

A Review of the Relationship Between Mineral Nutrition and Reproduction in Cattle

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Diet has a profound effect on growth, production, and reproduction in cattle (1, 2, 3). The more common nutritional problems encountered by the veterinarian are associated with insufficient consumption of total digestible nutrients and protein or depressed feed intake (2, 4, 5, 6). Although mineral requirements of cattle have been thoroughly investigated, the largest portion of this research has thus far dealt only with growth and production. The effects of dietary mineral intake on reproductive performance is a field which deserves additional investigation. When the nutritional plane does not meet the dietary requirements for the animal, the reproductive performance is compromised. Usually a decreased feed intake secondarily causes decreased mineral intake. The sparse information reporting the interrelationships between minerals and reproduction in the scientific literature is comprised primarily of case reports, clinical observations, and only a few controlled experiments.

Abnormal levels of the following mineral elements have been shown to be associated with decreased fertility or decreased reproductive performance: 1) calcium, 2) copper, 3) cobalt, 4) iodine, 5) manganese, 6) molybdenum, 7) phosphorus 8) selenium, and 9) zinc (Table 1).

Calcium and Phosphorus

Cattle fed calcium (Ca) deficient diets have delayed uterine involution (4). In such cases normal uterine involution occurred following the addition of Ca and Vitamin D to the diet (4). Decreased blood Ca concentration has been associated with an increased incidence of dystocia and retained placenta (4). Hypocalcemia has recently been associated with uterine prolapse in multiparous dairy cows (7). This may be due to delayed cervical and uterine involution during the hypocalcemic state (7, 8, 9).

Although several workers have demonstrated that excess dietary Ca intake decreased overall fertility in cattle (6, 10, 11, 12, 13), Littlejohn and Lewis failed to show this relationship (14). Hignett states that reproductive performance is inversely related to Ca:P ration, and directly related to dietary P intake (13). It has been observed that herds experience a depressed breeding efficiency when the

Ca:P ration is wide (13, 15, 16). Narrowing the Ca/P ration will usually improve the breeding efficiency (6, 12, 15, 16). Very high P intake and low Ca intake also results in infertility, which is reversed by the addition of Ca to the diet in normal proportions (12, 13). Hignett reports that in wide Ca:P feed ratios, affected cows have small corpora lutea, and "erect" uteri. When the Ca:P ration is extremely narrow, bordering on Ca deficiency, the corpora lutea may appear larger than normal or cystic and the uterus lacks proper tone (13). Russian workers reported optimum fertility occurred when the Ca:P ratio was between 1:1 and 3:1 with 3.5 to 3.9 gm P and 7 to 8 gm Ca/100 gm C.P. (17). Bulls fed rations with wide Ca:P ratios for prolonged periods of time may suffer testicular degeneration and sterility (6). Hignett suggested this is irreversible (6).

Carnahan stated that in cattle, the serum Ca:P ratio should be 2:1 (18). In cattle herds where the serum Ca:P ratio is not between 1.5:1 and 2.5:1, infertility may exist (18). Carnahan described two syndromes due to abnormal serum Ca:P ratio, one occurring if the ratio is $>2.5:1$ and the other when the ratio is $<1.5:1$. In beef herds where the serum Ca:P ratio was approximately 3:1, atonic uteri, small ovaries, decreased size of functional corpora lutea, and multiple small ovarian follicles were seen. This condition was correctible by supplementation. The other syndrome was seen in confined dairy cattle where the serum Ca:P ratio was very narrow ($<1.5:1$). Signs of narrow Ca:P ratio included: poorly involuted uteri, and increased incidence of retained placenta, luteal cysts, and pyometra. This condition was corrected by altering the dietary Ca:P intake to a more normal ratio (18).

The dietary Vitamin D concentrations, or its precursors, have been shown to greatly affect the Ca:P ratio's effect on reproduction (12, 13). Hignett suggested that when Vitamin D levels were high, fertility did not seem to be as dependant on P intake. When Vitamin D concentrations were low, an increase in dietary P was needed in order to maintain normal fertility (12, 13). Roberts reported that phosphorus is the most often deficient mineral in the diet of grazing cattle (19). Wagner reported P deficiency to be a cause of infertility in New York State dairy herds (5). Phosphorus deficient diets

may lead to pica, and decreased nutritional intake, which can contribute to depressed reproductive performance (1). Infertility due to P deficiency occurs after other signs of P deficiency are seen (i.e. poor hair coat, depraved appetite, and decreased appetite) (20). Phosphorus deficiency can cause irregular estrus, anestrus (4, 5, 20), decreased ovarian activity (21), depressed fertility.

In one study phosphorus responsive infertility became apparent at 0.16 to 0.20% P in the dry matter (DM) intake (23). Hignett suggested that cows of high fertility may have higher P requirements, than does the average cow (13). Low serum P concentrations are reportedly a good indicator of P deficient infertility (24). In instances where cattle were fed a deficient diet, an increase serum P was seen, along with a concurrent increased reproductive efficiency, when P was added to their diets (24). Morrow (25) described a dairy where P supplementation decreased services per conception from 3.7 to 1.3. This improved fertility was coupled with a rise in serum P from 3.9 mg/100 ml to 6.6 mg/100 ml. In one reported case, cows fed P deficient diets had calves only every 2 years, while heifers displayed signs of delayed onset of puberty (20). Adding 8 gms of P to the diet of beef cattle grazing P deficient Australian pastures, resulted in an improvement in fertility of those lactating (26).

The addition of disodium phosphate or other high P sources to diets deficient in P have been shown to increase the volume of both ejaculate and sperm output in adult bulls (17).

Manganese

Manganese (Mn) deficiency in cattle has been associated with silent estrus, infertility (13), abortion, and birth of calves with contracted tendons (4, 17). Wilson reported Mn responsive infertility in areas of the United Kingdom where forage Mn concentrations was <50 ppm. Common signs of Mn deficiency include anestrus, infertility, and cows having one or both ovaries of subnormal size (27). Annenakor (17) reported that when dietary Mn dropped below 10 mg/kg dry matter (D.M.) intake, increases in dystocia, infertility, and post-partum intervals occurred. Munro (28) found that supplementing cows fed forage containing 11-26 ppm Mn, with manganese sulfide solutions, increased first and second service conception rate from 48% to 72%.

Bentley and Phillips (27) stated a sterility problem existed in 10% of cattle where forage Mn fell below 20 ppm. They concluded that cattle fed a Mn deficient diet experience a delayed onset of first estrus, slightly reduced conception rate, and greater number of calves born with limb deformities. In a controlled experiment, Rojas and Dyer demonstrated that cows with low Mn intake (115 mg/day) had four services per conception versus two services/conception in cows on higher Mn diet (183 mg/day) (29).

Apparently, a relationship exists between the Ca:P ratio and Mn content of the diet and reproduction (10, 13, 15, 23). McClure, using data collected in Australia and New

Zealand, reported the tendency of excess dietary Ca to depress Mn utilization by the animal (11). With a deficient Mn intake and a normal Ca:P ratio, a high conception rate is retained, but if the Ca:P ratio is shifted to the right a decreased conception rate occurs, which is responsive to Mn (10, 15). High liming rates have been shown to depress Mn uptake by forages; and Mn is considered a sensitive index of soil pH (23). Russian workers reported bulls fed Mn deficient diets (<10 mg/kg D.M.) had degenerative testicles and depressed spermatogenesis (17, 30). These abnormalities were responsive to supplemental Mn (17, 30).

Molybdenum, Copper and Cobalt

Molybdenum (Mo) toxicity has been shown to be difficult to differentiate from Copper (Cu) deficiency (31, 32), and it has long been suspected that a close interrelationship between these two minerals exists (11, 23). Cattle grazing high Mo and low Cu pastures reportedly have delayed onset of puberty and experience anestrus (20). Copper deficiency in cattle has been associated with irregular estrous periods, anestrus, and depressed fertility (10, 17, 20, 33, 34). These abnormalities are responsive to orally administered Cu salts (10, 11, 17, 33). Aberrations in reproductive performance has been reflected in lowered blood Cu concentrations (23, 28), which is responsive to the addition of Cu to the diet (23). The Cu responsive anemia is not always a consistent finding in Cu deficient infertile herds (34).

Thomas and Moss (32) fed two holstein bull calves for 129 days (until age 217 days) a diet high in Mo (263 to 411 ppm Mo). The calves displayed a lack of libido, and had marked degenerative changes in their seminiferous tubules which were devoid of spermatids (32). Adult bulls fed less than 5 mg of Cu/kg feed had degeneration of testicular germinal epithelium (17).

An interrelationship between Co and Cu has been reported (23). Alderman reported on a dairy herd in the U.K. with a first service conception rate of 53%. The conception rate was increased to 67% by Cu injections and 93% by a combination of Cu injections and Co supplementation (23). Cobalt and/or Cu deficiencies have been associated with anemia (11), which could indirectly result in infertility of both bulls and cows (20). This has been manifested in cattle with estrus failure and delayed onset of puberty (20). Non-functional ovaries have been reported in New York State dairies where Co-deficiency anemia exists (5). This Co-deficiency reportedly results in abortion, birth of weak calves, and infertility (23). Wagner reported a case of a Co-deficient dairy herd, with a high incidence of silent heat. The affected cows had lowered hemoglobin concentrations (5). This problem of silent estrus was alleviated by the addition of Co supplements to the diet (5).

Iodine

Toxic iodine (I) intake has been associated with abortion (4), and calf deformities (35). In one case report of accidental

toxic I supplementation, abortions occurred during the first trimester of gestation (4). Deficient dietary I intake has been associated with impaired ovarian activity (4), anestrus (6) hairlessness, still-births, weak calves (4, 6, 17, 20, 36, 37), depressed libido in bulls (6, 22, 36), increased incidence of retained placenta (6, 38), lowered first service conception rates (38), embryonic mortality (23, 38) and infertility (10, 17) through its effect on the thyroid gland (4, 20, 36). Moberg reversed many of these signs (poor conception rates, retained placenta, and irregular breeding intervals) by the addition of supplemental I in diets of Finnish dairy herds (5). Decreased gonadotropic output by the anterior pituitary can occur with hypothyroidism (20).

Allcroft, et. al. reported a dairy herd in the U.K that had low plasma protein bound I and pathologic changes in fetal thyroids. This herd also had a high incidence of abortions, still-births, and weak calves. This condition was corrected by I supplementation (36). Goitrous calves have been born to I deficient dams (20, 22). Iodine supplementation increases the reproductive performance of cattle grazing I deficient pastures (17, 10). It has been reported that grazing beef cattle are more likely to experience I responsive infertility if they are not fed supplements (11).

Selenium

The interrelationship between Selenium (Se) and Vitamin E deficiency has been reviewed (39). Retained fetal membranes appears to be the most common sign noted in Se deficiency in cattle (4, 20, 37, 40, 41, 42). Abortion, or the birth of weak, premature or dead calves have also been reported as a consequence of Se deficiency (20, 37). These signs were shown to be responsive to Vitamin E and Se injections (37).

Trinder, et al. (42) demonstrated that an injection of Vitamin E and potassium selenite one month pre-partum reduced the incidence of retained placenta in dairy cows. They recorded a high incidence of retained placenta associated with forages containing <0.05 ppm Se. When a herd was divided into two groups, 42% of the non-treated controls retained their fetal membranes and none of the cows given 15 mg Se and 680 I.U. Vitamin E. Selenium injection alone only partially decreased the incidence of placental retention. Similar conclusions were reached on Michigan dairy farms (40, 41). Researchers reduced the incidence of retained placenta by including high concentrations of Se in the diet or by injections of Vitamin E and Se. Cows receiving 0.02 ppm Se in this diet had a 50% incidence of retained placenta versus 20% in cows ingesting 0.065 ppm (40). Raising selenium supplementations from .02-.05 ppm to .07 ppm Se reduced the incidence of placental retention from 38% to 0% (41).

Fifty mg of Se as selenite and 680 I.U. Vitamin E given 21 days pre-partum, or inclusion of .92 mg Se during the last few days of the dry period, decreased the incidence of retained placenta. In a herd of 193 dairy cows on a Se

FIGURE 1. Reproductive disorders due to abnormal mineral intake.

MINERAL	SIGNS	REFERENCE
Ca Deficiency	delayed uterine involution	4,13,18
	small corpora lutea	13
	cystic ovaries	13,18
	retained placenta	18
Ca Excess	decreased breeding efficiency	6,10,11,12,13
	testicular degeneration	6
P Deficiency	decreased breeding efficiency	13,15,16
	testicular degeneration	6
	decreased ovarian activity	21
	small corpora lutea	18
	anestrus	4,5,20
	weak or still born calves	20
	delayed onset of puberty	20
	depressed fertility	5,6,10,20,21,22,23,21
Mn Deficiency	anestrus	13,27
	infertility	13,27,29
	abortion	4,17
	calves born with contracted tendons	4,17
	small ovaries	27
	dystocia	17
Mo Excess	delayed onset of puberty	20
	anestrus	20
Cu Deficiency	anestrus	10,17,20,33,34
	depressed fertility	10,17,20,33,34
	depressed libido in bulls	32
	testicular degeneration	32
Co Deficiency	infertility of both sexes	20,23
	delayed onset of puberty	20
	nonfunctional ovaries	5
	abortion	23
	birth of weak calves	23
I Deficiency	impaired ovarian activity	4
	anestrus	6
	infertility	5,10,11,17,38
	retained placenta	5,6,38
	embryonic mortality	23,38
	birth of hairless calves	4,6,17,20,36,37
	birth of weak calves	4,6,17,20,36,37
	still births	4,6,17,20,36,37
	depressed libido in bulls	6,22,38
I Excess	abortion	4,35
	calves born with limb deformities	35
Se Deficiency	retained placenta	4,20,37,40,41,42
	abortion	20,37
	birth of weak or dead calves	20,37
Zn Deficiency	cystic ovaries	22
	abnormal estrus	22
	delayed testicular development	4
	small testicles	17,43,44
	testicular atrophy	17,30

deficient diet, the incidence of retained fetal membranes dropped from 51% in controls to 8.8% in those injected with 50 mg sodium selenite and 680 I.U. of Tocopherol (41).

Zinc

Several studies have concluded that Zinc (Zn) deficiencies resulted in reduced testicular size in growing bull calves (17, 43). Testicular size improved when the Zn deficiency was alleviated (43, 44). Although delayed testicular development in young bulls (4) and testicular atrophy in adult bulls (17, 30) has been described, one study demonstrated no irreversible effects on semen characteristics or quality (44). Decreased conception rates (30), cystic ovaries, and abnormal estrus have been reported in cows with Zn deficiency induced parakeratosis (22).

Conclusions

As discussed above, dietary mineral imbalances or deficiencies have been shown to play a role in decreased reproductive performance in cattle. When faced with a herd reproductive problem, the possibility of a mineral deficiency, mineral excess, or a mineral imbalance should be investigated. An analysis of pasture, hay, silage, grains, any supplemental feeds, and salt should be conducted. Steps which could be taken to alleviate a nutritional deficiency might include overall feeding changes or simply changing mineral supplements. Reproductive problems in cattle herds always present a diagnostic challenge to the clinician. Nutritionally related problems should be considered in a list of possible etiologies, and if present, corrected. Figure 1 is a summation of the clinical signs associated with infertility seen when the above minerals are deficient, excessive, or imbalanced in the diet.

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