pigs produced in 13 different locations in the United States. Sharp et al. (1970b) reported dietary E supplementation increased renal Se, but hepatic and myocardial Se were decreased.

This research was designed to investigate the influence of supplemental Se and/or E on tissue Se and serum tocopherol in growing pigs fed a cull pea diet low in Se and E.

**EXPERIMENTAL PROCEDURE**

Dietary regimes, blood and tissue collection procedures and housing and management of the pigs have been described (Piper et al., 1975). Performance data, clinical signs, pathology and selected blood parameters for this experiment have been previously reported (Piper et al., 1975). Ninety 3- to 4-week-old Yorkshire and Hampshire pigs of both sexes averaging 6.2 kg were fed an E depleting diet containing 8% rancid cottonseed oil and 50.3% cull peas (Ref. No. 5-08-480) for a 25-day depletion period. At the end of this period, 10 ml of blood were drawn from the anterior vena cava of 20 randomly selected pigs for blood Se analysis. Blood Se was determined on 10 randomly composited samples that represented two animals per determination. At this time the majority of the pigs had reached slaughter weight.

Blood samples were drawn from the anterior vena cava on the 123rd and 153rd days of the experiment. Some pigs fed the basal diet were bled at randomly selected times during the trial and at the time of death if it occurred before day 153. Total serum tocopherol was determined on serum collected on day 123 (Bieri et al., 1964). Tissue samples of liver and renal cortex were collected at the time of death or experimental termination. Both testicles were surgically removed from all boars on the 109th day of the experiment. Blood samples were collected on day 153 and at the time of death. Selenium concentration of feed, blood, liver, renal cortex and testicle was determined by neutron activation using 75Se as previously described (McDowell et al., 1974).

Three to 4 weeks prior to termination of the experiment, one female Yorkshire pig fed the basal ration (group 1) and three of four male Yorkshire pigs fed the basal ration supplemented with E alone (group 2) developed a mild dermatitis, characterized by erythema with a thickened, rough, scaly, occasionally fissured epidermis, over the dorsal cervical region with small patches extending over the back. The lesions, which were somewhat similar to those of a fatty acid deficiency, persisted
RESULTS AND DISCUSSION

Nine of the 10 pigs fed the unsupplemented basal diet had lesions attributed to a Se-E deficiency and eight of these pigs died between days 56 and 157 of the 160-day experimental period. Among the characteristics of the deficiency were sudden death with no prior signs of illness, massive hepatic necrosis, hemorrhagic nephrosis and to a lesser extent cholemic nephrosis, degenerative myopathy of cardiac and skeletal muscles, edema, icterus and acute terminal congestion and hemorrhage. Clinical signs, deaths or lesions attributed to Se-E deficiency were not observed in any of the pigs fed the basal diet supplemented with Se and/or E.

Selenium body stores of pigs fed the basal diet declined as the experiment progressed. Selenium concentrations in liver, renal cortex and blood in ppm (dry basis) of random pre-experimental animals were .50 ± .02, 3.27 ± .24 and .46 ± .02, respectively, whereas they were .16 ± .01, 1.86 ± .02 and .088 ± .01 ppm for pigs fed the basal diet at the time of death or slaughter. Blood Se concentration in pigs fed the basal diet declined as the experiment progressed, and in feed conversion the basal diet supplemented in any of the pigs fed the basal diet not supplemented in any of the pigs fed the basal diet had less efficient feed conversion than pigs fed the basal diet supplemented with Se and/or E.

Selenium concentrations in liver, renal cortex and blood in ppm (dry basis) of random pre-experimental animals were .50 ± .02, 3.27 ± .24 and .46 ± .02, respectively, whereas they were .16, 1.86 and .088 ppm for pigs fed the basal diet at the time of death or slaughter. Blood Se concentration in pigs fed the basal diet declined as the experiment progressed, and in feed conversion the basal diet supplemented in any of the pigs fed the basal diet had less efficient feed conversion than pigs fed the basal diet supplemented with Se and/or E.

Blood Se concentration of three male pigs fed the basal pea diet which were bled either 4, 35 or 54 days prior to death due to a Se-E deficiency was .145, .040 and .088, respectively, and averaged .091 ± .02 ppm (dry basis). At the time of death blood Se concentration was .058 ± .01, 0.012 ± .01 and .023 ± .01 for pigs fed the basal diet at the time of death or slaughter. Blood Se concentration of three male pigs fed the basal pea diet which were bled either 4, 35 or 54 days prior to death due to a Se-E deficiency was .145, .040 and .088, respectively, and averaged .091 ± .02 ppm (dry basis). At the time of death blood Se concentration was .058 ± .01, 0.012 ± .01 and .023 ± .01 for pigs fed the basal diet at the time of death or slaughter.

### TABLE 1. EFFECTS OF DIETARY TREATMENT ON TERMINAL TISSUE SELENIUM CONCENTRATIONS

<table>
<thead>
<tr>
<th>Diet</th>
<th>Liver (ppm, dry matter)</th>
<th>Renal cortex (ppm, dry matter)</th>
<th>Testicle (ppm)</th>
<th>Blood (ppm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Basal (96.8% cull peas)</td>
<td>.16 ± .020c (10)</td>
<td>1.86 ± .020d (10)</td>
<td>.67 ± .098 (3)</td>
<td>.05 ± .008c (8)</td>
</tr>
<tr>
<td>Basal + 100 ppm Se</td>
<td>.22 ± .017cd (6)</td>
<td>3.91 ± .316e (6)</td>
<td>.54 ± .109 (3)</td>
<td>.18 ± .039d (6)</td>
</tr>
<tr>
<td>Basal + 10 ppm E + .01 ppm Se</td>
<td>.29 ± .026f (6)</td>
<td>4.98 ± .303g (6)</td>
<td>.89 ± .188 (3)</td>
<td>.19 ± .032d (6)</td>
</tr>
<tr>
<td>Basal + .01 ppm Se</td>
<td>.37 ± .004s (6)</td>
<td>5.04 ± .408h (6)</td>
<td>.36 ± .046 (3)</td>
<td>.26 ± .004d (6)</td>
</tr>
<tr>
<td>Basal + .05 ppm Se</td>
<td>.68 ± .004c (6)</td>
<td>7.91 ± .616i (6)</td>
<td>1.57 ± .412 (3)</td>
<td>.45 ± .004e (6)</td>
</tr>
<tr>
<td>Basal + .10 ppm Se</td>
<td>1.26 ± .115j (6)</td>
<td>8.57 ± .533k (6)</td>
<td>1.40 ± .508 (3)</td>
<td>1.14 ± .128l (6)</td>
</tr>
</tbody>
</table>

aHepatic and renal cortical samples were collected at termination of the experimental period (day 160) or at time of death (eight pigs fed the basal diet from experimental days 56 to 157); blood samples were collected on experimental day 153 or at the time of death. Testicle samples were collected on experimental day 109.

bValues are means ± SE. Values in parentheses refer to number of observations.

c-d, Values within a column with different superscripts are significantly different (P<.05).

g-h, Values within a column with different superscripts are significantly different (P<.01).
decline in tissue Se levels of rats fed low Se
3torula yeast diets. Serum Se of cattle consum-
4ing a low Se diet for 90 days decreased from
5.022 to .013 ppm (Perry et al., 1976b).

Mean hepatic, renal cortical, testicular and
6blood Se concentrations of samples taken at
7death or at the end of the experiment are
8summarized in table 1. Individual pig values and
time of death of animals fed the unsupplemen-
tal basal diet are presented in table 2. Pigs fed
9the unsupplemental basal ration had lower
10mean Se concentration in renal cortex (P<.01)
11and blood (P<.05) than pigs fed the basal
12ration supplemented with various levels of Se
13and/or E. All three levels of Se supplementation
14without E also increased (P<.05) hepatic Se
15concentration as compared to pigs fed the basal
16diet. Higher tissue Se concentrations in pigs fed
17the basal ration supplemented with Se, E or
18both and the absence of clinical signs, death
19and lesions attributed to Se-E deficiency in
20these pigs, suggests that elevated tissue Se
21concentrations may have protected these pigs
22from the deficiency disease.

Within the basal ration group, the two
23Hampshire pigs slaughtered at termination of
24the experimental period tended to have higher
25mean Se concentrations in liver, renal cortex
26and blood than did the eight Yorkshire pigs
27that died during the experimental period (table
282). Mean hepatic, renal cortical, and blood Se
29concentrations (ppm, dry matter) for the two
30Hampshire pigs were .23, 3.23 and .06, respec-
tively, whereas terminal mean concentrations
for Yorkshire pigs which died were .14, 1.52
31and .04, respectively. The higher mean tissue Se
32concentrations in Hampshire pigs and the death
33of all Yorkshire pigs from Se-E deficiency
disease during the 160 days and the survival of
34both Hampshire pigs for the entire experi-
35mental period suggests a possible difference in
36breed susceptibility to Se-E deficiency disease.
Possible breed differences could be based, at
37least in part, upon differences in the ability of
38individual pigs or breeds to concentrate or to
39conserve body Se. It is also possible that the
40two Hampshire pigs had higher storage levels of
41Se prior to initiation of the experiment. The
42two Hampshire pigs sacrificed at the end of the
43depletion period (day 25) had mean Se levels of
44.49 and 3.56 ppm (dry basis) in liver and
45kidney, respectively, while the two Yorkshires
46sacrificed had similar levels of .51 and 2.97. In
47agreement, Obel (1953) previously had report-
ed a difference in breed susceptibility to hepa-
tosis dietetica.

Hepatic and blood Se concentrations in-
48creased (P<.05) with each increase in dietary Se
49supplementation level. These results are in
50agreement with those of Whanger and Weswig
51(1970), who reported hepatic Se concentrations
52increased linearly in rats when four dietary
53levels of Se ranging from .01 to .08 ppm were
54fed. Ku et al. (1972) in a survey of 13 states in
55the U.S.A., and a total of 40 pigs, found a
56significant linear correlation (r=.95) between
57dietary Se content and Se concentrations in
58tissue. The mean hepatic Se concentration of

<table>
<thead>
<tr>
<th>Identification of pigs</th>
<th>Disposition (day of experiment)</th>
<th>Selenium concentration (ppm, dry matter)</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. Breed Sex</td>
<td>Liver</td>
<td>Kidney</td>
</tr>
<tr>
<td>1 Yorkshire F</td>
<td>D (56)</td>
<td>.118</td>
</tr>
<tr>
<td>2 Yorkshire F</td>
<td>D (64)</td>
<td>.097</td>
</tr>
<tr>
<td>3 Yorkshire F</td>
<td>D (92)</td>
<td>.063</td>
</tr>
<tr>
<td>4 Yorkshire M</td>
<td>D (110)</td>
<td>.129</td>
</tr>
<tr>
<td>5 Yorkshire M</td>
<td>D (116)</td>
<td>.235</td>
</tr>
<tr>
<td>6 Yorkshire M</td>
<td>D (119)</td>
<td>.185</td>
</tr>
<tr>
<td>7 Yorkshire M</td>
<td>D (128)</td>
<td>.198</td>
</tr>
<tr>
<td>8 Yorkshire F</td>
<td>D (157)</td>
<td>.138</td>
</tr>
<tr>
<td>9 Hampshire M</td>
<td>E (160)</td>
<td>.256</td>
</tr>
<tr>
<td>10 Hampshire F</td>
<td>E (160)</td>
<td>.197</td>
</tr>
</tbody>
</table>

aF is for female and M is for male.
bD is for death and E is for euthanasia.
.16 ppm for pigs in the basal dietary group was similar to that reported by Ewan (1971) in pigs fed a predominantly torula yeast and glucose monohydrate diet. They reported mean hepatic Se concentrations of .140 ppm for two pigs which died and .084 ppm for six pigs which were killed after a 56-day feeding period.

Renal cortical Se concentrations increased (P<.01) when the basal diet was supplemented with .01 or .05 ppm Se. However, supplementation of the basal diet with .1 ppm Se did not result in significantly (P<.05) higher renal cortical Se concentrations than the basal + .05 ppm Se treatment. Renal Se concentrations in both these dietary treatments approached concentrations for "normal" pigs of 10.98 ppm as reported by Lindberg and Siren (1963, 1965). Lindberg and Lannek (1965) also noted no increase in renal Se concentrations with dietary Se supplementation of 1.2 ppm; however, both hepatic and muscular Se concentrations were significantly elevated. Groce et al. (1973b) postulated the existence of tissue and serum thresholds for Se from sodium selenite in pigs and that once physiological stores of Se are filled, the body tends to excrete any excesses and tissue and serum Se levels plateau. Their data indicated that myocardium Se was highest at .2 ppm supplemental Se, hepatic and longissimus muscle concentrations plateau at .1 ppm and renal Se plateaus at .05 ppm of supplemental Se. Jenkins and Winter (1973), likewise indicated that Se content of tissues tends to plateau. Further study is needed to determine if tissue levels continue to plateau with toxic levels of selenium as sodium selenite. Diehl et al. (1975) have suggested that the kidney and longissimus muscle become saturated, but that hepatic tissue was more flexible in its storage of Se and its Se concentration increases as the quantity injected increases. Our data support this view with respect to renal cortical Se levels and further suggests that renal Se concentration plateaus at lower supplemental Se levels than do blood and liver Se concentrations. Higher supplemental Se levels may have resulted in a plateauing of blood and liver Se concentrations in the present study.

In the eight pigs that died from Se-E deficiency, hepatic Se concentrations ranged from .06 to .24 ppm, renal cortical Se from 1.06 to 2.10 ppm and blood Se from .01 to .07 ppm (dry basis). Based upon these values, Se concentrations less than .25 ppm in the liver, 2.5 ppm in the kidney and .1 ppm in the blood would be considered "critical" for a Se deficiency status. When hepatic Se decreased to .04 ppm (wet weight) Ruth and Van Vleet (1974) correlated onset and progression of Se-E deficiency lesions in swine by sequential measurement of activity of several plasma enzymes: SGOT, creatine phosphokinase, alpha-hydroxy-butyric acid dehydrogenase, isocitric dehydrogenase and lactic dehydrogenase. Lindberg and Siren (1965) reported the "critical borderline" of porcine hepatic Se was between .25 and .5 ppm. If their "critical borderline" range was applied to our experimental pigs, hepatic Se concentrations of pigs fed either the basal ration supplemented with E, the basal ration supplemented with E and .01 ppm Se or the basal ration supplemented with .01 ppm Se would fall within or below their range. However, neither clinical signs nor lesions of Se-E deficiency were observed in any of the 30 pigs fed these three rations. Therefore, their "critical borderline" range appears to be slightly high. Pigs with hepatic Se concentrations of .25 ppm or more did not reach a critical level associated with clinical signs, death or lesions of Se-E deficiency. However, interpretation of tissue Se concentrations must take into consideration the age of the pigs, length of the feeding period and possible managerial and environmental stresses as well as concurrent diseases and biochemical interactions which could cause otherwise "normal" pigs with subclinical Se deficiency to suddenly develop a Se-E deficiency disease.

The renal cortex had much higher Se concentrations than liver, blood and testicles in all dietary groups. This is in agreement with Cousins and Cairney (1961), who concluded ovine kidneys were the organs most influenced by Se absorption and renal Se concentrations provided an adequate index of an animal's Se status. The mean renal cortical Se concentration of pigs fed the basal ration (1.86 ppm) was lower than renal concentrations reported by Lindberg and Siren (1963) for pigs with muscular (3.397 ppm) or hepatic (3.325 ppm) dystrophy.

Selenium and/or E supplementation had no effect (P>.05) on testicular Se concentration. However, Se supplementation levels of .05 and .10 ppm tended to increase testicular Se concentration. The observation that a Se deficiency does not result in a significant decrease in testicular Se when dietary levels are inadequate is consistent with work by Gunn et al. (1967).
who have demonstrated that the testes of rats and mice accumulated and retained a subcutaneous injection of $^{75}$Se while other organs lost 50 to 90% of the administered level by 7 days.

From the data of this experiment, the Se status of swine can be easily evaluated by determining Se concentrations in liver, renal cortex or blood. Each of these tissues accurately portrayed the Se status of the animal and the probability of its developing the deficiency disease. Mahan et al. (1975) presented data suggesting that hepatic and kidney tissue are perhaps the best tissues for evaluating the Se status in adult female swine and that the hepatic tissue is the more labile Se reservoir. From the present study, testicular Se concentration appears to be a poor indicator of the Se status of swine. Due to ease of sampling, blood would be the preferred indicator of body Se status in the live animal.

Supplementation of the basal ration with 100 ppm E increased renal cortical (P<.01) and blood (P<.05) Se concentrations and also tended to increase liver Se (table 1). These results are similar to those of Sharp et al. (1970b) and Groce et al. (1973b) for the kidney. However, others have shown no effect of supplemental E on porcine kidney Se (Ewan, 1971), blood Se (Groce et al., 1973b) or liver Se (Sharp et al., 1970b). Sharp et al. (1970b) reported slight decreases in porcine liver, heart and muscle Se concentrations resulting from dietary E supplementation. Whanger and Weswig (1970) and Burk et al. (1968) reported E had no influence on hepatic Se concentrations in rats. Supplementation of the basal ration with 10 ppm E and .01 ppm Se did not significantly affect tissue Se concentrations when compared to pigs fed the basal ration supplemented with .01 ppm Se alone. However, there was a tendency for Se concentration in blood, liver and renal cortex to decrease when 10 ppm E was added to the diet containing .01 ppm supplemental Se. These results partially support the observations of Wastell et al. (1967) who reported that E in the absence of supplemental Se resulted in a slight increase in renal Se concentrations; however, in the presence of supplemental Se a decrease (P<.01) was noted. Contrary to some reports our data supports the view that at least part of the function of E is to maintain concentrations of tissue Se above critical levels when no supplemental Se is included in the diet. How much longer than 135 days this protection would have lasted is an important question requiring further research.

Selenium, as sodium selenite, appeared to be well utilized by pigs in this study. Doubling the amount of supplemental Se from .05 to .1 ppm nearly doubled hepatic Se concentration and blood Se concentration increased two and one-half times. Addition of .1 ppm Se as sodium selenite to the basal ration resulted in approximately five- and eightfold increases in renal cortical and hepatic Se, respectively, and a 25-fold increase in blood Se concentrations over pigs fed the unsupplemented basal diet. Although Se as selenite was well utilized by pigs in this experiment, Lindberg and Lannek (1965) reported only a small increase in tissue Se concentrations when 1.2 ppm Se, as sodium selenite, was supplemented to a basal diet containing .126 ppm Se. Lopez et al. (1969) fed lambs supplemental selenium as selenite ranging from .5 to 5 ppm and found tissue retention of $^{75}$Se decreased with increasing dietary selenium levels. Investigations with pigs by Groce and co-workers (1971, 1973a) and Ku et al. (1973) have also suggested that supplemental Se (.1 ppm Se and above) in the form of selenite is less effective in elevating tissue Se levels and increasing Se retention than organic forms of Se. However, Cary et al. (1973) in studies with rats reported no significant differences in Se retention associated with different sources of Se when these were fed at levels less than .1 ppm Se. It appears that both organic Se compounds and Se as selenite are highly effective in elevating tissue Se concentrations when swine have low body Se stores. However, when tissue Se concentrations approach normal physiological levels, organic Se compounds may be biologically more available than selenite Se in increasing tissue Se concentration.

Pigs fed the unsupplemented basal ration had the lowest mean serum tocopherol concentration (table 2). Mean serum tocopherol was significantly higher (P<.05) in pigs fed the basal ration supplemented with 100 ppm E than in pigs fed the unsupplemented basal ration. However, serum tocopherol concentration of pigs fed the basal ration was not significantly (P>.05) different from that of those fed any of the Se supplemented diets. Wanntorp and Obel (1957) previously established that tissues of pigs with degenerative myopathy and hepatitis dietetica had lower serum tocopherol concentrations than did "healthy" pigs.

Analyses of the ration suggested that the dermatitis observed 3 to 4 weeks prior to
TABLE 3. EFFECT OF DIETARY TREATMENT ON SERUM TOTAL TOCOPHEROL AND LIVER AND BLOOD ZINC CONCENTRATIONS

<table>
<thead>
<tr>
<th>Diet</th>
<th>Serum tocopherol (µg/ml)</th>
<th>Zinc concentration (ppm, dry matter)</th>
<th>Liver</th>
<th>Blood</th>
</tr>
</thead>
<tbody>
<tr>
<td>Basal (96.8% cull peas)</td>
<td>1.8 ± .32c (4)</td>
<td>187.3 ± 6.34 (4)</td>
<td>14.2 ± .30 (4)</td>
<td></td>
</tr>
<tr>
<td>Basal + 100 ppm E/kg</td>
<td>2.9 ± .47d (8)</td>
<td>143.2 ± 11.85 (6)</td>
<td>17.8 ± .79 (6)</td>
<td></td>
</tr>
<tr>
<td>Basal + 10 ppm E/kg + .01 ppm Se</td>
<td>2.6 ± .25c (7)</td>
<td>181.5 ± 24.06 (6)</td>
<td>15.3 ± .71 (4)</td>
<td></td>
</tr>
<tr>
<td>Basal + .01 ppm Se</td>
<td>2.6 ± .35cd (7)</td>
<td>170.5 ± 22.37 (6)</td>
<td>17.2 ± 1.00 (6)</td>
<td></td>
</tr>
<tr>
<td>Basal + .05 ppm Se</td>
<td>2.7 ± .55cd (7)</td>
<td>154.2 ± 9.71 (6)</td>
<td>16.1 ± .73 (4)</td>
<td></td>
</tr>
<tr>
<td>Basal + .1 ppm Se</td>
<td>2.2 ± .31cd (7)</td>
<td>141.5 ± 10.23 (6)</td>
<td>16.2 ± .70 (6)</td>
<td></td>
</tr>
</tbody>
</table>

aSerum was collected on experimental day 123. Hepatic samples were collected at termination of the experimental period (day 160) or at the time of death (two pigs fed the basal diet). Blood samples were collected on experimental day 153 or at the time of death (two pigs fed the basal diet).

bValues are means ± SE. Values in parentheses refer to number of observations.

c,dValues within a column with different superscripts are significantly different (P<.05).

termination of the experiment was not caused by a Zn deficiency since the rations were relatively low in calcium (.61%) and high in Zn (90.4 ppm). Zinc analyses of liver and blood were not significantly different (P>.05) and tended to confirm that the dermatitis was not due to a Zn deficiency (table 3). Neither pigs in various dietary treatment groups nor pigs with or without dermal lesions had different (P>.05) hepatic or blood Zn concentrations. Lewis et al. (1957) reported severe Zn parakeratosis in pigs associated with hepatic Zn concentrations of 89 ppm while Morgan et al. (1969) reported no parakeratosis in pigs with hepatic Zn concentrations of 147 ppm. In our study, none of the pigs which developed dermatitis had a hepatic Zn level below 120 ppm. Welch et al. (1974) have shown that pea seeds are a good source of dietary Zn for rats. The cause of the dermatitis is therefore unknown although it does not appear to have been due to a Zn deficiency.

It is interesting to note that pigs fed rations with the highest concentrations of supplemental Se or E tended to have the lowest hepatic Zn concentrations. Ewan (1971) reported renal Zn concentrations were significantly decreased when supplemental E was fed and hepatic Zn tended to decrease with supplemental Se or E.


