Micronutrients and reproduction in farm animals

O.B. Smith, O.O. Akinbamijo

Abstract

Reproductive well-being and performance of farm animals is largely dependent on their nutritional status, which is often less than optimum in developing tropical countries. More often than not, they are malnourished, particularly with regards to micronutrients. Evidence was presented to show that because these micronutrients are involved in such functions as intracellular detoxification of free radicals, synthesis of reproductive steroids and other hormones, carbohydrate and protein and nucleic acid metabolism, their deficiencies and/or excesses may impair spermatogenesis and libido in the male, fertility, embryonic development and survival, post-partum recovery activities, milk production and offspring development and survival. A plea was made for intensified research efforts, farmer education and quality control of vitamin–mineral pre-mixes, in order to improve micronutrient nutrition, and, consequently, the reproductive performance and overall productivity of farm animals in developing tropical countries. © 2000 Elsevier Science B.V. All rights reserved.

Keywords: Reproduction; Vitamins; Microminerals

1. Introduction

Reproductive performance of livestock in the tropics as elsewhere is determined by four factors — genetic merit, physical environment, nutrition and management. Evidence from the literature and practical experiences suggests that nutritional factors are perhaps the most crucial, in terms of their direct effects on the reproductive phenomenon, and the potential to moderate the effects of other factors. Thus, adequate nutrition could encourage mediocre biological types to reach their genetic potential, alleviate the negative effects of a harsh physical environment, and minimise the effects...
of poor management techniques. Poor nutrition on the other hand, will not only reduce performance below genetic potential, but also exacerbate detrimental environmental effects. Moreover, nutritional factors more than all others, readily lend themselves to manipulations to ensure positive outcomes. Hence, there is a need to pay particular attention to the interactions between nutrition and reproduction particularly in the tropics, where, for a variety of reasons, nutritional inadequacies in terms of quantitative feed intake and qualitative nutrient imbalances remain prevalent. Failure to properly understand these interactions in order to minimize the negative, and enhance the positive impacts will adversely affect livestock production efficiency, since this depends largely on reproductive performance.

Several studies (Hamra and Bryant, 1982; Kirkwood et al., 1987; Manspeaker et al., 1989) and reviews (den Hartog and van Kempen, 1980; Short and Adams, 1988; Smith and Somade, 1994) have adequately examined the effects of quantitative feed and energy, as well as qualitative protein and macronutrients intake on livestock reproductive performance. In general, the results of such studies suggest that poor nutrition caused by inadequate, excess or imbalanced nutrient intake may adversely affect the various stages of the reproductive event, going from delayed puberty, reduced ovulation and lower conception rates, through high embryonic and foetal losses to excessively long post-partum anoestrus, poor lactation, high perinatal mortality and poor neonatal performance. It is only recently that attention has also been paid to similar potential effects of micronutrients intake, those elements needed in relatively minute amounts in the diet (< 1 ppm) and by the body (< 1%). This paper will therefore focus primarily on a review of the role and effects of vitamin A, vitamin E, selenium, copper, molybdenum and zinc, on reproductive events in farm animals.

2. Mechanisms of nutrients mediated impact on reproduction

In a recent review of studies on nutrition–reproduction interactions, Smith and Somade (1994) reported that several of the studies reviewed were, more often than not, contradictory in terms of the magnitude or even the direction (positive or negative) of nutritional effects on reproduction. They suggested that a better comprehension of the underlying mechanism of these interactions may shed some light on these variable and contradictory observations. The mechanisms are still not fully understood, because of the complexity of the neuro-hormonal dialogue and the equally complex nutrients partitioning involved. Nevertheless, some advances have been made, and interesting possibilities put forward. Some of these nutrient mediated effects act directly on the gonads and other reproductive organs, while others produce similar effects indirectly via the hypophyseal–pituitary–gonadal axis.

According to Gutteridge and Halliwell (1994), oxidative stress occurs at the cellular level, when reactive metabolites of oxygen are produced faster than they can be safely removed by antioxidant defence mechanisms. These reactive oxygen species are produced during normal metabolism, and can accumulate rapidly in actively reproducing cells. Vitamin E functions as an intra-cellular antioxidant scavenging for free reactive oxygen and lipid hydroperoxides, and converting them to non-reactive forms, thus
maintaining the integrity of membrane phospholipids against oxidative damage and peroxidation (Surai, 1999). Selenium, on the other hand, functions as cofactor of the glutathione peroxidase (GSH-Px) enzyme systems responsible for regulating extra and intra-cellular hydroperoxidase (Burk and Hill, 1993). In vitamin E and selenium deficiency conditions, these free radicals accumulate and not only damage cell membranes, but also disrupt several processes linked to the synthesis of steroids (Staats et al., 1988), prostaglandins (Hemler and Lands, 1980), sperm motility (Alvarez and Storey, 1989), and the development of the embryo (Goto et al., 1992).

It is not surprising therefore that negative impacts of vitamin E/selenium deficiencies have been observed on various components of the reproductive event, including ovulation rate (Harrison et al., 1984), uterine motility, sperm motility and transport (Segerson and Libby, 1982; McKenzie et al., 1998), conception rate and post-partum activities (Arechiga et al., 1994), foetal membrane expulsion (Wichtell et al., 1996), embryo survival, milk production and postnatal growth (Anke et al., 1989). Moreover, evidence exists that beta-carotene, vitamin A precursor, manganese and zinc are involved in steroidogenesis (Hurley and Doane, 1989; Corah and Ives, 1991). Their deficiencies may therefore directly impair ovarian activities or indirectly through a breakdown of the hypothalamo–pituitary feedback mechanism.

It is worth noting that although the emphasis in the literature is mainly on the negative impacts of deficiencies, excesses and/or imbalances of micronutrients may also result in reproductive disorders. For example, during the pre-pubertal stage of maturity, the episodic release of luteinizing hormone, responsible for follicular growth and the establishment of the ovarian cycle, is apparently inhibited by ovarian oestradiol, and a reduction of this inhibition and the accompanying release of the luteinizing hormone marks the passage into adulthood. Evidence exists (Wise and Ferrell, 1984; Phillipo et al., 1987) that higher-than-required dietary levels of molybdenum affect the frequency of luteinizing hormone episodes; hence, the well-known effect of delayed puberty of excess dietary molybdenum. It is through such and similar mechanisms that the effects of the micronutrients reviewed below are mediated on reproductive events in farm animals.

3. Vitamin A

Livestock, particularly ruminants, consume vitamin A, mainly in its inactive form — the carotenes or Provitamin A, except when it is fed as a supplement in cereal based concentrates. Provitamin A is converted into active vitamin A in the small intestines and together with preformed vitamin A supplement is stored in the liver, muscle, eggs, and milk to be used for a variety of functions, including those linked to the reproductive phenomena. Reproductive disorders observed with vitamin A deficiency in farm animals include delayed puberty, low conception rate, high embryo mortality, high perinatal mortality resulting from weak, blind offspring, and reduced libido in the male (Smith and Somade, 1994).

Litter size at birth and at weaning is an important economic index in multiparous species like the pig, and evidence exists that vitamin A has a positive effect on litter size
(Coffey and Britt, 1993; Whaley et al., 1997). In a recent study, Whaley et al. (1997) examined the mechanisms through which vitamin A supplementation improves litter size in pigs. The experimental model used in the study, involved feeding a high-energy diet known to reduce embryo survival (den Hartog and van Kempen, 1980), and then supplementing with vitamin A, to evaluate its corrective effect. Two groups of pre-pubertal gilts were fed a high- or low-energy diet from the 7th day of a second oestrus cycle. Fifteen days after the second oestrus cycle, each group was subdivided into two subgroups, and injected with either corn oil or vitamin A in corn oil, to constitute four dietary treatments: high energy + vitamin A (HEA), high energy (HE), standard energy + vitamin A (SEA), and standard energy (SE).

The authors reported that: (a) the HE diet, as expected, reduced the number of embryos recovered, and the embryo survival rates, while vitamin A supplementation (HEA) corrected this negative impact; (b) the number of CL in the vitamin A treated groups was similar to that in the control group, suggesting that the increased number of embryos obtained in the former was not due to an increased ovulation rate, but to a direct effect on the embryos. An interpretation confirmed by (c) the size and uniformity in size of embryos collected on the 11th or 12th day of gestation of the gilts. Apparently, larger size embryos (5.0–5.5 mm in diameter) were recovered from vitamin-A-treated gilts than from the controls (<3.5 mm). Moreover, vitamin-A-supplemented group had a more uniform group of embryos than their counterparts injected with corn oil. The authors pointed out that asynchrony of early embryonic development has been associated with increased embryonic mortality. (d) Vitamin A increased serum progesterone concentrations on day 3, 5 and 6 after oestrus. Others (Jindal et al., 1996) have linked higher levels of progesterone during this period to increased embryonic survival in gilts.

Based on these and other findings, the authors appropriately concluded that the observed litter size increases in pigs treated or supplemented vitamin A are primarily due to increased embryonic survival mediated via an improvement in early embryonic synchrony and increased progesterone levels during the early post-ovulatory period.

4. Vitamin E and selenium

A definite relationship exists between selenium and vitamin E. Both function as indicated earlier, as cellular antioxidants that protect cells from the harmful effects of hydrogen peroxide and other peroxides formed from fatty acids. According to Noguchi et al. (1973), selenium functions as a component of cytosolic GSH-Px, which reduces peroxides, while vitamin E functions as a specific lipid-soluble antioxidant in the cell membrane. GSH-Px therefore destroys peroxides before they attack cellular membranes, while vitamin E acts within the membrane preventing the chain reactive auto-oxidation of the membrane lipids. The relationship, however, extends beyond this antioxidant function as a deficiency of one or the other could lead to a number of well-described disease conditions, and evidence exists that some selenium-induced disease conditions respond to vitamin E therapy or supplementation, and vice versa (Trinder et al., 1969; Underwood, 1981).
Impaired reproductive performance in both males and females of all farm animals species has been attributed to a selenium deficiency. In cattle, reproductive disorders linked to selenium deficiency are erratic, weak or silent heat periods, delayed conception, poor fertility, cystic ovaries (Corah and Ives, 1991), reduced sperm motility (McKenzie et al. 1998), reduced uterine motility (Segerson and Libby, 1982), mastitis (Olson, 1995), and retained foetal membrane (RFM) (Trinder et al., 1969; Campbell and Miller, 1998).

RFM has been the most studied and documented of all these disorders. Olson (1995) reported that in a review of the literature that summarized over 60,000 calving, the incidence of RFM was about 10.3%, and that selenium and vitamin E deficiencies constituted the more important nutritional causes. In Table 1, we summarise a number of studies on selenium and vitamin E status and the incidence of RFM in dairy cattle.

A number of lessons can be drawn from these and other studies reported in the literature. The incidence of RFM in selenium deficient cows could be reduced by a pre-partum supplementation of either selenium alone or in combination with vitamin E. The supplementation could be by injection or via the feed, and under certain circum-

<table>
<thead>
<tr>
<th>Study no.</th>
<th>Incidence of retained foetal membrane (%)</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Selenium-supplemented group</td>
<td>Control group</td>
</tr>
<tr>
<td>1</td>
<td>0</td>
<td>42</td>
</tr>
<tr>
<td>2</td>
<td>subgroup a: 0</td>
<td>26.5</td>
</tr>
<tr>
<td></td>
<td>subgroup b: 7</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>0</td>
<td>38</td>
</tr>
<tr>
<td>4</td>
<td>subgroup a: 22</td>
<td>22</td>
</tr>
<tr>
<td></td>
<td>subgroup b: 22</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>14.7 multiparous cows; 7.0 primiparous cows</td>
<td>27.6 multi 16.5 primiparous</td>
</tr>
</tbody>
</table>
stances a positive response to a vitamin E supplementation alone may be obtained. The circumstances under which such responses to vitamin E are obtained are not yet clear.

A comprehensive study on the effect of selenium deficiency in goats, showed that this species is as susceptible as others to selenium deficiency (Anke et al., 1989). Detrimental effects of dietary selenium inadequacies were observed on several reproductive events from the onset of heat through conception rate to milk production and offspring performance. Some of these results are summarised in Table 2. We postulate that the considerable reduction in milk production, as well as the lower milk fat and protein contents in selenium deficient does, were, to a large extent, responsible for the lower number of kids reared to weaning at 91 days.

In pigs, selenium and vitamin E deficiencies adversely affect different aspects of the male reproductive processes, including testicular and spermatozoa development with the resultant consequences of low sperm concentrations, reduced sperm motility, and sperms with a high incidence of cytoplasmic droplets (Liu et al., 1982). It is evident that such disorders may in turn adversely affect fertilization rate in the female.

These findings were confirmed in a recent study by Marin-Guzman et al. (1997). The authors fed basal semi purified experimental diets fortified or not with selenium (0 or 0.5 ppm) and vitamin E (0 or 220 IU/kg diet) to four groups of boars ([Landrace × Yorkshire] × Duroc) during a growing phase from weaning to 25 kg, and during the a finishing phase from 25 to 150 kg. Semen samples collected over a 16-week period during the finishing phase were appropriately evaluated for semen quality. In addition, the collected semen was used to artificially inseminate gilts that had previously been successfully pen-mated to other boars. The authors reported that boars supplemented with selenium had a better sperm quality than the control boars. Thus, sperm motility (87.9% vs. 60.4%), normal sperm (61.9% vs. 24.2%), and number of accessory sperm (59.7 vs. 14.2) were significantly higher in the former. Consequently, significant differences were also observed in the fertilization rate of gilts inseminated with sperm from the two groups — 98.5% vs. 73.4%.

Some other interesting observations made by the authors were as follows:

(a) During the latter half of the 16-week sperm collection period from boars, boars fed control unfortified diets had the highest percentage of sperm with cytoplasmic droplets.

Table 2
Effect of selenium deficiency on reproductive events in does
From Anke et al. (1989).

<table>
<thead>
<tr>
<th>Reproductive events</th>
<th>Control does</th>
<th>Selenium deficient does</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heat</td>
<td>Recognizable</td>
<td>Recognizable but delayed</td>
<td></td>
</tr>
<tr>
<td>Conception rate, %</td>
<td>93</td>
<td>64</td>
<td>( P &lt; 0.05 )</td>
</tr>
<tr>
<td>Infertile does</td>
<td>7.4</td>
<td>36</td>
<td>( P &lt; 0.05 )</td>
</tr>
<tr>
<td>Weaned kids/doe at 91 days</td>
<td>0.89</td>
<td>0.36</td>
<td>( P &lt; 0.01 )</td>
</tr>
<tr>
<td>4% Milk production, l/day</td>
<td>1.02</td>
<td>0.91</td>
<td>( P &lt; 0.001 )</td>
</tr>
<tr>
<td>Milk fat, g/day</td>
<td>41</td>
<td>36</td>
<td>( P &lt; 0.001 )</td>
</tr>
<tr>
<td>Milk protein, g/day</td>
<td>32</td>
<td>28</td>
<td>( P &lt; 0.001 )</td>
</tr>
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</table>
(b) Sperm motility declined in unfortified groups of boar over the collection period, while it remained constant and high (87.9%) in selenium supplemented boars.

(c) The main type of sperm abnormalities observed were abnormal bent, shoehook and coiled tails, and the incidence of these abnormalities was higher in unfortified boar sperm.

The objective of the study was to evaluate the effects of feeding low levels of selenium and vitamin E on semen quality in boars, and the carry over effect on fertilization in female. The authors appropriately concluded that sow fertility rates can be influenced by the selenium status of boars, because fewer sperm cells are produced, reach and penetrate the outer layer of the oocyte in the oviduct. In contrast, vitamin E supplementation had no significant effect on any of the measured and observed parameters.

Such an association between vitamin E deficiency and a decreased fertilizing capacity of spermatozoa has, however, been observed in other farm animals, particularly in poultry by others (Friedrichsen et al., 1980; Surai, 1999). Friedrichsen et al. (1980) observed a decrease in fertility from 91.5% to 33.3% in cockerels fed a vitamin E deficient diet for 29 weeks, but not irreversibly, as inclusion of the vitamin in the diet restored the fertilizing ability. Apparently, the detrimental effect of vitamin E deficiency on the fertilizing ability of birds is associated with a very high level of sperm lipid unsaturation (high concentrations of C20 and C22 polyunsaturated fatty acids) and the necessity of their adequate protection against lipid peroxidation (Surai, 1999), provided by natural antioxidants such as vitamin E, which, as indicated earlier, acts as a lipid soluble intra-membrane antioxidant that prevents chain reaction oxidation of spermatozoa lipids.

5. Copper and molybdenum

It is convenient to treat copper and molybdenum together, because of the well known interactions between the two elements. Interactions that may lead to impaired copper utilization. Thiomolybdates are formed in the rumen following reactions between sulfur (sulfide) and molybdenum. Thiomolybdates, in turn, react with copper to form insoluble copper thiomolybdate, which is unavailable to cattle. This copper–molybdenum–sulfur complex is one of the limiting interactions affecting copper utilization, and the leading cause of secondary copper deficiency, i.e. in the presence of adequate dietary content, as opposed to a primary deficiency resulting from inadequate dietary content. According to Phillipo et al. (1987), copper reserves in dairy heifers were depleted following high iron and molybdenum intake. Other antagonistic relationships that may reduce the bioavailability of copper and lead to secondary deficiencies, sub-clinical symptoms and impaired reproductive efficiency are copper–iron (Suttle, 1986), copper–zinc, and copper–phytate (Smart et al., 1981) complexes. This susceptibility to the formation of biologically unavailable complexes is in part responsible for the high incidence of copper deficiency syndromes, particularly in grazing ruminants.
Reported reproductive disorders associated with a copper deficiency in grazing ruminants include: low fertility associated with delayed or depressed oestrus, and long post-partum return to oestrus period; infertility associated with anoestrus, abortion and foetal resorption (Annenkov, 1981; Corah and Ives, 1991). According to Corah and Ives (1991), data from the literature showed an inverse relationship between serum copper levels and important reproductive parameters such as days to first service (56 vs. 70 days), services per conception (1.1 vs. 4.4) and days to conception (56 vs. 183 in dairy cows with high and low serum copper levels, respectively. In other words, higher serum copper levels improved these parameters of economic importance.

According to Underwood (1981), the molybdenum requirement of animals are extremely low, and these requirements are likely to be met readily by normal diets, to the extent that unequivocal evidence of molybdenum deficiency in ruminants unrelated to copper is extremely rare (Underwood, 1981). On the other hand, many of the reported copper deficiencies syndromes are apparently secondary deficiencies related to high molybdenum concentration (Ward, 1978; Phillipo et al. 1987). Reported reproductive disorders such as decreased libido and sterility in bull calves caused by tissue damage and reduced spermatogenesis (Thomas and Moss, 1951); delayed puberty, reduced conception rate, and anoestru (Phillipo et al., 1987), have therefore, more often than not, been linked to high dietary intakes rather than to a deficient intake of molybdenum.

In a recent series of experiment, Phillipo et al. (1987) compared reproductive events in heifers fed a basal diet containing 4 mg copper/kg dry matter of feed, with those of heifers fed the same diet fortified with 5 mg of molybdenum or 500 or 800 mg iron/kg dry matter. They reported that molybdenum supplementation: delayed the onset of puberty by 8–12 weeks, and reduced fertility from 75% in the control group to about 14% in test heifers. They also observed an increase in the incidence of anovulation, 20% in test against 2.5% in control animals. An interesting observation reported earlier by Wise and Ferrell (1984) and also reported in this study, was a reduction in the LH ovulatory peak in molybdenum supplemented animals, to the extent that exogenous LH given to spike the peak had no effect on conception rates. It was therefore concluded that the observed effects of molybdenum may be mediated through an interference with the secretion of ovarian steroids, and/or the feedback of the steroids on the hypothalamo-pituitary system.

6. Zinc

As a constituent of several metallo-enzymes, zinc is involved in several enzymatic reactions associated with carbohydrate metabolism, protein synthesis and nucleic acid metabolism. It is therefore essential in cells like the gonads, where active growth and division are taking place. Consequently, reproductive functions are seriously impaired by zinc deficiency, and as indicated by Underwood (1981), spermatogenesis and the development of primary and secondary sex organs in the male, and all phases of the reproductive process in the female from oestrus through pregnancy to lactation, may be affected.
According to Pitts et al. (1966), a reduction in testicular size was observed in bull calves fed a zinc deficient diet for 13 weeks, from 8 to 21 weeks of age. The situation was reversed by the time the calves were 64 weeks old, by increasing dietary zinc intake. In another trial designed to evaluate the performance of Barki ewes under field conditions in Egypt, Ali et al. (1998) compared two groups of mature ewes fed either a control diet containing 23–25 ppm or a test diet supplemented with an additional 100 ppm of zinc as zinc sulphate. Supplementation started 1 month before mating, and continued until lambing. The authors reported that zinc supplemented ewes consumed about 15% more feed than the controls, had a higher fertility rate, were more prolific (89% vs. 40%), and produced heavier lambs at birth (4.0 vs. 2.9 kg) and at weaning (17.7 vs. 14.2 kg). In addition, supplementation increased serum zinc concentration, particularly during late pregnancy. These results confirmed the findings of others, who had shown the detrimental effects of a dietary zinc deficiency in farm animals on feed consumption and various phases of the reproductive cycle in the female (Masters and Fels, 1980; Masters and Moir, 1983; Underwood, 1981), perhaps through its involvement in steroidogenesis (Hurley and Doane, 1989) and carbohydrate and protein metabolism.

7. Concluding remarks

Some evidence has been provided to demonstrate the importance of micronutrients for the reproductive well being of farm animals. As summarised in Table 3, some of these functions are crucial to the overall biological well being of the animal, and

<table>
<thead>
<tr>
<th>Micronutrient</th>
<th>Mechanism/metabolic function</th>
<th>Deficiency consequences</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vitamin A</td>
<td>Steroidogenesis, embryonic synchrony</td>
<td>Delayed puberty, low conception rate, high embryonic mortality, reduced libido</td>
</tr>
<tr>
<td>Vitamin E</td>
<td>Intra-membrane free radical detoxification</td>
<td>Low sperm concentration and high incidence of cytoplasmic droplets, retained foetal membrane</td>
</tr>
<tr>
<td>Selenium</td>
<td>Component of GSH-Px</td>
<td>Reduced sperm motility and uterine contraction, cystic ovaries, low fertility rate, retained foetal membrane</td>
</tr>
<tr>
<td>Copper</td>
<td>Enzyme component and catalyst involved in steroidogenesis, and prostaglandin synthesis</td>
<td>Low fertility, delayed/depressed oestrus, abortion/foetal resorption</td>
</tr>
<tr>
<td>Zinc</td>
<td>Constituent of several metallo-enzymes; steroidogenesis, carbohydrate and protein metabolism</td>
<td>Impaired spermatogenesis and development of secondary sex organs in males, reduced fertility and litter size in multiparous species</td>
</tr>
</tbody>
</table>
deficiency consequences may well go beyond reproductive disorders, although emphasis has been placed on the latter in this paper, because the overall production efficiency of livestock depends largely on their reproductive performance. In the tropics, livestock reproductive performance is to a large extent mediocre, particularly in the traditional extensive and semi-intensive production systems, and is still below the optimum even in the modern intensive swine and poultry production units. As indicated earlier, poor nutrition is one of the four factors responsible for such mediocre performances, and is perhaps the one factor with the potential to positively influence the others. Efforts continue to be made in developing countries in the tropics, to improve livestock nutrition, but such efforts are concentrated on improving carbohydrate and protein nutrition, while micronutrient nutrition is more or less neglected.

It is, however, in the tropics that particular attention needs to be paid to the latter. During the dry season, available fodder is extremely low in minerals, because of the well-known phenomenon of mineral translocation to the root system during this period, and a part of what is present in aerial parts is tied up in fibre–mineral complexes. The situation is only marginally better during the wet season, because of the rapid growth and lignification of tropical grasses. In non-ruminant semi-intensive and intensive systems, the problem of micronutrient nutrition is no less serious, because to a large extent, supplementation is not done on a rational as required approach, and imported pre-mixes are used without due regard to the actual dietary content and needs of the animal. Moreover, no regulatory mechanisms are in place, to monitor the concentration and biosafety of such products, and we have presented evidence of how excesses of one micronutrient might interfere with the utilization of others. If all of the issues raised above are not addressed and resolved through research, farmer education and enforced regulations, micronutrient nutrition of livestock in developing tropical countries will continue to be sub-optimal, with predictable detrimental consequences on reproductive performance, and hence on overall productivity of farm animals.

References