



**Malnutrition in farmed red deer hinds**  
*(Cervus elaphus atlanticus)*  
**in Norway**

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**Key words:** Mineral deficiency, serum biochemistry, vitamin deficiency, unthriftiness, wasting, weight loss.

*Abstract:* Malnutrition was studied in farmed red deer. Malnourished animals did not recover from winter energy loss, remained in poor condition throughout the summer, and some died of starvation. Liver analysis of one hind that died of malnutrition showed selenium and cobalt deficiency. This hind was cachectic and heavily infected with lung-worm (*Dictyocaulus* spp). Serum levels of thirty-three parameters of hinds suffering from malnutrition (n=4) were compared to values from free-ranging reference hinds (n=10). Hinds suffering from malnutrition had significantly higher values ( $p<0.01$ ) of glutamate dehydrogenase, lactate dehydrogenase, globulin, alpha 2 globulin, beta globulin, gamma globulin, urea and copper. The farmed hinds, however, had been fed supplementary copper and did not suffer from deficiency. Malnourished hinds had significantly lower values of albumin, creatinine and zinc. These parameters can be used to diagnose malnutrition in red deer hinds. Hinds suffering of malnutrition recovered completely within two months after treatment with 20 mL Becoplex vet® i.m., 10 mL Selenevit vet® i.m. and 10 mL Ivomec® pour-on vet.

## **Introduction**

Malnutrition occurs in deer when there is an inadequate intake of required nutrients. This can happen even if the animals receive large amounts of food, but are unable to ingest, digest, absorb, or utilize this food. If animals are unable to obtain and metabolise food for an extended period of time they may suffer from malnutrition. The clinical signs are unthriftiness, reduction of weight and mortality. Red deer have a nocturnal hypometabolism as an overwintering strategy (Arnold *et al.*, 2004). Winter death syndrome, however, is part of the normal pattern of mortality of wild populations of red deer when range conditions deteriorate and nutritional stresses affect the animals (Ross, 1994). In farmed red deer it is called "inanition syndrome" (Haigh & Hudson, 1993). It may occur directly as a result of inadequate food intake, or may be caused by a variety of other disease conditions that occur in animals that are compromised by their nutrition condition. The high metabolic rate and low fat reserves of red deer relative to other domestic stock mean that they require more dietary energy in cold conditions and may be more vulnerable to cold stress (Ross, 1994). Clinical signs are non-specific. There may be features such as wasting, scouring and depression. Post mortem examination reveals lack of body fat reserves, which is evident from the lack of omental and perirenal fat, as well as the thin, pale pink gelatinous appearance of the bone marrow (Ross, 1994). Prevention of winter death syndrome, however, is possible. Preventative measures should start in the summer and autumn by ensuring that all deer lay down maximum fat reserves. For animals already suffering from malnutrition, however, preventative measures will not be successful. It is observed among farmed red deer in Norway that they do not recover from winter energy loss. They remain in a poor condition throughout the summer and some die of starvation.

In this study malnutrition was seen in the spring, summer and

autumn as a loss of condition. Animals lost weight despite the availability of silage and additional feeding with a special feed designed for red deer. This feed contained increased amounts of copper and added vitamins and micro minerals. The aim of this study was to diagnose the malnutrition and devise a procedure to prevent the animals from chronic weight loss.

### **Material and methods**

Necropsy of one hind with malnutrition that died 28 May 2003, was carried out the same day by the Section of Wildlife Disease, National Veterinary Institute, Oslo. On the same farm, blood samples were collected from four malnourished hinds, from two on 7 July and from one each on 9 July and 12 August. They were all in poor body condition. After the blood samples were taken they were injected i.m. with 20 mL of Becoplex vet.® (Boehringer Ingelheim Danmark A/S, Copenhagen, Denmark) that contains 20µg of cyanocobalamin, 5 mg of thiaminhydrochlorid, 3mg of riboflavin, 2 mg of pyridoxinhydrochlorid, 20 mg of nicotinamid and 12 mg of dexpanthenol per mL and with 10 mL Selenevit vet.® (Veterinaria AG, Zürich, Switzerland) containing 1mg of sodium selenite and 25 mg of tocoferolacetate per mL. Additionally the animals were treated with 10mL Ivomec® pour-on vet (Merial SAS, Lyon, France) containing 5mg of ivermectin per mL and 10g copper oxide (Copacaps®, Rhône Mérieux Limited, Harlow, Essex) per os. Blood samples from 10 immobilized free-ranging reference hinds were collected in February and March the same year. All hinds were darted and immobilized using a mixture of Rompun® dry powder (Bayer AG, Leverkusen, Germany) and Zoletil forte vet.® dry powder (Virbac International, Carros Cedex, France) dissolved in 5 mL of sterile water. The dosages used were approximately 250 mg of xylazine and 250 mg of tiletamine-zolazepam per 100 kg body mass

(Arnemo *et al.*, 2002).

For the biochemical and mineral analyses a 10 mL blood sample was collected in a Venoject® II Autosep® Gel+Clot Act, left to clot at room temperature for 1-2 hours and centrifuged at 3000 RPM for 5 minutes. The serum was removed and kept frozen at -80 °C before analysis.

The 22 biochemical constituents, aspartate amino transferase, alanine amino transferase, alkaline phosphatase, gamma glutamyl transferase, glutamate dehydrogenase, creatine kinase, lactate dehydrogenase, total serum protein concentration, albumin, globulin, alpha-1 globulin, alpha 2 globulin, beta globulin, gamma globulin, urea, creatinine, total bilirubin, cholesterol, triglycerides, free fatty acids and  $\beta$ -hydroxybutyrate were analysed using the automatic Advia® 1650 System (Bayer Corporation, Tarrytown, N. Y., USA) at the Central Laboratory, Norwegian School of Veterinary Science. Analysis of the 11 elements Na, K, Mg, Cu, Zn, P, Ca, Al and Fe was performed by atomic absorption spectrometry (Perkin Elmer® AAS 3100) at Telemark University College. The analysis of Co and Se was performed on ICP-MS, Fisons PQ11 by Miljølaboratoriet, Skien, Norway.

Blood serum values of the hinds suffering from malnutrition (n=4) were compared to the selected reference hinds (n=10) by using the Mann-Whitney U-test (Minitab Statistical Software). P-values less than 0.01 were considered significant.

## Results

The pathology report of the autopsied hind concluded with: cachexia, heavy infection with lungworm (*Dictioacaulus* spp) and deficiency of selenium and cobalt. The liver levels of selenium and cobalt were 0.07  $\mu\text{g/g}$  and 0.01  $\mu\text{g/g}$  wet weight respectively. Hinds suffering from malnutrition had significantly higher serum levels of glutamate dehydrogenase (p=0.004), lactate dehydrogenase (p=0.009), globulin

( $p=0.006$ ), alpha 2 globulin ( $p=0.006$ ), beta globulin ( $p=0.006$ ), gamma globulin ( $p=0.006$ ), urea ( $p=0.006$ ) and copper ( $p=0.006$ ) and significantly lower values of albumin ( $p=0.007$ ), creatinine ( $p=0.006$ ) and zinc ( $p=0.007$ ) than the reference hinds (Table 1). Three weeks after treatment the body condition of the affected hinds had improved and they had completely recovered after two months.

### **Discussion**

There are no data available concerning normal mineral and vitamin requirements for red deer. In spite of this, many animals are successfully raised using nutritional criteria established for other ruminants (Suttle, 1986; Haigh & Hudson, 1993). The effect of captures and chemical immobilization on levels of haematological and serum biochemical parameters have been documented by Cross *et al.* (1988) and Marco & Lavín (1999). In this study we used a mixture of xylazine-tiltamin-zolazepam because this combination is normally used for immobilization of red deer in Norway. Serum biochemical and mineral reference values in the free-ranging *Cervus elaphus atlanticus* subspecies have been established (Rosef *et al.*, 2004) and were compared to hinds with malnutrition in our study. Typically, malnourished and untreated hinds had periodical diarrhoea that ended with death (unpublished observations). Necropsy of one of the affected hind showed cachexia, deficiency of selenium and cobalt, and a heavy lungworm infection.

Copper deficiency is common in grazing red deer livestock. It can be manifested as a condition or disease in the absence of specific clinical signs or with typical clinical signs as enzootic ataxia. Copper deficiency with different clinical manifestations has been seen in the Norwegian red deer subspecies (Handeland & Flåøyen, 2000; Rosef *et*



*al.*, 2001). In the herd investigated, copper deficiency was diagnosed with typical clinical signs as enzootic ataxia and unthriftiness. Because of the copper deficiency the animals were daily given 0.2-0.5 kg supplementary red deer feed (Felleskjøpet, Rogaland). This feed consisted of 11.6% protein, 9.9% fiber, 4.4 % fat, 8.4 % ash, 11.9% water, 22.8% starch, 1.16% calcium, 0.55% phosphorus, 0.43% sodium, 0.41% magnesium and 27.4% neutral detergent fiber. Each kilo feed had additions of 5 IU vitamin A, 2 IU vitamin D3, 30mg alphas-tocopherol lactate, 15mg copper (II) oxide, 285mg copper (II) sulphate, 0.7mg sodium selenite, 3 mg cobalt (II) sulphate, 24 mg zinc oxide, 8 mg manganese oxide, and 1.2 mg of calcium iodate. Additionally the animals were given 10g of copper oxid per os. The mean blood value for copper in the treated animals was significantly higher than in the reference animals (Table 1). It can be concluded that the animals did not suffer from copper deficiency. Of the 50 hinds in the herd, 10 did not respond to the copper treatment but continued to show typical signs of malnutrition. The animals lost weight despite the availability of silage and the deer feed supplement. They appeared hungry but did not respond clinically to the feed. During spring and summer they were easily distinguished from the other members due to the delayed shedding of their winter coat, which gave them a moth-eaten appearance. Cobalt is essential because it is required for the synthesis of vitamin B12, and the only source of this vitamin in the animal body is from microorganisms in the rumen. Because cobalt is not stored in the tissues, it must be present continuously in the feed (Haigh & Hudson, 1993). Characteristic for cobalt deficiency is progressive intractable weight loss in the absence of any apparent disease, in spite of adequate care and feeding and even after administration of oral anthelmintic and copper treatments (Jones, 1994). Cobalt deficiency is a chronic disease with no specific signs that distinguish it from other conditions. Weight loss, poor performance and emaciation are seen. The absence

of vitamin B12 leads to starvation because it is essential for metabolism of volatile fatty acids, which are primary energy sources in ruminants. The main distinguishing sign at necropsy is extreme emaciation. This is in accordance with the finding in this study. It appears, however, that red deer are able to thrive on pasture with lower cobalt levels than sheep and cattle, and in two trials with red deer, no responses to treatment with injectable vitamin B12 (Clark *et al.*, 1986) were seen. On the other hand, red deer grazing in cobalt-deficient pastures in Scotland have shown evidence of response to vitamin B<sub>12</sub> therapy with increased growth (Jones, 1994). In our study extreme emaciation was confirmed by necropsy. The content of cobalt in the liver was low (0.01µg/g) which confirmed the diagnosis of cobalt deficiency. The blood levels of the diseased and the reference animals, however, were both below the detection level (0.17µmol/L) (Table 1). Sickness of moose caused by cobalt deficiency has been established (Frank *et al.*, 2004) with wasting as the main result. The liver values of cobalt were closely related with vitamin B<sub>12</sub> levels. Low cobalt values, however, have been reported to increase the zinc values (Vellema, 1996). The zinc values in the reference hinds were significantly higher than in the malnutrition hinds. Feed with enriched copper can also influence the digestion and metabolization of zinc. The malnourished hinds were injected with 400µg cyanocobolamin in July and August when the blood samples were taken. The herd, including the affected hinds, had been given supplementary feed as a part of standard procedure both before and after the treatment. The affected animals recovered totally, and no clinical signs of unthriftiness could be observed after two months.

A range of other conditions including unthriftiness and weight loss can be caused by parasitism and infectious diseases. Neither paratuberculosis nor chronic wasting disease was diagnosed, but a heavy infection with lungworm (*Dictiocaulus* spp.) was found. The herd, however, was

given Ivomec® pour-on vet as a prophylactic treatment against parasites. Animals in poor condition are normally more susceptible to parasites and the affected hinds had periodic diarrhoea. A lower level of albumin was found in these animals compared to the reference hinds. Hypoalbuminemia occurs in animals affected by intestinal parasitism (selective loss), and as a result of decreased synthesis of albumin in animals suffering from malnutrition (Kaneko, 1997). The liver is the only site of albumin synthesis and hypoalbuminemia is an important feature of chronic liver disease (Kaneko, 1997). The gamma, beta and alpha 2 globulin fractions were significantly higher in the malnourished hinds compared to the reference hinds (Table 1). Gamma globulines increase during infectious disease, connective tissue disease and liver disease. A rise in alpha 2 globulins is commonly seen during acute inflammatory disease. In acute nephritis and in the nephrotic syndrome it is typical to find increased alpha 2 globulin level and a decrease in the albumin blood concentrations. Increases in beta-globulines are seen in active liver disease and in the nephrotic syndrome (Kaneko, 1997). The low values of albumin and the high values of alpha 2 globuline and beta globulin cannot exclude kidney and liver diseases in the malnourished hinds. However, the animals recovered completely after treatment with minerals, vitamins and anthelmintic.

The characteristic expressions of selenium/vitamin E deficiency in cattle and sheep are nutritional myopathies. Unlike the more acute white muscle disease, the selenium responsive unthriftiness which occurs in lambs and weaners has different aetiologies and a condition which is often unrecognised. (Paynter *et al.*, 1979). Selenium is an important trace mineral in ruminant nutrition, being both an essential dietary element and potential toxic. The nutrition benefits of selenium and vitamin E are generally considered together, because several diseases of farm animals are caused by or associated with deficiency of either or both. These diseases, however, are often connected with

important predisposing factors such as unaccustomed exercise and rapid growth in young animals (Haigh & Hudson, 1993) Furthermore interactions of selenium and other trace minerals such as cobalt and zinc occur. Excessive amounts of any of these can lead to selenium deficiency. There was, however, a significant difference in the zinc blood concentration between the groups, with the highest value among the reference hinds, but the selenium blood values were slightly increased compared to the reference animals. Knox *et al.* (1987) reported an outbreak of selenium responsive unthriftiness in farmed red deer in Sussex. The animals were injected with barium selenate and responded clinically with weight gain, improved coat quality, increased appetite for the ensilage and greatly improved exercise tolerance. Mackintosh *et al.* (1989) failed to demonstrate what level of selenium concentrations in the pasture are necessary to cause selenium deficiency in young deer and suggested that a pasture concentration of <30ppb could result in selenium blood concentrations <250nmol/L in some individuals. The malnourished hinds in this study, however, had higher selenium blood values than the reference hinds ( $p=0.046$ ), and had higher blood values than the limit for deficiency. Selenium contents of the animal autopsied had a level of  $0.07\mu\text{g Se/g}$  (wet weight) and it was concluded that the value was below the limit for deficiency. Deficit of selenium and vitamin E can cause myopathy and significant losses of production in sheep (McDonald, 1975). When sheep are turned out to graze in spring they can develop acute myopathy, accompanied by large increases in the plasma activities of creatine kinase, that is an indirect functional marker of damage to muscle membranes, which can be prevented by the administration of selenium. There was no significant difference in the creatinine kinase value between the malnourished and reference group. It can therefore be concluded that there is no acute myopathy. Elevated lactate dehydrogenase, however, has been reported in selenium/vitamin E deficiency in cattle (Allen *et al.*, 1975),

sheep (Whanger *et al.*, 1970) and swine (Ruth & Van Vleet 1974). In the present study we found significantly higher blood values of lactate dehydrogenase in the malnourished hinds compared to the reference hinds. The hinds responded to the therapy including a mixture of selenium and vitamin E. Therefore, we cannot conclude that the selenium/vitamin E complex is not involved in the malnutrition syndrome.

A significantly higher level of glutamate dehydrogenase was found in the malnutrition hinds compared to the reference hinds. Glutamate dehydrogenase can be used to assess hepatic necrosis in sheep, goats and cattle and has been reported to be elevated in ruminants with hepatic necrosis and bile duct obstruction (Tennant, 1997). No good explanation can be given for the elevated blood values in this study.

Serum urea and creatinine concentration are used as indicators of retention of nitrogenous wastes by the kidneys. This reflects the intake of effective rumen degradable protein and its balance with fermentable metabolic energy. Increased levels of serum urea may be associated with high protein food catabolism and with the level of rumen degradable protein in the diet. The values in the malnourished hinds were significantly higher than in the reference hinds. Processes including protein catabolism can result in an increased blood urea concentration. Starvation is one of these cases described in man (Kumar *et al.*, 1972). An explanation of the higher levels can be that the fat reserves are used forcing the animals to metabolise muscle protein (Finco, 1997). The serum creatinine was significantly lower in the malnourished hinds than the reference hinds. Decrease in serum creatinine associated with loss of muscle mass has been seen in humans (Newman, 1971).

Other specific diseases that can cause unthriftiness and chronic weight loss is Johne's disease and chronic wasting disease. Necropsy and laboratory tests have been used to exclude these. Chronic wasting dis-

ease has so far not been seen in Europe, and the test was negative. Johne`s disease could also be excluded because of no typical finding by the autopsy, and *Mycobacterium paratuberculosis* was not isolated. The animals recovered after the treatment, a situation that confirms that these diseases were not involved.

The serum concentration of glutamate dehydrogenase, lactate dehydrogenase, globulin, alpha 2, beta, and gamma globulins, urea, albumin, creatinine and zinc can be used to diagnose malnutrition in red deer hinds. Treatment of affected hinds with a mixture of Selenevit vet®, Becoplex ® and an Ivomec® pour-on vet appears to solve the problem.

Table 1

Comparison of serum chemical values in free-ranging reference hinds ( $n=10$ ) and hinds suffering of malnutrition ( $n=4$ ).  $P$ -values  $< 0.01$  were considered significant.

Parameter	Constituent unit	Mean values reference hinds	Standard deviation	Median value reference hinds	Malnutrition hinds mean values	Standard deviation	Malnutrition hinds median value	$P$ -values
Aspartate aminotransferase	U/L	53.40	8.30	52.50	98.30	34.30	94.50	
Alanine amino transferase	U/L	54.30	8.92	54.00	57.00	18.51	50.00	
Alkaline phosphatase	U/L	178.20	91.20	158.00	337.00	186.80	353.50	
Gamma glutamyl transferase	U/L	18.60	6.92	16.00	38.25	16.56	30.50	
Glutamate dehydrogenase	U/L	1.44	0.50	1.20	11.0	5.53	10.50	0.004
Creatine kinase	U/L	166.50	70.10	150.50	238.50	65.60	257.50	
Lactate dehydrogenase	U/L	601.90	115.70	568.50	1183.00	378.00	1135.00	0.009
Total protein	g/L	66.10	7.52	67.00	69.25	2.63	68.50	
Albumin	g/L	38.60	5.93	39.50	24.25	1.50	24.00	0.007
Globulin	g/L	27.50	2.27	28.00	45.00	3.56	44.00	0.006
Globulin alpha 1	g/L	4.07	0.60	3.95	5.01	1.22	4.75	
Globulin alpha 2	g/L	5.71	0.72	5.65	8.78	0.59	8.80	0.006
Globulin beta	g/L	5.25	0.71	5.05	8.18	0.41	8.10	0.006
Globulin gamma	g/L	12.55	1.28	12.35	23.13	2.45	22.65	0.006
Urea	mmol/L	6.14	1.58	5.95	11.98	1.10	12.20	0.006
Creatinine	$\mu$ mol/L	178.80	25.13	185.00	94.75	10.69	92.00	0.006
Total bilirubin	$\mu$ mol/L	2.00	1.16	2.00	3.50	1.30	3.50	
Cholesterol	mmol/L	1.11	0.30	1.15	0.67	0.22	0.60	
Triglycerides	mmol/L	0.10	0.00	0.10	0.15	0.17	0.10	NP
Free lipid acids	mmol/L	0.11	0.06	0.10	0.10	0.8	0.10	
$\beta$ -hydroxybutyrate (B-HBA)	mmol/L	0.25	0.10	0.20	0.30	0.08	0.30	
Glucose	mmol/L	10.04	3.38	10.75	5.78	0.99	5.75	
Na	mmol/L	132.92	7.35	135.00	142.34	7.97	140.87	
K	mmol/L	5.10	0.49	5.12	4.74	0.14	4.72	
Mg	mmol/L	0.45	0.06	0.49	0.59	0.20	0.53	
Cu	$\mu$ mol/L	11.81	1.48	11.57	24.40	4.92	25.52	0.006
Zn	$\mu$ mol/L	7.78	1.07	7.69	5.39	0.39	5.28	0.007
Ca	mmol/L	1.89	0.24	1.85	2.08	0.36	2.06	
P	mmol/L	1.05	0.12	1.07	1.38	0.27	1.41	
Co	$\mu$ mol/L	<0.17*			<0.17*			NP
Se	nmol/L	890	186.3	899	1108	201	1102	
Al	mmol/L	0.77	0.92	0.37	1.10	1.07	0.78	
Fe	mmol/L	0.04	0.01	0.04	0.03	0.01	0.03	

NP= not possible to calculate \*below the detection limit

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*Abstract in Norwegian/Sammendrag:*

Et avmagringssyndrom har blitt observert blant hjortehinder (*Cervus elaphus atlanticus*) i oppdrett. Dyra kommer seg ikke etter energiunderskuddet fra vinteren og tar seg ikke opp igjen gjennom vår/sommer selv om tilgangen og kvaliteten på fôret er tilfredsstillende. En del av dem dør på våren eller utpå sommeren. Obduksjon av et dyr som hadde syndromet viste fullstendig avmagring (kakeksi). Dyret var infisert med lungeorm og hadde unormalt lave verdier av selen og kobolt i leveren. Blodverdier fra fire hinder som led av syndromet er sammenlignet med ti villlevende referansehinder. Hos hindene som led av avmagringssyndromet ble det funnet signifikant høyere serumverdier ( $p < 0,01$ ) av glutamat dehydrogenase, laktat dehydrogenase, globulin, alfa 2 globulin, beta globulin, gamma globulin, urea og kopper. Oppdrettsdyra hadde fått koppertilskudd. Det ble funnet signifikant lavere serumverdier av albumin, kreatinin og sink hos hindene som led av avmagringssyndromet sammenlignet med referansedyra. Disse blodparametrene kan bli brukt for å diagnostisere avmagringssyndromet hos oppdrettshjort når prøvene blir tatt av immobiliserte dyr. Etter behandling med 20 ml Becoplex vet® i.m., 10 ml Selenevit vet® i.m. og 10 ml Ivomec® pour-on vet restituerer de affiserte hindene i løpet av to måneder.