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To cite this version:

HAL Id: hal-00901418
https://hal.archives-ouvertes.fr/hal-00901418
Submitted on 1 Jan 1983

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A ZINC-DEFICIENT DIET FOR RUMINANTS:
DIAGNOSIS AND TREATMENT OF DEFICIENCY

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Résumé

RÉGIME CARENCÉ EN ZINC POUR RUMINANTS: DIAGNOSTIC ET TRAITEMENT DE LA CARENCE. — Un régime carencé en zinc et non semi-synthétique a été essayé sur 20 moutons mâles Limousin × Romanoff de trois mois et demi et pesant 36 kg en moyenne. Le régime, à base de foin pailleux, a été arrosé d'un caramel comportant de l'urée et des minéraux. Ce régime est plus appétent que le foin seul. Il est plus équilibré au point de vue énergétique et permet une meilleure utilisation de l'azote soluble. La carence a été visible en une semaine par le zinc plasmatique et a évolué cliniquement progressivement en 100 jours. Le zinc libre plasmatique et la phosphatase alcaline n'ont pas constitué de meilleurs critères que le zinc plasmatique total pour apprécier la carence. La supplémentation du régime à 50 ou 100 mg Zn/kg MS n'a pas supprimé la carence. En revanche l'injection de zinc a restauré la zincémie en dix jours. Le traitement per os de la carence en zinc est donc à revoir.

For practical purposes, most diets used for experimental zinc deficiencies are semi-synthetic (Miller and Miller, 1960; Mills et al., 1967, review of Schwarz and Kirchgessner, 1974). Ruminants can use soluble nitrogen, for instance purified urea, if the diet is rich enough in fermentable carbohydrates. They also require fiber in the diet, and preferably, long fiber. Industrial cellulose or cotton are unsatisfactory for proper ruminating activity. A semi-synthetic (powdered) diet should be pelleted in order to be ingested by the animals. But, making the pellets is in itself a cause of contamination. With unchopped hay, the preceding constraints can be satisfied. Late-harvested hay, cut after flowering, in other respects, is poor in nitrogen and trace-elements (Perigaud, 1971). It has low digestibility, is straw-like, and thus the zinc digestibility of these straws is very low (Lamand et al., 1977).

A late-harvested hay sprayed with caramel and enriched with urea and minerals was used to balance the zinc-deficient feed given to lambs. The development of the deficiency and its treatment were observed.

Materials and Methods

Twenty three and a half month-old male Limousin × Romanoff crossbred with an average weight of 36 kg received the deficient diet. The latter was based on timothy grass that had been harvested one month after flowering. The caramel composition was as follows: 250 g sugar (from a sugar refinery) dissolved in 60 ml distilled water and 4 ml 10 % acetic acid. This mixture was boiled until a yellow caramel was obtained. At this point, during boiling, the caramel was diluted by adding
125 ml distilled water that contained 12 g purified urea, 24 mg CuSO₄·5H₂O; 0.4 mg CoCl₂·6H₂O and 1 g NaCl. 250 ml of this mixture (with a density of 1.29; 63 % DM basis) were added per kg hay, i.e. 200 g DM/kg hay.

Plasma zinc was determined by atomic absorption spectrometry (Bellanger and Lamand, 1975) as were feed components (Bellanger, 1971). The caramel components had no zinc (signal did not differ from basic line). The hay was composed of 4 % total nitrogen (N x 6.25) and a content of 13 mg Zn/kg DM. In other words, the overall diet contained 10.8 mg Zn/kg DM, 8 mg Cu/kg DM, 0.12 mg Co/kg DM and about 48 g digestible protein/kg.

After a 43-day deficiency, the caramel was eliminated from the feed of 10 out of the 20 animals, and this group received hay alone. The hay was then sprayed with an aqueous solution (50 ml/kg) of 10 g urea, 24 mg CuSO₄·5H₂O; 0.4 mg CoCl₂·6H₂O and 1 g NaCl/kg hay. The dietary zinc was 13 mg/kg DM.

On the 69th day, all the animals (both caramel group and hay only group) were given a zinc-enriched (50 mg Zn/kg DM) diet. This same diet was increased to 100 mg Zn/kg DM on the 98th day. On the 114th day, all animals were given an intramuscular injection of 300 mg metal zinc in oil suspension (Prolontex zinc® Lamand, 1978).

Plasmonic alkaline phosphatase was measured using the Bessey et al. method (1946) (with Boehringer reagents). Free plasma zinc was determined. It was defined as zinc likely to reactivate the alkaline phosphatase of Escherichia coli that had been deprived of its zinc by chelation beforehand, using 5 mM/l nitriiloacetic acid (NTA); a concentration of 0.4 mM/l was used, which inhibited phosphatase by 50 % with a sheep plasma without zinc deficiency (zincemia: 15.4 μM/l).

Results

The decreased zincemia and alkaline phosphatase was significant the 7th day (P < 0.01) (fig.1 and 2). Free zinc did not decrease significantly until the 42nd day of the deficiency (fig.3).

Animal growth stopped, and very progressive weight losses were noted (fig.4). Zincemia in lambs that were fed hay alone was significantly higher in than lambs of the other group very soon after the change in diet (56th and 63rd days). This difference later disappeared. Supplementing diets with 50 mg Zn/kg DM on the 69th day relieved the zincemia only slightly and did not modify either the alkaline phosphatase or the free zinc. Similarly,

![Fig. 1](image-url) — Plasmonic total zinc evolution. Zn 50 or Zn 100: diet enriched to 50 or 100 mg Zn/kg DM. Injection: metal zinc in powder, in oil suspension intramuscularly injected.
Fig. 2. — Alkaline phosphatases evolution.

Fig. 3. — Plasma free zinc evolution.
increasing the dietary content to 100 mg Zn/kg DM did not increase zincemia or the other parameters measured. The hay sprayed with caramel seemed more appetizing to the lambs as opposed to the plain hay.

Clinically, the animals stature changed progressively, and they became thin and apathetic. Feed intake became much slower. Animals selected a lot, and consumed very little at a time. By the 100th day, crusty scabs appeared on the caramel batch lamb noses but not on the others. Zinc injection (300 mg intramuscular) brought on a rise in zincemia that returned to normal within ten days.

Discussion

Giving long hay sprayed with caramel is practical. Animals appeared to like this diet. This formula is very flexible for nitrogen and mineral supplementation. Caramel viscosity is such that additives which stay suspended can be included, stuck onto the hay without falling to the bottom of the trough. Refined sugar is inexpensive and practically exempt from trace-elements.

It might be possible to replace deficient hay with straw. Zincemia remains the most faithful criterion of the evolution of a zinc deficiency. As previously demonstrated (Lamand, 1982) alkaline phosphatase is not the plasmic parameter with a sensitivity that allows a subdeficiency to be detected as some authors have found in other species (Roth and Kirchgessner, 1979; Kasarskis and Schuna, 1980). These works attempted to show a drastic increase in alkaline phosphatase activity following zinc treatment. In the present case, the rise in the feed content of Zn did not modify alkaline phosphatase. This parameter, as opposed to zincemia, did not evolve in the six days following zinc injection. The same was true for free zinc. According to Donangelo and Chang (1981), this fraction of plasmic zinc available should be a good indication of the zinc status. The evolution of this parameter is not parallel to that of zincemia, and although the determination of this fraction does not require atomic absorption spectrometry, the method remains relatively long and tricky.

The attempt at treating the deficiency by supplying zinc to the diet failed: with straw-like hay whether supplemented or not with caramel, 50 mg Zn/kg DM or even 100 mg Zn/kg DM were insufficient to suppress the deficiency. In the present experiment, 100 mg Zn/kg DM proved to be below the animal requirements and the principle of treating a deficiency per os should be reconsidered. Zinc injection, by contrast, was efficient in restoring zincemia to normal.

Accepted for publication, 15th March 1983.

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Summary

A zinc deficient diet which is not semi-synthetic was tried on 20 male Limousin x Romanoff three and a half month-old sheep weighing 36 kg on average. This diet was composed of strawy hay sprayed with caramel containing urea and minerals. Compared to hay alone this diet is more appetizing to lambs and more equilibrated as to energy level and soluble nitrogen utilization. The deficiency appeared within a week in zinc plasma and progressed clinically within 100 days.

The plasmic free zinc and alkaline phosphatase were not better criteria for the deficiency diagnosis than total plasmic zinc. Supplementing the diet to 50 or even 100 mg Zn/kg DM was insufficient to suppress the deficiency. Zinc injection, by contrast, restored zincemia to normal in ten days. Zinc deficiency treatment by oral route should be reconsidered.

References