Iodine and deer calf survival

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Abstract:

Iodine deficiency in newborn and neonatal deer has been diagnosed clinically on a number of deer farms and a small number of laboratory reports confirm diagnosis. Thus, while Iodine deficiency is not commonly recognised, it may contribute sub-clinically to calf losses on a number of deer farms in New Zealand. Improvement in weaning percentage of sheep flocks has been reported but not all trials demonstrate responses, despite low forage iodine and/or blood thyroxine in some circumstances. Iodine deficiency is caused by low dietary iodine, or goitrogenic content of forage, namely glucosanolate, a precursor of thiocyanate that blocks uptake of iodine by the thyroid gland, or thiouracil, that blocks conversion of T4 to T3. The relative merits of diagnostic criteria are discussed.

Results of an Iodine supplementation field trial using iodised poppy seed oil on 3 farms in Hawkes Bay, and one in Wanaka involving 2600 red deer hinds showed no statistically significant increase in weaning percentage due to supplementation. This suggests that if iodine supplementation is to be used, it should be targeted at herds with known deficiency, those grazing high-risk diets, or used as a diagnostic tool to determine the possible influence of sub-clinical iodine deficiency in reducing calf survival to weaning.

Introduction

Prenatal losses on deer farms are common. Their incidence, potential causes, risk factors and attempts to reduce losses are summarised elsewhere in these proceedings (Wilson 2002). Clinical goitre has been observed in 1-2-month-old farmed deer in New Zealand (Wilson unpublished). The first laboratory report of the impact of iodine deficiency on calf survival described stillbirths in 4 of 20-30 calves born with oedema of the neck and enlarged thyroid glands, and evidence of thyroid hypoplasia (Anon 2000). A subsequent report described 10 of 70 first calving wapiti and 10 of 120 deer calves of unspecified species found dead without suckling (Anon 2001). Again histological evidence indicated thyroid hypoplasia. Abortion and stillbirth of fallow deer have occurred in Switzerland (Giacometti Personal Communication).

Evidence for the influence of Iodine on reproductive performance in sheep flocks in New Zealand has recently been reviewed (Clark et al 1998). That review indicated that supplementation with Iodine increased litter size in 2 of 10 flocks and perinatal survival in 3 of 10 flocks. Supplementation trials on one sheep property in two sequential years described by Sargison *et al.* (1997, 1998) indicated that this may be an ongoing problem on some farms. Indeed Sargison and West (1998) suggest that because modern varieties of white clover present in New Zealand pastures contain high concentration of thiocyanate precursors, iodine deficiency may be an emerging problem. Thus sub-clinical iodine deficiency contributing to reduced reproductive performance could be, or could become a common occurrence on New Zealand deer farms.

Sub-clinical iodine deficiency as a risk factor for reduced perinatal survival on deer farms is unknown. This paper reviews relevant aspects of iodine nutrition to deer production and presents data from the first reported iodine supplementation trial conducted on farmed deer.

Review of Iodine in livestock

Thyroid activity

The thyroid gland plays an important role in all kinds of metabolic processes, like lipid, carbohydrate, and nitrogen metabolism, calorigenesis, growth and development, nervous system function, and reproduction performance, due to the production of the thyroid hormones thyroxine (T4) and tri-iodothyronine (T3) (Bernal and Refetoff, 1977; Underwood 1977). Functioning of the thyroid gland depends on the availability of iodine (I). Insufficient

thyroid hormone (thyroxine) production by the thyroid gland as a result of iodine deficiency can reduce the progeny survival and therefore reproductive efficiency (Watkins *et al.*, 1983). Watkins *et al.* (1983) describe changes in hormone levels in relation to the season, age, lactation in white-tailed deer. No consistent seasonal patterns of T4 and T3 levels have been found for deer. Thyroid activity decreases with age and non-lactating animals show higher levels of T3 and T4 than lactating animals.

Effect of iodine deficiency on reproduction

The effects of Iodine deficiency on reproductive performance has been reported in several studies for sheep and cattle. Neonatal mortality of lambs associated with enlarged thyroids has been reported in Australia and New Zealand (Southcott 1945; Statham and Bray 1975; Setchell *et al.* 1960; George *et al.* 1966; King 1976; Caple *et al* 1980; Andrew and Sinclair, 1962). In Japan perinatal diseases as abortion, stillbirth, alopecia, or birth of weak young in cattle associated with goitre have been reported (Seimiya, *et al.* 1991). Insufficient intake of iodine, or eating feed containing goitrogens like thiocyanate from white clover (*Trifolium repens* L.) may cause serious loss in reproduction. (Alexander *et al.* 1990; Watkins *et al.* 1983). Alexander *et al.* (1990) found impaired thermoregulation and difficult or prolonged birth to be related with goitre in lambs. Enlarged thyroids have been diagnosed after autopsy of death born deer calves in the last 2 years in New Zealand (Anon 2000-2001).

Causes of Iodine Deficiency

Iodine deficiency is caused either by low iodine intake or the goitrogenic effect of some feeds. Barry (1983) describes iodine deficiency on pastures of 0.09 to 0.18 mg/kg DM. However, soil ingestion and the presence of goitrogenic compounds in forage confound the effect of iodine content of pasture *per se*.

There are two types of goitrogen. Thiocyanates block uptake of inorganic iodine by the thyroid gland and hence reduce T_4 synthesis (Barry 1983). Thiouracil goitrogens block the conversion of T_4 to the active T_3. The latter are found in some tropical legumes but are not detected in forage species used in the temperate New Zealand environment. Glucosinolates, the precursor of thiocyanates, in kale, cabbage, brussels sprouts and broccoli are broken down during chewing to organic thiocyante ions. However, glucosinolates in swedes and turnips produce only small quantities of goitrogenic thiocyanate. White clover contains cyanogenic glycosides that are goitrogenic (Coush and Caradus 1995)

Iodine concentrations in pasture were highest in March and August on some properties, but a seasonal pattern is not evident on all (Clark *et al* 1998). Iodine concentrations appear to be lower when pasture is growing rapidly and this often coincides with the season when clover, which contains goitrogenic compounds, are dominant.

Diagnosis of Iodine Deficiency

History of intake of goitrogenic containing feeds

History of grazing high-risk brassicas may be an important factor in diagnosis or determining the need for supplementation. However, clinical and sub-clinical iodine deficiency has occurred in sheep on ryegrass/ white clover pastures in New Zealand (Sargison et al 1997;1998).

Clinical and sub-clinical goitre

Enlargement of the neck caused by swollen thyroid glands is an obvious sign of iodine deficiency and has been observed in aborted deer (Giacometti Personal Communication), still births (Anon 2000/2001) and in young deer 1-2 months of age (Wilson unpublished). Histology is necessary to confirm the diagnosis. Mental retardation of progeny may be caused by low iodine concentrations in the dam during early pregnancy, but this has not been reported in deer. Progeny may be small, weak or hairless. However, some iodine

supplementation trials in sheep have shown responses in reproductive performance in the absence of clinical goitre, proving that sub-clinical goitre may be an important cause of loss.

Pasture iodine

While Barry (1983) suggested pastures below 0.18 mg I/kg DM may be deficient, the review by Clark *et al* (1998) proposed there was no reliable relationship between iodine responsiveness in sheep and iodine content of pasture. Those authors describe pasture iodine assays as " un-helpful". This is probably because of the confounding effects of soil and pasture species containing various thiocyante precursors.

Clark *et al* (1998) also review two studies where there was low T_4 concentration in sheep on pastures with high iodine concentration.

Serum thyroxine

Some trials indicated that T_4 can be a predictor of likely iodine supplementation responses (Clark *et al* 1998) but others did not. Comparison of absolute T_4 concentrations between studies is difficult because assay methodology varies. In some cases an iodine response, in terms of proportion of ewes carrying twins, was observed despite apparently normal T_4 concentrations in dams. This shows inconsistency between T_4 and likely responses. Clark *et al* (1998) concluded that "serum T_4 and T_3 concentrations appear to have no predictive value for determining the likelihood of increased prenatal mortality associated with sub-clinical iodine deficiency" in sheep. Thus in sheep there is a general relationship between T_4 and fertility of the dam but not in relation to lamb survival. This is probably because in the latter stages of gestation, thyroid hormone metabolism is a response to iodine available to the foetus and not to thyroxine concentrations in the dam.

No data is available for reference values in deer (Wilson and Grace 2001).

Thyroid:liveweight relationship

It is generally accepted for newborn lambs that a thyroid:liveweight (T/W) ratio (g/kg) of 0.4 or above is indicative of thyroid hypoplasia (Clark *et al* 1998). Those authors summarised trial data for 10 farms showing a significant difference in this ratio between progeny of treated and untreated ewes in 6 of 10 studies, although the means of only 3 progeny from untreated ewes were above 0.4. This measure was used by Grace et al (2001) as a measure of response to iodine supplementation to dams.

Four trials showed a difference in T/W ratios but not in perinatal survival, and in one case there was no difference in the ratio but a difference in prenatal mortality (Clark *et al* 1998). Sargison and West (1998) showed a curvi-linear relationship between T/W ration and prenatal mortality. While patterns exist between means, the ratio in an individual animal appears to have a poor predictive value. There is a poor relationship between T/W ratio and T4 concentration (Clark *et al* 1998)

In deer, a ratio of 0.42 was observed in one calf with a thyroid weight of 5.5g (Anon 2001). In another case the thyroid weight was 17g but no live weight was given (Anon 2000). However, if the weight of the calf was within the normal range of 8-15kg (depending on species) the T/W ratio would be 1.13-2.15.

Iodine supplementation response trials

Data on response trials for sheep have shown improvements in the number of foetuses at scanning rather than the number of ewes pregnant (Sargison *et al* 1997;1998). However, not all supplementation response trials showed this outcome. Thus the role of iodine in

conception rate in deer needs to be investigated where failure to conceive is a problem that cannot be attributed to other more common factors.

Improved survival of progeny has been observed in 3 of 10 supplementation response trials in sheep (Clark *et al* 1998).

Supplementation

Supplementation of ewes grazing a swede/kale/turnip crop with long-acting iodine injection prevented goitre (Grace *et al.* 2001), and increased weaning percentages in ewes on pasture (Sargison and West, 1998, Clark *et al.* 1998). Injectable iodine has been used by one of the authors (PW) to prevent goitre in young deer, by treatment of dams in late august-early September. When that became unavailable, oral potassium iodide was used for treatment in late August, followed by a second in October.

Summary

It is difficult to predict an iodine response based on pasture chemical and animal biochemical measurements. Previous history may provide some justification for supplementation but responsiveness may vary year to year due to forage, climate and management factors. Thus surveillance of neonatal losses at post mortem appears to be the best method to confirm the diagnosis of iodine deficiency. An alternative method of investigation is to undertake clinical trials where half of the dams are treated and half untreated.

An Iodine Supplementation Trial in Farmed Deer: Preliminary Findings

A field trial was conducted to test the hypothesis that some perinatal losses on commercial deer farms may be reduced by supplementation of hinds with iodine.

Farms and Animals

Deer from 4 farms were chosen to provide a minimum of 1292 for injection, being the number that a power analysis showed was necessary to achieve an 80% power if the difference between treated and non-treated hinds was three percentage points. Details of farms and deer numbers are presented in Table 1.

All hinds were red deer and farmed according to normal farming practice. Farms 1 to 3 were managed extensively and were not recorded in detail, whereas farm 4 was farmed intensively and a significant amount of data was collected (see paper by Bell and Wilson elsewhere these proceedings).

The latter farm scanned all hinds in June and culled non-pregnant hinds. Farms 1-3 scanned all yearling hinds but only a sample of adult hinds because pregnancy rates were 97-98%. They culled non-pregnant animals.

Herds were selected primarily on the desire of the owners to explore ways to improve weaning percentage based on previous figures (Table1). Large herds were chosen for logistical reasons but also to limit the number of confounding factors inherent in using large numbers of smaller herds.

Table 1: Description of study farms

Farm	1	2	3	4
Location	*Central HB*	*Central HB*	*Central HB*	*Wanaka*
Deer-fenced Area (ha)	262	165	540	146
No. Paddocks	20	27	25	26
Previous Weaning% *				
MAH	86-90	92	92-93	83-88
Yearling	80-84	?	N/A	60-83

Farm	1	2	3	4
Location	*Central HB*	*Central HB*	*Central HB*	*Wanaka*
Hinds mated 2001 Total Mixed-age Yearling	 646 504 142	 513 392 121	 1018 702 316	 617 427 190
Scanning % 2001 MAH	 95+	 97 +	 98 +	 94 (Elite) 97 (Commercial)
Yearling	84	88	62	93 (Elite) 95 (Commercial)
Drys culled	Yes	ylg only	Yes	Yes
Treatment Date	21 August	28 August	22 August	28 August

* Calves weaned/Hinds joined with stag

+ Sample only

No previous clinical evidence of Iodine deficiency had been reported on any of the study farms, but higher than acceptable calf losses were experienced in the past. Pasture iodine and blood thyroxine concentrations were not measured prior or during the trial, because previous data from similar trials using sheep showed these were not a good predictor of animal responses to iodine supplementation.

Iodine Treatment

The treatment was an iodised poppy seed oil "Flexidine" (Bomac Laboratories Ltd, Batch 906). This was given intramuscularly using 16G x 1/2 inch needles and a variety of delivery syringes under test. The dose rate was 3ml. Treatment dates ranged 21-28 August 2001 (Table1).

Experimental method and management

Farms 1 – 3

Deer were yarded and penned in small groups for injection. Every second deer was injected in the neck and their tag number, age and mob detail recorded. Between mid-February to weaning (See Table 2), deer were yarded and calves counted from each mob. Hind udders were examined and differentiated between wet/dry and dry/dry categories as possible. Dry hind identification was paired against identification of treated hinds and numbers of treated and control hinds not rearing a calf were calculated. In the event, it was not possible to accurately categorise all hinds as wet/dry or dry/dry, so data was pooled for statistical and descriptive analysis.

Farm 4

This farm underwent a more intensive management programme with "elite" hinds managed separately "commercial" hinds. All hinds were scanned with results presented in Table 1. Non-pregnant hinds were culled. Half of the hinds in each of the "elite" and in two of the "commercial" groups were treated as above and calved on flat paddocks with small sub-division. The remaining "commercial" hinds were allocated to three mobs (n = 109, 45, 45) with the larger group being treated with iodine. This was to permit collection of any dead progeny found for measurement of live weight and thyroid gland weights and for thyroid histology, allowing differentiation between progeny of known treated and untreated hinds.

Hinds on farm 4 were examined 19th October for pregnancy status by udder conformation and abdominal balloting as appropriate to estimate gestational loss rates. In mid January deer were yarded, calves counted and lactational status of hinds recorded.

Statistical analysis

Data for mixed-age and yearling hinds were combined within farms. Numbers of non-lactating hinds from each group were recorded against the total number of hinds available at calving. A Chi squared analysis was undertaken to examine treatment, farm and treatment by farm interactions using SAS. Data analysed was simply the number of animals not rearing a calf to weaning, without partitioning pregnancy status, pre-natal and post-natal losses.

Results

Weaning data are presented in Table 2.

Table 2: Weaning Data 2002

Farm	1	2	3	4
Date Checked	*March 5*	*February 13*	*April 24*	*January 15-17*
No. Hinds at weaning				
MA	} Not Counted	384	575*	399
Ylg		105	307	184
Weaning: No Dry				
MA	} 83	62	63	40
Ylg		10	60	44
Weaning: No Wet/Dry				
MA	} Not Done	23	} Not Done	33
Ylg		1		42
No Weaned/hinds mated (%)				
MA	} Not Done*	81	89	85
Ylg		80	80	69
No Weaned/hinds calving (%)				
MA	90+	} Not Done	} Not Done	87
Ylg	87	90		72
No Hinds Dry:				
Treated	34	31	60	37
Control	33	40	63	47

* 146 Hinds moved to a different property. Weaning 97%. Dry hinds not recorded for analysis

+ During matching, mobs became combined. Only data from 386 hinds presented

• Calculated from discrepancy between scanned pregnant and pre-calving check for pregnancy

Data shows that the "true" weaning percentage (Number weaned/hinds mated), ranged 81-90% for mixed age hinds and 69-82% for yearling hinds.

The numbers of treated and untreated hinds not lactating are given in Table 2. Statistical analysis showed there were no differences in numbers of calves not reared to weaning between treated and control hinds. There was a significant farm difference in overall weaning rate but there was no interaction between farm and treatment, indicating that the pattern of events was similar on each farm.

Two specimens were collected for thyroid weight and histology. One progeny from a treated hind had a thyroid weight of 10.82g and bodyweight of 8.4kg (Ratio T/B = 1.22) and the second from a control hind had a thyroid weight of 4.43g, and a weight of 9.2kg (ratio T/B = 0.48).

Discussion

Iodine treatment did not significantly increase reproductive efficiency on the study farms. This suggests that Iodine supplementation on farms chosen at random is unlikely to be effective in most cases. The chance of getting a response would be improved if. an attempt is made to diagnose iodine deficiency before Iodine supplementation is recommended in a given situation. However, paradoxically, one of the best means of diagnosing iodine deficiency is response to supplementation. This has been the case with sheep trials, where no clinical evidence occurred, yet a response to Iodine supplementation was achieved (Sargison and West 1998). As has been shown in other studies of deer neonatal mortalities, it can be extraordinary difficult to get good samples of neonatal tissue for confirmation of diagnosis. In this case an attempt was made to collect specimens from Farm 4 for analysis of live weight and thyroid weights, to enable calculation of thyroid over live weight ratios and for histology. Only two suitable specimens could be recovered. The thyroid:bodyweight ratios of both were

above 0.4g/kg, which is considered to be the indicative ratio for sheep, despite one being from a treated hind, and the other from an untreated hind. This suggests that the sheep ratio may not be appropriate for deer. More data from "normal" neonatal deer is needed.

Farms 1 and 3 had weaning percentages (calves weaned/hinds joined, or reproductive efficiency) above average for both mixed age and yearling hinds. Farm 2 had a weaning percentage in adult hinds below average, and there was a high number of dry/dry hinds. This was tentatively diagnosed as a late abortion problem caused by leptospirosis, on the basis of concurrent high titres, and diagnosis of leptospirosis in the farmer. Farm 4 had a weaning percentage in mixed-aged hinds below average but the weaning percentage in yearling hinds was average at 69%.

However, these results should not discourage veterinarians from using iodine supplementation trials as a means for diagnosing sub-clinical iodine deficiency on deer farms. Farms with consistently lower than average weaning percentage should be intensively monitored for clinical and sub-clinical iodine deficiency as a potential cause of sub-optimal performance. Iodine deficiency in deer has been described as a cause of neonatal and postnatal mortalities in laboratory reports from New Zealand. One author (PW) has clinical experience of goitre and reduced calf survival on a deer property which was prevented by iodine supplementation. The persistency of the problem on that property was confirmed subsequently by observation of calves with goitre at birth from a small group known to have missed their supplementation.

Additional to reproductive performance and previous history of goitre, the evaluation of the need for iodine supplementation should include an understanding of the diet of deer and location. It is likely that the goitrogenic compounds of brassica crops and possibly clover species affecting other domesticated ruminants in New Zealand influence thyroid activity in deer. Thus hinds grazing at risk crops during pregnancy, particularly the later stage of pregnancy, should be considered for preventative supplementation.

A more detailed presentation of experimental methods, results in discussion will be presented elsewhere in due course.

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