Trace Element Deficiencies and Fertility in Ruminants: A Review

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ABSTRACT

Various minerals (copper, cobalt, selenium, manganese, iodine, zinc, and iron) can influence reproductive performance of ruminants. Reproductive failure may be induced by deficiencies of single or combined trace elements and by imbalances. This review is focused on maladjustments of trace elements leading to impaired breeding performance. Opinion is diverse as to the existence of various reproductive disturbances from either a severe copper depletion or a marginal dietary copper deficiency. Field experience suggests that administration of cobalt to ruminants on cobalt-deficient diets improves their impaired breeding performance. Selenium infertility in ewes is more prevalent in some areas and in some seasons, but the actual cause of this malady and the continuing role of additional factors are unknown. Manganese is necessary for normal fertility in ruminants, and feeding low-manganese rations depresses conception rates. Lack of iodine impairs thyroid activity and also ovarian function. Reproductive failure in the female and in spermatogenesis are manifestations of zinc deficiency. Despite forages rich in iron, low availability in certain instances could affect adversely ruminant reproduction.

Knowledge of biochemical dysfunctions from trace element deficiencies is essential to determine the role which trace elements play in fertility of ruminant animals.

INTRODUCTION

Trace elements may function as cofactors, as activators of enzymes, or stabilizers of secondary molecular structure (103). Their study has evolved from a recognition of their essential function in cell metabolism. There has been special interest in effects of dietary trace element deficiencies on physiological functions in general and in reproduction in particular (56). Ruminants frequently are subject to severe dietary deficiencies of trace elements such as copper, cobalt, selenium, iodine, manganese, and zinc. Concomitant infertility in cattle is believed to be associated with enzymatic dysfunctions resulting from these deficiencies. Despite the accumulation of data suggesting that reproductive performance in ruminants depends upon adequate trace element nutrition, there has been no comprehensive review of this information.

Mineral deficiency usually involves several minerals as well as other conditioning factors; however, the deficiency symptoms of one mineral may predominate and affect the performance of the ruminant. The effects of the lack of a single element may include reproductive failure. In this respect effects of copper, cobalt, selenium, manganese, iodine, zinc, and iron malnutrition on reproduction will be reviewed.

Copper

A variety of clinical symptoms have been associated with copper deficiency. Hypocuprosis in cattle is associated with female reproductive disorders. Where such disorders have been attributed to copper deficiency, the most common symptom has been prenatal mortality, particularly early embryonic loss. Most of the relevant studies have been with sheep or goats.

A low copper content in the diet of the ewe either prevented implantation or induced
embryonic loss and fetal death (61, 62). Of nine ewes fed a severely copper-deficient diet and then bred, five did not become pregnant and died between the 23rd and 34th wk of the experiment; of the remaining four ewes, two aborted and two produced stillborn lambs. Feeding female goats a semipurified diet low in copper content lowered conception rates (4); of the pregnant goats affected by copper deficiency, 50% aborted. Fetal death occurred between the 2nd and 5th mo of pregnancy. The fetuses were mummified, and at abortion placenta had degenerated with hemorrhagic or necrotic lesions.

Contrary to expectations, concentration of copper in blood does not appear to be related directly to reproductive performance. Reproductive performance of rams and their plasma copper were unassociated (106). In the ewe, low copper in blood did not reduce the size of the lamb crop (45).

Copper deficiency may affect reproductive behavior as well as performance; for example, older female goats kept on a low copper diet for an extended period showed nymphomania (5). In these clinical field studies possible associated changes in gonadal steroid metabolism were not investigated.

Reliable information is limited for the influence of absolute or conditional copper deficiencies (such as results from a low copper diet with excess molybdenum and/or sulphur) on the fertility of cattle. One of the earliest signs of copper deficiency in cows is a decline in fertility. Marked improvement results from copper administration, provided there are no other deficiencies (42, 43). Opinions from casual observations in the field usually have been contradictory with few well planned studies yielding unequivocal data.

Generally, there is no clear relation between copper in the blood and liver of cattle and their fertility (54, 85). In some herds of cattle with low blood copper, female fertility has been impaired; yet in others conception rate has been high (78). Administration of copper orally or by injection has led to a rapid and dramatic improvement in breeding performance (58). In New Zealand in herds in which blood copper was marginally subnormal, a single administration of 400 mg of copper glycinate improved fertility. The conception rate for the copper treated cows was 72% versus 53% for the untreated cows; treated cows required 1.00 and untreated 1.15 services per conception (47).

Dietary supplement of copper has improved the reproductive performance of cattle (96). A high incidence of infertility in cattle in Australia (11) was associated with cases of "falling disease", anemia, and retarded growth in areas where pasture copper was less than 3 ppm. This was overcome by parenteral administration of copper. In Canada an increase in the copper:molybdenum ratio was associated with an improvement of fertility in cows fed silage (72), and in other studies infertile cows showed marked improvement in their conception rate when treated parenterally with copper glycinate (13, 80).

In bulls, the administration of copper improved semen quality (48). The improvement was associated with increased sperm mobility and fewer dead spermatozoa. However, situations have been described in which copper deficiency in young livestock was severe, but effects on breeding performance were not adverse (96).

The pituitary gland and ovaries of cows contained 9.5 and 4.8 µg copper/g dry weight (93). The average zinc and copper concentrations in the fluid of normal and cystic follicles of cows and heifers in (73) were 3 mg/liter Zn and 2.1 mg/liter Cu. No differences between cows and heifers in the follicular fluid content of either element were detected, nor were there significant correlations between liver and follicular fluid copper and zinc. Zinc and copper concentrations in fluid of follicular cysts from anestrus cows decrease as the volume of the fluid increases, indicating that content per follicle is constant and suggesting that these elements are intracellular in the follicle. The uterine fluid of cows with cystic endometrial hyperplasia contained 13 mg/liter zinc and 12 mg/liter copper. Again there was an inverse relation between volume of fluid and copper and zinc concentrations (73).

Metabolism of copper appears to be altered during pregnancy. Copper in blood plasma of ewes falls during pregnancy and rises after parturition (20). In pregnant cows, however, ceruloplasmin and plasma copper have increased, the highest attained in the 5th mo of pregnancy (22). Parturition also may be influenced by copper deficiency. Cows with retained placentas had lower serum copper than normal cows (105).
There seems to be an interaction between estrogen and copper. Diethylstilbestrol administration increased copper in the rat serum (98). In humans receiving diethylstilbestrol therapy, ceruloplasmin or copper, increased in plasma apparently in proportion to the dose of the estrogen administered (15). Changes in plasma copper in rats parallel changes in plasma estrogen during the estrous cycle, after ovariectomy, and during administration of exogenous estrogen (81). The relevance of these findings to similar interactions in ruminants, however, is not clear. The administration of estrogen to wethers and ewes for 3 mo did not affect copper in their blood or liver (69). Although the dosage of estrogen was physiological, the lack of any effect does not eliminate the possibility of nonphysiological interactions having pharmacologic interest.

Conclusions

Findings regarding the effect of copper deficiency on the fertility of ruminants conflict. It is difficult to ascertain whether reproductive function is affected directly by the lack of dietary copper or by some general dysfunction produced by copper deficiency. That otherwise normal cattle with a history of reproductive failure can be cured by copper administration suggests that the effect is specific. However, when cattle are affected by severe hypocuprosis, characterized by gross deficiency symptoms, copper therapy alleviates the physical conditions including infertility, thus suggesting that the effect is general. Nevertheless, there are reports of hypocupremic cows which appear normal and are also fertile. One might compare these differences in fertility to the variance in the clinical manifestations of copper deficiency. For example, there is a form of copper deficiency which is clinically silent and results in only marginally suboptimal fertility in cattle. This differs from the “persistent scouring” symptom common in Holland and from the “fibrosis of myocardium” which occurs in Australia.

Further study is needed to clarify the mechanism by which copper is involved in reproductive processes. Response to copper deficiency must take into consideration interactions with other dietary factors such as the availability of copper which may be influenced by proteins which, in turn, affect copper absorption (101). In addition, complex formation between copper, molybdenum, and sulfur may occur in the rumen which also will affect the availability of copper (100, 102) and produce a deficiency despite apparently normal copper intake.

Cobalt

Field experience suggests that cobalt deficiency impairs breeding performance in both cattle and sheep. The most common manifestation of cobalt deficiency is a marked reduction in conception rate. In tropical areas such as Central Africa (Zaire) or the northern part of South America (Northern Brazil, French Guyana) in which cobalt deficiency is endemic, the calving interval encountered is often greater than 24 mo instead of the usual 14 mo (Hidiroglou, unpublished). The reason for this is not yet known. However, several studies appear relevant. Involvement of the uterus is completed within 3 wk after calving in cows treated with cobalt but requires as much as 6 to 9 wk in cobalt-deficient cows. Moreover, the cobalt treated cows have stronger manifestations of estrus as well as a lower incidence of irregular estrous cycles and a higher conception rate (56). A Shorthorn herd grazing a cobalt deficient pasture had a conception rate at first service of 53% compared with 67% in a similar herd grazing in the same area but given copper therapy and 93% in a herd given cobalt as well as copper therapy (79).

In studies with sheep, cobalt deficiency was associated with a reduction in estrus during the normal breeding season. Dietary cobalt supplementation increased the incidence of estrous activity (110) and increased sperm counts of males (7).

Conclusions

Cobalt is required to ensure fertility in ruminants. In cobalt deficiency conception rate of cattle declines while in the ovine lack of estrus is one of the features of low dietary cobalt intake.

Selenium

There are conflicting opinions of the effect of selenium deficiency on reproductive performance in the ruminant. In those studies of
impairment of fertility associated with selenium deficiency, the locus of the impairment generally has been embryonic or fetal loss. In several cases of selenium deficiency, administration of selenium prevented certain reproductive problems. Lambing percentages have increased in controlled trials with ewes when selenium was administered orally in monthly doses beginning 1 mo before mating and continuing through pregnancy (33, 34, 35). Reduced fertility in the untreated animals was attributable to embryonic mortality at 20 to 30 days of pregnancy (3). Ewes grazing on pasture of low selenium and high estrogen content were given selenium 4 to 8 wk before breeding. Their conception rate increased from 49% to 76% (30). Significant improvement in lambing rate and incidence of twinning in ewes has been reported when the diet was supplemented with selenium and copper (46); the twinning rate was depressed by a selenium deficiency with fewer lambs born to the control ewes.

In contrast, studies on the effect of selenium given to ewes in another selenium-deficient area in Australia revealed no significant influence on conception rate (23). Similarly, in areas where white muscle disease in sheep had been diagnosed, selenium supplementation showed little or no effect on sheep fertility (29). Nine trials with ewes on low selenium pastures in Western Australia failed to show an improvement in reproductive performance when up to 50 mg of supplementary selenium was administered prior to breeding (60).

In other studies, there were no significant differences in rates of ovulation or embryonic loss between ewes fed selenium-deficient hay and those fed hay plus a selenium supplement. Overall fertility in selenium-deficient ewes was not lower than that of selenium treated ewes (67).

In cattle also, there are contradictory reports of a selenium requirement for the maintenance of fertility. Low selenium (less than 0.05 ppm) appears to account, at least in part, for infertility of dairy herds in some areas in Uganda (55). In other studies, however, prebreeding selenium treatment of beef cows in areas deficient in selenium did not improve subnormal pregnancy rates (83, 91).

Interactions between selenium intake and the availability of vitamin E have become apparent through the research of a number of workers who have indicated the difficulties of interpreting observations based on situations in which one of these factors is controlled. It is possible that the onset of infertility and its severity under field conditions are associated with seasonal changes in selenium and/or vitamin E in forages. Such fluctuations may account for some of the conflicting observations regarding the influence of selenium on fertility. Studies with purified diets have shown no differences in the fertility of selenium-depleted and of selenium-sufficient ewes, provided that adequate amounts of vitamin E are supplied (18, 19). In cattle, administration of vitamin E and/or potassium selenate 1 mo before calving greatly reduced the incidence of retained placenta in a herd of dairy cows fed little selenium during the precalving period (97).

Effects of selenium and vitamin E, alone or in combination, on fertilization of ova have been studied in beef cows on either a low or an adequate plane of nutrition (86); the fertilization rate was 100% for females on an adequate plane of nutrition given a supplement of selenium and vitamin E. However, rates of fertilization were lower when the diet was deficient in either selenium or vitamin E or was below maintenance requirement for protein and energy (86).

Little information is available concerning the influence of selenium on fertility in the male. Only one study deals with ruminants, and this indicates that selenium does not appear to influence testosterone secretion in rams (18). In rate, sperm mobility was poor in selenium-deficient males. The spermatozoa showed breakage near the mid-piece in most cases. Vitamin E supplementation, even high, did not alleviate these symptoms (108). The only studies of interactions of selenium with endocrine secretion, other than that just cited, concerns the rat in which selenium of blood fell between days 10 and 15 of gestation (9).

**Conclusions**

Reduced fertility sometimes is associated with selenium-deficiency in sheep and cattle. However, there are indications that other conditioning factors are involved. Low tocopherol in the roughage or the presence in the feed of certain antagonists of vitamin E and selenium may reduce fertility in animals raised
or maintained in selenium-deficient areas. The problem of selenium-related infertility appears to be more prominent in sheep than in other domestic ruminants.

Manganese

Although manganese occurs in most body tissues in low concentrations, only in extremely low dietary and tissue concentrations does deficiency result in manifestations of disturbed or depressed reproductive function (24, 101). The actual expression appears to depend on a variety of factors such as degree and duration of the deficiency.

While the most detailed and extensive investigation of the consequences of manganese deficiency have been with laboratory animals, a number of studies in cattle have yielded essentially similar results (5, 50). Principal among these are suppression of estrus, reduction in conception rates, increased incidence of abortions, and small birth weights. The main clinical sign of dairy cattle infertility caused by a low manganese intake is anestrus or irregular return to estrus, sometimes with extended periods of anestrus (14, 107). Follicular development is often poor, delayed ovulation is not infrequent, and signs of estrus when it does occur are less obvious. Conception rates of only 35 to 40% are not unusual (14, 107). Dairy cattle with a manganese intake of less than 40 ppm in the feed maintained high fertility when the calcium and phosphorus content of the diet were adequate and well balanced (43, 44), but when the calcium to phosphorus ratio was high and dietary manganese low (less than 40 ppm), fertility was depressed (45). However, when manganese content of the diet exceeded 100 ppm, the best breeding performances were with high calcium to phosphorus ratios. In Scandinavia, prophylactic administration of .25 g manganese per day to dairy cattle increased fertility (Sanstedt, cited by Kolb (50)).

Related reproductive observations have been in sheep and goats as well. More services per conception (2.5 vs. 1.5) were required for ewes fed a low manganese diet (less than 8 ppm) than were required for ewes fed a manganese supplemented diet (39). In female goats manganese deficiency resulted in a poor conception rate and increased embryonic mortality (5).

In South Australia manganese supplements increased the number of lambs born to a flock of Dorset-Horn ewes in which reproductive performance previously has declined (27); this further indicates the necessity for continued awareness of the possible need for providing the small grazing ruminant with manganese supplements.

Abortions and cystic ovaries are prevalent on the farms using feeds relatively rich in manganese while incidence of anestrus is relatively high in dairy herds with a manganese deficiency (28). Manganese content of corpora lutea from cows without ovarian cysts was generally higher than in luteal tissue from cows with cystic ovaries although the difference was not statistically significant (40). In the cortical stroma, which contains ovarian follicles in various stages of development and regression, the manganese concentration was significantly higher in cows without ovarian cysts than in those with cysts. The reason for the lower manganese content in cystic ovaries may be increased degeneration of granulosa cells since these are rich in mitochondria in which manganese is concentrated (40).

While the precise loci of specific manganese involvement in reproductive processes remain unknown, some evidence suggests that manganese plays a role in the activity of certain endocrine organs. The pituitary and ovary are relatively rich in manganese (5.9 and 5.2 μg/g). Ovarian content in particular is sensitive to dietary deficiencies of manganese. Manganese dropped by one-third of normal during a 6-mo imposition of a deficient diet (93). Large ovarian follicles and corpora lutea of ewes have taken up radioactively labeled manganese to a greater extent than other ovarian or extraovarian reproductive tissues (36). Uptakes also have differed in relation to the estrus cycle. Maximal
uptake occurred in the corpora lutea and this was greatest between days 4 and 11 of the estrus cycle when luteal progesterone production was greatest. This suggests that manganese is involved specifically in luteal metabolism and/or activity (36, 37).

Although manganese is well known as an activator of enzyme systems in many metabolic pathways and as a constituent of certain metalloenzymes (103), it also has been implicated explicitly in the synthesis of sterols, and, hence, of gonadal hormones (10). Gonadotropins such as HCG may influence manganese transport and modify its availability to different tissues of organs (38).

Conclusions

Manganese is necessary for normal fertility in ruminants, and a manganese deficiency depresses conception. Because of the generally low manganese content of corn silage there is a need for constant alertness to the possibility of reproductive failure caused by manganese deficiency in farm animals whose diets contain high proportions of corn silage, as well as in animals kept on land manganese-deficient.

Iodine

The need for iodine for the prevention of goiter in lambs long has been recognized. Its involvement in thyroid function and homeostasis through its incorporation with the thyroid hormones, thyroxine and triiodothyronine, is understood reasonably well. It generally has not been recognized, however, that iodine-deficiency in ruminants has repercussions in reproduction.

Iodine deficiency during pregnancy impairs fetal thyroid functions, and abortions are common in the iodine-deficient pregnant cow. Normal development of the reproductive organs and their proper functioning is dependent on thyroid status. Thyroidectomized dairy heifers cease to exhibit estrus at regular intervals (66, 92). An iodine-deficient diet fed to dairy cows resulted in anovulatory estrus caused by a disturbance of the thyroid and pituitary functions. This was corrected by the addition of iodine to the diet (88). For "repeat breeder" cows on iodine-deficient pasture, conception rate was improved by adding iodine to their feed (63). In Finland the administration of iodine to cattle by sublimation resulted in higher conception rate and lower incidence of retained placentas than in controls (68).

The beneficial effect of iodine is believed to involve stimulation of the anterior pituitary gonadotrophic secretion mediated through the thyroid gland. Intrauterine injections of iodine to cows caused distinct lengthening of the estrous cycle, but it was not ascertained whether this was physiologic, pharmacologic, or toxic (32). Rat uterus bound inorganic iodine specifically (101). Another phenomenon which may be involved in the lengthening of the estrous cycle is the effect of iodine on secretion of thyrotropin-releasing factor which in turn stimulates prolactin secretion (16). Infertility associated with iodine deficiency has been in sheep as well as in cattle. In areas in which a high incidence of goiter occurs among lambs, infertility problems in ewes have been alleviated by provided salt supplements containing .07% iodine (6).

Subnormal protein-bound iodine (PBI) in cows has been associated with infertility (2). In chronic repeat breeders, the average PBI was 37 μg/liter, significantly below the average of 46 μg/liter in normal cows. There was a significant inverse relation between serum PBI and interval between first breeding and conception as well as number of services per conception (53).

Improved reproductive performance was associated with higher PBI; for every 10 μg/liter increase in PBI, there was a decrease in the service interval of 7.4 days and a decrease of .2 services per conception. Low serum PBI also have been associated with abortion in cows (52).

During the estrous cycle in cattle, iodine and PBI in blood do not change (87). Blood iodine is depressed significantly by pregnant mare serum gonadotropin therapy but is not affected by administration of estrogens (82). In pregnant cows PBI was as high as 128 μg/liter (1). Iodine and PBI concentration of the thyroid are reduced in cows with cystic ovaries and also in cows in estrus as compared with pre-estrus (1). Other observations have suggested influence of estrogen on thyroid function; it is probable that the same pituitary factors (gonadotropin) which stimulate estrogen secretion have some direct action on the thyroid gland.
Conclusions

Iodine influences reproductive functions because of its vital role in thyroid function. Thus, reproductive failure in cases of iodine deficiency are likely to be secondary manifestations of thyroid dysfunctions resulting in anestrus of irregular estrus, retained placenta, abortion, and stillbirth.

Zinc

Zinc has been recognized for several decades as indispensable for normal growth and health in animals. Its lack causes various malformations and has deleterious effects on sexual functions, particularly in the male animal.

Although naturally occurring zinc deficiency is rare in livestock, there are numerous reports of improvements in the reproductive performance of animals given dietary zinc supplements. In general, information as to the roles zinc may play in reproductive processes has been obtained primarily from studies on experimentally induced zinc deficiencies.

In South Australia ewes given 140 mg of zinc weekly produced significantly more lambs than did untreated ewes. It is not clear whether this effect represents a remedy of a zinc deficiency. The zinc content of the pasture on which the flock had grazed varied between a subnormal 12 ppm dry grass and a normal or optimal 45 ppm over 2 yr (27). Some workers (84) have questioned an effect of zinc deficiency on fertility in ruminants although they have reported an apparent influence on the sex ratios of offspring in zinc-deficient animals.

Fertility of sheep has been improved when both rams and ewes received zinc supplementation (12, 51). Among the various factors responsible for the poor fertility of ewes on a low zinc ration was early embryonic mortality. The fertilized ovum of ewes fed low zinc diets does not implant in the uterine mucosa (8, 89). Feeding goats semipurified diets low in zinc caused low conception rate and reduced the number of kids per goat (5).

Cows given a zinc supplement had a 23% higher conception rate than controls. The ovarian zinc content of the treated cows was higher than that of controls. Discontinuation of the supplementation lowered fertility (71).

Zinc concentrations in plasma of nonpregnant heifers at various stages of the estrous cycle were little changed (25). Zinc in plasma decreased during parturition and was of greater magnitude in cows suffering from dystocia (25, 76). Although large transitory depressions of zinc have been recorded in some ewes with dystocia, fall in the zinc concentration has not been comparable to that in cattle (64). Possibly these changes in zinc are secondary to the dramatic increases in prostaglandin associated with parturition, inasmuch as prostaglandins bind zinc and facilitate its transport (90).

The highest zinc concentration in animals is in human semen and spermatozoa. The concentration of zinc in the semen of domestic animals is considerably less and variable among animals within species (26, 59). Considerable attention has been given to zinc in the accessory glands and in the semen of livestock in relation to fertility. In a study of seminal plasma of bulls of high and low fertility, the total zinc content was related inversely to fertility; zinc was lowest in the semen of highly fertile bulls and rose as fertility decreased (94). It is difficult to determine from available data whether high concentrations of zinc have an adverse effect on the fertilizing capacity of the semen or are themselves the consequence of other deleterious influences.

Zinc deficiency influences spermatogenesis in the ram. In rams fed a zinc deficient diet (2.4 ppm) atrophy of the seminiferous tubules accompanied complete cessation of spermatogenesis (99).

In lambs, a diet with 17 ppm of zinc supported satisfactory somatic growth but was insufficient for normal testicular growth and spermatogenesis compared with lambs fed a similar diet containing 32 ppm zinc. Zinc-deficient calves had considerably smaller testes than normal calves (74), and zinc-deficient goats had seminiferous tubules which were smaller than those of controls and contained an immature germinal epithelium (65). Other workers have found zinc-deficient goats had testicular atrophy and lacked libido (70).

It appears that male accessory sex gland is also dependent on zinc for normal function. The prostate, in particular, contains a relatively high concentration of zinc, and its uptake by this gland is stimulated by testosterone (41) which regulates prostate activity.

While zinc has been recognized as a cofactor or constituent of certain enzymes, for example...
carbonic anhydrase, activity of zinc-dependent enzymes in reproductive processes is unknown (17, 75). It has been postulated (77) that a possible role of zinc in ram seminal fluid might be to prevent destruction of spermatozoal DNA by inhibiting the DNase activity. Zinc is required for spermatogenesis during the final stage of maturation and also for maintenance of the germinal epithelium (99). Although some of these involvements may be mediated by influences on pituitary gonadotropin production (109), follicle stimulating and luteinizing hormones in serum of zinc-deficient cows are not altered by zinc supplementation (49).

Conclusions

Zinc deficiency in ruminants, as in other species, causes a more pronounced impairment of fertility in the male than in the female. The effect is severe and appears to be specific to the final stages of spermatozoa maturation. Testicular size and concentration of zinc in semen may be useful indicators of zinc deficiency as one of the possible causes of reproductive dysfunction in ruminants.

Iron

In view of the relatively large requirements for iron and its occurrence in major quantities in blood and muscle as well as in numerous enzymes, it is arguable whether iron properly is considered a trace element. Nevertheless, iron traditionally has been included among the essential trace minerals.

While there is no evidence of iron deficiency ever in grazing sheep or cattle except as a result of disease or infestation, a number of investigators have drawn attention to correlations between the iron content of body fluids and fertility.

Iron, manganese, copper, and zinc were significantly higher in the sera of regular breeders than in repeat breeders (57). Correlations were high between iron and zinc content of blood and number of inseminations required per conception (51).

In a study of iron and copper in the accessory gland fluids and semen of bulls, concentration of iron was much greater in the spermatozoa than in the seminal plasma (21). The average concentration of iron in ampullary fluid and in semen was 1.08 and .61 mg/ml, whereas in seminal vesicular fluid and seminal plasma it was .38 and .35 mg/ml. However, no comparisons have been made to allow deductions as to differences related to altered reproductive function.

Conclusions

Dietary iron is an essential element in ruminants although its deficiency rarely has been observed in grazing cattle or ovine. Iron is abundant in all feeds, and a deficiency in adult ruminant seems improbable. However, in certain instances because of low availability of iron in some roughages, ruminant reproduction could be affected adversely by iron deficiency.

GENERAL REMARKS

Deficiencies of single trace elements rarely occur in the field; combinations of mineral deficiencies are much more common. The problem is complicated further by close interrelations between functions of many trace elements and various metabolic processes so that an imbalance in the intake of one element may change the requirement for others. For example, excess calcium added to nonlegume rations without added trace elements has caused interference with the estrous cycle and with ovulation in cattle; when alfalfa hay or trace elements were added to the diet, fertility was improved (95). A general elevation of the mineral content of the diet appears to have beneficial effects on fertility. By analysis of feedstuffs fed to cows and blood samples periodically over several months, trace elements of the feed were reflected in the blood content, and this in turn was paralleled with fertility (104).

This review emphasizes the need for adequately controlled experiments to determine whether trace-element malnutrition, in itself, impairs fertility. Little is known about the way in which malnutrition of trace elements affects the complex function of the reproductive system. All metals in this review are involved in enzymatic functions, and some hormonal activities appear to be correlated with trace elements. In future studies, therefore, it may prove useful to examine the hormonal status of cattle and sheep in the light of possible deficiencies in reproductive dysfunction. Improved assay techniques for hormones and minerals

could lead to a better understanding of the interrelationships between reproductive abnormalities and deficiencies of specific trace elements.

The relationship between iodine, hypothyroidism, and cystic ovaries in cows and between zinc and spermatogenesis are particular interactions which need to be studied in detail as does the role of selenium in ruminant fertility. Other problems inviting further study include investigation of the possible involvement of zinc intake in the seasonal quality of ram semen and the physiological role of manganese in spermatogenesis of rams.

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