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VITAMIN K ABSORPTION IN THE HORSE; DOES ABSORPTION OCCUR FROM THE HINDGUT?

H.L. Regtop[A] A.M. Talbot [A] J.R. Biffin[A], J.E. Skinner[B], A.J. Cawdell-Smith[B] and W.L. Bryden[B]
[A]Agricure Pty Ltd, Braemar NSW, 2575
[B]The University of Queensland Equine Research Unit, School of Agriculture and Food Sciences, Gatton, 4343

Vitamin K consists of a group of structurally related compounds; phylloquinone (K1), that is synthesized by plants, and the menaquinones (MKs also known as K2) synthesized by bacteria. Menadione is the synthetic form of the vitamin and is routinely added to animal diets. It is designated K3 and does not have a side-chain. There is increasing evidence that vitamin K through its role as a cofactor for carboxylation of glutamate to gamma-carboxy-glutamic acid (GLA) residues in proteins in different tissues plays a significant role in bone metabolism, energy metabolism, spermatogenesis, apotosis and innate immunity, in addition to its recognized role in blood coagulation. The lack of a side chain restricts the activity of K3 to a role in blood clotting. The various forms of vitamin K are different not only with regard to their co-factor activity but also their absorption, transport, tissue distribution and turnover. Moreover, there are differences in vitamin K metabolism between species. In nature the general consensus appears to be that animals, including humans, meet their vitamin K requirements from plant and bacterial sources. Bacterial synthesis and subsequent absorption of K2 is considered by many authors to be an important source of vitamin K and yet there are no published reports of the extent and efficiency of these processes. The aim of this study was to determine the efficacy of vitamin K uptake from the hindgut of the horse. In this *in vivo* study vitamin K1 was coated with calcium alginate to prevent absorption in the small intestine thus allowing it to pass into the hindgut.

Four mature geldings were used in the study and each acted as its own control. Horses were dosed with a 200mg oral bolus (suspended in water) of either (1) KQ [QAQ, Quinaquanone™ a soluble form of K1 and K2 (10:1); (2) KQ coated with 1.5% Calcium alginate ; or (3) K1 oil .Blood sampling was undertaken at designated intervals for 12 hours and plasma samples were then analysed by fluorescent HPLC for K1, MK-4 and K3 concentrations.

In vitro studies with enzymes (Amylase, Protease, Lipase and cellulase) showed minimal release of K1 and breakdown of the alginate capsule over a period of 10hrs. In contrast, when incubated with a concentrated microbial fraction of horse faeces, breakdown of the alginate capsule was complete within 30minutes.

Plasma K1 concentrations differed significantly between treatments ($p<0.05$). Plasma K1 concentrations were 3-fold higher in the KQ treatment compared to K1 oil and K1 oil was higher that the KQ spheres; peak plasma values occurred for all three treatments at 4hours The results of this study questions the absorption of vitamin K from the hindgut of the horse .It shows that the intestines are partly responsible for the breakdown of the capsule with KQ spheres reaching a Cmax of 1.5ng/ml as opposed to 3.75ng/ml for KQ at 4hrs. There was no further uptake of K1 from the spheres in the hind gut. Studies in rats and humans have also demonstrated extremely poor absorption of vitamin K from the hindgut suggesting that bacterially synthesised vitamin K does not contribute substantially to vitamin K status.

There are two possibilities to explain the failure to observe an increase in circulating vitamin K after the coated vitamin reached the hindgut; 1) the bacteria in the hind gut metabolised K1 for their own use, or 2) the hind gut does not facilitate absorption of vitamin K1 as measured by plasma concentrations. . Moreover, 24% more Vitamin K1 was found in faeces of the Alginate KQ fed horses than those fed the unacapsulated KQ, again suggestive of poor uptake of vitamin K1 in the hindgut of a horse.