THE PREVENTION AND CURE OF WHITE MUSCLE DISEASE IN' LAMBS BY MEANS OF SELENIUM PELLETS

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Summary

Typical white muscle disease was produced within nine weeks in normal lambs fed pelleted, selenium-deficient meadow hay. Treatment with CSIRO 5% selenium pellets and grinders prevented the condition and provided adequate selenium for normal growth and health. All pellets were recovered at autopsy. Affected lambs given selenium pellets eventually recovered; in these, macroscopic lesions were barely discernible post-mortem and histologically, there was evidence of major repair in both cardiac and skeletal muscle.

I. INTRODUCTION

Since the initial demonstration that heavy pellets containing elemental selenium could provide physiological amounts of that essential element to sheep (Kuchel and Buckley 1969) further trials (unpublished) have confirmed that treatment with a single 5% Se pellet and a steel grinder will provide ewes with adequate selenium for several years and also improve the selenium status of their untreated lambs. Experiments to determine whether similar pellets given to lambs consuming selenium-deficient fodder would prevent the development of white muscle disease (W.M.D.) or cure the disease in advanced cases, are described below.

II. MATERIALS AND METHODS

In two separate experiments selenium deficiency was induced in normal ewe and wether lambs, newly weaned at seven to nine weeks of age, as described by Godwin, Kuchel and Fuss (1974). The deficient diet of pelleted meadow hay from Kangaroo Island, S.A., contained 0.019 p.p.m. Se D.M. in Expt I and 0.016 p.p.m. Se D.M. in Expt II.

In Expt I, 14 lambs were distributed as follows:-

1. no selenium (four lambs);
2. selenium drench: 3 mg Se twice weekly as a solution of sodium selenite (four lambs);
3. one selenium Pellet and one steel grinder at the outset (six lambs).

In Expt II, 17 lambs were treated'as follows:-

1. no selenium (ten lambs);
2. one selenium pellet and one steel grinder at the outset (four lambs);
3. treatment 2 was given to three grossly deficient lambs originally included in group 1. All had developed oedema of the upper hind limbs and forelegs; none could stand unaided and one of them (No. 516), not

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even with assistance.

The selenium pellets were pressed in the laboratory; they comprised 0.5 g Se powder (May and Baker) and 9.5 g Fe powder (B.D.H.)', S.G. = 5.9. The steel grinders of threaded mild steel, weighed 10 g, S.G. > 6. Liveweights and food intakes were recorded each week. Selenium was determined by the method of Watkinson (1966) and creatine phosphokinase (C.P.K.) in plasma by the method of Neilsen and Ludvigsen (1963) in Expt I or the Boehringer Mannheim kit (Cat. No. 15790) in Expt II. The lambs were killed for examination as changes in liveweight, appetite, blood selenium and plasma C.P.K. levels dictated.

III. RESULTS

In both experiments all lambs given selenium remained alert and active throughout whereas the untreated lambs became increasingly listless and in Expt II one died with advanced signs of W.M.D. in the ninth week.

(a) Food intake

In Expt I the depression of appetite in the untreated lambs was not significant but in Expt II the progressively declining food intake in the untreated group was significantly less than in the treated group ($P < 0.05$) from the seventh week (Figure 1).

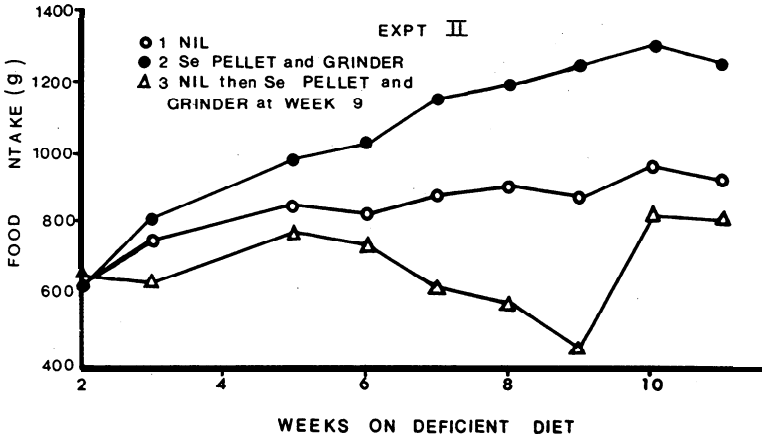

Fig. 1.-Group mean food intake (g/d).

The improvement in health and appetite of the lambs (group 3) given pellets was immediately obvious; two of them could rise unaided after two d but the worst of them, No. 516, only after 11 weeks.

(b) Liveweight

The liveweight response to selenium for the period tested, from week five to week eight of Expt I (Figure 2) was highly significant ($P < 0.001$); there was no difference between the selenium treatments. In Expt II, the difference due to selenium became significant ($P < 0.05$) after six weeks. The increase in wt of the lambs in group 3, Expt II, corresponds with their improved food intake after treatment.

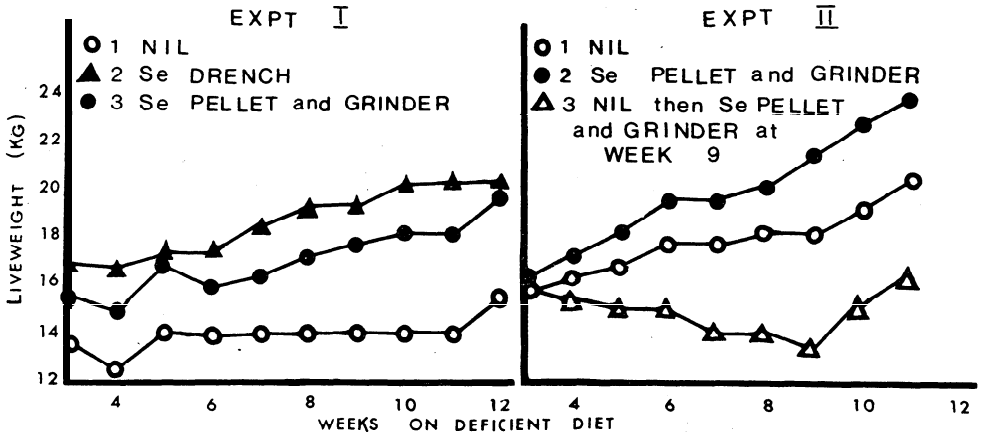

Fig. 2.-Group mean liveweights of lambs.

(c) Selenium in blood

In each experiment the mean concentration of selenium in the blood of the untreated lambs declined steadily and the immediate response to treatment was significant ($P < 0.01$) at the first sampling after pellet treatment (Figure 3).

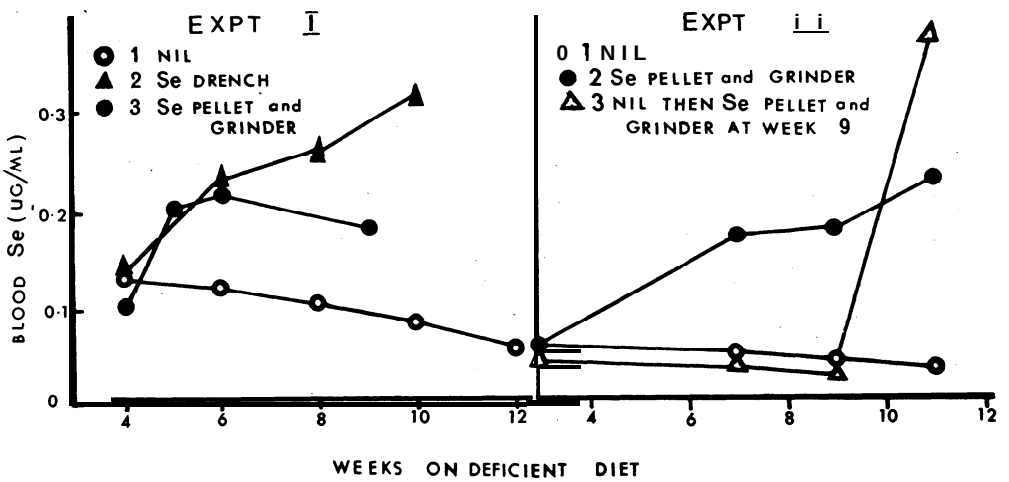

Fig. 3.-Group mean blood Se concentrations (µg/ml).

(d) C.P.K. in plasma

In Expt I, group mean differences were not significant whereas in Expt II the selenium effect was significant ($P < 0.01$) by the fourth week (group 1, 53 units C.P.K./100 ml; group 2, 5 units/l00 ml) and the differences increased thereafter. Extremely high values were recorded in lambs with advanced W.M.D.; the mean value for those in group 3, Expt II, reached 464 units/100 ml but had fallen to 49 units/ 100 ml five d after pellet treatment.

(e) Post-mortem findings

Bisymmetrical lesions of W.M.D. in the semitendinosus muscles and white plaques in the endocardium were found to a greater or lesser extent in all untreated lambs; widespread cell destruction and neutrophilic

invasion were apparent microscopically and the oedematous condition of the worst affected animals was associated with the release of serous exudate and a high moisture content (> 85%) of the skeletal muscles. By contrast, lambs treated continuously with selenium by drench or pellet showed none of these abnormalities. Residual macroscopic lesions were barely discernible in group 3, Expt II, in lambs killed 32, 46 or 88 d after pellets were given; histologically there remained only isolated abnormal muscle bundles with aggregated cell nuclei and invading neutrophils.

IV. DISCUSSION

Andrews, Grant and Brunswick (1974) reported that selenium pellets (early commercial prototypes made according to CSIRO specifications) increased the selenium status and wt gains of Romney lambs grazing selenium-deficient pasture in New Zealand but that, within a yr, an appreciable number of pellets had been lost by regurgitation and others had become coated with calcium phosphate.

In the present experiments with lambs fed a severely dystrophogenic, pelleted diet, selenium pellets maintained appetite, growth and adequate blood selenium concentrations during observations lasting about four months while fully protecting the lambs against W.M.D. All pellets were recovered at autopsy and none was coated. It is apparent from these and related observations, that Merino sheep retain pellets more effectively than do other breeds and that the administration of a steel grinder is always advisable to reduce the risk of the deposition of calcium phosphate on the surface of a remedial pellet while in the reticulum.

All of the untreated lambs developed gross signs of W.M.D. but three of them recovered completely when given selenium pellets. Mild cases of the disease may sometimes recover spontaneously (Underwood 1971) but these severely affected animals would undoubtedly have died had treatment been withheld.

V. REFERENCES

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