Milk production data shown in Table 1 appear somewhat erratic and with the exception of the low milk production of Lot 5, females, showed little difference among the treatments. Much more difference due to feed level has been reported elsewhere for older cows.

Summary

Winter feed levels that permit a weaner heifer calf to make little or no gain from fall to spring markedly retards the appearance of heat and delays the subsequent calf crop. Poor winter feeding during the second winter as a bred yearling seriously reduces milk flow the following summer and affects weaning weights. Both treatments, however, appear superior to a Low level both years, but of no advantage over the Moderate level. Maximum body size and weaning weight have been obtained when heifers are fed to gain at least 1 lb. per head daily as weaners, and lose little or no weight as bred yearlings from fall to spring after calving. Feed costs from this regime, however, were excessively high in this experiment, therefore the Moderate regime proved more profitable.

Mineral-Vitamin Interrelationship in Ruminant Nutrition

Allen D. Tillman

It has been estimated that fully 60 percent of the veterinary practice in the United States involves noninfectious diseases, most of which are of nutritional origin. In most cases, severe individual nutrient deficiencies can be identified and rectified by adding the missing nutrient, but the greatest loss in livestock production results from borderline deficiencies. In animals with borderline deficiencies, there are no definite clinical symptoms. Metabolic functions simply slow down to comply with the level of the limiting nutrient resulting in slower growth and less efficiency. To prevent these borderline or subclinical conditions, the nutrient requirements of animals must be met. This is sometimes much more difficult than is apparent to the layman. Recent changes in production methods in plants and animals, with emphasis upon faster and greater growth by the use of fertilizers and other adjuncts, have tended to increase the incidence of borderline deficiencies. Several mineral-vitamin interrelationships in ruminant nutrition are discussed in this article. They include nitrate and vitamin A, calcium and zinc, cobalt and vitamin B_{12}, vitamin E and selenium, and copper and molybdenum.

Nitrate and Vitamin A

Research and field reports have indicated that vitamin A deficiency symptoms occur in fattening beef cattle on rations formerly considered adequate in the carotenes. It has been suggested that either nitrates or some other factor (s) in ruminant rations are inhibiting the conversion of the carotenes to vitamin A by the intestinal walls.

It has been known for some years that the efficiency of carotene utilization decreases with increasing animal requirements. The National Research Council (NRC, 1955) Bulletin, "recommended nutrient allowances of beef cattle" established a ratio of 2.5:1 in favor of vitamin A over carotene at the minimum level of growth, 3.3:1 for reproduction, and 8.3:1 for a high level of lactation indicating that energy level of diet could be involved in the poor conversion of the carotenes to vitamin A.

Phosphorus Deficiency

Work at the Oklahoma Station (1)[1] indicated that a phosphorus deficiency in cattle rations decreased the efficiency of conversion of carotene to vitamin A. As there is widespread usage of phosphorus supplements in ruminant ration it is doubtful if this is a factor in the modern fattening rations.

Nitrates

Heavy nitrogen fertilization increases the nitrate content of some forages (2,3). In feeding tests with rats (4) it was shown that the addition of .3 percent KNO_3 to the diet resulted in a rapid destruction of vitamin E with subsequent rapid decrease in liver stores of vitamin A. High nitrate corn silage was found to exert an adverse effect upon the vitamin A status of rats (5). Similar results were obtained with sheep (6). Results with cattle are not conclusive but trends are in the same direction (7,8,9). The preliminary results indicate that liver storage is affected more than plasma levels (5,6,8,9). It appears that concentration of energy (8) in the diet may have more effect (negative) upon the conversion of carotenes than the nitrate content. A cattle fattening diet containing 71 percent TDN caused much more depletion of liver stores of vitamin A than a diet containing 54 percent TDN.

The conversion of carotene to vitamin A is lower in the vitamin A deficient animal (10) and in animals with hypothyroidism (11,12). It has also been shown that nitrates (13) interfere with the retention of iodine by the thyroid gland. Thus the mechanism of action of nitrates in reducing the conversion of the carotenes to vitamin A appears to be through their actions on the thyroid gland.

[1]Number in parenthes's refers to reference to original research data. These references are found at the end of each report covered in this article.

Vitamin A Requirements

High producing dairy cows weighing 1400 lbs. require a minimum of 34,000 I.U. (14) per day of vitamins (86 mg. carotene). These can be added in the dry form (vitamin A acetate or palmitate) at a cost of only $1.25 per year. Research at Purdue University (15) has shown that steers require additional vitamin A when fed rations containing 1 mg. of carotene per lb. The best results were obtained when 32,000 I.U. of supplemental vitamin A was provided daily. Steers receiving the high levels of vitamin A were observed to have sleeker coats and appeared to be more vigorous. Hale *et al.* (16) reported that the feeding 10,000 I.U. per day of vitamin A to steers failed to maintain liver stores of vitamin A when a fattening ration was fed. When the level of vitamin A was increased to 40,000 I.U. per day, the steers were just able to maintain the liver levels found when they were first placed on the fattening diets while levels above 40,000 I.U. per day promoted rapid storage of vitamin A in the livers of the steers. Beeson (17) recommends that fattening steers receive 30,000 I.U. of vitamin A daily. If the dry form of vitamin A (vitamin A acetate of palmitate) were used for a six months feeding period, the costs would be less than about 60¢ per steer.

Summary

It appears that ruminant animals are poor converters of carotene to vitamin A. Efficiency of conversion of the carotenes appears to be lowered by nitrates or by a greater concentration of energy. Hypothyroidism reduces the efficiency of conversion of the carotenes to vitamin A and it appears that nitrates reduce thyroid function. Some researchers recommend that fattening steers receive a daily allowance of 30,000 I.U. of vitamin A daily.

References Cited

1. Gallup, W. D., O. O. Thomas, O. B. Ross, and C. K. Whitehair, 1953. J. Animal Sci. 12:715.
2. Perez, C. B. and C. D. Staley, 1960. J. Animal Sci. 19:1311. (abstr.).
3. Breniman, G. W., A. L. Neumann, G. S. Smith, and J. E. Zimmerman, 1961. J. Animal Sci. 20:926 (abstr.).
4. O'Dell, B. L., Z. Erek, L. Flynn, G. B. Garner, and M. E. Muhrer, J. Animal Sci. 19:1280 (abstr.).
5. Smith, G. S., A. L. Neumann, and E. E. Hatfield, 1961. J. Animal Sci. 20:682 (abstr.).
6. Hatfield, E. E., G. S. Smith, A. L. Neumann, R. M. Forbes, U. S. Garrigus, and O. B. Ross, 1961. J. Animal Sci. 20:676 (abstr.).
7. Weichenthal, B. A., R. J. Emerick, L. B. Embry, and F. W. Whetzal, 1961. J. Animal Sci. 20:955 (abstr.).
8. Hale, W. H., F. Hubbert, and R. E. Taylor, 1961. J. Animal Sci. 20:934 (abstr.).

9. Mitchell, G. E., A. L. Neumann, R. R. Garrigus, W. M. Durdle, and K. A. Kendall, 1960. Illinois Feeders Day Report, p. 12.
10. Deuel, H. J., 1957. The Lipids. Vol. II. The Blakiston Co., New York.
11. Johnson, R. M. and C. A. Baumann, 1947. J. Biol. Chem. 171:513.
12. Mahtah, S. G. and P. R. Sundaresan, 1961. J. Nutr. 74:39.
13. Wyngaarden, J. B., B. M. Wright, and P. Ways, 1952. Endocrinology. 50:537.
14. Hibbs, J. W., 1961. Feedage. 11(12):38.
15. Perry, T. W., M. T. Mohler, W. H. Smith, and W. M. Beeson, 1961. J. Animal Sci. 19:1283 (abstr.).
16. Hale, W. H., F. Hubbert, R. E. Taylor, T. A. Anderson, and Bruce Taylor, 1961. J. Animal Sci. 20:668 (abstr.).
17. Beeson, W. M., 1961. Proceedings Texas Nutrition Conference.

Calcium and Zinc

A condition called parakeratosis in swine was observed for many years before Tucker and Salmon (1) showed that zinc supplementation prevented this condition. Results with swine and chicks (2,3,4,5) indicate that the severity of the parakeratoti symptoms increased as the calcium level in the diet increased.

Recently Miller and Miller (6) were able to produce a zinc deficiency in calves. When fed a low-zinc purified diet, the calves developed the following symptoms in about eight weeks: slightly red and inflamed nose and mouth, soft swelling above the rear feet in front of the fetlock, small areas of alopecia on the rear legs, breaks in the skin around the hoofs, rough and sealy skin on the rear legs, and a dull, listless appearance. Later symptoms, obtained 11 weeks after the animals were placed on the purified diets, were similar to those reported for hyperkeratosis. The zinc content and carboic anhydrase activity of the blood from the calves were much lower than those of the controls. Evidence that symptoms of zinc deficiency does occur in beef cattle kept under natural conditions is presented by Legg and Sears (7). They observed that cattle grazing on the Berbice savannahs had lesions resembling those described for rats receiving a zinc deficient diet. In the most severe conditions, parakeratosis spreads rapidly over about 40 percent of the body. Symptoms occur between March and June. Zinc supplementation administered either orally (2 gm. per week) or by injection (1 gm. per week) to affected cows caused new hair growth within one week and the animals were normal within three weeks. The unsupplemented control animals continually deteriorated. Later the control animals were also given zinc and responded rapidly. It was found that plants growing in the affected area also responded to supplement zinc.

In my travels through Finland in 1959, I saw many dairy cows suffering from an itch that covered much of their bodies. They had a scurfy skin and there was loss of hair. This condition caused lowered

production (11.4 percent) and reproduction (delayed an irregular estrus). There were increased incidences of uterine inflammation, retention of afterbirth, abortus, milk fever, and ketosis. Feeding 300 to 500 mg. of zinc for 1000 lbs. of body weight daily clears up the condition (8) and new hair growth starts within two weeks.

The high calcium requirement of the dairy cow undoubtedly increases the zinc requirement. No one has studied this, however, in systematic experiments.

Summary

Zinc deficiencies can be produced experimentally and occur in cattle under natural feeding conditions. Cattle deficiency symptoms are similar to those noted in swine, rats, and chicks. Deficiency symptoms in cattle can be alleviated by either feeding supplemental zinc or by subcutaneous injections. Feeding 300 to 500 mg. daily per 1000 lbs. body weight has been effective while injection levels have been about 150 mg. daily for each 1000 lbs. body weight.

References Cited

1. Tucker, H. F. and W. D. Salmon, 1955. Proc. Exp. Biol. Med. 88:613.
2. Lewis, P. K., W. G. Hoekstra, R. H. Grummer, and P. H. Phillips, 1956. J. Animal Sci. 15:741.
3. Lewis, P. K., R. H. Grummer, and W. G. Hoekstra, 1957. J. Animal Sci. 16:927.
4. Supplee, W. C., G. F. Combs, and D. L. Blamkerg, 1958. Poultry Sci. 37:63.
5. Lease, J. G., B. D. Barnett, E. J. Lease, and D. E. Turk, 1960. J. Nutr. 72:66.
6. Miller, J. K. and M. J. Miller, 1960. J. Dairy Sci. 43:1854.
7. Lagg, S. P. and L. Sears, 1960. Nature (London) 186:1061.
8. Haaranen, S. and K. Hyppula, 1961. Feedstuff 33 (46) :281.

Cobalt and Vitamin B_{12}

It has been 27 years since Underwood and Filmer (1) first reported that cobalt was required in ruminant diets and since that time much progress has been made in locating areas of acute deficiency. Cobalt deficiency in ruminants and its relation to vitamin B_{12} has been the subject of several excellent review papers (2,3,4,5). The experimental evidence supports the idea that rumen microorganism use cobalt to synthesize vitamin B_{12} and that the absorbed vitamin B_{12} is required by ruminant tissues in much the same way that non-ruminant tissues require this vitamin. Thus a cobalt deficiency in the ruminant is essentially a vitamin B_{12} deficiency which can be alleviated by either an oral or intravenous administration of vitamin B_{12} (5); however, the practical

control is through feeding supplemental cobalt. Australian workers have described a new procedure (6) for supplying cobalt to ruminants. The procedure makes use of the anatomy of the reticulum, which retains heavy foreign bodies. Cobalt oxide is pelleted and placed in the esophagus of the animal, who swallows it. It is subsequently trapped in the reticulum and there the cobalt is released at a slow rate to the rumen fluid. Our best proof of a cobalt deficiency in animals is the rapid recovery when cobalt is fed, thus many new cobalt-deficient areas have been found in this country by using this new procedure of cobalt administration. The requirement for cobalt is about .1 mg. per 100 lbs. body weight daily.

It has always been a mystery regarding why the vitamin B_{12} requirement in ruminants is so high and the answer had come only recently (7,8,9,10). Experimental evidence supports the idea that the coenzyme form of vitamin B_{12} is required for the metabolism of methylmalonate to succinic acid; methylmalonate is an intermediate in the pathway of propionic acid metabolism. Thus, cobalt, through vitamin B_{12}, is required for energy reactions typical of the ruminant animal only.

Summary

Ruminant animals have a high requirement for vitamin B_{12} because it is used in the metabolism of propionic acid. Dietary cobalt is used by rumen microflora in synthesizing vitamin B_{12}, thus ruminant requirements for vitamin B_{12} can be met by supplying .1 ppm of cobalt in the ration. This amounts to only .1 mg. per 100 lbs. body weight and can be supplied in pellets or in salt.

References Cited

1. Underwood, E. J. and J. F. Filmer, 1935. Australian Vet. J. 11:84.
2. Russell, F. C. and D. L. Duncan, 1956. Commonwealth Bureau Animal Nutr. Tech. Communication 15 (London).
3. Underwood, E. J., 1956. Trace Elements in Human and Animal Nutrition. Academic Press, New York.
4. Smith, S. E. and J. K. Loosli, 1957. J. Dairy Sci. 40:1215.
5. Fuller, R. G. and V. W. McAlpine. Cobalt in Animal Feeding. Centre D'Information in Cobalt, 35 rue des Colonies, Brussels.
6. Dewey, D. W., H. J. Lee, and H. R. Marston, 1958. Nature (London) 181:1367.
7. Flavin, M. and S. Ochoa, 1957. J. Biol. Chem. 229:965.
8. Beck, W. S., M. Flavin, and S. Ochoa, 1957. J. Biol. Chem. 229:997.
9. Barker, H. A., H. Weissbach, and R. D. Smyth, 1958. Proc. Nat'l Acad. Sci. 44:1093.
10. Lenhyal, P., R. Mazumder and S. Ochoa, 1960. Proc. Nat'l Acad. Sci. 46:1312.

Vitamin E and Selenium

Losses from White Muscle Disease (WMD) in certain areas of the western states have reached serious proportions (1). This disease has been recognized for many years (1,2) but there has not been an understanding of it. The disease has been described as Muscular Dystrophy, Nutritional Myopathy, Vitamin E Deficiency, or Stiff-lamb Disease. It causes varying degrees of stiffness when skeletal muscles are involved and sudden death when the heart muscles are involved.

For many years WMD was considered to be caused by a vitamin E deficiency (3); early work also indicating that the milk from cows or ewes consuming such diets were low in vitamin E (4). Also there was, in general, an improvement when vitamin E was administered to affected animals (5); however, evidence became available (6) that vitamin E was not the primary or sole cause of WMD. Experimental diets containing much lower levels of vitamin E than those found to produce WMD in the field produced normal animals (5,6,7). Authorities at this time were confused by these inconsistencies and attention was drawn to the brilliant researches of Schwarz and colleagues (8,9,10,11,12,13) in which they showed that selenium was an essential nutrient and that it was 500 times more active than vitamin E in preventing degeneration in rats and chicks. At this time it was also demonstrated that exudative diathesis was prevented in the fowl by selenium or vitamin E.

Interest in the possible beneficial effects of selenium in ruminant nutrition was aroused by the results of Muth *et al.* (2) and Proctor *et al.* (14) who, almost simultaneously, found that dietary selenium reduced the incidence of WMD in lambs. The Oregon results (2) showed that the feeding of .1 ppm of selenium as Na_2SeO_3, from 90 to 100 days prior to lambing showed a preventive effect against WMD, while vitamin E given orally or parentally to similar ewes was ineffective. When the New York workers fed ewes 1 ppm supplemental selenium 30 days prepartum, confirmatory results (14) were obtained. Later the Nevada workers (1) found that either oral or injectable selenium given to the ewe prior to lambing was effective in preventing WMD. These workers added .1 ppm (sodium selenite) to the diet during the last three months of pregnancy. They also injected some ewes with barium selenate ($BaSeO_4$). The selenium was suspended in a mixture of 98 percent peanut oil and 2 percent beeswax and enough selenium to supply .2 gm. $BaSeO_4$ per ml. and the ewes received .1 gm. barium selenate per 100 lbs. body weight. In order to determine the effectiveness of the treatments serum samples from their lambs were taken 10, 24, and 38 days of age and tested for its transaminase (serum glutamic-oxalacetic transaminase, SGOT) activity. The enzyme, transaminase, is liberated into the blood as a result of muscle damage or destruction.

The Nevada workers (1) also have shown that WMD can be prevented in susceptible lambs by feeding vitamin E in the creep feed. They provided each lamb with approximately 200 international units

of vitamin E daily and it was found that this method of feeding was effective in preventing WMD.

Much interest was aroused by the report (15) that WMD in lambs from susceptible areas was prevented by a subcutaneous injection of 1 mg. sodium selenate at birth and each 10 days thereafter until the lambs were weaned. Gains of the injected lambs were greater than their controls even in areas where no WMD has been observed. Results with calves in Scotland (16) are confirmatory.

It is interesting to note that, even though a direct comparison is impossible, protection from selenium in 1960 was similar to that afforded by vitamin E from 1953 to 1959. The single injection consisted of 15 mg. of selenium, while 5 mg. of selenium was either injected subcutaneously or given orally for three consecutive weeks beginning shortly after the calves were born. Evidence that selenium injected subcutaneously has both prophylactic and therapeutic effect in lambs was presented by Lagase (17).

Evidence is accumulating which indicates that the major if not only biochemical role of vitamin E is its inhibition of tissues lipid peroxide formation with the consequent effect of the free radicals. The association between vitamin E and selenium involves the antioxidant or antioxygenic properties of tissues but the exact relationship is not clear. Recent work (18) indicates that the antioxidant, 1,2-dihydro-6-ethoxy-2,2,4-trimethylquinoloine (Santoquin) prevents WMD in lambs and exudative diathesis in chicks. This opens up a new area in which several groups are actively investigating at this time.

Summary

Selenium fed to ewes in selenium-deficient areas at a level of .1 ppm for 3 to 4 months prepartum will prevent WMD in lambs; 1 ppm appears to be effective when given 30 days prepartum. Either vitamin E or selenium will prevent WMD if given to the lambs in selenium-deficient areas. Vitamin E is required at a level of 200 I.U. per lamb daily while selenium injected subcutaneously is required at a level of 1 mg. sodium selenate every 7 to 10 days. Either vitamin E (fed) or selenium was effective in preventing WMD in calves. Selenium was as effective when fed as when injected subcutaneously.

References Cited

1. Kuttler, K. L. and D. W. Marble, 1959. Nevada Agr. Exp. Sta. Bul. 207.
2. Muth, O. H. and J. E. Oldfield, J. R. Schubert, and L. F. Remmert, 1958. Sci. 128:1090.
3. Maynard, L. A., 1947. Animal Nutrition, 2nd edition. McGraw-Hill Book Co., Inc.
4. Whiting, F., J. P. Willman, and J. K. Loosli, 1949. J. Animal Sci. 8:234.

5. Willman, J. P., J. K. Loosli, S. A. Asdell, F. B. Morrison, and P. Olafson, 1946. Cornell Vet. 34:200.
6. Muth, O. H., 1955. J.A.V.M.A. 126:355.
7. Safford, J. W., K. F. Swingle, and D. E. McRoberts, 1956. Am. J. Vet. Res. 17:503.
8. Schwarz, K., 1944. Z. Physiol. Chem. 281:109.
9. Schwarz, K., 1951. Proc. Exp. Biol. Med. 77:818.
10. Schwarz, K., 1951. Proc. Exp. Biol. Med. 78:852.
11. Schwarz, K., 1952. Proc. Exp. Biol. Med. 80:319.
12. Schwarz, K., J. G. Bieri, G. M. Briggs, and M. O. Scott, 1957. Proc. Exp. Biol. Med. 95:621.
13. Schwarz, K., 1960. Nutr. Rev. 18:193.
14. Proctor, J. F., D. E. Hogue, and R. G. Warner. 1958. J. Animal Sci. 17:1183.
15. McLean, J. W., G. G. Thomason, and J. H. Claxton, 1959. Nature (London) 184:251.
16. Blaxter, K. L., E. S. R. McCallum, and R. S. Wilson, 1961. Proc. Nutr. Soc. 20:VI.
17. Lagase, A., 1961. J.A.V.M.A. 138:188.
18. Erwin, W. S., W. Sterner, R. S. Gordon, L. J. Machlin, and L. D. Tureen, 1961. J. Nutr. 75:45.

Copper, Molybdenum, and Sulfur

Ferguson *et al.* (1) reported that some pastures in certain areas of England caused severe scours in cattle and that the pasture grasses from these areas contained from 20 to 100 ppm of molybdenum. Later these workers (2) reported that the feeding or the drenching of cattle with copper sulfate prevented the excessive scouring and it was also found that increased levels of molybdenum (3) caused reduced levels of copper in the liver. The mechanism of the copper-molybdenum interaction is still not clear (4,5) even though it has been demonstrated that molybdenum toxicity symptoms are, in effect copper deficiency symptoms.

Inorganic sulfate is also involved (6,7) in the molybdenum-copper interaction. Molybdenum exerts its effect on copper only if inorganic sulfates are present at a sufficient level. The effectiveness of each of the elements is increased as the level of the other is increased, thus chronic copper poisoning can be obtained when copper intake is moderate but sulfur and molybdenum contents are low (8). It is also quite apparent that copper deficiency symptoms appear very quickly if the ration contains high levels of sulfur and molybdenum. The mechanism of action appears to have been elucidated by the researches of Mills *et al.* (9) and Halverson *et al.* (10). They observed that high levels of molybdenum in the presence of the sulfates caused a great reduction in the liver sulfide oxidase level, indicating that there is a great loss of copper through the formation of highly insoluble cupric sulfides in the liver.

In areas in which the pasture grasses contain 20 to 100 ppm of molybdenum, copper sulfate fed at levels of 2 gm. per day in adult and 1 gm. per day in young cattle overcomes the detrimental effects of the

excessive molybdenum. Daily drenching with 2 gm. of copper sulfate for four days also brings the condition under control.

Molybdenum is a part of an enzyme system (11), xanthine oxidase, thus is an essential mineral. Deficiency symptoms have been produced in chicks and rats but special treatment of the animals are necessary (12,13). Missouri workers (14) produced molybdenum deficiency symptoms in sheep, but these results were not confirmed by the Oklahoma workers (15).

Copper levels of 5 ppm meet the requirements of most animals in diets containing normal levels of molybdenum and sulfur. Molybdenum requirements of most animals are probably less than 1 ppm if normal copper and sulfur levels are used.

Summary

Molybdenum toxicity symptoms in ruminants appear to be copper deficiency symptoms. If adequate sulfur is present the excess molybdenum apparently causes precipitation of insoluble copper sulfide in the liver. Copper toxicity levels are much lower if inadequate levels of molybdenum or sulfur are present. Molybdenum toxicity symptoms can be alleviated by the feeding of copper sulfate.

References Cited

1. Ferguson, W. S., A. H. Lewis, and S. J. Watson, 1938. Nature (London) 141:553.
2. Ferguson, W. S., A. H. Lewis, and S. J. Watson, 1943. J. Agri. Sci. 33:44.
3. Dick, A. T., 1952. Australian Vet. J. 28:30.
4. Dick, A. T., 1956. Soils Sci. 81:229.
5. Allcroft, R. and G. Lewis, 1957. J. Sci. Food Agr. 8:596.
6. Dick, A. T., 1954. Australian P. Agr. Res. 5:511.
7. Dick, A. T., 1954. Australian Vet. J. 30:196.
8. Wynne, K. N. and G. L. McClymont, 1956. Australian J. Agr. Res. 7:45.
9. Mills, C. F., K. J. Monty, I. Ichihora, and P. B. Pearson, 1958. J. Nutr. 65:129.
10. Halverson, A. W., J. H. Phifer, and K. J. Monty, 1960. J. Nutr. 71:95.
11. Scaife, J. F., 1956. N. Z. J. Sci. Technol. A. 38:285.
12. Higgins, E. S., D. A. Richert, and W. W. Westerfield, 1956. J. Nutr. 59:539.
13. Leach, R. M. and L. C. Norris, 1957. Poultry Sci. 36:1176.
14. Ellis, W. C., W. H. Pfander, M. E. Muhrer, and E. E. Pickett, 1958. J. Animal Sci. 17:180.
15. Sheriha, G. M., R. J. Sirny, and A. D. Tillman, 1962. J. Animal Sci. 21:in press.