Effects of Nutrition on Reproduction (Fertility and Infertility) of Dairy and Beef Cattle

P. Miettinen
Department of Anatomy
University of Kuopio
Finland

Introduction

Infertility problems related to nutrition may be primary or secondary. An absolute deficiency or excess of a particular nutrient can affect reproductive performance in cows. However, most problems are associated with mismanagement of macronutrients, energy and protein. In addition, excess feeding before calving is associated with periparturient diseases that result in low fertility. After calving in dairy cows, energy deficiency as a result of limited capacity of feed intake greatly impairs fertility, whereas in beef cattle the quality rather than the quantity of feeding plays a major role.

Fertility Parameters

The importance of the herd manager for fertility is well known, and many other factors involved in cattle fertility must be excluded when nutritional factors affecting fertility are evaluated, e.g. conception rate at first insemination or non-return rate are widely used in the large field surveys. However, both measures of fertility in cows are affected by management decisions. Too early insemination results in a low pregnancy rate. On the other hand, a high conception rate can be obtained when the cows are not inseminated in uncertain estrus, but the number of days open might increase. In experimental studies, due to a low number of animals, the difference in conception rate might not be statistically significant, although the numerical difference is great. Moreover, the interval from calving to last insemination as a parameter of fertility should be avoided, since many cows are inseminated early, but are culled as non-pregnant. In addition, comparing fertility of herds with fertility of cows may skew the interpretation. For example, calving interval is longer in high yielding cows than in low yielding cows, but the average of calving intervals might be shorter in high yielding herds than in low yielding ones.

Energy Metabolism of Cows

Glucose plays a central role in the energy metabolism of dairy cows. High-yielding cows are unable to consume sufficiently food to meet their production and maintenance requirements in early lactation. Body fat is mobilized when the energy requirement exceeds the energy intake, resulting in shifts of energy metabolism. Since the liver has a limited ability to oxidize fatty acids during energy deficiency, excess of acetyl-CoA is converted into ketone bodies, acetoacetate, ß-hydroxybutyrate and acetone. Nevertheless, ketone bodies are not only waste, since they can replace carbohydrate as an aerobic fuel of muscular exercise. However, in contrast to other mammalians, cows cannot utilize ketone bodies in the brain. During the first days postpartum, carbohydrate, especially carbohydrate from muscle and liver glycogen, is utilized as an energy source and can balance the blood glucose concentration. In these stores, however, glycogen is a limiting factor for the metabolism, because it is present only in small amounts. In addition, glucose serves as a precursor for ascorbic acid, that is essential for optimal steroid hormone function.[1]

Fertility in Dairy and Beef Cattle

Postpartum bovine fertility is based primarily on two processes: involution of the uterus and the re-establishment of the estrous cycle. In cows, release of the fetal membranes occurs 3 to 8 hours after fetal expulsion, and within 3-5 weeks postpartum the uterus has returned to its pregravid size.[2] Unimpeded involution is a prerequisite for successful conception when insemination takes place soon after the end of the clinical puerperium.

Obviously many factors may influence the involution process, including the breed of cattle, parity, and postpartum diseases such as milk fever, dystocia and

Paper presented at the XVIII World Buiatrics Congress, Bologna, Italy August 29 - September 2, 1994

retained fetal membranes. Moreover, ovarian dysfunctions are more frequent after involutional disturbances.

Feeding Before Calving

Late lactation and the dry period is an important preparatory period which affects health and reproductive performance of cows in the next lactation. In many studies inadequate feeding before calving has increased the occurrence of periparturient diseases, e.g., cows with a fat body condition had up to 7-8 weeks postpartum (p.p.) higher levels of acetone in milk than cows with thin or moderate body condition at dry-up.[3] Moreover, excess feeding of protein has a detrimental effect on liver function. Nutritional deficiencies or imbalances have multiple manifestations, and a clinical disorder might have multifactorial etiologies. Puerperal disorders often are interrelated, thus one disease increases the risk for another disease. It is evident that periparturient diseases result in low fertility and increased rate of culling.

Although de Kruif and Mijten concluded in a recent review that feeding during late lactation and dry period does not adversely affect subsequent reproductive performance,[4] excess feeding before calving has been found to be associated with delayed involution of the uterus, disturbed ovarian activity and with long interval from calving to conception. For example, high levels of urea at dry-off was associated with delayed onset of ovarian activity.[3]

Periparturient Diseases

Retained placenta

No precise mechanism has yet been described which could account for nutrition induced retained placenta in cows, but evidently nutrition plays a major role; for example ketonaemia at calving,[5] metabolic imbalance before calving[6] and grass silage feeding[7] have been related to retained placenta which is a frequent predisposing factor for puerperal metritis or delayed uterine involution. Retained placenta has thus been found to be associated with poor fertility. Retained placenta is not an immediate cause of low fertility, but retained placenta is often associated with uterine diseases which have severe adverse effects on fertility.[8]

Among the mechanisms proposed for retained placenta is a disturbed synthesis of PgFα in the placentomas.[9] Consequently, retained placenta may be explained by a disturbance of prostaglandin metabolism in the placentomas and thus a connection with nutrition through PgF2α has been supported.[7] In field conditions, Barnouin and Chassagne found a high rate between high retained placenta in herds and feeding on rations high in grass silage with few cereals at the end of gestation.[7] The plasma level of PGFM, (main PgF2α metabolite) was lower in cows suffering retained placentas than in cows without retained placentas.[10]

A ration rich in grass silage is characterized by a rich linolenate fraction and a poor linoleate one. Linoleic acid is the alimentary precursor and linolenic acid an inhibitor of PagF2α synthesis via arachidonic acid.[11] Thus a high dietary linolenate/linoleate ratio would be unfavorable to the synthesis of the Pg2 and other arachidonic acid derivatives.

The etiological mechanism proposed for retained placenta via the dietary polyunsaturated fatty acid balance is consistent with the reported higher risk of retained placenta in the cow through a nutritional Se/vitamin E deficiency.[12] As antioxidants, they control the auto-oxidation of polyunsaturated fatty acids in the tissue. So a beneficial effect of antioxidant is exerted in the production of prostanoids while an uncontrolled peroxidation of polyunsaturated fatty acids through a vitamin deficiency results in tissue damage, alterations in prostaglandin balance and retained placenta.

Ketosis

After calving, obese cows eat less than cows with moderate or thin body condition, and more fat is mobilized for energy production, and thus fat cows especially tend to lose weight after calving. Clinical ketosis, which is a temporary energy imbalance, typically occurs spontaneously in susceptible high-yielding dairy cows between the 2nd and 7th week of lactation. Differences in the severity of ketosis might be one explanation for conflicting reports on the possible interactions between bovine ketosis and reproductive performance in dairy cattle. The low energy balance associated with ketosis causes a delay in the onset of ovarian activity, thus curtailing fertility. In addition, the adverse influence of ketone bodies on fertility depends on duration and timing of their increased levels; the longer and later the cow has ketone bodies, the lower is the fertility.[3,13]

Disturbed ovarian activity, delayed breeding and prolonged interval from calving to conception has been found to be associated with ketosis. A high incidence of ketosis has been correlated with a high incidence of infertility in herd level.

Fatty liver

A serious consequence of mobilization of body reserves is the development of fatty liver that is a part of the general fat mobilization syndrome, which is not specific to any organ or tissue. Fatty liver has many features in common with ketosis. Both involve a negative energy balance in early lactation. Fatty liver, however, is actually due to excessive mobilization of fat from the adipose tissue. Deposition of fat in the liver can occur before calving due to the mobilization of body fat. The mobilization of free fatty acids from adipose tissue reaches its maximum one week after calving, and infiltration of lipids in the liver 2-4 weeks postpartum, however, ketone bodies reach their maximum 3-4 weeks p.p.[14] Fatty liver and postpartum loss of body weight have been found to be associated with curtailed fertility.

Postpartum Energy Balance and Subsequent Fertility

Ovarian activity

A low energy level in early lactation depresses ovarian activity. Thus, low plasma glucose[13] and loss of body weight in the first three weeks p.p. are associated with delayed initiation of ovarian activity.[15] The earlier the ovarian activity begins (high energy balance) the higher is fertility. However, not only the initiation of ovarian activity but also the regularity of ovarian function and the level of luteal function play an important role, that are influenced by energy balance. Low conditioning cows indicating low energy balance, have fewer luteinizing hormone pulses and secretion of progesterone is reduced. Thus low level of progesterone secretion has been related to low fertility.

In dairy cattle a major problem is meeting the energy demand during the first weeks postpartum. Since overfeeding before calving causes diminished appetite after calving, the obesity at calving is a predisposing factor for low energy balance in early lactation and also for delayed onset of ovarian activity. In addition, excess feeding of protein in late lactation is associated with low energy balance in the next lactation.[3] Moreover, metritis, abomasal displacement, mastitis, traumatic reticuloperitonitis and foot diseases may be associated with poor appetite.

The degree of energy deficiency in early lactation should be as low as possible. Ketone bodies are widely used as an indicator of energy balance, however, due to the origin they can indicate only energy deficiency. On the other hand ketonaemia reduces milk production[16,17] and the energy and metabolic balance might equilibrate more rapidly before breeding time in cows with moderate hyperketonaemia and they could have high fertility, but low milk production.

Energy balance and conception

Degree, duration and timing of low energy balance influence the subsequent fertility. The later a cow reaches energy balance after calving the lower the fertility.[3,13,15,18]

Too early insemination results in a low rate of pregnancy.[3] The later a cow is inseminated the longer time the metabolic balance has to equilibrate and pregnancy rate can increase. Thus high energy balance, reflecting also as a gaining in body weight at the time of service, improves fertility.[15] Although about 10% of Finnish dairy cows are inseminated during the luteal phase, it is doubtful that metabolic balance affects only detection of estrus; moreover, all steps leading to ovulation, formation of corpus luteum and embryonic development may be improved by an adequate energy balance.

Effect of roughage

A detrimental effect of low roughage rations on fertility has been observed in many studies. However, the primary reason of low fertility is still unknown. Excess lactic acid, produced too rapidly, may temporarily exceed the capacity of the rumen to neutralize the acidity, resulting in indigestion and metabolic acidosis. Holtenius found a higher incidence of hyperketonaemia in cows fed with a high amount of concentrate (65%) than in cows fed with a moderate amount of concentrate (50%)[19] thus the low fertility could be due to the low energy balance reflected as a ketonaemia. A frequent feeding of a high amount of concentrate can stabilize rumen fermentation[20] and improve the utilization of energy.[21]

Nitrogen balance and fertility

In many studies excessive amounts of protein and high levels of urea have been found to be related to impaired fertility. However, in a recent review, Ferguson found that only conception rate was impaired by excess of rumen degradable protein, but not the number of days open.[22]

The balance between energy and nitrogen is important for high fertility (Table 1).

Table 1. Effect of metabolic balance two months postpartum on fertility.

Glucose (mmol/l)	Urea (mmol/l)	AI (days)	CC (days)	PR (%)	AIn (n)
<2.53	2.96	82.8±23.4	103.7±31.4	62	1.69
2.53<2.90	4.52	79.7±16.7	121.6±51.6	44	2.33
2.90<3.30	3.71	76.5±12.0	92.9±24.9	50	1.50
3.30≤	3.07	69.1±8.8	86.2±29.6	62	1.38

Thus, adequate nitrogen/energy balance is most important for high fertility. The negative association between protein intake, urea levels and fertility during the first two months after calving should be interpreted carefully:

1. by isocaloric feeding due to the limited feed intake, the protein intake does not meet the requirements,[23]
2. reduction in the first service conception rate as a result of low energy level is observed within 60 days of calving,[3]
3. in high yielding herds, the cows might be rebred

earlier than the herdmates in low yielding herds resulting in a low conception rate.
4. the urea content in milk varies widely during puerperium due to the changes in ration offered or consumed, and due to the type of feeding.[24]

Milk production and reproduction

In dairy cows, the selection for milk yield has changed endocrine pattern of metabolic hormones.[25] The directed flow of nutrients to lactation is controlled through homeorhetic mechanism.[26] On the other hand, for regulation of homeostasis, it is necessary that milk yield can be reduced when there is an energy deficiency. This ability to reduce milk yield may play a major role in the susceptibility of cows to clinical disease and to rapid recovery; therefore, e.g., a ketotic cow can recover without medical treatment if the milk yield is reduced.

There is no doubt that milk production (= energy output) plays a major role in infertility. Milk production leads to a high risk for energy deficiency, however, the most important factor affecting energy balance is feed intake.[27] Although the milk production is lower in beef cattle, the quality and quantity of feeding also might be lower than in dairy cattle. Thus energy level plays a central role in beef cattle also.

Controversy exists concerning the relationship between milk yield and reproductive performance. High milk yield has been related to anestrus, low conception rate and days open, whereas Harrison suggested that high milk yield is associated with suppression of estrous behavior rather than with a delayed interval to ovarian cyclicity.[28] Moreover, anestrous cows are culled at a higher rate than their herdmates.[29] In addition, there might be a negative association between high yield and reproductive efficiency in individual cows, whereas herds with higher average milk yields had shorter intervals to first insemination and to conception.

Indeed, the low energy balance in puerperium as a result of high milk production curtails fertility, thus the milk production *per se* is not the causative factor. If we attempt to reach high milk production together with good health and fertility, we must simultaneously improve environmental conditions and housing. Although high milk production might be associated with impaired fertility, we can allow a longer interval from calving to pregnancy for high yielding cows than for low yielding ones since the farmer is paid for the milk yield.

Effect of Season

It is well-known that there are seasonal variations in fertility. However, the casual factors are poorly known. The long period of natural light stimulates feed intake, and during exercise more fatty acids and ketone bodies can be used as a fuel which decreases the level of ketone bodies. Gain in body weight, which depended on season, was associated with subsequent improved reproductive performance.[30] Indeed, the effect of season on fertility is due to a better metabolic balance of cows on pasture.[31] The positive effect of pasture, in turn, may be due to exercise and better nutritional quality and quantity.

Monitoring Energy Balance in Field Practice

Many biochemical parameters (e.g., glucose, urea, ketone bodies, enzyme, bilirubin, albumin) and physical parameters (body condition score, body weight) have been used to indicate the energy status of cows, and have been found to be associated with low fertility. On the other hand, many times such a correlation has not been found.

Since dairy cows are energy deficient in early lactation, a high body reverse of glycogen at calving would be of great importance to meet the excessive demand for milk production. The rate at which fat is mobilized and used as fuel in the citric acid cycle might be crucial. Concerning the pre-calving degree of body fat reserve, the optimal amount may vary due to the farming system. Since more fatty acids can be used as fuel during exercise, cows in loose stalls might be able to mobilize more fat and can have more fat at calving.

Indeed, to prevent energy deficiency in early lactation a cow should have a high amount of glycogen in muscle and in the liver, and should be able to regulate the utilization of fat.

Summary

An absolute deficiency or excess of a particular nutrient can affect reproductive performance in cows. However, most problems are associated with mismanagement of macronutrients, energy and protein. In addition, excess feeding before calving is associated with periparturient diseases that result in low fertility. After calving, an energy deficiency as a result of high milk production impairs ovarian activity and conception.

References

1. Goralczyk, R., U.K. Moser, U. Matter & H. Weiser: 1992 *Ann. N.Y. Acad. Sci.*, 669, 349. 2. Miettinen, P.V.A.: 1990 *Acta Vet. Scand.*, 31, 181. 3. Miettinen, P.V.A., V.A. Rainio, S.A. Kukkonen & J.J. Setälä: 1992 Fertilitat., 8, 54. 4. De Kruif, A. & P. Mijten: 1992 Berl. Münch. tierärztl. Wschr., 105, 271. 5. Grunert, E.: 1983 Wien. tierärztl. Mschr., 70, 230. 6. Lotthammer, K.-H.: 1983 Dtsche tierärztl. Wschr., 90, 427. 7. Barnouin, J. & M. Chassagne: 1990 Prev. Vet. Med., 8, 231. 8. Pelissier, C.L.: 1976 *Theriogenology.*, 6, 575. 9. Leidl, W., D. Hegner & P. Rockel: 1980 Zbl. *Vet. Med. A.*, 27, 691. 10. Chassagne, M. & J. Barnouin: 1992 *Theriogenology.*, 38, 407. 11. Holman, R.T.: 1986 Prog. Lipid Res., 25, 29. 12. Trinder, N., C.D. Woodhouse & C.P. Renton: 1969 *Vet. Rec.*, 85,

550. 13. Miettinen, P.V.A.: 1990 *J. Vet. Med. A.*, 37, 417. 14. Furll, M., W. Deckert, M. Schäfer & A. Wehlitz: 1992 *Mh. Vet. Med.*, 47, 119. 15. Miettinen, P.: 1994 *Repr. Domest. Anim.*, 29, 121. 16. Miettinen, P.V.A. & J.J. Setälä: 1993 *Prev. Vet. Med.*, 17, 1. 17. Miettinen, P.V.A.: *J. Vet. A* (in press) 18. Miettinen, P.V.A.: 1991 *Acta. Vet. Scand.*, 32, 189. 19. Holtenius, P., G. Olsson & C. Björkman: 1993 *J. Vet. Med. A.*, 40, 118. 20. Kaufmann, W.: 1973 Kieler Milchwirtsch. Forschungsber., 25, 245. 21. Nielsen, T.K. & K. Jensen: 191 *Acta Vet. Scand.*, 32, 301. 22. Ferguson, J.D. & W. Chalupa: 1989 *J. Dairy Sci.*, 72, 746. 23. Miettinen, P.V.A.: 1992 Tierärztl. Prax., 20, 149. 24. Miettinen, P.V.A. & R.O. Juvonen: 1990 *Acta. Agri. Scand.*, 40, 289. 25. Bonczek, R.R., C.W. Young, J.E. Wheaton & K.P. Miller: 1988 *J. Dairy Sci.*, 71, 2470. 26. Bauman, D.E. & W.B. Carrie: 1980 *J. Dairy Sci.*, 63, 1514. 27. Villa-Godoy, A., T.L. Hughes, R.S. Emery, L.T. Chapin & R.L. Fogwell: 1988 *J. Dairy Sci.*, 71, 1063. 28. Harrison, R.O., J.W. Young, A.E. Freeman & S.P. Ford: 1989 *Anim. Prod.*, 49, 23. 29. Bartlett. P.C., J.H. Kirk & E.C. Mather: 1986 *Theriogenology.*, 26, 309. 30. Agyemang, K., D.A. Little, M.L. Bah & R.H. Dwinger: 1991 *Anim. Reprod. Sci.*, 26, 51. 31. Miettinen, P.V.A., V.A. Rainio, S. A. Kukkonen & J.J. Setälä: 1991 *J. Vet. Med. A.*, 38, 427.

Abstracts

Diagnosis of bovine fetal *Neospora* infection with an indirect fluorescent antibody test

B.C. Barr, M.L. Anderson, K.W. Sverlow, P.A. Conrad
The Veterinary Record, 1995 137: 611-613

Fetal fluids from 138 spontaneously aborted bovine fetuses were examined for the presence of antibodies against *Neospora* antigens by means of an indirect fluorescent antibody test (IFAT). The fetuses were divided into group 1, consisting of 74 fetuses with confirmed or presumptive fetal neosporosis, and group 2, consisting of 64 fetuses with either no aetiological diagnosis, presumptive diagnoses of non-*Neospora* infections or non-infectious diseases, or fetuses with confirmed diagnoses of other fetal diseases. Thirty-seven of the 74 fetuses in group 1 had detectable titres of antibody to *Neospora*; approximately 21 per cent of the fetuses between three and five months gestation, 56 per cent of these between six and seven months gestation, and 93 per cent of these between eight and nine months gestation, had detectable titres of antibody. Only one of the 64 fetuses in group 2 had a detectable titre of antibody to *Neospora*.

Application of real-time ultrasonography for the detection of tarsal vein thrombosis in cattle

J. Kofler, A. Buchner, A. Sendlhofer
Veterinary Record (1996) 138, 34-38

The clinical and ultrasonographic features of the thrombosis of three tarsal veins in a six-year old dairy cow are described. Thrombosis and metastatic abscessation developed on the left tarsus six days after the amputation of the lateral claw of the left hindlimb. Originally, the cow suffered from a severe purulent arthritis of the distal interphalangeal joint and a retroarticular abscess, caused by interdigital necrobacillosis. By means of real-time ultrasonography, using a 7.5 MHz transducer, a marked subcutaneous oedema, a subcutaneous abscess and a thrombosis of the ramus cranialis and ramus caudalis of the vena saphena lateralis reaching the confluence into the vena saphena lateralis and a thrombosis of the ramus caudalis of the vena saphena medialis could be identified. The thrombosed veins were not compressible, were oval and had an increased diameter of up to 9 x 12 mm. Intraluminal masses were visualized as hypoechoic structures and the veins distal to the thrombosis were distended up to 10 x 13 mm. The differential diagnosis and pathogenesis of the thrombosis and the abscessation are discussed, and the clinical course and the sonographic observations of the thrombosis during a six week period are described.