COBALT DELAYS THE DEVELOPMENT OF CLINICAL ANNUAL RYEGRASS TOXICITY IN SHEEP

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Annual ryegrass toxicity (ARGT) is a neurological disease of livestock caused by corynetoxins found in annual ryegrass infected by Clavibacter toxicus (Riley and Ophel 1992). We have previously shown that cobalt supplements offer partial protection against apparent liver damage resulting from doses of corynetoxins sufficient to cause sub-clinical ARGT in sheep (Davies et al. 1993). The current experiment investigated the response of cobalt supplemented sheep to doses that would cause the clinical disease.

Forty five Merino wethers maintained at 3.5 kg liveweight on a basal diet of oaten hay with 12% lupins were allocated to 9 treatment groups each of 5 sheep in a 3 x 3 factorial design. Toxic ryegrass seed was incorporated in the diet to provide either 0, 0.15 or 0.30 mg corynetoxins/kg liveweight.day. Sheep were drenched daily with an aqueous solution of CoSO₄·7H₂O to provide either 0, 4 or 16 mg cobalt/day. Toxin treatment was stopped at the first appearance of clinical signs (staggering gait, convulsions).

Sheep receiving cobalt supplements were able to ingest 30% more toxin before showing the clinical signs of the disease than those animals not supplemented with cobalt (P = 0.026). Increasing the level of cobalt from 4 to 16 mg/day did not confer any greater protection (P = 0.083). The cumulative dose of toxin required to induce clinical signs was slightly, but not significantly, greater for those sheep receiving toxin at the lower rate (P = 0.434).

![Figure 1. The total amount of toxin ingested before sheep showed clinical signs of annual ryegrass toxicity for (a) plus or minus cobalt supplementation with the low and high toxin treatments grouped and (b) nil cobalt (dark shading), 3 mg cobalt/day (diagonal hatch) and 16 mg cobalt/day (light shading) at each level of toxin](image)

The ability of the cobalt supplemented sheep to ingest 30% more toxin than the sheep not supplemented with cobalt, alongside the previous results of reduced liver damage due to cobalt supplementation (Davies et al., 1993), provides clear evidence that oral cobalt supplementation provides sheep with some protection against ARGT. However, this protection is not complete as all of the animals exposed to the toxin eventually developed the clinical signs of the disease. Cobalt supplementation may play a role in the alleviation of some of the sub-clinical losses thought to be associated with ARGT.